Social Evolution in Ants

Andrew F. G. Bourke and
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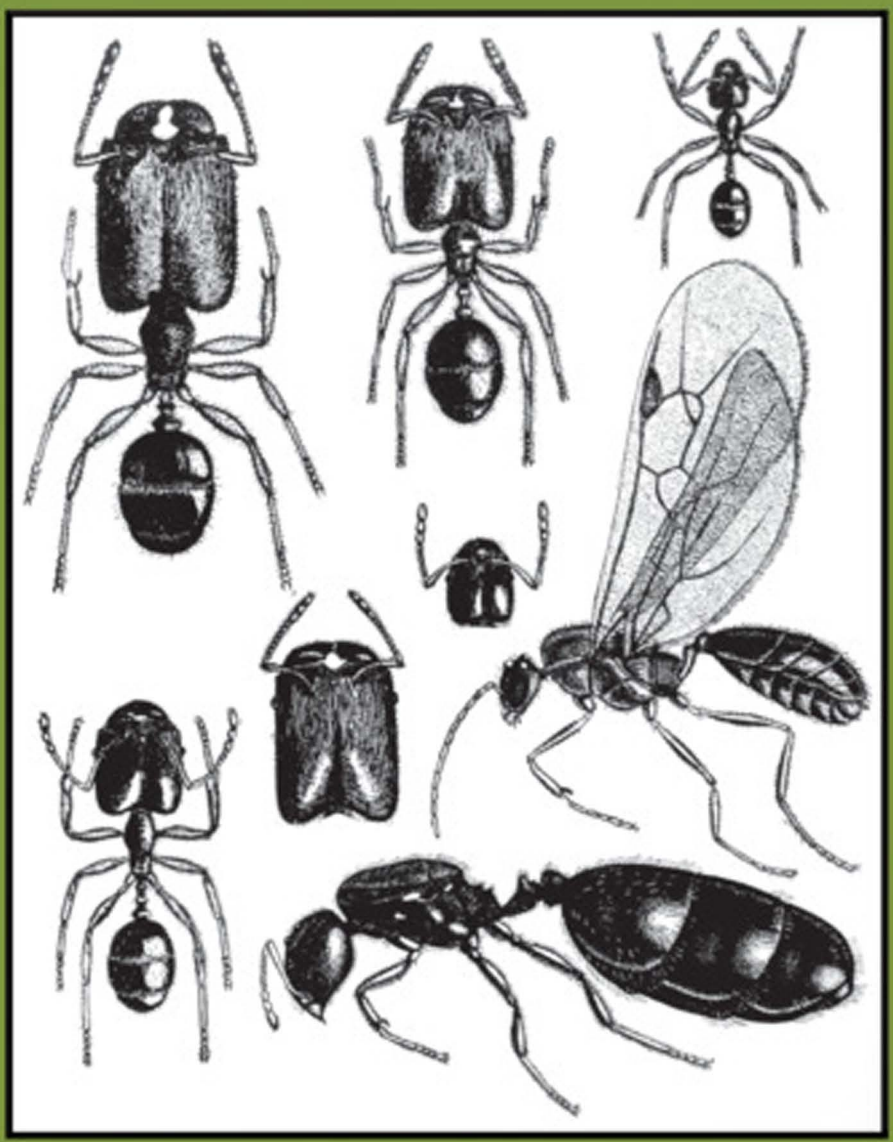

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Princeton University Press

Princeton, New Jersey

Published by Princeton University Press,
41 William Street,
Princeton, New Jersey 08540
In the United Kingdom: Princeton University Press,
Chichester, West Sussex

Library of Congress Cataloging-in-Publication Data

Bourke, Andrew F.G., 1961–
Social evolution in ants/Andrew F.G. Bourke, Nigel R. Franks.
p. cm. — (Monographs in behavior and ecology)
Includes bibliographical references (p.) and indexes.
ISBN 0-691-04427-9 (cl). — ISBN 0-691-04426-0 (pbk.)
1. Ants-Behavior. 2. Social evolution in animals. 3. Kin selection (Evolution) 4. Sex ratio. I. Franks, Nigel R. II. Title. III. Series.
QL568.F7B63 1995
595.79'6045248–dc20 95–5959

This book has been composed in 10/12 Times by Wearset, Boldon, Tyne and Wear

Princeton University Press books are printed on acid-free paper and meet the guidelines for permanence and durability of the Committee on Production Guidelines for Book Longevity of the Council on Library Resources

Printed in the United States of America by Princeton Academic Press

10 9 8 7 6 5 4 3 2 1

10 9 8 7 6 5 4 3 2 1
(Pbk.)

Contents

Preface and Acknowledgments

Ants have always provoked the amazement of people at large, and the intellectual curiosity of biologists in particular. To Darwin, their sterile castes posed "one special difficulty, which at first appeared to me insuperable, and actually fatal to my whole theory" (Darwin 1859 p. 236). In fact, Darwin found several difficulties in the sterile workers of ants (Cronin 1991 p. 298). The most pressing for modern biologists is the question of how sterility and self-sacrifice can evolve under natural selection. In 1964, W.D. Hamilton's theory of kin selection provided the radical and elegant solution to this problem. Hamilton's work revolutionized our understanding of natural selection and adaptation, particularly in the field of animal behavior (G.C. Williams 1966; Dawkins 1976). In addition, after being largely inspired by social insects, Hamilton's theory repaid the debt by sparking off an explosion in research on kin selection in ants and their relatives. In this, it was aided by the major synthetic works of E.O. Wilson (1971, 1975). Another fillip was provided by the paper of Trivers and Hare (1976), which linked kin selection with sex ratio evolution and the occurrence of conflicts of interest within insect societies. With these studies, social evolution in ants became a topic of concern to evolutionary biologists and behavioral ecologists of all kinds.

An additional facet of social evolution to which ant studies are central is the organization of work within societies. Prompted largely by the pioneering researches of Oster and Wilson (1978), the study of the division of labor in ants has also outgrown its original boundaries to become a topic of interest to many types of biologists. Its modern aspect includes, among other things, the study of self-organization in complex systems.

The inquiries initiated by Darwin, Hamilton, Wilson, Trivers, and others have created a rich and sophisticated body of theory and data on ant social evolution. In this book, we describe its present state. There are many recent books about ants. A sample includes those by Dumpert (1981), Passera (1984), Sudd and Franks (1987), and the magnificent treatise of Hölldobler and Wilson (1990). This book offers something that builds on these works, but is nevertheless different in purpose. Aided by the full panoply of modern adaptationist logic, we explore in depth the fundamental topics in evolutionary biology and behavioral ecology to which ant studies continue to make an important contribution. In full, these include sex ratio evolution, kin conflict, the

ecology of social systems, and self-organization. We also discuss the evolution of eusociality, along with the logic of kin selection theory and associated concepts of selection. In addition, we review two topics that have been relatively neglected in ants, namely life history strategies and mating biology. Throughout, we have aimed to make comparisons with other types of social insects, and other organisms altogether, where this seemed appropriate. Inevitably, we have not found space to consider several further, equally important facets of ant sociality. Examples are foraging systems, the evolution of social parasitism, and mutualisms between ants and plants. For these, and for a comprehensive, beautifully illustrated survey of ant biology in general, we refer readers to Hölldobler and Wilson (1990). In this book, we invite all those interested in evolutionary biology and behavioral ecology, including those whose favorite organisms are (for now) birds and mammals, to share in the knowledge and understanding that comes from the study of social evolution in ants.

We thank the many people who helped us produce this book. Laurent Keller and Peter Nonacs each read the whole work in draft, and made dozens of useful comments. The following people read and helped improve various chapters, passages, or groups of chapters: Tim Benton, Koos Boomsma, George Chan, Scott Creel, William Foster, Raghavendra Gadagkar, Charles Godfray, Jürgen Heinze, Ian Owens, Christian Peeters, Andrew Pomiankowski, Francis Ratnieks, and Mary Jane West-Eberhard. We thank them all; we always followed their advice, but from time to time we had second thoughts and changed the passage back to our version again. We particularly thank those colleagues who commented with good grace on passages opposed to their viewpoint. Many additional colleagues kindly sent unpublished or in press work. Simon Fraser and Ana Sendova-Franks prepared some of the figures.

A.F.G.B. thanks Jesus College, Cambridge, and the Zoology Department of the University of Cambridge, for support during the early stages of the writing. A.F.G.B. wrote the bulk of his contribution as a member of the Ecology Group of the Institute of Zoology, Zoological Society of London. Thanks go to Steve Albon and all the other members of the Group, especially Ian Owens, for their backing and encouragement. The following libraries are acknowledged for providing essential facilities: The Balfour Library, Department of Zoology, University of Cambridge; The Library of the Zoological Society of London, Regent's Park, London; and The Bloomsbury Science Library, University College London.

N.R.F. thanks the School of Biology and Biochemistry, University of Bath, and the Wissenshaftskolleg zu Berlin, Institute of Advanced

Study, Berlin, for their support. This book was partly written when N.R.F. was a Fellow at the Wissenschaftskolleg during the academic year 1993–1994. A debt of gratitude is owed to friends and colleagues at both Bath and Berlin for their encouragement and support. In particular, thanks go to Guy Blanchard, Don Braben, Scott Camazine, Jean-Louis Deneubourg, Melanie Hatcher, Mike Mogie, Glenda Orledge, Lucas Partridge, Alan Rayner, Stuart Reynolds, Tom Seeley, Ana Sendova-Franks, Lesley Smart, Tim Stickland, Chris Tofts, and Rüdiger Wehner.

Finally, we thank John Krebs and Tim Clutton-Brock for initiating this project, and for their professional help Emily Wilkinson and Kevin Downing of Princeton University Press, and Fisher Duncan, London. The order of authorship of this book was determined alphabetically.

Andrew Bourke, Nigel Franks
London and Bath, December 1994

Social Evolution in Ants

1 Kin Selection

1.1 Introduction

The basis of the modern evolutionary study of animal behavior is Darwin's theory of natural selection. But the more immediate foundations of the study of sociality and altruism in animals come from a development of Darwinian theory, W.D. Hamilton's theory of kin selection. Hamilton's theory is fundamental to the understanding of the social evolution of ants and their relatives. So the main purpose of this chapter is to explain the meaning and power of kin selection theory. Chapter 2 explores the relation between kin selection and other concepts of selection such as group selection and colony-level selection. Chapter 3 concentrates on specific models of social evolution in the Hymenoptera.

This book aims to interpret the social biology of ants from an adaptationist viewpoint, deploying the theory of natural selection as a tool of explanation. It therefore pursues what critics call the "adaptationist programme" (Gould and Lewontin 1979). However, embracing adaptationism need not mean that all evolutionary change must be regarded as adaptive, or all features of organisms as adaptations. Nor does it mean that natural selection, the mechanism of adaptive evolutionary change, is either omnipotent, or unconstrained, or a perfect optimizing agent. Lastly, it does not imply that other facets of evolutionary biology are irrelevant or uninteresting.

Instead, adaptationists hold that the evolution of adaptation by natural selection is a unique and pervasive feature of the living world. Therefore, it is a fruitful exercise, in terms of gaining understanding, to interpret biology in adaptive terms. This may not always be successful. On the other hand, the recent expansion of adaptationist thinking into animal behavior has been very successful. Many features of behavioral biology that previously seemed just natural history curiosities can be explained adaptively (for example, the kin conflicts within societies described in Chapter 7). After all, natural selection, though not omnipotent, is powerful; though not unconstrained, is not totally bound

by history; and though not a perfect optimizer, can achieve design good enough to give the illusion of creation. These are the features of natural selection that justify the adaptationist program. For full defences of adaptationism and associated concepts see, for example, Cain (1964), G.C. Williams (1966, 1985, 1992), Dawkins (1982a, 1986), Mayr (1983), Parker and Maynard Smith (1990), Cronin (1991), and Reeve and Sherman (1993).

This chapter also makes the case for a gene-centered approach to understanding natural selection. In other words, it advocates the gene selectionist or "selfish gene" perspective introduced to evolutionary biology by G.C. Williams (1966) and Dawkins (1976, 1982a, 1986). Gene selectionism is controversial (e.g. Wimsatt 1980; Wright 1980; Dawkins 1982a; Gould 1983a, 1992; Brandon and Burian 1984; Sober 1984a,b; Brandon 1985; Dover 1988; Ohta 1992). Nevertheless, a look at today's behavioral ecology textbooks suggests that it is already the dominant mode of evolutionary explanation. But the arguments for gene selectionism in general, and for its use in the study of social insects in particular, are still worth presenting.

First, since natural selection thinking underpins all of this book, what natural selection is taken to mean needs explaining. Second, regarding the main point of this chapter, kin selection theory is best understood as a logical consequence of gene selectionism. So a strong case for kin selection first has to set out its gene selectionist premises. Dawkins (1976, 1979, 1982a) has already clearly explained the gene selectionist logic of kin selection theory. Nevertheless, the necessity of kin selection for understanding social evolution has not been fully appreciated. Kin selection is still mistaken for an elaborate kind of selection on individuals, or suspected of being a mere scientific fashion. So another reason for a full explanation of gene selectionism is to justify kin selection theory from first principles, in the hope that this can dispel the lingering skepticism over the theory. In this we are reinforcing the message conveyed by earlier authors. In particular, a debt to the ideas and writings of Dawkins will be obvious and is acknowledged here.

An alternative to the gene selectionist understanding of natural selection focuses on the hierarchical organization of life and on the different levels at which selection is believed to act. The social insects, especially "advanced" ones like the ants, are often pressed into the service of this "levels-of-selection" perspective. This is because their colonies are held to represent prime examples of higher-level units subject to natural selection in their own right. As a result, the scientific literature on social insects is full of references to "colony-level selection" and "superorganisms." For this reason, the next chapter analyzes the issues surrounding levels-of-selection theory, colony-level selection, and the superorganism

concept. It concludes that, with qualifications, these ideas can be accommodated by the gene selectionist perspective. This is the final reason for initially concentrating on gene selectionism.

Therefore, this chapter starts very generally by setting out the case for gene selectionism (Section 1.2). It then explains how this underpins kin selection theory (Sections 1.3, 1.4). Next, Sections 1.5 to 1.9 elaborate on the structure and scope of the theory, covering topics such as inclusive fitness, how kin selection works at high frequencies of "genes for altruism," gene expression in Hamilton's rule, selection on loci other than loci for altruism, and parental manipulation theory. The conclusion is that kin selection theory is a logical corollary of gene-centered natural selection.

1.2 Natural Selection as Gene Selection

The world's living things are characterized by the detailed fit of their form and function to their way of life and environment – that is, by adaptive complexity. Darwin's (1859) theory of natural selection is biology's explanation for adaptation (e.g. Grant 1963; G.C. Williams 1966; Leigh 1971). Darwin framed natural selection theory in terms of individuals, stating the process to be a consequence of three properties. First, individuals vary. Second, they show heredity. And third, individuals with advantageous traits outreproduce those without such traits (Darwin 1859 pp. 80–81). Therefore, Darwin deduced, since the offspring of favorably endowed individuals would inherit their parents' beneficial features, favorable variations would accumulate in successful lineages, leading to the close fit of individuals to their lifestyle and environment that is organismal adaptation. Modern definitions of natural selection that characterize the process in terms of variation, heredity, and fitness differences (e.g. Lewontin 1970; Endler 1986) are essentially Darwin's individual-centered definition in more up-to-date language.

However, Dawkins (1976, 1978, 1982a,b, 1983, 1986) realized that the fundamental element in natural selection is a replicator, and so offered a new way of characterizing the process. A replicator is an entity of which copies are made, or which makes copies of itself (Dawkins 1982a p. 293, b). Replicators have two other important properties. The first is that each heads a potentially never-ending lineage of descendant replicators (Dawkins' [1982a p. 83] "germ-line" criterion). The second is that the copying process is exact to the point that alterations to replicator structure are preserved in successive replications (Dawkins 1978, 1982a pp. 97–99, b, 1989a pp. 273–274). Dawkins' "replicator version" of natural selection theory runs as follows. First, there are replicators

(structures that show high-fidelity copying). Second, there is a level of error, or mutation, in the copying process, producing undirected variation in each generation of replicators. Third, the structure and properties of replicators influence their survival and rate of replication. Given these conditions, the accumulation of favorable mutations within replicators will lead to the appearance of adaptive complexity benefiting the replicators.

Adaptation cannot arise in one stage, in what Dawkins (1986) calls single step selection, because this is too improbable. However, adaptation can arise through successive bouts of selection, in each of which new traits are added to – and if favorable preserved in – the existing set, yielding a complex, well-adapted final product. Dawkins (1986) calls this cumulative selection. Only this process can overcome the improbability inherent in adaptation that single step selection fails to address. This reasoning shows why the replicator concept is fundamental to natural selection theory: only replicators can form the basis of a process of cumulative selection, because only when mutations are conserved in replication can favorable ones accumulate. So Dawkins' argument is that adaptive complexity requires cumulative selection, which in turn requires replicators.

According to natural selection theory the world is presently full of units that exist because of their ancestors' success in leaving descendants. Therefore, going back in time, one would expect to find ancestral units that, though lacking foresight, were acting as if concerned to be successful in leaving descendants for the future. But the present time is just an arbitrary point in the continuum of history. So natural selection theory leads biologists to expect to find at all times a world inhabited by "units of self-interest" (Dawkins 1976 p. 12), where this means entities acting to ensure their successful propagation into the future. Such units must then persist over time, because they can only have inherited their "self-interest" from their ancestors if there is a correspondence between their past and present selves. Put another way, if these entities disintegrate, then they have no future to be successfully projected into. So entities undergoing natural selection, as units of self-interest, require what Dawkins (1986) terms durability.

Replicators have durability, but not in the sense of physical persistence. Instead, it follows from the earlier definition of replicators that their internal structure has the capacity to become sufficiently irregular to store information. It also follows that this information can be transmitted down the generations in the form of copies. Therefore, the durability of replicators is persistence in the form of copied messages (G.C. Williams 1985, 1992 p. 11; Dawkins 1986; Gliddon and Gouyon 1989), where the information preserved specifies adaptations. So replicators, with this kind of durability, can be units of self-interest.

In nature, the usual kinds of replicators are genes, lengths of DNA. Genes exist in aggregations embedded in bodies. Under gene selectionism, bodies are "vehicles" for replicators (Dawkins 1982a,b) – entities constructed by collectives of genes as dwelling places and aids to survival. (Hull's [1980] "interactor" broadly corresponds to Dawkins' vehicle. G.C. Williams [1992 p. 10] suggests the additional distinction between the "codical" [informational] and material "domains" inhabited by replicators and vehicles respectively; see also Cronin [1991].) Bodies are not replicators themselves because alterations to bodily structures are not transmitted in reproduction. This is the same as asserting that acquired characters are not inherited, or that inheritance is not Lamarckian. Bodies of course show heredity, but this is because of the genes they contain. Groups also are not replicators, because alterations to group structure are not conserved. Instead, groups are, like bodies, vehicles for replicators, because it may pay genes to instruct bodies to form societies for the genes' benefit. In addition, both bodies and groups lack durability, since no individual or group persists indefinitely, even in "message" form. Therefore, they are not units of self-interest in the sense that replicators are (Dawkins 1976).

Many species engage in sexual reproduction, which generally entails a fair meiosis (every gene in a body has the same 0.5 chance of getting into a gamete) and chromosomal crossing-over. The existence of crossing-over means that in species with sex it is impossible to say exactly what length of DNA constitutes a gene. Instead, an operational definition can be adopted of a gene as a piece of DNA small enough to have durability (persist down the generations in the form of copies), and large enough to encode meaningful information (G.C. Williams 1966, 1992 p. 18; Dawkins 1976, 1978, 1982b). The existence of fair meiosis also means that genes in sexual species are not usually in competition with genes at other loci (position on a chromosome) in the same individual (for exceptions see Section 1.8). Instead, genes in these species compete with each other in a highly structured way. In particular, they compete with their alleles in the population for representation at their shared locus (G.C. Williams 1966 pp. 57–58; Dawkins 1982a p. 283,b). This is made apparent by remembering that a rise in gene frequency, the definition of success for a focal gene, means a rise in the number of copies of a gene at its locus relative to the number of copies of its alleles. However, the selective background of a gene will still depend on genes at other loci in the body. A gene may even be favored on the basis of its success when matched with an allele, as in the case of heterozygote advantage (G.C. Williams 1966 p. 58; Dawkins 1976 p. 91, 1982a p. 52). Therefore gene selectionism needs to be applied in a qualified and sophisticated way to modern sexually reproducing species.

Another important point about modern genes is that some genes persist down the generations in groups within the larger collective (the genome) as linked "gene complexes" (Dawkins 1978). These complexes act like a large replicator. In addition, the entire genome of a sexual organism comes to represent a mutually stable set of replicators. This is because, as just mentioned, part of the selective environment of genes is made of the other genes it is likely to share a body with (Dawkins 1976). Therefore, in modern bodies adaptive complexity stems from the accumulation of favorable mutations both within the structure of individual replicators (by nucleotide substitution in genes, or gene substitution in gene complexes) and within the mutually stable community of replicators represented by the genome (by gene substitution).

Additional reasons exist for why, aside from being essential for cumulative selection, replicators are fundamental to any deep theory of natural selection. The first is that the arrow of causality points from genes to bodies (Dawkins 1983). Although this has been disputed (e.g. Gould 1983b; Sober 1984a), the causal priority of genes is evident from embryology and development, which involve a one-way flow of information from genes to bodies. So, since genes also carry information "vertically" (in heredity), an organismic trait cannot evolve by natural selection unless it is subject both to genetic control and genetic variation. Therefore, to regard gene frequency changes as simply the passive tracking of changes in the frequencies of traits (e.g. Bateson 1982; Gould 1983b, 1992), as "a kind of genetic bookkeeping" (Wimsatt 1980 p. 158), is to reverse the causal structure of natural selection theory. Traits are only worth considering as candidates for natural selection if they aid the survival and replication of genes (Dawkins 1983; G.C. Williams 1985).

The causal primacy of genes also has a temporal element, which becomes clear from considering the origin of life. Replicators occur in all living things (as RNA or DNA) and, as just discussed, control their development. From the earlier arguments, only replicators generate adaptive complexity. And, unlike bodies (which are too complex), replicators could have arisen spontaneously, by Dawkins' (1986) "single step selection." From these points, several authors have concluded that complex life originated as free replicators or "naked genes" (e.g. Dawkins 1982b p. 50), self-replicating molecules that were not embedded in bodies (Dawkins 1976, 1986). Replicators must then have evolved to acquire their complex cellular and bodily vehicles. In short, bodies exist because of genes, and not the other way round (Dawkins 1982a,b, 1983).

The other main reason why the replicator concept is so central to natural selection theory is that it has generality. Adaptive complexity must

be based on the natural selection of replicators, but these may be of many kinds (Dawkins 1983). Biologists typically regard nuclear, transcribed DNA as the prime genetic material. But the replicator concept also explains the existence and behavior of other kinds of replicating biological entity, for example (genes in) viral nucleic acids, mitochondrial DNA, "selfish" nuclear DNA, sex-ratio distorters, "killer" chromosomes, and plasmids (e.g. Doolittle and Sapienza 1980; Eberhard 1980, 1990; Orgel and Crick 1980; Cosmides and Tooby 1981; Dawkins 1982a; Werren et al. 1988; Hurst 1991, 1993a; Ebbert 1993).

The argument has now reached an important corollary of replicator-centered natural selection theory. This is the idea that all adaptations, including those seen in vehicles, ultimately exist for the benefit of the replicators responsible for them, and not for the good of the vehicles (Dawkins 1976, 1982a). This follows from the earlier demonstration that only replicators, not vehicles, have the durability to be units of self-interest. Put another way, adaptation arises from the accumulation of favorable mutations and – as argued earlier – this process can only occur in replicators. The idea that adaptations benefit replicators becomes important when the interests of replicators and vehicles, genes and bodies for example, do not coincide. An example comes from Hamilton (1967), who showed that a meiotic drive gene linked to the Y sex chromosome could produce an extreme male-biased sex ratio (its adaptation) that, in an unstructured population, could drive the population extinct. Therefore, recognizing the primacy of the replicator's interests shows that adaptation may not always involve "good design" at the organismic level. It may even fail to benefit the replicator itself in the long term. This merely emphasizes that natural selection is a mechanistic process without foresight.

Another example where the interests of replicators and vehicles do not coincide forms the main subject of this chapter – kin-selected altruism. The view that adaptations primarily benefit replicators suggests that when bodies behave in a self-destructive fashion, it could be for the selective advantage of a gene responsible for the behavior (Dawkins 1976, 1982a). This insight is central to the kin-selectionist explanation of self-sacrificial behavior (Section 1.4).

Lastly, these arguments imply that the traditional categories of individual selection, kin selection, and group selection are all, fundamentally, aspects of gene selection (Dawkins 1978). They do not rule out, however, a parallel approach to natural selection that focuses on the different levels at which selection acts, providing these levels are understood as levels of vehicles. This is the distinction between the individual- and replicator-centered views of natural selection (Chapter 2). In addition, in common with other behavioral ecologists, social insect biol-

ogists need to be aware that a gene selectionist, adaptationist approach presupposes a (simple) genetic basis to the traits they study (Grafen's [1984 p. 63] "phenotypic gambit"). Put another way, at some level of analysis all evolutionary hypotheses of adaptation in behavioral ecology have to be consistent with population genetics (Grafen 1984, 1988). To conclude, the arguments in this section show why we seek to understand adaptation in biology, including social insect biology, in terms of natural selection for adaptations serving the interests of genes.

1.3 The Problem of Altruism

The most obvious adaptive feature of ant biology is sociality, the habit of living in groups or colonies. The adaptiveness of sociality is demonstrated by the sophisticated design features shown by insect colonies, and by sociality's undoubted contribution to the ecological success of ants (E.O. Wilson 1987, 1990).

But ants are more than just social. All modern ants are eusocial ("truly social") or are workerless social parasites secondarily derived from eusocial species (Buschinger 1990a; Bourke and Franks 1991). The key trait of eusociality is that members of the society display a reproductive division of labor: some are fertile individuals (sexuals or reproductives, such as queens) and some are either completely sterile or show limited fertility (neuters or workers). The other defining features of eusociality (E.O. Wilson 1971 p. 4) are an overlap of adult generations in the society, and cooperative brood care, which together mean that the workers help raise the young of reproductives in the parental generation. Eusocial animals include the eusocial Hymenoptera (all ants, some bees, some wasps), termites, aphids (Aoki 1987; Itô 1989; Benton and Foster 1992), ambrosia beetles (Kent and Simpson 1992), thrips (Crespi 1992a), and some mole-rats (Sherman et al. 1991; Jarvis et al. 1994). According to how flexibly the definition is interpreted, they also include many other social vertebrates (Brockmann 1990; Gadagkar 1994; Crespi and Yanega 1995; Sherman et al. 1995).

From the definition of eusociality, it is apparent that another important biological trait of ants is altruism. Biological altruism can itself be defined in various ways (e.g. Alexander 1974; Orlove 1975; Crozier 1979; Starr 1979; Uyenoyama and Feldman 1980; Bertram 1982), but most definitions are framed in terms of effects, not motivation or psychology, and in terms of individuals, not genes. (Genetic altruism cannot evolve because a gene suffering a net loss in its representation in future generations cannot, by definition, increase in frequency [Alexander 1974]. The problem of altruism is the problem of how indi-

vidual-centered altruism evolves.) The definition to be followed here is that *altruism occurs when an individual behaves in such a way that the result is an increase in the survival or offspring production of another individual and a decrease in its own survival or offspring production.* So eusociality is "sociality-with-altruism," because workers give up their own chances of survival and reproduction to promote those of the brood they rear. Because of a focus of interest on the sacrifice in reproduction, this behavior is often specifically termed reproductive altruism (e.g. Trivers 1985 p. 169).

As already described, Darwin formulated natural selection theory in terms of selection acting on the individual. Altruism then emerged as a problem for the theory, which predicted that each individual should behave as if trying to maximize its number of offspring. In the language of the previous section, Darwin saw the individual organism as the "unit of self-interest." Altruism, and in particular extreme reproductive altruism involving sterility, contradicted this prediction, and so seemed to undermine the theory. This is the problem of altruism (see Cronin [1991] for a historical review).

Darwin's solution to this problem in the social insects was that workers could evolve if they were "profitable to the community," by which he meant the colony (Darwin 1859 p. 236). He also discussed, considering them greater problems than the problem of sterility, how workers could evolve to differ morphologically from queens and from one another. These passages (Darwin 1859 pp. 236–242) indicate that, as regards the evolution of sterility, Darwin envisaged colonies with workers benefiting by producing extra queens and males. So sexuals from these colonies would have been favored by selection, given that they would transmit to their own sexual offspring the profitable trait of worker-production. With hindsight, it is apparent that Darwin therefore closely anticipated modern, kin-selectionist explanations of worker sterility (E.O. Wilson 1975 p. 117; Alexander et al. 1991; Seger 1991; see also Cronin 1991 p. 298).

Nowadays, biologists recognize three ways in which altruism can evolve by natural selection acting on genes (e.g. Alexander 1974; West-Eberhard 1975; Ridley and Dawkins 1981; Trivers 1985). These are: (1) by kin selection (Hamilton 1963, 1964a,b, 1970, 1971a, 1972); (2) via "delayed benefits" (e.g. Bertram 1982 p. 257); and (3) by manipulation or social (or brood) parasitism. By "delayed benefits" is meant, for example, that an individual may join a social group as a helper in the hope of later inheriting the position of breeder. This kind of route to altruism is therefore sometimes called a "hopeful reproductive" or "mutualism" route (West-Eberhard 1978a; Seger 1991; Section 3.2). Another important kind of delayed benefits altruism occurs when an

individual makes a self-sacrifice in return for a future repayment by the beneficiary. This is termed reciprocal altruism (Trivers 1971; Axelrod and Hamilton 1981; Axelrod 1984; Ligon 1991; Dugatkin et al. 1992; Mesterton-Gibbons and Dugatkin 1992). Since reciprocal altruism may occur between relatives, this route to altruism and the kin selection one are not mutually exclusive (e.g. Nee 1989; Ligon 1991; Dugatkin et al. 1994). Over the time-scale of the individual's lifespan, delayed benefits altruism profits the "altruist" individual itself. It is therefore only altruism in a short-term sense (Alexander 1974), and so is more accurately classified as cooperative (see below) rather than altruistic behavior. Social parasitism typically involves the exploitation of preexisting altruistic behavior (generally evolved by kin selection) in a host of a different species, although it can be intraspecific. Parental manipulation of offspring has also been proposed as a promoter of altruism (Alexander 1974), but Section 1.9 argues that this idea falls within kin selection theory.

Another suggested mechanism for the evolution of altruism is group selection (e.g. Wynne-Edwards 1962). Group selection is controversial, partly because the term has been used to describe different processes (Maynard Smith 1976, 1982a; Wade 1978b; D.S. Wilson 1983; Grafen 1984). In one sense (not Wynne-Edward's), it is legitimate to say that kin selection for altruism involves group selection (e.g. Wade 1980). This topic is discussed in Chapter 2. The following sections consider the evolution of altruism by kin selection, and how kin selection theory solved Darwin's problem of altruism.

1.4 Kin Selection and Hamilton's Rule

Kin selection theory was formulated by Hamilton (1963, 1964a,b) as inclusive fitness theory, and termed kin selection by Maynard Smith (1964). Several earlier authors had appreciated that altruistic behavior could evolve via benefits to relatives (e.g. Darwin 1859; Fisher 1930; Haldane 1932, 1955; Williams and Williams 1957). But Hamilton was the first to develop kin selection as an evolutionary principle of far-reaching and radical importance. *Kin selection is the natural selection of genes for social actions via the sharing of these genes between the performer of the action and its relatives (kin).* For other definitions of kin selection, and reviews, see for example West-Eberhard (1975), Kurland (1980), Boorman and Levitt (1980), Michod (1982), and Trivers (1985).

To explain this definition of kin selection. First, by a gene "for" a trait is meant, as is conventional, the gene that makes the difference between whether the trait is shown or not. So if bearers of a gene G on

average show a trait T, and nonbearers do not show it, G is the gene "for" trait T (e.g. Dawkins 1979). Next, a social action occurs when an individual (the actor) behaves so as to increase or decrease the personal fitness (survival or offspring production) of other individuals (often termed neighbors, or recipients of the action). As a result, the actor may alter its own survival or offspring production. So genes for social actions are genes whose bearers, on average, perform social acts. It is evident from these definitions that there can be four mutually exclusive types of social action, according to whether the actor and recipient gain or lose personal fitness. These types – cooperation, selfishness, altruism, and spite – are defined in Table 1.1.

Table 1.1

Types of Social Action

		Effect on Recipient's Survival or Offspring Number	
		Gains	*Loses*
Effect on actor's survival or offspring number	Gains	Cooperation	Selfishness
	Loses	Altruism	Spite

After Hamilton (1964a, 1970), Trivers (1985), Gadagkar (1993).

Kin selection theory applies to the evolution of all four social actions. However, in practice it has been mostly used to explain altruism, because this created the greatest puzzle for individual selection theory. Another important feature of the definition of altruism is that it encompasses parental care (Clutton-Brock 1991), because parents who care reduce their own survival (compared to parents who do not care) while promoting that of their young. Correspondingly, since offspring are relatives, kin selection theory underpins the evolution of parental care, and was indeed formulated with parental care in mind (Hamilton 1963, 1964a). However, again, kin selection theory has not been widely invoked to explain parental care, because this seemed adequately explained by individual selection (Dawkins 1979).

Hamilton derived a condition for the spread of a gene for a social action now known as Hamilton's rule (Charnov 1977). Hamilton's rule is the mathematical embodiment of kin selection theory. It involves three terms: the magnitude of the change in the actor's personal fitness; the magnitude of the change in the personal fitness of the recipient; and

BOX 1.1 THE REGRESSION DEFINITION OF RELATEDNESS

Relatedness is a measure of genetic similarity, but its definition forms a complex subject that has been much debated in population genetics (e.g. Hamilton 1970, 1972; Orlove 1975; Michod and Anderson 1979; Michod and Hamilton 1980; Uyenoyama and Feldman 1981; Michod 1982; Pamilo and Crozier 1982; Grafen 1985, 1986, 1991; Bennett 1987a; Moritz and Southwick 1992 p. 237; Queller 1992a, 1994a; Gayley 1993). In kin selection theory (Section 1.4), relatedness is best defined as a regression coefficient (Hamilton 1970, 1972). Consider an outbreeding population consisting of groups or pairs of potential social interactants (potential donors of a social action, or actors, and potential recipients of the action). Regression relatedness at a locus equals the sign and slope of the regression line obtained when the focal gene's average frequency among the potential recipients within groups is regressed, across all the groups, on its within-group frequency in a random potential actor (e.g. Pamilo and Crozier 1982).

This is illustrated in Figure 1.1. The regression of the gene frequencies across groups is positive, so by definition the interactants within the groups are related. If they were not, then in Figure 1.1 relatedness would be represented by the flat dashed line at p on the Y axis. So a relatedness of zero does not imply that individuals have no genes in common, but instead that they share the focal gene with only random probability (p).

Clearly, positive relatedness means that if a potential actor has the focal gene with high (above-average) frequency, then so do the potential recipients, its group-mates. Say A is a gene for a social action (Section 1.4). Then the frequency of A in potential actors also determines the likelihood of the social action being performed (for example, in the extreme case of the frequency of A in actors being zero, the action will obviously not be performed). So positive relatedness at loci for social actions means that social actions are likely to be directed at individuals (recipients) that share the same gene with above-average frequency. This is why kin selection for altruism works, and why relatedness is central to it (Section 1.4).

Note also that the average frequency of gene A in the potential recipients and potential actors is the same, and equals p, the population average gene frequency (Figure 1.1). This condition forms a basic assumption of kin selection theory (e.g. Grafen 1984; Maynard Smith 1989 p. 173). For, as already described, the concept of relatedness does not involve average gene frequencies, but how individuals with frequencies other than the average associate. In kin selection theory, the roles of potential recipient and potential actor are not determined by gene frequency (except that individuals without the gene for the social action will not be actors), but are determined conditionally. In other words, contrary to some misunderstandings of kin selection, there is no genetic difference between actors and recipients (Section 1.7).

BOX 1.1 CONT.

Another important point is that relatedness and the population average gene frequency are independent of one another. In Figure 1.1, p was arbitrarily set fairly low. But p could be any value without the slope and sign of the regression line being different. This reemphasizes the point that relatedness concerns the chance of gene sharing independently of the average chance (set by p).

What is the link between regression relatedness and kinship? This becomes apparent if groups of potential interactants are families. Clearly, regression relatedness will then measure genetic similarity due to ties of pedigree. If parents have a focal gene, their offspring share it. If parents lack the gene, offspring lack it. So being members of the same family is a way of associating with individuals likely to share focal genes. Therefore kinship guarantees positive relatedness.

Let us now prove that applying the regression definition of relatedness yields the familiar relatedness value of $r = 0.5$ for diploid sibs. The method comes from Pamilo and Crozier (1982). Consider a population of outbred diploids divided into family groups. At a focal (autosomal) locus, the frequency of a gene A equals p, and that of its allele a equals q, where $p + q = 1$. The problem is to find the regression relatedness between sibs for gene A.

Each parent in the population can be of genotype AA, Aa, or aa. Assuming Hardy-Weinberg equilibrium, the frequencies of these genotypes are p^2, $2pq$, and q^2 respectively. So each family in the population will be headed by a parental pair in one of the possible combinations (mating types) shown in column 1 of Table 1.2.

The frequencies of these mating types are then calculated as the product of the parental genotype frequencies, summed over the possible ways of achieving each mating type (column 2 in Table 1.2). In addition, Table 1.2 gives, in each kind of family, the genotypes of the offspring and their ratios of abundance (column 3), the frequency of gene A in each potential actor (each genotype of sib) (column 4), and the frequency of A among potential recipients (all sibs) (column 5).

For calculating relatedness, the numbers in column 5 of Table 1.2 (Y values) are regressed on those in column 4 (X values). Each (X, Y) pair must also be weighted by its frequency (f) in the population, which is given by the product of X's genotype frequency among the offspring and the frequency of the family type. For example, the frequency of the pair ($X = 0.5$, $Y = 0.5$) is $1/2(4p^2q^2) + 2p^2q^2 = 4p^2q^2$. The plot of the X and Y values from Table 1.2, with the corresponding f values, is therefore as shown in Figure 1.2 (compare with Figure 1.1).

The next step is to find the slope of the line through the points in Figure 1.2. This equals the regression coefficient of Y on X, which is defined as

BOX 1.1 CONT.

$$\Sigma f(X - \bar{X})(Y - \bar{Y})/\Sigma f(X - \bar{X})^2$$

where $\bar{X}$ and $\bar{Y}$ are the mean values of X and Y respectively. These mean values must both equal p. This is known because all offspring can be potential actors or recipients, and if there is Hardy-Weinberg equilibrium the average frequency of a gene among offspring equals that among their parents, which was set at p. (This reasoning reflects the assertion above that the average frequencies of a focal gene in potential actors and recipients are the same, and equal the gene's population mean frequency.) Using the regression formula with the X, Y, and f values from Table 1.2 and Figure 1.2, and $\bar{X}$ and $\bar{Y}$ values of p, one finds after a lot of algebra that the regression coefficient equals $(1/4)/(1/2) = 0.5$. So sib-sib regression relatedness in diploids is 0.5. Similar calculations would yield all the familiar between-kin relatedness values.

Four more points about regression relatedness need making.

1. From the regression definition, relatedness can in theory take any value, positive or negative (Grafen 1991). But in most cases relatedness lies between 0 (no relatedness) and 1 (clonality). Negative relatedness means that bearers of a focal gene systematically associate with individuals bearing the gene with below-average frequency. The unusual conditions leading to this are discussed by Grafen (1985).

2. Relatedness can also be understood as the average proportion of genes at a locus in the recipient that is identical with any allele at the locus in the actor (Grafen 1986). This equals the probability that a gene in the recipient is present in the actor. The concept of relatedness as a proportion or probability appears frequently in the sociobiological literature. It is usually qualified by the statement that the gene copies in question must be "identical by descent," meaning "descended from the same copy of the gene in their most recent common ancestor" (Dawkins 1979 p. 191). However, the choice of most recent ancestor in the criterion for identity by descent is arbitrary, since every copy of a particular gene must descend from a common ancestral copy if one traces back through enough generations (Seger 1981; Grafen 1985). So the regression definition of relatedness allows biologists to discard the problematic idea of identity by descent (e.g. Gayley 1993).

3. The regression definition also underlies methods of estimating relatedness from gene frequency data obtained from social groups by, for example, electrophoretic allozyme analysis (Pamilo and Crozier 1982; Queller and Goodnight 1989; Pamilo 1989, 1990a). These measures assume that relatedness at neutral allozyme loci estimates relatedness at the putative loci for social actions (under weak selection). This is reasonable because kinship makes average relatedness between any two

BOX 1.1 CONT.

individuals the same across all their loci (Section 1.8).

4. Lastly, on a point of terminology, the regression definition explains why the relatedness between an actor A and a recipient R should strictly be termed b and phrased as the relatedness of R to A, $b_{R.A}$. This is because, by statistical convention, the regression of a dependent variable Y on an independent variable X is denoted $b_{Y.X}$ (Michod and Anderson 1979; Crozier and Pamilo 1980; Pamilo and Crozier 1982).

Boxes 1.3 and 3.1 discuss relatedness further. Box 1.3 describes Grafen's (1985) "geometric view of relatedness," which is formally equivalent to the regression definition. Box 3.1 deals with relatedness levels

the relatedness between the actor and the recipient. Relatedness is a measure of the genetic similarity between two individuals. In kin selection theory, it is formally defined as a regression coefficient, as Box 1.1 describes.

Why does relatedness feature in the gene selectionist explanation of altruism? Imagine a wildebeest carrying a gene that causes it to eat less

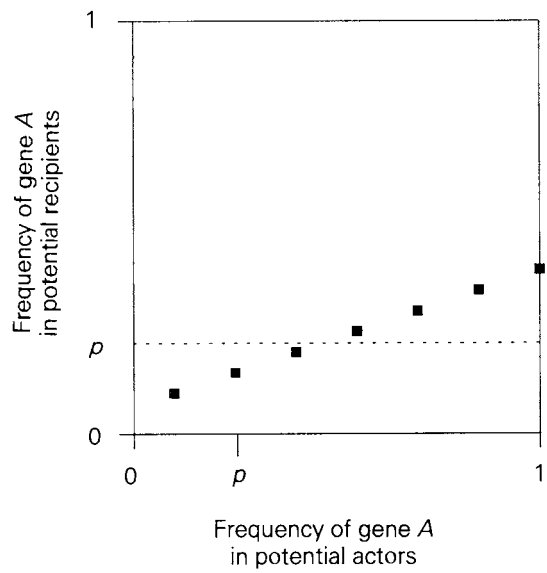


Figure 1.1 The regression relatedness between potential social interactants measured over a number of groups (represented by the black squares) for a gene A at a locus. Regression relatedness is given by the sign and slope of the line formed by these points. The average frequency of A in the population is p.

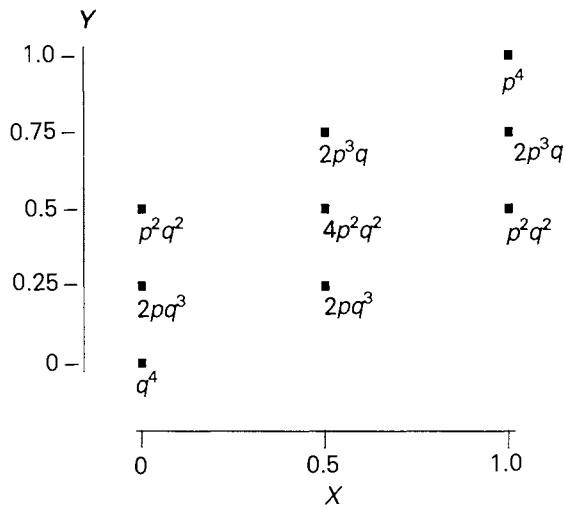


Figure 1.2 The regression of gene frequency in potential recipients (Y) on gene frequency in random potential actors (X) in a population of families of outbred diploids. The expressions below each point are the weights of the points (f).

Table 1.2

Genotype and Gene Frequencies in Families in Randomly Mating Diploids

1 *Parents Heading Family*	*2* *Frequency of Family*	*3* *Offspring (sib) Genotypes (with ratios if >1 type)*	*4* *Frequency of* A *in Random Potential Actor* (X)	*5* *Frequency of* A *in Potential Recipients* (Y)
$AA \times AA$	p^4	*AA*	1.0	1.0
$AA \times Aa$	$4p^3q$	*AA, Aa* (1 : 1)	1.0, 0.5	0.75
$AA \times aa$	$2p^2q^2$	*Aa*	0.5	0.5
$Aa \times Aa$	$4p^2q^2$	*AA, Aa, aa* (1 : 2 : 1)	1.0, 0.5, 0	0.5
$Aa \times aa$	$4pq^3$	*Aa, aa* (1 : 1)	0.5, 0	0.25
$aa \times aa$	q^4	*aa*	0	0

grass. By the earlier definition, this action is altruistic, because food loss decreases the actor's personal fitness and increases that of its neighbors (each remaining wildebeest in the herd has slightly more grass to eat). Will this gene spread through the wildebeest population by kin selection?

The answer is no, because wildebeest herds do not consist of groups of relatives. So the beneficiaries of the abstaining animal are a random section of the population, and share the focal gene with random probability, set by the gene's mean frequency in the population. When these individuals reproduce, their extra offspring will also bear the gene with random probability, because unless other factors intervene the average gene frequencies of offspring equal those of their parents. Therefore, the original animal's action brings about no increase in the frequency of the causative gene.

Suppose, however, that wildebeest lived in stable, sedentary family groups. (West-Eberhard [1975] discusses the influence of herd structure on kin selection.) Then the original animal's action would benefit a nonrandom section of the population, namely those with an above-average probability of bearing the focal gene via the sharing of genes inherent in kinship. Altruistic restraint in eating could then be positively selected, because the extra young produced as a result would also bear the gene with above-average frequency, resulting in an increase of the gene in the population. This example demonstrates why relatedness is crucial to the action of selection on a gene for altruism (see also Box 1.1). It also foreshadows the later discussion of Grafen's (1985) geometric view of relatedness (Section 1.6; Box 1.3).

In addition, this example illustrates the following point. Let a positive effect of a gene be one that increases its bearer's offspring production relative to the effect of the gene's allele, and a negative effect be one that does the opposite. Then all genes have either a positive effect on their bearer, or a negative effect, or no effect. Similarly, all genes have either a positive effect on the offspring production of a bearer's neighbors, or a negative one, or none. Therefore, excluding genes of null effect, all genes are genes for social actions. Dawkins (1979) made this point with particular reference to genes for selfishness and altruism. So there is nothing "special" about genes for social actions. On the contrary, they are probably very common. The issue is not whether these genes exist, but whether conditions are such that they can spread through populations. Hamilton's rule is the evolutionary principle specifying these conditions. Further, recognizing that genes for social actions potentially encompass many traits suggests that kin selection theory has very broad applicability. In fact, it is relevant whenever conspecific organisms interact in ways that influence personal fitness and are nonrandom with respect to relatedness. This means that kin selection thinking has justifiably entered many

fields that are not traditionally part of social biology at all, for example parasitology (Wickler 1976; Frank 1994), population ecology (Charnov and Finerty 1980), botany (Haig 1987; Queller 1989a), microbiology (Shub 1994), and medicine (Westoby 1994).

Box 1.2 gives a simple derivation of Hamilton's rule, especially as it applies to the evolution of altruism. Briefly, Hamilton's rule for altruism is that a gene for altruism spreads if the inequality $rb - c > 0$ is satisfied. In this expression, r is the regression relatedness between altruist and beneficiary (Box 1.1), b is the benefit of altruism in terms of the extra offspring the beneficiary gains, and c is the cost of altruism in terms of the offspring production lost by the altruist (Box 1.2).

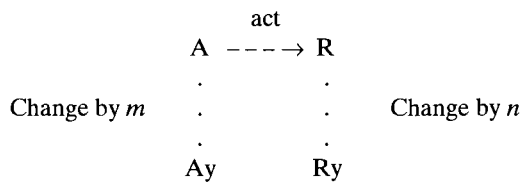
In the context of the study of social biology, Hamilton's rule implies that eusocial evolution is influenced both by genetical factors (affecting relatedness) and by ecological and ergonomic ones (affecting benefit and cost). However, the need to take account of both genetics and ecology in the study of eusociality was for a time neglected, despite warnings that it should not be (e.g. West-Eberhard 1975; Evans 1977). This was because, along with kin selection theory, Hamilton (1964b) proposed that haplodiploid sex determination in the Hymenoptera could lead to high relatedness levels especially favorable to eusocial evolution. This "haplodiploidy hypothesis" (Chapter 3) concentrated attention on genetic factors and, as noted by Dawkins (1989a p. 316), led many biologists to believe that kin selection theory required particularly high relatedness to account for eusociality. They therefore doubted the theory whenever such levels were found to be absent. This was a mistake. Kin selection theory states that altruist and beneficiary must show some level of relatedness, as opposed to none at all. In addition, high relatedness will certainly facilitate eusocial evolution. But Hamilton's rule shows that altruism can evolve if relatedness is very low (for example, if r and c are both low but b is very high). The preoccupation with genetic factors also obscured the common ecological factors such as nest-site shortage or a patchy distribution of food that promote eusociality in insects and similar phenomena (helpers at the nest, cooperative breeding) in birds and mammals. However, such ecological parallels are now being increasingly recognized (e.g. Vehrencamp 1979; Andersson 1984; Strassmann and Queller 1989; Brockmann 1990; Pamilo 1991a; Sherman et al. 1991; Crespi 1994).

Hamilton's rule suggests a powerful qualitative way of looking at kin selection. Exactly why does the gene for altruism spread through a population under kin selection? The answer is that the gene causes its bearer to care for individuals having an above-average probability of sharing the same gene, through relatedness. So, in terms of its representation in the next generation, what the gene loses in the sacrifice of the

BOX 1.2 DERIVING HAMILTON'S RULE

This box gives an informal derivation of Hamilton's rule (cf. West-Eberhard 1975; Craig 1979; Section 1.4). Michod (1982) and Grafen (1985) review the large population genetics literature on the rule. Papers formally proving it include Charnov (1977), Wade (1978a), Charlesworth (1980), Aoki (1981), Michod (1982), Queller (1984, 1985, 1992a), Grafen (1985), Goodnight et al. (1992), Gayley (1993), and Sibly (1994). Note that although several proofs deal with a single "gene for altruism," others are general inclusive fitness models or explicitly treat altruism as a quantitative trait influenced by many genes (e.g. Yokoyama and Felsenstein 1978; Boyd and Richerson 1980; Aoki 1982; Cheverud 1985).

Imagine an individual A (the actor) that carries a gene G for a social action. It influences another individual, the recipient R, with the result that A's expected offspring number is changed by m offspring, and R's is changed by n offspring. Let A's young be denoted Ay, and R's young, Ry. In addition, let the regression coefficient of relatedness between two individuals X (actor) and Y (recipient) be $r_{Y.X}$ (Box 1.1).



The change to gene G's frequency caused by A's social action is $(m \times r_{Ay.A})$ via effects on A's own young, and $(n \times r_{Ry.A})$ via effects on R's young. For example, if m were positive and n negative, m individuals each with a chance of $r_{Ay.A}$ of bearing the gene would be added to the population, and n individuals bearing the gene with a chance of $r_{Ry.A}$ would be lost from it.

By definition, gene G increases in frequency if the total change in its frequency is positive, that is if

$$(m \times r_{Ay.A}) + (n \times r_{Ry.A}) > 0.$$

This is Hamilton's rule in a general form, demonstrating the assertion (Section 1.4) that it applies to the spread of genes for all kinds of social actions. To adapt it for the evolution of altruism, note that in this case, by definition, m is negative (and is conventionally called c for cost), whereas n is positive (and is conventionally called b for benefit). So the rule becomes

$$(-c \times r_{Ay.A}) + (b \times r_{Ry.A}) > 0.$$

BOX 1.2 CONT.

This can be altered as follows. First, divide each side by $r_{Ay.A}$ and rearrange to give $[(r_{Ry.A}/r_{Ay.A}) \times b] - c > 0$. Second, note that, assuming $r_{Ay.A} = r_{Ry.R}$ (parent–offspring relatedness is uniform), then $r_{Ry.A}/r_{Ay.A}$ equals $r_{Ry.A}/r_{Ry.R}$. Finally, note that this last term equals $r_{R.A}$, since the relatedness of A with R ($r_{R.A}$) multiplied by the relatedness of R with R's young ($r_{Ry.R}$) equals the relatedness of A with R's young ($r_{Ry.A}$). Therefore, Hamilton's rule can be rewritten as

$$rb - c > 0$$

where r equals $r_{R.A}$. So, *Hamilton's rule states that a gene for altruism spreads if the genetic profit, calculated as the recipient's extra offspring production multiplied by the relatedness of the altruist and the recipient, minus the genetic loss, which is the fall in the altruist's offspring production, is greater than zero.*

Hamilton's rule is now a well-accepted theorem in population genetics (e.g. Grafen 1985). However, its validity depends on certain assumptions. These include additivity of gene effects (each separate instance of social behavior has the same effect on fitness), weak selection, and outbreeding (e.g. Grafen 1984, 1985; Queller 1984, 1985, 1989a; Maynard Smith 1989 pp. 172–173).

altruistic body it occupies, it can redeem many times over in the enhanced survival or reproduction of the beneficiaries. In effect, then, the gene spreads because it promotes care for copies of itself (Dawkins 1979). This perspective reveals why kin selection theory, as well as explaining the evolution of altruism towards nondescendant kin, also accounts for the evolution of parental care (Hamilton 1963, 1964a; Maynard Smith 1964; Dawkins 1979; Michod 1982). Both cases involve a gene promoting care for copies of itself in related individuals. Both are therefore aspects of the same, gene-selectionist phenomenon, namely kin selection.

Kin selection theory has had a major impact on evolutionary biology as a whole. With the theory, Hamilton solved Darwin's problem of how traits disadvantageous to their bearers in terms of personal fitness could evolve. In other words, he solved the problem that altruism posed for the individual-centered theory of natural selection. Further, Hamilton did this by recasting the problem in gene-level terms, through imagining a gene for altruism and then deriving a population genetics condition for the positive selection of such a gene. This revealed the inadequacy

of individualistic natural selection as an explanation of adaptive phenomena compared to the gene-level view. Hamilton's work therefore helped bring about the gene selectionist revolution in evolutionary biology of the past twenty-five years. This has involved the reformulation of natural selection theory in explicitly genetic terms (Section 1.2), most notably by G.C. Williams (1966) and Dawkins (1976). In fact, gene-level thinking was already present in the work of Fisher (1930), Wright (1931), and Haldane (1932), but it has taken modern gene selectionists to make it indispensable, especially in the field of behavior.

Many biologists use gene-centered language in discussing natural selection, and so clearly accept gene selectionism in some sense. But often their phraseology betrays a traditional individual-level perspective. For example, modern texts often make statements like "animals are naturally selected to pass on as many copies of their genes as possible to the next generation." Such statements fail to acknowledge the full radicalism of the gene selectionist revolution (Dawkins 1978, 1979, 1982a,b). This can be illustrated with regard to kin selection theory, where such statements translate into something like "individuals are selected to aid relatives because they share a high proportion of genes with them." This formulation is true in a sense (because a high average fraction of shared genes means that the gene for altruism is shared with high probability). But it fails to capture the essence of kin selection theory, because it distracts the focus from the party for whose benefit altruism has evolved, namely the gene for altruism. Clearly, the individual itself does not benefit from altruism in individual selection terms (by definition). Also, it cannot be taken for granted that all the other genes in the individual apart from the gene for altruism will benefit (Dawkins 1982a; Section 1.8). This means that statements of the above sort are not only theoretically unsatisfactory but also at times misleading. Such statements are admittedly sometimes used as a lazy shorthand, for example "Female workers can propagate their genes more effectively by raising their exceptionally closely related fertile sisters than by producing offspring themselves" (Franks and Bourke 1988 p. 48). But to represent kin selection as an elaborate kind of individual selection hampers understanding by obscuring its gene selectionist logic (Dawkins 1979).

One additional result of this half-hearted acceptance of gene selectionism is that biologists often use individual-level selection as a synonym for gene-level selection. This may not always matter, because in the largely nonsocial, sexual diploid organisms studied by many behavioral ecologists, the predictions of the two perspectives will often coincide. Nevertheless, this synonymizing is undesirable because it again loses the focus on causative genes and because it blurs Dawkins' (1982a,b) important distinction between replicators and vehicles

(Sections 1.2, 2.4). Natural selection is the process by which replicators promote their own survival and replication (Section 1.2). Commonly, this involves genes promoting their survival and replication through the agency of organisms (e.g. Dawkins 1978). This in turn may often involve individuals maximizing their gene transmission, and even their number of offspring, but need not.

Finally, kin selection theory is qualitatively very well supported, in that most animal societies with some degree of altruism are societies of relatives (examples in E.O. Wilson 1975). Certainly this is true in the eusocial insects, including the ants.

1.5 Inclusive Fitness

Now that the basic idea of kin selection has been presented, this and the following four sections discuss a number of issues arising from the theory, beginning with inclusive fitness. Fitness is the measure that evolutionary biologists use to gauge evolutionary success under natural selection within populations. But fitness can be measured in many ways (reviewed by Dawkins 1982a; Endler 1986). For example, field biologists may count the lifetime number of offspring of individuals, whereas population geneticists measure the average offspring production of individuals of given genotypes, relative to some standard. Both these measures refer to offspring number. In devising kin selection theory, Hamilton (1964a,b) recognized that a gene for a social action could be positively or negatively selected due to its effects on relatives other than offspring. Hamilton therefore invented an "inclusive" fitness, so-called because it included not just an individual's genetic representation in offspring, but in other classes of relative as well. Unfortunately, there has been much confusion over the precise definition of inclusive fitness (Grafen 1982, 1984, 1988, 1991; Creel 1990). For example, Grafen (1982) pointed out that inclusive fitness is often incorrectly defined as an individual's number of offspring, plus all the offspring of its relatives, with these being weighted by the appropriate coefficient of relatedness. The problem with this quantity is that it is theoretically almost never-ending (because every member of a population is connected by a pedigree link with a focal individual to some degree). It also fails to predict the direction of gene frequency change, as a measure of fitness should (Grafen 1982).

The correct definition of inclusive fitness is more subtle and complex. However, as Grafen (1984) demonstrated, this need not be worrying, because an inclusive fitness calculation and Hamilton's rule provide two ways of deciding the same thing, namely the direction of selection on a

gene for a social action. In inclusive fitness terms, Hamilton's rule is the statement that a gene for a social action spreads if bearers have above-average inclusive fitness (Grafen 1991). But Hamilton's rule has the advantage of a clearer logic (Grafen 1984). There may also be major practical problems in measuring inclusive fitness in the field (Grafen 1982, 1984, 1988; Queller and Strassmann 1989). So this section is designed to clarify inclusive fitness as a concept, not to present a methodology for its measurement.

An exact verbal definition of inclusive fitness can be constructed as follows (Hamilton 1964a; Grafen 1982; Creel 1990). Consider a population of animals. Each will experience what Hamilton (1964a) called the "social environment," which is that part of its environment represented by interactions with conspecific neighbors. These neighbors may increase or decrease a focal individual's offspring production (by help or hindrance), and a focal individual may in turn increase or decrease (by helping or hindering) the offspring production of neighbors.

In this population, one could in principle calculate the total number of extra offspring conferred by help, divided by the total number of individuals (helpers and breeders) in the population. This quantity will be termed the average per capita amount of help. Similarly, one could work out the total number of offspring lost due to hindrance, over the total population size. Let this be the average per capita amount of hindrance. The inclusive fitness of a focal individual then equals the following two-part expression:

(1) the number of offspring produced by the focal individual, minus the average per capita amount of help (if there is helping in the population), plus the average per capita amount of hindrance (if there is hindering in the population); plus

(2) the number of extra offspring the focal individual's help confers on its neighbors (if it does help them), minus the loss in offspring its hindrance causes to its neighbors (if it does hinder them), with these quantities being weighted by the respective coefficients of relatedness between the focal individual and its neighbors, and then summed over all affected neighbors.

Part (1) of this definition corresponds to Hamilton's (1964a p. 8) "production of adult offspring . . . stripped of all components which can be considered as due to the individual's social environment, leaving the fitness which he would express if not exposed to any of the harms or benefits of that environment." Part (2) corresponds to Hamilton's "certain fractions [i.e. coefficients of relatedness] of the quantities of harm and benefit which the individual himself causes to the fitnesses of his neighbours." Note, however, that in Hamilton's verbal definition, the

quantity "stripped" from the offspring production of a focal individual receiving help is *all* its offspring due to help, rather than the average per capita amount of help. Creel (1990) noticed that, to achieve consistency with Hamilton's (1964a) mathematical formulation of inclusive fitness, the first part of the verbal definition needed, as above, to be recast in terms of average per capita effects.

To see why this matters, consider a population of breeders and helpers in which the breeders cannot reproduce without help and in which helpers never reproduce. An example would be a population of queens of eusocial Hymenoptera with totally sterile workers. On the original definition, the inclusive fitness of all breeders in this population would be zero (Creel 1990). This is because in calculating part (1) of a breeder's inclusive fitness, all of its offspring would have to be "stripped" away, being entirely due to help received, leaving a part (1) of nothing; and because a breeder gives no help itself, meaning part (2) of its inclusive fitness is also zero. Creel's (1990) correction disposes of this paradoxical result, since now not all offspring due to help received are stripped. Instead, only the average number of extra offspring conferred per head of population is removed.

Creel's (1990) correction also shows why a breeder that produces many reproductive offspring in this kind of population would be fitter than one with few, as seems intuitively reasonable. Part (1) of a breeder's inclusive fitness is the only relevant quantity, since all breeders have a zero part (2). In other words, biologically, the inclusive fitness of breeders will depend on the amount of help they attract. A breeder attracting an above-average amount of help will have a high part (1) fitness when the average per capita amount of help received is stripped away. This means it will have above-average inclusive fitness. On the other hand, a breeder attracting a below-average amount of help will have a low part (1) fitness after the average per capita amount of help is removed, leaving a below-average inclusive fitness. Therefore, Creel's (1990) modified definition correctly gives productive breeders higher inclusive fitness than less productive ones, even though every breeder depends on help for all its offspring production.

Similar considerations apply to the helpers in this population. Now the relevant quantity is part (2) of inclusive fitness, since all sterile helpers have the same part (1) (they have no offspring and the other terms are all average values). If a helper confers a lot of assistance, many relatives bearing the helping gene will be added to the population. If it confers little, the opposite will be the case. Therefore, as well as giving and justifying a definition of inclusive fitness, this section has now also shown that for both breeders and helpers inclusive fitness is a satisfactory measure of fitness, in that it correctly predicts the direction

of gene frequency change. In effect, it has confirmed the earlier assertion that in the social environment an inclusive fitness calculation is equivalent to applying Hamilton's rule for determining the direction of selection on genes for social actions.

1.6 Kin Selection Works at All Gene Frequencies

An important question raised by recognizing kin selection to be an explicitly gene-level theory is the following. How does kin selection for altruism work when a gene for altruism becomes very common in a population? This appears to lead to universal, indiscriminate altruism, and yet phenomena such as nestmate recognition among ants (Section 7.3) show that this does not occur. But the logic described earlier suggests that, in a population with a high frequency of a gene for altruism, every individual should regard all others as related by a level approaching 1.0 at the altruism locus, and should therefore help them regardless of genealogical ties. So it seems that relatedness becomes a redundant concept at high frequencies of the gene for altruism, because all members of the population now share the gene with high probability.

There are two ways to resolve this problem, the first being an argument made by Dawkins (1979). Any gene for helping is likely to operate via a behavioral "rule of thumb," such as – in the social insects – "rear sexual forms in the nest in which you live." The statistical association between nest-sharing and relatedness means that a rule like this will usually lead to kin being aided. Therefore, when a gene for helping becomes very frequent, or even fixed in a population, the rule also becomes universal. The result is a population in which help is still directed at nestmates (kin), so help remains discriminating.

The second, more rigorous approach to this question is to demonstrate theoretically that kin selection can operate at all frequencies of the gene for altruism, and that relatedness remains meaningful at all frequencies. This was shown to be the case by Hamilton (1964a) and is implicit in the regression definition of relatedness (Box 1.1). However, the point is most clearly made using Grafen's (1985) "geometric view of relatedness", as Box 1.3 explains in full. In brief, regression relatedness can be regarded as the extra probability, over and above the average "background" probability (set by the gene's mean frequency in the population), that kinship adds to two individuals' chances of sharing a focal gene. Therefore, relatedness remains a valid concept whether the background level is high or low. From this follows the conclusion that kin selection can operate at all frequencies of a gene for a social action (Box 1.3).

BOX 1.3 THE GEOMETRIC VIEW OF RELATEDNESS

Grafen (1985) presented a formal proof of Hamilton's rule as a theorem in population genetics (Boxes 1.1, 1.2). This involved proving the identity between "Hamilton's relatedness" – the r term in Hamilton's rule – and "pedigree relatedness" – the concept of relatedness involving kinship. The geometric view of relatedness is Grafen's (1985) device for proving this identity, and hence the validity of Hamilton's rule. Both concepts of relatedness, as described below, also turn out to be strictly equivalent to regression relatedness (Box 1.1). This box explains Grafen's (1985) argument (see also Maynard Smith 1989 p. 172; Grafen 1991).

Hamilton's rule states that a gene for altruism spreads if $rb - c > 0$ (Box 1.2). (The present box refers specifically to a gene for altruism, but the proof applies to genes for social actions in general.) The relatedness term (r) can be viewed as the fractional, genetic valuation that the actor (altruist) places on one offspring of the recipient, compared to one offspring of its own (e.g. Hamilton 1972). For example, if r is 0.5, the actor would sacrifice one offspring to save at least two offspring of the recipient. So, to the actor, each offspring of the recipient is genetically worth 0.5 of one of its own. Generalizing, Hamilton's rule says that the actor values one offspring of the recipient at an "rth" of one of its own. This "r" is "Hamilton's r" and is the concept of relatedness needed for Hamilton's rule to work.

Hamilton's r can be represented geometrically by plotting on a one-dimensional scale the frequency of a gene for altruism in three classes of individual. The first of these is an actor. Let its gene frequency equal A and be set at $A = 1$. This is because the actor must have the gene for altruism by definition (i.e. $A > 0$), and for simplicity is assumed to be, say, a homozygous diploid (this does not affect the conclusions). The other two kinds of individual are a recipient (gene frequency = R), and a random member of the population. The gene frequency of the latter (μ), by definition, equals the average frequency in the population. No assumptions are made about the values of R and μ, except that $\mu < R < A$. Given this, λ is defined as the fraction of the distance between μ and A at which R lies, i.e. $\lambda = (R - \mu)/(A - \mu)$. When λ equals, say, 1/4, the plot is as shown in Figure 1.3.

Now imagine an individual at R which has, say, eight offspring. This will increase the frequency of the altruist gene, since R is higher than the gene's average frequency (μ), and the offspring of individuals at R will also contain the gene with frequency R. This increase can be decomposed into two elements: when $\lambda = 1/4$, an individual at R having eight offspring has the same positive effect on the altruism gene's frequency as if an individual at μ had six offspring and one at A had two offspring. To see why, consider Grafen's (1985) metaphor of the gene frequency scale as a lever

BOX 1.3 CONT.

with its fulcrum at μ. The turning moment is the same whether there are eight bricks at R, or six bricks at μ and two at A. For the six bricks at μ have no effect on the lever's tilt, being directly above the fulcrum. This leaves the equivalency between eight bricks at R and two bricks four times the distance from the fulcrum, at A (Figure 1.4).

Generalizing, an individual at R having N offspring when R is λ of the way from μ to A has the same positive effect on gene frequency as an individual at μ having $(1 - \lambda)N$ offspring, and one at A having the remaining λN offspring. For, as in the lever analogy, reproduction by the notional individual at μ has no effect on gene frequency, because it only involves adding to the population individuals with the population mean frequency. Therefore, production by an individual at R of N offspring increases gene frequency by the same amount as the production by an individual at A of λN offspring, because A is relatively $1/\lambda$ times higher than the average gene frequency than R.

Now set N to equal 1. It follows that if an individual at R has one offspring when it is λ of the way from μ to A, this raises gene frequency by as much as an individual at A having λ offspring. Therefore λ equals the fractional, genetic valuation that an individual at A places on the production of one offspring by an individual at R. So λ equals "Hamilton's r." Or, by the geometric view, relatedness equals $(R - \mu)/(A - \mu)$. In words, *relatedness equals the frequency of a focal gene in one individual (the potential recipient) minus the gene's population mean frequency, divided by the frequency of the gene in another individual (the potential actor) minus the gene's population mean frequency.*

It now needs to be shown that this is equivalent to the pedigree definition of relatedness. Consider a sexual diploid species with random breeding. What is "geometric" parent–offspring relatedness in this species? Say an individual bearing a focal gene with frequency A mates and has offspring. This individual's mate must, on average, have a gene frequency of μ, the population mean, because of the assumption of random breeding. The gene frequency of a typical offspring (call it R) will be the average of the parental frequencies, i.e. $(\mu + A)/2$. This is because in sexual diploids an offspring is genetically half its mother and half its father, since it receives an equal number of chromosomes from each parent. (Note that R will therefore lie between μ and A, matching the earlier assumption.) So geometric parent–offspring relatedness equals $(R - \mu)/(A - \mu) = [(\mu + A)/2 - \mu]/(A - \mu) = 0.5$. This is also the pedigree relatedness between parent and offspring in diploids. So geometric relatedness and pedigree relatedness are the same (Grafen 1985).

To recap, the geometric view of relatedness proves the identity of the relatedness term in Hamilton's rule and the relatedness deduced from

BOX 1.3 CONT.

pedigrees. Furthermore, it is the concept of relatedness that makes Hamilton's rule work, in that a potential altruist should be selected to value a potential beneficiary's reproduction according to their geometric relatedness. So the geometric view also provides a proof of Hamilton's rule.

On top of this, regression relatedness (Box 1.1) and geometric relatedness are the same, as Grafen (1985) formally demonstrated. One way to see this equivalence is to recall from Box 1.1 that regression relatedness also refers to a deviation from an average gene frequency. In addition, Box 1.1 showed that regression relatedness is the same as pedigree relatedness (and hence geometric relatedness). For regression relatedness is obtained by regressing gene frequencies across groups of potential interactants. So if these groups are families, it measures genetic similarity due to kinship. Lastly, regression relatedness equals the genetic valuation an actor places on a recipient's reproduction (and so again is the same as geometric relatedness). To understand this, remember that regression relatedness can be defined as the average proportion of genes at a locus in the recipient that is identical with any allele at the locus in the actor (Grafen 1986; Box 1.1). When they have young, both recipient and actor will pass on a random allele at a locus to each offspring. Therefore, regression relatedness must give the actor's valuation of the recipient as an offspring producer relative to the actor's valuation of itself as an offspring producer (Hamilton 1972; Grafen 1986).

Summing up, regression relatedness, pedigree relatedness, and the concept of relatedness as a genetic exchange rate are all the same thing, and are equivalent to geometric relatedness. A valuable feature of the geometric view of relatedness is to bring these connections to light.

Finally, the geometric view of relatedness shows that Hamilton's rule applies at all frequencies of a gene for altruism (Section 1.6). In deriving the formula for geometric relatedness, no assumption was made about the value of μ, the gene's mean population frequency, other than $\mu < R < A$. Imagine that relatedness equals 0.5, and that μ can freely slide up and down the gene frequency scale from 0 to 1. Then, even if μ is very high (close to A at 1), relatedness can still be represented as half of the (short) way from μ to A. Therefore, it remains a meaningful concept at high gene frequencies. Put more formally, the expression $(R - \mu)/(A - \mu)$ has a positive value for all values of μ provided that $0 < \mu < R < A \leq 1$. So, at all frequencies of a gene for altruism, relatedness guarantees an above-average chance of gene-sharing between potential interactants. Consequently, at high gene frequencies, altruists should still value the reproduction of relatives more highly than that of random members of the population.

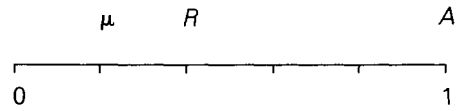


Figure 1.3 A gene frequency plot when the gene frequency in a recipient (*R*) lies a quarter of the way between the average gene frequency (μ) and the gene frequency in an actor (*A*, set at 1). (From Grafen 1985; by permission of Oxford University Press)

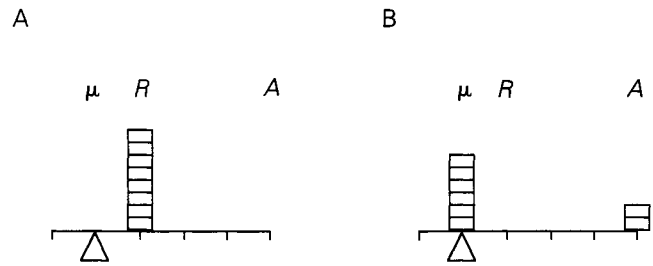


Figure 1.4 A diagram of Grafen's (1985, 1991) metaphor of gene frequency change as a system of weights on a lever. *R* lies a quarter of the way from the fulcrum (μ) to *A*. So placing eight bricks at *R* (Figure 1.4A) has the same effect as placing two bricks at *A* and six at the fulcrum (Figure 1.4B). (From Grafen 1991; by permission of Blackwell Scientific Publications Ltd)

1.7 Gene Expression in Kin Selection Theory

This section continues to explore facets of kin selection by considering the expression (penetrance) of genes for altruism under the theory. The essential point is that carriers of a kin-selected gene for altruism must include *both* helper phenotypes, such as workers, *and* reproducer phenotypes, such as queens. The reason is as follows. Hamilton's rule, as it applies to altruism, is about the spread through a population of a gene causing altruistic helping behavior in the gene bearers. But no gene can spread at all if no carriers of the gene ever have offspring. Therefore, if kin selection is to operate, some bearers of the gene for altruism must be reproducers. In other words, bearers must include both helpers and reproducers. This could come about in various ways. For example, the gene could have facultative expression (some adult bearers help, and others reproduce), or it could have obligate expression but be expressed at different life stages (for example, young bearers help and old ones reproduce). This point has been made by several previous authors (e.g. Orlove 1975; Charlesworth 1978; Crozier 1979; Dawkins 1979; Grafen 1984, 1985; Hamilton 1987a; Parker 1989).

To put it another way, imagine a gene for altruism with obligate expression such that all carriers were always helpers and none were

ever reproducers. Then if bearers helped reproductive individuals that they met, they would be directing their aid towards nonbearers, so such a gene could never spread. Kin selection works because bearers of the gene for altruism help reproducers who are also bearers of the gene (with above-average frequency). Such reproducers must therefore bear the gene but not express it, for example because the gene is expressed facultatively.

This reasoning is present in the scenario for the origin of eusociality in ants proposed in Section 3.3. Here one of its implications needs highlighting. If a facultative helping–reproducing gene spreads through a population of solitary nesters to fixation, the result is a population that is entirely social but in which the helper–reproducer dichotomy (worker–queen dichotomy) is nongenetic. In other words, kin selection reasoning implies that sexual-worker caste determination in eusocial insects should be nongenetic (Orlove 1975; Crozier 1979, 1992). This is indeed the case throughout the termites and the social Hymenoptera (e.g. Craig and Crozier 1978), with the possible exceptions of the stingless bees (Michener 1974) and two ant species (Winter and Buschinger 1986; Heinze and Buschinger 1989), where genetic control of caste is presumably secondary (Crozier 1977, 1979). In all other eusocial insects the factors affecting queen–worker caste determination are numerous and complex (Watson et al. 1985; Hölldobler and Wilson 1990), but, as far as is known, exclusively environmental.

These arguments reflect the assumption in kin selection theory that potential actors and recipients do not differ in their average gene frequencies (Box 1.1). They also rebut West-Eberhard's (1988) assertion that nongenetic caste determination in social insects is a contradiction of "allelic" versions of kin selection theory (see also Crozier 1992; West-Eberhard 1992). Lastly, they show that West-Eberhard's (1987a,b, 1988) "epigenetic" theory of the evolution of insect sociality is not unique in invoking the facultative nature of worker behavior. This last point is further discussed in Section 3.4.

1.8 The Gene for Altruism and the Interests of the Rest of the Genome

As has been emphasized, kin selection theory is an explicitly gene-level theory. It concerns the fate of genes for social actions – altruism in the present context. This raises the following question. If a gene for altruism is being positively selected, how should selection act on all the other genes in the genome of the altruist? A gene-level perspective cannot automatically assume that these genes and the altruism gene share the

same interests. So it needs explaining why in fact each individual altruist usually acts as a unit.

There is a reason why there is generally no conflict of interest within individuals between a gene for altruism and other genes. This is that, on average, the relatedness values between individuals at all loci on chromosomes sharing the same inheritance system are equal (Dawkins 1982a p. 149; Grafen 1984, 1985; Trivers 1985 p. 128). For example, in sexual diploids with chromosomal sex determination, all the chromosomes apart from the sex chromosomes (the autosomes) share a common inheritance system. Therefore, on average, the relatedness between two individuals is equal for all loci on the autosomes. To see why, imagine the chain of pedigree joining two individuals. The reason the probability of sharing genes changes at any link is meiosis. Further, at any meiosis, the probability of gene-sharing between parent and offspring is the same for all autosomal loci – one half. Since all loci in an individual are connected in the pedigree chain to all corresponding loci in another individual by the same number of links with the same probability of gene-sharing at each link, average relatedness between all loci must be equal.

This reasoning means that if at an autosomal locus for altruism the relatedness between the altruist and recipient is, say, 0.25, then relatedness at every other autosomal locus is also 0.25, on average. So if Hamilton's condition ($rb - c > 0$) is fulfilled for the gene for altruism, it is also fulfilled at every other locus. Therefore, complete agreement exists over the performance of the altruistic behavior between the rest of the autosomal genome and the gene for altruism. Put another way, when selection favors the altruism gene, all the other autosomal genes are indirect beneficiaries, and to the same extent, because all have the same chance of being present in the recipient. An altruist should therefore behave as a unit, as is observed. Of course, individuals also behave as units in most behavior, and the reason is the same. All genes on chromosomes having the same inheritance system share an equal probability (0.5 for autosomes) of being present in an individual's gametes, and hence in its offspring, because of a fair meiosis (Dawkins 1982a p. 135). Therefore, gene selectionism predicts that all autosomal genes should cooperate fully for the survival and reproduction of the body they inhabit, as long as meiosis remains fair (Section 2.5). (For simplicity, this argument ignores the possibility of genomic imprinting [Box 4.2].)

The condition that only genes on the same kind of chromosome share interests becomes important when exceptions are considered. Say a gene for altruism is on the Y chromosome of a species with human-style genetics (a diploid with chromosomal sex determination in which XX individuals are females, XY ones males, with no crossing-over between

the X and Y chromosomes). Then the relatedness between brothers at the altruism locus would be $r = 1.0$ (because each brother inherits the same Y chromosome from the father), but at all the autosomal loci relatedness would be the normal value for diploid sibs of 0.5. Therefore, the gene for altruism would be selected to make bearers more helpful to brothers than would be favored by selection on autosomal genes. For example, if the gene were for suicide to save brothers' lives, it would be selected if it caused its carrier to die on behalf of one or more brothers. But the autosomal genes would require two or more brothers to be saved, and dying for only one would cause more autosomal genes to be lost (1 unit) than gained (0.5 units). Mutant genes ("modifiers") at autosomal loci that rendered the "outlaw" altruism gene ineffective would therefore be selected (Alexander and Borgia 1978; Dawkins 1982a).

These considerations are not simply speculation. Selection for autosomal modifiers may account for the characteristic small size and inertness of the Y chromosome. This was suggested by Alexander and Borgia (1978) and Trivers (1985 p. 136), following Hamilton's argument attributing Y inertness to modifiers for suppressing Y meiotic-drive genes (Hamilton 1967; Leigh 1977; Cosmides and Tooby 1981; Dawkins 1982a p. 140). Whether these particular arguments are correct, the general point is that if relatedness between individuals differs systematically among loci, within-genome conflict is expected (Cosmides and Tooby 1981; Dawkins 1982a; Grafen 1991; Hurst et al. 1992). So the cooperation of genes for altruism and other genes cannot always be taken for granted. On the other hand, since autosomes outnumber sex chromosomes, genes will usually be selected to cooperate in social behavior, conferring the individual's characteristic unity of purpose. Therefore gene selectionism does not deny the integrity of individuals. But it does show that it flows from gene selectionist principles themselves, and according to these same principles can break down. So the integrity of individuals cannot be regarded as a given fact of nature (Dawkins 1982a, 1990; Hamilton 1987a; Section 2.5).

A final point is that kinship is about the only phenomenon in nature likely to bring about uniform average relatednesses between individuals over all loci in the genome (Grafen 1985). For example, say individuals shared a genetic preference for a particular habitat (e.g. Hamilton 1975a). Then those meeting at the habitat would be related at the locus for habitat preference, but not at their other loci, since they would not be kin. So altruism between individuals within the habitat could not be achieved without provoking within-genome conflict, despite the interacting individuals having greater than average genetic similarity. This is why relatedness due to kinship is by far the most plausible means of

achieving the genetic similarity represented by the relatedness term in Hamilton's rule (Dawkins 1982a; Grafen 1985).

1.9 Parental Manipulation Theory

Alexander (1974) proposed a parental manipulation hypothesis for the evolution of altruism in social insects. This suggests that workers help raise the queen's young because the queen manipulates them into doing so. For example, she may underfeed them (making them poor potential foundresses) or dominate them and so prevent their reproduction directly (Alexander 1974; Michener and Brothers 1974). This theory has strong appeal because social insect queens do appear to hinder worker reproduction (e.g. Brian 1980; Fletcher and Ross 1985; Bourke 1988a; Section 7.4).

Parental manipulation theory has frequently been presented as an alternative to kin selection as an explanation for the evolution of eusociality. Alexander (1974) himself maintained that parental manipulation was more powerful than kin selection alone because parents had an intrinsic, genetic advantage in conflicts of interest with offspring. His argument was that any gene in an offspring causing it to rebel against the parent would disadvantage the offspring when it became a parent itself, since the gene would be present in its own offspring. So only lineages with pliable offspring would persist, and offspring altruism could be best explained by parental manipulation.

However, this argument contained a flaw, pointed out by Dawkins (1976 pp. 145–148), which is that it works equally in reverse. The reversed argument is that a gene causing an adult to act against the interests of an offspring will be counter-selected when it is present in juveniles, through these juveniles having a parent bearing the gene. This suggests that offspring should always win in parent–offspring conflicts, the opposite of Alexander's conclusion. In reality, there is no inbuilt genetic advantage to either party, because there is no genetic asymmetry between them (parent–offspring relatedness equals offspring–parent relatedness) (Dawkins 1976). This point has since been conceded by Alexander (1979 pp. 38–39). Population genetics models have also shown that genes which make offspring behave selfishly at the parents' expense can spread (Blick 1977; Parker and Macnair 1978; Stamps et al. 1978). The basic reason is that, although a gene for offspring selfishness reduces litter size (parental fitness), it may be disproportionately frequent within the litter (Godfray and Parker 1992). So the status of parental manipulation as an independent evolutionary principle has been undermined. In general, models suggest that the out-

come of parent–offspring conflicts (which party wins, or whether a compromise results) varies with the costs to offspring and parents of, respectively, behaving selfishly and opposing the offsprings' demands (e.g. Trivers 1974; Blick 1977; Macnair and Parker 1978, 1979; Parker and Macnair 1978, 1979; Stamps et al. 1978; Harpending 1979; Parker 1985; reviewed by Clutton-Brock 1991) (Section 7.2).

Parental manipulation in the form of queen hostility to worker reproduction, or queen underfeeding of female larvae, is certainly feasible in social Hymenoptera. But it should not be considered to lie outside kin selection theory. First, as pointed out by several authors (e.g. West-Eberhard 1975, 1981; Maynard Smith 1982a; Michod 1982; Vehrencamp 1983a; Andersson 1984; Sudd and Franks 1987 p. 7), parental manipulation by definition involves social actions performed among kin, and so falls within the theory. Second, if parental manipulation occurs, offspring will not necessarily accept it passively. They may be selected to resist. The strength of this selection will then depend on the terms of kin selection theory, namely benefit, cost, and relatedness (Trivers 1974; Charlesworth 1978; Charnov 1978a; Craig 1979; Crozier 1979, 1982; Metcalf 1980a). The possibility of offspring counter-manipulation is particularly important given that there is no general (genetic) reason to assume parental victory. In fact, parental manipulation is best seen as part of kin conflict theory (Trivers 1974; Trivers and Hare 1976; Yamamura and Higashi 1992), the branch of kin selection theory dealing with evolutionary conflicts of interest between relatives. In this context, parental manipulation may well have been important in insect social evolution (Sections 3.9, 7.4).

1.10 Conclusion

Kin selection theory is not just a fashion in evolutionary biology, as it is sometimes perceived to be. Kin selection is a logical consequence of natural selection (Dawkins 1979; Ridley and Dawkins 1981). This is because kin selection follows from considering how natural selection will act on genes for social actions directed at co-bearers of these genes. In fact kin selection arguably includes conventional natural selection, since it is hard to see how a system of natural selection could exist without the appearance of the feedback phenomena implicit in the idea, from kin selection, that genes can influence their own spread via effects on copies of themselves in other individuals. The main purpose of this chapter has been to argue for the fundamental importance of kin selection theory.

A consequence of viewing kin selection as a logical corollary of nat-

ural selection theory is the conclusion that it is a deep-level theory, rather than the "local" hypothesis it is sometimes treated as. Deep-level theories are both more powerful, and harder to prove or disprove, than more superficial ones. This does not mean that kin selection is unfalsifiable (universal, unreciprocated altruism towards nonrelatives would falsify it). The point is that many ideas which previously have been regarded as inimical to kin selection theory can instead be incorporated into its deep-level gene-selectionist logic. This chapter has argued that this is the case for parental manipulation theory. The next one extends the argument to group selection, colony-level selection, and levels-of-selection theory.

1.11 Summary

1. Darwin's theory of natural selection forms the basis of the evolutionary study of animal behavior and sociality. The fundamental element of natural selection theory, the "unit of self-interest," is Dawkins' (1976, 1982a) replicator. Replicators make copies of themselves, head potentially immortal lineages, faithfully transmit mutations, and store information. Adaptation arises from the cumulative selection of favorable traits in replicators or mutually stable sets of replicators. Adaptations are therefore for the replicators' benefit. Genes are replicators. Genes inhabit "vehicles" such as bodies and groups over which they have causal priority. Adaptations therefore primarily benefit genes, and only benefit bodies and groups incidentally.

2. The eusocial insects (those with a reproductive division of labor) exhibit biological altruism, which involves one individual reducing its survival or offspring production and increasing those of a neighbor. Altruism poses a problem for individual-centered natural selection theory, which predicts that individuals should maximize their offspring number. This problem was solved by Hamilton's theory of kin selection, which is a replicator (gene)-centered theory.

3. Kin selection is the natural selection of genes for social actions (genes affecting the offspring output of their bearers and their bearers' neighbors) as a result of the sharing of genes among relatives. Kin selection theory states that a gene for altruism spreads if the condition, (relatedness) $\times$ (benefit) $-$ (cost) > 0, is met (Hamilton's rule). Here, benefit means the number of extra offspring gained by the beneficiary and cost means the number of offspring lost by the altruist. Relatedness is formally defined as the regression coefficient obtained when the gene frequency among potential recipients of a social action is regressed across groups of interactants against the gene frequency among the

potential actors. Informally, relatedness is the probability of gene sharing between individuals independently of the average probability, which is set by the gene's average frequency in the population. Therefore, according to kin selection theory, altruism can evolve because a gene for kin altruism promotes care to individuals with an above-average probability of sharing the same gene, through relatedness. That is, the gene promotes care for copies of itself.

4. Hamilton's rule implies that both genetic and ecological factors are important in explaining the evolution of altruism and eusociality. Hamilton's rule can also be expressed as the statement that a gene for altruism spreads if bearers have above-average inclusive fitness. A focal individual's inclusive fitness is its personal fitness (offspring number) in the absence of the average social effects of conspecifics, plus the individual's influence on neighbors' fitnesses devalued by its relatedness with neighbors.

5. Kin selection can operate at high frequencies of a gene for altruism because kinship always guarantees above-average levels of gene-sharing, wherever the average lies. This principle follows from Grafen's (1985) "geometric view of relatedness," which both demonstrates the validity of Hamilton's rule and emphasizes that relatedness is measured relative to a gene's mean frequency in a population. Geometric relatedness and the regression definition of relatedness are formally equivalent. Kin selection also requires that bearers of genes for altruism include both altruists and reproducers, and is therefore consistent with nongenetic caste determination in eusocial insects.

6. Genes for altruism and other genes within individuals are selected to cooperate if they lie on chromosomes with the same inheritance system, because this means that the average relatedness between all corresponding loci on these chromosomes in different individuals is equal. Kinship is about the only agent producing uniform average genetic similarity across all loci. The theory that altruism among offspring arises from parental manipulation is not an alternative to kin selection theory, but part of it, namely kin conflict theory.

7. Kin selection is a fundamental theory in evolutionary biology, and inseparable from natural selection theory. It is applicable whenever conspecific individuals interact in ways that affect offspring output and are nonrandom with respect to relatedness. In principle, it applies to all living things. Many other selectionist principles can be assimilated by its deep-seated logic.

2 Levels-of-selection Theory, Gene Selectionism, and Insect Societies

2.1 Introduction

The previous chapter discussed natural selection, kin selection, and the evolution of altruism in terms of gene selectionism. However, gene selectionist language is not universal in the scientific literature on social insects. For example, many authors invoke "colony-level selection" as a competing mode of evolution. This chapter examines whether colony-level selection is a legitimate concept, and whether it differs substantively from gene selection. To anticipate, our argument will be that there is no fundamental clash between gene and colony-level selection, with certain important qualifications.

Sections 2.2 and 2.3 reach this conclusion by considering the relation between colony-level, group, kin, and gene selection. Next, Section 2.4 puts the discussion in the wider context of "levels-of-selection" theory. Section 2.5 examines whether conflicts of interest in insect societies and other groupings can be analyzed with a single, shared set of principles. Lastly, Section 2.6 relates the chapter's findings to the idea, closely connected with colony-level selection, that insect societies are "superorganisms." The overall aim is to argue that recognizing ties between these concepts avoids unnecessary controversy over which is correct. Instead, they may represent correct, but different, ways of viewing gene selectionist natural selection theory. Similar conclusions, especially in the kin versus group selection controversy, have recently been reached by other authors (e.g. Wilson and Sober 1989; Ross and Carpenter 1991a; Queller 1992a,b; Ratnieks and Reeve 1992; Dugatkin and Reeve 1994).

2.2 Colony-level, Group, Kin, and Gene Selection

The concept of colony-level selection occurs in the social insect literature in both empirical and theoretical contexts (e.g. Darwin 1859; Sturtevant 1938; Wynne-Edwards 1962; Michener 1964; E.O. Wilson

1966, 1968, 1985a, 1990; Lin and Michener 1972; Crozier 1977; Oster and Wilson 1978; West-Eberhard 1981; Myles and Nutting 1988; Hillesheim et al. 1989; Page et al. 1989a,b; Robinson and Page 1989; Hölldobler and Wilson 1990 p. 336; Frumhoff and Ward 1992; Moritz and Southwick 1992). In addition, genetic (allele frequency) models of colony-level selection have been presented by, for example, Williams and Williams (1957), Crozier and Consul (1976), Owen (1986, 1989), and Moritz (1989). But colony-level selection appears to have several meanings. For example, it is sometimes equated with selection on queens (E.O. Wilson 1966; Oster and Wilson 1978 p. 98), or with the differential survival, multiplication, and extinction of whole colonies (West-Eberhard 1981). Alternatively, it is a force limiting the spread of selfishness (e.g. Sturtevant 1938; Hillesheim et al. 1989), or selection for features contributing to colony productivity (E.O. Wilson 1968), especially when these are properties expressed by whole colonies such as caste frequency distributions (Oster and Wilson 1978; Robinson and Page 1989; Frumhoff and Ward 1992). Owen (1989) explicitly defined colony-level selection as selection on the genotypes of the founding pair of the colony, acting via colony features determined by the genotypes of their worker offspring. Many authors who invoke colony-level selection also recognize the possibility of within-colony or "individual-level" selection. This is held to arise from the presence of multiple queens (Sturtevant 1938), reproductive workers (West-Eberhard 1981; Hillesheim et al. 1989; Frumhoff and Ward 1992; Tsuji 1994), nepotism in sexual rearing by workers (Page et al. 1989a), or more generally from genetic conflicts of interest within colonies (e.g. Leigh 1991; Ratnieks and Reeve 1992).

This and the following sections consider the validity of colony-level selection. The starting point is that colony-level selection is a special case of group selection, since a colony is a group of individuals. So much of this section examines the legitimacy of group selection. It does this in the context of the evolution of altruism, since this is the key theoretical issue. In addition, group selection's claim to explain altruism is the major source of controversy. This discussion concludes that a form of group selection (and hence colony-level selection) exists that is compatible with kin selection. Section 2.3 then shows how previous usages of colony-level selection, despite their superficial differences, fall within the framework that will be set out.

To begin, then, with group selection. This means entering the minefield of the group selection controversy, which has preoccupied evolutionary biologists for the past thirty years. For a comprehensive survey, see the reviews of Wynne-Edwards (1962, 1986, 1993), G.C. Williams (1966, 1971, 1992), Lewontin (1970), E.O. Wilson (1975), Dawkins