A geometric view of relatedness

ALAN GRAFEN

1. Introduction

The first objective of this paper is to introduce a geometric view of relatedness, as it is relevant to the evolution of social behaviour in the way pioneered by Hamilton (1963, 1964). The geometric view will suggest a new definition of relatedness. The exposition of this view will cover a number of facts about relatedness that have hitherto been available only to the mathematically competent (or at least adventurous) reader. The second objective follows on naturally from the first. It is to consider the status of Hamilton's rule as an evolutionary principle. There has been a steady trickle of interesting population genetical theory about the validity of Hamilton's rule and this will be briefly reviewed later. My main point will be that so far as the likely evolution of a character is concerned, this increasing body of theory confirms Hamilton's rule in a broad range of circumstances. The cases where Hamilton's rule does not apply will be explained with reference to the geometric view of relatedness.

In pursuit of these two aims, a number of other matters will come to our attention. An increasingly popular approach to population genetics, the covariance selection mathematics of Price (1970, 1972), will be explained and used in a new derivation of Hamilton's rule. A rough computation will be made of the frequency of unrelated individuals that happen to be as genetically similar to an animal as are its relatives of known degree. This is relevant to the suggestion of Hamilton (1971) and others that individuals may detect genetic similarity directly. We will also encounter, and eventually confront and solve, the 'paradox of inbreeding' of Seger (1981). The resolution will lead us to disagree with Jacquard (1974, p. 171), who suggested that relatedness is a measure of our information and not of anything real. If this relativism were true, then Hamilton's rule would be meaningless.

After that brief summary of my intentions, I wish now to give a fuller introduction to the paper. The readers for whom this introduction is intended have met the concept of relatedness and Hamilton's rule, and find them so unproblematic that they are surprised that any clarification, defence, or exposition is necessary. A fair sized literature, to which reference will be made later, deals with relatedness and Hamilton's rule, and its very existence is a good indication that there are problems with these ideas. However, this literature is mainly mathematical and I aim now to persuade the confident reader, using words only, that clarification, defence, and exposition are, after all, necessary for Hamilton's rule and the concept of relatedness. I will then give the plan of the rest of the paper, section by section.

Firstly, let us take the question whether relatedness is such an easy idea. A first attempt at showing that full, diploid sibs are related by one-half is to say that they share half their genes and that they do so because each gene in their parents has a 50 per cent chance of ending up in each sibling independently. However, we all share most of our genes, for, at least with the technology available to us a few years ago, the homozygosity in human populations was about 80 per cent (Cavalli-Sforza and Bodmer 1971). This means that two alleles chosen at random from the same locus from random human beings have an 80 per cent chance of being the same. If we really meant what we said about relatedness, we would conclude that 'unrelated' individuals are, in fact, closely related, with a relatedness of 0.8. The next step in the argument (Dawkins 1979) is to say that when we said 'share half their genes', we meant not only that the genes had to be identical, but also that they had to be identical by descent (Malécot 1969). If their parents are unrelated, then all identity by descent between the siblings must be by descent from their parents and our conclusion that siblings are related by one-half is vindicated.

Before proceeding, I should assure the reader that I do not doubt that the sibs are related by a half, but I am anxious that the argument that gets us there should be logically impeccable. When we ask if this idea of identity by descent can be taken seriously for our present purposes, we run straight into what Seger (1981) discussed under the heading 'The paradox of inbreeding'. The paradox is seen by first supposing that in the evolution of a particular locus, every mutation gives rise to a new allele. It then follows that any two identical alleles in the population now are descendants of the same, original, newly mutated allele. Therefore, any two identical alleles are identical by descent. (This conclusion applies in a weaker, but still vicious form if mutant alleles are not all unique.) We have come full circle and are faced again with knowing that sibs are related by one-half, but not being able to say why.

This problem with the meaning of relatedness is a crucially important point in trying to prove that Hamilton's rule is correct. (Hamilton's rule states that selection will favour an action by one animal that causes a loss to itself of c offspring, and a gain of b offspring to another animal to which it is related by r, provided $rb-c>0$.) Two approaches have been taken. One is to show that Hamilton's rule works for specific kinds of relatives. The other is to prove that Hamilton's rule works for any kind of relative, using some mathematical definition of relatedness. The first method leads to greater mathematical respectability, while the second is biologically more general.

The mathematical definitions used in the second method do not refer to kin connections at all, but only to the fraction of genes shared by interactants. It follows that these definitions do not in themselves run up against the paradox of inbreeding, but the paradox will not go away. If we wish to apply Hamilton's rule using relatednesses derived from our knowledge of common ancestry, then we need to be able to make the connection between common ancestry and the relatedness defined by the fraction of genes interactants share, and there the paradox creeps in.

I hope that I have explained why relatedness is a problematic concept. The confident reader may also be surprised that mathematical models are built to try to prove or disprove Hamilton's rule. Its truth may seem sufficiently obvious, but again this is not so. In the simplest, central case, there is little doubt that the rule does apply. Equally, there are now many cases known where there is no doubt that Hamilton's rule does not apply. Charlesworth (1978) gave one good example in which Hamilton's rule fails, which I shall recount briefly. Nestlings that are full sibs have the opportunity to commit suicide to increase the survival of their fellow nestlings. What is the condition for a dominant allele to spread that causes nestlings to take this opportunity? How large must the increase in survival of their fellow nestlings be to justify this sacrifice?

The answer is that this allele can never spread. All the bearers of the altruistic allele commit suicide. It follows that there will be no copies of the altruistic allele next generation. The point of this example is that a naive application of Hamilton's rule would give the wrong answer. We need principles that tell us when Hamilton's rule will fail, as it does here. That is the point of trying to prove Hamilton's rule. The interest is focussed on what assumptions we need to make to prove it. A general proof with few assumptions makes Hamilton's rule a general rule; if only proofs with many special assumptions can be found, this means Hamilton's rule is of limited applicability. A biologist who applies Hamilton's rule in the field should be most worried about its scope of validity, for only if it is true for all sorts of genetic systems can he hope that it applies to the usually unknown genetic system that governs the character he is studying.

Relatedness, then, is a concept that needs explaining and Hamilton's rule needs proving. The second method of modelling Hamilton's rule mentioned above consists of finding the right concept of relatedness to make Hamilton's rule work and that is what I shall do here.

Section 2 contains the new derivation of Hamilton's rule and, on the way, an exposition of the covariance selection mathematics of Price (1970, 1972). It concludes with a new definition of relatedness, which is the main point of the section. Section 3 is, by contrast, not at all mathematical and explains the geometric interpretation of the new definition of relatedness. It also explains how this definition of relatedness is connected to ordinary notions of kinship and why it is the right definition to make Hamilton's rule work. This is the section that contains the first main message of the paper and readers who are positively afraid of equations should start with it. Other sections can be viewed as showing that different parts of this message are true. Section 2 proves that the new definition makes Hamilton's rule work. Section 4 proves that the geometric definition of section 3 is the same as the algebraic one of section 2. Section 5 proves that the geometric definition of section 3 really is connected to ordinary notions of kinship under certain assumptions, which it goes on to spell out and explain.

The last three sections are almost free of mathematics. Section 6 contains the second main message of the paper, that Hamilton's rule is a good evolutionary principle. It does this by arguing that the assumptions of

section 5, while fairly restrictive for the application of Hamilton's rule to population genetics questions such as the rate of spread of particular alleles, are much less restrictive for the application of Hamilton's rule to broader evolutionary questions, such as the likely outcome of selection on a particular character. In the seventh section, I survey previous papers on Hamilton's rule and relatedness, and section 8 contains brief conclusions.

I have tried to make the paper intelligible to those to whom equations appear as a blur and to the extent that I have failed I apologize. As is often the case, the important conclusions and arguments are not mathematical, but a few essential points are. I hope that, provided these few essential points are taken on trust, the non-mathematically inclined reader can follow the important arguments and understand the important conclusions.

2. A new derivation of Hamilton's rule

This section contains a new derivation of Hamilton's rule, using the covariance selection mathematics of Price (1970, 1972). Price's method is also explained in some detail. Some previous derivations have used Price's method (Hamilton 1970, 1975; Seger 1981; Uyenoyama *et al.* 1981) to varying degrees. Applications of Price's method will be reviewed later in this section. Here I give a derivation that allows each individual to have its own ploidy, and whose formulation covers both single allele and multi-allele, and single-locus and multi-locus models. These extensions are natural within Price's method and their value is that they unify in one treatment results that would otherwise have to be proved separately. For the purposes of this paper, they mean that our definition of relatedness will be appropriate for arbitrary ploidies and any number of alleles. Other derivations will be discussed in section 7, when ideas to be developed later will allow more fruitful comments to be made.

What exactly, then, is the rule we are setting out to derive? It is a rule that tells us whether natural selection will favour any social action, where by a social action we mean an action that has consequences for the number of offspring of the animal performing it and also for the number of offspring of some other animal of the same species. According to the rule, we need to know only three numbers to decide if selection will favour a social action. Firstly, the effect of the action on the actor's number of offspring; because of the special interest in altruistic actions, this is conventionally measured as the decrease in the actor's number of offspring, is called a cost and denoted c. Secondly, the effect of the action on the recipient's number of offspring, conventionally measured as an increase, is called a benefit and denoted b. Thirdly, a quantity known as the relatedness of the actor to the recipient, denoted r. Hamilton's rule states that the social action is favoured by natural selection if

$$rb - c > 0. \tag{1}$$

The rule was first derived by Hamilton (1963, 1964) and it has a very

simple biological interpretation. The actor values one offspring of the recipient as a certain fraction of one offspring of its own. That fraction is the relatedness. The remarkable property of eqn (1) is its simplicity as a summary of, or prediction about, the results of quite complicated population genetics models. The dominance of the alleles involved is not mentioned in eqn (1), nor are the number of loci and the ploidies of actor and recipient. In many ways it looks too good to be true. Our aim in deriving Hamilton's rule, when we are not quite sure how relatedness is to be defined, is to find a condition that tells us whether a social action is favoured by natural selection, then to see if we can choose a definition of r to make this condition equivalent to eqn (1). This definition is the one we will adopt. Of course, we must then examine its properties to see what resemblance it bears to our notions of kinship and common ancestry.

This is a good place to introduce a distinction, made by Crozier (1970), between relatedness and relationship. We may explain r in eqn (1) in two ways. The first is to say that it measures the genetic similarity between donor and recipient, the extent to which they possess the same genes (Crozier's 'relatedness'). The second is to say that r is a measure of common ancestry and can be computed from a family tree (Crozier's 'relationship'). Now genetic similarity has many possible causes, of which common ancestry is only one. Genetic similarity makes Hamilton's rule work, but it is common ancestry that we are likely to know when we wish to apply it. For this and other reasons we shall come across in due course, Hamilton's rule is most useful when the genetic similarity is caused solely by common ancestry. In section 6, we shall see that common ancestry causes genetic similarity of a very special kind. The definition of r we uncover later in this section will be a measure of genetic similarity. Its connection with common ancestry is the topic of section 5. While the substance of the distinction is very important, it is confusing to use such similar words for concepts to be contrasted. In this paper, I have used the word relatedness for both concepts where confusion is unlikely, and explicitly refer to genetic similarity or common ancestry where necessary.

In the next subsection, Price's method is developed and explained and its uses reviewed. The following subsection uses Price's equation to derive Hamilton's rule.

PRICE'S COVARIANCE SELECTION MATHEMATICS

Now we turn to Price's method, which is a pleasing way of doing population genetics (Price 1970, 1972). I give here an account of one fairly general use of Price's method which I have found useful. Other accounts of aspects of Price's method are given by Hamilton (1975), Seger (1981), and Wade (1985). Assume discrete generations. We begin by taking all the individuals in one generation and indexing them, that is to say, giving each of them a number rather as houses in a street are given numbers for ease of reference. We then want to measure four things about each individual, considered as a potential parent. They will be four numbers and every

potential parent will have its own set of these four numbers. For the ith individual, they will be denoted:

l_i	the ith individual's ploidy;
w_i	the number of successful gametes of the ith individual, per haploid set;
p_i	the ith individual's 'p-score' (see below for definition);
$p_i + \triangle p_i$	the average p-score of the ith individual's successful gametes.

Let's take these in turn. An individual's ploidy is the number of haploid sets of chromosomes there are in its genome: this is one for a haploid, such as most male hymenopterans and human gametes; two for a diploid, such as hymenopteran females and humans; three for a triploid, such as some plant endosperm, and so on. We will be very general in our approach and allow each individual to have any ploidy. The usual circumstance is that all males have one ploidy and all females have another. For problems concerning other genetic entities, such as gametes or plant endosperm, it is useful to have a general result.

The number of successful gametes per haploid set is just what it says. A successful gamete is one that contributes to an offspring in the next generation at whatever stage we decide to count them. The reason for dividing by the ploidy of the individual parent is its significance in the theory. We want to know the average success of a haploid set. The sole parent of a haploid with two offspring is better represented genetically in the second generation than is one of the two parents of a diploid with three offspring.

The third and fourth numbers, p_i and $p_i + \triangle p_i$, lie at the heart of Price's method, and to explain them I must explain what I mean by a p-score. The p-score can be anything that an offspring inherits by averaging together the gametic contributions of the parents. The simplest example is where the p-score of an entity (individual or gamete, or group of individuals or gametes), is defined as the frequency within it of one particular allele. Thus, in a haploid gamete, the p-score is either 0 or 1. In a diploid individual, the p-score is either 0, 0.5, or 1. In a population of 50 diploids, the p-score can be any number out of 100. Let us check that this kind of p-score satisfies our requirements. If the p-score of the gametes that made you were both 0, then neither had the allele, so neither do you, so your p-score is 0. If one gametic p-score was 0, and the other was 1, then you are a heterozygote and your p-score is 0.5. If both p-scores were 1, then you have two copies of the allele and your p-score is also 1. In every case, your p-score is the average of the p-scores of the gametes that made you.

A more interesting choice for a p-score involves all the alleles at one locus. We can give every allele at this locus a separate number, any number we choose. Then the average of the numbers assigned to the alleles an entity possesses is a p-score. For example, suppose we have three alleles, A, B, and C, and we assign them the values 1, 3, and 11, respectively. A B gamete has a score of 3, an AC diploid has a score of 6, and an $AABC$

tetraploid would have a score of 4. The score of an entity is the average of the scores of the gametes that made it and so this score qualifies as a p-score.

One final extension will complete our range of p-scores. The genes in the previous example were alleles at the same locus. This was an unnecessary assumption, because even if they were genes at different loci, the defining property of a p-score would have been satisfied. Thus, the most general kind of p-score for a haploid entity is the sum of arbitrary values attached to any number of alleles at any number of loci. For other entities, it is the average p-score of the haploid sets that make it up.

Our derivation will proceed by following the evolutionary change in a p-score; because we will not specify which p-score, our conclusions will be true for any p-score. The range of p-scores for which Price's method works means that the same equations will serve for a two allele, single-locus model, when the p-score is interpreted as the frequency of one allele; for a polygenic model, in which the p-score is interpreted as the summation of the small allelic effects that combine to produce the polygenic character and, indeed, for a whole range of intermediate models. The additive genetic value of any character (Falconer 1981) can be represented as a p-score. All this is achieved at the same time as allowing each individual to have its own ploidy.

Having defined what a p-score is, the third and fourth numbers in the list above can now be explained. p_i is the p-score of the ith individual and the average p-score of its successful gametes is $p_i + \triangle p_i$. We write the p-score of the ith individual's successful gametes as $p_i + \triangle p_i$, because there is a presumption that an individual's gametes have the same gene frequencies as the individual has, and so $\triangle p_i$ will, on average, be 0. The randomness of meiosis will often make it non-zero in a particular case and meiotic drive (for an explanation see Wright 1968, 1969, 1977, 1978) would make it non-zero on average. Let us agree to ignore the effects of meiotic drive, and so assume where necessary that on average $\triangle p_i$ is 0.

There are two more notational devices to mention before we get down to work. The first is that a symbol without a subscript represents an average over all the haploid sets that make up the population. Thus, p is the average p-score of all the haploid sets in the parental population. Alternatively, we can think of it as the average p-score taken over individuals, with each individual weighted by its ploidy. The second device is that a prime ($'$) denotes the value among the offspring, so that p' represents the average p-score of all the haploid sets that make up the offspring. p' is also the p-score of the successful gametes of the parents and it is this equivalence that we now exploit.

We now go on to derive Price's basic equation. The number of successful gametes of the ith individual, and the sum of all the p-scores of those successful gametes, are

$$l_i w_i$$

and

$$l_i w_i(p_i + \triangle p_i).$$

So, using the $\sum$ notation to denote summing over i, that is adding up these values for all the individuals in the population, we obtain

$$\sum l_i w_i$$

and

$$\sum l_i w_i(p_i + \triangle p_i).$$

However, if these are the number of successful gametes and the summed scores, respectively, for all successful gametes, then the average p-score of the successful gametes must be given by

$$p' = \frac{\sum l_i w_i(p_i + \triangle p_i).}{\sum l_i w_i} \tag{2}$$

To produce Price's equation from this we need to use two statistical notions, the expectation and the covariance. The expectation is just an average, but I use the word to explain why I will use the symbol **E** to denote an average. It is an average of the sort described above, over haploid sets. The covariance of two quantities is the average product minus the product of the averages and will be denoted **Cov**. Putting these definitions into symbols gives us

$$p = \mathbf{E}p_i = \frac{\sum l_i p_i,}{\sum l_i}$$

$$w = \mathbf{E}w_i = \frac{\sum l_i w_i,}{\sum l_i}$$

$$\mathbf{E}(w_i \triangle p_i) = \frac{\sum l_i w_i \triangle p_i,}{\sum l_i}$$

$$\begin{aligned} \mathbf{Cov}(w_i, p_i) &= \mathbf{E}(w_i p_i) - \mathbf{E}(w_i)\mathbf{E}(p_i) \\ &= \frac{\sum l_i w_i p_i}{\sum l_i} - \frac{\sum l_i w_i}{\sum l_i} \frac{\sum l_i p_i.}{\sum l_i} \end{aligned}$$

With these definitions we can rewrite eqn (2) as follows, letting $\triangle p = p' - p$, in keeping with our notational conventions,

$$w \triangle p = \mathbf{Cov}(w_i, p_i) + \mathbf{E}(w_i \triangle p_i). \tag{3}$$

This is Price's equation, and it expresses in a very general and precise way an obvious truth. The left-hand side, $w\triangle p$, is the mean fitness of the parental population multiplied by the change in mean p-score. As the mean fitness is always positive, eqn (3) says that we can deduce the direction of evolutionary change by finding the sign of the right-hand side. The covariance term is positive if an individual's fitness is positively correlated with its p-score and negative if that correlation is negative. This just tells us that if a high p-score is correlated with a high number of successful gametes, then it will tend to be increased by selection. We could plot number of successful gametes against p-score, representing each individual in the population as one point. If the best fitting straight line (using ploidies as weights) slopes up, then the covariance term is positive; if the line slopes down, then the covariance term is negative.

The second term on the right-hand side of eqn (3) is the gametic discrepancy term, representing the effect of the difference between an individual's own p-score and the p-score of its successful gametes. If we were studying random drift, then this term would be important. We have already agreed to neglect meiotic drive and so the gametic discrepancy term is on average 0. We now further assume the population is large enough for us to be able to ignore random fluctuations in this term. We will deal only with the covariance term.

The essentials of Price's method have now been derived and the reader anxious to use it should skip ahead. The rest of this subsection is an aside about Price's method. Firstly, following Price (1972), Hamilton (1975), and Wade (1985), I show how powerful a tool Price's method can be in analysing higher levels of population structure, but expand a point made by Hamilton (1975) about a complication in interpretation that is sometimes missed. Secondly, I review the uses to which Price's method has been put.

The extension to population structure is done simply by changing the interpretation of the symbols we have used. Suppose the population is divided into groups and that i indexes groups, not individuals. Then l_i is the number of haploid sets in the ith group, p_i is the average p-score of the haploid sets in the ith group, and $p_i + \triangle p_i$ is the average p-score of the successful gametes of the ith group. Equation (3) is still true, by the same argument, for this new interpretation of the symbols. The covariance term now represents the effect of selection on groups and the second term is no longer gametic discrepancy. Rather, it is the effect of the difference between the p-score of the groups and the p-score of the successful gametes of the group. Part of this is the effect of individual selection within groups, by which individuals with, for example, high p-scores have a higher fraction of their group's reproduction than individuals with low p-scores.

This decomposition can be expressed formally, using a second level of subscripting. Let us use g to index groups, and gi to index the ith individual within the gth group. The expectation and covariance may also be subscripted, because we can ask for the average or covariance between groups, and this is what **E** and **Cov** stand for; we can also ask for the average within the gth group, and this is what $\mathbf{E}_g$ and $\mathbf{Cov}_g$ represent. The decomposition is as follows:

$$w\triangle p = \mathbf{Cov}(w_g, p_g) + \mathbf{E}(w_g \triangle p_g)$$

$$= \mathbf{Cov}(w_g, p_g) + \mathbf{E}\{\mathbf{Cov}_g(w_{gi}, p_{gi}) + \mathbf{E}_g(w_{gi}, p_{gi})\}.$$

The second line is obtained by noticing that the second term in the first line is the average over groups of an expression of the form $w\triangle p$ and so substituting for it using eqn (3). This recursive scheme could be repeated by substituting for the third term in the second line, and so on, as discussed by Hamilton (1975).

It is tempting to suppose that if the within group covariance is negative, then individual selection is acting against the trait; and if the between group covariance is positive, then group selection is acting in favour of the trait; and that in such a case the trait is altruistic in that it favours the group, but harms the individual who expresses it. It is important to realize that this is false, if we interpret altruistic as originally defined by Hamilton (1964). The reason is that the between groups covariance contains, as Hamilton (1975) put it, 'a group selection component which is not 0, but which is bound in unchanging subordination to the individual selection component'. The between group covariance must be greater than this subordinate component for the trait to be altruistic in Hamilton's sense.

It is not hard to understand the source of this subordinate component. Suppose a trait affects only the bearer's fitness and is simply advantageous. Then the within group covariance is positive, because bearers within the group have more offspring than non-bearers. However, the between group covariance is also positive, because the groups with more bearers have more offspring than groups with fewer bearers. This positive between group covariance is not the result of any help given by the individual to other members of the group, but occurs because an individual possessing the allele is himself a fraction (one over the group size) of the group. This is Hamilton's 'subordinate component'. In cases where there is help given to fellow group members, its influence could be measured by subtracting the subordinate component from the between group covariance. Better in my opinion is to follow Hamilton (1975) in analysing the trait in terms of Hamilton's rule.

I now turn to a brief review of the influence of Price's covariance selection mathematics. The essence of Price's method, as he expressed it in his 1972 paper, is the naming of individuals rather than the naming of genotypes. This means that a subscript refers to an individual rather than to a genotype or to a gene. This led naturally, in Price's hands at least, to the application of the statistical notions of expectation and covariance in his population genetics models. The method he devised has not been much used, but two results he derived from it are sometimes cited.

The first result for which he is often cited is that the change in a character resulting from selection is equal to the genetic covariance of that character with fitness. This result was derived in a very different way by Robertson (1966) and named by him (1968) the 'secondary theorem of natural selection'. Crow and Nagylaki (1976) develop this theorem further. For Robertson, this result was important because it allowed the effect of

selection on a character to be estimated from known quantities, no matter what incidental selection might be simultaneously carried out. This incidental selection could arise either because of a correlation of characters within the population, or just because there were also other selective forces at work. Other methods depended on the selective force studied being the only one at work and on the absence of correlation with other characters relevant for fitness. Price values the result for its conceptual clarity and generality of expression.

The second result for which Price is cited is the hierarchical decomposition of a population variance in a character, or population covariance between characters, in the way we have just seen. The variance (or covariance) has a within individual component, then a within group component, then a within deme component, and so on, to as many levels as desired. The decomposition is a general one, allowing individuals to have different ploidies, groups to have different numbers of individuals, and so on. Wright (references in Wright 1969) had already suggested this kind of decomposition for his *F*-statistics. Price used this second result as an illustration of the power of the first.

The only papers to my knowledge that use Price's method at any length, as opposed to citing one of these two results, are Hamilton (1970, 1975), Seger (1981), Wade (1985), and this paper. I know from as yet unpublished work of my own, and from comments of others who have used it, that the method is extremely useful over and above these two results, and I expect that there will soon be a number of papers that put the method itself to good use.

THE DERIVATION OF HAMILTON'S RULE

I now return to the main business of the section, deriving Hamilton's rule, and to our standard interpretation of i as indexing individuals. We have obtained Price's equation, which describes the evolutionary change in any character. The next step is to model the social interactions that are the subject of Hamilton's rule, and in particular, to say how the fitness of an individual depends on its phenotype and on the phenotypes of the individuals with which it interacts. In mathematical terms, we wish to model w_i. Suppose pairwise interactions take place, in such a way that one individual, the actor, has an opportunity to help another, the potential recipient. If the act is committed, we suppose that the actor's number of offspring is decreased by c, while the recipient's number of offspring is increased by b. (b and c are more precisely the changes in the number of haplotypes supplied in successful gametes to offspring. In the usual case, a parent provides one gamete containing one haplotype for each offspring.) Let m_i be the number of interactions in which the ith individual is the actor and n_i the number in which it is the potential recipient.

In order to know how many offspring an individual has, we need to know what happens in these interactions. On what fraction of the m_i occasions on which it was the actor did the ith individual commit the social act? Let it be

h_i. On what fraction of occasions on which the ith individual was the potential recipient did the actor commit the act, and thus the ith individual receive the benefit? Let it be y_i. I will refer to h_i as the phenotype of the ith individual, because h_i summarizes its actions. Similarly, y_i is the mean phenotype of the actor on the occasions on which the ith individual is the potential recipient. The net effect of all these social interactions on the number of offspring of the ith individual is

$$(n_i y_i)b - (m_i h_i)c.$$

Now this is the difference made by these social acts, but it is possible that the baseline fitness, to which this difference must be added, varies from individual to individual. The most obvious reason is that the baseline fitness of males and females may be different. If there are different ploidies within one sex, then the baseline fitness may be different for different ploidies. Finally, the baseline fitness may vary because selection of other characters is going on in the population or simply through random variation in number of offspring. Let the ith individual's baseline fitness be f_i. Then its number of offspring is

$$f_i + n_i y_i b - m_i h_i c.$$

w_i is the number of offspring of the ith individual, divided by its ploidy, and so we arrive at the following:

$$w_i = \frac{1}{l_i}(f_i + n_i y_i b - m_i b_i c). \tag{4}$$

This is the required model of w_i.

The next step is to combine our model of social interactions with Price's equation, so that we can look at the effect of social interactions on the systematic evolutionary changes in a p-score. We do this by using eqn (4) to substitute for w_i in eqn (3) without its last, gametic discrepancy, term. This gives

$$w\triangle p = \mathbf{Cov}(p_i, w_i)$$

$$= \mathbf{Cov}(p_i, \frac{1}{l_i}\{f_i + n_i y_i b - m_i h_i c\})$$

$$= \mathbf{Cov}(p_i, \frac{f_i}{l_i}) + b\mathbf{Cov}(p_i, \frac{n_i y_i}{l_i}) - c\mathbf{Cov}(p_i, \frac{m_i h_i}{l_i}). \tag{5}$$

The second step follows because covariances are distributive over addition.

The first of the three covariance terms is between the p-score and baseline fitness, and represents the effect on evolutionary change in the

p-score of forces other than the social interactions. It is the remaining two terms that we will study further. They are to be rearranged. At the moment they are covariances where each data point is an individual. Hamilton's rule is phrased in terms of occasions when help is or is not given, and the rearrangement is to convert the covariances across individuals into covariances across occasions. Our focus shifts from a list of individuals to a list of occasions. Just as each individual had a list of numbers associated with it, so each occasion has a list of numbers. Let us index occasions by j, just as we indexed individuals by i. Thus, we will speak of the jth occasion. We define the total number of occasions as J. The numbers for each occasion are

D_j	the p-score of the actor on the jth occasion;
R_j	the p-score of the potential recipient on the jth occasion;
H_j	the phenotype of the donor on the jth occasion, that is 1 if the act is committed and 0 if it is not. (h_i is the average of H_j for those occasions on which the donor was the ith individual.)

The expressions I will form from these variables are not strictly covariances, though they are covariance-like. I use the symbol **K** to represent them, and they are defined by:

$$\mathbf{K}(H_j,D_j) = \frac{1}{J} \sum H_j(D_j - p)$$

and

$$\mathbf{K}(H_j,R_j) = \frac{1}{J} \sum H_j(R_j - p).$$

Thus, the first **K** is the average value across occasions of the product of the donor's phenotype and the donor's deviation from the mean p-score of the population. The second is the average across occasions of the product of the donor's phenotype and the recipient's deviation from the mean p-score of the population.

The **K**'s would be covariances if p were the average p-score of actors in the first expression and the average p-score of potential recipients in the second. Instead, in both cases it is the average p-score of the population as a whole. This will be relevant later.

In order to assert the algebraic relationship between the covariances across individuals and the **K** forms across occasions, let α be the number of occasions in a generation divided by the number of haploid sets in the parental population. That is,

$$\alpha = \frac{\sum m_i}{\sum l_i} = \frac{\sum n_i}{\sum l_i} = \frac{J}{\sum l_i}.$$

The following identities are easily proved by expanding the covariances and **K** forms:

$$\mathbf{Cov}(\frac{m_i h_i}{l_i}, p_i) = \alpha \mathbf{K}(H_j, D_j)$$

$$\mathbf{Cov}(\frac{n_i y_i}{l_i}, p_i) = \alpha \mathbf{K}(H_j, R_j).$$

We can use these equations to substitute in eqn (5), and find that, for a p-score uncorrelated with baseline fitness,

$$w\triangle p = \alpha b \mathbf{K}(H_j, R_j) - \alpha c \mathbf{K}\ (H_j, D_j)$$

and so, providing $\mathbf{K}(H_j, D_j) \neq 0$,

$$w\triangle p = \alpha \mathbf{K}(H_j, D_j) \left\{ \frac{\mathbf{K}(H_j,R_j)}{\mathbf{K}(H_j, D_j)} \ b - c \right\} \quad . \tag{6}$$

This is the end of the derivation. Equation (6) is the expression we have been looking for; let us see why. The p-score increases from one generation to the next if $w\triangle p$ is positive and decreases if it is negative. The sign of $w\triangle p$ can be found from the signs of the three terms on the right hand side of eqn (6). α is positive by definition. The second term is positive for p-scores that are positively associated with committing the social action when the opportunity arises; let us restrict attention to those p-scores. Whether the p-score then increases or decreases depends on the sign of the third, bracketed term and it is to this we now turn.

If we allow ourselves to represent the ratio of **K** forms by one symbol and go so far as to use the symbol r, then we can rewrite the condition that the decisive third term is positive, so that the p-score increases, as

$$rb - c > 0.$$

However, this is exactly Hamilton's rule, as stated in eqn (1). We have proved Hamilton's rule, given that we are prepared to define r to be the ratio of **K** forms in the third term of eqn (6); that is

$$r = \frac{\sum H_j(R_j - p)}{\sum H_j(D_j - p)}. \tag{7}$$

The problem now is that having defined r in this way, as a measure of genetic similarity (Crozier's relatedness), we cannot assume that it has any connection with kinship (Crozier's relationship). Equation (7) is a particular way of measuring genetic similarity and its differences from previously suggested measures will be discussed in section 7. The most useful property of relatedness, as usually understood, is that it expresses a connection between two individuals that can be computed from ancestry.

Equation (7), on the other hand, looks as if it could be different for different p-scores and for different characters, which would be a serious inconvenience in applying Hamilton's rule. However, all we can do is to explore the properties of r as defined by eqn (7) and hope for the best; for that is the definition which makes Hamilton's rule work and so we are stuck with it, however inconvenient it may turn out to be. Much of the rest of the paper is concerned with exploring the consequences of defining relatedness by eqn (7).

I end this section by reviewing it. We set out to find a definition of relatedness that would make Hamilton's rule work and we found one. In the process, we encountered the very powerful technique of Price's method. Our derivation of Hamilton's rule is valid for populations in which different members have different ploidies, for asexual, bisexual, and trisexual populations, for varying numbers of social encounters per individual, and for cases where the interacting individuals are not genetically representative of the population. It is true for populations with any kind of geographical structure and any kind of inbreeding. This generality will prove to be somewhat illusory, however, owing to complications in the interpretation of r. Hamilton's rule is true for any p-score on which selection acts only through the social behaviour modelled, but for other p-scores it tells us the effect of the social behaviour on the direction of selection of the p-score. (Naturally, it cannot predict changes in p-scores that are the consequence of processes we have not modelled!) In the next section we start from quite different considerations to arrive at a way of measuring genetic similarity that turns out to be intimately connected with the definition of relatedness discovered in this section. Succeeding sections explore implications of this coincidence.

3. A geometric view of relatedness

Leave aside the newly discovered formula for r, to which we shall return, and concentrate instead on genetic similarity, which is what we intend relatedness to be a measure of. There are many senses of similarity, and to explain the particular kind of genetic similarity I have in mind in this section, it helps to have a picture. Figure 1 is a very simple picture indeed. The line represents the frequency of one particular gene, where one end is 0 and the other end is 1. We can represent individual animals on this line according to the frequency of the gene they possess. A haploid creature must lie at one end of the line or the other, because it either has the gene or it doesn't. A diploid creature must sit at one of the ends if it is a

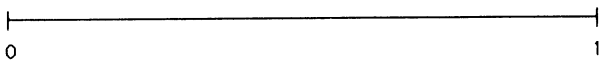

Fig. 1. A line representing a gene frequency, on which various entities can be represented. A haploid either has the gene or doesn't, and so its gene frequency is 0 or 1, and must lie at one end or the other of the line. A diploid's gene frequency is either 0 or 1 for a homozygote, or 0.5 for a heterozygote.

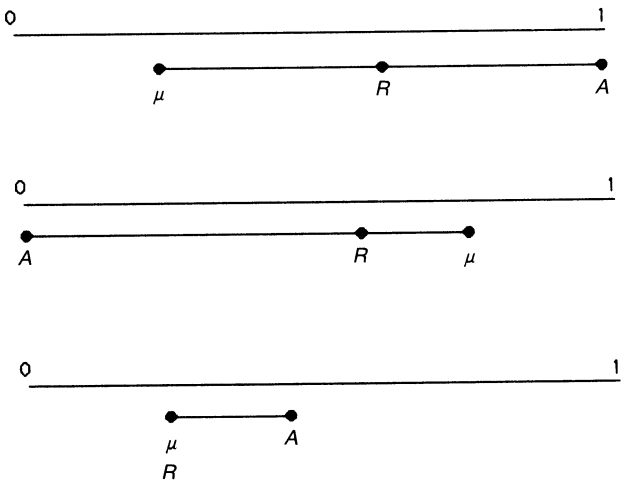

Fig. 2. Three examples of possible relationships between the gene frequencies of the population (μ), the actor (A) and the potential recipient (R). The relatednesses are, in order starting from the top, 0.5, 0.25, and 0. The population mean is not the same in each example.

homozygote, or in the middle if it is a heterozygote. We can also use a point to represent the average gene frequency in a population and, if the population is large, we can think of the population mean as lying at any position on the line.

Consider three points on the line. The point designated μ is the population mean, the point A is the actor, and R is the position of the average gene frequency of the potential recipients of the actor. This simple diagram contains all the necessary ingredients for the geometric view of relatedness, which is as follows. Take the line that runs from μ to A. Then the relatedness of the actor to the potential recipients is the fraction of the way along the line that R is found. If R is at the beginning of the line, at μ, then the relatedness is 0. If R is half-way in between the two points, then the relatedness is 0.5. If R is at the same point as A, then the relatedness is one. Some examples are illustrated in Fig. 2.

This is the geometric view of relatedness. I will develop it in a number of ways. I shall first explain how it can be extended into more dimensions so that it becomes a general picture of how whole genomes are related, rather than just of the presence of one allele at a single locus. I will then say how it relates to the more obvious ideas of relatedness such as sibship and other ancestral links, and thirdly why it is the right concept of relatedness to use in Hamilton's rule. These points can be seen as the formalization of the intuition that Hamilton's rule is obvious (at least in retrospect). In the next section I will make the connection between this simple picture of relatedness and the definition of r adopted in the previous section.

First the extra dimensions, to represent more than one allele, and more than one locus. The essentials of the picture, the points μ, R, and A stay

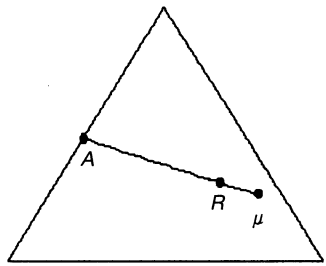

Fig. 3. The μ–*R*–*A* line in a triangle representing the allelic frequencies at a locus with three alleles. The relatedness is the fraction of the way from μ to *A* at which *R* is found.

the same: only the background changes. The simplest example is a locus with three alleles. The genotype at this locus can be represented as a point inside a triangle such as Fig. 3. The distance from each line represents the fraction of the corresponding allele. Haploid creatures must sit at one of the three corners. Diploid homozygotes also sit in a corner, but diploid heterozygotes sit at the midpoint of one of the sides. A triploid organism with one of each allele can sit in the middle of the triangle. Again, the mean of the population can be represented in the diagram and, if the population is large enough, it can be practically anywhere in the triangle.

Now think of the three points μ, *R*, and *A*, and in particular of the line from μ to *A*. Later a whole section is devoted to the possibility that *R* does not lie on this line, but assume for the moment that *R* does lie on the μ–*A* line. Again the relatedness is the fraction of the way along the line from μ to *A* at which *R* is found. The example can be extended to any number of alleles at one locus by imagining a tetrahedron for four alleles, then a tesseract for five, and so on.

Figure 4 shows a different extension to two dimensions. This is a square,

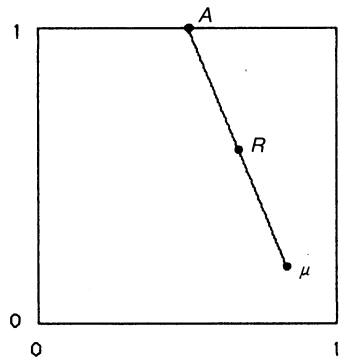

Fig. 4. The μ–*R*–*A* line in a square representing the frequency of one allele at each of two loci.

in which each axis corresponds to a different locus, each locus having two alleles. One point in the square represents the frequency of the alleles at each of two loci. Again individuals can be represented as points. Haploids sit at a corner. Diploids sit at a corner if they are double homozygotes, on the middle of an edge if they are homozygous at one locus and heterozygous at the other, and right in the middle of the square if they are double heterozygotes. The population can be represented by a point in the square, and so once again we can have the three points, μ, R, and A. Once more, the relatedness is the fraction of the way along the line from μ to A at which R is found. This same idea can easily be extended to any number of alleles at any number of loci. We can imagine a space in which one point represents the whole genome of an organism and the same three points could be found, and the same computation of relatedness made. μ, R, and A are what Jacquard (1974) called 'genic structures'.

Now what does this geometric picture of relatedness, of genetic similarity, have to do with ordinary notions of common ancestry, such as being someone's sister or second cousin? Suppose you are diploid, and you know your own position in genetic space and the position of the population mean as well. How can you compute the position of your offspring? Any particular offspring is at a particular place, of course, but where on average do you expect an offspring to be? Let's assume panmixia and an infinitely large population. One allele at each locus in your offspring comes from you and one from your mate. So your offspring are on average half-way between you and your mate in genetic space. Your mate is on average at the population mean, since this is the meaning of the assumption of panmixia. It follows that your offspring are, on average, half-way between you and the population mean. What then, in the geometric view, is your relatedness from you to your offspring? The answer is a half. Offspring themselves may have offspring, and their position will, by the same argument, be half-way between your offspring's position and the population mean. They are therefore three-quarters of the way from you to the population mean, and so they are one-quarter of the way from the population mean to you. You are therefore related to them by one-quarter.

A similar argument can be used for a sibling. Each of my two alleles at a locus came from a different parent, and so each allele had a separate 50 per cent chance of also being transmitted to my sibling, who shares both parents. So there's a 25 per cent chance of each of four possible outcomes: we share both alleles, only the paternal allele, only the maternal one, or neither. So the average fraction of alleles shared by copying from parents is a half and the average position of the shared part is my own position in genetic space. The unshared fraction of my sibling's genotype is filled with genes about which, because of the assumption of panmixia, I can assume that they are drawn at random from the population. Hence, the unshared fraction of a half has its average position in genetic space at the population mean. My sibling is on average half me, half the population mean, and so my sibling's average position is half-way between me and the population mean. So my relatedness to him is one-half.

Similar arguments can be constructed for other relationships. The point is not that these methods of computing relatedness are novel, far from it. Charnov (1977) applied them algebraically in his derivation of Hamilton's rule. Rather, the point is how easily the methods fit into the geometric view of relatedness. The picture is a helpful way of seeing how the arguments work.

Turning from how the geometric view accords with the concepts of common ancestry, we move on to the question of what connection it might have with the evolution of social behaviour. The mechanism of evolution is changes in gene frequency. If an individual at μ, the population mean, reproduces, then the gene frequencies are not changed because the addition to the offspring population has the same gene frequency as the adult population. On the other hand, if another individual, say the actor at A, reproduces, then this moves the offspring population slightly from μ to A, perhaps only a little way, but if everyone near A reproduces more than average, then this will cause the changes in gene frequency that underlie evolution.

Take A's view. Reproduction by an individual at μ, or equal reproduction by members of a group whose mean position is at μ, is irrelevant. However, someone else at A reproducing has exactly the same effect on gene frequencies as if A itself reproduced. If an individual half-way in between produces two offspring, then this will have the same effect on gene frequencies as if someone at μ had one offspring and someone at A had the other. Similarly, if an individual whose position in genetic space was one-eighth of the way from μ to A had eight offspring, then this would have the same effect on gene frequencies as if someone at μ had seven offspring and someone at A had one. The general drift must now be clear.

A physical analogy can be made. The process is rather like placing weights on a rod with a fulcrum at μ. To produce the same turning moment as one offspring at A, we would need two offspring at half the distance from μ to A, or four offspring at a quarter the distance from μ to A, and so on. The same turning moment corresponds to the same strength of effect on the gene frequency of the next generation. Hamilton (1971) used the analogy of different concentrations of liquid for the same purpose.

So if an individual reproduces who is a fraction λ of the way from μ to A, we can in our imaginations divide the set of offspring into two parts. The first, a fraction $1-\lambda$ of the offspring, at μ, and the second part, a fraction λ of the offspring at A. The combined average position of these two parts of the set of offspring is the same as the position of the original offspring, and so the effect on the gene frequency changes will be the same. However, the existence of the fraction at μ will not contribute to changes in the population gene frequency, because it is equal to the old population mean. The fraction at A, on the other hand, will change it and will change it to the same extent as if A had reproduced, but had only a fraction λ times as many offspring. Hamilton (1964) called the fraction $1-\lambda$ the 'diluting factor', because it does not change the direction of evolution, but does slow it down.

These arguments all show the same thing. That A should value R's

reproduction as a certain fraction of its own, because they would have the same effect on gene frequencies as a certain fraction of its own. Further, that fraction is the distance along the line from μ to A at which R can be found.

My hope is that the reader is now convinced that the geometric view of relatedness is simple, is connected in a straightforward way with ordinary notions of common ancestry and is plausibly the right kind of concept to make Hamilton's rule work. Before going on to the next section, though, there is one complication that should be mentioned because we will not return to it. It is the possibility of negative relatedness, whose relevance to the evolution of social behaviour was first explained by Hamilton (1970, 1971).

Negative relatedness means that one individual is genetically less similar to another than it is to a random member of the population. It has an obvious geometric interpretation. R is on the line that passes through μ and A, but it is not in between those two points. Rather it is on the other side of μ from A, as in Fig. 5. R deviates from μ in the opposite way to that in which A deviates from μ. The consequence of this is simple enough. A values R's offspring negatively and will be prepared to give up offspring of its own to prevent R reproducing. This is what Hamilton called spite (Hamilton 1964, 1970, 1971; Knowlton and Parker 1979).

We can also say a little about how negative relatedness might arise and the simplest plausible way involves a small population size. μ is the population mean and A is one of the individual in the population. If we ask what is the relatedness of A to a random member of the population, the answer is immediate: it is zero because μ is at a fraction zero of the way from μ to A. However, what is the relatedness of A to an individual chosen randomly from the other members of the population, excluding the possibility that it might be A itself? The answer is illustrated in Fig. 5. Let N be the population size. Take R to be the average position of the members of the population excluding A. μ is the mean of R weighted by $N-1$, and A weighted by 1. It follows, as Hamilton (1971) first showed, that the relatedness of A to R is $-1/(N-1)$. This is negligible if the population is not very small, but is the basis for the models of spite cited above. The alert reader may have noticed that a small population affects our calculations of relatedness to relatives as well, for similar reasons, but

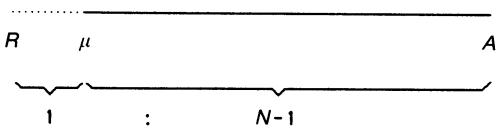

Fig. 5. The relatedness of an individual to the rest of the population is negative, because R lies on the other side of μ from A. The relative lengths of the line segments are 1:N-1 because μ is the average of R weighted by the N-1 other members of the population, and A weighted by one. This means that the relatedness is -1/(N-1) as Hamilton (1971) first showed.

again this has a negligible effect when the population is large. I have presented the geometric view of relatedness rather informally in the hope of being intelligible. The two connections of the geometric view, with social behaviour and kinship, can be made more rigorously, and this is the task of the next two sections. By making the connections in a more mathematical way, we prepare for answering the question of how generally Hamilton's rule applies, the topic of section 6.

4. Hamilton's rule and regression

The connection of the geometric view of relatedness proposed in the previous section on the one hand, with the definition of r from section 2, and with the notion of common ancestry, on the other, must be made within some formal framework. Regression is the framework I shall use. It is ideally suited because it has an algebraic side, in which to do things, and a geometric side, in which to think about what to do and how to do it. Relatedness was first treated as a regression coefficient by Hamilton (1970) and later by many others. However, in all cases they use only the algebraic aspect of a regression, as a covariance divided by a variance. The interpretation of these regressions in geometrical terms is the basis of the view of relatedness expounded in the previous section.

The first step is to find an algebraic way of saying that the average position of R, which is the average position of the potential recipients of A, lies on the line from μ to A. If it is a fraction β of the way from μ to A, then we can write

$$\mathbf{E}(R) = (1-\beta)\mu + \beta A,$$

or, after slight rearrangement,

$$\mathbf{E}(R) = \mu + \beta(A - \mu).$$

The average value of R depends on the position of A and, if we imagine that A can vary, formally that A is a random variable, then we should write the average value of R conditional on the value of A as the left-hand side of this, giving

$$\mathbf{E}(R|A) = \mu + \beta(A-\mu). \tag{8}$$

This is in statistical terms a regression equation, that is, it gives the expectation of one variable conditional on the value of another. It is a linear regression equation. We can compare this to the more familiar form, often used to represent the model in a simple, univariate regression of y on x in statistical textbooks:

$$\mathbf{E}(y|x) = \alpha + \beta x,$$

or

$$y = \alpha + \beta x + \text{error}$$

The differences between these two regressions are as follows. In the first, the dependent variable is R, and the independent variable is $A-\mu$, while in the second the dependent variable is y and the independent variable is x. Those are merely notational differences. Usually, α is unknown and needs to be estimated, while we regard μ in eqn (8) as known. Finally, μ, A, and R may be vectors in some large dimensioned space [the 'genic structures' of Jacquard (1974)], whereas α, x, and y are ordinary numbers. For ease of explanation, I want to discuss the simple case where μ, A, and R are ordinary numbers, and represent a p-score of the population, actor, and potential recipient, respectively. (The case where they must be treated as vectors is important when we want to estimate relatedness from electrophoretic data).

We have now expressed in an algebraic way the statement that R lies at a certain fraction of the way along the line from μ to A. The next step is to find a connection between this algebraic statement, and the algebraic definition of r in section 2. To do this, we will pretend we are trying to estimate β from our regression eqn (8), using the set of occasions in one generation as data. Of course, we don't have that data as a set of numbers in any example, but we can nevertheless find an algebraic expression for what we would estimate β to be if we did.

What form does this data take? Recall the notation of section 2. On the jth occasion, the actor's p-score was D_j and the potential recipient's p-score was R_j. There were J occasions in all. So the (x,y) data points we have with which to estimate β are the J pairs $(D_j,\ R_j-\mu)$. In order to see how to estimate β, it is convenient to rewrite eqn (8) in a more convenient form, as

$$R_j = \mu + \beta(D_j - \mu) + \epsilon_j, \tag{9}$$

where ϵ_j is the deviation of R_j from its expected value.

We could rearrange eqn (9) to give

$$\beta = \frac{R_j - \mu}{D_j - \mu} - \frac{\epsilon_j}{D_j - \mu}$$

and this suggests taking the first term on the right-hand side as an estimate of β. The reasons are that we know all the terms in it, whereas ϵ_j is unknown, and that the second term is on average 0 because ϵ_j is. However, this would give us a separate estimate of β for each occasion and, as our biological hypothesis implies that the set of occasions can be taken together, we want a combined estimate from all J occasions. Accordingly, we multiply eqn (9) by arbitrarily chosen constants z_j giving

$$z_j R_j = z_j\mu + \beta z_j(D_j - \mu) + z_j\epsilon_j,$$

and then adding up over all occasions to get

$$\sum z_j R_j = \sum z_j \mu + \beta \sum z_j (D_j - \mu) + \sum z_j \epsilon_j$$

and rearranging as before to give

$$\beta = \frac{\sum z_j (R_j - \mu)}{\sum z_j (D_j - \mu)} - \frac{\sum z_j \epsilon_j}{\sum z_j (D_j - \mu)}.$$

This suggests in the same way as before an estimate b of the form

$$b = \frac{\sum z_j (R_j - \mu)}{\sum z_j (D_j - \mu)}. \tag{10}$$

This is an unbiassed estimate of β provided only that the z_j are not correlated with the ϵ_j. Now I introduced the z_j as arbitrary constants and promised to return to them. A standard statistical argument goes on from eqn (10) to find the most efficient choice for these arbitrary constants, from the point of view of minimizing the sampling variance of b while maintaining its property of unbiassedness. However, we part company with the statistical argument here and choose to compare eqn (10) with our definition of r in section 2.

Let me repeat the definition, which was eqn (7), and set it alongside eqn (10).

$$b = \frac{\sum z_j (R_j - \mu)}{\sum z_j (D_j - \mu)}, \qquad r = \frac{\sum H_j (R_j - p)}{\sum H_j (D_j - p)}.$$

These two equations are very similar. One notational difference is that the population mean p-score is represented by μ on the left and by p on the right. Apart from that, we can make the two right-hand sides identical by choosing our arbitrary constants z_j to be equal to H_j.

The identity of these two formulae means that our algebraic definition of r from section 2 is an estimate of a regression coefficient. Further, it is an estimate of the regression coefficient that says how far R lies along the line from μ to A. Hence, our geometric picture of relatedness is the same as our algebraic formula, which was derived so as to make Hamilton's rule work. This is the formal version of the purely verbal argument made in section 3 that the geometric view of relatedness was also right for making Hamilton's rule work.

The main work of the section is now completed, but there is one important matter to discuss. We saw that r can be regarded as a special case of b, an estimate of the regression coefficient that expresses how far R lies along the line from μ to A. However, what if R does not lie on that line? In section 3 we assumed that it did, and now we return to look again at that assumption. The next section considers under what circumstances we expect the assumption to be fulfilled: here I want to explain the meaning

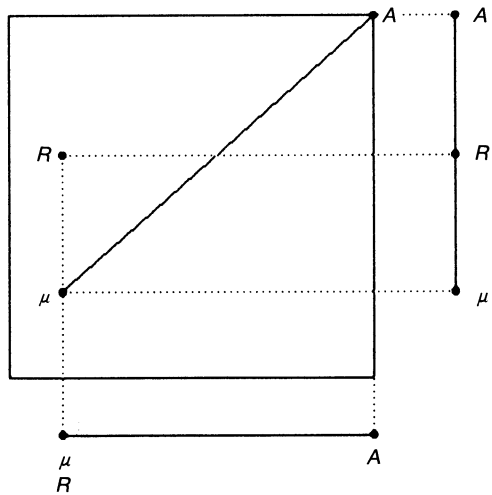

Fig. 6. When R lies off the μ–A line, the relatedness is different for different loci. In this example, the relatedness at the locus represented on the vertical axis is 0.5, while the relatedness at the locus represented on the horizontal axis is 0.

and consequences of its being false. The meaning is clear enough, and is illustrated in Fig. 6. R falls to one side of the line that joins μ and A. The immediate consequence is also illustrated in Fig. 6. We can compute r for any p-score. If we choose first the gene frequency at the locus represented on the horizontal axis, then we find that $r=0$, while if we choose the p-score to be the gene frequency at the other locus, then $r=0.5$. So, if R lies off the μ–A line, then r between the same sets of individuals is different for different p-scores, and therefore for different alleles and different loci.

One consequence of this is extremely important, and it is that we cannot expect to apply Hamilton's rule simply and usefully. Hamilton's rule is most simply applied when we know a group of individuals and some part of their genealogies. We then expect closer kin to be more co-operative and altruistic towards each other. This is because we hope to know r from common ancestry. However, if R lies off the μ–A line, then the r that makes Hamilton's rule work varies from locus to locus, and p-score to p-score, and so cannot depend on ancestry alone. The p-scores that are of special interest are those that control social behaviour, but it is beyond our current abilities to discover which loci are involved in those p-scores, to assess the influence of each allele at each locus, and to measure the presence of each allele in each individual. Even if we could do this, much of the value of Hamilton's rule would be lost. It is the rule's simplicity and range of applicability that make it so useful.

Another consequence is subtler. In the formula for b, our estimate of a regression coefficient, we have a set of arbitrarily chosen constants, namely the z_j. Consider to what extent the value of b depends on which set of arbitrary constants we choose. If R lies on the μ–A line, then the value of b

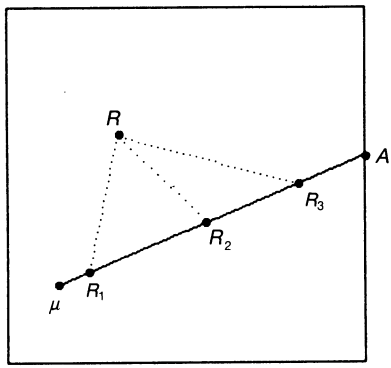

Fig. 7. The arbitrary constants determine the slope of the projection of R onto the μ–A line which is used in the computation of relatedness. If R lies on the μ–A line, then a projection with any slope will simply leave R where it is. However, when R lies off the μ–A line, as in the diagram, different projections take R to different points on the line, leading to different relatednesses.

does not depend at all on which constants we choose. If it lies close to the line, then the value of b depends weakly on the choice of arbitrary constants; and if R lies far from the μ–A line, then b will depend strongly on the choice. The reason for this is illustrated in Fig. 7. The regression involves projecting the position of R onto the μ–A line, and the choice of arbitrary constants determines the slope of the lines used in the projection.

Our definition of r from section 2 is, as we have seen, equivalent to a choice that the arbitrary constants should be chosen to be H_j. The H_j are the phenotypes of the actor on the jth occasion: H_j equals 0 if the actor does not perform the act and 1 if it does. It is the only item in the definition of r that contains information about dominance or about the way in which the p-score is related to phenotype at all. It follows that if R lies on the μ–A line, then dominance can have no effect on the direction of change of a p-score from one generation to the next. However, if R lies off the μ–A line, then dominance can affect the direction of the change. Queller (1984a) discusses a kin selection model where dominance has an effect and I will discuss briefly how this can be interpreted in terms of the geometric view of relatedness.

The example involves triploid endosperm in plants. This is a tissue that acts as an intermediary in the flow of resources between the parent plant and a seed. In the simplest of the genetic arrangements, the endosperm receives the same genetic contributions from each parent as the seed does, but it receives the maternal contribution in double dose. The endosperm is therefore triploid. The kin selection question is how the endosperm will be selected to act in its crucial role. Will it have the same interests as the parent, or as the seed, or somewhere in between?

This problem is the subject of a number of papers and this paper is not one of them. I want to concentrate on only one aspect of it and that is what

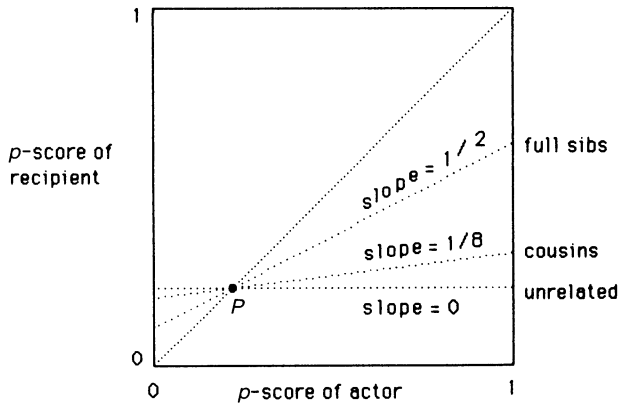

Fig. 8. The lines of best fit to regressions of recipient's p-score on actor's p-score, for sibs, cousins, and unrelated individuals.

is the relatedness of endosperm to seed? Let us use the simplest kind of p-score, the frequency of one allele. Figure 8 is helpful here and shows another side to the geometric view of relatedness. It has the actor's p-score on the x-axis, and the potential recipient's p-score on the y-axis. The large point marked P is on the 45° line, and is the point corresponding to the population mean on both axes. Imagine each occasion plotted on this graph, using the p-scores of the actor and the potential recipient, and calculating the line of best fit, on the condition that the line goes through the point P. (This is the same as redrawing the figure with P as the origin, and calculating the best fitting line that goes through the origin.) The slope of this line is the relatedness. Figure 8 shows lines of best fit (to imaginary data) for diploid siblings, cousins, and unrelated individuals. This graph is the basis of a method of estimating relatedness from electrophoretic data (Pamilo and Crozier 1982). Figure 8 is similar to Fig. 1 of Orlove (1975).

The statement that R lies on the μ–A line can be interpreted in terms of Fig. 9. For diploids, we can summarize the data that would appear in Fig. 8 as three points, each with its own weight: the mean p-score of potential recipients when the actor's p-score is 0, the mean p-score of the potential recipient when the actor's p-score is 0.5, and the mean p-score of the potential recipient when the actor's p-score is 1. The weights are the number of occasions contributing to each point. Now there always is a line of best fit, whether these three points are in a straight line or not. If R lies on the μ–A line, as in Fig. 9a, then these three points do lie on a straight line that passes through the point P. The consequence of this is that no matter how we weight the different points, the best fitting straight line is the same.

If, on the other hand, the three points do not lie on the same line, as in Fig. 9b, then how we weight them will affect which is the best fitting straight line and so its slope, and so will affect r according to our definition of section 2. Two factors determine the weights given to these three points.

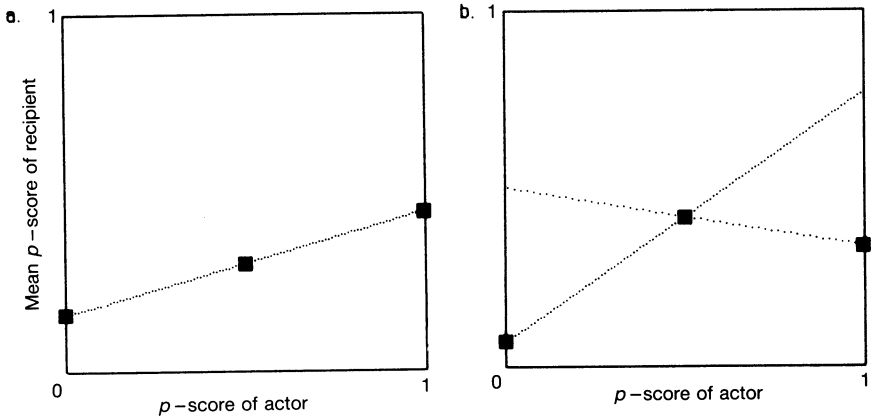

Fig. 9.(a) The points occupied by squares represent the average p-score of recipients when the actor's p-score is 0, 0.5, and 1. When they lie in a straight line, that line is the best fitting straight line. Therefore, the slope of the best fitting straight line does not depend on the weights given to the points in the regression. (b) Here the three squares do not lie on a straight line, and the slope of the best fitting straight line varies between the two lines illustrated. In this case, the slope of the best fitting straight line does depend on the weights given to the three points in the regression.

The number of occasions on which the actor has each of the three genotypes is one factor, and the other is the weights H_j which enter into the definition of r.

The case of the triploid endosperm is illustrated in Fig. 10. If the endosperm is homozygous, then the seed is also homozygous. If the endosperm is heterozygous, then the seed is too. However, both kinds of heterozygosity in the endosperm (one-third and two-thirds of an allele) are

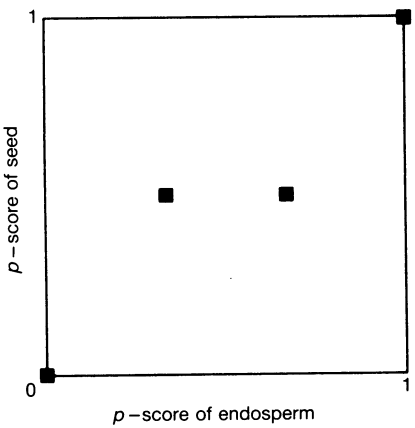

Fig. 10. A plot of the genotype of the seed (vertical axis) against the genotype of the endosperm (horizontal axis). The points do not lie on a straight line, so the best fitting straight line depends on how the points are weighted in the regression.

associated with the same kind of heterozygosity in the seed (one-half of the allele). Hence, the points do not fall on a straight line. Suppose that the frequency of an allele determines whether the endosperm transfers extra resources from the parent to the seed. Then H_j equals 1 if the endosperm does transfer extra resources, and equals 0 if it does not. H_j is a weight in finding the best fitting line and so the best fitting line depends on the dominance rule. This helps us to understand why, as Queller (1984a) and Bulmer (1985) show, whether such an allele spreads or not depends on its dominance. I am very grateful to Dr M. G. Bulmer for drawing my attention to this example.

The first lesson from this example is that it is important to know when R lies on the μ–A line. When it doesn't, as in the case of the triploid endosperm, it is not possible to predict the direction of selection of a character without knowing its genetics. This makes it much harder to see how natural selection will have acted on a character of whose genetics we are ignorant (which is to say nearly all characters). The second lesson is that our example is rather peculiar, and we may hope that this kind of difficulty is uncommon generally. We shall see that this example falls within the category of inbreeding, which turns out to be an important and general exception. The commonness of this difficulty is the topic of the next section.

5. When does R fall on the μ–A line?

To answer this question, we need a universe of possibilities. This will be provided by the mathematical machinery of identity by descent (Malécot 1969; Jacquard 1974). Within this set of possibilities, we will distinguish between those cases where R does and those in which R does not fall on the μ–A line. As we saw in the previous section, this is an important point in the usefulness of Hamilton's rule. To the extent that R does fall on the line, r between the same sets of individuals is the same for all loci and all alleles.

The plan of the section is to introduce the concept of identity by descent, to explain how this concept is used to describe in a precise way the genetic consequences of shared ancestry, and then to set out the assumptions needed to establish that for any non-inbred pattern of shared ancestry between actor and potential recipient, R will fall on the μ–A line. The plausibility of these assumptions in various circumstances will be discussed. We will then turn to the way in which genetic similarity between members of the same local population can be dealt with. The reason why inbreeding causes difficulties will be treated briefly. Then, as we have been using the machinery of identity by descent, we must confront the paradox of inbreeding of Seger (1981). The whole point of this section is concerned to avoid the conclusion that r varies between alleles and loci, and so at the end I explain briefly what would be the evolutionary consequences if it did.

Falconer (1981) defines identity by descent relative to a base population which existed at some time in the past. Two genes are said to be identical by descent if they are both descended from the same gene in that base

population. If we allow the base population to be two generations ago, then we can calculate how many genes at one locus are shared identically by descent by two outbred diploid sibs. There is a 50 per cent chance that they receive a copy of the same gene copy from their father. Even if their father is a homozygote at that locus, the two alleles he passes to his offspring are not identical by descent if one is a copy of his paternal allele and the other is a copy of his maternal allele. There is an independent 50 per cent chance that they receive a copy of the same gene copy from their mother. We can therefore assign an equal probability of 25 per cent to each of four possibilities: the sibs share no genes identically by descent at that locus, they share only the paternal gene, only the maternal gene, or both.

The genetic consequences of genealogical relationships are summarized by numbers of this sort. For non-inbred diploid relationships, a set of numbers called Cotterman's coefficients are used. They are the probabilities that the two individuals share exactly none, exactly one, or exactly two alleles identically by descent. For possibly inbred diploid relationships, a set of eight numbers is needed if the distinction between the individuals' maternal and paternal alleles is not kept. The details of these fascinating mathematical constructions can best be pursued by consulting Jacquard (1974), but only two points are essential to us here. They are that any specified genealogical relationship has a representation of this sort, that can be written down, and that one quantity of particular interest can be computed from this representation. That quantity is what Crozier (1970) called G, and is the average fraction of alleles in one individual (in our case the recipient) that are identical by descent with any of the alleles in the other (the actor). A proviso that will be important is that the probabilities which characterize a genealogical relationship are correct only in the absence of selection.

Let us denote by φ the average fraction of the potential recipient's genotype that is identical by descent with any of the alleles in the actor. Now in principle, φ could be different for different actors' genotypes, and this is indeed the case in relationships that involve inbreeding, as we shall see later. For the present, let us confine ourselves to non-inbred relationships and assume that φ is the same for all genotypes. We can think of two separate parts of the recipient's genotype, the IBD part and the non-IBD part, and in particular of the gene frequencies of those fractions. How do they compare with the population mean gene frequency and with the actor's gene frequency? Our route will be to assume that the IBD part of the recipient's genotype has on average the same gene frequency as the actor does and that the non-IBD part has on average the population mean gene frequency. Let us look at the consequences of these assumptions and then ask when we might expect them to be true.

If both these assumptions are true, then we can write the average gene frequency of the recipient as

$$\begin{aligned}\mathbf{E}(R \mid A) &= \varphi \text{ (average gene frequency of IBD part of genotype)} \\ &\quad + (1 - \varphi) \text{ (average gene frequency of non-IBD part of genotype)} \\ &= \varphi A + (1 - \varphi)\mu\end{aligned}$$

$$\mathbf{E}(R \mid A) \quad = \mu + \varphi(A - \mu) \tag{11}$$

This equation is similar to one of Jacquard (1974, p. 118) with notational differences and it is also familiar to us from above. It is the same as eqn (8) eqn (8) of section 4 except that here φ has taken the place of β. Furthermore, φ is determined by the genealogical relationship of the two individuals, and so is the same for all alleles and for all loci. Here we find a justification for assuming that R lies on the μ–A line. Equation (11) is true for any outbred genealogical relationship and also for mixtures of outbred genealogical relationships. If half of the potential recipients were sibs, a quarter were cousins and a quarter were unrelated, then the probabilistic statements of gene identities could be made for this mixture, and φ calculated in the same way as for 'pure' relatives.

Hence, it seems to be a general conclusion that if the genetic connection between the actor and the recipient arises through common ancestry with no inbreeding then R will lie on the μ–A line, but we must not forget the two assumptions we made to arrive at this conclusion. The first was that the IBD part of the recipient's genotype had the same gene frequencies as the actor did, and the second was that the non-IBD part of the recipient's genotype had the same gene frequencies as the population mean. Nor must we forget the proviso that the calculation of φ from the pattern of common ancestry assumes the absence of selection. Let us take the two assumptions in turn, and see why they should be true, and then decompose them into more fundamental assumptions. The justification of these more fundamental assumptions and of the proviso about the calculation of φ will be the task of the next section.

Why should the IBD part of the recipient's genotype have the same gene frequencies as the actor? If the actor is a homozygote at a locus, then the IBD part must be identical unless a mutation has occurred in the path through the genealogical tree that connects the recipient and the actor. After all, if it is identical by descent, then it is surely identical. If the actor is a heterozygote, then the recipient's IBD part must be identical (again, barring mutations) to one of the actor's alleles, but to which? If we can assume that it is equally likely to be to any of the actor's alleles, then it would again follow that the recipient's IBD part was the same on average, though not necessarily the same in any particular case, as the actor. We can sum up the assumptions needed to justify this in the statement that the only force at work is the random segregation of Mendelian genetics. Specifically, we need to assume no mutation and no selection.

Why does selection disturb our conclusion that the IBD part of the recipient's genotype is on average the same as the actor's genotype? Imagine tracing back along a diploid genealogical tree from the actor to a common ancestor and then forward again to the recipient, computing at each stage the chance that an allele in the actor was present. The rule we use is that there is a 50 per cent chance that a gene in a parent is passed on to an offspring, and that there is a 50 per cent chance that a gene in an offspring came from each parent. However, we know that each individual in the path survived to reproduce and if survival to reproduce is different for different genotypes, then this means that certain alleles had more than

a 50 per cent chance of being passed on, while others had less. The presence of selection means that Mendel's rules are not enough. Furthermore, the effect of selection will be different for different alleles and for different loci.

Noting that the first assumption has decomposed into the more fundamental assumptions of no mutation and no selection, we turn to the second assumption, which is that the non-IBD part of the recipient's genotype has the same gene frequencies as the population mean. This means that from the actor's point of view, the non-IBD part of the recipient's genotype is of equal importance to the genotype of a randomly chosen member of the population. This can fail in two ways. It may be that some genotypes are particularly prone to find themselves in the role of recipient. Alternatively, it may be that the population is not homogeneous, and that different parts of the population have different mean gene frequencies. For heterogeneous populations it may be that the non-IBD part of the recipient's genotype has the same gene frequencies as the local mean, not the global population mean. The actor is also likely to be genetically more similar to the local population, and so this is an additional source of genetic similarity between the actor and recipient, besides their common ancestry. Let us then add two more to our list of more fundamental assumptions: that the tendency to be a recipient is not affected by genotype and that the population is homogeneous.

While the strict truth of eqn (11) depends on no mutation and no selection, it will be approximately true provided the mutation or selection is small. What does small mean? The error in eqn (11) that is caused by a given mutation or selection pressure depends on the length of the genealogical routes connecting the recipient and the actor, because the opportunity for mutation and selection to act is proportional to the length of those paths. Only very strong selection could materially alter the conclusion that an offspring is equally likely to have either of its mother's alleles. Suppose, on the other hand, that an individual is known to have one gene identical by descent with a heterozygote ancestor a thousand generations ago. The accumulation of a thousand generations' worth of selection could easily make it much more likely that one rather than the other of the ancestor's genes is the one that is shared. Incidentally, we can also see how selection can affect the calculation of probabilities of identity by descent. Of all the individuals' ancestors a thousand generation's ago, it is more likely that the individual shares genes at a locus with those ancestors that possessed the selectively advantageous genes rather than the selectively disadvantageous ones. Which ancestors those are will be different for different loci.

Hence, both for the assumption that the IBD part of the recipient's gene frequencies is the same on average as the actor's gene frequencies, and for the calculation of probabilities of identity by descent, the mutation rates and selection pressures must be small in comparison with the lengths of the genealogical paths that connect the actor and the recipient. The justification for making this assumption about selection and mutation, in an argument that is intended to show how selection works on social behaviour, is one of the purposes of the next section.

Now the other fundamental assumption, of homogeneity, is also important and I want to consider two different ways of dealing with a structured population in the framework of the chapter. The first way is to assume that the non-IBD part of a recipient's gene frequencies is the same as the local population mean. Formally, we can let μ_L represent the mean of the local population to which the interactants belong and rewrite eqn (11) as

$$\mathbf{E}(R \mid A) = \mu_L + \varphi(A - \mu_L) \tag{12}$$

Now eqn (12) is no longer of the same form as eqn (8), and so we cannot identify φ and r so easily. The vital difference is that φ_L is no longer the same for every actor A. The derivation of r was valid for a population subdivided in any way: it does not change in parallel with the change in φ. Hence, this way of dealing with a subdivided population is analytically possible, but would imply a more complicated connection between r and φ, involving the correlation between A and μ_L. It would be different for different alleles and different loci, and so would lose the most attractive features of the simpler model.

The approach taken by Hamilton (1975) is equivalent to the introduction of a regression that says how μ_L varies with A, as follows:

$$\mathbf{E}(\mu_L \mid A) = \mu + \delta(A - \mu),$$

leading to

$$\mathbf{E}(R \mid A) = \mu + \{\delta + (1 - \delta)\varphi\}(A - \mu).$$

The problem with this is that δ, the regression coefficient that says how diverse groups are, may well differ from allele to allele and locus to locus, for the same reasons as we shall encounter below that impede the second way by which we might try to deal with local groups.

This second way is to view the extra similarity between group members as arising because of extra kinship ties between them. If a group is small, then random mating within the group must often be incestuous within the wider context of the population as a whole. Mating between relatives is one cause of extra genetic similarity between group members (another is adaptation to local conditions). Can we then not deal with social interactions between group members by calculating φ taking these extra kinship ties into account? The extra kinship ties are no doubt hard to specify in any particular case, but might this allow us in principle at least to rescue the identity between r and φ in the case of local groups?

Unfortunately not. The reason is that we fall foul of the fundamental assumption of no selection in a serious way. The paths that make fellow group members genetically more similar to each other than they are to members of other groups are likely to be long. The reason that selection influences the paths of descent is that it changes gene frequencies. It follows that random drift can have the same effect. Although random drift does not influence these paths averaged over all possible present universes,

we are interested in predicting selection in this particular present universe. Therefore, to apply in a simple way the machinery of identity by descent to model the effects of local grouping would be triple folly: the paths are long, significant drift and selection are very likely to have occurred, and inbreeding is almost certainly involved. An additional complication is that to the extent that groups are genetically isolated, the members of a group are likely to be competing particularly with each other for genetic representation in future generations. To that extent, the relevant relatedness will be relative to the local group and not to the whole population. I have discussed this at more length elsewhere (Grafen 1984). The application of the geometric view here is therefore rather complex, and I will pursue it no further here. The case of subdivided populations is nonetheless an important problem.

Now we tackle the problem of inbreeding, namely why φ should be different for different genotypes, that is the recipient lies at a different fraction of way from μ to A for different positions of A. Figure 11 illustrates the possibility.

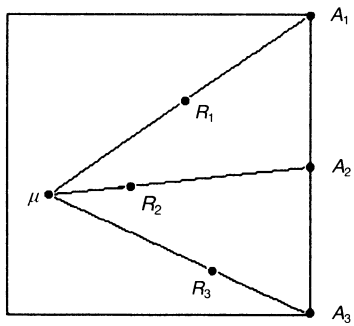

Fig. 11. Under inbreeding, the relatedness may depend on the genotype of the actor. Here the homozygotes have a higher relatedness than the heterozygote has to their respective recipients. It is likely in these circumstances that an actor will be differently related to the recipient at different loci.

Each actor represented there has a different value of φ, and if we considered more than one locus of one actor, we would find that it would have different values of φ at different loci. (As we saw in section 3, this implies that in the genetic space representing all the loci, R would not fall on the μ-A line.) The reason can be seen in Fig. 12a, which shows the various patterns of identity by descent associated with outbred sibship, and Fig. 12b, which shows the patterns for sibship when the parents were themselves outbred sibs. Each pattern has a value of φ associated with it, which is the fraction of the recipient's genotype that is identical by descent with any part of the actor's genotype at that locus. Each pattern also has a probability of occurring in a given genealogical relationship. The average value for the sibship is some weighted average of these φs. Now the three patterns for outbred sibship make no connections between the actor's two alleles and so place no restrictions on the actor's genotype. It follows,

conversely, that knowing the actor's genotype tells us nothing to make any one of the three patterns more likely than another.

Some of the inbred patterns, on the other hand, imply that the actor's two alleles are identical by descent and therefore that the actor must be a homozygote. Arguing backwards, if we know that the actor is a heterozygote, it follows that those patterns of identity by descent are not possible. Therefore, to find the average φ for an individual known to be a heterozygote, we must average over the patterns in which the actor's alleles are not identical by descent. A homozygote may or may not have its two alleles identical by descent, and so any of the patterns may occur. However, the knowledge that the actor is a homozygote makes it more likely that they are identical by descent. The balance of likelihoods depends on the frequency of the allele the homozygote possesses and so the relatedness will be different for the different types of homozygote. At a locus with three alleles, for example, there will therefore be four different degrees of relatedness: one for all heterozygotes and one each for the three different homozygotes (Elston and Lange 1976).

The difference between homozygotes and heterozygotes is important because dominant alleles are expressed in heterozygotes, while recessive alleles are expressed only in homozygotes. The spread of a recessive allele will therefore be determined by the homozygotic relatedness, while the spread of a dominant allele will be determined by some average of the homozygotic and heterozygotic relatednesses. The dominance of an allele may therefore affect whether it spreads or not. The relatedness of homozygotes depends on allele frequency, so whether an allele spreads or not may depend on its frequency. All these complications mean that alleles with the same phenotypic effect may be selected in opposite directions because they differ in dominance or frequency. Michod (1979) and Michod and Anderson (1979) have particularly clear discussions of this effect of inbreeding. The difficulties in applying Hamilton's rule to cases with inbreeding is discussed by Michod (1982), and a number of examples are worked through by Uyenoyama (1984). The appropriate definition of relatedness in terms of the nine identity coefficients of Fig. 12b, gene frequency and dominance, is given by Michod and Hamilton (1980, formula 3).

Our next topic is the paradox of inbreeding (Seger 1981). In the introduction, we dismissed the concept of identity by descent because we saw that if we look back far enough, most gene identity will be identity by descent. Yet for the whole of this section, we have been using identity by descent quite freely, refusing to see the paradox by looking no further back in time than some base population. This device serves the purposes of population geneticists well and involves no trickery, for they have a base population with respect to which they wish to measure identity by descent (Falconer 1981). We, on the other hand, have no such natural base population.

Our 'natural base' is not a population at all, but rather the behavioural rules that the population can adopt. If a nestling behaves in a particular way towards others in the same nest, but cannot discriminate between

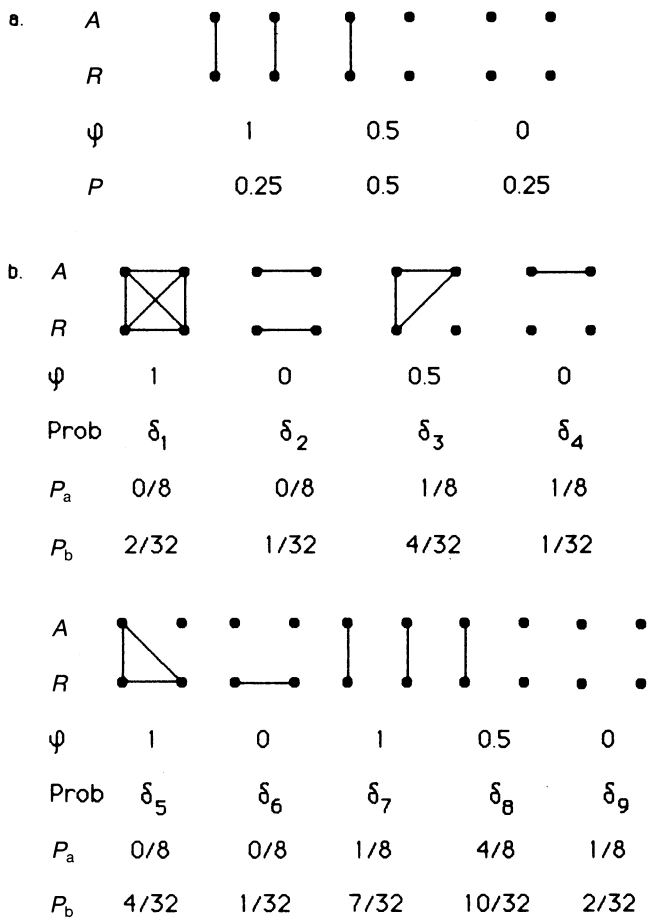

Fig. 12.(a) The top dots in each square represent the genotype of the actor, and the bottom two represent the genotype of the recipient. A line connects each pair of alleles that are identical by descent. Depicted here are the three patterns that are possible between outbred sibs and P is the probability with which they arise. φ is the fraction of alleles in the recipient that are identical by descent with any allele in the actor. With outbreeding, each genotype is distributed between the patterns in the same proportions. This allows us to calculate the relatedness between actor and recipient by averaging the φs weighted by the P's. (b) The nine possible patterns of identity by descent between diploids are illustrated, following Jacquard (1974). The δs represent the probability with which each pattern occurs and are his condensed identity coefficients ('condensed' because no distinction is made between an individual's maternal and paternal alleles). Two examples of the δs are given (P_A: the actor's two parents are parent and sibling of the recipient; P_B: the actor and recipient are sibs whose parents are outbred sibs). The top row contains those patterns in which the actor's two alleles are identical by descent, that is to say, the actor is inbred. The probability of this is therefore $\delta_1+\delta_2+\delta_3+\delta_4$. The bottom row contains the patterns in which the actor is not inbred. φ is the fraction of genes in

them in its actions, then the category of 'fellow nestling' is a natural one. For any particular nestling and fellow nestling, there will be a complex history of gene identities, but we shall divide it into two parts. One part will be correlated, in the sense that fellow nestlings are more likely to share it with each other than they are with nestlings from another nest; while the other part is uncorrelated, because fellow nestlings are no more likely to share it with each other than they are with those from another nest.

Take the simplest case, where all nestlings are full sibs, and there is random mating in a finite population. The correlated part of the relationship between nestlings is only sibship, because random mating destroys all other correlations. It does this in two steps. The random mating last generation prevents correlation between the alleles of one parent (that is, inbreeding of the parents), and the random mating this generation prevents any correlation arising from common ancestry between the parents, taking the set of sibships in the population as a whole. It may be that in a particular case, the mates are sibs, but this occasional event with a high positive correlation between the mates will be balanced by the much commoner slight negative correlation between the genotypes of mates guaranteed not to be sibs.

Of course, if nestlings can sense whether their parents are sibs or not, and can behave differently accordingly, then the systematic part of the relationship becomes more complex, but the same principles apply. The fact that parents are sibs, for example, would be a correlated part of the category 'Fellow nestling in a nest of mated sibs'. Any relationship that is uncorrelated with the distinctions that nestlings can make in their behaviour will cancel out when averaged over the population.

The resolution of the paradox of inbreeding is that relatedness is a property of 'action categories', and this has interesting implications. It does not make much sense to compute from their common ancestry the relatedness between two particular individuals. They will be related in very many ways, by distant routes, and the conclusion from adding them all together would probably be that the relatedness was one (Jacquard 1974, p. 171)! It makes more sense to ask how categories of individuals are related, such as nestlings or playmates or locals. Their relatedness can be assessed from common ancestry by knowing the paths that are correlated with the distinctions that individuals can make in their behaviour. It is

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the recipient that are identical by descent with any allele in the actor. The average value of φ for inbred actors is therefore $(\delta_1+\delta_3/2)/(\delta_1+\delta_2+\delta_3+\delta_4)$. The average value for outbred actors is $(\delta_5+\delta_7+\delta_8/2)/(\delta_5+\delta_6+\delta_7+\delta_8+\delta_9)$. Heterozygotes cannot be inbred, so are restricted to the bottom row, and therefore have the outbred relatedness. Homozygotes may be inbred or outbred, with a probability that depends on the frequency of the allele they bear. All the standard measures of relatedness can be defined in terms of the δs. Inbreeding coefficients for actor and recipient are $f_A = \delta_1+\delta_2+\delta_3+\delta_4$, and $f_R = \delta_1+\delta_2+\delta_5+\delta_6$. Malecot's 'coefficient de parente' is $f_{AR} = \delta_1+(\delta_3+\delta_5+\delta_7)/2+\delta_8/4$. Wright's coefficient of relationship is $r_A = 2f_{AR}/\{(1+f_A)(1+f_R)\}^{1/2}$. Hamilton's regression coefficient of relatedness is $b_{RA} = 2f_{AR}/(1+f_A)$.

important to stress that the centrality of those distinctions is quite natural. Our purpose in using relatedness is to analyse social behaviour, whose evolution must depend on the powers of discrimination animals possess. Jacquard (1974, p. 171) stated that 'It is obvious that, from this point of view, an inbreeding coefficient cannot be regarded as an estimate of a real quantity, but is simply a measure of information', and relatedness would fall into the same category as inbreeding. From our point of view, relatedness is a measure of the animal's information, and in the evolution of social behaviour this is something very real and very important.

Another consequence is that one individual can have a different relatedness to another, depending on the 'action-category' to which it belongs at the time. Suppose in a species with much brood parasitism that nestlings are related to each other by a quarter, because some are full sibs while others are unrelated; and that nestlings recognize each other only as fellow occupiers of the same nest and so cannot recognize each other after leaving the nest. Then an individual will treat sibs and unrelated individuals alike when in the nest, with a relatedness of one-quarter, and later will treat all of them as unrelated because he cannot distinguish them from any other individuals. Thus, relatedness is a property of 'action categories', not of individuals and not simply of patterns of common ancestry.

Finally, in this section whose main purpose is to argue that R will often fall on the μ–A line, I turn briefly to the evolutionary consequences of R's lying off that line. Recall that this means the relatedness as defined by eqn (7) of section 2, our definition of r that makes Hamilton's rule work, will be different for different p-scores. [It was Hamilton (1967) who first discussed this problem of genomic discord, with reference to the difference between the autosomes and the sex chromosomes, a cause I have entirely neglected here. He explored the consequences for the sex ratio of the fact that the X chromosome follows the same pattern of relatedness as haplodiploids, while the Y chromosome has an extreme pattern in which a male has a relatedness of one to sons and father.] Our formula for $w\triangle p$, eqn (6), is still correct for any p-score, so let us consider how it affects the frequency of an allele by choosing the p-score to be the frequency of that allele. In particular, let us consider those alleles that influence the performance of the social action. Then there is a critical value of r, say r', defined by $r'=c/b$, which is the relatedness at which the allele's frequency does not alter as a result of selection on the social action. The benefit and cost are such that with a relatedness of r', the actor is indifferent towards performing the act.

Now, if r varies between alleles and between loci, then alleles that contribute towards performance will increase in frequency if their r is greater than r' and decrease if it is less. Alleles that reduce performance of the act will increase in frequency if their r is less than r', and decrease if it is greater. Thus, selection will be acting in opposite ways on alleles with the same effect. The net effect of this genomic discord would depend on the relative numbers and size of effect of the alleles with different values of relatedness. Alleles with extreme values would tend to go to fixation or extinction. Some kind of average would prevail, but selection could stop

only when all alleles affecting the social behaviour, that had not gone to fixation or extinction, had the same relatedness. The position is more complicated than this, because relatedness is not a property of an allele by itself. If relatedness varies through the genome, then it is likely that the relatedness for one allele depends on its frequency and on the frequency of other alleles. This variation is described by eqn (7) in section 2, where the complication arises because the weights H_j matter and depend on these genetic complications. The conclusion that R falls on the μ–A line is useful because r does not then depend on those weights.

The next section picks up the fundamental assumptions on which we base our conclusion that R falls on the μ–A line and justifies them for our purposes. This forms part of a defence of Hamilton's rule as a general evolutionary principle.

6. Hamilton's rule as a general evolutionary principle

The justification of Hamilton's rule as an evolutionary principle has two parts to it. The first part was begun in previous sections and is to be completed in this. It is that we expect characters to evolve under conditions in which Hamilton's rule will be obeyed, and so if we understand a character correctly in nature, we should observe Hamilton's rule being obeyed. We justify the first part by showing that Hamilton's rule is a correct summary of a certain particularly relevant set of population genetics models. While the population geneticist is rightly interested in the exact analysis of a wide range of models, some models will be more relevant than others to the likely effect of selection in nature. The first part is permissive, it says that if we do use the rule we should get the right answer. The second part, not mentioned so far, is an argument that says we should want to analyse a character in terms of Hamilton's rule. For both models and data, this will enable us to understand the evolution of a character in a particularly interesting way; and for certain kinds of data it may be possible to analyse a character in terms of Hamilton's rule when it is not possible by rival methods. The second part of the justification may sometimes be so strong that it is worth using Hamilton's rule even when it is an incorrect summary of relevant population genetics models. These assertions will be argued later on.

Let us make sure of the starting point for our current argument by reviewing the relevant conclusions of earlier sections. On the way, we will come across points that need further discussion. The fundamental result is eqn (6) of section 2, which shows that Hamilton's rule correctly describes the effect of social interactions on the direction of selection of any p-score, provided we define relatedness as the genetic regression coefficient described in eqn (7). We must also remember that eqn (6) is based on an additive model of social interactions. The usefulness of eqn (6) depends on the relatedness being the same for all p-scores. Then, in section 4, we saw that this condition is fulfilled if the genetic similarity between interactants arises through links of common ancestry that do not involve inbreeding, provided there is weak selection and the population is homogeneous.

This leads us to the conclusion that Hamilton's rule, using the relatedness we would compute from ancestry, works under the conditions of (i) additivity, (ii) weak selection, (iii) homogeneity, and (iv) outbreeding. As we shall see in the next section, these conclusions are not new, and are the consensus of a fair-sized literature. If the third or fourth of these assumptions is broken, then alleles with the same effect may be selected in opposite directions because they differ in frequency or dominance. Although circumstances in which these assumptions are broken are very important, I have nothing further to say here about the outcomes of this genomic discord.

For most of this section, I shall concentrate on recent common ancestry as the main cause of the genetic similarity that influences the evolution of social behaviour. As a partial defence, I now consider a very special property of common ancestry in this respect. Common ancestry produces genetic similarity at every locus in the genome, and it produces the same genetic similarity (as measured by the relatedness of section 2) at every locus. Population structure as a cause of genetic similarity has already been discussed. Other possible mechanisms that have been suggested are genetic determination of micro-habitat choice, 'green beards', and the active detection of genetically similar individuals. Now I want to picture the effect

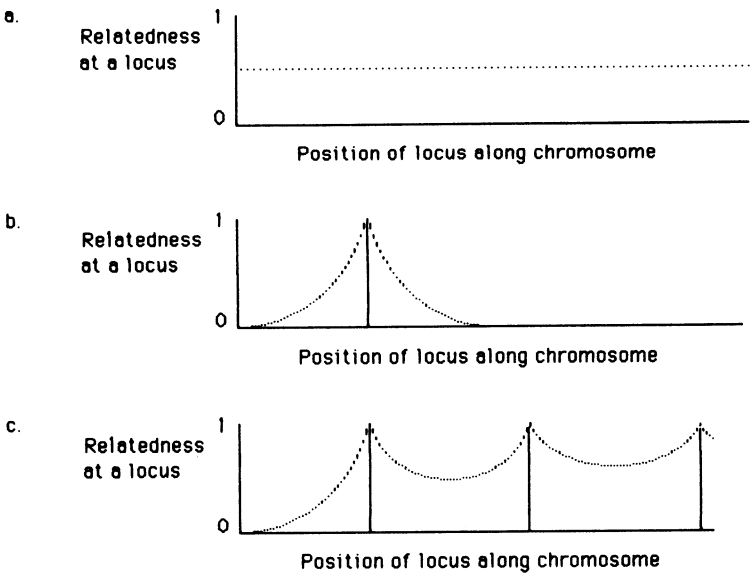

Fig. 13.(a) The relatedness between outbred diploid sibs under conditions of weak selection is the same at all loci and for all alleles at each locus. (b) When genetic similarity is caused by ensuring that one locus is identical, the relatedness falls off on either side of the 'guaranteed' locus. The rate of decay depends on the linkage disequilibrium between that locus and nearby loci. (c) To maintain a high relatedness along the chromosome, it is necessary to have 'guaranteed' loci at close intervals, so that all loci are in linkage disequilibrium with one or more of the 'guaranteed' loci, which can then act as telegraph poles.

of these possible mechanisms, and to consider their likely evolutionary consequences; I will not discuss the difficult problem of whether the mechanism is likely to evolve in the first place.

In the picture, each chromosome is a line, and the relatedness at each locus is plotted above it. Figure 13a shows the simple case of interacting sibs, where the relatedness at each locus is a half. Figure 13b shows the relatedness caused by any mechanism that ensures genetic identity at one particular locus. The relatedness at that locus is 1, and the relatedness at closely linked loci is increased above 0. The other loci must be so closely linked that they are in linkage disequilibrium with the locus that is guaranteed identical. The likely evolutionary consequence of one guaranteed identical locus is small. If a character could be affected only by very close loci, then it might evolve under a Hamiltonian regime with a relatedness appreciably different from 0. If a character can be affected by loci that are distant from the one guaranteed identical locus, then Hamilton's rule will continue to operate with 0 relatedness at distant loci and the net effect of selection will probably be settled in favour of the more numerous distant loci. This argument is the one given by Hamilton (1967) to explain the comparative inactivity of the sex chromosomes. To build up the kind of substantial average relatedness across the genome which is likely to be needed to produce an evolutionary effect, the mechanism would have to ensure identity at a number of loci, to be the 'telegraph poles' of Fig. 13c.

Hamilton (1964, p. 25) first suggested the possibility of direct recognition of possession of certain alleles [later termed 'green beard genes' by Dawkins (1976)] or even traits, and it has been further discussed by Dawkins (1976, 1982) and by Rushton *et al.* (1984). There are two possible effects of a direct recognition mechanism of this sort. The first is to distinguish between different kinds of relatives. Thus, in a mixed nest of sibs and half-sibs, the more similar individuals are likely to be sibs. This could be useful information. The other effect is to recognize from among unrelated individuals a subset who are as genetically similar as, say, cousins, but who are in fact genealogically unrelated. It is to this second possible effect that I now turn.

Rather than ask how a mechanism could ensure identity at enough loci to hold up the 'telegraph wire' of relatedness for the whole genome, I want to ask a logically prior question. It is, what fraction of unrelated individuals happen to be as genetically similar to an individual as its cousins? In other words, forget for the moment about how these individuals are to be recognized, how many of them are there to be discovered in the first place? If the number is very small, then genetic recognition mechanisms are probably unimportant for detecting genetically similar organisms from the population at large, but may still be important for distinguishing between different kinds of relative. We can answer this question in a rough way by recalling how many unrelated individuals we have met who are as phenotypically similar to us as our sibs (cousins, second cousins, and so on) are. We can also answer the question in a mathematical way, by calculating the probability distribution of relatedness [using eqn (7) from section 2 and weighting each locus equally] among unrelated individuals. Suppose there are L units in the genome that we can assume are in linkage equilibrium,

and that the mean probability of gene identity at a locus is f. Then, using the normal approximation to the binomial, we can obtain a normal deviate corresponding to a given relatedness. It is defined by the following formula:

$$z = r \sqrt{\left\{ L \ \frac{1-f}{f} \right\}}$$

For a species with L=50 000 and f=0.8 (plausible values for humans; Cavalli-Sforza and Bodmer 1971; Nevo 1978; Lewontin 1974), the square root is about 100. Hence, the normal deviate corresponding to an identical twin arising by chance among unrelated individuals is 100, for sibs is 50, and for cousins is about 12. These are very high normal deviates. A standard deviate of 4.8, corresponding to a relatedness of 0.048, would make the frequency of such individuals one in a million. A standard deviate of 3.1, corresponding to a relatedness of 0.031, would make their frequency 1 in 1000. If these values of L and f are reasonable, then it seems that there are no great advantages to be gained. The advantage from interacting with one individual with a relatedness of 0.03 would have to be offset against the cost of testing all 1000 individuals. I conclude that for genetic recognition to work other than by distinguishing between different kinds of relative, it is necessary that the relevant part of the genome be small, and this brings us back to green beards and the necessity for linkage; or alternatively that genetic variability be low. Unless the genetically similar, but unrelated individuals are of comparable frequency with corresponding relatives, selection is unlikely to favour complex or costly mechanisms for their detection. Of course, it may be that in some species these computations work out much more favourably.

I proceed by concentrating on common ancestry as the major cause of genetic similarity that is relevant to the selection of social behaviour. In the previous section, we saw that under weak selection, additivity, homogeneity, and outbreeding, common ancestry leads to unanimity of relatedness among the alleles and loci, and that this relatedness is derivable from knowledge of the ancestral links between interactants. I now wish to discuss how the assumptions of weak selection and additivity can be defended. The test is whether the r defined by eqn (7), a measure of genetic similarity I will call Hamilton's r because it makes Hamilton's rule work, is different from the relatedness that would be calculated from common ancestry, which I shall call the ancestral r.

An important point to begin with is that we are mostly interested in the evolution of a character, as distinct from the genetic changes that take place at a particular locus. The major part of the defence of the assumptions of weak selection and additivity is that they are likely to hold when selection has brought a character to a state in which there are no large improvements to be made. The argument is one given by Fisher (1958). Although strong selection pressures are to be expected when a character is changing rapidly, perhaps because of some change in the environment or in another species, once most of the required change has been made the possible improvements are small. It follows that the only

strong selection pressures in connection with the character are downwards pressures on strongly disadvantageous mutants. When the 'fine-tuning' of the character takes place, the only relevant selection pressures are weak ones. From now on we will assume where necessary that selection is weak. This is reasonable because we are interested in the conditions under which characters are perfected by natural selection. In this respect, Hamilton's rule and the Darwinian principle that animals are designed to maximize their reproduction, are in the same position. The Darwinian principle depends on fine-tuning under conditions of weak selection so that evolution will produce the precise optimum. Hamilton's rule depends on fine-tuning additionally because the weak selection implies equality between Hamilton's r and the ancestral r. Relying on the same condition twice introduces little extra burden of assumption for Hamilton's rule.

The simplest kind of character is one which can take any value in a continuum, and for which the fitness of the character is a smoothly varying, single peaked function of its value. (In the case of social interactions, the fitness function may depend on the state of the population in some way but this complication is unimportant.) I shall call this kind of character graded, and first discuss how Hamilton's rule works for them. Then we will go on to consider apparently ungraded characters.

In section 2 we proved Hamilton's rule using a model with additive fitness interactions and by agreeing to define r in a special way. We now wish to show that Hamilton's rule applies using ancestral r and with any kind of fitness interaction, on the assumption of weak selection. We saw in the previous section that the assumption of weak selection guarantees that Hamilton's r is the same as the ancestral r, and this leaves us with the additivity of fitness interactions. The meaning of additivity involves our model of the fitness of an individual as a function of its phenotype and of the phenotype of the individuals with which it interacts. Equation (4) from section 1 expresses the model as

$$w_i = \frac{1}{l_i}(f_i + n_i y_i b - m_i h_i c) \tag{4}$$

where w_i is the fitness per haploid set of individual i, l_i is its ploidy, f_i is its baseline fitness, n_i is the number of interactions in which it is the potential recipient, y_i is the average phenotype of the actors on the occasions when individual i is the potential recipient, m_i is the number of occasions on which it is the actor, and h_i is its phenotype. (For more details on the meaning of these symbols, the reader is referred back to section 2.) In our present application we are interested in whether a slight variant in a form of behaviour in a social interaction will spread or not. b is therefore the average effect on the recipient of interacting with the variant form rather than with the common form, and c is the average effect on the actor of adopting the variant form of behaviour rather than the common form. Examples of variants are to give slightly more or slightly less food, or to be slightly more or slightly less vigilant. b and c must be small if the social interaction is close to evolutionary stability, and the variant action is advantageous.

The important part of additivity is that w_i should be approximately a linear function of h_i and y_i. Alternative specifications for w_i are discussed by Scudo and Ghiselin (1975), Cavalli-Sforza and Feldman (1978), Charlesworth (1978), Maynard Smith (1980), Feldman and Cavalli-Sforza (1981), and others. The meaning of additivity is that the effects of separate occasions on an individual's fitness add up. Being a recipient once increases an individual's fitness by a certain amount. It is possible that being a recipient again does not increase the individual's fitness again by that same amount. This may be because, for example, the first gift of food rescued the individual from starvation, whereas the second merely allowed it to put on a little extra fat. Help in the form of warning calls when a predator is near does not add, but has diminishing returns, because the chance of survival is increased more by the first than by later calls.

However, when selection is weak the effects of occasions will approximately add up. This follows if fitness is a continuous and smooth function of the phenotypes of the interactants, which is part of the definition of a graded character. This convergence to additivity under weak selection is not a new point and has been made many times. One implication of this is that b and c may vary for a given kind of social interaction as the frequencies of those adopting it change [Uyenoyama and Feldman (1982) make this point more generally].

To find if Hamilton's rule fits a social interaction well, it is therefore necessary to consider the effects of slight variations in the behaviour of interactants. The total benefit and cost of the interaction are relevant to the question of whether the actor should abandon the interaction altogether, but marginal benefits and costs are relevant to the question of whether Hamilton's rule fits the precise form of the interaction.

Now this 'marginalization' of Hamilton's rule works well for graded characters, in which continuous changes in behaviour affect fitness continuously and there is only one local optimum. However, many characters seem to be not at all graded, and it is to these that we now turn. There are two main ways in which characters may fail to be graded. The first is that an action may be 'all or nothing', as in Haldane's famous example of a man saving a child from drowning by diving in and pulling him from a river (Haldane 1955). It is convenient here to suppose the victim to be an adult. The second way is that there may be 'multiple peaks' in the adaptive landscape (Wright 1977, 1978), and so it may be important to know whether a mutation of large effect will spread. When we need to predict the behaviour of a mutation of large effect, we lose the assumption of weak selection, and so lose the useful conclusion that Hamilton's r and ancestral r are equal.

My main strategy here is not to tackle ungraded characters directly, but rather to argue that apparently ungraded characters may be graded after all. This will not be a compelling case that all apparently ungraded characters are in fact graded, because that is largely a matter of fact. Instead I aim only to suggest that many apparently ungraded characters may be graded. After making this case, I will turn briefly to the question of how ungraded characters might evolve.

In Haldane's example, the action of the hero who dives in to save

someone from drowning seems to be 'all or nothing'. The problem this causes for Hamilton's rule is as follows. The potential hero makes an imaginary computation of the gene frequencies of the victim and, in particular, of the frequency of an allele that has a strong influence on the tendency of its bearer to be a hero in those circumstances when the opportunity arises. The obvious computation for the potential hero to make is the one from section 3. The gene frequency of the victim is in part the same as his own and the rest is the same as the population mean. That part is the relatedness.

The complication is that the potential hero has extra information about the genotype of the victim. The victim is still alive. This suggests that if the victim has had an opportunity to save others from drowning, he has not taken it. (We suppose a hero risks his own life.) This in turn suggests that the victim does not have alleles that are conducive to saving others. This information means that Hamilton's r at the loci affecting the tendency to save others from drowning is lower than their genealogical relationship alone suggests. If opportunities are rare, then this extra information is weak and the ancestral r will be only a little lower than Hamilton's r. If they are common, then the extra information would be stronger.

This illustrates the important point that genealogical relatedness affects the evolution of social behaviour only through the tendency of the same alleles to be present in actor and recipient, and so any other information about the presence or absence of those alleles is relevant in exactly the same way. We saw earlier that animals are unlikely to have information that makes a non-relative as genetically similar as a relative at all loci in the genome. The possibility shown in Haldane's example is that because of the phenotypic effects of particular loci, mere survival can give information about the genotype at those loci. In this case, the loci are those that affect willingness to risk one's own life. Selection through kin effects will be altered at those loci, in a way that Hamilton's rule predicts. Although Hamilton's rule is correct in this case, it loses its most appealing feature, because Hamilton's r is not the same as ancestral r.

Now the definition of a graded character was that the fitness of the character is a smoothly varying, single peaked function of its value. My aim now is to show how the character 'reaction when faced with the opportunity to dive in to a river to save someone else's life at some risk to one's own' might be graded, despite the all or none aspect of the decision in a particular case. These opportunities for heroism will not all be the same. In some cases the chance of success will be high, in some cases low. The risk will be great on some occasions and small on others. Various clues will be available to these chances, such as the temperature of the water, the light, the distance from shore, the swimming ability of the victim, the presence of other potential rescuers, and so on. The character can be graded once we accept that the behaviour of the potential hero depends on these circumstances. For any given set of circumstances, his decision is discrete – he dives or he doesn't – but to represent his decision rule we must say that in such and such sets of circumstances he will jump, and in so and so circumstances he will not.

Let us consider how those decision rules may differ among individuals. It

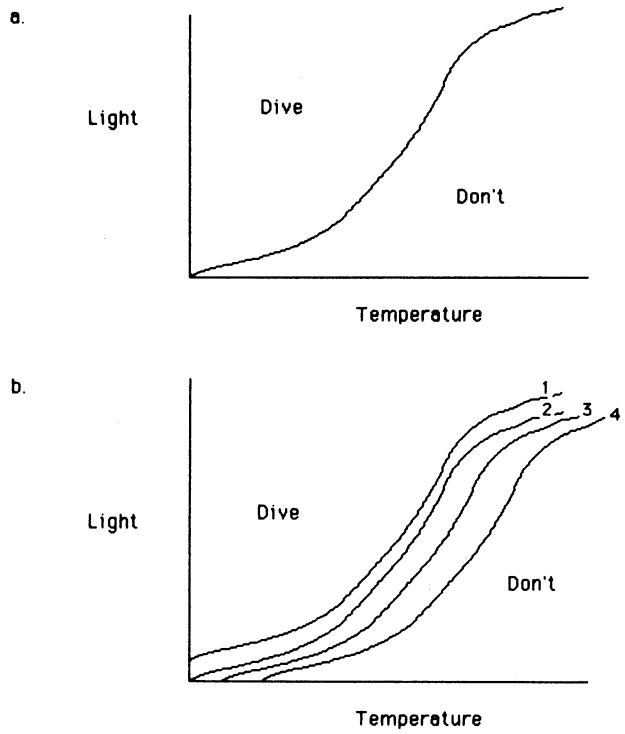

Fig. 14.(a) An imaginary decision rule that divides possible circumstances into two sets – those in which Haldane's man would dive and those in which he wouldn't. (b) The imaginary decision rules of four individuals. If the circumstances in which the rules differ are rare, then there is little information about genotype to be gained from observing the consequences of having dived or not (such as survival in the example in the text), even though diving may be common.

helps to have a picture and so Fig. 14a illustrates an imaginary decision rule that depends on temperature and light. The area to the left and above the line represents combinations of temperature and light at which he will dive, while the area to the right and below represents combinations at which he will not dive. Figure 14b illustrates the decision rules of a number of different individuals and I have assumed that they differ only slightly. For the sake of argument, assume that when an opportunity arises, it is equally likely to occur at any combination of circumstances in the figure. How does this affect the calculation of gene frequencies made by a potential hero?

The answer is that it tends to restore them to ancestral values. If individuals in the population have similar decision rules, then occasions on which they would disagree are rare. Most of the occasions will therefore fall into the unanimous regions of Fig. 14b, and so the survival to date of the victim gives very little information about the position of his decision

line. His survival therefore also gives very little information about his genotype at the loci affecting his decision line. It follows that Hamilton's r and the ancestral r will be much the same, because ancestry has again become the only guide to genetic similarity. If any shape of decision line is attainable by selection, then this means that the position of the line will be determined under a Hamiltonian regime and so at the circumstances on the line $rb-c=0$, while above it where the decision is to dive, $rb-c>0$, and below it where the decision is not to dive, $rb-c<0$. This is a strongly Hamiltonian conclusion, suggesting that in each circumstance the decision to dive or not will be determined by Hamilton's rule using ancestral r. This is possible because in the final evolved state of the character there is no genetic variation in the behaviour displayed in most circumstances. This is a good example of the divergence between the population geneticist who is interested in the genetics of a character, and the ethologist who is interested in the likely outcome of its evolution.

The crucial question for the equality of Hamilton's r and ancestral r is whether further information on genotype is available to the actor. There will be little information if the opportunities are rare on which the decisions of different individuals would be different.

This process of converting an all or nothing response into a graded character might be called parametrization, and is a generalization of the use to which Charlesworth (1978) put penetrance. The penetrance of a gene is the probability with which it is expressed, but usually there is no suggestion that the circumstances in which it is expressed are any different from the circumstances in which it is not. If the decision to dive in Haldane's example were made according to an irrelevant cue, then this would be equivalent to a random decision.

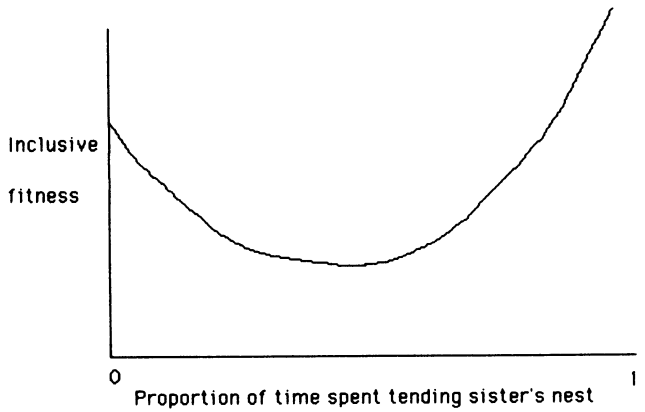

Fig. 15. A possible relationship between inclusive fitness (using the ancestral value of relatedness) and the extent of helping a sister. When a mutation for complete help arises in a population that doesn't help at all, the relatedness at that locus towards a sister is not the same as the ancestral relatedness, because the fact that the sister is not helping a previous sister makes it likely she does not share the gene for helping.

The second reason why a character may fail to be graded is that there are multiple peaks in its adaptive landscape (Wright 1977, 1978). An example of this is shown in Fig. 15. If the population is in the 'catchment area' of the highest peak, then our previous argument for graded characters applies, and the fine-tuning of the character is performed under Hamiltonian conditions in which Hamilton's r equals ancestral r. If the population is in the catchment area of a lower peak, then so far as small changes are concerned, the same argument applies again. The potential problem for Hamilton's rule occurs when a mutation occurs that could take the population from one catchment area to another. This could be an advantageous mutation of large effect, for which Hamilton's r and ancestral r might be different. If so, a mutation that is advantageous from the point of view of the ancestral r, and so from the point of view of a student of their behaviour, could fail to spread because Hamilton's r was lower than the ancestral r.

A hypothetical example of this, again borrowed from Charlesworth (1978), involves a hymenopteran female faced with a choice of how much of her time to allocate to her own reproduction, and how much to allocate to the reproduction of her sister, who, we may suppose, has nested nearby. It is convenient to suppose that a succession of sisters come to nest in an area. The value of $rb-c$ plotted against the fraction of time spent helping her sister might well be U-shaped, as in Fig. 15, on the grounds that dividing her time between two different nests would be inefficient. If the population is all at the lower, selfish peak, then a mutation of small effect would not spread. Consider the fate of a dominant mutation of large effect that caused a female to abandon her own attempt to reproduce and instead to become a full-time assistant to her sister. The fact that her sister has not abandoned her own reproduction and become an assistant to an earlier sister, shows that she does not share this mutation of large effect. Consequently, when this mutation of large effect arises, Hamilton's r will be 0 for this allele, despite the fact that the ancestral r is 0.75. (If the mutant had been recessive, Hamilton's r would have been 0.5. Notice how, when Hamilton's r and the ancestral r differ, genetic details such as dominance affect Hamilton's r.)

This character can be graded in the same way as Haldane's diving. If the mutation is of low penetrance, or if its expression is conditional on particularly favourable circumstances, then the fact that the sister has not helped another sister is much less informative about her genotype, and Hamilton's r will be close to ancestral r.

The conclusion is that the two most obvious reasons why a character may seem not to be graded are not decisive. Characters that have an all or nothing aspect and characters that have multiple peaks in their adaptive landscapes may after all be graded. If so Hamilton's r and the ancestral r will be equal when the character is finely-tuned by selection, and so the form of the character will be as predicted by Hamilton's rule using the ancestral r.

Even when characters are ungraded, fairly extreme circumstances must obtain to make the two r's very different. The fact of being a potential recipient must be very informative about an individual's genotype to

separate the r's, and this has strong implications for the size of the phenotypic effect, for the informative nature of its expression, and for the genetics of the character. It is for these reasons that I expect Hamilton's r and ancestral r to be the same for most characters. This is merely one opinion about an unknown matter of fact, but it is unfortunately important for the view we hold of the usefulness of Hamilton's rule.

We have reached the end of the exposition of the first part of the justification of Hamilton's rule. The rule is a reasonable summary of a particularly important set of population genetics models, namely those that seem most likely to be relevant to the outcome of the evolution of a character. Thus, if we use Hamilton's rule, we should not get the wrong answer about the evolutionary value of a character. If the first part says that it is safe, the second part of the justification says that it is desirable to analyse a character in terms of Hamilton's rule. Let us take models and data in turn.

To analyse a character in terms of Hamilton's rule means to work out r, b, and c. Now in some models, in particular those devoted to testing whether or not Hamilton's rule works, r, b, and c are taken as given, and in that case there is no analysing to be done. There are other models in which the character is described or defined in some other way, and it is in these models that Hamilton's rule can be used to advantage. The questions of particular interest for which this analysis is useful include: does this character spread because of its effect on the actor's own number of offspring, or does it spread only because of the effect of the action on the number of offspring of others? Is the action altruistic, or selfish, or spiteful? The way a character is defined may obscure these points. For example, the effects of an action may be described as, first, an effect on self and, second, an effect on every group member. b is the net effect of the action on self, and so if self is one of the group members, we must subtract the second effect from the first to compute b. Similarly, c is the total effect on others, and so to obtain c we must multiply the second effect by the number of group members (besides self) receiving it. This particular conversion from one description to another reconciles the trait group selection of Cohen and Eshel (1976), Matessi and Jayakar (1976), and Wilson (1975), with the Hamilton's rule approach.

Note that b and c are differences in number of offspring. Part of the discrepancy between the additive and multiplicative models pointed out by Cavalli-Sforza and Feldman (1978) is simply one of measurement. A physicist who decides to measure mass in a logarithmic scale can easily refute Newton's second law, if he fails to make the necessary concomitant adjustment to its algebraic expression. He deserves no attention because Newton's second law is framed with particular scales of measurement in mind. Hamilton's rule is designed to work on differences. It is not surprising that if they are measured as ratios instead, then Hamilton's rule fails. If the differences caused by the social action vary from occasion to occasion, then it will be necessary to find the average differences to compute b and c [Uyenoyama and Feldman (1982) explore this more rigorously]. However, simply measuring the costs and benefits as differences will not remove the discrepancies between the multiplicative and additive

models if some individuals partake twice in social actions, because then there is an added complication. The number of social actions an individual is involved in may depend on its genotype, because an altruist is more likely to have altruistic relatives. If, in addition, the effects of social actions combine multiplicatively, then the effect of the marginal social action when measured as a difference will depend in a systematic way on genotype. Thus, the benefit received by a recipient may depend on its genotype and this causes problems for inclusive fitness theory (Seger 1981; Queller 1984b). The main point here, though, is that even when social actions are thought to act multiplicatively, it is still possible and desirable to measure the costs and benefits as differences for the purposes of Hamilton's rule, averaging over occasions where necessary.

Having seen how and why we might want to analyse models in terms of Hamilton's rule, let us now turn to data. The questions Hamilton's rule will help us answer are the same – is the action selfish, spiteful, or altruistic, and is the action advantageous only because of the fitness effect on others? In applications to data, Hamilton's rule comes into its own. The great differences from models are that usually with data on social traits, the genotypes of individuals are unknown and the genetic system controlling the trait is unknown. This makes worries about dominance, number of loci, and mode of gene action purely academic. In modelling, the fundamental population genetics method of finding the number of offspring of each genotype is the main rival to Hamilton's rule. This alternative simply cannot be applied to data if the genotypes of individuals are unknown. Hamilton's rule can be applied, provided enough information is available to measure the effects of the social action. If this information is not available, we cannot discover by any means whether the action is altruistic or not. I have worked out an example in detail elsewhere (Grafen 1984, section 3.3.3), using data of Noonan (1981) on joint nesting in *Polistes fuscatus*.

Hamilton's rule, then, is the way to answer central questions of interest about a social action. It has the advantage that, unlike other methods, it can be applied to data that is available about social actions. For these reasons, the theoretical investigations of Hamilton's rule are unlikely to replace it with an alternative so far as data is concerned. A method that works well in a model in which individuals' genotypes are known can fail altogether when they are unknown. The theoretical investigations are valuable in finding the scope of validity of the rule, those circumstances in which it correctly predicts the direction of evolution of a trait. However, in a case where it is known to be incorrect, say where inbreeding is present, the practical response is to apply Hamilton's rule anyway, and treat the results with caution.

In conclusion, Hamilton's rule is useful because it tells us whether an action is selfish, spiteful, or altruistic, and because it tells us the value we expect one individual to place on another's reproduction. These are its important points, and they apply equally to models and to data. The current interest in altruism and social evolution has the rule at its centre, for it embodies the definitions of selfish, altruistic, and spiteful proposed by Hamilton (1964).

Many genetic details turned out not to upset the validity of Hamilton's rule. Dominance and the exact form of fitness interactions do not matter in a range of cases and even apparently discontinuous characters may be continuous underneath. However, there are limits in the range of possible genetic mechanisms for which Hamilton's rule does predict exactly the direction of evolution. The most important limitations are that when the population is structured, or when there is inbreeding, the relatedness which will make Hamilton's rule work will vary from allele to allele, and from locus to locus; so alleles with the same phenotypic effect will be selected in opposite directions because of differences in dominance or frequency. The likely outcome of these complications is not known, but it seems probable that some kind of average relatedness will prevail. The required modification to Hamilton's rule is likely to be a procedure for computing that average relatedness from knowledge of the population structure, nature of the inbreeding, and possibly the frequency of mutations of particular kinds.

The viewpoint of a population geneticist on the exceptions was expressed by Uyenoyama and Feldman (1982). They say:

> 'In summary, for additive kin selection models our analyses indicate that Hamilton's theory is remarkable precise' (p. 616).

> 'We show that by regarding the multiplicative model as an additive model with genotype-dependent benefit parameters, the multiplicative model can be reconciled with Hamilton's theory' (p. 626).

They do not think that the multiplicative model should be regarded as a minor variation on the additive model, however, and they give two reasons. The first is that multiplicative models allow strong internal equilibria, that is strongly stable polymorphisms, and the second is that the nature and identity of internal equilibria are affected by the use of approximations. These are good reasons why population genetic theorists should be interested in multiplicative models, but they are not reasons why an ethologist should be. The theory tells the ethologist that the behaviour of organisms should follow Hamilton's rule, and that is the only part of the theory he is likely to be able to test. Whether the population is genetically uniform for this behaviour (Uyenoyama and Feldman's 'viability-analogous equilibrium'), or polymorphic (their 'structural equilibria'), is less important, and will usually be beyond his ability to find out.

There are exceptions, in population genetics models, to Hamilton's rule. The lesson I draw from them is that, in order to make the rule work in those circumstances most relevant to the outcome of evolution on a character, we should find a suitable generalization of relatedness. The rule relates facts observable in the field to the evolutionary fate of a character, and its terms are a touchstone to the evolutionary significance of social behaviour. It is for these reasons, and not merely because it is a summary of certain population genetics models (though it is surprisingly good at that too), that Hamilton's rule is a general evolutionary principle.

7. A brief survey

In this section, a brief survey is presented of the work on Hamilton's rule and relatedness, and the ideas presented in previous sections will be placed in that context. For a full review of derivations of Hamilton's rule, see Michod (1982). The two authoritative sources for definitions of relatedness are Michod and Hamilton (1980), and Seger (1981). The first theme of this section is the confirmation of Hamilton's rule in a variety of models. The second theme is a division of claimed exceptions into three categories. The third is a comparison of the definition of r in this chapter with previous definitions.

Hamilton (1964) presented the derivation of his rule with care, and left the reader in no doubt about the qualifications and complications in the analysis. It was an additive model and so would work only for small effects; r was a good guide to the likely genotypic constitution of relatives only under weak selection. Since then, the rule has been rederived and these qualifications restated many times. Michod (1982) reviews these rederivations; here I wish only to mention the categories into which they fall. Some models have a single locus, while in others the tendency to altruism is a quantitative or polygenic character. Some ('inferential') models infer the gene frequency in recipients from the gene frequency of donors and the relatedness; while in others ('grouped') the population is divided up into mutually exclusive groups. These two classifications divide possible derivations into four, but to my knowledge only three of the possible types exist.

The original derivation was single locus and inferential, as were those of Hamilton (1970), Charnov (1977), Orlove and Wood (1978), Michod (1979), Harpending (1979), Charlesworth (1980), Charlesworth and Charnov (1980), Seger (1981), and the expository derivation of Maynard Smith (1982). The single locus grouped models include Hamilton (1975), Orlove (1975), Levitt (1975), Scudo and Ghiselin (1975), Charlesworth (1978), Cavalli-Sforza and Feldman (1978), Boorman and Levitt (1980), Uyenoyama and Feldman (1981, 1982), Uyenoyama *et al.* (1981), O'Donald (1982), Wade (1982), and Karlin and Matessi (1983). I know of no inferential quantitative models, but grouped quantitative models have been given by Yokoyama and Felsenstein (1978), Boyd and Richerson (1980), Aoki (1982), Crow and Aoki (1982), Engels (1983), and Cheverud (1984, 1985). All these models confirm the rule under the condition of weak selection, in the absence of inbreeding and additional population structure.

The derivations of Hamilton in 1964 and especially in 1970 have an extra generality, in allowing any number of interactions of any kind between the members of the population. Most others, including the derivation in this paper, consider only a single kind of action, involving only two individuals at a time, that may in some derivations be repeated. The 1970 derivation is in many ways still the most comprehensive of all, as it is also valid for inbreeding (though not mentioning exactly the problems this may cause). Almost all of the present paper can be regarded as a long, expository footnote to Hamilton's 1970 article.

The three kinds of approach have merits and disadvantages. The connections between the inferential and grouped models are discussed by Abugov and Michod (1981), and Michod (1982), who call them 'inclusive fitness' and 'family selection' models. The 'inferential' models are more general because the grouped models require that the social interactions take place within groups, and that within groups no discrimination is made between further and nearer relatives. The grouped models, on the other hand, allow greater rigour as a consequence of this simplicity.

The hallmark of a rigorous derivation, besides algebraic rectitude, is complete recursion. This means that when our 'generation-to-generation' equations are applied to one generation, they tell us enough about the next generation to allow us to apply to our generation-to-generation equations to that next generation. If they do, we can then move on to the third generation in the same way, and on to the fourth, fifth, and so on. Our derivation in section 2 is an example of an incomplete recursion, because eqn (6) needs to know about covariances in this generation, but tells us only about the mean p-score in the next. It follows that to obtain a complete recursion, we would need to find a way of calculating what those covariances will be in the next generation. Another name for completeness of recursion is dynamic sufficiency (Lewontin 1974).

Complete recursions are very demanding. Little vagueness is allowed, so it would be difficult, if not impossible, to prove a result that held for all different kinds of relatives. Each separate case must be analysed on its own. The joy of Price's method is its generality, and it is potentially a great advantage, but this raises an important question. Naively, it seems that either complete recursions are necessary to avoid error, in which case Price's advantage is illusory, or complete recursions are unnecessary. No doubt both methods have their part to play, but so far none of the important conceptual advances have been made by completely recursive methods. The important advances I have in mind are the original derivation (Hamilton 1964); the application of covariance methods and the 'backwards' definition of r as 'whatever will make Hamilton's rule work' (Hamilton 1970); the derivation of the rule to include inbreeding and the evaluation of relatedness from common ancestry in the presence of inbreeding (Hamilton 1970); the derivation of the rule for grouped populations (Hamilton 1975); and the rederivation that showed more explicitly the problems inbreeding may cause for Hamilton's rule (Michod 1979). The completely recursive methods tend to follow on in the rear, providing the comfort of a more rigorous derivation some time after the advance party has decided the problem and found the solution. When the incomplete methods lead to a result as simple as eqn (7), then for those interested in the fairly gross behaviour of the system it may be that there is little extra that complete recursions can do. Complete recursions are necessary to determine the exact nature of interior equilibria when the variations in r caused by selection are enough to change the sign of $rb-c$. These computations are unlikely to have any observational, empirical significance. Of course, it may be that complete recursions will play an important role in future in resolving the outstanding problems of inbreeding and heterogeneous populations.

The quantitative and polygenic models are all grouped models. They perform (essentially) an analysis of variance on the character of 'tendency to perform altruism to group members', and on its heritable component. There is, then, a within group variance and a between group variance in each. These imply certain correlations between group members and can be used to compute the covariances in terms of which relatedness can be defined (Michod and Hamilton 1980). The different models show, to varyingly explicit degrees, that Hamilton's rule applies under conditions of weak selection to indiscriminate social actions within groups in a grouped population.

Engels (1983), in addition, explicitly modelled what, in section 6, we argued by words, that a succession of small mutations will take a population to the equilibrium determined by Hamilton's rule. Cheverud (1984) developed quantitative genetics models that include maternal effects; he confirmed Hamilton's rule in the case with no maternal effects. Uyenoyama (1984) analysed exact models with specific patterns of inbreeding. She stated that the relatedness calculated from ancestry was a good guide to the models' results even when the rule strictly failed. The available literature confirms that the rule is a remarkably good summary of a wide range of population genetics models.

The derivation of section 2 is an inferential model, but is both single locus and polygenic, according to the interpretation of the p-score. It allows arbitrary ploidies, individuals may interact more than once, and the interactants need not have the same gene frequencies as the population. It thereby combines in one derivation results that would otherwise have to be proved separately.

The chief purpose of many of the papers cited above in support of Hamilton's rule was to prove it wrong in particular cases. The second theme of this brief review is a classification of the alleged exceptions to the rule. I divide them into three categories. The first are the strong selection exceptions that arise through breaking the weak selection assumption of section 6. These are real exceptions, but in section 6 I argued that they were likely to be unimportant from the point of view of the outcome of selection on a character. The second category are those that arise through a misinterpretation of Hamilton's rule, and these are unfair exceptions that we can set aside. The third category are exceptions within the assumptions of weak selection, which we may call important exceptions.

The multiplicative models begun by Scudo and Ghiselin (1975), and popularized by Cavalli-Sforza and Feldman (1978) are in danger of falling into the second, 'unfair' category because, in Hamilton's rule, the effects of an action are expressed as differences not ratios. This is well discussed by Uyenoyama and Feldman (1982). They regard the multiplicative model as an additive model with genotype dependent cost and benefit parameters, and this allows the results of their model to be compared with Hamilton's rule fairly. They found in their exact model of sib interaction that Hamilton's rule worked under weak, but not strong selection, and so we may place their exceptions, and by extension those of previous multiplicative models as well, in the first category.

Another strong selection exception is the deviation depending on heritability found by Boyd and Richerson (1980) in a quantitative model.

I will mention three examples of unfair exceptions. The first is claimed by Uyenoyama *et al.* (1981), in an additive model with two sexes. They define the benefits and costs relative to a mean fitness of unity, separately within each sex. The relative fitness of an unhelped, unhelping male is therefore 1 and that of a helped male is $1+\beta$. However, when all males are helped, the mean 'relative fitness' of the males is $1+\beta$, and so their fitnesses must be scaled down because the total male fitness is fixed by the number of females and the sex ratio of their offspring. Thus, in a population where virtually all males are helped, a rare selfish mutation reduces the male's number of offspring not by β, but by β normalized to the mean male relative fitness, i.e., $\beta/(1+\beta)$. This, together with a similar adjustment to the costs, brings the models' results into complete agreement with Hamilton's rule.

The second example of an unfair exception is from Cheverud (1984). He proposes that there is pleiotropy between two characters, namely some trait expressed as a juvenile and a maternal effect on that trait. He finds that Hamilton's rule, with costs and benefits derived from the effects of the juvenile trait alone, does not predict the effect of selection on the trait. A pleiotropic gene has two effects and it is hardly surprising that we cannot predict its fate with a rule applied to only one of those effects. It is certainly not a shortcoming in the rule.

The third example of an unfair exception is from Queller (1984b). He presents a two strategy, two player game theory model of interactions between relatives, where the strategy adopted by the recipient can affect the pay-offs to both interactants. This dependence of benefits and costs on the recipient's genotype is just the sort of thing that will plausibly cause exceptions to Hamilton's rule. In one of his two models (the continuous case), Queller supposes that the two strategies are played with a probability that depends on genotype, and he searches for an ESS probability which once common cannot be invaded by any other probability. Using a subscript 'Q' to distinguish Queller's notation where necessary, the pay-offs to strategy one are $b_Q-c_Q+d_Q$ when playing against strategy one, and $-c_Q$ when playing against strategy two. The pay-offs to strategy two are b_Q when playing against strategy one, and 0 when playing against strategy two. The idea is that strategy one is to perform a social act that costs c_Q, and has benefit b_Q, but that when both interactants perform this social act, there is a non-additive interaction so that instead of each receiving b_Q-c_Q, they each receive $b_Q-c_Q+d_Q$. He finds that the condition for a population playing strategy one with probability P to be invaded by a slightly higher probability of playing strategy one is

$$rb_Q - c_Q + Pd_Q + rPd_Q > 0$$

where r is the relatedness between interactants. This seems to differ from Hamilton's rule by the presence of the third and fourth terms which are caused by the interaction d_Q. However, the b of Hamilton's rule is the

average effect on the opponent's pay-off (in game theory terms), and this depends on the probability with which the opponent plays the two strategies. Similarly, c is the average effect on self and, in Queller's model, this also depends on the action of the opponent. Working out those average effects, we find that:

$$b = b_Q + Pd_Q$$

and

$$c = c_Q - Pd_Q.$$

When we express the condition for the spread of a slightly higher probability of strategy one in terms of b and c, we recover Hamilton's rule, for

$$rb_Q - c_Q + Pd_Q + rPd_Q = rb - c.$$

An equivalent conversion from game theory notation to Hamilton's rule notation was performed by Grafen (1979). The recovery of Hamilton's rule in this case is encouraging, because it may often be the case that the recipient's genotype affects the benefits and costs of social actions.

These unfair exceptions show that care must be taken in applying Hamilton's rule. The rule is based on a particular way of measuring costs and benefits, and on a particular concept of relatedness. If the rule works with one set of interpretations of its elements, then it is most unlikely to work with another. A result has no interest *as an exception to Hamilton's rule* if it is based on the wrong interpretation of r, b and c.

Now we come to the important exceptions, those where even with the correct interpretation of the rule and with weak selection the rule fails. The only example I am aware of is inbreeding and the reasons were first given in a slightly different context by Seger (1976). He was interested in explaining inbreeding depression as adaptive altruism by homozygotes, who by reason of their homozygosity were more highly related than average to those around them. The problems posed by inbreeding for Hamilton's rule were first explicitly discussed by Michod (1979), and Michod and Anderson (1979). The problem, as discussed in section 6, is that homozygotes and heterozygotes are differently related towards the same classes of relatives. This makes dominance important. The relatednesses depend on gene frequency, which adds to the complication.

Faced with this problem, a first approach is to try to place bounds on the deviation from the simple rule. This can be done using formula (3) of Michod and Hamilton (1980), although they did not do so. The relatedness in this context we may think of as the critical cost-benefit ratio at which the allele's frequency will not change. In one example of Michod (1979, in Fig. 2b with $h = 1$), the relatedness changed from seven-sixteenths to four-sixteenths as the gene changed in frequency from 0 to 1. (Michod and Hamilton pointed out that owing to a mistake in an earlier formula, Michod's figure is in error in showing the relatedness becoming negative as

the gene frequency approached 1.) This is an appreciable difference, of a size which could at some time matter in an application of Hamilton's rule to date.

One line of attack is to find bounds for a particularly relevant subset of inbred relationships, perhaps for those that are weakly inbred, or for those patterns that can arise in a system of regular mating. The second line of attack is to consider the consequence of these complications for a polygenic trait and find conditions under which the relatednesses at different loci will effectively average out to some central value. Michod and Anderson suggested simple averaging over loci and this could be right. The central value could then be compared with the simple relatedness that does not distinguish between homozygotes and heterozygotes.

There is one problem with the inclusive fitness approach which may be particularly acute in the case of inbreeding. In this approach, the nature of the relationship between interactants is taken as given, whereas it may be that the postulated social actions in one generation would change the relationship between interactants in the next. For example, a gene that caused females to mate with their brothers would create a situation in the next generation where some of the brother-sister pairs were themselves the product of a brother-sister mating. The relative frequency of the 'outbred' pairs and the 'inbred' pairs matters because they will have different relatednesses. Thus, it cannot be assumed that the pattern of relatednesses between interactants is a constant independent of gene action. This is not to say that the inclusive fitness approach is useless here, only that caution is required. Maynard Smith (1980) discusses the advantages of the inclusive fitness method in tackling this and other problems.

Uyenoyama (1984) analysed exact models with specific patterns of common ancestry that involve inbreeding. She stated that the relatedness calculated from ancestry was a good guide to the models' results even when the rule strictly failed. She searched for ESSs, in which a level of altruism was non-invadable by mutations of small effect. In some cases, the non-invadable level depended on the dominance of the mutations, which implies that there is no level proof against all mutations. There is, therefore, no ESS. In other cases, a whole range of levels was strongly stable against mutations of small effect, implying the coexistence of many strongly stable ESSs. Inbreeding is a major outstanding problem for Hamilton's rule and results about the likely outcome of selection on a character would be very interesting.

Summing up the survey of derivations of Hamilton's rule, the rule has been abundantly confirmed under weak selection. Inbreeding is the only important exception reported in the literature and, while the reasons for this exception are well understood, little has been established about the likely effect on the evolution of a character.

The next, briefer survey is of definitions of relatedness. The two authoritative surveys are by Michod and Hamilton (1980), and Seger (1981), and the early work was by Hamilton (1964, 1970, 1971, 1972, 1975), Orlove (1975), and Orlove and Wood (1978). The point of these definitions was to make Hamilton's rule work, and they all involve

variances and covariances of various sorts. Seger gave a particularly illuminating discussion of the assumptions under which some proposed definitions are valid. The definition in section 2 of the present paper is a slight improvement on previous definitions in three ways. Firstly, it is valid for arbitrary ploidies, thus uniting many cases in one formulation. Secondly, it allows different numbers of interactions per individual, and it allows the gene frequency of interactants to be different from the population mean. Thirdly, it allows the genotypic value to be any p-score. This draws our attention to the possibility that relatedness may be different for different alleles and shows how the relatednesses at loci in a many locus model should be combined.

Seger (1981) suggested a definition of relatedness (his R_4) which allowed actors and recipients to be genetically unrepresentative of the population. Comparison with eqn (7) shows that the reason for the complexity of his definition, and its dependence on b and c, is that it is expressed in terms of covariances around the actors' mean and recipients' mean. The simpler form of eqn (7) is achieved by taking moments about the population mean gene frequency, rather than about the actors' and recipients' means.

Michod and Hamilton (1980) gave the formula for relatedness from knowledge of common ancestry in the presence of inbreeding for diploids. They showed that all previous formulae were equivalent to it. I take this opportunity to present a particularly simple arrangement of their 'one pleomorphic coefficient'. Suppose α_{inb} and α_{outb} are the regressions of phenotype (tendency to perform the social action) on genotype (the fraction of a particular allele at a locus) within inbred actors and outbred actors, respectively, and that p_{inb} and p_{outb} are the probabilities of the actor being inbred and outbred, respectively, at that locus. Further, let φ_{inb} and φ_{outb} be the average fraction of genes at that locus in the recipient that are identical by descent with any gene at that locus in the actor, when the actor is inbred and outbred, respectively. The φs are therefore the relatednesses of inbred and outbred actors to recipients. Then, eqn (3) of Michod and Hamilton relatedness may be re-expressed as

$$\text{critical cost-benefit ratio} = \frac{2p_{\text{inb}}\alpha_{\text{inb}}\varphi_{\text{inb}} + p_{\text{outb}}\alpha_{\text{outb}}\varphi_{\text{outb}}}{2p_{\text{inb}}\alpha_{\text{inb}} + p_{\text{outb}}\alpha_{\text{outb}}}. \tag{13}$$

This is a weighted average of the relatedness of inbred actors and the relatedness of outbred actors. The idea of distinguishing these two relatednesses was suggested by Hamilton (1970). In terms of the coefficients of identity by Jacquard (1974), and as the reader can easily check from Fig. 12b,

$$\varphi_{\text{inb}} = \frac{\delta_1 + \delta_3/2}{\delta_1 + \delta_2 + \delta_3 + \delta_4}$$

and

$$\varphi_{outb} = \frac{\delta_5 + \delta_7 + \delta_8/2}{\delta_5 + \delta_6 + \delta_7 + \delta_8 + \delta_9}.$$

The weights in the weighted average are the product of three factors. The first factor is two for inbred actors and one for outbred actors, because the fate of genes in an inbred actor is twice as important from the point of view of one of his alleles. The second factor is the probability that the actor is inbred or outbred. The third factor, the regression of phenotype on genotype, is the only one that varies (explicitly at least) as a function of gene frequency and is the only one that depends on dominance. In fact it is only α_{outb} (equivalent to the α of Michod and Hamilton) that depends on gene frequency.

The form of eqn (13) makes very clear some of the properties of the critical cost-benefit ratio in the case of inbreeding. It is a constant if the two φs are equal, which means that inbred and outbred actors do not differ in their relatedness to the recipient; it does not depend on gene frequency when the heterozygotes are half-way between the homozygotes in their behaviour, for this makes α_{outb} independent of gene frequency; if the heterozygote lies between the two homozygotes in its behaviour (no over- or under-dominance) then the critical ratio lies between the two φs, because both weights are then always positive. I note that, with purely notational changes, eqn (13) is an immediate consequence of a result of Hamilton (1970).

In quantitative models, the population is divided into mutually exclusive and exhaustive groups, and the interactions have a very simple structure in which an individual affects all other group members equally. Relatedness is, in consequence, a much simpler concept and is defined as a correlation between group members. Thus, when Aoki (1982) claimed to have proved the rule 'independent of . . . inbreeding and under the action of selection', we must bear in mind that under inbreeding or selection the correlation between group members will vary between alleles and loci. Aoki had not, therefore, proved a stronger result in the quantitative case than is known for single locus models.

8. Conclusions

It may be of help to the reader to sum up the variety of material in the preceding seven sections and make some conclusions. The geometric view of relatedness was presented as a psychovisual aid in understanding the sometimes complex topics of relatedness and Hamilton's rule. Algebraic results parallel to the geometric intuition were derived in surrounding sections and these culminated in an explicit defence of Hamilton's rule as a general evolutionary principle in the study of social behaviour.

Hamilton (1964) derived his rule as a tool for understanding the evolution of social behaviour. In the central case of weak selection in an outbreeding, homogeneous population, later work has abundantly confirmed

the validity of the rule as a summary of relevant population genetics models. The social behaviour of inbred and heterogeneous populations is also of great interest, however. In 1970, Hamilton extended his rule to include inbreeding, while in 1975 he explored how population structure could increase relatedness between members of the same group. The problems caused by inbreeding have been explored by Michod (1979), and Michod and Anderson (1979), and some examples worked out by Uyenoyama (1984). The fundamental problem is that the relatedness needed to predict the direction of gene frequency changes differs for dominant and recessive alleles, and depends on gene frequencies. We have seen that this same problem arises in the case of heterogeneous populations.

Neither of these difficult cases has been fully explored, and it would not be surprising if the solutions proposed by Hamilton (1972, 1975) turn out to be close to the truth most or even all of the time. On the other hand, it is also possible that there are biologically significant exceptions, cases of special interest where the solution is materially different. To the extent that Hamilton's rule is applied to species with inbreeding or with a structured population, the speedy working out of these cases is of some importance.

The rule must be judged as a success. It expresses in a precise way the ideas of altruism, spite, and selfishness. It summarizes, in a readily intelligible and accurate way, a whole host of population genetics models relevant to the likely evolution a social trait. Neither of the two serious drawbacks is understood well enough to say what other relevant factors must be included in a more embracing rule. The effects of inbreeding or of a structured population would be best summarized, if possible, in a way of defining relatedness that would make Hamilton's rule apply to those cases as well. The relative value placed on the reproduction of another, compared to the value placed on one's own, is a clear and interesting way of summarizing the action of selection on a social characteristic. This is the reason for the importance of the rule, and why we should strive to express our conclusions in terms of it.

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