Confounding Factors for Hamilton's Rule

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May, 2002

Anthropology 137 Final Paper

1. Preliminaries

In his now classic 1964 paper on kin selection, W. D. Hamilton introduced a necessary and sufficient condition for the adaptiveness of altruistic behavior toward kin: namely, that the benefit conferred on the relative should exceed the cost assumed by at least a factor of the coefficient of relationship ($rb > c$). This criterion has since become known simply as "Hamilton's rule." In his derivation of the rule, Hamilton relied on certain assumptions, such as random mating, which from an intuitive standpoint may appear superfluous. This paper is concerned with identifying and analyzing conditions under which Hamilton's rule requires modification or restatement, with particular attention to situations not satisfying Hamilton's original hypotheses. The rule is found to be particularly sensitive to change in the assumption of random mating.

Many common objections to Hamilton's rule have their basis in fallacies or misunderstandings (Dawkins, 1979). Prior to any discussion of confounding factors for Hamilton's rule, then, a certain clarification of terms is in order. Central to the statement of Hamilton's rule is the notion of the coefficient of relatedness r between two individuals. Unfortunately, there is considerable confusion in the literature as to the exact definition of the coefficient r. Hamilton (1964) defines r as the proportion of genes identical by descent, that is, descended from the same copy of the gene in the closest common ancestor. The rather subtle distinction between genes that are merely identical and those that are identical by descent is the source of a common fallacy, labeled "Washburn's fallacy" by Dawkins (1979). Washburn (1978) argued that since even unrelated members of a species share the great majority of their genes, the logical extension of Hamilton's rule should be near universal altruism. As Dawkins points out, the error in this reasoning lies in the the fact that in unrelated individuals, the shared genes are not identical by descent.

To complicate matters further, r is often defined not as the proportion of genes identical by descent, but as the *probability* that a gene at a given locus will be identical by descent. These two definitions are employed more or less interchangeably by many authors, not least among them Hamilton (1964, 1975) and Dawkins (1979). Admittedly, in the absence of pleiotropy, the two definitions are equivalent. To use them interchangeably, however, invites a dangerous confusion between selection at the individual level and selection at the gene level. The notion of r as a *proportion* of genes identical by descent suggests an individual attempting to ensure that the greatest number of his

genes are replicated, without regard to which genes are replicated and in what multiplicity. On the other hand, the notion of r as a probability of finding a gene identical by descent at a given locus suggests a single gene attempting aid in the creation of replicas of itself.

When the emphasis is placed on the individual, rather than the gene, Hamilton's rule is generally stated as follows:

Hamilton's Rule (first version): If individual A shares a proportion of r genes identical by descent with individual B, then it is adaptive for A to perform an action which benefits B by b units of fitness at a cost of c units to himself if and only if $rb > c$.

This version of the rule, simple as it may seem, raises troubling philosophical questions. What, if any, role should by played by the *function* of genes? Should genes with more important function be given greater weight in the proportion? If so, what criteria should be used to determine the importance of function? Should junk DNA be counted in the proportion, despite its lack of function? If a gene undergoes a mutation which has no effect on its function (for example, by changing a nucleotide in such a way that it still codes for the same amino acid), should it still be considered "identical by descent?" The fundamental difficulty here is that it is not entirely clear why the action A is supposed to perform is actually adaptive.

The way around these philosophical pitfalls is to use a gene-based version of Hamilton's rule. We define a *Hamiltonian gene* as a gene that determines circumstances under which its carrier will act altruistically toward kin, in terms of the r, b, c, and possibly other parameters. Some simple examples of Hamiltonian genes are "always be altruistic," "always be selfish," "only be altruistic when $rb > c$," "only be altruistic when $rb > 2c$," "only be altruistic toward members of the opposite sex," etc. Of course, no single gene is solely responsible for determining behavior toward kin. A more realistic picture is that all other genes are being held constant, and the Hamiltonian gene influences some relatively simple behavior. For example, if birds have a tendency to feed things that squawk in their nest, the Hamiltonian gene might cause young birds to stay in the nest until after the hatching of the subsequent brood, with the result that elder brothers would help feed younger brothers (Dawkins, 1979). Now, fixing a particular locus on the genome and holding all other genes constant, various Hamiltonian genes will compete for the fixed locus. Thus we arrive at a gene-based version of Hamilton's rule:

Hamilton's Rule (second version): Under suitable conditions (slow selection, random mating, etc.), selection will favor the $rb > c$ gene over other Hamiltonian genes.

The r used here is the probabilistic r for the fixed locus under consideration. More precisely, if p is the probability that both Hamiltonian alleles are shared, and q is the probability that just a single Hamiltonian allele is shared, then r is defined as $p + \frac{1}{2}q$. The beauty of this second version of the rule is that it avoids making any claim about what is adaptive for individuals; instead, it simply makes a prediction about gene frequencies.

Though it vacillates between the two versions of the rule in its more qualitative sections, Hamilton's original (1964) treatment of kin selection is in fact concerned primarily with the second, gene-based version of the rule, as indicated in the paragraph beginning "Consider a single autosomal locus..." (p. 3). In particular, the mathematical treatment in Hamilton's original paper — still the most precise theoretical basis for the theory of kin selection — is derived entirely from considerations at a single locus, and thus applies exclusively to the gene-based version. Because the individual-based version of Hamilton's rule lacks a solid philosophical foundation, it is the gene-based version that lends itself best to rigorous analysis, and the present paper will concerned exclusively with the gene-based version.

2. Computation of r

Once the coefficient of relatedness r has been properly defined, the next logical question is one of computation. Using knowledge of the ancestry of two individuals A and B, how can their coefficient $r(A, B)$ be determined? Given the amount of attention that has been devoted to this question (Li & Sacks 1954; Kempthorne 1957; Haldane & Jayakar 1962), and the difficulty of adapting certain methods of computation to relationships involving inbreeding (Li & Sacks 1954), the following simple proposition should be of some interest. To compute coefficients of relationship, we employ an inductive algorithm. When a new individual, A_3, is born the offspring of A_1 and A_2, we compute his coefficients with all individuals currently living. Suppose B is one such individual. Because A_1, A_2 and B were all born before A_3, the coefficients $r(A_1, B)$ and $r(A_2, B)$ were computed previously, and we may

freely use these coefficients in the computation of the newborn's coefficient $r(A_3, B)$. As the following result shows, the newborn's coefficient is simply the average of the parents' coefficients.

Proposition 1. $r(A_3, B) = \frac{r(A_1,B)+r(A_2,B)}{2}$.

Proof. Let p_i $(i = 1, 2, 3)$ be the probability that individual A_i has both Hamiltonian alleles in common with B, and let q_i be the probability that A_i has only a single Hamiltonian allele in common with B. Of the nine possible scenarios obtained by allowing each parent to have either both alleles, one allele or no alleles in common with B, four can result in A_3 having both alleles in common:

$$p_3 = p_1p_2 + \frac{1}{2}p_1q_2 + \frac{1}{2}q_1p_2 + \frac{1}{4}q_1q_2.$$

Similarly, seven of the nine possibilities can result in A_3 having just one allele in common with B:

$$\begin{aligned}
q_3 &= p_1(1-p_2-q_2) + \frac{1}{2}q_1(1-p_2-q_2) + (1-p_1-q_1)p_2 \\
&\qquad + \frac{1}{2}(1-p_1-q_1)q_2 + \frac{1}{2}p_1q_2 + \frac{1}{2}q_1p_2 + \frac{1}{2}q_1q_2 \\
&= p_1 + \frac{1}{2}q_1 + p_2 + \frac{1}{2}q_2 - p_1p_2 - p_1q_2 - \frac{1}{2}p_1q_2 + \frac{1}{2}q_1p_2 - p_1p_2 - q_1p_2 \\
&\qquad - \frac{1}{2}q_1p_2 - \frac{1}{2}q_1q_2 + \frac{1}{2}p_1q_2 \\
&= r(A_1,B) + r(A_2,B) - 2p_1p_2 - p_1q_2 - q_1p_2 - \frac{1}{2}q_1q_2 \\
&= r(A_1,B) + r(A_2,B) - 2p_3.
\end{aligned}$$

Hence,

$$r(A_3, B) = p_3 + \frac{1}{2}q_3 = \frac{r(A_1,B) + r(A_2,B)}{2}. \quad \square$$

The standard values of r for brothers, nephews, cousins, etc. are easily derived from this proposition. First, assuming the parents A_1 and A_2 are unrelated, setting $B = A_1$ gives

$$r(A_3, A_1) = \frac{r(A_1,A_1) + r(A_2,A_1)}{2} = \frac{1}{2},$$

so parent and child are related by $\frac{1}{2}$, as expected. Similarly, setting $B = A_2$ shows that $r(A_3, A_2) = \frac{1}{2}$. Next, if A_3 has a brother, A_4, then setting $B = A_4$ gives

$$r(A_3, A_4) = \frac{r(A_1, A_4) + r(A_2, A_4)}{2} = \frac{\frac{1}{2} + \frac{1}{2}}{2} = \frac{1}{2}.$$

If A_3 has an uncle U, say the brother of A_1, then setting $B = U$ we obtain

$$r(A_3, U) = \frac{r(A_1, U) + r(A_2, U)}{2} = \frac{1}{4}.$$

If A_3 has a cousin C, the offspring of U, then A_1 is the uncle of C, so $r(A_1, C) = \frac{1}{4}$, giving

$$r(A_3, C) = \frac{\frac{1}{4} + r(A_2, C)}{2} = \frac{1}{8}.$$

Inbred relationships are easily computed as well. Suppose, for example, that the parents A_1 and A_2 are brother and sister. Then

$$r(A_3, A_1) = \frac{r(A_1, A_1) + r(A_2, A_1)}{2} = \frac{3}{4}$$

and

$$r(A_3, A_4) = \frac{r(A_1, A_4) + r(A_2, A_4)}{2} = \frac{\frac{3}{4} + \frac{3}{4}}{2} = \frac{3}{4},$$

showing that the inbred paternal and fraternal relationships have coefficient $\frac{3}{4}$.

3. Evolutionary Stability of Kin Altruism

Dawkins (1979) attempts to dispell, among other misunderstandings, the fallacy that "kin selection only works for rare genes." As Dawkins presents it, the fallacious argument runs as follows. If a gene encoding for kin selection is adaptive, it will spread to fixation; once this happens, the gene derives the same benefit from altruism toward unrelated individuals as it does from altruism toward kin; therefore, the natural consequence of a gene for kin selection spreading to fixation is universal altruism. Dawkins summarily disposes of this argument by showing that kin altruism is stable under invasion by universal altruism. There is, however, a more subtle line of reasoning

leading to the conclusion that "kin selection only works for rare genes," one which Dawkins fails to address. This second argument relies on an important feature of Hamilton's mathematical model: the treatment of fitness as a "conserved quantity." The resources comprising fitness are presumed to exist in fixed quantities, so that the population remains constant from generation to generation. One's gain in fitness, then, is another's loss. If a gene for kin altruism is common, then a gain in fitness for one carrier of the gene is likely to result in a loss for a different carrier, for a result of no net gain. It would seem, then, that the adaptiveness of a kin altruism gene decreases as the frequency of the gene increases.

If a gene for kin altruism spreads to fixation, then the conservation of fitness ensures that the altruistic behavior resulting from the gene can no longer confer on it any net benefit. If there is no longer any purpose to altruistic behavior in such a situation, perhaps selfish individuals could invade. The question, then, is not, as Dawkins asked, whether kin altruism can be invaded by universal altruism, but whether it can be invaded by *selfishness*. Intuitive considerations suggest that perhaps it cannot. Let G be a gene coding for kin altruism. Suppose that G has spread to fixation and consider the effect of the appearance of a selfish mutant H. The altruistic effects of G work only slightly to its benefit, since it comprises nearly the entire population. The mutant H, on the other hand, is a rare gene, hence ideally suited to the strategy of kin altruism; unfortunately, it is selfish, and fails to take advantage of this fact. In sum, neither G nor H employs an effective strategy. The slight benefits conferred to G by its kin altruism will in time permit it to repel the invading H, however.

If kin altruism cannot be invaded by selfishness, is there another strategy — perhaps some form of modified kin altruism, requiring, for example, that $rb > 2c$ — which can successfully invade? It is difficult to say. An argument that kin altruism is in fact evolutionarily stable might proceed as follows. If G, the gene fore kin altruism, has spread to fixation, then although kin altruism confers little benefit on the G itself, is the *ideal* strategy for mutants, who have rare genes. By definition, mutants must deviate from the kin altruists in some way, and therefore they are unable to employ what would be their ideal strategy. It is clear, then, that mutants are certainly at some disadvantage against G. It is not clear whether there exist mutants that are good enough to invade despite this disadvantage. If in fact kin altruism is an evolutionarily stable strategy, however, it appears to derive its stability not from any special benefit conferred on itself, but rather from depriving

mutants of *their* best strategy.

4. Gene Frequencies and Outbreeding

To make the above qualitative discussion somewhat more precise, let us find the ideal strategy for a Hamiltonian gene as a function not just of r, b and c, but also of its prevalence within the population. Denote this prevalence by λ. To be precise, if the Hamiltonian gene in consideration consists of two alleles, G_1 and G_2, we define λ to be the proportion of individuals in the population carrying both G_1 and G_2 plus one half the proportion carrying just one of the two alleles. If A is an individual carrying both alleles, then as we are considering just this particular locus, λ may be regarded as the ambient level of relatedness between A and the population at large. If λ is significantly greater than zero, this implies a high degree of ambient relatedness in the population, and random marriages (as Hamilton assumes in his model) will result in inbreeding, and hence in coefficients of relationship that are higher than expected. This possibility will be discussed in the following section; the present section will be concerned with outbreeding despite the nontrivial ambient relatedness λ.

It is often the case that strict outbreeding practices can be maintained only in relatively large populations with relatively low levels of ambient relatedness. This need not always be the case, however, as is demonstrated by the following example. Consider a hierarchy of three societies, A, B and C, constrained by the following marriage custom: Men in society A may marry women from any society, but men in societies B and C must marry women from C. Naturally, society A is polygynous, while B and C are monogamous. Descent is strictly patrilineal, so that children belong to the society to which their father belongs.

Suppose now that a Hamiltonian gene G not present in society C has frequency λ in society B. Notice that this gene may find its way into society A, but it can never be introduced into society C, since the men of C cannot marry women from A or B. Now, the marriage constraints are not sufficient to prevent inbreeding entirely, but they do prevent inbreeding on the part of male carriers of G in society B, since these men must marry women from C, who cannot carry G. It follows that the coefficient of relationship between two men in society B is exactly as given in Proposition 1. In particular, whereas inbred coefficients would vary with λ, the coefficients between men

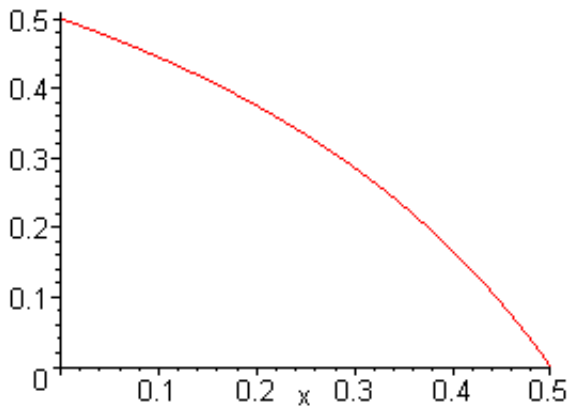

Figure 1: Effective r for brothers as a function of λ. When $\lambda = 0$, they are related by 0.5 as usual. At $\lambda = 0.5$, the brothers are effectively unrelated.

of society B are independent of λ. Consider now the effects of an action performed by a man M at a cost of c units to himself, and benefiting a second man N, related to M by the coefficient r, by b units. Since fitness is conserved, the net gain of $b - c$ units must be counterbalanced by a loss of $b - c$ units elsewhere; suppose for the time being that this loss is evenly distributed among the people of society B. Since these people are related to M on average by the coefficient λ, the corresponding loss in inclusive fitness to M is $(b - c)\lambda$. Thus M's net gain in inclusive fitness is

$$\Delta f = rb - c - (b - c)\lambda.$$

Therefore, M benefits from the action if and only if

$$\begin{aligned} & rb > c + (b - c)\lambda \\ \Leftrightarrow \quad & \frac{r - \lambda}{1 - \lambda}\, b > c. \end{aligned} \tag{1}$$

The quantity $\frac{r-\lambda}{1-\lambda}$ can be understood as the *effective* coefficient of relatedness between M and N, given the ambient relatedness λ. Figure 1 shows the effective relatedness of brothers as a function of λ, as λ varies from 0 to $\frac{1}{2}$.

The marriage customs of the three societies in this example violate Hamilton's assumption of random mating, and this example shows that Hamilton's rule can be quite sensitive to factors such as mating correlations. Even if we

allow random mating within the constraints of the marriage customs, the very fact that marriages between certain societies are prohibited is enough to drastically alter Hamilton's rule.

Admittedly, the assumption that the loss of fitness $b - c$ is distributed equally among the population is likely not always accurate. In many cases, especially if the population is large, the loss is probably distributed among a small number of neighbors of M, or at any rate, people who interact closely with M. In either case, given that most individuals live near and work with many of their kin, those who assume the loss $b - c$ are *more* likely to be close kin to M than someone in the population at large. Thus the estimate that the loss is distributed evenly throughout the population is a conservative one. In most real-world examples, the loss is probably concentrated more heavily on close kin, further reducing the benefit of the action in terms of inclusive fitness.

Before Hamilton's pioneering paper, Haldane had made similar observations concerning forms of kin selection other than parental care; but Haldane did not pursue his ideas because he assumed individuals would have no means of recognizing kin. Hamilton questioned this assumption, believing that factors such as proximity during youth could serve as rough unconscious estimators of r. If Hamilton's rule is to be modified to accommodate the gene frequency λ, however, Haldane's objection surely holds in full force. Gene frequencies are always in flux, and it is difficult to conceive of any means by which individuals could estimate λ. It follows that, for societies in which random mating does not hold perfectly (i.e., most real world societies), there may be no evolutionarily stable form of kin selection. If a given Hamiltonian gene is successful, its frequency in the population will increase until it no longer gives a good approximation to equation (1), at which point it will be superseded by another Hamiltonian gene. The dynamics of such fluctuations in gene frequencies may become quite complex.

5. Gene Frequencies and Inbreeding

If the previous section showed the fragility of Hamilton's rule, this section will demonstrate one of its resounding successes. Indeed, the following dramatic confirmation of Hamilton's rule came about despite the author's best intentions to find an exception to the rule. If, following Hamilton, we assume random mating, then in the context of a nontrivial ambient relatedness

λ, it follows that most matings will be inbred. Inbreeding results in coefficients of relationship that are higher than normal, and it happens that these higher coefficients serve to compensate precisely for the dampening effects prescribed by equation (1). To see this, we need the following result.

Proposition 2. *Suppose A and B are two individuals who, through kinship ties alone, would have coefficient of relatedness r in a society in which $\lambda = 0$. Then in a society in which $\lambda > 0$, they have coefficient $R = r + (1-r)\lambda$.*

Proof. We use an induction and Proposition 1. If A has parents A_1 and A_2 and the result is true for the parents, then

$$\begin{aligned} R &= \frac{R(A_1, B) + R(A_2, B)}{2} \\ &= \frac{r_1 + (1-r_1)\lambda + r_2 + (1-r_2)\lambda}{2} \\ &= \frac{r_1 + r_2}{2} + \left(\frac{2 - r_1 - r_2}{2}\right)\lambda \\ &= r + \left(1 - \frac{r_1 + r_2}{2}\right)\lambda \\ &= r + (1-r)\lambda. \quad \square \end{aligned}$$

Intuitively speaking, the first term, r, represents that portion of relatedness due to kinship ties, while the second term, $(1-r)\lambda$, represents that portion resulting from the ambient relatedness.

Now, setting $r = R$ in equation (1), we find that the *effective* coefficient of relatedness is exactly r:

$$\frac{R - \lambda}{1 - \lambda} = \frac{r + (1-r)\lambda - \lambda}{1 - \lambda} = \frac{r - r\lambda}{1 - \lambda} = r.$$

In other words, now that we have restored Hamilton's initial assumption of random mating, the effects of inbreeding precisely counteract those of ambient relatedness so that Hamilton's rule is again satisfied.

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