

# **THE EVOLUTION OF ALTRUISM BETWEEN SIBLINGS:**

## **WHY HAMILTON'S RULE WORKS<sup>1</sup>**

Robert Rowthorn

Economics Faculty  
Sidgwick Avenue  
Cambridge  
CB3 9DD  
UK

Tel (44) 1223-335230  
e-mail: [Bob.Rowthorn@econ.cam.ac.uk](mailto:Bob.Rowthorn@econ.cam.ac.uk)

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## Abstract

Hamilton's rule states that natural selection will cause an individual to value a sibling half as much as itself (50% altruism). The rule is normally justified on the grounds that siblings share half of their rare genes in common or that half of their genes are identical by descent. Neither of these arguments withstands close examination. This paper provides a more satisfactory justification for Hamilton's rule. Using a simple model of competition between altruistic alleles, it demonstrates mathematically how, under certain conditions, natural selection will lead to the 50% altruism between siblings predicted by this rule. Computer simulations are then used to explore what happens under more general conditions. In every case, the direction of evolution is towards 50% altruism. However, the pace of convergence is sometimes very slow, which suggests that in practice Hamilton's rule will be only a very rough approximation. These results are established without reference to the proportion of genes by descent or of rare genes that siblings share in common. The paper concludes by considering briefly some of the factors that might prevent the eventual victory of 50% altruism and allow alleles for more selfish and less selfish forms of behaviour to coexist permanently.

Keywords: Altruism, Kin-Selection, Hamilton's Rule

# THE EVOLUTION OF ALTRUISM BETWEEN SIBLINGS

Robert Rowthorn

In a celebrated paper Hamilton (1964) develops the theory of inclusive fitness as the basis for altruism between related organisms. In the case of siblings, he claims that natural selection will lead individuals to behave altruistically towards their siblings provided the cost to the donor does not exceed half the benefit to the recipient. This claim is justified on the grounds that siblings share half their genes in common, because they get half their genes from their mother and half from their father. If an individual carries  $x$  copies of a certain gene, Hamilton argues that its sibling will on average carry  $0.5x$  copies, and hence this individual will maximise the inclusive fitness of its genes by treating a sibling as if it were half as valuable as itself. Although at first sight appealing, this argument is wrong. It is not in general the case that "if an individual carries a certain gene the expectation that a random sib will carry a replica of it is...one half" (Hamilton p. 2). The fact that siblings inherit half their genes from each parent does not mean that the expectation that a random sib carries a replica of a particular gene is equal to one half. This is only true if the gene in question is rare. If a gene is widespread in the population, the number of copies carried by an individual and its siblings will on average be quite similar. With minor variations Hamilton's argument has been repeated in numerous academic writings, textbooks and lecture notes<sup>2</sup>. Most of these expositions modify this argument by casting it in terms of common descent. Genes are said to be "identical by descent" if one is descended from the other or they are both descended from a common ancestor. Except in the case of identical twins, triplets and the like, if parents are unrelated to each other then on average siblings share half of their genes by descent. Most expositions take it as self-evident that this fact alone explains why an individual should value a sibling half as much as itself. However, as the following highly artificial example shows, such an inference may not be justified. Suppose there is a gene for altruism between siblings which has a number of alleles,  $A$ ,  $B$ ,  $C$  etc. Suppose also that there are initially no heterozygotes in the population and that mating is fully assortative so that like breed only with like. Under these conditions, mating pairs are of the form  $AA \times AA$ ,  $BB \times BB$ ,  $CC \times CC$  etc. and siblings all carry the same number of each allele (2 or 0) as their parents. Siblings with  $AA$  parents are all  $AA$ -types, those with  $BB$  parents are all  $BB$ -types, and so on. From this point of view, siblings are genetically identical and it makes no difference to the propagation of an allele which siblings survive or reproduce. In terms of inclusive fitness, an offspring and its sibling are equally valuable and the allele that maximises the net benefit  $b-c$  will spread at the expense of the other alleles. This contradicts the genes by descent interpretation of Hamilton's rule, which implies that the allele with highest value of  $1/2b-c$  will triumph.

The coefficient of  $1/2$  in the conventional interpretation of Hamilton's rule is based on the proportion of genes which are identical by descent in an offspring and its sibling. This proportion is calculated using the method of labelled genes (alleles in this case). In the above example, consider a mating pair  $A_1A_2 \times A_3A_4$ , where each copy of  $A$  is labelled to distinguish it from the others. Let  $A_1A_3$  be an arbitrary offspring. Such an offspring has four equiprobable types of sibling containing the following distinct combinations of labelled alleles:  $A_1A_3$ ,  $A_1A_4$ ,  $A_2A_3$ ,  $A_2A_4$ . These siblings share 2, 1, 1 and 0 labelled alleles, respectively, in common with our  $A_1A_3$  offspring. Weighting these numbers by their probability of occurrence, our offspring contains exactly 1 *labelled* copy of  $A$  in

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<sup>2</sup> See for example Maynard Smith (1995, pp. 195-197), Williams (1996, pp. 43-51), Neal (2004, pp 322-326).

common with its average sibling. This is one half of the total number of labelled copies of *A* that this offspring carries. Thus, our offspring shares only 50% of its labelled alleles ('genes by descent') with its sibling but 100% of its unlabelled alleles. What matters for inclusive fitness are unlabelled alleles, and the genes by descent method may go wrong because it is based on labelled alleles.

Grafen (1985) has explored this issue at length. Basing himself on Crozier (1970), he points out that what matters for the theory of inclusive fitness is the degree of genetic similarity between donor and recipient<sup>3</sup>. Under some conditions, the genes-by-descent measure of relatedness provides a good indication of genetic similarity and can be taken as a proxy for the latter. However, as the above example shows, this is not always the case and the two should not be regarded as equivalent. Using the Price equation, Grafen derives a measure of genetic similarity and he explores the relationship between this measure and the conventional genes-by-descent measure.

The present paper accepts the standard proposition of kin selection theory with regard to sibling altruism. Its aim is to provide a more satisfactory explanation as to why such altruism should evolve through natural selection. To this end, we use a simple model of competition between altruistic genes, or as we shall call them from now on, alleles. Each allele codes for a particular degree of altruism towards siblings and the victor is the allele which most successfully propagates itself. Our major result is that the allele that expresses itself in a way consistent with Hamilton's rule will propagate at the expense of other alleles that are more or less generous towards their siblings. Our model is inspired by that of Hamilton, but there are a number of differences. Of these, the most important are our explicit analysis of the dynamics of allele competition and our use of genetic similarity, rather than relatedness, as our central concept. Our measure of similarity is identical to that suggested by Grafen (1985, p.41), although it is derived by a different method.

The structure of the paper is as follows. The first section lays out the model and derives the basic replicator equations when two alleles are competing against each other. The next section examines the long-run behaviour of allele frequencies and demonstrates how, under certain conditions, natural selection will lead to the 50% altruism predicted by the standard theory of kin selection. I was not able to prove this under more general conditions, so the issue is explored using computer simulations. In every simulation without exception, including many not reported in this paper, the direction of evolution is towards 50% altruism between siblings. Thus, even though the paper does not provide a general proof that siblings tend to display 50% altruism towards each other, it does show how altruism can be explained without relying on the fact that siblings share in common 50% of their genes by descent. It also provides strong, though not mathematically conclusive, evidence that natural selection will lead to the degree of altruism predicted by the standard theory of kin selection. The paper concludes by considering briefly some of the factors that may prevent the complete victory of 50% altruism and allow alleles for more selfish forms of behaviour to persist.

## **A Simple Model**

To analyse the evolution of altruism we shall use the following simple model. Mating is random and adults all breed simultaneously. They bring up their offspring and then die, at which point their offspring become adults and breed. Altruism between siblings is feasible either because offspring can identify their siblings or because they are segregated from non-offspring during their upbringing. There is no altruism between adult siblings, but there is a gene for altruism between juvenile siblings. The willingness of an offspring to help its siblings is genetically programmed and

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<sup>3</sup> Grafen's argument is summarised in Dawkins (1989).

varies from one offspring to another. One offspring may be willing to sacrifice a lot in order to help a siblings, whereas for another offspring even a minimal sacrifice may be too much. At any moment of time there are at most two alleles of the altruism gene present in the population ( $A$  and  $a$ ), and the extent of altruism exhibited by an offspring depends on which combination of these alleles it carries. Genetic transmission of altruism is assumed to follow Mendelian principles, so there is no genomic imprinting and the parental source of an allele is therefore irrelevant.

Depending on the nature of their parents, there are six distinct types of sibling group which are labelled  $m_1, m_2 \dots m_6$ . These types differ according to the proportions of offspring which contain 0, 1 and 2 copies of  $A$  (see table 1). Let  $d_i(s)$  be the average proportion of offspring which contain  $s$  copies of  $A$  in a group of type  $m_i$ . The absolute number of such offspring is equal to  $n_i(s) = d_i(s)n$ . All offspring are equally fit at birth, but their survival chances are influenced by their interactions with their siblings. Let  $b_s$  be the total altruistic benefit that an offspring of type  $s$  confers on the offspring in its sibling group – where the unit of measurement is survival probability. Summing over the group as a whole, the aggregate benefit which the offspring as a whole receive is equal to  $\sum_s b_s n_i(s)$ . It is assumed that all  $n$  offspring in the group share equally in this aggregate, including the offspring which is providing the benefit. Hence each offspring receives a total benefit equal to

$$\frac{\sum_s b_s n_i(s)}{n} = \sum_s b_s d_i(s) \quad (1)$$

Consider an offspring that contains  $r$  copies of  $A$ . Let  $c_r$  be the cost it incurs in contributing to group benefits. Taking both costs and benefits into account, the relative fitness of such an offspring is therefore as follows

$$f_i(s) = 1 + \sum_s b_s d_i(s) - c_r \quad (2)$$

Tables 1 and 2 lay out the mathematics of selection in the present model. In line with conventional notation,  $Q$ ,  $H$  and  $P$  denote the proportion of parents containing 0, 1 and 2 copies of the  $A$  allele,  $p = P + H/2$  denotes the frequency of this allele in the population,  $q = 1 - p$  is the frequency the other allele. The equivalent magnitudes following selection are indicated by an asterix.

**Table 1: Basic Features of Mating and Selection**

Name $m_i$	Mating Pair	W't $w_i$	Offspring Frequency in Sibling Group Before Selection			Offspring Frequency in Population Before Selection			Relative Fitness		
			$d_i(s)$			$w_i \times d_i(s)$			$f_i(s)$		
			s = 0	s = 1	s = 2	s = 0	s = 1	s = 2	s = 0	s = 1	s = 2
$m_1$	AA × AA	$P^2$	0	0	1	0	0	$P^2$	0	0	$(1 + b_2 - c_2)$
$m_2$	AA × Aa	2PH	0	½	½	0	PH	PH	0	$\left(1 + \frac{b_1}{2} + \frac{b_2}{2} - c_1\right)$	$\left(1 + \frac{b_1}{2} + \frac{b_2}{2} - c_2\right)$
$m_3$	AA × aa	2PQ	0	1	0	0	2PQ	0	0	$(1 + b_1 - c_1)$	0
$m_4$	Aa × Aa	$H^2$	¼	½	¼	$\frac{H^2}{4}$	$\frac{H^2}{2}$	$\frac{H^2}{4}$	$\left(1 + \frac{b_0}{4} + \frac{b_1}{2} + \frac{b_2}{4} - c_0\right)$	$\left(1 + \frac{b_0}{4} + \frac{b_1}{2} + \frac{b_2}{4} - c_1\right)$	$\left(1 + \frac{b_0}{4} + \frac{b_1}{2} + \frac{b_2}{4} - c_2\right)$
$m_5$	Aa × aa	2HQ	½	½	0	HQ	HQ	0	$\left(1 + \frac{b_0}{2} + \frac{b_1}{2} - c_0\right)$	$\left(1 + \frac{b_0}{2} + \frac{b_1}{2} - c_1\right)$	0
$m_6$	aa × aa	$Q^2$	1	0	0	$Q^2$	0	0	$(1 + b_0 - c_0)$	0	0
Sum		1				$p^2$	$2pq$	$q^2$			

Note:  $p = P + H/2$ ,  $q = Q + H/2$ ; also  $p + q = 1$ ,  $P + H + Q = 1$  and  $p^2 + 2pq + q^2 = 1$ .

**Table 2: Relative Frequencies after Selection**

Name $m_i$	Mating Pair	Offspring Frequency in Population <i>After</i> Selection $\frac{w_i \times d_i(s) \times f_i(s)}{K}$		
		s = 0	s = 1	s = 2
$m_1$	AA × AA	0	0	$\frac{P^2 (1 + b_2 - c_2)}{K}$
$m_2$	AA × Aa	0	$\frac{PH \left( 1 + \frac{b_1}{2} + \frac{b_2}{2} - c_1 \right)}{K}$	$\frac{PH \left( 1 + \frac{b_1}{2} + \frac{b_2}{2} - c_2 \right)}{K}$
$m_3$	AA × aa	0	$\frac{2PQ(1 + b_1 - c_1)}{K}$	0
$m_4$	Aa × Aa	$\frac{H^2 \left( 1 + \frac{b_0}{4} + \frac{b_1}{2} + \frac{b_2}{4} - c_0 \right)}{K}$	$\frac{H^2 \left( 1 + \frac{b_0}{4} + \frac{b_1}{2} + \frac{b_2}{4} - c_1 \right)}{K}$	$\frac{H^2 \left( 1 + \frac{b_0}{4} + \frac{b_1}{2} + \frac{b_2}{4} - c_2 \right)}{K}$
$m_5$	Aa × aa	$\frac{HQ \left( 1 + \frac{b_0}{2} + \frac{b_1}{2} - c_0 \right)}{K}$	$\frac{HQ \left( 1 + \frac{b_0}{2} + \frac{b_1}{2} - c_1 \right)}{K}$	0
$m_6$	aa × aa	$\frac{Q^2 (1 + b_0 - c_0)}{K}$	0	0
Sum		$Q^*$	$H^*$	$P^*$

Note:  $K = \sum_i \sum_s [d_i(s) \times w_i \times f_i(s)]$  is chosen so as to ensure that  $P^* + H^* + Q^* = 1$ . Post selection allele frequencies are given by  $p^* = P^* + H^*/2$ ,  $q^* = Q^* + H^*/2$ .

Let  $\Delta p = p^* - p$  and denote the changes in the frequency of the  $A$  allele from one generation of parents to the next. It is shown in the appendix that

$$\Delta p = \frac{\left[ \left( \frac{1-\varepsilon}{2} \right) b_A - c_A \right] - \left[ \left( \frac{1-\varepsilon}{2} \right) b_a - c_a \right]}{W} \quad (3)$$

where  $W$  is some positive number and

$$\varepsilon = \frac{H}{2pq} - 1 \quad (4)$$

This quantity indicates the extent to which the share of heterozygotes in the parental population exceeds the Hardy-Weinberg value of  $2pq$ . Such a deviation reflects the impact of sibling altruism on the differential survival of the existing generation of parents during childhood. Equation (3) indicates that natural selection favours the allele with the highest value of  $\left( \frac{1-\varepsilon}{2} \right) b - c$ .

### **Genetic Similarity**

Consider an offspring that exhibits altruism of type  $A$  and let  $E(\text{self} | A)$  and  $E(\text{sib} | A)$  be the expected number of copies of  $A$  carried by this offspring and a random sibling, respectively. It is shown in the appendix that

$$\frac{E(\text{sib} | A) - 2p}{E(\text{self} | A) - 2p} = \frac{1-\varepsilon}{2} \quad (5)$$

where  $2p$  is the number of copies of  $A$  that are carried by an average member of the population. A similar formula holds for the other allele  $a$ . The above ratio provides a measure of genetic similarity between an offspring and its siblings<sup>4</sup>. The numerator is the expected “excess” number of copies of  $A$  in an offspring that exhibits altruism of degree  $A$  (as compared to the number carried by an average member of the population). The denominator is the expected excess of  $A$  in an arbitrary sibling of this offspring. If mating is random and parents are in Hardy-Weinberg proportions, then  $\varepsilon = 0$  and the above measure of genetic similarity is equal to one half. Under these conditions, our measure of similarity coincides with the conventional measure of relatedness based on genes by descent. However, this is not always the case. For example, if heterozygotes all die before they can breed, then parents are not in Hardy-Weinberg proportions,  $\varepsilon = -1$  and our measure of similarity is equal to  $1$ . Conversely, if all homozygotes die before they can breed and  $p = 0.5$ , then parents are again not in Hardy-Weinberg proportion, but in this case  $\varepsilon = 1$  and our measure of similarity is equal to  $0$ . In all of these examples, siblings share exactly half of their genes by descent.

We can now give equation (3) an interpretation that links it directly to the standard literature on kin selection. Suppose that in calculating inclusive fitness the benefits of altruism are weighted according to the measure of similarity given in equation (5). Then  $\left( \frac{1-\varepsilon}{2} \right) b - c$  is the estimated

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<sup>4</sup> The definition of similarity given here coincides with that given by Grafen (1985, p.41).

net gain accruing to an offspring and its siblings resulting from altruistic behaviour by this offspring. Equation (3) tells us that the allele which yields the largest net gain, so measured, will increase in frequency. The equation is example of "Hamilton's Rule" in action, although it is based genetic similarity rather than relatedness. It holds even when parents are not in Hardy-Weinberg proportions.

### **An Approximate Formula**

The variable  $\varepsilon$  indicates the extent to which sibling altruism amongst parents caused the proportion of heterozygotes to deviate from the Hardy-Weinberg share that obtained when they were born. If the costs and benefits of altruism are sufficiently small, then  $\varepsilon$  is close to zero, and we can use the following approximate version of equation (5)

$$\Delta p = \frac{\left[ \left( \frac{1}{2} \right) b_A - c_A \right] - \left[ \left( \frac{1}{2} \right) b_a - c_a \right]}{W} \quad (6)$$

In this case, allele A will drive out allele a provided

$$\left[ \left( \frac{1}{2} \right) b_A - c_A \right] > \left[ \left( \frac{1}{2} \right) b_a - c_a \right] \quad (7)$$

This equation tells us that natural selection will tend to increase the value of  $\frac{1}{2}b - c$ . If there is a sequence of mutations affecting the degree of altruism, the ratchet effect will ensure that only those mutations which cause  $\frac{1}{2}b - c$  to increase will be successful. Thus, by a gradual hill climbing process altruism will converge toward the value which maximises  $\frac{1}{2}b - c$ .

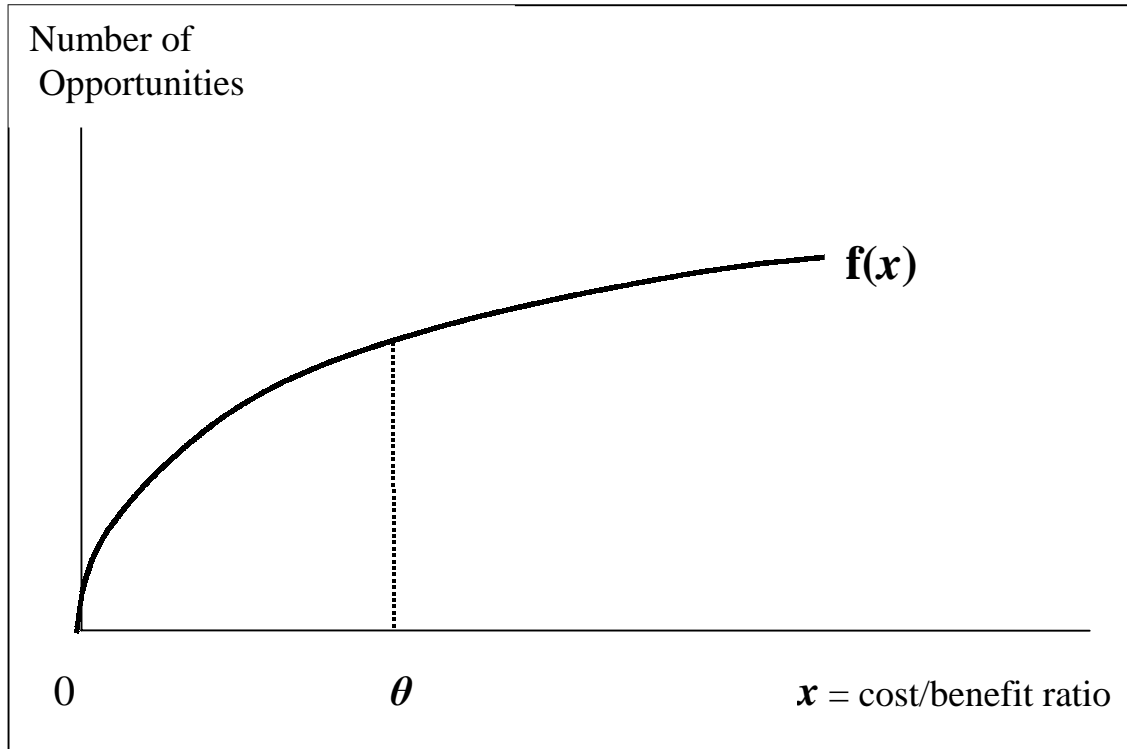
### **An Example**

To illustrate the above findings, let us be more specific about the nature of altruism. Suppose that each offspring faces a variety of opportunities to act altruistically towards its siblings, and that each such act confers a benefit of  $\lambda$  on the sibling group as a whole at a cost of  $\lambda x$  to the donor. An offspring will be said to exhibit altruism of type  $\theta$  if it behaves altruistically whenever  $x \leq \theta$  but not otherwise. Thus, the greater the value of  $\theta$  the more altruistic is the offspring in question.

We shall assume that the altruistic opportunities facing an offspring can be specified by a continuous function  $f(x)$  as shown in Figure 1. An offspring of type  $\theta$  will perform all acts of altruism for which  $x \leq \theta$  and hence the total number of such acts this offspring performs is equal to  $\int_0^\theta f(x) dx$  and the total benefit conferred on the siblings group is given by

$$b(\theta) = \lambda \int_0^\theta f(x) dx \quad (8)$$

**Figure 1: Altruistic Opportunities Ranked by Cost-Benefit Ratio**



The total cost to the donor of these acts is equal to

$$c(\theta) = \lambda \int_0^{\theta} x f(x) dx \quad (9)$$

Note that  $c(\theta)/b(\theta)$  is the average cost-benefit ratio of altruistic acts, whereas  $\theta$  is the marginal cost-benefit ratio beyond which an altruist of type  $\theta$  is unwilling to go.

The evolution of allele frequencies can be found by substituting the above expressions for  $b(\theta)$  and  $c(\theta)$  in equation (3). If  $\varepsilon$  is close to zero, natural selection will move  $\theta$  towards the value  $\theta^*$  that maximises the function  $\frac{1}{2}b(\theta) - c(\theta)$ . Since  $b$  and  $c$  are continuous, the first order maximum condition is then as follows:

$$\begin{aligned} 0 &= \frac{1}{2}b'(\theta^*) - c'(\theta^*) \\ &= \left(\frac{1}{2} - \theta^*\right) \lambda f(\theta^*) \end{aligned} \quad (9)$$

Thus,  $\theta^* = 1/2$ . Natural selection will therefore cause altruism to evolve to the point where each sibling counts half as much as the individual. Note that this conclusion assumes that  $\varepsilon$  is small enough to be ignored. This is likely to be the case where the scale of sibling altruism (as indicated by  $\lambda$ ) is small, since altruism will then have only a very minor effect on the proportion of heterozygotes. However, it is less obvious that  $\varepsilon$  can be ignored when the effects of altruism are large. It is then conceivable that evolution could lead to convergence on some value of  $\theta$  different from  $1/2$ . To explore what happens in this case we shall now present some simulation results. These results suggest that even when  $\lambda$  is extremely large,  $\varepsilon$  is still very small and  $\theta$  converges to approximately  $1/2$ .

## **Simulation**

To simulate the evolution of altruism, we assume that  $f(x) = x^\alpha$ . The benefits and costs of altruism are as follows:

$$b(\theta) = \lambda \int_0^\theta x^\alpha dx = \frac{\lambda \theta^{1+\alpha}}{1+\alpha} \quad (10)$$

$$c(\theta) = \lambda \int_0^\theta x.x^\alpha dx = \frac{\lambda \theta^{2+\alpha}}{2+\alpha} \quad (11)$$

If  $\alpha < 0$  there are frequent occasions when the cost to benefit ratio of altruism is low, but if  $\alpha > 0$  such occasions are comparatively rare.

The parameter  $\lambda$  determines the absolute scale of costs and benefits. The larger the value of  $\lambda$ , the greater is the impact of altruism on both donor and recipient. As an illustration, suppose that  $\alpha = 0$ , and consider a situation in which there is initially no altruism and each adult pair has six offspring. If the population is stationary, two of these offspring will on average survive, and the typical offspring will have a survival probability of 33.3%. Suppose that a lone altruist with  $\theta = 0.5$  appears amongst the offspring. If  $\lambda = 0.1$ , the effect of such altruism is to reduce the survival probability of the donor from 33.3 percent to 32.9 percent, whilst raising that of the average sibling to 33.6 percent. With  $\lambda = 1$ , the survival probabilities of donor and sibling become 29.1 percent and 36.6 percent respectively.

One striking feature of the simulations is how quickly Harvey-Weinberg proportions are established amongst parents. Starting from the most extreme initial position, within a couple of generations the value of  $\varepsilon$  is invariably very small and normally of the order  $10^{-4}$  or less. As a result, initial deviations from Hardy-Weinberg proportions have a negligible long-run effect on the direction of evolutionary competition which always selects the allele with the greatest value of  $1/2b(\theta) - c(\theta)$ . This is true no matter what the values of  $\alpha$  and  $\lambda$ , and it remains true even when the invader is recessive. The fact that every simulation without exception produces this outcome suggests that it must be mathematical necessity. However, I have not been able to prove this to be the case.

Table 3 shows how  $\lambda$  influences the course of evolution when a dominant allele  $A$  with  $\theta_A = 0.5$  invades a population with some given value of  $\theta_a$ . It is assumed that  $\alpha = 0$  and that the initial

frequency of the invader is 1.0%<sup>5</sup>. In each case, the invader takes over, but the pace of change varies greatly depending on  $\lambda$  and the incumbent value  $\theta_a$ . Against an allele which codes for complete selfishness ( $\theta_a = 0$ ) the allele  $\theta_A = 0.5$  takes over quite rapidly even when  $\lambda$  is small. For example, with  $\lambda = 0.1$ , the share of the invader reaches 99% in about ten thousand generations. If the difference between  $\theta_A$  and  $\theta_a$  is small, then it may require a long time for the invader to take over. For example, suppose a dominant invader has  $\theta_A = 0.5$  and a recessive incumbent has  $\theta_a = 0.45$ . Then, with  $\alpha = 0$  and  $\lambda = 0.1$ , it requires almost ten thousand generations for the invader to increase its allele frequency from 1% to 3%, and almost a million generations to reach 99%. With  $\lambda = 1.0$  the process is initially much faster and the invader's allele frequency reaches 78% after ten thousand generations. Even so, well over a hundred thousand generations are required to reach 99%.

To check on the robustness of these results, simulations were also made using different values of  $\alpha$ . As can be seen from Table 4, variations in this parameter have no effect on the ultimate outcome, and over the range considered they have little effect on the speed of convergence.

The evolution of altruism may be viewed as a hill-climbing process in which a sequence of mutations occur, in which each has a higher value of  $\frac{1}{2}b - c$  than its predecessor which is eventually driven to virtual extinction. In the course of time, the parameter  $\theta$  will eventually converge on the value of 0.5 predicted by the conventional theory of kin selection. Our simulations suggest that it may take hundreds of thousands, or even millions, of generations to get really close to the final equilibrium. However, even a few million generations is not so long when viewed against the broad sweep of history, since altruism is likely to be passed down from one species to the next in the evolutionary chain.

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<sup>5</sup> All simulations reported here assume that parents are initially in Hardy-Weinberg proportions. However, this is not an important assumption and simulations starting from other initial positions yield identical results.

**Table 3. Frequency of dominant  $\theta = 0.5$  after 10 thousand generations (for various  $\lambda$ )**

<u>Incumbent <math>\theta</math></u>	Selection Strength $\lambda =$		
	0.1	1.0	10.0
0.0	.9911	.9989	.9996
0.2	.9725	.9969	.9989
0.4	.4002	.9668	.9899
0.45	.0320	.7806	.9531
0.55	.0318	.7764	.9527
0.6	.3958	.9667	.9899
0.8	.9725	.9969	.9989
1.0	.9911	.9989	.9996

Note: this table assumes that  $\alpha = 0$  and that the initial frequency of  $\theta = 0.5$  is equal to  $0.01$ .

**Table 4. Frequency of dominant  $\theta = 0.5$  after 10 thousand generations (for various  $\alpha$ )**

<u>Incumbent <math>\theta</math></u>	Altruism opportunity parameter $\alpha =$		
	-0.5	0.0	0.5
0.0	.9995	.9989	.9975
0.2	.9973	.9969	.9951
0.4	.9651	.9668	.9548
0.45	.7495	.7806	.6855
0.55	.7145	.7764	.7128
0.6	.9587	.9667	.9615
0.8	.9958	.9969	.9668
1.0	.9984	.9989	.9990

Note: this table assumes that  $\lambda = 1.0$  and that the initial frequency of  $\theta = 0.5$  is equal to  $0.01$ .

## Polymorphism

It has been assumed so far that mating is random and that all genotypes are equally fit apart from the immediate effects of sibling altruism. Under these conditions, it is normally the case that one type of altruism will triumph in pair-wise competition. However, by relaxing these assumptions it is possible to generate a polymorphism in which multiple types of altruism coexist permanently. We shall consider briefly two mechanisms by which this may come about: dissortative mating and heterozygote advantage.

In the case of heterozygote advantage, heterozygotes have increased fitness either because they have a higher survival rate than homozygotes or are more fertile. Many examples of this phenomenon have been documented in the literature, including at least one associated a behavioural trait<sup>6</sup>. Thus, it is conceivable that such a phenomenon could arise in the case of sibling altruism. To model the effect of such behaviour we shall increase the fraction of heterozygotes by a fixed multiple using the following formula

$$\hat{H} = (1 + \mu)H \quad (12)$$

where  $\mu$  is a parameter indicating the degree of heterozygote advantage. Other types of couple are reduced in number accordingly.

In the case of dissortative mating we shall proceed as follows. The proportions of adults which manifest *a*-type and *A*-type altruism are  $Q$  and  $1-Q$  respectively. Under random-mating, a proportion  $M = 2Q(1-Q)$  of couples consist of one parent who is of type *a* and one parent of type *A*. We shall refer to these as “mixed” couples. Under dissortative mating, the proportion of mixed couples is higher than under random mating. This kind of behaviour does not seem to be very common, although a few cases have been documented in the literature<sup>7</sup>. In order to model the effect of such behaviour we shall increase the fraction of mixed couples by a fixed multiple using the following formula

$$\hat{M} = (1 + \phi)2Q(1-Q) \quad (13)$$

where  $\phi$  is a parameter indicating the departure from random mating. Other types of couple are reduced in number accordingly.

Figure 2 illustrates the effect of the above modifications. It is assumed that  $\alpha = 0$  and  $\lambda = 1$ . Under random mating without heterozygote advantage, the invading allele for altruism ( $\theta_A = 0.5$ ) eventually drives out the incumbent allele for selfishness ( $\theta_a = 0$ ) completely. This is indicated by the curve labelled “original”. Under dissortative mating, with  $\phi = 0.15$ , the share of the allele for selfishness initially falls but then stabilises at 48%. Since this allele is recessive, the long-run result is that around 23% ( $=100 \times 0.48^2$ ) of the offspring are selfish and the remainder are altruistic. Under heterozygote advantage, with  $\mu = 0.15$ , the share of the selfishness allele eventually stabilises at 39%. In the long-run around 15% ( $=100 \times 0.39^2$ ) of the offspring are therefore selfish and the remainder are altruistic. Thus, modest departures from the original assumptions yield

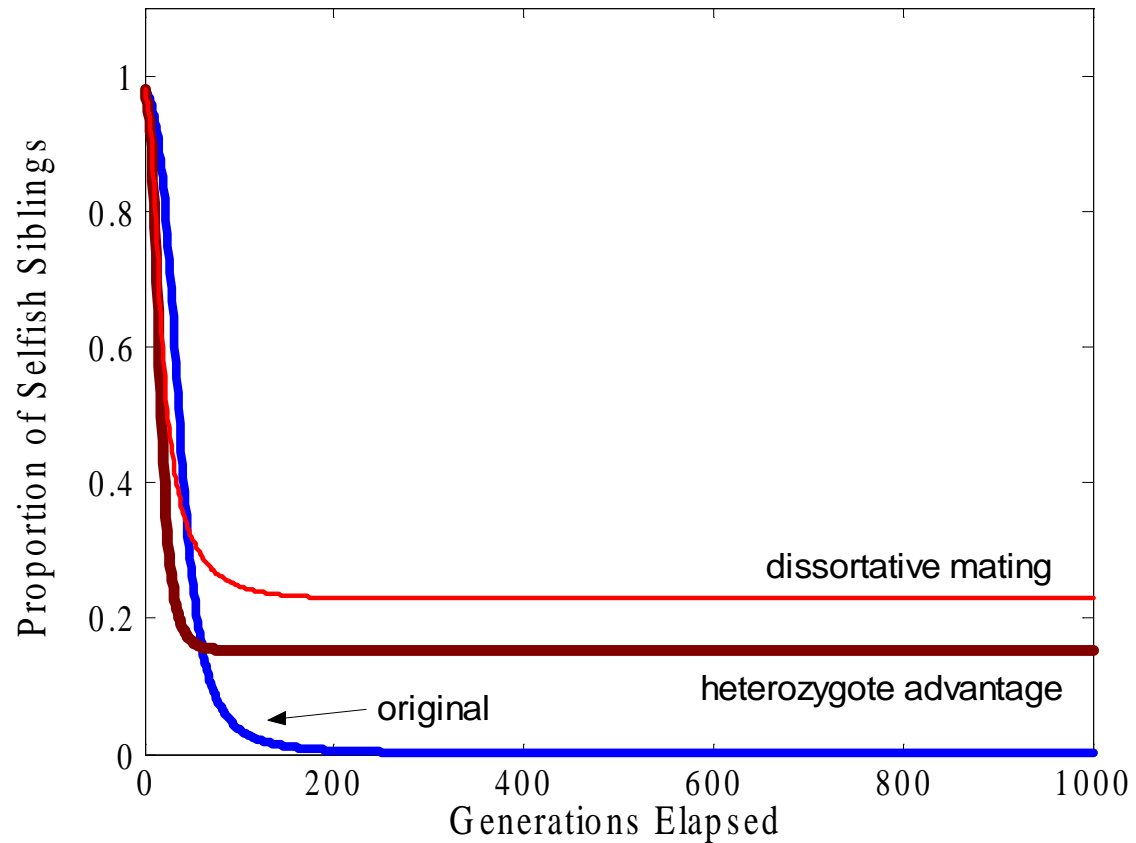
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<sup>6</sup> For example, sickle cell anaemia (Allison, 1955), birth weight (Saugstad, 1977), cystic fibrosis (Meindl, 1987), mating behaviour (Lank et al, 1995), plumage (Krüger et al, 2001), and deafness (Common et al, 2004),.

<sup>7</sup> For example, Laumann (1977), Wedekind et al (1995) and Irwin (2001).

polymorphisms in which a substantial proportion of offspring behave in a selfish fashion whereas other siblings are altruistic. This is not merely a transitory phenomenon during which natural selection works itself out, but is a permanent feature.

Figure 2: Coexistence of Altruistic and Selfish Behaviour



**Conclusions**

This paper has demonstrated how the evolution of sibling altruism can be explained without reference to the widespread, and misleading, proposition that siblings share in common half their rare genes or half their genes by descent. The paper has provided other grounds for believing, that natural selection will lead to the evolution of altruistic behaviour in which individuals behave as though siblings were half as valuable as themselves. Finally, it has shown how this finding may not hold if mating is non-random or there is heterozygote advantage. Under these conditions various degrees of altruism and selfishness may coexist permanently.

## Bibliography

- Allison, A. C. (19556), "The sickle-cell and haemoglobin C. genes in some African populations", *Annals of Human Genetic*, 21:67-89
- Common, J.E.A, Di, W-L, Davies, D. and D.P Kelsell, "Further evidence for heterozygote advantage of GJB2 deafness mutations: a link with cell survival", *Journal of Medical Genetics*, 41: 573-575.
- Crozier, R. H. (1970), "Coefficients of Relationship and the Identity of Genes by Descent in Hymenoptera", *American Naturalist*, 104: 216-7
- Dawkins, R. (1989), *The Selfish Gene*, 2nd ed., Oxford University Press.
- Grafen, A. (1985), "A Geometric View of Relatedness", R. Dawkins and M. Ridley (eds), *Oxford Surveys in Evolutionary Biology*, 2: 28-89.
- Hamilton, W. D. (1964), "The genetical evolution of social behaviour (I and II)", *Journal of Theoretical Biology*, 7: 1-16 and 17-52.
- Irwin, R. E. (2001), "Field and allozyme studies investigating optimal mating success in two sympatric spring-ephemeral plants, *Trillium erectum* and *T. grandiflorum*", *Heredity*, 87: 178-189.
- Krüger, O., Lindstrom, J. and Amos, W. (2001), "Maladaptive mate choice maintained by heterozygote advantage", *Evolution* 55: 1207- 1214.**
- Lank, D. B. C., Smith, C. M, Hanotte, O., Burker, T. and F. Cooker (1995), "Genetic Polymorphism for alternative mating behaviour in lekking male ruff *Philomachus pugnax*", *Nature*, 378: 59-62.
- Laumann, E. O. (1999), "Racial/Ethnic Group Differences in the Prevalence of Sexually Transmitted Diseases in the United States: A Network Explanation", *Sexually Transmitted Diseases*, 26(5): 250-261.
- Maynard Smith, J. (1995), *The Theory of Evolution*, Canto ed., Cambridge, Cambridge University Press.
- Meindl, R. (1987), "Hypothesis: A selective advantage for cystic fibrosis heterozygotes", *American Journal of Physical Anthropology*, 74:39-45
- Neal, D. (2004), *Introduction to Population Biology*, Cambridge, Cambridge University Press.
- Saugstad, L. F. (1977), "Heterozygote advantage for the phenylketonuria allele", *Journal of Medical Genetics*, 14: 20-24
- Wedekind C., T. Seebeck, F. Bettens and J. Paepke (1995), "MHC-dependent mate preferences in humans", *Proceedings of the Royal Society of London B*, 260: 245-249.
- Williams, G.C. (1996), *Plan and Purpose in Nature*, London, Weidenfeld and Nicholson.

## Appendix

### Derivation of Equation (3)

From Table 2 in the text

$$KQ^* = \left( Q^2 + QH + \frac{H^2}{4} \right) (1 - c_0) + \left( Q^2 + \frac{QH}{2} + \frac{H^2}{16} \right) b_0 + \left( \frac{QH}{2} + \frac{H^2}{8} \right) b_1 + \left( \frac{H^2}{16} \right) b_2$$

$$KH^* = \left( QH + \frac{H^2}{2} + 2PQ + PH \right) (1 - c_1) + \left( \frac{QH}{2} + \frac{H^2}{8} \right) b_0 \\ + \left( \frac{QH}{2} + \frac{H^2}{4} + 2PQ + \frac{PH}{2} \right) b_1 + \left( \frac{H^2}{8} + \frac{PH}{2} \right) b_2$$

$$KP^* = \left( \frac{H^2}{4} + PH + P^2 \right) (1 - c_2) + \left( \frac{H^2}{16} \right) b_0 + \left( \frac{H^2}{8} + \frac{PH}{2} \right) b_1 + \left( \frac{H^2}{16} + \frac{PH}{2} + P^2 \right) b_2$$

Note that

$$\frac{H^2}{4} + PH + P^2 = \left( \frac{H}{2} + P \right)^2 = p^2$$

$$\frac{H^2}{4} + QH + Q^2 = \left( \frac{H}{2} + Q \right)^2 = q^2$$

$$QH + \frac{H^2}{2} + 2PQ + PH = 2 \left( \frac{H}{2} + Q \right) \left( \frac{H}{2} + P \right) = 2pq$$

Hence

$$K = KP^* + KH^* + KQ^* \\ = q^2(1 - c_0) + 2pq(1 - c_1) + p^2(1 - c_2) + \left( Q^2 + QH + \frac{H^2}{4} \right) b_0 \\ + \left( QH + \frac{H^2}{2} + 2PQ + PH \right) b_1 + \left( \frac{H^2}{4} + PH + P^2 \right) b_2 \\ = q^2(1 + b_0 - c_0) + 2pq(1 + b_1 - c_1) + p^2(1 + b_2 - c_2)$$

Since  $p^* = P + H^*/2$  it follows that

$$Kp^* = pq(1-c_1) + p^2(1-c_2) + \left(\frac{QH}{4} + \frac{H^2}{8}\right)b_0 + \left(\frac{H^2}{4} + \frac{3PH}{4} + PQ + \frac{QH}{4}\right)b_1 + \left(\frac{H^2}{8} + \frac{3PH}{4} + P^2\right)b_2$$

Thus,

$$\begin{aligned} K(p^* - p) &= pq(1-c_1) + p^2(1-c_2) + \left(\frac{QH}{4} + \frac{H^2}{8}\right)b_0 + \left(\frac{H^2}{4} + \frac{3PH}{4} + PQ + \frac{QH}{4}\right)b_1 + \left(\frac{H^2}{8} + \frac{3PH}{4} + P^2\right)b_2 \\ &\quad - p\left[p^2(1+b_2-c_2) + 2pq(1+b_1-c_1) + q^2(1+b_0-c_0)\right] \end{aligned}$$

### A dominant

Suppose that  $A$  is dominant over  $a$ . Then

$$b_0 = b_a, b_1 = b_2 = b_A$$

$$c_0 = c_a, c_1 = c_2 = c_A$$

Substituting in the above equation yields

$$\begin{aligned} K(p^* - p) &= pq(1-c_A) + p^2(1-c_A) + \left(\frac{QH}{4} + \frac{H^2}{8}\right)b_a + \left(\frac{3H^2}{8} + \frac{3PH}{2} + PQ + P^2 + \frac{QH}{4}\right)b_A \\ &\quad - p\left[p^2(1+b_A-c_A) + 2pq(1+b_A-c_A) + q^2(1+b_a-c_a)\right] \\ &= pq(1-c_A) + p^2(1-c_A) + \left(\frac{qH}{4}\right)b_a + \left(p - \frac{qH}{4}\right)b_A \\ &\quad - p\left[p^2(1+b_A-c_A) + 2pq(1+b_A-c_A) + q^2(1+b_a-c_a)\right] \end{aligned}$$

After simplification,

$$\frac{K}{pq^2}(p^* - p) = \left[\left(1 - \frac{H}{4pq}\right)b_A - c_A\right] - \left[\left(1 - \frac{H}{4pq}\right)b_a - c_a\right]$$

which can be written in the form

$$\Delta p = \frac{\left[ \left( \frac{1-\varepsilon}{2} \right) b_A - c_A \right] - \left[ \left( \frac{1-\varepsilon}{2} \right) b_a - c_a \right]}{W}$$

where  $\Delta p = p^* - p$ ,  $\varepsilon = \frac{H}{2pq} - 1$  and  $W > 0$ .

Since  $q = 1 - p$  and  $q^* = 1 - p^*$  it follows that  $\Delta q = q^* - q$ , and hence

$$\Delta q = \frac{\left[ \left( \frac{1-\varepsilon}{2} \right) b_a - c_a \right] - \left[ \left( \frac{1-\varepsilon}{2} \right) b_A - c_A \right]}{W}$$

The above formulae for  $\Delta p$  and  $\Delta q$  have been derived on the assumption that  $A$  is dominant. Similar formulae can be derived when  $A$  is recessive.

### Derivation of Equation (5)

Let  $P$ ,  $H$  and  $Q$  denote the frequencies of  $AA$ ,  $Aa$  and  $aa$  at the time of mating. The frequencies of mating types are:

**Table A1. Mating pairs**

name	pairing	frequency
$m_1$	$AA \times AA$	$P^2$
$m_2$	$AA \times Aa$	$2PH$
$m_3$	$AA \times aa$	$2PQ$
$m_4$	$Aa \times Aa$	$H^2$
$m_5$	$Aa \times aa$	$2HQ$
$m_6$	$aa \times aa$	$Q^2$

Let  $s$  denote the number or copies of  $A$  in an offspring. The conditional probability  $\text{Prob}(s|m_i)$  is as follows

**Table A2.  $\text{Prob}(s|m_i)$**

	$m_1$	$m_2$	$m_3$	$m_4$	$m_5$	$m_6$
$s = 0$	0	0	0	1/4	1/2	1
$s = 1$	0	1/2	1	1/2	1/2	0
$s = 2$	1	1/2	0	1/4	0	0

The joint probability  $\text{Prob}(s, m_i)$  of  $s$  and  $m_i$  is as follows

**Table A3**  $\text{Prob}(s, m_i) = \text{Prob}(s|m_i)\text{Prob}(m_i)$

	$m_1$	$m_2$	$m_3$	$m_4$	$m_5$	$m_6$
$s = 0$	0	0	0	$H^2/4$	HQ	$Q^2$
$s = 1$	0	PH	2PQ	$H^2/2$	HQ	0
$s = 2$	$P^2$	PH	0	$H^2/4$	0	0

The conditional probability  $\text{Prob}(m_i|s)$  of  $m_i$  for given  $s$  is:

$$\text{Prob}(m_i|s) = \frac{\text{Prob}(s, m_i)}{\text{Prob}(s)}$$

where  $\text{Prob}(s)$  is the probability that an arbitrary offspring has  $s$  copies of  $A$ . Since mating is random, these probabilities satisfy the Hardy-Weinberg conditions:  $\text{Prob}(0) = q^2$ ,  $\text{Prob}(1) = 2pq$  and  $\text{Prob}(2) = p^2$  where  $p = P+H/2$  is the relative frequency of  $A$  and  $q = 1 - p$ . This yields,

**Table A4.**  $\text{Prob}(m_i|s) = \text{Prob}(s, m_i) / \text{Prob}(s)$

	$m_1$	$m_2$	$m_3$	$m_4$	$m_5$	$m_6$
$s = 0$	0	0	0	$(H/2q)^2$	$HQ/q^2$	$(Q/q)^2$
$s = 1$	0	$\frac{PH}{2pq}$	$\frac{PQ}{pq}$	$\frac{H^2}{4pq}$	$\frac{HQ}{2pq}$	0
$s = 2$	$\frac{P^2}{p^2}$	$\frac{PH}{p^2}$	0	$\frac{H^2}{4p^2}$	0	0

and

**Table A5.**  $\text{Prob}(r|s) = \sum_i \text{Prob}(r|m_i)\text{Prob}(m_i|s)$

	$r = 0$	$r = 1$	$r = 2$
$s = 0$	$\left(\frac{H}{4q}\right)^2 + \frac{HQ}{2q^2} + \left(\frac{Q}{q}\right)^2$	$\frac{1}{2}\left(\frac{H}{2q}\right)^2 + \frac{HQ}{2q^2}$	$(H/4q)^2$
$s = 1$	$\frac{H(H/4 + Q)}{4pq}$	$\frac{H}{4q} + \frac{Q[P + H/4]}{pq}$	$\frac{H}{4pq}\left[P + \frac{H}{4}\right]$
$s = 2$	$(H/4p)^2$	$\frac{1}{2}\left(\frac{H}{2q}\right)^2 + \frac{HP}{2q^2}$	$\frac{P}{p} + \left(\frac{H}{4q}\right)^2$

where  $\text{Prob}(r|s)$  is the conditional probability that a sibling carries  $r$  copies of  $A$  given that self carries  $s$  copies of the allele.

Note that the above probabilities are denoted in the text as follows:

$$\text{Prob}(m_i) = w_i, \text{Prob}(s|m_i) = d_i(s) \text{ and } \text{Prob}(s, m_i) = w_i \times d_i(s)$$

### A dominant

Suppose that  $A$  is dominant. Let  $\text{Prob}(s|A)$  be the conditional probability that an offspring exhibiting altruism of type  $A$  carries  $s$  copies of the allele  $A$ . Then

$$\text{Prob}(0|A) = 0$$

$$\text{Prob}(s|A) = \frac{\text{Prob}(s)}{\text{Prob}(1) + \text{Prob}(2)} \quad \text{for } s > 0$$

where  $\text{Prob}(0) = q^2$ ,  $\text{Prob}(1) = 2pq$  and  $\text{Prob}(2) = p^2$

Hence the expected number of copies of  $A$  carried by an offspring exhibiting altruism of type  $A$  is given by

$$\begin{aligned}
E(\text{self} | A) &= 0 \cdot \text{Prob}(0|A) + 1 \cdot \text{Prob}(1|A) + 2 \cdot \text{Prob}(2|A) \\
&= \frac{\text{Prob}(1) + 2\text{Prob}(2)}{\text{Prob}(1) + \text{Prob}(2)} \\
&= \frac{2pq + 2p^2}{2pq + p^2} \\
&= \frac{2}{q+1}
\end{aligned}$$

Thus,

$$\begin{aligned}
E(\text{self} | A) - 2p &= \frac{2}{q+1} - 2p \\
&= \frac{2q^2}{q+1}
\end{aligned}$$

Also,

$$\begin{aligned}
E(\text{sib} | A) &= \sum_r r \sum_s \text{Prob}(r|s) \text{Prob}(s|A) \\
&= \frac{\sum_r r [\text{Prob}(r|1)P(1) + \text{Prob}(r|2)P(2)]}{\text{Prob}(1) + \text{Prob}(2)} \\
&= \frac{\text{Prob}(1|1)\text{Prob}(1) + \text{Prob}(1|2)\text{Prob}(2) + 2[\text{Prob}(2|1)\text{Prob}(1) + \text{Prob}(2|2)\text{Prob}(2)]}{\text{Prob}(1) + \text{Prob}(2)} \\
&= \frac{\text{Prob}(1) - \text{Prob}(1|0)P(0) + 2[\text{Prob}(2) - \text{Prob}(2|0)\text{Prob}(0)]}{1 - \text{Prob}(0)} \\
&= \frac{2\{0.5\text{Prob}(1) + \text{Prob}(2) - [0.5\text{Prob}(1|0) + \text{Prob}(2|0)]P(0)\}}{1 - P(0)} \\
&= \frac{2\left\{pq + p^2 - \left[\frac{H^2}{8q^2} + \frac{HQ}{4q^2}\right]q^2\right\}}{1 - q^2} \\
&= \frac{2\left\{p - \frac{H}{4}\left[\frac{H}{2} + Q\right]\right\}}{1 - q^2} \\
&= \frac{2\left\{p - \frac{Hq}{4}\right\}}{1 - q^2} \\
&= \frac{2p\{1 - q^2(1 + \varepsilon)\}}{1 - q^2}
\end{aligned}$$

where  $\varepsilon = \frac{H}{2pq} - 1$ .

Thus,

$$E(\text{sib}|A) - 2p = \frac{pq^2(1-\varepsilon)}{1-q^2} = \frac{q^2(1-\varepsilon)}{1+q}$$

and

$$\frac{E(\text{sib}|A) - 2p}{E(\text{self}|A) - 2p} = \frac{1-\varepsilon}{2}$$

### A recessive

If A is recessive,

$$P(s|A) = 0 \quad \text{for } s < 2$$

$$P(2|A) = 1$$

Hence

$$E(\text{self}|A) = 2$$

$$E(\text{self}|A) - 2p = 2(1-p) = 2q$$

Also

$$\begin{aligned} E(\text{sib}|A) &= \sum_r r \sum_s P(r|s)P(s|A) \\ &= P(1|2) + 2P(2|2) \end{aligned}$$

From table A5 it follows that

$$E(\text{sib}|A) = \frac{1}{2} \left[ \frac{H}{2p} \right]^2 + \frac{Hp}{2p^2} + \frac{2p}{p} + 2 \left( \frac{H}{4p} \right)^2$$

Since  $p = P+H/2$  this can be written

$$\begin{aligned} E(\text{sib}|A) &= 2 - \frac{H}{2p} \\ &= 2 - \frac{2pq(1+\varepsilon)}{2p} \\ &= 2 - q(1+\varepsilon) \end{aligned}$$

Thus

$$\begin{aligned} E(\text{sib}|A) - 2p &= 2 - q(1+\varepsilon) - 2p \\ &= q(1-\varepsilon) \end{aligned}$$

and

$$\frac{E(sib|A) - 2p}{E(self|A) - 2p} = \frac{1 - \epsilon}{2}$$

This is the equation when A is recessive and  $a$  is dominant. The equivalent equation when A is dominant and  $a$  is recessive can be obtained by replacing A and  $p$  by  $a$  and  $q$  to yield,

$$\frac{E(sib|a) - 2q}{E(self|a) - 2q} = \frac{1 - \epsilon}{2}$$

as required.