Genetic Relatedness and the Evolution of Altruism*

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In their recent book, Elliott Sober and David Wilson (1998) argue that evolutionary biologists have wrongly regarded kinship as the exclusive means by which altruistic behavior can evolve, at the expense of other mechanisms. I argue that Sober and Wilson overlook certain genetical considerations which suggest that kinship is likely to be a more powerful means for generating complex altruistic adaptations than the alternative mechanisms they propose.

The purpose of this paper is to comment on some recent arguments of Elliott Sober and David Sloan Wilson (1998) concerning the evolution of altruistic behavior. The issue I am concerned with is the significance, or otherwise, of genetic relatedness in mediating the evolution of altruistic adaptations. I start by briefly outlining Sober and Wilson's position.

Altruism poses a familiar puzzle for the theory of natural selection, for an animal which behaves altruistically reduces its fitness relative to selfishly-inclined members of the population and should thus be *disfavored* by natural selection. So how can altruistic tendencies evolve? One traditional answer, first broached by Darwin himself, appeals to selection at the level of the *group.* Though altruistic individuals do worse than selfish ones, it is quite conceivable that *groups* of altruists will out-compete *groups* of selfish organisms. But since the 1960s group selection has been out of favor in mainstream evolutionary biology, due mainly to the powerful

*Received August 2000; revised July 2001.

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‡Thanks to the late William Hamilton, John Maynard Smith, Alex Rosenberg, Elliott Sober, and two anonymous referees for helpful comments and discussion.

Philosophy of Science, 69 (March 2002) pp. 138–149. 0031-8248/2002/6901-0007$10.00

attacks by G.C. Williams (1966) and John Maynard Smith (1964, 1976). Most contemporary biologists believe that the theory of *kin* selection, due originally to W.D. Hamilton (1964), offers a better explanation of altruism than group selection. The basic idea of kin selection is straightforward. A gene which codes for behavior that is costly to the individual who carries it, but benefits his genetic relatives, e.g. sharing food with siblings, will increase in frequency by natural selection—because the individual's relatives are likely to carry copies of the gene in question themselves. Altruism can evolve, Hamilton concluded, so long as the cost incurred by the altruist is offset by a sufficient amount of benefit to sufficiently closely related relatives. (This condition for the spread of an altruistic gene is known as *Hamilton's Rule.*) Thanks to the enormous influence of Hamilton's work, kin selection theory is widely accepted among evolutionary biologists as the most plausible way of explaining the evolution of altruism from a Darwinist perspective.

Sober and Wilson challenge this orthodoxy. They maintain that kin selection, far from being an alternative to group selection, is actually a special case of it. In support of this claim, Sober and Wilson offer a very general account of how altruism can evolve. Imagine a population containing two types of organism, altruistic and selfish. Assume for simplicity that reproduction is asexual and like always begets like—the offspring of altruists are altruistic, and similarly for selfish organisms. The population is subdivided into two groups. Group one contains mostly altruists with a few selfish; group two contains mostly selfish with a few altruists. *Within* each group, altruists are lower in fitness than their selfish counterparts (by definition). But this does *not* mean that altruism cannot evolve. For fitnesses are group-dependent: an organism's fitness depends not only on whether it is selfish or altruistic, but also on which group it is in. Fitnesses are higher in group one than group two, for the former contains a higher frequency of altruists. If the parameters are chosen appropriately, the global frequency of altruists can increase in the second generation. When this happens, the individual disadvantage of behaving altruistically is offset by the fact that altruists are grouped together, and thus tend to be the recipients of each others' help. So for an altruistic allele to increase in frequency over many generations, two conditions must be satisfied: (i) there must be a statistical tendency for altruists to find themselves grouped with other altruists; (ii) periodically, the groups must break-up and blend into the global population, then new groups form—to prevent the selfish allele spreading to fixation *within* each group. When these conditions are satisfied, altruistic adaptations can be expected to evolve. That is the central message of part I of *Unto Others.*

Sober and Wilson emphasise that the "groups" in the above model do not necessarily have to be groups in an ecological sense—they do not need

to be spatially discrete, nor reproductively isolated, nor to persist for any particular length of time. All that matters is that the organisms in question interact so as to affect one another's fitness: that is the sole criterion for being a group, for Sober and Wilson. Indeed in many of their examples, the duration of the groups is no longer than that of their constituent organisms—the groups are simply temporary collections of interacting organisms which break-up and reform every generation. Permitting groups to be transient entities allows Sober and Wilson to pre-empt one of the standard objections to group selection—that individual selection will eventually cause selfishness to spread to fixation *within* each group, undermining the prospects for altruism to evolve by between-group selection. They point out that this objection—known as "subversion from within"—rests on the assumption that groups hold together for many organismic generations, an assumption which they reject as unmotivated.

Given this conception of group selection, it is easy to see why Sober and Wilson regard kin selection as a special case of group selection. The evolution of altruism by kin selection proceeds exactly as in the basic model above: the individual disadvantage of behaving altruistically is offset by the fact that altruists are grouped together in kin groups, and thus tend to be the recipients of each other's help. The division of the population into kin groups (in Sober and Wilson's sense of "group", of course) is simply an efficient way of securing the statistical tendency of altruists to assort with each other that is needed for altruism to evolve. In other words, the basic model tells us that what is required for the evolution of altruism is that the recipients of altruistic actions tend to be altruists themselves—condition (i) above. This condition will obviously be satisfied, if altruists tend to direct their altruism at genetic relatives—as in kin selection. But Sober and Wilson insist that kin-directed altruism is only one among many possible ways of satisfying the condition. So it is a mistake, they argue, to think that kinship has an exclusive role in explaining the evolution of altruism, as most biologists do; rather kinship is simply a means to an end—a way of securing positive assortment of altruists, but not the only way. They write: "for all its insights, kin selection theory has led to the constricted view that genealogical relatedness is the one and only mechanism for the evolution of altruism" (Sober and Wilson 1998, 158).

Some critics have argued that Sober and Wilson's conception of group selection, in particular their definition of a group, is excessively liberal, and thus that they are mistaken in regarding kin selection as a type of group selection (Maynard Smith 1998). This issue is partly (though only partly) terminological; I have examined it elsewhere (Okasha 2001). My interest here is in a different question, which does not depend on whether we agree with Sober and Wilson or Maynard Smith on the definition of group selection. The question concerns Sober and Wilson's attempt to

dethrone kinship from the privileged role it is usually accorded in the explanation of altruistic adaptations. Are Sober and Wilson right to regard kinship as only one among many different ways in which altruism can evolve, or is the orthodox view—that kinship is of pre-eminent importance—correct? Detailed empirical research would be needed to answer this question conclusively, but I believe there are some important genetical considerations, of a relatively a priori nature, which favor the orthodox view, but which Sober and Wilson overlook.

Interestingly, Sober and Wilson cite W.D. Hamilton himself as an ally in support of their claim that genetic relatedness is only one among many factors that can promote the evolution of altruism. In his famous 1964 papers, Hamilton appeared to endorse the orthodox view: he argued that the spread of an altruistic allele depends on the "coefficient of relationship" between donor and recipient, a coefficient which measures their genealogical relatedness. However, in a lesser-known 1975 paper, Hamilton made the very point that Sober and Wilson stress: what matters is that the recipients of altruism should themselves be altruists, not that they be related to the donor. In his calculations, Hamilton (1975) replaced the coefficient of relationship of his 1964 papers with a *correlation* coefficient, which reflects the probability that donor and recipient both carry the altruistic allele, whether due to relatedness or not. He wrote: "it obviously makes no difference if altruists settle with altruists because they are related . . . or because they recognise fellow altruists as such, or because of some pleiotropic effect of the gene on habitat preference" (Hamilton 1975, 337). With this remark Hamilton appears to endorse the Sober and Wilson position—genetic relatedness is not the exclusive means by which unselfish behavior can evolve, but one among many possible causes. (Sober and Wilson set great store by this passage of Hamilton's, which they quote twice (1998, 77, 134)).

Hamilton cites *recognition of fellow altruists* and *a pleiotropic effect of an altruistic gene on habitat preference* as possible alternatives to kinship, for securing positive assortment among altruists. However, there is a simple genetical reason for thinking that both of these mechanisms, and indeed any other non-kin mechanisms, will be less effective than kinship in producing altruistic adaptations. The point is most easily seen in the case of pleiotropic effects. Imagine a gene in a baboon population which causes its bearers to behave altruistically towards neighbors, and also to favor a particular region of the forest to live in until adulthood. On reaching adulthood, the baboons blend into the global population and engage in population-wide competition for mates. In principle this gene can spread, for altruistic adolescent baboons will become grouped together in the given region of the forest, and so will tend to be the beneficiaries of each others' help; and the periodic break-up of the groups caused by the baboons' dispersal at adult-

hood prevents selfishness from spreading to fixation by within-group selection. So Sober and Wilson's two conditions for the spread of altruism are satisfied—over time, we should expect to see the altruistic gene gradually replace the selfish gene in the baboon population.

However, there is a complication. For there will be immediate selection pressure at virtually all[1] other loci in the baboon genome for suppression of the altruistic behavior. The reason for this is obvious: from the point of view of all the other genes in an altruistic individual, the altruistic behavior is a waste. For the altruism is directed at unrelated baboons. While the recipients of altruism are also likely to carry copies of the altruistic gene (thanks to the pleiotropic effect), *with respect to all other loci* donor and recipient are no more likely than average to share genes. So imagine a modifier gene at a different locus, which has the effect of suppressing the altruistic behavior, but leaving the habitat preference intact. Such a gene, if it arose, would clearly benefit any baboon possessing it. For possessors of the gene would spend their adolescence in the region of the forest with a high proportion of altruists, and thus still be beneficiaries of others' altruism, but without incurring any of the costs. The modifier gene would quickly spread within the population, undermining the evolution of the altruistic adaptation by frustrating the positive assortment on which it depends. So although a pleiotropic effect on habitat preference is a possible mechanism for generating the positive assortment among altruists necessary for altruism to spread, there are reasons for thinking that the mechanism is susceptible to breakdown via modifier genes at other loci.[2]

Note that the breakdown of altruism via modifier genes described above is not simply a case of "subversion from within," the textbook problem for group selection. In the standard case of subversion from within, a selfish allele gradually replaces an altruistic allele at a given locus within a group, thanks to the greater relative fitness of the selfish allele, and the assumption that the group lasts for many organismic generations. As noted above, Sober and Wilson's conception of group selection avoids the subversion from within problem by dropping the assumption that groups must last for many generations. But the problem of modifier genes does not depend on assumptions about group persistence—in the example above the baboon groups break up and disperse into the global population

1. "Virtually all" rather than "all" because at loci that are strongly linked to the altruistic gene's locus, there will be no selection pressure for suppression of the altruistic behavior. Genes at these linked loci benefit from the altruistic behavior, as there is a higher than average chance that the recipients of the altruism will also carry copies of these genes. (How much higher than average depends on how close the linkage is). This minor complication does not affect my basic argument however.

2. See Dawkins 1982, Ch. 8 for a good discussion of modifier genes in the context of the evolution of altruism.

each generation. The selection pressure for modifiers that suppress the altruism is simply a consequence of the fact that, from the point of view of all the genes in an altruistic organism except the altruistic gene itself, helping unrelated altruists is wasteful, so any way of preventing the waste without foregoing the associated benefit will be favored by natural selection. Intra-genomic conflict via the influence of genetic modifiers is the likely consequence of dispensing aid to non-kin.[3]

Matters are very different if the recipients of altruism are genetic relatives of the donor—as in standard kin selection. For then, there is no selection pressure at other loci for suppression of the altruistic behavior. The reason is straightforward. Donor and recipient are relatives, so have the same degree of relatedness at *every locus in the genome.*[4] This means that the altruistic behavior benefits all genes in the genome equally, not just the gene that codes for the altruism. Therefore, a modifier gene which suppresses the altruistic behavior will undermine its own replication prospects. This is because an individual possessing this modifier ceases to behave altruistically towards kin—thereby foregoing the opportunity of assisting other individuals *who have a greater than average chance of carrying a copy of the modifier gene themselves.* Since the altruistic behavior must initially have been obeying Hamilton's rule—or it would never have evolved in the first place—it follows that the modifier gene which causes suppression of the altruistic behavior cannot spread by natural selection.

This suggests that Hamilton's 1975 remark that "it makes no difference if altruists settle with altruists because they are related . . . or because they recognise fellow altruists as such, or because of some pleiotropic effect of the gene on habitat preference," needs qualification. Where altruists are brought together by pleiotropic effects, the increase in frequency of altruism is likely to be transitory—it will only occur until modifier genes have arisen at other loci that suppress the altruism. By contrast, in cases of orthodox kin selection, altruism can continue to increase in frequency generation after generation, unhampered by selective pressure at other loci. This difference is potentially vital. For the evolution of complex adaptations requires many rounds of cumulative selection—this is true for behavioral adaptations no less than for morphological and physiological ones. Hamilton's assertion that "it makes no difference" what causes the positive assortment of altruists is certainly true if we consider one or two generations; but if we consider a longer timespan—as we must do if we

3. See Hurst, Atlan, and Bengtsson 1996 for a good introduction to the subject of intragenomic conflict and the selection pressure for modifiers to which it gives rise.

4. See Uyenoyama and Feldman 1980 for a good discussion of the (tricky) degree of relatedness concept in population genetics, and its significance for the evolution of altruism.

are interested in complex adaptations—there are good reasons for thinking that kinship provides more robust a mechanism for the evolution of altruism than non-kin mechanisms for getting positive assortment. Hamilton (personal communication) has informed me that he regrets not substantially qualifying his 1975 remark, and that he regards genetical relatedness as by far the most effective means of producing complex altruistic adaptations.

What about the other way of getting positive assortment among unrelated altruists that Hamilton 1975 alluded to—recognition of altruists by each other? This, rather than pleiotropic effects on habitat preference, is actually the main focus of Sober and Wilson's attention, for they are especially interested in human evolution, and the possibilities for choosing whom to associate with are probably greater for humans than for other species. Of course, the ability to recognise other altruists and choose them as associates will not *necessarily* result in the positive assortment needed for altruism to evolve—for selfish organisms will no doubt prefer altruists as companions too, as Wilson and Dugatkin point out (1997, 347). But Sober and Wilson suggest that if joining a group requires the consent of other members of the group, then choice of associates can lead to altruists being grouped together (1998, 135). More generally, some mechanism for excluding the selfish organisms from joining an altruistic group is required, if the population structure necessary for altruism to evolve is to result from altruists' recognising each other and choosing each other's company. There is clearly an important issue about how any such mechanism could arise initially—which Sober and Wilson address (1998, 135–154). My concern here is not with this issue, so I propose to *concede* to Sober and Wilson that positive assortment among non-related altruists can be generated by altruists' recognising each other and choosing to assort. The question is: is positive assortment generated in this way susceptible to breakdown via modifier genes, or not? Prima facie the answer seems to be "yes." For in such a situation, the beneficiaries of altruism will be unrelated to the donors, so surely the above considerations about selection for modifier genes apply? Surely positive assortment via choice of associates is in exactly the same boat as positive assortment via pleiotropy? This *sounds* right, but in fact there is a complication.

The complication is this. In the pleiotropy example above, I imagined a modifier gene which switched off the altruistic behavior *but left the habitat preference intact.* That was essential to my argument, for if a modifier arose which cancelled both altruistic behavior *and* habitat preference, it would be at a selective disadvantage. The bearer of such a gene would not incur the cost of behaving altruistically, but neither would it receive benefit from other altruists—for it would spend its adolescence in the area of the forest where few altruists reside. And one cannot argue that the gain from

ceasing to be altruistic might outweigh the loss of benefit from other altruists. If that were so the altruistic gene would never have spread initially—for it would not have satisfied Hamilton's rule. So a modifier which suppresses both altruism and habitat preference would be counter-selected. Now consider a case where altruists are brought together not by pleiotropy but by recognising each other and choosing to co-associate. What would happen if a modifier gene arose at a different locus, that suppressed the altruistic behavior? Clearly, this would be analogous to the modifier which suppressed both altruism and habitat preference. Carriers of such a gene would no longer incur the cost of being altruistic, but neither would they receive benefits from other altruists—for their altruistic colleagues would no longer choose them as associates. Such a modifier would not be selected, and so would not cause the evolution of the altruistic adaptation to break down.

But this does *not* show that altruists choosing each other's company are immune from the influence of modifiers, and thus provide as robust a mechanism for the evolution of altruism as kin selection. For although modifiers which simply switch off the altruism will not spread, a situation where unrelated altruists choose each other as associates *is* susceptible to breakdown by modifiers which act more subtly. Imagine a modifier which causes its bearer to stop being altruistic but to continue behaving in a way that *appears* altruistic to others, i.e., to feign altruism. If the deception is successful, other altruists will continue to associate with the carrier of the modifier gene. So the latter is at an advantage: he receives a benefit from other altruists but does not incur the cost of dispensing altruism—for he only pretends to be altruistic. (Such a modifier is precisely analogous to the modifier which switched off the altruism but left the habitat preference intact.) Should such a modifier arise, it would quickly spread in the population, inhibiting the evolution of altruism by frustrating the positive assortment on which it depends. Since the altruists are by hypothesis unrelated, we should expect intense selection pressure at every other locus in the genome for modifiers which act in the manner described, i.e., which cause their carriers to pretend to be altruistic without really being so.

Sober and Wilson *are* aware that individuals who pretend to be altruistic while really being selfish can frustrate the evolution of altruism. Discussing a model where altruism evolves via assortative interactions among non-relatives, they admit that a problem for the model is that "it may be difficult to discover the altruistic tendencies of others, especially if individuals have evolved to conceal their selfish tendencies" (1998, 140). Their response to this problem is twofold. Firstly, they point out that in some cases it may be quite difficult to fake altruistic behavior. They cite predator inspection in guppies as an example of an altruistic action which can't easily be faked. It is hard to see how a guppy could fake the inspection of

a predator—either it swims up to the predator and observes it, or it does not. Secondly, they point out that in the case of humans, our cognitive sophistication may make cheating much harder. Humans can use their cognitive powers to "seek out trustworthy individuals and avoid cheaters in social interactions" (1998, 141). Both of these points are fair; they suggest that it is not *inevitable* that the evolution of altruism among non-relatives will be frustrated by cheaters. But what Sober and Wilson fail to point out is that where altruists are brought together by genetic relatedness, no sophisticated mechanisms for cheater-detection are necessary for altruism to evolve. This is because no selection pressure for cheating exists in the first place—since donor and recipient are relatives, their degree of relatedness[4] is the same at every locus in the genome. So a gene for cheating will undermine its own replication prospects. In kin-directed altruism the co-operation of all genes in the genome is automatic; in altruism directed at unrelated fellow altruists, co-operation needs to be enforced by special mechanisms and is in constant danger of breaking down. So to refer to kinship as "just one of several sorting processes" which can cause altruism to evolve, as Sober and Wilson (1998, 139–140) do, is misleading—it ignores the very factor which makes kin selection so robust a mechanism for evolving altruistic adaptations.

It is clear that the foregoing considerations, if correct, will apply to *any* mechanism for generating positive assortment among altruists apart from kinship, not just pleiotropic effects on habitat preference and the recognition of altruists by one another. All such mechanisms are potentially subject to undermining via modifier genes at other loci which act in an appropriate way. But is it likely that such modifiers will in fact arise? And if they do, is it true that they will simply sweep to fixation in the population and thus eliminate the altruistic behavior entirely? My argument above presupposes that the answer to both of these questions is "yes." Of course, it is impossible to *prove* that this presupposition is correct, but it is worth explaining why it is justified.[5]

The first point to note is that genes which modify or suppress the phenotypic effects of other genes are very common. Indeed, the modern 'interactive' view of gene action is that virtually all phenotypic traits, especially complex behavioral traits, are affected by genes at more than one locus, indeed often a very large number of loci.[6] (The various molecular mech-

5. Thanks to an anonymous referee for impressing on me the need to discuss this question.

6. Indeed, Dawkins (1982, 137) argues that the thoroughly interactive view of gene action accepted by modern geneticists means that the distinction between "major" genes which have "their own" phenotypic effects, and "modifier" genes which modify the phenotypic effects of major genes, becomes very blurred. So in a sense all genes are modifier genes, he argues.

anisms on which the action of modifier and suppressor genes is based are many; see Griffiths et al. 1999, 163–196 for discussion.) So my assumption above that if a gene for altruistic behavior arises, mutations at other loci will be capable of interfering with the effect of that gene is not an unreasonable one. But how likely is it that a mutation will arise *which acts in the required way,* i.e., which suppresses the altruistic behavior without altering the organism's chance of finding itself grouped with other altruists? It is difficult to say anything very specific about this, given how little is currently known about the causal pathways by which genes influence behavior. But a few general points are in order: (i) a priori, it seems no less likely that an appropriate modifier gene will arise than that the gene which codes for the altruistic tendency itself arose; (ii) empirically, there are many known cases where a gene which acts in a way deleterious for the other genes in the genome *has* led to the evolution of modifiers/suppressors at other loci (see Hurst, Atlan, and Bengtsson 1996); (iii) there is plenty of time for a modifier to arise, as many rounds of cumulative selection are required for the altruistic adaptation to evolve in the first place; (iv) there are plenty of suitable loci for a mutation to occur at; (v) even a mutation which has a very slight effect in the required direction will be favored by selection—as Darwin taught, evolution proceeds by the gradual accumulation of slight advantages.

Supposing that an appropriate modifier does arise, is it obvious that it will simply spread to fixation and thus eventually eliminate the altruism altogether, as I assumed above? An anonymous referee points out that there is another possibility: an *arms race.*[7] To see this point, suppose again that altruism has evolved by preferential assortment among unrelated altruists, and a modifier gene that codes for cheating (i.e. faking altruistic behavior) has arisen. One possibility is that the cheating gene sweeps to fixation. But suppose that the altruists have the ability to detect cheats, and they refrain from behaving altruistically towards anyone they suspect of cheating. If the altruists vary in their ability to detect cheats, and the variation is heritable, this could lead to selection for better cheater-detection. This in turn could induce selection for better faking among the fakers, and so on (see Trivers 1971). In theory such an arms race could have many different outcomes. However, there is actually an empirical reason for thinking that the eventual suppression of the altruism is the most likely outcome in a scenario of this type. For the altruistic gene will be 'outnumbered' by the other genes in the genome, as Dawkins (1982, 138) observes. To see this point, recall that altruist and donor are by hypothesis unrelated, so there will be selection pressure for cheating at loci

7. See Hurst, Atlan, and Bengtsson 1996 for a discussion of arms races in the context of intra-genomic conflict, and the various possible outcomes.

throughout the genome, whereas the selection pressure for cheater-detection will occur *only* at the locus of the altruistic gene (or at closely linked loci). If you are a randomly picked gene in an altruistic organism, it makes no odds to you whether your host organism is good at discriminating cheaters from true altruists or not—a copy of you is no more likely to be found in a true altruist than in a cheater. So you will channel your energy into trying to suppress the altruistic behavior altogether, not in trying to ensure that it is selectively directed toward true altruists. The loci at which there is selection pressure for cheating will thus far outnumber the loci at which there is selection pressure for cheater-detection. So even if an arms race does break out, the elimination of the altruistic behavior is the most probable final equilibrium.[8]

I believe, therefore, that Sober and Wilson's attempt to dethrone kinship from the privileged role it is usually accorded in the explanation of altruistic behavior is unlikely to succeed. Of course, the issue here is ultimately empirical. To determine whether kinship deserves its privileged role, one would have to find out whether positive assortment of unrelated altruists is in fact common in nature, and whether it has in fact led to the evolution of altruistic adaptations. Very little is known about these matters—as Wilson himself has admitted elsewhere (see Wilson and Dugatkin 1997, 342). Trying to pre-judge empirical issues is always risky, and future research may vindicate Sober and Wilson at the expense of the orthodox view. My aim has only been to show that the orthodox emphasis on kinship is not simply the result of a constricted outlook, as Sober and Wilson claim, but is underpinned by certain genetical considerations.

I'll end by noting an irony. An important theme of Sober and Wilson's book is the heuristic advantage of what they call "multi-level selection theory" over "selfish gene theory" for studying the evolution of behavior. Multi-level selection theory sees selection as potentially operative at all levels of the biological hierarchy, while selfish gene theory represents all selection processes as ultimately for the benefit of the genes. Although the two theories are not actually in conflict—they "offer different perspectives on the same set of processes"—Sober and Wilson (1998, 88) argue that multi-level selection theory has important heuristic advantages. Biologists overly impressed with the selfish gene picture of the world have lost the ability to see group-level adaptations in nature, they claim. Their arguments for this claim are compelling and should give pause to many a selfish

8. This argument assumes that the ability to detect cheats is heritable because it is genetically hard-wired. But if the ability is heritable for other reasons, e.g., cultural transmission, then the argument of this paragraph does not apply, and the likelihood that an arms-race will lead to an equilibrium outcome other than the suppression of altruism is correspondingly higher.

gene theorist. But the foregoing considerations illustrate the heuristic benefits of the selfish gene perspective—and the potential danger of not adopting it. It is because they do not consider the replication interests of each gene individually that Sober and Wilson fail to appreciate the intragenomic conflict, and the selection pressure for modifiers, that the dispensing of altruism to unrelated organisms inevitably entails. If the multilevel perspective has its heuristic advantages, so does the selfish gene perspective. The moral, surely, is that we should be pluralistic about the two perspectives and attempt to reap the heuristic benefits of both.

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