6

Comments on Maynard Smith's "How to Model Evolution"

Elliott Sober

1 *Units of Evolution*

Maynard Smith (1987) distinguishes *units of evolution* from *units of selection.* He claims that it is the first of these, not the second, that forms the conceptual center of the controversy that Wynne-Edwards (1962), Williams (1966), Hamilton (1964, 1967), Lewontin (1970, 1974), and Maynard Smith (1964, 1976) himself helped shape.

I agree that the process Wynne-Edwards postulated involves groups that exhibit heredity. David Wilson's (1975, 1980) trait groups require no such thing. Trait groups, according to Maynard Smith's proposed usage, are not units of evolution. Maynard Smith concludes that the trait group idea does not address the problem that stems from Wynne-Edwards's invocation of group selection.

My main disagreement with Maynard Smith arises here. Although Wilson's proposal is a handy example with which to distinguish Maynard Smith's way of carving up the problem from mine, our differences extend beyond the kind of process Wilson investigated. Our disagreement concerns what the biological debate of the last twenty some years has been about. It may seem more than a little odd that a philosopher should tell a biologist about the nature of a biological problem that that very biologist has done so much to illuminate. In self-defense, I can only say that it is the logic of the reasoning biologists pursue that leads me to these conclusions. Surely it is the merits of such arguments, not the disciplinary credentials of the people who put them forward, that really matter.

I take it that Maynard Smith uses the term "unit of evolution" to apply to anything that exhibits heritable variation in fitness. He also believes that an X-level adaptation requires that X's be units of evolution. This is why he says that group adaptations will be impossible if groups lack heredity. In this discussion, I shall adopt Maynard Smith's stipulation about what a unit of evolution is, but I shall question the connection he draws between this idea and the concept of adaptation. In particular, I shall claim that it is neither necessary nor sufficient for group adaptation that groups be units of evolution. Since I see group adaptation as the conceptual center of the

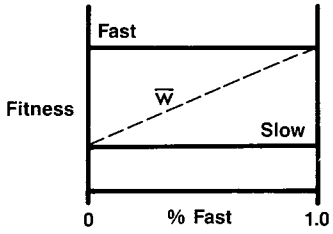

Figure 6.1
Fast deer are fitter than slow ones; the fitnesses are frequency independent. As *Fast* sweeps to fixation, the average fitness of individuals in the population, $\bar{w}$, increases.

biological controversy, I conclude that Maynard Smith's notion of a unit of evolution does not isolate the fundamental problem. This does not mean that the idea of group heredity is irrelevant, but that I assign to it a different importance.

The fundamental conceptual insight of Williams's (1966) *Adaptation and Natural Selection* was to see the difference between adaptation and fortuitous benefit. A trait of a group may be good for it, without the cause of its presence being that it is group beneficial. A group adaptation must have arisen by a process of group selection. This distinguishes group adaptations from group benefits that are artifacts of individual selection processes.

Williams (1966, p. 16) invented a simple example to illustrate this point. Suppose that speed helps deer escape from predators. Deer will therefore experience individual selection for being fast. As a result, slow deer are eliminated and the fleet survive. In this process of individual selection, the average level of speed found in the herd may increase. The process is illustrated by the frequency independent fitness function shown in figure 6.1.

Suppose for a group of deer that the slower it is, the more prone it is to extinction. If predators eat all the deer in a slow herd, the herd becomes extinct. It is therefore advantageous for a herd to be fast. Williams's point is that the survival of a fast herd is just a "statistical summation" of the facts of individual selection. Groups are benefited by containing fast deer; but it is false that groups are fast because they are so benefited.

Let us add to this picture the idea of group heredity. Imagine that groups not only go extinct but send out migrants to found new groups when the group reaches a threshold census size. Let the founders of a new group all come from the same parental population. Fast herds found more colonies and become extinct less often than slow herds. Groups thereby exhibit heritable variation in fitness. They are units of evolution, in Maynard Smith's sense. But the trait of fleetness is not a group adaptation, because it did not evolve by group selection.

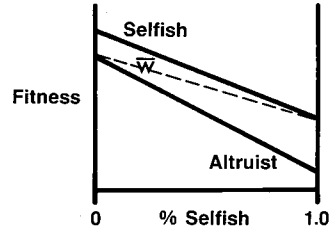

Figure 6.2
Within any group, selfish individuals are fitter than altruists, but groups of altruists will have a higher average fitness, $\bar{w}$, than groups of selfish individuals.

Slow deer are eaten while fast deer escape. As a result, slow herds survive and colonize less successfully than fast herds. But this difference between groups is just a "statistical summation" in Williams's sense. I conclude that groups can be units of evolution without there being group adaptations.

To establish the converse—that group adaptation does not require that groups be units of evolution—it is useful to think about the evolution of altruism. The relevant properties of an altruistic trait are exhibited in figure 6.2. Within any group, an altruist is less fit than a selfish individual. But groups in which altruism is common have a higher average fitness than groups in which altruism is rare.

In the kind of case I want to consider, individual and group selection will oppose each other. There will be individual selection for being selfish, but there also will be group selection for groups in which altruism is common. What will happen in this process depends on contingent properties of population structure. It cannot be determined a priori whether one trait will sweep to fixation in the ensemble of groups or selfishness and altruism will be maintained in a polymorphic equilibrium.

We can use Maynard Smith's idea of a unit of evolution to describe one way that altruism might evolve. Suppose that groups found colonies, where the founders of a daughter colony all come from the same parent. This means that we can identify for a colony in one generation its parent in the previous generation.

Let us suppose further that the organisms reproduce uniparentally, and that like always produces like. This does not imply that a daughter colony will always have exactly the same frequency of altruists as its parent. After all, the migrants from the parental colony are drawn by a sampling process; a daughter frequency may thereby differ from the parental frequency by sampling error. Nevertheless, I assume that groups have heredity in the requisite sense; although like does not always produce like, the *expected* frequency of altruists in a daughter colony (when it is founded) is just the *actual* frequency in the parental population (when sampling takes place).

Groups have heredity here just as organisms would, if the expected height of an offspring is the parent's height. So in the case before us, groups are units of evolution.[1]

The process begins with a set of populations exhibiting different local frequencies of altruism. Suppose one of these starting populations has 100% altruists. This population then founds offspring colonies, also with altruism at 100% (sampling cannot change *this* frequency). In each generation, a colony of pure altruists has a higher productivity than any other sort of colony. In the limit, we expect altruism to go to fixation in the ensemble of populations. In this group selection process, groups have heredity and a group adaptation—a trait that benefits the group at the individual's expense—thereby becomes universal. Here we have groups as units of evolution, in Maynard Smith's sense, and group adaptations as well.

For natural selection to produce evolution, heritability of some sort is essential. But for group selection to cause evolution, it is not essential that the heritability be *group* heritability.[2] Suppose that migrants are drawn by sampling from each population and then mixed in a global "migrant pool." After that, samples of ten organisms, say, are drawn from this pool, each such sample then founding a new colony. Suppose that the samples are drawn from the migrant pool, not at random, but on a principle of like associating with like. An extreme version of this procedure would have new colonies founded by either all altruists or all selfish individuals. A less extreme version might create colonies in which the ratio of altruists to selfish individuals is either 9:1 or 1:9. The point is that the distribution of local frequencies is more skewed to extreme ratios than would be expected if the sampling proceeded at random.

Altruism can increase in frequency under this regimen, even though the idea of group heritability has been destroyed. A given daughter colony may have founders drawn from many different parent colonies. Indeed, colonies do not form chains of descent (lineages) so much as densely connected webs. My point is that this does not mean that altruism—a group adaptation—cannot evolve.

A variant on this idea is Wilson's concept of trait groups. Suppose juveniles disperse into local groups, interact with each other during development from juvenile to adult, and then reassemble in a single global population to mate. If like associates with like in these local trait groups, altruism may increase in frequency. This may happen even though trait groups are entirely evanescent. A trait group in one generation cannot be said to be a parent of a trait group in the next.

In the first setup, groups have heredity—it is possible to say of a given colony, which colony in the previous generation was its parent. In the second setup, groups have many parents—conceivably the individuals in a colony may come from many (even all) of the colonies in the previous

generation. In the third, the idea of group heredity has all but disappeared. Yet in all these arrangements, altruism can evolve, if the parameters are right. Altruism, I take it, is a paradigmatic group adaptation. It cannot evolve by individual selection alone, since an altruistic characteristic helps the group at the expense of the individual who possesses it. The reason that group selection can cause altruism to evolve in these systems is that altruists associate mainly with altruists and selfish individuals mainly with selfish individuals. Group heritability is one device for making sure this is true, but it is only one.

I have argued that the fundamental biological issue has been whether group adaptation is common, rare, or nonexistent. Group adaptation requires group selection. Evolution by group selection requires some mechanism of heritability. Maynard Smith has emphasized the idea of group heritability. I do not contest its importance. However, group heritability is just one way to secure the heritability needed for selection to lead to evolution. In addition, the concept of a unit of evolution glosses over the distinction between group adaptation and fortuitous group benefit. The examples discussed so far and Maynard Smith's two concepts are represented in figure 6.3. I conclude that Maynard Smith's concept separates processes that belong together and unites processes that belong apart.

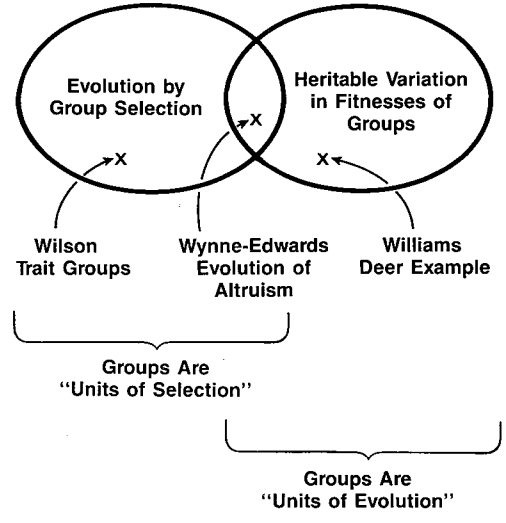

Figure 6.3
Maynard Smith's (1987) concepts of "units of evolution" and "units of selection." The controversy over group adaptation concerns whether groups are units of selection; although Wynne-Edwards's treatment required that they be units of evolution as well, this is neither necessary nor sufficient for the existence of group adaptations.

2 *Causality and Context Dependence*

If the difference between group adaptation and fortuitous group benefit is fundamental, then the central concept in the biological debate is the idea of a unit of selection. Maynard Smith does not characterize this concept. However, I take it that we can agree that X is a unit of selection in a given evolving system precisely when there is X selection in that system. This leaves it open whether there may be more than one unit of selection in a given biological system, whether some of those units may be more important than others, and whether different systems may have different units, depending on the details of their biology. This way of defining the idea does not mean that the kind of pluralism contemplated here is correct; it is just that the concepts should be understood in such a way that this possibility is not ruled out a priori. So the question of what a unit of selection is thereby boils down to the problem of understanding what distinguishes different sorts of selection processes.

For me, group selection is a distinctive sort of causal process. It is not definable by the existence of heritable variation in group fitness (as Williams's deer example shows); the question is not whether groups vary in fitness, but why they do so. I cannot do justice to this subject in the space of this reply, so I must refer the reader to the treatment I give of this issue in part II of *The Nature of Selection* (Sober, 1984).

Maynard Smith is quite right that Lewontin and I accorded no importance to the computational simplicity of the allelic versus the genotypic models of heterozyogote superiority (in Sober and Lewontin, 1982). This is because we wanted to say what the causal processes are that govern the evolution of this system. That question is properly answered by using some plausible account of what causality means and then consulting the biological facts. Since both the allelic and the genotypic models are correct as algebra, there is no notion of choosing between them, much less choosing between them on grounds of simplicity.[3]

Lewontin and I did not supply an explicit characterization of causality, but merely handled that sticky concept on an intuitive level. This is why I now regard some of the arguments we gave as less than conclusive. The account of causality suggested in my book underwrites the conclusions that Lewontin and I reached, but in what I think is a theoretically more satisfying way. In particular, we used the idea that context dependence can undermine a causal claim; this idea survives in the treatment of causality I develop. Maynard Smith objects to this, observing that "if I put a flame under a beaker of water, the water will expand if it is above 4°C, and contract if it is between 0°C and 4°C, yet in both cases I would wish to say that the flame caused the changes in density. Similarly, melanic moths are fitter in industrial areas, and less fit in rural areas, yet I want to say that the color difference causes the fitness differences."

To understand when context dependence is and is not relevant to the truth of a causal claim, we must distinguish two concepts of cause. There is *token causality* on the one hand and *property causality* on the other. The former describes what happens in a single unrepeated event (e.g., "Harry's smoking caused him to have a heart attack"), whereas the latter describes the causal role that properties play in some population (e.g., "Smoking is a positive causal factor in producing heart attacks among U.S. adults").

I agree that one moth may perish because it is melanic, whereas another moth, in a different area, may perish because it is nonmelanic. The fact that a trait has a given effect in one context is perfectly consistent with its having the opposite effect in another. So context dependence does not defeat claims of token causality. However, matters change when it comes to assessing the causal role that a property plays in a population. If, in a single population, melanism increases some individuals' chances of death while it decreases those of others, I do not think that the trait plays a univocal causal role in the population as a whole. Melanism is not a positive causal factor for mortality in that heterogeneous population. This does not mean that melanism is not a positive factor in one subpopulation and a negative factor in the other. Nor does it mean that melanism cannot be *correlated* with reduced mortality. This will be true if melanic individuals die less often than nonmelanic individuals.

So it is with respect to claims about the causal roles that properties play in populations that the context dependence of fitness becomes important. In the sickle cell case, having a copy of the S allele is not a positive causal factor for improved survivorship in the population as a whole. Having a copy of S on one chromosome improves fitness if there is a copy of A on the other, but it diminishes fitness if the other chromosome also has a copy of S. The real causal factors in this process are diploid genotypes; it is the pairwise gene combinations SS, SA, and AA that have determinate causal roles in the population as a whole.

Although there is no such thing as *the* causal role that melanism plays in the heterogeneous population imagined above, it is easy to divide that population in two—so that melanism is positive in one (industrial) subpopulation and negative in the other (rural) one. Why not do the same thing for heterozygote superiority, thereby showing how it can be described with single genes as causal factors?

There are different ways of trying to do this, each with its special failing. For example, we could say that each gene is such that having a second copy of it counts as a negative causal factor: if you have one, you are better off without a second. But this formulation does not show that single genes are causal factors, Having a second copy of a gene, I take it, is equivalent with having two copies of the gene, so we still are talking about diploid genotypes, not single genes, as causal factors.

Another proposal might begin by labeling the two chromosomes One and Two, and then listing the causal facts for each of four "subpopulations" —(1) the individuals who have *S* on chromosome One; (2) the individuals who have *A* on chromosome One; (3) the individuals who have *S* on chromosome Two; (4) the individuals who have *A* on chromosome Two.

Here are the causal facts concerning these four groupings: (1) For individuals who have *S* on chromosome One, having *A* on chromosome Two is a positive causal factor and having *S* on chromosome Two is a negative causal factor. (2) For individuals with *A* on chromosome One, having *S* on chromosome Two is positive while having *A* on Two is negative. (3) For individuals with *S* on chromosome Two, having *A* on chromosome One is positive and having *S* on chromosome One is negative. (4) For individuals with *A* on chromosome Two, having *S* on chromosome One is positive while having *A* on One is negative. I grant that within each of these four "subpopulations," having a single gene on the relevant chromosome counts as a positive or negative causal factor. The four "subpopulations" are shown in figure 6.4.

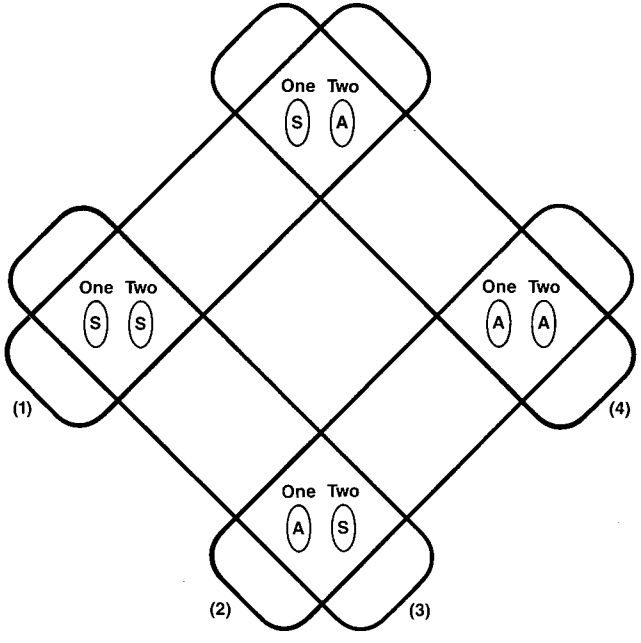

Figure 6.4
Heterozygote superiority can be analyzed so that single genes are causal factors for survival. To do so, however, requires that the population be segmented into four "subpopulations" in an entirely ad hoc way. See text for details.

Here we have heterozygote superiority from the point of view of the single gene as the unit of selection. Single genes are causal factors within "subpopulations," which in fact are four partially overlapping subsets of the single biological population under study. Notice that heterozygotes are split into two groups, depending on which allele occurs on which chromosome.

This procedure is entirely ad hoc; the "subpopulations" discerned are biologically meaningless. This creative storytelling obscures the fact that in this biological system it is entirely irrelevant whether a heterozygote has *S* on One and *A* on Two, or vice versa. Unless we deprive the word "population" of all content (so that it comes to mean any subset, no matter how arbitrary), we must grant that there is a single panmictic Mendelian population in this model, in which it is the three genotypes that are causal factors.

I conclude that genic selectionism can be formulated as a causal thesis, but at the price of inventing preposterous "subpopulations."[4] I should add that this strategy of splitting the population so that genic selectionism comes out true within artificially defined "subpopulations" will have the undesirable effect of making all selection processes cases of genic selection. Dawkins (1982) has embraced this result; for him even group selection is a kind of genic selection.[5] But this point of view deprives the group selection controversy of its empirical character. There was no need to build models or undertake detailed natural observations if the question could be dispatched so easily.

3 *Cause and Correlation*

In disposing of the "red herring" of genetic determinism, Maynard Smith notes that "all that is needed in a gene-centered view of evolution is the assumption that an animal with allele *A*, rather than *a*, is more likely to do *X* in environment *E*." This is entirely unobjectionable, provided that we are clear on what a gene-centered view can and cannot deliver. Maynard Smith's requirement is merely that the gene be correlated with the behavior, not that it cause it.

Whenever gene frequencies change via natural selection, there must be a correlation between genes and fitness. This is true whether the selection process occurs at the level of junk DNA, at the organismic level, or in Wynne-Edwards style group selection. In the model of heterozgyote superiority discussed before, a given allele will increase in frequency precisely when it is fitter than the alternative. Gene frequency equilibrium is reached when the allelic fitnesses are equal.

A gene-centered view of evolution, taken in this sense, is not a competitor with group selection, species selection, or with any other causal thesis

about evolutionary processes. This is because the view is an algebraic truism, consistent with all possible causal stories.

I very much doubt that the idea of the selfish gene would have been so influential if it had been clearly portrayed in this uncontroversial way. The reason biologists rightly took an interest in it is that they saw it as a correction to group selectionist thinking. Here I have in mind the reception of Williams (1966) and of Dawkins (1976). But to think of the problem in this way is to conflate cause and correlation.

Once the causal and the correlational theses are separated, it is possible to see that the former is vastly overstated, while the latter is, as I have said, a truism. The idea that genes are units of selection in the causal sense has limited validity. Junk DNA and meiotic drive are plausible examples. But heterozygote superiority and inferiority, epistasis, group selection, and species selection are all counterexamples to genic selectionism. A great deal of selection proceeds without its being *single genes* that are selected for and against.

Space does not permit a full documentation of my claim that gene-centered views of evolution conflate cause and correlation. I shall, however, mention a single piece of evidence. When Dawkins (1982, p. 12) takes up the question of what it means to say that a gene "causes" a trait, he produces this analysis:

> If, then, it were true that the possession of a Y chromosome had a causal influence on, say, musical ability or fondness for knitting, what would this mean? It would mean that, in some specified population and in some specified environment, an observer in possession of information about an individual's sex would be able to make a statistically more accurate prediction as to the person's musical ability than an observer ignorant of the person's sex. The emphasis is on the word 'statistically', and let us throw in an 'other things being equal' for good measure. The observer might be provided with some additional information, say on the person's education or upbringing, which would lead him to revise, or even reverse, his prediction based on sex. If females are statistically more likely than males to enjoy knitting, this does not mean that all females enjoy knitting, nor even that a majority do.

Having said all this, I should add that I do not think the genic point of view, taken as a truism about correlation, is useless. There can be a point to seeing evolution from the gene's point of view, even when one knows that genic selectionism is false as a causal claim. As noted above in the model of heterozygote superiority, the relationship of allelic fitnesses allows one to determine how gene frequencies will change. To see whether a model process will produce evolution (here identified with change in gene frequencies) in a certain direction, it can be useful to describe it in terms of allelic fitnesses. Although these models will often be misleading as to causal processes, they may have much the same utility as the idea of a net force in Newtonian physics. Describing just the net force acting on a billiard ball will fail to capture causal information about component forces; but the net force is a simple and relevant quantity for predicting the ball's trajectory.

4 *Group Selection and Female-Biased Sex Ratios*

In discussing altruism in section 2, I mentioned that within each group, an altruist will be less fit than a selfish individual. If this is true within each group, how can altruism increase in frequency under selection of any sort? The answer is that altruists can have *greater* average fitness in the ensemble of groups, even though they are less fit within each group. This may happen if like lives with like. The fact that the inequality within groups is reversed when we look at the global population is an example of Simpson's paradox (Sober, 1984).

Selection can make altruism increase in frequency only if altruists are on average fitter than selfish individuals in the global population. It may sound contradictory to some biologists that altruists can be fitter on average than selfish individuals; I conjecture that this is because they define altruism in such a way that it cannot evolve at all—not even by group selection. This way of understanding altruism hardly helps make the question of group selection the empirical matter that it should be.

Fisher (1930) provided the canonical statement of how individual selection judges the way a parent determines the sex ratio of her offspring. Roughly, the best sex ratio strategy is to produce offspring of the minority sex. This leads the population to evolve by individual selection to an equilibrium 1 : 1 sex ratio. A parent producing far more daughters than sons may help augment the population's productivity; but she will be less fit within that population than a parent who follows Fisher's advice.

Let us call organisms who always produce female-biased sex ratios *Hamiltonians* and individuals who do what Fisher recommended *Fisherians*. Suppose we have an ensemble of populations, each made of a mixture of Fisherians and Hamiltonians. What will happen? Within each group, Fisherians will do better than Hamiltonians. But this does not determine whether Fisherians will be fitter on average across the ensemble of groups (Simpson's paradox again). If the parameters are right, Hamiltonianism may evolve and be maintained.

Hamiltonians are *altruists* in the sense of figure 6.2. Groups of Hamiltonians do better than groups of Fisherians, but a Fisherian in a group does better than a Hamiltonian in that same group. This is why I agree with

Colwell (1981) and Wilson and Colwell (1981) that a female-biased sex ratio is evidence of group selection. Altruism (Hamiltonianism) is counter-predicted by individual selection acting alone.

Maynard Smith follows Hamilton's (1967) treatment of female-biased sex ratios. By reparameterizing the model a little, one can show that under certain conditions, Hamiltonians will be fitter on average than Fisherians, and this explains why Hamiltonianism increases in frequency. Maynard Smith believes this shows how to account for female-biased sex ratios within the confines of individual selection. My objection here is the one I registered against genic selection before.[6] It is entirely correct that when Hamiltonianism increases in frequency under selection, Hamiltonians must be fitter on average than Fisherians. But this is a fact about correlation, and so does not address the question of whether the process involves group selection.

5 *Conclusion*

Hamilton's (1967) ground-breaking work on sex ratio is an example of how models can provide fruitful insights even when they are misleading about the causal facts. For this reason, I entirely agree with Maynard Smith that there is every reason to investigate a plurality of models of a single process. Each may afford its own insights, even though each may have its special limitations.

However, biology also takes an interest in whether pluralism is plausible as regards the processes themselves. Does natural selection proceed almost entirely in the Darwinian mode, or do other forms of selection also make important contributions to the diversity we observe? To assess pluralism, either positively or negatively, it is essential to become clear on how selection processes differ from one another. It is at this point that the concept of cause becomes central.

Notes

1. Michael Wade's (1976) experimental work on group selection in the flour beetle *Tribolium* has the structure described above.
2. Here I correct a careless remark on p. 276 of my book *The Nature of Selection* (Sober, 1984).
3. The argument put forward by Williams (1966) and Dawkins (1976) that genic selectionism is preferable because it is more "parsimonious" is critically evaluated in Sober (1984, chapter 7).
4. Philosophers may wish to compare this conclusion with Davidson's (1966) observation that "gred" (= "green or red") is a natural predicate provided we apply it to emeroses (= emeralds or roses).
5. This is not the view disputed in Williams (1966) or in Dawkins (1976). There the dispute between genic selectionism and its rivals was substantive and empirical.

6. Other objections to the idea, not the algebra, of "local mate competition" are developed in Sober (1984, chapter 9).

References

Colwell, R. (1981) Group Selection Is Implicated in the Evolution of Female-Biased Sex Ratios. *Nature* 290:401–404.
Davidson, D. (1966). Emeroses by Other Names. In *Essays on Actions and Events*, Oxford: Oxford University Press, 1980, pp. 225–227.
Dawkins, R. (1976). *The Selfish Gene*. Oxford: Oxford University Press.
Dawkins, R. (1982). *The Extended Phenotype*. Oxford: W. H. Freeman.
Fisher, R. (1930). *The Genetical Theory of Natural Selection*. New York: Dover.
Hamilton, W. D. (1964). The Genetical Theory of Social Behavior: I and II. *Journal of Theoretical Biology* 7:1–52.
Hamilton, W. D. (1967). Extraordinary Sex Ratios. *Science* 156:477–488.
Lewontin, R. (1970). The Units of Selection. *Annual Review of Ecology and Systematics* 1:1–14.
Lewontin, R. (1974). *The Genetic Basis of Evolutionary Change*. New York: Columbia University Press.
Maynard Smith, J. (1964). Group Selection and Kin Selection. *Nature* 201:1145–1147.
Maynard Smith, J. (1976). Group Selection. *Quarterly Review of Biology* 51:277–283.
Maynard Smith, J. (1987). How to Model Evolution. In this volume.
Sober, E. (1984). *The Nature of Selection: Evolutionary Theory in Philosophical Focus*. Cambridge, MA: Bradford-MIT Press.
Sober, E., and R. Lewontin (1982). Artifact, Cause, and Genic Selection. *Philosophy of Science* 47:157–180.
Wade, M. (1976). Group Selection among Laboratory Populations of *Tribolium*. *Proceedings of the National Academy of Sciences USA* 73:4604–4607.
Williams, G. C. (1966). *Adaptation and Natural Selection*. Princeton: Princeton University Press.
Wilson, D. S. (1975). A Theory of Group Selection. *Proceedings of the National Academy of Sciences USA* 72:143–146.
Wilson, D. S. (1980). *The Natural Selection of Populations and Communities*. Menlo Park: Benjamin/Cummings.
Wilson, D. S., and R. Colwell (1981). Evolution of Sex Ratio in Structured Demes. *Evolution* 35:882–897.
Wynne-Edwards, V. C. (1962). *Animal Dispersion in Relation to Social Behavior*. Edinburgh: Oliver and Boyd.

7

Reply to Sober

John Maynard Smith

I disagree with almost everything Sober says. But I must confine myself to essentials. I shall argue that he does not understand what the "group selection" argument was about, and that he is wrong to say that there can be group adaptations without group heredity.

First, the group selection debate. In 1962, when Wynne-Edwards published his book, biology was riddled with "good-of-the-species" thinking. Again and again, one met in the literature explanations of some trait—of behavior, breeding biology, life history, or ecological interaction—in terms of the benefit that the trait conferred on the species, or even on the ecosystem as a whole. It was quite clear to me, as it must have been clear to George Williams, that no progress would be made toward understanding the evolution of such traits until this kind of thinking was ended. It was Wynne-Edwards's great merit that he saw that the evolution of such traits did require some special explanation, even if he was, in my view, mistaken as to what that explanation should be. In effect, his book brought the whole topic out into the open. Group-selection thinking could no longer be tacit and semiconscious: it had to be explicit.

The response that I and others attempted to make was a very simple one. Entities will evolve adaptations if, and only if, they have the properties of multiplication, heredity, and variation. For example, the eye is a group of cells, but it has been able to evolve as an adaptation for seeing because the cells that form it are part of a larger group, the organism, that does have heredity. (It is also important that the possibilities of between-cell, or between-gene, within-organism selection are very limited.) It is therefore perfectly justified to study eyes (or, for that matter, ribosomes, or foraging behaviors) on the assumption that these organs adapt organisms for survival and reproduction. But it would not be justified to study the fighting behavior of spiders on the assumption that this behavior evolved because it ensures the survival of the species, or to study the behavior of earthworms on the assumption that it evolved because it improves the efficiency of the ecosystem.

This point may seem so obvious as not to need stressing. I can only say that it was not obvious to everyone twenty years ago. If Sober's way of

describing the world is taken seriously, it will again cease to be obvious, and someone (not me, next time) will have the job to do over again.

But is it really true that groups without heredity do not have adaptations? Is it not the case that small groups of females whose offspring mate among themselves produce a female-biased sex ratio, which is advantageous for the group, even though, within a mixed group, "Fisher" females (producing a 1:1 ratio) are fitter than "Hamilton" females (producing a female-biased ratio)? Of course it is true, but it is not a group adaptation. If it were a group adaptation, we would expect the sex ratio produced to be that which is optimal for the group, but it is not. For the group, the best sex ratio would be more female biased than it is. In fact, the sex ratio that evolves is the one that is optimal for the individual, in terms of the number of genes passed on to grandchildren. To see this, look at table 5.2. In a mixed group, it is true that the Fisher female does better than the Hamilton one. But, and this is the point, in such a group the Fisher female would do better still if she switched over to the Hamilton ratio (24 instead of 21.3), and the Hamilton female would do worse if she switched to the Fisher ratio (16 instead of 18.7). The Hamilton ratio is the one that maximizes individual fitness: it does not maximize group fitness.

If individuals assort in groups, but those groups do not have heredity, then the traits that evolve will be those that maximize the fitness of individuals, and not of groups. Sober discusses at some length a model in which groups are formed assortatively, with like individuals coming together. Even in this case, if you wanted to know what traits would evolve, you would have to find the trait that maximized individual fitness, allowing for the fact that the individual is likely to assort with others of the same kind. Of course, this would not be the same trait that evolved if individuals assorted randomly, but so what? It need not be the case that the same trait maximizes fitness regardless of the environment in which an individual finds itself.

As it happens, I do not think that such assortment of like with like is common in nature, although it does occur. What is common is that neighbors are genetic relatives. As Hamilton has taught us, this does affect the course of evolution. As I explained in chapter 5, there are several ways in which one can analyze such cases: by inclusive fitness, or neighbor-modulated fitness, or by a more explicitly gene-centered approach. But the one thing one cannot do is assume that the trait that evolves will be the one that maximizes the fitness of some group—even if a group exists.

My central point, then, is that entities that do not have heredity do not evolve adaptations. I shall add a few words about "causation," although I accept that I am now playing on Sober's home ground. He thinks that "... context dependence can undermine a causal claim." I agree that if all explanations must be causal, and if causes must be independent of context,

then my way of seeing evolution is wrong. For a start, evolutionary game theory collapses, because it assumes that fitnesses are frequency-dependent. So much the worse, in my view, for Sober's concept of causation. Biology could not operate with context-independent causes. However, Sober does not really think that context-dependence undermines causal claims: if he did, he could not offer his explanation of sickle cell anemia. After all, AS heterozygotes are only fitter than homozygotes if the environment is malarial. His real objection, then, is to the artificality of my explanation of sickle cell anemia as a case of gene selection. But I think this is because he chose an unnecessarily complicated way of presenting the argument. If I say, "Allele S is fitter than allele A, given that (i) the world is malarial and (ii) the allele inherited from the other parent is A," I cannot see why the first kind of context-dependence is causally permissible, but not the second. Of course, I have in this case no objection (other than algebraic messiness) to the individual-selection way of seeing things. I discussed the case only because Lewontin and Sober chose it as their example to illustrate the causal inappropriateness of gene-centered models, whereas it seems to me that a causally sensible (and algebraically elegant) gene-centered model is possible.

I conclude by emphasizing that I do think there is a difference between causation and correlation. A correlation between A and B is evidence that there is some kind of causal link between them. It may be that A causes B, or that B causes A, or that some third factor, C, causes both A and B. In the third case, I would say that there is "only" a correlation between A and B. In the case of genes, characters, and fitness, I think that (dependent, of course, on context) a gene difference can cause a character difference, and that a character difference can cause a difference in fitness. It is not merely a correlation. Hence, when I wrote "... an animal with allele A, rather than a, is more likely to do X in environment E," I did mean that allele A causes the animal to do X.