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How to Model Evolution

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1 *Introduction*

In recent years, a number of arguments have occurred between evolutionists about the best way of modeling various evolutionary processes. There is agreement about the basic biology of the situation, and about the results to be expected. The disagreements concern such questions as whether a particular process is best seen as a case of individual selection, gene selection, group selection, or kin selection, or whether inclusive or classical fitness is the appropriate measure. Perhaps these arguments are trivial: if the deductions from the different models are the same, there can be no empirical choice between models, and a good Popperian can regard the whole matter as pseudoscience.

I do not think we can avoid the problems quite that easily, for the following reason. When confronted with a biological phenomenon, one first attempts to fit it into one of the categories with which one is familiar. For example, one might think such things as "This is a case of heterosis," or "Starling roosts are selfish herds," or "This biased sex ratio arises from local mate competition." Any such first idea is quite likely to be wrong. Further investigation would require a more precise formulation of the hypothesis, a search for alternatives, and an attempt to decide between them empirically. However, one does start out with a set of formal categories in terms of which one attempts to interpret phenomena. In the absence of such categories, one could not hope either to explain phenomena in terms of existing theory or, more important, to recognize that some phenomenon does not fit existing theory.

I think that, for most of us, phrases such as "group selection" or "mixed ESS" represent the causal structure of an explanation, rather than the

An earlier draft of this paper has been modified in the light of two sets of written comments. In response to Elliot Sober, I have tried to clarify why I think that causes can be context-dependent in their effects. In response to Richard Dawkins, I have explained the difference between the distinction I make here between units of evolution and units of selection, and his distinction between replicators and vehicles. I thank them both for forcing me to be more explicit.

algebraic details. If so, our choice of models, and to some extent our choice of words to describe them, is important because it affects how we think about the world. In saying this, I am agreeing with a view recently expressed by Sober and Lewontin (1982), who argue that what matters about a model is that it should have the correct causal structure, and that computational convenience is secondary. To give an example where I think this is true, consider the statement that light passes from one point to another by that path that minimizes the passage of time. This is correct and mathematically convenient, but it would be causally misleading if it led one to suppose that the light beam computed an optimal path. In physics, no one is likely to be misled, but the dangers are much greater in biology. As against this, if a model leads to mathematical simplicity, that implies that it is easy to reason about, which is greatly in its favor. I am uncertain about the importance of computational simplicity, but I do not think it can be rejected as confidently as do Sober and Lewontin.

In this essay, I aim to do the following. First, by discussing the term "adaptation," I shall illustrate how our choice of model decides what phenomena we regard as readily explicable, and which need further investigation. Second, I shall illustrate how two alternative methods of modeling the same process may be available, by giving two examples—heterozygous advantage and the sex ratio in subdivided populations. Finally, I shall discuss how far "gene selection" models of evolution are appropriate, and contrast several approaches to the modeling of interactions between relatives.

2 *Adaptation*

The problem solved by Darwin was not only to suggest a mechanism whereby populations would change in time—that is, would evolve; he also explained a particular characteristic of evolution, which was overwhelmingly apparent to him, as to all naturalists. This is the fact that organisms have a structure and behavior that adapts them to survive and reproduce. For Darwin, the task was not to prove that organisms are well adapted to particular ways of life: it was to explain how they came to be so.

The explanation he offered was, in effect, that if there is a population of entities with multiplication, variation, and heredity, and if some of the variations alter the probability of multiplying, then the population will evolve. Further, it will evolve so that the entities come to have adaptations in the sense of the last paragraph. For Darwin, the relevant entities were individual organisms. Hence his theory provided an explanation of adaptations ensuring the survival of individuals. He knew nothing of genes, and was clear that his theory did not predict species-level adaptations. He slipped up at least once, but later corrected himself. In the first edition of

The Descent of Man, he explained the 1 : 1 sex ratio by arguing that such a ratio would benefit the population. By the second edition, he had seen his error, and wrote, "I formerly thought that when a tendency to produce the two sexes in equal numbers was advantageous to the species, it would follow from natural selection, but I now see that the whole problem is so intricate that it is safer to leave its solution to the future." It is intriguing that his greatest nineteenth-century follower, August Weismann, had a similar experience. As is widely known, he interpreted senescence as a species adaptation, arguing that only if individuals died could the population evolve. However, he too saw this mistake, and later offered an explanation of the evolution of senescence that is not too far from the theory (Williams, 1957) that most of us today would accept.

Thus Darwin's theory predicts certain kinds of adaptation, but not others. For example, the statement "The emargination of the primary feathers of birds evolved because it has the effect of producing wing slots that reduce the stalling speed" is compatible with the Darwinian model, whereas "Earthworms eat dead leaves because this releases nutrients into the soil that nourish plants" is not. Note that I am not saying that the former statement is correct and the latter not, but only that Darwin's theory predicts adaptations of the former kind, but not of the latter. Note also that there is nothing in the theory to predict that adaptations will be perfect.

Today, we are asking whether there are entities other than individuals with the properties of multiplication, heredity, and variation, and that therefore evolve adaptations by natural selection. In particular, are genes, or populations, or species, such entities? I shall refer to such entities as "units of evolution." To qualify as a unit of evolution, it is not sufficient that an entity be selected for or against: it must have heredity. In contrasting units of evolution with units of selection, I am making a distinction different from that drawn by Dawkins (1976) between a "replicator" (i.e., an entity whose structure and information content is copied more or less precisely in the process of reproduction) and a "vehicle," whose structure is not replicated, but that is the object upon which selection typically acts. Thus organisms are units of evolution, but they are not replicators. What makes organisms into units of evoluion is that

> i. they have heredity, in the sense in which Darwin would have used the word—that is, offspring resemble their parents—and
> ii. the replicators, or genes, that are responsible for heredity behave in a way that, typically, does not permit within-individual, between-replicator selection; thus in typical cell division one copy of each gene present in the mother cell is transmitted to each daughter cell, and in meiosis each member of a pair of genes is equally likely to be transmitted.

There are, of course, exceptions to the second condition—for example, in "meiotic drive." However, if meiotic drive were the rule, organism-level adaptations would not have evolved, and there would be no organisms to qualify as units of evolution.

Thus whether an entity qualifies as a unit of evolution depends on the relationship between that entity and the replicators that are ultimately responsible for heredity. In this sense, the distinction between units of evolution and selection is less fundamental than that between replicators and vehicles. Nevertheless, it is important to distinguish between objects we can expect to evolve adaptations and those we cannot. In particular, selection may act between groups of organisms, but it does not follow that group adaptations will evolve.

Consider, for example, the following imaginary scenario. Each generation of a large, random-mating population breaks up into groups of 100 individuals. These groups are then selected according to some criterion—for example, their ability to defend themselves against predators—some groups being wiped out and others surviving. The members of the surviving groups then reenter the random mating pool. In this example, there is no question that selection is acting on the groups. But it would be dangerous to think of the groups as units of evolution. If we did, we would expect the groups to evolve adaptations ensuring their survival—for example, the existence of some individuals that sacrifice their lives to ensure the survival of the rest. Once we appreciate that the groups do not (at least, in the usual sense) have heredity, we shall be cautious about assuming that such suicidal individuals will evolve: if we observe them, we shall suspect that there is something about the situation we do not know. If we want to know whether self-sacrificing behavior will evolve, we shall treat the individuals as the units of evolution (while accepting that selection acts on the groups), and ask whether individuals with a genotype that makes them sometimes sacrifice themselves are on average more or less likely to survive—or, and I shall return to this later, we may prefer to treat the genes as the units of evolution.

The point made in the last two paragraphs is illustrated in table 5.1. I suppose that there are two kinds of individuals—S ("selfish") and A ("altruistic"). Groups of two are formed randomly, and individuals contribute numbers of offspring to the next generation as shown in the table. In table 5.1a, both altruism and selfishness are ESSs: that is, a population of altruists would be stable against invasion by selfish mutants, and vice versa. We might be tempted to argue that the stability of altruists is guaranteed by the fact that groups of altruists are, as groups, fitter than other groups (combined output of 8 offspring, as opposed to 4 offspring from AS and SS groups). However, this would be a mistake, as shown by table 5.1b. Here, selfishness, S, is the only ESS, although again groups of altruists are, as

Table 5.1
Fitnesses of "altruistic" (A) and "selfish" (S) individuals interacting in pairs

	Case (a)			Case (b)			Case (c)		
Group fitness	A,A	A,S	S,S	A,A	A,S	S,S	A,A	A,S	S,S
	4,4	1,3	2,2	4,4	1,5	2,2	4,4	3,5	2,2
		A	S		A	S		A	S
Payoff matrix	A	4	1	A	4	1	A	4	3
	S	3	2	S	5	2	S	5	2

groups, fittest. Hence selection between groups does affect the course of evolution, but it is better to think of such group selection as acting by altering individual fitnesses, because it is individual and not group fitness that is maximized. However, if groups had heredity, and produced groups like themselves, then altruism would evolve for the fitnesses of table 5.1b. For completeness, table 5.1c shows fitnesses for which neither altruism nor selfishness is an ESS, and a stable polymorphism results.

There has been some semantic confusion about the phrase "group selection," for which I may be partly responsible. For me, the debate about levels of selection was initiated by Wynne-Edwards's book, *Animal Dispersion* (1962). He argued that there are group-level adaptations—for example, "epideictic displays"—which inform individuals of the size of the population so that they can adjust their breeding for the good of the population. He saw clearly that such adaptations could evolve only if populations were units of evolution in the sense that I have used the term here. Perhaps unfortunately, he referred to the process as "group selection." As a consequence, for me and for many others who engaged in this debate, the phrase came to imply that groups were sufficiently isolated from one another reproductively to act as units of evolution, and not merely that selection acted on groups.

The importance of this debate lay in the fact that group-adaptationist thinking was at that time widespread among biologists. It was therefore important to establish that there is no reason to expect groups to evolve traits ensuring their own survival unless they are sufficiently isolated for like to beget like. It is, I think, not an accident that the present interest in evolution of sex, recombination, and breeding systems generally was initiated by Williams (1975) and myself (Maynard Smith, 1971, 1978), both of whom had been engaged in the attempt to correct group-selection thinking.

When Wilson (1975) introduced his trait-group model, I was for a long time bewildered by his wish to treat it as a case of group selection, and doubly so by the fact that his original model (which permitted only addi-

tive fitness interactions) had interesting results only when the members of groups were genetically related, a process I had been calling kin selection for ten years. I think that these semantic difficulties are now largely over, with the use of the terms "intrademic group selection" and "trait-group selection" for the process envisaged by Wilson, and "interdemic selection" and "species selection" for that envisaged by Wynne-Edwards (according to whether the "groups" are demes or species).

There remains the question whether it is really true, in the trait-group model, that there is no heredity. What is required for the Darwinian process is that there should be different kinds of entities, *A*'s, *B*'s, *C*'s, etc., and that when these replicate, *A*'s should give rise to *A*'s, *B*'s to *B*'s, and so on. For the imaginary example of trait groups of 100, a group with, say, 60 *A*'s and 40 *B*'s does not give rise to a group of the same composition. Instead, the individuals enter a random-mating pool and contribute genes to many different groups in the next generation. However, the same objection holds for individuals in a sexual population. Consider, for simplicity, changes in the frequency of a pair of alleles at a locus. Individuals can be thought of as a kind of trait group of two genes, upon which selection acts, before the genes reenter the gene pool, and pair up with new partners in the next generation. On this model, the entities with heredity are the genes: individuals are merely temporary trait groups.

There are two possible replies to this. One, made by Williams (1966) and Dawkins (1976), is to accept that the genes are indeed the units of evolution. To quote Dawkins, "I shall argue that the fundamental unit of selection, and therefore self-interest, is not the species, nor the group, nor even, strictly, the individual. It is the gene, the unit of heredity." (Note that Dawkins is using the term unit of selection in the sense that I am here using unit of evolution.) There is much to be said for this point of view, although it does not wholly solve the difficulty of identifying the unit of heredity. How small is a gene? If we think of the gene as the unit of function, it can be broken up by recombination. If we insist on a unit that is never broken up, we are left with the absurdity of a single base pair. Dawkins, following Williams, says, "A gene is defined as any portion of chromosomal material which potentially lasts for enough generations to serve as a unit of natural selection."

The alternative is to stick to the individual organism as the unit of evolution, on two grounds. First, with only two alleles at a locus, the correlation between parent and offspring is high (one-half, if we ignore environmental variance and non-additive gene effects). Second, and more fundamental, there is typically no analogue of within-group, between-individual selection, because the two alleles at a locus have equal chances of passing on a gamete. In our imaginary example of trait groups of 100, the reason why group adaptations could not readily evolve is that there was within-group selection: self-sacrificing individuals are less likely to

survive. As soon as Mendelian rules break down (as in meiotic drive, or transposable elements), the basis for the evolution of individual adaptation breaks down also.

Although there are two alternatives, I do not think we have to choose between them. In developing evolutionary game theory, I have adopted the individual as the unit, but there are many problems for which a gene's eye view is more appropriate. Since Dawkins wrote enthusiastically of game theory, I take it that he accepts that it can be useful to think of individuals as the units. I suspect, however, that he would argue that a gene-centered view is in some sense more fundamental, and I agree with him.

3 *Alternative Models of Evolution*

At this point, it will be useful to look at two examples of biological processes that can be viewed in different ways. I repeat that in neither case is there any argument about the biological facts, or about the results to be expected: it is simply an argument about viewpoints.

The first, used by Sober and Lewontin (1982), is sickle cell anemia, the classic example of heterozygous advantage. There are, in human populations, two allelic genes, A and S, coding for the β chain of hemoglobin. S/S homozygotes usually die from anemia. A/A homozygotes do not suffer from anemia, but may die from malaria if that disease is prevalent. The A/S heterozygotes do not die either from malaria or anemia: thus they are fitter than either homozygote. As a consequence, both alleles are maintained in populations at high frequency in malarial regions. Sober and Lewontin argue that it would be absurd to regard this as an example of selection at the genic level, although they concede that the approach has some computational advantages. Obviously, selection acts on the individual. The "fitness" of a gene depends on which other gene it finds itself with: an S gene is fit if it unites with an A gene, but unfit if with another S gene. Since Sober and Lewontin regard causal appropriateness as a more important feature of a model than computational convenience, they insist that the individual selection model is the right one.

At first, I was persuaded by this argument, but now I am less certain. Let me start with the computational side. The classic, individual selection approach is to ascribe fitnesses—say $1 - s$, 1, $1 - t$, respectively—to the three genotypes AA, AS, SS. If one wishes to find the equilibrium frequency, say p, of allele A, one writes down an equation for p', the frequency in the next generation, as a function of p, s, and t, and then solves the equation $p' = p$. The result is a cubic. This is not as bad as it sounds, because the equation can be factorized. However, I have seen generations of biology undergraduates discouraged by the messy algebra.

Let us now approach the problem by ascribing fitnesses to the alleles A and S. The fitness W(A) of allele A can be written as (probability that A unites with A) × (fitness of AA) + (probability that A unites with S) × (fitness of AS): that is, $W(A) = p(1 - s) + 1 - p = 1 - ps$. By a similar argument, the fitness of the S allele = $W(S) = 1 - t + pt$. Hence, at equilibrium, $1 - ps = 1 - t + pt$, or $p = t/(s + t)$.

This is computationally so much nicer than the textbook method that one is tempted to seek a causal justification for it. Such justification is not hard to find. After all, the A and S genes do cause the appearance of specific chains, and these in turn cause the death or survival of their carriers. This implies that the fitnesses of the A and S alleles depend on which other allele they find themselves with, and hence are frequency-dependent. It might be objected that the genes themselves cannot be thought of as the causes of fitness differences, because a given gene substitution is sometimes associated with an increase of fitness, and sometimes with a decrease. I do not think this objection is valid. 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 difference. Indeed, it is often the case that individual fitnesses are frequency-dependent. Evolutionary game theory was developed to analyze such when detailed genetic information is lacking. By analogy, I am tempted to say that the best way of representing the sickle cell problem is in the form of a payoff matrix to genes as follows:

	A	S
A	$1 - s$	1
S	1	$1 - t$

This is an example of the Hawk-Dove game: the ESS is given by $p = t/(s + t)$.

My second example, recently reviewed by Harvey, Partridge, and Nunney (1985), concerns the sex ratio with local mate competition (Hamilton, 1967). There is a choice of at least three methods: classical population genetics, a game-theoretic approach based on individual fitnesses, and intrademic group selection (Colwell and Wilson). Hamilton sought the stable sex ratio when the offspring of several females mate among themselves before dispersal. Using a game-theoretic approach, he showed that, if the offspring of n females interbreed, the stable proportion of males is $(n - 1)/2n$. The essence of the problem can be understood from the case $n = 2$. Consider two kinds of females, Hamilton (H) females, producing 1♂:3♀, and Fisher (F) females, producing 1♂:1♀. There are three possible groupings (table 5.2). The "fitnesses" in the table are the expected values of "number of genes passed on to grandchildren." From the payoff matrix, it is clear that, if H and F are the only possible strategies, then H is the only

Table 5.2
The sex ratio game, assuming four offspring per female

Type of female	H	H	H	F	F	F
Number of offspring	3♀, 1♂	3♀, 1♂	3♀, 1♂	2♀, 2♂	2♀, 2♂	2♀, 2♂
Grandchildren per child	4, 12	4, 12	4, 20/3	4, 20/3	4, 4	4, 4
Total grandchildren	12 + 12 = 24	12 + 12 = 24	12 + (20/3) = 18.7	8 + (40/3) = 21.3	8 + 8 = 16	8 + 8 = 16

Payoff matrix		H	F
	H	24	18.7
	F	21.3	16

ESS. It is not difficult to show that the ratio $(n - 1)/2n$ is stable against any other ratio.

This is the individual selection model. There are two alternatives. The first is to recognize that the above definition of "fitness" is unorthodox and potentially misleading, particularly when the genetic system is haplodiploid (as in the parasites considered by Hamilton). The classic population genetics approach is to consider a population with two alleles, determining the sex ratios S and s, respectively, and write down the recurrence equations for their frequencies (Maynard Smith, 1978). One then seeks a sex ratio allele S such that no alternative allele s can invade an S population. This is a kind of hybrid between classical population genetics and game theory: fortunately, it yields the same stable sex ratio as Hamilton's more intuitive approach.

The other possible departure (Wilson and Colwell, 1981) is to note that, in mixed groups, F females do better than H females, and that the success of the H phenotype arises because all-H groups do better than all-F or FH groups. This is the intrademic group selection approach. It is worth noting that in this example the fitnesses of groups are constant but of individuals frequency-dependent, whereas in the sickle cell example the fitnesses of individuals were constant but of genes frequency-dependent.

It would be easy to multiply examples from evolutionary genetics in which a variety of different approaches are possible. Is there any sense in which one of these is best? I am inclined to think not. Classical population genetics models, in which fitnesses are ascribed to genotypes (or to genotypes in specific environments), and recurrence equations for gene frequencies written down, have the virtues of clarity and correctness, but are often algebraically messy and lacking in perspicuity, as will become clearer when I discuss models of the evolution of altruism. Game-theoretic models are often algebraically simpler, and lend themselves to a kind of strategic

thinking—"If I were an animal—or a gene—what would I do?" They have an obvious justification in analyzing the evolution of traits of whose genetics we know little or nothing. But they can be misleading, and may need checking by more formal genetic models. The intrademic group selection model has the virtue of emphasizing that those factors that increase between-group genetic variance relative to within-group will favor the spread of altruistic traits, but seem needlessly tied to cases in which the population has a group structure, and not merely neighbors.

4 *The Evolution of Altruism*

One virtue of the trait-group model is that it brings out particularly clearly that there are two contexts in which traits that are advantageous to the group but disadvantageous to the individual may spread: the first is when fitnesses combine nonadditively, and the second when interactions are between relatives. It is the second of these possibilities that has been the greatest stimulus to the development of a gene-centered view of evolution. Before discussing this approach in detail, however, I want to meet an objection to it that has a political and philosophical origin rather than a biological one. This is the objection to "genetic determinism." It has been argued that to base a biological model on the idea that gene A causes an animal to do X leads too easily to the assumption that the economic backwardness of Africa, the low pay of women, or the small proportion of working class children entering Oxford has a genetic rather than a social cause. I think this is a red herring. 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. If this were not so, it is hard to see how behavior could evolve. It does not follow that the human inequalities listed above have a genetic cause, and I do not think that they do. Those who do hold hereditarian views about racial, sexual, and class inequalities usually imagine that heredity is carried in the blood: I doubt if they are much encouraged by the knowledge that altruism requires $B/C > 1/r$. Finally, it does not follow that biologists who espouse gene-centered models also think that genetic differences are more important in our species than environmental ones: for example, Dawkins is clearly not a genetic determinist.

There are, I think, three main approaches to the evolution of traits that have different effects on individual and group survival:

(i) Classical or "Neighbor-Modulated" Fitness

Let us consider a gene A causing its carriers, which we shall call type X, to be more likely to perform an altruistic act. If we want to know whether A

will increase in frequency, all we need to know is whether X individuals are "fitter"—that is, produce more offspring—than others (if there is dominance, there are three types whose relative fitnesses must be known). What happens to relatives of X individuals is irrelevant. However, when we calculate the fitness of X, we must remember that X individuals are more likely to have A-carrying relatives than are non-X, and hence are more likely to receive help. If we allow for this, we shall obtain what Hamilton (1964) called neighbor-modulated fitnesses, which will correctly predict genetic change.

(ii) "Inclusive Fitness"

Hamilton (1964) defined the inclusive fitness of type X as the expected number of offspring to X, not including any additional offspring to X because of help from others, plus any additional offspring to others because of help from X, weighted by the appropriate coefficient of relationship. He showed that the direction of genetic change, and its approximate rate, predicted if genotypes were given their inclusive fitnesses, correspond to the correct values predicted by neighbor-modulated fitnesses.

Cavalli-Sforza and Feldman (1978) have argued that, since the neighbor-modulated approach is exact, it is pointless to use inclusive fitness, which is hard to define, easily misunderstood (for an analysis of some misunderstanding, see Dawkins, 1979, and Grafen, 1984), and can in any case be applied only when fitnesses combine additively. Further, if intuitive ideas are going to be checked by computer simulation, there is no need to calculate inclusive fitnesses: the approach has to be classical (see, for example, Seger, 1983, which is the closest approach yet to an analysis of the relation between eusociality and haplodiploidy).

The short answer is that the classical approach is unusable except as a way of checking an insight gained in other ways. For example, I suspect that Seger, when thinking about the relations between bivoltine life histories, distorted sex ratios, and eusociality, used inclusive fitness as a guide, although this does not appear from his simulations. There are two reasons why neighbor-modulated fitnesses are hard to use. The less important is computational; one has to calculate, for all interacting relatives of X, the probabilities that they have particular genotypes, and not merely the gene frequencies in relatives (an example of how difficult this can be is given by Maynard Smith, 1982). The more serious difficulty is that one has to think causally backward. One has to say, "The reason why this bird is giving an alarm note is that the gene that is causing it to do so is likely to be present in other members of the flock, so those other members are also likely to give alarm notes, thus ensuring the survival of the gene we started with." I doubt whether many people can think that way.

(iii) Gene-Centered Models

In *The Extended Phenotype*, Dawkins argues that Hamilton's concept of inclusive fitness was a last-ditch attempt to treat the individual as the unit of evolution, and that he would have been wiser to abandon the attempt in favour of a fully gene-centered view. Grafen (1984) takes the same position, arguing that Hamilton's condition, $Br > C$, for the spread of a gene is a more useful approach than the calculation of inclusive fitnesses. In effect, one asks, for some allele A: "If allele A is expressed, and causes some action (or, more generally, some phenotype), are there as a result more or fewer copies of A than there would have been if A had not been expressed?" Such an intuitive approach can, in critical cases, be supported by a classical population genetics model, analytically or by simulation.

5 *Conclusion*

The issues discussed in this essay are conceptual rather than empirical. The most important distinction I have made is between what I have called "units of evolution," which must possess heredity, and "units of selection," which need not. The distinction is important because we can expect units of evolution to have complex properties ensuring their survival and reproduction. Since it is genes rather than individuals that replicate, how does it happen that most adaptations seem to be properties of individuals rather than of genes? The answer is that, so long as genes do not multiply horizontally, only those genes survive that ensure the survival of the individuals in which they find themselves. If gene selection within individuals (as in meiotic drive and transposition) were the rule rather than the exception, complex organisms would not evolve. Since, in higher level groups (demes, species, communities), there is pervasive between-individual, within-group selection, we cannot expect complex population-level adaptations.

The same biological process can often be modeled in more than one way. Different models may make the same predictions, yet give different insights: in such cases, we are not obliged to choose between them. There is much to be said for looking at a problem from different points of view. This plea for pluralism, however, differs from that recently made by Gould (1980). I am recommending a plurality of models of the same process: he is emphasizing a plurality of processes, and in particular selection at different hierarchical levels. His claim is in part an empirical one; for example, it includes the claim that differential survival and splitting of species as determined by species-level properties has been important in evolution. In the past, confusion between conceptual and empirical issues has generated needless heat. For example, the argument between Wynne-Edwards and others about "group selection" was ultimately empirical: are populations

sufficiently isolated, and free of intragroup selection, to evolve complex group adaptations? In contrast, my disagreements with D. S. Wilson have been partly semantic (what shall we mean by "group selection"?), and partly concerned with whether intrademic group selection is a useful way of modeling a process that we agree happens, but that can be modeled in other ways. Let us hope that similar confusions will not needlessly exacerbate the debates now taking place about selfish DNA, concerted evolution, and species selection.

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