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Units of Selection and the Structure of the Multi-Level Genome <sup>1</sup>

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1. Introduction: Williams' Concept of the Gene

The dominant view among evolutionary biologists today is that the gene is the only unit of selection. According to this view, larger units are too unstable in evolutionary time to act as units of selection. Chromosomes are broken up by sexual recombination. At the level of the individual organism, phenotypes pass on genes, but (it is claimed!) last themselves only one generation. Genotypes of individuals, collections of chromosomes, are rearranged by Mendelian independent assortment in sexual reproduction and so also persist for only one generation. Groups of individuals are still more ephemeral, or so we are told.

All of these points are argued at length by George C. Williams in his book Adaptation and Natural Selection which, since its publication in 1966, has been the watershed for the rise to dominance of this reductionistic vision of evolutionary theory. This vision has entered the popular consciousness through the rise of sociobiology, but it is endemic to the formulations of evolutionary theory which spawned that discipline.

In Williams' view, the apparent functional integration of the group is almost invariably to be explained away as the summed effects of adaptations of individuals (Williams 1966; Wimsatt 1970), and even the integration of the individual organism is usually to be explained as a means, a "survival machine" (Dawkins 1976) which is the joint product of a number of "selfish genes", each acting to maximize its own probability of survival.<sup>3</sup>

What then is a gene? Williams' characterizations of a gene are offered in cybernetic and general terms, in ways that suggest that genes might be found at any level of analysis, though his own more detailed arguments belie this impression. Thus he says:

- (1) "In its ultimate essence, the theory of natural selection deals with a cybernetic abstraction, the gene, and a statistical abstraction, mean phenotypic fitness." (Williams 1966, p. 33)
- (2) "In evolutionary theory, a gene could be defined as any hereditary information for which there is a favorable or unfavorable selection bias equal to several or many times its rate of endogenous change." (Williams 1966, p. 25)
- (3) "I use the term gene to mean 'that which segregates or recombines with appreciable frequency'." (Williams 1966, p. 24)

Williams' further comments (his "stability" arguments on pp. 22-24 against recognizing higher level units such as the genotype or phenotype as units of selection or adaptation) make it clear that he would add a further proviso to his third characterization: Not only is a gene that which segregates and recombines with appreciable frequency, but:

- (4) for an entity to be a gene, there must not be a significant degree of segregation or recombination within it.

If the gene is a "cybernetic abstraction", why must it be made of DNA? The cybernetic characterizations of the gene given in points (1) through (3) permit interpretation at a variety of levels. Dawkins (1976) uses this fact to argue in his last chapter that cultural evolution can be brought within the fold: his 'memes' are cultural genes, heritable through individual and cultural memory, and differentially replicated through processes operating at the cultural level. I believe that this insight is correct and allows higher level biological units of selection, as well as cultural ones.

It is the 4th criterion, applied to argue that sufficient stability for an entity to act as a gene is found only at the lowest possible level, which makes the difference. This criterion, as they apply (or misapply!) it forces both Williams and Dawkins (with the exception of Dawkins' last chapter) to a very reductionistic and material conception of the gene. In principle it is possible that there are higher level genes or units of selection according to criteria (1) and (2), they would say, but in fact these higher-level complexes are sufficiently unstable that they cannot count as genes (or units of selection)<sup>4</sup> and the only entities which are sufficiently stable to act as genes are relatively short strings of DNA. (The 3rd criterion is assumed, without argument, to apply only at the bottom level—an assumption which I will later show to be false.)

I believe that this reductionistic conclusion is fundamentally mis-

taken for a variety of reasons, only a few of which can be discussed here. Most important of these reasons is that the stability of genes is generally treated by Williams and Dawkins as a condition which must be met prior to and independently of any considerations of the reproductive rate of the entity in question. (Note that this is actually in conflict with Williams' 2nd criterion, which relates stability and reproductive rate. I will return to this below.) On their views, only the very stable entities survive for long enough to reproduce, and of these, only those with the highest reproductive rate survive the subsequent competition. But this separation of the effects of stability and reproductive rate ignores the fact that there is a direct tradeoff between stability and reproductive rate: things with a higher reproductive rate but lower stability may, in sum, reproduce at a far faster total rate than things with a higher stability and a lower reproductive rate, and thus be selected over them in spite of their lower stability. These tradeoffs are calculated and discussed in Wimsatt (1980b, pp. 244-246). The net effect of the existence of such tradeoffs is that the conditions for the existence of higher-level units of replication and selection are far less restrictive than imagined by Williams or Dawkins. Williams' own 2nd criterion suggests this result.

Another way of seeing this conclusion (and the one that will be pursued here) arises out of a discussion of Williams' claim that selection acting at any level can always be analyzed into components acting at the level of individual genes. This claim is motivated in the second section but is shown to be false in the third section of the paper. A discussion of the conditions under which it would be true in the fourth section suggest an operational criterion for detecting units of selection which is applicable at any level of organization. This definition and its consequences will be analyzed and defended in the fifth section of the paper. Finally, the existence of higher-level segregation analogues will be explicated in the sixth section, showing that the third criterion can also be met at higher levels of organization and that a multi-level selection theory need not always return to the lowest genetic level to find principles capable of yielding a dynamics for higher-level selection processes.

The philosophical and scientific consequences of the definition and existence of units of selection and analogues to Mendelian segregation at a variety of levels of organization will be explored in the closing section of this paper.

## 2. Multiple loci, interactions among genes, and the problem of complexity

The basis for the claim that the "selfish gene" can always be regarded as the unit of selection is again found in Williams' book. His view is that since the operation of any higher-level selection process can be mathematically expressed as resulting from the operation of selection coefficients acting independently at each locus to change the frequency of individual alleles or genes, there is no need to postulate the existence of any higher-level units of selection or selection

forces. This view will look most familiar to philosophers, since it bears the strongest resemblance to traditional philosophical accounts of theory reduction. Williams expresses it as follows:

Obviously it is unrealistic to believe that a gene actually exists in its own world with no complications other than abstract selection coefficients and mutation rates. The unity of the genotype and the functional subordination of the individual genes to each other and to their surroundings would seem, at first sight, to invalidate the one-locus model of natural selection. Actually these considerations do not bear on the basic postulates of the theory. No matter how functionally dependent a gene may be, and no matter how complicated its interactions with other genes and environmental factors, it must always be true that a given gene substitution will have an arithmetic mean effect on fitness in any population. One allele can always be regarded as having a certain selection coefficient relative to another at the same locus at any given point in time. Such coefficients are numbers that can be treated algebraically, and conclusions inferred from one locus can be iterated over all loci. Adaptation can thus be attributed to the effect of selection acting independently at each locus. (Williams 1966, pp. 56-57).

Williams goes on, in the next two pages, to illustrate how the algebraic manipulation can be accomplished in a simplified genetic environment of two alleles at each of two loci, and we are to imagine the extrapolation to cases of many alleles at many loci. Complicated it would be, but in principle, of course (we are told), it could be done, "by iterating over all loci." (See Wimsatt 1980b, on in principle claims.)

Before discussing this claim, it is useful to consider the motivations for making it. It is first of all natural to make it from the perspective of a reductionist—one influenced strongly by the developments in molecular biology and in population genetics. The history of genetics has shown a progression to smaller and smaller units of analysis (usually in smaller and faster-breeding organisms). This has led to the successive discovery of more and more about how genes replicate and are expressed in ways that appear to promise an ultimately molecular account of development and the production of character traits in the phenotype. It has even been possible in a very few cases (notably that of sickle-cell anemia) to trace the consequences of a single small change in the molecular structure of DNA up through the various levels of organization to assess and explain the consequences for selection of this single change, thus contributing to the view of the gene as a manipulator of the phenotype. The genes are clearly causally relevant to the production of the phenotype, and we think we see, at least in outline, how they are causally relevant.

By contrast, arguments that a given phenotypic feature would be selected for and thus cause the passing on of the genes responsible for its production (a change in perspective which makes this reductionistic vision harder to maintain) are usually much harder to make in a convincing fashion. (See, e.g., Lewontin 1978.) The effect of a given trait

is mediated by the complex of traits in the organism in which it occurs, and by traits in the organism's family, kin group, species, other species with which it interacts, and by its distribution and patterns of change in space and in time. (See, e.g., Levins 1968, and Wimsatt 1970, 1980b.) Their role must be included in a theory of its evolution.

In addition, the strong tendency to characterize the gene as the minimal unit of structure, function, recombination, or mutation owes much to the search which had occupied geneticists and cytologists since the 1880's for an understanding of how the genetic material functions. In that investigation, there were a number of strong biases favoring looking at the smallest units which can behave like genes: they are simpler to control, analyze, and understand (see Carlson 1966, for a history of this search). But the minimal unit (of, e.g., function), is not necessarily the usual unit of selection, which population biologists should try to model (if there is a usual unit of selection). The purposes of the investigation of the gene differ for molecular geneticists and for evolutionary biologists, but the remaining biases towards the gene as the minimal unit do not reflect this fact. The continued emphasis on the minimal unit is due, in part, to cultural inertia!

Another feature which makes the reductionistic vision attractive is the appearance of a universal language or vocabulary for analyzing and describing all similarities and differences in all organisms. Bacteria, viruses, slime molds, fungi, fish, fruit flies, flying squirrels, bats, baboons, men, elephants, whales, and sequoias have such an array of adaptations which they each employ in their own special environments that one could easily despair of ever comparing them at the level of their adaptive designs. Indeed, it is almost impossible to learn enough about any one of them to say with much certainty what its adaptive design and its selection pressures are (See Lewontin 1974, 1978).

But the simplicity of the language of DNA, with its 4-base alphabet, and the 20-base alphabet of amino acids in the proteins which it generates promises a tool of universal description and comparison, since all biologically meaningful "expressions" are but combinations of letters from these alphabets. All of these diverse organisms can be compared for the sequences of their proteins and DNA molecules, and this has already been done for a number of biologically important macro-molecules. (See, e.g., R. C. Dickerson 1972, for the phylogenetic comparison of cytochrome-c molecules of diverse organisms.)

This universality has promised so much that "molecular phylogenetics" has even threatened to displace the phenotypic character as the main tool in phylogenetic analysis, though there are increasing doubts about its ultimate usefulness. (See, e.g., Ayala 1976 and Throckmorton 1978, for some of the complexities facing "molecular phylogeneticists".) Genetic analysis provides at least a neat method of uniform bookkeeping, and has contributed in a variety of ways to our theoretical understanding, but can it provide all of the answers? Someone who thinks it does should ask a linguist how much understanding of the meaning and use of a

language comes with just a list of the alphabet of its basic symbols, and a "spelling list" of all of its words—each of which, after all, is "nothing more" than a combination of these basic symbols!<sup>5</sup> I will not pursue this argument further here, though this way of conceptualizing the problem is useful and I will discuss Williams' "genetic bookkeeping" again in the next section.

Probably the strongest reason for attempting to analyze complex selection forces at the level of individual genes has to do with the apparent intractability of almost any other way of proceeding, at least if we must proceed "from the bottom up." It is no accident that virtually all models of the evolution of traits characteristic of the recent "Sociobiological Revolution" employ only the simplest possible mathematical model of selection—that of 2 alleles at 1 locus. This is because the more complex and realistic models involving interactions of multiple alleles at multiple loci cannot be solved analytically. Even the case of 2 alleles at 2 loci has many unplumbed depths, and has been solved only for a variety of special cases (See Roughgarden 1979, chapter 5; and Nagylaki 1977). The case of the 3-body problem is legendary in classical physics. For the population geneticist, the analogous threshold of analytical intractability occurs at 2 loci, even though the species he studies have genotypes which contain many thousands of loci.

Important simplifications in these problems arise if certain kinds of interactions between genes can be ignored. These interactions arise from a variety of factors, all well-known to geneticists, including assortative mating, abnormal segregation ratios or gametic selection, a variety of types of inbreeding, sex-linkage, linkage, population structure which prevents random mating within the population, epistatic interaction of genes at different loci within a genotype, or of genotypes within a family or between families within a group. Since, I will argue, it is these interactions which are responsible for the existence of higher-level biological units of selection, we ignore the interactions at our peril when debating the existence of these higher-level units. But ignore them we must, it would seem, since to fail to do so is to produce models of evolutionary change which are too complex to analyze.

This problem of complexity is discussed by Lewontin (1974, pp. 281-283) and summarized in his table 56, from which Table 1 (below) (taken from Wimsatt 1980b) is derived. The dimensionality of a model is the number of independent variables it must contain to predict the consequences of selection, and is a measure of the complexity of the model. Table 1 shows how this complexity grows as a function of how the number of alleles per locus,  $a$ , or the number of loci in the system,  $n$ , increase under different simplifying assumptions which ignore different of the known interactions among genes. It can be seen that only under the strongest possible simplifying assumptions, assumptions which ignore all statistical associations or interactions of genes not at the same locus, does the problem remain of manageable complexity if more than a very small number of loci are considered.

TABLE 1  
Sufficient dimensionality required for the prediction of evolution of a single locus with  $a$  alleles where there are  $n$  segregating loci in the system

Level of Description:	Zygotic Classes	Gametic Classes	Allele Frequencies	Allele Frequencies
Dimensionality:	$\frac{a^n(a^{n+1})}{2} - 1$	$a^n - 1$	$n(a-1)$	$(a-1)$
Assumptions:	none	1	1, 2	1, 2, 3
$n$ :	$a$ :			
2	2	9	3	2
3	2	35	7	3
3	3	377	26	6
5	2	527	31	5
10	2	524799	1023	10
32	2	$9.22 \times 10^8$	$4.29 \times 10^9$	32

*Assumptions:*

- (1) random union of gametes (no sex linkage, no assortative mating)
- (2) random statistical association of genes at different loci (linkage equilibrium).
- (3) no epistatic interaction (inter-locus effects are totally additive).

(Table is adapted and extended from Table 56 of Lewontin 1974, p. 283.)

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This exponential growth in complexity occurs basically because if these interactions between genes cannot be ignored, we cannot use the frequencies of individual genes as variables in our models, but must use as variables the frequencies of the various higher level units within which the interactions occur. These higher-level units are combinations of basic genetic units, and the number of such possible units grows geometrically with the number of basic units, much as the number of words of length  $n$  constructable from an alphabet of  $a$  letters increases as a function of  $a$  and  $n$ . If the possible gametes are possible words, then the possible zygotes are the possible pairs of words. Lewontin's table does not even consider the still greater complexities arising if larger assemblages, the possible sentences and paragraphs of family and group structure, must be considered!

Thus our simple models appear to be forced upon us by the impossibility of dealing with the combinatorial explosion in number of variables and the complexity that results if we look at any but the smallest systems without ignoring all but the simplest interactions. (See Wimsatt 1979, for a more general discussion, and 1980a, 1980b, and 1981 for fuller development of this and related issues.) If we are faced with this apparently impenetrable thicket of ramifying complexities, Williams' promise that this baroque hierarchy of interactions can be decomposed into a set of algebraic selection coefficients, each operating on one gene at a time, is sufficient explanation why the "gene's eye view of evolution" has become the dominant perspective in a new and popular school of artistic representation. The question is —does it work?

3. The limitations of Genetic Bookkeeping: the failure of Williams' "mathematical decomposition" of selective forces to provide a theoretically or predictively adequate account of evolution

Williams claims (in the quote which began the preceding section) that all of the complexities of higher levels of interaction can be attributed to selection coefficients acting at the level of individual genes. Once the selection coefficients for the genes at a given locus are determined (by estimation from empirically observed changes in gene frequency), this procedure can be "iterated over all loci" (Williams 1966, p. 57).<sup>6</sup>

It is important to see that this is an iteration of an estimation procedure, and not a theoretically based claim about what will happen at all loci. But suppose for the moment that it is taken as a theoretical claim about the independence of selection acting at each locus.

A claim that evolutionary processes can be analyzed in the manner Williams suggests, as being of the lowest possible dimensionality, involves at least the claim that a deterministic theory of the change of gene frequencies at a given locus can be constructed using only the frequencies of the alternative alleles of that locus. In the simplest case of two alleles at one locus, this involved saying that it is a function only of the frequency of a single gene since if  $q$  is the frequency of gene a, then  $1-q$  must be the frequency of the other gene, A because there are no other genes at that locus. (It is a function also of the fitnesses,  $W_{11}$ ,  $W_{12}$  and  $W_{22}$  of the genotypes AA, Aa, and aa, but these are assumed to be constant parameters of the system in this discussion, though this is itself a common and serious, oversimplification.)

Consider Figure 1 as a graph of gene frequency from different initial points (.05 for the bottom curve, .95 for the top curve) as it changes in successive generations. If this were the graph of an actual case (Lewontin describes it as of a "hypothetical laboratory population") it would falsify Williams' claim. Why? Consider the topmost curve. At all points between the initial high value of gene frequency of .95 and the minimum value (of about .7, reached in generation 4) a population which is decreasing in gene frequency at that value (between generations 0 and 4) is later increasing in gene frequency at that value (in generations 5 and later). But if gene frequency can either increase or decrease from a given value, then gene frequency (of that gene or its allele) alone is not an adequate basis for a deterministic theory of evolutionary change.<sup>7</sup>

Williams gets into trouble at this point because his claim is neither a theory of evolution in terms of gene frequencies, nor even a schematic description of the form of such a theory. His statement that ". . . it must always be true that a given gene substitution will have an arithmetic mean effect on fitness in any population" (1966, p. 56) suggests the following procedure for evaluating this effect on fitness. Imagine a gigantic (non-interventive) DNA sequencer that, given a population, will determine all of the genes in that population and their

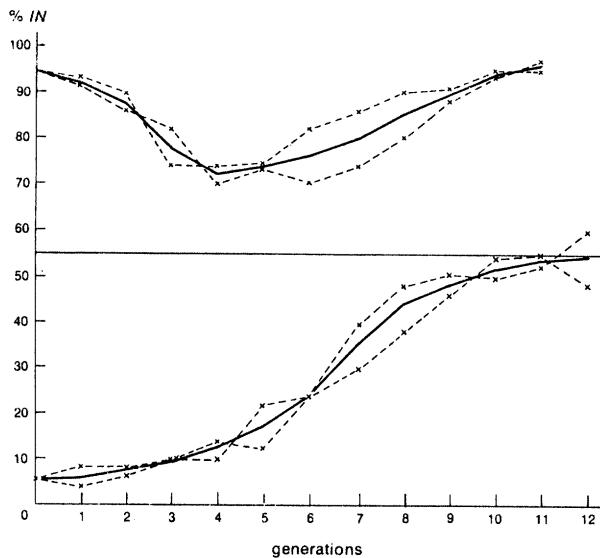


Fig. 1. The frequency of an inversion, IN, in hypothetical laboratory populations. The heavy lines represent the average behavior of replicates, while the X's represent individual data points. (Reprinted from Lewontin 1974, Figure 23, p. 274, with permission of Columbia University Press.)

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frequencies. Perform this genetic census at two points in time—or perhaps in each generation. For each gene, its frequency in the interval will either increase (in which case it is being selected for), decrease (it is selected against), or remain constant (it is neutral).

This data can then be used in one of two ways. It all can be used to describe the evolutionary trajectory of the population in its phase space. But then this is not a theory of evolutionary change but a description. The fitnesses,  $W_{11}$ ,  $W_{12}$ , and  $W_{22}$  inferred from this are merely biological redescriptions of what is happening in successive generations and may undergo arbitrary changes as the 'curve-fitting' parameters that they are. Or the changes observed in one generation may be used to estimate fitness values which are then used to predict future changes. This is more of a process of trend extrapolation using an assumed model rather than a theory itself, but it is at least not totally tautological.<sup>8</sup> To have a predictive tool or theory then, Williams must intend his remarks to describe a process of trend extrapolation.

But here is where the trouble arises. The graph of Figure 1 indi-

cates that local estimates of fitness values cannot be used in this way to extrapolate evolutionary trends. After all, gene *a* is apparently being selected against in generations 0-4, but subsequently it must be being selected for, as its frequency is then increasing. To put it more generally, local estimates of fitness or selective value are not valid globally, for other values of the frequency of that and other genes. Williams, in effect admits as much when he says that: "One allele can always be regarded as having a certain selection coefficient relative to another at the same locus at any given point in time."(p. 57, underlining added).

The reason why selection coefficients are only locally valid becomes apparent in Figure 2. Indeed, Lewontin's hypothetical laboratory popu-

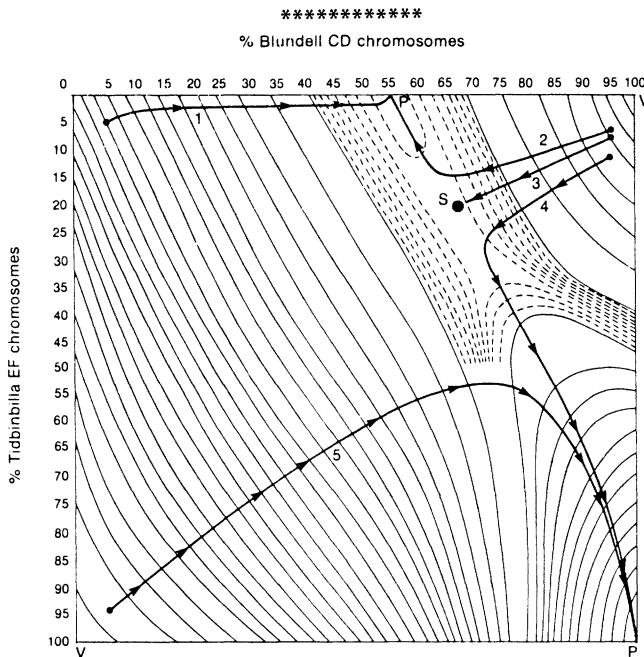


Fig. 2. Projected changes in the frequency of two polymorphic inversion systems in *Moraba scurra* from different initial compositions, based on fitness estimates from nature. The trajectories, shown by arrow-marked lines, are calculated by the solution of differential equations of gene frequency change. Lines crossing the trajectories are contours of equal mean population fitness, *W*. (Reprinted from Lewontin 1974, Figure 24, p. 280, with permission of Columbia University Press.)

lation of Figure 1 was not so hypothetical after all, but a description of changes that would be expected in a field population of the grasshopper, Moraba scurra, whose mean Darwinian fitness,  $\bar{W}$ , is estimated from the fitnesses of the 9 possible genotypes in natural circumstances, and is given as a function of the frequency of two alleles at each of two loci in the adaptive topography of Figure 2. An adaptive topography is a plot of contours of equal mean population fitness,  $\bar{W}$ , as a function of the gene frequencies (in this case, at two loci). Since in many simpler models (particularly where the genotypic fitnesses are constant) a population will tend to evolve in directions of increasing  $\bar{W}$ , the adaptive topography gives a visual means of making qualitative predictions about the direction and relative rates of local evolutionary change.

Indeed, the lower curve of Figure 1 is just trajectory 1 of Figure 2, and the upper (problematic) curve of Figure 1 is trajectory 4 of Figure 2. And the results that appeared as indeterministic in terms of the trajectory of gene frequencies of a single locus are seen to be deterministic once the frequencies at each of two loci are specified. Thus, the initial points of trajectories 1 and 4 should not have been specified as .05 and .95, the frequency of a gene at the first locus, as is implicit in Figure 1, but as (.05, .05) and as (.95, .12), the frequencies of the genes at both loci as in Figure 2. Note also that trajectory 2, with initial point (.95, .07)—in which the frequency at the first locus is the same as for trajectory 4, but that at the second locus is different—also shows a violation of the deterministic assumption if only the first locus frequency is looked at when the two trajectories are compared. Trajectory 2 is not by itself evidence of a violation, as is trajectory 4, however.

It is quite clear from this adaptive topography that what will happen in evolution is a function of the joint values of gene frequency at two loci, and no set of measurements or extrapolations looking at frequencies of just one locus at a time can provide an adequate basis for prediction. This is true in this case because of epistatic interactions between loci, which is a sufficient condition for having to go to a phase space of greater dimensionality for prediction.

Williams's proposal, then, fails in this case, in 3 ways:

- (1) It does not result in a deterministic theory of evolutionary change in terms of the gene frequencies of individual loci which can be "iterated over all loci" to produce a global solution.
- (2) It fails to do so because epistatic interactions among loci prevent local estimates of fitness at single loci from being predictable or extrapolatable if gene frequencies at other loci are free to change simultaneously. (The appearance of predictability usually arises when estimates are only done locally under conditions in which there is no change or no significant change at other loci. But this cannot be assumed in general.) Williams in effect errs by assuming that single locus fitnesses are independent of context, when in fact they are functions of

the context of other loci. Illegitimate assumptions of context-independence are a frequent error in reductionistic analyses. See Wimsatt 1976b p. 688 and 1980b, pp. 230-235 for further discussion.

- (3) In fact, Lewontin's data on Moraba scurra represents not gene frequency changes at single loci, but the frequencies of chromosomal inversions involving many loci. For reasons which I will not detail here, inversions can often act as units of selection, and Lewontin has devoted much of the earlier portions of his book to arguing that Williams' aim of measuring the fitness effects of single gene substitutions is bedevilled with a host of practical and theoretical problems. So Lewontin's one chromosome example of Figure 1 and the two chromosome counter-example of Figure 2 are already at a higher level of organization than that supposed by Williams' single locus genetic reductionism.

What goes for two loci or chromosomes, goes as well for many. In this light, Williams' remarks suggesting genetic reductionism are better seen as having more import as a kind of genetic bookkeeping than as promising a reductionistic theory of evolutionary change in terms of gene frequencies. The latter is a tempting mirage which vanishes upon closer inspection of the complexities of the actual theory.

The presentation of a 2-dimensional adaptive topography in Figure 2 not only clarifies why Williams' argument goes wrong at this point. It also allows one to see immediately that another assumption which is made almost universally in the arguments between genic or individual and group selectionists is generally false. This is the assumption that individual and group selection will generally be opposed, that they will operate to change gene frequencies in opposite directions (see Wade 1978, p. 102 and, e.g., Williams 1966, p. 115).

This assumption almost certainly has its origin not in any arguments about what would be true in nature, but in the joint action of a consideration of testability and a simplifying assumption. The consideration of testability is that, because of the complexity of interaction of fitness components and the difficulty of determining the relevant parameter values (see Lewontin 1974), it would be helpful in determining the efficacy of group selection if we could find a trait whose presence clearly signalled the operation of group selection, because it would be selected against at the individual level and thus could only owe its presence to group selection. This does not mean that group selection would usually or generally tend to be opposed to individual selection in nature. Nonetheless, it was probably responsible for the concentration of analytical models on circumstances designed to investigate this condition. In the context of models of two alleles at one locus, the natural way to implement this condition is to assume that the effect of selection at one level was to increase a given gene frequency and that of selection at the other level was to decrease it. In the context of such simplified models, the move from traits to genes or genotypes is easy—all too easy as recent sociobiology has shown—but this way is

fraught with error, as a longer discussion on another occasion will show.

It is worth noting only how implausible the assumption that individual and group selection are opposed becomes once multi-locus models such as that of Figure 2 are considered. The effects of selection may generally be described as a vector in which each component is the change in one of the state variables (e.g., frequencies of genes, gametic, or zygotic genotypes) describing the population. Only in a phase space of one dimension, such as that characterizing the model of two alleles at one locus, are change vectors constrained to lie in the same or in opposite directions. In spaces of any higher dimensionality, the probability that they will be identically or oppositely directed is of measure zero, and the resultant of the two vectors may similarly lie in any direction whatsoever. Any residual plausibility of this assumption is clearly an artifact of being guided only by the simplest possible model of evolutionary change.

But then if group selection no longer has to overcome forces of individual selection to which it must be opposed, it matters little what or how strong are the selective forces acting at the individual level in evaluating the possibility that group selection can be efficacious. This is particularly detrimental to many of Williams' (1966) arguments. In a multi-dimensional space, even relatively weak selective forces at the group level, when added to relatively strong forces at the individual level, can change the resultant selection vector sufficiently to cause evolution towards an alternative adaptive peak than that which might be achieved by individual selection acting alone.

Thus suppose Figure 2 is an adaptive topography determined solely by selection forces at the individual or genic levels. The addition of relatively weak selection forces at the group level would not modify the overall character of the topography much, but will be noticeable in moving "geographical" features which are nearly flat—i.e., where the change in fitness is small. Thus relatively weak selection against Tinbinbillia EF chromosomes at the group level ("tilting" the whole landscape slightly) could move the location of the "saddle point" a substantial distance "down the ridge", and could result in trajectory 4 going to the maximum at the top, rather than to one at the bottom. Indeed, a related point was urged against Lewontin and White in subsequent discussion of this case (see Wright 1978, pp. 127-145, for details). The richer dynamics and greater dimensions of a multi-dimensional phase space produces the possibility of a wide variety of interactions among selection forces at different levels, almost none of which are likely to be collinear, and the assumption that higher and lower-level selection forces will be opposed is untenable in general.

#### 4. Conditions necessary for the adequacy of Williams' account and the improbability of their being fulfilled in nature

The existence of a geometric tool, the adaptive topography, for the qualitative prediction of the outcome of selection simplifies the

presentation of conditions under which it would be possible to analyze selection processes one locus at a time as Williams suggests.

If one looks at the adaptive topography of Figure 2, the reason why the frequency of Blundell CD is not sufficient to predict its future changes is that the slope of the surface for a given value of the frequency of BD is different at different values of the frequency of EF. Since the direction of change is a product of this slope, a change in EF frequency produces a change in the selection coefficient for BD.

What condition is necessary to rule out this dependence? A sufficient condition would be that the fitness surface is a plane, since a plane has the same slope at all points in it. Such a situation is depicted in Figure 3. While motion in this plane produces changes in

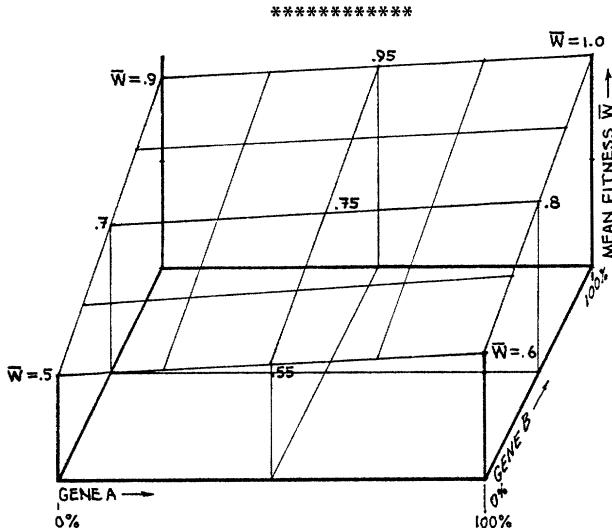


Fig. 3: A fitness surface or adaptive topography in which mean population fitness,  $\bar{W}$ , is totally linear or additive function of the frequencies of genes A and B. In this case, the fitness contributions or selection coefficients of genes A and B are totally independent of their genetic context.

mean population fitnesses, it does so in a particularly constrained fashion. A given increase in frequency of gene A confers the same increase in fitness for any value of the frequency of gene B, and conversely. Thus a change in  $f(A)$  from 0 to .5 produces a change in  $\bar{W}$  from .5 to .55 if  $f(B) = 0$  and from .7 to .75 if  $f(B) = .5$ . In each case the increment in  $\bar{W}$  is .05. Also, the same size increase in the frequency of gene A produces the same increase in  $\bar{W}$ , whatever its

initial frequency, and similarly for gene B. Thus, if  $f(A) = 0$ , a change in  $f(B)$  from 0 to .5 produces a change in  $\bar{W}$  from .5 to .7 and a change in  $f(B)$  from .5 to 1.0 produces a change in  $\bar{W}$  from .7 to .9, in both cases an increment in  $\bar{W}$  of .2. This is a particularly strong constraint on changes in fitness, in which total fitness is a totally additive function of the component fitnesses of the various genes. I will have more to say about this case later in this and in the next section.

While this is a sufficient condition for the decomposeability of the problem which Williams supposes, it is in fact stronger than we need. A necessary and sufficient condition is that the population fitness  $\bar{W}$  be a linear (or additive) function of the frequencies of genes or units at all other loci than the one under analysis, or, more generally, that  $\bar{W}$  depends non-linearly on at most one variable.

This case is diagrammed in Figure 4, where gene A shows heterozygote superiority in combination with gene S, and the population fitness

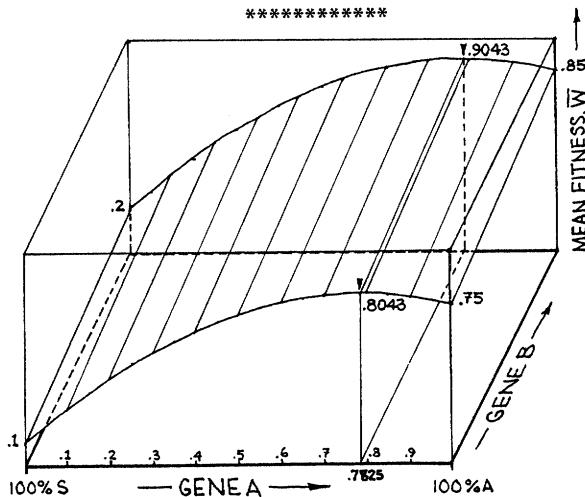


Fig. 4: A fitness surface in which  $\bar{W}$  is a non-linear function of gene frequency at the A-S locus, but a linear function of gene frequency at the B-b locus. In this case one can still analyze selection independently at each locus. A context-independent selection coefficient can be assigned to the B gene, but to get context-independent selection coefficients at A-S locus, one must rise to the level of the AA, AS, and SS genotypes, as the selection coefficients of A or S are now functions of their frequencies.

also changes with the frequency of gene B (allelic to gene b) but it changes so that the fitness differences between genotypes AA, AS, and

SS are invariant for any frequency of B.  $\bar{W}$  is thus a non-linear function of changes at the A-S locus, but a linear function of changes at the B-b locus. The fitnesses of AA, AS, and SS for  $f(B) = 0$  are .75, 1.00 and .10, approximate values for the fitnesses of the genotypes for homozygous "wild type" ( $Hb_A Hb_A$ ), sickle-cell heterozygotes ( $Hb_S Hb_A$ ), and sickle-cell homozygotes ( $Hb_S Hb_S$ ) given by Allison (1956). Under these conditions, a maximum value of  $\bar{W}$  of .8043 is found at a  $f(A)$  of .7825. I have supposed an imaginary gene B at another locus (not found in human populations) which confers an additive advantage of .05 in the heterozygote and .10 in the homozygote. Such a gene would obviously be selected for, and at the same rate, whatever the frequency of genes A and S. This is predicted in the imaginary adaptive topography for genes A and B of Figure 4, since each of the fitness lines for constant  $f(A)$  for different values of  $f(B)$  is parallel to each of the others. Thus the change from one value of  $f(A)$  to another does not affect the selection coefficient at the B locus. Similarly, a change in  $f(B)$  does not affect selection at the A-S locus, since all of the A-S curves for different values of  $f(B)$  are parallel, in this case parallel parabolas, rather than parallel straight lines! So the selection coefficients of AA, AS and SS are also constant, and independent of  $f(B)$ . As can be seen in Figure 4, the value of  $f(A)$  for which  $\bar{W}$  is a maximum is constant at .7825, no matter what the value of  $f(B)$ . The value of  $\bar{W}$  at this point changes, of course, according to the equation  $\bar{W}_{max}(B) = .8043 + .1 f(B)$ . In this case selection will proceed independently at each locus, and all populations in which all 4 alleles are present in frequencies greater than 0 will proceed (ignoring sampling error effects of finite population size) along deterministic trajectories to the equilibrium value at which the B allele is fixed at a frequency of 100%, the A allele is at a frequency of .7825 and  $\bar{W}$  has attained its maximum value for the fitness surface of .9043.

The relevant empirical question then becomes: How frequently would the conditions required for the truth of Williams' "decomposeability" claim be found in nature? A population whose members differ at all in fitness will have a computable variance in fitness if the fitnesses of the various genotypes and the frequencies of the various genes are known.<sup>8</sup> This variance in fitness may be partitioned into additive and non-additive components via a mathematical procedure which, given the n-dimensional array of fitnesses of all possible genotypes, and the frequencies of all alleles at the various loci, finds the best least-mean-squares fit n-dimensional hyperplane to the fitnesses of the genotypes, weighted by their frequencies. The slope of this hyperplane (basically the solution to an n-dimensional linear regression problem) is the additive component of variance, and deviations from this hyperplane contribute to the non-additive component. Since the expected frequencies of the genotypes changes as a function of the gene frequencies, the slope of the hyperplane will, in general, change from one point to another in the hyperspace, resulting in changes in the selection coefficients of individual genes, unless the variance in fitness is totally additive. In that special case, all genotypic fitness values lie on the regression plane which is then also identical with the

adaptive topography, and all genic selection coefficients are constants. For a fitness surface like that of Figure 3 which is a hyperplane, there is no non-additive variance in fitness. And in general, the larger the non-additive component of fitness variance, the less likely it is that the conditions for Williams' "decomposeability" picture will be true, since all of the non-additive variance would have to be in a single component.<sup>9</sup> How likely is it that most variance in fitness will be additive? This is a complex question which I can only begin to touch on here, but a number of considerations bear on it:<sup>10</sup>

(1) Even if a trait when it first appears shows a substantial component of additive variance in fitness, since selection tends to operate upon and "use up" this additive variance (see the discussion of "Fisher's fundamental theorem" in the next section), one would expect that after a time, additive variance would be greatly reduced or absent. Indeed, a population at equilibrium should have no remaining additive variance (ignoring stochastic effects of finite population size) (see also Lewontin 1970, p. 1, and Crow and Kimura 1970, pp. 236-239).

(2) However, since changes in gene frequency from a very small to a larger value at the loci under consideration or at other loci may make genotypes heretofore unrepresented in the population appear in appreciable frequencies, the above observation is not enough: these genotypes may contribute new variance in fitness which may be either additive or non-additive. With new additive variance, of course, consideration (1) applies again, but with the complication that the pool of additive variance cannot be regarded as a stable quantity, even if the external environment is unchanging. (This consideration also serves to partially undercut the widespread assumption that most populations are, most of the time, at or near equilibrium.)

(3) One must distinguish between additive variance in a phenotypic trait and additive variance in fitness. Many of the traits, such as size, studied by quantitative geneticists show additive variance but since there is presumably an intermediate optimum height, stabilizing selection would produce a distribution of heights centered on that optimum and at equilibrium there would be no additive variance in fitness.

(4) Population biologists often build separate models for various components of fitness.<sup>11</sup> Crow points out that substantial additivity in each of these components (e.g., viability, egg laying, fertility, predator avoidance, etc.) is consistent with there being no additive variance in total fitness when these components are all combined appropriately (letter dated December 26, 1979). In a fascinating paper to which Crow refers, D. S. Falconer (1977) considers a hypothetical "but realistic" model for the selection of an intermediate optimum size in mice. In this he assumes that number of offspring per litter increases linearly with size, but ability to escape predation decreases linearly with size. Variance in fitness is totally additive for each component, but since in the model, the total fitness is the product of

the two component fitnesses, the intermediate phenotype has the highest fitness and total fitness shows a substantial non-additive "epistatic" component.

This model is particularly suggestive, since multiplication of components of fitness to get total fitness is a very widely used assumption. Thus, since the probability of surviving to age X is frequently analyzed (particularly in demographic models) as the product of the probabilities of surviving through each of the stages from birth up to age X, additive components at these various stages would tend to produce increasing non-linearities in the function specifying total fitness, with, as a result, increasing components of non-additive variance in fitness. Levins' "coarse-grained adaptive function" for fitness in a changing environment (1966; 1968, pp. 17-18) is also a multiplicative model, in this case operating over successive generations. While multiplicative fitness models approach additivity (a) in the limit as fitness differences in each component get very small; (b) if the differences are all in the same direction (see Maynard Smith (1978), chapter 5), neither of these assumptions would be justified in general.

This particular model and its generalization suggest a particularly troublesome potential source of error in trying to determine whether additivity should be widely found in nature. Most models in population biology involve one or at most a small number of components of fitness, both because of our incomplete knowledge of the system and because of the analytical intractability of the more complex models. Extrapolations from models involving one or a few components of fitness could thus lead to a systematic overestimation of the additive component and a systematic underestimation of non-additive components of variance in fitness.

(5) A determination of the power and probable effects of this bias require an answer to a theoretical problem which to my knowledge has not been addressed: under what conditions can non-additive components of fitness combine to produce additivity in higher-level components of fitness or in total fitness? It seems unlikely to me that a general analysis of this problem can be given: fitness functions can be non-additive in a variety of ways, and can be combined in a variety of ways. Until we have some sort of answer, we will be unable to determine whether the additivity assumptions might not be justified after all. Three further considerations do however bear on the question of how likely we are to find additivity in total fitness: two against, and one for:

(6) In general, additivity is a very strong constraint. If we consider the space of possible fitness functions, however this is defined, it would seem likely that a vanishingly small proportion of these functions would be linear, and thus additive. A similar point applies to the combination of functions for components of fitness to yield a function for total fitness. If linear or nearly linear functions represent a very small proportion of the number of possible functions, then

arbitrary combinations of linear components (unless highly constrained, e.g., to combination by addition) would be far more likely to produce non-linearities than arbitrary combinations of non-linear components would be to produce linearities. A priori arguments would thus seem to make additivity an unlikely assumption.

But a priori arguments of this sort ignore constraints that usually apply in the real world. Thus there is no reason, apart from our ignorance, to assume that functions describing the dependence of components of fitness or total fitness on the presence and frequency of various genes are randomly distributed in an abstract function space. Nor is there reason to expect that the biologically meaningful ways of combining functions for components of fitness into a function for total fitness would produce arbitrary mappings from one part of function space to another.

(7) Happily, there are strong biological reasons for suspecting that total fitness should seldom show a strong additive component:

(a) Consideration (1) applies whether or not the components of fitness are additive. Thus even if they are additive, selection operating on total fitness will tend to remove additive variance at that level. (See the discussion of the next section.) And if they are not additive, but are combined in such a way to make a component of total fitness additive, selection on total fitness will again remove this additive component.

(b) As Crow has argued (in conversation) there are a variety of biological reasons to expect negative correlations among fitness components. (It was this negative correlation between components of fitness which produced non-additivity in total fitness in Falconer's example.) As Crow puts it, if all of these components were positively correlated, we would expect selection for phenotypes high in each, and substantially reduced variability in the population. The fact that observed variability is high in various fitness components thus argues against a positive correlation among the components.

The same conclusion can be arrived at from a consideration of engineering principles of optimal design or from arguments suggested by Darwin's use of the principle of correlation of growth.

(i) A consideration of practical problems in engineering design show that one is essentially never faced with a situation in which only one design constraint need be considered and optimal design can be considered as a maximization problem subject to a single constraint (see, e.g., Darwin (1859), pp. 147-148). The usual (indeed universal) situation is of optimization subject to a variety of constraints which usually (indeed invariably) conflict, such that one cannot meet all of them simultaneously and the optimal solution is some compromise among conflicting optima. (See Levins 1966, for an application of this to model-building in population biology.) This situation will produce non-additivity in total fitness.

(ii) Darwin made substantial use of a principle (e.g., 1859, pp. 11-12, 143-150) which he called "the laws of the correlation of growth". (See Mayr's index to the facsimile reprint of the first edition.) His primary use of this principle was to argue that, because of developmental interdependencies, changes in a selected character would usually or often be correlated with changes in other characters which were not under direct selection. When used in conjunction with the optimization argument of the preceding paragraph, however, these interdependencies suggested another point. Darwin believed that large variations would usually be disadvantageous, and for this reason argued that small variations were the primary materials of evolution. One reason why this would be so is that a major change in one character without correlative changes in other functionally related characters would throw the organism "out of balance" (Darwin 1859, p. 147) in a way that would reduce total fitness, even if, could the right correlated changes be made together, total fitness would increase. The probability that the correlations between characters that do exist would be the right ones for this adaptive change (producing an adaptive macro-mutation or "hopeful monster") would be very small, and this is one reason why Darwin pays special attention to the evolution of adaptations with complex interdependent component characters. This correlated interdependence produces non-additivity.

(8) On the other side, some additive variance in fitness at each of a variety of levels must be found, or else evolution as we know it would be impossible. This is the appropriate analytical reading of Lewontin's "principle of quasi-independence" (Lewontin 1978). If there did not exist additive variance in fitness for traits (at whatever level), selection for them would be impossible. The only way in which evolution could proceed would be to wait for (vanishingly improbable) "hopeful monsters", and evolution would proceed at a rate many orders of magnitude more slowly.

Hopeful monsters are improbable because their fitness is not nearly decomposeable. (See Simon 1969, chapter 4, and Wimsatt 1980a.) Their occurrence is a radically improbable event because combinatorial interactions are important, which necessitates finding the right combination of traits all at once, rather than by an accumulation of partial solutions.

Interestingly, for a "hopeful monster" to be selectively superior to the phenotype it replaces, there must again be a context-independent component of fitness—in this case between the whole phenotypes. Indeed, the selective exploitation of new niches accompanying adaptive radiations can be viewed as a manipulation of context which increases the size of the additive component. Thus the constraints imposed by additivity arise even here.

A further fact about quasi-independence is of supreme importance. Given the above advantage it confers (by allowing evolution of the phenotype to occur at a much more rapid rate), one would expect selection for the quasi-independence of traits, particularly those whose

selective value was highly variable in space and time as a function of spatial and temporal heterogeneity in the environment. (This fact is essential to the evolution of ontogenetic plasticity and learning as an adaptive mode. See Mayr 1974, for the best discussion of this I know in the literature.) Thus the evolution of genotypes and phenotypes which have components (genes or gene complexes and traits or trait-complexes, respectively) would be expected from genotypes or phenotypes which were not nearly decomposeable in this fashion, thus turning the adaptive problem into the simpler one of finding the correct gene or combination of genes at each locus independently. This fact was implicit in Darwin's remarks (1859, e.g., pp. 33, 148). It has the following further paradoxical consequence for the reductionist however: lower-level additive variance, rather than being an intrinsic property of lower level units (like genes) is most probably generally an emergent property of higher level gene-gene and phenotype-environment interactions. Thus in a way, one can be a reductionist only because of the existence and significance of higher-level units of selection.

This point also has a bearing on the "selectionist-neutralist" debate in contemporary evolutionary theory (see Lewontin 1974) which to my mind is decisive in favor of the selectionist. Instead of selective neutrality being an intrinsic property of most mutations or allelic substitutions, as supposed by most neutralists, it is probably in most cases an extrinsic property, a product of a variety of processes of genetic and developmental canalization, regulation, adaptation (in the physiological sense), and behavioral plasticity arising in and through development and learning. The neutralists may be correct in suggesting that the vast majority of allelic substitutions are neutral (though recent developments in the analysis of gene regulation in eucaryotes are raising new doubts on this score). They are almost certainly wrong in assessing its significance however. Genetic variability, sexual recombination, and variability in phenotype-environment interactions make the evolution of quasi-independence an adaptive necessity for the phenotype. Neutral substitutions are functionally neutral, as a product of the adaptive design of the phenotype, not intrinsically so.

Finally, a variety of considerations which cannot be discussed here (including the fact that neutrality is functionally designed and thus must be selectively induced and remaining problems of genetic load discussed by Lewontin (1974)) make it appear inevitable that the relative proportion of traits showing additive variance in fitness at any one level must be relatively small. The existence of a variety of levels of selection turns out to provide a heretofore unnoticed escape from the paradoxes of genetic load which Lewontin discusses. But that is a matter for another occasion.

##### 5. Darwin's principles and the definition of a unit of selection

I have discussed how the additivity of fitness is required if Williams' picture of the decomposeability of the genome is to be

correct, and how unlikely this condition is to be met in nature, but I have not yet discussed how central the notion of additivity is to theories of evolutionary change, and to the definition and individuation of units of selection. I will do so now, and then discuss the adequacy of this definition and its relation to that offered by Elliot Sober in his paper in this symposium (1981).

Charles Darwin's argument in The Origin of Species is adumbrated<sup>12</sup> by R. C. Lewontin (1970, p. 1) as a scheme involving three essential principles:

1. Different individuals in a population have different morphologies, physiologies, and behaviors (phenotypic variation).
2. Different phenotypes have different rates of survival and reproduction in different environments (differential fitness).
3. There is a correlation between parents and offspring in the contribution of each to future generations (fitness is heritable).

Where (and while)<sup>13</sup> these three principles hold, evolutionary change will occur. Lewontin argues not only that these requirements are necessary for evolution to occur, but also that they are sufficient. They also embody what is generally regarded as Darwin's major contribution over prior evolutionists in that they specify a mechanism, natural selection, which produces this change.

Mechanism or not, these principles specify very little about the units which must meet these conditions. Indeed, they have the same kind of generality as Williams' "cybernetic" characterizations of the gene. Although they are specified in terms of phenotypes and their properties (a form appropriate to Darwin's original theory, and one to which modern evolutionists still pay lip service), Lewontin immediately applies them to genes (the units of the neo-Darwinian theory, under the impetus of Weismannism). Lewontin exploits the fact that these requirements say little about the units which must meet them, to argue that selection can operate—simultaneously and in different directions—on a variety of units (the unspecified individuals) at a number of levels of organization. In his review he discusses selection processes at the micro- and macromolecular levels, and as operating on cell organelles, cells (in the immune system, in developmental processes, and, he could have added, in cancer), gametes, individual organisms, varieties of kin groups, populations, species, and even ecological communities. These units, I will suggest, are genes in the sense of Williams' generalized definitions discussed in the first section.

These principles give necessary conditions for an entity to act as a unit of selection, as well as necessary and sufficient conditions for evolution to occur. The three conditions must all be met by the same entity, in a way that can be summarized by saying that entities of that kind must show heritable variance in fitness.<sup>14</sup>

These conditions fail to be sufficient for the entity to be a unit of selection, however, for they guarantee only that the entity in question is either a unit of selection or is composed of units of selection. A further condition, which is sufficient, is given in the following definition:

A unit of selection is any entity for which there is heritable context-independent variance in fitness among entities at that level which does not appear as heritable context-independent variance in fitness (and thus, for which the variance in fitness is context-dependent) at any lower level of organization.

Much of population genetic theory involves the notion of additive variance in fitness. It is this quantity which, in Fisher's fundamental theorem of natural selection (Fisher 1930) determines the rate of evolution. To say that variance in fitness is totally additive is to say that the fitness increase in a genotype is a linear function of the number of genes of a given type present in it and of nothing else. But this entails that the contribution to fitness of a given gene whose effect on fitness is totally additive is independent of the genetic background in which it occurs, which is to say that the variance in fitness is context-independent. Additivity is thus a special case of context-independence. It is assumed for reasons of analytical tractability, but the properties which flow from this assumption derive from its relation to context-independence.

This relation is crucial in understanding why additivity has such a central role in evolutionary theory. If the fitness contribution of a component is independent of context, then it makes the same fitness contribution in whatever context it appears. If the component is a gene, and the context is the other genes in the genotype, then the gene has the same effect on fitness no matter how genotypes are reassorted by Mendelian segregation and recombination. But this is to say that its fitness contribution is heritable under such rearrangements. Conversely, fitness contributions which are non-additive, which are context dependent will not be heritable under rearrangements of (genetic) context. Additivity is thus important because it is equivalent to the third of Darwin's principles!

This is presumably recognized by most population geneticists. It does not arise in the discussion of many of the standard models of population genetics however because it is an insight derived from another theoretical formulation, that of quantitative genetics, which was developed largely as an aid to animal breeders, and often given at best cursory attention in standard "theoretical" accounts. Roughgarden however makes this clear in several points in his excellent discussion:

We shall see that the phenotype can be viewed abstractly as being produced by the sum of two kinds of terms and that it is the first kind, called 'the additive effect of a gene', which is the part of the phenotype that is inherited, assuming underlying Mendelian segregation. (1979, p. 149).

. . . it is the additive effects of the alleles that are transmitted to the offspring. (1979, p. 152).

And relating this to the (technical) notion of heritability (which is often discussed in textbooks):

Thus the heritability, that is the regression coefficient of offspring phenotype against midparent, equals the fraction of the phenotypic variance in the population attributable to the additive effects of the alleles. (1979, p. 154).

Fisher's theorem basically says that the rate of evolution is proportional to the heritability of a trait, and that with no heritability, there is no evolution, recapitulating Darwin's insight in mathematical terms. Additivity of fitness is thus central to the structure of evolutionary theory.

One very important result follows when this assumption of total additivity holds at a given level of organization. If variance in fitness is totally additive at a given level of organization over a given range of conditions on the environment and the system, then, under those conditions there are no higher-level units of selection! This is true because fitness of any higher level unit is then a totally aggregative or mass effect of the fitnesses of the individual entities at that level of organization. With no context-dependence of fitness, the organization of these units into higher-level units does not matter. There are no epistatic interactions to tie complexes of these entities together as units of selection. The higher level unit is totally reducible in its effects to the action of various lower level units, acting in a context-independent manner.<sup>15</sup>

The assumption of additivity is one of the major reasons contributing to the plausibility of Williams' reductionistic vision. It is clear that once this assumption is made, it becomes plausible to attribute adaptation (and thus fitness) "to the effect of selection acting independently at each locus" (Williams 1966, p. 57) and leads naturally to regarding fitness as a property of genes. This is an empirical claim and represents a view shared by few population geneticists for a variety of reasons, many of which were discussed in the preceding section. Sewall Wright has systematically argued throughout his professional life and his magisterial four-volume treatise that the opposite is true, that epistatic interactions are all pervasive and important (personal conversation; see e.g., Wright 1968, Chapter 5, especially pp. 71-105). Michael Wade's current research indicates the importance of epistatic interactions at the individual level (that is, between individuals in populations) in group selection (personal conversation; see Wade and McCauley 1980, and McCauley and Wade 1980). What is clearly true is that various biases would contribute substantially to failure to detect nonadditive variance if it exists because of artificially induced constancies in or ignorance of environmental conditions capable of producing nonadditive components of variance in fitness. (See Wimsatt 1980a, pp. 230-235.)

To summarize then, if variance in fitness at a given level is totally additive, the entities of that level are composed of units of selection, and there are no higher level units of selection. If the additive variance in fitness at that level is totally analyzable as additive variance in fitness at lower levels, then the entities at that level are composed of units of selection at these lower levels, rather than being units of selection themselves. To put it in terms of Salmon's (1971) analysis of statistical explanation, the higher level units of selection as causal factors are then 'screened off' by the lower level units of selection. In their causal effects, they are then 'nothing more than' collections of the lower level entities, and any independent causal efficacy is illusory. This is a necessary and sufficient condition for the truth of Williams' genetic reductionism.

The fact that, if additive variance in fitness at a given level is totally screened off by additive variance in fitness at any lower level, we do not recognize a higher level unit of selection, but instead attribute the action of selection to lower level units has a strong methodological significance. It is a more accurate version of Williams' (1966) "principle of parsimony", that we recognize selection as acting at no higher level than necessary. Sober (e.g., 1981) may be right in arguing for a general defense of considerations of parsimony in scientific hypotheses, but the principle is infamous for the ease with which it can be given different and conflicting interpretations, and it surely has been misapplied by those who follow Williams in arguing that higher level units of selection are unnecessary. (See Wimsatt 1979, pp. 260-261.) Rather than basing the demand not to go higher levels unless it is necessary to do so on dubious interpretations of a dubious principle, "Ockham's Razor", this principle can be seen to be anchored in a deeper metaphysical commitment: that we don't count entities or effects twice! To count higher-level additive variance as indicating a higher-level unit of selection when the variance is totally accounted for at lower levels would be as silly a mistake as to measure the weight of an entity by weighing its parts, then the whole, and adding the two. And we take the lower-level additive variance as prior to that measured at the upper level, when the two are equivalent, because the system is then simply decomposable and our reductionistic bias is justified.

Given this fact, it is still worth saying (as argued in the last section) that lower-level additivity is likely often to be a product of selection acting at higher-levels of organization—that selection for "quasi-independence" (Lewontin 1978) may generate lower level units of selection because near-decomposeability is adaptive. In this case, paradoxically, the origins of lower-level units of selection would require the explanatory invocation of higher-level units of selection, even if those higher-level units may no longer be acting as units under the conditions in which substantial additivity exists at the lower level. This picture is strongly consonant with Richard Levins' views (e.g., 1973) on the evolution of hierachial organization in complex systems.

But in general, we would expect this partitioning of variance in fitness into additive and nonadditive components at different levels to show a number of levels—genes, gene complexes, chromosomes, individuals, even groups—at which additive variance at that level appears only as nonadditive variance at lower levels. There are units of selection at each level at which this occurs, and if it does, genetic reductionism and single-locus determinism are false.

In his paper for this symposium, Elliott Sober (1981) criticizes the adequacy of accounts, including mine (Wimsatt 1980a), which attempt to analyze the existence of higher-level units of selection in terms of the context sensitivity (or dependence) of the fitness contributions of lower-level components. I believe that Sober's definition of a unit of selection and my own are at least *de facto* (and indeed probably theoretically) equivalent once the units in question and the relevant aspects of context are clearly stated, but demonstrating this requires some further discussion.

1) The first point to notice is that my definition does not entail that whenever fitness contributions of components at a given level are context sensitive, there are higher-level selection processes and units of selection. Rather, the additional requirement that there is additive or context-independent variance in fitness in these higher-level units must also be met, if this fitness variance is to be heritable. Suppose that the individual genes in individual genotypes are "tied together" by context-sensitive intra-genotypic epistatic interactions such that there is no residual additive variance in fitness at the genic level, but the fitness of a group is a nearly linear or additive function of the number or proportion of individuals of a given phenotype or genotype in that group. Suppose finally that the non-linearities of the individual level represent a context-independent component of fitness at the group level. Then group selection could proceed (at a rate determined by the intensity of selection and the heritability of the group trait according to a higher-level analogue of Fisher's fundamental theorem) even though individual selection was also proceeding (using up the additive variance at the group level) and genic selection would be impossible due to the lack of additive variance at the genic level. But non-additive variance in fitness at the lower level most emphatically does not guarantee the existence of a higher level with additive variance in fitness. Thus I agree with Sober that context sensitivity at the lower level does not guarantee the existence of group (or of any higher level) selection.

2) Secondly, we must look more closely at the source and kind of context sensitivity affecting fitness. R. C. Richardson has pointed out to me (personal communication) that context sensitivity due to variation in environmental factors external to any unit of selection ought not to be treated in the same way as context sensitivity due to variation in factors internal to some higher level unit of selection. The former kind of environmental variation is treated by theories of evolution in changing environments (see, e.g., Levins 1968). While such changes bear on the heritability of fitness of whatever units

there are (usually by decreasing it), it would seem at first sight not to bear on the question as to whether there are higher-level units. If taken as reiterating the importance of individuating units of selection and then treating differently factors producing context-dependence which are internal or external to these units, I would agree. But the matter cannot be left there, for the temporal and spatial patterns of environmental change are important factors in generating higher-level units of selection. In (Wimsatt 1980a), I discuss Lewontin's claim that a response to changes of a very low temporal (or spatial) frequency cannot be adapted to at the genetic level, and argue that higher-level units of selection, because of their slower dynamics, can in principle respond to these slower changes. Levins (1968) has argued that selection proceeds for adaptations to reduce the uncertainty of the environment of the relevant unit. This reduction in uncertainty proceeds via adaptations which convert "coarse grained" environmental heterogeneity into "fine grained" environmental heterogeneity, and heterogeneity which is "coarse grained" at a lower level of organization (or to smaller systems) will often be "fine grained" at a higher level (or to larger systems). The remarkable feature of Levins' analysis is that the fitness function for "coarse grained" heterogeneity is a non-linear function of the frequency of the various environments making up the complex pattern of environmental change, whereas the fitness function for "fine-grained" heterogeneity is a linear or additive function of the frequencies of the component environments. Here is another instance of "quasi-independence" (Lewontin 1978), a case of the adaptive conversion of non-linearities at lower levels into additive components at higher levels! (See Wimsatt 1980a, for further discussion of these concepts and examples of their application.)

3) Thirdly, total fitnesses can be context-sensitive without the relevant contributions to fitness being context-sensitive. Thus, suppose a change in the environment causes an increment (or decrement) of the same size in the fitnesses of all genotypes or phenotypes. Their fitnesses are then context sensitive but this would not affect the selection of alternative units, which depends upon their differences in fitness. This is exactly the case represented in figure 4, where the gene frequency at the B-b locus had an additive effect which did not affect selection at the A-S locus. If for the B gene we substituted an environmental factor which conferred an additive fitness advantage which affected all genotypes alike a similar conclusion would hold. And if the environmental factor were itself subject to selection (e.g., via selective choice by the organisms of what environments they would occupy), the B-environment or genes making its presence more likely by affecting the organisms' choice of environments would be selected for, independently of what was happening at the A-S locus, just as the B-gene would.

Conversely, if an environmental factor which could be present in different frequencies has a different effect on different genotypes, variance in fitness which is additive in its absence may become non-additive in its presence. Thus changes in the environment of a system may result in changes in how (or whether) it decomposes into units of selection!

It is now possible to turn to an analysis of Sober's criticisms of this approach. Sober (1981) argues that group selection can be defined in a variety of ways, some very conservative (like Williams' approach) which would seldom if ever recognize the existence of or need for group selection, and some (like the definitions he attributes to Wright, Wade,<sup>16</sup> and myself) which he sees as being too liberal in that they would see group selection as being nearly ubiquitous, and in particular would see it in cases which he would reject as being cases of group selection.

Though he discusses several cases, I will focus on the one which he regards as most damaging to my account. This is the case of the Arrowhead (AR), Chiricahua (CH) and Standard (ST) chromosomes of *Drosophila pseudoobscura* studied by Levene, Pavlovsky and Dobzhansky (1954). They found that with AR and CH in pairwise competition, AR was fitter, but that when all 3 chromosomes were present, AR was less fit than CH! Sober then asks us to imagine 2 population cages with AR and CH in one and CH and ST in the other:

Fitness values in the two pairwise competitions would be different from what would obtain if Arrowhead, Chiricahua, and Standard were all present in a single population. But that there would be a difference in no way implies that the two pair-wise competitions involve group selection. Group selection must involve more than the idea that the fitness values of organisms is influenced by the kind of groups they are in. (Sober 1981, p. 103.)

In the footnote to this passage (note 7), pp. 117-118 he continues:

I take it that this point undermines the definition of group selection presented in Wimsatt (1980b). . . . this definition would imply that in our two pair-wise competitions . . . we do not have a case of organismic, individual, selection. The reason is that the fitnesses of organisms in this situation is context-dependent.

In this passage, as in others, Sober interprets the relevant context in a way that I had never intended, but perhaps did not make sufficiently clear. The context which is appropriate in evaluating context dependence is the context that actually applies under the conditions of selection, not other possible contexts, or even other actual contexts of other actual populations which are isolated from the one in question. Thus the fact that the fitness of CH is different when it is in competition with AR than it is when it is in competition with ST or with both AR and ST is irrelevant unless these chromosomes which affect its fitness are actually present in the population in question. And in Sober's case, they are not. This also applies to Sober's remarks elsewhere (1981, p. 103) about the difficulty of "figuring out the character of the counterfactual situation [a supposed hypothetical breeding structure of a population] we are supposed to consider." The answer is simple: in this case, no counterfactual situations are to be considered, since we want to evaluate the context-sensitivity of fitness differences to features present in the actual context. (The issue is

perhaps confused by the fact that in order to determine what aspects of environment the fitness of a given chromosome depends upon and what aspects it is invariant over, one may need to look at a variety of different mixed populations in a variety of environments. But the relevant environment to use in determining whether its fitness is context-dependent or independent in this environment is still this environment.

But perhaps Sober has doubts whether any population or set of populations involving AR, CH, and ST chromosomes could exemplify group selection. In this case, it may or may not, since his imagined situation is not sufficiently specified. It depends upon what additional things we suppose to be true. He has, for example, described a situation in which intra-populational individual selection is occurring but in which nothing has been said about the conditions necessary to evaluate whether group selection is occurring. Thus, e.g., it might be that the AR-CH and CH-ST cages are being used as sources to found new populations. In this case we need to know not only the within cage relative fitnesses of AR and CH (in the AR-CH cage) and CH and ST (in the CH-ST cage), but also their absolute fitnesses or reproductive rates, or at least their between-cage relative fitnesses, since it is these which will determine the rates at which the AR-CH and CH-ST populations are producing flies which can be used in founding new colonies—thus affecting their group reproductive rates. Once this detail is added, there may well be group selection, and unless this detail is added, there cannot be. Evolution by group selection, after all, requires heritable variation in fitness of the groups, and Sober's example, as it stands, exemplifies only group variation and has no provision for group replication, much less for evaluating group fitness, or its heritability. Thus I think that Sober's criticisms do not undercut the soundness of my proposed definition.

Indeed, I am pleased that it does not because I see no real issue between Sober and myself. I believe that our definitions are closely linked—and on some interpretations, probably equivalent. Sober's definition is as follows (1981, p. 107)

Group selection acts on a set of groups if, and only if, there is a force impinging on those groups which makes it the case that for each group, there is some property of the group which determines one component of the fitness of every member of the group.

I will not discuss Sober's notion of a force. I agree with his views and find them illuminating. I will claim only that if there are higher level units of selection (which therefore must show heritable and thus additive or context-independent variance in fitness at this level), then there will be a causal factor generating this additive variance in fitness, resulting in a selection force at that level. Furthermore the fact that this additive variance does not appear as additive variance at any lower level means that it affects every "member" of the group-complex as Sober requires—indeed equally, which is more than Sober requires!

Consider the A and S genes at the A-S locus as the "members" of the group. Through Mendelian segregation, they will participate in the AA, AS, and SS "groups", and a property (or set of properties) of the group will determine a component of their fitness. Thus a member of an AS group in an environment with frequent malaria is a member of the choice group—the selected-for group. It inhabits an organism with much less severe anemia than those inhabited by SS groups and with malaria resistance not had by AA groups, both of whose organisms had lower fitnesses than its organism.

The A and the S members of the AS group are affected equally by being in the AS group. And so are the members of the AA and SS groups treated alike—though members of the different groups fare differently. For selection at the A-S locus then, the existence of selection coefficients for the AA, AS and SS genotypes which are independent of the frequencies of genes at other loci guarantees the existence of a unit of selection at that level—of the diploid 1-locus genotypes which are responding to distinguishable selection forces. The fitness of a genotype is also directly related to the fitness in that generation of its component genes, so this group force determines a component of the fitness of members of the group.

This case thus satisfies Sober's definition. Similar discussions would, I think show that any case fitting my definition would also fit Sober's definition. Furthermore, my definition shows an important way in which there may fail to be a higher level selection force: If a context-independent component of fitness at a given level is shown to be exhaustively accounted for by an additive component at a lower level, then the putatively upper level force is merely a summative redescription of lower-level forces, and not a "new" force. Finally, if it is not so analyzable, then lower level forces cannot account for the upper-level changes, and we are justified in positing a force or forces at the higher level, whose magnitude is related to the size of the higher level component of additive variance. Indeed, some accounts of heritability in quantitative genetics even define a quantity, the "intensity" or "strength" of selection, which can be thought of as the magnitude of the selection force, and which also relates directly to the additive variance in fitness. (See Hartl 1980, pp. 260-262, and Roughgarden 1979, pp. 141-142.)

If Sober's definition (suitably generalized to apply to various levels of selection, and not only to group selection) and my definition are equivalent, as I think they are, then they are strongly complementary. Sober's definition emphasizes the autonomy of higher level selection forces and the units they operate on in a way that I agree with. (I have argued for the dynamical and ontological autonomy of higher levels of organization in Wimsatt 1976a, in a manner consistent with Sober's views.) My definition is anchored in the theoretical structure of the modern mathematical theory of evolution as well as in the classical formulation of "Darwin's principles", and both explains why units of selection should have the properties Sober posits and provides, for the first time, an operational criterion for

individuating units of selection. If it turns out to be a "liberal" criterion, then so is Sober's. The feeling that it may be "too liberal" is just an expression of the reductionistic biases we have accepted too uncritically in recent years. I have argued elsewhere (elaborating on Wade 1978) that these biases have led to incorrect conclusions regarding the efficacy of group selection (Wimsatt 1980a), and I expect that the next few years will document that these biases have had a pervasive and systematic distorting effect in evolutionary biology generally. I agree with Sober that "all the facts are not yet in," but I think that the facts and theory that we now know make it exceedingly unlikely that Williams' and Dawkins' views will prove to be an adequate account of the matter.

## 6. Hardy-Weinberg equilibrium and analogues to Mendelian segregation at higher level units of selection

I have so far not discussed Williams' third criterion for an entity being a gene. In that criterion he characterizes a gene as "that which segregates and recombines with appreciable frequency" (Williams 1966, p. 24). In this section, I will argue that various higher-level units of selection show properties which are, in the relevant theoretical sense, analogues to Mendelian segregation and recombination.

This move, if successful, has two consequences:

(1) By showing that higher-level units of selection have all of the relevant properties of genes, it helps to further undercut the reductionistic vision Williams espouses, in which only the lowest level entities—short strings of DNA or RNA—are seen as genes.

(2) The formulation and development of Mendelian genetics was essential to the rebirth of Darwin's theory as the "neo-Darwinian" or "synthetic" theory of the 20th century (see, e.g., Provine 1971). Its impact has been so strong in the reformulation of evolutionary theory that some writers (e.g., John Maynard Smith 1973, 1975) have even held that evolutionary theory only became a truly scientific and testable theory through the incorporation of Mendelian genetics. (I think that this is too strong a conclusion, but it is clear that this incorporation immensely increased the predictive power and the testability of evolutionary theory.)

But the status of the modern theory for higher-level units of selection remains curiously anomalous. Various writers (Lewontin, Sober and myself among them) have argued that "Darwin's principles" could be met at higher levels than that of the molecular gene. But to have shown this much (if it has been shown, and I think it has!) is to leave the higher level theory in a pre-Mendelian state, one comparable to that of Darwin's original theory. What is needed, presumably, is principles of inheritance analogous to the laws of Mendel and Morgan for the generation of new higher-level genotypes (for example, of groups) from existing ones which could act as a basis for doing a higher-level population genetics with the inheritance and selection of

these higher level units. Without these, it seems reasonable to be more sceptical of the higher-level theory than of the lowest-level genetic theory: a crucial hurdle for it has not yet been passed.

The only alternative to developing such a higher-level machinery<sup>17</sup> seems to be to describe higher level interactions in lowest-level Mendelian terms. Until recently, the existing models of group selection appear to have done just this (see, e.g., Wade 1978, 1979). But this move leaves the higher-level theorist open to the claim that everything that is really happening is happening at the bottom level, and we are back to Williams' picture of evolution.

The construction of new higher-level theory is just what is needed, but it seems to be an insurmountable task. The biggest problem is that it is not even clear where to start. What is the group genotype other than the collection of genotypes of its parts or members?<sup>18</sup> How does one distinguish between genotype and phenotype at this higher level? Are there group "chromosomes" with "linkage" relations within them? What would be analogous to gene expression for groups, and how is it mediated? Can group genes be recessive, dominant, epistatic, and additive or non-additive in their effects?

New theory does need to be developed (and I will have more to say about this later) but the task is not nearly as big as it seems.

This is because many of the resources already exist in the classical population genetics of multi-locus systems, mating structure, and migration, and in the genetics and ecology of population differentiation and isolation mechanisms, and spatial and temporally heterogeneous environments. Much of the theory is already in place—it just needs to be looked at in a new way. This can be seen by focussing on the concept of Mendelian segregation, its historical and theoretical importance to the modern theory of evolution, and how it can be generalized to higher-level units of selection.

In 1868, Darwin proposed his 'provisional hypothesis of pangenesis' in which large numbers of gemmules secreted by the various cells of an organism were combined in sexual reproduction in such a way that the characters of the offspring (produced by equal contributions of gemmules from both parents) were an intermediate blend of the characters of the parents. This theory drew rapid and searching criticism (as it appeared in the fourth edition of Darwin's *Origin of Species*) by Fleeming Jenkin (1867), who pointed out that with such a 'blending' mechanism of inheritance, the variation in a population would be rapidly attenuated until the population was essentially homogeneous. With no variation for selection to act upon, evolution would rapidly come to a halt. With the rise of Mendelism (Mendelian segregation prevents significant blending in the 1-locus case, and limits its effects in the multi-locus case) blending theories were rapidly forgotten, and almost without exception (one being Wallace's excellent discussion in Wallace 1968, pp. 61-65), their characteristics and consequences are forgotten, ignored, and misunderstood.

To R. A. Fisher, however, the avoidance of blending inheritance and its consequences for the loss of genetic variance was a sine qua non for the possibility of evolution. He began his ground-breaking treatise (Fisher 1930, pp. 1-4) with a discussion of the character and consequences of a blending mode of inheritance. To Fisher, Mendel had clearly made the world safe for Darwinism.

Fisher makes the consequences of blending inheritance very clear. Fisher's fundamental theorem of natural selection says that the rate at which gene frequencies change is directly proportional to the additive variance in fitness. No additive genetic variance in fitness means no gene frequency change, and no evolution. In those beginning pages, he derives (or actually, claims the derivability of) a formula for the rate of loss of variance resulting from mating under blending inheritance. In each generation, the variance is attenuated by a factor of  $.5(1 + r)$ , where  $r$  is the correlation between parental genotypes. Fisher expected this correlation to be small ( $r$  ranges between -1 and +1, and is 0 in a randomly mating population). Thus, he argued that the existing genetic variance in a population would be approximately halved in successive generations. With no production of new variation, and weak selection, evolution would go roughly as far in a given generation as it would in all successive generations, in accordance with the series  $1 + 1/2 + 1/4 + \dots$  (see Wallace 1968). Evolution then would grind rapidly to a halt with no variation to act upon.

Suppose that variance is being created anew in each generation by some mechanism or mechanisms, at a rate  $V_0$ . Then the equilibrium pool of variance (which will determine the rate of evolution) is that amount of variance for which variance is being lost at the same rate at which it is being created, which happens when:

$$(1) \quad V = [1/(1 - \alpha)] V_0$$

where  $\alpha$  is the proportion of variance being lost in each generation. In Fisher's case, if  $r = 0$ ,  $\alpha = 1/2$  and the equilibrium pool of variance is just  $2V_0$ . (See Wimsatt 1980b, pp. 241-244.)

The model Mendel originally proposed for inheritance in his peas had pairs of factors which determined phenotypic characters and only one of which was transmitted at random from each parent to make up the pair of factors in the offspring. Crucial to this model is the appearance of segregation, in which matings between organisms both of which were heterozygous for the same factors, had an expectation of reproducing the homozygous "pure" types from which (in Mendel's experiments) the heterozygotes were derived in the initial cross. Ignoring environmentally induced variation, we have it that a phenotypically homogeneous stock (of heterozygotes) becomes phenotypically heterogeneous through segregation, thus increasing the variance of the character in question.

We have here the mechanism (Mendelian segregation) by which Mendelian inheritance prevents the loss of variance characteristic of blending

inheritance systems. Assuming an indefinitely large isolated random mating population in Hardy-Weinberg equilibrium (in which genotype frequencies are given by the products of the frequencies of their component genes) and with no selection, Fisher showed that genetic variance is conserved indefinitely with Mendelian (he says 'particulate') inheritance. If the variance is conserved, then it can accumulate as it is produced to a sufficient level for evolution to proceed at a reasonable rate. (It would accumulate up to its maximum possible value if selection were not operating, but as noted earlier, selection "uses up" additive variance.)

The major reason for the difference between these models is made clear in Wallace's discussion (1968) of blending vs. Mendelian inheritance. Blending inheritance is blending because there are no smallest units of genetic information, or at least that the units are much smaller than the size of existing differences between genotypes. In this case mating (or binomial sampling if there are very small units) will produce new genotypes which are an average (or in the latter case having an expectation of and small variance around the average) of parental genotypes. As Wallace points out, because of the production of new genotypes, there can be no Hardy-Weinberg equilibrium with blending inheritance. (In the case of the units much smaller than the differences found in the population, there is a Hardy-Weinberg equilibrium involving proportions of genotypes producable by all combinations of the very much smaller units, but the initially differentiated population is very far from it, and if there are very many units, the equilibrium will involve a very "peaky" distribution around the population mean with consequently low variance. The variance is much smaller than it would be for the Hardy-Weinberg equilibrium of 2 alleles at 1 locus. In the limit as the number of factors affecting a trait grows without limit, if these factors have an additive effect (and even in any "natural" case in which they do not), the variance approaches 0.

The Hardy-Weinberg equilibrium is an equilibrium not only of gene frequencies and genotype frequencies, but also of variance. At the equilibrium values of genotype frequencies for given values of gene frequencies, heterozygotes are being produced by matings of dissimilar homozygotes at the same rate as the various homozygotes are being produced through Mendelian segregation by matings between heterozygotes.

It is an equilibrium for a very deep reason, not to my knowledge ever mentioned by population biologists (but see Fisher 1930, pp. 38-40 for a related discussion): the Hardy-Weinberg equilibrium is a maximally mixed state for indivisible genes aggregated into genotypic units. *I* *is* *an entropic maximum, and the derivation of the Hardy-Weinberg equilibrium has a direct parallel with the earliest statistical derivations of the Second Law of Thermodynamics!* Just as the second law predicts the evolution of ordered or differentiated systems into less ordered or more thoroughly mixed systems, the Hardy-Weinberg principle for 1-locus systems, *because of the particularly strong character of its mixing assumption—random mating or panmixia—predicts the attainment of the maximally mixed state in 1 generation!*

Fisher (1930) and Wallace (1968) in their discussions emphasized that the amount of variance is conserved once H-W equilibrium is attained, but neglected to mention that, for an initially maximally differentiated population, one containing no heterozygotes, variance is lost in the first generation at the maximum possible rate! Indeed, in this first generation, the results under blending and Mendelian inheritance schemes are identical: half of the variance is lost. Because of panmixia, Mendelian inheritance produces maximal blending in the first generation. With blending inheritance however, this loss continues in successive generations because there are no smallest units of genotype. With Mendelian inheritance, it stops, because what is passed to the offspring at each locus is a genetic atom—an allele. Alternatively expressed, it is Mendelian segregation which preserves variance, but it is the atomicity of the genes which makes Mendelian segregation both possible and, given the haploidy of the gametes and the randomness of segregation and of mating, inevitable.

It is worth noting that in quantitative genetics, where a trait may be an additive function of alternative alleles at many loci (with  $n$  loci, there are  $2n$  genes affecting the character and  $2n+1$  possible character states) we have a closer analogue to blending inheritance. Here the behavior of a population consisting initially of individuals having only the 2 extreme character states will continue to lose variance and produce new types until a generation,  $G$  in which  $2^G$  is greater than  $2n$ . It will behave qualitatively as if its mode of inheritance is blending until that point, where its genes demonstrate their atomicity by not producing the next generation's expected new intermediate genotypes.

The point of this long discussion is to explore analogies with Mendelian segregation in higher level units of selection. To see how to do this, one must notice only two facts:

(1) The only role of Mendelian segregation in population genetics or for evolution (as opposed to in cytology or cytological genetics) is the effect it has on the variance in a population.

(2) When looking at "open" systems in which new variance is being created, the conservation of variance no longer has the central role that it does in "closed" systems in which no new variance is being added. This role is taken over by the relation between the rates of loss of variance and of creation of new variance, since these 2 rates determine (according to equation (1)) the magnitude of the steady-state equilibrium pool of variance.

In the light of these facts, I make the following claim: In a system which is "open" with respect to the production of variance, any factor which tends to retard the rate of loss of variance to a value below the maximum possible rate of loss (and thus increasing the size of the equilibrium pool of variance) is a segregation analogue.

What kinds of things produce segregation analogues? Here too, the

analogy holds. Just as the atomicity of genetic units produces segregation at the level of the Mendelian gene, so also the existence of units at higher levels produce constraints on mixing processes which results in analogues to Mendelian segregation at these higher levels.

But how do we detect these analogues, and how do we evaluate their retardation of the rate of loss of variance below the maximum rate possible? Here the concept of Hardy-Weinberg equilibrium, already implicated above in prior discussions of segregation, comes to our rescue. The Hardy-Weinberg equilibrium is an equilibrium of variance. Systems which are initially more differentiated (having more variance than the H-W value) spontaneously decrease their variance to the H-W value, and systems which are "too homogeneous" (e.g., a population of heterozygotes) spontaneously increase their variance to the H-W value. For more differentiated populations then, the rate of approach to Hardy-Weinberg equilibrium is a rate of loss of variance, and indeed, it is linear with this rate of loss. Furthermore, the 2 allele at 1 locus case provides the benchmark for the maximum rate of loss, since in this case any variance in excess of the H-W equilibrium value is totally lost in a single generation.

So to detect segregation analogues, we merely look for cases in the existing models of population genetics in which Hardy-Weinberg equilibrium is not approached instantaneously. Where this happens, look for the factor responsible for retarding the rate of approach to H-W equilibrium and it is a segregation analogue. This is a theoretically justified operational test for the existence of segregation analogues at higher levels of organization than that of 2 alleles at 1 locus. Corresponding to this segregation analogue will be a unit of genetic organization which shows some degree of stability and has its effect by preventing or retarding the rate of random mixing.

It is worth comparing this perspective with that of Williams and Dawkins. They place an enormous weight on stability and consider as genetic units only those things with a very high degree of stability. But if one is looking at the effect of stability on variance in an open system (in which new variance is being created in each generation) any degree of stability which prevents total random mixing of an allele throughout a whole population in one reproductive period (or one generation, in organisms with non-overlapping generations) is sufficient to generate a higher-level segregation analogue.

The effects of 2 such factors are represented in figure 5. This is a modification of a standard diagram found in virtually all population genetics textbooks. It represents the rate of approach to H-W equilibrium for 2 alleles at 2 loci as a function of the degree of linkage,  $r$ , between loci. If  $r = .5$ , genes at the 2 loci are statistically unlinked in the sense that their segregation is statistically independent. As such, they will meet Mendel's law of independent assortment for different factors (or genes). This will happen if they are on different chromosomes. It will also happen if they are sufficiently far apart on the same chromosome that sufficient numbers of crossovers are

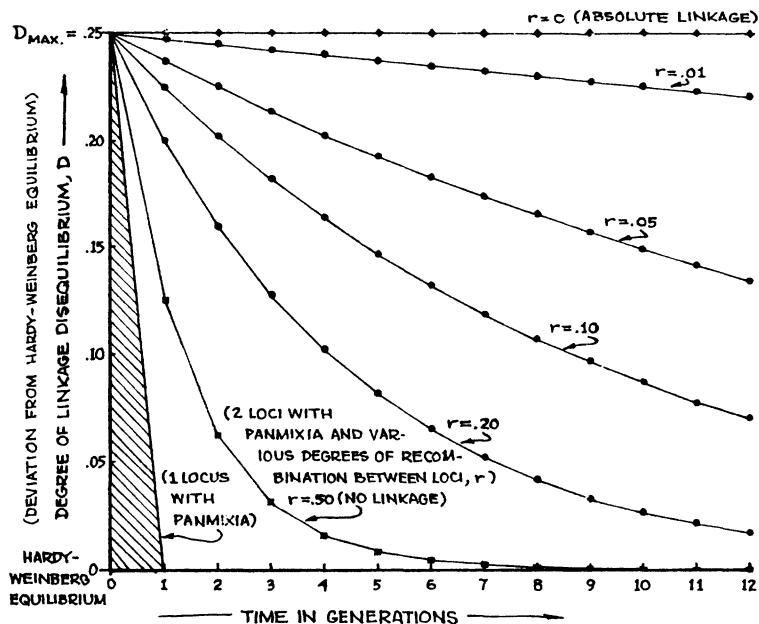


Fig. 5. The decay of linkage disequilibrium (or of the deviation from Hardy-Weinberg equilibrium) through time (measured in generations) caused by recombination between loci. The approach to H-W equilibrium of 2 alleles at 1 locus, the theoretical maximum rate (indicated by shading of the "forbidden region") is included for comparison.

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expected between them to make it a random occurrence (probability = .5) whether they will end up on the same or on opposite sides after synapsis in meiosis. Note that even in this (apparently maximally random) case H-W equilibrium is not achieved in 1 generation, but is approached by steps in a ratio given by the series  $1 + 1/2 + 1/4 + \dots$  It provides the benchmark of maximum rate of approach to H-W equilibrium for 2 alleles at each of 2 loci, but this benchmark is not as rapid as in the 2 allele at 1 locus case, indicating a segregation analogue. I will return to discuss this shortly.

The proportion of recombination between loci,  $r$ , indicates another factor—linkage. As 2 genes are taken at points closer and closer together, the probability of a recombination event occurring between them decays (ideally) to 0. As such, the stability of the chunk of chromosome between them under the effects of recombination increases. At  $r = 0$ , they might as well be the same gene, since they will never be separated. (This is an idealization, of course, since the existence of intra-cistronic recombination indicates that even the

"standard" genetic unit (probably that of Williams and Dawkins), the cistron, which produces a functional protein or protein subunit, does not meet this condition.)

Nonetheless, the presence of any degree of linkage (i.e., for  $r$  less than .5) indicates the presence of a segregation analogue. What unit does this correspond to? Obviously it is the chromosome. It is a curious unit, because we are accustomed to units having fixed and well-defined boundaries. The unit corresponding to the chromosome does not however, even though the chromosome itself does. The unit is characterized by a "coupling coefficient" (see Lewontin 1974, pp. 294-295) which is large in the immediate neighborhood of a given gene and falls off in both directions along the chromosome from that point.<sup>19</sup> The unit is thus defined by a center (at the gene under observation) and a coupling coefficient which defines a region around that point—that region in which the coupling coefficient is greater than a theoretically justified threshold value. Unlike genes, these units, although composed of genes, may overlap, so the same gene may be in more than one unit. The breakdown of well defined boundaries is characteristic of many higher level units, both of (analogous) segregation and of selection. Where theory specifies a critical value of a parameter for individuating units, the problem is partially ameliorated, but only partially. Being in the same unit is still a matter of degree, rather than a question with a simple "yes" or "no" answer. Often, however, the theory does not exist to specify a critical value of the parameter in question, and in any case the parameter will be a function of context and can be expected to fluctuate or to change systematically in time, so that the boundaries of the units would change accordingly.

This seems anomalous if we think of this unit as defined only by linkage and recombination forces, but it is not. Lewontin's "coupling coefficient" is a function both of the degree of linkage and of the amount of non-additive or epistatic variance in fitness attributable to the region in question. This raises another important point which is perhaps the major error in the vision of Williams and Dawkins: to some degree, linkage and selection forces are interconvertible in their effects on evolution! A gene complex can be highly stable in evolutionary time either by being intrinsically stable (as when 2 genes are closely linked) or by being such that any event which breaks up the complex results in phenotypes with significantly lowered fitness. Williams and Dawkins talk mostly about intrinsic stability, and consider selection as acting later to choose among intrinsically stable entities. (See Wimsatt 1980a, pp. 244-246, for further discussions.) They cannot legitimately make this separation.

Consider the case in which heterozygotes ( $Aa$ ) are lethal. An initial population composed of  $AA$  and  $aa$  homozygotes present in equal frequencies and with equal fitnesses will remain at equilibrium. (This is an idealization, of course, since the equilibrium is unstable.) Its gene frequencies, genotype frequencies and variance will remain the same through successive generations, though its intrinsic rate of increase (under random mating) will be halved, because half of the

offspring (all of the products of AA x aa matings) will die without reproducing. The AA and aa genotypes will remain stable in spite of segregation because of selection. Even more amazingly, this result is the same under either blending or Mendelian inheritance!

Similarly, suppose there is strong epistasis among genes at 2 loci such that the AAbb and aabb genotypes have high fitness, but all genotypes produced by reassortment or recombination (AaBb, AaBB, AABb, aaBB, AAbb, aaBb, and Aabb) are much lower in fitness. The evolutionary stability of the AB and ab gametes and the AABB and aabb genotypes will be much higher because of differential selection against the other gametes and genotypes than would be predicted from recombination forces alone.

This point relates to the one made in the first section and in the discussion of Table 2 in Wimsatt 1980a, pp. 244-246. It was there

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TABLE 2  
Ratio of intrinsic growth rates,  $r_b$ , of shorter-lived replicator ( $r_b$ ) to longer-lived replicator ( $r_a$ ) that shorter-lived replicator needs to offset shorter lifetime.\*\*

lifetime of replicator (generation)	Intrinsic growth rate (per generation) of a replicator with a lifetime of 1000 generations					
	1.001	1.01	1.1	2	10	100
1000	1	1	1	1	1	1
100	1.0062	1.0032	*	*	*	*
10	1.071	1.067	1.033	1.001	*	*
5	1.148	1.143	1.101	1.0062	*	*
4	1.188	1.183	1.139	1.015	*	*
3	1.259	1.254	1.205	1.040	1.0003	*
2	1.413	1.403	1.352	1.118	1.005	*
1	1.998	1.990	1.909	1.500	1.100	1.010
.9	2.158	2.149	2.062	1.611	1.141	1.017
.75	2.518	2.507	2.405	1.863	1.244	1.042
.50	3.997	3.980	3.816	2.914	1.733	1.210
.25	15.988	15.921	15.260	11.485	5.958	3.001
.10	1023.25	1018.92	976.46	728.44	345.95	133.18
.01	$1.27 \times 10^{30}$	$1.26 \times 10^{30}$	$1.21 \times 10^{30}$	$8.97 \times 10^{29}$	$4.04 \times 10^{29}$	$1.30 \times 10^{29}$

\* Added increase is less than  $1 \times 10^4$ , Fisher's rough lower limit for selective differences to be significant.

\*\* selection coefficient is  $r - 1$ .

$r$  short ( $r_b$ ) calculated from  $r_{1000}$  ( $r_a$ ) from the relationship  $r_b = \{[(r_a)^a - 1]^{b/a} + 1\}^{1/b}$  and from the approximation  $r_b = [(r_a)^b + 1]^{a/b}$  when  $(r_a)^a$  is greater than  $1 \times 10^{100}$ .

\*\*\*\*\*

argued that there is a tradeoff between stability and reproductive rate such that less stable entities having a higher reproductive rate could (and would if the changes are of the right orders of magnitude) be selected for over stabler entities having a lower reproductive rate. Fitness differences are differences in net reproductive rates, so the point made here indicating a tradeoff between epistatic or non-additive fitness differences and recombination probabilities is the same point in another form.

I wish now to return to the case of unlinked loci ( $r = .5$ ) depicted in Figure 5. What causes this retardation of approach to H-W equilibrium? It is simply a product of the fact that chromosomes do not float free in a gene pool any more than genes do. To produce a recombinant gamete (Ab or aB from AB or ab) requires the right combination of chromosomes in the genotype of the individual. Chromosomes do not recombine at random with other chromosomes in the chromosome pool. They can only recombine with their homologous chromosome in the same individual. Furthermore, not all recombinations between homologous chromosomes will produce gametic types different from the parental chromosomes. Thus, an individual with Ab and ab chromosomes can only produce Ab and ab gametes whether or not recombination occurs. There are 4 gametic types, AB, aB, Ab, and ab. Thus, in a diploid organism there are  $4 \times 4$  or 16 possible pairings. Of these pairings, only 4 can produce gametes carrying chromosomes different from the parents. This fact is responsible for the retarded rate of approach to H-W equilibrium. With 2 alleles at 1 locus, 2 of the 4 possible matings in a population containing AA and aa genotypes produce new Aa genotypes, in accord with Mendelian 1:2:1 ratios. With 2 loci, only 4 out of 16 chromosome pairings, or half as many, can produce new gametes. This is why 2 locus systems, even if the loci are unlinked, go only half way to H-W equilibrium in 1 generation. The segregation analogue exists because genes come packaged in diploid genotypes, and the unit of organization producing this effect is the individual so much maligned by Dawkins (and ambiguously, by Williams)! Two-locus systems reflect in their behavior the diploid organization of the genotype because recombination is an intra-genotypic event, and the distinction between being or not being in the same genotype becomes important.

A variety of other cases show retarded approach to Hardy-Weinberg equilibrium, and thus segregation analogues. If the gene-frequencies of a sex-linked trait are not the same in both sexes, then there is an oscillatory approach to H-W equilibrium produced by a trans-individual unit, the XX-XY system of sex-determination. In populations (like human populations) with overlapping generations, where individuals may reproduce several times, H-W equilibrium is approached slowly. Here we may assume that the offspring are produced in H-W proportions, but the parents continue as part of the breeding population. Here the unit is the genotype, but the segregation analogue is its persistence for more than one reproductive period. Assortative mating and inbreeding have similar effects, but these are better referred to a higher level of organization—that of population structure.

A variety of additional segregation analogues arise once population structure is taken into account. In a forthcoming paper (Wade and Wimsatt 1981, in preparation) Michael Wade and I have derived a formula for the rate of attenuation of variance due to processes of group inheritance in a simplified deterministic model. Here new groups are formed as collections of migrants from  $m$  parental populations. It is assumed that the effects of sampling error on the composition of both the parental populations and the offspring populations can be ignored. Where  $r_c$  is the (assumed uniform<sup>20</sup>) correlation between pairs of parental populations and  $w_i$  is the proportion of the offspring population

contributed by the  $i$ th parental population (also assumed constant over all offspring populations), then the fraction of variance lost in each group generation is given by:

$$(2) \quad \alpha = r_c + (1 - r_c) \sum_{i=1}^m w_i^2$$

This is a generalization of Fisher's formula for the case of  $m$  parents making possibly unequal contributions,  $w_i$ , and reduces to his if  $m = 2$  and  $w_1 = w_2 = 1/2$ .

In case  $r_c = 0$ , and all  $m$  parental populations make equal contributions,  $\alpha = 1/m$ . This case is interesting for two reasons:

(1) Variance is lost almost immediately—more rapidly in fact than with the classical blending theory of (individual) inheritance which Jenkins (1867) and Fisher (1930) criticize.

(2) It is in fact equivalent (neglecting sampling error) to (i) the assumption of a "migrant pool" (ii) to which all parental populations contribute equally. These assumptions are widely found in the mathematical models of group selection. (See Wade 1978. They are items 2 and 3 on his list of 5 incorrect simplifying assumptions of these models on p. 103.) It can be seen in the light of Fisher's discussion why previous writers have argued that group selection is virtually impossible. Without realizing it, they have assumed a particularly strong form of blending inheritance at the group level. (See Wimsatt 1980a, pp. 238-252, and Wade and Wimsatt 1981, for further discussion.)

Equation (2) suggests how a variety of other segregation analogues can arise. Variance is attenuated more slowly ( $\alpha$  is larger) if either there is more correlation between parents ( $r_c$  is greater) or as the contribution of parents becomes more unequal. ( $\sum w_i^2$  is a minimum when the  $w_i$ 's are equal and a maximum when everything is contributed by 1 parent.) If there are clines—systematic changes in gene frequencies throughout the species range, and the species range is significantly larger than the 1-generation migration distance,  $r_c$  will be positive, and in fact, quite large (near 1). If we assume (i) that groups are distributed randomly, but roughly uniformly in space (so that the chance of finding  $m$  groups in a given area is given by the Poisson distribution), (ii) that the 1-generation migration distance is not much larger than the mean distance between groups, and (iii) that the number of migrants declines exponentially or as some power of the distance (for geometrical reasons this power should be  $\geq 2$  and will plausibly be greater than 3) then 2 consequences follow:

(1) The number of populations,  $m$ , contributing migrants to a given population will be relatively small.

(2) Among these, the nearer populations will contribute substantially more than the farther ones.

TABLE 3: SEGREGATION ANALOGUES AT HIGHER LEVELS OF ORGANIZATION

<u>Case</u>	<u>Unit of segregation analogue:</u>	<u>Variance attenuation factor, <math>\alpha^*</math>:</u>
1) 2 (or n) alleles at 1 locus under standard H-W conditions	allele (gene) or locus**	0 (H-W equilib in 1 gen.)
2) 2 (or n) alleles at 2 loci w. no linkage ( $r = .5$ )	2 (or n) locus genotype	.5
3) 2 (or n) alleles at 2 loci w. linkage, where proportion of recombination, c is $0 \leq r < .5$	chromosome	(1-r)
4) blending inheritance (Fisher's model of Darwin's theory)	none (units are infinitely divisible)	a) .5 (but equilibrium variance = 0) b) $.5 / (1+r_c)$ , where $r_c$ is correlation between parents
5) Group inheritance (Wade-Wimsatt model)	group	$r_c + (1-r_c) \sum w_i^2$ , where $w_i$ are the proportions contributed by the i parent populations and $r_c$ is correlation between them
6) Other factors which may retard the loss of variance, and may indicate other units include sex-linkage, mating which is assortative by distance or by genotypic preference (complete or partial isolation of sub-populations), delayed mating or overlapping generations, and if the effects of selection are included; genetic selection, epistatic interaction (genotypic selection), family selection, group selection, sexual selection, frequency-dependent selection.		

\*\* ignores intra-cistrinic recombination. With recombination, this becomes a case of type 3.

\* $\alpha$  is defined as the proportion of the deviation of population variance from its equilibrium value at the maximally mixed state (at H-W equilibrium if it exists) which remains after 1 generation.

Both of these will increase  $\sum w_i^2$ , and thus  $\alpha$ .

Thus, we have 2 new segregation analogues. The first is something like the ratio of the 1-generation migration distance to the range of a species. The smaller this is, the stronger is the effect of the segregation analogue, which is manifested through increases in  $r_c$ . The second is the ratio of the 1-generation migration distance to the mean distance between groups, which also increases in its effects as it gets smaller and is manifested through changes in  $\sum w_i^2$ .

Both of these have assumed random diffusion of migrants to form new groups. A variety of behavioral isolating mechanisms (as well as genetic, ecological, or geological factors tending to increase isolation) would accentuate these effects, increasing  $r_c$  or  $\sum w_i^2$  and thus  $\alpha$  still further. These matters are discussed further in Wade and Wimsatt 1981.

Finally, just as selection can interact with linkage to increase the stability of certain genetic combinations within the individual and the "segregation effect" of higher level segregation analogues, so also selection at higher levels of organization—family selection, kin selection, group selection can enhance the effects of these higher-level segregation analogues. A variety of such factors are listed in Table 3, which summarizes the characteristics and effects of these higher-level segregation analogues, and lists a variety of other cases worth further study.

## 7. Closing Remarks on the Structure of a Multi-Level Selection Theory and Its Philosophical Implications

We have seen that it is possible to develop criteria for the existence of higher level units of selection and segregation analogues which are clear, unambiguous, and operationally usable, and which most importantly, have close theoretical ties with the structure of the mathematical theory of evolution. It is intriguing also that the units picked out by the segregation analogues are biologically meaningful units, and the same units we would expect to be picked out as units of selection. This consilience of two independent ways of picking out the units (assuming that it holds up, and more work must be done to demonstrate this theoretically) has a strong methodological significance. It shows that these units are robust, and robustness is of central importance to the testability, theoretical fruitfulness, and theoretical centrality of the entities in question. (See Wimsatt 1981.) Furthermore, robustness is widely used by scientists as a criterion for the reality of the entities in question—a matter with scientific as well as philosophical consequences for the reality of higher-level entities and properties. But to develop a higher level theory, the appropriate concepts and units are not enough. We need also laws relating these concepts and units with each other and to the causal and structural factors at the higher levels which give a dynamics for the higher-level evolutionary processes. This has yet to be done, but I wish to suggest one possible strategy for doing it which would dovetail neatly with the analysis presented here.

We have seen that additive variance in fitness can exist at a variety of levels, and that if the additive variance at a given level appears only as non-additive variance at lower levels, then there are units of selection at the level in question. Fisher's fundamental theorem has been applied at the lower level, and determines the rate of evolutionary change at that level. This rate of change is proportional to the additive variance in fitness. But there may be additive variance in fitness at several levels which does not appear as additive variance at lower levels. If so, then evolution of the traits which show additive variance in fitness at the higher level cannot proceed at the lowest level, according to Fisher's theorem. But there is no reason why one should not be able to derive analogues to Fisher's theorem for each of these higher levels, and these analogous theorems would determine the rate of evolution at these higher levels!

Some work already in print or in process already suggests this conclusion. In his (1979), Wade constructs more realistic 1 and 2-locus models of kin selection, in which the models reflect the fact that genes are in genotypes and that mating occurs by pairing of genotypes. (In Hamilton's seminal formulations (Hamilton 1964a, 1964b) kin selection is modelled for disembodied genes characterized only in terms of components of their selection coefficients corresponding to the benefits they confer on others and the cost to them of conferring these benefits and their degree of relatedness with the other genes on which they confer the benefits.) Mating pairs of genotypes can be thought of as "atomic families," and for his models, Wade is able to derive an analogue to Fisher's theorem: The rate of evolution is proportional to the between-family (additive) variance in fitness! This gives theoretical grounds for saying that the units of selection in Wade's model (and probably usually, in kin selection) is the family, rather than the gene, as is widely assumed. (See, e.g., Dawkins' (1976) discussions of kin selection.) In theoretical work now in process, Goodnight and Wade have further been able to demonstrate that, at least under some conditions, variance which is non-additive at the individual level is additive at the level of the group. I expect that further work at this and at other levels will produce further results in this direction.

What is needed to derive Fisher's theorem at the genetic level? Basically, 3 things are required (see the structure of Roughgarden's derivation in (1979), pp. 143-150):

- (1) There must be a set of basic units (genes in Fisher's case) which appear in different combinations to make up the units ("genotypes") in question, and whose frequencies determine the location of the population in its state space.
- (2) The fitnesses of these combinations must be specified. (Thus enters the phenotype, and its effects on selection.)
- (3) Laws of inheritance and "mating rules" must be specified to determine how the "genotypes" in one generation determine the types

and the proportions of the "genotypes" in the next generation. (At the genetic level, Mendelian inheritance and random mating determine (in the absence of selection) that the genotypes of the next generation will be found in Hardy-Weinberg equilibrium proportions determined by the gene frequencies.)

The satisfaction of these 3 basic structural requirements is sufficient to allow a formulation which partitions variance in fitness into additive and non-additive components. If, in addition the fitnesses of the "genotypes" are independent of their frequencies, it is possible to prove Fisher's theorem (that the rate of change of  $\bar{W}$ , the mean fitness of the population, is equal to the additive variance in fitness, and that the population will evolve to an equilibrium in which  $\bar{W}$  is a (local) maximum and there is no remaining additive variance in fitness.

If the fitnesses of units are functions of their frequencies, it should still be true that selection acts upon and uses up the additive variance in fitness. However the rate of evolution would no longer be proportional to the change in  $\bar{W}$ , and the population could no longer be expected to go to a maximum of  $\bar{W}$ , though it should still be true that at equilibrium there would be no remaining additive variance in fitness.<sup>21</sup>

There is no reason that I can see why these 3 basic structural requirements cannot be met by appropriate descriptions and specifications at higher levels. And given their satisfaction, it should be possible to perform the multiple linear regression which partitions fitness into additive and non-additive components. Similarly, the requirement that the fitnesses of the higher level entities ("genotype" analogues) be independent of their frequency should be satisfied as easily at higher levels as it is at lower levels. Indeed, it may be met more easily, because I suspect that at least some components of the frequency dependence of genic selection coefficients are products of the fact that genes are imbedded in higher-level units of selection and are thus a form of context-dependence that may be removed by going to a higher level of organization. Indeed, Michod (1981) has shown that it is a necessary consequence of the existence of group selection that the selection coefficients of genes and the fitnesses of individuals will show frequency dependence!

I hope that this speculative assessment of the situation is correct. If it is, then perhaps some writers with more mathematical facility than I will be able to derive higher-level analogues to Fisher's theorem for various levels of organization. I wonder even whether it would be possible to prove a general version of Fisher's theorem in which the 3 structural specification are given only very general specifications, applicable to classes of gene-genotype aggregation functions, phenotypic fitness functions, and rules for inheritance and mating. Such a result would produce a truly general (and generalizable) evolutionary theory. But all of this may be merely wishful thinking.

I have shown the existence of segregation analogues at higher levels of organization. But there are also analogues to many other properties of the gene, not only among higher-level biological units of selection, but for cultural units as well which I will discuss very shortly.

Given the existence of segregation analogues (which, given their development here may almost more productively be thought of as "linkage" analogues)<sup>22</sup> and the production of new units from existing ones according to rules of inheritance and mating, it follows that recombination analogues must exist as well. The existence of mutation analogues is unproblematic: changes in the things taken as "atoms" (whose combinations produce genotype analogues) are mutations. Something missing at the higher levels, however (particularly in applications of this perspective to cultural evolution), is the existence of constraints on the nature of possible mutations. This would require a canonical description of the structure of possible genes, analogous to the 4-base alphabet of DNA, and possibly the 20 amino-acid alphabet of proteins. Given this description, we could classify and place constraints on possible mutations. I assume that the prior discussions show how the concepts of heritability, additive and non-additive variance and dominance, recessiveness and epistasis can be generalized to higher levels. Wade and McCauley (1980) already speak of epistatic interactions between individuals and group phenotypes, so this process is already under way.

One distinction which demands more attention is the distinction between genotype and phenotype. I assume that for units of biological selection, this is unproblematic. Dawkins (1978) already speaks of an "extended phenotype" which is, roughly, anything in the environment of the genes manipulable by them for their selective and evolutionary advantage. This is the right idea, as far as it goes, but Dawkins' "extended phenotype" needs to be partitioned into a series of ever more encompassing adaptive structures from the gene outwards, comprising the adaptive structures of the various level units of selection.

The problem arises for accounts of cultural evolution. I am not aware of any account of cultural evolution which makes a distinction between genotype and phenotype at the cultural level. Writers on this topic have tended to regard anything that is replicated or heritable as a gene analogue. (See, e.g., Dawkins' (1976) account of "memes"—his cultural genes.) But this will not do: once Williams' argument that the phenotype is unstable and cannot be inherited is undercut (see note 2) it becomes apparent that phenotypes also replicate and are heritable. Indeed, the notion of heritability in quantitative genetics is a property of phenotypic traits or fitnesses, not a property of the gene. (See Roughgarden (1979), pp. 143-150.)

What has been ignored in these discussions is that the gene not only serves to replicate itself (its "autocatalytic function") but also serves to generate a phenotype (its "heterocatalytic function") which is differentially selected and whose success or failure determines whether genes will continue to survive to replicate themselves. Hull (1981b) has further suggested that in cultural evolution there may be

no analogue to the autocatalytic function of genes. Ideas do not replicate themselves directly, but only through the missionary activities of their phenotypic carriers, who convince others to take up these ideas and further propagate their cultural strain.

I propose that instead of focusing on the autocatalytic function of genes, we identify cultural genes through their role as producers of cultural phenotypes. A cultural entity is more gene-like in direct proportion to the degree to which it has a generative role in producing an adaptive structure whose success or failure has a role in its own replication and heritability. This generative role serves not only to identify cultural genes (the generators) but also to identify the cultural phenotypes (the generated structures which are differentially selected).

Thus, the axioms or assumptions of a theory may be thought of as generators of an interrelated set of laws, theorems, and theoretical consequences. The application of these consequences through interpretation to particular empirical situations produces predictions and tests of the theory. A variety of things relating to the success of the theory (*qua* phenotype) in the face of these tests determines its propagation or extinction, and thus determines whether future generations of teachers will teach the axioms to their students. In rough outline, this is the way to geneticize (and phenotize?) culture, not by premature and, I believe, erroneous extension of 1-locus models from population genetics to argue that cultural evolution is coded in biological genes (See, e.g., Lumsden and Wilson 1981), or even (if Hull is right) to look for cultural entities which replicate themselves directly.

Focusing on the generator-generated relation between genotype and phenotype has another nice consequence, in that it allows an intuitively pleasing account of the innate-acquired distinction. The distinction on this account is a distinction between properties of the phenotype, rather than a distinction between what is "coded in the genes" and what is not.

Two standard and intuitive criteria for calling something "innate" are as follows (see also Mayr 1974, for other criteria):

- (1) Something is innate in direct proportion to the variety of organisms in which it is found. Something found in every member of a given species is regarded as more likely to be innate than something which is not. And something found in a variety of related species is more likely to be innate than something which is not.
- (2) Something which appears early in development is regarded as more likely to be innate than something which appears later in development. (This is often given the justification that, since the organism acquires experience as it grows older, something which appears earlier is less likely to be a product of experience, or at least has a lesser contribution from experience.)

In his seminal work, Ontogeny and Phylogeny (1977), Stephen Gould discusses the relations between ontogeny and phylogeny. He argues that Haeckel's "biogenetic law" (that ontogeny recapitulates phylogeny, with earlier developmental stages of an organism being equivalent to the adult stages of its evolutionary ancestors) is false, but that a related law, that of von Baer, is true. Von Baer's (earlier!) law is that earlier developmental stages in different organisms resemble each other more strongly than later developmental stages. Von Baer glosses this law as: Differentiation (in development) proceeds from the general to the particular.

Traits which appear at earlier stages are more general in two respects:

- (1) They are found in a wider variety of organisms. As development proceeds, the first traits to appear are major phyletic distinguishing features, such as the distinction between vertebrates and invertebrates. Continuing development produces traits which mark off families, then classes, then species, then varieties, then individuals, moving down the taxonomic hierarchy in temporal order.
- (2) Development also proceeds from the general to the particular in a functional sense. Traits which appear earlier are more general in terms of their function—less specialized. Traits which appear later constitute adaptations to more and more specific features of the adaptive problems an organism will face in its ecological niche.

Note that in the first sense of generality, earlier traits will be more innate on the first criterion. But earlier traits are also more innate on the second criterion. Thus, von Baer's law asserts a law-like relation between these two criteria of innateness. The second sense of generality relates this to the ("free from experience") gloss given to the second criterion. Earlier traits are less appropriate to specific features of the experiential ecological niche of the organism. And since "fine tuning" of adaptations to specific features of the organisms' environment can sometimes only be accomplished through experience, since the more specific features of the environment are also more highly variable, and cannot be coded for in advance, the intuition that experience plays a larger role in the generation of later adaptations is a sound one. (See Mayr 1974, for further discussion.)

The interesting fact is that von Baer's law can be itself explained in terms of a very general feature of development which is a product of the generator-generated relationship. Let us assume that development begins with a set of generators which interact to produce features which are generated from them. Once a feature has been generated, it can itself act as a generator, interacting either with the primary generators or with other generated features to produce further features, and so on. The net effect of this mode of interaction is that the primary generators will have a particularly central generative role, and that features generated earlier will tend to be implicated in the

generation of more features than things which are generated later. This suggests a concept of innateness which is a matter of degree, with earlier things tending to be more innate, more involved in subsequent generations, even more "genetic" than later things.

Now consider the effect of a mutation in the primary generators which is expressed in terms of changing something which is produced at some stage in development. If the change is expressed late in development, it will tend to be less involved in the generation of other features. If it is expressed relatively early in development, however, the change in this feature may be expected to have far-reaching consequences, potentially affecting everything which that feature has a generative role in producing. The more far reaching the changes, the more likely it is that even if the feature produced is selectively superior at that stage of development, that the change will have consequences negatively affecting crucial features which it has a role in generating. A simple mathematical model of this relationship (which I will develop on another occasion) has the consequence that the probability that a mutation will be beneficial decays rapidly and exponentially as its expression occurs earlier and earlier in development.

The net effect of this is that mutations will be favorable more frequently as their effects are expressed later in development and that evolving entities will tend to be more "conservative" with respect to changes which are expressed earlier in development. But this produces the regularities of von Baer's law: Earlier developmental stages will resemble one another across wider ranges of types of organisms. And earlier things are also more general in a functional sense: they are involved in the generation of a wider array of more specific adaptations. Earlier things are more general and less subject to change because they are more "entrenched"—they are involved in the production of more later things.

The consequences of this model transfer directly to the problem of localizing the most probable and the most preferred changes in conceptual structures. Those assumptions which are more involved in the generation of the consequences of the theory and its application are more general, more entrenched, less likely to permit adaptive change, and farther reaching in the consequences of their change if a change is made. In terms of generative structure, theories and models also involve "differentiation from the general to the particular", as more specific models have more specific assumptions which delimit their range of applicability more severely and make it less likely that they will play a generative role in the production of still more specific models. For scientific theories as well as for organisms, one should attempt to change the structure at the most specific level possible, seeking changes at higher levels of generality only if lower-level changes are inadequate! While there are exceptions to this regularity, both for biological and for cultural phenotypes, it remains a regularity in the best sense, a tendency statement or Ceteris paribus law which flows directly from the relation between generator and generated.

The phenotypic approach to defining genes in terms of their heterocatalytic function promises a great deal. Aside from sidestepping problems which arise in cases where the autocatalytic function is absent because the replication of genes is not direct, it permits a natural treatment of the innate-acquired distinction and explains a very deep developmental regularity, von Baer's law, which applies to any functionally organized system, from organisms to scientific theories, in which a great deal of adaptive complexity is produced through the generative interaction of a relatively small number of elements. This analogy can be played out much further, but this is not the place to do so.

I have spent a great deal of time exploring scientific issues connected with the units of selection controversy, and the admittedly speculative (but I think, well founded) extension of existing theoretical apparatus and concepts to cover the evolution and interaction of higher-level biological and cultural units of selection. In the light of all the scientific detail discussion of these issues has required, one may reasonably ask what particularly philosophical import these conclusions might have.

In response to this, I would point out first of all that one of the roles of a philosopher of science is to act as a friendly critic of methodology and as a conceptual engineer or troubleshooter in the discipline chosen for study. As such, what has gone before is already philosophy of science, even though it may be at the same time virtually indistinguishable from some of the theoretical activities of some scientists. In this sense, notice that the philosopher has no monopoly on philosophy of science. This is as it should be.

But the philosopher of science should also be concerned with methodological and conceptual issues at a more general level than most scientists would find it productive for them to pursue, though I would hope that they would still find results at this higher level of generality to be sufficiently salient to their interests to find it interesting, even if they would not themselves pursue it. Indeed, I would urge that the perceived salience of philosophical work, even at this greater level of generality, to the activities and interests of scientists be adopted as at least one criterion (and possibly the most important one) of the adequacy of general analyses in philosophy of science.

Some of the more general points that emerge from this analysis are as follows:

- (1) Modern evolutionary biology provides one of the most current scientific paradigms of the success of a reductionistic research program. The approaches of Williams and Dawkins even look superficially a good deal like the classical picture of reduction as deductive derivability advocated by many philosophers. The inadequacy of this picture (see Wimsatt 1974, 1976a, 1976b, 1979, 1980a, 1980b, 1981) is further documented here. An alternative picture, in which adopting a reductionistic viewpoint is not inconsistent

with recognizing the reality of upper level entities (e.g., higher-level units of selection) the emergence of upper level properties (selection forces acting on higher level additive variance which does not appear as additive at any lower levels) and the dynamical autonomy of laws and processes at these higher levels (analogues to Fishers' theorem governing the rate of evolution at these higher levels) is far more adequate to the practices and conclusions warranted by a scientific reductionist. The earlier philosophical picture has misled many scientists. They need to see that on a more adequate view, they can be reductionists and emergentists too.

(2) Something that stands out clearly in this treatment is the role (and biases) adoption of a given perspective has in determining the perceived adequacy of a theoretical orientation and its metaphysical commitments, and even in determining the structure and assumptions of relatively specific lower level theorizing and model building. I have documented this more fully in my discussion of the heuristics and biases associated with adopting a reductionistic research strategy (1980b, pp. 230-235) and their effects in biasing the mathematical models for group selection (see Wade 1978, and Wimsatt 1980b, pp. 238-252). More generally, it is time that we recognize that the role and perspective of the observer must be included in the analysis of even the most "objective" sciences, since the perspective we take in approaching scientific problems leaves its mark in the structure of the models we build for the phenomena we are trying to explain. One of the most striking and productive aspects of the reductionistic vision of Williams and Dawkins is the change in perspective associated with "the gene's eye view of evolution". But as we have seen, to get a robust account of any phenomenon, we must look at it from a variety of perspectives, since only in this way can be transcend the biases associated with any given perspective. (See also Wimsatt 1981.)

(3) Another feature which stands out is the role of problems of computational complexity and the status of in principle claims in evaluating the adequacy of a theoretical position and in choosing the strategies and heuristics of problem-solving in a given area. This arises in this discussion, but is more fully treated elsewhere (Wimsatt 1979, 1980a, 1980b, 1981). Analyses of problem structure and the use (and abuse) of problem-solving heuristics in the analysis of scientific change and the products of scientific activity are coming into their own as one of the most fruitful and powerful "new waves" in philosophy of science (See Nickles 1980a, 1980b, 1981a, 1981b).

(4) Something implicit in the development in this paper (and which I expect to make more explicit in the near future) is the emerging generality of an evolutionary perspective on biological, social and even scientific activity. Under the banner of sociobiology, evolutionary imperialism has been strongly reductionistic. (See particularly Lumsden and Wilson 1981.) But the message of this paper is that it need not, and indeed, must not be so. Butler's statement

that "A hen is just an egg's way of making another egg." was intended as satire. With the rise of the gene's eye view of evolution, it was elevated to dogma. Now perhaps, we can see that there is some reason to reverse the order. DNA may well be the golden thread of molecular biology, but legend warns us that we had better take very good care of the goose. Should we do so, the future looks very bright for evolutionary studies. Physics has long been the philosophers' paradigm for the analysis of science. I believe that evolutionary biology is emerging as the next major paradigm. Not only do its complexities do more justice to the nature and processes of many sciences, but this paradigm is relevant in content as well as by example. I expect that the next decade will see a major growth in, and perhaps even a dominance of selectionist interpretations of scientific problem solving and evolutionary interpretations of scientific change. It is an exciting and a promising vision.

#### Notes:

<sup>1</sup>The debts I owe for this paper are similar to those for my earlier paper on the topic (1980b). Intellectually the viewpoints expressed here owe most to Richard Lewontin and Richard Levins, and more recently to Michael Wade. To James Crow and to Russ Lande, I owe special debts of thanks, for each prevented me (at places indicated in the text) from making major errors. Unfortunately, the errors avoided in this paper (my prior belief that most population geneticists believed that most variance in fitness is additive) is mistakenly attributed to Crow in 1980b, p. 237. I here admit my error, and thank him for his many useful comments on the topic, most of which have been incorporated in the present discussion. Others whose comments on the earlier paper were very useful to me in writing this one include James Griesemer, Jack Hirshleifer, David Hull, Marcy Lawton, Richard Michod, Bob Richardson, and Elliott Sober. Hull, Sober and I may not be in complete agreement, but the at least approximate consilience of our views (relative to those of the "reductionistic opposition") has been a productive spur to further work to reconcile our differences. Finally, the vast majority of this work was done with support from the National Science Foundation, under grant NSF-SOC78-07310. I thank them for this generous support. Frances LaDuke has again beautifully typed this paper.

<sup>2</sup>This argument is found widely in the literature, and apparently originates with Williams (1966, p. 23). It is thoroughly fallacious. Williams invites us to consider that however successful (as a biological parent or as a Greek philosopher) Socrates might have been, he could have been successful biologically only by passing on his genes. "His phenotype . . . was utterly destroyed by the hemlock, and has never since been duplicated. If the hemlock had not killed him, something else would have." (Ibid., p. 23.) But if something was killed by the hemlock, it was surely Socrates, the individual, not his type. If he had biological descendants, they surely had many of his characteristics, and in those respects, he passed on his phenotype. (He surely also has had many cultural descendants: the "Socratic method" is widely emulated and cultivated—by philosophers and others.) To

parallel the philosophers' type-token distinction, Socrates' phenotoken was killed by the hemlock, but his phenotype may well live on.

This should not be surprising, and is no different for genes. Gene-types may well be passed on, but in multi-cellular organisms, the probability of passing on a gene-token is negligible. The gene-tokens would be the relevant sections of the original DNA molecule in the zygote. But given the facts of DNA-replication, in which the molecule unwinds and each strand catalyzes the production of a copy of the other, each cell division halves the probability of finding one of the original strands in a given cell. With, say, 30 divisions in the germ line, there would then be 1 chance in  $2^{29}$  that a given germ cell would contain one of the original DNA strands—about 1 chance in 500 million!

If evolution had to depend upon the passing on of gene-tokens, it could not have happened. Genotokens and phenotokens are not inherited, but genotypes and phenotypes may be! Many of the remarks of modern evolutionists on the relative significance of genotype and phenotype for evolution are wrong as a result of the failure to make this distinction. In particular, if the phenotype can itself be inherited or passed on, it need not be regarded merely as a means of passing on its genes or genotype.

<sup>3</sup>Williams is ambivalent about whether the individual can be a unit of selection. He sometimes talks as if it is, though consistent application of his own arguments would force him to say that only genes will do, as does Dawkins, who bites the bullet and carries Williams' arguments to their logical conclusion.

<sup>4</sup>In a very interesting and useful paper, Hull (1980) argues that we must distinguish between 3 entities which are often confused in the literature on units of selection. These are the replicator, "an entity that passes on its structure directly in replication" (p. 318), the interactor, "an entity that directly interacts as a cohesive whole with its environment in such a way that replication is differential" (p. 318) and the lineage, "an entity that changes indefinitely thru time as a result of replication and interaction" (p. 327).

While these distinctions are useful for the questions Hull addresses, I have some doubts whether they will do for the problems I must address here. It is natural to say that the replicator should be taken as the gene or gene complex of the unit of selection, and the interactor as its phenotype. The genotype-phenotype distinction must be preserved for higher level units of selection, but if one talks, e.g., about group genotype, it would presumably be the genetic complement of the group. It is hard to see how this is to be "directly replicated", since its replication involves not only the replication of the genomes of all of the individuals in the migrant (reproduced) group, but also their collective migration and the manner in which they are "sampled" from the parent population. There would be less problem, I would think, with talking about the group phenotype, which would include not only the individual phenotypes of the members of the group, but also those stable structural and behavioral relations between them.

More generally, I have problems with the supposed "directness" of the replication of replicators and the appearance of a clear contrast with the "indirectness" of the replication of interactors. A DNA molecule replicates "directly" only by means of over 200 enzymes provided by the phenotype-interactor. And the phenotype replicates "indirectly" only if it is viewed as a single stage in the life cycle. While the phenotype is often incorrectly treated as a single stage (usually the adult), Bonner (1965) has argued that this is a theoretical mistake, and Williams himself (1966, p. 89) presupposes a view of the phenotype as including the whole life-cycle of the organism. But the only thing that intervenes between the adult stages of parents and that of their offspring is developmental stages of the latter. Reproduction of phenotypes is as direct as reproduction of genes, though it takes place at a higher level of organization.

While I think that Hull's distinctions mark changes in perspective in our views of reproduction, selection and evolution which it is essential to keep straight, I do not think that the distinctions between these units undercut the definition of or distinction between units of selection advanced in this paper. A detailed treatment of these issues will have to await another occasion.

<sup>5</sup>This analogy has been pursued (see, e.g., Masters 1970) and is well worth pursuing further.

<sup>6</sup>Williams actually speaks of "conclusions derived from one locus being iterated over all loci." (1966, p. 57, underlining added). This is crucially misleading, since it makes an iterated estimation procedure look like a theoretically based claim about what will happen "over all loci" which could act as a basis for explanation and prediction. There is no theoretical basis for such a claim and it cannot act as a basis for explanation or prediction.

That this is not a theoretical claim is argued further in the text. If it were, however, Williams would be open to another, more traditional criticism, for it seems that his claim that selection can always be analyzed as acting at individual loci is clearly unfalsifiable. Since any and all interactions at higher levels are described in terms of their effects on the gene frequencies at individual loci, and these frequencies must either increase, decrease, or remain the same, if this were an adequate procedure to demonstrate that higher level units of selection need not be invoked, then no possible state of affairs could demonstrate the need for individual, group, or any higher-level processes of selection!

<sup>7</sup>In his symposium talk, Sober remarked that the insufficiency of single gene frequencies to determine outcomes would not apply to stochastic versions of the single-locus theory, which Williams clearly espouses. While this may be true in some cases, it is not in the case Lewontin discusses. Lewontin's own figure 23 (or figure 1 here) represents the trajectories of different replicates as dotted lines. The solid line is the average trajectory of such replicates, and the argument can clearly be reformulated to apply to these averages.

Another point worth noting is that Williams' estimation procedure would lump all effects, including that of genetic drift, into his estimated selection coefficients. This reinforces the conclusion that Williams' proposal is just another means of "genetic bookkeeping" and not a claim about the structure of an adequate theory, as any adequate theory would distinguish between selection and drift.

<sup>8</sup> Strictly speaking, this assumes that the population is in Hardy-Weinberg and linkage equilibrium (if the fitness variance is to be computed from the genotype proportions at H-W equilibrium) or that the genotype frequencies are known independently if not. I thank Russ Lande for discussion of this case which eliminated a serious error in an earlier draft.

<sup>9</sup> The case of 2 alleles at 1 locus is discussed in Roughgarden (1979), chapter 9 (See especially pp. 145-152). A corresponding discussion for the n-locus case (without all of the explicit conclusions of Roughgarden's formulation) can be found in Kempthorne (1969). I thank Russ Lande for this reference.

<sup>10</sup> In much of the following remarks, I am drawing heavily on correspondence and conversations with Professor James Crow, who has been exceedingly helpful in correcting erroneous statements in an earlier draft.

<sup>11</sup> In this discussion of components of fitness, we are talking about the effects on fitness of different aspects of a given phenotypic trait, rather than different contributions to fitness of genes at different loci. Thus the multiplication of different additive components to give a non-additive component does not imply that additivity in fitness for genes at each of 2 loci will combine to give a non-additive fitness surface. Rather, the interaction of the components discussed will give a non-additive component in the variance in fitness for any genes affecting the trait in question. The fitness surface will be non-additive, but so will the components for one or more of the gene loci.

<sup>12</sup> These make no mention of the geometric rate of natural increase of organisms and the consequent inevitability of competition for resources (Malthus's observation). But this was a subsidiary argument employed by Darwin to establish the second principle—that different types of organisms had different fitnesses. Darwin needed this a priori argument because he had no direct observations of the occurrence of natural selection in nature.

<sup>13</sup> Lewontin applies these principles on a genetic micro-evolutionary scale, and points out that for a population in equilibrium of gene frequencies, however temporary, conditions 2, 3, or both are not met (1970, p. 1). And obviously, if there is only a single allele at a given locus in a population (violating condition 1), no change in gene frequency (or micro-evolution) is possible at that locus.

<sup>14</sup> I have analyzed these conditions and their ramifications in much greater detail in a book manuscript now in process and tentatively to be called Reductionism, Sociobiology, and the Units of Selection. Further excellent discussions of related issues can be found in Hull (1980), Sober (1981), and, less directly, Cassidy (1978).

<sup>15</sup> Mike Wade felt that this did not emphasize sufficiently strongly that whether an interaction was additive or epistatic is a function of the relation of the system to the environments in which it is studied. He feels that many studies which purport to show that variance in fitness is additive rather than epistatic suffer from looking at a restricted environment (usually in the laboratory) or range of environments, and that investigation of the system in a wide range of environments would show that many or most of the supposedly additive interactions are in fact epistatic.

It is worth pointing out that the term 'epistasis' is traditionally reserved for interactions between genes within a given genotype. But the discussion here naturally suggests an extension to interactions between higher level complexes of genes. Thus, when one speaks, as Wade does, of a group phenotype, it becomes natural to describe non-additive interactions between individuals in the group as epistatic.

The notion of additive variance has an implication that speaking of context-independent variance does not. Speaking of additive variance implies the context of a larger unit in which more than one of the smaller units which contribute to fitness will co-occur, so that their contributions will add. Thus genes which show additive variance will occur in genotypes. In the case of two alleles at one locus, this condition is met if the fitness of the heterozygote Aa is exactly half-way between the fitness of the two homozygotes, AA and aa. Talking about groups as units of selection may not imply a larger conspecific group whose fitness they contribute to, and in this case (and other similar cases), it is preferable to talk about context-independent fitnesses of groups rather than of additive contributions to fitness of groups.

<sup>16</sup> Sober's interpretation of Wade's definition is a narrow and uncharitable one, and, according to Wade, not what he had intended. Wade defines group selection as "that process of genetic change which is caused by differential proliferation or extinction of groups of organisms." (1978, p. 101). Sober claims that this fails to distinguish group selection either from individual selection or from drift. But if one takes "differential proliferation or extinction" to signal a causal tendency statement reflecting the action of causal forces, Wade's definition is equivalent to Sober's or mine and does distinguish group selection from both individual selection and drift.

<sup>17</sup> The only attempt to develop such machinery I know for biological units of selection is Lewontin's intriguing "continuous chromosome" model (1974, chapter 6). Lumsden and Wilson's (1981) attempt continues the development of lower level machinery for higher level (cultural)

phenomena. From what I understand, Cavalli-Sforza and Feldman's new book (1981) is an attempt to develop higher-level machinery for cultural evolution, but I have not yet seen it.

<sup>18</sup> Here I wish to indicate agreement with Hull (in this symposium) that in a sense, there can be no group selection, since if selection acts on groups of individual organisms in a way not reducible to selection at the organismal level, the group must in a relevant sense be acting as an individual. In this case, the individual organisms are better regarded as parts of an individual, rather than as members of a class of individuals, as Hull suggests.

<sup>19</sup> As indicated below, Lewontin's "coupling coefficient" is a function of selection forces as well as of linkage.

<sup>20</sup> This is of course an idealization. It is not even true, for sufficiently many populations, that they can all have the same correlations with each other, unless  $r$  is identically 1.

<sup>21</sup> This is suggested in the 2 allele at 1 locus case by Lewontin's (1958) treatment of frequency dependent selection, in which the genotypic fitnesses are replaced by frequency dependent "weights", but in which the form of the equation for the change in gene frequencies otherwise remains the same. But if the form of the equation remains the same, then the conditions of equilibrium should remain the same, and in particular, a regression on these weights should have a slope of 0 at equilibrium.

<sup>22</sup> The distinction between segregation and linkage is a product of the distinction between allelic genes (which are at the same locus) and non-allelic ones (which are at different loci). But the existence of loci is an organizational feature of the genotype, made significant by the fact that chromosomes come in copies (1 copy in the haploid genotype, 2 in the diploid, 3 in the triploid, etc.), and this feature may not be preserved in generalizations of segregation analogues to higher levels of organization. The stability feature which is preserved is most analogous to a linkage relation, even in the case of 2 alleles at 1 locus.

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