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ARTIFACT, CAUSE AND GENIC SELECTION*

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Several evolutionary biologists have used a parsimony argument to argue that the single gene is the unit of selection. Since all evolution by natural selection can be represented in terms of selection coefficients attaching to single genes, it is, they say, "more parsimonious" to think that all selection is selection for or against single genes. We examine the limitations of this genic point of view, and then relate our criticisms to a broader view of the role of causal concepts and the dangers of reification in science.

Introduction. Although predicting an event and saying what brought it about are different, a science may yet hope that its theories will do double duty. Ideally, the laws will provide a set of parameters which facilitate computation and pinpoint causes; later states of a system can be predicted from its earlier parameter values, where these earlier parameter values are the ones which cause the system to enter its subsequent state.

In this paper, we argue that these twin goals are not jointly attainable by some standard ideas used in evolutionary theory. The idea that natural selection is always, or for the most part, selection for and against single

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genes has been vigorously defended by George C. Williams (*Adaptation and Natural Selection*) and Richard Dawkins (*The Selfish Gene*). Although models of evolutionary processes conforming to this view of genic selection may permit computation, they often misrepresent the causes of evolution. The reason is that genic selection coefficients are *artifacts,* not causes, of population dynamics. Since the gene's eye point of view exerts such a powerful influence both within biology and in popular discussions of sociobiology, it is important to show how limited it is. Our discussion will not focus on cultural evolution or on group selection, but rather will be restricted to genetic cases of selection in a single population. The selfish gene fails to do justice to standard textbook examples of Darwinian selection.

The philosophical implications and presuppositions of our critique are various. First, it will be clear that we reject a narrowly instrumentalist interpretation of scientific theories; models of evolutionary processes must do more than correctly predict changes in gene frequencies. In addition, our arguments go contrary to certain regularity and counterfactual interpretations of the concepts of causality and force. To say that *a* caused *b* is to say more than just that any event that is relevantly similar to *a* would be followed by an event that is relevantly similar to *b* (we ignore issues concerning indeterministic causation); and to say that a system of objects is subject to certain forces is to say more than just that they will change in various ways, as long as nothing interferes. And lastly, our account of what is wrong with genic selection coefficients points to a characterization of the conditions under which a predicate will pick out a real property. Selfish genes and grue emeralds bear a remarkable similarity.

1. The "Canonical Objects" of Evolutionary Theory. The Modern Synthesis received from Mendel a workable conception of the mechanism of heredity. But as important as this contribution was, the role of Mendelian "factors" was more profound. Not only did Mendelism succeed in filling in a missing link in the three-part structure of variation, selection, and transmission; it also provided a canonical form in which *all* evolutionary processes could be characterized. Evolutionary models must describe the interactions of diverse forces and phenomena. To characterize selection, inbreeding, mutation, migration, and sampling error in a single predictive theoretical structure, it is necessary to describe their respective effects in a common currency. Change in gene frequencies is the "normal form" in which all these aspects are to be represented, and so genes might be termed the canonical objects of evolutionary theory.

Evolutionary phenomena can be distilled into a tractable mathematical

form by treating them as preeminently genetic. It by no means follows from this that the normal form characterization captures everything that is biologically significant. In particular, the computational adequacy of genetic models leaves open the question of whether they also correctly identify the causes of evolution. The canonical form of the models has encouraged many biologists to think of all natural selection as genic selection, but there has always been a tradition within the Modern Synthesis which thinks of natural selection differently and holds this gene's eye view to be fundamentally distorted.

Ernst Mayr perhaps typifies this perspective. Although it is clear that selection has an *effect* on gene frequencies, it is not so clear that natural selection is always selection for or against particular genes. Mayr has given two reasons for thinking that the idea of genic selection is wrong. One of the interesting things about his criticisms is their simplicity; they do not report any recondite facts about evolutionary processes but merely remind evolutionary theorists of what they already know (although perhaps lose sight of at times). As we will see, genic selectionists have ready replies for these criticisms.

The first elementary observation is that "natural selection favors (or discriminates against) phenotypes, not genes or genotypes" (1963, p. 184). Protective coloration and immunity from DDT are phenotypic traits. Organisms differ in their reproductive success under natural selection because of their phenotypes. If those phenotypes are heritable, then natural selection will produce evolutionary change (*ceteris paribus*, of course). But genes are affected by natural selection only indirectly. So the gene's eye view, says Mayr, may have its uses, but it does not correctly represent how natural selection works.

Mayr calls his second point *the genetic theory of relativity* (1963, p. 296). This principle says that "no gene has a fixed selective value, the same gene may confer high fitness on one genetic background and be virtually lethal on another." Should we conclude from this remark that there is never selection for single genes or that a single gene simultaneously experiences different selection pressures in different genetic backgrounds? In either case, the lesson here seems to be quite different from that provided by Mayr's first point—which was that phenotypes, not genotypes, are selected for. In this case, however, it seems to be gene complexes, rather than single genes, which are the objects of selection.

Mayr's first point about phenotypes and genotypes raises the following question: if we grant that selection acts "directly" on phenotypes and only "indirectly" on genotypes, why should it follow that natural selection is not selection for genetic attributes? Natural selection is a causal process; to say that there is selection for some (genotypic or phenotypic)

trait X is to say that having X causes differential reproductive success (*ceteris paribus*).[1] So, if there is selection for protective coloration, this just means that protective coloration generates a reproductive advantage. But suppose that this phenotype is itself caused by one or more genes. Then having those genes causes a reproductive advantage as well. Thus, if selection is a causal process, in acting on phenotypes it also acts on the underlying genotypes. Whether this is "direct" or not may be important, but it doesn't bear on the question of what is and what is not selected for. Selection, in virtue of its causal character and on the assumption that causality is transitive, seems to block the sort of asymmetry that Mayr demands. Asking whether phenotypes or genotypes are selected for seems to resemble asking whether a person's death was caused by the entry of the bullet or by the pulling of the trigger.

Mayr's second point—his genetic principle of relativity—is independent of the alleged asymmetry between phenotype and genotype. It is, of course, not in dispute that a gene's fitness depends on its genetic (as well as its extrasomatic) environment. But does this fact show that there is selection for gene complexes and not for single genes? Advocates of genic selection tend to acknowledge the relativity but to deny the conclusion that Mayr draws. Williams (1966, pp. 56–7) gives clear expression to this common reaction when he writes:

> 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 for one locus can be iterated over all loci. Adaptation can thus be attributed to the effect of selection acting independently at each locus.

[1]The "*ceteris paribus*" is intended to convey the fact that selection for X can fail to bring about greater reproductive success for objects that have X, if countervailing forces act. Selection for X, against Y, and so on, are component forces that combine vectorially to determine the dynamics of the population.

Dawkins (1976, p. 40) considers the same problem: how can single genes be selected for, if genes build organisms only in elaborate collaboration with each other and with the environment? He answers by way of an analogy:

> One oarsman on his own cannot win the Oxford and Cambridge boat race. He needs eight colleagues. Each one is a specialist who always sits in a particular part of the boat—bow or stroke or cox, etc. Rowing the boat is a cooperative venture, but some men are nevertheless better at it than others. Suppose a coach has to choose his ideal crew from a pool of candidates, some specializing in the bow position, others specializing as cox, and so on. Suppose that he makes his selection as follows. Every day he puts together three new trial crews, by random shuffling of the candidates, for each position, and he makes the three crews race against each other. After some weeks of this it will start to emerge that the winning boat often tends to contain the same individual men. These are marked up as good oarsmen. Other individuals seem consistently to be found in slower crews, and these are eventually rejected. But even an outstandingly good oarsman might sometimes be a member of a slow crew, either because of the inferiority of the other members, or because of bad luck—say a strong adverse wind. It is only *on average* that the best men tend to be in the winning boat.
>
> The oarsmen are genes. The rivals for each seat in the boat are alleles potentially capable of occupying the same slot along the length of a chromosome. Rowing fast corresponds to building a body which is successful at surviving. The wind is the external environment. The pool of alternative candidates is the gene pool. As far as the survival of any one body is concerned, all its genes are in the same boat. Many a good gene gets into bad company, and finds itself sharing a body with a lethal gene, which kills the body off in childhood. Then the good gene is destroyed along with the rest. But this is only one body, and replicas of the same good gene live on in other bodies which lack the lethal gene. Many copies of good genes are dragged under because they happen to share a body with bad genes, and many perish through other forms of ill luck, say when their body is struck by lightning. But by definition luck, good and bad, strikes at random, and a gene which is consistently on the losing side is not unlucky; it is a bad gene.

Notice that this passage imagines that oarsmen (genes) are good and bad pretty much *in*dependently of their context. But even when fitness is heavily influenced by context, Dawkins still feels that selection functions at the level of the single gene. Later in the book (pp. 91–2), he considers

what would happen if a team's performance were improved by having the members communicate with each other. Suppose that half of the oarsmen spoke only English and the other half spoke only German:

> What will emerge as the overall best crew will be one of the two stable states—pure English or pure German, but not mixed. Superficially it looks as though the coach is selecting whole language groups *as units*. This is not what he is doing. He is selecting individual oarsmen for their apparent ability to win races. It so happens that the tendency for an individual to win races depends on which other individuals are present in the pool of candidates.

Thus, Dawkins follows Williams in thinking that genic selectionism is quite compatible with the fact that a gene's fitness depends on context.

Right after the passage just quoted, Dawkins says that he favors the perspective of genic selectionism because it is more "parsimonious". Here, too, he is at one with Williams (1966), who uses parsimony as one of two main lines of attack against hypotheses of group selection. The appeal to simplicity may confirm a suspicion that already arises in this context: perhaps it is a matter of taste whether one prefers the single gene perspective or the view of selection processes as functioning at a higher level of organization. As long as we agree that genic fitnesses depend on context, what difference does it make how we tell the story? As natural as this suspicion is in the light of Dawkins' rowing analogy, it is mistaken. Hypotheses of group selection can be genuinely incompatible with hypotheses of organismic selection (Sober 1980), and, as we will see in what follows, claims of single gene selection are at times incompatible with claims that gene complexes are selected for and against. Regardless of one's aesthetic inclinations and regardless of whether one thinks of parsimony as a "real" reason for hypothesis choice, the general perspective of genic selectionism is mistaken for biological reasons.[2]

Before stating our objections to genic selectionism, we want to make clear one defect that this perspective does *not* embody. A quantitative genetic model that is given at any level can be recast in terms of parameters that attach to genes. This genic representation will correctly trace the trajectory of the population as its gene frequencies change. In a min-

[2]In the passages quoted, Williams and Dawkins adopt a very bold position: any selection process which *can* be represented as genic selection *is* genic selection. Dawkins never draws back from this monolithic view, although Williams' more detailed argumentation leads him to hedge. Williams allows that group selection (clearly understood to be an alternative to genic selection) is possible and has actually been documented once (see his discussion of the t-allele). But *all* selection processes—including group selection— can be "represented" in terms of selection coefficients attaching to single genes. This means that the representation argument proves far too much.

imal sense (to be made clear in what follows), it will be "descriptively adequate". Since the parameters encapsulate information about the environment, both somatic and extrasomatic, genic selectionism cannot be accused of ignoring the complications of linkage or of thinking that genes exist in a vacuum. The defects of genic selectionism concern its distortion of causal processes, not whether its models allow one to predict future states of the population.[3]

The causal considerations which will play a preeminent role in what follows are not being imposed from without, but already figure centrally in evolutionary theory. We have already mentioned how we understand the idea of *selection for X*. Our causal construal is natural in view of how the phenomena of linkage and pleiotropy are understood (see Sober 1981a). Two genes may be linked together on the same chromosome, and so selection for one may cause them both to increase in frequency. Yet the linked gene—the "free rider"— may be neutral or even deleterious; there was no selection *for it*. In describing pleiotropy, the same distinction is made. Two phenotypic traits may be caused by the same underlying gene complex, so that selection for one leads to a proliferation of both. But, again, there was no selection for the free rider. So it is a familiar idea that two traits can attach to exactly the same organisms and yet differ in their causal roles in a selection process. What is perhaps less familiar is that two sets of selection coefficients may both attach to the same population and yet differ in their causal roles—the one causing change in frequencies, the other merely reflecting the changes that ensue.

2. Averaging and Reification. Perhaps the simplest model exhibiting the strategy of averaging recommended by Williams and Dawkins is used in describing heterozygote superiority. In organisms whose chromosomes come in pairs, individuals with different genes (or alleles) at the same location on two homologous chromosomes are called heterozygotes. When a population has only two alleles at a locus, there will be one heterozygote form (*Aa*) and two homozygotes (*AA* and *aa*). If the heterozygote is superior in fitness to both homozygotes, then natural selection may modify the frequencies of the two alleles *A* and *a*, but will not drive either to fixation (i.e., 100%), since reproduction by heterozygotes will inevitably replenish the supply of homozygotes, even when homozygotes are severely selected against. A textbook example of this phenomenon is the sickle cell trait in human beings. Homozygotes for the allele controlling the trait develop severe anemia that is often fatal in

[3]Wimsatt (1980) criticizes genic selectionist models for being computationally inadequate and for at best providing a kind of "genetic bookkeeping" rather than a "theory of evolutionary change". Although we dissent from the first criticism, our discussion in what follows supports Wimsatt's second point.

childhood. Heterozygotes, however, suffer no deleterious effects, but enjoy a greater than average resistance to malaria. Homozygotes for the other allele have neither the anemia nor the immunity, and so are intermediate in fitness. Human populations with both alleles that live in malarial areas have remained polymorphic, but with the eradication of malaria, the sickle cell allele has been eliminated.

Population genetics provides a simple model of the selection process that results from heterozygotes' having greater viability than either of the homozygotes (Li 1955). Let p be the frequency of A and q be the frequency of a (where $p + q = 1$). Usually, the maximal fitness of Aa is normalized and set equal to 1. But for clarity of exposition we will let w_1 be the fitness of AA, w_2 be the fitness of Aa, and w_3 be the fitness of aa. These genotypic fitness values play the mathematical role of transforming genotype frequencies before selection into genotype frequencies after selection:

	$\underline{AA}$	$\underline{Aa}$	$\underline{aa}$
Proportion before selection	p^2	$2pq$	q^2
Fitness	w_1	w_2	w_3
Proportion after selection	$\frac{p^2w_1}{\overline{W}}$	$\frac{2pqw_2}{\overline{W}}$	$\frac{q^2w_3}{\overline{W}}$

Here, $\overline{W}$, the average fitness of the population, is $p^2w_1 + 2pqw_2 + q^2w_3$. Assuming random mating, the population will move towards a stable equilibrium frequency $\hat{p}$ where

$$\hat{p} = \frac{w_3 - w_2}{(w_1 - w_2) + (w_3 - w_2)}.$$

It is important to see that this model attributes fitness values and selection coefficients to diploid genotypes and not to the single genes A and a. But, as genic selectionists are quick to emphasize, one can always define the required parameters. Let us do so.

We want to define W_A, which is the fitness of A. If we mimic the mathematical role of genotype fitness values in the previous model, we will require that W_A obey the following condition:

> $W_A \times$ frequency of A before selection = frequency of A after selection $\times \overline{W}$.

Since the frequency of A before selection is p and the frequency of A after selection is

$$\frac{w_1p^2 + w_2pq}{\overline{W}},$$

it follows that

$$W_A = w_1 p + w_2 q.$$

By parity of reasoning,

$$W_a = w_3 q + w_2 p.$$

Notice that the fitness values of single genes are just weighted averages of the fitness values of the diploid genotypes in which they appear. The weighting is provided by their frequency of occurrence in the genotypes in question. The genotypic fitnesses specified in the first model are *constants;* as a population moves toward its equilibrium frequency, the selection coefficients attaching to the three diploid genotypes do not change. In contrast, the expression we have derived for allelic fitnesses says that allelic fitnesses change as a function of their own frequencies; as the population moves toward equilibrium, the fitnesses of the alleles must constantly be recomputed.

Heterozygote superiority illustrates the principle of genetic relativity. The gene *a* is maximally fit in one context (namely, when accompanied by *A*) but is inferior when it occurs in another (namely, when it is accompanied by another copy of itself). In spite of this, we can average over the two different contexts and provide the required representation in terms of genic fitness and genic selection.

In the diploid model discussed first, we represented the fitness of the three genotypes in terms of their *viability,* that is, in terms of the proportion of individuals surviving from egg to adult. It is assumed that the actual survivorship of a class of organisms sharing the same genotype precisely represents the fitness of that shared genotype. This assumes that random drift is playing no role. Ordinarily, fitness *cannot* be identified with actual reproductive success (Brandon 1978; Mills and Beatty 1979; Sober 1981a). The same point holds true, of course, for the fitness coefficients we defined for the single genes.[4]

Of the two descriptions we have constructed of heterozygote superiority, the first model is the standard one; in it, *pairs* of genes are the bearers of fitness values and selection coefficients. In contrast to this diploid model, our second formulation adheres strictly to the dictates of genic selectionism, according to which it is *single genes* which are the

[4]We see from this that Dawkins' remark that a gene that is "consistently on the losing side is not unlucky; it's a bad gene" is not quite right. Just as a single genotoken (and the organism in which it is housed) may enjoy a degree of reproductive success that is not an accurate representation of its fitness, so a set of genotokens (which are tokens of the same genotype) may encounter the same fate. Fitness and actual reproductive success are guaranteed to be identical only in models which ignore random drift and thereby presuppose an infinite population.

bearers of the relevant evolutionary properties. We now want to describe what each of these models will say about a population that is at its equilibrium frequency.

Let's discuss this situation by way of an example. Suppose that both homozygotes are lethal. In that case, the equilibrium frequency is .5 for each of the alleles. Before selection, the three genotypes will be represented in proportions 1/4, 1/2, 1/4, but after selection the frequencies will shift to 0, 1, 0. When the surviving heterozygotes reproduce, Mendelism will return the population to its initial 1/4, 1/2, 1/4 configuration, and the population will continue to zig-zag between these two genotype configurations, all the while maintaining each allele at .5. According to the second, single gene, model, at equilibrium the fitnesses of the two genes are both equal to 1 and the selection coefficients are therefore equal to zero. At equilibrium, no selection occurs, on this view. Why the population's *genotypic configuration* persists in zig-zagging, the gene's eye point of view is blind to see; it must be equally puzzling why $\overline{W}$, the average fitness of the population, also zig-zags. However, the standard diploid model yields the result that selection occurs when the population is at equilibrium, just as it does at other frequencies, favoring the heterozygote at the expense of the homozygotes. Mendelism *and selection* are the causes of the zig-zag. Although the models are computationally equivalent in their prediction of gene frequencies, they are not equivalent when it comes to saying whether or not selection is occurring.

It is hard to see how the adequacy of the single gene model can be defended in this case. The biological term for the phenomenon being described is apt. We are talking here about *heterozygote superiority,* and both terms of this label deserve emphasis. The heterozygote—i.e., the diploid genotype (not a single gene)—is superior *in fitness* and, therefore, enjoys a selective advantage. To insist that the single gene is always the level at which selection occurs obscures this and, in fact, generates precisely the wrong answer to the question of what is happening at equilibrium. Although the mathematical calculations can be carried out in the single gene model just as they can in the diploid genotypic model, the phenomenon of heterozygote superiority cannot be adequately "represented" in terms of single genes. This model does not tell us what is patently obvious about this case: even at equilibrium, what happens to gene frequencies is an artifact of selection acting on diploid genotypes.

One might be tempted to argue that in the heterozygote superiority case, the kind of averaging we have criticized is just an example of frequency dependent selection and that theories of frequency dependent selection are biologically plausible and also compatible with the dictates of genic selectionism. To see where this objection goes wrong, one must distinguish genuine from spurious cases of frequency dependent selection.

The former occurs when the frequency of an allele has some *biological impact* on its fitness; an example would be the phenomenon of mimicry in which the rarity of a mimic enhances its fitness. Here one can tell a biological story explaining why the fitness values have the mathematical form they do. The case of heterozygote superiority is altogether different; here frequencies are taken into account simply as a mathematical contrivance, the only point being to get the parameters to multiply out in the right way.

The diploid model is, in a sense, more contentful and informative than the single gene model. We noted before that from the *constant* fitness values of the three genotypes we could obtain a formula for calculating the fitnesses of the two alleles. Allelic fitnesses are implied by genotype fitness values and allelic frequencies; since allelic frequencies change as the population moves toward equilibrium, allelic fitnesses must constantly be recomputed. However, the derivation in the opposite direction cannot be made.[5] One cannot deduce the fitnesses of the genotypes from allelic fitnesses and frequencies. This is especially evident when the population is at equilibrium. At equilibrium, the allelic fitnesses are identical. From this information alone, we cannot tell whether there is no selection at all or whether some higher level selection process is taking place. Allelic frequencies plus genotypic fitness imply allelic fitness values, but allelic frequencies plus allelic fitness values do not imply genotypic fitness values. This derivational asymmetry suggests that the genotypic description is more informative.

Discussions of reductionism often suggest that theories at lower levels of organization will be more detailed and informative than ones at higher levels. However, here, the more contentful, constraining model is provided at the higher level. The idea that genic selection models are "deeper" and describe the fundamental level at which selection "really" occurs is simply not universally correct.

The strategy of averaging fosters the illusion that selection is acting at a lower level of organization than it in fact does. Far from being an idiosyncratic property of the genic model of heterozygote superiority just discussed, averaging is a standard technique in modelling a variety of selection processes. We will now describe another example in which this technique of representation is used. The example of heterozygote superiority focused on differences in genotypic *viabilities*. Let us now consider the way differential fertilities can be modelled for one locus with two alleles. In the fully general case, fertility is a property of a mating

[5]If the heterozygote fitness is set equal to 1, the derivation is possible for the one locus two allele case considered. But if more than two alleles are considered, the asymmetry exists even in the face of normalization.

pair, not of an individual. It may be true that a cross between an *AA* male and *aa* female has an expected number of offspring different from a cross between an *AA* female and an *aa* male. If fitnesses are a unique function of the pair, the model must represent nine possible fitnesses, one for each mating pair. Several special cases permit a reduction in dimensionality. If the sex of a genotype does not affect its fertility, then only six fitnesses need be given; and if fertility depends only on one of the sexes, say the females, the three female genotypes may be assigned values which fix the fertilities of all mating pairs.

But even when these special cases fail to obtain, the technique of averaging over contexts can nevertheless provide us with a fitness value for each genotype. Perhaps an *aa* female is highly fertile when mated with an *Aa* male but is much less so when mated with an *AA* male; perhaps *aa* females are quite fertile on average, but *aa* males are uniformly sterile. No matter—we can merely average over all contexts and find the average effect of the *aa* genotype. This number will fluctuate with the frequency distributions of the different mating pairs. Again, the model appears to locate selection at a level lower than what might first appear to be the case. Rather than assigning fertilities to mating pairs, we now seem to be assigning them to genotypes. This mathematical contrivance is harmless as long as it does not lead us to think that selection really acts at this lower level of organization.[6]

Our criticism of genic selectionism has so far focused on two forms of selection at a single locus. We now need to take account of how a multilocus theory can imply that selection is not at the level of the selfish gene. The pattern of argument is the same. Even though the fitness of a pair of genes at one locus may depend on what genes are found at other loci, the technique of averaging may still be pressed into service. But the selection values thereby assigned to the three genotypes at a single locus will be artifacts of the fitnesses of the nine genotype complexes that exist at the two loci. As in the examples we already described, the lower-level selection coefficients will change as a function of genotype frequencies, whereas the higher-level selection coefficients will remain constant. An example of this is provided by the work of Lewontin and White (reported in Lewontin 1974) on the interaction of two chromosome inversions found in the grasshopper *Moraba scura*. On each of the chromosomes of the EF pair, Standard (ST) and Tidbinbilla (TD) may be found. On the CD chromosome pair, Standard (ST) and Blundell (BL) are the two

[6]The averaging of effects can also be used to foster the illusion that a group selection process is really just a case of individual selection. But since this seems to be a relatively infrequent source of abuse, we will not take the space to spell out an example.

alternatives. The fitness values of the nine possible genotypes were estimated from nature as follows:

Chromosome EF	Chromosome CD		
	ST/ST	ST/BL	BL/BL
ST/ST	0.791	1.000	0.834
ST/TD	0.670	1.006	0.901
TD/TD	0.657	0.657	1.067

Notice that there is heterozygote superiority on the CD chromosome if the EF chromosome is either ST/ST or ST/TD, but that BL/BL dominance ensues when the EF chromosome is homozygous for TD. Moreover, TD/TD is superior when in the context BL/BL but is inferior in the other contexts provided by the CD pair. These fitness values represent differences in viability, and again the inference seems clear that selection acts on multilocus genotypic configurations and not on the genotype at a single locus, let alone on the separate genes at that locus.

3. Individuating Selection Processes. The examples in the previous section have a common structure. We noted that the fitness of an object (a gene, a genotype) varied significantly from context to context. We concluded that selection was operating at a level higher than the one posited by the model—at the level of genotypes in the case of heterozygote superiority, at the level of the mating pair in the fertility model, and at the level of pairs of chromosome inversions in the *Moraba scura* example. These analyses suggest the following principle: *if the fitness of X is context sensitive, then there is not selection for X; rather, there is selection at a level of organization higher than X.*

We believe that this principle requires qualification. To see why context sensitivity is not a *sufficient* condition for higher level selection, consider the following example. Imagine a dominant lethal gene; it kills any organisms in which it is found unless the organism also has a suppressor gene at another locus. Let's consider two populations. In the first population, each organism is homozygous for a suppressor gene which prevents copies of the lethal gene from having any effect. In the second population, no organism has a suppressor, so, whenever the lethal gene occurs, it is selected against. A natural way of describing this situation is that there is selection against the lethal gene in one population, but, in the other, there is no selection going on at all. It would be a mistake (of the kind we have already examined) to think that there is a single selection process at work here against the lethal gene, whose magnitude we calculate by averaging over the two populations. However, we do not conclude from this that there is a selection process at work at some higher level of organization than the single gene. Rather, we conclude that there

are *two* populations; in one, *genic* selection occurs, and in the other *nothing* occurs. So the context sensitivity of fitness is an ambiguous clue. If the fitness of X depends on genetic context, this may mean that there is a single selection process at some higher level, *or* it may mean that there are several different selection processes at the level of X. Context sensitivity does not suffice for there to be selection at a higher level.[7]

Thus, the fitness of an object can be sensitive to genetic context for at least two reasons. How are they to be distinguished? This question leads to an issue at the foundation of *all* evolutionary models. What unites a set of objects as all being subject to a single selection process? Biological modelling of evolution by natural selection is based on three necessary and sufficient conditions (Lewontin 1970): a given set of objects must exhibit variation; some individuals must be fitter than others; and there must be correlation between the fitness of parents and the fitness of offspring. Here, as before, we will identify fitness with actual reproductive success, subject to the proviso that these will coincide only in special cases. Hence, evolution by natural selection exists when and only when there is heritable variation in fitness.

Using these conditions presupposes that some antecedent decision has been made about which objects can appropriately be lumped together as participating in a single selection process (or, put differently, the conditions are not sufficient after all). Biologists do not talk about a *single* selection process subsuming widely scattered organisms of different species which are each subject to quite different local conditions. Yet, such a gerrymandered assemblage of objects may well exhibit heritable variation in fitness. And even within the same species, it would be artificial to think of two local populations as participating in the same selection process because one encounters a disease and the other experiences a food shortage as its principal selection pressure. Admittedly, the gene frequencies can be tabulated and pooled, but in some sense the relation of organisms to environments is too heterogeneous for this kind of averaging to be more than a mathematical contrivance.

It is very difficult to spell out necessary and sufficient conditions for when a set of organisms experience "the same" selection pressure. They need not compete with each other. To paraphrase Darwin, two plants may struggle for life at the edge of a desert, and selection may favor the one more suited to the stressful conditions. But it needn't be the case that some resource is in short supply, so that the amount expropriated by one reduces the amount available to the other. Nor need it be true that the

[7]The argument given here has the same form as one presented in Sober (1980) which showed that the following is not a sufficient condition for group selection: there is heritable variation in the fitness of groups in which the fitness of an organism depends on the character of the group it is in.

two organisms be present in the same geographical locale; organisms in the semi-isolated local populations of a species may experience the same selection pressures. What seems to be required, roughly, is that some common causal influence impinge on the organisms. This sameness of causal influence is as much determined by the biology of the organisms as it is by the physical characteristics of the environment. Although two organisms may experience the same temperature fluctuations, there may be no selective force acting on both. Similarly, two organisms may experience the same selection pressure (for greater temperature tolerance, say) even though the one is in a cold environment and the other is in a hot one. Sameness of causal influence needs to be understood biologically.

For all the vagueness of this requirement, let us assume that we have managed to single out the class of objects which may properly be viewed as participating in a single selection process. To simplify matters, let us suppose that they are all organisms within the same breeding population. What, then, will tell us whether selection is at the level of the single gene or at the level of gene complexes? To talk about either of these forms of selection is, in a certain important but nonstandard sense, to talk about "group selection". Models of selection do not concern single organisms or the individual physical copies of genes (i.e., geno*tokens*) that they contain. Rather, such theories are about groups of organisms which have in common certain geno*types*. To talk about selection for X, where X is some single gene or gene cluster, is to say something about the effect of having X and of lacking X on the relevant subgroups of the breeding population. If there is selection for X, every object which has X has its reproductive chances augmented by its possessing X. This does not mean that every organism which has X has precisely the same overall fitness, nor does it mean that every organism must be affected in precisely the same way (down to the minutest details of developmental pathways). Rather, what is required is that the effect of X on each organism be in the same direction as far as its overall fitness is concerned. Perhaps this characterization is best viewed as a limiting ideal. To the degree that the population conforms to this requirement, it will be appropriate to talk about genic selection. To the degree that the population falls short of this, it will be a contrivance to represent matters in terms of genic selection.[8]

[8]The definition of genic selection just offered is structurally similar to the definition of group selection offered in Sober (1980). There, the requirement was that for there to be selection for groups which are X, it must be the case that every organism in a group that is X has one component of its fitness determined by the fact that it is in a group which is X. In group selection, organisms within the same group are bound together by a common group characteristic just as in genic selection organisms with the same gene are influenced in the same way by their shared characteristic.

It is important to be clear on why the context sensitivity of a gene's effect on organismic fitness is crucial to the question of genic selection. Selection theories deal with groups of single organisms and not with organisms taken one at a time. It is no news that the way a gene inside of a single organism will affect that organism's phenotype and its fitness depends on the way it is situated in a context of background conditions. But to grant this fact of context sensitivity does not impugn the claim of causation; striking the match caused it to light, even though the match had to be dry and in the presence of oxygen for the cause to produce the effect.

Selection theory is about geno*types* not geno*tokens*. We are concerned with what properties are selected for and against in a population. We do not describe single organisms and their physical constituents one by one. It is for this reason that the question of context sensitivity becomes crucial. If we wish to talk about selection for a single gene, then there must be such a thing as *the* causal upshot of possessing that gene. A gene which is beneficial in some contexts and deleterious in others will have many *organismic* effects. But at *the population level,* there will be no selection for or against that gene.

It is not simply the averaging over contexts which reveals the fact that genic selection coefficients are pseudoparameters; the fact that such parameters *change* in value as the population evolves while the biological relations stay fixed also points to their being artifacts. In the case of heterozygote superiority, genotypic fitnesses remain constant, mirroring the fact that the three genotypes have a uniform effect on the viability of the organisms in which they occur. The population is thereby driven to its equilibrium value while genic fitness values are constantly modified. A fixed set of biological relationships fuels both of these changes; the evolution of genic fitness values is effect, not cause.[9]

[9]In our earlier discussion of Mayr's ideas, we granted that selection usually acts "directly" on phenotypes and only "indirectly" on genotypes. But given the transitivity of causality, we argued that this fact is perfectly compatible with the existence of genotypic selection. However, our present discussion provides a characterization of when phenotypic selection can exist without there being any selection at the genotypic level. Suppose that individuals with the same genotype in a population end up with different phenotypes, because of the different microenvironments in which they develop. Selection for a given phenotype may then cross-classify the genotypes, and by our argument above, there will be no such thing as *the* causal upshot of a genotype. Averaging over effects will be possible, as always, but this will not imply genotypic selection. It is important to notice that this situation can allow evolution by natural selection to occur; gene frequencies can change in the face of phenotypic selection that is not accompanied by any sort of genotypic selection. Without this possibility, the idea of phenotypic selection is deprived of its main interest. There is no reason to deny that there can be selection for phenotypic differences that have no underlying genetic differences, but this process will not produce any change in the population (ignoring cultural evolution and the like).

Are there real cases of genic selection? A dominant lethal—a gene which causes the individual to die regardless of the context in which it occurs—would be selected against. And selection for or against a phenotypic trait controlled by a single locus having two alleles might also be describable in terms of genic selection, provided that the heterozygote is intermediate in fitness between the two homozygotes. In addition, meiotic drive, such as is found in the house mouse *Mus musculus,* similarly seems to involve genic selection (Lewontin and Dunn 1960). Among heterozygote males, the proportion of t-alleles in the sperm pool is greater than 1/2. Chromosomes with the t-allele have enhanced chances of representation in the gamete pool, and this directional effect seems to hold true regardless of what other genes are present at other loci.[10] At this level, but not at the others at which the t-allele affects the population, it is appropriate to talk about genic selection.

We so far have construed genic selection in terms of the way that having or lacking a gene can affect the reproductive chances of organisms. But there is another possibility—namely, that genes differentially proliferate even though they have *no* effect on the phenotypes of organisms. A considerable quantity of DNA has no known function; Orgel and Crick (1980) and Doolittle and Sapienza (1980) have suggest that this DNA may in fact be "junk". Such "selfish DNA", as they call it, could nonetheless undergo a selection process, provided that some segments are better replicators than others. Although these authors associate their ideas with Dawkins' selfish gene, their conception is far more restrictive. For Dawkins, *all* selection is genic selection, whereas for these authors, selfish DNA is possible only when the differential replication of genes is not exhaustively accounted for by the differential reproductive success of organisms.

Standard ways of understanding natural selection rule out rather than substantiate the operation of genic selection. It is often supposed that much of natural selection is *stabilizing selection,* in which an intermediate phenotype is optimal (e.g., birth weight in human beings). Although the exact genetic bases of such phenotypes are frequently unknown, biologists often model this selection process as follows. It is hypothesized that the phenotypic value is a monotone increasing function of the number of "plus alleles" found at a number of loci. Whether selection favors the presence of plus genes at one locus depends on how many such genes exist at other loci. Although this model does not view heterozygote su-

[10]Genes at other loci which modify the intensity of segregator distortion are known to exist in *Drosophila;* the situation in the house mouse is not well understood. Note that the existence of such modifiers is consistent with genic selection, as long as they do not affect the *direction* of selection.

periority as the most common fitness relation *at a locus,* it nevertheless implies that a *heterogeneous genome* is superior in fitness. Exceptions to this intermediate optimum model exist, and the exact extent of its applicability is still an open question. Still, it appears to be widely applicable. If it is generally correct, we must conclude that the conditions in which genic selection exists are extremely narrow. Genic selection is not impossible, but the biological constraints on its operation are extremely demanding.

Although it is just barely conceivable that a critique of a scientific habit of thought might be devoid of philosophical presuppositions, our strictures against genic selectionism are not a case in point. We have described selection processes in which genic selection coefficients are *reifications;* they are artifacts, not causes, of evolution. For this to count as a criticism, one must abandon a narrowly instrumentalist view of scientific theories; this we gladly do, in that we assume that selection theory ought to pinpoint causes as well as facilitate predictions.

But even assuming this broadly noninstrumentalist outlook, our criticisms are philosophically partisan in additional ways. In that we have argued that genic selection coefficients are often "pseudoproperties" of genes, our criticisms of the gene's eye point of view are connected with more general metaphysical questions about the ontological status of properties. Some of these we take up in the following section. And in that we have understood "selection for" as a causal locution, it turns out that our account goes contrary to certain regularity analyses of causation. In populations in which selection generated by heterozygote superiority is the only evolutionary force, it is true that gene frequencies will move to a stable equilibrium. But this law-like regularity does not imply that there is selection for or against any individual gene. To say that "the gene's fitness value caused it to increase in frequency" is not simply to say that "any gene with that fitness value (in a relevantly similar population) would increase in frequency", since the former is false and the latter is true. Because we take natural selection to be a force of evolution, these remarks about causation have implications (explored in section 5) for how the concept of force is to be understood.

4. Properties. The properties, theoretical magnitudes, and natural kinds investigated by science ought not to be identified with the meanings that terms in scientific language possess. Nonsynonymous predicates (like "temperature" and "mean kinetic energy" and like "water" and "H_2O") may pick out the same property, and predicates which are quite meaningful (like "phlogiston" and "classical mass") may fail to pick out a property at all. Several recent writers have explored the idea that properties are to be individuated by their potential causal efficacy (Achin-

stein 1974; Armstrong 1978; Shoemaker 1980; and Sober 1982b). Besides capturing much of the intuitive content of our informal talk of properties, this view also helps explicate the role of property-talk in science (Sober 1981a). In this section, we will connect our discussion of genic selectionism with this metaphysical problem.

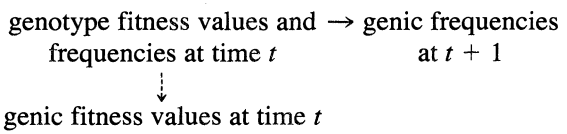
The definitional power of ordinary and scientific language allows us to take predicates which each pick out properties and to construct logically from these components a predicate which evidently does not pick out a property at all. An example of this is that old philosophical chestnut, the predicate "grue". We will say that an object is grue at a given time if it is green and the time is before the year 2000, or it is blue and the time is not before the year 2000. The predicate "grue" is defined from the predicates "green", "blue", and "time", each of which, we may assume for the purposes of the example, picks out a "real" property. Yet "grue" does not. A theory of properties should explain the basis of this distinction.

The difference between real and pseudoproperty is not captured by the ideas that animate the metaphysical issues usually associated with doctrines of realism, idealism, and conventionalism. Suppose that one adopts a "realist" position toward color and time, holding that things have the colors and temporal properties they do independently of human thought and language. This typical realist declaration of independence (Sober 1982a) will then imply that objects which are grue are so independently of human thought and language as well. In this sense, the "reality" of grulers is insured by the "reality" of colors and time. The distinction between real properties and pseudoproperties must be sought elsewhere.

Another suggestion is that properties can be distinguished from nonproperties by appeal to the idea of *similarity* or of *predictive power*. One might guess that green things are more similar to each other than grue things are to each other, or that the fact that a thing is green is a better predictor of its further characteristics than the fact that it is grue. The standard criticism of these suggestions is that they are circular. We understand the idea of similarity in terms of shared *properties,* and the idea of predictive power in terms of the capacity to facilitate inference of further *properties*. However, a more fundamental difficulty with these suggestions presents itself: even if grue things happened to be very similar to each other, this would not make grue a real property. If there were no blue things after the year 2000, then the class of grue things would simply be the class of green things before the year 2000. The idea of similarity and the idea of predictive power fail to pinpoint the *intrinsic* defects of nonproperties like grue. Instead, they focus on somewhat accidental facts about the objects which happen to exist.

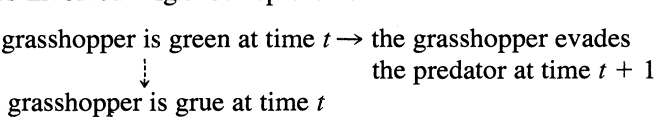
Grue is not a property for the same reason that genic selection coef-

ficients are pseudoparameters in models of heterozygote superiority. The key idea is not that nonproperties are mind-dependent or are impoverished predictors; rather, they cannot be causally efficacious. To develop this idea, let's note a certain similarity between grue and genic selection coefficients. We pointed out before that genotype fitnesses plus initial genotype frequencies in the population causally determine the gene frequencies after selection. These same parameters also permit the mathematical derivation of genic fitness values, but, we asserted, these genic fitness values are artifacts; they do not cause the subsequent alteration in gene frequencies. The structure of these relationships is as follows.



Note that there are two different kinds of determination at work here. Genic fitness values at a given time are not *caused* by the genotypic fitness values at the same time. We assume that causal relations do not obtain between simultaneous events; rather, the relationship is one of logical or mathematical deducibility (symbolized by a broken line). On the other hand, the relation of initial genotype fitnesses and frequencies and subsequent gene frequencies is one of causal determination (represented by a solid line).

Now let's sketch the causal relations involved in a situation in which an object's being green produces some effect. Let the object be a grasshopper. Suppose that it matches its grassy background and that this protective coloration hides it from a hungry predator nearby. The relationships involved might be represented as follows.



Just as in the above case, the object's color at the time *logically implies* that it is grue at that time but is the *cause* of its evading the predator at a subsequent time. And just as genic fitness values do not cause changes in gene frequencies, so the grasshopper's being grue does not cause it to have evaded its predator.

Our assessment of genic selectionism was not that genic fitness values are *always* artifactual. In cases other than that of heterozygote superiority—say, in the analysis of the *t*-allele—it may be perfectly correct to attribute causal efficacy to genic selection coefficients. So a predicate can pick out a real (causally efficacious) property in one context and fail to do so in another. This does not rule out the possibility, of course, that

a predicate like "grue" is *globally artifactual*. But this consequence should not be thought to follow from a demonstration that grue is artifactual in a single kind of causal process.

The comparison of grue with genic selection is not meant to solve the epistemological problems of induction that led Goodman (1965) to formulate the example. Nor does the discussion provide any *a priori* grounds for distinguishing properties from nonproperties. Nor is it even a straightforward and automatic consequence of the truth of any scientific model that grue is artifactual, or that the idea of causal efficacy captures the metaphysical distinction at issue. Instead, the point is that a certain natural interpretation of a biological phenomenon helps to indicate how we ought to understand a rather abstract metaphysical issue.[11]

5. Forces. Our arguments against genic selectionism contradict a standard positivist view of the concept of force. Positivists have often alleged that Newtonian mechanics tells us that forces are not "things", but that claims about forces are simply to be understood as claims about how objects actually behave, or would behave, if nothing else gets in the way. An exhaustive catalog of the forces acting on a system is to be understood as simply specifying a set of counterfactuals that describe objects.[12]

A Newtonian theory of forces will characterize each force in its domain in terms of the changes it would produce, were it the only force at work. The theory will take pair-wise combinations of forces and describe the joint effects that the two forces would have were they the only ones acting on a system. Then the forces would be taken three at a time, and so on, until a fully realistic model is constructed, one which tells us how real

[11]Another consequence of this analogy is that one standard diagnosis of what is wrong with "grue" fails to get to the heart of the matter. Carnap (1947) alleged that "green", unlike "grue", is purely qualitative, in that it makes no essential reference to particular places, individuals, or times. Goodman (1965) responded by pointing out that *both* predicates can be defined with reference to the year 2000. But a more fundamental problem arises: even if "grue" were, in some sense, not purely qualitative, this would not provide a fully general characterization of when a predicate fails to pick out a real property. Genic selection coefficients are "purely qualitative" if genotypic coefficients are, yet their logical relationship to each other exactly parallels that of "grue" to "green". Predicates picking out real properties can be "gruified" in a purely qualitative way: Let F and G be purely qualitative and be true of all the objects sampled (the emeralds, say). The predicate "(F and G) or ($-F$ and $-G$)" is a gruification of F and poses the same set of problems as Goodman's "grue".

[12]Joseph (1980) has argued that this position, in treating the distribution of objects as given and then raising epistemological problems about the existence of forces, is committed to the existence of an asymmetry between attributions of quantities of *mass* to points in space-time and attributions of quantities of *energy* thereto. He argues that this idea, implicit in Reichenbach's (1958) classic argument for the conventionality of geometry, contradicts the relativistic equivalence of mass and energy. If this is right, then the positivistic view of force just described, far from falling out of received physical theory, in fact contradicts it.

objects, which after all are subject to many forces, can be expected to behave. Each step in this program may face major theoretical difficulties, as the recent history of physics reveals (Cartwright 1980b; Joseph 1980).

This Newtonian paradigm is a hospitable home for the modelling of evolutionary forces provided in population genetics. The Hardy-Weinberg Law says what happens to gene frequencies when no evolutionary forces are at work. Mutation, migration, selection, and random drift are taken up one at a time, and models are provided for their effects on gene frequencies when no other forces are at work. Then these (and other) factors are taken up in combination. Each of these steps increases the model's realism. The culmination of this project would be a model that simultaneously represents the interactions of all evolutionary forces.

Both in physics and in population genetics, it is useful to conceive of forces in terms of their *ceteris paribus* effects. But there is more to a force than the truth of counterfactuals concerning change in velocity, or change in gene frequencies. The laws of motion describe the *effects* of forces, but they are supplemented by source laws which describe their *causes*. The standard genotypic model of heterozygote superiority not only says what will happen to a population, but also tells us what makes the population change.

It is quite true that when a population moves to an equilibrium value, due to the selection pressures generated by heterozygote superiority, the alleles are "disposed" to change in frequency in certain ways.[13] That is, the frequencies *will* change in certain ways, as long as no other evolutionary forces impinge. Yet, there is no force of genic selection at work here. If this is right, then the claim that genic selection is occurring must involve more than the unproblematic observations that gene frequencies are disposed to change in certain ways.

There is something more to the concept of force because it involves the idea of *causality,* and there is more to the idea of causality than is spelled out by such counterfactuals as the ones cited above. Suppose that something pushes (i.e., causally interacts with in a certain way) a billiard ball due north, and something else pushes it due west. Assuming that nothing else gets in the way, the ball will move northwest. There are two "component" forces at work here, and, as we like to say, one "net" force. However, there is a difference between the components and the resultant. Although something pushes the ball due north and something else pushes it due west, nothing pushes it northwest. In a sense, the re-

[13]For the purpose of this discussion, we will assume that attributions of dispositions and subjunctive conditionals of certain kinds are equivalent. That is, we will assume that to say that x is disposed to F is merely to say that if conditions were such-and-such, x would F.

sultant force is not a force at all, if by force we mean a causal agency. The resultant force is an artifact of the forces at work in the system. For mathematical purposes this distinction may make no difference. But if we want to understand why the ball moves the way it does, there is all the difference in the world between component and net.[14]

The "force" of genic selection in the evolutionary process propelled by heterozygote superiority is no more acceptable than the resultant "force" which is in the northwesterly direction. In fact, it is much worse. The resultant force, at least, is defined from the same conceptual building blocks as the component forces are. Genic selection coefficients, however, are gerrymandered hodgepodges, conceptually and dynamically quite unlike the genotypic selection coefficients that go into their construction. For genic selection coefficients are defined in terms of genotypic selection coefficients *and* gene frequencies. As noted before, they vary as the population changes in gene frequency, whereas the genotypic coefficients remain constant. And if their uniform zero value at equilibrium is interpreted as meaning that no selection is going on, one obtains a series of false assertions about the character of the population.

The concept of force is richer than that of disposition. The array of forces that act on a system uniquely determine the disposition of that system to change, but not conversely. If natural selection is a force and fitness is a disposition (to be reproductively successful), then the concept of selection is richer than that of fitness. To say that objects differ in fitness is not yet to say *why* they do so. The possible causes of such differences may be various, in that many different combinations of selection pressures acting at different levels of organization can have the same instantaneous effect on gene frequencies. Although selection coefficients and fitness values are interdefinable mathematically (so that, typically, $s = 1 - w$), they play different conceptual roles in evolutionary theory (Sober 1980).

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[14]This position is precisely the opposite of that taken by Cartwright (1980a), who argues that net forces, rather than component forces, are the items which really exist. Cartwright argues this by pointing out that the billiard ball moves northwesterly and not due north or due west. However, this appears to conflate the *effect* of a force with the force or forces actually at work.

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