ON THE ADAPTATIONS OF ORGANISMS AND THE FITNESS OF TYPES*

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We claim that much of the confusion associated with the "tautology problem" about survival of the fittest is due to the mistake of attributing fitness to individuals instead of to types. We argue further that the problem itself cannot be solved merely by taking fitness as the aggregate cause of reproductive success. We suggest that a satisfying explanation must center not on logical analysis of the concept of general adaptedness but on the empirical analysis of single adapted traits and their causal relationship to changes in allele frequencies.

The question of whether one of the major components of the theory of evolution, Natural Selection, is based on a tautology is a recurring theme in the recent literature on philosophical problems of biology. Many authors have attempted to deal with this question on many different levels. Most authors deny that a tautology is involved; but in light of the rarity of serious philosophical presentations of the tautology charge itself, the fact that a need is repeatedly felt to refute the charge once again seems to indicate a fundamental dissatisfaction with the refutations themselves.

The tautology criticism centers on the unfortunate phrase coined by Herbert Spencer and adopted by Darwin in later editions of *The Origin of Species*—"the survival of the fittest". The phrase, in Darwin's interpretation, asserts that the fitter forms tend to be reproductively more successful, that is, that selection leads to adaptation to the environment. However, since it also seems that fitness in the final analysis is nothing but the ability to be reproductively successful,[1] the statement appears to be without empirical content. But surely, Darwin's claim was something more than that fitter forms are fitter.

Although there have been many different attempts from widely differing points of view to explain why survival of the fittest is not tautological,

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[1]In recent discussions there are generally three *terms* involved—adaptedness, fitness, and reproductive success—but only two *concepts*. Some authors identify fitness with reproductive success and take adaptedness to be the cause of fitness; others identify fitness with overall adaptedness and take fitness to be the cause of success.

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all of them seem to have one common denominator. Many recent discussions of the logical problems involved with the concepts of fitness and adaptation in evolutionary theory use the same *thought experiment* and display the same *fundamental presupposition*. The thought experiment with various embellishments considers imaginary identical twins, one of which reproduces and the other of which is killed by some accident.[2] Because the two organisms are genetically identical, it would be counterintuitive to say that one twin is fitter than the other, even though it is reproductively much more successful. Two individuals can be equally fit without having equal reproductive success. Thus it is argued that fitness cannot be logically equivalent to actual reproductive success, but must be considered as a physical (probabilistic) cause of success. This sort of reasoning argues that if two individuals do not differ in fitness, they must be equal in fitness. This inference would in fact be legitimate if it were self-evident that individuals belong to the category of things that have a fitness value, or if a serious *argument* to this effect could be given. We don't find it self-evident that individuals can sensibly be said to be fit, and no one seems to have presented an argument in support of it. The thought experiment is, however, very important because it exposes an implicit presupposition underlying the entire discussion, which we think is worth explicating and questioning: the argument used to refute the tautology charge assumes that *individuals* are fit and that genetically *identical* individuals are *equally* fit. In the following we shall examine the concepts of adaptation and fitness with a view to their explanatory value.

1. Adaptation. One of the most influential contemporary formulations of the basic tenets of the theory of evolution (due to Lewontin 1978, 1984) enunciates the following three principles:

(a) The existence of variation in the population.
(b) Heritability of some of the variation.
(c) Differential reproductive success of different variants.

Lewontin considers these three necessary conditions to be sufficient for evolution by natural selection. In fact, however, they are sufficient only for unspecified change in gene frequencies. Natural selection is directive and operates only where there is a causal connection between the vari-

[2]Michael Scriven (1959) is credited with introducing this thought experiment into the philosophical discussion. He points out "that many organisms are killed by factors wholly unconnected with *any* characteristics they possess—for example, they happen to be sitting where a tree or a bomb falls. Of course, this is sometimes due to a habit or property they possess; but that is not always true, since even identical twins with identical habits do not always die together" (p. 478). Other writers (among those referred to in this paper) who have used this kind of thought experiment are Rosenberg (1985) and Mills and Beatty (1979).

ations and the reproductive success of the organisms possessing them. And in fact some philosophers who adopt Lewontin's formulation (for example, Brandon 1978) implicitly take this into account when they speak of "*significant variation*", which can only mean variation that is causally relevant to reproduction. However, Lewontin points out that Darwin also introduced a fourth principle into the theory, the principle of the struggle for existence, which explains why different organisms have different reproductive success. Darwin claimed that since variations favoring an individual's survival tend to become more common, they can provide the basis for further variations favoring the individual's survival and thus accumulate in a particular direction. The results of such directed accumulation may be called *adaptations*.

Adaptation is one of the most prominent phenomena in nature—the striking fit of organisms to their environments. It is a phenomenon that was described centuries before the era of Darwin and was even used as a premise in a proof for the existence of God, the argument from design. Since adaptation is still one of the major phenomena that evolutionary theory has to explain, it is obviously not very satisfying to dispose of the tautology problem simply by abandoning this concept altogether, as has been suggested recently (Krimbas 1984).

It will be useful to distinguish the *historical process* of "adaptation" from its cumulative *results* which may be the overall "adaptedness" of the organism to its environment or particular "adaptations" to the environment—by which we mean more or less discrete structural or behavioral traits. Whether such phylogenetic results remain "adaptive" in the future is an altogether different question. If we say that some trait is an adaptation we are referring to its causal past, while if we say it is adaptive we are referring to its causal present or future. Speaking of the overall "adaptedness" of an organism is ambiguous because it may mean one or the other or both depending on whether reference is made in the context to past or present. We shall be discussing adapted traits, that is, the results of the causal past.

An adaptation is always the result of natural selection; but whether it is also a trait that continues to favor an individual's survival in competition with other organisms and in the face of environmental stress depends on circumstances. For instance, if all organisms in a population have come to possess a particular adaptation with no meaningful variation,[3] then the trait is no longer relevant for *differences* in relative repro-

[3]Such "universal" traits are maintained in a population by stabilizing or purifying selection, that is, all new variants are eliminated from the population. The frequency of genetic changes affecting a universal trait (which is essential for the survival and reproductive success of all types in a population) depends on the frequency of mutation in the

ductive success in this population, although it might be essential to viability and reproduction. Thus it is obvious that we must define adaptations without relying on their *current* effects upon actual *differences* in reproductive success.

G. C. Williams (1966) has suggested two somewhat subjective rules for recognizing adaptations. Adaptations are, (a) organic systems that show a clear analogy with human implements; (b) organic systems that show a high degree of complexity and constancy between species.

We shall consider an adaptation to be a complex feature of an organism with a functional[4] significance whose structure is in some sense "complementary" to an aspect of its environment and whose origin is due to natural selection. Only when it is possible to show (or at least reasonable to infer) that the supposed adaptation has originated through or is being maintained by selection pressure, is the use of the term adaptation completely justified.

Most of the philosophical debate has concentrated on the search for criteria for defining adaptation, not in the sense of adapted traits but in the sense of general "*adaptedness*"; criteria are sought that are independent of actual reproductive success. This leads to confusion since an adaptation is *always* the *result* of differences in actual reproductive success and thus is causally dependent on success in past generations. On the other hand, it can be independent in the sense that it need not always lead to differences in reproductive success in present or future generations. It is admittedly sometimes difficult to circumscribe what exactly a "trait" is, and if pushed and stretched every "adaptation" expands to become an entire adapted organism or phenotype, but as it expands it loses all explanatory value. The overall adaptedness of an organism with reference to present or future is nothing other than the sum total of (non-accidental) causal factors influencing reproductive success. Thus the assertion that organisms of one kind are reproductively more successful than those of another because they are better adapted to their common environment has relatively little empirical content. Instead of being told that giraffes of a certain type are reproductively more successful because of their longer necks than those of other types, we are told that they are more successful because the sum total of causes of reproductive success in a certain unique environment favors them over giraffes of other types.

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relevant loci. However, the frequency of such mutations in the adult population will be nil since they will be lethal.

[4]A *function* can be defined as a property of a part of a self-reproducing system which the part has, due not to its own parts but to its relation to the system of which it is a part. In addition the property (or a functional equivalent) must be necessary for or must at least contribute to the self-reproduction of the system. Such a property of the part disappears when the part is removed from the system.

Furthermore, it becomes very easy to slip from adapted traits (past) to adaptive traits (future) by way of the overall adaptedness of the organism to its environment. In both cases seeming tautologies become very easy.

The main approaches to a definition of adaptation independent of actual success have concentrated on engineering design analysis and energy utilization considerations. Both of these use optimization models of systems in standard environments and thus try to establish an absolute measure of adaptation. Both are problematical. Brandon (1978) illustrates the problems of this kind of approach by arguing that it is possible to construct experiments in which one selects against organisms with lower energy requirements. And since artificial selection is only a kind of natural selection, the energy utilization definition of adaptation is found lacking. This is quite correct, but he maintains further that the same kind of argument can be used against any other success-independent definition. Brandon would have to deny that phenomena that have become universal such as the pathway of photosynthesis in green plants, the basic structure of the lung in mammals or the one to one sex ratio found in most sexually reproducing natural populations are adaptations, because a situation is conceivable in which they are no longer "adaptive". It thus becomes clear that what is actually being discussed under the title "adaptation" is merely overall adaptedness, that is, the ability to be reproductively successful, which can no more be separated *conceptually* from actual success than the ability to play basketball can be separated conceptually from the actual game.

2. Fitness. Unlike adaptation, fitness has a mathematically applicable definition which enables us to measure the fitness of one type of organism relative to another in a population in a certain environment on the basis of reproductive success. A definition of fitness that is universally accepted in biology, at least insofar as it is used as the basis of more detailed definitions, was given by Fisher in 1930 (1958). Fisher used a highly abstract model which allowed him to take survival to reproductive age as an adequate measure of reproductive success. He did not count descendants of individuals but rather survivors representing a type. Fisher's procedure makes no sense when applied to an individual, which either survives or does not survive; individuals can be quantified only as representatives of a type. And Fisher applied his concept of fitness explicitly to genotypes (1958, p. 50).[5] Although to our knowledge no one denies

[5]Although no doubt exists that fitness can be ascribed to genotypes, in some discussions philosophers seem not to realize that a genotype is a *type* and thus seem to believe that what is said to be fit is a pair of chunks of nucleic acid. Although since Fisher's days it has become common in genetics to use the term "genotype" not for the entire genetic information of an *organism* but rather for the information coding a particular *trait* that

that fitness is properly ascribed to genotypes it has often *also* been applied to other levels, a situation which has led to a lot of loose and metaphorical expressions. On different occasions, and by different authors, fitness has been assigned to alleles, traits, pieces of DNA, individuals, types and populations. Sometimes it has even been ascribed to sets, classes and lineages.

In order to clarify the concept of fitness we should ask what kinds of subjects the term can properly be predicated of, and whether its ascription to other sorts of things can be misleading. While it is true that only concrete entities (for example, individuals) reproduce, we shall argue that it is only abstract entities such as types, genotypes and alleles that can be fit. The term "allele" is used in contemporary genetics to refer both to *types* of genes and to *genes* of a particular type, that is, to the concrete representations of the type ("tokens" or "particular alleles"). We shall be using the term to refer to the abstract entity—the *type*.

We shall first show that it makes no biological sense to ascribe fitness to individuals, then argue that it also makes no philosophical sense, and finally define fitness in terms of types.

There seems to be no doubt that the fitness of an individual (should there be such a thing) cannot *actually* be *measured* or determined in any way on the basis of the individual's own actual reproductive fate; it can only be determined by taking the average success of a large sample of individuals of the same type. But it is thought that individual fitness can at least be *defined* or *conceived* independently of the fitness of types. One currently popular approach, which attempts to retain the ascription of fitness to individuals, defines fitness as a *propensity* of individuals for reproductive success. Mills and Beatty (1979, p. 275) define individual fitness as: "the [individual] *fitness* . . . of an organism x in environment E equals $n \equiv_{df}$ is the expected number of descendants which x will leave in E". However, a propensity can be ascribed to an individual only as an instance of a type and only on the basis of an analysis of the actual (or projected) reproductive success of a number of individuals that instantiate that type. In other words this approach implicitly uses types without explicitly mentioning them. Thus, whatever the categorial status of fitness and individuals, the empirical meaning of the "propensity fitness" of individuals is still conceptually dependent on the fitness of types.

However, individuals are not the proper entities for inspection in biology. If we actually attempt to conceive the fitness of an individual in terms of its (expected) number of descendants, we either implicitly treat

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interests us, nonetheless the term always refers to a *type* of physical entity (which carries the information) not to such an entity itself.

the individual as instantiation of a type (compare Mills and Beatty 1979) or we reduce descent to a purely formal relationship. If we say that the descendants must share some traits or genes with their ancestors in order to be counted towards its reproductive success (that is, that they be "sufficiently similar"), we assert that they must belong to the same type. If we don't implicitly appeal to types, then we take descent as a purely formal relationship: an organism remains the ancestor of its descendants even if all its genes have been shuffled out by natural selection. (For example, a brown-eyed individual may be the ancestor of an entirely blue-eyed population of individuals in some future generation.) The same argument can be extended to include all the hereditary traits of the ancestor, which none of the descendants may share. In such a case, the use of the future population to define the fitness of an *individual* ancestor makes no biological sense, since it is obvious that there are no common traits shared between the ancestor and its descendants. In fact *natural selection* has actually eliminated all the ancestor's traits. There is clearly a difference between formal descent and physical heredity and between ancestorship and genetic contribution. This argument would still hold even if we knew in intimate detail all the relevant causal factors and could distinguish precisely between "selective" and "accidental" elements. What biologists actually always do is to determine the fitness of types of organisms (for example, a brown-eyed type) by considering the numbers of individuals representing the types in later generations.

As we pointed out above, Fisher defined fitness in terms of genotypes, that is, types of allelic combinations. The philosophical question that arises is whether an individual which is an instance of a "fit" type can itself sensibly be said to have a fitness value. While it would obviously be a category mistake to attribute physical properties to an abstract entity (the *kingdom* of plants does not photosynthesize, the *class* of lions is not dangerous, a *type* of ring-tailed monkeys does not itself have a tail), it is not so apparent that the properties of types should not be ascribed to the individuals that instantiate them.

That some problems can occur when extrapolating from types to individuals can be made plausible with the following "Fisherian" example. According to our insurance company a white male American academic (type X) has a life expectancy of, say, 75 years. We say then that John, our colleague down the hall, has a life expectancy of 75 years if he instantiates this type. But, again according to our insurance company, an Irish Catholic polo player (type Y) has a life expectancy of only 69 years, and since John instantiates type Y as well, he has two incompatible life expectancies. Thus, it is clear that an *organism which is an instance of a type that has a particular fitness value cannot be said to have that fitness value.*

But does this apply to identical twins? Can we not say that in this case the *type* is comprehensive and therefore that an organism has the same fitness value as its *aggregate genotype* or that a particular piece of nucleic acid (token) has the fitness of the allele (type) that it instantiates?

Now, there is no question that the fitness of a type is somehow dependent on ("a function of", "derivative from", etc.) the properties of individual organisms: long necks, sharp horns, crooked beaks, etc. The question is: What sense does it make (what harm does it do) to say that this *type* of giraffe is fitter than that type *because the individual giraffes of the type are fitter,* (and these are fitter, say, because they have longer necks, which on the average effect a reproductive advantage). It is clear that we could *dispense* with the fitness of individuals and explain the fitness of the type directly by the long necks of the individuals, but there seems no immediate harm in it.

It may be revealing to take an example in which it intuitively seems to make sense to talk this way. A particular type of book binding is said to be stronger than another type because the bindings of the individual books of the types are differentially stronger.[6] Although we could here, too, ignore the strength of the individual bindings and refer directly to the various materials that make them strong, it is the fact that the individual bindings are strong that allows us to speak of a strong type of binding. However, imagine we were to say, "this binding is strong" only to have it fall apart in our hands; we would point out that, even under the best of circumstances and with the most rigorous quality control, accidental defects in the materials could mean that some instances of the type are not particularly strong. The relative strength of the type is compatible with the weakness of a token. However this entire analogy suggests that two individuals representing the same type (for example, identical twins) can have *different* fitness values just as they could have different "strength values". Not only is this empirically meaningless in the case of fitness as we have shown above, but it also contradicts the twins argument, which disposed of the tautology charge by assuming that identical twins must be *equally fit*.

The problem that comes to light here seems to lie in the use of the word "type". Whether or not a book binding is strong can *in principle* be ascertained by examining its material interactions with other bodies; the fitness of an individual organism cannot *in principle* be so determined. Furthermore, when we speak of "a strong type of binding" we don't actually mean that the *type* itself is strong any more than we mean that the *class* of lions is dangerous; "strong type" is just a way of referring to a *type of strong bindings*. But when Fisher says a *type* of organisms (type

[6]This comparison was actually suggested by an anonymous referee for this journal.

of allelic combination) is fitter, he means a fitter type of entity, not a type of fitter entities. It is the *pattern* of material entities that is said to be fit, not any particular system of such entities. Thus, it seems quite possible that some philosophers, when considering (and agreeing with) Fisher's definition of the fitness of types, might have assumed that "type" does not refer to an abstract entity instantiated by individuals but is just a collective name for the fit individuals themselves. But this is not the way "type" is actually used in biology, and it seems to us mistaken to substitute a colloquial figure of speech for a scientific concept.

Unlike individuals, types are abstract entities represented in the original generation whose representatives can actually be counted and their frequency compared over many generations. It is also possible to assign fitness to other abstract entities such as alleles and genotypes in a non-ambiguous manner. Fitness is assigned in such cases to specified alleles or allelic combinations, whose quantitative representation in the following generations can be measured.

These arguments lead us to define "fitness" as fitness of types:

> Type X is fitter than type Y in environment e if and only if the organisms representing X are on the average reproductively more successful in environment e than those representing Y and if, furthermore, we can infer a causal relationship between the relevant hereditary physical properties of the individuals representing the types and the differences in reproductive success.

There is, however, one fundamental difference between assigning fitness solely to alleles, and assigning it to genotypes, that is, to allelic combinations. In the latter case, fitness (which we might call genotypic fitness) incorporates part of the genetic context into the characterization of the entity that is fit. That is because, in a sense, the genotype (the allelic combination) is one of the determinants of the phenotype of an allele, that is, the expression of the allele in its genetic environment or background.[7] It is of course clear that every allele has both genetic and environmental context-dependent properties which can either reduce or enhance its selection coefficient[8] and which reflect the transfer of its genetic information to the phenotypic level. Such context-dependent properties can be causally relevant to the reproductive success of individuals. The discovery of the context-dependent properties of an allele is part of a research program carried out to decide which of two alternative alleles at a partiuclar locus is the fitter. Such a research program must distinguish

[7]On the concept of genetic background see Mayr (1970, p. 164).

[8]The selection coefficient gives a quantitative measure of the relative severity of selection on a particular type. It is ascertained by means of the proportion between the changes in frequencies of types in a population over time.

between allele frequencies changes due to adaptations and changes of allele frequencies due to random genetic drift and hitchhiking effects. The analysis of the relation of the frequencies of the alleles to their context-dependent properties allows, we believe, a conceptualization of fitness which avoids the tautology problem by making clear that fitness and reproductive success are assigned to different subjects. The assignment of fitness to genotypes, that is, to types of functioning allelic combinations, although legitimate, can be conceptually confusing because it skips a part of the causal chain. This can be demonstrated on the example of heterozygote superiority, where it seems at first obvious that the genotype should be the subject of fitness (Sober and Lewontin 1982). If we say that the genotype *Aa* is fitter than genotype *aa* or *AA*, we are implying that there is a physiologically significant and reproductively relevant interaction involving the gene products of the alleles. The physiological effect of an allele is context-dependent and thus the presence of another allele may be causally relevant to the connection of the fitness of that allele to the reproductive success of the individuals bearing it. With heterozygous superiority this is precisely the case—it shows how the presence of one gene changes the effects of another gene. In other words, by assigning fitness to genotypes we are already presupposing a part of the causal chain without spelling it out. For this reason, the ascription of fitness to alleles as starting points instead of to genotypes is more appropriate for formulating the general rule. Of course, an analysis of the allele level alone contains less information than the analysis of allele and genotype, or of allele, genotype and phenotype. But we argue that for logical reasons it is preferable to start by assigning fitness to single alleles.

The assignment of fitness to a single allele may seem like the adoption of Dawkins' "selfish gene" hypothesis (Dawkins 1976). We want to stress that despite the obvious similarities there is a fundamental difference between our approach and that of Dawkins. The part that causality plays in our definitions of fitness and adaptation demands that we follow the causal chain from allele to phenotype since selection leading to adaptation operates on the level of traits of individuals. Our claim that fitness is best ascribed to alleles does not, therefore, mean that the allele is "the unit of selection".

3. Causal Connections. To solve the "tautology" problem, it is not enough merely to attempt to show that the relation between fitness and actual reproductive success is causal instead of logical, that is, that survival of the fittest expresses a causal relationship. We must further show that the causal assertion has empirical content. To say that a body is accelerated by gravitational interaction with the earth is empirically meaningful; but

to say that it accelerates because the vector sum of all forces acting upon it is different from zero is true but trivially so. In order to show an empirically meaningful causal relationship, we need to link the organismic level (along with its relationships to the environment) to the allele level, namely, to link the adaptations (of individuals) to the frequency changes of allele (types).

In any biological experiment the concrete systems actually studied are taken as representatives of particular types of systems. In any experimental modeling of natural selection, whether by Darwin's breeders or by modern population geneticists, the types used are not defined by the sum total of reproductively relevant traits but rather by some particular trait which interests us and is supposed to be or is made to be causally decisive. The systems are not taken to be fitter in the strict sense described above; rather it is *hypothesized* that some particular phenotypic trait (assumed to have the same genetic basis) by which two types can be distinguished is causally responsible for differences in the reproductive success of the individuals representing them, and the experiment is tried. This is the main point of the responses in Ruse (1973) and Mayr (1982) to the tautology charge, responses which point to the kinds of empirical work actually done in evolutionary biology.

Sober (1984, pp. 97–102) points out that not every product of natural selection is an adaptation. With a view to the "hitchhiking effect", he makes a distinction between "selection of" (individuals) and "selection for" (traits). For instance, if green organisms happen also as a rule to be small, then the number of small organisms will increase if green individuals are selected. But if only the possession of greenness has been causally relevant, then only greenness can be considered an adaptation. "Selection of" individuals is an inevitable consequence of any selection process, and expresses a mere correlation between changes in allele frequencies, traits, types, etc., and the process of selection. The existence of a correlation is no proof that the possession of a given trait (for example, smallness) is or has been a cause of reproductive success. To claim that a trait is an adaptation, one must demonstrate that the correlation has been a causal one: only then can one say that selection of these individuals has been "selection for" this trait.

The context dependent properties of an allele also link the definition of fitness suggested by us to that of the process of adaptation, since adaptation can be understood only on the level of the functioning of interacting allelic systems (expressed ultimately as "traits"). However, this link is not trivial, nor does it follow automatically from our definitions of adaptation and fitness. It is possible for a certain allele to be selectively neutral (that is, to have no effect on the reproductive success of its bearers as compared to an alternative allele at the same locus) and yet to be adap-

tively significant in the long run, precisely because of its context-dependent properties.

There are still very few cases where the causal relationship between the presence of a particular allele and the reproductive success of its bearers can be demonstrated forcefully as, for example, in the case of the spread of antibiotic resistance in hospital populations of bacteria. There are, however, many cases in which an increase in the frequency of a particular allele is indeed correlated with higher reproductive success of the individuals bearing the gene, but in which it has been proved convincingly that the presence of this gene is not causally efficient in the individuals' relative reproductive success. The claim that the correlation between allele frequency changes and selective processes is a causal one raises empirical questions that can be answered only by observation and experimentation. Only by relating the ecological constraints of the environment to the action of different alleles, can we connect the adaptations of individuals to the fitness of alleles and convincingly claim that certain adapted and still adaptive traits, which are the physiological effects of particular alleles in certain environments, are the evolutionary causes for differences in reproductive success of individuals representing the types.

Evolutionary experiments have been performed in populations of self-replicating molecules, phages and bacteria (as, for example, Eigen and Schuster 1977; Chao et al. 1983). Such experiments make use of the relative simplicity, the known genetic structure, and the very short generation time of these systems, to test different evolutionary hypotheses about the relative importance in the evolutionary process of mutation rates, population sizes, recombination, allele polymorphism, selection pressures, etc. Recent advances in biology make it feasible to manipulate in a controlled and precise manner the genetic constitution of organisms and thus to test directly the effects of these alterations on interactions with aspects of the environment. Such manipulations are now also possible in multicellular organisms such as Drosophila, maize, nematode and many others, and therefore the claim, that the scope of evolutionary experimentation is too limited is outdated.

The construction of experiments with isolated systems in controlled environments is common practice in all experimental science. However, it can be argued that, since evolutionary biology is by definition a science of complex entities with a tangle of interactions on many levels, the modeling of evolutionary processes by laboratory experiments can reveal at most only trivial regularities and will miss essential aspects of complex interactions. Because biologists are well aware of this problem, the basic approach of most evolutionary research is to combine engineering modeling with detailed genetic analysis. In most cases engineering modeling

(which takes into account the relevant environmental constraints) fails to demonstrate causal relationships. However, it suggests ways of constructing experiments to test the involvement of the relevant genetic components in the adaptations studied. Engineering optimization models can be applied successfully, however, only to a rather narrow subclass of adaptations, mainly adaptations that have resulted from a long period of directional selection and therefore demonstrate very little variation. Traits like the aerodynamic structure of the bird wing or the structure of the eye yield to optimization criteria. Most traits, however, are a result of the compromise between many different selection pressures, and therefore it is very difficult to determine what is optimal.

One of the problems of assigning fitness (as defined above) to the alleles of interest is that selection coefficients of particular alleles are usually very small, but the assertion of causal relationships is most convincing when selection coefficients are high. It is no coincidence that there are very few cases among higher organisms (the most famous of which is "industrial melanism" (Kettelwell 1973)) in which the causal relationship has been demonstrated to the satisfaction of the majority of biologists (for a dissenting view see Lambert et al. 1986). Despite this difficulty it is possible to establish such a relationship by combining an engineering model (however limited), field observations on environmental constraints, and a detailed molecular analysis. Such a research program can be performed starting from any of these three specified approaches.

Part of the reason for the low selection coefficient of alleles is due to their being part of an intricate complex of genetic elements, the very complexity of which usually masks the effects of any particular allele. It is however possible to select for a trait whose continuous manifestation depends on a specific combination of genes and then to introduce a change in a single allele within this particular combination and study the effects. Such an approach can enable us to distinguish experimentally between the genetic background and the external environment (Waddington 1975). An adequate research program should therefore include a theoretical optimization model of the trait of interest, field observations and detailed genetic analyses which take into account the effects of the genetic background.

4. Some Biological Implications. The aim of the analysis above was to distinguish clearly between adaptation, fitness and reproductive success and to indicate that besides logical and conceptual problems there are problems to be solved by empirical research. We have tried to show that reproductive success refers to individuals within a population and that it could be due either to adapted traits or to other factors. Fitness refers to

types, and its value is always the result of differences in the physical properties of reproducing individuals representing the type. But while fitness always affects adaptations, not all adaptations must affect fitness differences.

This point can be relevant to the neutralist/selectionist debate in evolutionary biology. A major issue in this debate is the relative weight of random genetic drift and selective processes in shaping the genome. It is possible that many presently selectively equivalent (that is, neutral) alleles are in fact parts of adaptive complexes of genes. Our analysis suggests a distinction between two categories of neutral alleles: (a) Adaptively neutral alleles—alleles that do not affect the phenotype either because their sole phenotypic expression is a negligible copying cost or because their function is exclusively structural. (b) Selectively neutral alleles—alleles whose representatives participate in adapted complexes of alleles but from the point of view of natural selection are indistinguishable from some other alleles (Ettinger 1986). Examples for adaptively neutral alleles may be—selfish DNA (which, by definition has no phenotypic expression except for the cost of copying (Doolittle and Sapienza 1980, Orgel and Crick 1980)); or DNA whose sole function is structural (for example, the maintenance of the basic architecture of the chromosome).[9] In the latter case many changes in base sequence do not affect the structure and therefore are adaptively equivalent. Selectively neutral alleles are those alleles which in some ranges of environments or some genetic backgrounds can become selectively meaningful (such alleles were referred to by King 1984, Hartl et al. 1985).

Although Kimura, one of the main proponents of the neutralist theory explicitly states that selectively neutral alleles are merely selectively equivalent alleles and may become selectively meaningful in the future (Kimura 1983, pp. 34, 51), he actually treats them as *adaptively* neutral alleles. In his calculations of the rate of fixation of neutral alleles he assumes that they remain neutral for long evolutionary periods. If, however, some neutral alleles alternate between periods of neutrality and periods of "selectibility" such alleles may have a fixation rate different from that predicted by the neutralists. In this case even if most protein polymorphisms are "just a phase in molecular evolution" as the neutralists claim, it is still possible for most protein polymorphisms to be visible to natural selection.

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[9]Even adaptively neutral alleles can be significant in evolution but not on the level of adaptation. For example, repetitive DNA which is apparently selfish DNA may play a part in speciation (Dover 1982).

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