

Adaptation and Functional Explanation in Biology

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ABSTRACT: The evolution of adaptation by natural selection is held to legitimize functional explanations in biology. It cannot do so, not only because populations change in the absence of natural selection, but also because the maximization of mean population fitness is merely a restatement of such selection and has no additional empirical consequences. Thus natural selection is not such as to necessarily maximize any population parameter even if such a parameter is defined so as to encompass those environmental specifications embodied in the term "ecological niche." The process of evolution by natural selection provides no basis for the legitimacy of functional explanations in biology.

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Explanations in biology are of two kinds, causal and functional. Causal explanations (which are also used in the physical sciences) explain the state of a system by reference to prior states and to scientific process laws predicting future states from prior states (Hull 1974). Functional explanations (which have also been called Darwinian or compositionist (Dobzhansky 1968)) are of a different kind and explain form in terms of its purpose or function to the organism. They are thus implicitly teleological in that they make no reference to prior states. In science teleological explanations are not normally legitimate but it is held that in biology the process of evolution by natural selection gives to living organisms a property which is such that functional explanations are translatable into causal explanations. Such a translation would consist of a demonstration that the observed state forms a stable equilibrium in a dynamic model of any possible historical process leading to the present situation (Lewontin 1969). The name usually used for this property is adaptation.

Nevertheless, doubt exists as to the operational meaning of adaptation. Lewontin (1957) states, "There is virtually universal disagreement among students of evolution as to the meaning of adaptation", and Ehrlich and Holm (1963) conclude: "Because of the extremely loose application of the term adaptation in the biological literature, it might be wise to drop it completely." Some authors define the adaptation of an organism to its environment as a necessary (and, by implication, sufficient) condition for existence. Wallace and Srb (1964), for example, say "Imagine a species..... that is not adapted to its environment. By definition it could not exist", and more recently Dobzhansky, Ayala, Stebbins and Valentine (1977) say "The structure, function and behaviour of every species are adapted to its particular mode of life. In a way, this statement is a truism, for an organism lacking adaptiveness would not be alive."

This all-or-none meaning of adaptation is inconsistent with the more general use of the term as a continuous variable. As such, a criterion for assessing the adaptation of a population is suggested by Thoday (1953, 1958) and, in a modified form, by Slobodkin (1964, Slobodkin and Rapoport 1974). Thoday suggest that population adaptation could be measured by the probability of survival for around 10^8 years, and Slobodkin and Rapoport suggest that the probability of survival is what will be maximized by evolution, rather than any external pay-off to the species (in the games theory sense). The problems with such a criterion are two-fold. Firstly, as the authors realize, it is hard to imagine a way of measuring the probability of a species survival in future environments, and secondly, there

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seems to be no direct way in which natural selection will act to bring about the maximization of this probability.

Other authors, (for example Fisher 1930) however, while still regarding adaptation as a relative term, define it such that genetic types can be described as being better or less well adapted than others, adaptation here having the same meaning as genetic fitness. It is held (Stern 1970) that such an adaptation concept can be meaningfully applied to monomorphic populations. The main problem with such a concept is the difficulty of reconciling the observed existence of directional natural selection in wild populations (Georgiou 1972, Bradshaw, McNeilly and Gregory 1965, Kettlewell 1973) (and the, generally speaking, necessarily consequential increase in mean population fitness and thus adaptation) with the inability of functional explanations based on a criterion of optimality though to be derived necessarily from natural selection to either predict observed form (Lewontin 1978) or, by this inaccuracy, retrospectively refute the natural selection theory. That is to say, if the increase in mean population fitness in population genetics models is to justify the functional explanation of form, then a criterion for the acceptability of only one out of a range of conceivable functional explanations (such as those mentioned by Lewontin (1978) must be derivable from such models. The thoughtful response of Maynard Smith (1978a) that the empirical testing of functional explanation tests not the optimality of form but the nature of optimality in a given case indicates that such a derivation has not been possible. In this review, I propose to show that such a derivation is absolutely required before we can produce rigorous functional explanations (see McCleery 1978) which can be translated into causal explanations and that the possibility of such a derivation is not commensurate with what is known of evolutionary biology.

Integral to the idea that adaptation will increase with time is the assumption that the spread of alleles through populations takes place by natural selection. Of course, it is possible that even in large populations selectively neutral alleles may spread by genetic drift, but the neutrality of such gene frequency changes is probably due to the phenotypic equivalence of alleles. Thus genetic drift should not, in large populations, cause genetically determined phenotype changes. Processes which can cause such changes are not, however, limited to selective ones. Meiotic drive is well-documented in the *t*-locus of the mouse (Bennett 1975), the segregation distorter locus in *Drosophila melanogaster* (Peacock and Miklos 1973), the X-chromosome-borne "sex ratio" genes in *D. affinis* (Novitski 1947, Voelker 1972), *D. pseudoobscura* (Policansky 1974) and *D. melanogaster* (Peacock, Miklos and Goodchild 1975), and the rarer male-producing Y-chromosome "sex ratio" genes found naturally in *Aedes aegyptii* (Newton, Wood and Southern 1976) and experimentally produced in *Drosophila melanogaster* (Lyttle 1977). Further genes which are inherited other than in accordance with Mendel's rules are known in *Nicotiana tabacum* (Cameron and Moav 1957), *Triticum aestivum* (Loeagering and Sears 1963), *Neurospora* species (Raju 1979, Turner and Perkins 1979), *Zea mays* (Rhoades 1952), and *Lycopersion esculentum* (Rick 1970). In some fungi, such as *Ascobolus immersus*, gene conversion can produce effective selection coefficient (as measured by twice the proportional excess of wild-type spores over white spores of an experimentally-produced mutant allele in crosses) of 0.04 in some genetic backgrounds (Lamb and Chikas 1979) and thus gene conversion could be an important factor in causing gene frequency changes (Gutz and Leslie 1976).

The number of B chromosomes per cell is increased in germ line cells of *Crepis capillaris* by polarized non-disjunction in shoot meristems, and in a number of other plants, such as *Aegilops speltoides*, *Alopecurus pratensis*, *Secale cereale* and *Sorghum purpureo-sericeum* accumulation occurs by directed germ cell nondisjunction. Accumulation by other processes occurs in *Lilium callosum*, *Tradescantia virginiana*, *Phleum nodosum* and *Zea mays* (Rees and Jones 1977). In many Orthopterans B chromosomes are known, and can again show accumulation in transmission (Hewitt

1973). Recent papers by Orgel and Crick (1980) and Doolittle and Sapienza (1980) raise the possibility of the spread by transposition (in both prokaryotes and eukaryotes (Calos and Miller 1980) of genes causing a selective disadvantage to their carriers.

The fact that such a list of non-selective causes of genetic change can be made indicates, however, the rarity of such cases. If they are discounted it can be said that alleles certainly become more common as a result of their selective advantages over other alleles. The necessary consequence of this is that in single-locus models with fitnesses independent of genotypic frequencies, mean population fitness increases to a maximum, which is either global in simple heterozygote advantage models (Li 1955) or local in more complicated models (Wright 1970).

This increase in mean population fitness and thus adaptation cannot, however, legitimately be expected to cause populations to be optimally adapted. Ludicrously extreme optimal phenotypes, possessing for example, immortality or infinite reproductive rates, do not exist. Such adaptations cannot arise through natural selection as a result of the inavailability of appropriate mutations (Maynard Smith 1978a). Adaptation will arise only to the extent that the occurrence of mutations is not limiting. As pointed out by Lewontin (1979), however, the prediction of the spread of selectively advantageous alleles produced by mutation (and thus the increase in mean fitness) is just a formal analytic restatement of such selective advantages. The specification of the available genotypes along with their selective characteristics is not just a prerequisite for the prediction of evolutionary change. It is the entire synthetic content of the theory.

It is this problem which is the basis of the oft-repeated assertion of the untestability of the theory of natural selection (Popper 1974, Brady 1979). That individuals which survive and reproduce are fitter than those which do not is tautological and thus cannot predict, or (following Hempel (1965)) explain anything (unless this assertion is taken to imply that selective coefficients are constant (Hairston 1979, Williams 1970: Axiom D5)). However, observing whether or not natural populations are characterized by selectively important genetic variation is a perfectly valid scientific endeavour which renders unnecessary defenses that the tautological component of natural selection is nevertheless useful. These include that of Waddington (1960) who states: "Natural selection states that the fittest individuals in a population (defined as those which leave the most offspring) will leave the most offspring. Once the statement is made, its truth is apparent. This fact in no way reduces the magnitude of Darwin's achievement; only after it was clearly formulated, could biologists realize the enormous power of the principle as a weapon of explanation", a sentiment echoed by Mayr (1961): "One of the most important contributions to philosophy made by the evolutionary theory is that it has demonstrated the independence of explanation and prediction", and in a modified form by Lewontin (1972): "Even though natural selection might not be an epistemologically satisfactory hypothesis, it might nevertheless be true."

The consequence of the necessary nature of the selective increase in mean fitness is that the theory of natural selection has absolutely no formal consequences for the natures of the phenotypes resulting from such a process. The search for a parameter to be called adaptation which most accurately represents genetic fitness (see Stern 1970) must be fruitless. This can be illustrated by considering cases where the types of parameters which might be thought to be such measurements will not be increased by natural selection. It must be remembered that the mean relative genetic fitness of any genetically invariable population, including one becoming extinct, is defined as having the maximum value of one. The mean genetic fitness of a monomorphic population is not a measurement. It cannot represent population adaptation nor can any putative measurement of population adaptation represent it.

Consider a population containing both sexual and apomictic (ameiotic) parthenogens. A sexual female will, assuming a one-to-one sex ratio, anisogamy and

no parental care, produce half as many female offspring as a parthenogenetic female with equal fecundity (Maynard Smith 1971). The consequence will be that parthenogenesis will spread. However, a sexual population with a sex ratio heavily biased in favor of females and with a slight viability or fecundity advantage might outcompete a parthenogenetic population. In such a situation, the expected evolution of the equilibration of the sex ratio (Fisher 1930) would cause a diminution of the fitness of the sexual population in which it occurs.

A gene for aggressive behavior (giving rise to Maynard Smith and Price's (1973) "Hawk" strategy) can invade a population of non-aggressive individuals (showing the "Dove" strategy) even though the number of injuries to members of the population by violence will be thereby increased. Sexual selection is the name given to the process whereby a structure is selectively maintained in one sex by the second sex making the presence of the structure a prerequisite for mating (Fisher 1930). The choice is "self-reinforcing" (Maynard Smith 1978b) due to the low fitness of offspring of the first sex of an individual of the second sex which fails to make such a choice, as they will lack the structure. Provided that it is in some way manifest to the opposite sex, the nature of the structure referred to is irrelevant to the adequacy of the argument. The occurrence of sexual selection produces no consistent phenotypic effect other than a lack of agreement between observed phenotype and the maximization of some parameter thought to be maximized by other forms of selection.

This argument, of course, relies on the advantageousness of an allele in one sex being dependent upon the fixation or near-fixation of an allele favoring it in the other sex, which itself is favored by the near-fixation of the first allele. These advantages stably maintain the population in a state where its fitness is lower than if both alleles were absent. This being so, a formally analogous argument can be seen to apply even if the genes being considered act in the same sex, in which case its applicability, however, is so widespread that it is hardly even interesting, for from the obvious assertion that the selection acting on a locus is dependent upon the genetic background, it follows that it is possible that an observed genotype may form a "selective peak" (Wright 1970) (and thus be stable to gene frequency changes at individual loci) and yet have a considerably lower fitness than a genotype differing from it by simultaneous substitutions at a number of such loci. The consequence of this is that demonstrations that aberrant individuals differing from the population mean in some parameter (such as clutch size (Perrins 1964, Lack 1948, Lack, Gibb and Owen 1957)) have a lower fitness than more typical individuals do not demonstrate the cause of the mean being what it is, for the aberrant individuals will lack other genomic modification which, if present, could increase their fitness beyond that of typical individuals.

There remains the concept that there is an important (Cain and Sheppard 1954, 1956) and potentially measurable property of populations which might be called population fitness. Some authors have compared the fitnesses of populations by regarding various measurements, such as productivity, or biomass increase (Carson 1958, 1961a, 1961b, Smathers 1961, Battaglia and Smith 1961, Strickberger 1963a, Bert 1960), maximum specific rate of increase or $r_{\max}$ (Dobzhansky, Lewontin and Pavlovsky 1964, Birch, Dobzhansky, Elliott and Lewontin 1963), carrying capacity (Ayala 1965, 1966a, 1966b, 1968a, 1968b) fecundity (Buzzati-Traverso 1955, Wallace 1959), viability (Beardmore, Dobzhansky and Pavlovsky 1960, Beardmore and Levine 1963, Beardmore 1963), efficiency of energy utilization (Bock and Wahlert 1965) or control (Van Valen 1976, 1977) and competition to another species (Claringbold and Barker 1961, Barker 1963, 1965, Strickberger 1963b) or strain (Knight and Robertson 1957) as representing population fitness.

A problem with using population characteristics such as biomass or density as synonymous with fitness is the tremendous interspecific variation in these characters, which permits interspecific comparison of adaptation. The adoption of such a criterion should, in theory, make meaningful a ludicrous comparison between the

population fitness of an oak tree, a mole and *E. coli*, for example. Such a comparison would be ludicrous because the measurement of adaptation adopted would be strongly correlated with the obvious phenotypic differences between the species considered, such as size and longevity differences. However, even if it was true that accurate evolutionary predictions for one species could be made from a theory based on the maximization of any such measurement within that species, it could not be inferred that maximization of the same parameter occurred in a second species, if the second species was capable of living and competing successfully with a greatly different mean value of the parameter. One way to illegitimize such interspecific comparisons is to define the adaptation of a species in terms of its ecological niche (Lewontin 1978). The niche provides a sufficient condition for the existence of the species occupying it and defines an optimum phenotype towards which the species will evolve. This latter point can be translated into systems theory terms by saying that the niche defines a morphogenetic attractor towards which the species (defined by a series of state variables) will move (Bremermann 1973). Lewontin (1978) says that adaptation will only come about if phenotypic variables possess two properties; continuity, which means that small phenotypic changes do not radically change the niche to which the species is best adapted, and quasi-independence, which means that changes in one phenotypic variable do not necessarily produce changes in others. Taken together and translated into morphogenetic terms, these properties specify that a species can move unhindered towards an attractor.

The epistemological difficulty with a definition of adaptation based on the niche is the practical difficulty of detecting which niches exist and which optimal phenotypes they specify without reference to the species occupying them. Simply to read off the available niches in a one-to-one way from observed species is inadequate as the very legitimacy of this procedure relies on the adaptation which is yet to be demonstrated. This procedure could only usefully be attempted if the theoretical grounds for assuming near-perfect adaptation of species to their environment were strong.

They are not. Firstly, as pointed out by Cohen (1973), in changing environments species may well be unable to track by evolutionary changes such environmental changes. Secondly, and more importantly, there is the problem of evolutionary constraints (Gould and Lewontin 1979, Maynard Smith 1978a). A constraint on evolution is essentially some phenotypic parameter which is not variable by the evolutionary process in the time period being considered. Some constraints will be integral to the adaptation argument, being definitions of the niche in which the adaptation is occurring, but others, such as the properties of biological materials, for example, cannot be viewed in this light. If such information was to be included in niche specification this would preclude the demonstration of a constraint at all as the absence of a characteristic would not demonstrate that its existence was prevented by a constraint, but merely the absence of a niche in which it is required. The widespread occurrence of obvious evolutionary constraints, therefore, reveals that species do not conform to the optimal phenotypes for their niches, as such phenotypes cannot be achieved without transgressing such constraints.

Another difficulty with a concept of adaptation as the agreement between a species phenotype and the demands of its ecological niche is that the niche available for a species will be dependent upon the optimal or realized phenotypes of other species of the community. If predators, for example, are optimally adapted they will be such as to capture most efficiently the prey species available in their community. However, such prey species cannot be optimally adapted if they show the phenotype to which their coexisting predator species are best suited. This argument also applies to plant-herbivore and host-parasite relationships and demonstrates the impossibility of constructing communities of optimally adapted species which interact in the observed ways. The argument of the "Arms Race" of Dawkins and Krebs (1979) that predator-prey interactions will produce unidirectional

changes in each lineage does not follow from the presupposition of adaptive change.

Adaptation has been defined such that the adaptation of a species to its environment is a necessary consequence of its existence. Such all-or-none adaptation cannot arise through natural selection as the species must exist throughout the selective process. Nevertheless, even with a continuous scale of adaptation based on a parameter thought to represent genetic fitness it is frequently assumed that the probability of the survival of the species increases with adaptation. However, problems arise even with this seemingly innocent assumption. A considerable literature has been built up in attempts to solve the problem of the "prudent predator" (Slobodkin 1968). A predator genetically determined to eat a disproportionate amount of the species' food organisms would have a high fitness and its genes would spread through the population and possibly cause extinction of the prey and thus consequently the predator also. More generally, Roughgarden (1977, 1979) has shown theoretically that evolution of one species in a community can cause that species to increase its chance of becoming extinct. Natural selection simply will not systematically increase the chances of existence of a population in which it takes place and it would be wrong to define adaptation such that such maximization is regarded as being axiomatic.

"Adaptation" has thus been used to mean the existence of a population, the probability of its future existence, its mean relative population fitness, its adherence to the demands of an ecological niche itself read off from the populations phenotype, and a variety of population characteristics (such as biomass) or individual characteristics, (such as r_m). All of these meanings are logically distinct. Whether some or all of them are factually equivalent, or at least correlated, is an empirical question, the answer to which will be different in each case. The danger implicit in the variation in use of the term "adaptation" is that such distinct concepts may be thought to be related. What will be true is that different populations will have different stable densities in wild communities. The stabilities of these densities will be caused by density-dependent regulating factors. These will differ between populations. In other words, the populations will occupy different niches. Within each population evolutionary changes will occur which will, if fitnesses are frequency-independent and epistasis lacking, increase the mean population fitness. The effect of such changes on the size and stability of the population is an empirical matter which cannot be simply deduced from the fitness increase. Similarly, the direction and extent of evolutionary change within a population cannot be predicted from a knowledge of the density-dependent regulating factors acting upon such a population. The discovery of the relationship between the evolution of communities and the evolution of the species which they contain must be the major goal of the study of the evolutionary process. The use of the term "adaptation" for unrelated concepts belonging to these separate domains obfuscates the relevant questions which need to be addressed.

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