

The Bases of Conflict in Biological Explanation

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I believe that a large part of the problem of biological explanation is sociological rather than philosophical, for the renewed interest in the problem of explanation in biology is a result of the rise of a self-conscious and self-defined group of biologists who call themselves molecular biologists. This group feels, and explicitly states, that the reason biological explanation is in such a bad state is that biologists have by and large not asked the right kinds of questions and not used the right methods. There has been a setting up of a kind of dialectic of the molecular versus the evolutionary, the molecular versus the organismic, the reductionist as opposed to the synthetic theories of biology, a dialectic that is pretty much a false one. I would like to distinguish, from the standpoint of the biologists, wherein this conflict is a real one and wherein it is simply a sociological consequence of the excitement that some people have about what they do.

There is first the false conflict, which is really a conflict of *interest*. The difficulty has arisen in the context of everyday language because the two kinds of biologists have asked what seemed to be the same questions in the same way, but have really been asking different questions. They have all used the word "how," but there is a confusion of final and efficient causation, a confusion that biologists must be permitted because most of them have never heard of Aristotle and those who have heard of him have never read him. The confusion between final and efficient causation among biologists is contained in the following exemplar: "Why is the sex ratio in *Drosophila* 1:1?" It is a question typical of the biologist, for it is really two different questions even though it is stated as one. "Why is the sex ratio 1:1?" I can give a perfectly satisfactory answer to that question by discussing laws of genetics; by discussing chromosomal mechanics, which themselves are clearly reducible to physicochemical terms, although that has not yet been properly done; by reducing the explana-

tion to a series of statements about the chemistry of cells and about the chemical nature of genes. That is, I can answer this question by talking about the formation of gametes, and how it is that sex is determined by chromosomes. For the molecular biologist, that is the answer of interest to the question: "Why is the sex ratio 1:1?" For the molecular biologist an answer which is not framed in those terms is not very interesting; it does not say what one ought to say about the world.

There is a different answer to the question, "Why is the sex ratio 1:1?", and that is to explain why it is 1:1 in *Drosophila* but 1:2 in *Habrobracon*. We are not here interested in the question of the mechanism of the determination of the particular sex ratio in a particular organism, but rather in asking why there is a difference between two organisms in their sex ratio. We will not accept the answer that the mechanics of chromosomes are different in the two organisms; we will only accept an answer framed in terms of a functionalist or adaptive or evolutionary viewpoint. It will be an explanation that attempts to say that it is sensible that *Habrobracon* should have a 1:2 sex ratio while *Drosophila* should have a 1:1 sex ratio. The various ways in which this answer is given will be discussed later in this paper. Here it should be emphasized that one of the problems in biological explanation arises from the fact that there are two groups of biologists asking a question like "why is the sex ratio 1:1?" and satisfied with two different kinds of answers because they are really interested in two different kinds of questions. Efraim Racker recently asked me what I was doing in my laboratory. I began to tell him, and as time went on it became clear to me that he really thought what I was doing was very dull. It was not that he thought it was not well done, but rather that I was not asking anything very interesting about nature.

At the other extreme from the conflict of interest is what I will call the exclusive conflict between final and efficient cause, that is, the cases in which it is denied that an answer to the question "why?" in terms of final cause is even possible. Let me give an example of an evolutionary problem: R. A. Fisher asked, "Why is it that most mutations that occur in organisms are recessive?"—in other words, what is the origin of dominance of genes? Fisher gave an answer to this in terms of final causation. He talked about what happens in natural selection to new mutations as they arise and as they affect the fitness of organisms, and concluded by mathematical analysis that if mutations arose over and over again, then eventually

the genotype of organisms would evolve in such a way that any new mutation would be recessive in its phenotypic manifestations. What Fisher said was that dominance is a result of an evolutionary process in which the whole nature of the genome has been adjusted in order to hide the effects of new mutations. Sewall Wright opposed this notion, claiming that natural selection was too weak to accomplish the result predicted by Fisher, and that dominance is a physiological fact of life that does not need any explanation in terms of natural selection. Wright said that dominance arises because of the nature of the response-curve of enzymatic reactions. As is well known, when the concentration of enzyme is limiting, the amount of product is proportional to the amount of enzyme. As the amount of enzyme increases, the amount of product produced falls off and reaches an asymptote. Thus the phenomenon of dominance—because, for a doubling of the amount of enzyme, there is not a doubling of the amount of product. There will be complete dominance if in fact the amount of enzyme produced by one gene completely saturates the substrate.

Notice that I am making a distinction between my first case, “why is the sex ratio 1:1?”, in which there exist alternative explanatory systems, each designed to answer essentially different questions framed in the same terms, and a second case in which the two forms of explanation commonly used in biology are at variance with each other. In the latter case, these two explanations are juxtaposed and one is rejected as being, in fact, incorrect. This conflict also occurs in molecular biology. As an example, let us ask why it is that the genetic code looks as it does. Why does a certain set of triplets of nucleotides and not another set of triplets specify *phenylalanine*? Is it just a result of chance? Would it make any difference in the world if instead of UUU specifying phenylalanine it were UGC? There are two schools of thought about this matter. One says that the code we have is an historical accident, that there could have been (and may have been at one time) other codes. One of the interesting discoveries of modern molecular genetics is that the code is universal. It appears that all living organisms at present share the same relationship between the set of triplets and the amino acid that is produced. On the theory that many codes are *a priori* equally likely, this discovery proves the monophyletic origin of life. However, it can be argued that the set of triplets which specify particular amino acids are not at random with respect to each other. If you really look at them hard, you notice that the redun-

dancies are meaningful, that they are of such a form that the code is protected against any phenotypic effect of single-step mutations. It is adaptive to have UUG and UUU specify phenylalanine because a single step mutation in position three will not affect the amino acid specified. Among all the possible codes that could exist, evolution has, in fact, selected a code that has a set of redundancies with certain optimal properties.

Competing with this, there is a third theory, which is that the code is not random but that it has not evolved either. Rather, it is postulated that there is a quantum-mechanical relationship between the code elements and the amino acids produced. On this view, even if we allow an evolutionary argument for redundancy, it is not chance that phenylalanine is specified by this set of triplets and not by an utterly different but equally redundant set of triplets, let's say AUG and AGG. There may be quantum-mechanical reasons that a certain triplet will most easily code for a given amino acid because of the way in which this information is encoded in the messenger RNA. Here we see three explanations. The first is that the actual realization out of all the possible ones is that which has arisen by a process of natural selection, and indeed that there were other groups of organisms with other kinds of coding but that these groups were not so successful because their coding systems were in some sense less perfect, less optimal, and so died out. The second is that there were other groups of organisms with other codings and these just happened to die out by chance, the one remaining being in no sense optimal. The third is that there has been one code because in the time of prebiotic evolution the particular coding arrangement that arose was that which had the highest probability under certain rules of quantum chemistry.

A third conflict of explanation occurs in what may be called the problem of *exclusive versus complete* explanation. I am not going to say very much about this because it is fairly trivial and obvious to everyone, but it should at least be mentioned. Theoretical biology has as its goal the placing of bounds on the possible outcomes of given input conditions. That is to say, what theoretical biology is engaged in doing is taking a certain set of contingent statements about the world and constructing a machine, enabling one to say that if the inputs to this machine are of such and such a nature, the outputs must fall in a certain restricted subset. Theoretical biology is in fact nothing but a biology of exclusion. It says: if mutation rates are of the following nature, if the differences in organisms in rates of reproduction are such and such, then it

is not possible that outcome X will occur or the probability of outcome Y is less than 10^{-6} . A good deal of theoretical biology is misunderstood by molecular biologists, who by and large call themselves nontheoretical biologists and think of themselves as the archetype of the experimental biologist because they are concerned not with exclusion but with complete specification. If we were to agree that because somebody has worked out the complete base sequence of one of the transfer RNA's, we will not allow anyone to have any more money to work out any of the others, because it is just more of the same, molecular biologists would revolt. They would say that we have destroyed one of the most important tasks of the biologist, which is to specify completely the set of biological mechanisms. The theoretical biologists will say, on the other hand: "We set up a theory which predicted certain characteristics; we have allowed you to go off and spend a lot of money making one molecule to prove that it really works. Now go away and do something else useful." A conflict arises, then, between those who say that we do not explain an observation until we have filled in the complete picture of all the mechanical levers and gears, and those who are satisfied with an explanation which only says that the levers and gears must look "something like the following," must fall within a certain set.

This is a problem that arises not only in the conflict between theoretical biology and experimental molecular biology, but arises even within these disciplines. Related to it and of much more interest is what one might call the conflict between *sufficient* and *exact* explanation. Most of evolutionary theory has the following structure: there is some general framework of laws, and we know, from observations about nature, something about the values of the parameters that we put into the calculating engine. Thus we produce an allowable set of outcomes. We know something about mutation rates; we know about the differential rates of fertility of organisms. Given such bounds, the evolutionary biologist can then predict a possible set of outcomes. If he is really very clever, he can even set up a distribution function of the probabilities of the outcomes. The actual outcome is then observed, and we ask (a) whether it falls inside the set of allowable outcomes, and (b) if it does, what was the probability of that outcome. If the outcome is one of the highly probable ones, then we are happy, we have a sufficient explanation of the phenomenon and the evolutionary biologist goes no further. There is a very strong resemblance, then, between explanatory mechanisms in

evolutionary biology and the standard theory of hypothesis testing in statistics. What one does is to make deductions about the set of probabilities of possible outcomes, to look at a given event and ask whether that event falls within a critical set (which is constructed from the *a priori* distribution of the possible outcomes); if it does, we are happy and claim a sufficient explanation. Most evolutionary biologists are satisfied with this kind of explanation.

Let us take a classical case. There was a tremendous flowering of the architecture and ornamentation of the heads of the ceratopsian dinosaurs, a group of rhinoceros-like herbivorous dinosaurs. The question arises in evolutionary theory: why were there one-horned and three-horned ceratopsians; why is it that they all did not have the same ornamentation? There are two explanations for such a manifestation. One says that each one of the specific ornamentations was specially selected in an environment that was different from others and therefore was favored by natural selection. The assumption is that if a one-horned and a three-horned ceratopsian dinosaur were living in the same environment, three horns would be better than one and therefore three horns evolved. The alternative explanation is that these organisms evolved from somewhat different original pools of genes; that they evolved in isolation from each other, both temporally and spatially; that there was a tremendous time-scale involved, and that each of these outcomes represented a sufficient adaptive outcome of the evolutionary process. The question of comparing them or of demanding that there be specific environmental reasons for the different types is asking too much. In the multidimensional space of gene frequency, natural selection moved the population to one of a set of alternative singularities. Where it moved depended on its initial state and certain random elements. It is not even sensible to ask the question: is it better to be at one than at the other, or more adaptive? These are alternative outcomes of the evolutionary process, alternative probable realizations. It might be possible to compare these realizations if the organisms then become sympatric, but that is not at issue.

The second explanation follows from the existence of alternative stable states of a given dynamical system. This is a very important point which is not always understood. It is not true that, in a deterministic dynamical system in which everything is fixed except the initial starting values, the system will go to one and only one point. There exist, in any dynamical system of sufficient complexity, many, many alternative

stable states to which the system may go, and the determination of which state the system evolves to is a function of variation in the starting point. For example, if you are a mountain climber with a compulsion to go upward, then in the Rocky Mountains there are over fifty peaks above 14,000 feet, any of which will make you happy. It only depends on where you start. It is not true, therefore, that all the results of evolution are different from each other because the forces of natural selection were different. Many are different from each other because the starting conditions were different. This second explanation is sufficient; it says that what happened could happen. A difficulty arises when an event occurs which ought to have happened with a very, very low probability. Even in such cases, of course, since the probability-density function of all outcomes of evolution is almost flat anyway, there is a serious problem of deciding whether a thing could have happened or not. This is a serious methodological problem for the evolutionists. The evolutionist who wants to decide among explanations by using the principle of maximum likelihood is in serious trouble because the likelihood surface is so flat. For that reason, evolutionary explanatory hypotheses generally are not very specific. Rather, a set whose total likelihood is high is accepted.

I would like at this point to destroy what I think is an error in the writings about evolutionary theory, an error at least from our modern standpoint. That is the problem of whether or not the principle of natural selection, or survival of the fittest, is an *explanation* of evolution. I maintain that it is not an explanation of evolution and it should not be intended as such, whatever the intent of Charles Darwin and his disciples may have been. Evolution is the necessary consequence of three observations about the world, observations which are at this moment unchallengeable. They are: (1) There is phenotypic variation; the members of a species do not all look and act alike. (2) There is a correlation between parents and offspring. This statement says nothing about Mendelism, nothing about genetics. It just says that if the two parents are shorter than the average, their children will on the average be shorter. That is an observation which is true for every character to a different degree, but for every character that can evolve, it must be true. (3) Different phenotypes leave different numbers of offspring in *remote* generations. (If they have more grandchildren, that's good enough).

If these three propositions are true, there will be an unavoidable evolutionary change in the population. Does that

mean, then, that the principle of natural selection is an explanation of evolution?

These are three contingent statements, all of which are true about at least some part of the biological world, and it therefore follows that evolution must occur. The reason I list them is because there is a lot of confusion about the so-called "tautological" nature of the explanation of fitness and natural selection. There is nothing tautological here. These are three true statements about the world, and it follows from them that changes must occur in the population and that these changes can be predicted. If you like, these three statements are the explanation of evolution. They do not explain the origin of any particular event in evolution; they explain why there is a change, in general, in the forms of organisms in time.

Finally, I come to my main question: What do we mean by the historical elements in evolutionary explanation and in biological explanation? Let us consider first a generalized dynamical process in time, operating in some space. We can regard a system as being in some state E at time t and passing into some other state at E' at time $t + 1$, continuously or discontinuously. The system can be described as a point in a multidimensional state-space, and through time the system sweeps out a trajectory in the space. In this space there exist singularities. Think of the space as a vector field, a field showing the motion of the particle at every point. These singularities may be point singularities or they may be limit cycles, but more often in evolutionary context, they are not cycles but points. Arrows may be drawn showing the directions and the speeds with which the particle will move within the space at various points. Stable equilibria are points toward which all the vectors in a neighborhood point. If the system arrives at such a point, it will not come out again.

It is characteristic of a dynamical system in time that if the system is at such a point or in such a closed circle, there is no information about how it got there. That is, at equilibrium, there is *no historical information*. Of course it is possible to subdivide the space into regions from which the system would have fallen into one or the other of alternative equilibria. So it has some information about the set of possible starting points, but only that very weak kind of information. So, any explanatory hypothesis in biology which has explicit in it that the system is in an equilibrium state is ahistorical because it says that the biological world is in a stationary state in the dynamical sense. If you deliberately choose to concen-

trate your attention on the equilibrium aspect of biological systems, you are making a deliberate choice to ignore their historical aspect. In choosing to study only the equilibrium you destroy any possibility of information about history.

You can, on the other hand, choose to study the trajectories themselves. Since these trajectories can be described in general by some differential equations, if you have information about a number of points through which the trajectory passes, you can reconstruct the differential equations. That is to say, you then have historical information which allows many more deductions. For example, you can say where the system is going and where it has been.

Thus we must distinguish in a dynamical system two sets of points that make up the whole space. One set of points is a small set in which all historical information has been destroyed, the set of all singularities. The rest of the points lie in a set in which one can reconstruct some historical information. The argument among biologists as to the relevance of history in explanatory hypothesis revolves around the division among biologists into that camp which says it wishes to study the equilibrium states in biology and the camp which wishes to study the dynamical states in time. This division exists among evolutionary biologists and especially among ecologists. There are now two schools of ecologists: first, those who say that what is interesting is only the ergodic properties of biological systems—the properties of the ensembles which are invariant in time and about which no historical statements can therefore be made; and second, those who want to study properties which are not invariant in time but which are in fact unique in time and space.

I will give an example from modern ecology where these two alternative explanatory schemes are most in conflict. Let us ask the question: "Why is it that in a copse in England there are the following five species of birds—Species A, B, C, D, E—in the proportions P_A , P_B , P_C , P_D , and P_E , whereas in a copse in the United States which has life forms very similar to those in England, there are only four species of birds, F, G, H, and I, and in quite different proportions. This is a specific example of the whole problem of zoogeography and ecology; it becomes in fact the leading problem of ecological, evolutionary studies. There are two answers to this question. One is given by David Lack and represents the English school of explanatory hypothesis about zoogeography. The particular environmental situation is clearly different in England from that in America, and *therefore* there will be a different number of species and the identity of the

birds will be different. That is, American birds occupy their copse and English birds theirs. They are different sets of birds *because* the copses are different. This is equivalent to the argument that one-horned dinosaurs and three-horned ones were one-horned and three-horned because the environment was different. Under this hypothesis, if we want to understand why a particular set of bird species occupies a particular place in the world, we must know almost everything there is to know about the environmental circumstances in that place and about all the interactions between the birds.

Opposed to this view is the one typified by MacArthur and Wilson and the American school and given cogent form in their recent book, *Equilibrium Theory of Insular Zoogeography*. The authors suggest that we should forget about the names and identities of the species and ask why there are four or five species and not twenty-five species in this environment and whether we would predict more species in the English copse than in the American, on the theory that all the species are alike, or interchangeable. The equilibrium aspects of the occupation of woods by birds are such that, given a long time, such a copse will settle down to about four or five birds in frequencies predicted from a few variables. If all the birds of the species now occupying the copses became extinct, some other four or five species would get in and stabilize. In the case of islands, if one simply considers the size of the island, the distance from the nearest island, the distance from the mainland, and the number of species in the pool on the mainland, one can in fact give a fairly good description of how many species will be on each island. Moreover, one can predict the difference in the distribution of the number of species on islands that are close to the mainland and far out, and one can actually draw a graph of the relation between distance of the island to the mainland and the number of species. But in this equilibrium theory, the identity of the species is destroyed.

Thus, two alternative explanatory devices exist in zoogeography. One claims that the particular five species in a copse are only a sample of all the propagules that arrived there, and the interesting question is really how many there are and not what their particular names are. The alternative says that what is important is not how many there are but which particular ones there are, and that no other combination of species would be stable. I presume the real answer is—if I take all samples of five bird species that could have been in a copse—that they will be graded according to the probability with

which any particular group of five would be successful, or the length of time they would be resident before being replaced by another group of five. I do not know what the distribution of this probability looks like; I rather suspect that it is strongly modal—that is, that there are a few combinations which have very high probabilities because of the nature of the interaction in the species, and lots of combinations which have very low probabilities.

For the most part I have been concerned throughout this discussion with the sociology rather than with the philosophy of science. The two explanatory hypotheses, one of which includes an historical and specific element, and the other which includes a statistical and equilibrium element, have become for biologists mutually exclusive by the nature of the conflict between biologists as people. There is here a problem of historiography. Many of the very positive statements of reputable and intelligent scientists will be colored by the tendency of biologists, like everyone else, to polarize in some dialectical fashion. Historians have been taught from the cradle to be wary of what they read, not to confuse the record with what was. This caveat applies in science as in any other social endeavor. Each one of us has his preferred explanatory system and we tend to see the other explanatory systems as excluded by our own.