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Chasing Shadows: Natural Selection and Adaptation

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> The main task of any evolutionary theory is to explain adaptive complexity, i.e. to explain the same set of facts which Paley used as evidence of a Creator
>
> Maynard Smith (1969)

The origin of adaptive complexity is still one of the central issues in comparative biology. Currently the most popular approach to explaining the sources of adaptation takes its origin from Darwin. Darwin's question—remarkably like Paley's—is posed in Chapter 3 of *The Origin of Species*: 'How have all those exquisite adaptations of one part of the organisation to another part, and to the conditions of life, and of one distinct organic being to another, been perfected?' (Darwin, 1996, p. 51). Darwin's answer, remarkably *unlike* Paley's, invokes the process of natural selection.

> Natural selection is daily and hourly scrutinising,..., every variation, even the slightest; rejecting that which is bad, preserving and adding up all that is good; silently and insensibly working, whenever and wherever the opportunity offers, at the improvement of each organic being in relation to its organic and inorganic conditions of life. (Darwin, 1996, p. 133)

This passage, under a fairly natural interpretation, encapsulates the received view on the relation of natural selection and adaptation: natural selection in the organic realm is the *cause* of adaptations. In fact the appeal to natural selection is thought to give us more than merely an account of the causes of adaptations; it yields a reductive analysis of the concept of an adaptation. Sober's definition of adaptation is reasonably representative:

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> [Trait] *A* is an adaptation for task *T* in a population *P* if and only if *A* became prevalent in *P* because there was selection for *A*, where the selective advantage of *A* was due to the fact that *A* helped perform task *T* (Sober, 1984, p. 208).[1]

The significance of this reductive analysis of adaptation is difficult to overestimate. It seems that adaptation is indispensable to evolutionary biology not merely as explanandum but also as explanans. The presence of a trait is often explained by citing what it is an adaptation for. Examples of this form of explanation abound: the capacity of chameleons to match their colour to that of their surroundings is explained by the fact that this is an adaptation for avoiding predators; the presence of sonar in bats is explained by the fact that sonar functions as a device for navigation in dark environments. Taken at face value these adaptive, functional explanations appear to explain the presence of a trait by appeal to the way it subserves an organism's goals or purposes. They are teleological. Of course, in post-Galilean science, we are told, there is no room for such explanations. It is a feature of the 'anti-Aristotelian purge' of the natural sciences that no natural phenomenon can be explained by appeal to goals, ends or purposes.[2] There are no purposes in nature, only causes. The reductive analysis of adaptation allows us to recast adaptive explanations in the causal mode. If to be an adaptation is to be the product of natural selection, then to explain the presence of a trait by appeal to what it is an adaptation for is to explain the effect that the trait had *in the past* which caused it to be promoted by natural selection. For example, the capacity of chameleons to match their colour to that of their immediate environment is an adaptation for the avoidance of predators because this trait was promoted by selection in the past; it has the selectively salient effect of making chameleons difficult for predators to detect. This is a strictly efficient-cause type of explanation. There is no irreducible appeal to the purposes or goals of an organism. There is simply an appeal to the causal efficacy of natural selection: to cite Paul Griffiths' adage, 'where there is selection there is teleology' (Griffiths, 1993, p. 420). Natural selection makes available to us a sanitised, rehabilitated form of reductive, naturalised teleology.[3]

This is by now an extremely familiar story. One needs only to consult the recent collections of papers on adaptation, function and natural teleology to realise the impact it has had.[4] Its success relies upon the success of each of two constituent claims: i) that adaptations (and correlatively functions) are the causal consequences of natural selection; and ii) that explanations which appeal to adaptation (and function) may be reconstructed, giving them a strictly aetiological cast. The truth

[1]There are non-historical definitions of adaptation. See Brandon (1978); Reeve and Sherman (1993). For a marvellous account of the history of the concept of adaptation I recommend Amundson (1996).

[2]See Buller (1999, pp. 1–28) for an illuminating discussion of the difficulties posed by functional, adaptive explanations to the standard model of scientific explanation. The term 'anti-Aristotelian purge' originates with Charles Taylor (1989, p. 59). I thank Matthew Elton for directing me to it. See also Taylor (1964, ch. 1).

[3]See also Lennox (1995).

[4]See for instance Allen *et al*. (1998) and Buller (1999).

of the second claim is clearly dependent on that of the first. Much has been written about the latter phase of this reductive approach to biological teleology, but to my knowledge little has been said about the former. It has been more or less taken for granted—sanctioned by the orthodox reading of *The Origin of Species*. Nevertheless, I believe it to be false. Natural selection in the organic realm cannot causally explain adaptations because it is not the *cause* of adaptation. When we look more closely at the causes of evolutionary change we find that natural selection and adaptation are indeed related as explanans and explanandum, but the relation is the converse of that usually supposed. The process of adaptation causes, and hence explains, the particularly distinctive nature of selection in the biotic realm.

The source of the error, I believe, lies not in the *Origin* itself but in an erroneous metaphysical picture drawn from the Modern Synthesis theory of evolution. That theory explicitly construes selection as a force acting over populations of genes. I suggest that in the search for the *causes* of adaptation we should look not to the statistical structure of populations of genes but to the generic principles which determine the organisation and development of individuals.

1. The Forces of Evolution

Modern evolutionary theory, largely inherited from Darwin, is conceived as a theory of forces acting upon populations (Sober, 1984). Prior to Darwin biologists attempted to explain the diversity of form, and the prevalence of adaptation, in terms of the properties of individuals (Mayr, 1982). Typically, they invoked the inherent tendencies of organisms to strive to attain their ideal forms. Darwin's theory is a radical departure. It relies upon certain observations concerning the structure and behaviour of populations. Darwin noticed that the heritable variation in traits within a population determined differential rates of survival and reproduction within individuals. He surmised that over the course of time, because of this differential survival and reproduction, the constitution of a population changes. It comes to be constituted of individuals which possess those heritable characters which enhance survival and reproduction. Indeed it is thought that Darwin's most significant conceptual breakthrough was the shift from individual thinking to what Mayr has called 'population thinking' (Mayr, 1982). Sober (1980) expresses the view concisely:

> Darwin ...focussed on the population as a unit of organisation. The population is an entity, subject to its own forces, and obeying its own laws. The details concerning the individuals who are parts of this whole are pretty much irrelevant. Describing a single individual is as theoretically peripheral to a populationist as describing the motion of a single molecule is to the kinetic theory of gases. In this important sense, population thinking involves *ignoring individuals*. (Sober, 1980, emphasis in original. Quoted from Sober, 1984, p. 175)

There is a certain amount of scholarly debate concerning the degree to which Darwin himself thought along these lines (see Schweber, 1985; Depew and Weber, 1995). But this much is certain: the modern-synthesis theory of evolution which followed was developed expressly along the 'population thinking' model. It is conceived explicitly on the model of statistical dynamics (Fisher, 1958). The central idea is that a population is an 'aggregate of gene ratios'. The laws of evolution, like the gas laws, describe the behaviour of ideal populations of genes.[5] The fundamental forces affecting gene frequencies are taken to be natural selection and mutation. Variants of the modern synthesis theory differ over the relative importance to be placed upon those factors which divert a population from the ideal conditions—factors like the finite size of populations, non-random mating, epistatic interactions between genes. Nevertheless they appear in agreement over the view that natural selection is a force, or the principal force, acting on populations which alters gene frequencies and in turn produces adaptations. It will be worthwhile, I believe, to look a little more closely at this putative force of selection.

Natural selection, we are told, works on *heritable variation in fitness* (Lewontin, 1974). Fitness is usually taken to be a property of an individual, specifically its propensity to survive and reproduce (Mills and Beatty, 1979).[6] Whenever differences in these propensities between individuals are manifested there is selection. These propensities, in turn, are realised through the systematic causal contribution of an individual's traits to its survival and reproduction. This much, I think, should already be obvious from the reductive account of adaptation outlined above. If you want to explain why a trait is prevalent in a population you must cite the way traits of its type have contributed to the survival and reproduction of individuals in the past. For a trait to have been selected for in the past is just for it to have contributed (disproportionately) to the survival and reproduction of individuals in the past. The cause of the changes in trait frequency that natural selection is supposed to explain appears to be nothing more than the systematic contribution of heritable traits to survival and reproduction of individuals. This point is often overlooked, but it is amply demonstrated in the recent units-of-selection debate.

The units-of-selection debate, as it is usually interpreted, is a debate over the causes of evolutionary change. Do traits (genes) evolve because they benefit individuals or do they evolve because they merely benefit genes? Gene selectionists maintain that the unit of selection is always the gene; traits evolve only because they benefit genes and selection is a force which alters gene frequencies. Consequently, there is selection when *and only when* gene frequencies change. Individual selectionists maintain that the unit of selection is often the individual, perhaps sometimes the group (Sober and Lewontin, 1982; Sober and Wilson, 1994). Sober and Lewontin

[5]See Fisher (1958, p. 37ff.) and also Hodge (1992) for an extensive discussion of Fisher's analogy between natural selection theory and statistical dynamics.

[6]This is a notoriously tricky concept to state precisely; but nevertheless, the propensity account of fitness appears to be on the right track.

(1982) offer the example of individual selection without a change in gene frequencies. Consider a case of extreme heterosis. For some locus with two alleles *A* and *a*, the heterozygotes (*Aa*) are on average extremely robust, while the homozygous condition (*AA* or *aa*) is lethal. Only the heterozygotes survive long enough to reproduce. At mating all individuals will be heterozygotes, so the frequency of $A = a = 0.5$. After mating, however, not all individuals will be heterozygotes. The frequencies of genotypes will be $AA = 0.25$, $Aa = 0.5$, $aa = 0.25$. The gene frequencies, however, will remain constant throughout: $A = a = 0.5$. According to the gene selectionist there is no selection in this system because from one generation to the next, and even *within* a generation, there are no changes in gene frequencies. But, Sober and Lewontin protest, this conclusion misrepresents the *causes* of stasis in gene frequencies. There is selection of the heterozygotes; only they survive to breeding age. As a consequence, at segregation the proportion of *A* gametes is equal to the proportion of *a* gametes. At mating these gametes combine in the proportions given above. The reason they do not change is a combination of selection for the heterozygote individuals and the laws of Mendelian genetics.

Some gene selectionists take a slightly different tack (e.g. Kitcher and Stereleny, 1988; Williams, 1966). They respond that this situation can be reinterpreted or redescribed from the gene's point of view. In fact, they claim that, no matter what causal relations are involved, selection can always be 'represented algebraically' in terms of changes of gene frequency. Natural selection, according to this view, is merely a mathematical descriptor for changes in gene frequencies. In many ways I find this an unobjectionable position, but it is worth noting that it offers little succour to those who claim that natural selection *causes* changes in gene frequencies. Natural selection on this account is not a causal concept. It is merely a device for representing changes in gene frequencies.[7]

Sober and Lewontin conclude that if selection is to yield causal explanations we must construe it as a genuinely causal process which acts on individuals.[8] But what are the causal processes involved here? As I have said, selection is simply a consequence of traits making a systematic causal contribution to the survival and reproduction of individuals. Natural selection, it seems, is merely the consequence of an assemblage of causal processes taking place at the individual level. There is no need to invoke a *distinct* force operating over populations in order to explain the sort of changes in gene frequency thought to be explained by natural selection. Indeed, it is tempting to suppose that if there were such a force, it couldn't be natural selection. Suppose there were some force which caused a process *p* which is distinct from the assemblage of processes comprising the survival and reproduction of individuals. Could *p* explain the changes in trait frequencies which are the causal consequence of the contribution of traits to survival and reproduction?

[7]See Sober and Wilson (1994) for a discussion of the 'bookkeeping' argument for gene selectionism.

[8]I am restricting my attention here to cases of individual selection, prescinding from the issue of whether there is selection at other levels of organisation.

Clearly not. But if *p* couldn't explain that, it couldn't explain what natural selection is required to explain. Whatever process *p* might be, it couldn't be the process of natural selection. So, given the explanatory role selection is required to play it must be the consequence *only* of the systematic causal contribution of traits to the survival and reproduction of their bearers.

This argument has an air of sophistry about it. Natural selection might well be a genuine causal process which comprises an aggregate of distinct causal processes. There must surely be such aggregates of processes. The processes of metabolism and meiosis, for example, are genuine causal processes made up of distinct component causal processes. But it is worth noting that selection appears to be composed of its component processes in a quite distinctive way. The mere aggregate of individual survival and reproduction is not sufficient for selection. Selection only occurs as a consequence of the *difference* between individual-level processes. One could eliminate natural selection entirely by increasing the propensity of occurrence of one of its component processes. One could not, by contrast, eliminate the process of photosynthesis entirely by increasing the propensity of one of *its* component processes to occur. Perhaps by altering the propensity of a component process of photosynthesis one could affect the final outcome, perhaps even cause the discontinuation of the process, but that would not be the same as bringing it about that no such process occurs *at all*. It is tempting to suggest that, rather than being a population-level causal process, natural selection isn't a causal process at all. It bears the hallmarks of a pseudoprocess.

Imagine that you are chasing a shadow, perhaps one cast on the surface of the earth by the flight of an aeroplane. The motion of the shadow looks like an ordinary process, very much like your own motion. At each moment it has an instantaneous velocity; it has a trajectory, a history. But the movement of the shadow is no ordinary process: it's a *pseudoprocess*. It is simply the by-product of a set of other genuine causal processes which include: the flight of the aeroplane, the emission of photons from the sun, their collision with the aeroplane and their absorption by the surface of the plane, the collision of the photons with the ground around the shadow and the reflection of light off the earth's surface. The motion of a shadow—like the change in gene frequencies within a population—is a consequence of the *difference* between the effects of genuine causal processes. A shadow moves just when the boundary between the area of high illumination and low is shifted. Unlike your own motion, the motion of a shadow cannot be altered by imparting a force to it. To alter the trajectory of a shadow you must bring about a change in the differential rate of photon bombardment on the surface. Unlike the shadow, you have momentum and mass; changes to your shape, mass, and charge may be transmitted from one phase of your motion to a later phase.[9] Successive phases of your

[9]For various attempts to draw up a strict criterion for demarcating pseudoprocesses see Salmon (1984, 1998); Dowe (1992, 1995).

motion are directly causally connected. In contrast, successive phases of a shadow's motion are related as effects of a common cause. Your motion, unlike that of the shadow, is a genuine causal process.

I suggest that the process of selection is more like the motion of a shadow than it is like your own motion. It is simply the consequence of the differential rates of distinct causal processes occurring *within individuals*. Of course changes in trait frequencies are themselves simply the consequences of the differences in these same processes. Natural selection and changes in gene frequencies are not related as cause and effect; they are joint effects of a common cause. Natural selection does not cause changes in gene frequencies.

The causal inefficacy of selection should not be taken to imply that it is explanatorily idle. Pseudoprocesses may well be invoked in causal explanations. We may explain a sudden change in the temperature of a surface by saying a shadow passed over it. This explanation stands proxy for an explanation which involves a suite of genuinely causal processes. Closer to home, Elliott Sober (1987) has suggested that the inheritance of phenotypes is itself a pseudoprocess. It comprises the causal processes of meiosis, gamete fusion, transcription, protein synthesis etc.. Even so, the heritability of phenotypes is explanatorily important. One only needs to look at the recent Bell Curve debate to see this. Herrnstein and Murray (1994) argue that differences in IQ between various socioeconomic and ethnic groups are narrowly heritable. Detractors (e.g. Block, 1995) counter that IQ is *not* heritable. They manifestly do not claim that heritability is not explanatory. In the same way, I believe that natural selection may be explanatorily significant in evolutionary biology, even though it is not a force acting on populations. It stands proxy for a suite of genuinely causal processes operating on individuals.

This is not to say that the population-thinking model of evolutionary change is wrong. It is, after all, populations and not individuals which undergo adaptive evolution. Moreover, they do so simply by changing their constitution, just as Darwin surmised. The unit of evolution, we might properly say, is the population. However, it does not follow from the fact that the effect—change in trait frequencies—is a population property that the *cause* must be a population-level force. The modern synthesis theory does not sanction the metaphysical claim that selection causes changes in gene frequencies. Rather, it appears that the causes of changes in gene frequencies are the processes which determine the propensities of individuals to survive and reproduce. If we are interested in causal explanations of changes in trait frequency, we should look not to the statistical structure of 'aggregates of genes' but to the constitution of individuals.

2. The Source of Adaptation

Thus far I have discussed only the putative causal role of selection in changing trait frequencies within a population. The preliminary conclusion is that selection is not the cause of changes in trait frequencies; instead selection and changes in

trait frequencies are joint effects of those causal processes which determine the propensities of individuals to survive and reproduce. But selection is generally considered to be the cause of more than mere changes in trait frequencies. It also causes adaptations. The dual roles of selection are described by Ayala.

> Natural selection has been compared to a sieve which retains the rarely arising useful mutations and lets go the frequently arising harmful mutants. Natural selection acts in this way, but it is much more than a purely negative process, for it is able to generate novelty by increasing the probability of otherwise extremely improbable genetic combinations. Natural selection is creative in a way. (Ayala, 1970, p. 5)

Natural selection in the organic realm is both a winnower and, to borrow François Jacob's phrase, a tinkerer. Of course the two effects are related. The passage quoted from Ayala suggests that *by* changing trait gene frequencies, selection enhances the probability of new complex adaptations arising: selection tinkers by winnowing.[10] The view is put forward forcefully by Dawkins.

> Darwinism—the non-random selection of randomly varying replicating entities is the only force I know that can, in principle, guide evolution in the direction of adaptive complexity. The ingredients in a general recipe for Darwinian selection are replicating entities of some kind, exerting phenotypic 'power' of some kind over their replication successes. (Dawkins, 1998, p. 32)

Wherever selection occurs, so long as it is permitted to run for long enough, there will be an increase in adaptive complexity. The general picture, I take it, is that because selection causes changes in trait frequencies and these in turn are a positive causal factor in the origin of adaptations, selection itself is a positive causal factor in the origin of adaptations, a point made most explicitly by Neander.

> Selection does more than merely distribute genotypes and phenotypes...: *by* distributing existing genotypes and phenotypes it plays a crucial causal role in determining which new genotypes and phenotypes arise. (Neander, 1995, p. 585, emphasis in original)

The foregoing discussion should raise some doubts. If natural selection isn't the cause of changes in gene frequencies, it is probably not the cause of adaptive complexity either. Selection, as we have seen, is merely the consequence of differential propensities of individuals in a population to persist or replicate. It is difficult to see how these propensities *alone* should generate adaptive complexity.

As a matter of fact, they don't. Selection as a general phenomenon is widespread but in very few arenas does it lead to increases in adaptive complexity. Marc Bedau (1991) discusses an imaginary scenario: Suppose we have a population of crystals in solution which vary slightly in their structure in a way that determines that crystals of one sort more rapidly form stable lattice structures than do crystals of other sorts. One would naturally expect that crystals of this faster growing sort would come to be disproportionately represented in a population of growing crys-

[10]See Neander (1995) for a development of this position.

tals. This is a case of selection, but there is no adaptation here. The structures which confer on crystals a faster growth rate are not adaptations (Bedau, 1991; Matthen, 1997). More to the point, populations of crystals of this sort do not exhibit a tendency to become increasingly efficient at precipitating ions from solution. Selection does not increase the rate of formation or the stability of the lattice structures.

Another example of selection without adaptation occurs in systems of convective cells. When a pan of water is heated from beneath an energy gradient is set up between the hot water at the bottom of the pan and the cooler water at the top. When the energy gradient reaches a certain intensity, convective cells form. This is the most efficient arrangement for the dissipation of energy through the water column. There is both an optimal size and shape for a convective cell. The optimal size is determined by the viscosity of the fluid and the capacity of the cell to dissipate energy. The optimal shape is hexagonal, permitting the maximum packing of cells. Those cells which are closer to the optimum are more stable. Cells which are sub-optimal in size and or shape are less stable. Cells of smaller than optimal size fuse to become larger cells; larger than optimal cells break up. Non-hexagonal cells change their shape (Depew and Weber, 1995). The overall effect is a reduction of variation in selectable parameters. In its stable state, a population of convective cells (Benard cells) displays a remarkable uniformity in cell size and shape as a 'consequence' of selection. Even though there is selection, there is no adaptation here. Once a stable equilibrium is attained, the system does not improve its dissipative capacities in any way. Furthermore, we could keep the selection process going as long as we like by introducing perturbations of all sorts, but a population of convective cells would still manifest no tendency toward an increase in adaptive complexity.

Where selection occurs in populations of crystals or Benard cells it merely eliminates variants; it winnows. But when it operates over populations of organisms, we are told, it tinkers. What is so special about natural selection operating over populations of organisms that makes it 'able to generate novelty'? More to the point if, as I have suggested, natural selection is not a causal process at all, but the net effect of a series of individual-level processes in which individuals' traits make a systematic causal contribution to their survival and reproduction, the question becomes: 'What is so special about *organisms* which determines that their differential survival and reproduction leads to adaptation?'. A common answer, offered by Dawkins (1986); Stereleny (1988) and others is that only in populations of organisms is natural selection cumulative: 'Cumulative selection is the hallmark of biology, and is, I believe, the force underlying all adaptive complexity' (Dawkins, 1998). I am willing to accept that adaptive complexity comes about when selection is 'cumulative' and that only biological populations undergo cumulative selection, but I submit that this gets us no closer to uncovering the causes of adaptation. We still have no inkling of what makes selection operating over populations of *organisms* 'cumulative' in a manner

sufficient to produce adaptations, whereas selection operating over populations of crystals or convective cells is not.

It may be helpful in addressing this question to introduce the notion of an adaptive landscape. Since Sewall Wright (1932), it has become common to represent natural selection of the sort that leads to adaptative complexity in terms of the fitness landscape. The fitness landscape is a simple device for representing the comparative fitnesses of genotypes. Suppose we have a population of haploid organisms with N genes each, each of which has two alleles; there are, then, 2^N possible individual genotypes. We can imagine this assemblage of genotypes laid out on a multidimensional surface like a map. The distances between genotypes on the map correlates with their allelic differences. Each genotype has a fitness value which is represented as an altitude on this multi-dimensional landscape. Less fit individuals, those whose genotypes occupy lower altitudes on the fitness landscape, are eliminated by selection. Fitter individuals, those higher up, survive and reproduce. New mutations and combinations occasionally—but often enough—produce individuals which occupy places slightly higher on the landscape. Through the gradual accumulation of these fitter (higher ground) variants, and the elimination of less fit (lower ground) ones, selection gradually drives a population to local fitness maxima on the adaptive landscape.

The imagery of the adaptive landscape is a useful one. It demonstrates not that adaptive evolution is an inevitable consequence of selection, but that it requires specific sorts of landscapes. The first requirement for a landscape on which adaptive evolution can occur is that the fitnesses of neighbouring genotypes are reasonably highly correlated. Similar (neighbouring) genotypes must have quite similar fitnesses. If the fitness landscape is highly uncorrelated, then for any genotype, its neighbouring (similar) genotypes will be wildly different in fitness. In this case, the effect of any particular gene is so largely dependent upon its context (i.e. the other genes it is associated with) that no gene makes a systematic, relatively uniform contribution to fitness. Consequently selection could not be expected to drive any one gene or set of genes to fixation. It may eliminate various genotypes, but it will not tend to favour any particular genes over the long term. Populations will not be driven gradually by selection toward local maxima because these adaptive landscapes are not made up of gently undulating hills and valleys. They are random and jagged; and adaptive evolution—the gradual accumulation and combination of beneficial traits—cannot occur on them (Kauffman, 1995). There is selection, but no adaptation; winnowing, but no tinkering.

Nor would adaptive evolution occur on a very smooth landscape with only a single global maximum—a Mt Fujiyama landscape.[11] There are two reasons to suppose that adaptive evolution does not occur on Fujiyama landscapes: one is a reason to suppose it doesn't, the other a reason to suppose it couldn't. Darwin and

[11]The imagery is taken from Kauffman (1995, ch. 8).

his predecessors, recall, were struck so profoundly by the staggering diversity of adaptive forms. There isn't just one solution to the problems of surviving and reproducing, there are enormously many. The biological world is replete with all sorts of adaptations. Intermediate states between these adaptations are not adaptations at all.[12] So it looks like an explanation of adaptive diversity requires us to think of the adaptive landscape as multiply peaked. A second reason is that on a Fujiyama landscape, even very slow rates of mutations would cause a population not to climb to a peak, but to seep down the slope. The vast majority of mutations would represent a lower altitude on the adaptive landscape. Every mutation would cause the population to move on average downhill. Where the selection coefficients are low—where the slopes are shallow—the effect of the accumulation of mutations would not be new adaptations but a decrease in the average adaptedness within a population. Populations would exhibit little tendency to cluster around the single adaptive peak (Wright, 1932).[13] If adaptive evolution is to occur, then populations must inhabit reasonably highly-correlated, multiply-peaked landscapes.

The adaptive landscape that a population inhabits determines its capacity to undergo adaptive evolution. But what determines the sort of landscape a population inhabits? The landscape is just an abstract representation of the relative fitnesses of each (total) genotype. Fitness, recall, is the propensity of an individual to survive and reproduce as determined by the systematic causal contributions of its heritable traits. Ultimately, it is the nature of the processes which determine the propensity of *individuals* to survive and reproduce which determines the kind of adaptive landscape a population inhabits. So, ultimately it is the make-up of individuals in a population that determines whether the population is capable of undergoing adaptive evolution.[14]

How far have we got? We started with Darwin's question: 'how have all these magnificent adaptations arisen?'. So far we have the following answer:'through the differential survival and reproduction of individual organisms, populations of which inhabit multiply-peaked, highly-correlated landscapes'. Now it seems we have a corollary question: '*What sort of things are individual organisms such that populations of them inhabit these fitness landscapes*?'. In a sense this latter question is more fundamental than Darwin's—an answer to it is a prerequisite to an answer to Darwin's question. I shall call it 'Kauffman's Question'.

3. Kauffman's Question

Kauffman lays out his views of the source of adaptation in his *Origins of Order* (1993) and later in his more accessible book *At Home in the Universe* (1995). The

[12]This point is amply demonstrated by the studies of morphological variation in Darwin's finches. See Weiner (1995) for a synopsis.

[13]I thank Alexander Bird for helpful discussion on this point. There is a further (better) reason to suppose that adaptive evolution cannot occur on Mt. Fujiyama landscapes. I discuss this below.

[14]There are other contributing factors, particularly mutation rate. I shall discuss this below.

earlier book begins with a question remarkably reminiscent Darwin's. Kauffman asks 'What are the sources of the overwhelming and beautiful order which graces the living world?' (p. xiii). His answer, very roughly, is that the order found in the organic world, the diversity of adaptive responses to evolutionary problems, is largely a consequence of the intrinsic natures of individual organisms. Organisms, according to Kauffman, are self-organising, complex dynamical systems. Order and adaptation are consequences of the basic principles of self-organisation in these sorts of complex systems.

Kauffman accepts much of the machinery of the modern-synthesis theory of evolution: mutation, selection, drift. He even construes selection as a force acting upon populations. But he sees the principles of self-organisation in complex systems as both a constraint on and a prerequisite to the creative power of selection. Acknowledging that the complex adaptations of organisms such as neural and immune systems are the result of mutation and selection, he asks:

> How can such wonderful systems emerge merely through random mutation and selection? For if Darwin told us that adaptation occurs through the gradual accumulation of useful mutations, he has not yet told us what kinds of systems are capable of accumulating useful mutations. (Kauffman, 1993, p. 173)

The systems in question, of course, are organisms. I take it that one may paraphrase Kauffman's question as: *What sorts of things are organisms, such that selection of and mutation within them eventuates in adaptive evolution*?

This is a striking departure from the approach we find in the standard accounts of the modern-synthesis theory of evolution. Rather than starting from the structure and the statistical dynamics of populations of genes, a perspective taken by the modern synthesis theory, Kauffman's approach focuses on the organisation and development of individual organisms. Far from being 'pretty much irrelevant' to the explanation of adaptive evolution, the nature of individuals is one of its principal determinants. Whereas on the traditional modern-synthesis view, the order we see emerge during the development of organisms is strictly the causal consequence of selection, Kauffman maintains that this order is more properly understood as a consequence of the self-organising properties of extremely complex systems.

> I present a countering thesis: most of the beautiful order seen in ontogeny is spontaneous, a natural expression of the stunning self-organization that abounds in very complex regulatory networks. We appear to have been profoundly wrong. Order, vast and generative, arises naturally.... I propose that much of the order in organisms may not be the result of selection at all, but of the spontaneous order of self-organised systems. (Kauffman, 1993, p. 25)

If we want an account of biological order, then, we should look not to some force driving a population, but to the processes of self-organisation found in the development, survival and reproduction of individual organisms.

The principles of self-organisation are becoming increasingly well understood, largely through the study of Artificial Life. As Boden characterises it:

> The central concept of A-life, excepting *life* itself, is *self-organization*. Self-organization involves the emergence (and maintenance) of order, or complexity, out of an origin that is ordered to a lesser degree. (Boden, 1996, p. 3, emphasis in original)

Of course, being a self-organising system isn't sufficient for being the kind of system populations of which can undergo adaptation evolution. The crystals and Benard cells discussed above are examples of the spontaneous emergence of structured entities from less structured, simple components, but again there is no adaptation here. What more is needed to make a self-organising system the kind of thing populations of which are susceptible to adaptive evolution? Here again, Kauffman offers us a distinctive answer:

> To engage in the Darwinian saga, a living system must first be able to strike an *internal* compromise between malleability and stability. To survive in a variable environment, it must be stable, to be sure, but not so stable that it remains forever static. Nor can it be so unstable that the slightest internal chemical fluctuation causes the whole teetering structure to collapse. (Kauffman, 1995, p. 73)

The key concept here is *homeostasis*. An organism must be able to maintain a stable state despite perturbations, both internal and external. But mere stability is not enough. Homeostasis involves responding to internal and external perturbations by means of compensatory changes. If a system is too robust it cannot implement compensatory changes; if it is too labile any internal change or external perturbation will cause the complete collapse into disorder. Those systems best able to withstand and compensate for perturbations are neither too rigidly ordered nor chaotic. They are 'poised on the edge of chaos'. Such poised systems, says Kauffman, 'appear to be best able to co-ordinate complex, flexible behavior and best able to respond to changes in their environment'. (Kauffman, 1993, p. 29)

In fact, among self-organising systems, living things seem to be distinguished by the possession of an extremely finely-honed capacity for maintaining homeostasis, of withstanding and adapting to internal and external perturbations. This distinctive capacity has led Marc Bedau to propose that this is what constitutes being a living thing:

> The essential principle that explains the unified diversity of life seems to be the *suppleness* of the adaptive processes—its unending capacity to produce novel solutions to unanticipated changes in the problems of surviving, reproducing, or, more generally, flourishing. Some forms of adaptations are rigid, such as those exemplified by artefacts like street lights or thermostats which have strictly limited options. By contrast, supple adaptation involves responding appropriately to an indefinite variety of ways to an unpredictable variety of contingencies. (Bedau, 1996, p. 338)

Organisms are, in short, *complex, self-organising, adaptive systems*. It is this capacity to maintain and produce order through adaptive responses which makes an organism an organism.

A-life models can simulate the conditions required for a self-organising system to be capable of supple adaptation. Return to the model of haploid organisms having N genes, each with two alleles, so that there will be 2^N possible genotypes. We may

define a parameter K as the degree of interaction between genes within a genotype: *epistasis*.[15] Where K is equal to 0, there are no interconnections between genes, and the phenotypic effects of each gene are entirely independent of all others. Where $K = N - 1$, the phenotypic effects of each gene are influenced by every other. Where there is no epistasis, developmental systems are robust and ordered, incapable of adaptation. These systems settle into stable configurations from which they are not deflected by perturbations (in the form of mutations). Where there is high epistasis, developmental systems are chaotic; once perturbed, these systems do not settle into a stable configuration. Where the degree of epistasis is small, systems are capable of undergoing adaptation. They can sustain the development of reasonably stable structures, despite the presence of a considerable number of mutations. Some perturbations cause these systems to settle into new stable states. 'This is a first hint of something like a construction requirement to make complex systems with many interacting parts which remain perfectible by mutation and selection. Each part should directly impinge on rather few other parts'. (Kauffman, 1993, p. 67).

Studies in molecular genetics bear this out. Most enzymes exhibit an enormous amount of mutational variation, most of which has very little effect on fitness (Kimura, 1968). Only occasionally will these mutations bring about phenotypic differences. Most of these will be deleterious, but some will enhance fitness. Lewontin (1974) calls this variant on the modern theory of selection which allows for the accumulation of numerous neutral mutations, the *neoclassical* theory. The importance of the neoclassical theory is that it is precisely what we should expect if individual organisms are complex, self-organising systems with low (but not *very* low) degrees of epistasis.

Finally, there is a connection between these developmental systems which are able to sustain and adapt to mutations in their developmental programmes and the sorts of populations which are capable of undergoing adaptive evolution. Populations of such individuals are precisely those which inhabit the adaptive landscapes propitious for adaptive evolution.

> We know that there is a clear link between the stability of the dynamical system and the ruggedness of the landscape over which it adapts. Chaotic ...networks ... are structurally unstable. Small changes wreak havoc on their behavior. Such networks adapt on very rugged landscapes. In contrast, ... networks in the ordered regime are only slightly modified by mutations to their structure. These networks adapt on relatively smooth fitness landscapes. (Kauffman, 1995, p. 187).

The lesson to be learned from all this is that the sort of fitness landscape which is propitious for a population to undergo adaptive evolution is determined by the generic properties of individuals which compose the population. It is not a matter

[15]Again these models are taken from Kauffman. They are a response to yet another question that Kauffman addresses: 'We must ask that which Darwin did not broach: what kinds of integrated dynamical systems harbor the ability to adapt?' (Kauffman, 1993, p. 209).

of population structure. It is a matter of the degree of integration in the development of organisms. In short, it is only populations of individuals whose developmental programmes allow them to adapt to internal and external perturbations which can undergo adaptive evolution. *The source of adaptation is the generic properties of self-organisation of individual organisms*.

4. Adaptation and Natural Selection

Where does that leave us in our initial problem of determining the relation between selection and adaptation? I think we can discern three distinct positions.[16]

The traditional modern-synthesis view is that natural selection is a force operating on an assemblage of genes. This force alters gene frequencies and by doing so *causes* adaptations to occur in a population. *Adaptation is a causal consequence of selection*. I have adduced a few reasons for supposing this picture is flawed. Natural selection seems not to have any causal efficacy of its own, at least with respect to changes in gene frequencies. If selection does not cause changes in trait frequencies then it cannot cause adaptations either, at least in the way envisaged by the traditional modern-synthesis view. As we have seen, in order to explain how selection could lead to adaptation, we need to take account of the nature, particularly the development, of the individuals it operates over.

A second view, championed by Kauffman and his commentators (Burian and Richardson, 1996; Weber and Depew, 1996), is that the principles of self-organisation somehow constrain the efficacy of natural selection; the principles of self-organisation determine the conditions propitious for selection. Yet, at the same time, selection operates as a force which tends to drive a population toward adaptive peaks. On this picture selection and self-organisation are joint causes of adaptive evolution, or, equally likely, self-organisation and selection work antagonistically in the production of adaptations. This view is a hybrid; it countenances distinct forces at two distinct levels. There is the force of selection operating over assemblages of genes—as per the traditional modern-synthesis picture—and the forces of development which determine the trajectories of individual organisms. Its most notable feature is that much of the order in the biological world, much of the adaptive complexity that we observe in individuals 'comes for free'; it arises in the *absence* of any population-level force.

This approach corrects at least one of the deficiencies of the standard modern-synthesis view by incorporating the principles of self-organisation into the explanation of adaptation. But it still accords a significant and *distinct* causal role to natural selection (Weber and Depew, 1996). In fact, natural selection is given two causal roles: i) it differentially retains in a population the better-adapted forms; and ii) it 'molds' the adaptive landscape, producing a population capable of climb-

[16]Depew and Weber (1995); Weber and Depew (1996) offer roughly similar, but more finely split taxonomies of possible positions.

ing adaptive peaks (Kauffman, 1995, p. 185). We have already seen that the first of these effects is not a consequence of selection *per se*, but simply the consequence of differential rates of survival and reproduction of individuals. If the considerations outlined in Sections 1 and 2 above are correct, this effect needs no population-level force to bring it about. But what of the second effect; how might selection 'mold' an adaptive landscape? The consequence of molding is a population which inhabits a highly-correlated, multiply-peaked landscape. But, as we have seen, the nature of the landscape inhabited by a population is determined by the nature of the individuals *in* the population. So, again, a landscape is 'molded' simply by means of the differential retention in a population of individuals capable of mounting adaptive responses to external and internal (including mutational) perturbations. The 'molding' of a fitness landscape is simply a concomitant of the first role of selection. And the first role of selection, as we have seen, is merely a consequence of the differential survival and reproduction of individuals. Here again, we need posit no distinct population-level force, or process of selection.

This presents us with a third picture of the relation of adaptation to natural selection. The discussion in Sections 1 and 2 suggests that natural selection could not be a force or causal process operating over aggregates of genes. The discussion of Section 3 suggests that no such causal process is required to explain the origin of adaptations or the tendency of populations to undergo adaptive evolution. The picture, in outline, is as follows. There is no distinct process or force of selection which causes changes in trait frequencies or adaptations. There is merely the differential survival and reproduction of individuals. The processes which operate at the individual level—those which determine the development, survival and reproduction of individuals—are themselves the causes of adaptations. I believe there is much to credit in this view but it requires a reversal of the traditional conception of the relation between natural selection and adaptation. Natural selection in the organic realm is merely a shadow cast by the differential effects of the processes of survival and reproduction which occur within individual organisms. Attempting to explain adaptation as the causal consequence of natural selection is as futile as chasing shadows. Natural selection in the organic realm, it would appear, is not the cause of adaptation. Quite the reverse, the distinctive features of natural selection in the organic world—its cumulative nature, its tendency toward increased adaptive complexity—are consequences of the fact that the individual organisms it operates over are complex, self-organising, *adaptive* systems.

5. Conclusions

There are at least three sorts of general conclusions which may be drawn from this discussion. I shall merely gesture toward them briefly.

Evolutionary theory, as I mentioned, has been formulated expressly along the model of statistical dynamics. It considers a population as an aggregate of genes. Selection works on the population by manipulating the distribution of genes in the

population. This approach to evolutionary theory abstracts away from the nature of individuals. Yet it is the nature of individual organisms which distinguishes between selection as a mere winnower and selection as a tinkerer. It is the principles according to which individuals are organised which explain the nature and origin of adaptation. For the purposes of explaining the tendency of life to exhibit 'marvellous adaptations' the basic unit of organisation should not be seen as the population but the individual. Individual thinking—not population thinking—is crucial to any understanding of adaptation. The statistical dynamics model of the modern synthesis should be replaced by the more modern complex-systems dynamics model.

Secondly, natural selection is not the cause of adaptations. It is merely the consequence of the differences between the various causal contributions made by traits to the survival and reproduction of individuals. The process of natural selection in the organic realm has some distinctive features to be sure; it is 'cumulative', it is 'creative'. But we should not be lured into supposing that it is these features which permit selection to cause adaptations in populations of individual organisms, but not elsewhere. On the contrary, natural selection in the organic realm manifests these features simply as a consequence of the generic, self-organising properties of individual organisms. In particular, natural selection occurs the way it does in the organic realm because individuals are capable of mounting adaptive responses to perturbations. This capacity to adapt allows individuals to survive in unpredictable environments and to reproduce with startling fidelity, despite the presence of mutations. It is adaptation which explains the distinctive features of natural selection in the organic realm and not the other way round.

Finally, this view has implications for naturalised teleology in biology. If selection does not cause adaptations then the programme of reductive teleology cannot be successfully carried out. That programme, recall, rests on two theses: i) that natural selection is the cause of adaptation; and ii) that to explain the presence of a trait by appeal to what it is an adaptation for is merely to advert to some effect of natural selection. The first of these theses, I have suggested, is false. If so, the second probably is too. There is no obvious non-teleological reduction of adaptive explanations to be had. Faced with unreduced teleology, we have a couple of options. One is to deny that adaptive explanations are teleological at all (cf. Cummins, 1975). The other is to accept the account of adaptation suggested by complex systems dynamics, viz. that adaptation emerges as a consequence of the principles of self-organisation in complex systems. This yields an account of the origins of biological teleology—adaptation—without a reduction of teleological explanation. We have unreduced, naturalised biological teleology. Perhaps it is time to re-evaluate the anti-Aristotelian purge in the natural sciences.

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