

ry rates are similar, either the characters are controlled by the effects of the same genes or they contribute to adaptive changes that are subject to the same or similar selection pressures.

This modal theme is important because it reverses an emphasis on constancy of evolutionary rates that, in the past, many evolutionists have accepted almost intuitively. In addition to seeking explanations for differential rates of evolution, we should also explain as well as possible why certain organs, tissues, cells, or molecules evolve at similar rates. Clearly, neither dissimilarity nor similarity can be ascribed necessarily to different rates of mutation, although these may sometimes be involved. The stability over millions of years of bodily form in horseshoe crabs and lungfishes, of leaf structure in lycopods and ferns, of cell structure in blue green bacteria, and of amino acid sequences in histone molecules must have persisted in spite of mutations, because mutant forms have been continuously rejected by normalizing selection. Similar rates of evolution on the part of different structures need not always have a basis in natural selection, but at least the possibility of its action must be explored. At the macroevolutionary level, this kind of exploration requires close collaboration among population geneticists, developmental morphologists, comparative cytologists and biochemists, and paleontologists. The ultimate syntheses will not be the products of a single mind.

CONCLUSION

The following conclusions may be reached. First, evolutionary biology is so complex that attempts in the near future to build syntheses around the framework of rigid, all-inclusive generalizations or laws will continue to be self defeating and will lead to disputes and confrontations that generate more heat than light. Second, the framework of modal themes possesses a flexibility that enables more realistic syntheses to be elaborated. Third, this framework has been operating intuitively in the evolution of concepts of Mendelian genetics. Fourth, current disputes, such as adaptiveness vs. neutrality, geographic vs. sympatric speciation, gradualism vs. punctuated equilibria, and evolution of regulatory vs. structural genes, will be resolved more successfully on the basis of modal themes than by polarization in defense of rigidly held generalizations. Finally, at the beginning of the 1980s evolutionists find themselves in one of the most exciting fields of expanding knowledge.

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ADAPTATION AND THE INTEGRATION OF EVOLUTIONARY FORCES

Alan R. Templeton

INTRODUCTION

Evolution is often subdivided into anagenesis, or evolution within a phyletic line, and cladogenesis, or the splitting of one phyletic line into two or more lines. The major feature of anagenetic evolution is adaptation: that is, the process by which a population acquires traits that tend to enhance survivorship, mating success, and/or fertility with respect to a particular environment. (I use "adaptation" in the evolutionary sense rather than in the physiological sense.) Although Darwin entitled his book *Origin of Species*, his volume dealt primarily with the origin of adaptations. Of course, the phrase "origin of species" as used by Darwin referred primarily to the phyletic transformation of a species through time (rather than cladogenetic processes), and he viewed the process of adaptation as being primarily, but not exclusively, responsible for this transformation. Considerable attention has been paid to cladogenetic origins of species during this century, and one of the more common views regards speciation as a pleiotropic side-effect of adaptation occurring independently in two populations separated by some extrinsic barrier to gene flow (usually geographical). Under both of these views of origin of species, adaptation is clearly the primary anagenetic and cladogenetic agent. This idea has recently been challenged, with many evolutionists now claiming that both speciation and the origin of major features in macroevolution

...tle, if anything, to do with adaptation (Lewin, 1980). The major ... of evolution under this view are attributed instead to the pro- ... of speciation (which is regarded as involving mechanisms that ... qualitatively different from those occurring during evolution within a species) and higher level processes such as "species selection" (Stanley, 1979).

In order to evaluate these claims, the mechanisms underlying adaptation and their implications must be examined very carefully. I feel the rejection of the importance of adaptation in speciation and macroevolution has often been based on an overly simplistic view of the adaptive process. I also firmly believe that speciation can under some circumstances be effectively decoupled from adaptation, but the implications of these circumstances are such that they represent a most unlikely explanation for most macroevolutionary trends (Templeton, 1980a). Consequently, I will argue that the interment of adaptation is premature and that adaptation is still alive and well and playing a critical role in the origin of species in both the anagenetic and cladogenetic senses.

SOME RARELY TOLD TALES OF SICKLE-CELL

I begin my argument by examining the mechanisms underlying adaptive evolution. To aid this examination, I will use an example—sickle-cell anemia. Although sickle-cell has become a standard example of adaptation, there are aspects of this system that are rarely told but which nonetheless offer critical insight into the mechanisms of adaptive processes.

Sickle-cell refers to an allele at the β -chain hemoglobin locus in man. This allele confers resistance to malaria when either homozygous or heterozygous (with the predominant "A" allele) and hence can greatly reduce preadult mortality (most deaths due to malaria occur in infants or children) in the malarial regions of the world. Unfortunately, another pleiotropic consequence of this allele when homozygous is a severe hemolytic anemia, known as sickle-cell anemia, which kills most homozygotes before adulthood. These effects of the sickle-cell allele (*S* allele) are reflected in the relative viability fitness estimates given in Table 1 for a West African population (based on data from Cavalli-Sforza and Bodmer, 1971). This table also gives the viabilities of the genotypes associated with a third allele at the β -chain locus, the *C* allele. The *C* allele also provides malarial resistance, but unlike *S*, it is a recessive allele for malarial resistance relative to the *A* allele. However, homozygosity for *C* is not associated with a severe anemia, and perhaps such homozygosity confers a degree of malarial resistance far superior to that displayed by *AS* heterozygotes (the fitnesses are measured with error, but for illustrative purposes, I will

treat the values in Table 1 as known constants hereafter). As a result, the *CC* genotype has the highest fitness: 1.3 compared to 1.0 for *AS*, the next highest. Finally, the *C* allele when coupled with *S* does lead to a hemolytic anemia that significantly lowers viability, although not nearly as severely as sickle-cell anemia.

Before the Bantu-speaking peoples of Africa expanded into west-central Africa, malaria was not an important selective agent in their environment; but with the Bantu expansion and with the introduction of slash-and-burn agriculture an extreme malarial environment was rapidly established (Weisenfeld, 1967). Initially, these Bantu populations most likely had near fixation of the *A* allele, with *S* and *C* both being very rare. However, the fitness alterations induced by the introduction of malaria lead to evolutionary changes that greatly altered these initial allele frequencies. The following model quantifies these alterations. Letting p = frequency of *A*, q = frequency of *S*, r = frequency of *C*, and W_{ij} = fitness of genotype ij , and assuming a single, infinite-sized population with random mating and discrete generations, the equation for the change in the frequency of *S* over one generation (Δq) is:

$$\Delta q = \frac{pqW_{AS} + q^2W_{SS} + qrW_{SC}}{\bar{W}} - q = \frac{q}{\bar{W}}(a_S) \tag{1}$$

where \bar{W} is the average fitness of the population at this locus and $a_S = pW_{AS} + qW_{SS} + rW_{SC} - \bar{W}$. Note that whether *S* increases or

TABLE 1. Relative fitnesses of some genotypes at the β -globin locus in West-African populations of man. The fitness of the *AS* heterozygote is arbitrarily set to one. Fitness differences are primarily due to the listed conditions affecting preadult viability. The fitnesses are estimated from data given in Cavalli-Sforza and Bodmer (1971).

Genotype	Fitness	Condition
AA	0.9	Malarial susceptibility
AS	1.0	Malarial resistance
SS	0.2	Anemia
AC	0.9	Malarial susceptibility
SC	0.7	Anemia
CC	1.3	Malarial resistance

S is dominant
C is recessive

decreases is determined only by the sign of a_S . The biological meaning of a_S is straightforward. Given a gamete bearing an S allele in a random mating population, that S gamete will pair with an A -bearing gamete with probability p to produce an AS genotype with fitness W_{AS} . Similarly, with probability q it will be in homozygous state with fitness W_{SS} , and with probability r that S gamete will be in an SC genotype with fitness W_{SC} . Therefore, a_S represents the average fitness that an S -bearing gamete will display after fertilization, minus the average fitness of the population. More technically, a_S is the conditional expected fitness deviation given that one gamete involved in zygote formation bears the S allele. The quantity a_S allows us to perform a rather remarkable trick—with it one can assign a fitness value to a haploid gamete as a function of the fitness effects it will display in diploid genotypes. This is a very important step, for, as Darwin pointed out, the only aspects of fitness that are important in adaptation are those aspects which can be transmitted to the next generation—or in more modern terminology, only the fitness effects that can be transmitted through a haploid gamete to the next diploid generation. It is precisely these fitness effects transmissible through a gamete that are measured by a_S .

The quantity a_S also has a well-defined meaning in terms of classical quantitative genetics: it is the average excess of the S allele. This definition emphasizes the fact that fitness is a phenotype and not some special entity that must be treated as qualitatively different from other phenotypic measures. Moreover, like any other phenotype at the level of whole-organism organization, fitnesses are not inherited; rather, responses to environments are inherited. Because fitness differences can alter the genetic composition of a population through natural selection, the external environment plays a direct role in shaping the evolutionary fate of the population. Indeed, this is the very essence of adaptation, as shown very clearly by equation (1). If fitness phenotypes were determined solely by the genotype rather than being inherited environmental responses, there could be no adaptation.

A final advantage of representing the allele frequency changes in terms of average excesses is that the resulting equation is very general. Virtually all classical population-genetic equations describing natural selection can be regarded as special cases of this formulation. Thus, it is not necessary to teach 1,001 Δq equations: one is sufficient. Moreover, the average excess formulation makes it clear that any factor influencing the manner in which phenotypes at one generation are transmitted through gametes to produce phenotypes in the next generation can play an active role in the adaptive process. Natural selection obviously is an important determinant of this process as it can greatly influence the types and quantities of the gametes passed

on to the next generation. Moreover, in the absence of different genotypic fitnesses, there can never be nonzero average excesses of fitness phenotypes. Thus, natural selection is *necessary* for adaptive evolution, but it is *not sufficient* to define an adaptive process, since other factors can and do influence the average excess. Thinking of adaptation only in terms of natural selection can be erroneous and misleading. I will now illustrate this by returning to the sickle-cell example.

As argued earlier, the premalarial Bantu population most likely had $p \approx 1$, $q \approx 0$ and $r \approx 0$. When the malarial environment was induced, these initial conditions yield $\bar{W} \approx 0.9$. Hence, the average excess of the S allele under random mating is

$$a_S \approx (1)(1.0) - 0.9 = 0.1 \quad (2)$$

Thus, from equation (1) the frequency of S should increase, and because equation (2) is so large this initial increase will be quite rapid. This occurs because under random mating and with S initially rare virtually all S -bearing gametes become incorporated into AS genotypes with malarial resistance. On the other hand, consider the average excess of the C allele under these same initial conditions:

$$a_C \approx (1)(0.9) - 0.9 = 0 \quad (3)$$

Because the C allele is recessive for malarial resistance, virtually all C -bearing gametes are incorporated into malarial susceptible genotypes when C is rare. Actually, the average excess of C is slightly positive when no approximations are used, but the average excess of S will still be many orders of magnitude greater than that of C . Therefore, the initial adaptive response to malaria is a rapid and large increase in the frequency of S , but practically no change in the frequency of C . Obviously, dominance and recessiveness are playing critical roles in the initial adaptive response.

S increases so rapidly, that soon a quasi-equilibrium is established with A and S in a balanced polymorphic state ($p \approx 0.89$, $q \approx 0.11$) but with C still extremely rare ($r \approx 0$). At this point, the average excesses of the S and C alleles become

$$\begin{aligned} a_S &\approx (0.89)(1.0) + (0.11)(0.2) - 0.91 = 0 \\ a_C &\approx (0.89)(0.9) + (0.11)(0.7) - 0.91 = -0.03 \end{aligned} \quad (4)$$

Note that the average excess of the S allele is zero, reflecting its equilibrium situation with A . At this point, although the genotypes associated with the A and S alleles certainly display different fitnesses, A - and S -bearing gametes transmit identical fitness effects to the next generation. Because of this, natural selection now operates

as a force preventing evolution rather than causing it. On the other hand, the increase in frequency of *S* has changed the average excess of the *C* allele from 0 in equation (3) to a negative value in equation (4). This occurs because now *C* alleles are frequently incorporated into *SC* heterozygotes which have a very low fitness due to anemia. Since the *C* allele is still rare, its beneficial homozygous effects are virtually irrelevant. The net result is that now natural selection is operating to eliminate the *C* allele. Thus, starting with the initial conditions described earlier, the adaptive outcome to a malarial environment is to evolve a balanced polymorphism of the *A* and *S* alleles and to eliminate the *C* allele.

Recall that the *C* allele is associated with the genotype with highest fitness in this environment; yet natural selection insures that the fittest genotype is eliminated in this case. So much for the phrase "survival of the fittest." Moreover, note that there are basically two adaptive options to malaria: (1) become polymorphic for *A* and *S*, in which case only about 20 percent of the population is protected from malaria with the remaining 80 percent either susceptible to malaria or afflicted by an extremely deleterious genetic disease; or (2) become homozygous for *C*, in which case all of the population enjoys a malarial resistance greatly superior to that of *AS* heterozygotes and without suffering from a genetic disease. Option (1) increases average fitness from 0.9 to 0.91; option (2) from 0.9 to 1.3. I am sure most people would regard option (2) as the optimal one; yet, this is precisely the option that is eliminated during adaptation. So much for equating adaptation to "optimization."

The elimination of the *C* allele illustrates very well the fact that adaptation cannot be understood solely in terms of individual phenotypes. We must never forget that *adaptive processes are manifest only at the level of a population reproducing through time*, and individual fitnesses are only one factor of many influencing these processes. This point can be made more clearly by considering a hypothetical inbred Bantu population.

I will now assume a nonrandom mating population with an inbreeding coefficient of $f = 0.05$. All other assumptions will be retained. Thus, at the individual level, nothing has been altered. Under inbreeding, an *S*-bearing gamete will join with another *S*-bearing gamete identical-by-descent with probability f . Even if not identical-by-descent (an event of probability $1-f$), an *S*-bearing gamete can still pair with another *S* allele with probability q . Any heterozygotes involving *S* obviously must be from the noninbred portion $(1-f)$ of the population. Hence, under these rules of pairing,

$$\begin{aligned} a_S &= (1-f)pW_{AS} + [f+(1-f)q]W_{SS} + (1-f)rW_{SC} - \bar{W} \\ a_C &= (1-f)pW_{AC} + (1-f)qW_{SC} + [f+(1-f)r]W_{CC} - \bar{W} \end{aligned} \quad (5)$$

Under the initial conditions of $p \approx 1, q \approx 0, r \approx 0$ and $\bar{W} \approx 0.9$ with $f = 0.05$, equations (5) become

$$\begin{aligned} a_S &\approx (0.95)(1)(1.0) + (0.05)(0.2) - 0.9 = 0.06 \\ a_C &\approx (0.95)(1)(0.9) + (0.05)(1.3) - 0.9 = 0.02 \end{aligned} \quad (6)$$

In this case the initial adaptive response is to increase the frequencies of both the *S* and *C* alleles. As *C* increases in frequency, more and more weight is placed upon its highly advantageous homozygous effects, but as *S* increases more and more weight is placed upon its highly deleterious homozygous effects. Hence, initially a_C increases but a_S decreases with time. Moreover, the increased frequency of *C* soon pushes the value of \bar{W} beyond the maximum obtainable by an *A-S* polymorphism, causing the *A* and *S* alleles to acquire negative average excesses. Thus, in this case, natural selection results in the fixation of the *C* allele and the elimination of the *A* and *S* alleles.

Note that in both the inbreeding and random-mating examples, the initial gene pools were identical, the environmental alteration was identical, and the individual genotypic fitness responses were identical, but the adaptive outcomes were totally different. Obviously, the course of adaptation cannot be predicted from even a total knowledge of how individual genotypes respond to environments to produce fitness phenotypes. This result is not surprising in light of the average excess formulation, because system of mating is patently a critical determinant of the types and frequencies of the various genotypes into which a particular gamete type becomes incorporated. Thus, the course of adaptation cannot be explained in terms of natural selection alone.

Another way of expressing this idea is in terms of Wright's (1932) adaptive topography concept. A plot of \bar{W} for all possible values of p, q and r would define a topography with two adaptive peaks under random mating: one of height 0.91 at $p = 0.89, q = 0.11$ and $r = 0$, and a second of height 1.3 at $p = q = 0$ and $r = 1$. The contours of the landscape are such that natural selection insures that any population starting at a point near $p = 1$ climbs the lower peak. Under inbreeding, the lower peak is altered to a height of 0.907 and a location of $p = 0.93, q = 0.07$ and $r = 0$, but more importantly the contours near $p = 1$ are also altered so as to insure that natural selection now causes the population to climb the higher peak. Thus, the adaptive topography is a function not only of the genotypes' fitness responses to an environment, but also of the population's system of mating. An alteration of the system of mating can potentially modify an adaptive topography as much as an alteration of the external environment. This is an important conclusion that I will use later.

As mentioned before, system of mating is not the only factor besides natural selection that is an active determinant of adaptive processes; any factor influencing phenotypic transmission through gametes is also part of the adaptive process. I will now turn to some of these other factors, starting with genetic architecture and following with the joint interactions of genetic drift and gene flow.

GENE AND SUPERGENE

An important determinant of what aspects of the phenotype are transmissible through a gamete is the genetic architecture underlying the phenotype. The number of loci, their linkage relationships, pattern of epistasis, and so forth are not just trivial complications of adaptive evolution—they are often critical determinants. A good example of the importance of genetic architecture is provided by polymorphic mimicry systems in several species of butterflies (Charlesworth and Charlesworth, 1975). In these mimicry systems, it is commonly observed that the genetic basis underlying the phenotype is a group of several alleles at different but closely linked loci that interact with one another to produce the mimetic phenotype. Such gene complexes are known as supergenes.

Charlesworth and Charlesworth have considered how such complexes could have evolved. The process begins with a major locus causing a noticeable but imperfect degree of mimicry. Suppose, for example, such a mutation causes a change in ground color from black to orange. Even though imperfect mimicry results, natural selection will often favor the increase of such an allele. Now consider, for example, a mutation at a second locus that causes a row of white spots to appear on the forewing. Further assume that if these spots appear upon a black wing they merely make the organism more conspicuous, but if they appear on an orange wing they make the mimicry more perfect. Thus, on the black background this allele is deleterious, on the orange it is beneficial. Charlesworth and Charlesworth (1975) showed that the necessary conditions for increase of such an allele when rare depend critically upon the recombination frequency with the first locus: the tighter the linkage, the more likely the second allele will be favored by natural selection. If linkage is too loose, the allele at the second locus is eliminated by natural selection, despite the fact that it increases the perfection of mimicry. Needless to say, the second allele must arise in (or recombine into) coupling with the allele for orange color.

It is also commonly observed that mimetic phenotypes in different species have totally different biochemical or developmental bases. Thus, consider the situation of a major mimicry locus with modifier mutations occurring at loci scattered throughout the genome. Each of

these mutations will be associated with its own set of pleiotropic consequences, some potentially deleterious. Suppose a mutation at a modifier locus closely linked to the major mimicry locus occurs, and a mutation at an unlinked modifier locus also occurs that results in the same mimetic phenotype. Suppose further the linked locus is associated with some deleterious physiological effects whereas the unlinked locus is not. Nevertheless, the model of Charlesworth and Charlesworth (1975) predicts that the modifier at the linked locus may have a much greater chance of being incorporated into the adaptation than the more "optimal" unlinked modifier.

Charlesworth and Charlesworth (1975) therefore concluded that supergenes evolve because "the only modifier mutations which can escape elimination are ones which occur at loci which are fairly closely linked to the mimicry locus. This would obviously lead to a condition in which one region of a particular chromosome, around the original mimicry gene, contained all the loci affecting the mimetic pattern." Moreover, they show that the scheme in which loosely linked modifiers are put together by translocations and recombination modification is extremely unlikely. If appropriately placed loci did not exist a priori, such an adaptation would be impossible. Thus, these adaptive mimicry complexes can only evolve *because* of the genetic architecture.

The genetic architecture also interacts with the intensity of selection in determining adaptive outcomes. Most mutations with major phenotypic effects have many pleiotropic effects that are primarily deleterious. Indeed, sickle-cell is a good example of this. However, if selection upon one particular pleiotropic effect is strong enough, it may be sufficient to cause the allele frequency to increase, despite the deleterious nature of the other pleiotropic effects. Once again, the increase of the *S* allele because of its malarial resistance is a good example of this phenomenon. Once such a major allele has increased in frequency, selection will favor modifiers that suppress or circumvent the deleterious pleiotropic effects. Sickle-cell provides an excellent example of this phenomenon as well.

Malaria probably became an important selective agent in Central Africa only about 1,500 to 2,000 years ago (Weisenfeld, 1967). However, malaria has probably been selectively important in parts of the Middle East and India for considerably longer. The interesting fact is that the *S* allele is found in high frequency in these areas of the world as well; it is not, as commonly thought, an allele confined to black populations of African origin. However, the *S* allele has certainly attracted more attention in these black populations than in the Caucasian populations because the deleterious effects associated with sic-

selection for recombination rate

kle-cell anemia have been either eliminated or greatly reduced in clinical severity, apparently due to the action of alleles at other loci in these Caucasian populations (Pembrey et al., 1980; Roth et al., 1980). Similarly, that other classic example of adaptive evolution—industrial melanism—also falls into this same pattern: the initial adaptive response is through a major locus whose expression is subsequently modified by the action of other loci (Kettlewell, 1973). Additional examples of this pattern are discussed in Templeton (1982).

The course of adaptive evolution suggested by the above examples has been confirmed by computer simulations performed by Drs. Edward Spitznagel and Theodore Reich at Washington University (personal communication). In their simulations, the initial response to selection was almost solely at the major locus which underwent rapid alteration of allele frequency until it reached a quasi-equilibrium state. Subsequent evolution of the system was more gradual and less dramatic and occurred primarily through the minor modifier loci.

One major locus has a considerable dynamic advantage over many minor loci affecting the same phenotype in the face of intense selection. The average excess at any particular locus will be quite small if the selected trait is determined by many loci, each with small additive effects, even with strong selection. Hence, the allele frequencies will change very slowly. However, with a major locus, the fitness effects are channeled primarily through a single gamete type, and hence the average excess can be quite large even in the face of deleterious pleiotropic side-effects. Hence, under intense selection, small additive polygenes often lose out to major genes as the primary cause of adaptive change (given, of course, that such genetic alternatives exist), even though the major genes often bring with them deleterious side-effects which must be eliminated or circumvented by the subsequent accumulation of modifiers. However, if selection is not intense, it is unlikely that a single pleiotropic trait of the major locus could overcome all the deleterious pleiotropic traits. In this case, the polygene architecture would most likely evolve. Note that in both of these cases the genetic architecture underlying the adaptation arises, not because it is the only genetic basis allowable for the adaptive trait, but because it is the genetic basis that interacts with natural selection to allow that particular course of adaptation. Hence, genetic architectures can play an active, not passive, role in adaptive evolution.

THE PEAKS AND PITS OF ADAPTATION

Genetic drift and gene flow are two other evolutionary forces that can have a major impact on the types and frequencies of genotypic combinations that a particular gamete-type enters into. The role of gene flow and genetic drift as agents in adaptive evolution—not neutral

evolution—was emphasized most strongly and effectively by Wright (1932) in his shifting balance theory. Since many misconceptions exist concerning this theory, I will now briefly discuss it.

Wright's knowledge of developmental genetics convinced him there are generally many ways of adapting to an environment, although pleiotropy insures that they are rarely equivalent in a fitness sense (recall the two unequal fitness peaks for malarial adaptation in man). Wright illustrates this conclusion with the adaptive landscape concept with its adaptive peaks of unequal height separated by adaptive valleys. His theory was primarily concerned with how balanced genetic systems could effect a shift from one peak to another in light of the fact that such peak shifts would be opposed by natural selection. However, the phrase "shifting balance" can also be used to describe the manner in which basic evolutionary forces—natural selection, genetic drift, and gene flow—interact with one another upon an adaptive landscape. To illustrate this, I will draw an analogy that is in some ways the inverse (quite literally) of Wright's original analogy of the adaptive landscape, but one for which a physical model can be easily constructed as an aid to understanding. [M. J. Kottler has brought to my attention the fact that Wright (1960) also used a similar, but not identical, analogy to the one I give below.]

Consider turning an adaptive landscape upside down, thereby transforming peaks into pits and valleys into ridges. Now let a ball correspond to a deme (*not* an individual or locus). When this ball (deme) is placed upon the inverted adaptive landscape and released, gravity will cause it to roll down to the bottom of the nearest pit, not the deepest, just as natural selection causes the demes to climb the nearest adaptive peak, not the highest. (Recall the example of sickle-cell.) Now, put some lateral motion into the balls by randomly shaking the inverted landscape. This causes the balls to roll around, even up the sides of the pits against the force of gravity, just as random genetic drift causes demes to move around the adaptive landscape, even in directions opposed by natural selection. The intensity of the shaking corresponds to the strength of genetic drift; that is, the more the inverted landscape is shaken, the smaller the deme sizes in Wright's model. During this shaking process, some balls will actually roll up the side of a pit and over a ridge, at which point gravity once again causes the balls to roll to the bottom of a new pit. This corresponds to an adaptive peak change.

As the shaking process continues, the balls preferentially come to be located in the deeper pits. The reason for this is very straightforward—it is harder to roll out of a deep pit than a shallow pit; hence,

as the balls roll around the inverted landscape, the ones in shallow pits are very likely to continue rolling into different pits, but the ones landing in deep pits are unlikely to make any further transitions. Another way of saying this is that there is a shift in the balance between the relative importance of gravity and random shaking in determining the movement of the balls, depending upon whether the balls are in a shallow or a deep pit. Similarly, there is a shift in the balance between natural selection and genetic drift as a deme makes transitions from peaks of unequal height and steepness. The result of this shifting balance is that populations go preferentially from lower to higher peaks through time.

Many people portray Wright's theory as if genetic drift induces peak transitions at random, and uniformly through time. However, what Wright realized is that genetic drift plus natural selection would consistently cause evolution from low to high adaptive peaks. Hence, adaptive evolution is far more efficient when natural selection is *not* in sole control, even though, paradoxically, natural selection is the only force actually necessary for adaptation. Similarly, gravity plus random shaking is a far more effective procedure than just gravity alone in getting all the balls to the bottoms of deep pits in my inverted landscape; yet gravity is the only force that actually causes the balls to roll to the bottom. It took the genius of a mind like Wright's to come up with such a simple yet subtle insight into the nature of adaptive evolution.

Finally, note that in shaking such an inverted landscape, pit changes (adaptive peak transitions) are very common at the beginning of the process, but as more and more of the balls come to lie in deep pits such transitions become less likely, often until there are no pit changes at all, even though the amount of random shaking may be constant throughout the entire process. This, of course, is also a result of the progressive shift in the favor of gravity in the balance between gravity and shaking. Similarly, in Wright's theory, peak shifts become less likely as the shifting balance process operates through time even if the deme sizes remain constant.

Moreover, there are further attributes of the shifting balance model that accentuate this tendency toward stasis that are not readily modeled by balls rolling in an inverted landscape. Demes are on many peaks during the initial phases of the process; hence, gene flow between them frequently acts as a random perturbing factor aiding genetic drift. But as the shifting balance process proceeds and more and more demes end up on the highest peak or a small set of high peaks, gene flow becomes more and more of a deterministic force attracting demes to the highest peaks. As more demes are brought to a single peak by the action of this type of gene flow, the more gene flow between demes acts as a factor in maintaining all the demes on

that single peak, particularly if the demes on the high peaks produce most of the emigrants (Wright's "interdemic selection"). In other words, gene flow becomes more and more of a static force reinforcing natural selection and less and less of a perturbing force reinforcing genetic drift as the shifting balance process operates through time. This, of course, accentuates the shift in the balance between selection and drift, and thereby accentuates the tendency to go from dynamism to stasis. Wright (1932) has also argued that as the demes move onto the higher adaptive peaks their population sizes might also tend to increase, thereby decreasing the importance of genetic drift.

Thus, the shifting balance theory predicts that periods of evolutionary transition will be intense but brief and lead directly to a very static adaptive situation. This stasis will only be broken if the environment (and thus the landscape) is altered, if the system of mating is altered (which, as previously pointed out, also determines the topology of the adaptive landscape), if the population structure is altered to shift the balance back toward genetic drift or disruptive gene flow, or if new genetic variability occurs that adds a new, unexplored dimension to the adaptive landscape. The shifting balance theory therefore explains why adaptive evolution is rapid and why it is static; both are caused by the same underlying mechanism. There has been a tendency to emphasize the dynamic aspect in most accounts of the theory, including Wright's, but the progressive shift toward stasis inherent in the shifting balance theory is equally important in making sense out of the relationship of adaptation to speciation and macroevolutionary patterns.

THE ORIGIN OF SPECIES—ADAPTATION DEFENDED

I will now return to the original problem outlined at the beginning of this chapter: the challenge to the position that adaptation is the primary determinant of speciation and macroevolutionary trends. In essence, the basic challenge rests upon the observation of "punctuated equilibrium" (Eldredge and Gould, 1972); that is, the observation that many species apparently remain morphologically static for long periods of time, with most evolutionary changes occurring during relatively brief periods of time. It is critical to realize that punctuated equilibrium is merely a description, not a mechanism or a process. Is this description of evolution incompatible with the implications of the adaptive process? Although some recent evolutionists have answered this question in the affirmative (Lewin, 1980; Stanley, 1979), Darwin himself predicted just such a pattern of macroevolution repeatedly

throughout his *Origin of Species* (Templeton and Giddings, 1981). It has been argued that these statements of Darwin represent a mere handful of sentences and are not really important (Gould, personal communication, and Chapter 5.) However, the bulk of the *Origin* was devoted to the proximate causes and implications of adaptation, and Darwin did not devote much space to making explicit macroevolutionary predictions. Nevertheless, whenever he did, Darwin clearly stated that rates of evolution are unequal over long periods of time and that periods of adaptive transition are short in comparison to periods of adaptive stasis. Admittedly, these represent only a handful of explicit statements, but there are *no* statements by Darwin in which he predicts that adaptive processes should occur uniformly and continuously over long periods of geological time. Thus, the description of punctuated equilibrium is certainly compatible with Darwin's view of adaptive evolution. It is also compatible with the view of adaptive evolution outlined in this chapter.

To illustrate this point, consider the classic model of speciation in which an ancestral population is subdivided into two or more isolated subpopulations by some extrinsic barrier to gene flow. Under this model, intrinsic isolating barriers then arise as a pleiotropic consequence of the adaptive processes occurring separately within the subpopulations. However, most extrinsic geographical or ecological barriers to gene flow are temporary in nature, so the chances for speciation under this model are often a function of how rapidly adaptive divergence occurs. Unless intrinsic isolation arises rapidly enough, the extrinsically isolated subpopulations will simply fuse together upon secondary contact.

I have discussed more fully elsewhere (Templeton, 1982) the factors influencing the speed of adaptive divergence; here, I will only mention two. Adaptive divergence will be most rapid if one or both of the extrinsically isolated subpopulations is subjected to novel and intense selective pressures—a proposition with considerable empirical support (Templeton, 1981). New and intense selective pressures not only make speciation more likely in the quantitative sense that they simply speed up the rate of adaptive change, but also in a qualitative sense because of the types of genetic architecture that emerge under intense selection.

Recall that intense selection favors major genes that often have many deleterious pleiotropic effects that induce secondary adaptive processes at modifier loci. On the other hand, a less intense selective regime favors polygenes of small phenotypic effect. Hence, for the same degree of phenotypic response on a selected trait, a regime of intense selection not only achieves the response more rapidly than the less intense regime, but it also induces more major pleiotropic alterations while achieving this response. Moreover, intense selection on

a major locus creates conditions optimal for hitch-hiking effects. A potential example of this is provided by malarial adaptation on the island of Sardinia. In this case, intense selection favoring the X-linked allele causing glucose-6-phosphate dehydrogenase deficiency (another malarial adaptation) might have caused a hitch-hiking effect at the closely linked locus causing Protan color-blindness (Filippi et al., 1977). Thus, with intense selection, many more direct and indirect phenotypic alterations are expected on traits other than those being selected as compared to more moderate selective regimes. Hence, intense selection is far more likely to result in pleiotropic isolating barriers, and thereby speciation, because of both quantitative and qualitative considerations.

Recall also the dynamic response to intense selection: the bulk of the adaptive change occurs extremely rapidly, followed by a long period of near stasis with only minor adaptive adjustments. Moreover, the extrinsic factors that split a population in the first place are often the very same factors that induce environmental changes (e.g., see Axelrod, 1981, on the role of climatic shifts upon plant speciation in California). Therefore, the bulk of adaptive divergence under this scheme occurs shortly after the extrinsic split, followed by relative stasis. This, of course, fits the descriptive pattern of punctuated equilibrium very well.

A second determinant of the speed of adaptation is population structure. Wright (1932) predicted that adaptive evolution would be most rapid when the population is subdivided into small demes with restricted gene flow between demes. Such a structure allows his shifting balance process to operate. However, as I argued earlier, most species displaying the appropriate population structure for shifting balance would still be characterized by stasis and not continual peak transitions. Therefore, for rapid adaptive divergence to occur and lead to speciation, one or more of the extrinsically isolated subpopulations must undergo the transition from the static phase of shifting balance to the dynamic. Such a transition is quite likely for many reasons.

First, as mentioned earlier, the factors that split the population are often associated with environmental changes, and hence will induce an altered adaptive topography. Moreover, such environmental changes often induce a reduction in numbers and density of the affected species. Density changes can directly alter the system of mating, and, moreover, the nature of the relationship between density and system of mating is often nonlinear (Anderson, 1980), so that drastic alterations can sometimes occur even with a relatively modest change in density. As illustrated with sickle-cell, alterations in the system of

mating also alter the adaptive topography. In addition, density changes will often directly affect gene flow patterns and deme sizes, and thus could also aid in the transition to dynamic shifting balance by altering the balance between selection, drift, and gene flow.

Finally, the very act of splitting the ancestral species via extrinsic barriers obviously alters gene flow patterns. The implications of such a split is particularly important when one realizes that population structure is rarely homogeneous throughout the ancestral species. For example, Wright (1978) discusses studies on the Mojave Desert plant, *Linanthus parryae*, which indicate the neighborhood sizes in the center of the species range are of the order of 100, but in the periphery they are of the order of 10. In general, ecologically peripheral populations will often show lower deme sizes and more extinction and recolonization than central populations. As a result, the central populations should be very static under the shifting balance theory, whereas the marginal populations have far more potential to enter the dynamic phase of shifting balance. Nevertheless, this potential is often not realized because gene flow from the more numerous central populations represents a strong conservative force that can maintain the static phase of shifting balance even in the marginal areas and that can interfere with local adaptation. However, this static situation can be radically altered if gene flow from the central populations is severed, and it would also seem reasonable to assume that most geographical and ecological barriers that split a species would preferentially occur between marginal and central populations. Once gene flow from central to marginal areas has been severed, the marginal (but not the central) population could enter into the dynamic phase of shifting balance, particularly since the severing of gene flow also allows more effective local adaptation and thus the exploration of a somewhat novel adaptive landscape. Hence, ecologically and/or geographically marginal populations will often play a critical role in the speciation process resulting in a peripatric pattern of species distributions.

Note that in all of the above cases the agents causing the extrinsic split of the ancestral species are also the very same agents that can trigger the transition from static to dynamic shifting balance in one or more of the isolated subpopulations. Such a triggering should then lead to a phase of very rapid adaptive transition shortly after the split that quickly evolves into stasis. By concentrating most of the adaptive changes into a short period of time following the extrinsic split, the shifting balance mode of adaptive evolution greatly increases the chances for speciation, given a temporary extrinsic barrier. Moreover, it is patent that the resulting temporal pattern of evolutionary change is once again very consistent with the descriptive pattern of punctuated equilibrium. However, speciation is a pleiotropic by-product of adaptive processes in all the speciation models I have discussed above.

Consequently, the inference that the pattern of punctuated equilibrium indicates that adaptive processes are unimportant in speciation or macroevolution is not valid. This inference is based upon a simplistic caricature of the adaptive process called "phyletic gradualism" (Stanley, 1979). When adaptive processes are treated in a more rigorous and detailed fashion, no incompatibility is apparent. Consequently, I agree with Darwin that adaptation has indeed played a critical role in the origin of species.