Environmental Stress as an Evolutionary Force

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Stressful environmental conditions can be defined as those that lead to a sharp reduction in fitness in populations. That is, when changed environmental conditions cause a drastic reduction in reproductive output, and when persistence of the conditions leads to permanent damage, these conditions constitute an environmental stress. Physical stresses that are encountered rarely in populations—such as periods of drought or extreme cold—or that are encountered by a minority of a species—such as in populations that are located at distribution borders or are exposed to local chemical stresses arising from human activities—can, through their direct or indirect effects, lead to marked reductions in the size of populations (Glynn 1988, Hoffmann and Parsons 1991) and to repeated cycles of colonization and extinction (Andrewartha and Birch 1954).

Because of their effects on fitness, stressful conditions can be extremely effective in shifting the mean of a trait by imposing directional selection. There are many examples of such shifts in natural populations (Hoffmann and Parsons 1997), including responses to selection arising from human activities, such as the evolution of pesticide resistance in insects and the evolution of resistance to heavy metals in plants and invertebrates (Macnair 1993, McKenzie and Batterham 1994). In addition, there is evidence for rapid shifts in morphological traits due to periodic exposure to climatic stresses, particularly in bird populations. For instance, selection led to increased body size in Darwin's finches after approximately 80% of the population died during a drought (Grant and Grant 1989); selection also increased body size in cliff swallows during an extreme cold spell that resulted in more than 50% mortality (Brown and Brown 1998).

However, although stressful conditions can in some instances lead to rapid evolution, stress is not necessarily required to explain the observed rates of evolutionary change in the fossil record and in historic times. Selection experiments performed in the laboratory have shown that most traits can respond fairly rapidly to directional selection, even when selection intensities are only moderate. The experiments of Weber on a range of traits in Drosophila (e.g., Weber 1990, Weber and Diggins 1990) illustrate the large phenotypic changes in morphological and physiological traits that can be achieved through stress selection. Selection responses in such laboratory experiments involve evolutionary rates that far exceed those seen in the fossil record (Gingerich 1983). Thus, moderate or even weak selection (i.e., selection imposed by factors other than stress) may be sufficient to account for observed rates of evolution in the fossil record and in historic times.

In this article, we explore ways in which stressful conditions may stimulate evolutionary change and explain evolutionary patterns other than by simply increasing the selection intensity on some traits. Although stresses can be physical or biotic, we focus on physical stresses. We first argue that evolutionary stasis may be common in populations in the absence of periodically stressful conditions. Populations may be prevented from adapting to environmental changes because of the effects of gene flow, deleterious mutations, tradeoffs, and lack of variability. We then show how stressful conditions can provide a way of overcoming this stasis and promoting adaptive changes. They do so by promoting the expression of variability in traits, influencing adaptive changes by restricting gene flow, and...
ternary showed that few beetles exhibited morphological changes over this time period, whereas there were marked changes in the distribution of beetle species, suggesting that species shifted distributions instead of evolving when climatic changes occurred. For mammals in the Eocene and lower Oligocene (37–30 million years ago), when marked climatic change took place, only 3 out of 177 species showed continual morphological changes; for most species there was no morphological change throughout this period, despite shifts in distribution (Prothero and Heaton 1996). There are many additional examples of fossil assemblages that show little morphological change and virtually the same species composition over extended periods (Schopf 1996).

Of course, the absence of morphological change in a lineage does not necessarily imply the absence of evolutionary change, particularly because physiological adaptation can occur independently of morphology and therefore not be reflected in the fossil record. One way of discerning the extent to which physiological evolution has occurred is to consider whether changes in distributions involve entire species assemblages (Coope 1979). If so, then large-scale physiological evolution seems unlikely because not all species possess the same ability to adapt physiologically, Schopf (1996) and Coope (1979) emphasized that groups of organisms in the fossil record tend to occur in the same assemblages despite environmental changes and that even the dominance of certain species appears constant over an extended time. However, other authors have emphasized that species tend to respond individually rather than as an assemblage; such responses may reflect physiological adaptation without morphological change (Graham 1992, Nowak et al. 1994).

Limits to morphological and physiological adaptation are evident in extant populations as well as in fossil studies. Although many pest species have evolved resistance to agricultural chemicals, others have failed to evolve resistance, even when chemicals have been applied for many years. Limits are also evident at species borders, which have tended to be relatively constant over many decades (Bull 1991). In the absence of evolutionary limits to adaptation, borders would be expected to continually shift in response to evolutionary changes in geographically marginal populations. Instead, ecological range expansion appears to be rare.

Given the evidence for adaptive limits, how can the apparent stasis of fossil assemblages and the inability of populations to overcome marginal conditions be explained? One possibility is that there is insufficient genetic variability for adaptive changes to occur. This hypothesis appears to run counter to both the efficacy of laboratory selection in changing population means and the high level of genetic variability that is common in natural populations (Nevo 1988). Nevertheless, a limitation of laboratory selection experiments is that they have tended to focus on traits that can respond to selection. Many traits exhibit an extremely low level of variability and do not respond readily to selection. Some examples include floral morphology in Linanthus, vibrissa number in mice, and embryonic developmental rates in Drosophila (see references in Scharloo 1991, Hoffmann and Parsons 1997).

Moreover, even when genetic variability is present in populations, it may not be used. For example, it may be of the wrong type, as illustrated by the evolution of insecticide resistance (Roush and McKenzie 1987). In field populations of insects, resistance is usually determined by a single gene. However, there are situations in which resistance has not evolved or has not persisted, despite the presence of genetic variability. In such cases, it appears that resistance is determined by several genes, each of which has relatively small effects—polygenes (McKenzie et al. 1980). A high intensity of selection provides an explanation for the finding that resistance does not evolve when it is determined polygenically. Selection for resistance is intense in the field, where mortality levels need to approach 100% to achieve effective control of pests. When the selection intensity is extremely high, only individuals carrying major genes may have sufficient high resistance levels to survive pesticide application, whereas under weak selection all genes can contribute. A similar argument has been made for the evolution of resistance to other toxicants, such as heavy metals (Macnair 1991). That is, resistance is more likely to evolve when it is determined by major genes than when it is determined by several minor genes.

Major genes are also more likely to confer resistance to chemical stresses than are minor genes because of gene flow (Roush and McKenzie 1987). When susceptible individuals migrate into a population that had previously been exposed to chemicals, the influx of susceptible alleles will dilute the frequency of resistance genes. This dilution effect is likely to be much greater when resistance arises from a combination of many minor genes (i.e., polygenic
When individuals with polygenic resistance mate with susceptible individuals, the gene combination needed for resistance will be lost in the ensuing generations, and the probability of recovering offspring with polygenic resistance is therefore low. This dilution effect has a much lower impact when resistance is conferred by one or a few major genes. For instance, when a single recessive allele controls resistance, one-fourth of the F2 progeny of a cross between resistant and susceptible individuals will be homozygous for the resistance allele. However, when four genes are involved, only 1 in 256 offspring will be resistant. Thus, for traits where variation in populations is polygenic, it may be difficult to achieve a response to selection, even when ample genetic variation exists.

Gene flow may also prevent adaptive divergence among populations. The role of gene flow in limiting adaptation in a population has been recognized for a number of traits and organisms (Storfer 1999), including color pattern variation in snakes and moths, survival in fish, clutch size in birds, and anti-predator behavior in salamanders. Moreover, even when selection produces differences among populations, these differences may not persist. Futuyma (1989) has suggested that local selective forces change over time, which, in combination with gene flow, breaks down differences between populations. Geographical differences may persist only if strong reproductive isolation develops between populations.

The effect of gene flow in limiting adaptive changes is likely to be particularly important at the periphery of a species' distribution. Gomulkiewicz and Holt (1995) and Holt and Gaines (1992) have emphasized that natural selection acts as a conservative force, increasing fitness in the environments commonly encountered by organisms but not in more marginal environments. Marginal conditions are experienced by the minority of members of widespread species, particularly because the density of species decreases toward the margins (Gaston 1990). As a consequence, margins act as sinks for centrally distributed populations. Most populations therefore fail to adapt to marginal conditions, becoming well adapted only to common conditions experienced away from the margins.

Moreover, the effects of gene flow in limiting adaptation can be influenced by tradeoffs. In general, increased fitness in unfavorable environments is associated with decreased fitness in favorable environments. For instance, resistance to desiccation and starvation stresses in *Drosophila* is often associated with an increase in lipid and glycogen storage or with a decrease in metabolic rate (Hoffmann and Parsons 1989, 1991, Chippindale et al. 1996, Djawdan et al. 1996). These physiological changes lead to a decrease in early fecundity, an increase in larval development time, or both, decreasing the overall fitness of organisms under optimal conditions. There is supporting evidence for such tradeoffs in insects and other animals (Hoffmann and Parsons 1991, 1997), although few field tests have been undertaken. In plants, the ability to persist in infertile soils is often associated with low growth rates, which reduce competitive ability under favorable field conditions (Grime 1979, Lambers and Poorter 1992). The fact that genes favored under marginal conditions are often associated with low fitness under favorable conditions, and vice versa, will increase the impact of gene flow on the inability of marginal sink populations to adapt.

Finally, recent evidence indicates that the effects of deleterious mutations may be expressed in only some environments (Kondrashov and Houle 1994); as a result, mutations that are not deleterious in the original environment may be so in a new environment and contribute to genotype × environment interactions. Although selection will remove deleterious mutations, this process is much more efficient in environments that are commonly encountered by organisms than in those that are rarely encountered. Thus, mutations that are detrimental in a novel environment accumulate, leading to a decrease in the adaptedness of populations to rare, marginal conditions (Kawecki et al. 1997).

The outcome of all of these processes is a population that is well adapted to common conditions but poorly adapted to novel conditions or to conditions that are experienced by a minority of members of a species. That is, although natural selection can be extremely effective in causing genetic divergence, its effects are often obliterated by gene flow and fitness interactions among environments. Processes that affect evolutionary change in a population are summarized in Figure 1. The left side of Figure 1 depicts processes with negative impacts on evolutionary changes in a population. These negative impacts include gene flow from nearby populations, an increase in the load of deleterious mutations, and the presence of tradeoffs between traits and environments. Adaptation can also be limited by a lack of genetic variation. But the right side of Figure 1 shows that stressful conditions can also have a potentially positive impact on evolutionary change, as we now discuss.

**Stress and the expression of phenotypic and genetic variation**

The idea that evolution mainly involves sudden and large phenotypic shifts because of mutations with large effects ("hopeful monsters") was promoted by Goldschmidt (1940) and by some developmental biologists. However, this view has been dismissed by neo-Darwinists as incorrect (Charlesworth 1982) because mutations with large effects are often rapidly selected against in populations due to their low fitness.

Nevertheless, it does appear that large phenotypic shifts can be triggered in populations by repeated exposure to stressful conditions. It has long been known that specific stresses can lead to the increased expression of phenotypic variability (Figure 2), particularly in morphological traits that are normally invariant. Classic examples include
the duplication of the thoracic segment and alteration of wing venation patterns in Drosophila triggered by exposure to chemical and high-temperature stresses (Milkman 1960, Waddington 1961). Phenotypic variants can be selected for and increase in frequency in a population as long as they have a genetic basis, and they can eventually become expressed even in the absence of the stress, a process Waddington (1961) referred to as "genetic assimilation." In the case of one phenotypic variant, in which a duplicated thoracic region is triggered by ether, the molecular changes underlying this assimilation process have been found to involve polymorphisms in the Ultrabithorax gene (Gibson and Hogness 1996).

A simple mechanism by which genetic assimilation might occur (Bateman 1959) involves a threshold model (Figure 3), whereby a phenotypic variant is expressed only under a certain level of stress, which then allows alleles producing the variant to be selected. As these alleles increase in frequency, they pass another threshold (the "normal" threshold in gene frequency in Figure 3), which results in the expression of the phenotypic variants in the absence of the stress. Thus, stressful conditions can serve to expose phenotypic variants that can then be selected in populations, leading to evolutionary change.

One problem with making general conclusions based on these early experiments is that stresses involving particular chemicals or heat applied to specific life cycle stages were required to create phenotypic shifts by genetic assimilation. However, Rutherford and Lindquist (1998) have recently proposed a more general mechanism for producing morphological variants under stress. They investigated the effects of mutations in one of the Drosophila heat-shock protein genes, hsp83, on morphological abnormalities. This gene codes for the Hsp90 protein, which normally acts to ensure that signaling proteins in cells remain stable. The authors showed that these mutations (as well as geldanamycin, a biochemical inhibitor of Hsp90) release hidden morphological variation that can then be exposed to selection; once morphological variants are selected, their expression no longer depends on the presence of mutant hsp83 alleles.

The mechanism by which this process could occur is as follows: cellular stress may cause a transient decrease in Hsp90 levels because these proteins are titrated by stress-damaged proteins. The reduction in Hsp90 would then lead to the increased expression of morphological variants; eventually, the variants would be expressed even in the absence of changing Hsp90 levels if alleles that increase the expression of Hsp90 accumulate. This mechanism is similar to the threshold model for genetic assimilation in Figure 3, except that any environmental variable affecting Hsp90 levels could lead to increased variability. Rutherford and Lindquist (1998) suggested that this process could facilitate rapid morphological radiation. However, as in the case of genetic assimilation, this process would still require the morphological variants to have a fit-
Figure 3. A threshold model to account for genetic assimilation. (a) Before selection, a phenotypic variant is expressed under stressful conditions when a genetic threshold is exceeded, but not under normal conditions, which are associated with a different threshold. The genetic threshold is determined by the number of alleles present that lead to the expression of the variant. (b) Continued selection for the individuals expressing this variant leads to an increase in the frequency of the alleles responsible for the expression of the variant. The distribution of the population is altered, and the variant is eventually expressed even when stress is not experienced.

ness advantage. In addition, processes that act against evolutionary change, such as gene flow, deleterious mutations, and tradeoffs between traits and environments, would still need to be overcome.

Stressful conditions can also influence evolution by increasing mutation and recombination rates. In the laboratory, organisms exposed to stressful conditions can produce more variable offspring than organisms not exposed to such conditions because of an increased incidence of mutants and new combinations of genes (Parsons 1988, Hoffmann and Parsons 1991). There is also evidence that mutation rates differ between natural populations exposed to different levels of stress. For instance, Lamb et al. (1998) studied mutation rates in a fungus (Sordaria fimicola) from two sides of a canyon. The south-facing slope is warmer, drier, and much more variable than the north-facing slope, which experiences mild and relatively constant conditions. Mutation rates were threefold higher in fungal strains originating from the southern slope than in strains from the northern slope; some of this variation was environmentally induced, but a component was genetically determined, persisting through two generations of selfing. Thus, both induced and inherent mutation rates were higher in strains from the more stressful environment. Evidence that recombination frequencies in natural populations may also be associated with stress levels comes from the finding that, in the mole rat, Spalax ehrenbergi, increasing aridity stress is associated with increasing levels of recombination, as measured by chiasma frequency (Nevo et al. 1996).

Increases in the rates of both mutation and recombination could potentially enhance rates of evolutionary change. When populations are under continuous directional selection, there is good evidence that new mutations play a major role in the response to selection (Frankham 1980). Moreover, increased recombination rates are also associated with rapid evolutionary responses in the laboratory (Flexon and Rodell 1982). Thus, increases in mutation and recombination rates under stress could enhance rates of evolutionary change.

However, changes in mutation and recombination rates under stress are not necessarily adaptive. Some changes in mutation rate have been linked with the activation of transposons that insert into the DNA of a host organism and thereby affect gene expression. There is evidence that the rate of transposition events increases under some forms of environmental stress (Ratner et al. 1992). In addition, the increase in mutation and recombination rates may be an indirect consequence of organisms having fewer resources available for DNA repair processes when they are under stress.

Moreover, costs, as well as potential benefits, are associated with an increase in mutation rate. Because most mutations are deleterious, an increase in mutation rate generates a mutational load in a population. One way that organisms decrease this load is via directed mutagenesis, a process that produces mutations that increase fitness specifically in response to the stressful conditions an organism is experiencing. Evidence that such mutations exist has come mainly from microorganisms that have auxotrophic mutations and are therefore unable to grow without a particular nutrient unless a reversion event occurs. Whether the available data support the existence of a process that produces these mutations has, however,
been hotly debated (Lenski and Mittler 1993, Hall 1998). The general consensus is that mutations are probably not directed in the sense of arising to deal specifically with particular environmental conditions. Instead, it appears that stressful conditions increase mutation rates to a greater extent in specific classes of genes that sometimes, but not always, include those under selection. A number of mechanisms could explain this process. For instance, Wright (1997) has proposed that stress increases the rate of transcription of some genes, particularly those under selection; these genes are in turn more likely to mutate because DNA that is actively transcribed is particularly vulnerable to mutagenesis as a consequence of being single stranded for much of the time.

Although these results and others (see Hoffmann and Parsons 1997) demonstrate that stress affects phenotypic and genetic variation, there remains a large gap between demonstrating these effects and showing that they have a role in adaptive evolution. Almost all of the abnormal phenotypes generated in laboratory experiments are unlikely to survive in nature. In addition, a few abnormal phenotypes produced by localized stresses are likely to be diluted by gene flow in populations, unless the phenotypes have large positive effects on the fitness of organisms.

One way to explore whether stress has more general effects on phenotypic variation and hence on adaptive change is to consider stress effects on variation in quantitative traits known to be under selection. To this end, a number of experiments have investigated the heritability of morphological and life-history traits under both stressful and optimal conditions. “Narrow-sense” heritability expresses the proportion of variation in a trait that contributes to phenotypic similarity between parents and their offspring; it is therefore an important measure for predicting the effects of natural selection across generations. Narrow-sense heritability is defined as $V_p/N_p$, where the additive genetic variance is $V_A$ and the phenotypic variance is $V_p$. Another commonly used measure is the “broad-sense” heritability of a trait, which is defined as $V_c/N_p$, where $V_c$ is the genetic variance, which includes nonadditive as well as additive genetic effects. The broad-sense heritability of a trait provides an indication of the proportion of phenotypic variance that is genetic, but this measure is less useful than narrow-sense heritability for predicting evolutionary change because not all sources of genetic variation contribute to the similarity between parents and offspring.

If the heritability of a trait under directional selection is increased by stressful conditions, then that trait is likely to evolve more rapidly than a trait whose heritability is not increased by stress. Unfortunately, the results of experiments testing this idea have been inconclusive (Hoffmann and Merila 1999). Several studies in Drosophila suggest that the broad-sense heritability of traits may be increased under high temperature or nutritional stress, but there are also exceptions to this pattern. Other studies, particularly in birds and plants, suggest that both the broad- and narrow-sense heritability of morphological traits may decline under unfavorable conditions. For life-history traits, the data are also inconclusive. In a Drosophila study that considered the effects of a complex stress arising from a combination of cold stress, poor nutrition, and ethanol (Sgrò and Hoffmann 1998), heritable variation for fecundity increased under stressful conditions. However, other studies have shown a decrease in the heritability of fecundity and development time under stressful conditions (Kasule 1991, Imasheva et al. 1998).

One limitation of studies on stress and heritability is that they have tended to focus on traits with moderate heritabilities and fairly high levels of phenotypic variation. Studies on characters with low heritabilities and low phenotypic variances should produce more consistent changes in $V_A$ with stress. It would also be informative to test whether traits that have reached a selection plateau (i.e., traits that fail to respond to further directional selection) can continue to evolve after stress exposure. Ideally, such experiments need to be undertaken using traits that are known to have undergone adaptive shifts in natural populations rather than morphological traits of unknown adaptive significance.

Overall, the effects of stress on variation suggest a much more dynamic interaction between the environment and genetic systems than has previously been appreciated. This environmental emphasis is also becoming apparent in epigenetic inheritance systems (Jablonka and Lamb 1998). These systems are associated with cell memory and allow cell phenotypes that are induced by environmental changes to be transmitted across generations. Examples of epigenetic inheritance systems include the many genes influenced by methylation patterns. The environment can influence the methylation patterns of these genes, and their methylation state in turn influences their transcription. Methylation patterns can be stable and inherited across many generations. The environment therefore not only acts on variation via natural selection but also helps to determine the expression of genetic and phenotypic variants that can then be exposed to selection.

In summary, some processes can result in stress having a positive effect on evolutionary rates (Figure 1). When a stressful environment is encountered, it can trigger an increase in the rate of mutation, transcription, and recombination, all of which, in turn, can lead to increased phenotypic variation exposed to selection. Moreover, stress can trigger the expression of variation in invariant traits that can then be exposed to selection.

**Stress, gene flow, and population size**

In addition to influencing levels of variability, stress may also indirectly influence rates of evolution by countering the limiting effects of gene flow on adaptation. Stressful conditions are likely to decrease gene flow among populations because these conditions result both in species
becoming increasingly restricted to favorable habitat patches and in fewer migrants (i.e., because of a declining reproductive output). Genotypes with a high fitness in marginal conditions may then increase in frequency because they are no longer being diluted, leading to a positive effect on adaptation. García-Ramos and Kirkpatrick (1997) have modeled a situation in which a quantitative trait is under clinal selection in a species whose density decreases toward the periphery of a population. They showed that once peripheral populations become isolated, selection can act within a few generations to shift the population mean several standard deviations away from that of the original population. Therefore, restricting gene flow can have a positive impact on adaptation.

Unfortunately, there have been few attempts to rigorously test the importance of this process. As mentioned earlier, many examples exist of gene flow apparently restricting adaptation. In addition, there is direct evidence that restricting gene flow can lead to rapid evolutionary shifts, albeit not in the context of stress. For instance, Riechert (1993) found that desert spiders (Agelenopsis aperta) from a riparian habitat appeared to behave maladaptively in that they showed low levels of prey discrimination, even though prey were abundant and high levels of prey discrimination were therefore expected. The authors hypothesized that the evolution of discrimination was limited by asymmetric gene flow with a much larger population in the surrounding desert region, an environment where prey were scarcer and low levels of discrimination were therefore expected. When gene flow was artificially restricted between the desert and riparian populations for a generation, prey discrimination in the riparian population increased markedly. That is, the restriction of gene flow triggered an adaptive response.

The effects of stress on gene flow could help to account for the high rates of evolution and diversification that are often seen in disturbed environments. Examples include the transitional zone in southwestern Australia (Hopper 1979) and the arid Succulent Karoo Region in southern Africa (Ihlenfeldt 1994). Such high-diversity regions are associated with long-term disturbance resulting from periodically stressful conditions, which in turn are likely to have led to marked changes in the degree of isolation and size of populations. However, it is also possible that other direct and indirect effects of stress have contributed to diversification in such regions.

The effects of stress on gene flow and population size can have negative as well as positive effects. If isolated populations become too small, then genetic variation can become lost and \( V_A \) is expected to decrease. The only exception is if epistatic interactions occur, which can result in a release of genetic variability following a population bottleneck (Goodnight 1988). A small population size will also lead to inbreeding depression, which occurs when there is an increase in the frequency of genotypes homozygous for recessive deleterious alleles in a population. There is good evidence from agricultural studies (Barlow 1981) that inbreeding depression is increased under stressful conditions. This pattern is also apparent in more recent experiments with other organisms, including Drosophila, Tribolium flour beetles, birds, and plants (Miller 1994, Bijlsma et al. 1997, Keller et al. 1998).

In summary, stressful conditions may indirectly increase rates of adaptation when gene flow is disrupted by the fragmentation of populations but decrease adaptive responses if there is a persistent reduction in population size (Figure 1). More information is needed to test whether adaptive changes are commonly enhanced by a reduction in gene flow. For instance, it would be interesting to test whether isolating populations at the margins of species distributions results in their adapting to marginal conditions.

**Stress and the persistence of genetic variability**

Periodically stressful conditions can increase evolutionary rates by enhancing the expression of fitness differences among genotypes and phenotypes. Several studies, particularly in bacteria, have shown that variation among genotypes coding for enzymes appear to have no fitness consequences under optimal conditions, whereas fitness differences among these genotypes become evident under more stressful conditions (Hartl et al. 1985). In addition, it appears that the positive association between the heterozygosity of individuals (as measured at several enzyme loci) and fitness, which is often weak (David 1998), can become stronger under moderate levels of stress. Examples include an increase in the effect of heterozygosity on the fitness of the earthworm Eisenia fetida under moisture stress (Audo and Diehl 1995) and on the fitness of the clam Mulinia lateralis under temperature and salinity stress (Scott and Koehn 1990).

The fitness effects triggered by stressful conditions can influence the persistence of genetic variation in populations and thereby indirectly affect evolutionary rates. Stress effects on persistence can occur in two ways: heterozygote advantage and, perhaps more important, genotype \( \times \) environment interactions. When heterozygote advantage occurs at a particular locus, multiple alleles are maintained in a population. Because the positive association between heterozygosity and fitness tends to be stronger under stressful conditions than under favorable ones, stress may promote the persistence of genetic variation. However, heterozygous advantage can maintain multiple alleles at a locus only under fairly restrictive conditions (Karlin and Feldman 1981).

Stressful conditions can also promote persistence when genotype \( \times \) environment interactions result in different genotypes having relatively higher fitness in different environments (Gillespie and Turelli 1989). The evidence for tradeoffs between fitness in unfavorable and favorable environments, as mentioned above, suggests that genotype
As mentioned earlier, microevolutionary processes document the evolution of organisms (Nevo 1998). For instance, in subterranean and climatically unpredictable areas (Nevo et al. 1994), genetic variation, there should be an association between levels of genetic variation and the likelihood of populations encountering such conditions. Nevo and coworkers have accumulated evidence of this association in a number of organisms (Nevo 1998). For instance, in subterranean mole rats, allozyme diversity is positively correlated with aridity stress, increasing from a stable area toward an arid and climatically unpredictable area (Nevo et al. 1994).

However, it should be emphasized that there are likely to be negative as well as positive effects of stressful conditions on genetic variation. If these conditions persist and population numbers are reduced, levels of genetic variation may be lowered via genetic drift, decreasing the adaptive potential of a population. Thus, some of the arrows in Figure 1 highlight potential negative as well as positive effects of stressful conditions on genetic variation.

**Patterns in the fossil record**

As mentioned earlier, microevolutionary processes documented within populations are sufficiently rapid to account for rates of evolutionary change in the fossil record (Gingerich 1983) without the need to invoke a role for stressful conditions. Nevertheless, although patterns in the fossil record are notoriously difficult to interpret, there is some evidence that periodic stresses effect evolutionary changes and probably help to counter evolutionary stasis.

There is good evidence, particularly from marine environments extending from the shoreline (Jablonski and Bottjer 1990). New invertebrate taxa originated far more frequently in disturbed habitats close to the shore, where food was not limiting, than in the more stable but more resource-limited habitats offshore. The new taxa then expanded to occupy the other areas.

As well as being linked with the emergence of evolutionary novelties, stress has also been linked to patterns of evolution in the fossil record. Debate is ongoing about whether evolution within lineages follows punctuated equilibrium patterns (in which periods of rapid change are followed by periods of stasis) or patterns of gradual evolutionary change. Sheldon (1992) has suggested that these patterns probably depend on the nature of environmental changes. When environmental conditions change gradually and the environment does not fluctuate too greatly (i.e., periodically stressful conditions are rare), evolutionary changes will probably be gradual, whereas stasis and, occasionally, rapid evolutionary change will occur when environments fluctuate widely.

Finally, extremely stressful conditions have been implicated in promoting evolution because of the evolutionary diversification that often follows extinction events. Although these events often wipe out a large proportion of all species, they tend to be followed by the emergence of new taxa and periods of rapid evolution. A number of hypotheses (see box this page) have been proposed to explain how evolutionary diversification might occur (Vermeij 1987, Hoffmann and Parsons 1997). One possibility is that stressful conditions weaken interactions among organisms, particularly predation and competition, because population densities are drastically reduced and many populations become extinct. This phenomenon may allow novel evolutionary forms to survive stressful conditions. Another possibility is that areas that are vacant after a mass extinction event can become occupied by new evolutionary forms that are normally excluded by the presence of other organisms. In addition, the direct effects of stress in generating new phenotypic variants by the mechanisms discussed earlier may be important during periods of mass extinction. Under these hypotheses, the intense stresses leading to mass extinction events have a role in releasing constraints that normally limit adaptation.

Although fossil patterns suggest that evolutionary change is directly or indirectly affected by stressful conditions, it is nevertheless difficult to link fossil patterns with stress effects on microevolutionary patterns in extant populations because of the different time scales involved. That
is, because rapid changes in the fossil record would appear as relatively slow changes in extant populations, it is difficult to make connections between these levels. However, it does seem that major evolutionary shifts are concentrated in some types of environments, specifically environments that are relatively unstable and likely to be intermittently stressful. Direct effects of stress on phenotypic variability and indirect effects on gene flow may well play a role in promoting those shifts.

**Concluding remarks**

At this stage, the picture of how stressful conditions affect evolutionary change is still incomplete. These conditions can undoubtedly influence the expression of variation at the DNA and phenotypic levels and the expression of fitness differences among phenotypes. However, it remains to be seen if such influences can be linked to the types of stresses that occur in nature as well as to the development of adaptive differences among populations. The current focus on the molecular genetic basis of differences among species (Carroll 1995) should help to at least clarify the nature of the genes involved in adaptive differences among species and their susceptibility to processes such as genetic assimilation.

The role of the indirect effects of stress on evolutionary change will be difficult to evaluate because of the fact that stress can cause both positive and negative effects on evolutionary change. If population sizes remain small due to persistent stress, there is a danger that levels of genetic variation can be reduced and inbreeding effects increased; however, a reduction in gene flow under stressful conditions may increase rates of adaptation. One way of tackling this issue is to undertake experiments to test whether evolutionary limits in natural populations are commonly associated with restricted gene flow. Populations at species boundaries provide excellent systems with which to undertake such tests. Another approach is to take advantage of the fragmentation currently being imposed on natural populations because of human activities to see if this process influences adaptive changes in populations.

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