Role of Stress in Evolution: From Individual Adaptability to Evolutionary Adaptation

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INTRODUCTION

Environments outside the range normally experienced by a population, and the associated changes in organisms' morphological, physiological, or behavioral homeostasis (stress), accompany most evolutionary changes (Bijma and Lenschke, 1997; Hoffmann and Parsons, 1997; Hoffmann and Hercos, 2000). Depending on the intensity, predictability, and recurrence of stress, responses might range from stress tolerance and avoidance at organismal level to the rapid
appearance of novel traits or extinction at population level. Yet, moderate stress is essential for normal growth and differentiation of metabolic, physiological, neurological, and anatomical systems of an organism (Hedner, 1996; Clark and Fustic, 1998; Muller, 2003). For example, a large part of skeletal development is directed by exposure to tension and mechanical overloads in excess of those norm-
ally experienced by the organism (Halt, 1986; Carter 1987). Stress plays an important role in facilitating local adaptations by enabling better adjustments, time-
chronization, and functioning of many organ systems (Simons and Johnston, 1997; Emlen et al., 2003; Wingfield, 2003). Anyone who has experienced the
irritating effects of diving into icy-cold water after a sauna (both of which are extreme environments), the health benefits of rigorous exercise (which by definition
exceeds the range of everyday environments), or analgesic and attention-sharpening
effects accompanying stressful encounters (McEwen and Sapolsky, 1995; Shors and
Sersaous, 1997) will testify to these effects of stress. On the other hand, response to
an acute and unfamiliar stressor precludes normal organ functions (Sidky and Calos, 1989), and the high cost of stress tolerance or lack of evolved stress
response strategies can lead to evolutionary stasis (Parsons, 1994).

Extreme environments not only disrupt normal development and induce large phenotypic changes in novel directions, but they also simultaneously exert strong phenotypic selection that favors changes in these directions (Vedel, 1991; Schmalhausen, 1949; Bradshaw and Hatwick, 1989; Jablonski et al., 1995; Fishel and Mateos, 1996). Not surprisingly, evolutionary diversification, the appearance of phenotypic novelties, and mass extinction are all closely associated with extreme environmental changes (Howarth, 1993; Giese, 2001; Nicolais et al., 2003). Yet, there exists a remarkable gap in our understanding of the mechanisms behind the evolutionary importance of stress. Whereas it is widely recognized, especially in physiological and neurological studies, that stress plays an important role in directing and organizing the adaptive adjustment of an organism to ever-changing environments, very little is known about the mechanisms that enable the organismal accommodation of stress-induced effects and the evolution of a response to stress.

Lack of a developmental change or an evolutionary change of stress has led us with several unresolved questions. First, how can organisms prepare for novel and intense environmental change? The organismal ability to mount an appropriate reaction to a stressor requires recognition and evaluation of the extreme environment. How can this ability evolve in relation to stressors that are short and rare in relation to a species generation time? Second, numerous studies have documented an increase in phenotypic and genotypic variance under stress, and it is suggested that this variance is a source of novel adaptations under ever-changing environments. Yet, for stress-induced modifications to have evolutionary importance they have to be inherited and persist in a sufficient number of individuals within a population. This requires an organism to survive stress and reproduce at least once; thus, stress-induced variation has to be accommodated by an organism without reducing its functionality. How is such accommodation accomplished? Moreover, could existing organismal systems channel accumulation of stress-induced variance in some directions, but not others and thus direct evolutionary change in response to stress? The perspective outlined here, with specific focus on the effect of stress during development in animals, suggests that these questions are resolved by considering: (1) the organization of developmental systems that enable accommodation and channeling of stress-induced variation without compromising organ-
mal functionality; (2) the significance of phenotypic and genetic assimilation of neurological, physiological, morphological, and behavioral responses to stressors; as well as (3) multiple inheritance systems that transfer the wide array of develop-
mental resources and conditions between the generations enabling long-term per-
sistence and evolution of stress-induced adaptations.

I. EVOLUTION OF RESPONSE TO STRESS

A. DETECTION AND AVOIDANCE

Stress occurs when changes in the external or internal environment are interpreted by an organism as a threat to its homeostasis (e.g., Greenberger et al., 2001; McEwen and Wingfield, 2003). The ability of an organism to mount an appropriate response to potentially stressful environmental changes requires correct recognition of environmental change and the activation of a stress response (e.g., Johnson et al., 1992). The costs and benefits of stress-detection and stress response implementation and the costs and benefits of maintaining stress resistance strategies vary among environments and individuals, favoring multiple solutions of dealing with stress. Crucial to these solutions is an organism’s familiarity with the strength and types of stressors. This familiarity is determined, in turn, by the recurrence of a particular stressor in relation to a species’ generation time (Lively, 1986; Luechmann and Jablonski, 1996; Maynard, 2001; Piersma and Dent, 2003). Yet, it is unclear how the ability to recognize and assess potentially stressful environments can evolve. How can organisms judge the appropriate reaction to a stressor, such as is required to select between stressor avoidance and stress tolerance? Are the mechanisms of avoidance and avoidance specific to a particular stressor?

1. Familiarity with Stressor: Cognitive and Physiological

Assimilation of a ‘Rare Event’

The response to stress depends crucially on prior experience and a “memory” of response to a stressor. Generally, repeated exposure to a particular stressor favors the evolution of mechanisms that suppress an organism’s acute stress reaction and, instead, activate stress-specific responses (Johnson et al., 1992; Wernery et al., 2003).
For example, in higher vertebrates, stress-induced activation of the neuroendocrine system increases its reactivity to internal and external stimuli, facilitating the processing of sensory information, and ultimately enables the formation of a behavioral or physiological strategy for dealing with a stressor. Furthermore, stress-induced activation of neuroendocrinological systems facilitates long-term retention of information about a stressful event and corresponding organismal response after the stressor is gone. Interestingly, once formed, the maintenance of such "memory" can be accomplished by peripheral exposure to different stressors. For example, hormones associated with stress detection and avoidance also play a major role in modifications of neural circuits (Gold and McGaugh, 1978; once the stress-avoidance strategy is formed, exposure to even low concentrations of these hormones maintains the strategy (McGaugh et al., 1982).

Physiological studies of animals show that the repeated experience of successfully overcoming social stresses during ontogeny is a prerequisite for the acquisition of a normal repertoire of behavioral strategies (Huether, 1996; see also Gans, 1979). An insightful example comes from experiments that show that individuals exposed to repenable but consistently unfamiliar (and thus "uncontrollable") by an animal) stressors develop "stressful helplessness" i.e., they lose their ability to react to my stressor (Katz et al., 1981; Johnson et al., 1992; Avissur et al., 2001).* On the contrary, individuals that were allowed to develop a stress-avoidance strategy by exposure to a previously encountered stressor not only developed a stress tolerance to a particular stressor but also actively sought out other mild stressors. In the absence of other stressors, their stress-avoidance abilities diminished (Katz et al., 1981; Johnson et al., 1992; Avissur et al., 2001). These results suggest that, once originated, a stress-response strategy can be maintained by other environments and that adaptation to one type of stressor, at least in "social" stresses, may facilitate adaptation to other stressors.

Phenotypic assimilation of the appropriate stress response is facilitated when social roles and hormones related to the stress response are also involved in other physiological functions (Aston-Jones et al., 1986; Greenberg et al., 2001). In such cases, even a single stressful experience during development is often enough to induce changes that, in the future, will prevent organism-wide stressful reactions and activate stress-specific behavioral and physiological responses (Levine et al., 1967, 1989). Generally, stress-induced reorganization of developmental pathways and organismal function rather than the production of novel stress-specific pathways is thought to account for the ease with which individuals and populations lose and gain the ability to resist stress in laboratory populations (Chapin et al., 1993).

*Organisms "stressful helplessness" from lack of opportunity to develop stress-specific avoidance strategy is conceptually analogous to "morphological stress" of images that occur in environments with frequent acute and diverse stresses that prevent the evolution of stress-specific adaptations.

**FIGURE 13.1. Conceptual outline of the acquisition of ability to accommodate stress (solid black line) and gray area across a life span of an individual (A) normal (i.e., non-stressed) environment; (B) novel strong stressor, (C) stressful episode; (D) resilient, novel, and strong stressors; (E) "living below" stress tolerance; "Gen" (solid gray line) indicates genetic effects on acquisition of ability to accommodate stress; "exp" (dotted black line) indicates the effects of individual experience with stressor across the generation time; "age" is a duration of a single generation. gray areas show the timing and strength of a stressor.
networks (Clark and Fucito, 1998), and challenges to skeletal tissues caused by mechanical overload during growth lower developmental errors (see also Simons and Johnston, 1997; Graham et al., 2000). Wingfield 2003; Wingfield and Sapolsky, 2003) stresses stress avoidance and where suppression of organism-wide stress responses is accomplished by a blockage of either neural system providing a stressor or sensitivity of individual organism systems to stress-induced increase in circulating glucoconotrollins. Generally, when the environment during growth is a good predictor of the environment to be experienced as an adult, developmental plasticity in morpholgy and behavior can enable the accommodation of internal and external environmental fluctuations (Levine et al., 1967). Consequently, organisms activate stress reactions when there is a discrepancy between environments during their development and their current external and internal environments (Seaver, 2001; Barson et al., 2004; Weaver et al., 2004).

On the longer time scale, avoidance of a predictable stressor can be accomplished by changes in an organism's life history, especially by altering the timing of reproduction or duration of development. Common cases include stress-induced modification of the timing of metamorphosis in amphibians, changes in the duration of gestation in mammals, and the timing of flowering and seeding in plants (e.g., Bradshaw and Hardwick, 1989; Sæther et al., 2000). For example, tadpoles of several species accelerate metamorphosis when environmental changes indicate a greater probability of dehydration; this sensitivity to stressors was regulated by the corticotropin-releasing hormone signaling system (Rover, 1997). Hell et al. (2004) described evolutionary establishment of environmentally induced stress avoidance in invertebrates. In sum, initial behavioral accommodation of stress (e.g., hiding, relocation, lowering metabolism) may set the stage for the evolution of adaptive stress-avoidance strategies (e.g., periodic hibernation, migration, torpor). When a stressor is reliably preceded by other environmental changes, the mutual reinforcement facilitates the establishment of stressor recognition, assessment and avoidance strategies, and an evolved stress-specific strategy does not involve an activation of an organism-wide stress response. When individuals vary in their reaction to stress and when stress-induced strategies are favored by natural selection during and after stressful events, these strategies can become phenotypically and genetically assimilated in a population (Baldwin, 1896; Hinton and Nolan, 1980; Oyama, 2000; West-Eberhard, 2003; Figure 13-1).

II. EVOLUTIONARY CONSEQUENCES OF STRESS

A. STRESS-INDUCED VARIATION

A stress-induced increase of phenotypic and genetic variance in a population has three main sources. First, directional selection imposed by a stressor can result in faster rates of mutation and recombination. Second, stress challenges to regulatory mechanisms can release and amplify previously accumulated, but unexpressed, genetic and phenotypic variation. Third, stressful environments can facilitate developmental expression of genetic variance that had accumulated, but was phenotypically neutral, under normal range of environments. These sources of variation can be adaptive under stressful conditions when they facilitate the population's persistence through a stressful event by the development of novel adaptations to changed environments.

1. Generated Variance

Organismal reaction to a stressor is often associated with generation of variation in a directional and locally adaptive manner (Jablonka and Lamb, 1995; West-Eberhard, 2003). In some cases, such directionality is attributed to the channeling effects of complex developmental networks (e.g., Walker, 1979; Bush and Waelke, 1983). In other cases, it is associated directly with a stressful environment (e.g., Williams, 1983) or with stress effects on organismal fitness (Fridlyand and Beher, 2003). Some studies documented that an extreme environment increases genetic variation because of the increase in mutation and recombination rates (Imahara, 1999, reviewed in Hoffmann and Parson, 1997). When such mutations are directional (or "focused"), sensitive Carapace, 1990) in relation to a stressor—that is when the stressful environment both causes a mutation and favors phenotypic change associated with this mutation—such an increase in mutation rate results in greater similarity among individuals in response to a stressor facilitating evolutionary adaptation to novel environments (Shapira, 1992; Jablonka and Lamb, 1995; Wright, 2000). For example, exposing Chlamydomonas to a stressful ultraviolet irradiation increased mutation rates in traits affecting fitness (Goh and Bell, 2000). Similarly, stress-induced directional and locally appropriate mutations in bacteria (Cairns et al., 1988; Szewiowski et al., 2000; Wright, 2000; Bjedov et al., 2003). Exposure to acute stress was associated with rapid adaptive evolution of a gene family, primarily because of gene duplications, in corynbacteria (Dovermy et al., 2002), with rapid amplification of a gene in humans (Prody et al., 1989), and with greater frequency of sexual recombination in Vicia (Nielde and Michod, 2003). Other examples include long-term effects of stress on gene expression and DNA sequence, activation of previously unexpressed genes by stressful events, and stress-induced transposition in plants (Belveny and Borodin, 1982; Ruvinsky et al., 1983; McClintock, 1984; Wessler, 1996). At the level of phenotype, induction of a phenotypic trait by a stressor and concurrent selection on the induced trait are common (Jablonka et al., 1995; Oyama, 2000; Nicolakis et al., 2003; Price et al., 2003; West-Eberhard, 2003).

2. Hidden Variance

Stressful environments often reveal greater phenotypic and genetic variability than is seen under normal environments. It is commonly suggested that such hidden variation results from stress-induced challenge of preexisting genetic and developmental
architects of organismal homeostasis (Scharloo, 1991). In turn, an increase in variation in individual organismal systems and their subsequent reorganization is thought to enable the formation of novel adaptations (Bradshaw and Hardwick, 1989; Eshel and Marais, 1999; Bloom and Wagner, 2000; Lipson et al., 2002; Schlüchting and Smith, 2002; Badayev, 2004c). The idea that the extreme environment’s challenge to previously canalized systems is the source of such hidden variation is corroborated by observations of the stress-induced sudden appearance of phenotypically ancestral forms in some lineages (Gaue, 2003); by studies of phenotypic responses to stress that mimic the expression of mutation (Goldschmidt, 1940; Chow and Chan, 1990; Schlüchting and Smith, 2002); by documentation that phenotypically neutral genetic variation in ancestral forms of cultivated plants becomes highly adaptive in the hybrid backgrounds of domesticated forms (Jauker and Doebley, 2002; Riedberg et al., 2003); and by numerous examples of environment dependency in expression of genetic variation (Kondrashov and Hoyle, 1994; Leips and Mackay, 2002; Badayev and Qvarnström, 2002; Keller et al., 2002; Badayev, 2004d).

Yet, despite these examples, it is not clear how genetic and developmental systems accumulate and store phenotypically neutral genetic variation while not expressing it (Eshel and Marais, 1999; Wagner and Metz, 2000; Hermisson et al., 2003; Masel and Bergman, 2004). Specifically, the discussion has focused on the existence of “evolutionary capacitors” (Bathford, 2000) and “adaptively inducible canals” (Møller and Harl, 2002), which are specific mechanisms that buffer and accumulate developmental variation, producing “hidden reaction norms” of a phenotype. A debated question is whether “evolutionary capacitors” are stress-specific regulatory systems or whether evolutionary capacity is a property of any complex and locally adapted organismal system. Rutterton and Lindquist (1998) describe that mutations at the gene for the stress-induced chaperone protein (Hsp90) harbor abundant but normally unexpressed genetic variation that when selected leads to the appearance and assimilation of novel phenotypes in the population (Rutten et al., 2003). Thus Hsp90 might be a specialized evolutionary capacitor that buffers developmental variation but under stressful conditions facilitates adaptation (Møller and Harl, 2002). However, recent studies suggested that “evolutionary capacity” is a property of most adapted developmental systems (that when challenged by a novel environment (external or internal) reveal large genetic variation (Könchle and Gerhart, 1998; Bathford, 2000; Bergman and Siegel, 2003; Badayev, 2004a). For example, Milton et al. (2003) showed experimentally that Hsp90 is involved in buffering of only some developmental pathways and not others. Similarly, Schmidtke et al. (2001) found that as long as mutant effects are not expressed, many complex and redundant developmental systems enable accumulation of mutational variation. Thus complex developmental processes and genetic networks can constrain variation in individual traits (Rice, 2004), and phenotypically neutral genetic variation can accumulate in such systems given sufficient time and population size (Hermisson and Wagner, 2005).

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Interestingly, in many complex social networks, a stress-induced decrease in integration accelerates acquisition of a new optimum phenotype. An interesting example is the stress-induced modification of foraging and nest site searches in social insects. In some ant species, a destruction of the nest site leads to the breaking of the strict hierarchical social structure and rapid proliferation of random individual nest search routes and patterns. When a few individuals find a new suitable site, their recruitment of other individuals to follow them to the site rapidly leads to crystallization of the relocation route and movement patterns and reinstatement of the social integration of the colony (Britton et al., 1998; Costanz and Frankl, 2003).

In sum, stress resistance might be a by-product of an organism’s complexity, and accumulation of unexpressed variation by genetic and phenotypic developmental systems facilitate evolutionary change under extreme environments. Organismal homeostasis can be compromised by either novel directional selection on some organismal systems but not others, by organism-wide effects of a stressor, resulting in weaker organismal homeostasis and greater phenotypic plasticity (Schlüchting and Pignutti, 1998; Newman and Muller, 2002). Under the former scenario, a more directional and faster response to a stressor at the population level is expected because stress-induced variation will be channeled and amplified by existing functional complexes. The latter scenario should produce a greater opportunity for the evolution of morphological novelty. Overall, the weakening of complex phenotypic regulatory systems and accumulation of neutral genetic variation provides a link between diversification, evolutionary change, and extreme environments.

B. BUFFERING, ACCOMMODATING, AND DIRECTING STRESS-INDUCED VARIATION

Organisms can maintain functionality in stressful environments by channeling and accommodating stress-induced variation. This is accomplished by buffering some organizational functions while increasing the flexibility of others (Alberch, 1980; Nibert, 2002). How can such an organization evolve?

1. Stress Buffering: A By-Product of Complexity in Development or an Evolved Strategy?

Organisms functions most closely related to fitness are thought to be the most buffered against internal and external stressors (Waddington, 1941; Schmalhausen, 1949; Stearns and Kawecki, 1994). Yet, an organism’s functioning in changing environments requires the ability to track and respond to these environments. Consequently, evolved systems that shield an organism from stressors restrict an organism’s ability and capacity to adapt continuously to changing environments (Wagner et al., 1997; Eshel and Marais, 1998; Ance, 1999; Schlüchting}
and Smith, 2002). For example, suppression of stress-induced activation of the sensory systems limits an organism’s ability to acquire and retain the sensory cues and behavioral strategies necessary for stress avoidance (see preceding text: Hauter, 1996). On the one hand, a lack of phenotypic plasticity results in population extinction under stress (Gavillet and Scheiner, 1993; Ancel, 1999). On the other hand, extensive phenotypic variability in organizational functions weakens the effects of directional selection imposed by stressful environments and thus increases the opportunity for genetic assimilation and evolution of adaptations to stress (Pear and Price, 1998; Ancel, 2000; Hoesy et al., 2003). Thus, for functioning of organizational systems that are most closely related to fitness, intermediate levels of phenotypic plasticity and environmental sensitivity should be the most optimal (Behrend and Nanjundiah, 1993; Wagner et al., 1997; Ancel, 2000; Price et al., 2003). Yet, it is unclear how an optimal level of stress buffering can evolve. Specifically, is it shaped by natural selection exerted by extreme environments or by internal stabilizing selection for the cohesiveness of an organism?

Recent studies suggest that buffering is an emerging property of developmental complexity rather than an evolved stress-resistance mechanism (see preceding text): the increasing complexity of developmental pathways and networks leads directly to environmental and genetic stability and canalization (Hanz et al., 1997; Clark and Fuctio, 1998; Rice, 1998; Waxman and Peck, 1996; Meiklejohn and Hartl, 2002; Siegel and Bergman, 2003; Baden et al., 2003; Rice, 2004). Complex generic and developmental networks can accommodate the effects of stressful perturbations without the loss of function or structure, while building up neutral genetic variation (Batherford, 2000; Bergman and Siegel, 2003; Mauel, 2004).

An organism’s resistance to extreme environments depends on the historical recurrence of stressors as well as the ability of existing developmental processes to accommodate stress-induced changes (Gams, 1979, Lively, 1986; Jablonka and Lamb, 1995; Chippman, 2001; Arthur, 2002; Emelen et al., 2003). Thus differences among organisms and organizational systems in response to stress may reflect different histories of past selection. Some traits (such as foraging or sexual traits) may experience recurrent and fluctuating directional selection that favors rapid transformation in response to changing environments, whereas other parts of a phenotype might be under concurrent stabilizing selection favoring canalization (Olsen and Miller, 1998; Wagner, 2001). A combination of long-term stabilizing selection on the entire organism with strong and variable directional selection imposed by a stressor on a few organisal components should favor the evolution of modular organization where stress-induced modifications of traits can be accomplished with minimum interference with the rest of the phenotype (Simpson, 1953; Berg, 1960; Kirschner and Gerhart, 1988; Wagner and Meezy, 2004; Wagner et al., 2005; Figures 13-2 and 13-3). Persistence of such modular organization under fluctuating selection pressures is enabled by developmental complexity of its components (Baldwin, 2004a; c): such organization channels stress-induced variation while buffering
organismal components and thus enables a greater and more similar response to a stressful environment among individuals.

2. Stress Accommodation by Changes in an Organism's Integration

Organisms might accommodate stress-induced variation without the loss of function by lessening homeostasis of individual systems. Such a decrease in an organism's integrative under stress allows exploration of new environmental niches and novel solutions to adapt to these environments (Holloway et al., 1990, 1997; Rabinberg et al., 1998; Badger and Forceman, 2000; Hoffmann and Woods, 2001). For example, individual hormonal systems have a greater potential range of performances and can remain functional under a wider range of environments than is allowed by homeostasis under normal range of environments (Dixon, 1988; Johnson et al., 1992; Greeneberg et al., 2001). In other words, organism-wide homeostasis is accomplished at the expense of the potential of individual systems and components (e.g., Smith-Gill, 1983), and organisms might react to a stressor by actually weakening homeostasis. For example, frequently documented suppression of immunocompetence under stressful conditions might facilitate novel adaptations to a stressor by realizing full capacities of individual immune systems (Rabinberg et al., 1998; Avisor et al., 2001). When stress is associated with damage of tissues and accumulation of heat shock proteins, as is the case with hypertension and greater activity, suppression of immunological functions enables individual organismal systems to respond to a stressor without activation of organism-wide immunological response (Dixon, 1988; Rabinberg et al., 1998; Avisor et al., 2001).

However, there are examples of stress-induced increases in organismal integration and corresponding suppression of random genetic and developmental variation under stress (e.g., Siegel and Doyle, 1975; Bennington and McGraw, 1996; Badger and Forceman, 2000). For example, exposure to stress prevented the expression of deleterious mutations in *Escherichia coli* (Hiscoy and Lehrer, 2003). Similarly, fluctuating asymmetries of developmentally independent forelimbs and hindlimbs because integrated in bumblebees raised under stressful, but not under control conditions, apparently as a result of a greater resource exchange between different tissues under stress (Klingenberg et al., 2001). An increase in overall integration accounted for lesser phenotypic variation in the foraging structures of several mammalian species raised under stressful conditions (Badger, 1998; Badger et al., 2000). Similarly, when breeding opportunities are limited or when the benefit of the current breeding attempt exceeds the costs of stress response, organisms can "buffer" reproductive systems by blocking or reducing their sensitivity to stress or by increasing compensatory interactions within reproductive systems to counteract the stress effects on the organism (Wingfield and Sapolsky, 2003).

When stresses are mild and occur during ontogeny, individual organismal systems often accommodate stress-induced variation without the reduction in functionality.
3. Accommodation of Stressor by Channeling Stress-Induced Variation

Natural selection favors organismal homeostasis that maintains some developmental variation for adjustment of the organism to its external and internal environments (Simons and Johnston, 1997; Wigner et al., 1997; Exel and Matsui, 1998; Emlen et al., 2003; Nanjundiah, 2003). Stressed conditions can increase this variation and differences among organismal systems in their reaction to a stressor (and the corresponding channeling of stress-induced variation) might bias the introduction and expression of variation available for selection and thus bias evolutionary change (Bottner, 1965; Roth and Wake, 1985; Jablonski and Lamb, 1995; West-Eberhard, 2003).

Empirical studies show that the coordinated development of morphological traits leads to their similarity in expression of stress-induced developmental variation (Leamy, 1993; Smits et al., 1996; Badyaev and Foresman, 2000; Klingenberg and Zaklan, 2000; Klingenberg et al., 2001, 2004; Badyaev et al., 2005). Our studies of four species of shrews showed that stress-induced variation was largely confined to the directions delimited by groups of traits involved in the same function (muscle attachment) (Badyaev et al., 2000; Badyaev and Foresman, 2004). Interestingly, this channeling was concomitant with the direction of species divergence—species differed most in the traits that were most sensitive to stress within each species (Badyaev et al., 2000). These results not only confirm a strong effect of functional complexes on directing and incorporating stress-induced variation during development, but also might explain the historical persistence of complex groups of traits despite the effects of stressful environments.

C. INHERITANCE

For a stress-induced modification to be preserved in a lineage, it needs to be accommodated by an organism, and if conditions favoring this modification recur transmitted between generations, i.e., inherited. This presents two problems. First, can environmentally induced effects become inherited? Second, if an organism accommodates a stressor by different adjustments, then how can this diversify enable directional evolution of a stress-response strategy?

Stress-induced phenotypic changes commonly persist across several generations; such across-generations carry-over effects (e.g., Jablonski et al., 1995) can be caused by the transfer of physical substances, inheritance and developmental incorporation of a stressor, hormonal effects that influence expression of genetic variance in subsequent generations, epigenetic inheritance of stress-induced variation and structures, as well as behavioral effects (Jablonski and Lamb, 1995; Cyana, 2000; West-Eberhard, 2003). For example, inheritance of dominance-subdominant relationships in groups of many social mammals is accomplished by mechanisms different from original stressful encounters that established the dominance structure (Cree et al., 1996; Goymann and Wingfield, 2004). Similarly, maternal care often
sets the stage for a lifelong reaction to stressors, by modifying the expression of genes that regulate behavioral, physiological and endocrinological responses to stressors (Moura and Fox, 1998; Meaney, 2001; Bulyev, 2002; Weaver et al., 2004). Direct and indirect effects of stress on endocrinological systems often occur with significant delay after the exposure to stress and persist for a long time. This led to the suggestion that the primary function of such delayed changes is integration of past stress-induced responses and sensitization of the organism to future occurrences of similar stressors (Huether, 1996). In turn, within-generation and between-generation maintenance of stress-induced changes in neural and physiological systems is accomplished by similar hormonal mechanisms (McCaugh et al., 1982; Meaney, 2001). Poststress fluctuating environments are often different from both the environment before the stressor and the stressful environment itself and have few predictable cues to organisms that survived stressful event. Under such conditions, a short-term inheritance of developmental resources is highly advantageous (Jablonska et al., 1995). More generally, short-term and nongenetic inheritance is beneficial when the frequency of stress recurrence is greater than the generation time, but shorter than is necessary for the spread and fixation of adaptive mutation (i.e., the evolution of genetic adaptation) (Levins, 1964; Ansel Meiners and Bull, 2002).

In sum, accommodation of stress-induced variance by an organism can be facilitated by recurrent developmental stressors; genetic assimilation replaces stress-induced developmental modification, if this modification has a fitness advantage in both stressful and pestless environments (Schmalhausen, 1949; Waddington, 1952). Even when the short-term organisational responses to a stressor are not genetically heritable, differences among organisms in the ability to survive stress and the recurrence of stressful environments will catalyse stress-induced responses developmentally (Baldwin, 1886; Schlichtring and Pigliucci, 1998; Ansel, 1999; West-Eberhard, 2003).

III. EVOLUTIONARY ADAPTATION

Close association between extreme environments and the pattern and rate of adaptive evolution is one of the best-documented patterns in evolutionary biology; stressful environments unceasingly generate and amplify phenotypic and genetic variation among individuals in the population and facilitate population divergence (Hoffmann and Parsons, 1997). Unlike environmental fluctuations within a range normally experienced by a population, stressful environments modify and reorganize integrated developmental and genetic networks simultaneously in a large group of individuals, directional change produced by these networks in combination with strong and novel directional selection by stressful environment facilitates rapid evolution and diversification (Jablonska and Lamb, 1995; West-Eberhard, 2003). Moreover, when a stressor compromises an organisal trait, releasing accumulated

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and unexpressed genetic variation associated with the trait's function, such variance enhances the organisal response to selection acting on this trait (Zahavi, 1993; Robinson and Dallai, 1999; Bergman and Siegal, 2003). Example include stress-induced cartilage changes during development of bird skeletons that lead to the formation of novel structures (Muller, 2003), and stress-induced modifications in integration of foraging structures that facilitate diversification of cichlid jaw morphology (Chapman et al., 2000; Alherson et al., 2003). Moreover, extreme environments cause evolutionary change by modifying populations in processes such as immigration, population size, inbreeding and competition (Kawata, 2002; Gavrilets, 2004). For example, in shrews, periods of environmental stress are accompanied by increased food competition and extensive mortality (Zahavi et al., 1991; Bulyev et al., 2000). In turn, greater interspecific competition for food amplified and extended the effects of stress exposure on the ostegony of morphological structures (Foresman and Bulyev, 2003).

A. STRESS-INDUCED EVOLUTION VERSUS STRESS-INDUCED STASIS

Stress specificity, intensity, and recurrence are of fundamental importance for its evolutionary consequences (Bradshaw and Hardrick, 1989; Parsons, 1994; Ansel Meyers and Bull, 2002). Parsons (1994) suggested that only some subsets of stressful environments — narrowly fluctuating and slowly changing in relation to generation time — are associated with a rapid evolutionary change, whereas extreme and rapidly changing environments promote morphological stasis because of the costs associated with stress tolerance (see also Hoffmann et al., 2003). Furthermore, only stressors specific to an organisal system are expected to enable assimilation and evolutionary persistence of stress-induced adaptations, because more general stressors favor stress tolerance by increasing homeostasis, in turn leading to a reduction in organisational size and fitness. Thus, among the array of organisational responses to stressful environments, only accommodation of stress-induced variation and stress avoidance leads to evolutionary change (Parsons, 1993). In turn, because of its association with lower metabolism and stronger regulatory systems, stress tolerance is unlikely to be associated with greater organisational plasticity, thus leading to stability under extreme environments, which is observed in “living fossils” (Parsons, 1993, 1994; Figure 13-1).

IV. CONCLUSIONS

Several themes and approaches in recent studies significantly further our understanding of the relationship between stressful environments and evolution. First, stressful environments modify (most often induce) the accumulation of nervous, endocrinological, morphological, and behavioral regulatory systems. Second, such
reduced integration and subsequent accommodation of stress-induced effects by complex developmental systems enables organizational "memory" of a stressful event, as well as phenotypic and genetic assimilation of the response to a stressor. Third, this is not correct. In complex functional systems, a stress-induced increase in pheno- typic and genetic variance is often directional, channeled, and amplified by the existing developmental system, which accounts for similarity among individuals in stress-induced change and thus significantly facilitates the rate of adaptive evolution. Fourth, accumulation of phenotypically neutral genetic variance might be a prop- erty of any locally adapted and complex developmental system; novel or extreme environments facilitate the phenotypic expression of this variance. Fifth, stress-induced effects are enabled by hormonal effects on learning and gene expression and are facilitated by maternal inheritance of either a stressor or a stress-induced response. These transgenerational effects along with the complexity of developmental systems and stressor recurrence might lead to genetic assimilation of stress-induced effects. Accumulation of neutral genetic variance by develop- mental systems and phenotypic accommodation of stress-induced effects, together with the inheritance of stress-induced modifications, ensures the evolutionary persistance of stress-response strategies and provides a link between individual adaptability and evolutionary adaptation.

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