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Review

Stress-induced variation in evolution: from behavioural plasticity to genetic assimilation

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Extreme environments are closely associated with phenotypic evolution, yet the mechanisms behind this relationship are poorly understood. Several themes and approaches in recent studies significantly further our understanding of the importance that stress-induced variation plays in evolution. First, stressful environments modify (and often reduce) the integration of neuroendocrinological, morphological and behavioural regulatory systems. Second, such reduced integration and subsequent accommodation of stress-induced variation by developmental systems enables organismal 'memory' of a stressful event as well as phenotypic and genetic assimilation of the response to a stressor. Third, in complex functional systems, a stress-induced increase in phenotypic and genetic variance is often directional, channelled by existing ontogenetic pathways. This accounts for similarity among individuals in stress-induced changes and thus significantly facilitates the rate of adaptive evolution. Fourth, accumulation of phenotypically neutral genetic variation might be a common property of locally adapted and complex organismal systems, and extreme environments facilitate the phenotypic expression of this variance. Finally, stress-induced effects and stress-resistance strategies often persist for several generations through maternal, ecological and cultural inheritance. These transgenerational effects, along with both the complexity of developmental systems and stressor recurrence, might facilitate genetic assimilation of stress-induced effects. Accumulation of phenotypically neutral genetic variance by developmental systems and phenotypic accommodation of stress-induced effects, together with the inheritance of stress-induced modifications, ensure the evolutionary persistence of stress–response strategies and provide a link between individual adaptability and evolutionary adaptation.

Keywords: adaptation; developmental plasticity; genetic and phenotypic variation; stress

1. INTRODUCTION

Environments outside the range normally experienced by a population, and the associated changes in organisms' morphological, physiological or behavioural homeostasis, accompany most evolutionary changes (Bijlsma & Loeschcke 1997; Hoffmann & Parsons 1997; Hoffmann & Hercus 2000). Depending on the intensity, predictability and recurrence of stress, responses might range from stress tolerance and avoidance at the organismal level to the rapid appearance of novel traits or extinction at a population level. Yet, moderate stress is essential for normal growth and differentiation of many organismal systems (Huether 1996; Clark & Fucito 1998; Muller 2003). For example, a large part of skeletal development is directed by exposure to tension and mechanical overloads in excess of those normally experienced in the organism's functioning (Hall 1986; Carter 1987; Herring 1993). Stress plays an important role in facilitating local adaptation by enabling better adjustments, synchronization and functioning of many organismal systems (Simons & Johnston 1997; Emlen *et al.* 2003; Rutherford 2003; Wingfield 2003; Robertson 2004). Anyone who has experienced the invigorating effects of diving into icy-cold water after a sauna, the health benefits of rigorous exercise, as well as analgesic and attention-sharpening effects accompanying stressful encounters (McEwen & Sapolsky 1995; Shors & Servatious 1997) will testify to these effects of stress. On the other hand, response to an acute and unfamiliar stressor precludes normal organismal functions (Sibly & Calow 1989), and the high cost of stress tolerance or lack of evolved stress response strategies leads to evolutionary stasis (Parsons 1994).

Extreme environments not only disrupt normal development and induce large phenotypic changes in novel directions, they also simultaneously exert strong phenotypic selection that favours changes in these directions (Waddington 1941; Schmalhausen 1949; Bradshaw & Hardwick 1989; Jablonka *et al.* 1995). Not surprisingly, evolutionary diversification, the appearance of phenotypic novelties and mass extinction are all closely associated with extreme environmental changes (e.g. Howarth 1993; Guex 2001; reviewed in West-Eberhard 2003). Although it is widely recognized (especially in physiological and embryological 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 evolution of mechanisms that enable the organismal accommodation of stress-induced variation.

The lack of a developmental perspective in evolutionary studies of stress has left us with several unresolved

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Table 1. Empirical patterns of the stress-induced phenotypic and genetic variation.

general phenomenon	specific pattern (organisms)	references
stress-induced generation of novel genetic variation	exposure to stress induces directional and locally adaptive mutations (green alga, bacteria, yeast, *Daphnia*)	Ruvinsky *et al.* (1983), Cairns *et al.* (1988), Hall (1990), Steele & Jinks-Robertson (1992), Foster (2000), Goho & Bell (2000), Sniegowski *et al.* (2000), Bjedov *et al.* (2003) and Wright (2004)
	increase in evolutionary rate of a gene (cyanobacteria; human)	Prody *et al.* (1989) and Dvornyk *et al.* (2002)
	increase in frequency of sexual recombination (*Volvox*)	Nedelcu & Michod (2003)
	increase in mutation and/or recombination rates (many species)	Belyaev & Borodin (1982), Parsons (1988), Selker (1990) and Imasheva (1999)
	increase in stress-induced transposition (plants, *Drosophila*)	McClintock (1984), Bownes (1990), Rather *et al.* (1992) and Wessler (1996)
	appearance of primitive, ancestor-like forms	Guex (2001)
stress challenge of general homeostasis releases hidden variation	phenotypic responses to stress mimic the expression of mutation	Goldschmidt (1940), Waddington (1941, 1953), Milkman (1965), Ho *et al.* (1983), Chow & Chan (1999) and Schlichting & Smith (2002)
	phenotypically neutral genetic variance in ancestral forms of domesticated organisms becomes adaptive in the hybrid backgrounds, including domesticated forms (soybeans, maize, sunflowers) and other organisms	Roth *et al.* (1989), Lauter & Doebley (2002), Rieseberg *et al.* (2003), Innan & Kim (2004), Pelabon *et al.* (2004), Piffanell *et al.* (2004) and Seehausen (2004)
	environment dependency and context dependency in expression of genetic variation	Kondrashov & Houle (1994), Threadgill *et al.* (1995), Leips & MacKay (2000) and Keller *et al.* (2002)
	complex and redundant developmental systems enable accumulation of mutational variance	Szafraniec *et al.* (2001)
stress challenge of specific buffering mechanisms releases hidden variation	stress-induced changes in regulation of chaperone proteins releases normally unexpressed genetic variation	Rutherford & Lindquist (1998), Queitsch *et al.* (2002) and Ruden *et al.* (2003)
	release of cryptic genetic variation by artificial selection (*Drosophila*)	Gibson & Hogness (1996) and Dworkin *et al.* (2003)
	epigenetic regulation of genes uncovers normally unexpressed phenotypic variation	Rutherford (2003), Sollars *et al.* (2003) and True *et al.* (2004)

questions. First, how can organisms prepare for novel and extreme environmental change? 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 changed environments. Yet, for stress-induced modifications to have evolutionary importance, they have to be inherited and must persist in a sufficient number of individuals within a population. This requires an organism to survive stress and reproduce; that is, to accommodate stress-induced variation without reduction in the organism's 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 a particular focus on the effect of stress during development in animals, suggests that these questions are resolved by considering: (i) the organization of developmental systems that enable accommodation and channelling of stress-induced variation without compromising organismal functionality; (ii) the significance of phenotypic and genetic assimilation of neurological, physiological, morphological and behavioural responses to stressors; and (iii) multiple inheritance systems that transfer the wide array of developmental resources and conditions between the generations enabling long-term persistence and evolution of stress-induced adaptations.

2. SOURCES OF STRESS-INDUCED VARIATION

A stress-induced increase of phenotypic and genetic variation 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 homeostatic mechanisms can release and amplify phenotypic variation. Third, stressful environments can facilitate developmental expression of novel genetic variation that had accumulated, but was phenotypically neutral under a normal range of environments. These sources of variation can be adaptive under stressful conditions when they facilitate the development of novel adaptations to changed environments.

(a) *'Generated' variance*

Organismal reaction to a stressor is often associated with generation of variation in a directional and locally adaptive manner (Jablonka & Lamb 1995; West-Eberhard 2003; see table 1). In some cases, such directionality is attributed to the channelling effects of complex developmental networks (e.g. Walker 1979; Roth & Wake 1985). In other cases, it is associated directly with a stressful environment (e.g. Wills 1983). An extreme environment often increases genetic variation because of the elevation in mutation and recombination rates (Hoffmann & Parsons 1997; Hoffmann & Merilä 1999; Imasheva 1999; Wright 2004). When such mutations are directional (or 'focused'; Caropale 1999) in relation to a stressor—that is, when the stressful environment causes a mutation and favours phenotypic change associated with this mutation—such an increase in mutation rate might result in greater similarity among individuals within a population in response to a stressor facilitating evolutionary adaptation to novel environments (Shapiro 1992; Jablonka & Lamb 1995; Foster 2000; Wright 2000; Brisson 2003; Rutherford 2003; Wright 2004). Table 1 shows examples of stress-induced generation of novel genetic variation. At the level of phenotype, induction of a phenotypic trait by a stressor and concurrent selection on the induced trait are common (Price *et al.* 2003; West-Eberhard 2003).

(b) *'Hidden' variance*

Stressful environments reveal greater phenotypic and genetic variability than is seen under normal conditions, and it is commonly suggested that such hidden variation results from stress-induced challenge to organismal homeostasis (Scharloo 1991). In turn, an increase in variation and subsequent reorganization of organismal systems are thought to enable the formation of novel adaptations (Bradshaw & Hardwick 1989; Eshel & Matessi 1998; Gibson & Wagner 2000; Schlichting & Smith 2002). The idea that the extreme environments' challenge to previously canalized systems is the source of such hidden variation is corroborated by numerous empirical observations of environment- and context-dependency in expression of novel genetic variation (table 1). However, it is not clear how genetic and developmental systems can accumulate and store phenotypically neutral genetic variation while not expressing it (Wagner & Mezey 2000; Hermisson *et al.* 2003; Masel & Bergman 2003; Gibson & Dworkin 2004). Specifically, the discussion has focused on the existence of 'evolutionary capacitors' (Rutherford 2000) and 'adaptively inducible canalizers' (Meiklejohn & Hartl 2002)—specific mechanisms that buffer and accumulate developmental variation, producing 'hidden reaction norms' of a phenotype under stress. A debated question is whether these 'evolutionary capacitors' are stressor-specific regulatory systems or whether evolutionary capacity is a property of any complex and locally adapted organismal system. Rutherford & Lindquist (1998) found that mutations at the gene for the stress-induced chaperone proteins harbours abundant but normally unexpressed genetic variation, which, when selected, leads to the appearance and assimilation of novel phenotypes in the population (Queitsch *et al.* 2002; Ruden *et al.* 2003; Rutherford 2003; Sollars *et al.* 2003). However, other studies suggested that 'evolutionary capacity' is a property of most locally adapted developmental systems that, when challenged by a novel environment, reveal large genetic variation (Kirschner & Gerhart 1998; Rutherford 2000; Bergman & Siegal 2003). Complex developmental processes and genetic networks can constrain variation in individual traits (Rice 2004), and phenotypically neutral genetic variation is commonly accumulated in such systems given sufficient time and population size (Hermisson & Wagner 2004).

3. EVOLUTION OF RESPONSE TO STRESS

Stress occurs when changes in the external or internal environment are interpreted by an organism as a threat to its homeostasis (e.g. Greenberg *et al.* 2001; McEwen & Wingfield 2003). Thus, 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). However, 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, as is required in order to select between stressor avoidance and stress tolerance? Are the mechanisms of assessment and avoidance specific to a particular stressor?

(a) *Insights from cognitive and physiological assimilations of a stressful event: what does not kill you makes you stronger*

Generally, repeated exposure to a particular stressor favours the evolution of mechanisms that suppress an organism–wide stress reaction and, instead, activate stress-specific responses (Johnson *et al.* 1992; Barclay & Robertson 2001; Veenema *et al.* 2003). For example, in mammals and birds, stress-induced activation of the neuroendocrinological system increases its reactivity to internal and external stimuli, facilitates the processing of sensory information, and ultimately enables the formation of a behavioural or physiological strategy of dealing with a stressor. Furthermore, stress-induced activation of neuro-endocrinological systems facilitates long-term retention of information about a stressful event and corresponding organismal response. Once formed, the maintenance of such 'memory' can be accomplished by periodic exposure to different stressors. For example, hormones associated with stress detection and avoidance also play a major role in modifications of neural circuits (Gold & McGaugh 1978). Once the stress-avoidance strategy is formed, exposure to even a low concentration of these hormones maintains the strategy (McGaugh *et al.* 1982; for similar examples, see Sockman *et al.* 2002, 2004).

Animal physiology studies show that the repeated experience of successfully overcoming social stresses during ontogeny is a prerequisite for the acquisition of a normal repertoire of behavioural strategies (Huether 1996; e.g. Gans 1979). An insightful example is provided by experiments that show that individuals exposed to repeatable, but consistently unfamiliar (and thus 'uncontrollable' by an animal) stressors develop 'stressful helplessness' (i.e. they lose their ability to react to any stressor; Katz *et al.* 1981; Johnson *et al.* 1992; Avitsur *et al.* 2001). In contrast, individuals that were allowed to develop a stress-avoidance strategy by exposure to a previously encountered stressor not only developed 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; Avitsur *et al.* 2001). Similarly, in insects, exposure to temperature stress facilitated not only subsequent long-term tolerance of extreme temperatures, but also performance under anoxia and various motor stresses (Karunanithi *et al.* 1999; Ramirez *et al.* 1999; Wu *et al.* 2002). The interchangeability of distinct stressors in maintaining and exerting organismal stress response, and long-term persistence of stress effects, have also been documented in the studies of amphibians, where organismal stress from food deprivation and from a water level reduction exerted similar endocrinological effects during larval metamorphosis (Denver 1999; Boorse & Denver 2004). These results suggest that, once originated, a stress–response strategy can be maintained by other environments, and adaptation to one type of stressor facilitates adaptation to other stressors.

Phenotypic assimilation of the appropriate stress response is further facilitated by a common involvement of neural and endocrine pathways of the stress response in other organismal functions (Aston-Jones *et al.* 1986; Greenberg *et al.* 2001; Robertson 2004). 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 will instead activate stress-specific behavioural and physiological responses (Levine *et al.* 1967, 1989). Generally, stress-induced reorganization of existing developmental pathways and organismal function, rather than the production of novel stress-specific pathways, accounts for the ease with which individuals and populations lose and gain the ability to resist stress in laboratory settings (Chapin *et al.* 1993; Hoffmann & Merilä 1999).

(b) *Stress-avoidance strategies*

The ability actively to remove a stressor by either relocation or avoidance requires an evolved ability to detect or anticipate stressful changes and the 'knowledge' or 'memory' of stress avoidance strategies or adjustments (Bradshaw & Hardwick 1989; Jablonka *et al.* 1995; Denver 1999). Therefore, the evolution of stress avoidance is more probable when stressful events are predictable, prolonged and frequent in relation to generation time (Ancel Meyers & Bull 2002). Alternatively, the short-term avoidance of a frequent but mild stressor might be accommodated by behavioural or physiological plasticity of an organism (Schlichting & Smith 2002; Nicolakakis *et al.* 2003; Piersma & Drent 2003; Wingfield & Sapolsky 2003). For example, repeated challenges of an organism's immune system and challenges to skeletal tissues caused by mechanical overload during growth lower developmental errors and enable a more precise reaction to a specific pathogen (e.g. Graham *et al.* 2000; Hallgrímsson *et al.* 2004). Generally, organisms can activate stress reactions when there is discordance between environments during their development and their current external and internal environments (Meaney 2001; Bateson *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 & Hardwick 1989; Denver 1999; Stanton *et al.* 2000; Boorse & Denver 2004).

In sum, initial behavioural accommodations 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, their mutual recurrence facilitates the establishment of stressor recognition, assessment and avoidance strategies, such that 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 favoured by natural selection during and after a stressful event, these strategies can become phenotypically and genetically assimilated in a population (see below; Baldwin 1896; Hinton & Nolan 1980; Oyama 2000; West-Eberhard 2003).

(c) *Stress buffering as a by-product of developmental complexity*

Organismal functions most closely related to fitness are thought to be the most buffered against internal and external stressors (Waddington 1941; Schmalhausen 1949; Stearns & Kawecki 1994). However, an organism's functioning in changing environments requires the ability to track and respond to these environments, and evolved buffering from stressors would also restrict an organism's ability and capacity to adapt to continuously changing environments (Wagner *et al.* 1997; Eshel & Matessi 1998; Ancel 1999; Schlichting & 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 behavioural strategies necessary for stress avoidance (Huether 1996). On the one hand, a lack of phenotypic plasticity results in population extinction under stress (Gavrilets & Scheiner 1993; Ancel 1999). On the other hand, extensive phenotypic variability in organismal functions weakens the effects of directional selection imposed by stressful environments, and thus lessens the opportunity for genetic assimilation and evolution of adaptations to stress (Fear & Price 1998; Ancel 2000; Huey *et al.* 2003). Recent studies suggest that buffering is an emerging property of developmental complexity rather than an evolved stress-resistance mechanism (see above). The increasing complexity of developmental pathways and networks leads directly to environmental and genetic stability and canalization (Baatz & Wagner 1997; Rice 1998, 2004; Waxman & Peck 1998; Meiklejohn & Hartl 2002; Siegal & Bergman 2002; Ruden *et al.* 2003). Complex genetic and developmental networks can accommodate the effects of stressful perturbations without the loss of function or structure, while building up neutral genetic variation (Rutherford 2000; Bergman & Siegal 2003; Masel 2004).

(d) *Accommodation of stress-induced variation by changes in an organism's integration*

Organisms might accommodate stress-induced variation without the loss of function by lessening homeostasis of individual systems. For example, individual hormonal systems have a far greater potential range of performances

Table 2. Empirical patterns of the relationship between stress and homeostasis.

general phenomenon	specific pattern	references
stress increases homeostasis	stress-induced increase in resource exchange leads to a greater developmental integration among normally independent organismal components in animals raised under stress	Sciulli *et al.* (1979), Schandorff (1997) and Klingenberg *et al.* (2001)
	exposure to stress prevents the expression of deleterious mutations in *Escherichia coli*	Kishony & Leibler (2003)
	increased homeostatic interactions within reproductive systems counteracts the stress effects on the organism when breeding opportunities are limited	Wingfield & Sapolsky (2003)
stress decreases homeostasis	increase in developmental noise and variation in numerous organismal traits	Parsons (1990), Hoffmann & Parsons (1997), Møller & Swaddle (1997), Polak (2003) and Badyaev (2004)
	destruction of the nest site leads to temporary breaking of the strict hierarchical social structure and rapid proliferation of random individual search routes that facilitate finding of a new nest site in social ant species	Britton *et al.* (1998) and Couzin & Franks (2003)

and can remain functional under a wider range of environments than is allowed by homeostasis under a normal range of environments (Dickinson 1988; Johnson *et al.* 1992; Greenberg *et al.* 2001; Robertson 2004; Romero 2004). Frequently documented suppression of immunocompetence under stressful conditions might facilitate novel adaptations to a stressor by realizing full capabilities of individual immune systems (Råberg *et al.* 1998). When stress is associated with damage of tissues, suppression of immunological functions enables individual organismal systems to respond to a stressor without activation of organism-wide autoimmunological response (Dickinson 1988; Råberg *et al.* 1998; Avitsur *et al.* 2001). Examples of stress-induced increases in organismal integration, mostly in response to mild stressors, and corresponding suppression of random developmental variation under stress are shown in table 2.

The importance of the timing of stress for directing the evolution of morphological traits is well documented. When stressors are mild and occur during ontogeny, individual organismal systems often accommodate stress-induced variation without the reduction in functionality (Bradshaw & Hardwick 1989; Herring 1993; Huether 1996; Schandorff 1997; Hallgrímsson 1999; Badyaev & Foresman 2004). For example, when components of foraging structures differ in patterns of ossification, morphological variation in later ossified components can be directed by stress-induced modifications of earlier ossified components (Mabee *et al.* 2000; Badyaev *et al.* in press). In addition, when stress occurs early in ontogeny, accommodation and channelling of stress-induced variation by existing organismal structures causes similar reorganization in many individuals simultaneously (Roth & Wake 1985; Chapin *et al.* 1993), thus facilitating adaptive evolution (Goldschmidt 1940; West-Eberhard 2003).

(e) *Channelling of stress-induced variation*

Natural selection favours organismal homeostasis that maintains some degree of developmental variation necessary for adjustment of the organism to its environments (Simons & Johnston 1997; Wagner *et al.* 1997; Eshel & Matessi 1998; Emlen *et al.* 2003; Nanjundiah 2003). Stressful conditions can increase this variation, and differences among organismal systems in their reaction to a stressor might bias the introduction and expression of variation available for selection and, thus, ultimately, affect evolutionary change (Bonner 1965; Roth & Wake 1985; Jablonka & Lamb 1995; West-Eberhard 2003). Empirical studies show that the coordinated development of morphological traits often leads to their similarity in expression of stress-induced developmental variation (Leamy 1993; Badyaev & Foresman 2000, 2004; Klingenberg *et al.* 2001; Badyaev *et al.* in press). These examples not only confirm a strong effect of functional complexes on directing and incorporating stress-induced variation during development, but might also explain the historical persistence of complex groups of traits through stressful environments.

4. INHERITANCE OF STRESS-INDUCED VARIATION

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

Stress-induced phenotypic changes commonly persist across several generations. Such across-generation carry-over effects (*sensu* Jablonka *et al.* 1995) can be a result of 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 behavioural effects (Jablonka & Lamb 1995; Rossiter 1996; Oyama 2000; Odling-Smee *et al.* 2003; West-Eberhard 2003). For example, inheritance of dominant–subdominant relationships in groups of many social mammals is accomplished by mechanisms different from original stressful encounters that established the dominance structure (Creel *et al.* 1996; Fairbanks 1996). Similarly, maternal care often sets the stage for a lifelong reaction to stressors by offspring by modifying the expression of

genes that regulate behavioural, physiological and endocrinological responses to stressors (Mousseau & Fox 1998; Meaney 2001; Badyaev in press). At the individual level, stress-induced changes in neuroendocrinological systems often occur with significant delay after the exposure to stress, and persist for a long time. This observation 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; Ramirez *et al.* 1999; Romero 2004). In turn, within- and between-generation maintenance of stress-induced changes in neurophysiological systems is accomplished by similar hormonal mechanisms (see above; McGaugh *et al.* 1982; Meaney 2001). More generally, short-term and non-genetic inheritance might be 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 1963; Ancel Meyers & Bull 2002; Gavrilets 2004).

In sum, accommodation of stress-induced variance by an organism can be facilitated by recurrent developmental stressors and genetic assimilation replaces stress-induced developmental modification if this modification has a fitness advantage in both stressful and post-stress environments (Schmalhausen 1949; Waddington 1953). Even when the short-term organismal responses to a stressor are not genetically heritable, differences among organisms in their ability to survive stress and the recurrence of stressful environments will canalize stress-induced responses developmentally (Baldwin 1896; Schlichting & Pigliucci 1998; Ancel 1999; West-Eberhard 2003). An excellent example is a recent comprehensive review by Palmer (2004), where he shows that genetic assimilation enables the evolutionary establishment of environmentally induced novelty—directional asymmetry (for other examples of genetic assimilation of stress-induced developmental variation see Ho *et al.* 1983; Chapman *et al.* 2000; Heil *et al.* 2004).

5. STRESS-INDUCED EVOLUTION VERSUS STRESS-INDUCED STASIS

Only a subset of stressful environments—narrowly fluctuating and slowly changing in relation to generation time—are thought to be associated with a rapid evolutionary change, whereas extreme and rapidly changing environments might result in morphological stasis because of the costs associated with stress tolerance (Parsons 1994). Furthermore, only stressors specific to an organismal system are expected to enable phenotypic assimilation and evolutionary persistence of stress-induced adaptations because a more general stressor favours stress tolerance by increasing homeostasis, which, in turn, leads to a reduction in organismal metabolism and fitness. Thus, among the array of organismal responses to stressful environments reviewed above, only accommodation of stress-induced variation and stress avoidance are expected to lead to significant evolutionary change (Parsons 1993). On the contrary, because of its association with lower metabolism and stronger regulatory systems, stress tolerance is unlikely to be associated with greater organismal plasticity, and instead, leads to stasis under extreme environments which is observed in 'living fossils' (Parsons 1993, 1994).

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