CHAPTER 8

Beyond Allostatic Load

The Stress Response System as a Mechanism of Conditional Adaptation

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HISTORICAL CONTEXT

THE STRESS RESPONSE SYSTEM (SRS) has a central role in orchestrating physical and psychosocial development of both humans and nonhuman species (Ellis, Jackson, & Boyce, 2006; Korte, Koolhaas, Wingfield, & McEwen, 2005). For many organisms, the SRS contributes crucially to responding flexibly to environmental opportunities and challenges. One of the most remarkable features of the SRS is the wide range of individual variation in physiological parameters. Some respond quickly and strongly even to minor events, whereas others show flat response profiles across situations. Furthermore, the balance of activation among primary SRS subsystems—the sympathetic nervous system (SNS), the parasympathetic nervous system (PNS), and limbic-hypothalamic-pituitary-adrenal (LHPA) axis—can vary considerably across individuals.

It is difficult to overstate the real-world relevance of such individual variability. Decades of research demonstrate not only that physiological patterns of stress responsivity constitute a primary integrative pathway through which *psychosocial environmental factors* are transmuted into the behavioral, autonomic, and immunologic manifestations of human pathology (reviewed in Boyce & Ellis, 2005), but also that patterns of stress responsivity regulate variation in a wide range of adaptive processes and behaviors including (but not limited to) growth and metabolism, reproductive status and fertility, aggression and risk taking, pair bonding and caregiving, and memory and learning (reviewed in Del Giudice, Ellis, & Shirtcliff, 2011). Clearly, understanding the causes of such individual differences and their development over the life course has important implications for medicine, psychology, and psychiatry, among other disciplines.

One approach has been to view individual differences in stress reactivity through a pathology lens. Indeed, a common assumption in the stress literature is that there

is an optimal level of stress responsivity and that overly heightened or dampened SRS reactivity is dysfunctional and tends to undermine emotional and behavioral regulation (e.g., Evans & English, 2002). This purported biological dysregulation of the SRS is typically interpreted in an allostatic load framework (e.g., Juster, McEwen, & Lupien, 2010), whereby wear and tear of chronic stress is presumed to impair SRS functioning (see extended discussion below).

This allostatic load model (ALM) has recently been promoted in a double Special Issue of *Development and Psychopathology* (Cicchetti, 2011). In this chapter, however, we argue that enthusiasm for the ALM may have been overstated. Specifically, we contend that that the ALM has strengths, but also serious limitations when evaluated from the vantage point of evolutionary biology; that the ALM does not address the role of allostasis in regulating adaptive developmental plasticity; that, in most instances, the core metaphor of ''stress dysregulation'' could usefully be replaced by ''adaptive calibration'' of SRS parameters; and that explicit modeling of biological fitness trade-offs, as instantiated in life history theory (LHT), is needed to more fully explain the complex relations between developmental exposures to stress, stress responsivity, behavioral strategies, and health. As an alternative to the ALM, we present the adaptive calibration model of stress responsivity (ACM; Del Giudice et al., 2011). We begin by reviewing the concepts of adaptive calibration and miscalibration more generally. We then summarize key ACM concepts and discuss their implications for developmental psychopathology. We conclude by comparing the ALM and ACM explicitly and suggest that it is time for the field to move beyond the ALM perspective.

CONDITIONAL ADAPTATION AND MALADAPTATION

A large body of scientific work has sought to explain the relations between developmental exposures to stress, stress responsivity, behavioral strategies, and health. Unfortunately, this work has been hampered by patchy, inconsistent, and sometimes confusing usage of the concepts of adaptation and maladaptation. Here we attempt to define and explain these concepts from an evolutionary–developmental perspective.

Conditional Adaptation

Developmental exposures to stress have always been part of the human experience. For example, almost half of children in hunter-gatherer societies—the best model for human demographics before the agricultural revolution—die before reaching adulthood (Kaplan & Lancaster, 2003). Thus, from an evolutionary-developmental perspective, stressful rearing conditions, even if those conditions engender sustained stress responses that must be maintained over time, should not so much impair SRS functioning (''dysregulation'' in the ALM) as direct or regulate it toward response patterns that are adaptive under stressful conditions, even if those patterns are harmful in terms of the long-term welfare of the individual or society as a whole (e.g., Ellis, Boyce, Belsky, van Ijzendoorn, & Bakermans-Kranenburg, 2011; Mead,

Beauchaine, & Shannon, 2010). From an evolutionary perspective, there is no optimal level of stress responsivity; adaptation is context-specific.

Consider the extensive experimental work conducted by Michael Meaney and colleagues showing that putatively low quality maternal care in the rat (i.e., low levels of maternal licking and grooming) alters pups' stress physiology and brain morphology. Although such changes seem disadvantageous (i.e., higher corticosterone levels, shorter dendritic branch lengths, and lower spine density in hippocampal neurons), they actually enhance learning and memory processes under stressful conditions (e.g., Champagne et al., 2008). Moreover, such physiological and morphological changes mediate the effects of maternal behavior on central features of defensive and reproductive strategies: behavior under threat, open-field exploration, pubertal development, sexual behavior, and parenting (Cameron et al., 2005, 2008).

In total, enhanced learning under stressful conditions, increased fearful and defensive behaviors, accelerated sexual maturation, increased sexual behavior, and reduced parental investment in offspring apparently represent strategic—that is, functional—ways of developing when the young organism is relatively neglected. In such contexts, neglect itself can be regarded as a behavioral mechanism through which rat parents guide their offspring's development toward optimal survival and reproductive strategies under conditions of adversity. It would seem mistaken, therefore, to view diminished licking and grooming as ''poor maternal care'' or the development induced by such care as ''disturbed,'' even though this is how they are often characterized. From an evolutionary perspective, altered care provided by parents may be appropriate preparation of their offspring for expected ecological conditions.

Accordingly, the evolutionary perspective emphasizes *conditional adaptation*: ''evolved mechanisms that detect and respond to specific features of childhood environments, features that have proven reliable over evolutionary time in predicting the nature of the social and physical world into which children will mature, and entrain developmental pathways that reliably matched those features during a species' natural selective history'' (Boyce & Ellis, 2005, p. 290; for a comprehensive treatment of conditional adaptation, see West-Eberhard, 2003). From this perspective, variation in SRS functioning results largely from individuals tracking different environmental conditions and altering their SRS profiles to match those conditions. Presumably, this matching process promoted fitness—survival and ultimately reproduction—across heterogeneous environmental contexts over human evolution.

However, an evolutionary history of exposure to such heterogeneous contexts, in which the fitness of different phenotypes varied across time and/or space, is a necessary but not sufficient condition for the evolution of conditional adaptations. The fitness of the alternative phenotypes must also be predictable on the basis of reliable cues that can be observed by the individual (Pigliucci, 2001). For example, tadpoles (*rana sylvatica*) alter their size and shape based on the presence of dragonfly larvae in their rearing environment (Van Buskirk & Relyea, 1998). These alterations involve development of smaller and shorter bodies and deep tail fins. Although tadpoles that do not undergo these morphological changes are highly vulnerable

to predation by dragonflies, those that do but end up inhabiting environments that are not shared with dragonflies have relatively poor developmental and survival outcomes. In short, the predator-induced phenotype is only conditionally adaptive. This process highlights that in many cases, natural selection favors a primary phenotype that yields high payoffs under favorable circumstances and a secondary phenotype that ''makes the best of a bad situation'' (West-Eberhard, 2003).

The Meaning of Adaptive

The foregoing discussion highlights that the term *adaptive* has different meaning when viewed from evolutionary and mental health perspectives (see also Ellis et al., 2012; Mead et al., 2010). Because evolution by natural selection is driven by differences among individuals in reproductive success, the evolutionary significance of any behavior, or its ''adaptive value,'' depends ultimately on its costs and benefits with respect to the organism's fitness (i.e., the contribution of offspring to future generations by an individual and its relatives). Even high-risk behaviors that result in net harm in terms of a person's own phenomenology and well-being (e.g., producing miserable feelings or a shortened life), the welfare of others around them, or the society as a whole, can still be *adaptive* in an evolutionary sense. Consider, for example, risky behaviors that expose adolescents to danger and/or inflict harm on others but increase dominance in social hierarchies and leverage access to mates (Ellis et al., 2012). On the other hand, from a mental health perspective, different patterns of behavior are regarded as ''adaptive versus maladaptive'' depending on the extent to which they promote versus threaten people's health, development, and safety. Adaptive developmental outcomes are thus equated with ''desirable'' outcomes (as defined by dominant Western values; e.g., health, happiness, secure attachment, high self-esteem, emotion regulation, educational and professional success, stable marriage), whereas maladaptive developmental outcomes are equated with ''undesirable'' outcomes constituting the opposite poles of these traits and variables. For the remainder of this paper, we use ''adaptive'' only in the evolutionary sense of the term. In contrast the word *desirable* is used to connote adaptiveness from a mental health perspective.

Maladaptation

The converse of adaptation is maladaptation. Biological maladaptation can occur for many reasons. Sometimes, an evolved mechanism ceases to perform its intended function because of, for example, harmful genetic mutations, accidents, or manipulation by other organisms (e.g., pathogens). Even when biological mechanisms perform normally, an organism may develop a phenotype that is poorly suited for its environment and as a consequence experiences a diminution in fitness (often accompanied by other ''undesirable'' outcomes). Thus, maladaptation is closely connected to the concept of developmental miscalibration or mismatch (see Frankenhuis & Del Giudice, 2012, for an extended discussion). There are a number of causes of such developmental miscalibration or mismatch. First, an individual may experience

novel environments that are outside the range recurrently encountered over evolutionary history. In this case, all developmental bets are off and the person may experience abnormal outcomes. For example, Romanian or Ukrainian orphanages (Dobrova-Krol, Van IJzendoorn, Bakermans-Kranenburg, & Juffer, 2010; Nelson et al., 2007) constitute genuinely substandard, novel environments that are beyond the normative range of conditions encountered over human evolution. Children's brains and bodies simply could not have responded adaptively to collective rearing by paid, custodial, nonkin caregivers with minimal human contact (Hrdy, 1999). Exposures to such challenging and (evolutionarily) unprecedented conditions are likely to induce pathological development, not evolutionarily adaptive strategies.

Second, individuals may become maladapted to their environments because of a lack of behavioral plasticity. As discussed later, one of the responsivity profiles highlighted by the ACM is the unemotional pattern, which is characterized by low susceptibility to environmental influence (i.e., dampened physiological stress reactivity), which generally inhibits social learning and sensitivity to social feedback. One hypothesized pathway here is a genetic disposition toward hypoarousal of stress systems. Such a disposition could translate into a wide distribution of unemotional phenotypes across a range of familial and ecological conditions, including supportive and well-resourced rearing environments. Maladaptation may occur in this context because unemotional phenotypes are not susceptible to environmental influence and thus may not adjust their behavioral strategies to match the high levels of support and resources available to them (e.g., they may not adequately detect positive opportunities and learn to capitalize on them, such as seeing a teacher as a prospective mentor or taking advice from a loving parent; and/or they may develop a manipulative, antagonistic social strategy when trust and cooperation would better fit their social context). In total, increased probability of mismatch is a clear cost of low developmental plasticity.

Third, mismatch can occur because the validity of environmental cues that guide conditional adaptation is limited spatially, so such cues become invalid in other contexts. For example, according to LHT, children's brains and bodies tend to respond to dangerous or unpredictable environments by growing up fast and ''living for the here and now'' (e.g., Belsky, Steinberg, & Draper, 1991; Ellis, Figueredo, Brumbach, & Schlomer, 2009). This ''get it while you can'' strategy often translates into high-risk activities such as early initiation of sexual behavior, greater numbers of sexual partners, infrequent contraceptive use, delinquency, substance use, violence, and risky driving. These high-risk behaviors may only be locally adaptive, however. The research of Gibbons et al. (2012) on African American males is instructive in this context. Youth who were exposed to greater stress while growing up (e.g., more dangerous neighborhoods, lower quality parental investment, greater racial discrimination) developed ''fast'' life history strategies that may have been adaptive in their local context (e.g., participation in risky behaviors that leveraged positions in dominance hierarchies, increased access to mates) but clearly undesirable—and probably biologically maladaptive—in the wider U.S. society (e.g., dropping out of school, high rates of arrest and incarceration).

Fourth, mismatch can occur because the validity of environmental cues that guide conditional adaptation is temporally limited, so that those cues may become invalid at later times. One hypothesis is that individuals calibrate to environmental parameters early in life, even prenatally. When these values differ from those experienced later in life, normative processes of developmental plasticity can become maladaptive, resulting in a mismatched phenotype with increased likelihood of physical health problems (e.g., Gluckman, Low, Buklijas, Hanson, & Beedle, 2011). For instance, prenatal exposure to undernutrition may result in the development of metabolic processes designed to retain and store insulin and fatty acids (Barker, 1994). However, if resources are plentiful in the postnatal environment, the individual may be at increased risk for obesity and metabolic syndrome throughout life. This hypothesis is supported by data showing that detrimental effects seem absent when the postnatal environment continues to be lacking in resources (Stanner & Yudkin, 2001), suggesting that mismatch (rather than undernutrition) may be the root cause.

Finally, mismatch can occur due to a restricted range of niches that undermine the ability of organisms to choose environments that match their phenotypes. For example, in a study of semi-free ranging rhesus macaques (Boyce, O'Neill-Wagner, Price, Haines, & Suomi, 1998), the troop lived in a 5-acre wooded habitat in rural Maryland, on the grounds of the National Institutes of Health Primate Center. In 1993, the troop encountered a 6-month period of protective confinement to a small, 1,000-square-foot building, during a construction project on the habitat grounds. The confinement proved highly stressful, however, and the incidence of violent injuries increased fivefold during the 6-month period. During this period, when behavioral strategies available to troop members were severely curtailed, monkeys who had been previously characterized as high in biobehavioral reactivity to stress suffered dramatically higher rates of violent injuries than their less reactive peers. In the free-ranging wooded habitat, however, where a wide range of behavioral strategies could be employed, including escape from conflict, highly reactive monkeys suffered comparatively low rates of violent injury.

In summary, processes of conditional adaptation and phenotype-environment matching are fallible, and a number of circumstances can lead to maladaptation. Understanding this set of circumstances is critical to understanding the developmental origins of psychopathology.

FUNCTIONS OF THE STRESS-RESPONSE SYSTEM

Environmental events signaling threats to survival or well-being produce a set of complex, highly orchestrated responses within the neural circuitry of the brain and peripheral neuroendocrine pathways regulating metabolic, immunologic, and other physiological functions. The SRS comprises primarily three anatomically distinct neuroendocrine circuits: the sympathetic (SNS) and parasympathetic (PNS) branches of the autonomic nervous system and the LHPA axis. Activity of these circuits is integrated and cross-regulated, so that they can be considered as partially independent yet interrelated components of a single functional system despite

their anatomical and physiological diversity (e.g., Boyce & Ellis, 2005; Porges, 1995; Schlotz et al., 2008).

The general function of the PNS is to promote vegetative functions and reduce physiological arousal (see Del Giudice et al., 2011; Porges, 2007). However, when a stressor is encountered, the PNS responds quickly by withdrawing this inhibitory influence (Lovallo & Sollers, 2007), allowing the SNS to operate unopposed, thereby causing rapid increases in physiological arousal. More extreme defense reactions associated with ''freeze/hide'' behaviors also involve PNS activation, albeit via different efferent fibers (Porges, 2007). If parasympathetic deactivation is not sufficient to cope with the present challenge, activation of the SNS occurs within 20 to 30 seconds, providing a second layer of response in this hierarchy. Sympathetic activation mediates fight/flight responses and is coordinated by the locus coeruleus-norepinephrine (LCNE) system. SNS activation follows a fast, direct pathway via the noradrenergic innervation of visceral organs and a slower, hormonal pathway through innervation of the adrenal medulla (see e.g., Goldstein & Kopin, 2008; Gunnar & Vazquez, 2006). Following SNS activation, the adrenal medulla secretes epinephrine (E) and smaller quantities of norepinephrine (NE) to increase heart rate, respiration, blood supply to skeletal muscles, and glucose release in the bloodstream.

The third component of the SRS is the LHPA axis, which mounts a delayed, long-term response to environmental challenges. The end point of the LHPA response is cortisol release by the adrenal cortex, typically within 5 minutes after the triggering event, with a cortisol peak between 10 and 30 minutes. The effects of cortisol secretion may be observed for several hours or more (Sapolsky, Romero, & Munck, 2000). Cortisol binds to nuclear receptors and regulates gene transcription. Its main effects are to (1) mobilize physiological and psychological resources (e.g., energy release, alertness and vigilance, memory sensitization; e.g., Flinn, 2006; van Marle, Hermans, Qin, & Fernández, 2009), and (2) counter-regulate physiological effects of SNS activation, facilitating stress recovery (Munck, Guyre, & Holbrook, 1984). The joint effects of the SNS and LHPA axis are complex (Hastings et al., 2011), and can be synergistic (especially in the short term) or antagonistic (especially at later phases of responding).

Biological Sensitivity to Context

The foregoing summary of the SRS is a widely accepted description of how peripheral neuroendocrine responses prepare the organism for challenge or threat. However, according to the theory of Biological Sensitivity to Context (BSC; Boyce & Ellis, 2005), these ''stress response'' systems also function to increase susceptibility to resources and support in the ambient environment (e.g., positive social opportunities, cooperative information; see also Porges, 1995, 2007). This dual function signified the need to conceptualize stress reactivity more broadly as biological sensitivity to context, which Boyce and Ellis (2005) defined as neurobiological susceptibility to both cost-inflicting and benefit-conferring features of the environment and operationalized as a biological property indexed by heightened reactivity in one or more of the stress response systems (PNS, SNS, LHPA). Depending on

levels of nurturance and support versus harshness and unpredictability in their developmental environments, highly reactive children experience either the best or the worst of psychiatric and biomedical outcomes within the populations from which they are drawn (reviewed in Ellis et al., 2011). BSC theory therefore posits that individual differences in the magnitude of biological stress responses function to regulate openness or susceptibility to environmental influences, ranging from harmful to protective (see Sijtsema et al., in press, for a review and critical analysis of BSC assumptions).

Given past evidence that early trauma increases stress reactivity and newer evidence that high reactivity may enhance developmental functioning in highly supportive settings, Boyce and Ellis (2005) postulated a curvilinear, U-shape relation between levels of early support-adversity and the magnitude of biological response dispositions. Specifically, we hypothesized that (1) exposure to acutely stressful childhood environments up-regulates BSC, increasing the capacity and tendency of individuals to detect and respond to environmental dangers and threats; (2) exposure to especially supportive childhood environments also up-regulates BSC, increasing susceptibility to social resources and support; and (3) by contrast, and typical of the majority of children, exposure to childhood environments that are not extreme in either direction down-regulates BSC, buffering individuals against the chronic stressors encountered in a world that is neither highly threatening nor consistently safe. Exploratory analyses in two studies offered confirmatory evidence that the lowest rates of high reactivity phenotypes were found in conditions of moderate stress and that both tails of the support-adversity distribution were associated with higher proportions of reactive children (Ellis Essex, & Boyce, 2005; see also Bush, Obradovic, Adler, & Boyce, 2011; Gunnar, Frenn, Wewerka, & Van Ryzin, 2009).

Although BSC theory has moved the field toward a new conceptualization of stress responsivity, it has a number of significant limitations. First, BSC theory does not systematically link the different stress reactivity patterns to functional variation in behavior, such as individual differences in social and reproductive behaviors that are specified by LHT. Second, although BSC theory advances a general developmental prediction (the U-shape curve), it does not model the developmental trajectories leading to individual differences in a more fine-grained way (e.g., by discussing the development of stress responsivity at different life stages and identifying ''switch points'' when plasticity is preferentially expressed). Third, BSC does not address the adaptive meaning and developmental origins of sex differences in responsivity. Fourth, BSC focuses on explaining heightened reactivity to stress and does not afford a theory of hypoarousal (or dampened reactivity), in terms of its development or functional significance. Fifth, BSC does not address the development or functions of basal (tonic) levels of activity of the SRS. Finally, BSC theory does not advance discriminative predictions regarding PNS, SNS, and LHPA. The ACM, an extension and refinement of BSC, was formulated to address these issues.

The Adaptive Calibration Model of Stress Responsivity

Goals of the ACM are to provide (1) a coherent, systematic account of the biological functions of the SRS; (2) an evolutionary-developmental theory of individual

differences capable of explaining adaptation—and maladaptation—of stress physiology and behavior to local environmental conditions; and (3) a functionally valid taxonomy of stress response profiles, including neurobiological correlates (e.g., serotonergic function), behavioral correlates (e.g., aggression, self-regulation), and developmental trajectories, which integrates across baseline activity and responsivity measures of the SRS (Del Giudice et al., 2011). Achieving these goals would enable scientists to move beyond the purely inductive theory-building that now dominates the field and increase their ability to advance targeted hypotheses about individual differences and their development. The ACM has its main theoretical foundations in LHT, an evolutionary biological framework for describing the developmental ''decisions'' of organisms and their allocation of resources over the life course (Ellis et al., 2009; Kaplan & Gangestad, 2005), and the theory of adaptive developmental plasticity (West-Eberhard, 2003). In the ACM, individual differences in the functioning of the SRS are thought to result largely (though not exclusively) from the operation of evolved mechanisms that match the individual's physiology and behavior to local environmental conditions (i.e., calibration to the environment). Thus, patterns of stress responsivity are seen as *adaptive* in the biological sense, as they function in a way that ultimately tends to maximize the individual's survival and reproduction.

The ACM can be summarized in seven points (see Del Giudice et al., 2011, for complete explication of the model):

1. The SRS has three main biological functions: to coordinate the organism's allostatic response to physical and psychosocial challenges; to encode and filter information from the environment, thus mediating the organism's openness to environmental inputs; and to regulate a range of life history-relevant traits and behaviors.
2. The SRS works as a mechanism of conditional adaptation, regulating the development of alternative life history strategies (i.e., suites of reproductively relevant traits such as sexual maturation, intrasexual competitive behaviors and risk taking, and patterns of mating and parenting). Different patterns of baseline activity and responsivity in early development modulate differential susceptibility to environmental influence and shift susceptible children on alternative pathways, leading to individual differences in life history strategies.
3. Activation of the SRS during the first years of life provides crucial information about life history-relevant dimensions of the child's environment, namely, danger and unpredictability (see Ellis et al., 2009). This information is used to adaptively regulate stress responsivity and associated development of life history strategies.
4. At a general level, a nonlinear relation exists between exposures to environmental stress during development and optimal levels of stress responsivity (see Figure 8.1). This nonlinear relation can be characterized by a taxonomy of four prototypical responsivity patterns (labeled sensitive [I], buffered [II], vigilant [III], and unemotional [IV]). The four patterns constitute combinations of physiological parameters indexing functioning of the PNS, SNS, and LHPA axis (see Figure 8.1 and Table 8.1) and include neurobiological indicators,

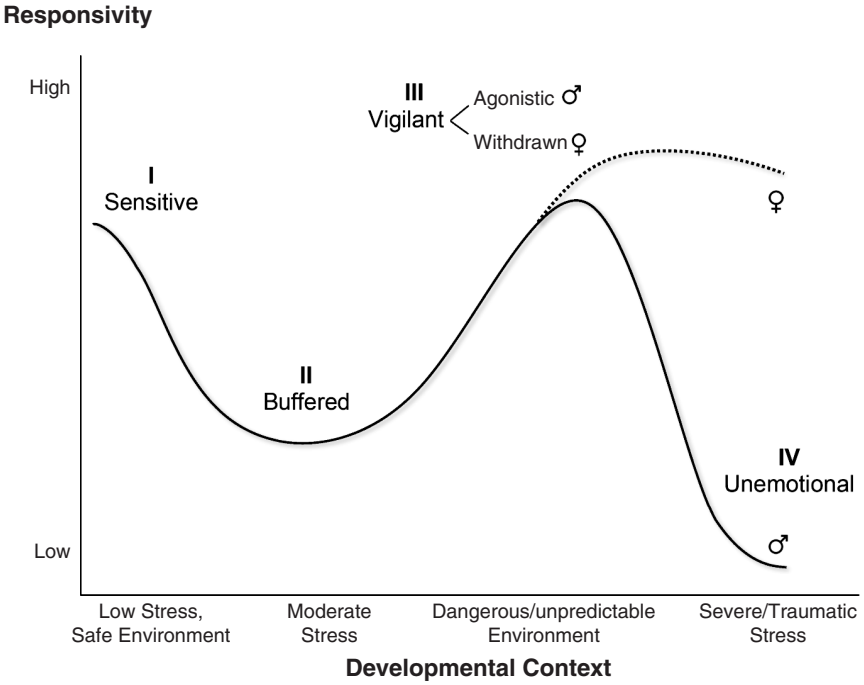

Figure 8.1 The Adaptive Calibration Model of individual differences in development of stress responsivity.

At a general level, a nonlinear relation exists between exposures to environmental stress and support during development and optimal levels of stress responsivity. Although this nonlinear relation is specified for the stress response system (SRS; see Table 8.1), it may apply to other neurobiological systems as well. The figure does not imply that all components of the SRS will show identical responsivity profiles, nor that they will activate at the same time or over the same time course. Male/female symbols indicate sex-typical patterns of responsivity, but substantial within-sex differences in responsivity are expected as well. From Del Giudice et al. (2011).

Table 8.1
Predicted Physiological Profiles of the Four Responsivity Patterns

		Responsivity patterns			
Physiological profile		**I Sensitive**	**II Buffered**	**III Vigilant**	**IV Unemotional**
PNS	*responsivity*	High	Moderate	Low/moderate	Low*
	basal	High	Moderate	Low	Low
SNS	*responsivity*	High/moderate	Low/moderate	High	Low*
	basal	Moderate	Low/moderate	High	Low
HPA	*responsivity*	High	Moderate	High	Low
	basal	Moderate	Moderate	High/moderate	Low

*Unemotional individuals may display autonomic activation when faced with immediate physical threats and during agonistic confrontations, in contrast with their general pattern of unresponsivity to nonagonistic stressors.

From Del Giudice et al. (2011).

behavioral outcomes, and developmental trajectories. Note that environment-responsivity relations need not be the same for all the components of the SRS (see Table 8.1 for detailed predictions).

5. Sensitive and vigilant individuals display relatively high responsivity to the environment, whereas buffered and unemotional individuals display relatively low responsivity. Although comparisons between the two patterns of high responsivity (sensitive vs. vigilant) and the two patterns of low responsivity (buffered vs. unemotional) show substantial *convergence* in SRS baseline activity and responsivity (Table 8.1 and Figure 8.1), there is marked *divergence* in both antecedent environmental conditions and behavioral outcomes.
6. Because of sex differences in optimal life history strategies, sex differences are expected in the distribution of responsivity patterns and in their specific behavioral correlates. Sex differences should become more pronounced at increasing levels of environmental stress; in particular, contexts characterized by severe/ traumatic stress should favor the emergence of a male-biased pattern of low responsivity (the unemotional pattern) and a female-biased pattern of high responsivity (the vigilant-withdrawn pattern).
7. Pre- and early postnatal development, the transition from early to middle childhood, and puberty are likely ''switch points'' for the calibration of stress responsivity. Individual and sex differences in the functioning of the SRS are predicted to emerge according to the evolutionary function of each developmental stage.

ENVIRONMENTAL INFORMATION

One of the crucial functions of the SRS is to collect and integrate information about changing states in the environment (including the presence of threats, dangers, and opportunities) to adjust the state of the whole organism accordingly. This information can be encoded by the SRS and, in the long run, provides the organism with a statistical ''summary'' of key dimensions of the environment. In the ongoing process of physiological adjustment, the system's level of responsivity acts as an amplifier (when highly responsive) or filter (when unresponsive) of various types of contextual information. In this section we consider this function of the SRS in more detail, and take a closer look to the ecological information that can be encoded through repeated SRS activation.

Key Dimensions of the Environment

Life history theory is a general framework for understanding biological trade-offs involved in development, such as those between growth and reproduction, current and future reproduction, and quality and quantity of one's offspring. According to LHT (Charnov, 1993; Stearns, 1992), variation in life history traits results from trade-offs in distribution of resources to competing life functions: bodily maintenance, growth, and reproduction. Due to structural and resource limitations, organisms cannot maximize all components of fitness simultaneously and instead are selected to make trade-offs that prioritize resource expenditures, so that greater investment

in one domain occurs at the expense of investment in competing domains. For example, resources spent on an inflammatory host response to fight infection cannot be spent on reproduction. Thus, the benefits of an inflammatory host response are traded off against the costs of lower fertility. Each trade-off constitutes a decision node in allocation of resources, and each decision node influences the next decision node (opening up some options, foreclosing others) in an unending chain over the life course (Ellis et al., 2009).

Most important for the present discussion, LHT can be used to predict how organisms adjust their developmental trajectories according to variable ecological conditions. The key dimensions of the environment relevant to life history development are availability of resources, level of unavoidable danger (or, more precisely, extrinsic morbidity-mortality rate), and predictability of environmental change. Energetic resources—caloric intake, energy expenditures, and related health conditions—set the baseline for development, slowing growth and delaying sexual maturation and reproduction under energetic stress (i.e., favoring a ''slow'' life history strategy). When energetic resources are adequate, cues of extrinsic morbidity-mortality and unpredictability gain importance (Ellis et al., 2009). Given adequate bioenergetic resources to support growth and development, individuals should detect and respond to proximal cues of danger (e.g., exposures to violence, harsh childrearing practices) and unpredictability (e.g., stochastic changes in ecological context, economic conditions, family composition, parental behavior) by entraining faster life history strategies (see Belsky, Schlomer, & Ellis, 2012; Simpson, Griskevicius, Kuo, Sung, & Collins, 2012, for supporting longitudinal data). Fast life history strategies are comparatively high risk (taking benefits opportunistically with little regard for long-term consequences), focusing on mating opportunities (e.g., competitive risk taking, aggression), reproducing at younger ages, and producing a greater number of offspring with more variable outcomes. As discussed later, trade-offs incurred by the fast strategy include reduced health, vitality, and longevity—of self and offspring.

The SRS is attuned exquisitely to the life history-relevant features of the environment. Of particular interest, the level of extrinsic morbidity-mortality is conveyed both by frequent SNS activation (signaling a potentially dangerous ecology) and by repeated LHPA activation. Because it responds strongly to uncontrollable challenges and novel situations, the LHPA axis also encodes information about environmental unpredictability/uncontrollability, thus giving LHPA functioning a central role in the regulation of life history strategies (see Del Giudice et al., 2011). Across development, environmental information collected by the SRS (in interaction with the child's genotype) canalizes physiological and behavioral phenotypes to match local ecological contexts.

The SRS as an Information Filter/Amplifier

If the SRS encodes environmental information as a statistical aggregation of repeated responses to challenge, it follows that SRS responsivity can function as an information filter. Low SRS responsivity results in a number of potential costs (e.g., reduced

alertness, reduced sensitivity to social feedback) and potential benefits (e.g., resource economization, avoidance of immune suppression). A highly responsive SRS, by contrast, amplifies the signal coming from the environment and maximizes the chances that the organism will be modified by current experience. Potential costs of a highly responsive system include adverse physiological events, hypersensitivity to social feedback, and exposure to psychological manipulation. In addition, the organism's action plans can get interrupted easily by minor challenging events, and the ability to deal with future events may be reduced if physiological resources are already overwhelmed. On the other hand, a highly responsive system facilitates social learning and social bonding, enhances mental activities in localized domains, focuses attention, and primes memory storage, thus tuning cognitive processes to opportunities and threats in the environment.

Empirical studies (e.g., Pruessner et al., 2010) illustrate how SRS thresholds for responding to environmental stimuli differ dramatically from one person to another. It is also intriguing that such thresholds may show domain-specificity, as when challenges related to competition or achievement are more salient for males, but challenges related to social exclusion or rejection are more salient for females (Stroud et al., 2009; Stroud, Salavey, & Epel, 2002). Individual differences in the functional parameters of the SRS depend on complex causal chains across genetic, epigenetic, and neuroendocrine levels. Close social relationships can also filter/amplify more distal environmental factors, such as when cortisol reactivity is suppressed in the presence of a warm, supportive caregiver (e.g., Fries, Shirtcliff, & Pollak, 2008).

Although ACM terminology tends to emphasize the role of responsivity, components of the SRS operate at both state (situation-specific) and trait (basal) levels. Basal functioning indicates a level of physiological preparedness or anticipation of the individual's context (Pruessner et al., 2010), exerting a permissive effect on the individual's ability to respond to novel events and encode environmental information (e.g., Gunnar & Quevedo, 2007). It may also provide a rough index of the physiological accumulation of prior stressful events. High basal SRS activity is expected when the individual anticipates or needs to be engaged, aroused, or active in that context. High basal activation of the PNS promotes calm, concentration, and self-regulation (e.g., Fabes & Eisenberg, 1997; Porges, 2007), whereas high SNS baseline relates to anxiety (El-Sheikh, Erath, Buckhalt, Granger, & Mize, 2008), and baseline cortisol secretion regulates energy mobilization and engagement with the physical and social environment (Booth, Granger, & Shirtcliff, 2008). This role of the SRS in relation to anticipation is emphasized, for example, in an extensive literature demonstrating high cortisol in contexts characterized by unpredictability (Dickerson & Kemeny, 2004). Empirical findings that cortisol levels elevate prior to laboratory arrival (e.g., Ellis et al., 2005; Hastings et al., 2011) or in anticipation of challenges of the day (e.g., Fries, Dettenborn, & Kirschbaum, 2009; Schmidt-Reinwald et al., 1999) further bolster the interpretation that basal SRS activity serves an anticipatory or preparatory function.

Over time, repeated SRS responses to environmental challenges may accumulate so that state-specific activity patterns become part of the individual's trait-like

functional parameters (Shirtcliff, Granger, Booth, & Johnson, 2005). Basal functioning of the SRS achieves set-points that calibrate the individual's physiology with the expected environmental demands, but as the environment changes, so, too, may the optimal set-point (McEwen & Wingfield, 2003). This implicates one of the most important functions of the SRS: to change according to anticipated or current context, using those changes to optimize physiological functioning for the expected future conditions.

Implications for Developmental Psychopathology

Looking at the SRS through the lens of information filtering and encoding provides useful insights into the developmental processes that ultimately lead to psychopathological outcomes. First, and foremost, this reconceptualization of the functioning of the SRS as a mechanism of susceptibility to environment influence (Boyce & Ellis, 2005) helps to explain the bivalent effects of stress responsivity on mental and physical health, whereby highly reactive children experience either the best or the worst of psychiatric and biomedical outcomes depending on levels of stress and support encountered over development (see earlier, BSC). A radical implication of this theory is that the very children whose heightened responsivity appears to make them vulnerable to developing psychopathology may also be most able to benefit from positive, supportive environments and interventions. For example, consider the results of two studies of naturally occurring environmental adversities and stress reactivity as predictors of respiratory illnesses in 3- to 5-year-old children (Boyce et al., 1995). Results revealed, first, that children showing low cardiovascular or immune reactivity to stressors had approximately equal rates of respiratory illnesses in both low and high adversity settings. Second, and consistent with the prevailing ALM of developmental psychopathology, highly biologically reactive children exposed to high adversity child care settings or home environments had substantially higher illness incidences than all other groups of children. The third finding, however, was that highly reactive children living in lower adversity conditions—that is, more supportive child care or family settings—had the *lowest* illness rates, significantly lower than even low reactivity children in comparable settings, supporting a BSC interpretation. Thus, the very qualities that appear to increase children's frailties may also be their strength, given supportive contexts, thus inspiring the metaphor of ''orchid children'' (Boyce & Ellis, 2005).

In addition, LHT delineates basic dimensions of environmental stress and support—underscoring resource availability, morbidity-mortality risk, and unpredictability as key dimensions of the environment that regulate development of SRS responsivity patterns and their behavioral correlates (see the next section). This has already proven to be a valuable tool in empirical research (e.g., Belsky et al., 2012; Simpson et al., 2012), given the confusing abundance of environmental/contextual variables that might be measured and correlated with developmental outcomes. Furthermore, LHT provides organizing principles needed to understand the broad network of interactions between the SRS and other physiological response systems, such as the immune system (see Miller, Chen, & Parker, 2011).

Another important implication of the concepts reviewed in this section is that both high- and low SRS responsivity can be adaptive precisely because they modulate the organism's openness to environmental information. As discussed earlier, there is no optimal level of responsivity; rather, the value of high versus low informational openness varies depending on the local ecology, and in some cases an unresponsive system can be highly functional in the context of an individual's life history strategy. This idea is developed in the next section.

PATTERNS OF RESPONSIVITY

The ACM builds on the theoretical principles outlined in the previous sections to derive a taxonomy of four prototypical responsivity patterns. Each pattern describes an integrated mode of SRS functioning, life history-relevant behavioral tendencies, and plausible neurobiological correlates. Three of the patterns correspond to regions on the U-shape curve of the BSC theory; the fourth pattern is a novel addition, and accounts for the development of hypoarousal in severely stressful conditions.

The Logic of Hypoarousal

Some individuals show a persistent pattern of markedly reduced SRS basal activity and responsivity, even following stimuli that elicit strong physiological reactions in most people. So-called hypoarousal or hyporesponsivity is reliably associated with externalizing behaviors, conduct disorders, and psychopathic traits (especially from middle childhood on; e.g., Ortiz, & Raine, 2004), which makes it especially interesting from the perspective of developmental psychopathology. Hypoarousal is usually treated as a sign of physiological dysregulation (e.g., Lupien et al., 2006); interestingly, chronic early adversity can lead to both hyper- and hypo-responsivity of the SRS (e.g., De Bellis et al., 1999; Gustafsson et al., 2010; Tarullo & Gunnar, 2006; Yehuda, 2002). The ACM suggests that dampened responsivity may actually follow an adaptive logic, as a way to maximize the fitness benefit/cost ratio in severely dangerous and unpredictable environments (see Gatzke-Kopp, 2011, for a similar account regarding dopaminergic responsivity). In moderately dangerous contexts, a responsive SRS enhances the individual's ability to react appropriately to dangers and threats while maintaining a high level of engagement with the social and physical environment. Moreover, engaging in fast life history strategies should lead the individual to allocate resources in a manner that discounts the long-term physiological costs of the stress response in favor of more immediate advantages. In this context, the benefits of successful defensive strategies outweigh the costs of frequent, sustained LHPA and SNS activation.

When danger becomes severe, however, engaging in high levels of risk taking (e.g., antagonistic competition, impulsivity and extreme discounting of the future) can become the optimal response from an evolutionary perspective (see Ellis et al., 2012). Note that such strategies require outright *insensitivity* to threats, dangers, and social feedback. An unresponsive SRS has a higher threshold for letting environmental signals in: Many potential threats will not be encoded as such, and many

potentially relevant events will fail to affect physiology to a significant degree. For an extreme risk taker, however, informational insulation from environmental signals of threat is an asset, not a weakness (see also Korte et al., 2005). In particular, adopting an exploitative/antisocial interpersonal style requires one to be shielded from social rejection, disapproval, and feelings of shame (all amplified by heightened LHPA responsivity). In summary, generalized low responsivity can be evolutionarily adaptive (i.e., fitness-maximizing) at the high-risk end of the environmental spectrum, despite the possible negative consequences for the social group and for the individual's subjective well-being. This type of chronic low responsivity should be carefully distinguished from temporary ''exhaustion'' periods, usually arising after prolonged SRS activation in highly responsive individuals exposed to enduring stressors (Miller, Chen, & Zhou, 2007).

The Logic of Sex Differences

In sexually reproducing species, the two sexes differ predictably on life history-related dimensions. They are thus expected to employ different strategies in response to the same environmental cues (e.g., Geary, 2002; James, Ellis, Schlomer, & Garber, 2012). In mammals, including humans, males tend to engage in higher mating effort and lower parental effort than females (Geary, 2002; Kokko & Jennions, 2008; Trivers, 1972). In addition, males usually undergo stronger sexual selection, that is, their reproductive success is more variable than that of females, leading to higher risk propensity (Trivers, 1972; see also Frankenhuis & Del Giudice, 2012). The extent of sex differences in life history-related behavior, however, is not fixed but depends in part on the local environment.

At the slow end of the life history continuum, both sexes tend to engage in high parental investment, and male and female interests largely converge on long-term, committed pair bonds; sex differences in behavior are thus expected to be relatively small. As environmental danger and unpredictability increase, males benefit by shifting to low-investment, high-mating strategies; females, however, do not have the same flexibility since they benefit much less from mating with multiple partners and incur higher fixed costs through childbearing. Thus, male and female strategies should diverge increasingly at moderate to high levels of danger/unpredictability. In addition, sexual competition takes different forms in males and females, with males engaging in more physical aggression and substantially higher levels of risk-taking behavior. As life history strategies become faster, sexual competition becomes stronger, and sex differences in competitive strategies become more apparent.

For these reasons, sex differences in responsivity patterns and in the associated behavioral phenotypes should be relatively small at low- to moderate levels of environmental stress, and increase as the environment becomes more dangerous and unpredictable. In particular, males should be more likely to develop unresponsive phenotypes in highly stressful contexts. Also, the behavioral correlates of both high- and low responsivity in dangerous environments can be expected to differ between the sexes. As we discuss later, we do not expect sex differences in responsivity to be present from birth, but rather to emerge gradually during development.

The Four ACM Patterns

It is now possible to present a brief outline of the four ACM patterns (see Del Giudice et al., 2011, for a detailed description). Each pattern represents a stable configuration of SRS activity. Later, we discuss developmental pathways leading to establishment of these configurations.

Sensitive pattern (Type I). Sensitive patterns are hypothesized to develop in safe, predictable conditions and warm, family environments. High stress responsivity in sensitive individuals increases their openness to social and physical environments. Physiological profiles of those with this pattern (high LHPA and PNS responsivity, moderate SNS responsivity) favor sustained but flexible attention and sensitivity to social feedback. Sensitive individuals are reflective, self- and other-conscious, and engaged with the environment. They are high in inhibitory control, delay of gratification, and executive function. These traits promote sustained learning and cooperation. Other plausible correlates are high serotonergic function and slow sexual maturation.

Buffered pattern (Type II). Buffered patterns are predicted to develop preferentially in conditions of moderate environmental stress, where they strike a balance between costs and benefits of responsivity. Compared to Type III and IV patterns, buffered individuals should be lower in anxiety, aggression, and risk taking.

Vigilant pattern (Type III). Vigilant patterns develop in stressful contexts, where they enable people to cope effectively with dangers and threats in the physical and social environment. Their SNS-dominated physiological profile mediates heightened attention to threats and high trait anxiety. Increased SRS responsivity in dangerous environments can be expected to go together with increased responsivity in other neurobiological systems. For example, hyperdopaminergic function may contribute to the vigilant phenotype by boosting attention to threat-related cues and fast associative learning (Gatzke-Kopp, 2011). In the ACM, vigilance is not associated with a single behavioral pattern, but rather with a *distribution* of patterns involving different mixtures of aggressive/externalizing (''fight'') and withdrawn/internalizing (''flight'') behaviors. In males, vigilant responsivity should be associated more often with increased risk taking, impulsivity, agonistic social competition and reactive aggression (the vigilant-agonistic subtype). In females, the typical pattern should involve social anxiety and fearful/withdrawn behavior (the vigilant-withdrawn subtype). Vigilant children who display high levels of both agonistic and withdrawn behaviors (typically females; Zahn-Waxler et al., 2006) may be best described as belonging to a third subtype, the vigilant-agonistic/withdrawn pattern.

Unemotional pattern (Type IV). Unemotional patterns are marked by a profile of low stress responsivity, with the possible exception of strong autonomic responses when facing immediate physical threats. Generalized unresponsivity inhibits social learning and sensitivity to social feedback; it can also increase risk taking by blocking information about dangers and threats in the environments. Predicted correlates of this pattern are low empathy and cooperation, impulsivity, competitive risk taking, and antisocial behavior, including high levels of proactive/instrumental aggression, especially in males. Based on LHT, the distribution of Type IV is expected to be male-biased, and its behavioral correlates are expected to differ between sexes.

For example, one key feature of unemotional responsivity in females may be a generalized pattern of aloof social relationships with parents, siblings and peers. Low serotonergic and dopaminergic activity are likely neurobiological correlates of Type IV.

Preliminary empirical evidence supports the ACM taxonomy in children aged 8 to 10 years (Del Giudice, Hinnant, Ellis, & El-Sheikh, 2012), showing the presence of four major classes that provide a satisfactory match to the patterns described here. However, the sample size of this initial study is comparatively small; larger samples spanning a wider age range are needed to fully evaluate the ACM taxonomy, especially in view of the gradual emergence and consolidation of responsivity patterns and their distribution by sex (see next section).

Implications for Developmental Psychopathology

The logic sketched in this section has several implications for developmental psychopathology. First, it provides a functional account of hypoarousal that goes beyond ''dysregulation,'' begins to explain why early adversity can have divergent outcomes (hyper- versus hypoarousal), and suggests that sex-related factors (such as sex hormones) may play an important role in determining the behavioral and physiological outcomes of early stress. Second, an evolutionary focus permits a better understanding of comorbidity patterns. For example, many superficially different traits and behaviors (e.g., aggression, early and promiscuous sexuality, substance abuse, reduced empathy) can be seen as manifestations of high-risk life history strategies that discount the future and increase mating effort. Consistent with this perspective, externalizing problems and precocious sexual behaviors in children not only covary, but also share many etiological factors (see Lévesque, Bigras, & Pauzé, 2010). Similarly, sex differences in the development of vigilant versus unemotional phenotypes may help to explain why girls show higher comorbidity between aggression and anxiety/depression: when exposed to highly stressful conditions, boys more than girls tend to develop an unemotional phenotype, characterized by high externalizing and low internalizing behaviors.

Finally, the ACM helps clarify complex relations between psychosocial environmental factors and stress responsivity patterns. Psychosocial stress and adversity over development can either up-regulate or down-regulate levels of SNS, PNS, and LHPA responsivity. The empirical literature on this topic remains highly conflicted: For every study linking stressful rearing experiences to hyperarousal (e.g., De Bellis et al., 1999; Essex, Klein, Cho, & Kalin, 2002; Yehuda, 2002) another study links such experiences to hypoarousal (e.g., Gunnar & Vazquez, 2006; Gustafsson et al., 2010; Tarullo & Gunnar, 2006). The ACM potentially explains both hyperarousal and hypoarousal by specifying nonlinear relations between environmental conditions and development of stress responsivity (Figure 8.1). According to the theory, developmental exposures to low- to moderate levels of stress either up-regulate (in the sensitive pattern) or down-regulate (in the buffered pattern) responsivity. Likewise, developmental exposures to high levels of stress either up-regulate (in the vigilant pattern) or down-regulate (in the unemotional pattern) responsivity.

Thus, if one considers the environment-responsivity curves shown in Figure 8.1, it is apparent that the results of any single study looking at linear statistical relationships can range from positive to null to negative, depending on the portion of the curve sampled in each case (Boyce & Ellis, 2005; Ellis et al., 2005). The many inconsistent results in the stress literature may depend, at least in part, on the failure to consider nonlinear relationships between environmental factors and SRS parameters, or the failure to assess the full range of environmental variance necessary to capture all four patterns of responsivity and associated behavioral strategies specified by the ACM.

DEVELOPMENTAL PATHWAYS AND TRANSITIONS

Individual differences in stress responsivity are likely present prenatally, but patterns we described in the previous sections emerge as outcomes of long-term developmental processes. In these processes, the SRS collects environmental information and is shaped by that information, in a dynamic interplay with other key neurobiological systems. Developmental pathways are thus an integral aspect of the ACM, and they connect our model to a broader evolutionary theory of human development. Central to our discussion is the concept of a *developmental switch point*, the elementary unit in the development of plastic organisms.

Developmental Switch Points

West-Eberhard (2003) proposed that developmental change is coordinated by regulatory switch mechanisms, which serve as transducers (mediators) of genetic, environmental, and structural influences on phenotypic variation. These switch mechanisms control *developmental switch points*: ''a point in time when some element of phenotype changes from a default state, action, or pathway to an alternative one—it is activated, deactivated, altered, or moved'' (West-Eberhard, 2003, p. 67). This can involve a discrete structural change or a change in the rate of a process. Genetic and environmental inputs interact with phenotypic qualities to determine the functioning of regulatory switch mechanisms and influence their thresholds. Once a threshold is passed (i.e., the switch occurs), the regulatory mechanism coordinates expression and use of gene products and environmental elements that mediate the species-typical transition to the new phenotypic stage, as well as individually differentiated pathways within that stage. A concrete example of a switch-point in human development is puberty (see Ellis, 2011, for more details).

Critically, regulatory switch mechanisms provide a common locus of operations for genetic and environmental influences on phenotypic development; that is, these mechanisms are the vehicle through which gene-gene, environment-environment, and gene-environment interactions occur (see Chapter 3). These inputs structure the operation of regulatory switch mechanisms and may affect thresholds necessary for a developmental switch to occur and/or the organism's ability to cross that threshold (West-Eberhard, 2003). This is also the most crucial difference between

a developmental switch point and a *sensitive* or *critical period*. A sensitive period is defined by increased susceptibility to environmental input, whereas a developmental switch point is marked by increased susceptibility to both environmental *and* genetic effects. For example, the activation of sex hormone-related biochemical pathways at puberty induces expression of genetic variation that was previously silent. This, in turn, creates new opportunities for gene-environment interactions, which may alter the direction of individual developmental trajectories.

Stages and Transitions in Human Life History

Human life history can be described as a sequence of stages and transitions (Bogin, 1999). Life history strategies unfold progressively, according to the evolutionary function of each life stage. Del Giudice and Belsky (2011) proposed that the major switch points in human life history strategies occur during (a) pre- and early postnatal development, (b) the transition from early to middle childhood or *juvenile transition*, and (c) puberty. The juvenile transition (Del Giudice, Angeleri, & Manera, 2009) takes place at around 6 to 8 years in Western societies, and is marked by ''adrenal puberty'' or *adrenarche*, whereby the cortex of the adrenal glands begins to secrete increasing quantities of androgens, mainly dehydroepiandrosterone (DHEA) and its sulfate (DHEAS).

The onset of human juvenility (i.e., middle childhood) witnesses massive changes in children's social behavior, cognitive abilities, and the emergence or intensification of sex differences in aggression, attachment, play, language use, and so forth (reviewed in Del Giudice et al., 2009). In an evolutionary perspective, the main functions of juvenility are learning (including social and practical skills) and competition for status and social resources in the peer group. With the onset of puberty, sexual behavior and romantic attachment come to the forefront, and social competition further intensifies. Puberty affords another opportunity to revise one's life history strategy, depending for example on the success enjoyed—or the level of competition experienced—during juvenility.

Developmental Pathways in the ACM

The development of stress responsivity begins prenatally, with interactions between fetal genes and maternal hormones (see Pluess & Belsky, 2011). The first years of life are also important because they provide the child with information about their local environment, both directly and indirectly through their parents' behavior. In this phase, high levels of stress are expected mainly to increase SRS responsivity (vigilant phenotypes), whereas moderate, repeated activation of the SRS leads to the development of buffered responsivity.

In the ACM, we argue that the juvenile transition is the first critical turning point in the development of stress responsivity. We predict that sex differences in responsivity patterns and their behavioral correlates should emerge from the beginning of middle childhood, with a further increase at puberty. Second, we

expect individual changes in responsivity to be especially frequent in the transition from early to middle childhood, possibly driven by adrenal androgens. Some children (especially males) who grow up under conditions of severe stress may display a highly responsive profile in early childhood, then shift to low responsivity as social competition becomes a central developmental task. Thus, we expect a number of individuals—likely the most aggressive ones—to display a transition from Type III- to Type IV during juvenility or adolescence. The development of unemotional responsivity may also follow a different pathway, dependent on strong genetic dispositions. In this pathway, unemotional traits may appear in childhood, even in low-stress environments (Del Giudice et al., 2011; see discussion earlier).

Finally, it is noteworthy that we predict sensitive patterns to develop in infants and young children who are emotionally labile initially and characterized by difficult or inhibited temperament. As they grow up in a protective environment, however, they may become less anxious, more stable emotionally and more confident socially, possibly even more so than their temperamentally stable peers (reviewed in Ellis et al., 2011). At the physiological level, this shift is likely to be marked by increased PNS tone and responsivity (see Del Giudice et al., 2011).

Implications for Developmental Psychopathology

Much work needs to be done before we will achieve a principled, comprehensive description of developmental pathways, and many important details (for example the mechanics of gene-environment interactions and epigenetic regulation) are yet to be filled in. However, we feel that our approach to human development has important heuristic advantages. At a general level, the life history perspective, with its emphasis on underlying trade-offs as explanations for manifest traits, is a natural framework for understanding equifinality and multifinality in development (Pickles & Hill, 2006), as well as the related issue of homotopic and heterotopic continuity (Costello & Angold, 2006). In a nutshell, the three main insights are that superficially similar behaviors may actually be serving different strategies (and reflect different patterns of responsivity); that the same strategy may require different behaviors at different times; and that the same feature of the environment may have very different biobehavioral implications for individuals engaging in different strategies with different cost-benefit balances.

Just as important, a biologically informed view of developmental stages and transitions provides content and specificity to the study of how stress exposure can have different effects according to its developmental timing (e.g., Ganzel & Morris, 2011). Also, empirical findings from developmental epidemiology (Costello & Angold, 2006) acquire more meaning when put in a proper evolutionary-developmental framework (see Del Giudice et al., 2009). As a specific example, the hypothesized transition from vigilant to unemotional patterns under the influence of adrenal androgens may explain the puzzling finding that externalizing and aggressive behavior are associated with high cortisol levels in preschoolers, but low cortisol levels from middle childhood on (Alink et al., 2008; Shirtcliff et al., 2005).

ADAPTIVE CALIBRATION AND THE ALLOSTATIC LOAD MODEL

With the ACM we are seeking an integrative theoretical framework for the study of stress and stress responsivity across development. Presently, the main contender for this role is the allostatic load model (ALM; McEwen & Stellar, 1993). The ALM has become quite popular in developmental psychopathology (e.g., Beauchaine et al., 2011; Lupien et al., 2006), and in recent years researchers have started adopting it as a foundation for interdisciplinary integration (e.g., Ganzel, Morris, & Wethington, 2010; Juster et al., 2011). In this section, we review key points of convergence and divergence between the two models and explain why we consider the ACM to have many advantages and to be a more viable alternative in some important domains of inquiry.

The Allostatic Load Model

The key concept of the ALM is that of allostasis, or ''stability through change.'' Allostasis comprises processes through which an organism adapts to environmental challenges by modifying its regulatory parameters (e.g., by increasing or decreasing the set point of a homeostatic physiological mechanism). The term is typically used to describe the ''moment to moment process of establishing a new homeostatic equilibrium in the face of challenge'' (Ganzel & Morris, 2011, p. 956) or, stated differently, ''how continuous reevaluation and readjustments create new set points that maximize the organism's resources (e.g., increased cardiac output when running)'' (Juster et al., 2011, p. 727). Here allostasis enables accommodation to current stressors (Lupien et al., 2006). However, some authors (e.g., Beauchaine et al., 2011) restrict the meaning of allostasis to long-term, potentially permanent changes in the system's parameters in contexts of protracted stress—what McEwen and Wingfield (2003) labeled ''allostatic states.'' The SRS is a crucial mediator of allostasis, though many other central and peripheral structures initiate and sustain allostatic responses (see Ganzel et al., 2010).

Allostatic load is a label for the costs of allostasis and is often described as the ''wear and tear'' that results from repeated allostatic adjustments, exposing the organism to adverse health consequences. The central tenet of the ALM is that the stress response is usually adaptive in the short term (i.e., acute stress responses mobilize biological resources that permit fight or flight responses that are normally protective against danger), but maladaptive and damaging in the long term (see Lupien et al., 2006; McEwen & Stellar, 1993). As eloquently stated by Juster et al. (2011, p. 725):

> Central to this biological damage is altered stress hormone functioning that inexorably strains interconnected biomarkers that eventually collapse like domino pieces trailing toward stress-related endpoints.

Among other adverse outcomes, allostatic load is thought to cause SRS dysregulation, resulting for example in excessive or insufficient responses to stressors and increasing the risk for psychopathology. The idea of physiological dysregulation

is integral to the ALM, and both ''hyperarousal'' and ''hypoarousal'' are routinely described as dysfunctional deviations from the norm (e.g., Beauchaine et al., 2011; Juster et al., 2011; Lupien et al., 2006), usually caused by a combination of excessive stress exposure and genetic or epigenetic vulnerability.

ACM Versus ALM

It should be noted at the outset that there are significant points of convergence between the ACM and the ALM. First, the ACM explicitly embraces the concept of allostasis and describes the coordination of allostatic responses as one of the main biological functions of the SRS. The ACM also acknowledges that chronic SRS activation does carry substantial costs, in terms of biological fitness as well as subjective well-being. Finally, whereas the ACM focuses on conditional adaptation, it leaves open the possibility that—for a number of reasons—some developmental outcomes are biologically maladaptive (see earlier discussion).

From an evolutionary standpoint, the biggest limitation of the ALM is that no distinction is made between the two meanings of ''adaptive'' (and maladaptive) described above: positive versus negative biological fitness outcomes, on the one hand, and desirable versus undesirable mental and physical health outcomes, on the other. Maladaptation is inferred whenever there are costs for the organism. For example, if elevated cortisol levels in children are associated with a negative outcome, such as reduced working memory, then elevated cortisol is classified as a marker of allostatic load; Juster et al., 2011). This reasoning ignores the crucial fact that biological processes are adaptive when their fitness benefits outweigh the costs, *not* when they are cost-free. As discussed earlier, even large costs can be offset by large enough expected benefits. For example, in dangerous and unpredictable environments, organisms often accept the risk of severe damage in exchange for a chance of improving their condition (see Ellis et al., 2012; Frankenhuis & Del Giudice, 2012). Similarly, when health and reproductive success conflict, natural selection favors the latter at the expense of the former (see Nesse, 2001).

Because of the failure to distinguish between (mal)adaptive and (un)desirable outcomes, most applications of the ALM do not address the trade-offs involved in the development of physiological and behavioral phenotypes; as a consequence, the ALM literature often lacks a theory of adaptive individual variation in stress responsivity (but see Korte et al., 2005, for a notable exception). Although the ALM is sophisticated in explaining the costs of allostasis, it only captures the short-term benefits of allostasis and does not consider the long-term benefits in terms of regulating conditional adaptation to varying environmental conditions. As a result, the development of enduring individual differences is usually traced to pathogenic processes.

In contrast, the ACM is built on life history theory, which is a theory of inherent trade-offs in the life cycle of organisms, and explicit consideration of these trade-offs is at the heart of the ACM taxonomy of responsivity patterns. For example, consider heightened SRS responsivity in vigilant patterns (Type III). In the ACM, it is hypothesized that the costs of repeated SRS activation are offset by improved

management of danger. Although the system is on a hair trigger, with the resulting burden of anxiety and/or aggression, few instances of actual danger will be missed. In addition, engaging in a ''fast,'' present-oriented life history strategy makes it optimal to discount the long-term health costs of chronic SRS activation if the immediate benefits are large enough (for in-depth discussion, see Del Giudice et al., 2011). In the ALM framework, the same pattern of responsivity would be treated as dysfunctional, because the stress response is deployed even in absence of true dangers (''excessive'' response, ''unnecessary'' triggering; see Beauchaine et al., 2011; Lupien et al., 2006) and because of the associated unpleasant states and health risks. This approach, however, fails to consider that natural defenses are usually designed by natural selection to accept a high rate of false positives (the so-called smoke detector principle; Nesse, 2005). Moreover, adaptive defenses, from environmentally triggered surges in catecholamines and glucocorticoids to development of fever in response to an infection, are often aversive, disabling, and occasionally harmful (or even fatal); but mistaking them for diseases because of these superficial features is a fallacy, though one that is exceedingly common in the psychopathology literature (see Nesse & Jackson, 2006).

A related point of divergence between the ACM and the ALM concerns responses to acute versus chronic stress. In the ALM, adaptive responses to acute stress are contrasted with the biological ''wear and tear'' caused by chronic stress and the resulting long-term modifications of SRS regulatory parameters. In the ACM, responses to both acute and chronic stress can be adaptive (though not cost-free); and, as a rule, the long-term adjustment of SRS parameters is seen as adaptive calibration rather than maladaptive dysregulation. Indeed, we anticipate that many of the allegedly ''toxic'' effects of chronic stress (e.g., its effects on immune function, brain physiology, memory, learning, and so forth) will ultimately find a better explanation as mediators of biological fitness trade-offs. In total, the ALM, relative to the ACM, overemphasizes the costs of allostasis and underappreciates its benefits.

For example, the ALM and ACM both recognize that childhood exposures to stress and adversity often result in physiological, cognitive, behavioral, and maturational changes in the developing organism (e.g., new SRS set points, elevated sensitivity to threat cues, attenuated delay of gratification, early puberty), and that over time these changes may mediate undesirable mental and physical health outcomes. From an evolutionary perspective, however, these outcomes reflect the costs of life history strategies—strategies instantiated in a chain of resource allocation decisions over the life course—that ''make the best of a bad situation'' by trading off survival for reproduction. According to the ACM, the SRS collects environmental information, is shaped by that information (allostasis), and uses it to adaptively the match development of life history strategies to local conditions. Physiological, cognitive, behavioral, and maturational changes in response to environmental harshness and unpredictability function to accelerate life history strategies, shifting resource allocations toward more risky and aggressive behavior, earlier pubertal timing and sexual debut, less stable pair bonding, more children, and less parental investment per child (e.g., Ellis et al., 2009)—at the price of reduced health and longevity

(i.e., allostatic load; see especially Allsworth, Weitzen, & Boardman, 2005). These biological fitness trade-offs are discussed further below.

Implications for Developmental Psychopathology

The ALM and the concept of allostatic load have become remarkably popular in developmental psychopathology. Here we argue that the ALM has a number of limitations, and that the ACM provides researchers with a theory of stress responsivity that is broader and more consistent with the principles of evolutionary biology. We recognize that the ALM may be especially attractive because it conforms to the implicit assumptions of the standard mental health approach, particularly regarding stress-disease relationships, and therefore does not require any fundamental shift in thinking and logic. However, it also fails to deliver the insight and heuristic power of a modern evolutionary-developmental framework. In the long run, the field of developmental psychopathology may be better served by a model informed by life history theory, modeling of strategic trade-offs, and a more sophisticated consideration of the relations between adaptation, health, and well-being.

We believe that the ACM embodies the main insights of the ALM without sharing its limitations. Even more importantly, most of the work that is presently carried out under the ALM umbrella could be easily reframed in the perspective of the ACM. For example, the ACM taxonomy of responsivity patterns already contains predictions on likely neurobiological correlates such as dopaminergic, serotonergic, and noradrenergic function (Del Giudice et al., 2011). This makes the ACM a natural catalyst for the integrative work initiated by Beauchaine and colleagues (2011) and Ganzel and colleagues (2010). Similarly, the theory of developmental stages and switch points embodied in the ACM might serve as a detailed, biologically-grounded foundation for the analysis of the effects of stress exposure at different points in the life cycle (Ganzel et al., 2011).

Finally, the ACM addresses major anomalies in the field regarding complex relations between psychosocial environmental factors, stress responsivity, life history relevant traits and behaviors, and health. In the ALM, both hyperarousal and hypoarousal are considered indicators of stress dysregulation resulting from allostatic load, and the developmental pathways leading to systematic up-regulation versus down-regulation of SRS parameters are not theoretically modeled (rather, hyperarousal and hypoarousal are grouped together as dysfunctional deviations). We believe that this is a substantial limitation of the ALM. Valid explanatory models of the developmental pathways leading to both hyper and hyporesponsivity are critical to explaining the development of psychopathology because both heightened and dampened responsivity can look either good or bad in terms of behavioral adjustment and health. Such bivalent effects of the SRS have been documented in SNS, PNS, and LHPA studies focusing on both baseline arousal and responsivity (e.g., Bauer, Quas, & Boyce, 2002; Burke, Davis, Otte, & Mohr, 2005; Evans & English, 2002).

A vast body of research has shown that children exposed to higher levels of psychosocial stress and adversity tend to develop more mental and physical health

problems, and that these relations are often moderated by variation in stress responsivity. However, for every study showing that heightened stress reactivity operates as a *risk factor* that increases children's vulnerability to psychosocial stress (e.g., Boyce et al., 2006; Obradovic, Bush, & Boyce, 2011; Obradovic, Bush, Stamperdahl, Adler, & Boyce, 2010) another study shows that heightened stress reactivity operates as a *protective factor* that reduces children's vulnerability to psychosocial stress (e.g., Calkins & Keane, 2009; Degnan, Calkins, Keane, & Soderlund, 2008; El-Sheikh, Harger, & Whitson, 2001). The ACM potentially explains these anomalous findings by specifying two patterns of heightened stress reactivity (sensitive and vigilant phenotypes) and two patterns of dampened stress reactivity (buffered and unemotional phenotypes). Most importantly, each phenotype is characterized by different developmental histories and behavioral and health trajectories. Accordingly, *heightened* reactivity may look like a protective factor in sensitive phenotypes and a risk factor in vigilant phenotypes, while *dampened* reactivity may look like a protective factor in buffered phenotypes and a risk factor in unemotional phenotypes.

As discussed earlier, some people develop slower life history strategies characterized by later reproductive development and behavior. The slower strategy also involves greater allocation of resources toward enhancing growth, vitality, and long-term survival. By contrast, others develop faster strategies characterized by the opposite pattern. Because slower life history strategists, by definition, allocate more bioenergetic resources to somatic effort, meaning growth and maintenance of one's body (*soma* in Greek), life history theory predicts that they will generally experience better physical and mental health than will faster life history strategists, and more so as the life course progresses. This prediction has been supported by empirical research, both cross-sectional and longitudinal, showing that individuals who pursue faster life history strategies suffer from more mental health problems, medical ailments (e.g., thyroid disease, high blood pressure or hypertension, ulcers), and physical health symptoms (e.g., sore throat or cough, dizziness) (Brumbach, Figueredo, & Ellis, 2009; Figueredo, Vasquez, Brumbach, & Schneider, 2004; Sefcek & Figueredo, 2010). Furthermore, exposures to higher levels of psychosocial stress during childhood reliably predict the development of faster life history strategies in adolescence and beyond (reviewed in Ellis et al., 2009).

When viewed in the context of the ACM, these links between psychosocial stress, life history strategy, and health potentially explain why both heightened and dampened stress reactivity can either act as risk factors for or protective factors against psychiatric and biomedical disorder. Both sensitive phenotypes (↑ responsivity) and buffered phenotypes (↓ responsivity) are associated lower levels of psychosocial stress and concomitant development of slower life history strategies (and thus better mental and physical health outcomes), whereas both vigilant phenotypes (↑ responsivity) and unemotional phenotypes (↓ responsivity) are associated with higher levels of psychosocial stress and concomitant development of faster life history strategies (and thus worse mental and physical health outcomes, especially in the long run).

CONCLUSION

In this chapter we presented and elaborated an evolutionary-developmental theory of individual differences in stress responsivity—the ACM—that reorganizes many empirical findings from different research fields, weaves them together in a theoretically coherent manner, and advances novel and testable predictions about behavior, development, and neurobiology. Built explicitly on the foundation of modern evolutionary biology, the ACM provides a framework for research on stress and development that takes us beyond the ALM. We are not arguing that the ALM is *wrong* per se, nor that the extensive body of research documenting the negative effects of allostatic load on health is incorrect, but rather that the overemphasis of the ALM on the costs of allostasis weakens its conceptual power. The ALM does not address the adaptive role of allostasis in regulating developmental plasticity, which is the main objective—and strength—of the ACM. Because of their divergent focus and underlying assumptions, especially regarding adaptive calibration versus stress dysregulation, the ACM and ALM are only partially complementary. Nonetheless, conceptual differences between the two models should not be irreconcilable, and greater integration of the ACM and ALM in the future could potentially strengthen both approaches. Most relevant to the current volume, the ACM and ALM have rather different implications for understanding the development of psychopathology and, consequently, may support different intervention strategies.

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