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CHAPTER 8

The Adaptive Calibration Model of Stress Responsivity

Concepts, Findings, and Implications for Developmental Psychopathology

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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 challenges and opportunities in the environment. One of the most remarkable features of the SRS is the wide range of individual variation in its physiological parameters. Some individuals respond quickly and strongly even to minor events, whereas others show flat response profiles across most situations. Furthermore, the balance of activation among primary SRS subsystems—the parasympathetic nervous system (PNS), the sympathetic nervous system (SNS), 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;

Ellis & Del Giudice, 2014). 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 the wear and tear of chronic stress is presumed to impair SRS functioning (see extended discussion below). Although the allostatic load model (ALM) has proven useful in predicting health-related endpoints, it is not consilient with current theory and research from evolutionary biology. The key limitation of the ALM and related theories (e.g., toxic stress, Shonkoff & Bales, 2011) that employ a pathology lens is that they do not provide a theory of adaptive individual differences in physiological mediators and related patterns of social and physical development.

As an alternative approach, we present the adaptive calibration model of stress responsivity (ACM; Del Giudice et al., 2011; Ellis & Del Giudice, 2014). We begin by reviewing concepts of developmental programming and adaptive calibration more generally. We then summarize key ACM concepts, including the theory of biological sensitivity to context, upon which the ACM builds, and discuss implications for developmental psychopathology. At this juncture, 5 years after it original publication, we review the current empirical status of the ACM and highlight potential updates and revisions that may be needed going forward. We conclude by comparing the ACM and ALM explicitly, arguing that the field needs to expand beyond allostatic load to incorporate an adaptive calibration framework that addresses the functional role of stress response systems in regulating alternative developmental pathways. Central to the ACM is the assumption that gaining a better understanding of the functional developmental changes that occur under stressful conditions will enable us to gain a better understanding of the costs of these changes (e.g., allostatic load and its consequences) and thus develop more effective interventions for the crucial goals of risk prevention and management.

CONDITIONAL ADAPTATION AND MALADAPTATION

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—died 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 biologically adaptive (i.e., tend to increase an

individual's fitness) 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, Bakermans-Kranenburg, & van IJzendoorn, 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, which shows that low-quality maternal care in rats (i.e., low levels of maternal licking and grooming) alters pups' stress physiology and brain morphology. Although such changes seem disadvantageous (e.g., higher corticosterone levels, shorter dendritic branch lengths, lower spine density in hippocampal neurons), they actually enhance learning and memory processes under stressful conditions (e.g., Champagne et al., 2008; Oomen et al., 2010). 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, play behavior, pubertal development, sexual behavior, and parenting (Cameron et al., 2005; Cameron et al., 2008; Franks, Champagne, & Curley, 2015).

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 functional ways of developing when the young organism is neglected. In such contexts, neglect itself may be regarded as a behavioral mechanism through which rats guide their offspring's development toward optimal survival and reproductive strategies under conditions of adversity. It would seem mistaken, therefore, to simply 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 (at least in part) function to prepare offspring to survive and reproduce under harsh ecological conditions.

Conditional Adaptation

The evolutionary perspective thus 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 in ways that are likely to enhance survival and reproductive success.

For conditional adaptations to evolve, the fitness of the alternative phenotypes must be predictable on the basis of reliable cues that can be observed by the

individual (Pigliucci, 2001). Reliable cues to adversity are especially relevant because they may signal the need to develop a secondary (alternative) phenotype that is adversity-adapted, with resulting benefits *and* costs that are often reflected in health trade-offs. 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).

Developmentally, conditional adaptation is often implemented through physiological and neurobiological "switches," or mechanisms that integrate environmental and genetic information to steer developmental trajectories along alternative trajectories. Developmental *switch points* are junctures during which those mechanisms become activated; they are typically located at the transition between different life stages and are regulated by hormonal signals (see Del Giudice, 2014; Ellis, 2013; West-Eberhard, 2003). For example, puberty is a critical switch point in the development of sexual, reproductive, and social behavior, including individual and sex differences in risk taking (Ellis et al., 2012).

The Meaning of Adaptive

The foregoing discussion highlights that the term *adaptive* has different meaning when viewed from an evolutionary perspective (with its functional lens) versus a public health or standard psychological perspective (with its pathology lens; 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). Even high-risk behaviors that result in net harm in terms of a person's own well-being or long-term survival (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). Yet from a public 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 outcomes that are typically viewed as "adaptive" from a public health perspective. That *adaptive* is not equivalent to *desirable* is an important distinction: It clarifies that hypotheses rooted in evolutionary biology do not, by default, imply that adaptations are "good" or should never be targeted for intervention. The use of evolutionary models, however, allows for adaptations to be precisely targeted based on environmental inputs and their developmental consequences.

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, non-kin caregivers providing minimal human contact (Hrdy, 1999). Exposures to such challenging and (evolutionarily) unprecedented conditions are likely to induce pathological development rather than evolutionarily adaptive strategies.

Second, individuals may become maladapted to their environments because of a lack of behavioral plasticity. For example, one of the responsivity profiles highlighted by the ACM is the *unemotional* pattern (described in detail below); this pattern 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 SRS hypoarousal. 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 (Del Giudice, Hinnant, Ellis, & El-Sheikh, 2012). Maladaptation may occur in this context because unemotional phenotypes are relatively unsusceptible to environmental influence and thus may not adjust their behavioral strategies to match the high levels of support and resources that might be 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 developmental models based on life history theory (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, violence, and, in contemporary societies, behaviors such as substance use and risky driving. These high-risk strategies may only be locally adaptive, however. Research by Gibbons et al. (2012) on African American males is instructive in this context. Youth who are exposed to greater stress while growing up (e.g., more dangerous neighborhoods, lower quality parental investment, greater racial discrimination) develop "fast" life history strategies that may be adaptive in their local context (e.g., participation in risky behaviors that leverage positions in dominance hierarchies, increased access to mates) but clearly undesirable—and possibly biologically maladaptive—in wider American society (e.g., dropping out of school, high rates of arrest and incarceration). A similar logic may apply to effects of early stress on cognitive processes (Frankenhuis & de Weerth, 2013).

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 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 are often absent when the postnatal environment continues to be lacking in resources

(Stanner & Yudkin, 2001), suggesting that mismatch (rather than undernutrition per se) 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 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 used, 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 can be critical to understanding the developmental origins of psychopathology. More importantly, these forms of maladaptation are comparatively rare; the organism most commonly responds to environmental conditions by adapting to its local circumstances, regardless of whether this adaptive process is desirable for the individual or society.

FUNCTIONS OF THE STRESS RESPONSE SYSTEM

Environmental events that signal 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 that regulate metabolic, immunologic, and other physiological functions. The SRS comprises primarily three anatomically distinct ~~neuroendocrine~~ systems: the PNS and SNS branches of the autonomic nervous system, and the LHPA axis. Activity of these systems is integrated and cross-regulated, so they can be considered as partially independent yet interrelated components of a coordinated functional system, despite being anatomically distinct and physiologically diverse (e.g., Boyce & Ellis, 2005; Porges, 1995; Schlotz et al., 2008). All the components of the SRS are regulated by top-down cognitive and affective processes; conversely, SRS activation modulates brain activity at multiple levels (through direct neural connections and indirect hormonal effects) in a neuroendocrine feedback loop.

Additional components of the SRS may be discovered as neurobiological methods become more sophisticated, as long as—following the ACM—the component (a) coordinates the organism's allostatic response to physical and psychosocial

challenges; (b) encodes and filters information from the environment, thus mediating the organism's openness to environmental inputs; and (c) functions to shift physical or behavioral endpoints. Epigenetic, cellular, immune, or neuropeptide-related processes may, for example, emerge as SRS components. This does not imply that all new neurobiological measures index components of the SRS. Without these three criteria, new measures may instead be better conceptualized as moderators of the SRS or substrates of other systems. Here we focus on the most common SRS components.

In the absence of stress, the PNS promotes vegetative functions (i.e., rest and restorative behavior), inhibits cardiac activity and cardiac output, and enables sustained attention as a consequence of regulatory mechanisms that occur in the prefrontal cortex (see Beauchaine & Thayer, 2015; Del Giudice et al., 2011; Porges, 2007). When a stressor is encountered, the PNS responds quickly by withdrawing this inhibitory influence (i.e., *vagal withdrawal*), allowing the excitatory SNS to operate unopposed, which results in rapid increases cardiac output to cope with the stressor (Lovallo & Sollers, 2007). PNS withdrawal promotes rapid, flexible responding to stress and coping with mild to moderate stressors (such as solving a difficult puzzle). More extreme defense reactions associated with freezing and fainting also involve changes in PNS activity, albeit via different brainstem nuclei and efferent fibers (Porges, 2007).

In most stressful situations, ranging from mild to severe, increases in cardiac output are effected via coupled PNS withdrawal and SNS activation. However, SNS effects are delayed by a few seconds because they are mediated through a second messenger system. PNS withdrawal and SNS activation also facilitate fight/flight responses via noradrenergic innervation of visceral organs and a slower, hormonal pathway through innervation of the adrenal medulla (e.g., Goldstein & Kopin, 2008; Gunnar & Vazquez, 2006). Following SNS activation, the adrenal medulla secretes epinephrine (E) and 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 more delayed, long-term responses to environmental challenge (although traditional distinctions between rapid and delayed responding have become increasingly blurred; Joëls & Baram, 2009). The endpoint 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 (Sapolsky, Romero, & Munck, 2000). The main effects of cortisol release are to (a) 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 (b) counterregulate physiological effects of SNS activation, thereby facilitating stress recovery (Munck, Guyre, & Holbrook, 1984). Joint effects of the SNS and LHPA axis are complex (Hastings et al., 2011) and they 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 provides a brief 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 signifies a 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—operationalized biologically by heightened reactivity in one or more components of the stress response system (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., 2013, 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-shaped relation between levels of early support-adversity and the magnitude of biological response dispositions. They hypothesized that (a) exposure to acutely stressful childhood environments upregulates BSC, increasing the capacity and tendency of individuals to detect and respond to environmental dangers and threats; (b) exposure to especially supportive childhood environments also upregulates BSC, increasing susceptibility to social resources and support; and (c) by contrast, and typical of the majority of children, exposure to childhood environments that are not extreme in either direction downregulates BSC, buffering individuals against chronic stressors 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 are found in conditions of moderate stress, and that both tails of the support-adversity distribution are associated with higher proportions of reactive children (Ellis et al., 2005; see also Bush, Obradovic, Adler, & Boyce, 2011; Gunnar, Frenn, Wewerka, & Van Ryzin, 2009).

Although BSC theory has helped move the field toward a new conceptualization of stress responsivity, it has a number of significant limitations. First, BSC theory does not systematically link 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-shaped 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 (a) a coherent, systematic account of the biological functions of the SRS; (b) a theory of individual differences capable of explaining adaptation of stress physiology and behavior to local environmental conditions; and (c) 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, integrating across baseline activity and responsivity measures of the SRS (Del Giudice et al., 2011). Achieving these goals would enable scientists to move beyond the 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 developmental "decisions" of organisms and their allocation of resources over the life course (Ellis et al., 2009; Del Giudice, Gangestad, & Kaplan, 2015), as well as the theory of adaptive developmental plasticity (West-Eberhard, 2003). In the ACM, individual differences in SRS functioning are thought to result, at least in part, 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 generally *adaptive* in the biological sense, as they function in a way that ultimately tends to maximize the individual's survival and reproduction in specific environmental contexts.

The ACM can be summarized in seven points (see Del Giudice et al., 2011, for complete explication of the model, and Ellis & Del Giudice [2014] for extended discussion of its theoretical background). The first three points make broad statements about the functions of the SRS which constitute the backbone 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 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 autonomic, neuroendocrine, metabolic, and immune system responses during the first years of life (including the prenatal phase) provides crucial information about life history–relevant dimensions of the child's environment, especially danger and unpredictability (see Ellis et al., 2009). This information is used to adaptively regulate stress responsivity and associated development of life history strategies.

The following four points rely on additional assumptions about the behavioral correlates of SRS functioning to make specific predictions about the development of individual differences:

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 gives rise to 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 and include neurobiological indicators, behavioral outcomes, and developmental trajectories.
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 (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. Prenatal and early postnatal development, the transition from early to middle childhood, and puberty are likely "switch points" for calibration of stress responsivity. Individual and sex differences in SRS functioning emerge according to the evolutionary functions of each developmental stage.

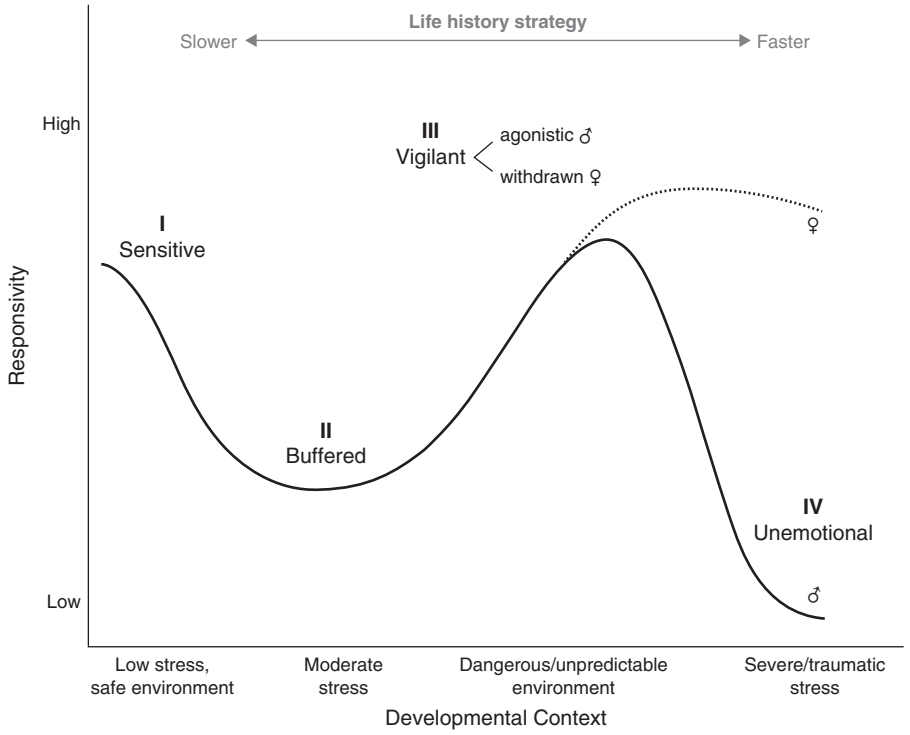

Figure 8.1 Predicted curvilinear relation between developmental context and optimal levels of stress responsivity.

Developmental context refers to variation in rearing experiences (i.e., individual differences in developmental exposures to key dimensions of environmental stress and support). 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 ~~(see Table 8.1 for specific predictions regarding different SRS parameters).~~ Male/female symbols indicate sex-typical patterns of responsivity, but the model also predicts substantial within-sex variation.

Adapted from Del Giudice, Ellis, and Shirtcliff, 2011, p. 1577.

ENVIRONMENTAL INFORMATION

A crucial function of the SRS is to collect and integrate information about changing states in the environment—including presence of threats, dangers, and opportunities—to adjust the state of the whole organism accordingly. This information can be encoded by the SRS in its functional parameters 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 ecological information that can be encoded through repeated SRS activation.

Key Dimensions of the Environment

The conceptualization of key dimensions of environmental influence in the ACM is based on LHT—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 allocation of resources to competing life functions: bodily maintenance, growth, and reproduction. Because of 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).

At the broadest level, these trade-offs result in covarying sets of traits (i.e., life history strategies) that generally fall along a dimension of "slow" versus "fast." Fast life history strategies are comparatively high risk and present oriented (taking benefits opportunistically with little regard for long-term consequences) and prioritize mating effort (e.g., competitive risk taking, aggression); they are also characterized by earlier ages of sexual development and reproduction, and focus on producing of a greater number of offspring with less investment of resources, time, and energy in each. In contrast, slow life history strategies are comparatively long-term oriented and low risk (e.g., longer time horizons, more delay of gratification, better self-regulation and behavioral control), characterized by later timing of sexual development and reproduction, and focus on producing a smaller number of offspring and investing heavily in each of them. As discussed below, trade-offs incurred by the fast strategy include reduced health, vitality, and longevity—of self and offspring.

Most important for the present discussion, LHT can be used to predict how organisms adjust their life history strategies according to ecological conditions. Key dimensions of the environment relevant to life history development are availability of resources, extrinsic morbidity-mortality (i.e., external sources of disability and death that are relatively insensitive to the adaptive decisions of the organism), 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 bioenergetic resources

are adequate to support growth and development, however, cues to extrinsic morbidity-mortality and unpredictability gain importance (Ellis et al., 2009). In this context, LHT predicts that individuals will respond to extrinsic morbidity-mortality cues (e.g., exposures to violence, premature disability, death of people around you) and unpredictability (e.g., stochastic changes in ecological context, resource availability, family composition) by entraining faster life history strategies (see Belsky, Schlomer, & Ellis, 2012; Simpson, Griskevicius, Kuo, Sung, & Collins, 2012, for supporting longitudinal data). Developmental models based on LHT emphasize that these ecological factors tend to operate indirectly on children through more proximal processes, including those mediated by family characteristics (e.g., harsh parental discipline vs. warm and supportive parenting behaviors, family chaos vs. routines). For example, parental investment can buffer the impact of extrinsic mortality cues and heighten controllability or predictability by providing a stable, caregiving environment. Indeed, much of the effect of "toxic stress" on children's development works through the mechanism of both exposure and failure of supportive parental relationships to buffer the child from stress exposure (Shonkoff & Bales, 2011).

The SRS is attuned exquisitely to 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 regulating 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 an 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). In contrast, a highly responsive SRS amplifies signals coming from the environment and maximizes the chances that the organism will be modified by current experience. Potential costs of a highly responsive SRS 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 challenges, 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, Salavey, & Epel, 2002; Stroud et al., 2009). Moreover, sex is not the only individual difference factor capable of influencing which domain shows a low threshold for activation (e.g., Wobber et al., 2010). Close social relationships can also filter/amplify more distal environmental factors, such as when cortisol reactivity is buffered by the presence of a warm, supportive caregiver (Hostinar & Gunnar, 2013a; Hostinar, Johnson, & Gunnar, 2015).

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 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, which reflects upstream regulation from prefrontal areas, promotes calm, concentration, and self-regulation (e.g., Beauchaine & Thayer, 2015; Fabes and 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 reactivity in contexts characterized by unpredictability (Dickerson & Kemeny, 2004). Empirical findings that cortisol levels elevate prior to laboratory arrival (e.g., Ellis, Essex, & Boyce, 2005; Hastings et al., 2011) or in anticipation of challenges of the day (e.g., Fries, Dettenborn, & Kirschbaum, 2009; Schmidt-Reinwald et al., 1999) 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 biologically embedded as 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 process 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 SRS functioning as a mechanism of susceptibility to environment influence (Boyce & Ellis, 2005) helps to explain 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 above, 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 benefit most from positive, supportive environments and interventions. Thus, the very qualities that appear to increase children's frailties may also constitute 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 proved 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 local ecologies, and in some cases an unresponsive system can be highly functional in the context of an individual's life history strategy. This idea will be 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-shaped 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 hyporesponsivity of the SRS (e.g., Gustafsson, Anckarsäter, Lichtenstein, Nelson, & Gustafsson, 2010; De Bellis et al., 1999; Tarullo & Gunnar, 2006; Yehuda, 2002). The ACM suggests that dampened responsivity may actually follow an adaptive logic, as a way to maximize fitness benefit/cost ratios in severely dangerous and unpredictable environments.

When danger becomes severe, 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 can be 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 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 use 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, i.e., 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. Thus, 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 as 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/or 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, we predicted that males should be more likely to develop unresponsive phenotypes in highly stressful contexts. Another possibility is that the behavioral *correlates* of high and low responsivity in dangerous environments may differ between the sexes. Finally, we do not expect sex differences in responsivity to be present from birth, but rather to emerge gradually during development, as social and mating competition become more biologically salient (see Del Giudice, 2014; Ellis, 2013).

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.

Sensitive Pattern (Type I)
Sensitive patterns are hypothesized to develop in safe, predictable conditions and warm family environments. High stress responsivity among 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 (for details see Del Giudice et al., 2011).

Buffered Pattern (Type II)

Buffered patterns (marked by moderate to low SRS responsivity across the board) 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)

Highly responsive 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 co-occur 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, Crick, Shirtcliff, & Woods, 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 across systems, 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. As explained above, we predicted the distribution of Type IV to be male-biased; moreover, we anticipated that behavioral correlates of this pattern would differ between sexes. For example, one key feature of unemotional responsivity among females

may be a generalized pattern of aloof social relationships with parents, siblings and peers. Low serotonergic activity is a likely neurobiological correlate of Type IV.

Empirical Investigations of the ACM Responsivity Patterns

Many empirical studies have attempted to link child and adolescent physiological profiles with the kinds of contextual factors and behavioral outcomes theorized in ACM (reviewed in Del Giudice et al., 2011; Obradović, 2012; see also below, Implications for Developmental Psychopathology). Although not explicitly designed to test the ACM, some of these studies have used the model as a framework for interpreting their results (e.g., Essex et al., 2011; Peckins, Susman, Negriff, Noll, & Trickett, 2015). Most relevant, a small number of studies have attempted to test for the ACM responsivity profiles across multiple SRS subsystems.

Del Giudice, Hinnant, Ellis, and El-Sheikh (2012) was the first explicit attempt to empirically validate the four-pattern classification of the ACM. In this study, which examined stress responsivity patterns in an at-risk sample of 8-to 10-year-old children, we identified four classes of autonomic nervous system activity during resting conditions and in response to a stressful task. SNS activity was indexed by skin conductance level and PNS activity was indexed by respiratory sinus arrhythmia. Physiological differences between the classes were dominated by SNS activity and (to a lesser extent) PNS basal activity. Furthermore, the four patterns were associated with different levels of family stress. Two components of environmental stress emerged as significant predictors of class membership: (1) negative family relationships and (2) family warmth/predictability. As predicted, high-responsivity and low-responsivity patterns were found under both low-stress and high-stress conditions.

Although the study by Del Giudice et al. (2012) provided a first step toward testing the ACM responsivity patterns, it had several limitations. First, measures of physiological activity were limited to the autonomic nervous system; however, LHPA axis functioning is central to the ACM and needs to be taken into account when determining responsivity patterns. Second, Del Giudice et al. (2012) used the star-tracing task (a cognitive challenge) to elicit stress responsivity. Although the star-tracing task is a valid procedure, social-evaluative threats—and particularly exposure to challenging conditions that reliably elicit LHPA-axis activation—are necessary to obtain all of the responsivity data needed to classify individuals into the four responsivity patterns of the ACM. Finally, and most critically, Del Giudice et al. (2012) did not examine links between the identified responsivity patterns and indicators of life history strategy. Testing for these links is necessary to evaluate the theory, and especially to distinguish sensitive from vigilant phenotypes and buffered from unemotional phenotypes, which are hypothesized to display overlapping patterns of stress physiology but different life history strategies.

Two studies since Del Giudice et al. (2012) have attempted to address some of these limitations. Both Quas et al. (2014; Peers and Wellness Study [PAWS]) and Ellis, Oldehinkel, and Nederhof (in press; Tracking Adolescent Lives Study [TRAILS]) conducted a latent profile analysis (LPA) that incorporated all three SRS subsystems, assessed relations with environmental conditions, and elicited stress responses based on socio-emotional or social-evaluative threat. TRAILS also included indicators of life history strategy. These studies further differed from Del Giudice et al. (2012), and from each other, regarding age of the participants at the time of the stress physiology assessments (PAWS: 5 years of age; TRAILS: 16 years of age) as well as levels of environmental risk that characterized the samples (PAWS: moderate risk; TRAILS: low risk), and sex (TRAILS included only boys). Given that the ACM is a developmental theory that posits changes in responsivity over child and adolescent development, differences in responsivity patterns under different levels of environmental stress and support, and sex differences, these three studies are inherently difficult to compare. Nonetheless, a discussion of similarities and differences between the results of these studies should provide a useful first step toward evaluating the ACM.

The ACM predicts that a buffered pattern (without either hyperresponsivity or hyporesponsivity) emerges among most children who develop in normative environments that are not characterized by extremes of either nurturance and support or adversity and trauma. In all three empirical studies, the largest number of participants fit the buffered response pattern, displaying roughly average levels of psychosocial stress, stress responsivity, and (in TRAILS) behavioral indicators of life history strategy. TRAILS constituted a relatively low-risk sample, and the LPA placed about three quarters of its participants into the buffered profile (74%). In contrast, PAWS had more diverse socio-demographic and ethnic characteristics than TRAILS, and the LPA resulted in a buffered group with 52% of participants. Finally, the at-risk U.S. sample studied by Del Giudice et al. (2012) had a buffered group with only 45% of participants. Thus, there is emerging empirical support for the high prevalence of the buffered pattern among low-risk samples, as well as variation across samples in the relative proportion of this pattern depending on background stressor exposures. Consistent with the ACM, the LPAs of both the TRAILS and PAWS data sets resulted in two patterns of high stress responsivity: One profile characterized by heightened multisystem reactivity across PNS, SNS, and LHPA axis parameters and the other characterized by PNS-specific reactivity (i.e., strong vagal withdrawal). In the TRAILS analyses, the pattern of multisystem reactivity was labeled sensitive because it was characterized by significantly elevated scores on quality of family environment (i.e., more warmth/support and less stress/rejection in the family environment) and the lowest levels of aggressive/rule-breaking behavior (indicating a slow life history strategy), whereas the PNS-dominated responsivity pattern was labeled vigilant because it was characterized by the highest levels of prenatal/perinatal risk factors and childhood stress,

the highest levels of depressed/withdrawn behavior, and moderately elevated levels of aggressive/rule-breaking behavior. In contrast, in the PAWS analyses, multisystem reactivity was labeled vigilant because it was associated with high family adversity.

These opposing findings regarding the environmental contexts of multisystem reactivity will need to be addressed in future research. However, the two studies were markedly different, especially in the age of the participants (early childhood vs. adolescence) when stress responsivity was assessed. The substantial reorganization of neurobiological stress responses at puberty (Dahl & Gunnar, 2009; Forbes & Dahl, 2010), as emphasized in the ACM, may be especially relevant to explaining these divergent results. More generally, these initial findings call into question some of the original predictions of the ACM concerning the prevalence of SNS reactivity in vigilant pattern versus PNS reactivity in sensitive patterns (Del Giudice et al., 2011). Those predictions were based on the assumption that high PNS reactivity is a consistent marker of self-control and positive social engagement. However, this assumption may need some revision in light of later research showing that strong PNS withdrawal in response to emotional stimuli correlates with indices of behavioral problems (see Beauchaine, 2015; Beauchaine & Thayer, 2015). Moreover, tonic activity and reactivity of the PNS increase markedly with age from infancy to adulthood; PNS parameters in child and adolescent samples may be confounded with the rate of physical and sexual maturation (Beauchaine & Webb, in press; Graziano & Derefinko, 2013), which is especially problematic from the standpoint of evaluating life history models.

Consistent with the ACM, the LPA of TRAILS data resulted in two responsivity patterns that were characterized by relatively high levels of environmental stress and faster life history strategies, with opposing patterns of stress responsivity. One of these profiles was characterized by high stress responsivity (the vigilant pattern, as described above) and the other by low stress responsivity (labeled unemotional); both were PNS dominated. The unemotional profile was associated with strong vagal augmentation, as well as low LHPA axis reactivity. This profile was clearly linked to a fast life history strategy (highest scores on aggressive/rule-breaking behavior; lowest scores on effortful control) and to low scores on withdrawn/depressed behavior. In addition, membership in this profile was predicted by low scores on quality of family environment and associated with elevated scores on various childhood adversity measures. Again, the centrality of the PNS in distinguishing these two higher-risk profiles highlights the need, as the ACM is further developed and revised, to more fully delineate the role of PNS activity, especially in relation to the vigilant profile.

The contrast between vigilant and unemotional profiles converges with past developmental research showing that a pattern of strong vagal withdrawal in response to social or cognitive challenges (as in the TRAILS vigilant responsivity pattern) is associated with internalizing symptoms or co-occurring internalizing-externalizing behavior problems whereas weak vagal withdrawal or

vagal augmentation (as in the TRAILS unemotional responsivity pattern) is associated with externalizing behavior problems (Boyce et al., 2001; Calkins & Keane, 2004; Calkins, Graziano, & Keane, 2007; El-Sheikh, Hinnant, & Erath, 2011; Gazelle & Druhen, 2009; Hinnant & El-Sheikh, 2009). These relations are complex, however, and not always consistent, particularly when comparing clinical and normative samples (see Zisner & Beauchaine, in press). For example, Hinnant and El-Sheikh (2013) found that vagal augmentation in boys was associated with co-occurring high internalizing and high externalizing trajectories across middle to late childhood, and Pang and Beauchaine (2013) documented excessive vagal withdrawal specifically in response to an emotionally evocative video in 8- to 12-year-old children who were extremely high in conduct problems. In the ACM, both vigilant and unemotional patterns are associated with higher rates of externalizing behavior, but they reflect different patterns of SRS activity.

One of the developmental hypotheses of the ACM is that boys who grow up under highly stressful conditions will initially display a vigilant profile of heightened stress responsivity—but following chronic severe stress, shift toward a male-biased pattern of low responsivity (the unemotional pattern) under the influence of adrenal androgens in middle childhood. This pattern is then expected to further intensify in adolescence in relation to the pubertal transition. Consistent with this prediction, a clear unemotional profile did not emerge in the PAWS analysis of 5-year-olds but did emerge in Del Giudice et al.'s (2012) analyses (8- to 10-year-olds) and in the TRAILS analysis (16-year-olds). This age trend converges with past research showing that, over the course of development from childhood to young adulthood, females with histories of child sexual abuse shift from initially upregulated to downregulated morning cortisol levels (Trickett, Noll, Susman, Shenk, & Putnam, 2010), and to blunted feedback of the HPA axis (see e.g., Beauchaine, Crowell, & Hsiao, 2015).

On the other hand, Del Giudice et al. (2012) failed to find the predicted male-biased distribution in unemotional patterns. Most of the sample was still prepubertal, however, so caution is warranted interpreting any sex differences or lack thereof. Nonetheless, many studies have shown that both men and women become hyporesponsive under conditions of severe stress (e.g., Bruce et al., 2009; Gustafsson et al., 2010; Miller, Chen, & Zhou, 2007; Tarullo & Gunnar, 2006; Vigil et al., 2010). A possibility to be explored in future research is that, even if unemotional patterns are equally frequent in males and females, similar physiological profiles may have different manifestations in behavior in the two sexes, as discussed above (see Del Giudice et al., 2011 for a more in-depth treatment of sex differences across behavioral domains).

In conclusion, theoretical models such as the ACM are useful insofar as they explain known facts and make novel, testable predictions. The ACM is a complex model, and it can be used to derive dozens of predictions at different levels of analysis, including hypotheses about relations between childhood stress and stress responsivity, stress responsivity and behavior, individual differences in neuromodulation, Gene × Environment interactions, sex differences in life history strategies,

and responses to domain-specific stressors (such as agonistic confrontations). Clearly, no single study can address all of these predictions, and multiple studies will be required even to evaluate the more basic ones. This first set of three studies (Del Giudice et al., 2012; Ellis et al., in press; Quas et al., 2014) provide initial empirical tests of the four responsivity patterns of the ACM. To varying degrees, these investigations provide support for the ACM by documenting complex, nonlinear relations between (a) childhood indices of familial and ecological conditions and life stress, (b) multisystem/multiphasic patterns of stress responsivity in adolescence, and (c) behavioral indicators of life history strategy in adolescence. Since its original publication, many other studies have also used the ACM as a guiding framework to explain biobehavioral links, yielding results that are consistent with the theory even within studies not originally designed to tease apart complexities of the ACM. Thus, the ACM has emerged as a useful theory in the field, helping us to move toward a more coherent "big picture" of the biosocial processes involved in developmental adaptation to the environment. At the same time, each of the three empirical studies that have specifically tested for the ACM responsivity patterns displays substantial limitations; their results both show support for and deviations from the ACM predictions, highlighting important theoretical challenges and empirical issues for future research.

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. For example, the hypothesis that some children shift from vigilant to unemotional patterns across middle childhood and adolescence 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, Granger, Booth, & Johnson, 2005). 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 co-vary but also share many etiological factors (see Lévesque, Bigras, & Pauzé, 2010).

Finally, the ACM helps clarify complex relations between psychosocial environmental factors and stress responsivity patterns. Although the theory is rooted in biology and evolution, in practice the ACM emphasizes the importance of the

environment for shaping children's biosocial development. Specific predictions can be made about the effects of key dimensions of the environment—resource availability, extrinsic morbidity-mortality, and unpredictability—rather than catchall concepts such as "cumulative stress" or "lifecourse adversity." Moreover, moderators such as supportive caregiving can be specified within each key dimension (i.e., Does the moderator change environmental unpredictability? Does it shield the child from external morbidity or mortality threats? Does it provide necessary bioenergetic resources?), rather than tautologically defining a buffer as something that acts as a buffer. Making sense of these key dimensions of environmental risk and support has tremendous implications for treatment and prevention efforts, as the theory helps sort through the wide range of possible stressors to focus on the most likely targets for successful intervention.

The ACM can be criticized for suggesting that stress and adversity over development can either upregulate or downregulate levels of SNS, PNS, and LHPA responsivity and thus that "any outcome" can be consistent with the ACM. Nonetheless, this situation is reflective of the state of the empirical literature on this topic: 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 (as reviewed above). 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 upregulate (in the sensitive pattern) or downregulate (in the buffered pattern) responsivity. Likewise, developmental exposures to high levels of stress either upregulate (in the vigilant pattern) or downregulate (in the unemotional pattern) responsivity. Thus, if one considers the environment-responsivity curves shown in Figure 8.1, it is apparent that results of any single study that examines 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). Many inconsistent results in the stress literature may depend, at least in part, on failures to consider nonlinear relationships between environmental factors and SRS parameters, the tendency to view SRS functioning as divergent from some optimal base-point rather than a wide range of starting points, or difficulties with assessing the full range of environmental variance necessary to capture all four patterns of responsivity and associated behavioral strategies specified by the ACM.

Of equal importance, the ACM predicts that it will be difficult to discriminate between functionally different profiles of responsivity without including information about life history–relevant traits such as risk taking, self-regulation, sexual maturation, and so on. What distinguishes the sensitive and vigilant patterns in the model is not LHPA reactivity per se, but rather the constellation of traits that go with it and clarify its functional meaning (increased social sensitivity in one case versus readiness to face social or physical danger in the other). If the model is correct, attempts to discriminate between meaningful individual types based

exclusively on SRS parameters will yield weak and inconsistent findings, as SRS profiles are only one component of the predicted responsivity patterns.

ADAPTIVE CALIBRATION AND THE ALLOSTATIC LOAD MODEL

With the ACM, we are seeking an integrative theoretical framework for the study of stress, stress responsivity, and health across development. The ACM both complements and provides a counterpoint to the ALM, which has become quite popular in developmental psychopathology (e.g., Beauchaine, Neuhaus, Zalewski, Crowell, & Potapova, 2011; Lupien et al., 2006). In recent years, researchers have started adopting the ALM as a foundation for interdisciplinary integration (e.g., Ganzel, Morris, & Wethington, 2010; Juster et al., 2011). As Hostinar and Gunnar (2013b, p. 400) conclude: "The field has two major theories for talking about stress and health: the Allostatic Load Model, which grew out of biological and neuroscience approaches to understanding health and disease, and the Adaptive Calibration Model, which developed out of an explicitly evolutionary-developmental framework." In this section, we review key points of convergence and divergence between the models, in order to clarify some of the advantages of the ACM (for a more detailed exposition, see Ellis & Del Giudice, 2014).

The Allostatic Load Model

The process by which the regulatory parameters of the SRS (as well as other neurobiological systems) are modified in the face of challenge is termed *allostasis* (i.e., "stability through change"; Sterling & Eyer, 1988). Allostasis is a key concept of the ALM; it refers to the moment-to-moment process of increasing or decreasing vital functions (i.e., adaptively adjusting physiological parameters within the organism's operating range) to new steady states in response to the demands of the environment and the organism's resources (McEwen & Stellar, 1993; see also Lupien et al., 2006). Allostasis functions to help the organism cope with challenging events or "stressors," enabling short-term adaptation to environmental perturbations. However, the term *allostasis* is not always used consistently; for example, 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* and is now more commonly referred to as *biological embedding*). 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 long-term costs of allostasis; it is often described as "wear and tear" that results from repeated allostatic adjustments (i.e., adaptation to stressors), exposing the organism to adverse health consequences. The ALM emphasizes that biological responses to threat, while essential for survival, have negative

long-term effects that promote illness. Thus, short-term *benefits* of mounting biological responses to threat are traded off against long-term *costs* to mental and physical health, and these costs (allostatic load) increase as the organism ages. Among other adverse outcomes, allostatic load is thought to cause SRS dysregulation, resulting for example in excessive or insufficient responses to stressors and increasing vulnerability to mental and physical health problems (e.g., Juster et al., 2010; Juster et al., 2011). The idea of physiological dysregulation is integral to the ALM, which assumes that there is an optimal level of biological responsivity to social and environmental challenges. Accordingly, both "hyperarousal" and "hypoarousal"—recurring overactivity or underactivity of physiological mediators—are routinely described as dysfunctional deviations from the norm (e.g., Adam, 2012; Juster et al., 2011; Lupien et al., 2006), usually caused by a combination of excessive stress exposure and genetic or epigenetic vulnerability. Sometimes, models based on allostatic load assume that these response patterns evolved to meet the demands of more dangerous ancestral environments, but are mismatched to less perilous modern environments, thus setting in motion pathogenic processes that eventuate in mental and physical illness (e.g., Miller et al., 2011).

ACM Versus ALM

We note 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 among 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 their costs, *not* when they are cost-free. As discussed above, 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), as illustrated by the high levels of risk taking and aggression

that characterize the adversity-adapted unemotional responsivity pattern. Similarly, when health and reproductive success conflict, natural selection favors the latter at the expense of the former (see Nesse, 2001).

Because of persistent confusion underlying the distinction 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 the notion of inherent trade-offs in the life cycle of organisms; 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 discounts 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 manner in which the ALM framework is often applied, the same pattern of responsivity is 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 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, long-term adjustment of SRS parameters (as in the development of different responsivity profiles) 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 (such as the well-documented trade-offs between faster life history strategies and health; reviewed in Ellis & Del Giudice, 2014). In total, the ALM, relative to the ACM, overemphasizes the costs of allostasis and underappreciates its benefits. A comparison and contrast between the core assumptions of the two theories is presented in Table 8.1.

In conclusion, we are not arguing that the ALM is *wrong* per se, nor that the extensive body of research documenting 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. Be that as it may, conceptual differences between the ACM and ALM should not be irreconcilable, and greater integration of the two models 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 (Ellis & Del Giudice, 2014).

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 substantive limitations, especially regarding the current manner in which it uses a pathology lens to explain influences on human development. In practice, this focus has moved the field away from the roots of the ALM, which began in evolutionary biology and an exploration of allostasis and allostatic states, toward a context-free view about optimal health outcomes or pathological deviations from normative SRS profiles. The ACM attempts to swing the pendulum back to be more consilient with theory and research in evolutionary biology, providing researchers with a broader theory of stress responsivity that acknowledges the central importance of calibration to local environmental conditions.

We recognize that the ALM is attractive because it conforms to implicit assumptions of the standard mental health approach, particularly regarding stress-disease relationships, and therefore does not require a 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 that is informed by life history theory, modeling of strategic trade-offs, and a more explicit consideration of the relations between adaptation, health, and well-being. In total, we believe that the ACM embodies the main insights of the ALM while addressing some of its key limitations. Even more importantly, most of the work that is presently carried out under the ALM umbrella could be reframed in the perspective of the ACM. For example,

Table 8.1
Comparison of Adaptive Calibration Model (ACM) and Allostatic Load Model (ALM)

Responses to Psychosocial Stress/ Unpredictability	Examples of Response	ACM	ALM
Activation of autonomic, neuroendocrine, metabolic, and immune systems	• Acute SNS and LHPA responses mobilize energy reserves, protect against septic shock and nutrient deprivation, permit fight or flight responses that are normally protective against danger. • Inflammation accelerates the healing of wounds.	Central to theory	Central to theory
Changes in allostatic mechanisms	• Increased inflammatory tone • Elevated cortisol and catecholamines • Muted cardiovascular responses to stress	Central to theory	Central to theory
Cognitive, behavioral, and emotional impairments in children	• Reduced scores on standard tests of intelligence, language, memory, and other abilities • Early onset and increased prevalence of psychopathology	Not inconsistent with theory	Central to theory
Cognitive, behavioral, and emotional adaptations to stress in children	• Tailoring of emotion systems, arousal responses, and perceptual abilities to the detection and monitoring of danger • Development of insecure attachments, mistrustful internal working models, opportunistic interpersonal orientations, oppositional-aggressive behavior	Central to theory	Not inconsistent with theory
Long-term deleterious outcomes	• Cognitive and physical impairments • Depression • Increased risk of cardiovascular disease and all-cause mortality	Not inconsistent with theory	Central to theory
Long-term adaptive changes in biobehavioral systems	• Adaptive calibration of autonomic, neuroendocrine, metabolic, and immunological systems • Regulation of alternative life history strategies to match ecological conditions	Central to theory	Beyond the scope of the theory

Note. Light shading indicates a difference in emphasis between the ACM and ALM. Dark shading indicates a qualitative divergence between the two theories. SNS: sympathetic nervous system; LHPA: limbic-hypothalamic-pituitary-adrenal axis.
Adapted from Ellis and Del Giudice, 2014.

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 & Morris, 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 upregulation versus downregulation of SRS parameters are not theoretically modeled (rather, hyperarousal and hypoarousal are grouped together as dysfunctional deviations from an optimal setpoint). Valid explanatory models of developmental pathways leading to both hyper- and hyporesponsivity are critical to explaining the development of psychopathology because both heightened and dampened responsivity can appear either good or bad in terms of behavioral adjustment and health. Such bivalent effects of the SRS have been documented in PNS, SNS, and LHPA studies focusing on both baseline arousal and responsivity (e.g., Bauer, Quas, & Boyce, 2002; Burke, Davis, Otte, & Mohr, 2005; Evans & English, 2002). 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 appear to be a protective factor in sensitive phenotypes and a risk factor in vigilant phenotypes, whereas *dampened* reactivity may appear to be a protective factor in buffered phenotypes and a risk factor in unemotional phenotypes. This contrast highlights the critical importance of examining larger responsivity patterns in the context of environmental antecedents and life history outcomes.

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; it delineates coherent, functional responses to stress, including regulation of alternative life history strategies, which reliably emerge in given developmental contexts. These responses have to be taken into account to more fully and accurately capture child and adolescent development under conditions of psychosocial stress and unpredictability. Ultimately, our ability to translate research on stress-health relationships into effective interventions for the

crucial goals of risk prevention and management depend on understanding when and how adaptations to stress emerge and can be changed.

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