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Making Sense of Stress: An Evolutionary-Developmental Framework

Marco Del Giudice [1]

Bruce J. Ellis [2]

Elizabeth A. Shirtcliff [3]

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[1] Center for Cognitive Science, Department of Psychology, University of Turin.

Address: Via Po 14, 10123 Torino, Italy.

Email: marco.delgiudice@unito.it (corresponding author)

[2] John and Doris Norton School of Family and Consumer Sciences, University of Arizona.

Address: McClelland Park, 650 North Park Ave, Tucson, AZ 85721-0078, USA.

Email: bjellis@email.arizona.edu

[3] Department of Psychology, University of New Orleans.

Address: 2000 Lakeshore Drive, New Orleans, LA 70148, USA.

Email: birdie.shirtcliff@uno.edu

Abstract

In this chapter we present an evolutionary-developmental framework for individual differences in stress responsivity, the Adaptive Calibration Model (ACM). We argue that the core propositions of the ACM provide a context for the integrative biological analysis of the stress response system, exemplified by Tinbergen's "four questions" of mechanism, ontogeny, phylogeny, and adaptation. We then show how the ACM can be used to generate novel predictions on responsivity profiles in humans and their development across the life span.

Keywords

Individual differences; life history theory; sex differences; stress response system

1. Tinbergen's Four Questions in Stress Research

If anything qualifies as a complex biological mechanism, the stress response system certainly does. The stress response involves the hierarchical, co-ordinated action of the autonomic system and the hypothalamic-pituitary-adrenal axis (HPA), as well as multilevel feedback loops with cortical brain structures. Be it by direct innervation or endocrine signaling, the stress response system (henceforth SRS) regulates an astonishing range of physiological and behavioral processes, including bodily growth, metabolism, reproductive functioning, attention and memory, learning, aggression, risk-taking, caregiving, and so forth.

Whereas the basic design of the SRS has been worked out reasonably well (see **CHAPTER 3 IN THIS VOLUME**), the intricate details of its functioning in ecological contexts still pose formidable puzzles to researchers. First, the ubiquity of SRS involvement adds to the difficulty of making sense of the system's function(s). Second, stress physiology exhibits remarkable individual

variation, which is still not well understood and often interpreted from a pathologizing standpoint, especially in the case of humans (see Ellis, Del Giudice, & Shirtcliff, in press b). Third, the logic by which individual patterns of stress responsivity develop over time, and the role played by genes and environments in the process, remain elusive despite the hundreds of empirical studies carried out every year.

Complex biological mechanisms like the SRS can be fully understood only by approaching them from multiple interlocking perspectives. Nikolaas Tinbergen (1963) famously described the four main types of explanation required for a complete understanding of a biological system. Tinbergen's "four problems" or "four questions" have since become a standard heuristic device in evolutionary biology. With an updated terminology, the four problems of biology can be described as: *mechanism* (what is the structure like? How does it work?); *ontogeny* or *development* (how does the structure come to be over developmental time, and how does it change across the lifespan?); *phylogeny* (what is the evolutionary history of the structure? How did it change across generations and species?); and *adaptation* (why is the structure the way it is? What selective advantages does it confer, or did it confer, to the organism?). Ontogenetic and mechanistic explanations concern the way an organism works in the present, without reference to evolution and adaptation; collectively, they are called *proximate* explanations. In contrast, *ultimate* explanations (phylogenetic and adaptationist) consider the organism in relation to its past and to the evolutionary forces that shaped its body and behavior (Mayr, 1963). It should be obvious that, as already stressed by Tinbergen, the four types of explanation are pragmatically distinct, but not logically independent from one another. Even more important, they are not mutually exclusive but complementary and synergistic: adaptive function crucially informs the study of mechanism and development, while development and mechanism constrain the range of adaptive explanations (McNamara & Houston, 2009; Scott-Phillips et al., 2011). A similar interplay occurs between adaptationist and phylogenetic questions.

Starting a virtuous cycle between different levels of explanation is the best way to build a satisfactory model of a complex biological system.

1.1. The Need for an Integrative Framework

Precisely because Tinbergen's four questions produce the best answers when they are asked synergistically rather than in isolation, it is extremely useful to possess an integrative theoretical framework. An adequate framework for stress research should involve all four types of biological explanation, tying them together in a coherent narrative. Moreover, it should address both the species-specific functioning of the SRS and the origin of individual and sex differences. Ideally, it should be possible to apply it to different species (with the necessary changes and refinements), a reasonable requirement given the ancient and highly conserved structure of the SRS (Nesse, 2007; Porges, 2001, 2007). In recent years, considerable progress has been made toward this goal, and a number of evolutionary models of stress responsivity have appeared in the literature (e.g., Carere et al., 2010). Among the most notable are the hawk-dove model by Korte and colleagues (2005); the social plasticity model by Flinn (2006); the polyvagal theory by Porges (2001, 2007); the tend-and-befriend hypothesis by Taylor and colleagues (2000); and the theory of Biological Sensitivity to Context (BSC) by Boyce and Ellis (2005). While each of these models provides crucial insights in the function, development, and phylogeny of the SRS, none of them has the scope of a truly integrative theory.

1.2. The Adaptive Calibration Model

In the remainder of this chapter, we will introduce the Adaptive Calibration Model (ACM), our recent attempt to provide the field with a comprehensive evolutionary-developmental framework (Del Giudice, Ellis, & Shirtcliff, 2011). The ACM extends and refines the BSC theory (Boyce & Ellis, 2005; Ellis, Essex, & Boyce, 2005), while incorporating several key elements of other evolutionary models (e.g., Flinn, 2006; Korte et al., 2005; Porges, 2007; Taylor et al., 2000).

From the standpoint of Tinbergen's four questions, the main focus of the ACM is on adaptation and development, but the model also makes several novel predictions about the mechanism of the stress response. The main elements of the ACM are: (a) an evolutionary analysis of the functions of the SRS, defined as an integrated, hierarchically organized system comprising the autonomic nervous system and the HPA axis; (b) a theory of the adaptive match between environmental conditions and stress responsivity; and (c) a taxonomy of four prototypical responsivity patterns to be found in humans, their behavioral and neurobiological correlates, and their hypothesized developmental trajectories. Whereas (b) and (c) are tailored to human ecology and physiology, our evolutionary analysis (a) is based on general biological principles and has the potential to be applied (with minor adjustments) to many different species. Thus, the ACM in its present form is best conceived as consisting of a general theoretical "core", with a detailed theory of human development built on top of it.

In keeping with the broad scope of this volume, here we will mainly focus on the theoretical core of our model. After providing a succinct overview of some key concepts in evolutionary biology (section 2), we will present an evolutionary analysis of the functions of the SRS and argue that it operates as a mechanism of conditional adaptation and a central mediator of the development of life history strategies (section 3). Then, we will briefly outline our main predictions about the development of individual and sex differences in humans (section 4). Interested readers can refer to Del Giudice and colleagues (2011) for an extended treatment of the ACM, including many additional details and empirical predictions.

2. Biological foundations

2.1. Life History Theory

Life history theory is a branch of evolutionary biology dealing with the way organisms allocate time and energy to the various activities that comprise their life cycle (see Ellis et al., 2009; Hill, 1993; Roff, 2002). All organisms live in a world of limited resources; the energy that can be

extracted from the environment in a given amount of time, for example, is intrinsically limited. Time itself is also a limited good; the time spent by an organism looking for mates cannot be used to search for food or care for extant offspring. Since all these activities contribute to an organism's evolutionary fitness, devoting time and energy to one will typically involve both benefits and costs, thus engendering trade-offs between different fitness components. For example, there is a trade-off between bodily growth and reproduction because both require substantial energetic investment, and thus producing offspring reduces somatic growth. Life history theory concerns optimal allocation of time and energy toward competing life functions – bodily maintenance, growth, and reproduction – over the life cycle.

Life history *strategies* are adaptive solutions to a number of simultaneous fitness trade-offs. The most basic trade-offs are between *somatic effort* (i.e., growth, body maintenance, and learning) and *reproductive effort*; and, within reproductive effort, between *mating* (i.e., finding and attracting mates, conceiving offspring) and *parenting* (i.e., investing resources in already conceived offspring). From another perspective, the critical decisions involved in a life history strategy can be summarized by the trade-offs between *current* and *future reproduction*, and between *quality* and *quantity of offspring* (see Ellis et al., 2009).

In sexual species, the two sexes predictably differ on life history-related dimensions; they thus can be expected to employ somewhat different strategies in response to the same cues in the environment (James et al., in press). In most species, 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) and tend to mature more slowly in order to gain the competitive abilities and qualities needed for successful competition for mates. Sexual asymmetries in life history strategies can be attenuated in species with monogamous mating systems and when both parents contribute to offspring care.

One of the most important implications of life history theory is that no strategy can be optimal in every situation; more specifically, the optimal (i.e., fitness-maximizing) strategy for a given organism depends on its ecology and on a series of factors such as resource availability, mortality and environmental uncertainty. Indeed, organisms usually embody mechanisms that allow them to fine-tune their life histories according to the environmental cues they encounter during development. For this reason, life history traits and strategies tend not to be genetically fixed, but rather evolve to show developmental plasticity (Ellis et al., 2009). Developing organisms assess their local environments and adjust their strategic allocation choices, following evolved rules that maximize expected fitness in different ecological conditions. To the extent they result from evolved mechanisms of plasticity, individual differences in life history are examples of *conditional adaptation* (see below).

2.1.1. Factors in the development of life history strategies

The key dimensions of the environment that affect the development of life history strategies are *resource availability, extrinsic morbidity-mortality*, and *unpredictability,* as signaled by observable cues. As explained in detail by Ellis and colleagues (2009), energetic stress (i.e., malnutrition, low energy intake, negative energy balance, and associated internal stressors such as disease) tends to cause the developing organism to shift toward *slow* strategies, characterized by slower growth and maturation and delayed reproduction. In contrast, both extrinsic (i.e., uncontrollable) morbidity-mortality and unpredictable fluctuations in environmental parameters tend to entrain the development of *fast* strategies, accelerating sexual maturation, promoting early reproduction and reducing the amount of parental investment provided to the young.

Of course, genetic factors also contribute to determine individual life history strategies. Theoretical models suggest that one should often expect a balance between genetic and environmental determination of phenotypic individual differences. At the individual level, regulatory mechanisms should often evolve so as to integrate both genetic and environmental

information in phenotypic determination (Leimar et al., 2006). At the population level, the opportunity for habitat choice plus heterogeneous environmental conditions can maintain a diverse population composed of both "specialists" (fixed phenotypes) and "generalists" (plastic phenotypes), as shown by Wilson and Yoshimura (1994). In a similar vein, differential susceptibility theory (Belsky, 1997, 2005) maintains that, because the cues driving the development of conditional phenotypes are not completely reliable, children vary in their susceptibility to rearing influences. Such differential susceptibility underlies pervasive person-by-environment interactions, whereby individuals with given genotypes or phenotypes show higher sensitivity to environmentally-induced effects on development (see Belsky, 1997, 2005; Belsky & Pluess, 2009; Boyce & Ellis, 2005; Ellis et al., 2011).

2.1.2. Life history strategies and the organization of behavior

When interpreted in a narrow sense, life history strategies refer mainly to growth- and reproduction-related traits such as maturation timing, age at first reproduction, fertility, and number of sexual partners. However, it is easy to see that the choice of a specific strategy will affect a much broader range of traits and behaviors (e.g., Belsky et al., 1991; Figueredo et al., 2004, 2005, 2006; Meaney, 2007; Wolf et al., 2007). Imagine an organism that, following cues of extrinsic morbidity-mortality and unpredictability, adopts a strategy characterized by early reproduction and high mating effort. To succeed, the organism needs to outcompete same-sex conspecifics and be chosen by members of the other sex. Especially for males, this is likely to involve dominance-seeking behavior, plus considerable investment in traits and displays that the other sex finds attractive. The cues of environmental risk that drive the choice of the strategy will also prompt higher risk-taking in other domains (e.g., exploration, fighting, dangerous sexual displays), preference for immediate over delayed rewards, and impulsivity (Wolf et al., 2007).

Thus, life history strategies play a powerful role in the organization of behavior. Traits and behaviors that covary along life history dimensions form a broad cluster which includes

exploration/learning styles, mating and sexual strategies, pair-bonding, parenting, status- and dominance-seeking, risk-taking, impulsivity, aggression, cooperation, and altruism. Correlations within this cluster have been documented in both humans (e.g., Del Giudice, 2009a; Figueredo et al., 2004, 2006; Kruger et al., 2008) and other animals (e.g., Dingemanse & Réale, 2005; Korte et al., 2005).

2.2. Conditional Adaptation and Developmental Switch Points

Conditional adaptation is the evolved ability of an organism to modify its developmental trajectory (and the resulting phenotype) to match the local conditions of the social and physical environment. Conditional adaptation is a manifestation of adaptive developmental plasticity (Pigliucci, 2001; West-Eberhard, 2003), and is closely related to the concept of a *predictive adaptive response* (e.g., Gluckman et al., 2007). Mechanisms of developmental adaptation can be guided both by external environmental factors (e.g., predation pressures, quality of parental investment, seasonal change, diet) and by indicators of the individual's status or relative competitive abilities in the population (e.g., age, body size, health, history of wins and losses in agonistic encounters).

How do genetic and environmental factors drive conditional adaptation? West-Eberhard (2003) proposed that developmental change is coordinated by regulatory switch mechanisms, which serve as transducers (mediators) of genetic, environmental, and structural influences on phenotypic variation. These switch mechanisms control *developmental switch points:* "...[points] in time when some element of phenotype changes from a default state, action, or pathway to an alternative one – it is activated, deactivated, altered, or moved" (West-Eberhard, 2003, p. 67). This can involve a discrete structural change or a change in the rates of a process. Genetic and environmental inputs interact with the extant phenotype to determine the functioning of regulatory switch mechanisms and influence their thresholds. Once a threshold is passed (i.e., the switch occurs), the regulatory mechanism coordinates the expression and use of gene products and environmental elements that

mediate the species-typical transition to the new phenotypic stage as well as individually differentiated pathways within that stage.

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

[FIGURE 1 ABOUT HERE]

3. The Stress Response System as a Mechanism of Conditional Adaptation

3.1. Functions of the Stress Response System

The SRS has three main biological functions (see Figure 1). We will now examine these functions in light of the biological concepts presented in the previous section.

3.1.1. Allostasis

A key function of the SRS is to coordinate the organism's physiological and behavioral response to environmental threats and opportunities. This includes any event that may have important (i.e., fitness-relevant) consequences for the organism and requires the organism to modify its current state in order to be dealt with effectively. In addition to threats and dangers, environmental opportunities may be represented by unexpected or novel events, and even highly pleasurable situations (e.g., signs of sexual availability in a potential mate). The whole-organism adjustment to environmental challenge is often termed *allostasis* (McEwen, 1998; McEwen & Wingfield, 2003; Sterling & Eyer, 1988). The SRS mediates allostasis by coordinating brain/body changes in response to environmental challenges, both in the short and in the long term. Because allostasis is a broader concept than "stress response", and because many of the challenges that activate the SNS and HPA are not "stressors" in the classical sense, the label "stress response

system" is not entirely adequate to describe the function of the SRS. Here we employ it for lack of a widely accepted alternative; however, we want to make it clear that the SRS is a general interface with the environment, mediating the organism's adjustment to both positive and negative events (Boyce & Ellis, 2005; Koolhaas et al., 2011).

3.1.2. Information encoding and filtering

The second function of the SRS, closely connected to the first, is that of encoding and filtering *information* coming from the social and physical environment. The SRS receives complex information about the external environment through limbic structures, and complex information about the organism from interaction with other neuroendocrine systems (e.g., the HPG axis and the immune system; see Herman et al., 2003). Activation of the SRS components thus carries information about the likelihood of threats and opportunities in the environment, their type, and their severity. This information can be encoded by the SRS and, in the long run, provides the organism with a statistical "summary" of key dimensions of the environment, including the crucial life history-relevant dimensions of extrinsic morbidity-mortality and unpredictability. Indeed, unpredictable and uncontrollable events elicit the strongest SRS responses across species, especially at the level of the HPA axis (Dickerson & Kemeny, 2004; Koolhaas et al., 2011).

The amount of information encoded by each component of the SRS depends on the specificity of its response. Parasympathetic withdrawal occurs frequently and is a relatively non-specific response, so it comparatively conveys relatively little information about the local environment. Sympathetic activation, in contrast, is more specifically tied to challenges requiring fight-or-flight responses; patterns of SNS activation may thus provide reliable information about the dangerousness (or safety) of one's environment. The most information-rich response (and the one with the longest lasting effects) is that of the HPA axis, which is strongly activated in unpredictable and/or uncontrollable situations.

An important corollary of this informational view of SRS functioning is that the system's level of responsivity acts as an amplifier (when highly responsive) or filter (when unresponsive) of various types of environmental information. A highly responsive system makes an individual more informationally open and enhances his/her sensitivity to contextual influences, both "positive" and "negative" (Boyce & Ellis, 2005; Ellis et al., 2006). An unresponsive system has a higher threshold for letting environmental signals in: many potential challenges will not be encoded as such, and many potentially relevant events will fail to affect the organism's physiology to a significant degree. This will result in a number of potential costs (e.g., reduced alertness, reduced sensitivity to social feedback) as well as potential benefits (e.g., resource economization, avoidance of immune suppression). In fact, many of the possible consequences of low responsivity can be read as either costs or benefits depending on context. Reduced sensitivity to feedback, for example, can be optimal in highly competitive contexts, or when taking deliberate risks. More generally, sometimes organisms do well to partially or totally shield themselves from the effects of environmental information.

A highly responsive SRS, by contrast, amplifies the signal coming from the environment and maximizes the chances that the organism will be modified by current experience. This, too, can have both costs and benefits. Potential costs of a highly responsive system include high physiological costs, hypersensitivity to social feedback, and exposure to psychological manipulation; in addition, the organism's action plans can get easily interrupted by minor challenging events, and the ability to deal with future events may be reduced if physiological resources are already overwhelmed. On the other hand, a highly responsive system facilitates some forms of learning, enhances mental activities in localized domains, focuses attention, and primes memory storage, thus improving cognitive processes for dealing with environmental opportunities and threats (e.g., Barsegyan et al., 2010; Flinn, 2006; Roozendaal, 2000; van Marle et al., 2009).

3.1.3. Regulation of life history-relevant traits

The role of the SRS extends way beyond mounting responses to immediate challenges. Profiles of SRS baseline activity and responsivity are associated with individual differences in a range of life history-relevant domains including competitive risk-taking, learning, self-regulation, attachment, affiliation, reproductive functioning, and caregiving. In the next paragraphs we will discuss some examples (for an extended treatment see Del Giudice et al., 2011).

To begin with, the HPA is crucially involved in the regulation of metabolism, and chronic stress has been linked to individual differences in growth patterns (e.g., Hofer, 1984; Schanberg et al., 1984). Physical growth is an important component of somatic effort but, from the biological point of view, *learning* can also be conceptualized as a form of investment in "embodied capital." A learning organism spends time and energy accumulating knowledge and developing skills that may become useful in the future (e.g., Kaplan et al., 2000). The SRS modulates learning in a number of different ways: In humans, HPA and autonomic profiles have been associated with individual differences in cognitive functioning (e.g., Staton et al., 2009), memory (e.g., Stark et al., 2006), and self-regulation/executive function (e.g., Blair et al., 2005; Shoal et al., 2003; Williams et al., 2009).

The autonomic systems, HPA, and hypothalamic-pituitary-gonadal (HPG) axes are connected by extensive functional cross-talk (e.g., Ellis, 2004; Viau, 2002), and cortisol is a major regulator of fertility and sexual development. Given adequate bioenergetic resources to support growth and reproduction, exposures to chronic psychosocial stressors generally provoke early or accelerated development of the HPG axis but suppressed ovarian functioning in mature individuals (reviewed in Ellis, 2004). The effects of *acute* response to challenge are much more variable; males and females do not respond in the same way, and whether acute stress suppresses or enhances fertility depends on individual characteristics such as dominance status (e.g., Chichinadze & Chichinadze, 2008; Tilbrook et al., 2000). Especially in females, reproductive suppression can be an evolved response to temporary shortages of social or energetic resources (e.g., Brunton et al., 2008; see Wasser &

Barash, 1983), and there is evidence linking HPA functioning to fertility and pregnancy outcomes in human females (e.g., Nepomnaschy et al., 2004, 2006; Wasser & Place, 2001).

Competition among same-sex individuals is the inevitable outcome of sexual reproduction. Dominance-seeking, aggression, and risk-taking are all functionally connected to mating competition, and all are associated with SRS functioning. In humans, there is a huge literature linking HPA and autonomic functioning to aggression, antisociality, and externalizing behavior (e.g., Alink et al., 2008; Lorber, 2004; Shirtcliff et al., 2009; van Goozen et al., 2007). Given the centrality of risk-taking and impulsivity in life history models of behavior, it is noteworthy that HPA functioning has also been linked to risk-taking behavior in standardized laboratory tasks (e.g., Lighthall et al., 2009; van den Bos et al., 2009). Moreover, executive function and self-regulation have a key role as (negative) mediators of risky and impulsive behavior (Figueredo & Jacobs, 2009). Stress exposure can also regulate mating behavior more directly by, for example, altering mate preferences and affecting the perceived attractiveness of potential sexual partners (e.g., Lass-Hennemann et al., 2010).

In the modulation of risky competition, the SRS interacts with sex hormones, serotonin (5-HT), and dopamine (DA). Studies of aggression and antisocial behavior often report interactions between cortisol, testosterone (T) and adrenal androgens such as DHEA and DHEAS (e.g., Popma et al., 2007; van Goozen et al., 2007). The general function of 5-HT is to regulate avoidance of threat, withdrawal from dangerous or aversive cues, and behavioral inhibition/restraint. Serotonergic activity is thus crucially involved in risk aversion and self-regulation (Cools et al., 2008; Fairbanks, 2009; Tops et al., 2009). Serotonin is an upstream modulator of SRS activity through its action on the amygdala and hypothalamus; serotonergic neurotransmission, in turn, is reciprocally affected by cortisol (Porter et al., 2004; van Goozen et al., 2007). Dopaminergic activity is also tightly linked to SRS functioning (Alexander et al., 2011); Gatzke-Kopp (2011) recently argued that reduced dopaminergic activity can be adaptive in highly dangerous and

unstable environments (and especially so for males) by promoting sensation-seeking, risk-taking, and preference for immediate rewards.

Finally, the SRS is involved in the regulation of parental investment, both directly (e.g., caregiving) and indirectly by affecting the mechanisms of pair-bonding. In humans, individual differences in SRS functioning have been associated with differences in romantic attachment styles (e.g., Quirin et al., 2008; Laurent & Powers, 2007; Oskis et al., 2011; Powers et al., 2006); in turn, romantic attachment predicts relationship stability, commitment and investment (reviewed in Del Giudice, 2009a). The key molecules that can be expected to interact with the SRS in the regulation of pair-bonding and parental investment are sex hormones, vasopressin, oxytocin, serotonin, and endogenous opioids. Oxytocin secretion, in particular, has been related to individual differences in romantic attachment styles (e.g., Marazziti et al., 2006). Differences in SRS functioning (as well as in oxytocin- and serotonin-related genes) have been also linked to individual differences in maternal sensitivity and parenting behavior (e.g., Bakermans-Kranenburg & van IJzendoorn, 2009; Martorell & Bugental, 2006).

3.2. The Developmental Role of the Stress Response System

As discussed in the last section, the SRS has a pervasive role in the regulation – and, most importantly, the *integration* – of physiology and behavior across the whole spectrum of life history-relevant traits. In a life history framework, this is no coincidence: we argue that – together with sex hormones and relevant neurotransmitter systems – the SRS is a critical mediator of life history development, gathering information from the environment and translating it into broad-band individual differences in behavior and physiology (Figure 1; see also Korte et al., 2005; Worthman, 2009). In other words, the SRS interacts with other neurobological systems so as to enable conditional adaptation. Following the logic of West-Eberhard's theory (section 2), it should be possible to identify a number of developmental switch points in an organism's life cycle when plasticity is preferentially expressed and environmental cues are integrated with genotypic

information to adjust the organism's developmental trajectory. Of course, long-lived organisms can be expected to have more switch points than short-lived ones, so as to permit sequential adjustment of life history decisions as environmental conditions change (Del Giudice & Belsky, 2011).

Crucially, different strategies may require different calibrations of the SRS itself (curved arrow in Figure 1); for example, a slow strategy in a safe environment could be optimally served by a responsive HPA axis and parasympathetic system, coupled with moderate sympathetic reactivity. SRS calibration can be expected to depend on the system's previous history of activity (Adam et al., 2007), in interaction with factors such as the individual's sex and developmental stage (Miller et al., 2007). The analysis presented in this section can be summarized in the first three points of the ACM (adapted from Del Giudice et al., 2011), which embody the model's theoretical core:

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 broad range of life history-relevant traits and behaviors.
2. The SRS works as a mechanism of conditional adaptation, regulating the development of alternative life history strategies. Different patterns of activation and responsivity in early development modulate differential susceptibility to environmental influence and shift susceptible individuals on alternative pathways, leading to individual differences in life history strategies and in the adaptive calibration of stress responsivity (Figure 1).
3. Activation of the SRS during the initial life stages provides crucial information about life history -relevant dimensions of the environment. Frequent, intense SNS/HPA activation carries information about extrinsic morbidity-mortality and environmental unpredictability; consequently, it tends to shift life history strategies toward the fast end of the life history continuum. In contrast, a safe environment (and/or the buffer

provided by investing parents and alloparents) results in infrequent and low-intensity activation of the SNS and HPA axis, and shifts development toward slow strategies oriented to high somatic effort and parental investment.

4. The Development of Stress Responsivity in Humans

The theoretical core of the ACM (Figure 1) can be employed as the foundation for a detailed model of the development of stress responsivity in humans. We tried to accomplish this in two steps: first, we put forth some general predictions on the relation between environmental conditions and responsivity; second, we described a (provisional) taxonomy of four prototypical patterns of SRS responsivity labeled *sensitive* [I], *buffered* [II], *vigilant* [III], and *unemotional* [IV]. The four patterns are characterized by combinations of physiological parameters indexing the functioning of the parasympathetic and sympathetic branches of the autonomic system and of the HPA axis. Our predictions can be summarized in the remaining four points of the ACM, as follows:

4. At a very general level, a nonlinear relationship exists between environmental stress during ontogenetic development and the optimal level of stress responsivity (Figure 2). Note that the environment-responsivity relationship need not be the same for all the components of the SRS (for details see Del Giudice et al., 2011). Furthermore, stress responsivity is expected to show domain-specific effects; for example, a generally unresponsive component of the SRS may respond strongly to some particular type of challenge.
5. Because of sex differences in life history trade-offs and optimal strategies, sex differences are expected in the distribution of responsivity patterns and in their specific behavioral correlates. Sex differences should become more pronounced toward the fast end of the life history continuum; in environments characterized by severe/traumatic stress, we predict the emergence of a male-biased pattern of low responsivity.

6. Pre- and early postnatal development, the juvenile transition (see below), and puberty are likely switch points for the calibration of stress responsivity. Individual and sex differences in SRS functioning are predicted to emerge according to the evolutionary function of each developmental stage.
7. Responsivity profiles develop under the joint effects of environmental and genetic factors. Genotypic variation may have directional effects on stress responsivity and associated strategies, thus predisposing some individuals to follow a certain developmental trajectory. Genotypic variation, in part through effects on the SRS, may also affect their sensitivity to environmental inputs, resulting in gene-environment interactions whereby some individuals display a broader range of possible developmental outcomes (i.e., broader reaction norms) than others.

[FIGURE 2 ABOUT HERE]

4.1. Environmental Stress and Responsivity

In safe, protected, low-stress environments, a highly responsive SRS enhances social learning and engagement with the external world, allowing the child to benefit more fully from social resources and opportunities, thus favoring development of a *sensitive* phenotype (pattern I). A sensitive phenotype in this context may make children better at detecting positive opportunities and learning to capitalize on them (e.g., seeing a teacher as a prospective mentor, taking advice from a parent). Social learning and sensitivity to context are especially adaptive in the context of slow life history strategies, as a form of protracted somatic investment. It is important to note that in very safe and protected settings, sensitive individuals will *rarely* experience strong, sustained activation of the sympathetic and HPA systems; precisely because of the high quality of the environment, they will most likely experience a pattern of low-key, short-lived activations followed by quick recovery. Thus, the individual enjoys the benefits of responsivity without paying significant fitness costs (e.g., immune, energetic, and so on). At moderate levels of environmental stress, however, the

cost/benefit balance begins to shift; the optimal level of HPA and sympathetic responsivity falls downward, leading to *buffered* phenotypes (pattern II).

The benefits of increased responsivity rise again when the environment is perceived as dangerous and/or unpredictable. A responsive SRS enhances the individual's ability to react appropriately to dangers and threats while maintaining a high level of engagement with the social and physical environment. Moreover, engaging in fast strategies should lead the individual to allocate resources in a manner that discounts the long-term physiological costs of the stress response in favor of more immediate advantages. In this context, the benefits of successful defensive strategies outweigh the costs of frequent, sustained HPA and sympathetic activation, leading to *vigilant* phenotypes (pattern III). High HPA and sympathetic responsivity, however, can be associated with rather different behavioral patterns, leaning toward the "fight" (*vigilant-agonistic*, III-A) or "flight" (*vigilant-withdrawn*, III-W) side of the sympathetic response. Furthermore, evolutionary theory provides reasons to expect males and females to differ in the distribution of agonistic versus withdrawn patterns (see below). Increased SRS responsivity in dangerous environments can be expected to go together with increased responsivity in other neurobiological systems; for example, hyper-dopaminergic function may contribute to the vigilant phenotype by boosting attention to threat-related cues and fast associative learning (Gatzke-Kopp, 2011).

What happens in extremely dangerous environments characterized by severe or traumatic stress? We argue that the balance shifts again toward low responsivity, especially for males who adopt a fast, mating-oriented strategy characterized by antagonistic competition and extreme risk-taking. Such a strategy requires outright *insensitivity* to threats, dangers, social feedback and the social context. For an extreme risk-taker, informational insulation from environmental signals of threat is an asset, not a weakness. 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 HPA responsivity). In summary, an *unemotional* pattern of generalized low responsivity (pattern IV) can be evolutionarily adaptive (i.e., fitness-maximizing) at the high-risk end of the environmental spectrum, despite its possible negative consequences for the social group and for the individual's subjective well-being. The same principle applies to other neurobiological systems involved in the regulation of risk-taking; for example, hypodopaminergic function is likely adaptive in severely stressful environments (Gatzke-Kopp, 2011).

Figure 2 depicts the overall predicted relations between developmental context and stress responsivity, extending the original BSC curve to the right and showing the male-biased pattern of low responsivity in high-risk environments. This broad-band analysis can be supplemented with a more fine-grained description of the profiles of basal activity and responsivity of the various SRS components (see Del Giudice et al., 2011).

4.2. Sex Differences

Because the costs and benefits associated with life history trade-offs are not the same for males and females, life history strategies show consistent differences between the sexes (section 2). On average, men engage in faster strategies and invest more in mating effort (and less in parenting effort) than women. 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 engage in high parental investment, and male and female interests largely converge on long-term, committed pair bonds; sex differences in behavior are thus expected to be relatively small. As environmental danger and unpredictability increase, males benefit by shifting to low-investment, high-mating strategies; females, however, do not have the same flexibility since they benefit much less from mating with multiple partners and incur higher fixed costs through childbearing. Thus, male and female strategies should increasingly diverge at moderate to high levels of environmental 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 (e.g., Archer, 2009; Byrnes et al., 1999; Kruger & Nesse, 2006; Wilson et al., 2002). As life history strategies become faster, sexual competition becomes stronger, and sex differences in competitive strategies become more apparent. For these reasons, sex differences in responsivity patterns and in the associated behavioral phenotypes should be relatively small at low to moderate levels of environmental stress (patterns I and II) and increase in stressful environments (pattern III). Finally, males should be over-represented as high-risk, low-investment strategists (pattern IV) because of the larger potential benefits of extreme mating-oriented behavior.

4.3. Developmental Stages and Switch Points in Human Development

The human life history can be described as a sequence of stages and transitions (Bogin, 1999). Life history strategies unfold progressively, according to the evolutionary function of each life stage. Del Giudice and Belsky (2011) proposed that the major switch points in the development of human life history strategies are (a) pre- and early postnatal development, (b) the juvenile transition, and (c) puberty. The juvenile transition (Del Giudice et al., 2009) is the transition from early to middle childhood, taking place at around 6-8 years of age in Western societies. This developmental transition is marked by the event of "adrenal puberty" or *adrenarche* (Auchus & Rainey, 2004; Ibáñez et al., 2000), whereby the cortex of the adrenal glands begins to secrete increasing quantities of androgens, mainly dehydroepiandrosterone (DHEA) and dehydroepiandrosterone sulfate (DHEAS). The onset of human juvenility (i.e., middle childhood) witnesses massive changes in children's social behavior, cognitive abilities, and the emergence or intensification of sex differences in aggression, attachment, play, language use, and so forth (reviewed in Del Giudice et al., 2009).

The juvenile transition can be expected to be a critical turning point in the development of stress responsivity. First, we predict that sex differences in the developmental trajectories of stress responsivity will become apparent starting from the beginning of middle childhood, with a further

increase at puberty. Second, we expect that individual changes in responsivity will be especially frequent in the transition from early to middle childhood. Early childhood affords an "evaluation" period in which the child can sample the environment – both directly and through the mediation of parents. With juvenility, however, stress responsivity becomes an integral component of the child's emerging life history strategy. Indeed, the SRS is crucially involved in the biological functions of juvenility – including social learning and peer competition. For this reason, it may be adaptive for some children to adjust their levels of responsivity when transitioning from early to middle childhood, possibly under the effect of adrenal androgens.

With the onset of puberty, sexual behavior and romantic attachment come to the forefront, and social competition further intensifies (see Ellis et al., in press a; Weisfeld, 1999). Puberty affords another opportunity to "revise" one's strategy, depending for example on the success enjoyed – or the level of competition experienced – during juvenility. The activation of sex hormone pathways also provides a source of novel genetic effects on life history-related behavior. Thus, adolescence is expected to witness the further intensification of both individual and sex-related differences.

5. Conclusion

The ACM offers an integrative view of the evolved functions of the SRS and its role in development. We believe this perspective will prove useful, both in organizing and systematizing existing knowledge and in suggesting novel questions for empirical research. In our opinion, what the field needs is more fundamental theory, rather than a multitude of alternative micro-models without a common frame of reference. Although the original model was developed to capture individual differences in humans, we are excited at the prospect of extending the core of the ACM to deal with different species and different ecologies. Adding a phylogenetic and comparative dimension to the ACM would be extremely valuable, in keeping with the spirit of Tinbergen's four

questions. Many stimulating reflections on the ACM from the perspective of animal behavioral ecology can be found in Sih (2011).

To conclude, we wish to stress that the ACM is a work in progress, and that many theoretical and empirical gaps still have to be filled in. For example, much more work is needed on domain-specificity in SRS functioning, and on the mechanistic basis of genetic effects and GxE interactions in development. Furthermore, the initial focus of our model was skewed toward adaptive variation; while the ACM recasts many supposedly "pathological" processes in an adaptive framework, it still lacks an explicit treatment of actual dysfunction and pathology (Ellis et al., in press b). Mathematical models of the developmental processes hypothesized in the ACM would also help refine the theory and test the robustness of its assumptions. We anticipate that, in the near future, substantial portions of the model (especially the more specific ones) will have to be updated, revised, and possibly rejected. If so, the model will have served his goal of moving the field forward, promoting theoretical advance, and increasing the vitality of an important and exciting field of research.

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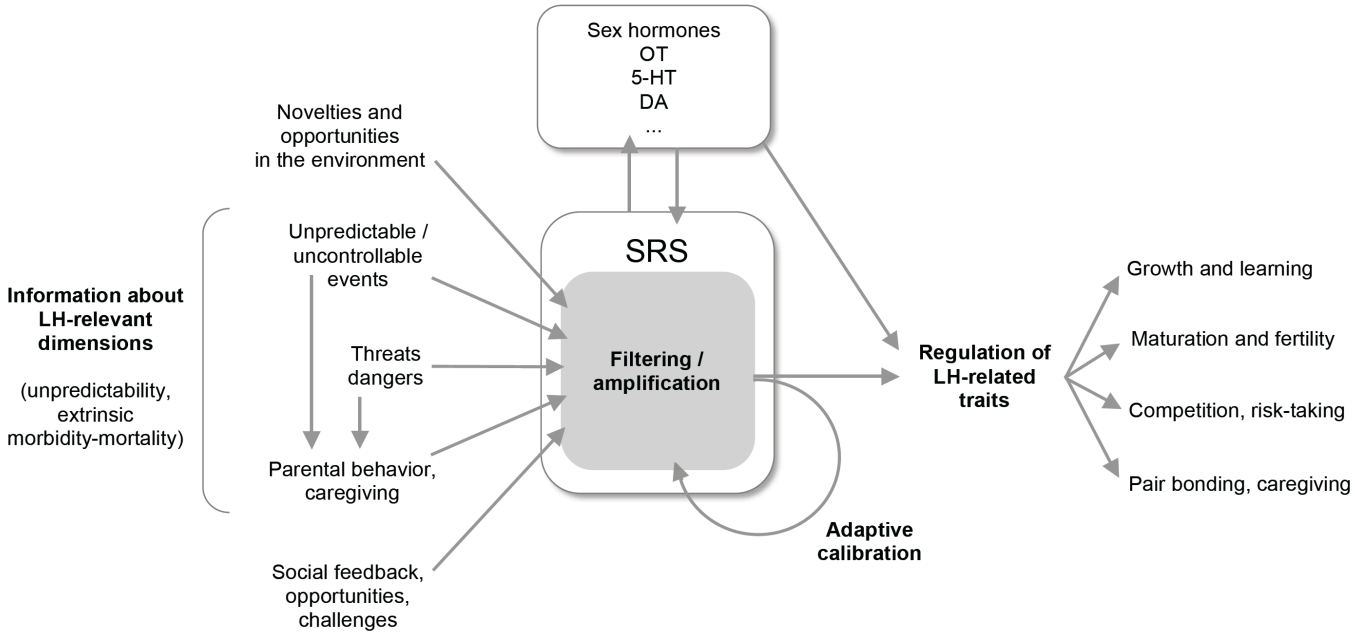

Figure 1. The core theoretical structure of the Adaptive Calibration Model. SRS: stress response system; LH: life history; OT: oxytocin; 5-HT: serotonin, DA: dopamine.

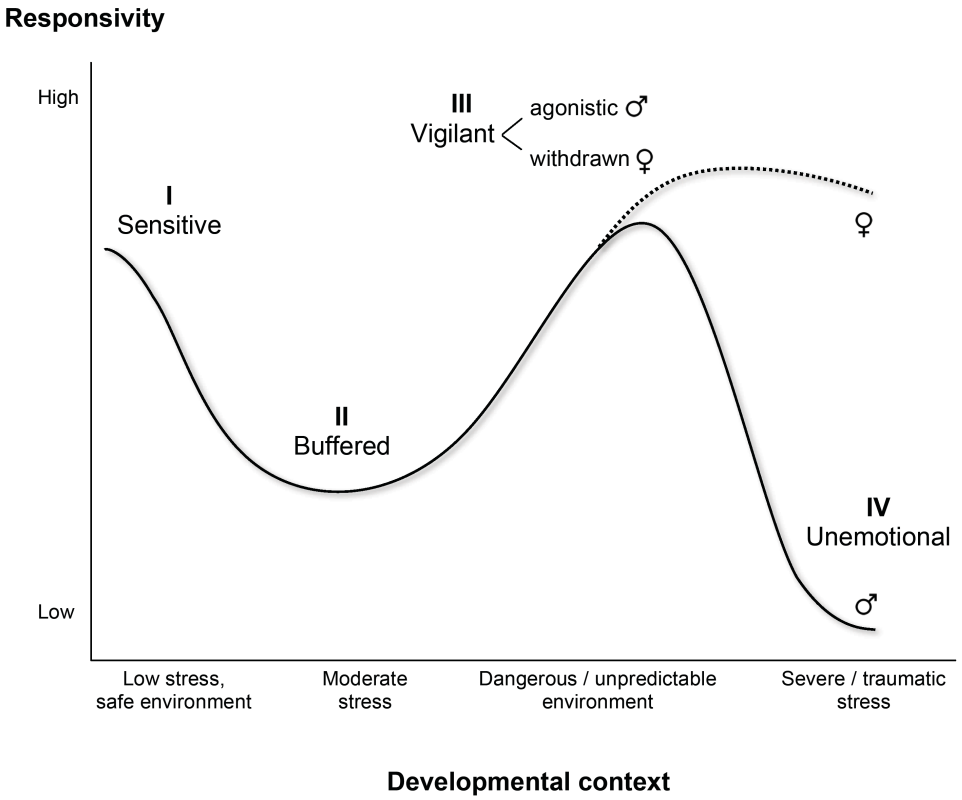

Figure 2. Environmental effects on the development of stress responsivity in humans, according to the Adaptive Calibration Model. At a very general level, a nonlinear relation exists between exposures to environmental stress and support during development and optimal levels of stress responsivity. The figure does not imply that all components of the SRS will show identical responsivity profiles, nor that they will activate at the same time or over the same time course. Male/female symbols indicate sex-typical patterns of responsivity, but substantial within-sex differences in responsivity are expected as well.