Chapter 5
The Biobehavioral Legacy of Early Attachment Relationships for Adult Emotional and Interpersonal Functioning

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One of the chief contributions of attachment theory to relationship science is that it provides a coherent framework for understanding how individuals' earliest ties to their caregivers during infancy and childhood influence the quality of their adult romantic bonds. Historically, these linkages have been understood chiefly in terms of individuals' internalized, psychological models of relationships—the constellation of cognitions and expectations regarding attachment figures which provide the template for individuals' perceptions of, feelings about, and behaviors toward romantic partners. Yet an important development in attachment research over the past several decades is the increased attention to the *biological* implications of early attachment experiences. Specifically, we now know that early attachment experiences shape not only individuals' cognitions and emotions, but also a range of basic physiological systems involved in stress reactivity and regulation. The functioning of these systems, in turn, influences the development of multiple interpersonal processes which are critically implicated in the formation and maintenance of adult attachment bonds. The purpose of this chapter is to review this basic model. Specifically, (1) the quality of early caregiving, in interaction with genetically based temperament and overall stress exposure, calibrates the infant's basic stress-regulatory systems, most notably the autonomic nervous system (ANS) and the hypothalamic–pituitary–adrenocortical (HPA) axis; (2) the infant's resulting profile of ANS and HPA reactivity shapes his/her developing capacity for stress- and emotion-regulation and, as a result, his/her emerging interpersonal skills; (3) by adulthood, this variability in interpersonal and stress-regulatory skills affects the formation and maintenance of adult attachment bonds, by shaping individuals' abilities to seek and provide the safe haven and secure base components of attachment with their romantic partners during times of stress.

Yet importantly, this is not a biologically determinist model. One of the most exciting recent developments in research on the role of ANS and HPA functioning in child development is the emerging view that children's profiles of stress reactivity

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do not represent global vulnerabilities, but rather *differential susceptibilities* to environmental input (Belsky et al. 2007; Belsky and Pluess 2009; Del Giudice et al. 2011; Ellis et al. 2011). Hence, when the environmental input changes from bad to good, individuals with highly reactive HPA and ANS systems might be disproportionately likely to benefit, in essence "absorbing" more developmental strengths and skills from highly nurturant attachments than their low-reactive counterparts. One intriguing possibility is that this differential susceptibility extends to early adulthood, potentially rendering highly reactive individuals more likely to develop enhanced interpersonal and self-regulatory skills if some of their earliest adult attachment bonds are particularly nurturant and supportive. Toward the end of this chapter, I outline promising directions for future research on such possibilities.

Attachment and the Biology of Stress and Emotion Regulation

Although attachment theory has historically been viewed as a theory of interpersonal functioning, Bowlby (1977) placed considerable emphasis on the role of the attachment system in governing *overall* responses to danger and threat. Confirming Bowby's view, researchers have demonstrated robust associations between attachment experiences and individuals' emotional responses to major and minor stressors (Ditzen et al. 2008; Maunder et al. 2006; Mikulincer and Florian 2004; Simpson et al. 2002), and studies have increasingly investigated the specific behavioral, cognitive, and physiological processes underlying the emotion-regulation functions of the attachment system. *Emotion regulation,* in this context, refers to the range of internal and transactional processes through which individuals consciously or unconsciously modulate the experience or expression of emotions elicited by environmental events (Gross 1999; Thompson 1994). Adaptive versus maladaptive patterns of emotion regulation shape the ways in which individuals perceive, appraise, and react to emotionally relevant experiences (reviewed in Diamond and Hicks 2004), and they are also fundamentally integrated with broader self-regulatory processes including executive functioning, response inhibition, and the regulation of attention (Koole 2009; Lewis et al. 2006; Ochsner and Gross 2007; Posner and Rothbart 2007). Hence, effective emotion regulation is critical for state regulation, social competence, and interpersonal behavior (Cicchetti et al. 1995; Frick and Morris 2004; Silk et al. 2003), and is considered a core developmental achievement for both children and adolescents (Collins et al. 1998; Denham 2006; Eisenberg et al. 2002).

Attachment figures foster the development and maintenance of the infants' emotion regulation abilities by continuously modulating the infant's affective and attentional state in response to changing situational demands. They achieve this on an ongoing basis by adjusting their own facial expressions, vocalizations, and physical touch during routine interactions with the infant, and by regulating the infant's direct engagement with the environment. This routine regulatory "scaffolding"

supports the development of the infant's own self-regulatory capacities, as strong positive and negative emotions are repeatedly elicited, experienced, expressed, and modulated dyadically from situation to situation and moment to moment (Dickson et al. 1998; Fogel 2001; Fogel et al. 1999; Tronick 2007). Importantly, the attachment figure need not be perfectly attuned to the infant at each and every moment in order for these normative developmental processes to occur. Rather, even the periodic "mismatches" between the infant's and the caregiver's expressions, intensions, and affects are developmentally beneficial, as they provide opportunities for coordinated communicative "repair" that supports the infant's developing ability to cope with routine irritants and stressors (DiCorcia and Tronick 2011; Tronick and Beeghly 2011).

The cognitive and behavioral aspects of these normative dyadic processes have received extensive attention over the years, yet these processes also have physiological manifestations. Specifically, the processes by which attachment figures regulate their infants' ongoing emotional experiences is now understood to play a central role in "tuning" stress regulatory systems in the orbitofrontal cortex that provide the foundation for effective self- and emotion-regulation (Schore 1996; Siegel 2001). This emerging body of work is quite consistent with Bowlby's original formulation of the attachment system as fundamentally psychobiological. Specifically, he posited two different "rings" of homeostasis that assist the individual in responding to major and minor stressors so that emotional security could be maintained and environmental exploration fostered (Bowlby 1973). The inner ring comprises life-maintaining biological systems that govern ongoing physiological adaptation to external demands. The outer ring comprises behavioral (and particularly, interpersonal) strategies for coping and adaptation. From Bowlby's perspective, the integrated functioning of these two levels is critical for optimal self-regulation.

Extensive research confirms Bowlby's view. We now know that deficits in the quality of the infant–caregiver relationship disrupt not only children's social and behavioral development, but also their biological capacities for maintaining homeostasis in the face of threat (reviewed in Repetti et al. 2002). To understand these processes, developmentalists have increasingly adopted "biosocial" perspectives on the family (Booth et al. 2000), aimed at identifying the reciprocal influences among environmental, interpersonal, behavioral, psychological, and biological processes that unfold within family relations over time (Cairns et al. 1990; Gottlieb 1991). In this view, profiles of biological reactivity—established by interactions between genetic predispositions and early infant/caregiver interactions—set the stage for certain biological, cognitive, emotional, and behavioral responses to stress and challenge. These response patterns become regularized over time, especially as individuals self-select themselves into and out of environments that "fit" their patterns, and over time these patterns exert enduring influences on individuals' psychological functioning and the quality of their intimate relationships. Although such dynamics involve numerous biological processes, we focus below on two systems which have particular relevance for affect regulation: the autonomic system and the HPA axis of the endocrine system. We provide a brief review of these systems, followed by evidence for the critical role of early caregiving (including the quality of

infant–caregiver attachment) in establishing enduring reactivity profiles. We then address the implications of these reactivity profiles for emotion regulation and, by extension, *adult* romantic attachment functioning. We conclude by highlighting recent research on the plasticity of these developmental processes, and identifying promising areas for future research on how individuals' earliest adult attachment bonds might prove differentially formative for individuals with different physiological reactivity profiles.

The HPA Axis

Our bodies regulate responses to psychological stress through two primary pathways: The hypothalamic–pituitary–adrenal (or HPA) axis, characterized by activation of the pituitary gland and release of adrenocorticotropic hormone (ACTH) and cortisol, and the sympathetic–adrenal medullary (SAM) axis, characterized by activation of the adrenal medulla (which is part of the autonomic nervous system, or ANS), release of catecholamines (such as norepinephrine and epinephrine) and immediate effects on cardiovascular functioning. Hence, both HPA and ANS activation provide markers of stress reactivity, but they represent distinct "channels" through which stress is regulated in the body, with different antecedents, different effects on other stress-induced biological processes (such as cellular immune function), and different long-term consequences for physical and mental health (Cacioppo 1994). Research has found that the manner in which individuals appraise the stressor at hand shapes the degree to which his/her physiological response is characterized by combined SAM/HPA activation versus SAM activation alone (Blascovich and Tomaka 1996; reviewed in Cacioppo 1994).

In cases where stressors are primarily appraised as challenges (i.e., in which one's resources are viewed as adequate for meeting the demand), the hypothalamus activates the adrenal medulla to release catecholamines, which activate the sympathetic nervous system (SNS) and inhibit the parasympathetic nervous system (PNS), producing increased heart rate, blood pressure, and respiration. In cases where stressors are primarily appraised as threats (i.e., in which one's resources are not viewed as adequate for meeting the demand), the hypothalamus activates the anterior pituitary in addition to the adrenal medulla. The pituitary is signaled to release ACTH, which in turn triggers the release of glucocorticoid hormones, primarily cortisol, into the bloodstream. The release of cortisol facilitates the body's response to stress by regulating glucose metabolism, inflammatory responses, localized blood flow, and the maturation of lymphocytes (Sapolsky et al. 2000). Hence, although most responses to stress involved combined patterns of HPA and SAM activation, the differences between the antecedents and consequences of these two different stress pathways (Blascovich and Tomaka 1996; Cacioppo 1994) makes it important to discus each system separately.

HPA Activation

Studies examining patterns of HPA reactivity to stress have generally followed two different approaches: The first involves measurement of increases in cortisol in response to a laboratory stress task, relative to a pre-task baseline (for a comprehensive review of published research using such paradigms, and a synthesis of the task characteristics most strongly associated with reactivity, see Dickerson and Kemeny 2004). Yet studies have also investigated how acute and chronic stressors influence sustained patterns of cortisol release over one or several days. Cortisol release follows a diurnal pattern, peaking in the first half hour after waking and then declining over the rest of the day.

Extensive research has found that exposure to major and minor stress can produce both transient and lasting alterations in this pattern of secretion (reviewed by Miller et al. 2007). Yet importantly, whereas laboratory studies of momentary HPA reactivity typically detect transient *increases* in cortisol in response to psychological stress, studies of longer-term patterns of cortisol release have found that sustained exposure to stress can elicit chronic increases *or* decreases in cortisol. Hence, stress-related dysregulation of the HPA axis appears to take two forms: Exaggerated cortisol release (paralleling the transient increases found in laboratory studies) and dampened or "blunted" cortisol release, in which the pattern of diurnal secretion is lowered or "flattened," lacking the pronounced morning rise or the evening fall that characterizes normal HPA functioning.

The processes through which chronic stress produces each pattern are thought to be somewhat different. Exaggerated HPA activity is thought to result from stress-related disruption of the normal feedback processes through which HPA activation is typically "shut down" once sufficient levels of cortisol are present in the bloodstream to meet environmental demands. Chronically low or "blunted" HPA activity, in contrast, has been interpreted as a potentially adaptive mechanism for protecting the brain from the detrimental effects of sustained stress-related exposure to cortisol. Awareness of both patterns of dysregulation is important, given that both patterns have been linked to early stress and caregiving experiences (Miller et al. 2011).

The Autonomic Nervous System

The classic "fight-or-flight" response to stress, with its well-known manifestations of increased heart rate, blood pressure, and sweat production, is part of a larger syndrome of physiological changes produced by the ANS, including increased cardiac output, widespread vasoconstriction, and changes in blood flow to the skeletal muscles, myocardium, brain, kidneys, gastrointestinal tract, and skin. All of these changes serve the purpose of redistributing metabolic energy throughout the body so that the organism can either "fight" or "flee" threats.

The ANS has two branches, the PNS and the SNS, which have antagonistic effects on autonomic functioning. Heightened activation of the SNS produces the physiological changes most commonly associated with fight-or-flight responses: acceleration in heart rate, increased blood pressure, increased sweating, etc. In contrast, the PNS is responsible for maintaining normal growth and restoration of internal organs, processes that are suspended in times of intense stress. Thus, stress-induced activation of the SNS is usually accompanied by some degree of "withdrawal" or "suppression" in the PNS, which functions to redistribute metabolic resources to cope with the external threat. Post-stress reengagement of the PNS channels metabolic energy back toward normal maintenance of internal organs and reestablishes homeostasis. Thus, PNS engagement produces the types of physiological changes associated with relaxation rather than arousal, such as decreased heart rate and blood pressure.

Correspondingly, each and every change in ANS activity must be understood as the product of parasympathetic and sympathetic influence, and thus stress responses such as heart rate acceleration can be brought about by activation of the SNS, withdrawal of the PNS, or some combination of the two. The specific balance of SNS and PNS control over cardiovascular functioning varies from situation to situation (Berntson et al. 1996) as well as from person to person (Berntson et al. 1994; Cacioppo et al. 1994).

ANS stress responses that involve a greater degree of PNS withdrawal than SNS activation appear to be more rapid, more flexible, and easier to disengage than SNS-dominated responses (Berger et al. 1989; Saul 1990; Spear et al. 1979), and thus individuals with more parasympathetically mediated patterns of cardiovascular reactivity are conceptualized as having nervous systems that more flexibly react to and recover from environmental stressors than those with sympathetically mediated patterns (Calkins 1997; DeGangi et al. 1991; Porges 1992; Porges et al. 1994).

Individual Differences in HPA and ANS reactivity: Links to Early Caregiving

Multiple studies of animals and humans have documented stable individual differences in both HPA and ANS stress reactivity that appear to have both genetic and environmental determinants (Kirschbaum et al. 1992; Piha et al. 1994; Propper et al. 2008; Snieder et al. 1997). Importantly, for both systems the major environmental determinants appear to be early adversity and early caregiving.

Specifically, research indicates that variations in stress and caregiving at sensitive periods of development can be particularly formative and can result in permanent alterations in the functioning of both the HPA-axis and the ANS (de Kloet et al. 2005; Gunnar and Quevedo 2007; Oitzl et al. 2010; Taylor et al. 2011). With respect to HPA functioning, inadequate parental care appears to have enduring detrimental effects on HPA regulation and broader neurodevelopment (Buss et al. 2007; Heim and Nemeroff 1999, 2001; Heim et al. 2008), particularly during the

earliest years of life when brain systems such as the hippocampus and prefrontal cortex, which play key roles in the regulation of the HPA axis, undergo major development (Sanchez et al. 2001; Teicher et al. 2003). Notably, negative effects have been detected for *both* maternal and paternal care: Recent studies have found that paternal negativity is associated with heightened cortisol release to stress in infancy (Mills-Koonce et al. 2011) and heightened basal and reactive cortisol (in response to peer interactions) in adolescence (Byrd-Craven et al. 2012). In contrast to the detrimental effects of inadequate parental care, high levels of physical affection and warmth between a caregiver and his or her infant during stressful circumstances have been tied to normal HPA activation profiles in response to environmental demands (Chorpita and Barlow 1998; Gunnar 1998; Spangler et al. 1994), which is thought to promote overall biobehavioral regulation and well-being (Gunnar 2003; Gunnar and Donzella 2002).

Regarding ANS activity, studies have found that individual differences in PNS reactivity to stress among children are associated with the quality of parenting practices (Blandon et al. 2010; Calkins et al. 1998). In infants, PNS reactivity is associated with the degree of synchrony and symmetric responsiveness of mother–infant interaction (Moore and Calkins 2004; Porter 2003). Aspects of the home environment, such as marital conflict, are also significantly associated with PNS functioning (Porter et al. 2003). Links between early caregiving and ANS functioning appear to be preserved into adolescence and adulthood. For example, Luecken (1998) found elevated blood pressure reactivity among young adults who had undergone the loss of a parent as children, coupled with poor relationship quality in the family, and has documented interaction effects (between parental loss and parental caring) in predicting adult blood pressure reactivity and recovery (Luecken et al. 2009; Luecken et al. 2005).

Implications of HPA and ANS Activity for Emotion Regulation

Although the influence of early caregiving relationships on enduring profiles of HPA and ANS functioning is interesting and important in its own right, it is also important because dysregulated patterns of stress reactivity lead to deficits in social and interpersonal functioning that in turn have important implications for adult attachment relationships. In essence, individual differences in HPA and ANS functioning provides a potent biological mechanism through which deficits in childhood attachment bonds carry forward to reproduce deficits in *adult* attachment bonds, mediated by deficits in emotion regulation that make it difficult for adults to manage day-to-day interpersonal challenges that call for effective emotion regulation in the service of adaptive relationship functioning.

Accordingly, studies have found that individuals who show exaggerated HPA hyperreactivity to stress show deficient coping strategies and exaggerated experiences of negative affect (reviewed in Scarpa and Raine 1997; Stansbury and Gunnar

1994). Additionally, individuals whose HPA reactivity fails to habituate to *repeated* stressor administration are characterized by low self-esteem, low extraversion, high neuroticism, and multiple physical complaints (Kirschbaum et al. 1995). Similar findings were reported by Gerra et al. (2000), who found that adolescents with sustained HPA reactivity were more depressive and harm-avoidant. Other studies have found that youths with heightened HPA reactivity show greater inhibition (Kagan et al. 1987), and more rumination during stress anticipation (Roger and Najarian 1998). Links between HPA activity and emotion regulation have also been found for basal profiles: For example, elevated tonic cortisol levels have been found to be associated with general anxiety among adolescents girls (Schiefelbein and Susman 2006). Notably, research has found that dysregulation of cortisol can also manifest as chronically suppressed or "blunted" cortisol levels (reviewed extensively by Miller et al. 2007). For example, adult men with high levels of cynical hostility show flattened 24-hour cortisol profiles (Pope and Smith 1991) and tonically suppressed cortisol has been found to be associated with callous-unemotional traits in adolescent boys (Loney et al. 2006). Another study assessing diurnal profiles of cortisol release found that adolescents with higher levels of depressive symptoms had slightly lower basal cortisol levels, whereas adolescents with higher levels of trait anger had a significantly stronger cortisol response to awakening (Adam 2006).

Individual differences in ANS functioning are also associated with emotion regulation deficits. For example, excessive reactivity in the SNS system, typically indexed by skin conductance (SCL) reactivity to laboratory stressors, has been posited as a potential marker of children's hypersensitivity to environmental challenges (Boucsein 1991). Accordingly, heightened SCL reactivity predicts a variety of child and adolescent psychosocial outcomes, including reactive aggression (Hubbard et al. 2004, 2002), anxiety (Weems et al. 2005), shyness and inhibition (Kagan et al. 1987), emotional disorders (Garralda et al. 1991), and internalizing and externalizing problems (El-Sheikh 2005; El-Sheikh et al. 2007). In contrast to these reactivity effects, low SNS activity at baseline has been found to predict heightened risk for outcomes such as aggression and conduct problems (Beauchaine et al. 2007; Crowell et al. 2006; Lorber 2004; Raine et al. 1990).

The role of PNS functioning for emotion regulation is outlined in Thayer and Lane's (2000) neurovisceral integration model and Porges' polyvagal theory (Porges 2003), both of which suggest that PNS regulation of heart rate undergirds the constellation of self-regulatory and emotion-regulatory processes that are fundamental to adaptive socioemotional functioning. Generally, vagal activity provides a constant "brake" on cardiovascular functioning ("vagal" refers to the functioning of the 10th cranial nerve, which provides inhibitory input to the heart and plays a critical role in regulating metabolic output in response to environmental events). This tonic inhibitory control permits rapid and efficient modulation of cardiovascular activity in the service of changing environmental demands. Whereas SNS influences on heart rate are relatively slow-acting, typically taking several seconds, vagal inhibition can be suspended in a matter of milliseconds (Saul 1990). Hence, individuals with greater tonic PNS regulation of heart rate (often denoted *vagal tone,* and assessed via resting levels of respiratory sinus arrhythmia, or RSA) are

conceptualized as having nervous systems that flexibly react to and recover from environmental stressors (Calkins 1997; DeGangi et al. 1991), which facilitates effective coordination of expressive and affective behavior in the service of social engagement (Porges 2003).

Numerous studies have provided empirical support for this model. For example, infants with low vagal tone (i.e., low baseline levels of RSA) show poor emotional control (Fox 1989; Porges 1991) and high behavioral inhibition (Snidman 1989). Children and adults with low vagal tone show ineffective behavioral coping in response to stress (Fabes and Eisenberg 1997; Fabes et al. 1993), as well as higher levels of anger, hostility, mental stress, and generalized anxiety (reviewed in Brosschot and Thayer 1998; Friedman and Thayer 1998; Horsten et al. 1999). Complementing these findings, recent research also suggests that vagal tone is associated with tonic positive affectivity (Oveis et al. 2009), which may provide a relational "building block" promoting approach-oriented behavior and fostering social resources (following Fredrickson 1998).

Although the majority of research on affect regulation and the PNS has focused on vagal tone (operationalized as resting levels of RSA), an increasing body of research assesses changes in PNS activity during stress and other environmental demands (operationalized as baseline-to-task changes in RSA). Polyvagal theory (Porges 2001) suggests a normative, adaptive pattern of reduced RSA during stress (often denoted vagal *suppression* or withdrawal, given that reductions in RSA represent reductions in PNS activity). Although most individuals typically think of stress as provoking heightened rather than reduced reactivity, reductions in PNS activity actually function to promote mobilization of energy and attention toward the task at hand because the PNS normally has an inhibitory influence on heart rate. Hence, suppressing this inhibitory influence produces a rapid and efficient increase in heart rate *without* requiring energy-costly SNS mobilization. Vagal suppression (indexed as baseline-to-task *reduction* in RSA) has been observed in numerous studies of children, adolescents, and adults engaged in stressful or affectively negative tasks (Beauchaine 2002; Beauchaine et al. 2001; Pieper et al. 2007), and studies suggest that individuals with greater reductions in RSA during stress have more adaptive patterns of emotional and interpersonal functioning (El-Sheikh and Buckhalt 2005; El-Sheikh and Whitson 2006; Hessler and Katz 2007; Huffman et al. 1998; Moore and Calkins 2004).

Yet it is not quite appropriate to characterize reduced RSA as "the" singular adaptive pattern of PNS activity during stress, given that an increasing body of research has found that PNS activity sometimes *increases* during stress (producing an increase in the inhibitory effect of the PNS on heart rate), especially in tasks that call for active *regulatory effort* (Beauchaine 2001; Kettunen et al. 2000; Segerstrom and Nes 2007; Thayer and Lane 2000). It has been theorized that this pattern (often denoted "vagal engagement" in contrast to "vagal suppression/withdrawal") may serve to facilitate attention and vigilance to environmental demands by slowing down cardiovascular activity. Hence, both vagal suppression and vagal engagement might be viewed as adaptive stress responses, albeit in different contexts, and researchers have not yet identified a stable set of criteria by which we might view one

pattern or the other as "more appropriate." In addition to situational variation in PNS responses to stress, there also appear individual differences. Katz (2007) has argued that individuals exposed to *chronically* stressful environments may develop an *enduring* pattern of vagal engagement which may help them to monitor their environment and maintain control over their emotions and behavior. Yet although such a pattern might prove adaptive in the short term, it may prove taxing over the long term. Many researchers have begun to conceptualize regulatory capacity as relatively finite, analogous to a muscle that tires upon repeated use (Muraven and Baumeister 2000). Hence, individuals who show chronic patterns of increased RSA in response to stress might experience chronic regulatory "fatigue," leaving them vulnerable to frequent failures of self-control (Vohs et al. 2005). This may explain why individuals who show heightened RSA during stress show multiple indices of emotion dysregulation, such as depression, anxiety, and hostility (Hessler and Katz 2007; Neumann et al. 2004; Vella and Friedman 2007). It also bears noting that heightened RSA *during stress* is independent of—and appears to have quite different psychosocial correlates—than heightened RSA *during rest* (i.e., vagal tone). Hence, although the extant literature consistently suggests that high vagal *tone* is adaptive, the nature of stress-induced vagal engagement versus suppression remains a topic of ongoing debate and research.

Implications for Adult Attachment Functioning

How do these biologically based deficits in HPA and ANS functioning influence adult attachment functioning? Currently, there are no long-term longitudinal studies testing associations between childhood profiles of HPA and ANS reactivity and adults' feelings and behaviors in their romantic attachments. Yet as reviewed earlier, individuals with highly stress-sensitive ANS and HPA systems (due to early caregiving deficits, genetic factors, or their combination) are likely to manifest chronic difficulties regulating stress and negative emotions. These difficulties, in turn, may impede not only the quality of adults' attachment bonds, but their basic propensity to *form* such bonds. This is due to the fact that normative processes of attachment formation, at all stages of life, depend critically upon repeated experiences of comfort-seeking and security-provision via contact with the attachment figure. It is through repeatedly seeking contact with the attachment figure when distressed, and experiencing downregulation of distress as a result of this contact (and, in adulthood, through participation in specifically comforting and supportive behaviors) that individuals come to develop a conditioned, internalized representation of the attachment figure as a fundamental base of security.

Hence, individuals whose HPA and ANS systems show chronic difficulties with stress regulation, and who may consequently fail to experience rapid and reliable downregulation of stress as a result of contact with their romantic attachment partner, may be slower to internalize the partner as a fundamental base of security, and in some cases may never do so. Furthermore, their *partners* may have difficulties

drawing emotional security from *them.* Previous research suggests that individuals with stress-regulatory deficits are often poor providers of support and security, since their own unregulated emotions can interfere with their capacity to accurately perceive and sensitively respond to the emotions of others, particularly during times of stress (reviewed in Diamond et al. 2012). Hence, because adult attachment formation and maintenance depends on repeated experiences of receiving *and* providing emotional security during major and minor stressors, individuals with biologically mediated deficits in emotion and stress regulation may show deficits in basic adult attachment dynamics.

The dual nature of these deficits (i.e., difficulties providing and benefiting from care and support) are particularly important, given that the bidirectional nature of adult attachment bonds is a fundamental characteristic of such ties which distinguishes them from infant/caregiver attachments. Specifically, whereas parents provide emotion regulation for their children but do not seek it in return, longstanding romantic partners have reciprocal influences on one another's emotional states, through a series of intentional and unintentional processes denoted as "coregulation" (reviewed in Butler 2011; Sbarra and Hazan 2008). This is not to say that the ties between caregivers and infants are wholly unidirectional, and that they do not, as well, show reciprocal coregulaton. Yet the asymmetrical nature of infant/caregiver attachment, and the immature state of the infant's physiological, cognitive, and affective systems, suggests that the chains of influence from caregiver to infant—especially when informed by conscious intent, such as when the caregiver attempts to soothe the child—are more robust and developmentally salient than vice versa.

Coregulation in the context of adult attachment can be broadly defined as a state of mutual interconnectedness in which both partners in an adult attachment relationship provide subtle forms of regulatory scaffolding for one another. Research has provided consistent evidence for emotion coregulation in cohabiting couples, most typically in the form of one partner's emotions or physiological responses influencing the other's, either through specific disclosures or through simple time spent together in close proximity (Butner et al. 2007; Helm et al. 2012; Hicks and Diamond 2008; Reed et al. 2012; Saxbe and Repetti 2010; Schoebi 2008; Semin and Cacioppo 2008). Not only does effective coregulation provide a potential pathway through which well-functioning attachments influence individuals' physical and mental health (Diamond and Fagundes 2012), but Sbarra and Hazan (2008) have suggested, expanding on the seminal work of Hofer (1984), that the establishment of coregulation may in fact be a distinguishing characteristic of attachment bonds that distinguishes them from other affiliative ties. Accordingly, to the degree that individual differences in ANS and HPA reactivity to stress may interfere with the establishment of coregulation or bias its functioning (such that one partner has a greater influence on the other than vice versa, or such that emotional states coordinate in a manner that facilitates escalation rather than attenuation of negative affect), basic processes of attachment formation and functioning may be disrupted, potentially interfering with the ability of the bond to provide a reliable base of security for each partner.

The quality of the bond is also likely to suffer, due to the critical role of ongoing mutual emotion regulation in the course of effective relationship functioning. For example, effective maintenance of a well-functioning romantic relationship, in the face of day-to-day stress and conflict, requires inhibiting impulses to reciprocate negative affect or behavior, and sometimes even constraining one's own expressive behavior in order to avoid escalation; it requires active efforts at maintaining positive, relationship-enhancing interpretations of partner behavior; it requires periodically sacrificing one's immediate needs in the service of compromise and forgiving the partner's transgressions. Accordingly, researchers have increasingly acknowledged the importance of self-regulation and self-control in romantic relationship functioning (Finkel and Campbell 2001; Halford et al. 2007). Finkel and Campbell, for example, demonstrated that dispositional capacities for self-regulation are critical to couples' abilities to engage in *accommodation* (Rusbult et al. 1991), i.e., to respond constructively to one another's potentially destructive behavior and to resist reciprocating and escalating negative affect. As they noted, "all partners in romantic relationships behave badly at times…. should the nonoffending individual act on the self-interested, gut-level impulse to 'fight fire with fire,' or should he or she instead resist the temptation to retaliate, choosing to behave in a constructive, prorelationship manner?" (p. 263). Accommodation might take the form of apologizing to a partner, forgiving him/her for a transgression, introducing humor or affection into a potentially difficult interaction, or simply "letting go" of a complaint or a perceived slight. Accommodation is important because it can *interrupt* potentially negative chains of interaction, ensuring that periodic transgressions remain periodic (Arriaga and Rusbult 1998; Gottman 1993; Gottman and Levenson 1992; Rusbult et al. 1998). Yet it is not easy: Most individuals' immediate responses to negative interpersonal behavior tend to be self-centered, self-protective, and potentially destructive (Thibaut and Kelley 1978), and individuals must draw upon self-regulatory resources in order to consistently inhibit these impulses (Finkel and Campbell 2001; see also Robins et al. 2000). Not surprisingly, deficits in emotion regulation have been related to maladaptive aggression in intimate relationships (Marshall et al. 2011; McNulty and Hellmuth 2008; Shorey et al. 2011a; Shorey et al. 2011b).

Notably, heightened physiological reactivity may render self- and emotion-regulation in intimate relationships more difficult. Studies of HPA reactivity in particular have found that the excessive amounts of cortisol produced by HPA hyperreactivity to stress have detrimental effects on in situ cognitive processing, executive functioning, attention, and memory (Abercrombie et al. 2003; Cahill et al 2003; Lupien et al. 1994; McEwen et al. 1992) in a manner that may immediately interfere with the adoption of adaptive interpersonal strategies in the face of stress and threat. Hence, in the face of either generalized or relationship-specific stress (such as conflict), individuals with heightened HPA activity may prove less able to identify, select, and implement mature problem-solving approaches that call for heightened impulse control, self-awareness, and self-regulation, and might more quickly resort to more primitive, defensive, aggressive, and escapist approaches. Over time, the repeated adoption of such maladaptive coping strategies can introduce significant

strains and burdens on one's romantic relationship, potentially leading to dissolution.

This scenario (in which deficits in biologically based capacities for emotion regulation have implications for adult romantic relationship functioning) applies to ANS functioning as well. Numerous researchers have highlighted the specific relevance of tonic PNS activity (vagal tone) for *social* functioning, given the critical role of affective and behavioral regulation in guiding children's reactivity and responsiveness to increasingly complex social situations as they mature (Beauchaine 2001). For example, researchers investigating links between vagal tone and tonic positive affect (Oveis et al. 2009) have argued that the positive and flexible disposition of individuals with high vagal tone may serve as a relational "building block" which promotes approach-oriented behavior and fosters social resources (following Fredrickson 1998). In contrast, the interpersonal hostility and defensiveness associated with low vagal tone (Brosschot and Thayer 1998; Demaree et al. 2004; Movius and Allen 2005; Sloan et al. 1994) directly hinders interpersonal functioning.

Supporting this view, one recent study (Diamond et al. 2011) found that individual differences in PNS regulation related to couples' day-to-day interpersonal behavior. Specifically, men with higher vagal tone (indexed by higher baseline RSA, and suggestive of more robust emotion regulation) were rated by their female partners as interacting more positively (i.e., expressing more connectedness and understanding) over 3 weeks of day-to-day assessment than were men with lower vagal tone. This study also found that on days when men with either low vagal tone (i.e., low baseline RSA) or high vagal reactivity (i.e., baseline-to-task increases in RSA in response to stress) reported high negative affect, their female partners described them as more critical and argumentative. Yet this was not the case for men with high vagal tone or low vagal reactivity. As for women, those with higher vagal tone interacted more positively with their partners (according to their partners' reports) on days when the women reported greater positive affect. This effect was not observed among women with lower vagal tone. These findings may suggest that women with higher vagal tone are better able to mobilize positive affect in the service of sensitive and responsive interactions with their romantic partners, or they might suggest that women with higher vagal tone experience stronger positive emotions *in response* to positive interactions with their romantic partners. Either interpretation is consistent with the notion that robust vagal regulation, potentially established by individuals' earliest interactions with caregivers, may allow adults to adaptively regulate negative emotions, foster positive emotions, and sustain adaptive interpersonal interactions with their romantic partners in the face of day-to-day stressors.

Directions for Future Research

Up until now, I have emphasized normative attachment processes such as the basic provision of safety, security, and mutual caregiving and coregulation. Yet there is, of course, an extensive body of research on individual differences in attachment

security, commonly known as *attachment styles*. One promising direction for future research would involve bridging the conventional gap between research on normative attachment processes and research on individual differences in attachment styles by investigating how the (normative) activity of the HPA and ANS in response to stress contributes to the development and maintenance of adult attachment styles. Hence, although much adult attachment research has focused on the consequences of individual differences in attachment style for multiple aspects of couple functioning, an equally important question concerns the normative biobehavioral attachment processes that *give rise* to variation in adult attachment styles, and what these processes tell us about the total psychobiological complex of the attachment system.

Historically, attachment styles were conceived as trait-like expectations concerning the responsiveness of attachment figures, established through early infant/caregiver interactions, which function as mental prototypes for future relationship experiences (Ainsworth et al. 1978). Over the years, however studies have increasingly shown a key role for attachment styles in organizing the encoding, storage, retrieval, and manipulation of information related to affective states (Shaver and Mikulincer 2007), and attachment styles are now generally viewed as influencing not only interpersonal functioning, but psychosocial approaches to stress and to negative emotions more generally (reviewed in Mikulincer and Florian 2004; Nolte et al. 2012).

According to attachment theory, infants who did not receive adequate "external" emotion regulation from their attachment figures came to rely on secondary—and suboptimal—regulatory strategies. Specifically, individuals with high attachment *anxiety* have been found to maximize the experience and expression of negative affect, to be hypervigilant to threat cues, and to show patterns of spreading emotional reactivity such that one negative thought or memory triggers many others (Shaver and Mikulincer 2002). Individuals with high attachment *avoidance*, to the contrary, tend to minimize experiences of negative affect and to direct attention away from threat cues (Mikulincer et al. 2003). These "deactivating" strategies involve the denial or suppression of affective experience, the inhibition of affective expression, and distortion of encoding of affective experiences (Becker-Stoll et al. 2001; Kobak et al. 1993; Mikulincer et al. 2003). Importantly, both types of attachment insecurity are associated with the inability to derive emotion regulating benefits from contact with attachment figures. (Feeney 1999).

The cognitive, behavioral, affective, and interpersonal consequences of these two divergent strategies have received extensive attention over the years (reviewed in Mikulincer and Shaver 2008). Yet research has increasingly investigated their physiological manifestations. After all, if sensitive and responsive caregiving is responsible *both* for the development of attachment security *and* for the calibration of the stress regulation functions of the HPA axis and the ANS, one might expect to find that individual differences in attachment style relate directly to the functioning of these systems. Some research supports this view: Specifically, children with insecure patterns of attachment show heightened HPA responses to acute stress (Hertsgaard et al. 1995; Nachmias et al. 1996; Spangler and Grossman 1993; van

Bakel and Riksen-Walraven 2004), and a recent study found that attachment insecurity in adolescence is linked to heightened basal levels of cortisol (Oskis et al. 2011). As for adults, Quirin et al. (2008) found that adults' attachment anxiety was associated with heightened HPA reactivity to a standardized laboratory stressor, and both attachment anxiety and avoidance have been associated with lower vagal tone (Diamond and Hicks 2005; Maunder et al. 2006).

A shortcoming of this emerging body of research is that most studies investigate HPA or ANS functioning in response to relationship-specific rather than generalized stressors, and correspondingly interpret the physiological results as indicative of anxious or avoidant individuals' emotional responses to relationship events. Yet the model I have outlined in this chapter suggests that anxious and avoidant individuals may have *started out in childhood* with particularly reactive HPA or ANS systems, which may have developed due to deficits in early caregiving, genetic factors, or a combination of the two, and that this heightened reactivity may have contributed to the development of their attachment anxiety and avoidance. Yet research paradigms assessing associations between attachment style and physiological reactivity to relationship-specific stressors cannot reliably disentangle "pre-relationship" patterns of HPA/ANS reactivity from "relationship-triggered" HPA/ANS reactivity. For example, Powers et al. (2006) found that insecurely attached individuals showed greater HPA reactivity to (laboratory-induced) romantic conflict than did securely attached individuals. Specifically, attachment avoidance in female participants was related to increased HPA reactivity to couple conflict, whereas men showed elevated reactivity if they had high levels of anxiety combined with high avoidance. Individuals with high attachment anxiety also showed poorer recovery of HPA levels after the conflict (Laurent and Powers 2007). Dewitt and colleagues (Dewitte et al. 2010) led individuals to believe that they were going to have to watch a tape of their partner being interviewed about previous sexual and romantic relationships by an attractive opposite-sex experimenter. Participants with higher attachment anxiety showed heightened HPA reactivity while anticipating this stressful event, with the highest increases found among women with high anxiety *and* avoidance. Finally, during an *actual* 4–7-day physical separation from their romantic partner, individuals with high attachment anxiety showed tonically elevated levels of cortisol (Diamond et al. 2008).

All of these findings are consistent with the notion that anxiety is associated with a lower threshold for attachment-related threats (conflict, partner unavailability, jealousy, etc.), which manifests itself in a heightened physiological response. Yet it is also possible that, as outlined in this chapter, insecurely attached individuals possessed a *generalized* predisposition for heightened HPA or ANS stress reactivity, to relationship-specific as well as other stressors, and that this heightened reactivity contributed to the development of their attachment insecurity over the course of their development. This interpretation is consistent with the limited body of findings cited above on associations between attachment security and overall patterns of ANS and HPA functioning (Diamond and Hicks 2005; Maunder et al. 2006; Oskis et al. 2011; Quirin et al. 2008), but considerably more research is needed. In particular, future research should conduct comprehensive comparisons between anxious

and avoidant individuals' physiological, cognitive, and emotional reactivity to relationship-related stressors *as well as* generalized stressors, at multiple points in time, in order to try and determine the degree to which patterns of reactivity reflect stable features of individuals (which "travel" from relationship to relationship and from situation to situation) versus the degree to which they reflect features of individuals' current relationship experiences (i.e., the degree of hostility or avoidance or support in this particular interaction). Such studies have enormous potential to contribute to our understanding of the complex links between early attachment experiences, biologically based patterns of emotion regulation, and adult attachment functioning.

Finally, one of the most intriguing areas for future research concerns the role of the HPA and the ANS systems in potentiating *plasticity* in attachment style and basic attachment-related interpersonal functioning over the lifespan. Historically, research on links between ANS and HPA functioning and socioemotional development adopted a diathesis-stress or "dual-risk" perspective, positing that children with heightened ANS and HPA responses to stress were disproportionately vulnerable to stressful rearing environments, due to their basic deficits in stress regulation (reviewed by Boyce and Ellis 2005). Yet recently an alternative perspective has emerged, denoted *differential susceptibility* (Belsky et al. 2007; Belsky and Pluess 2009; Del Giudice et al. 2011; Ellis et al. 2011) positing that the same factors that render children highly sensitive and reactive to negative environments also render them highly sensitive and reactive to positive environments. Hence, both dual-risk and differential susceptibility perspectives predict that children with certain patterns of ANS functioning will show disproportionately negative outcomes in negative environmental contexts (as shown by Boyce et al. 1995; Bubier et al. 2009; Cummings et al. 2007; El-Sheikh et al. 2007, 2009; Katz 2007). However, the differential susceptibility perspective additionally predicts that these children will show disproportionately positive outcomes in positive environments (reviewed in Belsky et al. 2007; Belsky and Pluess 2009), or might particularly show improvements in general socioemotional functioning when their environmental input changes from bad to good, due to the fact that they may be better able to "absorb" developmental strengths and skills from highly nurturant environments than their low-reactive counterparts.

Although most research testing the differential susceptibility model has focused on environmental inputs experienced during childhood (reviewed in Belsky and Pluess 2009), some researchers have argued that environmental inputs experienced at later ages also play a potentially important role. Specifically, Del Giudice et al. (2011) have argued that there may be additional "windows" of heightened susceptibility which open up during later developmental transitions, such as puberty (Del Giudice et al. 2011), which function to ensure that individuals adjust appropriately to the key environmental ingredients relevant to their changing developmental tasks. During adolescence, of course, mating and reproduction emerge as newly important life tasks, and one of the hallmarks of this transition involves the normative transfer of emotional attachment from parents to romantic partners (Diamond and Fagundes 2008; Hazan and Zeifman 1994; Trinke and Bartholomew 1997).

Most existing research on youths' first significant romantic attachments has focused on the degree to which the quality of these ties can be predicted from youths' preexisting interpersonal skills and deficits (see reviews in Crouter and Booth 2006), but little research has specifically investigated whether youths' first substantive romantic attachments have a particularly formative role with respect to adult attachment style. In light of the differential susceptibility model, one possibility is that youths' entry into mature adult attachments represents a normative window of heightened sensitivity to environmental input, *and* that highly reactive individuals will be disproportionately sensitive to these experiences, rendering their early romances particularly influential on their developing attachment cognitions and behaviors. For example, highly reactive youths whose first substantive romantic attachments are extremely positive and supportive might be particularly likely to develop robust interpersonal and self-regulatory skills as a result, even if they *began* these relationships with compromised skills. Moving forward, their improved social and regulatory capacities may improve the quality of their future adult attachment bonds, fostering an enduring sense of security and potentially attenuating the levels of anxiety and avoidance that they may have started with.

In contrast, highly reactive individuals whose first substantive ties are troubled, conflictual, and unsupportive might be particularly likely to become highly anxious or avoidant *as a result*, and to settle into maladaptive patterns of interpersonal behavior and emotion regulation that impede their abilities to form and sustain nurturant adult attachment bonds. Hence, although early appearing profiles of biological stress-regulation are likely to have important and enduring influences on attachment formation and functioning, these influences are not theorized as deterministic. Rather, the seeds for change in attachment-related skills and capacities may be a fundamental component of these profiles, and may be why they have been fundamentally preserved in our species as engines for lasting developmental adaptation.

Conclusion

The increasing body of psychobiological research on attachment and emotion regulation underscores the critical role of attachment relationships in fostering psychological, physical, and interpersonal functioning at all stages of the life course. Understanding the role of early attachment relationships in calibrating critical stress-regulatory systems, and the long-term implications of these systems for emotion regulation and interpersonal functioning, provides important new ways to understand the fundamental linkages between infant–child attachment and adult attachment, and potentially to bridge the long-standing bifurcation between these two separate research traditions. The development of integrative, lifespan, biobehavioral models of the attachment system should be a priority for future research, and greater emphasis on the emotion regulation functions of attachment, as they are manifested in different types of social and interpersonal challenges at different stages of life, can make an important contribution to this goal. After all, emotion regulation within

attachment relationships is not a developmental task to be mastered at a certain age (after which attention turns to the psychological and behavioral implications of one's relative success or failure at this task), but rather a "moving target" that is continually sensitive to changing goals and contexts. The quality of individuals' parental attachments clearly has critical implications for both subjective and physiological aspects of emotion regulation and hence long-term interpersonal functioning, opening up a host of fascinating questions regarding the basic biopsychology of the attachment system, its multiple manifestations, and its potential developmental changes over the lifespan. Addressing these questions can help to integrate the increasingly sophisticated bodies of knowledge on social relationships and multiple domains of mental and physical functioning which have developed within the social-psychological, developmental, and behavioral medicine traditions—such an integration is critical for elucidating how and why attachment bonds play such a fundamental role in well-being over the life course.

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