

Contributions of Psychophysiology to Research on Adult Attachment: Review and Recommendations

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Despite the increasing use of psychophysiological measures to investigate social and interpersonal phenomena, few studies of adult romantic attachment have taken advantage of this approach. In this article I argue for a biologically-specific, theory-based integration of psychophysiological measures into adult attachment research. This approach would help elucidate the normative psychobiological properties of the attachment system, which have received little study in humans. Specifically, it would allow researchers to test targeted hypotheses regarding affect and arousal regulation in attachment relationships. I provide a general introduction to 2 biological systems that hold particular promise for adult attachment research: the parasympathetic branch of the autonomic nervous system and the hypothalamic-pituitary-adrenocortical axis of the endocrine system. I highlight the relevance of these systems for attachment phenomena and review findings from selected social psychophysiological research. I conclude by outlining a tentative theoretical model of the psychobiology of adult attachment and identifying specific directions for future research.

The notion that romantic relationships are adult “versions” of infant and caregiver attachment bonds has generated hundreds of studies since it was first postulated over a decade ago (Hazan & Shaver, 1987; Shaver, Hazan, & Bradshaw, 1988). Nearly all of these studies have relied exclusively on self-reports, observational methods, or both. Although psychophysiological measures have been used in studies of infant and caregiver attachment (reviewed in Fox & Card, 1999), they have not generally been applied to adult attachment research. The small number of adult attachment studies that have integrated psychophysiological measures (Carpenter & Kirkpatrick, 1996; Feeney & Kirkpatrick, 1996; Fraley & Shaver, 1997; Mikulincer, 1998) have used them as objective indexes of emotional arousal and have not considered deeper questions about the basic psychobiological properties of adult attachment bonds (discussed by Reite & Boccia, 1994).

These deeper questions have received increasing attention in recent years. Animal research has made notable strides in understanding the basic biopsychology of mammalian attachment and its underlying neural circuitry (for comprehensive reviews from diverse perspectives, see Carter, Lederhendler, & Kirkpatrick,

1997). Yet this biopsychological approach has not generally been extended to studies of human attachment. This is not to say that research on human social relationships has ignored physiological processes. To the contrary, social, health, and clinical psychologists have conducted hundreds of studies investigating physiological responses to social interactions between strangers, friends, family members, and spouses (e.g., Gottman & Levenson, 1992; Levenson & Gottman, 1983; Uchino, Cacioppo, & Keicolt-Glaser, 1996). Yet, because such studies were not designed to address basic questions about affectional bonding, the implications of their findings for research on the biopsychology of adult attachment have gone largely unexplored.

In this article I directly address these implications and make the case for a more biologically specific, theory-based integration of psychophysiology into adult attachment research. I begin by outlining the functional properties of the attachment system and then focus on two specific biological systems that have been found to be associated with attachment-related phenomena in prior social psychophysiological research: the autonomic nervous system (ANS) and the hypothalamic-pituitary-adrenal (HPA) axis of the endocrine system.

These are certainly not the only biological systems related to attachment. For example, there is mounting evidence based on animal research that the neuropeptides oxytocin and vasopressin play particularly important roles in mammalian attachment forma-

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tion (Carter, 1998), along with endogenous opioids and catecholamines (Nelson & Panksepp, 1998). However, I will touch on these systems only briefly, primarily because there are already several comprehensive reviews available that explicitly address their relevance for attachment processes (Carter et al., 1997; Insel, 2000). Furthermore, these systems are more difficult to study in humans than are ANS and HPA activity, which explains why nearly all extant research on the neuroendocrinology of attachment has been conducted with animals.

For this reason, a review of attachment-relevant research on ANS and HPA activity in humans appears more immediately useful to researchers interested in exploring the psychophysiology of attachment. I provide a basic introduction to the ANS and the HPA axis for the benefit of researchers new to psychophysiology and review research findings highlighting their roles in attachment processes. Based on these findings, I lay out a preliminary model outlining how these two systems may be specifically implicated in both normative attachment processes and individual differences in attachment. I conclude by pinpointing specific questions for future investigation. Because there is insufficient space to address the numerous theoretical, conceptual, and methodological issues concerning the appropriate use of physiological measures in social-psychological research, readers new to these topics should consult the excellent treatments of them that appear elsewhere (Blascovich, 2000; Cacioppo, Tassinary, & Berntson, 2000).

Attachment Theory: Basic Concepts

Bowlby (1958, 1982) conceptualized attachment as an evolved behavioral system designed to regulate infants' proximity to a protector and thereby maximize chances for survival. Because human infants are born developmentally immature relative to the infants of other species, they require a prolonged period of vigilant care and feeding following birth to ensure survival. The attachment system is activated whenever the child experiences distress or perceives danger. This results in immediate attempts to gain proximity to the attachment figure, whether through crying, reaching, following, or clinging. In normative cases, contact with the attachment figure soothes the infant, allowing him or her to continue exploring the environment.

Although any competent adult can provide care for a child, attachment theory posits that infants usually develop a unique, exclusive, and irreplaceable relationship with one, or only a few, primary caregivers, such that these individuals become preferred targets for security-seeking. These preferences develop gradually, as a result of repeated intimate interactions with

caregivers. However, the fact that the infant preferentially seeks security from his or her caregiver does not mean that the caregiver consistently provides it. Rather, some infants fail to derive consistent distress-alleviation and emotional security from their caregivers, leading them to develop insecure patterns of attachment (addressed in more detail later). Yet all such infants succeed in forming attachments, even if the quality of these relationships is low.

Normative attachments are characterized by the presence of four distinct forms of behavior: seeking and maintaining physical closeness to the attachment figure ("proximity seeking"); turning to the attachment figure for comfort and reassurance ("safe haven behavior"); experiencing distress as a result of separations from the attachment figure ("separation distress"); and using the attachment figure as a reliable, dependable base of support from which to explore the world ("secure base behavior"; Ainsworth, Blehar, Waters, & Wall, 1978; Bowlby, 1982). These four components also characterize adult romantic relationships, and thus these relationships have been conceptualized as adult attachment bonds serving the same security-provision functions as infant and caregiver attachments (arguments for and against this view are reviewed in Hazan & Zeifman, 1999, and Kirkpatrick, 1998).

Attachment as Regulation

In recent research, the security-provision and distress-alleviation functions of attachment relationships have been increasingly conceptualized in terms of *affect and arousal regulation* (Brennan & Shaver, 1995; Feeney, 1995; Field, 1991; Field & Reite, 1984; Kobak & Sceery, 1988; Reite & Boccia, 1994; Simpson, Rholes, & Nelligan, 1992). These processes involve the modulation of emotional and physiological reactivity to external and internal stimuli so that individuals can respond appropriately to the demands of everyday life and accomplish their goals (Porges, Doussard-Roosevelt, & Maiti, 1994; Thompson, 1994). Although both positive and negative feeling states are subject to regulation, negative feeling states have received the most attention from attachment researchers, given that distress-alleviation is a core function of the attachment system (Bowlby, 1958, 1982). However, the role of positive emotions in day-to-day functioning should not be overlooked. Specifically, enhancement of positive feeling states has been suggested as one of the mechanisms through which negative feeling states are managed (Fredrickson, 1998; Fredrickson & Levenson, 1998). Thus, affect and arousal regulation should be broadly conceptualized as involving the modification of both positive and negative emotions and their physiological correlates.

Although affect and arousal regulation are often discussed as processes that originate and terminate within the individual, they are also accomplished through interactions with others. In infancy, distress-alleviation is typically provided by direct contact with caregivers (Spangler & Grossman, 1993), but children and adolescents gradually acquire the ability to regulate their own responses to distress over the course of development (Thompson, 1994), typically through strategies such as attention shifting, approaching or avoiding certain stimuli, emotion-focused coping, and physical self-comforting (Derryberry & Rothbart, 1988; Kobak, Cole, Ferenz-Gillies, Fleming, & Gamble, 1993). Nonetheless, social partners continue to play an important role in distress-alleviation throughout the life course by communicating empathy, providing comfort, extending a listening ear, providing an alternative perspective, making us laugh, or simply distracting us from our problems (Gross & Munoz, 1995; Thompson, 1994). These functions have been conceptualized in both the social psychology and behavioral medicine literatures as *social support*. Thus, notions of “security provision,” “social support,” and “affect and arousal regulation” share a common emphasis on the attenuation (and in some cases prevention) of emotional and physiological responses to distress through social contact.

Yet despite this functional overlap, attachment and social support are not quite the same thing. Most notably, a range of different partners might provide social support, whereas attachments are inherently selective social ties in which the attachment figure is considered fundamentally unique and irreplaceable. Consistent with this formulation, studies of bereavement (Stroebe, Stroebe, & Abakoumkin, 1999; Stroebe, Stroebe, Abakoumkin, & Schut, 1996) have found that the loss of an attachment figure brings about distinct psychological deficits that cannot be attributed solely to the loss of partner support and that cannot be remedied by drawing on ancillary sources of support. Thus, whereas attachment figures are important and often preeminent support providers (Antonucci & Jackson, 1987; Cassidy, 1994; Hazan & Zeifman, 1994; Thompson, 1994; Trinke & Bartholomew, 1997), their influence on psychosocial and physical functioning should be considered independently from that of other support providers (Berlin & Cassidy, 1999).

Normative Versus Individual-Difference Perspectives on Attachment

As Simpson and Rholes (1998) elucidated, attachment theory has two main components: a normative component specifying species-typical patterns of

affectional bonding and an individual-difference component specifying variations on these patterns. In the social and personality literature, these variations are called *attachment styles* and are conceived as trait-like expectations concerning the responsiveness of attachment figures (Ainsworth et al., 1978; Hazan & Shaver, 1987). Ainsworth and her colleagues identified three attachment styles that, for clarity, are here described as prototypes. *Secure* infants experienced sensitive, responsive caregiving, and as a result they expect their attachment figures to be dependable sources of comfort and emotional security in times of distress. *Anxious and ambivalent* infants experienced inconsistent caregiving, and as a result they do not feel secure in the knowledge that their attachment figure will be available and responsive to their needs in times of distress. *Avoidant* infants did not receive adequate, sensitive care from their attachment figure and may have been directly rebuffed in their bids for close contact and security. As a result, they learned not to seek such contact when distressed.

Hazan and Shaver (1987) argued that the same basic attachment patterns also influence feelings and behaviors toward adult attachment figures—that is, romantic partners. For example, just as avoidant infants resist contact with caregivers when distressed, adults with an avoidant romantic attachment style resist contact with romantic partners when distressed (Simpson et al., 1992). More than a decade of research supports the notion of stable adult attachment styles (reviewed in Simpson & Rholes, 1998), although the precise number of these styles and the extent to which they are carried forward from infant and caregiver styles remains debated (Weinfield, Sroufe, Egeland, & Carlson, 1999). Also, researchers have increasingly conceptualized these styles as regions in a two-dimensional space (anxiety-by-avoidance) and have therefore gravitated toward assessing continuous variation in both anxiety and avoidance rather than sorting individuals into discrete categories (Brennan & Shaver, 1995).

Whereas both childhood and adult attachment styles were originally viewed as cognitive and affective expectations regarding the availability of attachment figures, this view eventually broadened to incorporate the role of attachment relationships in affect and arousal regulation. Specifically, attachment styles came to be conceptualized as distinct *capacities* and *strategies* for affect and arousal regulation (Kobak & Sceery, 1988; Mikulincer & Florian, 1998), and researchers have found extensive support for this view (Kobak et al., 1993; Mikulincer & Florian, 1998; Rholes, Simpson, & Orina, 1999; Simpson et al., 1992). For example, insecurely attached individuals have difficulty regulating negative affect and do not effectively learn regulatory skills from other sources (Kobak, Sudler, & Gamble, 1991; Rosenstein &

Horowitz, 1996), whereas securely attached adults report more frequent positive emotions (Simpson, 1990) and engage in more support-seeking with romantic partners (Simpson et al., 1992).

Unanswered Questions

There has been substantially more research on the individual-difference component of attachment than the normative component (Berlin & Cassidy, 1999; Marvin & Britner, 1999; Simpson & Rholes, 1998). As a result, many basic questions about the nature and ontogeny of the attachment system remain unanswered (however, see Hazan & Diamond, 2000; Hazan & Zeifman, 1999). For example, through what mechanisms do new romantic relationships develop into enduring attachment bonds? How long does it take, and what factors moderate this process? Without knowing the answers to these questions, how can adult attachment researchers establish whether the romantic couples in their samples are, in fact, even attached to each other to begin with (a question that is rarely explicitly addressed in the adult attachment literature)?

Accordingly, researchers have increasingly called for more systematic investigation of the psychological, interpersonal, and physiological processes underlying normative attachment formation and functioning (Fraley & Shaver, 2000; Main, 1999; Marvin & Britner, 1999). This nascent shift in emphasis has been fueled by the growing body of animal research, cited previously, that has demonstrated that mammalian attachment processes have clear-cut neurobiological substrates (Carter, 1998; Insel, 2000; Nelson & Panksepp, 1998), the human analogs of which remain largely unexplored (for exceptions, see Fleming, Ruble, Krieger, & Wong, 1997; McCarthy & Altemus, 1997).

Given our increasingly sophisticated understanding of the interconnections between human psychological and biological processes (Berntson & Cacioppo, 2000), social psychophysiology has much to contribute to these questions. Yet for social psychophysiological research to yield meaningful information about these processes, researchers should focus on those biological systems that are demonstrably (or at least plausibly) related to specific attachment phenomena. Furthermore, in addition to using psychophysiological measures to index the conscious or unconscious emotional “output” of the attachment system, such measures should also be used to elucidate the psychobiological processes underlying these outputs. In this manner, researchers can begin to address basic questions about the process of attachment formation; the state of “being attached”; and the mechanisms through which individual differences in attachment style shape adults’ social cognitions, emotions, and behaviors. Toward this end, two of the

best candidates for study are the ANS (particularly the parasympathetic branch) and the HPA axis of the endocrine system. I provide an introduction to the basic functioning of each system and review research highlighting their potential roles in attachment processes.

The ANS

When most people think of the physiological changes brought about by psychological stress, they think of the classic “fight-or-flight” response with its well-known manifestations of increased heart rate, blood pressure, and sweat production. These changes are part of a larger syndrome of physiological changes 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.

Countless social psychological studies have assessed components of the fight-or-flight response to measure individuals’ reactivity to psychological stress. Measures of cardiovascular function such as heart rate and blood pressure are most common (for a review of these processes, see Brownley, Hurwitz, & Schneiderman, 2000), along with measures of electrodermal activity, commonly known as skin conductance (reviewed in Dawson, Schell, & Filion, 2000). Stress-induced activation in these domains is often used as an index of generalized autonomic stress reactivity. Yet the notion of generalized stress reactivity has largely fallen out of favor because it overlooks the diverse patterns of up-regulation and down-regulation in the sympathetic and parasympathetic branches of the ANS that characterize different stress responses (Berntson, Cacioppo, & Quigley, 1991).

The parasympathetic and sympathetic branches of the ANS have antagonistic effects on the physiological processes involved in stress reactivity. Heightened activation of the sympathetic nervous system (SNS) produces the physiological changes most commonly associated with fight-or-flight responses: acceleration in heart rate, increased blood pressure, increased sweating, and so forth. In contrast, the parasympathetic nervous system (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 deactivation in the PNS, both of which function to redistribute metabolic resources to cope with the external threat. Poststress 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, meaning that a physiological index of “generalized arousal” such as heart rate acceleration is not so general after all. Rather, social psychophysicologists now generally conceptualize cardiovascular reactivity in terms of a two-dimensional “autonomic space” in which PNS and SNS activity vary along independent axes (Berntson et al., 1994; Cacioppo, Uchino, & Berntson, 1994). By decomposing any particular stress response into its independent SNS and PNS determinants, one can represent the response as SNS-dominated (i.e., more sympathetic activation than parasympathetic withdrawal), PNS-dominated (i.e., more parasympathetic withdrawal than sympathetic activation), or equally determined by both branches.

Assessing these differences is important for two main reasons. First, PNS withdrawal produces more rapid and flexible changes in heart rate than SNS activation (Berger, Saul, & Cohen, 1989; Spear, Kronhaus, Moore, & Kline, 1979), and thus PNS-dominated stress responses are generally considered more adaptive and efficient when it comes to day-to-day environmental demands. Furthermore, sustained SNS activation is associated with patterns of neuroendocrine and immunological response that have negative implications for long-term health (Cacioppo et al., 1995; Kamarck & Jennings, 1991). Accordingly, much attention has focused on determining which types of environmental demands typically elicit PNS-dominated versus SNS-dominated responses (e.g., Berntson, Cacioppo, & Fieldstone, 1996).

Second, there are stable individual differences in patterns of SNS and PNS functioning that emerge within and across different environmental demands (Allen & Crowell, 1989; Berntson et al., 1994; Cacioppo, Uchino, & Berntson, 1994). These individual differences have received increasing empirical attention, largely because they have shown associations with an array of other individual differences ranging from neuroendocrine and immune function (Uchino, 1995) to personality (Kamada, Miyake, Kumashiro, Monou, & Inoue, 1992; Schweiger, Wittling, Genzel, & Block, 1998) to clinical conditions such as depression and panic anxiety (Carney et al., 1988; Friedman & Thayer, 1998; Light, Kothandapani, & Allen, 1998) to “lifestyle” factors such as marital status and household size (Horsten et al., 1999). Thus, investigating individuals’ underlying patterns of autonomic reactivity across a variety of environmental demands, rather than just the magnitude of reactivity, elucidates multiple as-

pects of cognitive, affective, and physiological functioning that have direct relevance for physical and mental well-being.

To investigate such patterns, researchers have gravitated toward the use of psychophysiological measures that can differentiate between PNS and SNS activity. Of these measures, the most well-validated are preejection period (PEP) and respiratory-sinus-arrhythmia (RSA), respectively (Cacioppo, Uchino, & Berntson, 1994). RSA (discussed in greater detail later) represents the degree of beat-to-beat variability in heart rate attributable to respiration, with greater RSA representing greater parasympathetic engagement. PEP represents the time between ventricular depolarization and the ejection of blood into the descending aorta, with shorter PEPs representing greater sympathetic activation (Berntson et al., 1994; Cacioppo et al., 1994). Measurement of RSA requires sustained, fine-grained measures of heart rate that are analyzed with specially designed software. PEP can only be measured with impedance cardiography, a technique that measures systolic time intervals, cardiac output, and contractility in the cardiac muscle. Although impedance cardiography has become increasingly common in social psychophysiology, it might nonetheless be unfamiliar to the average psychologist. Thus, assessment of electrodermal activity—small changes in the electrical activity of the skin attributable to SNS-mediated sweat production—might prove a more accessible, albeit less precise, index of SNS activity.

Attachment and ANS Activity

As noted earlier, attachment relationships have been hypothesized to have a regulatory effect on individuals’ affective and physiological responses to distress (at least for those with secure attachment styles). If this is the case, one might expect the presence of attachment figures to attenuate stress-induced ANS activity. As it happens, this exact phenomenon (traditionally called *stress-buffering*) has been the subject of hundreds of psychophysiological studies published in the social psychological, behavioral medicine, and social neuroscience literatures (many of which are reviewed in Gardner, Gabriel, & Diekmann, 2000). Many of these studies have found that supportive social interactions attenuate individuals’ ANS reactivity (and often endocrine activity) to psychological stress (e.g., Gerin, Pieper, Levy, & Pickering, 1992; Kamarck, 1990; Lepore, Allen, & Evans, 1993), and this phenomenon has been proposed as a key mechanism through which social support promotes mental and physical health over the life span (Uchino et al., 1996).

Yet a number of studies have unexpectedly failed to find stress-buffering effects of social support (Allen,

Blascovich, Tomaka, & Kelsey, 1991; Sheffield & Carroll, 1994; Snydersmith & Cacioppo, 1992), and much attention has therefore focused on identifying the “boundary conditions” for such effects (Kamarck, Annunziato, & Amateau, 1995). For example, are they dependent on specific supportive behaviors such as verbal reinforcement, positive facial expressions, or comforting physical contact? Are they “canceled out” when the participant feels that he or she is being evaluated by the support provider (see especially Allen et al., 1991)?

One of the reasons it has been so difficult to disentangle these factors is that the average stress-buffering experiment ends up manipulating far more than psychological stress per se, sometimes intentionally and sometimes not. The types of tasks used to induce psychological stress vary in difficulty, controllability, and performance demands, and each of these factors bears its own pattern of associations with different forms of physiological reactivity. For example, both the level and the autonomic patterning of cardiovascular reactivity is influenced by (a) whether the task requires active effort, watchful attention, or passive participation (Smith, Ruiz, & Uchino, 2000; Wright, Tunstall, Williams, Goodwin, et al., 1995); (b) the incentive or penalty used to elicit task performance (Smith, Limon, Gallo, & Ngu, 1996; Waldstein, Bachen, & Manuck, 1997); (c) the difficulty and effort requirements of the task (Kamarck et al., 1998; Wright, Tunstall, Williams, Goodwin, & Harmon, 1995); and (d) the extent to which individuals can control their own performance and the administration of rewards and penalties (Peters et al., 1998).

Social psychophysicists have become increasingly sophisticated in identifying and manipulating these different determinants of physiological reactivity to isolate and test the effects they are interested in. Yet surprisingly, the same level of attention is less frequently devoted to the preexisting relationship between the participant and the support provider. Participants are typically asked to bring “a friend” or “a close friend” to the laboratory, or are “supported” by a total stranger. This leaves many questions about social support and relationship quality unanswered. For example, although some research findings (reviewed in Uchino et al., 1996) suggest that an individual’s most important relationships—such as spouses and family members—might be the most effective in moderating ANS reactivity to stress, this has not been definitively established.

In Uchino and colleagues’ (Uchino et al., 1996) review of laboratory studies of social support and ANS activity, they cited only one study in which researchers directly compared cardiovascular function in the presence of attachment (or attachment-like) figures versus other supportive ties. Using ambulatory blood pressure

monitors and self-report diaries, this study (Spitzer, Llabre, Ironson, Gellman, & Schneiderman, 1992) found that individuals had significantly lower blood pressure in the presence of family members than in the presence of friends or strangers. However, the authors noted a number of potential confounds complicating interpretations of this finding and highlighted the need for more research. Furthermore, several studies have found that the presence of supportive strangers is just as effective at buffering physiological stress reactivity as the presence of good (or best) friends, even when friends are subjectively judged as more supportive (Fontana, Diegnan, Villeneuve, & Lepore, 1999; Sheffield & Carroll, 1994; Snydersmith & Cacioppo, 1992). Research on animals, too, has found that, although attachment figures are preferentially sought out over other social partners by infants in distress, they are not necessarily more effective than other social partners in alleviating the physiological indexes of this distress (Polan & Hofer, 1999).

These conflicting findings highlight how much remains to be learned about the circumstances under which different types of relationships influence ANS reactivity to stress. In particular, they demonstrate that our thoughts and feelings about important social partners, our behavior toward them, and our corresponding physiological responses do not always concord in a predictable fashion. Identifying and investigating such disjunctures is important not only for understanding general attachment processes, but for exploring how these processes are moderated by individual differences in attachment security, a question that has already received some study using psychophysiological measures (Carpenter & Kirkpatrick, 1996; Feeney & Kirkpatrick, 1996). For example, Carpenter and Kirkpatrick subjected college-age women to a psychological stressor at two separate laboratory sessions: once in the presence of their romantic partners and once by themselves. In contrast to securely attached individuals, avoidantly attached individuals showed greater blood pressure and heart rate reactivity when their romantic partners were present than when they were absent, demonstrating that individuals’ expectations regarding the availability of attachment figures moderate stress-buffering effects.

Notably, securely attached individuals showed no difference between reactivity in the partner’s presence versus absence, suggesting that secure attachment might entail more successful “internalization” of the attachment figure’s stress-buffering capacities and therefore less dependence on concrete displays of support. Perhaps, then, it is the presence of a secure attachment figure in an individual’s life, rather than in the experimental room, that buffers stress. These are clearly promising areas for future study that require replication in different contexts, with different stress-

ors, and with more specificity in psychophysiological assessment.

In particular, future research on associations between attachment and ANS functioning would profit by focusing particular attention on the parasympathetic branch of the ANS. This is because the conceptualization of attachment relationships as regulators of affect and arousal implicitly suggests a homeostatic function, which is the domain of the PNS. Furthermore, there is extensive empirical evidence that the PNS is directly related to the processes of affect and arousal regulation that are hypothesized to play such a critical role in attachment. I therefore turn to a more extensive discussion of the functioning and measurement of the PNS and review extant social psychophysiological research suggesting its relevance for attachment processes.

Measuring and Interpreting PNS Activity

Measures of the PNS often focus on the functioning of the vagus nerve, which is a critical component of the PNS that is responsible for maintaining chronotropic control of the heart. Fluctuations in vagal activity are observed not only during intense stress, but also on a moment-by-moment basis as the body responds to both major and minor environmental demands. The result of these ongoing adjustments is *heart rate variability* (HRV). HRV refers to the continuous fluctuations in heart rate that occur in response to respiration, vasomotor factors, thermoregulatory factors, and environmental challenges (Akselrod, Gordon, Snidman, Shannon, & Cohen, 1985; Berntson et al., 1997). The component of variability that is directly attributable to respiration—which is called *respiratory sinus arrhythmia* (RSA)—is thought to be directly-mediated by vagal influence, and is therefore used to measure tonic levels and phasic changes in PNS activity (Porges, McCabe, & Youngue, 1982).

To briefly explain, heart rate accelerates slightly with each inhalation and decelerates slightly with each exhalation. This regular oscillation reflects the repeated withdrawal and subsequent reinstatement of vagal influence and produces a regular sinusoidal waveform in the pattern of intervals between successive heartbeats. The greater the vagal regulation of metabolic activity, the more heart rate will accelerate and decelerate in response to respiration, producing an RSA waveform with larger amplitude. This amplitude serves as a continuous numerical measure of vagal functioning. Measures of RSA taken at rest are typically used to index overall vagal tone, whereas phasic changes in RSA (such as those observed during and after stress) index vagal withdrawal and subsequent reengagement.

It bears noting that there is considerable variation in the terminology used to describe measurements of vagal activity and the techniques used to derive them. Some researchers measure and report RSA, whereas others refer to vagal tone, HRV, or high-frequency HRV. To make things more complicated, differences in terminology do not always correspond neatly to differences in measurement. Two different studies might both report data on RSA, yet use different techniques to derive these measures. Similarly, two researchers might measure RSA using the same method, but one might describe it as RSA and the other as vagal tone. The potential for confusion is clearly high, but the common dimension underlying all of these terms and techniques is that they assess parasympathetically mediated fluctuations in heart rate.

The different methods of measuring HRV, their technical requirements, and their appropriateness for different research aims are comprehensively reviewed in a recent task force report on these topics (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). Presently, most social psychophysiologicals use spectral analysis (Porges, 1986) to extract periodicity in the heart period pattern that occurs at the typical respiratory frequency (.12–40 Hz, corresponding to approximately 7.5–24 breaths per min). The variance of the heart period pattern in this bandwidth is calculated as the estimate of RSA. Another technique, called the “peak-to-valley” method (Grossman & Svebak, 1987), uses time series data on heart rate and respiration to isolate respiration-induced variability in heart rate on a breath-by-breath basis. There is very high correspondence between measurements obtained using these two methods (Grossman, van Beek, & Wientjes, 1990), but to apply and interpret them correctly, interested readers should consult the aforementioned task force recommendations.

Baseline Vagal Tone and Vagal Withdrawal

The mean amplitude of RSA taken during a baseline period is frequently used to classify individuals as having high or low vagal tone. Individuals in the latter group are generally conceptualized as having nervous systems that respond less quickly and flexibly to attention-demanding or stressful tasks than those in the former group. They are also conceptualized as recovering less effectively than the former group from stress-induced emotional arousal. Support for this view comes from research demonstrating that individuals with low baseline levels of vagal tone show heightened emotional reactivity and ineffective behavioral coping in response to stress (Fabes & Eisenberg, 1997; Fabes,

Eisenberg, & Eisenbud, 1993). Given that attachment styles have been conceptualized as affect regulation capacities and strategies, one might therefore expect insecurely attached individuals to have low vagal tone. Thus far, infant studies on this topic have yielded mixed results (reviewed in Fox & Card, 1999), and associations between vagal tone and attachment style have never been tested among adults. Related research, however, suggests that it deserves more extensive investigation. Adolescents and adults with secure attachment styles show more effective emotional and behavioral responses to stress (Cooper, Shaver, & Collins, 1998; Mickelson, Kessler, & Shaver, 1997), and the same has been found among adults with high vagal tone (Fabes & Eisenberg, 1997). Similarly, adolescents and adults with insecure attachment styles show higher rates of depression, anger, stress, and anxiety (Cooper et al., 1998; Mickelson et al., 1997), as have adults with low vagal tone (reviewed in Brosschot & Thayer, 1998; Friedman & Thayer, 1998). This is clearly a promising direction for future adult attachment research.

However, it may be equally important to focus on individual differences in stress-induced vagal withdrawal, which is typically represented as the baseline-to-task change in RSA. In general, effective physiological regulation is manifested by rapid, transient declines in RSA during highly stressful or attention-demanding tasks, which allows metabolic and attentional resources to be channeled toward the stressor without requiring over-activation of the SNS (Calkins, 1997; DeGangi, DiPietro, Greenspan, & Porges, 1991). Rapid increases in RSA after the termination of the stressor index rapid reinstatement of vagal control, which reestablishes homeostasis. As noted earlier, researchers have found notable individual differences in the extent to which cardiovascular reactivity to particular stressors is sympathetically versus parasympathetically mediated (Cacioppo, Uchino, & Berntson, 1994), and assessing individual differences in vagal withdrawal is one way to examine such differences. Although variation in vagal withdrawal is not wholly independent of variation in baseline vagal tone (individuals with low vagal tone tend to show less vagal withdrawal partly because of a floor effect), these measures nonetheless provide different types of information regarding physiological regulation that have different implications for attachment phenomena.

As summarized by Huffman and colleagues (Huffman et al., 1998), baseline vagal tone appears to index one's basic capacity to maintain an organized behavioral state, whereas vagal withdrawal appears to index one's ability to appropriately engage and disengage with features of the environment to manage environmental demands. For example, one study (Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996)

found that, whereas lower baseline vagal tone in 9-month-old infants was related to problems with basic state regulation, such as sleeping difficulties, low levels of vagal withdrawal in response to an attentional task at 9 months of age predicted socioemotional problems such as depression, social withdrawal, and aggression 2 1/2 years later. Another study of 2- and 3-year-old children found that baseline vagal tone was significantly related to reactivity during an emotion elicitation task, whereas vagal withdrawal was associated with coping behaviors during the task (Calkins, 1997). Of importance, this study also found that children with negative, controlling mothers showed less vagal withdrawal, but not lower baseline vagal tone. Thus far, potential links between individual differences in vagal withdrawal and individual differences in temperament and social functioning have received substantially more attention by researchers studying children than adults (Keltikangas Jaervinen, Kettunen, Ravaja, & Naeaetaenen, 1999). Yet given the increasing emphasis on parsing adults' autonomic reactivity into its parasympathetic and sympathetic determinants, this is a natural direction for adult attachment research to pursue. Toward the end of this article I outline specific hypotheses regarding PNS activity and attachment phenomena that are amenable to future testing. Presently, I turn attention to stress-induced reactivity in the HPA axis, another system with particular relevance for attachment phenomena.

The HPA Axis

Neuroendocrine responses to stress are observed in two different systems: the HPA axis and the sympathetic-adrenal medullary (SAM) axis. Activation of these two systems has 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). Stress reactivity in the SAM axis is associated with the release of catecholamines such as norepinephrine and epinephrine, whereas reactivity in the HPA axis is associated with the release of corticotropin releasing hormone (CRH), adrenocorticotrophic hormone, and cortisol (reviewed in Baum & Grunberg, 1995).

Much research has focused on differences between SAM and combined SAM and HPA patterns of stress reactivity (for reviews, see Blascovich & Tomaka, 1996; Cacioppo, 1994). The general consensus is that SAM reactivity represents an adaptive response to challenge, or situations in which the individual feels he or she has sufficient resources to meet task demands. In contrast, combined SAM and HPA activation represent responses to threat, or situations in which the indi-

vidual feels he or she cannot capably meet task demands. Not surprisingly, combined SAM and HPA reactivity is typically observed when stressors involve high levels of negative affect and is more strongly associated with increased cardiovascular disease risk and compromise immunological functioning than pure SAM activation (reviewed in Cacioppo, 1994). Thus, researchers investigating the health consequences of neuroendocrine stress reactivity have devoted increasing attention to HPA reactivity, and especially to individual differences in this domain. There are several reasons for adult attachment researchers to adopt this emphasis as well.

Attachment and HPA Activity

Research on both animals and humans has found that HPA activity—particularly cortisol release—is highly sensitive to social stimuli, making it a good candidate for investigations of the attachment system. For example, maternal touching reduces HPA stress responses in rats (Pihoker, Owens, Kuhn, Schanberg, & Nemeroff, 1993; Wang, Bartolome, & Schanberg, 1996) and ventral contact after a stressor has the same effect in rhesus monkeys (Gunnar, Gonzalez, Goodlin, & Levine, 1981; Reite, Short, Seiler, & Pauley, 1981). In fact, researchers developing animal models of mammalian social bonding (such as Carter, 1998) have argued that the HPA axis is critically implicated in attachment formation, an issue that will be revisited later. Not only do the hormones released by stress-induced HPA activity facilitate the formation of social bonds, but their subsequent release is directly influenced by both proximity to and separation from social partners (Hennessy, 1997). Of importance, the nature of these effects depends on the nature of the relationship between partners. Mendoza, Lyons, & Saltzman (1991) found that, whereas the presence of attachment figures appeared to provide a stress-buffering effect for squirrel monkeys that was manifested in reduced cortisol secretion, the presence of social affiliates (typically same-sex age-mates) was associated with patterns of cortisol secretion that were inconsistent with stress-buffering models. They concluded that the standard stress-buffering interpretation of social contact among nonhuman primates might only apply to attachment-like relationships, a conclusion seconded by Sachser, Durschlag, and Hirzel (1999).

This perspective has some support from research in humans, but requires considerably more investigation. Kirschbaum, Klauer, Filipp, and Hellhammer (1995) examined stress-induced cortisol reactivity among men and women receiving no social support, social support from a stranger, or social support from their romantic partner. Among men, only partner support was

associated with reduced cortisol reactivity, consistent with the notion of attachment figures as more effective regulators of stress-induced reactivity. However, this pattern was not observed among women; rather, partner support was associated with greater cortisol reactivity, an unexpected result that mirrors some of the conflicting findings reviewed earlier regarding stress-buffering effects on ANS reactivity. This further demonstrates that researchers must attend to both the relational context of support provision and individuals' interpretations of particular relational contexts to meaningfully interpret reactivity to different types of tasks performed in different social circumstances.

Individual Differences in HPA Reactivity

There is considerable evidence for individual differences in stress-induced HPA activation from research on nonhuman primates (Suomi, 1991), human infants (Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996; Spangler & Grossman, 1993), and human adults (Berger et al., 1987; Brandtstaedter, Baltes-Goetz, Kirschbaum, & Hellhammer, 1991). The general picture is that individuals whose stress-induced cortisol secretion fails to habituate over time tend to be highly reactive to negative emotions and to be socially inhibited and withdrawn (Gerra et al., 2001; Kirschbaum, Prussner, et al., 1995). Studies of cortisol secretion in fraternal versus identical twins suggest that these individual differences are partially heritable (Kirschbaum, Wust, Faig, & Hellhammer, 1992; Maxwell, Boyle, Greig, & Buchanan, 1969; Meikle, Bishop, Stringham, Ford, & West, 1989), but Kirschbaum and his colleagues noted that the extent of variability within twin pairs indicated a notable environmental component to such differences. Notably, one such component appears to be infant stress. Research on both normal and maltreated infants suggests that the routine process of down-regulating distress through comforting interactions with the caregiver helps to “tune” the infant’s developing neuroendocrine stress system (reviewed in Glaser, 2000; Schore, 1996), providing a critical foundation for effective coping and emotion regulation. For example, rhesus monkey infants who are reared with same-age peers instead of their mothers show exaggerated HPA reactivity to social separations later in life (Suomi, 1999).

Considering that insecure attachment patterns are thought to derive from inconsistent experiences of caregiver-mediated distress-alleviation, one might therefore expect insecurely attached infants and adults to show exaggerated HPA reactivity to stress. There is some support for this hypothesis from research on human infants. For example, several stud-

ies have found that infants with distress-prone temperaments and anxious attachment styles show exaggerated HPA reactivity to separations and reunions with attachment figures (Gunnar, Brodersen, Nachmias, Buss, & Rigatuso, 1996; Nachmias et al., 1996; Spangler & Grossman, 1993; Spangler & Schieche, 1998), although other studies have failed to detect such effects (Gunnar, Mangelsdorf, Larson, & Hertsgaard, 1989; Hertsgaard, Gunnar, Erickson, & Nachmias, 1995). This is an area in which interactions between temperament and attachment style appear particularly important (Belsky & Rovine, 1987; Stansbury, 1999) and warrant close scrutiny. Notably, associations between attachment style and HPA reactivity have never been tested in adults, and this area is clearly ripe for future study.

Links Between HPA and ANS Functioning

Future research should also focus on links between individual differences in HPA and ANS reactivity. Experimental research has found that individuals with high sympathetic ANS reactivity to laboratory stressors also show exaggerated HPA reactivity (Cacioppo et al., 1995). Cacioppo and his colleagues noted that these correspondences might be attributable to the joint role of corticotropin releasing hormone (CRH) in both HPA and ANS activity. In addition to functioning as a primary activator of the HPA axis, CRH stimulates both the sympathetic and parasympathetic branches of the ANS, especially the former (reviewed in Dunn & Berridge, 1990). Furthermore, there is evidence from animal research that CRH might mediate the influence of early rearing environments on individual differences in HPA and SNS stress reactivity (reviewed in Francis, Caldji, Champagne, Plotsky, & Meaney, 1999). Thus, although I have devoted separate attention to the role of the HPA axis and the ANS in attachment-related processes, it is important to note the functional interconnectedness of these systems. Given the role of CRH in both systems and the capacity for early rearing environments to alter CRH gene expression, future research might devote particular attention to testing links between CRH-related processes and attachment processes.

A Different Conceptualization of Regulation

In discussing the regulatory functions of attachment relationships, I have emphasized the alleviation of affective and physiological distress. Yet a somewhat different conceptualization of attachment and

regulation has also circulated in the literature. Although it has received far less theoretical and empirical attention, its provocative tenets are directly amenable to psychophysiological investigation and are worthy of closer study. This alternative conceptualization of regulation does not focus on the attenuation of discrete stressor responses, but rather on an assortment of longer-term processes by which two organisms reciprocally influence one another's physiological states (Field, 1985, 1994; Hofer, 1984, 1994; Mendoza et al., 1991). Field called the resulting state of biopsychological coregulation "attunement" and suggested that it might be a defining feature of attachment bonds.

Hofer (1994, 1987) was the first to systematically describe psychobiological coregulation in attachment relationships. His research demonstrated that in rats, contact with attachment figures regulates multiple physiological systems, not just those related to distress alleviation. He found that infant rats' sleep states, activity levels, metabolic processes, oxygen consumption, and endocrine activity were all directly regulated by contact with their mothers, and this regulation was effected through diverse sensorimotor stimuli ranging from maternal body warmth to milk nutrients to caregiving behaviors to olfactory cues. Correspondingly, Hofer found that prolonged separation from the mother triggered systemic disorganization in many of the infant's biological and behavioral systems, and that this dysregulation persisted for several days after the infant's initial, acute phase of separation distress had subsided. He interpreted this long-term separation response as the product of physiological dysregulation in each of the independent systems that was previously regulated by contact with the mother.

Hofer (1984) cautiously extended his model to human adults as well, suggesting that adult spousal bereavement might also be conceptualized as physiological dysregulation. As he pointed out, there are numerous correspondences between the long-term biobehavioral sequelae of adult bereavement and those of maternal separation, including depressed affect, social withdrawal, decreased food intake, sleep disturbance, and changes in ANS, endocrine, and immune system activity (reviewed in Hofer, 1984). Rather than attributing these diverse effects solely to the profound psychological stress of bereavement, Hofer proposed that they might also represent slow-developing biological changes resulting from the "withdrawal of patterns of sensorimotor stimulation that had been exerting an imperceptible regulating action on the subjects' minds and on their internal biologic systems" (p. 191). In other words, the bond between attached romantic partners might be physiological as well as psychological, and disruptions in the bond might have consequences for both domains.

Yet this compelling and provocative notion has never been empirically tested. It is therefore not surprising that Main's (1999) recent discussion of future directions in attachment research called for systematic investigation of hidden psychobiological regulators in human attachment bonds on par with those discovered by Hofer (1984). It is likely that regulatory processes take notably different forms in humans than in other mammals, and that they undergo change over the course of development. For example, both Hofer (1987) and Kraemer (1992) argued that in species capable of complex cortical functions and higher order symbolic associations, biobehavioral regulators may become increasingly internalized from infancy to adulthood, transduced "not only by sensorimotor and temporal patterning of the actual interactions, but also by the internal experiences of the relationship as it is carried out in the mind of the person involved" (Hofer, 1984, p. 192). Yet nonetheless, physical proximity and concrete interpersonal interactions with attachment figures appear to play an important ongoing role. In fact, intense distress arising from physical separation is the closest thing available to a "marker" of attachment formation in animals (Carter, 1998) and human infants (Field, 1991), and similar forms of distress have been observed among married couples undergoing war-time or job-related separations (reviewed in Vormbrock, 1993). Such studies raise fascinating questions about what exactly proximity and separation "do" in attachment relationships and how their effects are manifested in individual's minds and bodies on conscious and unconscious levels.

These are among the most basic and fundamental questions one can ask about human attachment, and they are best answered by developing and testing rigorous psychobiological models of attachment formation and functioning. Toward this end, I now introduce a tentative theoretical model outlining how the ANS and the HPA systems might be fundamentally integrated into the basic processes underlying the normative and individual-difference components of human attachment. Given the lack of extant research on the psychobiology of human attachment, this model is largely speculative at present and draws heavily from studies of animal attachment that have not been replicated with humans. Despite these limitations, it helps identify some of the most promising directions for future research on adult attachment.

The Psychobiology of Adult Attachment: A Preliminary Model

As noted earlier, animal research has found that the neuropeptide oxytocin plays a key role in mammalian attachment and affiliation (reviewed in Carter, 1998;

Insel, 2000), and it might also be implicated in integrating ANS and HPA processes related to human attachment. Oxytocin is a neuropeptide hormone produced in the hypothalamus that is released into circulation from the posterior pituitary, and which is also released directly into the brain from neurons in the paraventricular nucleus. Oxytocin is most well-known for stimulating the contractions of labor and facilitating milk let-down in nursing mothers, but animal studies have found that oxytocin is also released during (and subsequently reinforces) physical contact between social affiliates (Knox & Uvnäs-Moberg, 1998; Martel, Nevison, Rayment, Simpson, & Keverne, 1993; Uvnäs-Moberg, Bruzelius, Alster, & Lundeberg, 1993; Witt, Winslow, & Insel, 1992) and has been shown to influence maternal feeding behavior, maternal and infant bonding, and kin recognition (Carter, 1998; Nelson & Panksepp, 1996; Uvnäs-Moberg, 1994). Oxytocin also facilitates conditioning (Liberzon, Trujillo, Akil, & Young, 1997; Ostrowski, 1998) and plays a role in the formation of stable preferences for both places and social partners (Nelson & Panksepp, 1996; Nelson & Panksepp, 1998; Williams, Insel, Harbaugh, & Carter, 1994). Most notably, research on monogamous prairie voles has demonstrated that administration of central oxytocin facilitates adult pairbonding, whereas administration of oxytocin antagonists interferes with it (Cho, DeVries, Williams, & Carter, 1999; Williams et al., 1994).

Oxytocin has pronounced antistress effects, and this has been suggested as a mechanism through which it facilitates social bonding. In rats, chronic central infusions of oxytocin over a 5-day period produced sedation, reductions in blood pressure, reductions in stress reactivity, and reductions in stress hormone levels, and these changes persisted for several weeks after oxytocin treatments were terminated (Uvnäs-Moberg, 1997a). Such effects appear to be attributable to the fact that oxytocin attenuates HPA and sympathoadrenal activity (Chiodera et al., 1991; Uvnäs-Moberg, 1997b) and enhances parasympathetic activity (Uvnäs-Moberg, 1997a). On the basis of these effects, Uvnäs-Moberg (1998) and Carter (1998) proposed models of attachment formation that emphasize three main processes: (a) the triggering of oxytocin release by physical contact or sustained proximity to a potential attachment figure; (b) the subsequent, oxytocin-mediated down-regulation of stress reactivity in the HPA axis and the SNS, accompanied by enhanced PNS activity; and (c) the development of conditioned associations between the potential attachment figure and these effects.

Thus, these models posit that reactivity and recovery in the ANS and the HPA axis are critically implicated in the formation of attachment bonds, which might explain why mild to moderate stress has long been observed to

facilitate social bonding in both primates (Mason & Mendoza, 1998) and humans (Simpson & Rholes, 1994). Uvnäs-Moberg and her colleagues (Knox & Uvnäs-Moberg, 1998; Uvnäs-Moberg, 1998) have gone on to suggest that, as a result of repeated exposure to oxytocin release, participation in social bonds might gradually bring about long-term changes in stress reactivity (specifically, attenuated SNS reactivity and enhanced PNS regulation) that promote not only psychological feelings of comfort and security, but also long-term cardiovascular health.

I would argue that these enduring changes offer some of the most interesting and provocative possibilities for adult attachment research. If Carter and Uvnäs-Moberg are correct, then perhaps such socially induced changes in physiological functioning might serve as “markers” of attachment formation. Specifically, one might hypothesize that adults who form and maintain secure, long-term attachments will experience greater cumulative exposure to centrally released oxytocin, eventually resulting in faster down-regulation of HPA reactivity and more parasympathetically dominated patterns of ANS reactivity. In other words, the enduring presence of a secure attachment figure in an adult’s life might gradually “retune” his or her pattern of physiological reactivity in a manner that facilitates effective emotional and physiological regulation both in and out of the partner’s presence.

Existing Evidence

Is this “retuning” possible? The overall extent of such changes is no doubt limited by genetic factors, but research demonstrates that there is room for environmentally-induced modification in patterns of autonomic and neuroendocrine reactivity. As noted previously, early experiences of stress and soothing appear to shape patterns of HPA reactivity in both humans and nonhuman primates (Glaser, 2000; Schore, 1996; Suomi, 1999). With regard to the ANS, longitudinal research has found that patterns of vagal withdrawal (to identical stressors) vary over time—more so, notably, than baseline levels of vagal tone (Bornstein & Suess, 2000). This is consistent with twin data demonstrating that, although there is evidence for partial heritability in baseline vagal tone, there is no evidence for heritability in patterns of stress-induced vagal withdrawal (Snieder, Boomsma, Van Doornen, & De Geus, 1997). Such findings suggest that patterns of both HPA and PNS reactivity might be influenced by early childhood experiences of socially-induced distress-alleviation (or the lack thereof).

Additional changes may take place in adulthood, as well. Studies have found that short-term therapeutic in-

terventions aimed at reducing anxiety and enhancing relaxation are associated with enhanced parasympathetic functioning (Friedman, Thayer, & Borkovec, 1993; McCraty, Atkinson, Tiller, Rein, & Watkins, 1995; Sakakibara, Takeuchi, & Hayano, 1994; Toivanen, Laensimies, Jokela, & Haenninen, 1993) and reductions in cortisol release (Cruess et al., 1999; Cruess, Antoni, Kumar, & Schneiderman, 2000). McCraty and colleagues (McCraty et al., 1995; McCraty, Barrios-Choplin, Rozman, Atkinson, & Watkins, 1998) specifically argued that such relaxation interventions work by reprogramming the patterns of neuroendocrine and autonomic stress-reactivity that individuals first establish through childhood emotional experiences. Perhaps, then, attachment formation might be likened to an emotional “intervention” that gradually shifts individuals’ patterns of physiological reactivity in a more adaptive direction. On this point, it is particularly notable that the relaxation intervention used by McCraty and his colleagues to effect changes in ANS functioning involved focusing on feelings of “care and appreciation” for loved ones.

Additional support for this model comes from a study finding that strong feelings of affection for spouses predicted attenuated ANS stress-reactivity 2 years later (Uchino, Kiecolt-Glaser, & Cacioppo, 1994), and a study demonstrating that women who cohabitated with a spouse or romantic partner had significantly higher levels of tonic HRV (i.e., greater tonic parasympathetic regulation of heart rate) than women who were living alone, even after controlling for overall social support (Horsten et al., 1999). Collectively, such findings support Cacioppo’s (1994) claim that an individual’s current pattern of cardiovascular and neuroendocrine activity might be conceptualized as a function of his or her most important interpersonal relationship.

Attachment Versus Social Support

But which relationships “count?” Obviously, social influences on ANS and HPA reactivity are not limited to romantic attachments. Research has detected general attenuation of stress reactivity in individuals perceiving high emotional support from their overall social networks (Knox, 1993; Tardy, Thompson, & Allen, 1989), and Light et al. (1998) found lower levels of tonic HRV and exaggerated sympathetic nervous system reactivity in women with low perceptions of social support and high depressive symptoms. As for the HPA axis, research has documented that both stress and loneliness are associated with greater HPA activity (Cacioppo et al., 2000; Hawk, Dougall, Ursano, & Baum, 2000; Lovallo, Pincomb, Edwards, Brackett, & Wilson, 1986; Steptoe, Cropley, Griffith, & Kirschbaum, 2000),

whereas perceptions of social support are associated with lower HPA activity (Goodkin et al., 1998; Seeman et al., 1994; Turner Cobb, Sephton, Koopman, Blake Mortimer, & Spiegel, 2000).

This suggests that the processes through which social bonds are hypothesized to retune patterns of physiological reactivity might operate in all important social bonds, reintroducing the ever-present question of how exactly attachment and social support differ from one another. Of importance, researchers studying animals have suggested that both attachment and affiliative relationships are mediated by common neural circuits (DeVries, Johnson, & Carter, 1997; Keverne, Nevison, & Martel, 1999; Nelson & Panksepp, 1998), suggesting that the oxytocinergic mechanisms through which social support and attachment hypothetically influence long-term HPA and ANS functioning might be largely the same. Nonetheless, these mechanisms might be activated more frequently within attachment relationships, might become more powerfully conditioned to specific individuals in these contexts, and might have more enduring regulatory effects on psychological and physiological functioning. Clearly, future study of these issues can significantly advance our understanding of the basic processes underlying all human social ties.

Individual Differences

If individual differences in attachment style interfere with individuals' ability to derive emotional security from contact with attachment figures, might they moderate the hypothesized psychobiological processes underlying attachment formation? Furthermore, might attachment style differences themselves be conceptualized as fundamentally psychobiological as well as cognitive–affective–behavioral predispositions? These are perhaps some of the most interesting questions that psychophysiological research on adult attachment might address.

As noted earlier, it is important to remember that insecure attachment styles do not prevent individuals from forming attachments (i.e., developing a selective orientation toward a specific person as the primary target for distress-alleviation), but rather influence individuals' strategies for distress-alleviation and the effectiveness of these strategies. Thus, one testable possibility is that the basic psychobiological mechanisms underlying attachment formation are the same for adults with insecure versus secure attachment styles, but that the process is more protracted in the former group or might not be associated with the same degree of change in physiological functioning that I have hypothesized to occur within normative, secure attachments. In other words, not only might insecurely attached individuals fail to derive immediate stress-buffering benefits from their partners' presence, as demonstrated by Carpenter

and Kirkpatrick (1996), but they might also fail to derive as many long-term benefits from their close relationships as would securely attached individuals. This is consistent with research demonstrating that heightened physiological stress reactivity is better predicted by individuals' satisfaction with the social support available to them than the overall amount of available support (Carels, Blumenthal, & Sherwood, 2000).

An additional issue warranting further study involves differences between anxious and avoidant “versions” of insecurity. Some research suggests that, at least among infants and children, attachment anxiety more strongly resembles a biologically-mediated form of psychosocial vulnerability than does attachment avoidance (reviewed in Cassidy & Berlin, 1994). Specifically, attachment anxiety may represent a deficient capacity for emotion regulation, whereas avoidance may represent a nonoptimal but serviceable strategy for emotion regulation (see also Fraley & Shaver, 1998). If so, then psychophysiological manifestations of attachment anxiety should be observed in notably different contexts than psychophysiological manifestations of attachment avoidance. For example, one might expect that, regardless of their partner's presence, highly anxious individuals would show poor down-regulation of HPA reactivity and more sympathetically-dominated patterns of ANS reactivity. In contrast, one might only find such patterns in avoidant individuals if they were “forced” to deviate from their preferred emotion-regulation strategy—that is, if they were required to maintain close proximity to their partners when they might prefer distance (as in Carpenter & Kirkpatrick, 1996). Studies combining self-report, observational, and physiological data would help clarify these possibilities.

Directions for Future Research: Summary

Based on the aforementioned model, I propose the following four areas for future study on the psychobiology of adult attachment. Most of these research questions have already been discussed previously, but the following summary clarifies how they fit together in an organized investigative approach to attachment and psychophysiology.

How do individuals “become attached,” on a physiological level? Are the aforementioned physiological pathways valid? If they do contribute to attachment formation, is their role mediated by affective–cognitive processes (i.e., you are less physiologically reactive because you feel secure), or do they mediate affective–cognitive processes (i.e., you feel secure because you are less physiologically reactive)? Alternatively, do the cognitive–affective components of

attachment develop independently of—or at different rates than—the hypothesized physiological component?

Are there physiological markers of secure attachment formation? Do individuals with long-term, secure attachments show the gradual changes in ANS and HPA reactivity that have previously been hypothesized? Are such changes sensitive to proximity to, and distance from, the attachment figure? How might physiological markers of secure attachment resemble or differ from markers of high social support? Does the loss of (or prolonged separation from) an attachment figure have different physiological effects than the loss of social support?

Are the physiological aspects of attachment moderated by attachment style? Does attachment formation take longer in individuals with insecure attachment styles? Does it follow different physiological pathways? Are the hypothesized physiological markers of attachment less evident among insecurely attached individuals? Are the physiological effects of the partner's actual versus imagined presence moderated by attachment style? Do insecurely attached individuals show greater disjunctions between the physiological and affective–cognitive aspects of attachment?

Do attachment styles have physiological underpinnings? Is attachment anxiety associated with less adaptive patterns of ANS and HPA stress-regulation? If so, are such associations independent of temperamental variation in these domains, or are there specific temperament and attachment style combinations that are particularly physiologically vulnerable? Is avoidance also associated with physiological vulnerability, or does it more commonly reflect a behavioral rather than a physiological profile?

Some of these questions will prove more difficult to answer than others. For example, hypotheses about the physiological profiles of different attachment styles can be tested fairly easily with readily available methods and measures. Testing hypotheses about attachment formation is more problematic. The gold standard, of course, would be a multimethod longitudinal study that tracked the physiological, affective, cognitive, and behavioral sequelae of attachment formation over a number of years. Given the obvious logistical problems presented by such a study, it makes more sense to begin with cross-sectional comparisons of new and established couples aimed at identifying the most promising physiological processes and markers for future study. All such investigations must involve careful, differentiated assessment of autonomic and neuroendocrine activity both at rest and in response to multiple, diverse stressors experienced in different contexts. As noted previously, careful attention must be paid to the task

characteristics of each stressor (i.e., controllability, incentive, performance demands) and its documented autonomic and neuroendocrine response profile to meaningfully interpret the results. Finally, it would be ideal to supplement laboratory measures of reactivity with those obtained in naturalistic settings using ambulatory monitors (e.g., Sloan et al., 1994).

Conclusion

In recent years, social psychological research has increasingly broadened its models and methods to explore the biological constituents of social behavior (Berntson & Cacioppo, 2000), and research on adult attachment should follow suit. Human affectional bonding is a complex phenomenon whose diverse psychological, behavioral, and physiological manifestations require a multilevel analytical approach (Cacioppo & Berntson, 1992). Psychophysiological measures are a critical component of this approach, but must be selected and interpreted with care if we are to make conceptual and empirical advances in understanding the biobehavioral processes underlying the attachment system.

I have argued that, given the role of attachment relationships in regulating negative affect and arousal, initial forays into the psychophysiology of adult attachment should focus on physiological systems that are involved in the regulation of psychological distress and that are demonstrably influenced by social stimuli. Both the parasympathetic branch of the ANS and the HPA axis of the endocrine system meet these criteria and are promising starting points for future study. Hopefully, research of this nature will expand to include additional biological processes that have shown links to the attachment system in other mammalian species, such as oxytocinergic and opioid mechanisms, and will contribute to the growing body of research into the physiological mechanisms through which marriage and social integration promote long-term physical health (Berkman, 1995; House, 1988; Uchino et al., 1996). Such research has the potential to advance our understanding of the powerful effects of human love on our bodies as well as our minds.

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