Personality and Social Psychology Review

Coregulation, Dysregulation, Self-Regulation: An Integrative Analysis and Empirical Agenda for Understanding Adult Attachment, Separation, Loss, and Recovery

David A. Sbarra and Cindy Hazan

The online version of this article can be found at:
http://psr.sagepub.com/cgi/content/abstract/12/2/141

Published by:
$SAGE Publications
http://www.sagepublications.com

On behalf of:

SP
SP

Society for Personality and Social Psychology, Inc.

Additional services and information for *Personality and Social Psychology Review* can be found at:

Email Alerts: http://psr.sagepub.com/cgi/alerts

Subscriptions: http://psr.sagepub.com/subscriptions

Reprints: http://www.sagepub.com/journalsReprints.nav

Permissions: http://www.sagepub.com/journalsPermissions.nav

Citations (this article cites 156 articles hosted on the
SAGE Journals Online and HighWire Press platforms):
http://psr.sagepub.com/cgi/content/refs/12/2/141

Coregulation, Dysregulation, Self-Regulation: An Integrative Analysis and Empirical Agenda for Understanding Adult Attachment, Separation, Loss, and Recovery

David A. Sbarra
University of Arizona

Cindy Hazan
Cornell University

An integrative framework is proposed for understanding how multiple biological and psychological systems are regulated in the context of adult attachment relationships, dysregulated by separation and loss experiences, and, potentially, re-regulated through individual recovery efforts. Evidence is reviewed for a coregulatory model of normative attachment, defined as a pattern of interwoven physiology between romantic partners that results from the conditioning of biological reward systems and the emergence of felt security within adult pair bonds. The loss of coregulation can portend a state of biobehavioral dysregulation, ranging from diffuse psychophysiological arousal and disorganization to a full-blown (and highly organized) stress response. The major task for successful recovery is adopting a self-regulatory strategy that attenuates the dysregulating effects of the attachment disruption. Research evidence is reviewed across multiple levels of analysis, and the article concludes with a series of testable research questions on the interconnected nature of attachment, loss, and recovery processes.

Keywords: adult attachment; coregulation; separation; loss; recovery; oxytocin; stress; cytokines; psychophysiology

In response to loss, several different processes may be at work having different biological mechanisms. . . . They suggest that we look carefully at the relationship *before* the loss took place and try to understand more precisely who and what has been lost, rather than beginning our investigation with the disruption of the emotional bond or tie between the two individuals, as if bereavement were simply a stress that was suddenly imposed.

Hofer (1984, p. 194)

Not infrequently after a person has been bereaved the situation with which he has to deal is unique, for the death entails the loss of the very person in whom he has been accustomed to confide. Thus, not only is the death itself an appalling blow but the very person towards whom it is natural to turn in calamity is no longer there. For that reason, if mourning is to follow a favorable course, it is essential that the bereaved be able to turn for comfort elsewhere.

Bowlby (1980, p. 232)

Social attachments are a fundamental human need. Although this observation has spurred a wealth of research attention (e.g., Baumeister & Leary, 1995; Mikulincer & Shaver, 2003; Reis, Collins, & Berscheid, 2000) and

141

is hardly novel, many aspects of the thesis remain unexplored. If we thrive—mentally and physically—most completely when socially connected (House, Landis, & Umberson, 1988; Stack & Eshleman, 1998), it follows that we are pained when relationships end not merely because we miss companionship or are suffering from the adverse consequences of stress, but because humans need social ties to function best. When long-term mate relationships end, many adults lose the person who helps them maintain psychological and physiological homeostasis. In human attachments, the core of this homeostatic set point is the experience of felt security, a sense that the world is safe and nonthreatening and that exploratory activities can be pursued without the risk of danger (Mikulincer & Shaver, 2007; Sroufe & Waters, 1977). Accordingly, when relationships dissolve, it is this state of security that must be regained as individuals recover from separation and loss experiences. In this article, we propose an integrative framework for understanding how multiple biological and psychological systems are regulated by relationships, dysregulated by separation and loss, and, potentially, reregulated through individual recovery efforts. We believe that separation, loss, and recovery are best understood by first considering the nature and function of intact attachment bonds (Hofer, 1984, 1987, 2006; Mikulincer & Shaver, 2007; Shear & Shair, 2005). In other words, to fully appreciate what is lost when human social bonds are disrupted and how adults recover from such experiences, it is essential to know more precisely what ongoing attachments provide.

An attachment perspective on social loss is not new. Bowlby's (1969/1982, 1973, 1980) theory was developed to explain the harmful effects associated with maternal separation and deprivation. In the first book of his trilogy, *Attachment* (Bowlby, 1969/1982), he argued, "Understanding the response of a child to separation or loss of his mother-figure turns on an understanding of the bond that ties him to that figure" (p. 177). In adulthood, attachment-related concepts play a large role in contemporary theories of bereavement and divorce (Bonanno et al., 2002; Bonanno & Kaltman, 1999; Emery, 1994; Fraley & Shaver, 1999; Hazan & Shaver, 1992; Kitson & Holmes, 1992; Parkes, 2001; Parkes & Weiss, 1983; Shaver & Tancredy, 2001; Shear & Shair, 2005; Vormbrock, 1993; Weiss, 1975, 2001). With only few exceptions (e.g., Mikulincer & Shaver, 2007, in press; Shear & Shair, 2005), however, most of this research either relies on Bowlby's (1969/1982) initial observations concerning a child's tie to his or her caregiver or is focused on individual differences in responses to loss. Although useful, these approaches are insufficient for understanding the experience of pair bond dissolution; neither

approach addresses the underlying function of the relationship that was disrupted. Due largely to the introduction of the strange situation paradigm for investigating patterns of mother–infant attachment (Ainsworth, Blehar, Waters, & Wall, 1978) and the subsequent development of self-report and interview measures of adult romantic attachment (George, Kaplan, & Main, 1984; Hazan & Shaver, 1987), individual differences are the near exclusive focus of human attachment research (Marvin & Britner, 1999; Mikulincer, Birnbaum, Woddis, & Nachmias, 2000; Mikulincer & Shaver, 2003; Shaver & Mikulincer, 2002; Simpson & Rholes, 1998). The accumulation of knowledge on adult attachment styles has come at a cost to understanding how attachment relationships (in adulthood) develop more generally. The field still lacks a fully developed theoretical account of what it means to be attached to another person and the functional mechanisms that maintain adult pair bonds (Diamond, 2001; Fraley, Garner, & Shaver, 2000; Hazan, Gur-Yaish, & Campa, 2004; Hazan & Zeifman, 1999; Main, 1999). Normative models of adult attachment certainly exist, but they are in need of extension and elaboration, particularly with respect to the ways in which two people become attached and how attachment relationships provide protective health benefits beyond general stress buffering. Although Milkulincer and Shaver's (Mikulincer & Shaver, 2003, 2007; Mikulincer, Shaver, & Pereg, 2003; Shaver & Mikulincer, 2002) research and theory development have shed much light on the normative regulation of felt security within the attachment behavioral system, the secondary strategies used to maintain and regain this state of felt security (e.g., hyperactivating and deactivating behaviors), the nature of working models of attachment, and individual differences in the use of these strategies, their model says little about the underlying physiological systems that are associated with felt security and the process through which two people become attached.

One way to profitably extend Mikulincer and Shaver's work on normative attachment is to examine individuals' responses to separation and loss events. Indeed, many investigators believe that separation distress is the strongest marker of the existence of an attachment relationship (Bowlby, 1980; Fraley & Shaver, 1999; Hazan et al., 2004; Parkes, 1998; Weiss, 2001), and Hofer (1996, 2006) has argued that our understanding of normative attachment can be advanced by answering the question of what exactly is lost when a loved one dies (also see Shear & Shair, 2005). Understanding more about how adults become attached can provide deeper insights into the functional elements of human coregulation and shed new light on biobehavioral reactions to attachment figure loss.

ORGANIZATIONAL AND
THEORETICAL OVERVIEW

The organization of this article follows directly from our main aims of describing the normative functions of attachment relationships (coregulation), what happens when relationships are disrupted or severed in adulthood (dysregulation), and what strategies individuals invoke to regain homeostasis following separation and loss events (self-regulation). The analysis begins with some orienting remarks on attachment across the lifespan and then, drawing heavily from animal studies, reviews evidence for a cross-species model of attachment that involves a conditioned physiological state called coregulation, which is defined as the reciprocal maintenance of psychophysiological homeostasis within a relationship. In this model, coregulation is the physiological instantiation of felt security within an attachment relationship; coregulation describes how felt security operates at the level of physiology. Although we hypothesize that coregulation is a unique and emergent property of all clear-cut attachment relationships (see Hazan et al., 2004), the focus of this article is on adult pair bonds, which, relative to child–caregiver attachments, are defined by reciprocity (in the provision of felt security) between partners. As described below, in romantic partners, sexual and physical intimacy provides the strongest and fastest conditioning of physiological reward systems. In friendships (or nonromantic attachments), the same processes are likely operating, but in a much slower fashion and with less potent physiological correlates. Understanding how coregulation emerges in adulthood is critical to a nuanced account of why and how loss and separation experiences confer risk for poor physical health outcomes. Moreover, elaboration of the concept of human coregulation highlights ways in which attachment relationships may confer health benefits above and beyond interpersonal stress buffering alone (see Uchino, 2004).

From the outset, two points of order are critical. First, the discussion of coregulation rests heavily on a model of commingled physiology between romantic partners, but this does not imply that the entirety of an attachment bond is physiological or that physiological changes are the causal mechanisms driving the emergence of felt security or secure base behaviors. Rather, because there is a great deal of research on psychological dynamics of attachment, our goal is to echo and expand on the work of others who suggest that understanding the biological correlates of attachment will provide a richer appreciation of the functional, normative components of adult relationships (Carter, 1998; Coan, in press; Diamond, 2001; Hofer, 1984, 2006; Insel, 2000; Shear & Shair, 2005). The notion that an attachment relationship is more or less of a psychological or physiological entity is reification of the dualism that has fractured psychological science for much of the past 100 years (cf. Cacioppo et al., 2000); therefore, a complete account of attachment and loss needs to address both psychology and physiology. As attachments form they are evident and operative at multiple levels, including the physiological level (likely because of the unique physical intimacy of mate relationships). Second, the account of adult coregulation presented here is one of the first attempts to integrate the entirety of this literature, much of which is in the context of animal research on the psychobiology of affiliation. We recognize that caution should be used in generalizing from animals to humans. Moreover, although the goal of this article is to describe the physiological dynamics operating in adult pair bonds, some of the best evidence for coregulation comes from animal studies of dam–pup (mother–infant) interactions (e.g., Polan & Hofer, 1999), which may function in quite different ways than within mate relationships. Although evidence from animal studies is strong, there exists only limited evidence from human studies for an attachment-as-coregulation model. Although the data are clearly limited, discussions of coregulation, attunement, and social zeitgebers are emerging with increasing prevalence in the social psychology literature on close adult relationships (Diamond, 2001, Diamond, Hicks, & Otter-Henderson, in press; Mikulincer & Shaver, 2003; Pietromonaco, Barrett, & Powers, 2006; Shear & Shair, 2005; Uchino, 2004). Without a synthesis of the available evidence and a working definition of precisely what coregulation is (e.g., how it differs from stress buffering, an important element of social support) and how it operates in humans in a manner that is consistent with Hofer's (1984) initial observations in animals, the field runs the risk of suffering a paralyzing lack of precision in understanding the biological bases of normative attachment and, by extension, responses to separation and loss. There is little doubt that our detailing of these processes is incomplete, but there is clear need for integrating many of the points raised by Bowlby (1969/1982, 1973, 1980), Hofer (1984, 1987, 1996, 2006), and other researchers (Carter, 1998; Diamond, 2001; Hazan et al., 2004; Insel, 2000; Panksepp, Nelson, & Bekkedal, 1997). Organizing this literature under an integrative framework allows for a critical taking stock of what we know and what we need to know about the normative components of attachment and loss.

In the second section of the article, we argue that separation and loss experiences, by definition, disrupt individuals' sense of felt security, which is tantamount to losing the functional components of the attachment relationship. This disruption of coregulation can lead to

a state of *biobehavioral dysregulation* ranging from mild or diffuse physiological arousal to a full-blown and highly organized stress response. At its core, this model of dysregulation has two components, the loss of reward and the onset of distress, which emerge along a continuum based on perceived threats to felt security and the existence of the relationship itself. This is not a new model of physiological stress responses unique to attachment figure loss; rather, our goal is to connect, in a new way, what is known about the biology of attachment with the biology of loss experiences. After outlining this model of dysregulation, we reinterpret the available physiological research on loss in terms of the known parameters of the mammalian stress response and briefly summarize key findings on the association between attachment disruptions and health outcomes. The third section of the article concerns self-regulation, and we argue that in cases of successful recovery following a significant social loss, multiple cognitive, emotional, and behavioral strategies serve the function of attenuating physiological arousal and moving individuals from a state of dysregulation to individually maintained homeostasis (i.e., felt security). This perspective on self-regulation focuses on the functional aspects of recovery efforts rather than the specific content of the coping strategies. We suggest that multiple coping strategies can serve the same end state: restoring individuals' sense of felt security. Finally, the last section of the article outlines an empirical agenda for filling gaps in the framework; we raise looming questions and present testable hypotheses about the integrated nature of attachment, loss, and recovery.

NORMATIVE ATTACHMENT AND HUMAN COREGULATION: FORMATION, FUNCTION, AND PHYSIOLOGY

Our account of normative pair bonding begins with a hypothesis: The emergence of an attachment relationship is a developmental process that unfolds over time. Understanding how adults form a pair-bond is critical for understanding the functional, normative elements of an attachment relationship. In both infancy and adulthood, repeated social contact with a rewarding other results in a conditioned response pattern whereby one particular person (above many others) is reliably associated with a state of psychological security and physiological calm (Depue & Morrone-Strupinsky, 2005). As attachments form, they are evident and operative at multiple levels, including—in mates at least—the level of physiology (likely because of the unique physical intimacy of mate relationships). In the remainder of this

section, we describe this attachment formation process and the resulting physiological coregulation in detail.

Attachment Formation and Functionality Across the Lifespan

Drawing from a combination of psychoanalysis, evolutionary theory, ethology, and control systems theory, Bowlby's (1958, 1969/1982) initial conceptualization of attachment was as a biologically based behavioral system designed to promote infant survival across the life course. Four classes of behaviors, which are preferentially directed toward one person (i.e., an attachment figure), signify the presence of an attachment bond: proximity maintenance, safe haven activities, secure base activities, and separation distress (see Hazan et al., 2004, for a discussion of these behaviors in adults). Infant-to-caregiver attachment has immediate survival value because immature infants are in considerable danger unless their signaling can reliably elicit care. According to Bowlby (1969/1982), the formation of an attachment bond in infancy results from an increasingly complex dialectic between infant and caregiver that progresses through four relatively distinct phases, beginning with a general readiness to become attached to any available caregiver and ending with preferential signaling and distress on separation from a specific caregiver (Ainsworth, 1967; Bowlby, 1969/1982). Although the attachment system is present at birth, attachment bonds are not. By 6-8 months of age, infants behave in a way that reflects the emergence of "clear-cut" attachment—they reliably seek contact comfort from a specific caregiver and experience distress when their efforts are thwarted (Cassidy, 1999). The development of this bond is fueled by behavioral conditioning, which results from repeated distress alleviation and pleasure induction by the primary attachment figure. Bowlby (1969/1982) noted that the development of attachment toward particular figures emerges from

> exposure learning, which results in an infant learning the perceptual attributes of whoever is caring for him and discriminating that person from all other persons and things . . . and that well-known form of learning through which, as a result of feedback of certain consequences of behaviour, that behaviour can become augmented (reinforced). (p. 314)

It is important to note that this "exposure learning" is a product of both negative and positive reinforcement. In times of distress, infants' affective or physiological arousal can be most readily attenuated by an attachment figure. As infants learn that their primary caregiver is the person who most consistently extinguishes noxious

states and provides pleasurable interactions, proximity maintenance is reinforced, and the infant is more likely to seek repeated contact with and comfort from one particular caregiver.

Over time, both distress alleviation and pleasure induction contribute to infants' feelings of security within the relationship. Attachment behaviors serve the proximal purpose of regulating the experience of emotional security and keeping children in proximity to their attachment figures (Bowlby, 1973). Sroufe and Waters (1977) developed these ideas more completely by positing that the inherent goal or "set point" of the attachment system is not physical proximity per se but rather "felt security." Attachment behaviors (crying, signaling, approaching) could be activated by internal (anxiety, fatigue, illness) or external (strangers, a novel environment) threats to felt security and deactivated by the perception of safety.

In concert with this progress in understanding the regulatory dynamics of attachment, Ainsworth et al. (1978) demonstrated that differences in caregiver responsiveness and sensitivity to infants' bids for comfort resulted in systematic differences in the ways infants organized their attachment behaviors and used their attachment figures during times of stress. The function of these different patterns is essentially the same—that is, to effectively regulate felt security (Crowell et al., 2002; Weinfield, Sroufe, Egeland, & Carlson, 1999). Hazan and Shaver (1987) demonstrated that Ainsworth et al.'s patterns of infant–caregiver attachment could be applied to adult romantic relationships. Like the patterns observed in infancy, adult attachment styles reflect individuals' general approach to romantic relationships.

As in infant–caregiver attachment, the process of funneled conditioning is essential to understanding adult pair-bond development. Hazan and Zeifman (1999; Zeifman & Hazan, 1997) proposed a process model of attachment formation in adulthood that parallels Bowlby's (1969) phases of attachment development in infancy. Within this model, adults (like infants) move through successive phases of attachment formation. As romantic partners fall in love and enter what Ainsworth called the *attachment-in-the-making* phase, they engage in many behaviors typical of infant–caregiver pairs, such as cuddling, nuzzling, prolonged gazing, mutually ventral (i.e., front-to-front) contact, and baby talk (Zeifman & Hazan, 1997).

Two differences between infant–caregiver attachment and pair bond development are especially notable. First, the latter typically begins with the conditioning of pleasure and ongoing positive reinforcement. Unlike infants, who are almost entirely dependent on adults to regulate

their affect and reduce aversive states of arousal, adults are able to maintain emotional homeostasis and are less dependent on others to alleviate their distress. Instead, the positive aspects of social contact and closeness dominate the earliest phases of pair bond development. A second major difference is that pair bonding by definition involves sex. As detailed below, sexual behavior activates and, over time, conditions the physiological systems associated with an attachment relationship in adulthood. Sexual behavior is strongly associated with the endogenous physiological reward systems within the mammalian brain, and there is compelling evidence that the neuropeptide oxytocin plays a vital role in birth, nursing, and mating behaviors in mammals (Carter & Altemus, 1997; Carter, Williams, Witt, & Insel, 1992; Uvnäs-Moberg, 1998). Furthermore, the opioid theory of social affiliation (Depue & Morrone-Strupinsky, 2005; Nelson & Panksepp, 1998; Panksepp et al. 1997) posits that the rewarding effects of social contact facilitate learning and funnel attention toward social stimuli and may produce social "addiction" or attachment. Together, the oxytocin and opioid systems provide the physiological basis of felt security and serve the primary purpose of making attachment bonds inherently pleasurable and also capable of reducing distress. In adults, sexual and other intimate behaviors (e.g., cuddling, nuzzling) activate these systems and facilitate pair bond formation. As in infant–caregiver attachment, the result of repeatedly activating and ultimately conditioning these physiological systems is a specialized bond that serves the primary purpose of regulating psychophysiological arousal.

Oxytocin and the Endogenous Opioids Induce Pleasure and Alleviate Distress

Oxytocin, which is found exclusively in mammals, is a small neuropeptide produced in the paraventricular nuclei of the hypothalamus and stored in the posterior pituitary (Insel, 2000). It serves the primary roles of triggering labor and facilitating milk ejection during lactation, and it has been implicated in a wide range of mammalian behaviors, including birth, sexual behavior, maternal behavior, social bonding, and pair bonding (Lim & Young, 2006). (Vasopressin is a closely related nine-amino-acid neuropeptide that is also synthesized in the paraventricular nuclei; although both hormones regulate pair bond development, vasopressin is more highly associated with typically male social behaviors, such as aggression and mate guarding; see Lim & Young, 2006.) Oxytocin attenuates responses to social separation, is released by intimate social stimulation, plays a central role in the formation of social preferences, and modulates

affiliative behavior across a wide range of social contexts (Carter, 1998; Nelson & Panksepp, 1998; Panksepp et al., 1997). In rats, for example, actions taken to block the effects of this hormone (by lesion or receptor antagonists) inhibit the onset of nesting and maternal behaviors (Insel, 1997), and it is widely believed that oxytocin may act centrally to integrate maternal behavior (Carter, 1998; Insel, 2000; Pedersen, 1997). In one of the first human studies of this hypothesis in humans, Feldman, Weller, Zagoory-Sharon, and Levine (2007) studied 62 pregnant women and found that high levels of oxytocin during pregnancy (and during the first postpartum month) were positively correlated with a constellation of clearly defined maternal behaviors, including gazing, vocalization, affectionate touch, positive affect, and maternal checking behavior.

In terms of pair bond development, it is hypothesized that centrally circulating oxytocin may enhance sexual arousal, that the peripheral release of oxytocin may coordinate the phenomenology of orgasm and facilitate sperm transportation (via uterine contractions), that oxytocin may be implicated in the satiety effects of sexual behavior, and that oxytocin released during sexual behavior could function to reinforce social bonds between sexual partners (Carter et al., 1992; Carter & Altemus, 1997). Much of the support for the role of oxytocin in pair bond development comes from comparative investigations of prairie and montane voles, which are small rodents native to the American Midwest. Prairie and montane voles are highly similar in appearance and nonsocial behavior but differ considerably in terms of social behavior, with prairie voles manifesting the classic features of monogamy and montane voles showing a promiscuous pattern of mate preference (Carter, DeVries, & Getz, 1995). Centrally administered oxytocin facilitates the development of partner preference among female prairie voles in the absence of mating, and oxytocin antagonists block the formation of partner preference without interfering with mating, which causes the prairie voles to resemble the montane voles (Insel, 1992; Insel & Hulihan, 1995). Insel (2000; Insel & Young, 2001) argued that cellular differences in oxytocin receptor distributions within the brain that are associated with reward (e.g., nucleus accumbens and prelimbic cortex) are responsible for the phenotypic differences between the two species of voles. For prairie voles, mating may activate these reward systems and thus reinforce a social bond; for montane voles, no oxytocin receptors are found in these brain regions, suggesting that mating does not confer such physiological rewards. Indeed, pair bond formation in prairie voles is moderated by dopaminergic neurotransmission in the nucleus accumbens (Aragona et al., 2006). Young and Wang (2004) recently reviewed the animal literature on

pair bonding and concluded that ample evidence exists to suggest that the positive association between oxytocin and increased mesolimbic and prefrontal dopamine activity released in the context of mating behaviors points to the strong role of social learning and behavioral conditioning in the emergence of a pair bond (see Depue & Morrone-Strupinsky, 2005). This evidence also is consistent with experimental addiction research in rats demonstrating that oxytocin plays a neuromodulatory role in the dopamine reward circuitry of the brain, attenuating naloxone-induced withdrawal reactions (Kovacs, Sarnyai, & Szabo, 1998).

Although the biological bases of these attachment-promoting mechanisms are not well understood in humans, the available animal literature suggests that one explanation for oxytocin's role in facilitating human attachment formation is that the release of this hormone during sexual arousal, genital stimulation, and orgasm is highly rewarding (Young & Wang, 2004). The conditioned association between the rewards of oxytocin release and a specific person bears much resemblance to the opioid theory of social attachment, which is premised on the neurochemical similarities between opioid dependence and social dependence. There is evidence that, in addition to powerfully attenuating reactions to social separation and being released during social contact, endogenous opioids induce states of euphoria in animals and have powerful antinociceptive properties (Nelson & Panksepp, 1998; Panksepp et al., 1997; Panksepp, Siviy, & Normansell, 1985). It is posited that the rewarding effects of social contact facilitate learning (Belluzzi & Stein, 1977) and funnel approach toward social stimuli and may produce social "addiction" or attachment (Nelson & Panksepp, 1998). Rat pups quickly develop preferences for social stimuli associated with opioid activation (Kehoe & Blass, 1986), and opioid antagonists are known to increase social solicitations in young rhesus monkeys (Martel, Nevison, Simpson, & Keverne, 1995). Similar results have been found in guinea pigs and adult rats in which morphine was found to disrupt social cohesiveness and reduce bids for social contact (Nelson & Panksepp, 1998).

In sum, the pleasurable aspects of both increased oxytocin release and endogenous opioid system activity have clear potential to operate as conditioned stimuli when paired with an attachment figure (Lim & Young, 2006; Young & Wang, 2004). An attachment figure, who was initially a neutral stimulus, comes to elicit a strong biological response via the repeated pairing of pleasure induction and distress alleviation with this person. Sexual and other social contact elicits oxytocin and endogenous opioid activity, which when repeatedly paired with a specific person results in that person, above all others, emerging as an attachment figure. This framework is consistent with

evidence indicating that adults can have multiple attachment figures (e.g., Trinke & Bartholomew, 1997), typically organized hierarchically. The sexual and intimate behaviors that are characteristic of romantic partners should operate as a fast track toward a pair bond, but this does not preclude the closeness conferred in friendships and adult child–parent relationships from being associated with the same physiological reward and distress alleviation processes.

Attachment Attenuates the Human Stress Response

Oxytocin and the endogenous opioids are clearly implicated in the brain reward systems designed to reinforce social affiliative behavior in animals. In addition to the physiological reward systems activated by both caregiving and pair bond behaviors, evidence points to the important roles of oxytocin and the endogenous opioids in regulating the autonomic nervous system (ANS) and alleviating stress (Diamond, 2001; Insel, 2003; McCubbin, 1993; Uvnäs-Moberg, 1998). Most literature in this area focuses on oxytocin release during lactation (Carter & Altemus, 1997) and suggests that oxytocin-induced milk ejection is associated with decreases in sympathoadrenal activity (e.g., blood pressure and heart rate), cortisol levels, plasma level catecholamines, and enhanced activity of the vagus nerve, causing upregulation of parasympathetic nervous system activity in rats (Light, Smith, Johns, Brownley, & Hofheimer, 2000; Uvnäs-Moberg, 1997). It is hypothesized that the noted antistress effects of breastfeeding (e.g., calm, sedation, temporary changes in personality profiles) are integrated by centrally circulating oxytocin (Nissen, Gustavsson, Widstrom, & Uvnäs-Moberg, 1998; Pedersen, 1997).

Bonding behaviors reduce sympathoadrenal activity and can enhance parasympathetic–vagal activity. Uvnäs-Moberg (1998) noted that because it is widely known that the antistress effects of oxytocin are easily conditioned and become more pronounced after repeated exposures (see Insel & Young, 2001; Young & Wang, 2004), human memory and attachment representations may serve to reactivate physiological processes originally induced by positive social interactions. Thus, as adult pair bonds form, partners become internalized at the level of psychology and at the level of biology; when an adult is faced with a threatening situation, calling on a mental representation of an attachment figure can lead to psychological security and physiological calm. Diamond (2001) speculated that adults who "form and maintain secure, long-term attachments will experience greater cumulative exposure to centrally released oxytocin, eventually resulting in faster downregulation of [hypothalamic–pituitary–adrenal] reactivity and more

parasympathetically dominated patterns of ANS activity" (p. 287). This contention has received recent empirical support. Using a double-blind placebo controlled trial investigating whether oxytocin and social support work in conjunction to buffer stress responses, Heinrichs, Baumgartner, Kirschbaum, and Ehlert (2003) found that intranasally administered oxytocin attenuates cortisol responses in healthy men (women were not studied) during the Trier Social Stress Test; furthermore, the stress-buffering effects of the presence of participants' best friends were significantly stronger among those in the oxytocin administration group. Similar socially relevant effects were observed in a small pilot study by Meinlschmidt and Heim (2007), who demonstrated that intranasally administered oxytocin led to a decrease in cortisol (relative to placebo) among young men who had experienced an early parental separation but not among control participants who had not experienced these events. Other research in humans has demonstrated that increases in plasma oxytocin follow from warm contact among couples in the laboratory and that this holds for both men and women (Grewen, Girdler, Amico, & Light, 2005), and that intranasal administration of oxytocin increases feelings of trust (Kosfeld, Heinrichs, Zak, Fischbacher, & Fehr, 2005) and decreases fear and amygdala activation in males, which suggests one route through which oxytocin attenuates cortically driven appraisals of stress (Kirsch et al., 2005). Although data on this topic are limited and only newly emerging, the research on oxytocin is consistent with other work on stress buffering in humans. Gump and colleagues (Gump, Polk, Kamarck, & Shiffman, 2001), for instance, demonstrated that daily interactions with partners are associated with significantly lower levels of systolic and diastolic blood pressure compared with social interactions with nonpartners and time spent alone. In a similar study of 102 men and women, interactions with family members and spouses were associated with lower ambulatory blood pressure compared with other social interactions (Holt-Lunstad, Uchino, Smith, Olson-Cerny, & Nealey-Moore, 2003). In other primates, this pattern of downregulated arousal is well documented (Reite & Boccia, 1994). In infant rhesus monkeys, for example, ventral–ventral contact with the mother is associated with rapid decreases in both sympathetic–adrenal–medullary (SAM) and hypothalamic–pituitary–adrenal (HPA) axis activity (Suomi, 1999).

In reviewing this literature, a caveat on gender differences is in order. At this time, and with very little empirical data in humans, it is not clear whether the processes described above differ between men and women. From animal studies, there is clear evidence that oxytocin and vasopressin have gender-specific effects (Insel & Hulihan, 1995). In outlining their tend-and-befriend

model of stress response, Taylor, Klein, et al. (2000) reviewed evidence indicating that oxytocin output in the face of stress may be greater among females than among males, that androgens tend to inhibit oxytocin release, and that the estrogen-enhanced antianxiolytic effects of oxytocin are particularly pronounced (see McCarthy, 1995). The sexually dimorphic features of oxytocin and vasopressin may be associated with the development of different social behaviors (Carter, 2007), and there is little reason to regard pair bond development any differently. Although gender differences in the processes that underpin pair bond development are likely to be realized, such differences need not preclude a common end point of coregulated physiology. For instance, in their recent review of the neurobiology of pair bonding, Young and Wang (2004) noted distinct gender differences in the secretion of oxytocin and vasopressin in response to mating but were also clear in illustrating that both neuropeptides are connected to the dopaminergic reward system and thereby have reinforcing properties.

Attachment Involves Physiological Coregulation

Evidence from the literature on both animals and humans indicates that attachment relationships are correlated with distinct neurobiological responses. Over time, and primarily through the pleasurable aspects of adult sexual and other intimate behaviors, physiological systems associated with reward and distress alleviation provide the glue for an attachment relationship by associating a romantic partner with the experience of felt security. We argue below that attachment involves not only this state of conditioned stress buffering (i.e., the psychological and physiological experience of felt security in a time of stress), but also a more complex commingling of physiological states, or coregulation, whereby each individual within the relationship serves as the primary physiological regulator for their partner. From this perspective, individual homeostasis—both psychological and physiological—is maintained and regulated in the context of the relationship and by the presence and availability of one's attachment figure. Perhaps the best example of this process in humans is recent work by Feldman and Eidelman (2007), who demonstrate that mother–infant and father–infant gaze synchrony is associated with greater parasympathetic vagal tone in infants. When parents were better able to coordinate their gaze to their infant's, the infant evidenced higher levels of heart rate variability, which the authors described as reflecting greater autonomic maturity. Behavioral synchrony with caregivers has physiological benefits for human infants. We argue that in adults, coregulation emerges as an attachment forms and can be

defined more specifically as the ways in which one person up- or downregulates the partner's psychophysiological arousal. This regulation is a property of the relationship itself (not either individual alone) and can occur through any of several modalities (e.g., touch, smell, eye contact, cognition). Because we humans are a social species, other humans can buffer us from the potentially harmful biological effects of stress, and most evidence suggests that the better the relationship, the greater the protective benefit. This general protection or stress buffering, however, is not the same as coregulation, and we argue below that the latter is a defining feature of normative attachment, whereas the former is a necessary but not a sufficient element of clear-cut attachment relationship.

The notion of an interwoven physiology within social relationships—variously labeled *synchrony* (Feldman, 2007; Sander, Stechler, Burns, & Julia, 1970), *attunement* (Field, 1985, 1994), *social entrainment* (Ehlers, Frank, & Kupfer, 1988), or an "emergent property of the mammalian autonomic nervous system" (Porges, 1998, p. 837)—was first examined in detail by Hofer (1984, 1987, 1994, 1995; Polan & Hofer, 1999) in a series of programmatic studies with mother–pup rat dyads. On the basis of multiple experiments in which mothers were separated from their pups, Hofer (1987) offered a new understanding of the commonly observed biphasic separation response (i.e., a period of agitated "protest" followed by a period of passive "despair") by discovering that specific components of the mother–pup interaction regulated the infant rat's behavioral and physiological systems, and that different components of the interaction provided by the mother (e.g., warmth, nutrients, olfactory or tactile stimulation) regulated different behavioral and physiological systems in the pup independently. For example, by experimentally exploring cardiac and behavioral reactions to separation of the infant rats from their dams, the researchers revealed that the dam's warmth served as the primary regulator of cardiac activity (with no effects on behavioral responses) whereas the provision of milk prevented lethargy or inactivity (with no effects on cardiac activity). Hofer (1987, 1995) called these individual components "hidden" regulators because their specific functions were not evident until removed. He determined that the mother provided a combination of thermal, olfactory-alerting, and tactile stimuli that act in concert to exert a long-term control over the pup's behavioral responsiveness, providing both up- and downregulation of these systems. It is important to note that the distress resulting from removal of the components was directly attributable to their powerful regulatory properties and homeostatic mechanisms (Hofer, 1987).

The observation that the regulatory functions of relationships are hidden was, arguably, the most startling

aspect of Hofer's (1987) discoveries: Only when attachments are disrupted can the powerful, homeostasis-maintaining functions of the bond be observed. Beyond the study of dam–pup dyads, other research in the animal literature supports this observation. Disruption of attachment bonds in primates is associated with a similar pattern of physiological disturbance. In a wide range of studies on the separation responses of 4- to 6-month-old pigtail and bonnet monkey infants, both the initial agitation (expressed through frantic locomotor and vocalization efforts to reestablish contact with their mothers) and the ensuing phase of despair (expressed in disrupted sleeping and eating patterns, cardiac arrhythmias, reduced lymphocyte activation by mitogens, and reduced antibody response to a foreign protein antigen) are consistent with a disturbance of autonomic homeostasis precipitated by the separation (Reite & Boccia, 1994). These changes also are observed in isolation and separation experiences among infant rhesus monkeys, including upregulation of the catecholamine norepinephrine in cases of extended separation (Kraemer, 1992; Kraemer, Ebert, Schmidt, & McKinney, 1989). In human caregiver–infant dyads, both physical and emotional unavailability of the caregiver has similar effects, including decreased play, heightened autonomic activity, and disturbed sleep patterns in infants (Field, 1985). Based on the entirety of this literature, Reite and Boccia (1994) surmised, "We believe that separation has these consequences because one function of attachment is to promote concordant regulation of physiological and behavioral systems. The disruption of this attachment, then, permits dysregulation of the systems it supports" (p. 113).

DYSREGULATION: BEHAVIORAL AND PHYSIOLOGICAL RESPONSES TO SEPARATION AND LOSS

Hofer (1984) suggested that his detailed studies of coregulation in infant rats separated from their mothers could potentially shed new light on bereavement in human adults and, in particular, the commonly observed biphasic response to loss. He posited that because many human systems are under environmental control, it was plausible that bereavement resulted from "withdrawal of specific sensorimotor regulators hidden within the many complex interactions of the relationship that has ended" (p. 188). Why did Hofer believe this to be so? First, there was clear experimental evidence that this process operated across several mammalian and primate species (e.g., Hofer, 1987; Reite & Capitanio, 1985). Second, he noted the uncanny resemblance between the symptoms of human bereavement

and the dysregulating consequences of sensory deprivation and desynchronization of biological rhythms. These observations led him to conclude that romantic partners, like physiological regulators in the natural world, serve a similar homeostasis-maintaining function. For example, he observed that sleep disturbance, restlessness and anxiety, decreased food intake, and hallucinations were among the core symptoms of bereaved individuals and of individuals who were (in a series of military studies on the acute effects of social isolation) deprived of sensory stimulation and confined to light- and soundproof cubicles for days at a time (see Hofer, 1984).

Hofer (1984) also reviewed evidence from the field of chronobiology demonstrating that external physical stimuli, particularly light, play an important role in regulating mammals' biological rhythms and synchronizing circadian pacemakers (see Ehlers et al., 1988). In the absence of these physical regulators or, *zeitgebers,* humans' two major biological pacemakers run free, become disengaged from a 24-hour daily cycle, and ultimately become desynchronized. The resulting symptoms are, again, similar to the withdrawal symptoms of bereavement: sleep disturbance, malaise, decreased vigilance, depression, hostility, and cognitive impairment. Based on the similarity between the consequences of losing physiological regulators and the psychophysiological symptoms of bereavement, Hofer (1984) conjectured that the diverse responses to losing a partner could be most fully understood in terms of the removal of interpersonal regulators rather than the psychological stress of bereavement alone. In other words, the removal or loss of the regulatory components of the relationship disrupts the systems—physiological and psychological—that are maintained within homeostatic limits by the attachment bond, and the resulting state can be considered one of biobehavioral dysregulation.

Several investigators, in addition to Hofer (1984), have argued that regularly occurring social interactions may function as a *social zeitgeber* serving to regulate and synchronize bodily rhythms (Ehlers et al., 1988; Field, 1985, 1994; Reite & Capitanio, 1985). For mammals, repeated and regular contact with an attachment figure has the potential to maintain physiological homeostasis in a fashion that approximates potent nonsocial (environmentally mediated) regulators. Bowlby (1969/1982, 1980) contended that the attachment behavioral system, like other homeostatic systems, operates effectively only within certain limits. Outside this goal-corrected operational range, dysregulation occurs, and behavioral and emotional actions are elicited to preserve the bond, increase felt security, and restore emotional and physiological homeostasis.

Dysregulation: What Is It?

One immediate obstacle for understanding how dysregulation operates in adult humans is that the subtleties of separation and loss responses are rarely measured. Few human studies of loss provide detailed measurements of sleep disturbance, food intake, or temperature regulation, yet these are the precise systems Hofer (1984) proposed are regulated in the context of relationships. Instead, when physiological and health outcomes are considered, adults' reactions to loss are studied in terms of large-scale autonomic and neuroendocrine changes, indices characteristic of the mammalian stress response (e.g., Hall & Irwin, 2001). From the perspective of animal studies, dysregulation is not simply a biological stress response. To be sure, separation and loss experiences can and often do precipitate a stress response, but to fully understand normative attachment, it is critical to look closely at more-subtle biological responses that result from threats to felt security and the relationship itself. We argue that dysregulation results from the removal of the homeostasis-maintaining functions of relationships and can therefore range from mild affective, behavioral, and physiological disturbance (e.g., nonspecific malaise, psychomotor agitation, or disturbances in sleep architecture) to a full-blown stress response that includes prolonged activation of both the SAM and HPA axes.

Given the range of responses individuals experience when relationships are threatened or disrupted, dysregulation can be conceptualized in terms of two components that often but not necessarily co-occur: a disorganized response and an organized stress response. The former response is driven by the loss of coregulatory reward processes and is similar to a state of physiological withdrawal (see Insel, 2003). As described above, attachments form through a process of both pleasure induction and distress alleviation. The rewarding features of an attachment serve to entrain the physiological systems that are associated with pleasure and soothing, and this is how the physiological regulation of felt security occurs. On an experience of separation or loss, these systems run free and can lead to biological disorganization. Over time, as the distress of separation and loss sets in, a classic stress response can follow. Bowlby (1980) initially observed that children and adults move through two well-defined (though sometimes overlapping) stages following attachment disruptions. The first stage, which he called protest, is characterized by yearning search behavior, apprehensiveness, and a high level of arousal centered on reuniting a person with his or her attachment figure. When protest fails, withdrawal and despair set in, and this stage is typically characterized by psychomotor retardation, depression, and sadness. Both of

these responses, Bowlby believed, were stress reactions. In the model presented here, the disorganized/withdrawal response is conceptualized as a precursor to Bowlby's (1973) protest reaction, which is born of an emergency attempt to reunite an individual with an absent attachment figure.

Figure 1 provides a simple diagram for understanding this continuum of dysregulation. The x-axis is can be understood as threats to felt security. The regulation of felt security plays a large role in Mikulincer and Shaver's (2003, 2007) model of attachment dynamics, as well as other theories on the maintenance of close relationships, such a Murray, Holmes, and Collins's (2006) concept of *assurances* within their risk–regulation model. Within our continuum of physiological reactions, *disorganization* is the first phase or response to a separation experience. In contrast to the stress response, which is a highly organized reaction provoked by an environmental challenge (Chrousos & Gold, 1992), the less pronounced dimensions (e.g., restlessness, sleeplessness, nonspecific dysphoria) of dysregulation can be viewed as a disorganization of the physiological systems supported by the attachment relationship. This state of disorganization, emerging from the loss of coregulatory reward, is akin to a withdrawal reaction (Insel, 2003; Kovacs et al., 1998), which results in the free running of biological and psychological systems under homeostatic control. In animals, these symptoms often involve problems of extrapyramidal motor coordination, increased salivation, loss of body weight, core temperature fluctuations, and irritability (Kovacs et al., 1998). These effects are consequent to the development of physical dependence in which biological systems require external regulation. The removal of external regulation is not a stress response per se. Consider, for instance, a regular coffee drinker who delays morning caffeine intake. The known symptoms of caffeine withdrawal (e.g., headache, poor concentration, dysphoria, nausea, muscle stiffness) are not the immediate result of an HPA axis cascade. Continued withdrawal may precipitate an organized stress response, but it is important to recognize that the initial physiological consequences of removing a biologically conditioned stimulus are not a stress response.

As indicated in the leftmost panel of Figure 1, at relatively low levels of threat to felt security and the attachment bond, the resulting state includes generalized dysphoria, restlessness/agitation, disruptions of sleep architecture, changes in appetite and body temperature, and decreases in vagal tone/parasympathetic control of heart rate.[1] The physiological underpinnings of these responses result from the physical dependence and tolerance effects instantiated in the neural systems for reward (Bruijnzeel, Repetto, & Gold, 2004; Kelley &
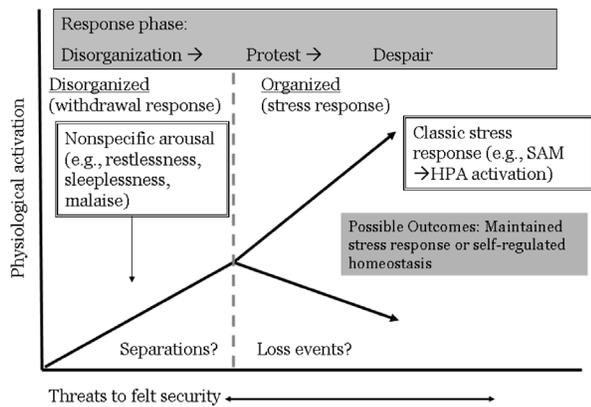
Figure 1   A continuum of biobehavioral dysregulation depicts the distinction between disorganized and organized responses to a social separation and loss experience. In contrast to the stress response, which is a highly organized reaction provoked by an environmental challenge, the less pronounced dimensions of dysregulation can be viewed as a disorganization of the physiological systems supported by the attachment relationship. This state of disorganization, emerging from the loss of coregulatory reward, is akin to a withdrawal reaction, which results in the free running of biological and psychological systems under homeostatic control. As threats to felt security increase, dysregulation becomes organized and can evolve into a classic stress response. The double-headed arrow along the x-axis indicates that the regulation of felt security is a dynamic process in constant operation (cf. Mikulincer & Shaver, 2007) and that threats to felt security can decrease, which moves people away from a loss-related stress response.

Berridge, 2002). From this perspective, differences are expected in how individuals progress through this state of disorganization to a more organized and full-blown stress response. Disorganization can be fleeting or nonexistent if, for instance, one learns that a partner has just died in a car accident, which would, presumably, be a major threat to the regulation of felt security and immediately provoke an organized stress response. Alternatively, the state of disorganization can last much longer when a partner is frequently absent, such as during business trips, military deployments, or marital separations. If marital conflicts disrupt the experience of felt security, we would expect disorganized responses to follow. As the probability of a true loss event increases, dysregulation can become organized by co-opting the primary components of the mammalian stress response. Bowlby (1980) explicitly recognized protest behaviors as an acute physiological stress response; the inability to restore the broken bond resulted in repeated priming of the attachment system, which can result in a state of chronic stress.

As noted, almost all of the human literature on social separations is represented in the right panel of Figure 1.

The disorganized responses that follow from less pronounced threats to felt security are difficult to study and rarely addressed. The only empirical test of these ideas in human adults is a recent study by Diamond et al. (in press), which investigated changes in behavior and affect associated with temporary physical separations among romantic partners. The results revealed a sharp increase in sleep disturbances for both members of a couple (the homebound and the traveling partner) during the travel period, and the researchers found that these disturbances quickly abated on reunion (Diamond et al., in press). Unfortunately, that study did not examine whether changes in felt security explained these associations; the model of dysregulation presented here holds that the observed magnitude of sleep disturbances during a separation period should be statistically mediated by changes in felt security. In the animal research, Hofer (1987) was able to make the coregulation–dysregulation connection in rats by experimentally removing a single homeostatic function of the dam (e.g., anesthetizing her, thus allowing only for the provision of body heat) and showing that such manipulations had a disorganizing effect on the pup's regulation. For obvious reasons, it is difficult to disentangle human reactions to loss from the stress of loss. No physiological experience of divorce or bereavement can be reliably separated from the stress induced by this event.

Given these conceptual and practical complications, why not simply consider all loss reactions as stress reactions and forgo the concept of disorganized dysregulation? The disruption of felt security and the subsequent loss of coregulation illustrate the powerful regulatory functions of an attachment relationship. Although the specific biological responses to separation and loss many not differ from reactions to other stressful life events (see our discussion of this matter later, under Empirical Question 5), a close study of disorganized responses suggests that true attachment relationships hold a unique and preeminent status in providing for the systematic regulation of physiological functioning. Once felt security becomes regulated within a specific relationship, biological systems of reward and distress alleviation are under homeostatic control. Conceptualizing a continuum of dysregulation allows for a demonstration of the subtle ways in which social separations can disturb this homeostasis and, as with Hofer's (1984) rats, this approach is the most powerful demonstration of coregulation. Moreover, the model of dysregulation proposed here distinguishes between responses characterized by the loss of reward (which disrupts homeostasis and leads to withdrawal-like symptoms) and the presence of a classic stress response. This distinction holds value for understanding the health consequences of separation experiences. The loss of coregulation permits the free running of the

biological systems that are involved in the regulation of felt security, and without a means of maintaining this set point, dysregulation can be maintained in time. As the loss of reward evolves into the onset of distress, an organized stress response can follow.

Organized Dysregulation: The Stress of Loss

Although the notion that humans move through discrete phases or stages following loss is questionable (Bonanno et al., 2002; Bonanno, Keltner, Holen, & Horowitz, 1995; Stroebe, Stroebe, Schut, Zech, & Bout, 2002; Vormbrock, 1993; Wortman & Silver, 1989, 2001), substantial evidence indicates that nonhuman primates and other mammals show biphasic behavioral reactions to separations, characterized first by active protest that slowly evolves into a passive despair (Hofer, 1994; Reite & Boccia, 1994; Seay, Hansen, & Harlow, 1962). Bowlby (1980) was explicit in contending that the behavioral and emotional reactions to separation and loss were manifestations of a physiological stress response, which was designed over the course of evolutionary history to solve environmental challenges and deal effectively with threatening situations. Despite Bowlby's (1980) conjectures, few analyses have considered the biphasic protest–despair sequence within the known parameters of the mammalian stress response (see Hennessy, Deak, & Schiml-Webb, 2001). In this section, we do so by describing the similarity between the behavioral reactions to separation and loss and the known (and highly organized) properties of the mammalian stress response. By connecting levels of analysis in this way, new perspectives on loss responses emerge. In particular, interactions between the psychology and the biology of loss responses suggest that reorganization and recovery are not driven exclusively by top-down changes in psychological adjustment to the separation event; many of the psychological and behavioral symptoms of grief (e.g., depression, social withdrawal, mental slowing) are known consequences of the inflammatory processes associated with a stress response. There is good reason to believe that biological responses themselves can lead to changes in behaviors, thoughts, and subjective emotional experience (e.g., Maier & Watkins, 1998). This perspective highlights the importance of integrating across levels of analysis and points to new ways of understanding attachment figure loss reactions.

When attachments are disrupted, protest and search behaviors all function to solve the adaptive challenge of separation by facilitating reunion and restoring felt security. Bowlby (1980) described these initial reactions as a state of emergency, and protest behaviors can be understood in the context of the first phase of Selye's

(1956) general adaptation syndrome (GAS), which he termed an alarm reaction. As mentioned previously, the two central outflows from the brain that control peripheral organs (including those of the immune system) are the SAM and HPA axes. During the alarm stage, the activation of the sympathetic branch of the ANS, primarily mediated through direct and indirect release of epinephrine and norepinephrine, results in a "fight or flight" behavior characterized by increased heart rate, blood pressure, and skeletal muscle activity designed to free fatty acids and promote glycogenolysis (i.e., the breakdown of glycogen) in the liver and skeletal muscles (Stratakis & Chrousos, 1995). Sympathetic activity is widely believed to be goal directed and motivating (Toates, 1995), and the alarm stage can confer benefits by quickly mobilizing bodily resources to respond to an adaptive challenge or threat (see Uchino, Smith, Holt-Lunstad, Campo, & Reblin, 2007). Though usually short-lived, these biological responses serve to maintain a state of agitated protest, which allows for search and recovery efforts. It is well known, however, that the prolonged activation of the autonomic and neuroendocrine systems associated with an alarm reaction also has negative consequences for health (Chrousos & Gold, 1992; McEwen & Stellar, 1993; Munck & Guyre, 1991; Uchino, Kiecolt-Glaser, & Glaser, 2000).

If alarm reactions fail to solve an immediate environmental demand, the body initiates a second or resistance stage of the stress response by working to regain homeostasis while continuing to deal with a challenge or threat. Selye (1956) argued that the HPA and glucocorticoid response activated during this stage are adaptive for countering stress by mobilizing energy stores.[2] Although the overproduction of glucocorticoids can downregulate immune responses and ultimately compromise health (see McEwen & Stellar, 1993; Stratakis & Chrousos, 1995; Uchino et al., 2000), resistance can be adaptive by promoting inflammatory processes (Hennessy et al., 2001; Leu & Singh, 1992; Sapolsky, Rivier, Yamamoto, Plotsky, & Vale, 1987). Recently, the understanding of the bidirectional stress–immune response has been advanced by studies investigating the production and activity of the proinflammatory cytokines (Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002; Maier & Watkins, 1998). Cytokines are protein substances secreted by immune cells to organize cellular interactions leading to antibody production and maximizing host defense against infection. As the HPA cascade is set into motion, corticotropin-releasing factor is synthesized by the hypothalamus and leads to cytokine production (Black, 2002; Hennessy et al., 2001; Larson & Dunn, 2001).[3] Negative emotions also are thought to induce cytokine activity by activating physiological stress systems (Kiecolt-Glaser, McGuire, Robles, &

Glaser, 2002). As the name implies, the proinflammatory cytokines promote inflammation (primarily through the secretion of interleukins) by altering metabolism and temperature regulation (Maier & Watkins, 1998). The global cytokine response operates to combat infection and injury and leads to what is often called sickness behavior or an acute-phase response (Dantzer, 2001), which is one of the body's main mechanisms for fighting invading microorganisms or defending against physical trauma and tissue damage (Black, 2002). Maier and Watkins (1998) posited that the beneficial symptoms of sickness behavior (e.g., fever, shifts in liver metabolism, increases in white blood cell count) represent an evolved pattern or central motivational state designed to enhance the immune response and conserve energy for fighting infection. It is important to note that psychological stressors and associated cognitions can activate the same neural immune circuitry as infectious agents can, and they also lead to sickness behavior characterized by fever, increased sleep, reduced activity and exploration, depressed mood, and cognitive distortions (Maier, Grahn, & Watkins, 1995; Maier & Watkins, 1998).

How does all this relate to the physiology of loss? In both animal and human studies, the similarities between sickness behavior and passive withdrawal following loss are substantial. Hennessy et al. (2001) argued that the psychological state of "despair" poses an interpretive dilemma for understanding the common elements of this passive response because similar responses are observed across a wide range of animal taxa, including both primates and nonprimate mammals. Because it is unlikely that guinea pigs, for example, possess the cognitive abilities commonly associated with despair in humans (e.g., pining, realization that loss is permanent), a more parsimonious explanation for the commonality in reactions between guinea pigs and adult humans may be that deactivated, withdrawal reactions to loss are mediated primarily by stress-induced sickness behavior. Indeed, Hennessy et al. (2001) noted that "behaviors typical of primate infants during the second stage of separation such as reduced social behavior and general activity as well as the assumption of a characteristic crouched or hunched stance are classic sickness behaviors" (p. 78). Social withdrawal, cognitive alterations and depressed mood, disturbed sleep, general weakness, and heightened endocrine activity are all characteristic of both sickness and loss reactions (see Hofer, 1984; Maier & Watkins, 1998). Moreover, malaise, fatigue, and apathy are among the cardinal symptoms of both depression and sickness (Dantzer, 2001; Dantzer, Wollman, & Yirmiya, 1999; Meyers, 1999). With respect to grief, the malaise, depression, lethargy, and apathy that typically follow loss may thus be reinterpreted in terms of the functional value of sickness (see Hennessy et al., 2007;

Schiml-Webb, Deak, Greenlee, Maken, & Hennessy, 2006). From this perspective, grief responses can be considered part of a state that aids in physiological reorganization and recuperation during times of stress. The evidence for this argument is only newly emerging and comes mainly from animal studies. Although the research on inflammatory processes suggests that the physiology of loss reactions may drive the observed psychological and behavioral response that frequently follows attachment disruptions, human data are needed before definitive conclusions can be drawn.

Although HPA activity can initially enhance immune system activity (Dhabhar & McEwen, 1996) and cytokine-induced sickness can be highly adaptive for mounting defensive responses, the neuroendocrine cascade initiated by the HPA axis and resulting in corticosteroids (especially cortisol in humans) can inhibit many aspects of cellular immunity when it is chronically activated (Bremner & Vermetten, 2001). Over time, as endocrine and cytokine activity persists, one avenue through which the known increases in morbidity following loss result is impaired immune functioning (Kiecolt-Glaser et al., 2002). From this perspective, the physiological reactions to loss experiences may be best understood in terms of an acute-phase response that can slowly evolve into compromised immune functioning via the necrotic effects of stress hormones on immune organs. These effects may be especially evident when attachment disruptions involve threats to one's social standing. Recent meta-analytic findings (Dickerson & Kemeny, 2004; Miller, Chen, & Zhou, 2007) indicate that threats to the social self are positively correlated with HPA responses. Variability across this dimension is a key moderator of biological responses to loss events.

Several empirical studies have documented the immune consequences of social separations, but most of this research is not wed to a larger theory about the nature of attachment and loss reactions. Divorced adults who continue to struggle with their separation evince significantly higher antibody titers to Epstein–Barr virus and a lower percentage of natural killer (NK) cell activity, both of which indicate compromised immune functioning (Kiecolt-Glaser et al., 1987, 1988; Powell et al., 2002). A much larger literature indicates that the death of a partner is associated with a range of disrupted endocrine and immune functioning, increasing susceptibility to illness following partner loss. Hall and Irwin (2001) concluded that a close relation exists between nonsuppression of cortisol and bereavement, suggesting that bereavement-related stress may precipitate dysregulation of the HPA axis (Bartrop, Luckhurst, Lazarus, Kiloh, & Penny, 1977; Irwin, Daniels, Risch, Bloom, & Weiner, 1988; Irwin, Daniels, Smith, Bloom, & Weiner, 1987; Jacobs et al., 1986, 1987). In response to a loss

experience, Goodkin et al. (1996) proposed that decrements in NK cell cytotoxicity activity emerge early and even before loss experience and are primarily mediated by the SAM axis, whereas later-emerging immunological changes, such as decreased mitogen responses, are driven by the combined activation of the SAM and HPA axes and emerge more slowly. Kemeny et al. (1995) also reported substantial changes in immunological parameters among 39 gay men whose partners had died of AIDS in the previous year, compared with age- and serostatus-matched nonbereaved men.

SELF-REGULATION AND PSYCHOPHYSIOLOGICAL RECOVERY

In the research described above, what is the common denominator for understanding why some people succumb to dysregulating effects of a loss experience but other people quickly regain their normative levels of functioning? In this section, we attempt to answer this question and argue that the primary task for successful recovery following attachment figure loss is adopting a behavioral, emotional, or cognitive strategy that attenuates the physiological consequences of the relationship disruption. This perspective emphasizes the underlying function of any given regulatory strategy rather than the manifest content of one's actions, thoughts, or feelings. Thus, whereas coregulation can be defined by the maintenance of physiological homeostasis within the attachment relationship (and rests within the systemic functioning of the dyad), adaptive self-regulation hinges on individuals' finding a way to provide themselves the regulatory functions typically conferred by an attachment relationship. Bowlby (1980) described the end point of successful mourning as a psychological reorganization of one's thoughts and feelings about a lost attachment figure (see Mikulincer & Shaver, in press). In many ways, the process of reorganization is consistent with a model of gradual extinction through which the regulatory benefits conferred by mental representations of the relationship slowly diminish. One's attachment figure is no longer available to alleviate distress and induce pleasure; thus, although it may be an effective short-term strategy to call on representations of a former partner to do so, it is critical to establish a new means of regulating felt security (or re-establish old means of doing so; e.g., Vormbrock, 1993). Reorganization can happen in many different ways. For instance, the gradual reappraisal of a loss or separation as less threatening can restore one's sense of individually maintained felt security. Similarly, felt security can be restored by reaching out to others for support; to the extent that social

coping mechanisms allow individuals to adjust their perceptions of a situation, feel psychologically calm and secure, and reestablish physiological homeostasis, social support enhances self-regulation (cf. Cohen & Wills, 1985). Maladaptive responses to loss, in contrast, maintain dysregulation by not providing a suitable means of downregulating the physiological dysregulation that follows from threats to felt security. This section focuses on the strategies that counteract the dysregulation associated with losing one's primary external regulator. In short, how do people deal with the loss of coregulation?

An array of strategies have proven adaptive following loss experiences (Bonanno et al., 1995, 2002; Stroebe & Schut, 1999; Wortman & Silver, 2001). Some people "work through" the pain of grief, finding meaning and support through disclosing their most intimate feelings; others avoid negative emotion altogether. Some people invoke problem-focused coping; others engage in more emotion-focused strategies. Recently, Mikulincer and Shaver (in press) proposed that adaptive coping responses (i.e., those that work for achieving a reorganization of the loss) involve a combination of hyperactivating and deactivating strategies that help people titrate their engagement with and detachment from the painful feelings that accompany a loss (also see Stroebe, Schut, & Stroebe, 2005). All of these regulatory strategies can be viewed as adaptive if they are conceptualized as serving the same underlying purpose: restoring individuals' sense of felt security and downregulating states of physiological dysregulation. This perspective is consistent with the concept of *equifinality,* which is commonly used in developmental psychopathology literature to describe the ways in which different start points can converge on a single outcome (e.g., Cicchetti & Rogosch, 1996). In cases of attachment figure loss, the equifinal outcome of interest is the restoration of individually maintained felt security, and the functional utility of one's coping responses for achieving this end point, rather than the overt content of the behaviors, is the most important aspect of self-regulation (regardless of whether coping involves support-seeking, emotional venting, cognitive reappraisal, or simple avoidance).

Regulatory Strategies

Bowlby's (1980) observation that the natural course of mourning, when uncomplicated, is to move from a state of agitated distress to one of reorganization implies that an underlying restorative process facilitates this transition. Most nonhuman primates, children, and adults ultimately adapt to loss and do so without getting stuck in states of pathological grief (Amato, 2000; Bonanno, 2004). A variety of cross-species regulatory

strategies have been observed to quell the physiological dysregulation of an attachment figure loss. Various modes of self-regulation were evident in Harlow's monkeys (Seay et al., 1962; Seay & Harlow, 1965), including rhythmic rocking and other self-soothing behaviors. Bowlby (1973) described the work of Kaufman and Rosenblum (1967), who observed that infant pigtail monkeys separated from their mothers gradually resumed their affiliative and exploratory behavior (typically within a week of separation). Depressed behavior abated as the monkeys started interacting with peers (see Bowlby, 1973). Infant bonnet macaques who were separated from their mothers with an alternative juvenile attachment figure (friend support) evidenced fewer behavioral indices of dysregulation and consequently were buffered against the negative immune changes observed in infants not accompanied by (or housed with) juvenile friends (Boccia et al., 1997). Both the supported and nonsupported infants increased allomaternal behavior (including increased ventral contact), suggesting that the infants attempted to alleviate the distress of maternal separation through social support that mimicked the comfort provided by mothers (Boccia et al., 1997). Similar results are observed for young adult rhesus monkeys; Suomi, Eisele, Grady, and Harlow (1975) reported that monkeys separated from their nuclear families who were housed with friends were relatively unaffected by the separation in comparison to individually housed monkeys who exhibited a host of depressive-like behaviors. Although it presumed that these regulatory strategies all service the underlying function of reducing environmental threat (i.e., restoring felt security), the animal research provides little information to this end; on this topic, human studies provide more useful information.

Adult humans also invoke a variety of strategies to cope with the distress of loss. Seeking support from other social partners and moving toward the establishment of new romantic relationships are among the most reliable predictors of postloss adjustment following both partner death and divorce (see Goodkin et al., 2001; Hetherington & Kelly, 2002). During both wartime- and job-related routine marital separations, homebound spouses (typically women) sought increased social support from family members, and increased connectedness with family is associated with better overall adjustment (Vormbrock, 1993). Social support is among the most effective sources of both psychological and physiological stress buffering (Robinson-Whelen, Kim, MacCallum, & Kiecolt-Glaser, 1997; Uchino, Cacioppo, & Kiecolt-Glaser, 1996), and it is likely that one of the key psychological benefits conferred by supportive relationships is

providing an individual with a sense of control over seemingly uncontrollable events (Cohen & Herbert, 1996; Goodkin et al., 2001; Kiecolt-Glaser et al., 2002). Taylor, Kemeny, et al.'s (2000) tend-and-befriend model suggests that women are more likely than men to engage in affiliative behaviors in the face of stress. This gender difference is hypothesized on the basis of differences in oxytocin levels in the face of stress: Estrogen enhances the effects of oxytocin, which may operate to selectively influence women's tending and befriending behaviors. The tend-and-befriend analysis suggests that oxytocin can signal both positive relationship states and the need for repair behaviors, some of which may fail and lead to maintained stress responses over time (for a description of the positive correlation between oxytocin and stress responses, see Taylor, 2006). Overall, this analysis indicates that when attachments are disrupted or permanently severed, adults seek alternative routes for the provision of security by rekindling filial attachments or seeking to establish new attachments. Support seeking may be particularly common among women (Taylor, Kemeny, et al., 2000).

How adults cognitively adapt to attachment figure loss plays a critical role in either maintaining distress or restoring psychophysiological homeostasis (Kemeny, 2003; Taylor, 1983). The notion of cognitive adaptation suggests a process that moves individuals from a state of cognitive uncertainty to feeling more control, optimism, and self-efficacy (Helgeson, 2003). This process, often involving the search for meaning, is among the best predictors of physiological arousal modulation following stressful events in general and loss experiences in particular. Fundamentally, the regulation of felt security is a subjective, appraisal-based coping strategy (see Mikulincer & Shaver, 2007), and there exist multiple routes to achieving this end point. Humans disarm nonphysical threats by creating meaning and coherence from a difficult experience. For example, following divorce, a prolonged period of emotional mourning may help some adults reorganize their thoughts and feelings about the separation, which, in turn, helps them downregulate concomitant physiological arousal. Others, in contrast, may avoid strong emotions and say, "It's over and done with; let me just get on with life and forget about this mess." Unless this person is suppressing strong emotional reactions, which is associated with increased autonomic arousal (Gross & Levenson, 1993), this reaction is as adaptive as any deeper form of mourning (provided it is effective in extinguishing physiological dysregulation). In the remainder of this section, we review two areas of study pointing to the importance of cognitive adaptation for regulating the physiological effects of loss.

Adaptive Responses: Cognitive Processing, Narratives, and Meaning Making

Among the many possible strategies for coping with stressful life events, the study of cognitive processing, or how individuals think about, come to understand, and appraise their social disruption experiences, has received considerable research attention. Several studies have found that the positive beliefs typically associated with control are linked to positive physiological states (Futterman, Kemeny, Shapiro, & Fahey, 1994; Segerstrom, Taylor, Kemeny, Reed, & Visscher, 1996; Sgoutas-Emch et al., 1994; Sieber et al., 1992; Taylor et al., 1992). Individuals with a sense of personal control have more social support and may be more effective in mobilizing social support during times of stress (Taylor & Brown, 1994). In a study of HIV-positive bereaved men, positive expectations regarding one's health, feelings of confidence and optimism, and greater perceived control over one's disease were associated with a slower progression of HIV-related illness (Reed, Kemeny, Taylor, & Visscher, 1999). Alternatively, numerous laboratory studies have found that both experimentally induced unpredictability and low control, as well as stable individual differences in personal mastery, affect physiological arousal to acutely stressful events (Pham, Taylor, & Seeman, 2001; Thompson, Cheek, & Grahma, 1988).

Work on the development of narratives following loss and other stressful life events suggests that individuals who are able to construct an organized and coherent account of a painful event benefit because thoughts and feelings can be more fully integrated (Capps & Bonanno, 2000; Neimeyer, 2000; Pennebaker, Mayne, & Francis, 1992; Pennebaker & Seagal, 1999; Stein, Folkman, Trabasso, & Richards, 1997). In this sense, a seemingly meaningless and complicated event can become understandable and simplified through narrative (Affleck & Tennen, 1996; Affleck, Tennen, Croog, & Levine, 1987; Bower, Kemeny, Taylor, & Fahey, 1998; Folkman, 2001; Taylor, Kemeny, Reed, Bower, & Gruenewald, 2000), which renders the loss experience less threatening to felt security. Bower et al. (1998) reported that finding meaning by making a major shift in values, priorities, or perspective in response to loss was associated with less rapid immune declines and rates of AIDS-related mortality among 40 HIV-seropositive men who had lost a close friend or partner to AIDS within the previous year. For these men, finding meaning following AIDS-related bereavement proved biologically protective, buffering against stress-related changes in the ANS and HPA axis (Bower et al., 1998). In the wake of attachment figure loss, finding meaning may induce a state of peacefulness or calm that has salutary effects on the ANS and is thereby physiologically protective (Kemeny, 2003; Taylor, Kemeny, et al., 2000).

Distress-Maintaining Mechanisms

In contrast to the salutary benefit of successful meaning making, evidence indicates that several regulatory strategies maintain or augment physiological arousal following separation and loss. Kemeny and Gruenewald (2000), for instance, have found that relative to adaptive grief responses (which do not involve depressive symptoms other than sadness), bereaved individuals who reported elevated depression were more likely to show immune changes consistent with more rapid HIV progression. In particular, their studies indicate that the self-reproach aspect of depression is a significant predictor of immune system decline in a bereaved, HIV-positive sample, although other components of depressed mood, such as sad affect, confusion, and sleep disturbance, are not (Cole & Kemeny, 2001). These findings are consistent with other work by Kemeny demonstrating that negative beliefs about the future predicted shorter survival time among men with AIDS (Reed et al., 1999), that men with stable negative self-attributes (i.e., attributing negative life events to one's personal character) were more likely to demonstrate immune system declines over time (Segerstrom et al., 1996), and that HIV-positive gay men who were particularly sensitive to social rejection showed an accelerated time to an AIDS diagnosis and to mortality during a 9-year follow-up period (Cole, Kemeny, & Taylor, 1997). The relations between these negative cognitive states and immune functioning were not mediated by generalized distress of negative mood, suggesting that negative beliefs have biological correlates apart from their association with emotional distress (Kemeny & Gruenewald, 2000). Numerous other studies indicate that dysphoric cognitive–affective rumination is particularly maladaptive for dealing with social loss and other stressful events. Individuals who focus on thoughts and feelings associated with depressive symptoms and on the causes and consequences of these symptoms report exacerbated and prolonged distress following both naturally occurring and laboratory-induced stressful events (Lyubomirsky & Nolen-Hoeksema, 1993; Nolen-Hoeksema, 2001; Nolen-Hoeksema, McBride, & Larson, 1997; Nolen-Hoeksema, Parker, & Larson, 1994). It is clear from this analysis that negative psychological states exert a powerful effect on maintaining physiological dysregulation. What is not yet clear is the extent to which these states are correlated with disturbances in felt security. We presume that psychological variables such as the fear of social rejection interrupt felt security, but future research needs to measure the ways in which changes in felt security are correlated with negative appraisals of a loss experience, as well as the consequent physiological responses.

LOOKING FORWARD: AN EMPIRICAL AGENDA FOR THE STUDY OF NORMATIVE ADULT ATTACHMENT, SEPARATION, LOSS, AND RECOVERY

Although there was a clear need to integrate research on attachment, loss, and the recovery process, the model proposed here is incomplete, largely because much of the available evidence in support of coregulated attachment and biobehavioral dysregulation comes from animal studies. Human studies on these topics are needed, and the primary goal of this article is to spur empirical advances. In this section, we discuss six questions emerging from this analysis, all of which are open for continued study and empirical debate. Answers to these questions will provide deeper insight into precisely how normative adult attachments operate, why social attachments confer physical health benefits, and the pathways through which individuals adapt or succumb to the dysregulating effects of attachment figure loss.

1. Is coregulation adaptive for adults?

Many of human infants' self-regulatory capacities hinge on the presence and behaviors of a caregiver (e.g., soothing, feeding, temperature regulation), and there are obvious benefits of having linked biological systems whereby infants' physiology can be synchronized and quickly attuned to the biology of a caregiver: There is an immediate adaptive value to coregulation in this context (Hazan & Diamond, 2000). Because adults are entirely capable of independent physiological self-regulation regardless of whether they are in or out of a relationship, an obvious question emerges: What is the adaptive value of having commingled physiology within adult pair bonds? If coregulation emerged in the context of a highly dependent infant–caregiver relationship, a reasonable question is whether this process was simply preserved over the course of evolutionary history within any attachment relationship. In this sense, coregulation within adult relationships may be a vestigial by-product of the infant–caregiver attachment system.

There is reason to believe that the by-product argument is false. One interesting way of thinking about the adaptive value of coregulation within adult attachment relationships was recently discussed in a study by Coan, Schaefer, and Davidson (2006). Using functional MRI, this study investigated neurophysiological responses to environmental threat and demonstrated that holding the hand of one's partner, relative to a stranger or the waiting-alone condition, attenuates the neural response to the threat condition; perhaps more strikingly, the degree of neural threat response varied according to

reported marital quality. The Coan et al. study was largely about stress buffering and did not focus explicitly on coregulated attachment; however, in discussing why social attachments attenuate neural responses to threat, these investigators reasoned that the adaptive value rests in the ability of the threatened person to borrow from the partner's emotional and physiological stability. Rather than using top-down reappraisal or some other intra-individual regulatory strategy, the ability to quickly use the resources of a close other may represent a so-called fast route of emotion regulation. In a recent review of the neuroscience of attachment, Coan (in press) describes this process in terms of a *social baseline model* positing that social regulation provides the most efficient and metabolically cost-effective means of regulating affect. Coregulated physiology describes the underpinnings of this process and can provide adaptive value by enabling individuals the ability to quickly regulate affective distress by synching their physiological systems to their attachment figure. If this hypothesis is correct, studies can be designed to determine whether dyadic emotion regulation is indeed less effortful and more automatic than individual emotion regulation. It is true that adults are fully capable of intra-individual emotion regulation; however, relationships—and attachment relationships specifically—may enhance emotion regulation capabilities and provide a less effortful route for downregulating affective distress.

2. What empirical evidence would support the existence of coregulation in humans?

The first portion of this article offers a strong and empirically testable hypothesis: Although stress buffering (i.e., the downregulation of psychophysiological arousal) is observed in many social contexts and varies largely with the degree of relationship closeness, coregulated physiology is an emergent property of an attachment relationship and, therefore, should vary according to the extent to which individuals report using their partner as a true attachment figure (see Hazan et al., 2004). If a relationship involves clear-cut attachment behaviors, then coregulation should follow. One feasible and straightforward way of testing this hypothesis would be to model the physiological functioning (e.g., indices of cardiovascular responses) of each person in a relationship as a bivariate system in which changes in one person's physiology (in response to any task demands) are dependent on, not only their own prior physiological state, but their partner's prior physiological state as well.

In studies designed to assess coregulation, the key parameter of interest is the extent to which the degree of normative attachment (measured via an instrument such as Hazan & Zeifman's [1994] WHO-TO scale, which

asks individuals to rate the degree to which they use their partner to meet a variety of normative attachment needs) predicts the coupling between each individual's physiological responses to a task demand. Analyses of this kind can be accomplished using a variety of currently available tools, such as cross-lagged regression models, Kenny and colleagues' (Kenny, Kashy, & Cook, 2006; Laurenceau & Bolger, 2005) actor–partner interdependence model, structural equation growth models (Newsom, 2002), or more-recent advances such as bivariate latent difference score modeling (Ferrer & McArdle, 2003; McArdle & Hamagami, 2001). The latter approach represents a particularly innovative means of assessing coregulatory influences because changes in one person's physiological states can be modeled as a function of the partner's physiological state at the prior occasion. If this coupling of physiological responses varies according to degree of reported attachment to a romantic partner, this information would provide strong evidence that attachment (rather than relationships in general) involves physiological coregulation. Research of the kind described here also is important for understanding how coregulation operates across multiple attachment relationships in adulthood. Evidence suggests that adults have multiple attachment relationships that vary in the degree to which they fulfill all attachment functions (i.e., the extent to which they are full-blown attachments; Doherty & Feeney, 2004). Again, to the extent that indices of coregulation vary according to degree of attachment, synchrony should be evidenced across multiple adult attachment relationships, albeit in varying degrees.

In keeping with the large animal literature suggesting that coregulatory influences are "hidden" and not fully evident until removed (Hofer, 2006), human studies can follow suit in two primary ways. First, the available animal evidence provides reason to believe that the coupling processes described above may not be evident until the attachment system is phasically activated by some type of environmental challenge or threat. This is the precise methodology employed by Coan et al. (2006); it was expected that the neurophysiological effects of holding a partner's hand would be evident only under a condition of threat. A key assumption of infant attachment research is that the functional components of the attachment system are best observed by tasks that activate and potentially stress the system (Ainsworth et al., 1978), and the same logic can be applied to studies of adult relationships: If you want to see the normative components of adult attachment, develop paradigms that evoke attachment behaviors. Within-person studies examining the coupling of physiological systems under resting baseline and threat conditions are uniquely suited to evaluate the hypothesis

that coregulatory processes are not evident until the attachment system is phasically active. Second, human studies need to develop selective "knockout" paradigms that conceptually replicate those used in the animal literature. This is one of the primary challenges for demonstrating coregulation in humans. Hofer's studies (e.g., Polan & Hofer, 1999) are particularly elegant because they selectively remove one component of the rat dam's physiological functioning (e.g., the dam's body temperature) and track the causal response in the pup (e.g., cardiac activity). Investigators have argued that conceptually similar studies would be difficult in adult humans because individuals need only to invoke mental representations of an attachment figure to achieve the regulatory benefits conferred by the relationship (see Uvnäs-Moberg, 1998). However, Simpson, Rholes, and Nelligan (1992) developed an experimental paradigm similar in several respects to the laboratory procedure created by Ainsworth et al. (1978) to assess infant attachment, and variants of this approach can be used to selectively manipulate the degree of attachment figure availability when individuals are faced with a stressful task. Another approach to addressing this issue would be to put adults under some form of cognitive load during an attachment-related threat. The cognitive load would interfere with the ability to invoke security-providing mental representations of an attachment figure (cf. Mikulincer, Gillath, & Shaver, 2002). If individuals' ability to sync their physiology to an attachment figure's physiological state is mediated by the ability to call on a mental representation for felt security, dysregulation should be observable once individuals are under cognitive load. The same logic can be applied to selectively knock out other aspects of the adult attachment system, including the ability to physically see or smell an attachment figure.

3. In the model proposed here, coregulation is described as a conditioned physiological state defined by the reciprocal maintenance of psychophysiological homeostasis within a relationship. What are the defining features of homeostasis?

It is not uncommon for research on personal relationships to argue that relationships are systems and, as such, include many of the basic elements of other naturally occurring dynamical systems, although many of the propositions emerging from this perspective have been difficult to document empirically (see Boker & Laurenceau, 2006). The model of coregulation presented here offers testable hypotheses about how these dynamics may operate as attachment relationships emerge.

Two elements are critical for understanding how attachment relationships maintain the physiological equilibrium, or homeostasis, for a given individual. The first element is a demonstration that the physiological responses of each person in the relationship can, in fact, be mathematically modeled as a single system rather than individual systems that are correlated over time. From this perspective, in a hypothetical attachment relationship, person A's physiological responses at any given time are not merely associated with person B's physiological responses; instead, they are dependent on person B's functioning. (Stress buffering, in contrast, would be evidenced by an attenuation in the response to stress by the presence of a supportive other; in this case, person A's physiology is not dependent on person B's, but the magnitude of the stress response is limited by B's presence.) As stated above, the key test here is one of standard statistical moderation: Do these processes operate differently based on degree of reported normative attachment? The second element is a demonstration that one person's physiology can serve as an attractor or set point toward which the system can evolve over a prolonged time. When one partner experiences an environmental or psychological stressor, the partner's physiology may serve as the set point for restoring a state of resting equilibrium. This set point and associated compensatory responses are among the hallmarks of homeostatic systems (Bernston & Cacioppo, 2000). Set-point ideas can be examined in the context of dyadic psychophysiological laboratory studies in which one person faces a threatening or stressful task in the presence of the partner (cf. Coan et al., 2006). If, for instance, cardiovascular responses are recorded from both individuals, a variety of models can be specified to examine whether the physiological responses of person under threat are attenuated by the partner's physiology. A stress buffering model would hold that significant others will limit the degree of observed reactivity. From the coregulatory perspective, the parameters of interest would be the degree to which the physiological responses of a person under threat covary or move toward their partner's physiological responses. For instance, relative to couples who have not yet become attached, does the threatened person's heart rate demonstrate a faster recovery toward the partner's heart rate after the stress has passed? This is only one approach to illuminating coregulatory set-point ideas, and many more await development. Of course, it is possible that there is nothing unique about adult attachments beyond stress buffering alone, but until this is demonstrated, the available evidence suggests many reasons to believe that coregulation exists. Regulatory set points and individual physiological responses considered as part of a single system are measurable dimensions of an attachment relationship that can be mathematically defined and modeled using dyadic social psychophysiological laboratory studies (Gottman, Swanson, & Swanson, 2002).

4. When does dysregulation emerge? Is there a difference in the expected physiological responses when a partner is psychologically unavailable and when there is an actual physical loss?

Bowlby (1980) suggested that behaviors designed to restore the attachment bond would emerge following any real or perceived separation or loss experience. As a separation experience evolved into a full-blown loss, an emergency stress response should follow (Bowlby, 1980). Understanding of the boundaries between what we call disorganized and organized dysregulation will be advanced by studies that measure, not only the extent to which person A is attached to person B, but also the extent to which person A appraises person B's behaviors as enhancing felt security and, in cases of true separation and loss experiences, the extent to which individuals feel they have developed strategies for meeting those needs outside the relationship. When person A is highly attached to person B but also reports that person B does not or cannot meet A's attachment needs (e.g., items such as, "I would very much like to turn to my former partner during periods of stress, but I don't feel she can comfort me when I am upset"), there should be a consequent loss of coregulation and dysregulation to follow. This type of finding may be observed among dissatisfied couples who have become attached but are no longer capable of regulating each other's sense of felt security—psychologically or physiologically.

The magnitude of the difference between wanting to use a partner or ex-partner as a secure base and feeling that one can in fact do so should predict (a) the extent of coregulated physiology in the context of a dyadic measurement paradigm and (b) the extent of physiological dysregulation in response to some type of attachment-related threat. The larger the discrepancy between these indices, the greater the loss of regulatory benefits conferred by the relationship. In cases of actual physical loss, dysregulated responses would be associated with the degree of continued attachment to a former partner and the extent to which individuals report having a strategy in place that leads to individually maintained felt security (e.g., items such as, "Since this loss experience, I feel as if I have a good means of calming myself down when I am distressed"). It is easy to envision a situation in which individuals remain highly attached to a former partner but also have a clear strategy for calming themselves in times of distress—that is, they would

like to turn to the ex-partner but have found other effective means of coping with an environmental challenge. On the other hand, someone may remain highly attached to a former partner without an efficient, functional means of meeting any of those attachment needs. The ideas outlined here provide a handful of testable means of studying dysregulation, as well as potential boundary conditions between psychological unavailability and true loss experiences.

5. How is the dysregulation that follows a real or perceived social loss different from the dysregulation that might accompany any other routine disruptions (e.g., a job loss, a major move, a day without light exposure)? Are attachment relationships different from any other form of environmental regulation of biological rhythms?

The main goal of this article was to underscore the breadth of evidence suggesting that mammalian biological systems are open to external regulation and that one of the most potent of these regulators is another member of our species to whom we are closely attached. From the cradle to the grave, attachment relationships involve the regulation of felt security; attachment relationships provide what is arguably the most efficient and metabolically effective context for affect regulation (Coan, in press; Diamond & Hicks, 2004). This fact suggests that, relative to other major life events, attachment disruptions represent a fundamental threat to how felt security will be regulated, which can lead to a potent dysregulation of the physiological systems associated with a state of calm and well-being. Understanding how coregulation operates at the level of physiology not only sheds light on the normative functioning of attachments but also calls for a reframing of how we view responses to loss. When considering separation and loss experiences, the literature reviewed here suggests that understanding the loss of coregulation is at least as important as understanding the onset of a stress response. We have argued that a complete account of separation and loss includes a consideration of both disorganized (loss of coregulation) and organized (onset of a stress response) reactions to the separation experience. Although it is likely that any routine disruption can be understood from this perspective—as long as it is clear what two elements in a system are regulating each other and what biological processes are associated by this regulation—it is unlikely other classes of routine disruptions have as potent an influence on felt security. Job loss, for instance, may disrupt circadian rhythms because of the regulation conferred by a daily schedule and a habitual routine. Moreover, the loss of a job can be major adaptive challenge associated with many psychological stressors that will likely lead to an organized biological stress response. As with a relationship loss, a distinction can be made between the removal of the regulatory benefits of the one's daily routine (associated with the job) and the onset of a biological stress response (following from the perceived demands associated with the job loss). It is important to note that we have argued that it is the regulation of felt security that will prove most critical in predicting the magnitude of the consequent physiological dysregulation. Because attachment relationships provide a unique context in which felt security is regulated, loss experiences are among life's most potent dysregulators. Understanding ways in which attachment disruptions are similar to and different from other routine disruptions will cast a brighter light on the boundaries between the loss of coregulation and the onset of a stress response.

6. Where do individual differences fit into the proposed framework?

The role of individual differences is an inescapable question and one that will prove very useful for extending understanding of the normative processes described above. A vast body of evidence indicates that adult attachment styles play a critical role in how individuals seek to maintain felt security when proximity seeking is not a viable option (Mikulincer & Shaver, 2007), and there is clear evidence that individual differences play a role in coping with loss experiences (Mikulincer & Shaver, in press). Moreover, new research suggests that attachment styles moderate the neural responses to threat observed in the Coan et al. (2006) study described above (see Coan, in press) and that adults high in attachment anxiety evidence greater HPA axis activity during short-term separation experiences (Diamond et al., in press). As this area of study grows, answers to four primary questions will be especially informative. First, are there gender differences in coregulation? Marriage confers different health benefits for men and women (Kiecolt-Glaser & Newton, 2001), and basic questions can be asked about whether men and women differ in the ways in which their partner serves as a physiological regulator. In addition, attention should be paid to gender differences in self-regulation. Taylor, Klein, et al.'s (2000) tend-and-befriend model suggests that women are more likely than men to engage in affiliative behaviors as a regulatory strategy in the face of stress. Are these effects observed postloss? Second, how does relationship satisfaction alter coregulation, dysregulation, and self-regulation? In the child attachment literature, it has been recognized for a long time that although insecure and secure children differ in the way they regulate felt security, children who are classified as insecure are nonetheless still attached to their caregivers

(Cassidy, 1999). We believe the same logic can be applied to understand coregulation and dysregulation in the context of distressed couples. Among couples who have reached the level of a true attachment relationship, are coregulatory processes moderated by relationship satisfaction? Do distressed couples experience less coregulatory benefits? Is dysregulation less or more severe among couples experiencing distress (or in cases of relationship violence)? Third, most people have more than one attachment relationship, which raises the question of how these processes operate between relationships. For example, a married woman may be attached to her partner, her mother, her best friend, and her adult child. Does she experience coregulation with all of these people? Will dysregulation follow from the loss of any of these relationships? In this review, we have proposed a strong hypothesis: When a true attachment exists, so should coregulation and dysregulation on the occasion of the partner's loss. This is a reasonable starting point for investigating these processes across multiple relationships. Finally, how do early experiences with stress and adversity moderate coregulation and dysregulation? Meaney and colleagues' (e.g., Meaney, 2001) research demonstrates that early adversity and individual differences in maternal care alter rats' ability to cope with stress later in life. Is the emergence of coregulation delayed, or does it operate differently in adults with a history of early adversity? In what specific ways are self-regulatory efforts at restoring felt security impinged among these individuals? Answers to these questions will prove useful as social psychologists pursue the integration of psychology and biology in the study of attachment, loss, and recovery.

CONCLUSION

Mammalian biological systems are open to external influence, and research suggests that human attachment relationships can serve as a potent environmental regulator. The main thesis of this article is that a deeper understanding of social separations, loss, and recovery can be achieved by considering the normative physiological processes operating within adult attachment relationships. Evidence from animal studies indicates that pair bonding is associated with a distinct neurobiology operating to induce pleasure, alleviate distress, and maintain psychophysiological homeostasis through a conditioned state called coregulation. Coregulation represents the physiological instantiation of felt security, which is a critical element of normative attachment. Although evidence for coregulation in humans is limited and only newly emerging, conceptualizing attachment relationships as a system in which the physiological

functioning of one person is highly associated with the physiological functioning of the partner provides a new vantage point for understanding responses to relationship disruptions and the experience of attachment figure loss. When relationship separations and losses occur, adults lose an efficient means for maintaining homeostasis and quickly downregulating potentially harmful physiological arousal; as threat to adults' perceived sense of felt security increases, the resulting state can be described as one of biobehavioral dysregulation, which ranges from diffuse physiological arousal and disorganization to a full-blown—and highly organized—stress response. We proposed a continuum of dysregulation to highlight the importance of not equating all loss reactions with a stress response, and thus we distinguished between two forms of physiological dysregulation: the loss of coregulation (termed disorganized dysregulation) and the onset of a biological stress response (representing an organized response to an environmental challenge that requires active adaptation). Although almost all research on the biology of human separation and loss focuses on SAM and HPA activity (with the available evidence indicating that chronic stress negatively impacts both neuroendocrine and immune functioning following divorce and bereavement), understanding the subtle ways in which separation events can disturb biological homeostasis may prove useful for illuminating the nature of coregulation and, therefore, what is lost when attachment relationships end. From this perspective, outcome variables such as sleep quality and architecture, appetite, and body temperature are among the core regulatory components disturbed when relationships are disrupted or dissolved. Inquiry into these areas is beginning to emerge, and many questions about relationships, regulation, and loss are ripe for investigation. Finally, we have argued that the chief task in coping with loss is managing one's state of dysregulation. The underlying function of adaptive self-regulation is to move individuals from a state of biobehavioral dysregulation to self-maintained homeostasis, which involves adopting a coping strategy that provides for the sense of felt security formerly conferred by one's attachment figure. This framework suggests that our understanding of the complexities of loss and recovery is only as good as our understanding of normative attachment. The final section of this article posed six research questions and outlined an empirical agenda for continued study, including testable hypotheses concerning how coregulation in humans can be evaluated. Further inquiry into the psychology and biology of human coregulation will make clear what is lost when relationships are disrupted and what must be regained in the process of recovery. Answers to the questions emerging from this analysis will provide deeper insight into precisely how adult

attachments operate, why social attachments confer physical health benefits, and the pathways through which individuals adapt or succumb to the potentially dysregulating effects of attachment figure loss.

NOTES

1. Strictly speaking, if a classic stress response is a combination of activity along the SAM and HPA axes (Chrousos & Gold, 1992), vagal activity is independent of these systems (in other words, parasympathetic and sympathetic modes of cardiac control are orthogonal; see Bernston, Cacioppo, & Quigley, 1991) and may precede or co-occur with increases in sympathetic activity. The vagus nerve exerts tonic, inhibitory control of the heart and serves to slow heart rate; thus, it is reasonable to speculate that increases in heart rate can follow from vagal withdrawal without associated increases in sympathetic activity. This fits well with our model of disorganized responses; in contrast, when these disorganized forms of dysregulation become organized, it is expected that decreases in parasympathetic control of the heart (i.e., vagal withdrawal) will be coupled with increases in sympathetic activity (e.g., increased cardiac output, shortened pre-ejection period, reflecting faster mechanical activity of the myocardium). The same logic can be applied to understanding sleep disturbances. The extrapyramidal symptoms of withdrawal responses often involve extreme restlessness, which can impede sleep efficiency and quality. We consider this a disorganized feature of attachment loss. At the same time, sleep problems are common to many psychiatric conditions and frequently follow both major and minor stressors. Therefore, there can be an overlap between these disorganized responses and a more classic stress response, described in detail in the next section, but this fact does not preclude a discussion of sleep or vagal activity as part of a disorganized response.

2. A recent meta-analysis conducted by Miller et al. (2007) suggests that neuroendocrine responses and immunological health risk may not follow the classic stress response pattern outlined under Seyle's GAS. Miller et al. (2007) demonstrate that the nature and timing of HPA glucocorticoid response depend on several key moderators and that hypocortisolism is as associated with chronic stress as hypercortisolism is. In the analysis presented here, we describe loss reactions in the immediate aftermath of a separation event. Thus, the experience is ongoing and involves threats to the social self, both of which lead to hypercortisolism (Dickerson & Kemeny, 2004; Miller et al., 2007). Miller et al. (2007) found that loss experiences were sometimes associated with flattened cortisol patterns, which the authors argued may represent psychological disengagement over time. It is arguable that these responses occur after the initial sequence of protest–despair described here. Therefore, we discuss the available evidence in terms of Selye's GAS model but acknowledge that the Miller et al. framework calls for a careful assessment of the important moderators of neuroendocrine responses to loss.

3. Although the glucocorticoid activity is typically believed to be immunosuppressive (see Miller, Cohen, & Ritchey, 2002), especially in cases of chronic activation, emerging evidence indicates that corticotropin-releasing factor and associated stress hormones also can initiate a physiological response characterized by cytokine production and adaptive inflammation (Black, 2002; Maier & Watkins, 1998).

REFERENCES

Affleck, G., & Tennen, H. (1996). Construing benefits from adversity: Adaptational significance and dispositional underpinnings. *Journal of Personality*, 64, 899-922.

Affleck, G., Tennen, H., Croog, S., & Levine, S. (1987). Causal attributions, perceived benefits, and morbidity after a heart attack: An eight-year study. *Journal of Consulting & Clinical Psychology*, 55, 29-35.

Ainsworth, M. D. S. (1967). *Infancy in Uganda: Infant care and the growth of attachment.* Baltimore, MD: Johns Hopkins University Press.

Ainsworth, M. D. S., Blehar, M. C., Waters, E., & Wall, S. (1978). *Patterns of attachment: A psychological study of the Strange Situation.* Hillsdale, NJ: Lawrence Erlbaum.

Amato, P. R. (2000). The consequences of divorce for adults and children. *Journal of Marriage & the Family*, 62, 1269-1287.

Aragona, B. J., Liu, Y., Yu, Y. J., Curtis, J. T., Detwiler, J. M., Insel, T. R., et al. (2006). Nucleus accumbens dopamine differentially mediates the formation and maintenance of monogamous pair bonds. *Nature Neuroscience*, 9, 133-139.

Bartrop, H. R., Luckhurst, E., Lazarus, L., Kiloh, L. G., & Penny, R. (1977). Depressed lymphocyte function after bereavement. *Lancet*, 1, 834-836.

Baumeister, R. F., & Leary, M. R. (1995). The need to belong: Desire for interpersonal attachments as a fundamental human motivation. *Psychological Bulletin*, 117, 497-529.

Belluzzi, J. D., & Stein, L. (1977). Enkephalin may mediate euphoria and drive-reduction reward. *Nature*, 266, 556-558.

Berntson, G. G., Cacioppo, J. T., Binkley, P. F., Uchino, B. N., Quigley, K. S., & Fieldstone, A. (1994). Autonomic cardiac control: III. Psychological stress and cardiac response in autonomic space as revealed by pharmacological blockades. *Psychophysiology*, 31, 599-608.

Berntson, G. G., Cacioppo, J. T., & Quigley, K. S. (1991). Autonomic determinism: The modes of autonomic control, the doctrine of autonomic space, and the laws of autonomic constraint. *Psychological Review*, 98, 459-487.

Black, P. H. (2002). Stress and the inflammatory response: A review of neurogenic inflammation. *Brain, Behavior, and Immunity*, 16, 622-635.

Boccia, M. L., Scanlan, J. M., Laudenslager, M. L., Berger, C. L., Hijazi, A. S., & Reite, M. L. (1997). Juvenile friends, behavior, and immune responses to separation in bonnet macaque infants. *Physiology & Behavior*, 61, 191-198.

Boker, S. M., & Laurenceau, J.-P. (2006). Dynamical systems modeling: An application to the regulation of intimacy and disclosure in marriage. In T. A. Walls & J. L. Schafer (Eds.), *Models for intensive longitudinal data* (pp. 195-218). Oxford, UK: Oxford University Press.

Bonanno, G. A. (2004). Loss, trauma, and human resilience: Have we underestimated the human capacity to thrive after extremely aversive events? *American Psychologist*, 59, 20-28.

Bonanno, G. A., & Kaltman, S. (1999). Toward an integrative perspective on bereavement. *Psychological Bulletin*, 125, 760-776.

Bonanno, G. A., Keltner, D., Holen, A., & Horowitz, M. J. (1995). When avoiding unpleasant emotions might not be such a bad thing: Verbal-autonomic response dissociation and midlife conjugal bereavement. *Journal of Personality & Social Psychology*, 69, 975-989.

Bonanno, G. A., Wortman, C. B., Lehman, D. R., Tweed, R. G., Haring, M., Sonnega, J., et al. (2002). Resilience to loss and chronic grief: A prospective study from preloss to 18-months postloss. *Journal of Personality & Social Psychology*, 83, 1150-1164.

Bower, J. E., Kemeny, M. E., Taylor, S. E., & Fahey, J. L. (1998). Cognitive processing, discovery of meaning, CD4 decline, and AIDS-related mortality among bereaved HIV-seropositive men. *Journal of Consulting & Clinical Psychology*, 66, 979-986.

Bowlby, J. (1958). The nature of the child's tie to his mother. *International Journal of Psycho-Analysis*, 41, 89-113.

Bowlby, J. (1982). *Attachment and loss: Vol 1. Attachment* (2nd ed.). New York: Basic Books. (Original work published 1969)

Bowlby, J. (1973). *Attachment and loss: Vol 2. Separation.* New York: Basic Books.

Bowlby, J. (1980). *Attachment and loss: Vol 3. Loss: Sadness and depression.* New York: Basic Books.

Bremner, J. D., & Vermetten, E. (2001). Stress and development: Behavioral and biological consequences. *Development and Psychopathology*, 13, 473-489.

Bruijnzeel, A. W., Repetto, M., & Gold, M. S. (2004). Neurobiological mechanisms in addictive and psychiatric disorders. *Psychiatric Clinics of North America*, 27, 661-674.

Cacioppo, J. T., Burleson, M. H., Poehlmann, K. M., Malarkey, W. B., Kiecolt-Glaser, J. K., Berntson, G. G., et al. (2000). Autonomic and neuroendocrine responses to mild psychological stressors: Effects of chronic stress on older women. *Annals of Behavioral Medicine*, 22, 140-148.

Capps, L., & Bonanno, G. A. (2000). Narrative bereavement: Thematic and grammatical predictors of adjustment to loss. *Discourse Processes*, 30, 1-25.

Carter, C. S. (1998). Neuroendocrine perspectives on social attachment and loss. *Psychoneuroendocrinology*, 23, 779-818.

Carter, C. S. (2007). Sex differences in oxytocin and vasopressin: Implications for autism spectrum disorders? *Behavioural Brain Research*, 176, 170-186.

Carter, C. S., & Altemus, M. (1997). Integrative functions of lactational hormones in social behavior and stress management. In C. S. Carter, I. I. Lederhendler, & B. Kirkpatrick (Eds.), *The integrative neurobiology of affiliation* (pp. 164-174). New York: New York Academy of Sciences.

Carter, C. S., DeVries, A. C., & Getz, L. L. (1995). Physiological substrates of mammalian monogamy: The prairie vole model. *Neuroscience & Biobehavioral Reviews*, 19, 303-314.

Carter, C. S., Williams, J. R., Witt, D. M., & Insel, T. R. (1992). Oxytocin and social bonding. In C. A. Pederson, J. D. Caldwell, G. F. Jirikowski, & T. R. Insel (Eds.), *Oxytocin in maternal, sexual, and social behaviors. Annals of the New York Academy of Sciences: Vol. 652* (pp. 204-211). New York: New York Academy of Sciences.

Cassidy, J. (1999). The nature of the child's ties. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (pp. 3-20). New York: Guilford Press.

Chrousos, G. P., & Gold, P. W. (1992). The concepts of stress and stress system disorders: Overview of physical and behavioral homeostasis. *Journal of the American Medical Association*, 267, 1244-1252.

Cicchetti, D., & Rogosch, F. A. (1996). Equifinality and multifinality in developmental psychopathology. *Development and Psychopathology*, 8, 597-600.

Coan, J. A. (in press). Toward a neuroscience of attachment. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (2nd ed.). New York: Guildford Publications.

Coan, J. A., Schaefer, H. S., & Davidson, R. J. (2006). Lending a hand: Social regulation of the neural response to threat. *Psychological Science*, 17, 1032-1039.

Cohen, S., & Herbert, T. B. (1996). Health psychology: Psychological factors and physical disease from the perspective of human psychoneuroimmunology. *Annual Review of Psychology*, 47, 113-142.

Cohen, S., & Wills, T. A. (1985). Stress, social support and the buffering hypothesis. *Psychological Bulletin*, 98, 310-357.

Cole, S. W., & Kemeny, M. E. (2001). Psychosocial influences on the progression of HIV infection. In R. Ader, D. L. Felten, & N. Cohen (Eds.), *Psychoneuroimmunology: Vol. 1* (pp. 1-20). New York: Academic Press.

Cole, S. W., Kemeny, M. E., & Taylor, S. E. (1997). Social identity and physical health: Accelerated HIV progression in rejection-sensitive gay men. *Journal of Personality & Social Psychology*, 72, 320-335.

Crowell, J. A., Treboux, D., Gao, Y., Fyffe, C., Pan, H., & Waters, E. (2002). Assessing secure base behavior in adulthood: Development of a measure, links to adult attachment representations and relations to couples' communication and reports of relationships. *Developmental Psychology*, 38, 679-693.

Dantzer, R. (2001). Cytokine-induced sickness behavior: Where do we stand? *Brain, Behavior, and Immunity*, 15, 7-24.

Dantzer, R., Wollman, E. E., & Yirmiya, R. (Eds.). (1999). *Cytokines, stress, and depression.* New York: Kluwer-Academic/Plenum.

Depue, R. A., & Morrone-Strupinsky, J. V. (2005). A neurobehavioral model of affiliative bonding: Implications for conceptualizing a human trait of affiliation. *Behavioral and Brain Sciences*, 28, 313-395.

Dhabhar, F. S., & McEwen, B. S. (1996). Stress-induced enhancement of antigen-specific cell-mediated immunity. *Journal of Immunology*, 156, 2608-2615.

Diamond, L. M. (2001). Contributions of psychophysiology to research on adult attachment: Review and recommendations. *Personality and Social Psychology Review*, 5, 276-295.

Diamond, L. M., & Hicks, A. M. (2004). Psychobiological perspectives on attachment: Implications for health over the lifespan. In W. S. Rholes & J. A. Simpson (Eds.), *Adult attachment: Theory, research, and clinical implications* (pp. 240-263). New York: Guilford.

Diamond, L. M., Hicks, A. M., & Otter-Henderson, K. (in press). Every time you go away: Changes in affect, behavior, and physiology associated with travel-related separations from romantic partners. *Journal of Personality and Social Psychology*.

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, 130, 355-391.

Doherty, N. A., & Feeney, J. A. (2004). The composition of attachment networks throughout the adult years. *Personal Relationships*, 11, 469-488.

Ehlers, C. L., Frank, E., & Kupfer, D. J. (1988). Social zeitgebers and biological rhythms. *Archives of General Psychiatry*, 45, 948-952.

Emery, R. E. (1994). *Renegotiating family relationships: Divorce, child custody, and mediation.* New York: Guilford Press.

Feldman, R. (2007). Parent–infant synchrony: Biological foundations and developmental outcomes. *Current Directions in Psychological Science*, 16, 340-345.

Feldman, R., & Eidelman, A. I. (2007). Maternal postpartum behavior and the emergence of infant–mother and infant–father synchrony in preterm and full-term infants: The role of neonatal vagal tone. *Developmental Psychobiology*, 49(3), 290-302.

Feldman, R., Weller, A., Zagoory-Sharon, O., & Levine, A. (2007). Evidence for a neuroendocrinological foundation of human affiliation: Plasma oxytocin levels across pregnancy and the postpartum period predict mother–infant bonding. *Psychological Science*, 18, 965-970.

Ferrer, E., & McArdle, J. J. (2003). Alternative structural models for multivariate longitudinal data analysis. *Structural Equation Modeling*, 10, 493-524.

Field, T. (1985). Attachment as psychobiological attunement: Being on the same wavelength. In M. Reite & T. Field (Eds.), *The psychobiology of attachment and separation* (pp. 415-454). Orlando, FL: Academic.

Field, T. (1994). The effects of mother's physical and emotional unavailability on emotion regulation. *Monographs of the Society for Research in Child Development*, 59, 208-277.

Folkman, S. (2001). Revised coping theory and the process of bereavement. In M. S. Stroebe, R. O. Hansson, W. Stroebe, & H. Schut (Eds.), *Handbook of bereavement research: Consequences, coping, and care* (pp. 563-584). Washington, DC: American Psychological Association.

Fraley, R. C., Garner, J. P., & Shaver, P. R. (2000). Adult attachment and the defensive regulation of attention and memory: Examining the role of preemptive and postemptive defensive processes. *Journal of Personality and Social Psychology*, 79, 816-826.

Fraley, R. C., & Shaver, P. R. (1999). Loss and bereavement: Attachment theory and recent controversies concerning "grief work" and the nature of detachment. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (pp. 735-759). New York: Guilford Press.

Futterman, A. D., Kemeny, M. E., Shapiro, D., & Fahey, J. L. (1994). Immunological and physiological changes associated with induced positive and negative mood. *Psychosomatic Medicine*, 56(6), 499-511.

George, C., Kaplan, N., & Main, M. (1984). *Adult attachment interview protocol.* Unpublished manuscript, University of California–Berkley.

Goodkin, K., Baldewicz, T. T., Blaney, N. T., Asthana, D., Kumar, M., Shapshak, P., et al. (2001). Physiological effects of bereavement and bereavement support group interventions. In M. S. Stroebe, O. Hansson, W. Stroebe, & H. Schut (Eds.), *Handbook of bereavement research: Consequences, coping, and care* (pp. 671-703). Washington, DC: American Psychological Association.

Goodkin, K., Feaster, D. J., Tuttle, R. S., Blaney, N. T., Kumar, M., Baum, M. K., et al. (1996). Bereavement is associated with time-dependent decrements in cellular immune functioning in asymptomatic human immunodeficiency virus type 1-seropositive

homosexual men. *Clinical and Diagnostic Laboratory Immunology*, 3, 109-118.

Gottman, J., Swanson, C., & Swanson, K. (2002). A general systems theory of marriage: Nonlinear difference equation modeling of marital interaction. *Personality and Social Psychology Review*, 6, 326-340.

Grewen, K. M., Girdler, S. S., Amico, J., & Light, K. C. (2005). Effects of partner support on resting oxytocin, cortisol, norepinephrine, and blood pressure before and after warm partner contact. *Psychosomatic Medicine*, 67, 531-538.

Gross, J. J., & Levenson, R. W. (1993). Emotional suppression: Physiology, self-report, and expressive behavior. *Journal of Personality and Social Psychology*, 64, 970-986.

Gump, B. B., Polk, D. E., Kamarck, T. W., & Shiffman, S. M. (2001). Partner interactions are associated with reduced blood pressure in the natural environment: Ambulatory monitoring evidence from a healthy, multiethnic adult sample. *Psychosomatic Medicine*, 63, 423-433.

Hall, M., & Irwin, M. (2001). Physiological indices of functioning in bereavement. In M. S. Stroebe, R. O. Hansson, W. Stroebe, & H. Schut (Eds.), *Handbook of bereavement research: Consequences, coping, and care* (pp. 473-492). Washington, DC: American Psychological Association.

Hazan, C., & Diamond, L. M. (2000). The place of attachment in human mating. *Review of General Psychology*, 4, 186-204.

Hazan, C., Gur-Yaish, N., & Campa, M. (2004). What does it mean to be attached? In W. S. Rholes & J. A. Simpson (Eds.), *Adult attachment: New directions and emerging issues* (pp. 55-85). New York: Guilford Press.

Hazan, C., & Shaver, P. (1987). Romantic love conceptualized as an attachment process. *Journal of Personality & Social Psychology*, 52, 511-524.

Hazan, C., & Shaver, P. R. (1992). Broken attachments: Relationship loss from the perspective of attachment theory. In T. L. Orbuch (Ed.), *Close relationship loss* (pp. 90-110). New York: Springer-Verlag.

Hazan, C., & Zeifman, D. (1994). Sex and the psychological tether. In K. Bartholomew & D. Perlman (Eds.), *Attachment processes in adulthood* (pp. 151-178). London: Jessica Kingsley.

Hazan, C., & Zeifman, D. (1999). Pair bonds as attachments: Evaluating the evidence. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (pp. 336-354). New York: Guilford Press.

Heinrichs, M., Baumgartner, T., Kirschbaum, C., & Ehlert, U. (2003). Social support and oxytocin interact to suppress cortisol and subjective responses to psychosocial stress. *Biological Psychiatry*, 54, 1389-1398.

Helgeson, V. S. (2003). Cognitive adaptation, psychological adjustment, and disease progression among angioplasty patents: 4 years later. *Health Psychology*, 22(1), 30-38.

Hennessy, M. B., Deak, T., & Schiml-Webb, P. A. (2001). Stress-induced sickness behaviors: An alternative hypothesis for responses during maternal separation. *Developmental Psychobiology*, 39, 76-83.

Hennessy, M. B., Schiml-Webb, P. A., Miller, E. E., Maken, D. S., Bullinger, K. L., & Deak, T. (2007). Anti-inflammatory agents attenuate the passive responses of guinea pig pups: Evidence for stress-induced sickness behavior during maternal separation. *Psychoneuroendocrinology*, 32, 508-515.

Hetherington, E. M., & Kelly, J. (2002). *For better or for worse: Divorce reconsidered*. New York: Norton.

Hofer, M. A. (1984). Relationships as regulators: A psychobiological perspective on bereavement. *Psychosomatic Medicine*, 46, 183-197.

Hofer, M. A. (1987). Early social relationships: A psychobiologist's view. *Child Development*, 58, 633-647.

Hofer, M. A. (1994). Hidden regulators in attachment, separation, and loss. *Monographs of the Society for Research in Child Development*, 59, 250-283.

Hofer, M. A. (1995). Hidden regulators: Implications for a new understanding of attachment, separation, and loss. In S. Goldberg & R. Muir (Eds.), *Attachment theory: Social, developmental, and clinical perspectives* (pp. 203-230). Hillsdale, NJ: Analytic Press.

Hofer, M. A. (1996). On the nature and consequences of early loss. *Psychosomatic Medicine*, 58, 570-581.

Hofer, M. A. (2006). Psychobiological roots of early attachment. *Current Directions in Psychological Science*, 15, 84-88.

Holt-Lunstad, J., Uchino, B. N., Smith, T. W., Olson-Cerny, C., & Nealey-Moore, J. B. (2003). Social relationships and ambulatory blood pressure: Structural and qualitative predictors of cardiovascular function during everyday social interactions. *Health Psychology*, 22, 388-397.

House, J. S., Landis, K. R., & Umberson, D. (1988). Social relationships and health. *Science*, 241, 540-545.

Insel, T. R. (1992). Oxytocin: A neuropeptide for affiliation: Evidence from behavioral, receptor autoradiographic, and comparative studies. *Psychoneuroendocrinology*, 17, 3-35.

Insel, T. R. (1997). A neurobiological basis of social attachment. *American Journal of Psychiatry*, 154, 726-735.

Insel, T. R. (2000). Toward a neurobiology of attachment. *Review of General Psychology*, 4, 176-185.

Insel, T. R. (2003). Is social attachment an addictive disorder? *Physiology & Behavior*, 79, 351-357.

Insel, T. R., & Hulihan, T. J. (1995). A gender-specific mechanism for pair bonding: Oxytocin and partner preference formation in monogamous voles. *Behavioral Neuroscience*, 109, 782-789.

Insel, T. R., & Young, L. J. (2001). The neurobiology of attachment. *Nature Neuroscience*, 2, 129-136.

Irwin, M., Daniels, M., Risch, S. C., Bloom, E. T., & Weiner, H. (1988). Plasma cortisol and natural killer cell activity during bereavement. *Biological Psychiatry*, 24, 173-178.

Irwin, M., Daniels, M., Smith, T. L., Bloom, E. T., & Weiner, H. (1987). Impaired natural killer cell activity during bereavement. *Brain, Behavior, & Immunity*, 1, 98-104.

Jacobs, S. C., Mason, J., Kosten, T. R., Kasl, S. V., Ostfeld, A. M., & Wahby, V. (1987). Urinary free cortisol and separation anxiety early in the course of bereavement and threatened loss. *Biological Psychiatry*, 22, 148-152.

Jacobs, S. C., Mason, J., Kosten, T. R., Wahby, V., Kasl, S. V., & Ostfeld, A. M. (1986). Bereavement and cetecholamines. *Journal of Psychosomatic Research*, 30, 489-496.

Kaufman, I. C., & Rosenblum, L. A. (1967). Depression in infant monkeys separated from their mothers. *Science*, 155, 1030-1031.

Kehoe, P., & Blass, E. M. (1986). Opioid-mediation of separation distress in 10-day-old rats: Reversal of stress with maternal stimuli. *Developmental Psychobiology*, 19, 385-398.

Kelley, A. E., & Berridge, K. C. (2002). The neuroscience of natural rewards: Relevance to addicitve drugs. *Journal of Neuroscience*, 22, 3306-3311.

Kemeny, M. E. (2003). The psychobiology of stress. *Current Directions in Psychological Science*, 12, 124-129.

Kemeny, M. E., & Gruenewald, T. L. (2000). Affect, cognition, the immune system and health. In E. A. Mayer & C. Saper (Eds.), *The biological basis for mind body interactions. Progress in brain research series* (pp. 291-308). Amsterdam: Elsevier Science B.V.

Kemeny, M. E., Weiner, H., Duran, R., Taylor, S. E., Visscher, B., & Fahey J. L. (1995). Immune system changes after the death of a partner in HIV-positive gay men. *Psychosomatic Medicine*, 57(6), 547-554.

Kenny, D. A., Kashy, D. A., & Cook, W. L. (2006). *Dyadic data analysis*. New York: Guilford Press.

Kiecolt-Glaser, J. K., Fisher, L. D., Ogrocki, P., Stout, J. C., Speicher, C. E., & Glaser, R. (1987). Marital quality, marital disruption, and immune function. *Psychosomatic Medicine*, 49(1), 13-34.

Kiecolt-Glaser, J. K., Kennedy, S., Malkoff, S., Fisher, L., Speicher, C. E., & Glaser, R. (1988). Marital discord and immunity in males. *Psychosomatic Medicine*, 50, 213-229.

Kiecolt-Glaser, J. K., McGuire, L., Robles, T. F., & Glaser, R. (2002). Emotions, morbidity, and mortality: New perspectives from psychoneuroimmunology. *Annual Review of Psychology*, 53, 83-107.

Kiecolt-Glaser, J., & Newton, T. L. (2001). Marriage and health: His and hers. *Psychological Bulletin*, 127, 472-503.

Kirsch, P., Esslinger, C., Chen, Q., Mier, D., Lis, S., Siddhanti, S., et al. (2005). Oxytocin modulates neural circuitry for social cognition and fear in humans. *Journal of Neuroscience*, 25, 11489-11493.

Kitson, G. C., & Holmes, W. M. (1992). *Portrait of divorce: Adjustment to marital breakdown*. New York: Guilford Press.

Kosfeld, M., Heinrichs, M., Zak, P. J., Fischbacher, U., & Fehr, E. (2005). Oxytocin increases trust in humans. *Nature*, *435*, 673-676.

Kovacs, G. L., Sarnyai, Z., & Szabo, G. (1998). Oxytocin and addition: A review. *Psychoneuroendocrinology*, *23*, 945-962.

Kraemer, G. W. (1992). A psychobiological theory of attachment. *Behavioral and Brain Sciences*, *15*, 493-541.

Kraemer, G. W., Ebert, M. H., Schmidt, D. E., & McKinney, W. T. (1989). A longitudinal study of the effects of different rearing environments on cerebrospinal fluid norepinephrine and biogenic amine metabolites in rhesus monkeys. *Neuropsychopharmacoloy*, *2*, 175-199.

Larson, S. J., & Dunn, A. J. (2001). Behavioral effects of cytokines. *Brain, Behavior, & Immunity*, *15*, 371-387.

Laurenceau, J.-P., & Bolger, N. (2005). Using diary methods to study marital and family processes. *Journal of Family Psychology*, *19*, 86-97.

Leu, S. J., & Singh, L. K. (1992). Stimulation of interleukin-6 production by corticotropin-releasing hormone. *Cellular Immunology*, *143*, 220-227.

Light, K. C., Smith, T. E., Johns, J. M., Brownley, K. A., & Hofheimer, J. A. (2000). Oxytocin responsivity in mothers of infants: A preliminary study of relationships with blood pressure during laboratory stress and normal ambulatory activity. *Health Psychology*, *19*, 560-567.

Lim, M. M., & Young, L. J. (2006). Neuropeptidergic regulation of affiliative behavior and social bonding in animals. *Hormones and Behavior*, *50*, 506-517.

Lyubomirsky, S., & Nolen-Hoeksema, S. (1993). Self-perpetuating properties of dysphoric rumination. *Journal of Personality & Social Psychology*, *65*, 339-349.

Maier, S. F., Grahn, R. E., & Watkins, L. R. (1995). 8-OH-DPAT microinjected into the region of the dorsal raphe nucleus blocks and reverses the enhancement of fear conditioning and the interference with escape produced by exposure to inescapable shock. *Behavioral Neuroscience*, *109*, 404-413.

Maier, S. F., & Watkins, L. R. (1998). Cytokines for psychologists: Implications of bidirectional immune-to-brain communication for understanding behavior, mood, and cognition. *Psychological Review*, *105*, 83-107.

Main, M. (1999). Epilogue. Attachment theory: Eighteen points with suggestions for future research. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (pp. 845-888). New York: Guilford Press.

Martel, F. L., Nevison, C. M., Simpson, M. J. A., & Keverne, E. B. (1995). Effects of opioid receptor blockade on the social behavior of rhesus monkeys living in large family groups. *Developmental Psychobiology*, *28*, 71-84.

Marvin, R. S., & Britner, P. A. (1999). Normative development: The ontogeny of attachment. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (pp. 44-67). New York: Guilford Press.

McArdle, J. J., & Hamagami, F. (2001). Latent difference score structural models for linear dynamic analyses with incomplete longitudinal data. In L. M. Collins & A. G. Sayer (Eds.), *New methods for the analysis of change* (pp. 139-175). Washington, DC: American Psychological Association.

McCarthy, M. M. (1995). Estrogen modulation of oxytocin and its relation to behavior. In R. Ivell & J. Russell (Eds.), *Oxytocin: Cellular and molecular approaches in medicine and research* (pp. 235-242). New York: Plenum Press.

McCubbin, J. A. (1993). Stress and endogenous opioids: Behavioral and circulatory interactions. *Biological Psychology*, *35*, 91-122.

McEwen, B. S., & Stellar, E. (1993). Stress and the individual: Mechanisms leading to disease. *Archives of Internal Medicine*, *153*, 2093-2101.

Meinlschmidt, G., & Heim, C. (2007). Sensitivity to intranasal oxytocin in adult men with early parental separation. *Biological Psychiatry*, *61*, 1109-1111.

Meyers, C. A. (1999). Mood and cognitive disorder in cancer patients receiving cytokine therapy. *Advances in Experimental Medicine and Biology*, *461*, 75-81.

Mikulincer, M., Birnbaum, G., Woddis, D., & Nachmias, O. (2000). Stress and accessibility of proximity-related thoughts: Exploring the normative and intraindividual components of attachment theory. *Journal of Personality & Social Psychology*, *78*, 509-523.

Mikulincer, M., Gillath, O., & Shaver, P. R. (2002). Activation of the attachment system in adulthood: Threat-related primes increase the accessibility of mental representations of attachment figures. *Journal of Personality and Social Psychology*, *83*, 881-895.

Mikulincer, M., & Shaver, P. R. (2003). The attachment behavioral system in adulthood: Activation, psychodynamics, and interpersonal processes. In M. P. Zanna (Ed.), *Advances in experimental social psychology: Vol. 35* (pp. 53-152). San Diego: Elsevier Academic Press.

Mikulincer, M., & Shaver, P. R. (2007). *Attachment in adulthood: Structure, dynamics, and change*. New York: Guilford Press.

Mikulincer, M., & Shaver, P. R. (in press). An attachment perspective on bereavement. In M. Stroebe, H. A. Hansson, H. Schut, & W. Stroebe (Eds.), *Handbook of bereavement research and practice: 21st century perspective* (2nd ed.). Washington, DC: American Psychological Association.

Mikulincer, M., Shaver, P. R., & Pereg, D. (2003). Attachment theory and affect regulation: The dynamics, development, and cognitive consequences of attachment-related strategies. *Motivation & Emotion*, *27*, 77-102.

Miller, G. E., Chen, E., & Zhou, E. S. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological Bulletin*, *133*, 25-45.

Miller, G. E., Cohen, S., & Ritchey, A. K. (2002). Chronic psychological stress and the regulation of pre-inflammatory cytokines: A glucocorticoid-resistance model. *Health Psychology*, *21*, 531-541.

Munck, A., & Guyre, P. M. (1991). Glucocorticoids and immune function. In R. Ader, D. L. Felten, & N. Cohen (Eds.), *Psychoneuroimmunology* (pp. 447-474). San Diego, CA: Academic Press.

Murray, S. L., Holmes, J. G., & Collins, N. L. (2006). Optimizing assurance: The risk regulation system in relationships. *Psychological Bulletin*, *132*, 641-666.

Neimeyer, R. A. (2000). Searching for the meaning of meaning: Grief therapy and the process of reconstruction. *Death Studies*, *24*, 541-558.

Nelson, E., & Panksepp, J. (1998). Brain substrate of infant–mother attachment: Contributions of opioids, oxytocin, and norepinephrine. *Neuroscience and Biobehavioral Reviews*, *22*, 437-452.

Newsom, J. T. (2002). A multilevel structural equation model for dyadic data. *Structural Equation Modeling*, *9*, 431-447.

Nissen, E., Gustavsson, P., Widstrom, A. M., & Uvnäs-Moberg, K. (1998). Oxytocin, prolatin and cortisol levels in response to nursing in women after Sectio Caesarea and vaginal delivery: Relationship with changes in personality post partum. *Journal of Psychosomatic Obstetrics and Gynaecology*, *19*, 49-58.

Nolen-Hoeksema, S. (2001). Ruminative coping and adjustment to bereavement. In M. S. Stroebe, R. O. Hansson, W. Stroebe, & H. Schut (Eds.), *Handbook of bereavement research: Consequences, coping, and care* (pp. 545-562). Washington, DC: American Psychological Association.

Nolen-Hoeksema, S., McBride, A., & Larson, J. (1997). Rumination and psychological distress among bereaved partners. *Journal of Personality & Social Psychology*, *72*, 855-862.

Nolen-Hoeksema, S., Parker, L. E., & Larson, J. (1994). Ruminative coping with depressed mood following loss. *Journal of Personality & Social Psychology*, *67*, 92-104.

Panksepp, J., Nelson, E., & Bekkedal, M. (1997). Brain systems for the mediation of social separation-distress and social-reward: Evolutionary antecedents and neuropeptide intermediaries. In C. S. Carter & I. I. Lederhendler (Eds.), *The integrative neurobiology of affiliation. Annals of the New York Academy of Sciences: Vol. 807* (pp. 78-100). New York: New York Academy of Sciences.

Panksepp, J., Siviy, S. M., & Normansell, L. A. (1985). Brain opioids and social emotions. In M. Reite & T. Field (Eds.), *The psychobiology of attachment and separation* (pp. 3-39). Orlando, FL: Academic Press.

Parkes, C. M. (1998). *Bereavement: Studies of grief in adult life* (3rd ed.). Madison, CT: International Universities Press.

Parkes, C. M. (2001). A historical overview of the scientific study of bereavement. In M. S. Stroebe, R. O. Hansson, W. Stroebe, & H. Schut (Eds.), *Handbook of bereavement research: Consequences, coping, and care.* (pp. 25-45). Washington, DC: American Psychological Association.

Parkes, C. M., & Weiss, R. S. (1983). *Recovery from bereavement.* New York: Basic Books.

Pedersen, C. A. (1997). Oxytocin control of maternal behavior: Regulation by sex steroids and offspring stimuli. In C. S. Carter, I. I. Lederhendler, & B. Kirkpatrick (Eds.), *The integrative neurobiology of affiliation. Annals of The New York Academy of Sciences: Vol. 807* (pp. 126-145). New York: New York Academy of Sciences.

Pennebaker, J. W., Mayne, T. J., & Francis, M. E. (1997). Linguistic predictors of adaptive bereavement. *Journal of Personality and Social Psychology*, *72*, 863-871.

Pennebaker, J. W., & Seagal, J. D. (1999). Forming a story: The health benefits of narrative. *Journal of Clinical Psychology*, *55*, 1243-1254.

Pham, L. B., Taylor, S. E., & Seeman, T. E. (2001). Effects of environmental predictability and personal mastery on self-regulatory and physiological processes. *Personality & Social Psychology Bulletin*, *27*, 611-620.

Pietromonaco, P. R., Barrett, L. F., & Powers, S. I. (2006). Adult attachment theory and affective reactivity and regulation. In D. K. Snyder, J. Simpson, & J. N. Hughes (Eds.), *Emotion regulation in couples and families: Pathways to dysfunction and health* (pp. 57-74). Washington, DC: American Psychological Association.

Polan, H. J., & Hofer, M. A. (1999). Psychobiological origins of infant attachment and separation responses. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (pp. 162-180). New York: Guilford Press.

Porges, S. W. (1998). Love: An emergent property of the mammalian autonomic nervous system. *Psychoneuroendocrinology*, *23*, 837-861.

Powell, L. H., Lovallo, W. R., Matthews, K. A., Meyer, P., Midgley, A. R., Baum, A., et al. (2002). Physiologic markers of chronic stress in premenopausal, middle-aged women. *Psychosomatic Medicine*, *64*(3), 502-509.

Reed, G. M., Kemeny, M. E., Taylor, S. E., & Visscher, B. R. (1999). Negative HIV-specific expectancies and AIDS-related bereavement as predictors of symptom onset in asymptomatic HIV-positive gay men. *Health Psychology*, *18*, 354-363.

Reis, H. T., Collins, W. A., & Berscheid, E. (2000). The relationship context of human behavior and development. *Psychological Bulletin*, *126*, 844-872.

Reite, M., & Boccia, M. L. (1994). Physiological aspects of adult attachment. In M. B. Sperling & W. H. Berman (Eds.), *Attachment in adults* (pp. 98-127). New York: Guilford Press.

Reite, M., & Capitanio, J. P. (1985). On the nature of social separation and attachment. In M. Reite & T. Field (Eds.), *The psychobiology of attachment and separation* (pp. 415-454). Orlando, FL: Academic.

Robinson-Whelen, S., Kim, C., MacCallum, R. C., & Kiecolt-Glaser, J. K. (1997). Distinguishing optimism from pessimism in older adults: Is it more important to be optimistic or not to be pessimistic? *Journal of Personality & Social Psychology*, *73*, 1345-1353.

Sander, L., Stechler, G., Burns, P., & Julia, H. (1970). Early mother–infant interaction and 24-hour patterns of activity and sleep. *Journal of the American Academy of Child Psychiatry*, *9*, 103-123.

Sapolsky, R. M., Rivier, C., Yamamoto, G., Plotsky, P., & Vale, W. (1987). Interleukin-1 stimulates the secretion of hypothalamic corticotropin releasing hormone. *Science*, *238*, 522-544.

Schiml-Webb, P. A., Deak, T., Greenlee, T. M., Maken, D., & Hennessy, M. B. (2006). Alpha-melanocyte stimulating hormone reduces putative stress-induced sickness behaviors in isolated guinea pig pups. *Behavioural Brain Research*, *168*, 326-330.

Seay, B., Hansen, E., & Harlow, H. F. (1962). Mother–infant separation in monkeys. *Journal of Child Psychology and Psychiatry*, *3*, 123-132.

Seay, B., & Harlow, H. F. (1965). Maternal separation in the rhesus monkey. *Journal of Nervous & Mental Disorders*, *140*, 434-441.

Segerstrom, S. C., Taylor, S. E., Kemeny, M. E., Reed, G. M., & Visscher, B. R. (1996). Causal attributions predict rate of immune decline in HIV-seropositive gay men. *Health Psychology*, *15*, 485-493.

Selye, H. (1956). *The stress of life.* New York: McGraw-Hill.

Sgoutas-Emch, S. A., Cacioppo, J. T., Uchino, B. N., Malarkey, W., Pearl, D., Kiecolt-Glaser, J. K., et al. (1994). The effects of an acute psychological stressor on cardiovascular, endocrine, and cellular immune response: A prospective study of individuals high and low in heart rate reactivity. *Psychophysiology*, *31*, 264-271.

Shaver, P. R., & Mikulincer, M. (2002). Attachment-related psychodynamics. *Attachment & Human Development*, *4*, 133-161.

Shaver, P. R., & Tancredy, C. M. (2001). Emotion, attachment, and bereavement: A conceptual commentary. In M. Stroebe, R. O. Hannson, W. Stroebe, & H. Schut (Eds.), *Handbook of bereavement research* (pp. 63-88). Washington, DC: American Psychological Association.

Shear, K., & Shair, H. (2005). Attachment, loss, and complicated grief. *Developmental Psychobiology*, *47*, 253-267.

Sieber, W. J., Rodin, J., Larson, L., Ortega, S., Cummings, N., Levy, S., et al. (1992). Modulation of human natural killer cell activity by exposure to uncontrollable stress. *Brain, Behavior, & Immunity*, *6*, 141-156.

Simpson, J. A., & Rholes, W. S. (1998). Attachment in adulthood. In J. A. Simpson & W. S. Rholes (Eds.), *Attachment theory and close relationships* (pp. 3-24). New York: Guilford Press.

Simpson, J. A., Rholes, W. S., & Nelligan, J. S. (1992). Support-seeking and support-giving within couples members in an anxiety-provoking situation: The role of attachment styles. *Journal of Personality and Social Psychology*, *62*, 434-446.

Sroufe, L. A., & Waters, E. (1977). Attachment as an organizational construct. *Child Development*, *48*, 1184-1199.

Stack, S., & Eshleman, R. (1998). Marital status and happiness: A 17-nation study. *Journal of Marriage & the Family*, *60*, 527-536.

Stein, N., Folkman, S., Trabasso, T., & Richards, T. A. (1997). Appraisal and goal processes as predictors of psychological well-being in bereaved caregivers. *Journal of Personality and Social Psychology*, *72*, 872-884.

Stratakis, A., & Chrousos, G. P. (1995). Neuroendocrine and pathophysiology of the stress system. In G. P. Chrousos, R. McCarty, K. Pacak, G. Cizza, E. Sternberg, P. W. Gold, & R. Kvetnansky (Eds.), *Annals of the New York Academy of Sciences: Vol. 771* (pp. 1-18). New York: New York Academy of Sciences.

Stroebe, M., & Schut, H. (1999). The dual process model of coping: Rationale and description. *Death Studies*, *23*, 197-224.

Stroebe, M., Schut, H., & Stroebe, W. (2005). Attachment in coping with bereavement: A theoretical integration. *Review of General Psychology*, *9*, 48-66.

Stroebe, M., Stroebe, W., Schut, H., Zech, E., & Bout, J. (2002). Does disclosure of emotions facilitate recovery from bereavement? Evidence from two prospective studies. *Journal of Consulting & Clinical Psychology*, *70*, 169-178.

Suomi, S. J. (1999). The emotional dynamics of disruptions in attachment relationships: Implications for theory, research, and clinical intervention. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (pp. 21-43). New York: Guilford Press.

Suomi, S. J., Eisele, C. D., Grady, S. A., & Harlow, H. F. (1975). Depressive behavior in adult monkeys following separation from family environment. *Journal of Abnormal Psychology*, *84*, 576-578.

Taylor, S. E. (1983). Adjustment to threatening events: A theory of cognitive adaptation. *American Psychologist*, *38*, 1161-1173.

Taylor, S. E. (2006). Tend and befriend: Biobehavioral bases of affiliation under stress. *Current Directions in Psychological Science*, *15*, 273-277.

Taylor, S. E., & Brown, J. D. (1994). "Illusion" of mental health does not explain positive illusions. *American Psychologist*, *49*, 972-973.

Taylor, S. E., Kemeny, M. E., Aspinwall, L. G., Schneider, S. G., Rodriguez, R., & Herbert, M. (1992). Optimism, coping, psychological distress, and high-risk sexual behavior among men at risk for Acquired Immunodeficiency Syndrome (AIDS). *Journal of Personality & Social Psychology*, *63*, 460-473.

Taylor, S. E., Kemeny, M. E., Reed, G. M., Bower, J. E., & Gruenewald, T. L. (2000). Psychological resources, positive illusions, and health. *American Psychologist*, *55*, 99-109.

Taylor, S. E., Klein, L. C., Lewis, B. P., Gruenewald, T. L., Gurung, R. A. R., & Updegraff, J. A. (2000). Biobehavioral responses to stress in females: Tend-and-befriend, not fight-or-flight. *Psychological Review*, *107*, 411-429.

Thompson, S. C., Cheek, P. R., & Grahma, M. A. (1988). The other side of perceived control: Disadvantages and negative effects. In S. Spacapan & S. Oshkamp (Eds.), *The social psychology of health* (pp. 69-93). Newbury Park, CA: Sage.

Toates, F. (1995). *Stress: Conceptual and biological aspects*. New York: John Wiley.

Trinke, S. J., & Bartholomew, K. (1997). Hierarchies of attachment relationships in young adulthood. *Journal of Social and Personal Relationships*, *14*, 603-625.

Uchino, B. N. (2004). *Social support and physical health: Understanding the health consequences of relationships*. New Haven, CT: Yale University Press.

Uchino, B. N., Cacioppo, J. T., & Kiecolt-Glaser, J. K. (1996). The relationship between social support and physiological processes: A review with emphasis on underlying mechanisms and implications for health. *Psychological Bulletin*, *119*, 488-531.

Uchino, B. N., Kiecolt-Glaser, J. K., & Glaser, R. (2000). Psychological modulation of cellular immunity. In J. T. Cacioppo, L. G. Tassinary, & G. G. Berntson (Eds.), *Handbook of psychophysiology* (2nd ed., pp. 397-424). New York: Cambridge University Press.

Uchino, B. N., Smith, T. W., Holt-Lunstad, J. L., Campo, M., & Reblin, M. (2007). Stress and illness. In J. Cacioppo, L. Tassinary, & G. Berntson (Eds.), *Handbook of psychophysiology* (3rd ed., pp. 78-100). New York: Cambridge University Press.

Uvnäs-Moberg, K. (1997). Physiological and endocrine effects of social contact. In C. S. Carter & I. I. Lederhendler (Eds.), *The integrative neurobiology of affiliation. Annals of The New York Academy of Sciences: Vol. 807* (pp. 146-163). New York: New York Academy of Sciences.

Uvnäs-Moberg, K. (1998). Oxytocin may mediate the benefits of positive social interaction and emotions. *Psychoneuroendocrinology*, *23*, 819-835.

Vormbrock, J. K. (1993). Attachment theory as applied to wartime and job-related marital separation. *Psychological Bulletin*, *114*, 122-144.

Weinfield, N. S., Sroufe, L. A., Egeland, B., & Carlson, E. A. (1999). The nature of individual differences in infant–caregiver attachment. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (pp. 68-88). New York: Guilford Press.

Weiss, R. S. (1975). *Marital separation*. New York: Basic Books.

Weiss, R. S. (2001). Grief, bonds, and relationships. In M. Stroebe, R. O. Hannson, W. Stroebe, & H. Schut (Eds.), *Handbook of bereavement research: Consequences, coping, and care* (pp. 47-62). Washington, DC: American Psychological Association.

Wortman, C. B., & Silver, R. C. (1989). The myths of coping with loss. *Journal of Consulting and Clinical Psychology*, *57*, 349-357.

Wortman, C. B., & Silver, R. C. (2001). The myths of coping with loss revisited. In M. S. Stroebe, R. O. Hansson, W. Stroebe, and H. Schut (Eds.), *Handbook of bereavement research. Consequences, coping, and care* (pp. 405-430). Washington, DC: American Psychological Association.

Young, L. J., & Wang, Z. (2004). The neurobiology of pair bonding. *Nature Neuroscience*, *10*, 1048-1054.

Zeifman, D., & Hazan, C. (1997). A process model of adult attachment formation. In S. Duck (Ed.), *Handbook of personal relationships* (pp. 179-195). Chichester, UK: Wiley.