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# Adult Attachment and Coregulation of Stress in Romantic Couples

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UNIVERSITY OF MIAMI

ADULT ATTACHMENT AND COREGULATION OF STRESS IN ROMANTIC  
COUPLES

By

Daniel L. Hall

A THESIS

Submitted to the Faculty  
of the University of Miami  
in partial fulfillment of the requirements for  
the degree of Master of Science

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ADULT ATTACHMENT AND COREGULATION OF STRESS IN ROMANTIC  
COUPLES

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Adult Attachment and Coregulation of Stress  
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In moments of stress, adults often turn to their romantic partners as regulatory agents. Literature suggests that qualities of one's relationship to their romantic partner will influence the magnitude of one's physiological activation during moments of stress. Both attachment theory and findings to date suggest that insecure attachment is predictive of greater reactivity to stress. Moreover, whether the HPA-axis activity of a romantic partner - versus that of a stranger - who is present influences one's physiological arousal during moments of acute stress, called coregulation, is largely unknown. This study tested the effects of physiological coregulation in romantic partners using a repeated-measures design with four samplings of salivary cortisol as outcome and one between-group factor (pairing of partner) to determine whether one's HPA-regulated acute stress response is in part dependent on that of their romantic partner.

The present study recruited young dating couples (N=40; Mean age=23; Mean relationship length=2 years; 48% Hispanic) from the University of Miami who provided valid data including demographic and physiological data. Participants were randomized into two group conditions: couple (paired with romantic partner) or stranger (paired with a study confederate). The experimental stress task asked participants to respond to a scenario wherein romantic partner was experiencing acute physical pain. Attachment (Measure of Attachment Qualities) and demographic data was collected before stress

task. Salivary cortisol was sampled before stressor task onset (T1), immediately after 5-minute stress task (T2), and at eight (T3) and 18 minutes (T4) after.

It was hypothesized that H1a) higher cortisol levels at T3 than T2 (controlling for T1) will be positively associated with attachment anxiety, followed by attachment avoidance, then attachment security; H1b) attachment avoidance scores would be associated with highest cortisol levels at T4, followed by attachment security, and then attachment anxiety; H2a) cortisol levels at T3 would be predicted by partners' cortisol levels at T2, but only when paired with romantic partner (versus stranger); H2b) cortisol levels at T4 would be predicted by partners' cortisol levels at T3, but only when paired with romantic partner (versus stranger); H2c) the influence of partner's prior cortisol levels on one's cortisol levels would be strongest when paired with romantic partner (versus stranger) and when one is high in attachment anxiety, followed by security, then avoidance.

Overall, participants were relatively high in attachment security ( $M=3.79$ ,  $SD=.40$ ) and low in attachment avoidance ( $M=1.50$ ,  $SD=.44$ ) and attachment anxiety ( $M=1.50$ ,  $SD=.49$ ). General linear modeling indicated that participants' cortisol levels increased during the stress task and decreased after cessation,  $F(1,39)=15.20$ ,  $p < .001$ . A series of hierarchical linear regression models were conducted to test study hypotheses, controlling for relevant covariates such as age, gender, and time of day. Overall, none of the hypotheses were empirically supported. Results fail to relate individual cortisol levels during acute stress to their attachment to romantic partner or the cortisol levels of their study partner. Future studies would benefit from a larger sample size and adding measurements of cortisol at 10, 20, 30, 45, 60, 90, and 120 minutes after cessation of

stress task in order to ensure reactivity and recovery processes are captured.

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## CHAPTER 1: INTRODUCTION

### *Overview of Stress Physiology: Role of the HPA Axis and SAM Processes*

As described by Kudielka and colleagues (2004) and Sapolsky, Romero, and Munck (2000), once the brain detects the presence of a stressor or perceives a threat, an elaborate series of cascading events occurs in body. The hypothalamus-pituitary-adrenal cortical (HPA) axis and the sympathetic nervous system (SNS) are engaged immediately. The hypothalamus is the first area initiated in the stress response. Neurons in the paraventricular nucleus (PVN) of the hypothalamus secrete corticotrophin-releasing hormone (CRH), also known as corticotrophin-releasing factor (CRF), and arginine vasopressin (AVP). Vasopressin plays a role in homeostasis, including the regulation of blood pressure through vasoconstriction. However, it is CRH that initiates the next step of the HPA axis.

After its release by the hypothalamus, CRH travels to the anterior pituitary gland (Miller, Chen, & Cole, 2009). Exposure to CRH stimulates the pituitary to engage in two important processes: activating noradrenergic neurons in the brain and initiating the release of adrenocorticotropin hormone (ACTH). The noradrenergic neurons are part of the locus caeruleas/norepinephrine (LC/NE) system, a component of the Sympathetic Adrenomedullary System (SAM). This system stimulates the adrenal medulla to release norepinephrine and epinephrine, critical neurotransmitters for the SNS that drive the immediate “fight or flight” response. While this process occurs, the pulse of ACTH released by the pituitary moves through peripheral circulation to the adrenal cortex. The adrenal cortex releases corticosteroids, including mineralocorticoids, glucocorticoids, and dehydroepiandrosterone (DHEA).

The story of how the body responds to stress does not end there. The major product of HPA axis activation, cortisol, plays a key role in mediating systemic responses in the body both to mobilize it immediately and to return it to a homeostatic set point (Elenkov & Chrousos, 2006).

### *Stress, Cortisol, and Its Health Implications*

Of the glucocorticoids released by the adrenal cortex, cortisol has received the most attention by researchers. This hormone regulates cellular activity through glucocorticoid receptors and mineralocorticoids receptors found in most tissues within cells (Buckley & Schatzberg, 2005). Once cortisol is released by the adrenal cortex, it binds to one of these receptors. The resulting complex translocates to the cell nucleus, modifies genetic expression routes, and modifies cellular activity (Miller, Chen, & Cole, 2009). One such activity might be the breakdown of lipids and proteins by cells (Miller, Chen, & Cole, 2009).

Cortisol is a popular subject of research due to its widespread influence in regulating important bodily processes. For instance, cortisol is involved in learning, memory, and emotion (via effects it has on glucose transport and utilization in the brain); on appetite, metabolism, and reproductive behavior and physiology (decreasing both the release of growth hormone and insulin sensitivity); and on inflammatory and immune responses (Sapolsky, Romero, & Munck, 2000). In addition to these processes, cortisol also triggers the liver to produce additional glucose (Miller, Chen, & Cole, 2009). In this capacity, cortisol can promote the release of additional energy for hours, as opposed to the immediate, yet short-lasting bursts of energy provided through the SAM (Denson,

Spanovic, & Miller, 2009).

Acute cortisol responses to a laboratory-induced stressor are reflective of overall HPA-axis health and functioning (Matthews, Gump, & Owens, 2001). In their study of acute stress reactivity, Matthews and colleagues (2001) measured cortisol before, during, and after a speech task in 61 adults (mean age 35 years old) in addition to taking self-report measures of chronic stress. The authors found that acute cortisol reactivity and recovery was blunted in participants who endorsed high levels of chronic stress relating to work and relationships. Porter and colleagues (2003) support this finding in a cohort of breast cancer survivors in the days before, during, and after a mammogram. The survivors, who endorsed elevated chronic stress, evidenced blunted cortisol reactivity and recovery in comparison with healthy, age and race-matched controls. A blunted acute cortisol response in the chronically-stressed has been suggested to play a role in the etiology of stress-related bodily disorders (Heim, Ehlert, & Hellhammer, 2000). However, elevated acute cortisol responses may also be present and have been associated with chronically depressed mood and anxiety (Kara et al., 2000; Kirschbaum & Hellhammer, 1994). Overall, cortisol changes in response to a laboratory stressor are associated with chronic mental and physical health, though patterns of under and overactivation may relate to different outcomes.

Cortisol is a useful proxy for indexing stress objectively (Sapolsky et al., 2000). More specifically, salivary cortisol has been found to reliably increase in response to acute laboratory-induced stressors (Dickerson & Kemeny, 2004; Foley & Kirschbaum, 2010). In their meta-analysis of cortisol responses to acute stressors, Dickerson and Kemeny (2004) found that the peak cortisol response to an acute psychological stressor

occurs in general 21-40 minutes after onset of the stress. In tasks involving uncontrollability and a socio-evaluative threat, increased cortisol elevations at both 0-20 and 21-40 minutes post-stressor were found. Foley and Kirschbaum (2010) published an updated review and concluded that a) cortisol levels increase within 10 minutes of stress onset and b) peak within 10-30 minutes after stress cessation. Overall, these studies show that cortisol levels can be detected in saliva as early as 10 minutes after a stress task.

Cortisol output has important long-term health implications. Although short-term HPA axis activations are adaptive for functioning, extreme, frequent, or chronic activation is related to negative health outcomes (Adam & Kumari, 2009). Chronic overactivation of the stress system and the resulting prolonged exposure to stress hormones has been referred to as *allostatic load* (McEwen & Wingfield, 1993). It has been established that high allostatic load from chronic stress is positively associated with poor immune functioning (for review, see Herbert & Cohen, 1993). Frequent or sustained increases in cortisol levels may make the body more susceptible to developing diseases such as hypertension, cardiovascular disease, diabetes, and cancer (McEwen, 1998). Several biopsychosocial models have been proposed to explain the pathways among stressor types, psychological responses, neuroendocrine activity, and disease progression (Antoni et al., 2006; Carney, Freedland, Miller, & Jaffe, 2002; Peralta-Ramirez, Jimenez-Alonso, Godoy-Garcia, & Perez-Garcia, 2004). These models illustrate possible ways in which cancer tumor biology, rheumatic disease, and heart disease develop and, in some instances, create new sources of psychophysiological stress. Central to these models are CNS stress responses that trigger HPA axis activation and sustained secretion of cortisol by the adrenal cortex.

### *Types of Stress and Cortisol*

These internal physiological processes are complemented by psychological factors involved in the stress response. Cognitive and interpersonal processes of appraisal and emotion regulation can ameliorate or aggravate the stress response (Denson, Spanovic, & Miller, 2009). Currently, the interaction between psychosocial and ongoing physiological stressors is an emerging field among emotion researchers (Denson, Spanovic, & Miller, 2009; Dickerson & Kemeny, 2004; Saxbe & Repetti, 2010). Denson and colleagues (2009) offer a comprehensive review of this literature and relate it to research on stress and the HPA axis. Drawing from appraisal theory, the authors propose a model to describe the process by which an emotional stressor affects HPA axis up or down-regulation. For instance, if a stressor is immediate or imminent, associated appraisals may be surprise, worry, or anticipation. If however a stressor is appraised as a threat to one's social status, associated appraisals may be rumination, submissiveness, or self-conscious emotions. The authors employed meta-analytic techniques to determine that cortisol and immune responses to acute stress inductions differ depending on the appraisals and emotions induced.

Denson and other researchers have concluded that the HPA-axis is especially sensitive to conditions of social threat and interpersonal rejection (Denson, Spanovic, & Miller, 2009; Dickerson & Kemeny, 2004; Kaplan, Manuck, Clarkson, Lusso, & Taub, 1982; Sapolsky, 1993). For example, Miller's (2008) glucocorticoid-resistance model has received support from literature on glucocorticoid receptor sensitivity in persons experiencing social isolation (Cole et al., 2007; Hermes et al., 2009; Lutgendorf et al., 2009). Furthermore, interpersonal and social stressors have a reliably demonstrated



deleterious effect on our health (i.e., Schmaling & Sher, 2000). For instance, Kiecolt-Glaser and Newton (2001) summarized in a meta-analysis that poor marital functioning is related not only to greater depression and perceived stress but also impaired cardiovascular and endocrine health.

As mentioned earlier, Dickerson & Kemeny (2004) found that perceived social hierarchy and evaluative social threat predicted cortisol secretion in an acute laboratory stress paradigm. In their meta-analysis of 208 studies, they determined that socio-evaluative threat was a major component of stressors that elicit the largest HPA-axis activation and result in the most prolonged recovery (Dickerson & Kemeny, 2004). Their conclusion indicates that interpersonal dynamics and perceived social roles are core features of our stress response mechanisms.

#### *Adult Attachment Theory*

The exploration of couples' interactions in the context of stress lends itself to Adult Attachment Theory, a theoretical framework, interpersonal in nature, described as one of the most powerful to describe the way in which our relationships with others affect the way we regulate stress (Schmidt, Nachtigall, Wuethrich-Martone, & Strauss, 2002). Attachment theory (Bowlby, 1969, 1973, 1980, 1982) posits that the attachment system, which is formed during infancy and early childhood through interactions with a primary caretaker, develops our expectations of how likely others will respond to our needs. In early childhood, these expectations are determined by the perceived availability of one's caretakers, most often their parents. These expectations, based on our perception of the reliability of help from these objects of attachment, form an "internal working model," a

collection of cognitive schemata that has significant implications for how we interact with those closest to us. In adulthood, these expectations are based on both our prior set of internal working models and our interactions with new friends, family, and romantic partners.

The attachment system is a stress-mitigation system, wherein an individual under duress uses relationships with others to return to a homeostatic level of comfort (Bowlby, 1988; Mikulincer & Shaver, 2003). In other words, it is a behavioral and affective regulation system that, once activated by environmental threat or psychological distress, maintains activation until the stress is resolved. Moreover, individual differences exist in the way adults address attachment activation.

Although different prototypes of adult attachment orientations have been proposed, there exists evidence to suggest that they fall on two dimensions. Early two-dimensional conceptualizations of adult attachment included one “anxiety” dimension and one “avoidant” dimension (Bartholomew & Horowitz, 1991; Brennan et al., 1991). People could therefore be described as being high or low in each dimension. Carver (1997) examined this conceptualization and found support for a modified two-dimensional description of attachment, with these dimensions described as secure-avoidant and anxious-ambivalent. If the quality of interactions with the attachment figure in times of need is favorable, and the interaction ameliorates distress, then an individual’s schema about interactions with that attachment figure will reflect high attachment security (Bowlby, 1973).

However, sometimes the attachment figure is not perceived to be available, physically or emotionally, in times of need. When this consistently occurs, an individual

in distress is not able to engage in proximity seeking. Instead, an individual employs a secondary attachment strategy of either hyperactivation or deactivation of the attachment system. This is similar to the fight-or-flight response system in stress. One attachment system is to become hypersensitive to threats and become excessively dependent on one or more people in times of need. It may involve being hypervigilant in terms of proximity seeking and need for reassurance. It would be the “fight” activation in the fight-or-flight analogy. This reflects high attachment anxiety-ambivalence, and is categorized by intense self-focus, clinginess, and overall hyperactivation of the attachment system (Mikulincer & Shaver, 2003). High attachment anxiety-ambivalence is associated with high distress, manifesting in clinical and sub-clinical levels of depression (Kim, Kashy, & Evans, 2007). On one end of the anxious-ambivalent dimension is Anxious-Ambivalence, Worry type (AAW). High AAW is associated with a focus on worrying about future abandonment. On the other end of the dimension, Anxious-Ambivalence, Merger type (AAM) describes a strong desire to reunite with and cling to an attachment figure.

Another attachment system is to deactivate the attachment system, becoming desensitized to threats and avoid intimacy altogether. This tendency is associated with high attachment avoidance, described by an underutilization social support and self-isolation in response to distress instead of seeking outside help or assurance. It is similar to the “flight” activation in the fight-or-flight analogy. High attachment avoidance is associated with avoiding expressing one’s distress and having strained interpersonal relationships, especially romantic relationships (Mikulincer & Shaver, 2007).

The applicability of adult attachment theory to understanding psychosocial and psychophysiological phenomena cannot be overstated. The adult attachment system impacts an array of life events (Hazan & Shaver, 1987). Once the “internal working model” is developed within an individual, emotions, expectations, goals and behavioral strategies can be organized conceptually in terms of attachment orientation along attachment dimensions. Attachment-related behaviors and goals cut across all aspects of relationships within the social world (see Dykas & Cassidy, 2011; Mikulincer & Shaver, 2003 for reviews). Since the primary goal of the attachment system is to reduce distress, it is not surprising that attachment-related attitudes, expectations, and behaviors also influence stress and stress-regulation processes. Attachment activation is in essence an affect regulation strategy; attachment insecurity has been associated with emotion dysregulation (Allen & Miga, 2010). Allen and Miga (2010, p187) go so far as to say, “almost certainly, adaptive emotion regulation capacities grow and develop most effectively in the context of secure attachment relationships.” Researchers have established that the attachment system is activated in response to negative affect (Sroufe & Waters, 1977) and distress (Shaver & Mikulincer, 2002), but we are only beginning to understand how specific attachment dimensions relate to physiological markers of stress, including cortisol levels, heart rate, blood pressure, and electrodermal activity (Diamond & Hicks, 2004; Kim, 2006; Maunder & Hunter, 2001).

A growing body of research is evaluating whether the hyperactivating or deactivating strategies of the anxious-ambivalent and avoidant attachment styles (respectively) are associated with parallel physiological arousal responses in the body. To answer questions about attachment insecurity and physiology in response to stress,

researchers have turned to a well-researched system involved in stress and emotion regulation: the HPA axis. Since HPA activity is particularly sensitive to interpersonal stressors, its regulation is likely connected to the attachment system (Powers et al., 2006). Therefore, cortisol levels may provide information about not only HPA functioning, but of attachment system activation as well. The relevant findings to date was summarized first from studies that recruited at individual level, then from those that recruited participants at the couple level.

### *Attachment and Stress Regulation*

One question researchers have asked is if individuals high in attachment anxiety have elevated physiological responses to stress (see review by Diamond & Fagundes, 2010). Attachment theory would suggest that individuals higher in attachment anxiety are hypervigilant when responding to cues, reacting strongly when exposed to stress but recovering quickly when stimuli ceases (Mikulincer, M., & Shaver, 2007). At the individual level, attachment anxiety does indeed appear to be associated with elevated levels of cortisol in response to stress, however this trend persists both during and after exposure (Brooks, Robles, & Schetter, 2011; Diamond, Hicks, & Otter-Henderson, 2008; Powers et al., 2006; Quirin, Pruessner, & Kuhl, 2008). Quirin and colleagues (2008) used an acute laboratory stressor to activate participants' attachment styles. The authors used a startle probe consisting of a sound recording of an electric shock to induce acute stress in 48 women. Before and after the stress procedure, the researchers obtained salivary cortisol samples from participants. Results showed that individuals high in attachment anxiety had on average a larger net increase in cortisol concentration in

response to stress than did participants who were low in attachment anxiety. Diamond and colleagues (2008) found a similar effect of attachment anxiety on cortisol levels in individuals who were physically separated from their romantic partners over 4-7 days. In an acute stress paradigm, Powers and colleagues (2006) asked young adult romantic partners to discuss and resolve a topic of conflict they had endorsed arguing about in the past. Salivary cortisol was sampled before, during and after the 15-minute stress task. Attachment anxiety was positively associated with an increase in individuals' salivary cortisol levels. Specifically, men high in attachment anxiety (vs. low attachment anxiety) were found to have faster cortisol release during stress and longer recovery to baseline levels following the stress task. Similarly, Brooks and colleagues (2011) recently found that attachment anxiety was positively associated with heightened cortisol release in men during exposure to an acute, laboratory-induced stressor.

Similar to findings on attachment anxiety, attachment avoidance has been found to be associated with elevated cortisol levels in response to acute laboratory-induced stressors. Findings from one study indicate that individuals who are high in attachment avoidance have elevated cortisol levels when presented with abandonment-related imagery in a controlled laboratory setting (Rifkin-Graboi, 2008). Powers and colleagues (2006) reported that women high in attachment avoidance (vs. low attachment avoidance) had higher cortisol levels before entering the laboratory and during the task. They also had lower cortisol levels post-stress task than did participants low in attachment avoidance. Brooks et al. (2011) also found that attachment avoidance was positively associated with higher cortisol release in during acute stress exposure. It should be noted

that findings on attachment security and individual cortisol responses to an acute stressor are lacking.

In adults, the attachment system is activated not only when individuals face personal threat but also when an individual observes a loved one who is in distress (Mikulincer & Shaver, 2003). Bowlby (1982) argued that attachment security is a foundation for caregiving, because a sense of security (comfort with closeness and interdependence) allows individuals to attend more responsively to the partner's needs. Similarly, in adulthood, relationships involve being an attachment object to the partner – offering comfort, reassurance, help, and safety. That is, partners in a romantic dyad have individual attachment orientations and are also each other's objects of attachment. If an individual is in a romantic relationship, her ability to regulate stress may therefore be partially dependent on the attachment orientation of her partner.

Recent research has found support for this assertion (see Collins, Guichard, Ford, & Feeney, 2006). For instance, adults in a romantic relationship who have high attachment anxiety concerning their partner tend to be overinvolved, controlling, and pushy in how they care for their partner and also report higher levels of personal distress in response to observing their partner's distress (Feeney & Collins, 2001). Additionally, individuals who have high avoidant attachment to their partners are both less sensitive to their partner's needs and less willing to provide comfort in response to their partners' expressions of distress (Feeney & Collins, 2001). These findings indicate that attachment plays a role in explaining caregiving attitudes and behaviors. Kim and Carver (2007) explored this idea in spousal caregivers of cancer patients. Four hundred caregivers were surveyed and assessed for attachment in addition to several aspects of providing care.

The authors found that attachment and gender of caregivers predicted the frequency with which they provided care. All caregivers high in attachment avoidance reported providing less frequent tangible support, and several interactions with gender were noted. Additionally, attachment of a caregiver predicted the degree of difficulty they have in their caregiving role, with caregivers high in avoidance reporting the greatest difficulty and those high in security reporting less difficulty (Kim & Carver, 2007).

When couples are exposed to an acute stressor, one's attachment appears to predict cortisol output in their partner's response to stress (Porter et al., 2012; Powers et al., 2006). Powers and colleagues (2006) reported that, among male participants exposed to a laboratory-induced acute stressor, men whose partners were high in attachment security had the lowest cortisol reactivity to stress. Conversely, men whose partners were high in attachment avoidance and/or anxiety had higher cortisol levels throughout the task and during the recovery period.

In summary, adult attachment theory provides a dynamic framework for conceptualizing stress regulation between two persons in an intimate relationship. Attachment can describe patterns of cognitions, emotions, and behaviors that have the potential to either downregulate or upregulate perceived stress. To date, the literature indicates that attachment avoidance and attachment anxiety are positively associated with cortisol levels during reactivity; findings are mixed about these associations during recovery from stress. It is also not clear how cortisol levels of individuals high in attachment security change in response to acute stressors. Additionally, the paucity of published findings on couples' attachment and cortisol responses to an acute laboratory-induced stressor suggests a need for further exploration in this area. It would be good to



know, for instance, if Powers' (2006) findings replicate in another sample. Only in laboratory designs can momentary reactivity and recovery be measured, and the relative contribution of attachment thus identified, while controlling for potential confounds. Still, existing literature indicates that when exploring stress in adults who are in a relationship, both partners' attachment orientations can provide important information as each partner has their own reaction to observing the other under stress.

### *Attachment, Coregulation of Stress, and Cortisol*

How does a significant other influence one's own stress regulation? Is there reciprocity of social exchange that can attenuate or exaggerate an emotional exchange? These questions have forged into a recently burgeoning area of research with contributions from the areas of social, emotion, and health psychology (reviewed by Larson & Almeida, 1999). This reciprocal, momentary exchange of emotional and physiological arousal between two individuals in a relationship is referred to as "coregulation" (Sbarra & Hazan, 2008). In their review of coregulation and adult attachment, Sbarra and Hazan (2008) offer what they describe as a coregulatory model of normative attachment. The authors posit that coregulated physiology between romantic couples results in part from the emergence of felt security between romantic partners. That is, the perceived availability of an adult individual's attachment figure, often their romantic partner, plays a major role in their maintenance of autonomic homeostasis (Sbarra & Hazan, 2008).

Coregulation does not merely refer to synchrony, wherein two persons would have the same physiological arousal level. It also does not refer to merely stress-

buffering; the effect of coregulation on physiological arousal can hypothetically also amplify the magnitude of an individual's stress response (Sbarra & Hazan, 2008).

According to the negative affect reciprocity model, this amplification effect may be most salient when partners of a dyad are experiencing acute stress or alarm (Gottman, Coan, Carrere, & Swanson, 1998; Levenson & Gottman, 1983). This model explains that partners escalate in stress as they seek to interpret and address their partner's corresponding stress. Therefore, one's acute stress response may be apt to amplification, rather than dampening, when observing a loved one experience distress. Other research suggests that the presence of a loved one may increase fears of evaluation, thus heightening the stress response (Lepore, 1998). The degree to which coregulation is beneficial, and in what context, is largely unexplored.

Coregulated physiology refers to the momentary up-or-down regulation of one's arousal as a function of the perceived availability of their attachment object, such as a romantic partner (Hazan, Gur-Yaish, & Campa, 2004). When physiological arousal is coregulated, each partner in the dyad will have arousal levels that are in part dependent on being in the presence of the other. Sbarra and Hazan (2008) offer a way to test for coregulation. The authors say that physiological functioning should be modeled as a bivariate system, wherein physiology in one partner is (partially) dependent on their partner's prior physiology.

Physiological coregulation has been documented in several studies, including studies of parent-child physiological arousal in animals (Hofer, 1984) and humans (Feldman & Eidelman, 2003; Neu, Laudenslager, & Robinson, 2009; Schrieber et al., 2006) as well as within adult human dyads (Berg & Wynne-Edwards, 2002; Helm,

Sbarra, & Ferrer; 2011; McClintock, 1971; Saxbe & Repetti, 2010; Schrieber et al., 2006). Perhaps the most well known study in this area found that college roommates' menstrual cycles become synchronized over time (McClintock, 1971). Overall, findings from these studies have evidenced that the phenomenon occurs across a variety of close relationships, though very few examine coregulation in stress reactivity paradigms.

Findings from three studies that explored the stress coregulation phenomenon with adults manifested in cortisol values have been mixed, as are saliva sampling and analytic methodology. For instance, Berg and Wynne-Edwards (2002) found no association between spouses' cortisol levels in a sample of nine couples that were expecting and later caring for their first child. Saliva was sampled weekly at participants' homes. Only the data collected from both persons within 90 minutes interval on the same or adjacent day were included in the analysis. Correlations of time-matched samples within couples were compared against the null hypothesis that the average Pearson correlation coefficient would not be different from zero. Alternatively, Schreiber and colleagues (2006) reported positive correlations in concurrent cortisol levels between spouses in two separate studies (individual  $n$ 's = 221 and 107). Saliva sampling occurred once per day over three days. A Student  $t$  test was used to examine significance of Pearson's  $r$  correlations.

Only one other study, conducted by Saxbe and Repetti (2010), has investigated the association of cortisol levels between romantic partners. In this study, saliva was sampled four times per day over three days. This study reported positive correlations in concurrent levels of cortisol between 30 married couples. Hierarchical linear modeling analyses to represent the change of cortisol values throughout the day and to analyze

trait-level (between-person) and state-level (within-person) factors indicated that increases in cortisol (above their average cortisol value for that sampling occasion over three days) in one spouse were associated with similar increases in their spouse. Moreover, coregulation of cortisol was evidenced when couples were together, as opposed to when they were physically separated. Finally, less satisfied spouses in their marital relationship had greater similarity in change patterns of cortisol with their partner over time, supporting the moderating effect of marital satisfaction.

This last finding suggests that adult attachment, a similar construct to relationship quality or marital satisfaction, may have similar effects on coregulation of cortisol. As Sbarra and Hazan (2008, p157) suggest, “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... . . . 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.”

Adult attachment has already been evidenced to moderate coregulation of affect and cardiovascular activity in romantic partners (Butner et al., 2007; Helm et al., 2011). The next step would be to test this moderation effect with cortisol as the outcome. Helm, Sbarra, and Ferrer (2011) recently explored coregulation and attachment in 32 heterosexual couples. Participants were asked to engage in three sequential tasks: rest with no communication, maintain three minutes of eye contact, and “mirror each other’s physiology.” Respiration and heart rate were measured as outcomes, and adult attachment was a primary predictor. The authors found greatest evidence for dyadic

coregulation during the gazing task, and attachment avoidance predicted less coregulation. Whether similar patterns exist with partners' cortisol levels in response to an acute stressor have yet to be asked. Additionally, the authors suggest that future studies should also establish that dyadic coregulation occurs only between attached partners, as opposed to between two strangers (Helm et al., 2011, p12).

Overall, these recent findings indicate that not only do romantic dyads coregulate, but also that attachment is highly relevant to understanding this phenomenon. No study to date has explored the role of attachment in predicting coregulated cortisol levels between partners. An investigation in this area is warranted, especially because the HPA-axis is particularly sensitive to interpersonal dynamics. Moreover, no study to date has explored coregulation of cortisol in response to an acute, laboratory-induced stressor; only results from less structured, naturalistic designs are currently available.

A laboratory design is desirable for testing coregulation for several reasons. First, this setting allows the researcher to control for confounding variables that may influence physiological changes, such as environmental factors, time of day, and extent and quality of interpersonal interaction during the experiment. Controlling for extraneous variables allows the researcher to test hypotheses about cause-and-effect. Moreover, participants are exposed to the exact same stressor, allowing for psychometric properties of the stressor to be evaluated. A laboratory design is also important for testing acute stress, because physiological measures can be synchronized with precise times of baseline, and induction and cessation of a stressor. This methodological consideration allows researchers to investigate momentary fluctuations in physiological arousal and attribute those levels/changes to specific environmental cues.

## *Study Hypotheses*

### I. Attachment and Individual Stress Response

Individuals' attachment orientations will predict physiological stress response.

- a) All individuals will show increased cortisol levels from before stress onset to cessation of the stressor. However, higher cortisol levels at cessation of the stressor will be positively associated with attachment anxiety, followed by attachment avoidance, then attachment security.
- b) All individuals will show decreased cortisol levels from stress cessation to the end of resting. However, attachment avoidance will be positively associated with more elevated cortisol levels at the end of resting, followed by attachment security, then attachment anxiety.

### II. Coregulation of Stress

Couples will coregulate their physiological stress responses.

- a) Condition where individuals paired with their romantic partner (couple condition) vs. with a stranger (stranger condition) will influence cortisol changes of individuals. Specifically, in the couple condition, person A's cortisol level after stress cessation will be positively related to the partner, person B's immediately prior cortisol level. Such association in the stranger condition will not be significant.
- b) Condition where individuals paired with their romantic partner (couple condition) vs. with a stranger (stranger condition) will influence

cortisol changes of individuals. Specifically, in the couple condition, person A's cortisol level at the end of the resting period will be positively related to the partner, person B's immediately prior cortisol level. Such association in the stranger condition will not be significant.

Attachment will predict coregulation of reactivity and recovery.

- c) In the couple condition, the associations stated in Hypothesis 2a and 2b will be more pronounced with higher attachment anxiety scores, followed by higher attachment security scores, then lower attachment avoidance scores. These relationships will not be significant in the stranger condition.

## CHAPTER 2: METHODS

### *Participants*

Enrolled participants consisted of 26 young adult couples (total N=52) at least 18 years of age who were in a current heterosexual romantic relationship at the time of study participation. “Romantic relationship” in this study is defined as “a committed, monogamous, emotionally-intimate relationship”. Minimum three months in the relationship was selected to screen out couples whose relationship is more transient than established. Eligible participants were identified from the Introductory Psychology courses or flyers posted throughout the campus. Both the student and their partner were invited to the study. None of the participants evidenced any major health concerns; however, one participant had jaw surgery the day before study participation, and thus their saliva data could not be collected.

### *Study Design*

*Experimental Session Overview.* After being seated, each participant began the study. The study involved a resting period lasting 28 minutes, a task preparation period lasting three minutes, a stress task period lasting five minutes, and a recovery period lasting 19 minutes. The entire procedure lasted approximately 70 minutes, which included time for task instruction and debriefing. For an overview of the study timeline, see Study Timeline: Appendix 1.

Overall, saliva was sampled four times. T1 occurred after 28 minutes of resting, before any stress task was introduced. T2 occurred immediately after the stress task ends, which was 11 minutes after T1. T3 occurred eight minutes after the stress task ends, and



T4 occurred 19 minutes post-stress task. At each sampling, participants answered a measure of subjective stress.

The study employed a design with one between-group factor: stranger condition vs. couple condition.

*Procedure for Randomization.* Upon arrival of the couple at the laboratory, a research assistant (RA) briefly introduced the study and obtained the informed consent form. Participants were then asked to rinse their mouth with water at a nearby restroom. Next, the couple was asked to pick a piece of paper folded in a jar, indicating to which group (stranger vs. couple) they were assigned. This between-group condition, however, was predetermined, so that two RA's were on-hand to play the "partner" role in the stranger condition. Predetermined randomization occurred upon couple enrollment such that an equal number of couples were assigned to each group.

If a pair was assigned to the couple condition, both romantic partners were retained in the same room. If a couple was assigned to the stranger condition, a dyad of confederates was brought in and introduced themselves as study participants. Each participant was paired with an opposite-sex confederate. One pairing was ushered to a separate room along with an experimenter. Unless otherwise noted, all study pairings are referred to as "partners" hereafter. The following procedures are identical for both the stranger and couple conditions.

*Study Procedure.* Participants were seated facing 90 degrees to each other while they completed the study measures. After completing the measures, partners were instructed to sit quietly. This pre-task period lasted 28 minutes in total. At the completion of the pre-task period, each participant was asked to provide a brief saliva

collection (T1). The experimenter instructed participants on proper saliva collection with a salivette. T1 saliva collection measured baseline cortisol level. After the T1, the participants were told they are now beginning the experimental phase of the study.

Participants were assigned to either the speaker participant (SP) or listener participant (LP). Participants in the speaking condition were asked to give a five-minute speech; and participants in the listening condition listened to the speech, imagining as if they were the person who was looking for help for their romantic partner (Stress Manipulation Scenario: Appendix 2). Seats of the study partners were turned to face each other at 45-degree angles.

The RA asked study partners to play close attention and to imagine being in a scenario. The Stress Manipulation Scenario was read by the RA. The RA was trained to read the scenario with crescendoed emotional intensity. After the scenario was read, SP's were given the copy of the scenario along with suggested aspects to be included in the speech. They were reminded that they had three minutes to prepare a continuous speech lasting five minutes.

During the three minutes preparation time for SP, LP's were asked to write down descriptions of their typical class agenda and how these classes will help them reach their academic and professional goals. This activity is to let LP's engage in a neutral activity and to prevent potential interference by talking to or staring at the participants in the speaking condition.

While SP's delivered their speech, LP's were asked to listen to the speech carefully. Engaging in conversation with the participant in the speech condition was not

allowed. If SP's completed the speech sooner than five minutes, the RA instructed them to reiterate some of the suggested points until five minutes was reached.

Immediately following the stress manipulation, the second saliva collection (T2) occurred. After the T2 saliva collection was completed, participants were asked to remain seated calmly and comfortably as much as possible while closing their eyes. After eight minutes, the third saliva collection (T3) occurred. Participants were asked to continue sitting quietly. Ten minutes after T3, the fourth saliva collection occurred (T4). Following T4, a debriefing session was held, followed by providing incentives for participating in the study.

*Debriefing.* At the conclusion of the stress manipulation and recovery sessions, all participants were fully debriefed. Participants in the couple condition were debriefed about the purpose of the study; that is, to examine the psychological and physiological co-regulation among dating couples. For participants in the stranger condition, they were told that their study partner who they were told as another study participant was a research assistant of the study, not an actual participant, in order to compare the correlations of stress reactivity to the task (making a speech or listening to the speech about the stressful situation the couple was imagining) between the couple with between the strangers. All concerns or questions raised with regard to any study procedure and materials were fully answered. All study participants were also given the information to obtain psychological services that they may require due to participating in this study, such as the Psychological Service Center and the Counseling Center.

*Incentives for Study Participation.* Each couple from the current Introductory Psychology pool received 6 course credits (for the partner who was enrolled in the

Introductory Psychology course). In addition, each couple selected an additional incentive option, either \$20 in cash for current Introductory Psychology pool/\$30 in cash for former Introductory Psychology pool (or general student population) or a raffle ticket for the chance to get \$200, for their time and the cost involved in traveling to the study location. The exact time, date, and location for the drawing of the raffle tickets was specified. There was one raffle ticket selected for the \$200 cash prize. The notification was sent to participating students' emails, blind copied to all the study participants so that all participants were notified about the results of raffle ticket drawing.

### *Measures*

#### *Adult Attachment: Psychosocial Predictors*

The Measure of Attachment Quality (MAQ; Carver, 1997) is a 14-item self-report measure of four attachment dimensions: security (*e.g.*, “*It feels relaxing and good to be close to someone.*”), avoidance (“*I prefer not to be too close to others.*”), anxious-ambivalent merger (AAM) type (“*My desire to merge sometimes scares people away.*”), and anxious-ambivalent worry (AAW) type (“*I often worry that my partner doesn't really love me.*”). Each item is presented in the form of a statement. Respondents are instructed to endorse the degree to which they agree with each item, using a four-point Likert-scale, ranging from 1 to 4. Response options are: Strongly Disagree, Somewhat Disagree, Somewhat Agree, and Strongly Agree. Each sub-scale was scored by averaging responses (after appropriate reversals).

### *Psychological Stress*

*Subjective stress.* Three adjectives (stressful, unpleasant, and strained) were used to measure psychological stress. Participants were asked to rate the extent to which they currently feel. Response options were: Not at All, A Little Bit, Somewhat, Quite a Bit, and Very Much, scored 1 to 5, respectively. An overall subjective stress score was calculated for each participant at each timepoint by averaging their three responses. Overall subjective stress scores therefore had a minimum value of 1 and a maximum value of 5, with higher scores indicating greater subjective stress.

*Daily life stress.* Daily life stress was measured using the Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983). The PSS asks respondents to rate the frequency of having 10 thoughts or feelings during the past month. Response options were: Never, Almost Never, Sometimes, Fairly Often, and Very Often, scored 0 to 4, respectively. An overall score was calculated by summing responses after appropriate reversals.

### *Salivary cortisol: Biological outcome.*

Salivary cortisol was sampled. Corticosteroid binding globulin levels, which may fluctuate within an individual, mediate differences in unbound and total (unbound and bound) cortisol levels within individuals; however, this is not a factor when measuring salivary cortisol since only the unbound levels are obtained (Foley & Kirschbaum, 2010). Moreover, unbound (free) cortisol levels among plasma, serum, and saliva are highly correlated ( $r > .90$ ; Foley & Kirschbaum, 2010). For this reason, in addition to being a less intrusive sampling method, salivary cortisol is a good outcome for investigation of HPA activity.

Salivary cortisol was collected in salivettes using a cotton swab (Sarstedt, Rommelsdorf, Germany). Samples were stored in a secure freezer at -80 degrees Fahrenheit until shipment for analysis. Assays were conducted at the Technical University of Dresden, Germany. A commercial chemiluminescence immunoassay (IBL, Hamburg, Germany) was used (lower detection limit of 0.41 nmol/L). Additionally, inter- and intra-assay covariance is < 10% across the expected range of cortisol levels (Rohleder, Beulen, Chen, Wolf, & Kirschbaum, 2007).

A total of 51 participants provided saliva samples. One participant had jaw surgery the day before study participation; her jaw had limited mobility and thus saliva was not collected. Saliva from the remaining participants was collected at four occasions (Timepoints 1-4), yielding a potential total sample of 204 cortisol values. However, six samples were un-analyzable by the processing lab due to insufficient saliva. Overall, 198 cortisol values (97%) were available for use in statistical analyses.

*Covariates.*

*Time of sampling.* Cortisol has a naturally occurring diurnal cycle, peaking 30-40 minutes after waking and gradually declining throughout the day (Fries, Dettenborn, & Kirschbaum, 2009; Hall et al., 2011; Posener, Schildkraut, Samson, & Schatzberg, 1996). Therefore, time of first sample was entered as a covariate in all hypothesis testing.

*Participant role.* The stress task was designed such that both SP's and LP's experience stress. To control for any effects of role assignment, this factor was entered as an additional covariate in cortisol analyses.

*Gender.* Literature on cortisol indicates that while females have lower cortisol levels throughout the typical day than do males, cortisol changes in reactivity are comparable between the genders (Kudielka, Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004; Van Cauter, Leproult, & Kupfer, 1996). Therefore, gender was entered as a covariate.

## CHAPTER 3: RESULTS

### *Preliminary Analyses*

#### *Sample Characteristics.*

Out of a total of 52 participants, 40 provided valid data for study variables. Chi-square and independent samples t-tests for all demographic variables and study predictors between participants with complete data (n=40) and those with partial data (n=11) were conducted. No significant differences were found between the two groups ( $ps > .08$ ). Therefore, the 40 participants with complete data were used in all subsequent analyses. As shown in Table 1, the sample was predominantly Hispanic or White and in their young adulthood (mean age 23 years old). Half of participants were female. Couples were in the current romantic relationships for an average of two years.

#### *Testing Reliability of Measures.*

*Adult Attachment.* The MAQ subscale scores were composed following the scoring guideline (Carver, 1997), resulting in unacceptable internal consistency for all the four subscales:  $\alpha$ 's = .53, .68, .69, and .61 for attachment security, avoidance, attachment anxiety merger (AAM), and attachment anxiety worry type (AAW), respectively. Seeking to improve reliability of primary predictors, an exploratory factor analyses (EFA) with principal axis factoring extraction method was conducted. Results of the EFA suggested a three-factor solution (57% total variance explained) after excluding one item ("Being close to him/her gives me a source of strength for other activities"), which failed to load reliably on any factor (factor loadings  $< .300$ ). The first factor included three items measuring preference for and comfort with closeness to other, thus labeled Attachment Security. The second factor included three items measuring discomfort with



being close to other, thus labeled Attachment Avoidance. The final factor included six items measuring anxiety as an underlying character of the interpersonal relationship. Thus, this factor was labeled as Attachment Anxiety. The three refactored subscales had good face validity and internal consistency ( $.72 < \alpha's < .74$ ). As shown in Table 2, attachment security scores had a ceiling effect, whereas attachment avoidance and attachment anxiety scores had a floor effect.

*Subjective stress and daily life stress.* As presented in Table 1, the three-item subjective stress measure across four assessments had variable reliability ( $.55 < \alpha's < .83$ ). Daily life stress, measured using the PSS, demonstrated high reliability ( $\alpha=.91$ ).

*Cortisol.* Cortisol values for all participants were graphed across each sampling time to visually inspect data for outliers and overall trends. Cortisol values for each timepoint were analyzed for mean free cortisol concentration, range of values, and normality. Normality of values—skewness and kurtosis— was assessed by timepoint. Skewness values at T1, T3, and T4, were more than double their standard errors, indicating non-normality of raw cortisol values (Brown, 1997). A  $\log_{10}$  transformation was therefore applied to establish normality (Howell, 2012). Descriptive statistics for raw and log-transformed cortisol values are presented in Table 3. Statistical analyses produced similar associations among study variables using either log-transformed or raw cortisol values. Log-transformed values were therefore used in all reported analyses. Baseline (T1) cortisol levels in morning and afternoon sessions were similar to those reported in the literature for resting baseline cortisol levels (Kudielka, Schommer, Hellhammer, & Kirschbaum, 2004).

*Lagged effect of cortisol.* The lagged effect of cortisol secretion must also be taken into account when interpreting cortisol levels at different timepoints. In our study design, T3 saliva sampling occurred approximately 20 minutes after stress onset and eight minutes after cessation (see Appendix 1). This measurement was most likely to capture stress levels during the peak of stress, considering the minimum 10-minute lag for detecting acute cortisol change in after stress onset (Foley and Kirschbaum, 2010). Therefore, T3 values were used as reflecting the cortisol level at peak of stress. Additionally, T4 cortisol values, which were sampled approximately 30 minutes after stress onset and 18 minutes after cessation, were predicted to be lower than those at T3, representing a recovery from the peak of stress. As show in Table 3, cortisol levels at T3 and T4 were indeed the highest and lowest among all four sampling occasions, respectively.

*Testing Validity of Experimental Design.*

*Stress manipulation check.* A quadratic trend of subjective stress values was expected to reflect the study design. In other words, subjective stress was expected to be the highest after engaging in the stress task (T2) and to decrease during baseline and recovery measurements. To test whether changes in stress levels reflected the experimental phases, a general linear model with four repeated Subjective Stress Measures was conducted. As expected, subjective stress through the experiment evidenced a quadratic trend with an inverted u-shape,  $F(1,39)=47.05, p < .001$ . A series of alpha-adjusted paired t-tests revealed that T2 scores were significantly higher than those at T1, T3, and T4,  $ps < .001$ .

*Cortisol across experiment.* It was expected that log-transformed cortisol values would follow the study design in a lagged fashion, and evidence a quadratic trend. Using general linear modeling with four repeated cortisol measures, a significant quadratic trend was found,  $F(1,39)=15.20$ ,  $p < .001$ . The quadratic trend for cortisol was associated with an inverted u-shaped parabola, indicating that cortisol increased from T1 to T3 and decreased from T3 to T4. A series of paired t-test revealed that T2 and T3 cortisol levels were significantly higher than those at T4, ( $p < .05$ ), and that T1 and T4 cortisol levels were not significantly different ( $p > .21$ ).

*Cortisol and subjective stress.* To determine whether participants' subjective experiences of distress correlated with timepoint-lagged cortisol levels (i.e., T1 subjective stress with T2 cortisol levels), bivariate correlations were calculated between scores on the Subject Stress Measure and cortisol values at each subsequent timepoint. Overall, subjective stress scores were not significantly associated with lagged cortisol values ( $r_s < .30$ ;  $p_s > .31$ ), so they were not included as study covariates.

*Cortisol and daily life stress.* To rule out the potential confounding effect of stress in daily life in the association between the laboratory-induced acute stress and cortisol values, the extent to which daily life stress was associated with cortisol values across study phases was tested using zero-order Pearson correlational analysis. The daily life stress score was not correlated with any cortisol values across study phases ( $r_s < .08$ ;  $p_s \geq .62$ ). Daily life stress was therefore not considered as a study covariate in subsequent analyses.

*Cortisol and time of study participation.* Next, since cortisol values have a naturally occurring diurnal pattern, the potential relationship between time of study

participation and levels of cortisol values was explored. Three-fourths of participants providing saliva (n=29) began study participation in the morning (9:40am to 12:00pm), while the remaining quarter of the sample (n=11) participated in the study after 12:00pm (12:01pm to 4:27 pm). Spearman rho correlations between time of the first saliva sampling and cortisol values at each timepoint were calculated. The time of study participation was “0” if participation in study began at or before 12pm or “1” if participation in study began after 12:00pm. As shown in Table 4, results revealed negative correlations between time of the first saliva collection and cortisol values, as expected ( $ps \leq .01$ ). Therefore, time of initial cortisol sampling was used as a covariate in subsequent analyses.

*Hypothesis Testing: Individual Differences in Stress Response*

The first series of hypotheses test individual differences in cortisol levels after stress exposure. **Hypothesis 1a** states that attachment anxiety would be positively associated with higher cortisol levels at T3, followed by attachment avoidance, and attachment security. A hierarchical regression was conducted to test this hypothesis with cortisol values sampled eight minutes after end of stress task (T3) as the indicator of stress reactivity. The outcome was thus T3 cortisol levels. Step 1 included cortisol levels at T2 to control for prior cortisol level. Step 2 included other covariates: time of sampling, participant role, group condition, and gender. Step 3 included primary predictor variables: individuals’ scores of attachment security, attachment avoidance, and attachment anxiety.

Results of regression analyses are presented in Table 5. Among covariates, prior (T2) cortisol level and participant role were significantly associated with T3 cortisol,

( $p < .05$ ). Cortisol levels at T2 were significantly positively associated with those at T3, and being randomized to listen to the stress speech was related to lower T3 cortisol levels. A marginally significant ( $p < .10$ ) association was also found between group condition and T3 cortisol, such that being randomized to the stranger condition was associated with lower T3 cortisol levels.

Adult attachment orientations were not significantly associated with T3 cortisol ( $p > .74$ ), after controlling for covariates. Beta weights of attachment scores also did not suggest that higher scores on attachment anxiety, attachment avoidance, then attachment security, in that order, predicted higher T3 cortisol levels. The results failed to support Hypothesis 1a.

**Hypothesis 1b** states that lower cortisol levels at T4 will be associated with higher attachment anxiety scores, higher attachment security scores, and lower attachment avoidance scores. A hierarchical regression was conducted to test this hypothesis with cortisol values sampled eighteen minutes after end of stress task (T4) as outcome. Step 1 included cortisol levels at T3 to control for cortisol levels prior to T4. Step 2 included other covariates: time of sampling, participant role, group condition, and gender. Step 3 included primary predictor variables: individuals' scores of attachment security, attachment avoidance, and attachment anxiety.

As presented in Table 6, regression analyses showed that cortisol levels at T3 and time of day were significant predictors of cortisol values at T4 ( $p \leq .05$ ). Cortisol levels at T3 were positively associated with T4 cortisol levels, and study participation in the afternoon predicted lower T4 cortisol levels. Attachment scores were not associated with T4 cortisol levels ( $p > .30$ ). Beta weights of attachment scores also did not suggest that

higher scores on attachment avoidance, attachment security, then attachment anxiety, in that order, predicted more elevated T4 cortisol levels. Therefore, Hypothesis 1b was not supported.

*Hypothesis Testing: Partner Effects on Individual Differences in Stress Response*

The second set of hypotheses test for the influence of individuals' study partners on their own stress response. **Hypothesis 2a** states that T3 cortisol levels would be predicted by partners' T2 cortisol levels, but only in the couple condition, when individuals are paired with their romantic partner (versus a stranger). To test this hypothesis, a hierarchical linear regression model was conducted with cortisol levels at T3 as the outcome. Step 1 included cortisol levels at T2 to control for cortisol levels prior to T3. Step 2 included other covariates: time of sampling, participant role, and gender. Step 3 included main effects of partner's cortisol levels at T2 (centered) and group condition. Step 4 included an interaction term of partner's cortisol levels at T2 (centered) x group condition.

Results of regression analyses are presented in Table 7. Two covariates were (marginally) significantly related to T3 cortisol levels. Prior cortisol level (T2) was positively associated with cortisol level at T3,  $p < .001$ . Additionally, being randomized to listen to the stress speech tended to be related to lower T3 cortisol levels,  $p < .10$ . For main effects, partner's T2 cortisol levels were not associated with the outcome variable ( $p = .14$ ), while group condition was borderline associated with own T3 cortisol values ( $p = .10$ ). The interaction term was not significant, failing to support Hypothesis 2a.

**Hypothesis 2b** states that T4 cortisol levels would be predicted by partners' T3 cortisol levels, but only in the couple condition, when individuals are paired with their

romantic partner (versus a stranger). To test this hypothesis, a hierarchical linear regression model was conducted with cortisol levels at T4 as the outcome. Step 1 included cortisol levels at T3 to control for cortisol levels prior to T4. Step 2 included other covariates: time of sampling, participant role, and gender. Step 3 included main effects of partner's cortisol levels at T3 (centered) and group condition. Step 4 included an interaction term of partner's cortisol levels at T3 (centered) x group condition.

Results of regression analyses are presented in Table 8. Two covariates were significantly related to T4 cortisol levels: cortisol levels at T3 and time of day ( $p \leq .05$ ). Cortisol levels at T3 were positively associated with T4 cortisol levels ( $p < .001$ ), while study participation in the afternoon predicted lower T4 cortisol levels ( $p < .05$ ). The main effects of partner's T3 cortisol levels and group condition were not significant ( $p > .32$ ). The interaction term was also not significant, failing to support Hypothesis 2b.

**Hypothesis 2c** states that the influence of partner's prior cortisol levels on one's cortisol levels would be evident when paired with romantic partner and this association would be more pronounced by one's higher attachment anxiety scores, followed by higher attachment security scores, and then lower attachment avoidance scores. This hypothesis was tested by two hierarchical regression models.

The first model tested this hypothesis for cortisol at T3. Step 1 included cortisol levels at T2 to control for cortisol levels prior to T3. Step 2 included other covariates: time of sampling, participant role, and gender. Step 3 included main effects of partner's cortisol levels at T2 (centered), attachment security (centered), attachment avoidance (centered), attachment anxiety (centered), and group condition. Step 4 included two-way interaction terms: partner's cortisol levels at T3 (centered) x group condition, partner's

cortisol levels at T3 (centered) x attachment security (centered), partner's cortisol levels at T3 (centered) x attachment avoidance (centered), and partner's cortisol levels at T3 (centered) x attachment anxiety (centered). Step 5 included three-way interaction terms: partner's cortisol levels at T3 (centered) x group condition x attachment security (centered), partner's cortisol levels at T3 (centered) x group condition x attachment avoidance (centered), and partner's cortisol levels at T3 (centered) x group condition x attachment anxiety (centered).

Regression analyses presented in Table 9 revealed no significant associations between primary predictors and T3 cortisol levels ( $ps > .35$ ), other than covariates that have been previously reported. Therefore, Hypothesis 2c regarding cortisol levels at T3 was not supported.

The second model tested Hypothesis 2c for cortisol at T4. Step 1 included cortisol levels at T3 to control for cortisol levels prior to T4. Step 2 included other covariates: time of sampling, participant role, and gender. Step 3 included main effects of partner's cortisol levels at T3 (centered), attachment security (centered), attachment avoidance (centered), attachment anxiety (centered), and group condition. Step 4 included two-way interaction terms: partner's cortisol levels at T4 (centered) x group condition, partner's cortisol levels at T4 (centered) x attachment security (centered), partner's cortisol levels at T4 (centered) x attachment avoidance (centered), and partner's cortisol levels at T4 (centered) x attachment anxiety (centered). Step 5 included three-way interaction terms: partner's cortisol levels at T4 (centered) x group condition x attachment security (centered), partner's cortisol levels at T4 (centered) x group condition



x attachment avoidance (centered), and partner's cortisol levels at T4 (centered) x group condition x attachment anxiety (centered).

Table 10 presents results from this analysis. Covariates associated with T4 cortisol levels from prior analyses remained significant. No other primary predictors were associated with cortisol levels at T4 ( $ps > .13$ ), so Hypothesis 2c was not supported for cortisol levels at T4.

## DISCUSSION

This study examined the extent to which individual differences in attachment orientations to one's romantic partner influence one's as well as their study partner's response to a laboratory-induced stressor. The first set of hypotheses predicted that individuals' responses to an acute stressor would be associated with their adult attachment orientations. Results revealed that attachment security, avoidance, and anxiety orientations were not significantly associated with their cortisol levels at T3 (eight minutes post-stress task) or T4 (18 minutes post-stress task). It was established that individuals' cortisol levels increased from before stress onset to stress cessation, then decreased from stress cessation to the end of resting. However, results suggest that one's attachment may not significantly influence their cortisol levels in response to acute, interpersonal stress. These results are not consistent with existing literature that had shown attachment anxiety and avoidance as being positively associated with one's cortisol levels during and after cessation of a stress task (Brooks, Robles, & Schetter, 2011; Powers et al., 2006; Quirin et al., 2008; Rifkin-Graboi, 2008). One explanation for our null findings concerns the nature of the stress task. The stress task created for this study encouraged participants to imagine a hypothetical scenario, relying heavily on use of one's imagination. In contrast, other studies reporting significant findings have used different stress paradigms to activate participants' attachment systems, including an auditory startle probe or discussion with study partner about recent romantic conflict or personal concerns (Brooks, Robles, & Schetter, 2011; Powers et al., 2006; Quirin et al., 2008). These stressors, in contrast to the one we used, do not require participants to imagine a scenario, which may require creativity, imaginativity, and cognitive ability that

detract from the potency of the stress task. Stressors involving high cognitive demand may not trigger the attachment system; tasks relying heavily on short-term memory, as ours was, have not triggered stress responses differentiated by attachment scores (Rifkin-Graboi, 2008). In a separate paradigm, Rifkin-Graboi (2008) asked participants to imagine an interpersonal scenario while measuring salivary cortisol. The authors only found a link between cortisol and “dismissing” attachment, and no other attachment aspects, when asking participants to imagine being abandoned by their parents. It may be the case, then, that if asking participants to imagine a scenario, abandonment imagery needs to be used in order to trigger activation of one’s attachment system. While our scenario included aspects of abandonment, it is possible that this theme could have been emphasized more strongly.

It may also be possible that participants were responding to other aspects of the stress task; perhaps adult attachment was not relevant in explaining why or how participants responded to the stressor. It is possible that since our sample was quite young (average age 22), attachments to their romantic partner are not as salient as in older couples. Schemas of abandonment, mutual trust, and emotional intimacy may not be as relevant to young adults as to older adults. Another explanation is that the stress stimulus we used may not have involved attachment dynamics. The stress task involved speech delivery and being in the presence of another. These qualities are known to elicit a more pronounced HPA axis response (i.e. higher cortisol levels during reactivity and recovery periods) to acute stress than in stress tasks lacking these elements (Dickerson & Kemeny, 2004). Perhaps individual variability in cortisol levels in our study is attributable to individuals’ differences in levels of performance anxiety, social anxiety,

perceptions of self-efficacy in response to stress, or other similar characteristics we did not measure.

A second set of hypotheses tested the notion that romantic partners engage in a process of coregulation, wherein one's partner influences the autonomic regulation of the other. Results failed to support this hypothesis both at T3 (eight minutes post-stress task) and T4 (18 minutes post-stress task). Our null results are inconsistent with findings that individuals look to their romantic partners for cues, and observing one's partner in distress may cause subsequent distress in the other (Gottman, Coan, Carrere, & Swanson, 1998; Larson & Almeida, 1999; Lepore, 1998; Levenson & Gottman, 1983; Mullen, Bryant, & Driskell, 1997). Our lack of findings may be partially due to the time required to measure coregulated HPA activity. Investigations of coregulation of the HPA axis thus far have measured cortisol levels over a broad stretch of time, such as weekly, once per day for three days, or four times per day for three days (Berg & Wynne-Edwards, 2002; Saxbe & Repetti, 2010; Schreiber et al., 2006). Our study design sought to measure coregulation with only two measurements: partners' cortisol levels spaced eight minutes (T2-T3) and 10 minutes (T3-T4) apart. Documentation of coregulation of HPA activity thus may require a longer measurement period with more than two measures. Suggestions for timing of these measurements are offered under Limitations and Future Directions.

It may also be the case that coregulation does not occur via the HPA axis when one is faced with an acute stressor. The HPA axis, while effective in providing short-term bursts of energy to the body, is relatively slow in comparison with the SAM axis, which can stimulate peripheral changes in heart rate and blood pressure immediately after

stress appraisal (Kimura, Isowa, Ohira, & Murashima, 2005). When an acute stressor is present, and immediate action is required of one's partner, it may be more adaptive to coregulate using this autonomic pathway. Still, both HPA and SAM axes are stimulated during a stress appraisal, so one would anticipate seeing coregulation evidenced via both sympathetic pathways.

We also sought to determine the extent to which coregulation was predicted by individuals' attachment orientation scores. Overall, our results did not yield significant findings to elucidate such associations. While attachment literature theorizes that coregulated physiology should be a manifestation of one's attachment to another, it may be the case that coregulated HPA arousal during acute stress is not best described through the adult attachment framework. Instead, coregulated arousal in acute stress may be more a function of relationship satisfaction (Helm et al., 2011; Saxbe & Repetti, 2010). Self-efficacy may also moderate coregulation, such that individuals more confident in their ability to manage stress rely less on others for regulatory cues. Personality factors such as extraversion, hostility, or agreeableness may also influence the degree to which one uses another to regulate arousal (Gross, 1998). As no study to date has related HPA axis coregulation during acute stress with one's attachment to another, this presumed relationship still requires empirical support.

### *Strengths*

This study had several strengths, including stressor type, outcome measurement, and experimental design. The HPA-axis is particularly sensitive to interpersonal stress, and our stress induction was interpersonal in two ways. It required participants to imagine a scenario involving both romantic partners, and asked them deliver or listen to a

speech in the presence of another individual. The content and social context of the stress task were both interpersonal and socio-evaluative, thus maximizing our ability to elicit a cortisol response. Measurement via cotton swabs was non-invasive, and provided the investigators with an outcome measure of stress that is both objective and implicated in broader health literature. Finally, the experiment was conducted in a laboratory setting. This method allowed us to control the setting and timing of stress induction and cessation, which enabled us to attribute changes in cortisol levels to specific predictors.

#### *Limitations and Future Directions*

This study had several limitations, which future studies should aim to address. Time as well as number of saliva measurements could be modified in future protocols. In terms of time of assessment, cortisol levels exhibit a diurnal pattern, so this variability may have influenced the magnitude of stress response we observed in individuals (Robles, Shaffer, Malarkey, & Kiecolt-Glaser, 2006). However, other literature indicates that cortisol levels in response to acute stress are comparable in the morning and afternoon (Kudielka, Schommer, Hellhammer, & Kirschbaum, 2004). As findings are mixed, it would be prudent for future studies to be consistent in time of day experimental sessions are held.

Future studies would benefit from including more measures of salivary cortisol before and after the stress task. Participants were asked to complete questionnaire for 28 minutes before the first measure of salivary cortisol was sampled. Had salivary cortisol been sampled immediately after consent was obtained, one might argue that our baseline measure may have reflected a more valid reading of their naturalistic baseline. However, experimental sessions were held at a medical facility most participants had never visited,

and which required travel. Cortisol levels in participants were thus likely elevated immediately upon consent, so baseline measurement was delayed until participants quietly completed questionnaires. The timing of our baseline was meant to capture a resting baseline, and indeed cortisol levels at T1 were very similar to those reported at baseline in the literature, accounting for time of day (Kudielka, Schommer, Hellhammer, & Kirschbaum, 2004). Still, future studies should consider obtaining multiple baseline measurements in light of this consideration.

Furthermore, added measurements of salivary cortisol after the stress task could have provided a more refined picture of participants' autonomic reactivity and recovery. Our study design limited the number of measurements for measuring participants' response to stress. Future studies would benefit from adding additional measurements, as coregulation between two individuals may require multiple measurements to reliably detect (Sbarra & Hazan, 2008). Cortisol changes during the recovery period may be gradual, and detection may be maximized by sampling every 10 to 15 minutes for up to two hours after stress task is complete (Dickerson & Kemeny, 2004; Foley & Kirschbaum, 2010; Kudielka, Schommer, Hellhammer, & Kirschbaum, 2004; Loving et al., 2009; Powers et al., 2006). Future studies measuring acute changes in cortisol should therefore include measurements 10, 20, 30, 45, 60, 90, and 120 minutes after cessation of stress task in order to ensure reactivity and recovery processes are captured, especially since full return to baseline may not occur until after 60 minutes (Dickerson & Kemeny, 2004). Another consideration is duration of stress stimulus. Future studies should consider how long participants should endure a stressor in order to minimize participant burden. As effect sizes are comparable for tasks ranging from three to 60

minutes (Dickerson & Kemeny, 2004), future studies may consider using brief stress tasks such as ours (three minutes of speech preparation plus five minutes of speech delivery).

Additional measurements of cortisol would beget the use of more powerful statistical techniques for measuring inter-partner covariations in their stress responses (Butler, 2011; Helm, Sbarra, & Ferrer, 2011). Multilevel modeling approaches with individual and dyadic levels could provide researchers with information about overall coregulation over multiple measurements, test moderators of these associations, such as both partners' adult attachment scores, and also examine individual-level factors influencing such associations, which could yield valuable insight into the way partners use one another as regulatory agents. In our study, we were interested in modeling cortisol of an individual, so only attachment scores of that individual were used. If however future studies wish to examine cortisol of both partners as outcomes, a dyadic multilevel modeling approach would be warranted.

Also, our participants were young, heterosexual, and students of the university, so our findings can only be generalized this population. Future studies may explore the degree to which our results apply to other population with romantic partners, including those of older age and sexual orientation. Our small sample size also limited our ability to detect significant effects ( $\alpha = .05$ , two tailed) among primary predictor variables and cortisol levels in each analysis of study hypotheses. Post-hoc power analyses revealed that with 40 participants and one between-subjects factor (group assignment), our power to detect effects for all hypothesis testing ranged from .05 to .11. Post-hoc power analyses also indicated that a minimum sample of 657 participants would be



required to find a significant association among attachment, coregulation, and cortisol levels at T3. Future studies should therefore aim to recruit a larger sample.

Finally, future work in this area could explore the coregulation phenomenon in individuals whose physiological health is at risk and their relatives, such as medical patients and their caregivers. Coregulation amplifying one's stress response may be adaptive in the short-term, when individuals need to respond with vigilance to their partner's distress. The real world is ripe with scenarios similar to what we asked participants imagine, where one partner is in distress and the other is called to action. A responsive, aroused partner would be beneficial in this circumstance, if subsequent behavior served to ameliorate the upset partner's distress. For instance, a responsive spouse could be helpful to a patient who becomes unresponsive, falls, or has a fever. Caregivers for the chronically ill are relied upon for many forms of support (i.e., instrumental, emotional, functional) that require vigilance to the patient's cues (Kim & Schulz, 2008). However, a hypervigilant partner may not always be beneficial. When a loved one becomes a hindrance and their attempts to help worsen the situation, they may in fact cause more distress in their loved one (Rafaeli, Cranford, Green, Shrout, & Bolger, 2008). Additionally, a partner who habitually responds to their partner's stress with heightened stress may be at risk for high levels of chronic stress, and subsequent health and emotional problems (Mancini & Blieszner, 1989; Adam & Kumari, 2009).

The coregulation phenomenon may help explain why caregivers of the chronically ill are at risk for not only emotional but also health disturbance (Kim & Schulz, 2008; Miller, Cohen, & Ritchey, 2002; Miller et al., 2008; Pinguart & Sörensen, 2007; Vitaliano, Zhang, & Scanlan, 2003). As previously described, chronic HPA activation is

found in both patients and caregivers, which may involve coregulatory processes when responding to frequent acute stressors. Compared to non-caregivers, caregivers have higher morning cortisol levels after controlling for perceived stress (Wahbeh, Kishiyama, Zajdel, & Oken, 2008). Glucocorticoid resistance in monocytes, thought to occur in response to chronically elevated levels of cortisol in circulation, has been related to poorer immune functioning in the caregiving population (Miller et al., 2008).

Additionally, the cortisol levels and physical health in patients and caregivers have been related. One recent study linked caregivers' cortisol levels to the health status of the patient (Gonzalez-Bono, De Andres-Garcia, & Moya-Albiol, 2011). The relationship between HPA activity, physical health, and dyadic processes in patients and caregivers is ripe for exploration. Future studies investigating health outcomes in both populations would thus benefit from considering potentially coregulated stress responses between these dyads.

Table 1. *Descriptives of study sample and predictor variables. Part 1.*

Variable	Mean (SD)/ N (%)	Actual Range	
<b>Demographic Variables</b>			
Age (years)	22.91 (4.04)	19 – 36	
Length of Romantic Relationship (months)	26.95 (21.36)	3 – 68	
Gender (Female)	20 (50.00%)		
<b>Ethnicity</b>			
Hispanic	19 (47.50%)		
White	13 (32.50%)		
African American/Black	6 (15.00%)		
Asian/Pacific	2 (5.00%)		
American Indian	1 (2.50%)		
<b>Psychological Stress Variables</b>			
Psychological Stress Variables	Mean (SD)/ N (%)	Scale Range	$\alpha$
Subjective Stress at T1	1.31 (.44)	1 – 5	.55
Subjective Stress at T2	2.45 (.98)	1 – 5	.83
Subjective Stress at T3	1.43 (.66)	1 – 5	.83
Subjective Stress at T4	1.38 (.55)	1 – 5	.72

Note: N=40. Group Condition (Stranger) = 0 if randomized to couple condition, 1 if randomized to stranger condition; Participant Role (Listener) = 0 if randomized to deliver speech, 1 if randomized to listen during speech delivery; Time of Day = 0 if participation in study began at or before 12pm (9:40am to 12pm), 1 for participants who began study between 12:01pm and 4:27pm. PSS=Perceived Stress Scale.

Table 2. *Descriptives of study sample and predictor variables. Part 2.*

Study Predictors	Mean (SD)/ N (%)	Scale Range	$\alpha$
Group Condition (Stranger)	18 (45.00%)		
Participant Role (Listener)	14 (35.00%)		
Time of Day (Afternoon)	11 (27.50%)		
Attachment Security	3.79 (.40)	1 – 4	.74
Attachment Avoidance	1.28 (.44)	1 – 4	.72
Attachment Anxiety	1.50 (.49)	1 – 4	.73
Daily Life Stress (PSS)	16.57 (7.41)	1 - 30	.91

Note: N=40. Group Condition (Stranger) = 0 if randomized to couple condition, 1 if randomized to stranger condition; Participant Role (Listener) = 0 if randomized to deliver speech, 1 if randomized to listen during speech delivery; Time of Day = 0 if participation in study began at or before 12pm (9:40am to 12pm), 1 for participants who began study between 12:01pm and 4:27pm. PSS=Perceived Stress Scale.

Table 3. *Raw and log-transformed cortisol values by assessment timepoint.*

	Timepoint	Mean (SD)	Actual Range	Skewness (SE)	Kurtosis (SE)
Raw	T1	7.83 (4.90)	1.28 - 21.16	.85 (.37)	.19 (.73)
Values	T2	7.81 (4.46)	1.60 - 19.26	.62 (.37)	-.20 (.73)
	T3	8.30 (5.37)	1.42 - 21.24	.93 (.37)	.29 (.73)
	T4	7.38 (5.02)	1.22 - 21.49	1.22 (.37)	1.22 (.73)
Transformed	T1	.80 (.30)	.11 - 1.33	-.68 (.35)	.45 (.69)
Values	T2	.81 (.28)	.20 - 1.28	-.33 (.35)	-.64 (.69)
	T3	.82 (.31)	.15 - 1.33	-.22 (.35)	-.46 (.69)
	T4	.77 (.31)	.09 - 1.33	-.18 (.35)	-.30 (.69)

Note: N=40. T1=Baseline; T2=At the end of stress task; T3= 8 minutes post-stress task; T4= 18 minutes post-stress task. SE=Standard Error.

Table 4. *Pearson correlation coefficients (p-value) among cortisol values and time of study participation.*

	T1 Cortisol	T2 Cortisol	T3 Cortisol	T4 Cortisol	Time of Day
T1 Cortisol	--	.958 (<.001)	.853 (<.001)	.852 (<.001)	-.513 (.001)
T2 Cortisol	--	--	.922 (<.001)	.915 (<.001)	-.455 (.003)
T3 Cortisol	--	--	--	.988 (<.001)	-.422 (.007)
T4 Cortisol	--	--	--	--	-.467 (.002)

Note: N=40. Time of Day = 0 if participation in study began at or before 12pm (9:40am to 12pm), 1 for participants who began study between 12:01pm and 4:27 pm.

T1=Baseline; T2=At the end of stress task; T3= 8 minutes post-stress task; T4= 18 minutes post-stress task.

Table 5. Hierarchical regression predicting cortisol levels at T3.

	$\beta$	$t$	$p$
<u>Step 1: Covariate</u>		$R^2 = .85$	<.001
T2 Cortisol Level	.92	14.68	<.001
<u>Step 2: Covariate</u>		$\Delta R^2 = .03$	.12 <sup>†</sup>
Time of Day	.03	.46	.65
Listener	-.15	-2.38	.02
Stranger	-.12	-.172	.09
Female	.05	.86	.40
<u>Step 3: Attachment Orientations</u>		$\Delta R^2 = <.01$	.93 <sup>†</sup>
Attachment security	.02	.25	.80
Attachment avoidance	-.02	-.20	.84
Attachment anxiety	-.03	-.33	.74

Note.  $N=40$ . T3=8 minutes post-stress task; T2=At the end of stress task. <sup>†</sup> =  $p$ -value for  $R^2$  change. Time of Day = 0 if participation in study began at or before 12pm (9:40am to 12pm), 1 for participants who began study between 12:01pm and 4:27 pm. Listener = 0 if randomized to deliver speech, 1 if randomized to listen during speech delivery. Stranger = 0 if randomized to couple condition, 1 if randomized to stranger condition. Female = 0 if male participant, 1 if female.  $\beta$  = Standardized coefficient.

Table 6. Hierarchical regression predicting cortisol levels at T4.

	$\beta$	$t$	$p$
<u>Step 1: Covariate</u>		$R^2 = .98$	<.001
Cortisol level at T3	.99	39.55	<.001
<u>Step 2: Covariate</u>		$\Delta R^2 = .01$	.07 <sup>†</sup>
Time of Day	-.06	-2.06	.05
Listener	-.04	-1.62	.11
Stranger	-.03	-.99	.33
Female	.02	.87	.39
<u>Step 3: Attachment Orientations</u>		$\Delta R^2 = <.01$	.63 <sup>†</sup>
Attachment security	-.01	-.26	.80
Attachment avoidance	-.01	-.20	.85
Attachment anxiety	.03	1.02	.32

Note.  $N=40$ . T4= 18 minutes post-stress task; T3= 8 minutes post-stress task. <sup>†</sup> =  $p$ -value for  $R^2$  change. Time of Day = 0 if participation in study began at or before 12pm (9:40am to 12pm), 1 for participants who began study between 12:01pm and 4:27 pm. Listener = 0 if randomized to deliver speech, 1 if randomized to listen during speech delivery. Stranger = 0 if randomized to couple condition, 1 if randomized to stranger condition. Female = 0 if male participant, 1 if female.  $\beta$  = Standardized coefficient.



Table 7. Hierarchical regression predicting cortisol levels at T3 as a function of partner's prior cortisol levels and group condition.

	$\beta$	$t$	$p$
<u>Step 1: Covariate</u>		$R^2 = .85$	<.001
Own cortisol level at T2	.92	14.68	<.001
<u>Step 2: Covariate</u>		$\Delta R^2 = .02$	.22 <sup>†</sup>
Time of Day	-.02	-.26	.80
Listener	-.12	-1.90	.07
Female	.06	.91	.37
<u>Step 3: Main Effects</u>		$\Delta R^2 = .02$	.08 <sup>†</sup>
Partner's cortisol level at T2 (PT2)	.09	1.52	.14
Stranger	-.12	-1.72	.10
<u>Step 4: Interaction</u>		$\Delta R^2 = <.01$	.52 <sup>†</sup>
PT2 x Stranger	-.05	-.65	.52

Note.  $N=40$ . T3=8 minutes post-stress task; T2=At the end of stress task. <sup>†</sup> =  $p$ -value for  $R^2$  change. Time of Day = 0 if participation in study began at or before 12pm (9:40am to 12pm), 1 for participants who began study between 12:01pm and 4:27 pm. Listener = 0 if randomized to deliver speech, 1 if randomized to listen during speech delivery. Stranger = 0 if randomized to couple condition, 1 if randomized to stranger condition. Female = 0 if male participant, 1 if female.  $\beta$  = Standardized coefficient.

Table 8. *Hierarchical regression predicting cortisol levels at T4 as a function of partner's prior cortisol levels and group condition.*

	$\beta$	$t$	$p$
<u>Step 1: Covariate</u>		$R^2 = .98$	<.001
Own cortisol level at T3	.99	39.55	<.001
<u>Step 2: Covariate</u>		$\Delta R^2 = .01$	.05 <sup>†</sup>
Time of Day	-.07	-2.61	.01
Listener	-.03	-1.40	.17
Female	.02	.93	.36
<u>Step 3: Main Effects</u>		$\Delta R^2 = <.01$	.46 <sup>†</sup>
Partner's cortisol level at T3 (PT3)	.02	.80	.43
Stranger	-.03	-1.01	.32
<u>Step 4: Interaction</u>		$\Delta R^2 = <.01$	.34 <sup>†</sup>
PT3 x Stranger	-.03	-.97	.34

Note.  $N=40$ . T4= 18 minutes post-stress task; T3= 8 minutes post-stress task. <sup>†</sup> =  $p$ -value for  $R^2$  change. Time of Day = 0 if participation in study began at or before 12pm (9:40am to 12pm), 1 for participants who began study between 12:01pm and 4:27 pm. Listener = 0 if randomized to deliver speech, 1 if randomized to listen during speech delivery. Stranger = 0 if randomized to couple condition, 1 if randomized to stranger condition. Female = 0 if male participant, 1 if female.  $\beta$  = Standardized coefficient.

Table 9. Hierarchical regression predicting cortisol levels at T3 as a function of individual factors, partner's prior cortisol levels, and group condition.

	$\beta$	$t$	$p$
<u>Step 1: Covariate</u>		$R^2 = .85$	<.001
Own cortisol level at T2	.92	14.68	<.001
<u>Step 2: Covariate</u>		$\Delta R^2 = .02$	.22 <sup>†</sup>
Time of Day	-.02	-.26	.80
Listener	-.12	-1.90	.07
Female	.06	.91	.37
<u>Step 3: Main Effects</u>		$\Delta R^2 = .02$	.37 <sup>†</sup>
Partner's cortisol level at T2 (PT2)	.11	1.50	.15
Stranger	-.12	-1.65	.11
Attachment security	-.04	.47	.65
Attachment avoidance	.01	.16	.88
Attachment anxiety	-.06	-.75	.46
<u>Step 4: Two-way Interactions</u>		$\Delta R^2 = <.01$	.95 <sup>†</sup>
PT2 x Stranger	-.06	-.63	.54
PT2 x Attachment security	<.01	.03	.98
PT2 x Attachment avoidance	-.04	-.39	.70
PT2 x Attachment anxiety	.05	.49	.63
<u>Step 5: Three-way Interactions</u>		$\Delta R^2 = .01$	.44 <sup>†</sup>
PT2 x Stranger x Attachment security	.19	1.14	.27
PT2 x Stranger x Attachment avoidance	.17	1.00	.33
PT2 x Stranger x Attachment anxiety	.21	1.56	.13

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Note.  $N=40$ . T3=8 minutes post-stress task; T2=At the end of stress task. † =  $p$ -value for  $R^2$  change. Time of Day = 0 if participation in study began at or before 12pm (9:40am to 12pm), 1 for participants who began study between 12:01pm and 4:27 pm. Listener = 0 if randomized to deliver speech, 1 if randomized to listen during speech delivery. Stranger = 0 if randomized to couple condition, 1 if randomized to stranger condition. Female = 0 if male participant, 1 if female.  $\beta$  = Standardized coefficient.

Table 10. *Hierarchical regression predicting cortisol levels at T4 as a function of individual factors, partner's prior cortisol levels, and group condition.*

	$\beta$	$t$	$p$
<u>Step 1: Covariate</u>		$R^2 = .98$	<.001
Own cortisol level at T3	.99	39.55	<.001
<u>Step 2: Covariate</u>		$\Delta R^2 = .01$	.05 <sup>†</sup>
Time of Day	-.07	-2.61	.01
Listener	-.03	-1.40	.17
Female	.02	.93	.36
<u>Step 3: Main Effects</u>		$\Delta R^2 = <.01$	.60 <sup>†</sup>
Partner's cortisol level at T3 (PT3)	.03	1.01	.32
Stranger	-.04	-1.23	.23
Attachment security	-.02	-.71	.48
Attachment avoidance	.01	.16	.87
Attachment anxiety	.02	.61	.55
<u>Step 4: Two-way Interactions</u>		$\Delta R^2 = <.01$	.14 <sup>†</sup>
PT3 x Stranger	-.02	-.58	.57
PT3 x Attachment security	-.04	-.86	.40
PT3 x Attachment avoidance	.06	1.46	.16
PT3 x Attachment anxiety	.01	.23	.82
<u>Step 5: Three-way Interactions</u>		$\Delta R^2 = <.01$	.21 <sup>†</sup>
PT3x Stranger x Attachment security	.01	.32	.75
PT3 x Stranger x Attachment avoidance	-.02	-.40	.70
PT3 x Stranger x Attachment anxiety	-.07	-1.58	.13

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Note.  $N=40$ . T4= 18 minutes post-stress task; T3= 8 minutes post-stress task. † =  $p$ -value for  $R^2$  change. Time of Day = 0 if participation in study began at or before 12pm (9:40am to 12pm), 1 for participants who began study between 12:01pm and 4:27 pm. Listener = 0 if randomized to deliver speech, 1 if randomized to listen during speech delivery. Stranger = 0 if randomized to couple condition, 1 if randomized to stranger condition. Female = 0 if male participant, 1 if female.  $\beta$  = Standardized coefficient.

## REFERENCES

- Adam, E. K., & Kumari, M. (2009). Assessing salivary cortisol in large-scale, epidemiological research. *Psychoneuroendocrinology*, *34*(10), 1423-1436.
- Allen, J. P., & Miga, E. M. (2010). Attachment in adolescence: A move to the level of emotion regulation. *Journal of Social and Personal Relationships*, *27*(2), 181-190. doi: 10.1177/0265407509360898
- Antoni, M. H., Lutgendorf, S. K., Cole, S. W., Dhabhar, F. S., Sephton, S. E., McDonald, P. G., ... Sood, A. K. (2006). The influence of bio-behavioural factors on tumor biology: Pathways and mechanisms. *Nature Reviews Cancer*, *6*, 240-248.
- Berg, S.J., Wynne-Edwards, K.E. (2002). Salivary hormone concentrations in mothers and fathers becoming parents are not correlated. *Hormones and Behavior*, *42*, 424-436.
- Bower, J. E., Ganz, P. A., Dickerson, S. S., Petersen, L., Aziz, N., & Fahey, J.L. (2005). Diurnal cortisol rhythm and fatigue in breast cancer survivors. *Psychoneuroendocrinology*, *30*, 92-100.
- Bowlby, J. (1969/1982). Attachment and loss: Vol. 1. Attachment. New York, NY: Basic Books.
- Bowlby, J. (1973). Attachment and loss: Vol. 2. Separation. New York, NY: Basic Books.
- Bowlby, J. (1980). Attachment and loss: Vol. 3. Loss. New York, NY: Basic Books.
- Bowlby, J. (1988). A secure base: Parent- child attachment and healthy human development. London, England: Routledge
- Brooks, K. P., Robles, T. F., & Schetter, C. D. (2011). Adult attachment and cortisol responses to discussions with a romantic partner. *Personal Relationships*, *18*(2), 302-320.
- Brown, J. D. (1997). Statistics corner: Questions and answers about language testing statistics: Skewness and kurtosis. *Shiken I*(1), 20-23.
- Buckley, T. M., & Schatzberg, A. F. (2005). Review: On the interactions of the hypothalamic-pituitary-adrenal (HPA) axis and sleep: Normal HPA axis activity and circadian rhythm, exemplary sleep disorders. *The Journal of Clinical Endocrinology & Metabolism*, *90*(5), 3106-3114.

- Butler, E. A. (2011). Temporal interpersonal emotion systems: The "TIES" that form relationships. *Personality and Social Psychology Review*, Advance online publication. doi:10.1177/1088868311411164
- Butner, J., Diamond, L. M., & Hicks, A. M. (2007). Attachment style and two forms of affect coregulation between romantic partners. *Personal Relationships*, *14*, 431–455. doi:10.1111/j.1475-6811.2007.00164.x
- Carney, R. M., Freedland, K. E., Miller, G. E., Jaffe, A. S. (2002). Depression as a risk factor for cardiac mortality and morbidity: A review of potential mechanisms. *Journal of Psychosomatic Research*, *53*, 897–902.
- Carver, C. S. (1997). Adult attachment and personality: Converging evidence and a new measure. *Personality and Social Psychology Bulletin*, *23*, 865–883.
- Cohen, S., Kamarck, T., Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior*, *24*, 385–396.
- Cole, S. W., Hawkey, L. C., Arevalo, J. M., Sung, C. S., Rose, R. M., Cacioppo, J. T. (2007). Social regulation of gene expression: inflammation and the human transcriptional response to loneliness. *Genome Biology*, *8*(9), R189.
- Collins, N. L., Guichard, A. C., Ford, M. B., & Feeney, B. C. (2006). Responding to need in intimate relationships: Normative processes and individual differences. In M. Mikulincer & G. S. Goodman (Eds.), *Dynamics of romantic love: Attachment, caregiving, and sex* (pp. 149–189). New York: Guilford Press.
- Denson, T. F., Spanovic, M., & Miller, N. (2009). Cognitive appraisals and emotions predict cortisol and immune responses: A meta-analysis of acute laboratory social stressors and emotion inductions. *Psychological Bulletin*, *135*(6), 823-853.
- Diamond, L. M., & Fagundes, C. P. (2010). Psychobiological research on attachment. *Journal of Social and Personal Relationships*, *27*(2), 218-225. doi: 10.1177/0265407509360906
- Diamond, L. M., & Hicks, A. M. (2004). Psychobiological perspectives on attachment: Implications for health over the lifespan. In J. A. Simpson & W. S. Rholes (Eds.), *Adult attachment: New directions and emerging issues* (pp. 240-263). New York, NY: Guilford.
- Diamond, L. M., Hicks, A. M., & Otter-Henderson, K. D. (2008). 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*, *95*(2), 385-403. doi: 10.1037/0022-3514.95.2.385



- 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.
- Dykas, M. J., & Cassidy, J. (2011). Attachment and the processing of social information across the life span: Theory and evidence. *Psychological Bulletin*, *137*(1), 19-46. doi: 10.1037/a0021367
- Elenkov, I. J., & Chrousos, G. P. (2006). Stress System – organization, physiology and immunoregulation. *Neuroimmunomodulation*, *13*(5-6), 257-267.
- Feeney, B. C., & Collins, N. L. (2001). Predictors of caregiving in adult intimate relationships: An attachment theoretical perspective. *Journal of Personality and Social Psychology*, *80*, 972–994.
- Foley, P., & Kirschbaum, C. (2010). Human hypothalamus–pituitary–adrenal axis responses to acute psychosocial stress in laboratory settings. *Neuroscience & Biobehavioral Reviews*, *35*(1), 91-96. doi: 10.1016/j.neubiorev.2010.01.010
- Fries, E., Dettenborn, L., & Kirschbaum, C. (2009). The cortisol awakening response (CAR): Facts and future directions. *International Journal of Psychophysiology*, *72*(1), 67-73. doi: 10.1016/j.ijpsycho.2008.03.014
- Gonzalez-Bono, E., De Andres-Garcia, S., & Moya-Albiol, L. (2011). The cortisol awakening response in caregivers of schizophrenic offspring shows sensitivity to patient status. *Anxiety, Stress, & Coping*, *24*(1), 107-120.
- Gottman, J. M., Coan, J. A., Carrere, S., & Swanson, C. (1998). Predicting marital happiness and stability from newlywed interactions. *Journal of Marriage and the Family*, *60*, 5–22.
- Gross, J. J. (1998). The emerging field of emotion regulation: An integrative review. *Review of general psychology*, *2*(3), 271-299.
- Hall, D. L., Blyler, D., Allen, D., Mishel, M. H., Crandell, J., Germino, B. B., & Porter, L. S. (2011). Predictors and patterns of participant adherence to a cortisol collection protocol. *Psychoneuroendocrinology*, *36*, 540-546.
- Hazan, C., & Shaver, P. R. (1987). Romantic love conceptualized as an attachment process. *Journal of Personality and Social Psychology*, *52*, 511–524.
- Hazan, C., Gur-Yaish, N., & Campa, M. (2004). What Does It Mean to Be Attached? *Adult attachment: Theory, research, and clinical implications*. (pp. 55-85): New York, NY, US: Guilford Publications.

- Heim, C., Ehlert, U., & Hellhammer, D. H. (2000). The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders. *Psychoneuroendocrinology*, 25(1), 1-35. doi: 10.1016/s0306-4530(99)00035-9
- Helm, J. L., Sbarra, D., & Ferrer, E. (2011). Assessing cross-partner associations in physiological responses via coupled oscillator models. *Emotion*, Advance online publication. doi: 10.1037/a002503
- Herbert, T. B., & Cohen, S. (1993). Stress and immunity in humans: A meta-analytic review. *Psychosomatic Medicine*, 55, 364-379.
- Hermes, G. L., Delgado, B., Tretiakova, M., Cavigelli, S. A., Krausz, T., Conzen, S. D., McClintock, M. K. (2009). Social isolation dysregulates endocrine and behavioral stress while increasing malignant burden of spontaneous mammary tumors. *Proceedings of the National Academy of Sciences, USA*, 106(52), 22393-22398.
- Howell, D. C. (2012). *Statistical methods for psychology*: Wadsworth Publishing Company.
- Larson, R. W., & Almeida, D. M. (1999). Emotional transmission in the daily lives of families: A new paradigm for studying family process. *Journal of Marriage and the Family*, 61, 5–20.
- Kaplan, J., Manuck, S., Clarkson, T., Lusso, F., & Taub, D. (1982). Social status, environment, and atherosclerosis in cynomolgus monkeys. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 2(5), 359-368. doi: 10.1161/01.atv.2.5.359
- Kara, S., Yazici, K.M., Gulec, C., Unsal, I. (2000). Mixed anxiety–depressive disorder and major depressive disorder: comparison of the severity of illness and biological variables. *Psychiatry Research*, 94, 59–66.
- Kiecolt-Glaser, J. K., & Newton, T. L. (2001). Marriage and health: His and hers. *Psychological Bulletin*, 127(4), 472-503.
- Kim, Y. (2006). Gender, attachment, and relationship duration on cardiovascular reactivity to stress in a laboratory study of dating couples. *Personal Relationships*, 13(1), 103-114. doi: 10.1111/j.1475-6811.2006.00107.x
- Kim, Y., & Carver, C. S. (2007). Frequency and difficulty in caregiving among spouses of individuals with cancer: effects of adult attachment and gender. *Psycho-Oncology*, 16(8), 714-723. doi: 10.1002/pon.1110
- Kim, Y., Kashy, D., & Evans, T. (2007). Age and attachment style impact stress and depressive symptoms among caregivers: a prospective investigation. *Journal of Cancer Survivorship*, 1(1), 35-43. doi: 10.1007/s11764-007-0011-4

- Kim, Y., & Schulz, R. (2008). Family caregivers' strains comparative analysis of cancer caregiving with dementia, diabetes, and frail elderly caregiving. *Journal of Aging and Health, 20*(5), 483-503.
- Kimura, K., Isowa, T., Ohira, H., & Murashima, S. (2005). Temporal variation of acute stress responses in sympathetic nervous and immune systems. *Biological Psychology, 70*(2), 131-139.
- Kirschbaum, C., & Hellhammer, D.H. (1994). Salivary cortisol in psychoneuroendocrine research: Recent developments and applications. *Psychoneuroendocrinology, 19*, 313– 333.
- Kudielka, B. M., Buske-Kirschbaum, A., Hellhammer, D. H., & Kirschbaum, C. (2004). HPA axis responses to laboratory psychosocial stress in healthy elderly adults, younger adults, and children: impact of age and gender. *Psychoneuroendocrinology, 29*(1), 83-98. doi: 10.1016/s0306-4530(02)00146-4
- Kudielka, B. M., Schommer, N. C., Hellhammer, D. H., & Kirschbaum, C. (2004). Acute HPA axis responses, heart rate, and mood changes to psychosocial stress (TSST) in humans at different times of day. *Psychoneuroendocrinology, 29*(8), 983-992.
- Lepore, S. J. (1998). Problems and prospects for the social support-reactivity hypothesis. *Annals of Behavioral Medicine, 20*(4), 257-269.
- Levenson, R. W., & Gottman, J. M. (1983). Marital interaction: Physiological linkage and affective exchange. *Journal of Personality and Social Psychology, 45*, 587–597. doi:10.1037/0022-3514.45.3.587
- Lutgendorf, S. K., Degeest, K., Sung, C. Y., Arevalo, J. M., Penedo, F., Lucci, J., III, ... Cole, S. W. (2009). Depression, social support, and beta-adrenergic transcription control in human ovarian cancer. *Brain, Behavior, and Immunity, 23*, 176–183.
- Mancini, J. A., & Blieszner, R. (1989). Aging parents and adult children: Research themes in intergenerational relations. *Journal of Marriage and the Family, 51*(2), 275–290.
- Manning, W.H., & DuBois, P.H. (1962). Correlation methods in research on human learning. *Perceptual and Motor Skills, 15*, 287–32.
- Matthews, K. A., Gump, B. B., & Owens, J. F. (2001). Chronic stress influences cardiovascular and neuroendocrine responses during acute stress and recovery, especially in men. [Research Support, U.S. Gov't, P.H.S.]. *Health Psychology, 20*(6), 403-410.

- Maunder, R. G., & Hunter, J. J. (2001). Attachment and psychosomatic medicine: Developmental contributions to stress and disease. *Psychosomatic Medicine*, *63*, 556- 567.
- McEwen, B. S. (1998). Protective and Damaging Effects of Stress Mediators. *New England Journal of Medicine*, *338*(3), 171-179.  
doi:10.1056/NEJM199801153380307
- McEwen, B. S. and Wingfield, J. C. (2003). The concept of allostasis in biology and biomedicine. *Hormones and Behavior*, *43*(1), 2-15.
- 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). New York, NY: Academic Press.
- Mikulincer, M., & Shaver, P. (2005). Attachment theory and emotions in close relationships: Exploring the attachment-related dynamics of emotional reactions to relational events. *Personal Relationships*, *12*, 149 – 168.
- Mikulincer, M., & Shaver, P. R. (2007). *Attachment in adulthood: Structure, dynamics, and change*. New York, NY: Guilford Press.
- Miller, G. E., Chen, E., & Cole, S. W. (2009). Health psychology: Developing biologically plausible models linking the social world and physical health. *Annual Review of Psychology* *60*, 501-524.
- Miller, G. E., Chen, E., Sze, J., Marin, T. J., Doll, R. M., ... Cole, S. W. (2008). A functional genomic fingerprint of chronic stress in humans: decreased glucocorticoid and increased NF- $\kappa$ B signaling. *Biological Psychiatry*, *64*(4), 266-272.
- Miller, G. E., Cohen, S., & Ritchey, A. K. (2002). Chronic psychological stress and the regulation of pro-inflammatory cytokines: a glucocorticoid-resistance model. *Health Psychology*, *21*(6), 531-541.
- Mullen, B., Bryant, B., & Driskell, J. E. (1997). Presence of others and arousal: An integration. *Group Dynamics: Theory, research, and practice*, *1*(1), 52-64.
- Neu, M., Laudenslager, M. L., & Robinson, J. (2009). Coregulation in Salivary Cortisol During Maternal Holding of Premature Infants. *Biological Research For Nursing*, *10*(3), 226-240. doi: 10.1177/1099800408327789

- Peralta-Ramirez, M. I., Jimenez-Alonso, J., Godoy-Garcia, J. F., Perez-Garcia, M. (2004). The effects of daily stress and stressful life events on the clinical symptomatology of patients with lupus erythematosus. *Psychosomatic Medicine*, 66, 788–794.
- Pinquart, M., & Sörensen, S. (2007). Correlates of physical health of informal caregivers: A meta-analysis. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, 62(2), P126-P137.
- Porter, L. S., Keefe, F. J., Davis, D., Rumble, M., Scipio, C., & Garst, J. (2012). Attachment styles in patients with lung cancer and their spouses: associations with patient and spouse adjustment. *Supportive Care in Cancer*, 1-8.
- Porter, L. S., Mishel, M., Neelon, V., Belyea, M., Pisano, E., & Soo, M. S. (2003). Cortisol levels and responses to mammography screening in breast cancer survivors: A pilot study. *Psychosomatic Medicine*, 65(5), 842-848. doi: 10.1097/01.psy.0000088595.91705.c5
- Posener, J.A., Schildkraut, J.J., Samson, J.A., Schatzberg, A.F. (1996). Diurnal variation of plasma cortisol and homovanillic acid in healthy subjects. *Psychoneuroendocrinology*, 21, 33-38.
- Quirin, M., Pruessner, J.C., Kuhl, J., 2008. HPA system regulation and adult attachment anxiety: individual differences in reactive and awakening cortisol. *Psychoneuroendocrinology*, 33, 581-590.
- Rafaeli, E., Cranford, J. A., Green, A. S., Shrout, P. E., & Bolger, N. (2008). The good and bad of relationships: How social hindrance and social support affect relationship feelings in daily life. *Personality and Social Psychology Bulletin*, 34(12), 1703-1718.
- Rifkin-Graboi, A. (2008). Attachment status and salivary cortisol in a normal day and during simulated interpersonal stress in young men. *Stress: The International Journal on the Biology of Stress*, 11(3), 210-224. doi: 10.1080/10253890701706670
- Robles, T. F., Shaffer, V. A., Malarkey, W. B., & Kiecolt-Glaser, J. K. (2006). Positive behaviors during marital conflict: Influences on stress hormones. *Journal of Social and Personal Relationships*, 23, 305 – 325.
- Rohleder, N., Beulen, S., Chen, E., Wolf, J. M., & Kirschbaum, C. (2007). Stress on the dance floor: The cortisol response to social-evaluative threat in competitive ballroom dancers. *Personality and Social Psychology Bulletin*, 33, 69–84.

- Sapolsky, R. M. (1993). The physiology of dominance in stable versus unstable social hierarchies *Primate social conflict*. (pp. 171-204): Albany, NY, US: State University of New York Press.
- Sapolsky, R. M., Romero, L. M., & Munck, A. U. (2000). How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. *Endocrine Reviews*, *21*(1), 55-89.
- Saxbe, D., & Repetti, R. L. (2010). For better or worse? Coregulation of couples' cortisol levels and mood states. *Journal of Personality and Social Psychology*, *98*(1), 92-103.
- Sbarra, D. A., & Hazan, C. (2008). Coregulation, dysregulation, self-regulation: An integrative analysis and empirical agenda for understanding adult attachment, separation, loss, and recovery. *Personality and Social Psychology Review*, *12*(2), 141-167. doi: 10.1177/1088868308315702
- Schmaling, K.B., & Sher, T.G. (2000). The psychology of couples and illness: Theory, research, & practice. Washington, DC: American Psychological Association.
- Schmidt, S., Nachtigall, C., Wuethrich-Martone, O., & Strauss, B. (2002). Attachment and coping with chronic disease. *Journal of Psychosomatic Research*, *53*(3), 763-773. doi: 10.1016/s0022-3999(02)00335-5
- Shaver, P. R., & Mikulincer, M. (2002). Attachment-related psychodynamics. *Attachment and Human Development*, *4*, 133-161.
- Smyth, J. M., Ockenfels, M. C., Gorin, A. A., Catley, D., Porter, L. S., Kirschbaum, C., et al. (1997). Individual differences in the diurnal cycle of cortisol. *Psychoneuroendocrinology*, *22*, 89-105.
- Sroufe, L. A., & Waters, E. (1977). Attachment as an organizational construct. *Child Development*, *48*, 1184 - 1119.
- Van Cauter, E., Leproult, R., & Kupfer, D. J. (1996). Effects of gender and age on the levels and circadian rhythmicity of plasma cortisol. *Journal of Clinical Endocrinology & Metabolism*, *81*(7), 2468-2473. doi: 10.1210/jc.81.7.2468
- Vitaliano, P. P., Zhang, J., & Scanlan, J. M. (2003). Is caregiving hazardous to one's physical health? A meta-analysis. *Psychological Bulletin*, *129*(6), 946.
- Wahbeh, H., Kishiyama, S.S., Zajdel, D., & Oken, B.S. (2008). Salivary cortisol awakening response in mild Alzheimer disease, caregivers, and noncaregivers. *Alzheimer Disease and Associated Disorders*, *22*, 181-183.

Appendix 1. *Timeline of experimental procedures, example beginning at 9:00am.*

<u>START</u>	<u>STOP</u>	<u>DURATION (min)</u>	<u>EVENT</u>
9:00 AM	9:02 AM	2.0	Meet and greet
9:02 AM	9:04 AM	2.0	Informed Consent
9:04 AM	9:06 AM	2.0	Mouth rinse
9:06 AM	9:34 AM	28.0	Questionnaire session
9:34 AM	9:37 AM	3.0	<b>T1 saliva collection</b>
9:37 AM	9:38 AM	1.0	Random assignment procedure for speaker-listener
9:38 AM	9:39 AM	1.0	Read the scenario to participants
9:39 AM	9:42 AM	3.0	Task Preparation Period
9:42 AM	9:47 AM	5.0	Stress Task Period
9:47 AM	9:48 AM	1.0	<b>T2 saliva collection</b>
9:48 AM	9:55 AM	7.0	Resting Period
9:55 AM	9:56 AM	1.0	<b>T3 saliva collection</b>
9:56 AM	10:06 AM	10.0	Resting Period
10:06 AM	10:07 AM	1.0	<b>T4 saliva collection</b>
10:07 AM	10:10 AM	3.0	Debriefing
10:10 AM	10:11 AM	1.0	Credit and Incentive

Appendix 2. *Stress manipulation scenario.*

“It was a birthday of a mutual friend of you (speaker). The weather was just perfect, so you decide to walk to the friend’s birthday party. Your friend lives in an off-campus house in a very quiet area where there are not many houses around. You and your partner got there around 9 p.m. The walk took about 30 minutes from the dormitory where both of you live. It was very pleasant and romantic walking with your partner. At the party, you and your partner had a great time catching up with many friends. It was 2 a.m. and you and your partner were leaving the party. You were the last people to leave.

It was about 15 minutes after you were walking back from the party, which is half way home and once again very pleasant. You see a car approaching, and suddenly you realize that the car is out of control. Leaving no time to react, the car slams into the sidewalk, missing you but hitting your partner. The driver stumbles out of the car, hardly able to walk, approaching you and your partner. You realize that the driver is under the influence of alcohol. As the driver sees your partner bleeding on the ground, the driver gets back in the car and drives away, leaving you and (*name of listener*) helpless in the middle of the road.

You reach for your phone and realize that you have left it at the party, you try to reach for your partner’s phone and you realize that it has no battery. There is no one around, and the neighborhood is not familiar to you. Your partner is bleeding from a wound and is unconscious on the pavement. You yell for help, and no one answers. Your partner is unresponsive. You begin to panic, knowing that you need to get him/her to a hospital immediately...”