November 2017

Mediators and Moderators of Childhood Family Adversity and Adult Cortisol Response: The Role of Marital Conflict Behavior

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MEDIATORS AND MODERATORS OF CHILDHOOD FAMILY ADVERSITY AND ADULT CORTISOL RESPONSE: THE ROLE OF MARITAL CONFLICT BEHAVIOR

A Dissertation Presented

by

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Submitted to the Graduate School of the
University of Massachusetts Amherst in partial fulfillment
of the requirements for the degree of

DOCTOR OF PHILOSOPHY

September 2017

Clinical Psychology

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DEDICATION

This dissertation is dedicated to the memory of Gloria and Joseph Borstelmann.

ABSTRACT

MEDIATORS AND MODERATORS OF CHILDHOOD FAMILY ADVERSITY AND ADULT CORTISOL RESPONSE: THE ROLE OF MARITAL CONFLICT BEHAVIOR

SEPTEMBER 2017

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Childhood family adversity influences behavioral and physiological response processes to acute interpersonal stress. Additionally, conflict behaviors in marriage are primary determinants of stress response and related psychological problems in adulthood. As little research has examined these two important literatures simultaneously, further work is warranted to clarify the role of marital conflict behavior in the relation between childhood family adversity and adult cortisol response to conflict. The current study examined relations between childhood family adversity, observed marital conflict behaviors, and salivary cortisol in response to acute marital conflict among 228 different-sex newlywed couples. We examined intrapersonal "actor" effects as candidate mediators of the relation between childhood adversity and cortisol response; and examined interpersonal "partner" effects as candidate moderators of the relation between childhood family adversity and cortisol response. Path analysis using Actor-Partner Interdependence Modeling demonstrated that wives' childhood family adversity was negatively associated

with wives' cortisol. Wives' negative conflict behavior (e.g., hostility and distress maintaining attributions) was negatively associated with wives' cortisol. In the context of higher levels of wives' negative conflict behavior, husbands' experiences of childhood family adversity were positively associated with husbands' cortisol in response to conflict. Results demonstrate the potential lasting impacts of childhood family adversity on later cortisol response to conflict and the important role of wives' negative conflict behaviors on *both* husbands and wives. This study adds to the developmental psychopathology and close relationships literature, and further clarifies how stressful childhood experiences and conflict behaviors in marriage "get under the skin" in the form of physiological stress response to conflict.

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CHAPTER 1

INTRODUCTION

It is becoming increasingly evident that childhood family adversity and related allostatic load (the cumulative ‘‘wear and tear’’ on the body due to long-term activation of stress-mediating physiological systems) is a major risk factor predicting adult health outcomes (e.g., McEwen, 2008; McLaughlin, 2016). While the link between more extreme cases of childhood adversity (e.g., child maltreatment; institutional rearing) has been relatively well documented as a predictor of stress-related psychological health problems in adulthood (Doom & Gunnar, 2013), research is much more sparse regarding how childhood family adversity found more commonly across a broad range of community contexts (e.g., interpersonal family conflict, household disorganization) influences later stress processes in adulthood (e.g., Shonkoff & Phillips, 2000).

Additionally, the research that has attempted to explore the relations between less extreme experiences of childhood adversity and later functioning is nearly silent on how these effects unfold in the context of arguably the most common and important relationship of adult life, marriage. As behavior patterns for managing stress and conflict in close relationships have their origins in earlier development (e.g., Roisman, 2007), and adaptive and maladaptive dyadic conflict behaviors in marriage are primary determinants of stress dysregulation and related health problems in adulthood (e.g., Kiecolt-Glaser & Newton, 2001; Robles, Slatcher, Trombello, & McGinn, 2013), further research is warranted to unravel these complex relations. A better understanding of these associations adds to the extant developmental psychopathology and close relationships literature, and further clarifies how stressful childhood experiences and conflict behaviors

in marriage “get under the skin” in the form of physiological stress response during conflict.

The present project examined the relations of family adversity experienced during childhood and adolescence (referred to here as “childhood family adversity”) on later functioning of individuals and their spouses. This project specifically examined the intrapersonal and interpersonal effects of childhood family adversity on stress responses during a laboratory-based marital conflict discussion, where the hormone cortisol was indexed as a biological marker of stress response. Spouses’ observed behaviors during the conflict were examined as hypothesized mediators (i.e., intrapersonal *actor* behaviors) and moderators (i.e., interpersonal *partner* behaviors) of the relation between childhood family adversity and adult stress response.

Salivary Cortisol as a Measure of Stress Response

Researchers have long been interested in understanding the biological mechanisms by which both acute and chronic social stress influence health outcomes (e.g., McEwen, 2008). One potential mechanism that has received widespread and persistent attention in studies of acute and chronic psychosocial stress is the hypothalamic-pituitary-adrenocortical (HPA) axis (Doom & Gunnar, 2013). This hormonal response system is present across many organisms and can be activated by a broad array of mental and physical stressors (McEwen, 1993). In humans, cortisol has received significant research attention because it is the major hormonal outcome of the HPA system, exerts regulatory influences on the system, and is a well-recognized indicator of HPA axis functioning and reactivity (Stansbury & Gunnar, 1994).

Activation of the HPA axis occurs when neurons in the periventricular nucleus of the hypothalamus secrete corticotropin-releasing hormone (Smyth, Hucklebridge, Thorn, Evans, & Clow, 2013). This molecule travels through the hypophyseal portal circulation to the anterior pituitary gland, which responds to its presence by secreting a pulse of adrenocorticotropin hormone (ACTH) (Miller, Chen, & Zhou, 2007). The ACTH signal is carried through the peripheral circulation to the adrenal glands, which synthesize and release cortisol into the tissue layer, *zona fasciculate*, and ultimately into the bloodstream (Miller et al., 2007). Glucocorticoids (cortisol and other steroid hormones) in the hippocampus, hypothalamus, and pituitary then operate via a negative feedback loop to regulate further hormone release, suppressing HPA activity and restoring basal cortisol levels. In healthy individuals, daily cortisol patterns exhibit a marked circadian rhythm characterized by peak levels of cortisol following morning awakening and declining levels thereafter, reaching lowest levels in early sleep (e.g., Edwards, Clow, Evans, & Hucklebridge, 2001). Cortisol and its pattern of homeostatic regulation is essential for life and is involved broadly in learning, memory, and emotion; in the metabolic system, and in the immune system (Hostinar & Gunnar, 2013; Miller et al., 2007; Smyth et al., 2013).

Cortisol Dysregulation and Health Risk: Adverse Childhood Experiences

Short-term activation of the HPA axis is adaptive and essential for supporting normal daily functioning in everyday life, however, long-term damage to the body may occur if the HPA axis is chronically activated (Miller et al., 2007). Furthermore, both high and low levels of cortisol are implicated in physical and psychological disorders (for reviews see, Kyrou & Tsigos, 2009; McEwen & Gianaros, 2011). Flattened cortisol circadian rhythms (due to either abnormally high or low levels) are associated with a

wide range of physical and mental health problems (e.g., Hostinar & Gunnar, 2013; Miller et al., 2007). In adults, abnormally high and abnormally low overall cortisol levels are associated with depression (Burke, Davis, Otte, & Mohr, 2005; Holsboer, 2000), while chronic fatigue is consistently associated with lower cortisol levels (e.g., Crofford et al., 2004). Importantly, dysregulation of the HPA axis, other than that attributed to a specific endocrine disorder (e.g., diabetes), is often attributed to either acute or chronic stress effects and this dysregulation may be characterized by both hyper- or hypo-secretory patterns (e.g., Miller et al., 2007).

Children who grow up in adverse, highly stressful family environments, particularly those environments defined by high levels of abuse, neglect, family conflict, and household disorganization/dysfunction (all instances of potential trauma), are at increased risk for later mental health problems and associated stress dysregulation (e.g., Dube et al., 2001; Dube, Anda, Felitti, Edwards, & Williamson, 2002; Felitti et al., 1998). One hypothesized mechanism that may explain these relations is that the developing HPA axis of children who experience consistent and immediate threats to their livelihood may be repeatedly over-activated and ultimately dysregulated (e.g., Repetti, Taylor, & Seeman, 2002). This is evidenced because the developing HPA axis is highly sensitive to environmental risks and resources and children's physiological development unfolds within the context of multiple psychosocial systems. Of these systems, the most impactful on healthy development is the family environment (e.g., Hagan, Roubinov, Purdom Marreiro, & Luecken, 2013; Hostinar & Gunnar, 2013).

The vast majority of models exploring the relation between childhood stress and later health posit that stress triggers vulnerability to health risk (e.g., psychopathology) by

increasing output of cortisol, thereby exposing bodily tissues to elevated concentrations of the hormone (e.g., Kalmakis, Meyer, Chiodo, & Leung, 2015; Taylor, Lerner, Sage, Lehman, & Seeman, 2004; Taylor, 2010). If sustained, this process is thought to lead to tissue damage and subsequent dysregulation of diverse biological systems. Additionally, high levels of chronic childhood stress may produce a physiological negative feedback loop, whereby sustained high levels cortisol down-regulates the HPA system leading to attenuated cortisol functioning. This blunted cortisol response may be a key culprit in later health risk (e.g., Heim, Ehlert, & Hellhammer, 2000). These processes potentially explain how stress could exacerbate conditions in which deficient cortisol functioning contributes to health risk, which could be the case in individuals with posttraumatic stress disorder (PTSD) (e.g., Raison & Miller, 2003). As such, current theories view cortisol deviations in both directions (e.g., too high or too low) as potentially detrimental for health. Given these findings, the current project aims to examine relations between higher levels of actors' childhood family adversity and their own cortisol response such that dysregulation during stressful experiences (e.g., conflict discussion) can take the form of either hypo-activation (lower levels of cortisol) or hyper-activation (higher levels of cortisol).

There are now a multitude of studies showing associations between adverse childhood experiences and alterations in the functioning of the HPA axis, whether predicting reactivity (Carpenter et al., 2007; Fisher, Kim, Bruce, & Pears, 2012; Goldman-Mellor, Hamer, & Steptoe, 2012; Gump et al., 2009) or basal circadian secretion of cortisol (Nicolson, 2004; Trickett, Noll, Susman, Shenk, & Putnam, 2010; van der Vegt, van der Ende, Kirschbaum, Verhulst, & Tiemeier, 2009). Researchers have

examined relations of adverse childhood experiences on cortisol functioning in youth who have experienced low socioeconomic status/poverty (e.g., Evans & Kim, 2007; Lupie, King, Meaney, & McEwen, 2001), neglect and abuse (e.g., Harkness et al., 2011; Heim et al., 2000; MacMillan et al., 2009; Oosterman et al., 2010), institutional rearing followed by adoption (e.g., Fries et al., 2008; Gunnar, Morison, Chisholm, & Schuder, 2001; Gunnar et al., 2009; van der Vegt et al., 2009), or a collection of other adverse family factors (e.g., Ellis, Essex, & Boyce, 2005; Essex, Klein, Cho, & Kalin, 2002).

As previously noted, while some studies exploring relations between childhood adversity and cortisol document hyperreactivity of the HPA axis following adversity (e.g., Fries, Shirtcliff, & Pollak, 2008; Heim et al., 2000; Kaufman et al., 1997; Oosterman, De Schipper, Fisher, Dozier, & Schuengel, 2010), others report blunted HPA axis reactivity (e.g., Gordis, Granger, Susman, & Trickett, 2006; Gunnar, Frenn, Wewerka, & Van Ryzin, 2009; Harkness, Stewart, & Wynne-Edwards, 2011; MacMillan et al., 2009). Furthermore, in spite of these many studies, evidence is still inconclusive as to the candidate moderators and mediators that further influence how stressful childhood experiences impact functioning across development (Doom & Gunnar, 2013; McLaughlin et al., 2015).

As documented above, the majority of past research on the relations between stressful childhood experiences and later outcomes has primarily, yet understandably, focused almost exclusively on high-risk clinical populations (Hostinar & Gunnar, 2013; McLaughlin et al., 2015). Although adverse family environments may include extreme cases of maltreatment, varying levels of adverse family dysfunction are present and common across families in the United States. In fact, we know much less about how

more common childhood family stressors (e.g., family conflict, harsh parenting, disorganized home environment) impact later adult HPA functioning (e.g., Doom & Gunnar, 2013). The current study, therefore, specifically examines variations of family risk in a community sample of married couples that were not specifically targeted as "high risk." This project is well situated, therefore, to further facilitate a better understanding of broad family dynamics and dysfunction.

Clarifying this aspect of the extant literature is important because childhood experiences of family conflict and household dysfunction (as opposed to court-documented child maltreatment or institutional rearing) are not only more common and potentially more relevant in community populations, but also may assert insidious and subtle effects on long-term development that are often underexplored in both health risk etiology and intervention development. By examining how childhood family adversity impacts both individuals and their partners' total cortisol response, the current project sheds light on how these relations play out individually and within close relationship dyads. Furthermore, the current research not only examined direct associations between adversity and stress response, but notably examined candidate behavioral mediators (i.e., intrapersonal *actor* behavior) and moderators (i.e., interpersonal *partner* behavior) via observed marital conflict in order to further elucidate these complex relations.

Childhood Family Relationships and Adult Close Relationships

Family conflict is one of the most common and consist behavioral manifestations of family stress (e.g., Hagan et al., 2013; Smith, Knoble, Zerr, Dishion, & Stormshak, 2014). Across development, in the process of establishing, maintaining, and ending close relationships, people develop distinct behavioral strategies with which they engage in

conflict (e.g., Repetti et al., 2002; Taylor, Lerner, Sage, Lehman, & Seeman, 2004). Increasingly, researchers recognize adult romantic relationships as an important context that has roots in the interpersonal family experiences of childhood and adolescence (e.g., Andrews, Foster, Capaldi, & Hops, 2000; Conger, Cui, Bryant, & Elder, 2000; Roisman, Collins, Sroufe, & Egeland, 2005; Roisman, 2007). Attachment theory emphasizes how childhood experiences with close family members influence both adaptive and maladaptive behaviors that unfold in the context of later close relationships (Mikulincer & Shaver, 2007). Through experiences with parents, siblings, and other close relationships in families, people develop behavioral strategies for managing stress and conflict (e.g., Kiecolt-Glaser, Gouin, & Hantsoo, 2010). These learned experiences consist of an array of thoughts, emotions, and behavioral strategies that help individuals navigate conflict and regulate distress in the face of actual or perceived threat with loved ones.

Studies consistently show that interpersonal conflict between parents and within family systems (e.g., one common aspect of childhood family adversity) impacts the adult relationships of their children (Doucet & Aseltine, 2003). Growing up in a family with poor parenting skills (e.g., Capaldi & Clark, 1998), experiencing divorce (e.g., Amato, 1996; Glenn & Kramer, 1987), or experiencing chronic parental conflict (Conger et al., 2000; Repetti et al., 2002) are all associated with adult children's maladaptive conflict behavior and related dyadic maladjustment.

Past research suggests that children who grow up in families with consistent family discord are more likely to have an interpersonal style marked by problematic behaviors, including frequent and violent arguments, hostility, jealousy, difficulty

discussing disagreements calmly, and a lack of dyadic cooperation (e.g., Amato, 1996; Doucet & Aseltine, 2003) – all attitudes and behaviors that may threaten the success and stability of marriages. Other research suggests that individuals who experience childhood family adversity are more likely to exhibit stronger negative perceptions of others (e.g., Colman & Widom, 2004) and/or are more likely to report current marital problems than individuals without adverse childhood backgrounds (e.g., Dube et al., 2005).

Of note, the current study of different-sex married couples contains individuals who *have* been able to establish and maintain close relationships. The sample does not represent individuals so troubled that pair bonding is not possible, but rather this sample represents a group that *made it* to marriage, and display a range of adaptive and maladaptive behaviors based on a diverse past experiences. Thus, this project specifically examines the associations between childhood family adversity and both positive (e.g., statements of acceptance, relationship enhancing attributions about one's partner) and negative (e.g., hostile statements or gestures) behaviors within the context of marital relationships. Understanding these relations further clarifies how childhood experiences map onto a range of marital conflict behaviors and associated stress outcomes.

A Focus on Different-Sex Marriages

For adults in western and/or primarily industrialized countries, marriage is generally considered the most common form of close relationship in adulthood. In the United States, 73% of adults report they have been married at some point during their lives (United States Census Bureau, 2010). A broad literature demonstrates that adults in healthy marriages (e.g., high marital quality, low levels of spousal conflict, etc.) have better physical and mental health than those who never marry or who have highly

conflictual marriages (e.g., Robles et al., 2013). Within marriages then, spouses are uniquely placed to impact one another's health and wellbeing: to buffer against the stresses and adversity of life, or to augment risk when maladaptive processes develop (e.g., Laws, Sayer, Pietromonaco, & Powers, 2015).

From a life course perspective, the context of marriage, and in particular, the early years of marriage, represents a critical transition in psychosocial health and development. Successes and failures in transitions to or from work, shifts in living structure, often preparations for childbirth and/or parenting, and further establishing an intimate relationship outside the family of origin may all set the stage for social and emotional functioning in the decades that follow (Huston & Caughlin, 2001; Kiecolt-Glaser, Bane, Glaser, & Malarkey, 2003). Disrupted shifts in these role domains (e.g., marital distress and conflict) may be an associated outcome of previous childhood family adversity (e.g., Summers, Forehand, Armistead, & Tannenbaum, 1998), and may represent a major link between family functioning in childhood to family functioning in adulthood (e.g., Kessler, Davis, & Kendler, 1997; Taylor, 2010).

Although often joyous, new marriage may additionally be a time of great stress and emotional health disturbance (e.g., Brock & Lawrence, 2011; Brock & Lawrence, 2014; Lavner, Karney, & Bradbury, 2014). Importantly, psychological functioning in the first years of marriage are essential factors in predicting later marital satisfaction and divorce, as well as physical and mental health outcomes for both men and women (Birditt, Brown, Orbuch, & McIlvane, 2010; Caughlin, Huston, & Houts, 2000; Huston & Caughlin, 2001; Kiecolt-Glaser et al., 2003; Whitton et al., 2007). Newlywed marriages are of particular interest because it is thought that certain dyadic processes (e.g., the

impact of conflict behaviors on health) may be more salient in certain developmental stages of marriage (e.g., Rehman, Gollan, & Mortimer, 2008). Interestingly, decreases in marital satisfaction across the early years of marriage are quite drastic for some couples; about a third of divorces occur within the first four years of marriage (Kiecolt-Glaser et al., 2003; Kurdek, 1991). Thus the first years of marriage are a particularly rich time to measure relational processes between spouses, as it is a key and potentially vulnerable transition in relationship development (e.g., Carrere, Buehlman, Gottman, Coan, & Ruckstuhl, 2000).

Dyadic Close Relationship Behavior and Cortisol Response

Higher stress within marriage has been associated with higher waking cortisol levels and a flatter decrease over the day, and this is consistent with the broad health risks related to psychosocial stress as discussed above (Robles et al., 2013). Although some studies have found cortisol dysregulation for only one spouse within different-sex dyads (e.g., Rodriguez & Margolin, 2013), numerous studies have shown links between dyadic marital functioning and HPA response in both husbands and wives (for reviews see, Kiecolt-Glaser & Newton, 2001; Robles et al., 2013). These links between stressful dyadic interactions (e.g., conflict discussion) and physiological indicators are important to consider because these physiological measurements are distinct from self-reported distress (Powers, Pietromonaco, Gunlicks, & Sayer, 2006), and constitute potential pathways through which dyadic behavioral processes might influence stress related health outcomes (e.g., Kiecolt-Glaser & Newton, 2001).

Of key relevance to the current study, dyadic behaviors in close relationships have been linked to individuals' cortisol responses in situations that provoke a relationship-

salient threat, such as the discussion of an unresolved relationship issue with a spouse (e.g., Beck, Pietromonaco, DeBuse, Powers, & Sayer, 2013). Furthermore, several studies of romantic relationships indicate that romantic partners' HPA functioning is associated with specific types of adaptive and maladaptive conflict behavior (Beck, Pietromonaco, Devito, Powers, & Boyle, 2013; Laurent, Powers, & Granger, 2013; Liu, Rovine, Klein, & Almeida, 2013). Furthermore, evidence has emerged that both an individual's behavior (i.e., actor effects) and his or her spouse's behavior (i.e., partner effects) may impact cortisol response (e.g., Kordahji, Bar-Kalifa, & Rafaeli, 2015), however, classification of these complex behavior patterns is still in its infancy (e.g., Beck et al., 2013; Liu et al., 2013). Extending the current literature, the present project examined relations between actors' and partners' conflict behaviors on stress response. Actors' own behaviors were thought to function as one candidate mechanism through which adverse childhood experiences impact adult cortisol functioning. Partners' behaviors were conceptualized as candidate moderators, in so far as partners' contextual positive behaviors buffer the relation between actors' childhood family adversity and cortisol, and partners' negative behaviors amplify the relation between actors' childhood family adversity and cortisol.

In addition to work that demonstrates the impact of both actor and partner effects, some work has shown that men and women may display distinct behavioral and cortisol profiles in the context of dyadic conflict (e.g., Brooks, Robles, & Schetter, 2011; Gunlicks-Stoessel & Powers, 2009; Laurent et al., 2013; Powers et al., 2006). One review of the literature concluded that physiological changes during spousal conflict were reliably stronger for women than for men (Kiecolt-Glaser & Newton, 2001). These physiological gender differences may therefore reflect women's greater sensitivity to

negative marital interactions and other relationship conflict events (Kiecolt-Glaser & Newton, 2001). Yet this summary of the literature is challenged by work showcasing that diffuse physiological arousal may be more problematic for husbands than wives, and represents a candidate mechanism as to why men are more likely to avoid or withdraw from conflict than women (e.g., Gottman, Coan, Carrere, & Swanson, 2013; Gottman, 1999). As specific gender differences are still mixed and relatively unclear in the reviewed literatures, the current project does not purport gender-based differences in hypotheses but acknowledges the importance of investigating such differences.

In sum, this compelling but multifaceted literature warrants further investigation to determine if and when behavioral and physiological gender differences emerge in the context of dyadic conflict, and to further elucidate potential contextual or etiological factors that may alter how these processes unfold. The current study was well positioned to take on this research challenge – investigating the interplay of childhood experiences, observed dyadic behavior, and cortisol response in a sample of different-sex newlyweds. The current project had the following aims.

Aim 1: Examine the Actor Effects of Childhood Family Adversity on Cortisol.

Hypothesis 1: There will be a negative association between actors' childhood family adversity and cortisol as measured by Area Under the Curve with respect to ground (AUCg). There will be a positive association between actors' childhood family adversity and cortisol as measured by Area Under the Curve with respect to increase (AUCi). These different directions of effect for childhood adversity on AUCg and AUCi were proposed because AUCg is thought to measure a broad non-specific cortisol response whereas AUCi measures a more acute increase or decrease from cortisol

baseline. As chronic stress (childhood family adversity) is more often associated with an attenuated cortisol response, we proposed a negative relation for AUCg; as acute stress more often engenders an increased cortisol response, we proposed a positive relation for AUCi. For further information on cortisol assessment with AUCg and AUCi see methods section below. Analyses determined whether these effects were different for husbands and wives, but no specific hypotheses were purported.

Aim 2: Examine the Partner Effects of Childhood Family Adversity on Cortisol.

Hypothesis 2: There will be a positive association between partners' childhood family adversity and actors' cortisol (AUCg and AUCi), such that higher levels of partners' childhood family adversity will be related to higher levels of actors' cortisol. Again, analyses determined whether these effects were different for husbands and wives, but no specific hypotheses were purported regarding gender effects.

Aim 3: Examine Actor Effects of Childhood Family Adversity on Marital Conflict Behavior.

Hypothesis 3: There will be a positive association between actors' childhood family adversity and negative conflict behavior (e.g., hostility), such that higher levels of childhood family adversity will be associated with higher levels of negative conflict behavior. Additionally there will be a negative association between actors' childhood family adversity and positive conflict behavior (e.g., relationship enhancing attributions), such that higher levels of actors' childhood family adversity will be associated with lower levels of positive conflict behavior. No specific hypotheses regarding gender differences were purported.

Aim 4: Examine Actor Effects of Marital Conflict Behavior on Cortisol.

Hypothesis 4: There will be a positive association between actor's negative conflict behavior and cortisol (AUCg and AUCi). Additionally, there will be a negative association between actor's positive conflict behavior and cortisol (AUCg and AUCi). No specific hypotheses regarding gender differences were purported.

Aim 5: Investigate Intrapersonal Mediators (Actor Effects) of Childhood Family Adversity on Cortisol.

Hypothesis 5: Actors' conflict behaviors will mediate and partially explain the relation between their own childhood family adversity and cortisol. That is, negative behavior will partially explain the relation between childhood family adversity and cortisol (AUCg and AUCi). Conversely, positive behavior will partially explain the relation between childhood family adversity and cortisol. No specific hypotheses regarding gender differences were purported.

Aim 6: Investigate Interpersonal Moderators (Contextual Partner Effects) of Childhood Family Adversity on Cortisol.

Hypothesis 6: Partners' behaviors during the conflict discussion will moderate the relation between actors' childhood family adversity and cortisol. Specifically, higher levels of partners' negative behavior will amplify the relation between actors' childhood family adversity and cortisol (AUCg and AUCi). Conversely, partners' positive behavior will buffer the relation between actors' higher levels of childhood adversity and cortisol, such that the strength of the positive relation between actors' childhood family adversity and cortisol will decrease in the context of partners' positive behavior. No specific hypotheses regarding gender differences were purported.

CHAPTER 2

METHODS

Participants

Data for the present study were obtained from a larger short-term longitudinal study, the Growth in Early Marriage Project. 228 couples (456 individuals) who were married for the first time, did not have children, and were living in New England (wives M_{age} = 27.70, *SD* = 4.80, 93% White; husbands M_{age} = 29.13, *SD* = 5.27, 96% White) were recruited from marriage license records to participate in a study investigating mental and physical health in early marriage. The first study visit, which is the focus of the current project, occurred within the first seven months of marriage. Couples were ineligible if either partner had an endocrine disorder (e.g., Cushing's disease, diabetes) or worked overnight shifts, which can disrupt cortisol patterns (e.g., Federenko, Nagamine, Hellhammer, Wadhwa, & Wüst, 2004; James, Cermakian, & Boivin, 2007). Of the 228 couples assessed at Time 1, three couples did not complete the study; two couples were excluded because one partner could not provide saliva and one couple decided not to participate.

Procedures

Following IRB approval, all research sessions were conducted at the University of Massachusetts Amherst during the late afternoon and early evening hours (between 4 p.m. and 7 p.m.) to control for the diurnal patterns of cortisol (e.g., Dickmeis, 2009; Liu, et al., 2013). All participants gave informed consent based on the approved IRB protocol. Study sessions lasted approximately three hours. At the start of each laboratory session, a trained research coordinator described the tasks that participants would perform and gave

both husbands and wives the opportunity to ask questions. Prior to the study, all participants knew that they would be engaging in a conversation about an unresolved disagreement they were having with their partner, and that this conversation would be digitally video-recorded.

Throughout the laboratory visit, husbands and wives individually completed questionnaires. During this time, couples sat in a stimulus-neutral room and although separated by a divider, were also asked not to speak to each other while completing questionnaires. Participants provided five saliva samples during the laboratory visit at times intended to reflect cortisol levels before, during, and after the conflict discussion task. Participants also provided another saliva sample at home on a separate day.

After completing a first battery of questionnaires and providing one saliva sample approximately 30 minutes after arriving to the laboratory, each partner independently reported on three important and unresolved areas of disagreement or conflict in their relationship. Partners then rated the intensity of each on a 7-point scale from 1 (not at all intense [i.e., calm]) to 7 (extremely intense [i.e., heated]). To select the topic for discussion, the research coordinator then chose a topic that both partners had listed and that had the highest combined intensity rating. When this was not possible, the research coordinator chose a topic that had the highest intensity rating or chose a topic at random. Next, the research coordinator provided information about the upcoming conflict discussion by reminding participants that they would discuss an important topic that they had *disagreed* about recently and had not resolved. The research coordinator also specifically stated that "we would like you to clearly understand that we are asking you to discuss a topic you disagree about that might take the form of an argument." Husbands

and wives provided another saliva sample 15 minutes after they were reminded of the upcoming discussion. Immediately following this, the research coordinator took the couple into a separate room, designed to look like a living room (furnished with a small sofa and several lamps). The room also contained three small, but visible, cameras to record the conflict discussion. The research coordinator asked couples to attempt to resolve the conflict they had been assigned over the next 15 minutes. Ten, 30, and 60 minutes after the conflict discussion ended, saliva samples were collected from each partner. At the session's end, participants were provided with snacks and returned to the mock living room to discuss positive aspects of their relationship to end with a positive interaction. After this final discussion task, the research coordinator debriefed the couples and gave each participant $50.

Measures

Perceived Childhood Family Adversity. The 13-item Risky Families Questionnaire (Taylor et al., 2004), was used to retrospectively assess perceived abuse, neglect, family conflict, and household dysfunction from ages 5 through 15. The Risky Families Questionnaire, which was adapted from the Adverse Childhood Experiences (ACE) instrument, was designed to assess the relation of childhood experiences, including childhood family adversity, to mental and physical health outcomes in adulthood (Felitti et al., 1998). The Risky Families Questionnaire is a gold standard for parsimonious retrospective assessment of adverse childhood experiences when prospective data are unavailable (e.g., Carroll et al., 2013; Cho, Bower, Kiefe, Seeman, & Irwin, 2012; Maleck & Papp, 2015). The Risky Families Questionnaire and the broader ACE measure have been reliably correlated with adverse mental and physical health

outcomes in adulthood across diverse samples (Carroll et al., 2013; Dube et al., 2001; Dube et al., 2009; Dube et al., 2005). The Risky Families Questionnaire has been additionally validated against clinical interviews of individuals' experiences during childhood conducted and coded by trained clinical interviewers (Taylor, Lehman, Kiefe, & Seeman, 2006; Taylor et al., 2004).

Participants rated aspects of their childhood family environment on a series of 5-point Likert scales ranging from 1 (not at all) to 5 (very often), with items related to *neglect* (e.g., "How often would you say you were neglected while you were growing up, that is, left on your own to fend for yourself?), *family conflict* (e.g., "How often would you say there was quarreling, arguing, or shouting between your parents?"), *abuse* (e.g., "How often did a parent or other adult in the household push, grab, shove, or slap you?"), and *household disorganization* (e.g., "Would you say the household you grew up in was chaotic and disorganized?"). Positively worded items were reverse coded (i.e., "How often did a parent or other adult in the household make you feel that you were loved, supported, and cared for?"). Total scores on the RFQ can range from 13 to 65 and are then divided by total number of answered items (i.e., 13). In the current sample Cronbach's alpha was excellent and measured at .86 (husbands $\alpha = .85$, wives $\alpha = .87$).

Marital Conflict Behavior. The fifteen-minute conflict discussion task was recorded and coded using the Rapid Marital Interaction Coding System (RMICS) (Heyman & Vivian, 2000; Weiss & Heyman, 2004). The RMICS is an event-based system designed to code observed dyadic behavior. Behavior is defined broadly to include all observable actions (i.e., affective, motoric, paralinguistic, and linguistic). The

RMICS was designed to measure frequencies of behavior and behavioral patterns between intimate partners during conflicts (Heyman, 2011).

The RMICS is the second-generation extension of the Marital Interaction Coding System (MICS) developed at the University of Oregon in the late 1960s, the oldest and most widely used couples observational system (Heyman, 2001). The RMICS behavioral coding system was developed on the basis of a factor analysis of 1,086 couple interactions coded with the MICS over a 5-year period (Heyman, Weiss, & Eddy, 1995). Based on the comprehensive factor analysis, the current version of the RMICS distilled the original 37 microbehavioral MICS codes down to 5 more general negative codes (psychological abuse, distress maintaining attribution, hostility, dysphoric affect and withdrawal), 4 positive codes (acceptance, relationship enhancing attribution, humor, and self-disclosure), 1 neutral code (constructive problem discussion/solution), and 1 other code (other; discussing something other than a personal or relationship topic) for more reliable and valid use. For more detailed information about behaviors associated with each code, refer to the Appendix.

The basic coding unit of the RMICS is the speaker turn. In RMICS terminology, the speaking individual "has the floor" until that individual completes a statement or the other speaker interrupts (Heyman, 2000; Heyman, 2011). If a speaker turn lasts longer than 30 seconds it is broken down into 30-second intervals with each one given a code. Coders assign only one of the eleven codes to each speaker turn or unit; if two or more codes are present, a theoretically derived hierarchy is applied to determine which code to use (i.e., with the more negative code assigned) (Heyman, 2000; Heyman, 2011).

The RMICS discriminates between distressed versus non-distressed dyads in numerous samples and demonstrates convergent validity with the Dyadic Adjustment Scale (Spanier, 1976). The RMICS predicts improvement and dropout in-group treatment for partner-aggression (e.g., Heyman, Brown, Feldbau-Kohn, & O'Leary, 1999; Weiss & Heyman, 2004) and future marital declines including separation/divorce (Heyman, 2001). Previously published internal consistency values for the RMICS codes are above .90 and inter-rater agreement coefficients are above 0.70 (Heyman, 2011; Weiss & Heyman, 2004).

To date, the RMICS has been used in approximately 20 separate investigations with a range of ages (primarily with married couples, but also preteen siblings, adolescent dating couples, and engaged couples), populations (e.g., general married population, couples in relationship counseling, cancer patients and their spouses, families at risk for adolescent substance abuse, war veterans), and research purposes (for full list of RMICS studies both published and on-going, see Heyman, 2011).

In the present study, following prior convention (Crowell et al., 2002; Kiecolt-Glaser et al., 2015; Testa, Crane, Quigley, Levitt, & Leonard, 2014), we created two variables based on total global positive behavior and total global negative behavior. The numerical value for each RMICS individual code is calculated as the percent of that person's total coded behaviors, meaning the code is divided by the total frequency of all codes in the recorded interaction and multiplied by 100. Global positive codes are total percent frequency of all positive behaviors, and global negatives codes are total percent frequency of all negative behaviors. For example, an RMICS negative global behavior code of 5.00 would indicate that of total coded behaviors, 5% of such behaviors observed

were negative behavior codes. Importantly, summing negative and positive global codes will not equal 100% – typically a large portion of behaviors in the RMICS are coded as "Constructive Problem Solving/Solution" which are not included in either global positive or negative codes. In past research studies, the constructive problem solving variable accounts for more than 50% of observed behaviors (e.g., Miller et al., 2013; Heyman & Vivian, 2000). As a result, negative and positive codes represent more precise instances of truly negative or positive behavior. In some past work, researchers do not include the negative code "dysphoric affect" in global negative behavior scores because it is "self" rather than "other" focused (Kiecolt-Glaser et al., 2015; Testa et al., 2014). We ran all models with dysphoric affect included and not included in the global negative behavior code and found equivalent results.

In the current study, couple conflict discussions were digital-video recorded and batch shipped to Dr. Richard Heyman's laboratory at New York University where they were coded using the RMICS by trained undergraduates. For all interactions coded with the RMICS, Cohen's kappa, a measure of inter-rater agreement, is calculated on a random subset of couples to determine inter-rater reliability. Two undergraduate coders were randomly assigned to code the same recording, and remain blind as to which recordings are used for reliability testing: 25% of interactions were coded for reliability testing. The average overall Cohen's kappa per couple for 17 previously published RMICS studies was .59 (SD = .17, n = 469), which has been considered good for a complex behavioral coding system (Heyman, 2011). Consistent with this data, Cohen's kappa per couple in the current study was measured at .54 (SD = .15).

Salivary Cortisol. To assess HPA response patterns before, during, and after the conflict discussion, salivary cortisol samples were collected across the laboratory visit. As cortisol takes between 15 and 20 minutes to enter saliva after secretion from the adrenal gland, each sample reflects participants' cortisol reactions 15 to 20 minutes prior to the actual collection (Smyth et al., 2013; Stansbury & Gunnar, 1994). In total, saliva samples were obtained five times during the laboratory session and once at home. The first saliva sample was provided approximately 30 minutes after participants arrived to the research laboratory. This sample was the first anticipatory sample because all participants were aware before the session that they would discuss an area of continued disagreement with their spouse, and they were reminded of this task when they completed the informed consent paperwork. The second anticipatory sample was provided 15 minutes after husbands and wives had received further detailed instructions about the conflict discussion task and had reported three areas of unresolved conflict in their relationship. The third sample (the conflict discussion sample) was provided 10 minutes after the conflict discussion task ended and reflected cortisol during the actual conflict discussion. The fourth sample (post-discussion sample 1) was provided 30 minutes after the discussion task; the fifth sample (post-discussion sample 2) was provided 60 minutes after the discussion task. In addition, to obtain a baseline cortisol reading outside of the laboratory setting, a home saliva sample (the home sample) was collected on a separate day. This was usually collected 1 week after the laboratory session at the same time of day that participants provided their first saliva sample in the laboratory. For the purposes of our Area Under the Curve calculations, the home sample is "set" 30 minutes prior to the first laboratory saliva sample. Although this setting is somewhat arbitrary,

conceptually this sample should reflect cortisol at the same time of the day as the first laboratory sample (regardless of the exact date on which the sample was provided.) This entire procedure gives access to six total saliva samples (5 from the laboratory visit and 1 from home).

Following parameters outlined by Salimetrics, LLC, husbands and wives were asked to "passively drool down a straw and into a small plastic vial" with their heads tilted forward until the necessary amount of saliva was collected. The vial was then sealed and immediately placed in frozen storage (-85°C) until samples were shipped on dry ice to Salimetrics for analysis. All saliva samples were divided into two vials and separately assayed for salivary cortisol with a highly sensitive enzyme immunoassay. As a result, each cortisol sample contained two values, leading to a total of 12 values for all six samples per participant. The cortisol assay used 25 μL of saliva per determination and had a lower limit of sensitivity of .003 μg/dl, a standard curve range from .012 μg/dl to 3.0 μg/dl, an average intra-assay coefficient of variation of 3.5%, and an average inter-assay coefficient of variation of 5.1%. Method accuracy determined by spike and recovery averaged 100.8%, and linearity determined by serial dilution averaged 91.7%. Values from matched serum and saliva samples show the expected strong linear relationship, $r(47) = .91$, $p < .001$. Additionally, several other procedures were followed to ensure the accuracy of cortisol assays.

Couples received instructions (both verbally and written) asking them to (1) avoid brushing their teeth, using any salivary stimulants (e.g., gum), and eating a large meal within 1 hour prior to the session; (2) avoid eating sugary or acidic foods and smoking within 30 minutes before the session; (3) refrain from consuming alcohol for 12 hours

prior to the session; and (4) not visit the dentist within 48 hours of the session. Participants were asked to call to reschedule if either they or their spouse had an elevated temperature or felt ill. At the laboratory session, it was confirmed that participants were not currently ill, and all participants took their own temperature with an ear thermometer. If either partner had an elevated temperature they were asked to return on another date. Approximately 10 minutes before couples provided their first saliva sample in the laboratory, they each drank a small bottle of water (or thoroughly rinsed their mouths with the water). This procedure was designed to minimize the potential for saliva contamination (e.g., from food, drinks, or other particles). During the laboratory session, participants were required not eat or drink anything (other than the water provided earlier in the session) until all five saliva samples were collected. Couples were provided with snacks and drinks once all saliva samples were successfully collected.

When conducting research with salivary cortisol, it is important to be aware that medications can potentially affect cortisol through different pathways (Granger, Hibel, Fortunato, & Kapelewski, 2009). To assess the potential effects of different medications on cortisol levels, participants listed all medications (prescription and nonprescription) and supplements they had taken in the 24 hours prior to the laboratory session; they were provided with a reference guide of common medications and supplements if they needed help remembering specific names. Medications were categorized by type, and dummy variables (0 = no, 1 = yes) were created for each of the following medications: hormonal birth control (for wives), corticosteroids, allergy medications, antianxiety or antidepressant medications, attention-deficit/hyperactivity disorder medications,

analgesics, proton pump inhibitors, and anti-inflammatories. This information was then used in the calculation of cortisol Area Under the Curve scores.

Cortisol Area Under the Curve Calculation. Following Pruessner and colleagues' methodological guidelines (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003), Area Under the Curve (AUC) was examined with respect to both *ground* (AUCg) and *increase* (AUCi) to evaluate cortisol output (e.g., Rodriguez & Margolin, 2013). As is suggested in literature, all statistical models were run twice, once with AUCg as the dependent variables, and once with AUCi as the dependent variables (Pruessner et al., 2003; Rodriguez & Margolin, 2013). While the two AUC measures are sometimes conflated or used interchangeably (see Khoury et al., 2015 for a review), it is important to understand their distinction and specify their use in capturing different aspects of cortisol response (Fekedulegn et al., 2007; Khoury et al., 2015; Pruessner et al., 2003).

Both AUC formulas are derived from a trapezoid formula (Pruessner et al., 2003). The AUCg formula calculates the total area under the curve of all the measurements as the area of interest. It takes into account the difference between the single measurements from each other (i.e., the change over time) and the distance of these measures from the ground, or zero (i.e., the level at which the changes over time occur). AUCi is calculated with reference to the first measured value. In contrast to AUCg, AUCi ignores the distance from zero for all measurements, thereby emphasizing the changes or "reactivity" (positive or negative) over time. Thus the primary difference in the measurements is that AUCg *includes* and AUCi *removes* the area between ground and the first measure (baseline) for all time points. As a result, AUCg may be thought of as a broader measure

of cortisol functioning and response, and may more accurately capture the initial level of cortisol as well as response to a specific event. On the other hand, AUCi is a more specific measure of cortisol reactivity assessing acute reactivity to a discrete event regardless of initial level of cortisol (Fekedulegn et al., 2007; Khoury et al., 2015). For a visual representation of the similarities and differences between AUCg and AUCi, modeled off of an example from Pruessner and colleagues (2003), see Figure 3.

Prior to calculating AUCg and AUCi, variability in cortisol scores that might be attributed to medications that husbands and wives were taking were removed (Granger et al., 2009). Only those medications that five or more individuals were taking were included in models, regressing the cortisol value from each time point on all such medications separately for husbands and wives. Then, medications were trimmed that did not exhibit significant or marginal relationships with cortisol from the models and were fit again. Finally, the intercept values (means) and the residuals obtained from fitting these models were added together. The resulting corrected cortisol scores were used to compute AUCg and AUCi. While all individuals provided multiple saliva samples that were used for AUC calculation, any individuals missing four or more saliva samples were not included in AUC calculation. For individuals who were missing three or less samples we used imputation by multiple regression to estimate missing value(s).

CHAPTER 3

DATA ANALYSIS AND RESULTS

Hypothesis testing was carried out with path analysis based on the Actor–Partner Interdependence Model (APIM; Cook & Kenny, 2005). APIM is a method of dyadic data analysis that distinguishes between actor effects (involving associations among within-subject variables) and partner effects (involving the influence of one member of a dyad on the other). APIM provides separate tests of actor and partner paths. Path effects are estimated while controlling for the other path. With this approach, the dyad is treated as the unit of analysis, and actor and partner effects are tested with the proper degrees of freedom (Ben-Naim, Hirschberger, Ein-Dor, & Mikulincer, 2013; Campbell & Kashy, 2002; Kashy & Kenny, 2000). In the current study, the direct and indirect effects of childhood family adversity and conflict behavior on cortisol (in both husbands and wives) were simultaneously estimated. As a result, all models controlled for all paths concurrently. Additionally, consistent with APIM, variables across partners were allowed to covary: husbands' and wives' childhood family adversity, husbands' and wives' conflict behaviors, and husbands' and wives' cortisol response.

Path analysis was performed with the Mplus statistical modeling software (version 7.3; Muthén & Muthén, 2012) using a maximum likelihood estimator (ML) to account for the non-normal distributions of some variables. The few cases of missing cortisol data (7 in total) were not eliminated but instead modeled under maximum likelihood estimation. Models were evaluated using standard fit indices and cut-off scores recommended by Hu & Bentler (1999). Specifically, we examined: (a) the chi-square test of model fit (which should be small and non-significant, although in large applied

datasets this is often difficult to achieve); (b) the root mean square error of approximation (RMSEA; values less than or equal to .06 are consistent with good model fit); (c) the standardized root mean squared residual (SRMR; values less than or equal to .08 are consistent with good model fit); (d) the comparative fit index (CFI; values of .90 and greater are consistent with adequate model fit with values of .95 or greater suggestive of good model fit); and (e) the Tucker–Lewis index (TLI; interpreted in the same fashion as the CFI).

The relative fit of competing models was evaluated using the chi-square difference test (corrected for the use of the ML estimator); nested models contain fewer free parameters (i.e., are more parsimonious) and if the reduction in free parameters does not result in significantly degraded model fit, then the more parsimonious model is preferred. These population-based fit indices favor model parsimony and fit. Once the most parsimonious model was selected, the direction and magnitude of all paths were examined in line with proposed hypotheses.

Descriptive Statistics

A summary of descriptive statistics and the corresponding correlation matrix of the primary study variables can be found in Table 1. Husbands' and wives' self-reported childhood family adversity were positively associated. Husbands' and wives' conflict behaviors were positively and negatively associated as anticipated. Specifically, husbands' positive behavior was positively associated with wives' positive behavior, husbands' negative behavior was positively associated with wives' negative behavior, husbands' positive behavior was negatively associated with wives' negative behavior, and husbands' negative behavior was negatively associated with wives' positive

behavior. Additionally, husbands' and wives' cortisol patterns were positively correlated when calculated with AUCg but husbands' and wives' AUCi were not correlated. With regards to relations across cortisol calculations, husbands' AUCg was positively correlated with husbands' AUCi and wives' AUCg was positively correlated with wives' AUCi. Descriptive statistics and correlation matrices of individual RMICS variables (i.e., individual behavior codes used to create global behavior scores) can be found in Tables 2 and 3. Means and standard deviations of individual cortisol samples assessed at each time point can be found in Table 4.

Frequency and distributions of study variables were consistent with past research using these constructs, suggesting variables were well sampled. Descriptive statistics of the Risky Families Questionnaire total score are consistent with past research with adult community samples (e.g., Edge et al., 2009; Raposa, 2015), and the larger prevalence of positive codes to negative codes is typical and expected in research using the RMICS (e.g., Heyman, 2011). Cortisol patterns demonstrating a general cortisol decline across sampling (e.g., attenuation over time) is anticipated with afternoon sampled conflict discussion tasks (Kiecolt-Glaser et al., 2003).

Model Testing

We first examined the full saturated model with all actor, partner, moderator, and mediator effects (See Figure 1). Based on these results we then constrained all nonsignificant paths to zero to improve model fit and parsimony. As this project investigated two cortisol outcome variables, AUCg and AUCi, we conducted this full process with husbands' and wives' AUCg as the outcome variables of interest and

conducted the full process with husbands' and wives' AUCi as the outcome variables of interest.

AUCg Model 1 Results

We first ran the full saturated model with AUCg as the outcome variable of interest for husbands and wives – this model included all actor, partner, moderator, and mediator paths with no constraints (See Table 5, Model 1). All proposed direct and indirect effects were estimated in this model. The fit indices of this model (RMSEA, SRMR, CFI, and TLI) demonsrtated an adequate to good fit to the data.

AUCg Model 2 Results

In pursuit of the most parsimonious model, and to further probe the trend relation between wives' negative behavior and wives' cortisol response to conflict, we compared Model 1 to Model 2, a model where trending paths to and from wives' negative conflict behavior were freely estimated. Model 2 also continued to include freely estimated actor and partner effects (a requirement for using the Actor-Partner Interdependence Method), but all other nonsignificant paths were constrained to zero. Because of non-significant results, equivalency constraints across parallel paths between husbands and wives were not indicated in model testing. Model 2 was compared to Model 1 where all paths were freely estimated (See Table 5, Model 2). Constraints imposed in Model 2 did not significantly deteriorate model fit; therefore, the more parsimonious model was retained.

AUCg Model 3 Results

In an attempt to continue to improve model fit we adjusted Model 2 by constraining the remaining nonsignificant trend effect (p =.09) of wives' childhood family adversity on wives' negative conflict behavior to zero (See Table 5, Model 3).

Model 3 also included freely estimated actor and partner effects and all other nonsignificant paths were constrained to zero. Constraints imposed in Model 3 did not significantly deteriorate model fit; therefore this more parsimonious model was retained. In Model 3, a direct actor effect demonstrated wives' negative behavior was significantly negatively related to wives' cortisol response to conflict (β = -.14, p = .04). A direct partner effect demonstrated wives' childhood family adversity was negatively associated with husbands' cortisol (β = -0.14, p = .03). Finally, husbands' childhood family adversity was positively associated with husbands' cortisol when moderated by wives' negative conflict behavior (β = 0.16, p = .02). Specifically, in the context of wives' negative conflict behavior the relation between husbands' childhood family adversity and cortisol response to conflict was amplifed. See Figure 2 for a visual representation and see Table 7 for path estimates of this best fitting model. Interpretation of these findings are reviewed in the discussion section.

AUCg Model 4 Results

In a final model, Model 4, we constrained the path from wives' negative conflict behavior to cortisol in response to conflict to zero (See Table 5, Model 4). Although this path was significant in Model 3, this path was measured at the trend level in Model 1. This test, therefore, provided further evidence as to whether including this path improved model fit. Significant and non-significant actor partner paths were retained in line with APIM model testing (Cook & Kenny, 2005). Model comparision tests (comparing Model 4 to Model 3) revealed that Model 4 did significantly deteriorate model fit; therefore, Model 3 was reatained as the most parsimonius model.

AUCi Model 1 Results

We first ran the full saturated model with AUCi as the outcome variable of interest for husbands and wives – this model included all actor, partner, moderator, and mediator paths with no constraints (See Table 6, Model 1). All proposed direct and indirect effects were estimated in this model. The fit indices of this model (RMSEA, SRMR, CFI, and TLI) demonsrtated a good to adequate fit to the data. It is likely that this good to adequate model fit was due to the multiple statistically significant covarying paths – no hypothesized paths reached statistical signifigance.

AUCi Model 2 Results

To further improve the fit of the model to the data, all nonsignificant paths were removed (i.e., set to zero) but we retained all significant *and* non-significant actor and patner paths (See Table 6, Model 2). As described above, this procedure is consistent with the Actor-Partner Interdepence Model as actor and partner paths must be retained to accurately interpret nested dyadic data (Cook & Kenny, 2005). Constraints imposed in Model 2 did not significantly deteriorate model fit; therefore the more parsimonious model was retained. Inspite of model improvement, the results remained essentialy the same as Model 1 – no significant paths emerged.

Results of Hypothesis Testing Based on Proposed Study Aims

Aim 1: Examine the Actor Effects of Childhood Family Adversity on Cortisol.

Hypothesis 1: We hypothesized that there would be a negative association between actors' childhood family adversity and cortisol as measured by Area Under the Curve with respect to ground (AUCg). We hypothesized that there would be a positive association between actors' childhood family adversity and cortisol as measured by Area

Under the Curve with respect to increase (AUCi). We would determine whether these effects were different for husbands and wives, but no specific hypotheses were purported.

Results to Hypothesis 1: Contrary to the hypothesized relations there were no significant direct associations between actor's childhood family adversity and actor's cortisol (AUCg or AUCi) for husbands or wives.

Aim 2: Examine the Partner Effects of Childhood Family Adversity on Cortisol.

Hypothesis 2: We hypothesized that there would be a positive association between partners' childhood family adversity and actors' cortisol (AUCg and AUCi), such that higher levels of partners' childhood family adversity would be related to higher levels of actors' cortisol. We would determine whether these effects were different for husbands and wives, but no specific hypotheses were purported.

Results to Hypothesis 2: There was a partner effect of wives' childhood family adversity on husbands' cortisol. Specifically, higher levels of wives' childhood family adversity predicted husbands' lower cortisol (AUCg) in response to conflict ($\beta = -.14$, $p = .03$). This noted, the direction of the effect was in the opposite direction as hypothesized. Higher levels of wives' childhood family adversity were associated with lower levels of husbands' cortisol (AUCg). There was no significant relation between wives' childhood family adversity and husbands' cortisol (AUCi). There was no significant direct association between husbands' childhood family adversity and wives' cortisol (AUCg or AUCi).

Aim 3: Examine Actor Effects of Childhood Family Adversity on Marital Conflict Behavior.

Hypothesis 3: We hypothesized that there would be a positive association between actors' childhood family adversity and total negative conflict behavior, such that higher levels of childhood family adversity would be associated with higher levels of negative conflict behavior. Additionally, we hypothesized that there would be a negative association between actors' childhood family adversity and total positive conflict behavior, such that higher levels of actors' childhood family adversity would be associated with lower levels of positive conflict behavior. It would be determined whether these effects were different for husbands and wives, but no specific hypotheses were purported.

Results to Hypothesis 3: Contrary to the hypothesized relations there were no significant direct associations between actors' childhood family adversity and actors' positive or negative behavior for husbands or wives.

Aim 4: Examine Actor Effects of Marital Conflict Behavior on Cortisol.

Hypothesis 4: We hypothesized that there would be a positive association between actors' negative conflict behavior and cortisol (AUCg and AUCi). Additionally, we hypothesized that there would be a negative association between actors' positive conflict behavior and cortisol (AUCg and AUCi). It would be determined whether these effects were different for husbands and wives, but no specific hypotheses were purported.

Results to Hypothesis 4: An actor effect emerged for wives but not husbands – higher levels of wives' negative conflict behavior was negatively associated with wives' cortisol (AUCg) in response to conflict ($\beta = -.14$, $p = .04$). Interestingly, the direction of the effect was in the opposite direction as hypothesized. No other significant relations

between conflict behavior (positive or negative) and cortisol (AUCg or AUCi) for husbands or wives emerged.

Aim 5: Investigate Intrapersonal Mediators (Actor Effects) of Childhood Family Adversity on Cortisol.

Hypothesis 5: We hypothesized that actors' conflict behaviors would mediate and partially explain the relation between their own childhood family adversity and cortisol. That is, negative behavior would partially explain the relation between childhood family adversity and cortisol (AUCg and AUCi) and conversely, positive behavior would partially explain the relation between childhood family adversity and cortisol (AUCg and AUCi). It would be determined whether these effects were different for husbands and wives, but no specific hypotheses were purported.

Results to Hypothesis 5: Contrary to the hypothesized relations there were no significant mediational relations of actors' childhood family adversity and cortisol (AUCg or AUCi) through actors' positive or negative behavior for husbands or wives.

Aim 6: Investigate Interpersonal Moderators (Contextual Partner Effects) of Childhood Family Adversity on Cortisol.

Hypothesis 6: We hypothesized that partners' behaviors during the conflict discussion would moderate the relation between actors' childhood family adversity and cortisol. Specifically, higher levels of partners' negative behavior would amplify the relation between actors' childhood family adversity and cortisol (AUCg and AUCi). Conversely, partners' positive behavior would buffer the relation between actors' higher levels of childhood family adversity and cortisol, such that the strength of the positive relation between actors' childhood family adversity and cortisol would decrease in the

context of partners' positive behavior. It would be determined whether these effects were different for husbands and wives, but no specific hypotheses were purported.

Results to Hypothesis 6: Confirming the hypothesis, husbands' childhood family adversity predicted husbands' greater cortisol response (AUCg) to conflict when moderated by wives' negative behaviors ($\beta = .16$, $p = .02$). Specifically, the relation between husbands' childhood family adversity and cortisol (AUCg) was stronger when wives displayed highly negative behavior (e.g., output of cortisol increased). The same interaction was not significant when AUCi was used as the outcome variable. Wives' positive behavior did not act as a contextual moderator for husbands' childhood family adversity and husbands' conflict behaviors (positive or negative) did not moderate the relation between wives' childhood family adversity and cortisol (AUCg or AUCi).

In summary, we found some support for hypotheses proposed in Aim 6. Significant results also emerged in line with Aims 2 and 4, but the direction of effects were in the opposite direction as hypothesized. We did not find support for hypotheses proposed in Aims 1, 3, and 5.

CHAPTER 4

DISCUSSION

The current study examined pathways between childhood family adversity (e.g., abuse, neglect, interpersonal conflict, and household disorganization), positive and negative marital conflict behavior, and cortisol in response to conflict in a community sample of individuals and their different-sex spouses. Specifically, we examined effects of observed intrapersonal (i.e., "actor") and interpersonal (i.e., "partner") behaviors during marital conflict on relations between childhood family adversity and stress response during a laboratory-based conflict discussion task.

Results demonstrated several key findings. (1) Wives' childhood family adversity was negatively associated with husbands' cortisol: as wives' experiences of childhood family adversity increased, husbands' levels of cortisol in response to marital conflict decreased. (2) Wives' negative conflict behavior (e.g., hostility and distress maintaining attributions) was negatively associated with wives' cortisol: as wives' negative behavior increased, wives' cortisol in response to marital conflict decreased. (3) Interestingly, in the context of higher levels of wives' negative conflict behavior, husbands' experiences of childhood family adversity predicted higher levels of cortisol in response to marital conflict. Importantly, husbands' childhood family adversity was not directly associated with husbands' cortisol response. Rather, this relation only emerged in the context of wives' negative behavior.

Overall, these findings indicate that wives' childhood family adversity and negative conflict behavior were *significantly* associated with husbands' cortisol, whereas husbands' own childhood family adversity and conflict behavior were not directly related

to husbands' own cortisol in response to conflict. Only in the context of wives' negative behavior did husbands' adverse childhood experiences predict cortisol. These findings add to and advance the developmental psychopathology and close relationships literature in several ways.

While it is well established that very high levels of childhood adversity engender significant changes in cortisol functioning into childhood and adolescence (Doom & Gunnar, 2013; McLaughlin et al., 2015), the effects resulting from less extreme environments (e.g., non-clinically referred samples) and the long term relations between childhood family adversity and much later adult outcomes are less well established (e.g., Miller et al., 2007; Taylor et al., 2000). While the current study did not find significant direct relations between individuals' own experiences of childhood family adversity and later significant increases or decreases in cortisol in response to conflict, a partner effect did emerge – wives' adverse childhood experiences predicted husbands' attenuated cortisol response. This direct partner effect in the absence of a parallel actor effect is somewhat surprising, and this direct partner effect is likely accounted for by other processes that were not directly examined in the current study (e.g., other types of interpersonal behavior not measured, cumulative effects of both partners history of adversity, current depression and anxiety symptoms, relationship satisfaction etc.)

One possible explanation for this significant result is that as women grow up in adverse family environments they may develop interpersonal behavioral patterns for managing high conflict contexts that impact their later partners in complex and potentially deleterious ways. While these strategies may have been adaptive for women in certain high-stress childhood contexts, these behaviors may be less adaptive in marital

dyads, and may prompt a dysregulated cortisol response in their husbands during acute conflict. For due diligence, simple follow-up analyses were run to clarify whether wives' negative conflict behavior mediated the relation between wives' childhood family adversity and husbands' cortisol – results were nonsignificant. Further work is needed to clarify other possible explanatory pathways between wives' childhood family adversity and husbands' attenuated cortisol response.

Another possible explanation for our finding is that because individuals are more likely to select a partner who is similar to themselves (i.e. assortative mating, see Caspi & Herbener, 1990), individuals who experienced greater childhood family adversity may be partnered with individuals who also experienced greater adversity. Our sample supports this idea as husbands' and wives' childhood family adversity scores were positively correlated. In the current study, an individual's own adversity may not predict his or her cortisol outcomes, but a partner's adversity may act as a tipping point for long term effects on cortisol functioning. Further research could investigate this process.

Expanding from childhood family adversity, negative conflict behavior (e.g., hostility) appears to be one of the strongest drivers (e.g., effect size accounted for in physiological change) of physiological change during acute marital conflict (Kiecolt-Glaser & Newton, 2001). Additionally, there is ample evidence that psychological health problems that emerge in married couples with high levels of negative conflict behavior are at least partially accounted for by the consistent dysregulation of the HPA axis experienced across repeated or chronic negative dyadic conflicts (e.g., Laurent, Hertz, Nelson, & Laurent, 2016). Laboratory-based conflict discussion tasks, like the one in the

current study, are thought to represent a window into the broader, reoccurring patterns of interpersonal behavior exhibited between couples during interpersonal conflict.

Our findings demonstrating that wives' negative conflict behavior plays an important role in both wives' and husbands' cortisol functioning in response to conflict is consistent with past biopsychosocial models of marital conflict and health (e.g., Robles et al., 2013). Specifically, with regards to the often problematic role of negative behavior in marital conflict (Gottman & Levenson, 1992; Robles & Kiecolt-Glaser, 2003), some researchers have argued that *not displaying* negative conflict behaviors is in fact more important for psychophysiological health and well-being than *displaying* positive behaviors (e.g., Ewart, Taylor, Kraemer, & Agras, 1991). Simply put: in martial conflict, the presence of negative behavior is worse than the absence of positive behavior. Consistent with this idea, wives' negative behavior in the present study had a direct relation to wives' own cortisol functioning. That same negative behavior may have also provided a toxic climate elevating the cortisol response of husbands at risk for interpersonal stress dysregulation (e.g., higher levels of childhood family adversity). Our laboratory conflict task may have provided a window into the common psychophysiological responses of husbands with childhood family risk, and further demonstrated the powerful contextual role intimate partners may play in amplifying past adversity's deleterious effects.

Interestingly, why did wives' negative conflict behavior influence an increase in husbands' cortisol but a decrease in wives' cortisol? Some past research has demonstrated that women with a history of past and current depression display attenuated cortisol profiles during relationship conflict, whereas men with a history of past and

current depression display raised cortisol profiles during conflict (e.g., Powers, et al., 2016). As a result, it is possible that, like depression, negative behaviors and interpersonal stress experienced by wives more commonly leads to decreased cortisol levels, whereas negative behaviors and interpersonal stress experienced by husbands more commonly leads to increased cortisol levels. Additionally, it is also possible that wives' low cortisol is a function of greater chronic developmental stress, whereas husbands' high cortisol is a function of the acute stress brought on by the laboratory conflict task. Although husbands' and wives' rates of childhood family adversity were quite similar in the current sample, it is possible that other types of chronic stress impact wives more systematically than husbands (e.g., sexual assault, institutional sexism and patriarchy). As a result the difference in direction of effect may be partially accounted for by different experiences of chronic stress versus acute stress in men and women.

Alternatively, some researchers purport that husbands may simply be more physiologically activated than wives in response to marital conflict, and this process may partially explain why men commonly withdraw and disengage during marital conflict (Gottman, Jacobson, Rushe, & Shortt, 1995; Gottman, 1999). However, other scholars argue that women are in fact more vulnerable to close relationship conflict as evidenced by diverse physiological outcome differences and women's broader mental health risk in the context of high conflict marriages (where low cortisol is often a culprit) (e.g., Kiecolt-Glaser & Newton, 2001; Kiecolt-Glaser et al., 1996).

While our research does not clearly point to one specific psychophysiological framework over another, it does demonstrate (at least in our current sample) that wives' negative conflict behaviors may be especially impactful on cortisol for both husbands and

wives in early marriage. This finding is consistent with past work demonstrating that women in different-sex pair bonds often "take the lead" in close relationship conflict (e.g., Christensen & Heavey, 1990; Kelley et al., 1978). Specifically, women are generally more likely to initiate and promote problem discussions with romantic partners, and men are more likely to attempt to withdraw from such discussions (Powers et al., 2006). Given the social history of men's oppression of women in close relationships, in order for women to counteract gender-based inequities, women must often speak up against oppressive forces to get their needs met (Nolen-Hoeksema, 2001). As is the case in conflict between agents with different levels of power, the individual with historically less power has both more to gain and more to lose. This in turn may drive more passionate and intense interpersonal exchanges (e.g., hostility) and may help explain why wives' negative behavior predicted both wives' and husbands' cortisol. While interesting, this possible phenomenon requires further investigation.

In an attempt to place our work among more contemporary findings and synthesize existing literatures, our work is consistent with other marital conflict scholarship that proposes husbands are most negatively impacted by wives' hostile behavior, whereas wives are most negatively impacted not by hostile behavior, but from a lack of emotional closeness (e.g., Brock & Lawrence, 2014; Brock & Lawrence, 2011). As emotional closeness was not behaviorally measured in the current study, there may have been certain behaviors that husbands exhibited that were associated with wives' cortisol dysregulation that were not captured in the current study. Future work could further clarify this possible pathway.

The current research demonstrated some significant paths between childhood family adversity and marital conflict behavior on cortisol in response to conflict. Significant relations emerged when total cortisol was calculated using Area Under the Curve with Respect to ground (AUCg) but we found no significant results when cortisol was calculated with Area Under the Curve with Respect to increase (AUCi) as the dependent variable of interest. Overall, these results are not that surprising. The majority of salivary cortisol research using area under the curve techniques is able to determine significant results using AUCg, whereas examining significant results with AUCi appears more elusive (Fekedulegn et al., 2007; Hagan et al., 2013; Lamers et al., 2012; Saxbe, Negriff, Susman, & Trickett, 2014). Our study likely continues this trend because our laboratory conflict discussion task differs from standard stress paradigms such as the Trier Social Stress Task (Kirschbaum, Pirke, & Hellhammer, 1993), which generally elicits a *peak* cortisol response relative to baseline among most participants. In our conflict discussion task, individuals vary widely in the extent to which they show decreased or increased cortisol in response to anticipating the conflict, the conflict itself, and after the conflict. AUCi likely does not fully capture cortisol response in our laboratory paradigm because it "presumes" that there is a peak reactivity point relative to baseline, and that increases in cortisol over time reflect stress in response to the task. As there is no clear average pattern of cortisol responding in our paradigm, and just as both higher and lower cortisol responses may index stress dysregulation (e.g., Miller et al., 2007), we anticipate that both increases and decreases at points along the cortisol trajectory in our paradigm may be associated with maladaptive psychological outcomes.

As such, AUCg is likely the best measure of cortisol functioning in the context of our laboratory-based conflict discussion task.

Limitations of this work suggest several directions for future research. Although our measure of childhood family adversity was designed to be used retrospectively and possesses high reliability with related prospective assessment procedures (e.g., Taylor et al., 2004), prospective, long-term longitudinal designs in future studies that span early childhood through the early years of marriage would eliminate the possibility that participants' current situations bias their retrospective memories of childhood experiences. This study was not experimental; thus, although significant relations were found between study variables, formal causal conclusions cannot be drawn.

The current study sample was limited to married women and men, the majority of whom were White. To evaluate the generalizability of our findings to newlywed couples more broadly, it would be important to examine these questions among more diverse samples (e.g., across different race, ethnicity, and income groups, and among couples in same-sex marriages) as the construct of marriage and marital conflict may have differential effects based on race, ethnicity, and related contextual understandings of close relationships (e.g., Pietromonaco & Perry-Jenkins, 2014). Additionally, future research could determine if the relations documented in this study are further modified by the quality of marital relationships (e.g., reported relationship satisfaction).

In the present research, we specifically sought out a community sample to explore common adversity and relationship processes, and, this noted, associations between childhood family adversity, marital conflict, and cortisol may differ in clinical samples (e.g., couples presenting for couples therapy) so generalizations to clinical populations

should be tempered. The current data are drawn from a single study visit during the first 7-months of marriage. While this provides a clear and unique window during early marriage, it would be important to further examine how these processes remain or evolve as marriages age over time. How stable are marital conflict behaviors and associated cortisol responding, and do any of these processes map onto divorce? Future longitudinal research should examine these important questions.

We found significant dyadic associations between husbands' and wives' childhood family adversity, positive conflict behavior, and negative conflict behavior. Future research could reduce our reliance on covarying pathways for good model fit and could investigate composite variables (i.e., "couple's total childhood family adversity" or "couples total negative conflict behavior") to determine if different significant pathways emerge when these variables are combined. Additionally, RMICS positive and negative conflict behavior scores were not statistically transformed. Future work could transform these variables to further account for possible skewness. The current study also examined linear relations between variables – as higher or lower levels of cortisol are the potential result of stressful experiences, suppression effects are possible, and future research could also examine cortisol as a curvilinear outcome variable. Finally, it is possible that couples at greatest risk for high marital conflict may be less likely to respond to recruitment efforts for a study on marital health and, consequently, were excluded from the community sample.

These limitations noted, our findings document unique associations between childhood family adversity, marital conflict, and cortisol in response to conflict in a sample of newly married different-sex couples. Our findings may be useful for life course

developmental psychopathology researchers by providing further evidence for the potential lasting impact of childhood family adversity on cortisol functioning during marital conflict. Additionally, the important role of wives' negative conflict behavior (e.g., hostility, distress maintaining attributions) is noted as a key construct associated with wives' attenuated cortisol in response to conflict. Furthermore, wives' negative conflict behavior is also an important interpersonal context that may be especially damaging for husbands with a history of adverse childhood experiences. For clinicians and treatment developers interested in couple and family mental health, close attention should be paid to both husbands' and wives' current conflict behavior and history of childhood family adversity when developing and implementing depression, anxiety, and transdiagnostic behavioral health interventions.

CHAPTER 5

TABLES

Table 1.

Descriptive Statistics and Correlation Matrix of Study Variables

Variables	*n*	*SD*	M	range	1	2	3	4	5	6	7	8	9	10
1. RFQ.H	225	.663	2.07	1.00 – 4.31	-									
2. RFQ.W	225	.726	2.16	1.08 – 4.23	.20**	-								
3. RMICS_Neg_H	225	1.59	1.29	0.00 – 7.69	.03	-.09	-							
4. RMICS_Neg_W	225	2.06	1.82	0.00 – 9.68	.10	.04	.64**	-						
5. RMICS_Pos_H	225	2.73	6.95	.34 – 15.00	.02	.03	-.24**	-.15*	-					
6. RMICS_Pos_W	225	2.92	6.58	1.34 – 14.84	-.12	-.02	-.20**	-.37**	.56**	-				
7. AUCg.H	218	11.70	40.96	17.09 – 97.88	-.01	-.13	-.05	-.04	.05	.04	-			
8. AUCg.W	218	10.39	37.61	18.79 – 80.60	.06	-.08	-.07	-.15*	.001	.10	.26**	-		
9. AUCi.H	218	11.65	-1.13	-40.36 – 38.56	-.09	-.008	-.03	-.04	-.06	-.001	.31**	-.08	-	
10. AUCi.W	218	11.60	-1.85	-39.03 – 47.74	.02	-.05	-.12	-.13	.03	.06	-.05	.36**	.04	-

Note. H = Husbands. W = Wives. RFQ = Risky Families Questionnaire. RMICS = Rapid Marital Interaction Coding System (Neg = negative behaviors; Pos = positive behaviors). AUC = Area Under the Curve salivary cortisol calculation based on five laboratory and one home sample. AUCg = Calculated with respect to ground. AUCi = Calculated with respect to increase.
*$p < .05$, **$p < .01$.

Table 2.

Descriptive Statistics and Correlation Matrix of Husbands' Individual RMICS Behavior Codes

Variables	*n*	*SD*	M	range	1	2	3	4	5	6	7	8	9	10	11
1. PA.H	225	.17	.01	0.00 – 2.31	-										
2. DA.H	225	3.32	1.98	0.00 – 20.69	-.009	-									
3. HO.H	225	6.32	4.41	0.00 – 33.85	.29**	.25**	-								
4. DY.H	225	.26	.04	0.00 – 2.94	-.01	.29**	-.05	-							
5. WI.H	225	.23	.02	0.00 – 3.28	-.007	.003	.08	-.01	-						
6. AC.H	225	1.77	.81	0.00 – 10.71	-.01	.002	-.13*	.19**	.08	-					
7. RA.H	225	5.55	6.38	0.00 – 29.55	-.04	.18**	-.04	-.03	-.03	.06	-				
8. SD.H	225	4.05	3.60	0.00 – 20.83	-.001	.08	-.04	.08	-.04	.12	.16*	-			
9. HM.H	225	9.60	13.01	0.00 – 50.75	-.04	-.20**	-.24**	.003	.02	.06	-.19**	-.20**	-		
10. PD.H	225	12.72	66.42	32.86 – 94.34	-.16*	-.30**	-.29**	-.12	-.12	-.20**	-.28**	-.18**	-.49**	-	
11. OT.H	225	6.60	3.30	0.00 – 42.76	.11	-.10	-.010	.01	.13	.04	-.20**	-.15*	.09	.38**	-

Note. All variables represent the percentage of specified behavior out of total RMICS behaviors. H = Husbands. PA = Psychological Abuse. DA = Distress Maintaining Attribution. HO = Hostility. Dysphoric Affect = DA. WI = Withdrawal. AC = Acceptance. RA = Relationship Enhancing Attribution. SD = Self-Disclosure. HM = Humor. PD = Constructive Problem Solving. OT = Other.

*$p < .05$, **$p < .01$., ***$p < .001$.

Table 3.

Descriptive Statistics and Correlation Matrix of Wives' Individual RMICS Behavior Codes

Variables	*n*	*SD*	M	range	1	2	3	4	5	6	7	8	9	10	11
1. PA.W	225	0.00	0.00	0.00 – 0.00	-										
2. DA.W	225	3.94	2.91	0.00 – 23.53	0.00	-									
3. HO.W	225	8.40	5.93	0.00 – 44.09	0.00	.30**	-								
4. DY.W	225	1.33	.28	0.00 – 10.58	0.00	-.04	-.06	-							
5. WI.W	225	.08	.005	0.00 – 1.20	0.00	.05	.09	-.01	-						
6. AC.W	225	2.10	.95	0.00 – 18.18	0.00	-.05	-.17**	.11	-.031	-					
7. RA.W	225	5.93	6.97	0.00 – 36.00	0.00	.15*	-.16*	.02	-.011	.14*	-				
8. SD.W	225	4.21	3.81	0.00 – 23.81	0.00	-.04	-.21**	.08	-.041	.23**	.30**	-			
9. HM.W	225	9.49	14.60	0.00 – 51.43	0.00	-.19**	-.30**	0.00	-.069	-.01	-.14*	-.20**	-		
10. PD.W	225	13.00	61.31	26.13 – 90.16	0.00	-.34**	-.28**	-.11	-.006	-.19**	-.32**	-.15*	-.42**	-	
11. OT.W	225	6.21	3.24	0.00 – 44.22	0.00	-.13	-.13	.02	.004	.04	-.20**	-.12	.17*	-.36**	-

Note. All variables represent the percentage of specified behavior out of total RMICS behaviors. W = Wives. PA = Psychological Abuse. DA = Distress Maintaining Attribution. HO = Hostility. Dysphoric Affect = DA. WI = Withdrawal. AC = Acceptance. RA = Relationship Enhancing Attribution. SD = Self-Disclosure. HM = Humor. PD = Constructive Problem Solving. OT = Other. *$p < .05$, **$p < .01$., ***$p < .001$.

Table 4.

Means and Standard Deviations of Cortisol Samples for Husbands and Wives

	Salivary Cortisol Sample Time Points (ug/dL)					
	Home Sample	Anticipatory Sample 1	Anticipatory Sample 2	Conflict Discussion	Post-Discussion Sample 1	Post-Discussion Sample 2
Husbands	.08 (.06)	.11 (.08)	.08 (.06)	.07 (.05)	.06 (.04)	.05 (.05)
Wives	.08 (.06)	.10 (.07)	.09 (.07)	.07 (.06)	.07 (.06)	.06 (.05)

Table 5.

Path Model Comparisons with Cortisol AUCg as Outcome Variables

	# of Parameters Estimated	**Minimal Fit χ^2**	**χ^2Df**	**χ^2 *p***	**RMSEA**	**SRMR**	**CFI**	**TLI**	**$\Delta\chi^2$**	**Δdf**	**$\Delta\chi^2$*p***
Model 1	40	65.39	36	.002	.06 (.04 - .08)	.06	.90	.86	--	--	--
Model 2	31	71.47	45	.007	.05 (.03 - .07)	.06	.91	.90	6.08	9	.73
Model 3	**30**	**74.27**	**46**	**.005**	**.05 (.03 - .07)**	**.06**	**.91**	**.90**	**2.79**	**1**	**.10**
Model 4	29	78.5	47	.01	.06 (.03 - .08)	.06	.90	.89	4.24	1	.04

Note.
Model 1: Full saturated model with no constrained paths.
Model 2: Includes actor and partner paths but all other nonsignificant paths were constrained to zero except trending paths to and from wives' negative conflict behavior.
Model 3: Includes actor and partner paths but all other nonsignificant paths were constrained to zero.
Model 4: Using results from Model 1, includes actor and partner paths but all other nonsignificant paths were constrained to zero.

Table 6.

Path Model Comparisons with Cortisol AUCi as Outcome Variables

	# of Parameters Estimated	**χ^2**	**χ^2Df**	**χ^2 *p***	**RMSEA**	**SRMR**	**CFI**	**TLI**	**$\Delta\chi^2$**	**Δdf**	**$\Delta\chi^2$*p***
Model 1	40	61.87	36	.005	.06 (.03 - .08)	.06	.91	.87	--	--	--
Model 2	**28**	**73.39**	**48**	**.01**	**.05 (.02 - .07)**	**.06**	**.91**	**.90**	**11.52**	**12**	**.48**

Note.
Model 1: Full saturated model with all paths (nothing constrained).
Model 2: Model includes actor and partner paths but all other nonsignificant paths are constrained to zero.

Table 7.

Standardized Path Estimates for AUCg Model 3 (Best Fitting Model)

Variable	Path		Standardized Estimate	SE
Actor Effects				
RFQ.H	→	AUCg.H	.02	.07
RFQ.H	→	RMICS_Neg_H	0.00	0.00
RFQ.H	→	RMICS_Pos_H	0.00	0.00
RMICS_Neg_H	→	AUCg.H	0.00	0.00
RMICS_Pos_H	→	AUCg.H	0.00	0.00
RFQ.W	→	AUCg.W	-.09	.07
RFQ.W	→	RMICS_Neg_W	0.00	0.00
RFQ.W	→	RMICS_Pos_W	0.00	0.00
RMICS_Neg_W	→	AUCg.W	-.14*	.07
RMICS_Pos_W	→	AUCg.W	0.00	0.00
Partner Effects				
RFQ.H	→	AUCg.W	.10	.07
RFQ.W	→	AUCg.H	-.14*	.08
Moderation Effects				
RFQ.H X RMICS_Neg_W	→	AUCg.H	.16*	.07
RFQ.H X RMICS_Pos_W	→	AUCg.H	0.00	0.00
RFQ.W X RMICS_Neg_H	→	AUCg.W	0.00	0.00
RFQ.W X RMICS_Pos_H	→	AUCg.W	0.00	0.00
Mediation Effects				
RFQ.H → RMICS_Neg_H	→	AUCg.H	0.00	0.00
RFQ.H → RMICS_Pos_H	→	AUCg.H	0.00	0.00
RFQ.W → RMICS_Neg_W	→	AUCg.W	0.00	0.00
RFQ.W → RMICS_Pos_W	→	AUCg.W	0.00	0.00
Covarying Paths/Residual Correlations				
RFQ.H	WITH	RFQ.W	.10**	.003
RMICS_Neg_H	WITH	RMICS_Pos_H	-1.02*	.30
RMICS_Neg_W	WITH	RMICS_Pos_W	-2.30***	.43
RMICS_Neg_H	WITH	RMICS_Pos_W	-.90*	.32
RMICS_Neg_W	WITH	RMICS_Pos_H	-.81*	.38
RMICS_Neg_H	WITH	RMICS_Neg_W	2.10***	.26
RMICS_Pos_W	WITH	RMICS_Pos_H	4.50***	.61
AUCg.H	WITH	AUCg.W	28.20**	8.14

Note. H = Husbands. W = Wives. RFQ = Risky Families Questionnaire. RMICS = Rapid Marital Interaction Coding System (Neg = negative/maladaptive behaviors; Pos = positive/adaptive behaviors). AUC = Area Under the Curve salivary cortisol calculation based on 5 laboratory and one home sample. AUCg = Calculated with respect to ground. AUCi = Calculated with respect to increase. *p <.05, **p <.01, ***p <.001

CHAPTER 6

FIGURES

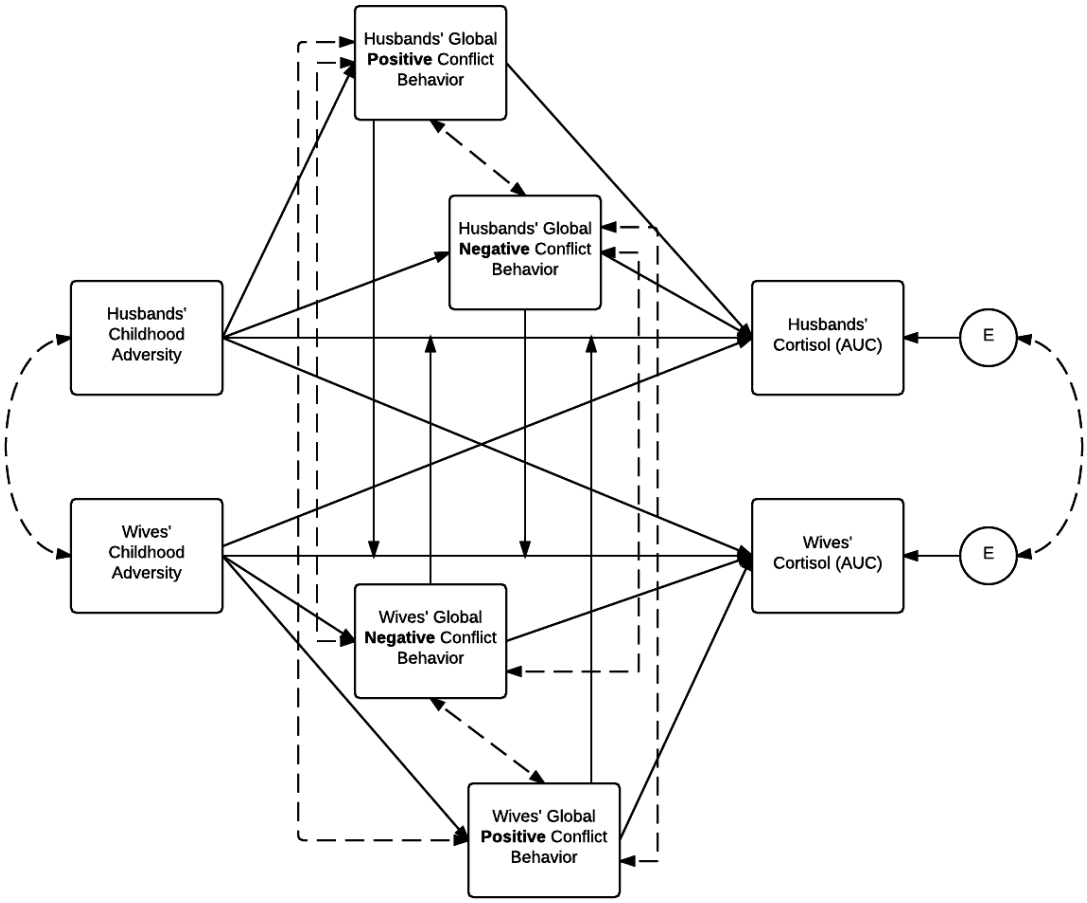


Figure 1. Proposed Full Saturated Model with All Paths Estimated

Note. Black lines = Estimated paths. Dashed black lines = Covarying paths.

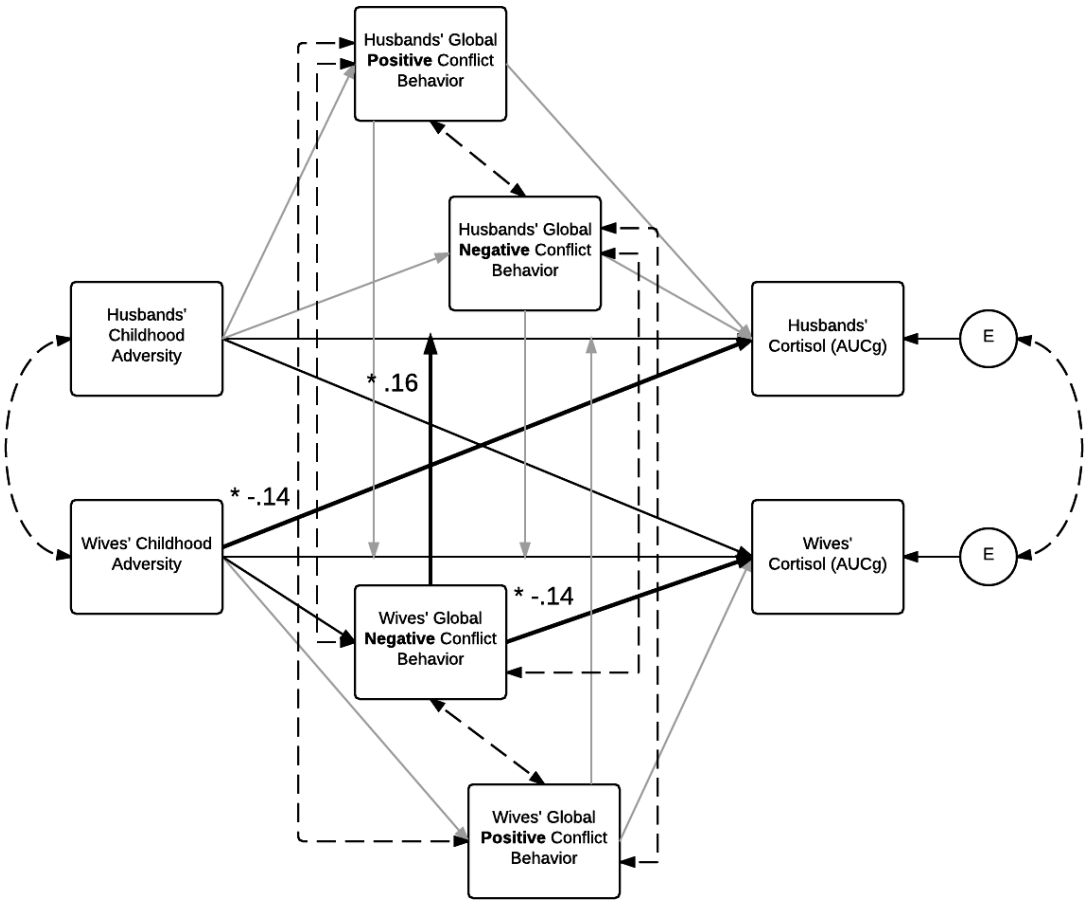
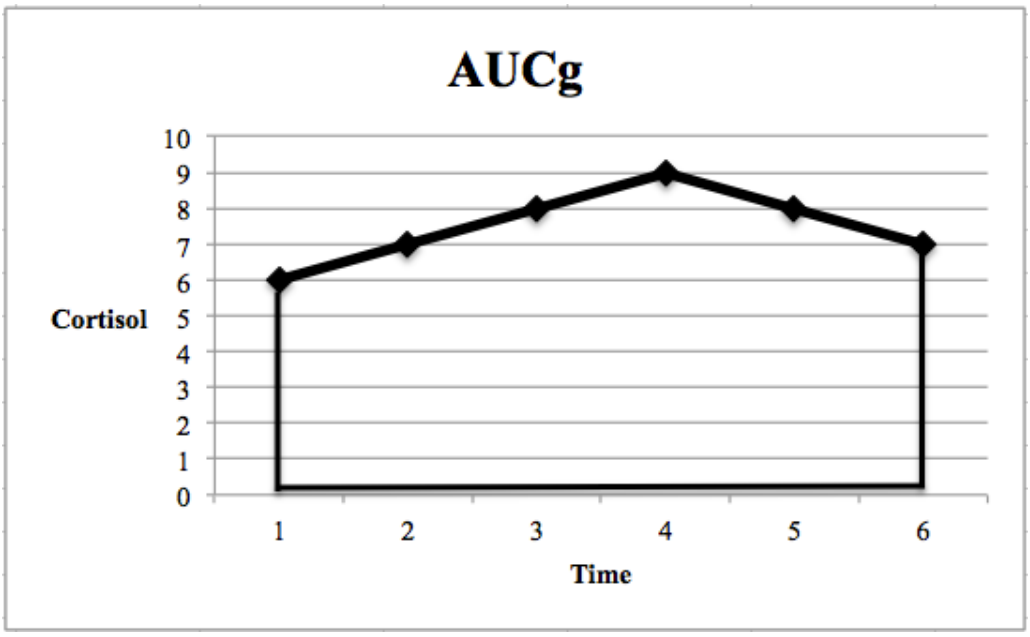


Figure 2. Best Fitting Dyadic Path Model, AUCg Model 3.

Note. Bolded black lines = Significant paths ($p < .05$). Non-bolded black lines = actor partner paths retained in model. Dashed black lines = Covarying paths. Grey lines = Paths constrained to zero.



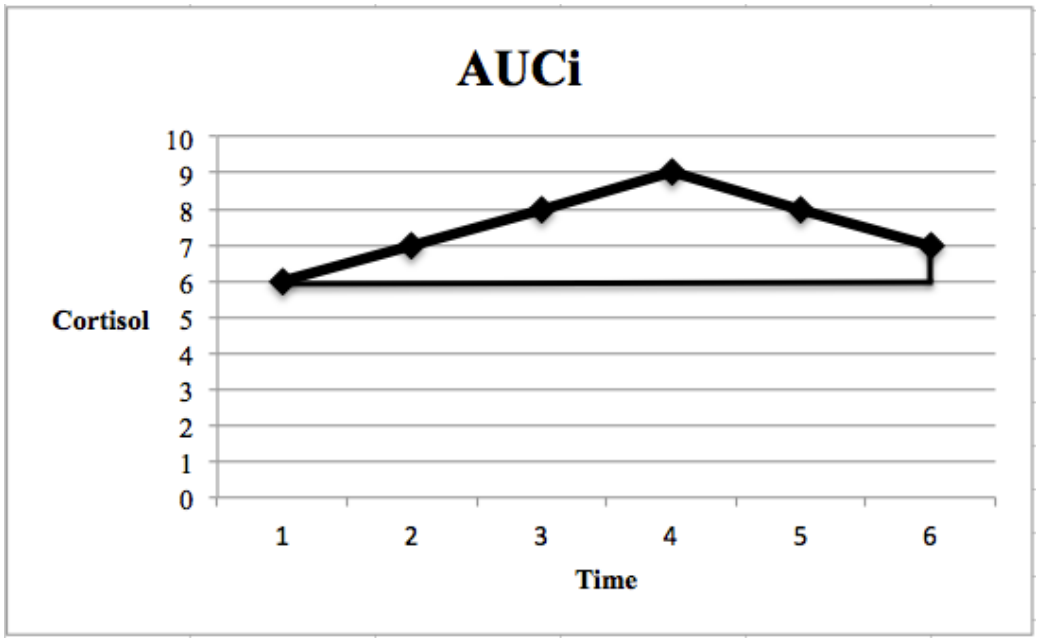


Figure 3. Visual Comparison of AUCg and AUCi Calculations

Note. This figure includes sample data. Notice that while the data points are the same, the total area of calculation is distinct. AUCg measures the total response with respect to zero (ground). AUCi measures the total reactivity with respect to the first data point (increase).

APPENDICES

APPENDIX A
RISKY FAMILIES QUESTIONNAIRE

These are questions about your childhood and early adolescence (age 5 – 15). Please think over your family life and answer these questions.

1. How often did a parent or other adult in the household make you feel that you were loved, supported, and cared for?

1	2	3	4	5
Not at All				Very Often

2. How often did a parent or other adult in the household swear at you, insult you, put you down, or act in a way that made you feel threatened?

1	2	3	4	5
Not at All				Very Often

3. How often did a parent or other adult in the household express physical affection for you, such as hugging, or other physical gestures of warmth and affection?

1	2	3	4	5
Not at All				Very Often

4. How often did a parent or other adult in the household push, grab, shove, or slap you?

1	2	3	4	5
Not at All				Very Often

5. In your childhood, did you live with anyone who was a problem drinker or alcoholic, or who used street drugs?

1	2	3	4	5
Not at All				Very Often

6. Would you say that the household you grew up in was well-organized and well-managed?

1	2	3	4	5
Not at All				Very Often

7. How often would you say that a parent or other adult in the household behaved violently toward a family member or visitor in your home?

1	2	3	4	5
Not at All				Very Often

8. How often would you say there was quarreling, arguing, or shouting between your parents?

1	2	3	4	5
Not at All				Very Often

9. How often would you say there was quarreling, arguing, or shouting between a parent and you?

1	2	3	4	5
Not at All				Very Often

10. How often would you say there was quarreling, arguing, or shouting between a parent and one of your siblings?

1	2	3	4	5
Not at All				Very Often

11. How often would you say there was quarreling, arguing, or shouting between your sibling(s) and you?

1	2	3	4	5
Not at All				Very Often

12. Would you say the household you grew up in was chaotic and disorganized?

1	2	3	4	5
Not at All				Very Much

13. How often would you say you were neglected while you were growing up, that is, left on your own to fend for yourself?

1	2	3	4	5
Not at All				Very Often

APPENDIX B
RAPID MARITAL INTERACTION CODING SYSTEM CODE DEFINITIONS

The Rapid Marital Interaction Coding System (RMICS) contains 11 behavior codes. The following summary of individual codes is derived from Heyman & Vivian (2000).

Negative Codes:
PA – Psychological Abuse

PA is defined as a communication intended to cause psychological pain to another person, or a communication perceived as having that intent. Context is especially important when coding PA. The cultural context is one such element: individualistic PA involves a personal attack on the spouse (e.g., belittling, mocking), whereas collectivist PA involves attacking a cultural group with which the victim closely identifies.

DA – Distress Maintaining Attribution

Attributions are the basic explanations that people give regarding the factors that cause a particular event. Attribution statements will often contain "because." The RMICS codes two types of attributions — distress maintaining and relationship enhancing. DAs are negative causal explanations. DAs explain negative behaviors as due to personality traits, or to voluntary or intentional causes. DAs explain positive behaviors as due to circumstances, or to involuntary or unintentional causes. A DA that is a self-derogatory statement is not a DA, but is coded as a DY. For example, "we can't afford to send the kids to camp because I am too stupid to get a good job" (said with sad voice tone).

HO – Hostility

Hostility comprises all negative affect, and statements with a negative content. The RMICS hostility code could include, a nonverbal response that communicates hostility, displeasure, disapproval, or disagreement, and is usually in reaction to something the other partner is saying or has just said. A hostile voice tone that accompanies nonnegative statements. A disapproval of the other's behavior. Negative inferences and assumptions made by one person about the spouse, including thoughts, beliefs, and intents that are not offered as attributions. Disagreements said with negative affect or that do not further the discussion. Note that disagreements that further discussion or explain a partner's point of view in a nonnegative way are coded as PD.

DY – Dysphoric Affect

Any of the following four conditions should be coded as DY. Any self–statement that indicates the subject has experienced, is currently experiencing or will experience some negative condition. Negative conditions can refer to physical problems, psychological problems, or a degrading/derogatory self–evaluation. Self– complaints of a psychological nature can be expressed in such specific terms as fear, anger, depression, or anxiety, or they can be expressed using more global terms such as down, grumpy, out of it, spacey,

irritable. Whiny voice tone or affect communicating sadness, despondency, or depression. Crying and tearfulness are included in DY. Persons who communicate dysphoric affect may show signs of sadness or distress such as speaking in a low, slow tone, becoming tearful, and verbally expressing their sadness. A DA that is a self-derogatory statement is not a DA, but in coded as a DY. For example, "we can't afford to send the kids to camp because I am too stupid to get a good job" (said with sad voice tone).

WI – Withdrawal

WI is coded for behaviors that imply pulling back from the interaction, walling off the partner, or not listening to the speaker. Withdrawal does not consist of any one behavior, nor is it cued by any set cluster of behaviors of affective signs. Rather, the coder must make a judgment, based on the flow of the conversation and the verbal and nonverbal cues, if someone is withdrawing. WI can be used as either a speaker or listener code. A verbal WI is when one partner expresses a desire to end the discussion in a non–neutral voice tone. The following are cues to non–verbal WI. Closed–off body language (e.g., folded arms, moving body away from partner), especially when there is a change from a more open position during a turning point in a discussion. Failure to respond (verbally or nonverbally) to the partner's question. Muscular tenseness and/or rigidity. Facial and verbal indications of holding back emotions. Nonverbal expressions that indicate that the listener is not listening (e.g., no eye contact, direct but glazed eye contact, turning away from speaker). A sudden decrease in listener backchannel behaviors.

Positive Codes:
AC – Acceptance

Acceptance comprises active listening skills that help the partner feel understood and validated. This code includes all utterances that demonstrate understanding and acceptance of the partner. AC is characterized by the speaker trying to put her/himself in the partner's place, so s/he can comprehend the other's feelings and emotions better. AC includes statements that involve paraphrasing (Restating partner's statement in your own words) and reflecting feelings (voicing what you thought the partner's underlying feelings were), giving positive feedback, and expressing caring, concern, or understanding of the partner's experience.

RA – Relationship Enhancing Attribution

Attributions are the basic explanations that people give regarding the factors that cause a particular event. Attribution statements will often contain "because." The RMICS codes two types of attributions — distress maintaining and relationship enhancing. RAs are positive causal explanations. RAs explain negative behaviors as due circumstances, or to involuntary or unintentional causes. RAs explain positive behaviors as due to personality traits, or to voluntary or intentional causes.

SD – Self-Disclosure

Self-Disclosures are statements about the speaker's feelings, wishes or beliefs. Generally, SDs include "I" statements, which should be either explicit or strongly implied. SDs should reveal something about the person. Moreover, if a speaker talks about a self–disclosure which occurred in the past, the self–disclosure is still coded as a SD. 1. Direct expressions of feelings are also coded as SDs. This includes all speech contributions which mention a positive or negative feeling by name. These can be feelings about specific things. Excluded are global negative feelings (e.g., anger, hate, dislike, disgust) expressed with "you" as an object and meant to hurt or criticize the receiver (these are coded as HO or PA). Also excluded are disclosures of depressive thoughts or feelings, which are coded as DY.

HM – Humor

Any statement that is clearly intended to be humorous will be coded HM. An HM is usually made in a lighthearted tone and is almost always accompanied by laughter from the person making the statement. Furthermore, HM statements will often (but not always) evoke laughter from the other. HM is also coded for each turn of a laugh or a smile (excluding nervous laughter or smiling). Humor with even slight undertones of sarcasm (directed at the spouse) is coded as HO; sarcastic humor that is mean or hurtful would be coded as PA.

The following codes are used in calculating total codes but are not individually included in proposal (as is standard in RMICS research).

Neutral Code:

PD – Constructive Problem Discussion/Solution

This code comprises all constructive approaches to discussing or solving problems. The RMICS code includes the following: All proposals of constructive plans/contracts to solve problems; suggestions for problem resolution that entail increases in the target behavior; suggestions for problem resolution that entail decreases in the target behavior; verbal inquiries toward the partner; verbal or nonverbal signs of accord.

Other Code:

OT – Other

OT is most often coded when the experimental situation itself is discussed. OT is coded conservatively; the statement must be clearly out of bounds. If the couple strays from the appointed topic, but is talking about anything relevant to their lives or marriage, use a richer code.

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