# AFFECTIVE ATTUNEMENT AND ADRENOCORTICAL ATTUNEMENT AS PATHWAYS BETWEEN INTIMATE PARTNER VIOLENCE AND CHILD BEHAVIOR PROBLEMS

By

Lia Field Martin

A DISSERTATION

Submitted to
Michigan State University
in partial fulfillment of the requirements
for the degree of

Psychology – Doctor of Philosophy

2013

## ABSTRACT

AFFECTIVE ATTUNEMENT AND ADRENOCORTICAL ATTUNEMENT AS PATHWAYS BETWEEN INTIMATE PARTNER VIOLENCE AND CHILD BEHAVIOR PROBLEMS

By

Lia Field Martin

Children exposed to intimate partner violence [IPV] are at increased risk for experiencing deficits in emotional and behavioral self-regulation. One pathway between children's IPV-exposure and the development of behavior problems is the effects of IPV on the mother-child care-giving relationship, specifically on mother-child attunement, a process that is thought to underlie children's development of self regulation skills. Mother-child dyads that are affectively attuned have mothers who can accurately read their children's emotional states and respond to them sensitively and appropriately; children are then able to use their mothers' responding to successfully regulate their affect. Research with non-IPV exposed populations indicates that some mother-child dyads also exhibit attuned adrenocortical stress responses, and that mothers from more physiologically attuned dyads are more affectively attuned to their children. However, it remains unclear whether there is a causal relationship between these two forms of attunement. It is also unclear whether the relationship between affective and adrenocortical attunement is the same for dyads exposed to IPV. The current study hypothesized that affective and adrenocortical attunement functions differently in IPV-exposed dyads due to the negative psychological and physiological effects of IPV. For example, IPV may pose a threat to affective attunement by undermining the mother's sense of efficacy as a care-giver for her child, and infringe upon the child's sense of emotional security in the care-giving relationship. IPV may also pose a threat to physiological attunement because it can alter women's and children's adrenocortical stress reactivity. This study sought to more fully elucidate the nature of affective and physiological attunement in IPV-exposed populations, as well as the contribution of attunement to child behavior problems. The sample included143 mother-child dyads recruited from a Midwestern Head Start program. Dyads participated in a lab stress task, providing baseline, peak and recovery cortisol measures; difference scores for the three cortisol values were used to assess adrenocortical attunement. During a reunion episode dyads were coded in-vivo for quality of affective attunement. Mothers also reported on IPV exposure, their children's behavior problems, and their own ability to mentalize about their children's emotional states. Variable-centered statistics (structural equation modeling) and person-centered statistics (predictive configural frequency analysis; PCFA) were used to examine associations between IPV, adrenocortical attunement, affective attunement and child behavior problems. Structural equation modeling indicated that IPV was not associated with adrenocortical attunement, but more IPV was associated with less affective attunement in the dyads, and more behavior problems in children. Additionally, more adrenocortical attunement predicted less affective attunement; however, affective attunement did not predict child behavior problems. Results of this study suggest that IPV exposure can interfere with successful affective attunement, and that affective attunement can also be impeded upon if dyads are too physiologically attuned, perhaps as a result of an emotional contagion effect. Although affective attunement did not predict child behavior problems, it may be important for other aspects of child social-emotional functioning, such as empathy or theory of mind skills. Additionally, structural equation modeling suggested that there are both cognitive and behavioral components of attunement. These findings have implications for future research on attunement in IPV-exposed populations, as well as for clinical intervention with IPV-exposed mothers and their children.

## Dedication

This manuscript is dedicated to the many women and children I have met throughout the course of my training, the brave survivors of unthinkable violence who have inspired this work through their stories and their strength.

ACKNOWLEDGEMENTS

A huge, heartfelt thank you goes out to all of my friends and family who provided me with support, love, and guidance, without which I would not have been able to complete this work. Specifically to my mother, Susan Field, I thank you for the endless evening phone calls, and for always being there to share in my triumphs as well as my tribulations. To my father, Mark Field, thank you for reminding me that dissertating is as much an exercise in perseverance as it is intelligence, just when I needed to hear it most. Special thanks goes to my friend and partner in crime, Audie Black, whose focus, drive and attention to detail helped to keep the Healthy Moms Healthy Kids Study steadily on course. Thanks also to my original cohort, Parker Huston, Jesse Bledsoe and Alexia Spanos, without whom I might not have survived this crazy, beautiful journey. To my advisor and mentor, Alytia Levendosky, I give my thanks for your guidance, patience and wisdom, all of which allowed me to discover my true self as an academic, thinker and psychologist. As Galileo said, “You cannot teach a man anything; you can only help him find it within himself.” And finally, to my husband, Liam Martin, I give my endless gratitude for your steadfast support and love which helped me endure during one of the most difficult experiences of my life. Liam, you are my heart’s true companion, always.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

## Introduction

Intimate partner violence (IPV; defined as male perpetrated physical or sexual violence, threats of violence, and/or use of coercive control with a female partner) is a significant and costly social issue, resulting in serious mental and physical health problems for IPV-exposed individuals. The negative mental health consequences for adult female survivors of IPV are well documented (e.g., Bogat, Levendosky, DeJonghe, Davidson & von Eye, 2004; Hill, Schroeder, Bradley, Kaplan & Angel, 2009; Levendosky, Leahy, Bogat, Davidson & von Eye, 2006; Martinez-Torteya, Bogat, von Eye, Davidson & Levendosky, 2009). Additionally, the increased risk for psychopathology in IPV-exposed women is of concern given the high prevalence of IPV exposure among adult females in the U.S. One study estimated that approximately 20% of women have a lifetime history of violence by a current or former partner (Tjaden & Thoennes, 2000), while a more recent national survey suggested that as many as 1 in 4 women report a lifetime experience of IPV (Breiding, Black & Ryan, 2008). Additionally, research indicates that these estimates may be suppressed by as much as 8-13% depending on the methodology used when assessing IPV prevalence (Waltermaurer, Ortega & McNutt, 2003).

Although women are frequently the direct victims of IPV, children living in households where relationship violence occurs are also adversely affected by IPV-exposure. Research suggests that the prevalence of child exposure to IPV is alarmingly high, with estimates ranging between 10 and 15 million children witnessing IPV in their homes annually (Jaffee, Wolfe & Wilson, 1990; McDonald, Jouriles, Ramisetty-Mikler, Caetano, & Green, 2006; Straus, 1992). An examination of 5,000 substantiated police reports of IPV collected over a two-year period of time from one Northeastern county indicated that children were physically present for approximately

50% of the events (Fantuzzo, Fusco, Mohr, & Perry, 2007). Of particular note is the prevalence of clinically significant internalizing (e.g., depression, anxiety, fear, phobias, self-esteem problems) and externalizing (e.g., fighting, tantrums, defiance, aggression) problems observed in children exposed to IPV (e.g., Fantuzzo & Mohr, 1999; Grych & Fincham, 1990; Holden & Ritchie, 1991; Katz, Hessler & Annest, 2007; Malik, 2008; Onyskiw & Hayduk, 2001; Sternberg et al, 2006). For example, Malik (2008) found that even when controlling for income, ethnicity, child age and child gender, increased exposure to IPV was associated with a greater number of externalizing behavior problems in children. It has been estimated that IPV-exposed children are almost 5 times more likely to exhibit psychopathology characterized by deficits in emotion regulation than those not exposed to IPV (Sternberg et al, 2006). It has also been estimated that children exposed to IPV are 1.6 times more likely than non-exposed children to have clinically significant problems with externalizing behavior, such as aggression and delinquency (Kernic, Wolf, Holt, McKnight, Huebner & Rivara, 2003).

In addition to behavior problems, children exposed to IPV also exhibit deficits in prosocial behaviors and problems with peer relationships. For example, compared to children from nonviolent families, IPV-exposed children have poor social problem-solving skills and lower levels of empathy for others than non-exposed children (e.g., Fantuzzo & Mohr, 1999). IPV-exposed children report feeling lonelier and report more conflict in their close peer relationships (McCloskey & Stuewig, 2001). Children exposed to IPV also have less emotional awareness (e.g., ability to identify, express, regulate and resolve emotional states) and thus have fewer intimate peer relationships and a greater likelihood of developing friendships based on superficial characteristics (Katz, Hessler, & Annest, 2007). The current study aims to explore one potential pathway by which IPV exerts these negative effects on children’s social-emotional development and the development of child behavior problems: the effects of IPV on mother-child attunement. More specifically, the current study will examine mother child attunement as it occurs at both the physiological and behavioral levels to better understand how these dyadic processes may be related to one another, and whether such mother-child attunement processes are impacted by IPV exposure, thus impeding upon children's social-emotional development.

Research examining how IPV is related to these social-emotional problems in children has often focused on maternal parenting practices such as warmth, involvement, and use of harsh discipline techniques (e.g., Krishnakumar & Buehler, 2000; Levendosky, Leahy, Bogat, Davidson & von Eye, 2006; Onyskiw & Hayduk, 2001) and maternal mental health problems such as depression and posttraumatic stress disorder as mediating variables (e.g., Levendosky et al., 2006; Whitaker, Orzol, & Kahn, 2006; Street, King, King & Riggs, 2003). However, there is only partial evidence to suggest that maternal parenting and psychopathology account for the relationship between IPV exposure and subsequent child behavior problems. For example, longitudinal research has indicated that even when controlling for potential confounds such as maternal age, race, level of education, employment status, substance use problems, physical health, social support and child temperament, the relationship between IPV and child outcomes is only partly mediated by maternal depression and maternal parenting practices (e.g., Huang, Wang & Warrener, 2010). These findings highlight the role of the mother-child relationship for the development of behavior problems in the IPV-exposed child, but also suggest that other more nuanced aspects of the mother-child relationship beyond the broad dimensions of parenting warrant exploration. One such aspect of the mother-child relationship that may contribute to the development of behavior problems in IPV-exposed children is affective attunement.

Affective attunement is defined as a mother's ability to decode her child's affective state and then respond to such in a way that is both empathic and that aids the child in affect-regulation. This construct is of particular interest because of the important role it plays in children's development of emotional regulation and social skills (e.g., Eisenberg, Spinrad & Eggum, 2010; Goldberg, MacKay-Soroka & Rochester, 1994; Kochanska, 1997; Healey, Gopin, Grossman, Campbell & Halperin, 2010). When healthy affective attunement is present, the mother's responses communicate to the child that she is sharing in the child's internal experience of the interaction. In doing so she acts as an external regulator of the child's affective state, helping the child to develop the internal capacity for self-regulation. Furthermore, because of its dyadic nature, affective attunement promotes children's ability to regulate affective behavior within the context of interpersonal relationships. Finally, affective attunement teaches children how to decode the internal states and processes of others, thus laying the framework for children's competence in the realms of interpersonal intimacy, empathy, moral behavior and other psychosocial domains (Feldman, 2007a; Kochanska & Murray, 2000). This is evidenced by research suggesting that mother-child affective attunement is associated with better child functioning and fewer externalizing behavior problems (e.g., Healy et al., 2010).

Although affective attunement may be influenced by a variety of individual factors such as mothers' early attachment security (Haft & Slade, 1989), maternal stress and social support (e.g., Crnic, Greenberg, Robinson & Ragozin, 1984), and child temperament (e.g., Lee & Bates, 1985), as well as contextual factors such as socioeconomic disadvantage (e.g., Cezero & Pons, 1996), IPV can also pose a unique threat to the development of mother-child affective attunement during early childhood. IPV is a type of intentional harm that occurs at the hand of a trusted romantic partner, and unlike other types of relational traumas experienced during adulthood (e.g., rape, assault) IPV tends to be chronic and recurring in nature. The chronic nature of IPV and the dynamics of power, control and coercion that often occur in abusive relationships have the potential to influence the way a woman thinks about herself and others with whom she shares significant relationships. A woman's beliefs about herself as a care-giver, nurturer and protector may be particularly vulnerable to the effects of IPV, and as IPV undermines a woman's sense of self as a care-giver, the way she relates to her child may also be negatively impacted.

Although there is a great deal of research on the general parenting practices of IPV-exposed women, there exists only a handful of research studies regarding affective attunement in violence-exposed samples, and these have yielded inconsistent findings. Johnson and Lieberman (2007) examined associations between IPV exposure, mother-child attunement, and child internalizing and externalizing problems and found no direct association between IPV-exposure and mother-child attunement. Conversely, research examining parental emotional coaching, (a construct that shares many similarities with affective attunement) indicated that victims of marital aggression are less able to coach their children through experiences of negative emotion compared to non-exposed parents (Katz & Windecker-Nelson, 2006). Finger, Hans, Bernstein and Cox (2009) also found that IPV-exposure shared a significant negative relationship with maternal warmth and attunement behavior, such that greater IPV was associated with mothers' use of fewer attunement behaviors.

The limited research regarding the influence of IPV on affective attunement and child outcomes has also focused primarily on attunement at the affective level, for example, evaluating mothers' ability to successfully read their children's affective cues and appropriately respond (e.g., Johnson & Lieberman, 2007). However, recent research suggests that mother-child attunement can also occur at the physiological level. For example, some mother-child dyads demonstrate coordinated changes in cortisol levels in response to typical daily stressors (e.g., Sethre-Hofstad, Stansbury & Rice, 2002; van Bakel & Riksen-Walraven, 2008). Furthermore, this attuned stress response appears to be related to maternal sensitivity, such that only dyads with highly sensitive mothers (e.g., mothers who were consistently responsive and able to accurately perceive and address their children's cues) exhibited this coordinated stress response with their children (Sethre-Hofstad et al., 2002). Despite the fact that this association between behavioral and physiological attunement has been replicated in research by van Bakel and Riksen-Walraven (2008), the functional relationship between these two phenomena and the significance of this association remains largely unclear.

It is also important to note that the majority of research examining associations between affective attunement and physiological attunement has been conducted with low-risk, non-IPV-exposed samples. It is of critical importance to examine associations between affective attunement and physiological attunement in IPV-exposed populations for two reasons. First, the chronic, interpersonal nature of IPV and the power and control dynamics of IPV can lead to enduring changes in how mothers interpret and respond to the affective cues of their children, thus impeding upon affective attunement processes. Second, IPV is associated with alterations in adrenocortical stress reactivity in both women and children (e.g., Davies, Sturge-Apple, Cicchetti, Manning & Zale, 2009; Griffin, Resick & Yehuda, 2005; Inslicht, Marmar, Neylan, Metzler, Hart, Otte, et al., 2006; Pico-Alfonso, Garcia-Linares, Celda-Navarro, Herbert, & Martinez, 2004; Schechter, Zeanah, Myers, Brunelli, Liebowitz, Marshall et al., 2004). Thus, the nature and function of both physiological and affective attunement may be altered in IPV-exposed dyads. Furthermore, physiological attunement may share a different association with affective attunement in IPV-exposed populations.

To date, only one known study has explored the relationship between affective and physiological attunement in a violence-exposed population (Hibel, Granger, Blair & Cox, 2009). The results of this study yielded several important findings including: (a) IPV moderates the presence/absence of mother-child adrenocortical (physiological) attunement, such that only those dyads exposed to IPV exhibited attuned cortisol reactivity in response to a stressor, (b) mothers' use of restrictive and punitive parenting behavior were significantly associated with adrenocortical attunement, and (c) maternal parenting behavior did not moderate attunement of adrenocortical response for IPV mother-child dyads (and vice versa), suggesting that the association between the two types of attunement may not function the same way in IPV-exposed dyads as it does for non-exposed dyads (Hibel et al., 2009). While the results of this study represent an important starting place for understanding the nature of physiological and affective attunement in IPV populations, there are several methodological limitations in this research pertaining to the measurement of maternal parenting behavior and IPV. Finally, although there have been studies examining the contribution of affective attunement to IPV-exposed children's externalizing and internalizing problems (e.g., Johnson & Lieberman), no known study to-date has examined the role of both physiological and affective attunement together. Previous research suggests that adrenocortical attunement is adaptive or positive because it is related to increased parental sensitivity and more attuned parental responding, but it is unclear whether or how this may be related to child outcomes in general, and for IPV-exposed populations.

Building upon prior research, the current study seeks to address five basic science questions regarding the nature of attunement in IPV-exposed mother-child dyads:

1. Is physiological dysregulation (e.g., impairment in any aspect of the HPA-axis, as indicated by cortisol levels outside the normal range) observed in IPV exposed mothers and children in response to acute stress?
2. Is affective dysregulation (e.g., impairment in the modulation of emotional reactions; Thompson, 1994) observed in IPV-exposed mothers and children in response to acute stress?
3. How is IPV related to mother-child attunement both at the affective and physiological levels?
4. Is attunement in one system (affective, physiological) meaningfully associated with attunement in the other?
5. Finally, how are physiological and affective attunement (or lack thereof) related to child outcomes in terms of internalizing and externalizing behavior problems?

The remainder of this dissertation includes several chapters that review both the theoretical and empirical literature pertaining to the aims of this study. The topics reviewed include the influence of IPV on children's behavior problems, the significance of IPV as a psychological stressor, affective attunement and the impact of IPV on affective attunement, the impact of IPV on physiological stress reactivity in mothers and children, and physiological attunement. The review will conclude with a synthesis of this work, as well as critique of the only two known empirical studies conducted on this subject matter to-date, forming the rationale for the current study. This will be followed by a description of the study hypotheses, research questions and research methodology. The results of the data analysis will then be provided. Finally, a discussion of the study's main findings, as well the study limitations and clinical and research implications will be discussed.

## Chapter 1: Behavioral Outcomes of IPV-Exposed Preschool Age Children

### *1.1 Clarifying the Definition of IPV*

IPV has often been thought of as a single unidimensional construct characterized by male perpetrated acts of physical and sexual violence toward a female partner. However, more recently, it is becoming increasingly acknowledged that women's experiences of IPV are heterogeneous and that the behaviors that characterize IPV have many different forms and communicate many different meanings. More specifically, work by Johnson and others (1995; Johnson & Leone, 2005; Kelly & Johnson, 2008; Leone, Johnson, Cohan, & Lloyd, 2004; O'Leary, 1999) has highlighted the importance of recognizing psychological aggression and emotional abuse as components of IPV. Psychological aggression and emotional abuse can include, but are not limited to isolating the victim from loved ones, using threats and intimidation, blaming, threatening to use the children against the partner, and cutting off the victim from economic resources. An individual's use of physical aggression and psychological tactics can be fluid and changing throughout the course of the romantic relationship (Johnson, 2005; Kelly & Johnson, 2008), and research indicates that both aspects of IPV can have deleterious effects on women (e.g., Aguilar & Nightingale, 1994; Arias & Pape, 1999; Johnson & Leone, 2005; Leone, Johnson, Cohan, & Lloyd, 2004) and children (e.g., Huston et al., 2010; Litrownik, Newton, Hunter, English & Everson, 2003; Martinez-Torteya et al., 2009). In some cases the effects of psychological abuse can be more profound than those of physical violence (O'Leary, 1999). Thus, in order to capture the full heterogeneity of the IPV construct, it is critical to consider exposure to physical and sexual violence as well as psychological and emotional aggression.

*1.2 The Prevalence of Exposure to IPV in Preschool Age Children*

It is estimated that approximately 20% of women have a lifetime history of intimate partner violence by a current or former partner (Tjaden & Thoennes, 2000). Estimates from the U.S. Census suggest that approximately 1.5 million women become victims of IPV annually in the U.S. (Tjaden & Thoennes, 1998). However, when violence occurs in the home women are often not the only ones exposed, with studies showing that the prevalence of IPV is greater in homes with children than in homes without children (McDonald, Jouriles, Ramisetty-Mikler, Caetano, & Green, 2006). Research using nationally representative samples suggests that the prevalence of child exposure to IPV is high, with estimates ranging between 10 and 15 million children witnessing IPV in their homes annually (Jaffee, Wolfe & Wilson, 1990; McDonald et al., 2006; Straus, 1992). Further, of the approximately 15 million children who witness IPV each year, 7 million are exposed to severe violence, including kicking, biting, hitting with a fist, hitting with an object, choking, burning/scalding, forced sexual contact, threats of violence by knife or gun, and/or actual violence with a knife or gun (McDonald et al., 2006).

Often when IPV occurs, children are direct witnesses of the violence. An examination of 5,000 substantiated police reports of IPV collected over a two-year period of time from one Northeastern county indicated that children were physically present for approximately 50% of the IPV events (Fantuzzo, Fusco, Mohr, & Perry, 2007). In a similar examination of approximately 1,500 police-investigated IPV incidents, over 40% of the altercations occurred while children were present in the household, and in 95% of these cases the children had some kind of sensory exposure to the violence (e.g., physically seeing or hearing the event). Three percent of the children in this study incurred some kind of injury while witnessing the IPV event, and a disproportionate number of IPV-events that were witnessed by children were experienced by child under the age of 6 years (Fusco & Fantuzzo, 2009). Finally, in nearly 40% of the cases children were physically involved in the IPV event either passively (e.g., being held in the victim's arms while she was assaulted) or actively (e.g., trying to pull the perpetrator away), while in 28% of the cases a child placed the call for help to the police (Fusco & Fantuzzo, 2009).

*1.3 Behavioral and Social-emotional Outcomes Associated with Child IPV-Exposure*

The rates of child exposure to IPV are concerning, as research has repeatedly documented the deleterious effects of IPV exposure on child outcomes (e.g. Chan & Yeung, 2009; Evans, Davies & DiLillo, 2008; Huang et al., 2010; Kernic et al., 2003; Kitzmann, Gaylord, Holt & Kenny, 2003; Margolin & Gordis, 2000; McDonald, Jouriles, Briggs-Gowan, Rosenfield & Carter, 2007; McFarlane, Groff, O'Brien & Watson, 2003; Meltzer, Doos, Vostanis, Ford & Goodman, 2009; Wolfe, Crooks, Lee, McIntyre-Smith & Jaffe, 2003). For example, a study of 167 IPV-exposed children ranging in age from 2 to 18 years indicated that children exposed to IPV (without co-morbid exposure to child maltreatment) were 1.6 times more likely than children in a non-IPV control group to exhibit borderline and clinically significant externalizing behavior problems (Kernic et al., 2003). In a similar study of 258 IPV-exposed children aged 18 months to 18 years violence-exposed children had significantly higher mother-reported scores of internalizing, externalizing, and total behavior problems as compared to children from non-IPV households (McFarlane et al., 2003). IPV-exposed children scored significantly higher on all three scales of behavior problems when compared to clinically-referred norms, as well (McFarlane et al., 2003). In terms of specific psychopathology and clinical disorders, research has indicated that even when controlling for risk factors such as race, single parenthood, parental unemployment, poverty and maternal mental health, children exposed to IPV are almost two times more likely to meet for a diagnosis of conduct disorder when compared to non-exposed children (Meltzer et al., 2009). Similarly, another study found that one-third of children between the ages of 4 and 10 residing in battered-women's shelters exhibited clinical levels of conduct problems (Ware, Jouriles, Spiller et al., 2001).

With regard to preschool-aged children specifically, research has shown that IPV exposure experienced when children were age 1 year was directly and positively associated with both internalizing and externalizing child behaviors problems at age 5, even after accounting for maternal mental health and parenting practices (Huang et al., 2010). In another preschool-age sample, children exposed to IPV were significantly more likely to exhibit atypical/maladaptive behavior problems (e.g., perseverative play, making odd noises) and externalizing behavior problems when compared to children from homes characterized by non-violent verbal adult conflict (McDonald et al., 2007).

Though research repeatedly indicates that IPV is associated with increased child behavior problems, much of this research is confounded by methodological limitations, like failing to examine child age at first exposure to IPV, not accounting for differences in the chronicity and severity of IPV, and not accounting for co-morbid exposure to child maltreatment. Meta-analytic studies examining the overall effect of IPV on child behavior problems have been able to control for some of these methodological limitations. For example, a meta-analysis conducted by Chan and Yeung (2009) found a moderate but significant effect of IPV on both children's internalizing and externalizing problems (.209 and .230 respectively), and determined that these effects were not moderated by factors such as study design, IPV reporting source, co-morbid child maltreatment exposure, child age or child gender. Similar moderate effect sizes were found in meta-analyses conducted by Kitzmann et al. (2003) and by Wolfe et al. (2003), while Evans and colleagues (2008) found larger but also moderate effect sizes of .48 for internalizing problems and .47 for externalizing problems.

In addition to behavior problems, children exposed to IPV also exhibit deficits in emotional competence, prosocial behavior and peer relationships. For example, compared to children from nonviolent families, IPV-exposed children have poor social-problem solving skills and lower levels of empathy for others (e.g., Fantuzzo & Mohr, 1999). IPV-exposed children also report feeling lonelier and experiencing more conflict in their close peer relationships (McCloskey & Stuewig, 2001). Furthermore, children exposed to IPV have less emotional awareness (e.g., ability to identify, express, regulate and resolve emotional states), fewer intimate peer relationships and an increased likelihood of developing friendships based on superficial characteristics (Katz, Hessler, & Annest, 2007). In a study exploring IPV-exposed children's responses to peer provocation (e.g., being teased or mocked, listening to another child brag), IPV-exposed children were more likely to exhibit odd behaviors such as laughing or making nonsensical statements compared to non-exposed children, suggesting that these children may become emotionally dysregulated when confronted with negative affect in others (Katz, Hunter & Klowden, 2008). In a study of college students, those who experienced IPV during childhood demonstrated more difficulty with the encoding of emotional expressions (expressing a specific emotion through verbal and non-verbal behavior) compared to those without an IPV history (Hodgins & Belch, 2000). These findings suggest that growing up in the context of IPV may somehow deprive children of opportunities to learn how to appropriately and effectively communicate emotions (Hodgins & Belch, 2000).

In trying to understand this association between children's IPV exposure and increased risk for behavior problems and social-emotional difficulties, a number of potentially mediating and moderating variables have been examined, including maternal mental health (e.g., Owen, Thompson, Shaffer, Jackson & Kaslow, 2009; Huang et al., 2010), parenting stress (e.g., Levendosky & Graham-Bermann, 1998; Huth-Bocks & Hughes, 2008; Owen, Thompson & Kaslow, 2006), maternal social support (e..g, Owen, Thompson, Mitchell et al., 2008), and children's appraisals of violence (e.g., Fosco, DeBoard & Grych, 2007; Kerig 1998). While there is empirical support that these variables all influence children's outcomes in the context of IPV, one variable which is repeatedly identified as a significant mediator of children's outcomes is the quality of the mother-child relationship (e.g., Hazen, Connelly, Kelleher, Barth & Landsverk, 2006; Levendosky, Huth-Bocks, Shapiro & Semel, 2003; Skopp, McDonald, Jouriles & Rosenfield, 2007). One aspect of the mother-child relationship that may be particularly important for children's social-emotional outcomes in the context of IPV is mother-child affective attunement. As will be discussed in more detail in the following section, affective attunement plays a critical role in how children learn to manage and regulate their emotional experiences, particularly within relational contexts (e.g., Feldman, 2010; Healy, Gopin, Grossman, Campbell & Halperin, 2010; Kochanska, Aksan, Prisco & Adams, 2008), thus it is of particular interest in the current study.

One reason IPV may pose a threat to attunement is because partner violence can have a *spillover effect* to other interpersonal relationships and relational processes within the family system (e.g., Engfer, 1988; Magolin, Gordis, Medina & Oliver, 2003; Repetti, 1987). From the family systems perspective, each dyadic relationship within a family is nested within a larger system of familial relationships which can have reciprocal effects upon one another. The spillover effect occurs when negative affect occurring within one relationship becomes transferred to another relationship as a result of the interconnectedness of the family system.

One of the more common ways in which spillover is observed within families is from the parent-parent relationship to the caregiver-child relationship. For example, research has shown that in families experiencing high rates of marital conflict, there is an increased likelihood for spillover of aggression and conflict to the parent-child relationship (e.g., Margolin et al., 2003). This is particularly true if the family system is made vulnerable by additional stressors, such as economic hardship or parenting stress (e.g., Margolin et al., 2003). Furthermore, because IPV occurs within the context of an adult attachment relationship, IPV can activate the attachment system thus allowing negative representations of the romantic partner to essentially spill over into other primary attachment-based relationships (e.g., the mother-child care-giving relationship). For example IPV is associated with changes in the way that mothers represent the mother-child relationship, relate to their children, and construe their children's behavioral and affective cues (e.g., Stephens, 1999; Sokolowski, Hans & Bernstein, 2007). These alterations may subsequently interfere with mothers' ability to appropriately attune and respond to such cues. The next chapter will provide a more detailed exploration of the ways in which IPV may exert negative psychological effects on the mother-child dyad and attunement processes.

# Chapter 2: The Psychological Significance of IPV as an Interpersonal Trauma

Throughout an individual's lifetime, he/she may be exposed to a number of different kinds of events which could potentially result in a traumatic stress reaction (e.g., developing symptoms such as re-experiencing, avoidance, increased affective arousal or increased physiological stress response) (please note: such types of events will be referred to as *traumas* for the remainder of this paper). It is important to note, however, that IPV exposure represents a fundamentally different type of experience when compared to other types of acute traumas (e.g., car accident, natural disaster) and long-term traumas (e.g., environmental deprivation) because it tends be *both* chronically occurring and relational in nature. IPV is also a type of trauma that is carried out with intent and that occurs at the hand of a trusted attachment figure (the romantic partner). Because of these characteristics, IPV can undermine a woman's belief in her capacity to be an adequate care-giver for her child, as well as her perceived ability to provide physical and emotional safety for herself and the children in her household. In this way, IPV can exert deleterious psychological effects on both women and children alike.

## *2.1 Psychological Effects of IPV on Women through the Care-giving System*

From an attachment theory perspective, the mother-child relationship is composed of two complementary psychological systems, one of which functions to guide the behavior of the mother within the dyad (the care-giving system), the other of which functions to guide the behaviors of the child (the attachment system), and both of which together contribute to the quality of the mother-child relationship. From an evolutionary standpoint, these complementary systems are adaptive and evolved both to promote the survival of the child, as well as to ensure the mother's reproductive success (Bowlby, 1969). Survival of the child is enhanced by the attachment system, which becomes activated in times of distress or perceived threat, and drives the child to seek protection and proximity to his/her care-giver. The complimentary care-giving system also serves to enhance child survival by guiding the mother's behavioral responses when the child's safety is threatened, prompting her to provide protection and comfort for her child (George & Solomon, 2008). The mother's ability to respond to the child appropriately during these moments allows trust and security to develop within the dyad.

Early experiences within the care-giving relationship also give rise to internal representations, or an individual's "template" (e.g., beliefs, ideas, feelings and expectations) for how she thinks about the self in relation to others. Internal representations derived from the care-giving system specifically pertain to the mother's own expectations and beliefs about herself as a care-giver, including her ability to provide adequate protection and comfort to her child. It is primarily through an undermining of the care-giving system and a woman's representations of herself as a caregiver that IPV exerts negative psychological effects on the mother (George & Solomon, 2008; Levendosky, Bogat, & Huth-Bocks, 2011).

It has been suggested that the care-giving system first begins to emerge during adolescence and early adulthood, but also becomes flexible and open to change at times when the individual takes on new relationship roles, for example when a woman becomes a mother for the first time (Solomon & George, 2008). An individual's "psychological plasticity" during these times works somewhat like a double-edged sword: it both allows the individual to develop new internal representation of the self (e.g., that of mother), but also makes her internal representations more vulnerable to the influence of other important relationships in her life (e.g., her own relationship with her parents or the relationship she shares with her romantic partner)

(Levendosky, Bogat & Huth-Bocks, 2011). For women whose romantic relationships are characterized by IPV this is particularly problematic, as IPV can create both real and perceived barriers to mother's ability to protect their children. For example, an abusive partner may verbally damage a woman's sense of self as care-giver by insulting her competency as a mother and belittling her parenting skills. Abusive partners may also make threats to harm the children, take the children away from the mother, or report the mother to the police as a way of asserting control in the relationship. Such use of intimidation, ridicule and threats, in addition to use of physical and sexual violence, can leave a mother feeling powerless to protect herself and her child from the perpetrator. Additionally, physical injury to the mother or child during the course of a violent altercation can pose an actual threat to the mother's ability to protect her child.

George and Solomon (2008) suggest that when a woman feels helpless to protect her child (and herself) from the threat of danger in this way, she may essentially abandon her role of care-giver, nurturer and protector. For women who have experienced relational traumas such as childhood maltreatment or IPV, the relinquishment of the care-giver role may not be a conscious act, but the result of painful memories and emotions associated with relational trauma causing a break-down in care-giving behavior (George & Solomon, 2008). For example, activation of such memories and experiences may compete with the mother's ability to remain emotionally present with the child and/or make it difficult for her to differentiate her internal representations of her child from that of others (e.g., the perpetrator). These processes may directly and negatively influence maternal parenting behavior, particularly when the child exhibits proximity seeking behaviors (e.g., crying). In these moments the child's distress may be experienced by the mother as a "posttraumatic trigger", activating memories of emotional trauma and leaving the mother emotionally dysregulated and unable to engage in appropriate care-giving behaviors

(Levendosky, Bogat & Huth-Bocks). As a result, such mothers may look emotionally dysregulated or disorganized, helpless, or disengaged in these moments (Levendosky, Bogat & Huth-Bocks). These mothers may also experience distortions in the way they relate to their children, for example engaging in projective identification (e.g., viewing the child as a victim like oneself), role confusion (e.g., being unable to differentiate between the child and the self, seeking comfort from the child as if they were the protective parent), and/or projection (e.g., viewing and treating the child as if they were the abuser).

The next section will present evidence for the deleterious effects of IPV on the care-giving system, including damage to representations of the self as care-giver, damage to mothers' representations of their children, and disruptions in maternal care-giving behavior. This evidence comes from both qualitative (e.g., Stephens, 1999) and quantitative research conducted with IPV survivors (e.g., Sokolowski, Hans and Bernstein, 2007; Theran, Levendosky, Bogat & Huth-Bocks, 2005).

*2.2 Evidence from Qualitative and Quantitative Research with IPV Survivors*

Stephens (1999) conducted semi-structured interviews with IPV-exposed women and then coded the interviews for themes regarding cognitive distortions of their children and the care-giving relationship. One recurring theme was that of adultification of children, for example projecting adult motives onto normative child behavior, or viewing the child as embodying the same malicious characteristics as the IPV perpetrator. An illustration of this comes from a woman who described her young daughter's behavioral problems and inability to concentrate at school as a "…four going on four and a half year-old who can be manipulative kind of like her dad, and you don't know whether [her behavior is] the truth or not" (Stephens, 1999, p. 735). A second kind of adultification that was observed in the women's descriptions of their children was the projection of their own shame and self-loathing onto their children, and an inability to differentiate between the identity of the child and the self. For example, one woman described her young daughter as unattractive, yet no different from herself in that both needed to dress prettily because "it's so ugly inside that I don't want anyone to know" (Stephens, 1999, p.736).

Quantitative research examining the impact of IPV on maternal representations of their unborn children during pregnancy also illustrates the pernicious effects of IPV on the care-giving system (Huth-Bocks, Levendosky, Theran & Bogat, 2004). In this study, a community sample of women, approximately half exposed to IPV, were given the Working Model of the Child Interview (WMCI) during their third trimester of pregnancy to assess for maternal representations of the infant and of themselves as mothers. Based on their responses, the women's representations were classified into one of three categories: balanced (characterized by rich detail about the infant and the care-giving role, ability to imagine the infant's subjective experience and coherent integration of both positive and negative aspects of the infant), disengaged (characterized by emotional distance, a lack of detail, integration and flexibility, and a lack of regard for the infant's subjective experience), or distorted (characterized by inconsistent, unrealistic, or incoherent descriptions, emotional flooding and emotional dysregulation). The results indicated that women who were exposed to IPV during pregnancy were more likely than non-exposed women to be classified as distorted or disengaged. Furthermore, IPV-exposed mothers' narratives were generally less flexible, less coherent, less sensitive, less accepting of the infant, and included fewer feelings of self-efficacy as a caregiver compared to non-exposed mothers (Huth-Bocks et al., 2004). One year later, these classifications predicted the mothers' parenting behaviors with their children: mothers classified as disengaged during pregnancy acted more controlling with their children, while mothers classified as distorted were more hostile with their children (Dayton, Levendosky, Davidson & Bogat, 2010).

Another study utilizing this same sample of women examined the stability/instability of the women's representations between pregnancy and the end of infants' first year of life (Theran, Levendosky, Bogat & Huth-Bocks, 2005). In this study, the WMCI interview was again used to assess the women's representations of their children and themselves as caregivers, this time when their infants were one year of age. Using the WMCI categories from both pregnancy and the end of the first year of life, mothers were coded into four groups based on whether they demonstrated stability in their representations (remained balanced, remained non-balanced, shifted to from non-balanced to balanced, shifted from balanced to non-balanced). While a large number of women demonstrated stability in their internal representations, 21% of the women exhibited a shift from having balanced to non-balanced representations. Importantly, this shift in internal representations was associated with a number of factors, including low income and single parenthood, as well as exposure to significantly more IPV. Although this shift cannot be attributed to IPV exposure alone, this finding suggests that IPV exposure can not only shape the way women view and experience their children as the care-giving system develops, but IPV can also exert deleterious changes upon the care-giving system over time.

Finally, cross-sectional work by Sokolowski, Hans and Bernstein (2007) suggests that IPV is associated with maladaptive maternal representations of the self and of the child not only during the prenatal period and infancy, but during early childhood, as well. Mothers in this study reported on exposure to verbal or physical aggression from their romantic partners using the Conflict Tactics Scale (Straus, Hamby, Boney-McCoy, & Sugarman, 1996) and mothers' representations of their children (between 17-20 months in age) were assessed using the WMCI. Results indicated that for every unit increase in amount of verbal and aggressive conflict with a romantic partner, the odds of a mother having a distorted representation of her child increased significantly (by a factor of 1.84). Mothers with higher scores of IPV-exposure tended to provide narratives of their children characterized by more guilt, less sensitivity, and less openness to change (Sokolowski et al., 2007). As a whole, these findings provide ample evidence to suggest that IPV exerts psychological effects on women by damaging the care-giving system. IPV undermines the woman's belief in herself as a successful protector and nurturer, increasing the likelihood that she will perceive the mother-child relationship in distorted ways, thus impacting the quality of her care-giving behavior.

### *2.3 Psychological Effects of IPV on Children through Attachment, the Social Defense System and Emotional Security*

As mentioned earlier, women are not the only individuals exposed to and affected by IPV. Similar to adults, IPV may be experienced by children as an interpersonal trauma with the capacity to alter the way they view and behave within the attachment relationship. Schore (2002) and van der Kolk (2005) have offered a theory to explain why children exposed to interpersonal traumas such as IPV may be vulnerable to attachment disturbances. In a securely attached mother-child dyad, the "good-enough" mother allows the child appropriate distance to interact with the environment, has the capacity to respond both promptly and appropriately to the child's emotional bids, and uses interaction with child to teach appropriate affect regulation (Schore, 2002). However, in a rearing environment where violence is commonplace, the care-giver is often the source of negative affect rather than the one to help the child manage feelings of distress. For example, in a home where IPV occurs, the child may not experience his/her mother as a source of safety who can protect him/her from the abuser.

Furthermore, the IPV-exposed mother's ability to help her child manage emotional distress when conflict occurs within the family system may be limited, particularly if the mother is not emotionally or physical available to the child (e.g., as a result of injury sustained during the IPV). This may leave the child in a prolonged state of negative affective arousal without opportunities for recovery or repair, resulting in the child's experience of the traumatic event becoming disorganized. If this happens often enough, the child does not develop the capacity to integrate the physical sensations and emotional responses experienced during times of distress. The child is thus left without a systematic way to manage his/her affect (Schore, 2002; van der Kolk, 2005).

Furthermore, in the context of IPV, children may also develop a disturbed sense of self in relation to others. Pre-school age children in particular may be vulnerable to this, as their still immature cognitive capacities may lead them to make misappraisals of both the causes and meaning of IPV. For example, children may develop a fundamental misunderstanding of the cause of the violent events that they witness, viewing themselves as the source of the problem (Lieberman & Knorr, 2007). The tendency for children to view their own behavior, thoughts or wishes as the cause of the violent events around them can contribute to feelings of shame and other disturbances of the self in relation to others (Lieberman & Knorr, 2007). Recent research has generated empirical support for this theory. For example, Schechter et al. (2007) examined IPV-exposed children's internal representations of the self and of their mothers as care-givers. Mothers who endorsed having filed a restraining order against a dangerous romantic partner, or endorsed having experienced at least one violent physical or sexual assault in adulthood were classified as IPV-exposed. Children were between 8 months and 2 years old when mothers initially reported on experiences of IPV. Four years later, children were read story stems and asked to provide narratives regarding the adult and child characters. The narratives were coded for attachment related themes (e.g., caregiver protection, caregiver containment of fear/anxiety) and aspects of self representation (e.g., exclusion of self, boundary confusion, spacing out). Results indicated that the narratives of children from IPV homes were characterized by significantly greater dysregulated aggression and attentional biases towards danger/distress. Additionally, the narratives provided by children of violence-exposed mothers contained significantly less coherent internal representations of their mothers and of the self, suggesting that child exposure to IPV may result in disturbances of self-concept and the self in relation to the caregiver (Schechter et al., 2007).

Davies and colleagues also suggest that IPV exposure can have significant psychological consequences for children; however, they propose that IPV affects children not through its effects on attachment but rather by influencing feelings of emotional security (e.g., Davies & Cummings, 1994; Davies & Cummings 1998; Davies & Wotiach, 2008). Davies and Woitiach (2008) assert that humans have evolved to have two innate systems, both of which serve to promote survival during the earliest years of life when we are most vulnerable and unable to physically protect ourselves. The first system is the attachment system, the system which becomes activated in times of danger or distress and promotes proximity to the caregiver in order to ensure safety. The second system proposed by Davies and Wotiach, the *social defense system*, evolved in order to protect the developing child from threats to safety specfically posed by members of one's family or other social networks. This system is selectively sensitive to social signals of threat (e.g., dominant posturing), and is thought to be the system which helps organize children's behavioral responses in times of intense family conflict such as IPV (Davies & Woitach, 2008).

The emotional security hypothesis (Davies & Cummings,1994; Davies & Cummings, 1998) suggests that when threats to safety come from outside the family system, the attachment system will activate and children will demonstrate proximity promoting behaviors such as bids for soothing and contact with the caregiver.  However, when the threat is internal, children will develop a different repertoire of distress responses, including fear, vigilance, protest behaviors, flight/escape behaviors or camouflaging of emotion (e.g., avoidance, inhibiting overt affective displays) (Davies & Cummings, 1994; Davies & Woitach, 2008).  These types of behaviors can be viewed as an adaptive attempt to achieve physical and emotional security in the context of extreme family conflict or family violence.

There is some preliminary evidence that children's feelings of emotional security are jeaopardized in the context of IPV, thus shaping their behavioral repertoire in time of distress. For example, using prospective longitudinal research, Cummings, Schermerhorn, Davies, Goeke-Morey and Cummings (2006) found that exposure to parental conflict in 5 to 7 year-old children predicted less child emotional security one year later, as indicated by more child attempts at involvement and more emotional dysregulation during subsequent parental conflicts.  Davies and Cummings (2002) examined children's reports of their emotional and behavioral reactions to simulated parental conflict and found that children experienced anger when the conflict was characterized by only verbal hostility, while children predominately felt fear when the conflict involved physical aggression.  Research also suggests that children's emotional security guides their emotional and behavioral responses to interpersonal interactions. For example, Davies and Forman (2002) compared school-aged children's emotional and behavioral reactions to a conflictual exchange between their mother and a confederate and found three groups with different patterns of responding: (a) a balanced group, who exhibited a moderate degree of concern but expected a successful conflict resolution, (b) an insecure-preoccupied group who responded with heightened distress and avoidance, and (c) an insecure-dismissing group who reported low levels of distress but momentarily acted with distress or avoidance.  Notably, these groups of children were differentiated by the degree of conflict in their own parents' relatonships, with children from the proccupied and dismissing groups exposed to significantly greater parental conflict than those in the balanced group.  These findings suggest that exposure to extreme parental discord influences children's expectancies for how their caregivers will respond to conflict in general.  In turn, these expectancies shape children's own responses to witnessing conflict between individuals both within and outside of the family system.

In sum, IPV can exert deleterious psychological effects on both women and children alike by threatening the percieved and actual safety (physical and emotional) of the child, as well as the mother's perceived and actual ability to protect the child from such dangers.  On a cognitive level, child distress can activate the mother's traumatic memories and feelings associated with IPV, thus interfering with her ability to accurately decode the emotion and intent behind her children's behavior.  This is reflected in research finding that mothers exposed to IPV are more likely to be disengaged or distorted in how they represent their children internally.  In a parallel process, children exposed to IPV can come to view their mothers as emotionally and physically unavailable to them during times of distress, leaving them without a means to approproiately manage fear and arousal.  These cognitive changes then influence the affective communication strategies utilized by both mothers and children when distressing events occur.  The IPV-exposed mother, for example, will possibly withdraw from the child, react with hostility, become intrusive and controlling, or emotionally disorganized. The IPV-exposed child may similarly withdraw from the mother, hide his emotion, become hypervigalent, become affectively dyregulated, or even try to take on an adult role such caring for the mother or intervening in a parental conflict in an attempt to preserve a sense of emotional security. These types of affective responses to distress are particularly problematic for attunement processes for both the mother and the child, as will be discussed in the next chapter.

# Chapter 3: Affective Attunement

## *3.1 Defining the Construct and its Role in Self-Regulation*

The construct of affective attunement was first suggested by Stern (1974; 1987), and is defined as a dyadic phenomena characterized by the occasional partial matching of the mother's emotional behavior to that of her infant, with the sole purpose of communicating to the infant that she is sharing in his/her internal emotional experience. Stern considered attunement to be a component of mother-infant interaction that first emerges in typically developing infants between 9 and 12 months of age, though more recent research indicates that attunement can be observed as early as 2 months after birth (e.g., Jonsson et al., 2001). During infancy, Stern believed that babies took a passive role in attunement processes by simply expressing emotional or physical states, to which their caregivers responded with a mirroring of affect, thus creating the sensation of shared experience (Stern, 1974; 1987). While Stern thought that attunement was present in all mother-infant dyadic interactions, not all attunement was considered "healthy"; for adaptive and healthy attunement to be present, Stern argued that the mother's reflective behaviors should not be perfectly matched to the infant's in either timing, intensity or modality, but reflect only some degree of similarity. For example, according to Stern, when an infant cries it would not be appropriate for the mother to respond with crying as well, as this would be an example of exact mirroring instead of partial mirroring. Instead, the mother would utilize care-giving behaviors that were comforting and soothing, which would communicate shared affect of the infant's state through use of a different affective modality (Jonsson, Clinton, Fahrman, Mazzaglia, Novak & S□rhus, 2001). Though Stern asserted that mother-child attunement played an important role in children's socialization and emotional develop, the exact mechanism by which this occurred was not well understood. Additionally, Stern's definition of attunement was specific to the mother-infant dyad; he did not discuss whether or how attunement behaviors might change as children grew and developed.

Gergely and Watson (1996, 1999) expanded upon Stern's construct of affective attunement in infancy in their concept of a*ffective-reflective mirroring*, which is defined as the social reflection of the child's states and properties through facial expressions and vocalizations. Gergerly and Watson hypothesized that in infancy, the baby's primary "emotions" are pre-wired automatic responses to external stimuli and internal states such as pain, hunger, etc. Mirroring of affect by the care-giver provides the infant with reflections of these internal states, and over time this mirroring allows the infant to recognize these states and eventually develop the capacity to self-regulate these states. Both mothers and infants are thought to be highly motivated to engage in this mirroring during dyadic interaction, because the imperfect contingency of the mother's mirroring behavior is a rewarding process for an infant to engage in. When an infant detects a repeated stimulus response contingency between his/her cue and the maternal response through her mirroring behavior (e.g., infant cries, mother soothes using a calming voice) he/she experiences a sense of efficacy which in itself may be as soothing to the infant as the mother's calming techniques (Gergely & Watson, 1999). When operating effectively, these attunement processes help the infant learn to indentify and group together similar sets of stimulus-response mirroring contingencies, and eventually consolidate these into discrete types of feeling states, laying the foundation for emotional knowledge and early self-regulation, or the capacity to monitor, evaluate, and modify internal emotional states and emotional reactions in order to accomplish one's goals (Thompson, 1994). The mirroring aspect of affective attunement also aids in the social-emotional development of children by helping them to learn that the caregiver has a separate mind from his/her own. For infants, this is perhaps the very first step in the development of theory of mind (also called mentalization), or the ability to understand the emotional experience of others and to make predictions about their behaviors and intentions (Jonsson et al., 2001).

Though Gergely and Watson's ideas regarding the role of affective attunement in children's achievement of emotional self-regulation were largely theoretical, there is a growing body of research supporting the theory. For example, research by Feldman and Greenbaum (1997) found that attuned affect between mothers and their infants predicted children's ability to use internal state talk at 2 years of age, even after controlling for child IQ. The capacity for internal state talk suggests the ability to view feelings as coming from the self and the ability to reflect on affective experiences, thus the emergence of internal state talk is viewed as an important step in the development of theory of mind and empathy skills in young children. Lindsey, Cremeens, Colwell and Coldera (2009) found that in sample of mothers and their preschool-aged children, mother-child affective attunement predicted children's ability to demonstrate self-control and restraint during a lab task. In this study, children from dyads who demonstrated more attunement at 18 months of age were better able to delay playing with a desired toy at 3 years of age, an indicator of more developed self-regulation skills (Lindsey et al., 2009). Another study using a community sample of healthy mothers and normally developing infants found that affective attunement during a face-to-face unstructured play paradigm at both 3 and 9 months of age directly predicted children's self-regulation at age 6 and empathy at age 13 years (Feldman, 2007a).

While both Stern's and Gergely and Watson's theories suggest that attunement occurs during all kinds of emotionally-valenced interactions, attunement when the child is experiencing distress is thought to be particularly important for children's development self-regulation (Gianino & Tronick, 1992). This is because it is during these times that it is most challenging, but also most important, for the child to be able to manage their emotions and generate prosocial and adaptive behavioral responses that will result in their physical and/or emotional needs being met. Research supports this idea. For example, mothers who are unable to attune to their infants' expressions of negative affect tend to have insecurely attached babies, while mothers who are able to attune to a range of affect (including negative affect) in their infants tend to have more securely attached babies (Haft & Slade, 1989). Furthermore, research finds that children who have mothers that can successfully attune to their feelings of sadness and anger exhibit fewer externalizing behavior problems (suggesting they have better self-regulation skills) as compared to children whose mothers are less successful at attunement to negative affect (Johnson & Lieberman, 2007).

### *3.2 The Neurobiology of Affective Attunement*

Our understanding of the neurobiology underlying maternal care-giving behavior such as attunement comes from the study of both human and non-human mammal species. These studies indicate that the neurobiological circuitry of parenting consists of a complex network of connections among a number of areas of the brain. Swain and colleagues (2012) argue that these areas of the brain each belong to one of three subsystems that together form a larger system responsible for coordinating parental care-giving behavior. These include one subsystem that is responsible for detecting the need for care-giving in the child, one that creates a motivational state for providing care-giving, and a last subsystem that controls the performance of the care-giving response (Swain et al., 2012). Support for this idea comes from both animal research and human research using neuroimaging techniques.

Brain imaging studies suggest that the first subsystem, which functions to detect child behaviors that signal the need for parental responding, shares commonalities with some of the neural networks implicated in empathy, namely the cingulate and the insular cortices (Swain, 2011). For example, an FMRI study examining neural activity in adults when they received signals that a loved one was experiencing pain showed activation of the both the insula and the anterior cingulate (Singer et al., 2004). Similar activity was observed in the insula and cingulate, as well as the basal ganglia and amygdala, when first-time mothers in an fMRI study listened to the cries of their own babies compared to the cries of other non-biologically related infants (Swain et al., 2003).

Frith and Frith (2003) suggest that role of the insula and cingulate in parenting go beyond simple cue detection; they propose that these brain structures are critical in the capacity to understand the internal states of others, as well as the intentions of others' relational behavior. Similarly, it has been suggested that the maternal capacity for reflective functioning and face-to face mother-child regulatory processes are influenced by a mirror neuron system found in areas of the brain such as the ventral premotor cortex, inferior frontal gyrus and posterial parietal cortex, all of which interact with the limbic system through the anterior insula (Lenzi, Trentini, Pantano et al., 2009; Newman et al., 2011). fMRI studies lend some support for these hypotheses, indicating the anterior cingulate and anterior insula are both implicated in mothers' ability to read emotional cues which specifically signal another individual's physical or emotional suffering (e.g., Carr, Iacoboni, Dubeau, Mazziotta & Lenzi, 2003; Singer, Seymour, O’Doherty, Kaube, Dolan & Frith, 2004). Thus, these two areas of the brain, the cingulate and the insula, may be particularly critical for mother-child attunement.

Swain (2011) suggests that the insula and cingulate are implicated in parents’ capacity to detect children’s affective cues partly because these areas are involved in anxiety-driven behavior. However, neuroimaging research indicates that other areas of the brain are also involved in affective cue detection, as well. For example, studies using positron emission tomography (PET) suggest that the thalamus, hypothalamus and fusiform gyrus are implicated in transmitting information to the amygdala, which then perceives facial emotional cues that are read in order to generate a care-giving response (e.g., Davidson, Putnam & Larson, 2000). In an fMRI study where women were shown images of children’s faces, mothers showed increased activation in the dorsal and ventral striatum, thalamus, orbitofrontal cortex, and medial prefrontal cortex when looking at the faces of their own children compared to the faces of children that were not biologically related (Bartels & Zeki, 2000).

Similar areas of the brain have also been implicated in mothers’ responses to auditory affective cues, such as crying. For example, in several fMRI studies mothers showed greater activation of the thalamus, hypothalamus, amygdala, fusiform gyrus, as well as the anterior and posterior cingulate, when listening to their own infants’ cries versus a generated crying sound (e.g., Lorberbaum, Newman, Dubno, Horwitz, Nahas, Teneback et al., 1999; Lorberbaum, Newman, Horwitz, Dubno, Lydiard, Hamner, Bohning & George, 2002). Similarly, Swain, Leckman, Mayes, Feldman, Constable & Schultz (2004) found that as first time mothers learned to read and differentiate between the specific crying cues of their infants over time, they exhibited less activation of the cingulate and insula, and greater activation of the medial prefrontal cortical and hypothalamic regions. Together, these findings suggest that maternal affective cue reading can be localized in anxiety circuitry such as the cingulate and insulate for some parents, or in more regulatory circuitry such as the hypothalamus, thalamus and prefrontal cortex for other parents. Notably, the hypothalamus helps to regulate both the nervous and endocrine systems, and therefore is also a component of the subsystem that is responsible for the physical implementation of parenting behavior. Thus, although there are different aspects of parenting behavior (cue detection, motivation to respond, and performance of care-giving behavior), the neural subsystems which regulate these functions are somewhat overlapping.

The last of the subsystems identified by Swain involves areas of the brain that are involved in reward processing/motivation. For example, research has shown that the ventral tegmental area/substantia nigra, striatum and prefrontal cortex (implicated in the motivation system) work in combination with information processing parts of the brain such as the amygdala and hypothalamus, essentially hardwiring mothers to find reading the emotion cues of their children rewarding (Strathearn, Li, Fonagy & Montague, 2008). For example, in the previously mentioned study by Bartels and Zeki (2000), activation of the substantia nigra was observed when women viewed images of their own children's faces. Swain, Leckman, Mayes, Feldman and Schultz (2006) found that when parents viewed photographs of their own infants versus non-related infants, they demonstrated activation in the ventral tegmental area, superior temporal lobe, orbitofrontal cortex, frontal lobe and thalamus. It is thought that communication between these neural networks promotes enhanced processing of children's affective cues and thus more empathic responding in mothers (Newman, Harris & Allen, 2011).

Bartels and Zeki (2004) found that in addition to the motivation and reward system of the brain (e.g., the mesolimbic pathway, the ventral tegmental area, and the nucleus accumbens), there are complimentary cortical regions that also work to suppress negative emotion systems and social judgment (e.g., the middle prefrontal cortex, superior temporal sulcus, and paracingulate cortex) in order to produce positive feelings from the care-giver for the child and the desire to provide the care-giving response. Thus it seems that both the reward processing/motivational systems of the brain and the negative emotion system of the brain work in a delicate balance to result in care-giving behavior.

Animal studies of care-giving have also been useful for identifying neural systems underlying parenting behavior, and have yielded results to support findings from the human neuroimaging research. These studies further highlight the role of structures such as the hypothalamus, amygdala, cingulate, ventral tegmental area and prefrontal cortex in parental care-giving behavior (e.g., Afonso, Sison, Lovic & Fleming, 2007; Ferreira, Dahlöf & Hansen, 1987; Fleming, Miceli & Moretto, 1983; Hernández-González, Navarro-Meza, Prieto-Beracoechea & Guevara, 2005; Numan, Numan & English, 1993; Slotnick & Nigrosh, 1975). For example, it was found that female rats with lesions to the amygdala were more likely to exhibit care-giving behaviors toward pups by allowing the odor of the pups to be perceived as rewarding rather than aversive (Fleming et al., 1983; Numan, Numan & English, 1993). Animal research also indicates the importance of the cingulate and thalamus in care-giving behavior through their impact on selective attention and the organization of behavior. In particular, when female mice are induced with lesions to the cingulate they demonstrate slower pup retrieval (e.g., Slotnick & Nigrosh, 1975) and care-giving behavior that in general appears to be disorganized (Slotnick, 1967). Additionally, when lesioned in the cingulate and thalamus, female rat dams exhibit a significant decrease in maternal protective aggression (e.g., protecting the nest against a male intruder; Ferreira, Dahlöf & Hansen, 1987). Finally, the importance of the prefrontal cortex and ventral tegmental area for care-giving has also been illustrated through animal studies. For example, female rats lesioned in the medial prefrontal cortex engage in fewer retrieval and licking behaviors compared to non-lesioned dams (e.g., Afonso, et al., 2007). An EEG study of female rats indicate greater electrical activity in both the prefrontal cortex and ventral tegmental area when the rats engage in pup retrieval and licking, as compared to when they engage in other non-maternal behavior (Hernández-González et al., 2005). Functional mapping of rat dam brains suggests that these regions are more so activated for care-giving directly after birth; over time and with experience, activation of these areas becomes more integrated with other parenting circuitry such to the medial prefrontal cortex (Pereira & Morrell, 2011). Taken as whole, this body of research, in combination with the human neuroimaging findings summarized earlier, suggest that many of care-giving behaviors that constitute attunement behavior are localized in specific areas of the brain, such as but not limited to the cingulate, ventral tegmental area, prefrontal cortex, and limbic areas such as the amygdala.

Importantly, research has also demonstrated that exposure to adverse, stressful environments (as may be posed by IPV), is linked with alterations in the limbic system and other brain structures involved in sensitive care-giving. For example, neuroimaging studies show decreased prefrontal cortex volume in individuals who have experienced sexual abuse and who meet criteria for PTSD (Vermetten & Bremner, 2002). This decreased volume of the prefrontal cortex has implications for subsequent amygdala arousal and may thus be linked with decreased emotion regulation skills (Weiss, 2007), impeding on attunement processes. Similarly, many individuals who have experienced trauma such as IPV demonstrate hyper-responsivity of the amygdala likely due to dysfunction in the prefrontal cortex (Shin, Rauch & Pitman, 2006).

Research conducted with women exposed to IPV suggest that alterations in other specific brain structures related to mothers' capacity for attunement, such as the anterior cingulate, can be found, as well (e.g., Seedat, Videen, Kennedy & Stein, 2005). For example, Seedat and colleagues (2005) examined the neuronal integrity of the anterior cingulate in women exposed to IPV and found evidence of altered anterior cingulate functioning in the form of increased choline and decreased *N*-acetylaspartate in those women with IPV exposure and a PTSD diagnosis. This type of metabolite profile is suggestive of neuronal and axon loss, and is thought to be associated with difficulties in attending to external stimuli and a heightened fear response (Seedat et al., 2005), both of which have implications for which affective cues mothers respond to in their children and the quality of that response. In a similar study, Fonzo, Simmons, Thorp, Norman, Paulus and Stein (2010) utilized fMRI to compare the functioning of the anterior cingulate/medial prefrontal cortex, anterior insula and amygdala in IPV-exposed women with PTSD when conducting an emotional face-matching task. Results indicated that the IPV-exposed women exhibited greater activation of the anterior insula, amygdala, and anterior cingulate, but decreased connectivity between the three brain areas, compared to non-IPV-exposed controls. The difference in brain activation between the two groups of women was greater when the participants viewed faces displaying fearful and angry emotion, as opposed to positive emotions. The authors concluded that the IPV-exposed women demonstrated hyper-active cognitive –appraisal networks, particularly when detecting threat-related emotional cues (Fonzo et al., 2010). This may be particularly important in determining the sensitivity and accuracy with which mothers' detect their children emotional cues in times of distress.

There is also research to suggest linkages between the neurobiological systems involved in maternal care-giving behavior and the neurobiological systems involved in the physiological stress response. For example, some studies suggest that cortisol plays a mediating role in the functioning of the medial prefrontal cortex and the amygdala. A study by Veer, Oei, Spinhoven, van Buchem, Elzinga, and Rombouts (2012) found that higher levels of baseline cortisol were related to greater negative functional connectivity between the medial prefrontal cortex and the amygdala in adults, suggesting decreased ability to modulate amygdala activity using cognitive strategies such as reappraisal. This link between the two neural systems has also been found when examining mothers' responses to their children's affective cues. For example, Laurent, Stevens & Ablow (2011) found that mothers who exhibited a lower cortisol response to the sound of their infants' crying also exhibited increased activation of the limbic system and prefrontal cortical circuitry, allowing for more sensitive maternal responding. Given this impact of cortisol on the brain structures and neural circuitry underlying maternal care-giving, mother-child cortisol attunement may represent the neural process directly underlying disruptions in attunement behavior. In support of this idea, recent research has found that mother-infant dyads classified with resistant attachment have more attuned cortisol patterns than those from other classifications, and demonstrate an attuned profile of high cortisol reactivity with non-recovery (Laurent & Ablow, 2011).

In summary, there are specific systems of brain structures implicated in maternal care-giving behavior, and even more specifically associated with empathy, the capacity for reflective functioning and emotional cue detection, all of which contribute to mother-child attunement. Research indicates that both functional and structural alterations in some of the brain structures implicated in attunement can be observed in women exposed to IPV, suggesting that this is one pathway by which IPV may pose a threat to mother-child attunement, in addition to psychological processes. Finally, there is some evidence from both animal and human research to suggest that these neurobiological systems underpinning maternal care-giving behavior and the physiological stress response are closely associated with one another, suggesting that cortisol attunement may be the neural basis for difficulties with affective attunement in mothers and children.

### *3.3 Affective Attunement in the Preschool-Age Years*

While attunement is most often discussed as a dyadic process occurring in infancy, it is important to note that affective attunement continues to occur within the mother-child relationship throughout toddlerhood and early childhood, as well. As the infant develops into a child and acquires new and more complex communication skills such as language, the form of attunement behavior so too changes and becomes more nuanced. For example, research suggests that while maternal imitation and mirroring constitute the majority of mother-child attunement interactions during very early infancy (2 to 3 months of age), by the time their infants are 6 months of age mothers are modifying aspects of their mirrored emotional responses, (e.g., varying the intensity, rhythm and/or duration of vocalizations and facial expressions) when engaging in dyadic interactions (Jonsson et al., 2001). By toddlerhood and early childhood, affective attunement takes on even more new characteristics, and may look very similar to what is often referred to as *emotion coaching* (Johnson & Lieberman, 2007). Attunement at this age continues to be based in the parent's ability to read and reflect their child's emotional experiences in order to help them better understand what they are feeling and how to manage those feelings. Attuned parenting behavior at this time also continues to include some aspects of mirroring the child's affect, but is not limited to mirroring alone. For example, according to Johnson and Lieberman (2007) an attuned mother of a toddler or young child will communicate empathy for the child's emotion by also validating or showing respect for the child's experience, comforting and calming the child, talking about the child's emotions, and encouraging the child to talk with them about their emotions. Attunement to cues of negative emotion at this stage in child development continues to be particularly crucial for the development of self-regulation skills, as well (Johnson & Lieberman, 2007).

*3.4 Evaluating the Current Definitions of Affective Attunement*

Although the current definitions of early childhood affective attunement include some important elements, they are also somewhat problematic because they do not differentiate between the subcomponents of the construct well, making measurement difficult. Essentially, successful affective attunement in the preschool years is comprised of three components: (1) the cognitive/decoding component where the mother must first adequately perceive and interpret the child's affective experience, (2) the behavioral component where the mother must respond in a way that both communicates her empathic understanding *and* helps her child manage emotional arousal (and/or behavior), and (3) the child's ability to utilize the mother's responses to regulate his/her affect. Currently, most studies use the mother's behavioral response as an indicator of whether or not she has been successful at the cognitive component of attunement. This approach assumes that if a mother accurately decodes her child's emotional state, she will then utilize an appropriate behavioral response; however, this assumption is problematic. Take for example a child who is distressed and whose mother responds by making a joke. In one instance this may reflect that the mother has adequately perceived that her child is distressed, and thus she decided to use humor to try to help her child recover from the negative affect and shift to a positive state. In another instance this may reflect that the mother has misinterpreted the child's affective cues and has thus selected a behavioral response that is inappropriate and invalidating. This example illustrates how it is difficult to understand the true nature of these kinds of dyadic interactions if we do not assess both the mother's ability to mentalize, or perceive her child's emotional state, as well as her behavioral response.

Another problem with the current definition of affective attunement in preschool-age children is that it focuses heavily on the role of the mother and continues to view the child as a passive recipient of attunement processes. While this definition may be well-suited to attunement in infancy, this view of childhood attunement fails to take into account the enhanced verbal and communication skills that children have developed by toddlerhood. These increased cognitive capacities and verbal communication skills allow pre-school children to move from passive recipient of mother's affective mirroring to active participant in the attunement process. For example, at this age children can purposely initiate certain types of interactions in order to elicit desired affective responses from the mother (crying, reaching out to mother, making verbal bids for attention, etc). They can also actively limit their involvement with the caregiver when the care-giver response in not sufficiently helping them to modulate their emotional experiences (e.g., disengaging from the parent if the parent is too emotionally aroused, or not sufficiently engaged in the interaction). Additionally, because children are no longer passive recipients of affective attunement during toddlerhood and early childhood, an important aspect of attunement at this age is how well children are able to utilize the mother's response to help regulate his/her emotional state. For example, a child who withdraws his/her involvement or rejects the mother's comforting behaviors during a dyadic interaction may be refusing to utilize the mother's response to aid in self-regulation.

### 3.5 *The Effect of IPV on Affective Attunement*

The effect of IPV on both mothers' and children's experience of the care-giving relationship has significant implications for mother-child affective attunement. When mothers experience significant distortions in their interpretations of children's emotional behavior, their ability to attune to those emotions and respond appropriately may be compromised. Likewise, children who do not perceive their mothers as available to them for emotional support may disengage from their mothers or camouflage their emotional experiences. In both these ways mother-child attunement processes in IPV-exposed mother-child dyads may become disrupted. The next set of studies that will be reviewed are particularly important because similar to the aims of the current study, they sought to empirically examine the relationship between exposure to interpersonal trauma and mother-child affective attunement (or aspects of attunement such as sensitivity and mother-child synchrony). Consequently, the methodology and findings from these studies will be reviewed in greater detail in order to facilitate methodological critiques.

Though there is little empirical work directly examining affective attunement in IPV-exposed mother-child dyads, work by Casanueva, Martin, Runyan, Barth and Bradley (2008) has generated some preliminary evidence for impaired attunement in IPV-exposed populations. Casanueva and colleagues conducted a study of parenting behavior in women with a history of IPV exposure and women currently experiencing IPV, identified through a previously conducted national survey of child and adolescent well-being (NSCAW). The participants were selected to participate in this follow-up study if their children were under 10 years of age and still living in the home with the mother. Women's parenting behaviors were assessed with an observational rating system that included harsh parenting and parental sensitivity. The results indicated that women currently experiencing IPV engaged in less adaptive parenting, including less sensitive responding to their children as compared to women who had experienced IPV in the past, but were not currently enduring IPV.

Reid-Cunningham (2009) assessed the quality of the mother-child relationship in women with children between the ages of 4 and 6 years who had experienced interpersonal trauma (e.g., sexual assault or rape in adulthood). Controlling for mothers' exposure to non-interpersonal trauma, the authors found that mothers who had experienced adulthood sexual assault had more impaired relationships with their children as compared to mothers who had not. These relationships were significantly less reciprocal and attuned, and they were more likely to be characterized by conflict or disorganization. Although this study did not specifically distinguish between sexual assault by a stranger versus a romantic partner, because the experience of rape shares many features with IPV and is often part of the constellation of behaviors characterizing IPV, these results provide valuable insight into how interpersonal violence can impact mother-child interaction. Katz and Windecker-Nelson (2006) examined IPV-exposed mothers' capacity to engage in emotion coaching with their 4-5 year-old children. Although emotion coaching is not completely analogous to affective attunement, it shares many features with the construct, such as how well mothers are able to help regulate their children's affect. The results of this study indicated that children from families characterized by IPV and low maternal emotion coaching were more likely to exhibit aggression, withdrawal, anxiety or depression.

In the only published study examining mother-child affective attunement in an IPV-exposed sample, Johnson and Lieberman (2007) studied mothers' attunement to children's sad and angry emotions but found no association between mothers' experience of IPV and their attunement to children's negative emotions. However, these results must be interpreted with caution due to the fact that attunement was measured using maternal report, rather than *observed* *use* of attunement behavior when children were distressed. Finally, in the only other known study of affective attunement in IPV-exposed dyads (Field, Levedosky, Bogat & von Eye, unpublished manuscript), mothers and their 7-year old children were coded into categories of attunement (e.g., unattuned, or attuned for positive, neutral, negative or mixed emotion) which were examined for differences in the degree of IPV exposure. The results indicated that although all of the attunement groups had some degree of IPV exposure, dyads that were attuned for mixed affect (affect that shifted rapidly between intense positive and negative emotion) had experienced the highest and most severe levels of IPV. Children in this group also demonstrated significantly more antisocial and aggressive behavior than children from all other groups, including the unattuned group. These results suggest that while IPV-exposure may not prevent mothers and children from attuning, exposure to more severe IPV may result in affective attunement that may not be adaptive. For example these children may end up attuning to an affectively labile mother, resulting in a similarly dysregulated state in the child and an absence of skills for managing such emotions. In such cases, it may actually be more adaptive for the child to be unattuned from the mother.

As a whole, the results of the above studies provide some preliminary evidence for the influence of IPV on mother-child affective attunement. Together the studies suggest that interpersonal trauma, including but not limited to IPV, are associated with less sensitive parenting and more observed impairment to mother-child dyadic interaction (e.g., less reciprocal and less synchronous). Although sensitivity, reciprocity and synchrony can all be considered aspects of attunement, these results must be interpreted cautiously as these studies do not directly assess the complete construct of affective attunement.

In the two studies that specifically examined the impact of IPV on mother-child affective attunement, methodological limitations made it difficult to understand the true nature of the relationship between partner violence and attunement.  For example, one of these studies (Johnson & Lieberman, 2007) did not find a direct relationship between IPV and affective attunement, but the heavy reliance on maternal report in this study told us more about how IPV influences whether mothers *think* they can attune to their children, rather than how successfully they *do* attune to their children. The other study (Field, Levendosky, Bogat & von Eye) found an association between attunement such that greater IPV was associated with attunement for mixed affect.  However, because the mother-child interaction paradigm used in this study did not generate much negative emotion in the dyads, the presence and function of attuned negative affect could not be assessed by this study.  Additionally this study only defined affective attunement based on the degree of matched affective states in the mother and child.  Thus, there is still a need for research that examines the nature of mother-child attunement in IPV populations that (1) uses an appropriate paradigm to provoke negative affect, (2) that measures affective attunement using observational methods, and (3) that measures all aspects of the affective attunement construct (e.g., maternal decoding, maternal behavioral responses, and involvement of both mother and child in the interaction).

# Chapter 4: Physiological Stress Responses to IPV

## *4.1 The Physiological (Adrenocortical) Stress Response*

Stress can be defined from either a psychological or biological perspective; however the term stress itself is often viewed in terms of biological reactivity. For example, McEwen (1994) defines stress or trauma as the perceived threat to the self which results in arousal of the autonomic nervous system (as cited in Cicchetti & Walker, 2001). However, Cicchetti & Walker (2001) note that the very inclusion of the phrase "perceived threat" in this definition suggests that stress has an important psychological component where the individual's construal of an event alone may be sufficient for evoking a physiological response.

Most people experience some type of acute stressor or trauma in their lifetime. According to the National Comorbidity Survey, approximately 60.7 percent of American men (age 15-54 years) and 51.2 percent of American women have experienced at least one type of traumatic event, including witnessing someone being killed or badly injured, surviving a natural disaster, being involved in a life-threatening accident and/or combat (Kessler, Sonnega, Bromet, Hughes & Nelson, 1995). Many of these individuals experienced numerous traumas, for example, nearly 10 percent of men and 6 percent of women reported exposure to four or more traumas across the lifespan. Individuals can also be exposed to prolonged stressors, such as chronic poverty and environmental deprivation.

Research indicates that the experience of intense acute stress or prolonged stress can have observable effects on the physiological reactivity of both adults and children. Specifically, studies with both humans and animals suggest that prolonged exposure to stress/trauma can have measurable affects on the hypothalamic-pituitary-adrenal (HPA)-axis, the component of the neuroendocrine system that controls the release of stress hormones as well as many other bodily processes. The HPA-axis is receiving increasingly greater attention in stress/trauma research because it is responsible for the release of cortisol, a steroid hormone emitted by the adrenal gland, that when secreted for prolonged periods of time is thought to result in significant alterations in stress reactivity.

The release of cortisol into the system begins when the hypothalamus, the component of the brain that acts as a link between the nervous and endocrine systems, becomes activated. Activation of the hypothalamus results in the release of corticotropin-releasing hormone (CRH) (Bartels,Van den Berg, Sluyter, Boomsma & de Geus, 2003) and can be triggered by a variety of both internal and external cues, including circadian sleep/wake cycles, levels of various hormones in the blood, olfactory cues, sensory signals such as pain or blood pressure changes, and signals from other areas of the brain including the hippocampus and the amygdala (the part of the brain responsible for processing emotional events and signaling fear responses) (Stansbury & Gunnar, 1994). Release of CRH then initiates the release of adrenocorticotrophic hormone (ACTH) by the pituitary gland, which consequently stimulates secretion of cortisol from the outer cortex of the adrenal gland (Bartels et al., 2003). Thus, the release of cortisol is the result of a chain of events in the HPA-axis which are initiated by various internal biological rhythms or environmental stimuli, including stressful or fear-inducing events. Cortisol levels are thought to begin increasing approximately 10 to 15 minutes following the initiation of a stress response and peak within 20 to 30 minutes, but may take hours to be reabsorbed from the bloodstream (Stansbury & Gunnar, 1994).

The HPA-axis also functions as a negative feedback loop whereby the release of cortisol is both regulated by and serves to regulate HPA-axis activity through interactions between the hippocampus, hypothalamus and pituitary. After cortisol has been released into the blood stream, it eventually binds to receptors on the hypothalamus, triggering the down-regulation of the system and reducing the release of CRH and ACTH, helping to bring the system back to homeostasis. Thus cortisol also plays a critical role in helping the body to return to a balanced state following stressful events which trigger the “fight or flight” response of the sympathetic nervous system. Under typical conditions (e.g., when confronting a normative stressor such as navigating busy roadways to avoid a car accident) activation of the HPA-axis and release of cortisol helps the body to perform critical survival functions, including generating the energy needed for self-defense or to flee from a dangerous situation, and enhancing concentration and focus (Stansbury & Gunnar, 1994). Furthermore, glucocorticoids like cortisol raise sensory thresholds, allowing for more efficient interpretation of sensory stimuli (Stansbury & Gunnar, 1994). In resting states, the HPA-axis and cortisol also help to regulate the immune system, as well as influence learning, memory and emotional functioning. Despite these ways in which cortisol helps the body to achieve internal homeostasis, prolonged secretion of cortisol may result in substantial alterations to the functioning of the HPA-axis, or dysregulation of the HPA-axis. Dysregulation in this sense refers to impairment in one or more components within the regulatory system of the HPA-axis (e.g., impairment in the functioning of one of the organs itself, impairment in the functioning of the receptors, impairment in the feedback mechanism, etc.) and is thought to be related to a variety of negative physical outcomes (e.g., weakening of the immune system by inhibiting growth of T-cells, interfering with bone formation often resulting in osteoporosis). Prolonged cortisol secretion may also interfere with emotion regulation processes through its effects on specific brain organ functioning (e.g., hippocampal functioning), through alterations of brain structures, and even by altering the expression of genes which influence brain processes (Stansbury & Gunnar, 1994; Cicchetti & Walker, 2001).

Alterations in HPA-axis functioning can occur in the form of *hypercortisolism*, or an excess of cortisol in the system. Hypercortisolism is characterized by elevated basal levels of cortisol. In normally functioning individuals, cortisol levels follow a normal diurnal pattern, peaking not long after waking in the morning and then decreasing throughout the day. Individuals with hypercortisolism, however, do not experience this normative decrease and tend to have cortisol levels that remain heightened throughout the day (Gunnar & Vasquez, 2001). Hypercortisolism may be the result of impairment in the negative feedback loop of the HPA-axis, where cortisol reuptake does not occur as efficiently and impedes upon the down-regulation of the system. Hypercortisolism is often associated with disturbances of affect such as depression (Gunnar & Vasquez, 2001).

HPA-axis dysregulation can also occur in the form of *hypocortisolism*. Hypocortisolism is thought to occur as a result of repeated exposure to interpersonal trauma which influences the functioning of the HPA-axis though a *priming effect* (or blunting) of basal cortisol secretion levels (DeBellis, 2001; Heim, Ehlert, & Hellhammer, 2000). Two different explanations have been offered to account for how hypocortisolism may develop. The first suggests that increased activity in or number of glucocorticoid receptors results in greater feedback of cortisol and thus the suppression of the ACTH pituitary response (Gunnar & Vasquez, 2001). An alternative explanation suggests that frequent elevations in cortisol over time lead to the down-regulation of adrenal ACTH receptors as a protective mechanism (Gunnar & Vasquez, 2001). The ultimate result is that individuals exposed to trauma appear to have basal cortisol levels that are set lower than normal, as well as blunted cortisol reactivity when coping with acute stress. (Bevans, Cerbone & Overstreet, 2005). Children may be particularly vulnerable to the priming effect due to neural plasticity in the early years of development and the capacity for cortisol to bind to nuclear receptors and alter the expression of RNA, which codes for proteins that influence the growth, structure and function of neurons proteins (Bevans et al.,2005; Cicchetti & Walker, 2001).

### *4.2 Prenatal Programming of the HPA-axis*

An alternative way in which HPA-axis functioning can become altered is through prenatal programming. The prenatal programming hypothesis suggests that during pregnancy, the developing fetus is extremely sensitive to the characteristics of its in *utero* environment. Due to the extreme neural plasticity of the fetal brain, alterations in the mother's adrenocortical functioning could have potentially significant effects on the developing infant's brain organization and structure (Kaplan, Evans & Monk, 2008; Kapoor, Dunn , Kostaki , Andrews & Matthews, 2006). Women's experiences of extreme mood disturbance or chronic stress exposure during the prenatal period, for example, may result in HPA-axis dysregulation, exposing the developing fetus to increased levels of glucocorticoids through transplacental transfer and putting the development of the infant HPA-axis at risk. To illustrate, high fetal glucocorticoid concentrations have been linked with changes in central nervous system functioning, such as CNS sensitivity to dopamine and serotonin (which may increase risk for depression and other psychiatric disorders), delayed myelination of the central nervous system, and the down-regulation of IGF-II mRNA expression in the adrenal gland (Bertram & Hanson, 2002).

A vast amount of empirical support for the prenatal programming hypothesis has been generated using various animal models (e.g. Banjanin, Kapoor & Matthews, 2004; Lui, Li &

Matthews, 2001; Pryce, Aubert, Maier, Pearce & Fuchs, 2011; Weinstock, 2001; Welberg & Seckl, 2001). These studies indicate that when intrauterine levels of glucocorticoids are increased either by injection of synthetic glucocorticoid into the pregnant female (e.g., Banjanin, Kapoor & Matthews, 2004; Lui, Li & Matthews, 2001) or by creating environmental adversity for the pregnant mother (e.g., Weinstock, 2001; Welberg & Seckl, 2001) the offspring exhibit observable short-term and long-term differences in their HPA-axis structure and functioning. For example, guinea pigs whose mothers were treated with dexamethasone during pregnancy have an increased number of glucocorticoid receptors in the anterior pituitary (Banjanin et al., 2004). The offspring of rat dams who were injected with dexamethasone during pregnancy exhibit increased HPA-axis activity after birth, as well as increased "anxious" looking behaviors, when compared to offspring of non-treated control dams (Seckl, 2004). Finally, the offspring of female rhesus monkey who were exposed to stressful environments during pregnancy (e.g., repeated startle by the sounding of a horn) have offspring with higher basal cortisol, as well as higher cortisol levels following dexamethasone injection, compared to offspring of non-stressed control mothers (Pryce et al., 2011).

Despite the vast amount of evidence for prenatal programming in the animal literature, our understanding of whether prenatal programming occurs in humans is still somewhat limited. Some evidence for the effects of prenatal programming in humans comes from studies using low infant birth weight as an indicator of greater prenatal environmental stress exposure (e.g., Phillips & Jones, 2006; Seckl, 2004). These studies find that infants exposed to greater prenatal stress demonstrate exaggerated physiological responses following exposure to acute stress paradigms, as indicated by higher levels of post-task salivary cortisol and increased atrial pressure (e.g. Jones, Godfrey, Wood et al., 2005; Phillips & Jones, 2006). In a study examining women's subjective experiences of stress during pregnancy, mothers who reported high levels of prenatal stress and anxiety had children with high cortisol levels and a flattened diurnal pattern (van den Bergh, van Calster, Smits, van Huffel & Lagae, 2008). Finally, a study examining the effects of prenatal cortisol exposure (as measured from amniotic fluid) found that higher cortisol levels were associated with delayed cognitive development in 17-month old infants (Bergman, Sarkar, Glover & O'Connor, 2010). Together these results suggest that prenatal glucocorticoid exposure can have widespread effects on the developing fetus's brain, as well as specific effects on the structure and functioning of the HPA-axis after birth. Thus, when examining HPA-axis functioning in children, fetal exposure to environment stress should always be taken into account.

*4.3 Physiological Stress Responses to IPV*

Tjaden and Thoennes (2000) estimate that 1.3 million U.S. women are victims of male-perpetrated partner violence each year. Between 21 to 34% of all adult women report that they have been physically attacked by a romantic male partner at some point during their lifetime (Browne, 1993) and among populations such as impoverished and homeless women, as many as 61% report having experienced severe violence at the hand of a male partner (Browne & Bassuk, 1997). Prolonged exposure to IPV can result in chronic activation of the physiological stress response which ultimately can result in substantial and enduring alterations in the functioning of the HPA-axis for both women and children. Importantly, because IPV is a chronic interpersonal trauma, repeated IPV-exposure may exert a different effect on HPA-axis functioning as compared to other stressors that are acute and are not of an interpersonal nature (e.g., a car accident, natural disaster, etc.) (e.g., Meewisse, Reitsma, De Vries, Gersons & Olff, 2007).

Evidence for the effect of IPV on adrenocortical stress reactivity comes directly from studies examining IPV-exposure, as well as from studies examining the relationship between other types of interpersonal traumas (e.g., child maltreatment) and physiological stress reactivity.

### *4.4 Evidence from IPV Research*

Although there is a paucity of research regarding HPA-axis functioning in IPV-exposed adults, there are several studies which suggest an association between IPV and altered functioning of the HPA-axis (e.g., Griffin, Resick & Yehuda, 2005; Inslicht, Marmar, Neylan, Metzler, Hart et al., 2006; Pico-Alfonso, Garcia-Linares, Celda-Navarro, Herbert & Martinez, 2004; Schechter, Zeanah, Myers, Brunelli, Liebowitz et al., 2004; Seedat, Stein, Kennedy & Hauger, 2003). Of these studies, several found evidence of hypercortisolism in IPV-exposed women (Griffin et al., 2005; Inslicht, et al., 2006; Pico-Alfonso, et al., 2004), one found evidence suggesting a link between IPV and hypocortisolism (Seedat et al., 2003), and one produced findings to suggest that IPV-exposed women exhibit heterogeneous patterns of HPA-axis functioning (Schechter et al., 2004).

For example, Pico-Alfonso et al. (2004) found evidence of heightened circadian cortisol levels in a sample of women exposed to either physical or psychological IPV during their lifetime. When controlling for a number of confounds, women who had experienced physical IPV exhibited significantly higher levels of evening cortisol when compared to women from a non-abused control group (but their cortisol levels did not significantly differ from those in the psychological IPV group). Inslicht and colleagues (2006) also found that IPV-exposed women demonstrate HPA-axis alterations in the form of circadian cortisol levels. Specifically, their results indicated that women who had experienced IPV *and* had current or lifetime history of clinically significant PTSD had significantly elevated circadian cortisol levels when compared to women who had experienced IPV without PSTD. Griffin, Resick and Yehuda (2005) used a low-dose dexamethasone suppression test to determine whether dysfunction of the negative feedback inhibition loop of the HPA-axis was observed in women exposed to IPV with PTSD alone and/or IPV with comorbid PTSD and depression, as compared to non-exposed controls. Results of the study indicated that the control group had significantly higher baseline cortisol levels as compared to both groups of IPV survivors. These results were inconsistent with the authors' hypothesis that IPV-exposure would be associated with suppressed activation of the feedback loop, with IPV survivors exhibiting enhanced negative feedback inhibition. Notably, greater feedback inhibition (lower baseline cortisol) was observed in the IPV with PTSD only group, compared to the group with both PTSD and depression, suggesting a unique relationship between the presence of PTSD and blunting of the HPA-axis response (Griffin et al., 2005).

While the above studies all found evidence of hypercortisolism in trauma-exposed samples, other studies have generated evidence to suggest a different relationship between interpersonal trauma and HPA-axis activity. For example, utilizing plasma cortisol Seedat and colleagues (2003) found that women with a lifetime history of either physical or sexual IPV exposure had significantly lower levels of morning cortisol when compared to a non-IPV exposed control group. They also examined whether cortisol differences were observed based on whether IPV-exposed women met diagnosis for PTSD and found that PTSD was not associated with differences in cortisol functioning. Furthermore, cortisol levels were not significantly correlated with any PTSD symptoms. These results have to be considered with caution, however, because the group sizes utilized in this study were considerably small. Finally, in the only known study to-date to examine acute stress reactivity in an IPV-exposed population, cortisol was collected from women with histories of childhood maltreatment and/or adult IPV after engaging in stressful separation episodes with their children. Results indicated that IPV exposure was associated with lower baseline levels of cortisol prior to separation, while IPV exposure combined with greater PTSD symptomatology in the women was associated with a blunted cortisol reactivity in response to the stress task, as well (Schechter, Zeanah, Myers, Brunelli, Liebowitze, Marshall et al., 2004). Thus, while the results generally indicated that interpersonal trauma was associated with baseline levels of cortisol, changes in cortisol reactivity were specific to the presence or absence of PTSD symptoms.

In sum, women with a history of IPV exposure demonstrate heterogeneous patterns of HPA-axis dysregulation, with some exhibiting hypercortisolism and some exhibiting hypocortisolism depending on the whether there is a presence of PTSD diagnosis or symptomatology. Although the associations between HPA-axis functioning and PTSD are somewhat inconsistent, the findings from the studies with the strongest methodology (e.g.,Griffin et al., 2003; Schechter et al., 2004) suggest that the presence of IPV exposure in combination with PTSD symptoms resulting from that exposure is specifically associated with a blunting of the cortisol response. These results are consistent with the findings of a meta-analysis conducted by Miller, Chen and Zhou (2007), which suggest that differences in cortisol reactivity are associated with both the nature of the trauma, as well as the subjective degree to which the individual finds the traumatic event to be stressful. More specifically, results of this study indicate that exposure to chronic stress (including abuse/maltreatment, assault, unemployment and combat/war exposure) is associated with suppressed cortisol response following dexamethasone challenge, as well a flattened diurnal rhythm. This blunting of the HPA-axis response is specific to events that pose a threat to the physical integrity of the victim and that are perceived as uncontrollable in nature, characteristics both shared by IPV. Furthermore, although greater HPA-axis reactivity was observed when the traumatic event resulted in feelings of shame (as may occur with IPV), when the trauma was associated with a resulting PTSD diagnosis, blunting of the HPA-axis was again observed (Miller et al., 2007).

### *4.5 Evidence from Childhood Maltreatment Research*

IPV and childhood maltreatment share several common features, including the fact that they are both relational traumas where physical and/or psychological harm is afflicted at the hands of a trusted attachment figure. Due to the similar nature of these traumas, it is not unreasonable to believe that some of the physiological and psychological sequelae resulting from childhood maltreatment are also observed in individuals exposed to IPV. Some research on adult female survivors of childhood abuse has yielded findings that suggest interpersonal trauma is associated with dysregulated physiological stress reactivity due to alterations of the HPA-axis (e.g., Elzinga, Schmahl, Vermetten, van Dyck & Bremner, 2003; Meewisse et al., 2007). Elzinga et al. (2003) conducted a study of women with childhood histories of maltreatment, collecting salivary cortisol before and after the women recorded written vignettes of abuse events from their past. Results indicated that those women with trauma histories and current PTSD symptomatology had significantly higher (60% greater) baseline cortisol levels, significantly greater cortisol reactivity post-stressor, and significantly faster recovery than those women without PTSD symptoms. Brand and colleagues (2010) found a similar pattern of rapid cortisol recovery after women with childhood maltreatment histories watched their infants participate in an arm restraint task. The results of these two studies suggest a complex relationship between interpersonal trauma and cortisol functioning. More specifically, women with a maltreatment history did not exhibit a consistent pattern of either hypercortisolism (over-elevated cortisol levels) or hypocortisolism (suppressed cortisol levels), rather they showed a rapid and exaggerated increase in cortisol during arousing events, followed by a rapid decrease in cortisol functioning.

*4.6 Physiological Stress Responses to IPV in Children*

A majority of the research examining physiological stress reactivity in IPV-exposed children focuses on HPA-axis dysfunction as a mediating factor or risk factor for the development of affective /behavioral problems (e.g., El-Sheikh, 2005; El-Sheikh, Keller & Erath, 2007; El-Sheikh & Whitson, 2006). For example, both cross-sectional and longitudinal research has found that increased basal cortisol levels predict the development of children's internalizing and externalizing behavior problems following exposure to parents' marital conflict (El-Sheikh, Erath, Buckhalt, Granger & Mize, 2008; El-Sheikh, Keller & Erath, 2007). Little research has been done, however, to determine whether IPV exposure could result in long-term functional changes to the HPA-axis. Despite this paucity of research, findings from studies of children exposed to other types of relational traumas (e.g., child maltreatment) suggest that HPA-axis changes have been observed. For example, when compared to a group of non-abused controls, DeBellis, Chrousos, Dorn and colleagues (1994) found blunted ACTH responses and blunted free cortisol levels in a sample of sexually abused girls (7-15 years in age). Elevated levels of norepinephrine, epinephrine and dopamine (all catecholamines released during HPA-axis activity) have been found in urinary samples of abused children with PTSD (Nemeroff, 2004). Studies of women who experienced sexual abuse in childhood have indicated increased ACTH levels during stress-inducing lab tasks, as well as higher levels of 24-hour urinary cortisol

(Nemeroff, 2004). Finally, DeBellis, Baum, Birmaher, Keshavan, Eccard, Boring, Jenkins and Ryan (1999) conducted a cross-sectional study to examine circadian levels of urinary cortisol in violence-exposed children (including IPV, sexual and/or physical abuse) and found that regardless of clinical diagnosis, violence-exposed children had higher levels of urinary cortisol as compared to the non-exposed controls. Together these studies provide evidence of altered HPA-axis functioning, mainly in diurnal rhythm, among violence-exposed and maltreated children.

One of the only three studies to have tested for alterations in acute stress reactivity (challenged cortisol) in violence-exposed children was conducted by Ellis, Essex and Boyce (2005). This study did not focus on the effects of IPV alone, but rather examined the impact of exposure to cumulative stressors from the home environment on children's adrenocortical stress functioning. Indicators of home stress including but not limited to marital discord and IPV were measured during the first year of life, and again when children were 3 and 4 years of age. Generally, the results indicated that children from more stressful environments demonstrated higher anticipatory adrenocortical activation prior to performing a stressful task, and decreased activation following stress tasks, when compared to children from less stressful homes. The longitudinal approach of this study demonstrated that changes occured to the HPA-axis as a function of chronic environmental stress exposure (including as IPV exposure), as opposed to pre-existing qualities of children's HPA-axis functioning predisposing them to certain kinds of stressful experiences.

In the second study acute stress reactivity, Saltzman, Holden and Holahan (2005) assessed IPV-exposed and non-exposed children ranging between 5 and 13 years of age with clinically significant depression, anxiety and externalizing symptomatology. Both groups were assessed for a history of physical abuse as well as current PTSD symptoms. These are two important improvements upon earlier research in that they allowed the researchers to examine the unique effects of IPV by controlling for the presence of child abuse and PTSD symptomatology resulting from other non-interpersonal traumatic experiences. After controlling for parent education, family income and severity of abuse, Saltzman et al. found that IPV-exposed children, when compared to the non-exposed children, had higher baseline cortisol levels prior to participating in a stress task.

Finally, Hibel, Granger, Blair and Cox (2011) examined the impact of IPV on cortisol reactivity as children over time from infancy to toddlerhood. Children were exposed to in-lab stress tasks at age 7, 15, and 24 months. Mothers' care-giving behavior was coded for sensitivity at the same intervals. Although the results indicated that there was no main effect of IPV on children's cortisol reactivity or recovery, IPV-exposed and non-exposed children exhibited different trajectories of cortisol reactivity across the three time points. More specifically, although exposed and non-exposed children did not differ in their levels of cortisol reactivity at 7 months or 15 months, by 24 months of age, those children from IPV households demonstrated a significant increase in cortisol in response to the stress task, while non-exposed children did not. This difference appeared to be predicted by cumulative exposure to IPV, as opposed to current IPV-exposure. Furthermore, maternal sensitivity moderated the effects of IPV on cortisol reactivity, where only those IPV-exposed children with insensitive mothers demonstrated significant cortisol reactivity responses at age 24 months (Hibel et al., 2011).

In sum, there is evidence of altered HPA-axis functioning and adrenocortical reactivity in both women and children exposed to interpersonal traumas such as IPV. However, the nature of these alterations remains somewhat unclear as a result of the different methodologies used in the extant research. For example, some studies differentiate IPV-exposed women with childhood maltreatment histories from women without maltreatment histories; others do not make this distinction. Some studies differentiate IPV-exposed women with PTSD symptoms from IPV-exposed women with no history of PTSD; others do not make this distinction. Some studies utilize non-exposed comparison groups while others do not make this distinction either. Furthermore most studies examining the association between IPV and HPA-axis functioning in adult women focus predominately on alterations to circadian cortisol levels and patterns. There are few studies examining adrenocortical reactivity to acute stress in IPV-exposed women; additional information must be extrapolated from studies of women with childhood maltreatment exposure. The research regarding HPA-axis functioning in IPV exposed children is also diminished by the same methodological limitations. Despite these problems, some overarching patterns of HPA-axis functioning in IPV exposed populations have emerged. Three of the four studies examining challenged cortisol indicated that women exposed to interpersonal trauma have lower baseline cortisol levels. Two of these studies also indicated that these women exhibit exaggerated peak cortisol levels. With regard to children, four of the five studies examining challenged cortisol indicated higher baseline cortisol levels (anticipatory). One study found that IPV children exhibit lower peak responses compared to non-exposed children, while a second found greater reactivity in IPV-exposed children, but only in the context of insensitive parenting.

### *4.7 Physiological Attunement*

While individuals exhibit different patterns in their diurnal and challenged cortisol levels, recent research has indicated that these patterns of HPA-axis activity can become coordinated, or attuned, between individuals who share close relationships, such as a mother and her child.

Although there is a great deal of research on the characteristics of *affective* attunement in mother-child dyads, far less is known about physiological attunement between mothers and their children. Similar to the aim of the current study, the following studies sought to determine whether physiological attunement can be observed and measured in mother-child dyads. Additionally, some of these studies examine physiological attunement using similar methodology as is used in the current study. Thus, the methodology and findings of these studies will be reviewed in detail.

There is some evidence from normative samples that mothers and children experience synchrony in their biological rhythms before infants are even born. For example, by 33 weeks gestation, mothers and their unborn fetuses demonstrate attunement of their vagal tone and heart rhythms (Feldman, 2007a; Feldman 2007b), and within the first few weeks of birth many mother-child dyads exhibit attunement in their sleep-wake cycles (Feldman, 2006). It has been suggested that this early attunement of biological rhythms lays the foundation for later attunement of affect between mothers and their children by creating patterns of contingent responding (Feldman 2006; Feldman; 2007a; Feldman 2007b). For example, in a study of mothers and their infants during the still face paradigm, mothers and babies who engaged in more synchronous and attuned interactions during play (e.g., higher mirroring of the infants' affect by the mothers) also exhibited coordinated heart rates, with mothers' heart rates becoming more or less accelerated in response to their infants' heart rates (Feldman, 2006).

In another recent study conducted by Ham and Tronick (2009), mothers and their 18-month old infants demonstrated attuned biological and physiological rhythms both during and after the still-face paradigm, a laboratory task designed to induce distress in infants. In this study, mothers' and infants' respiratory sinus arrhythmia (RSA; an index of the parasympathetic system's influence on heart rate variability) was measured as an indicator of emotion regulation during the interactions, with lower RSA indicating a stronger stress response (Ham & Tronick, 2009). Skin conductance, which measures the electrical conductivity of the skin and is used as an index of the sympathetic nervous system "fight or flight" response, was also measured in the dyads. Concordance of skin conductance between the mothers and infants was of particular interest in this study, as previous studies have shown that this type of concordance is directly associated with the capacity for empathy in the dyad (Marci, Ham, Moran & Orr, 2007, as cited in Ham & Tronick, 2009). Results of the study indicated that during the still-face paradigm the dyads demonstrated concordance of skin conductance. Furthermore, greater mother-infant concordance in skin conductance was associated with increased displays of infant negative affect, suggesting that mothers were "empathically sharing" in the infants' physiological experience of stress. During the reunion episode of the still-face paradigm, mothers and infants continued to exhibit concordance in skin conductance; however greater concordance at this time was associated with greater attunement in the dyad (e.g., more calming and soothing behaviors in response to infant distress). Furthermore, in response to this physiological and affective attunement during the reunion episode, the infants demonstrated an increase in RSA, suggesting that they were using the affective attunement to help regulate their physiological state (Ham & Tronick, 2009).

More recently research has also suggested that mothers and children also experience attunement at the physiological level. For example, in a low-risk community sample of mothers and their children aged two to four years, dyads demonstrated attunement of salivary cortisol levels following participation in lab stress task (Sethre-Hofstad, Stansbury & Rice, 2002). Cortisol levels were measured at baseline before the children performed a stressful beam task while mothers watched from another room, and peak cortisol levels were measure 30 minutes later. Mothers who were better able to assess their children's emotional experience of the beam task exhibited changes in cortisol from baseline to peak that were more highly correlated with their children's cortisol changes, as compared to less sensitive mothers (Sethre-Hofstand, Stansbury & Rice, 2002). In a similar study by van Bakel and Riksen-Walraven (2008) mothers and their 1-year old infants exhibited attunement of physiological stress reactivity after engaging in stressful lab task involving a confrontation with a stranger and a novel moving robot. In this study, salivary cortisol was collected from mothers and their 15-month-old infants prior to and 20 minutes following the lab tasks and analyzed for concordance in the amount of change from baseline to the peak stress response. In a separate lab interaction task, mothers' behaviors were also coded for sensitivity of responding. Again, more attuned cortisol responses were observed in dyads with mothers who were rated more highly on sensitivity and attuned care-giving.

Attunement of diurnal (circadian) cortisol levels is also observed in mothers and their children. For example, in a study of middle-income families with adolescent children, mothers and their children exhibit attunement of salivary cortisol both in times of heightened conflict and stress, and also generally throughout the day (Papp, Pendry & Adam, 2009). In this study, the families provided seven samples of salivary cortisol across the day, one at waking, one 30 minutes post waking (when a peak is typically experienced), one before bed, and 4 that occurred throughout the day based on a timed pager system. The families also kept diaries of events and interactions they experienced throughout the day. A first pager signal was used to alert the participants to record their current experiences in their diary, and then a second pager signal sounded 30 minutes after the first to signal that cortisol collection should occur. In this way, cortisol levels could be examined in response to specific momentary experiences, as well as based on diurnal pattern. The results of the study indicated that a subgroup of the sample exhibited a strong positive association between mothers and adolescent cortisol levels even after controlling for diurnal rhythm of cortisol, within-day error, demographic variables, and health. Mother-child dyads who exhibited attuned diurnal cortisol values were distinguished from those who were unattuned by amount of time spent together (attuned dyads spent more time together), engagement in shared activities (attuned dyads spent more time doing shared-activities), and parental monitoring (mothers from attuned dyads engaged in more monitoring). Finally, the diary recordings indicated that mothers and their adolescents tended to exhibit more attuned levels of cortisol during moments when either one or both members of the dyad was exhibiting negative affect (Papp, Pendry & Adam, 2009).

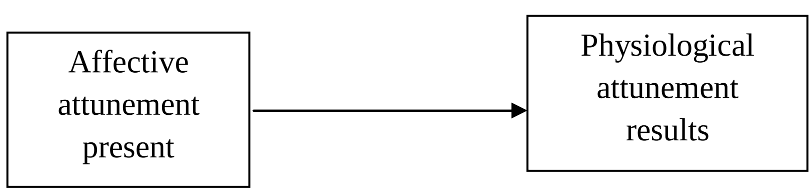
*4.8 The Link between Physiological Attunement and Affective Attunement*

As indicated above, research on physiological attunement in isolation from affective and behavioral attunement is rare. Feldman (2006) observed that coordination in mothers' and infants' heart rates corresponded to the degree of affective attunement during their play. Ham and Tronick (2009) found that attuned skin conductance was related to attuned maternal responding during the reunion episode of the still-face paradigm. Sethre-Hofstad and colleagues (2002), as well as van Bakel and Riksen-Walraven (2008) found that mothers who were more attuned to their children's affect during play and stressful lab tasks showed attuned cortisol reactivity with their children in response to acute stressors. Even adolescents and their parents exhibited coordinated stress reactivity when both members of the dyad shared in negative affect during interpersonal exchanges (Papp, Pendry & Adam, 2009).

Though there appears to be some kind of association between attunement at the physiological and affective levels, to date this association is not well understood. Sethre - Hofstad et al. (2002) have offered a few interpretations of their finding that mother and child cortisol changes are more strongly correlated in dyads with highly sensitive and attuned mothers. The first of these is that adrenocortical attunement shares a direct one-to-one association with affective attunement, and that lack of physiological attunement in mothers may be indicative of a failure to accurately detect or interpret infant cues. Put differently, the presence of affective attunement is defined by an ability to detect infant cues, and in turn this results in the presence of adrenocortical attunement (one will not occur without the other). The implication of this is that adrenocortical attunement results if and only if affective attunement occurs, and that affective attunement is both necessary and sufficient for adrenocortical attunement to occur. This idea is illustrated below:

Figure 4.1

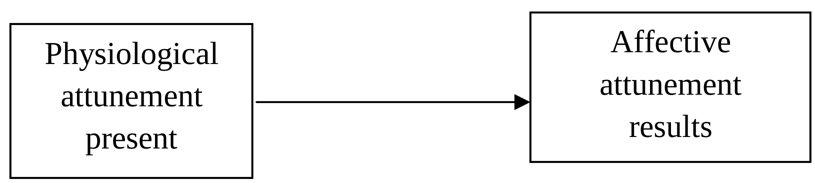
*Physiological Attunement Resulting from Affective Attunement*




An alternative explanation is that although almost all mothers are able to detect obvious emotional cues in their children (e.g., crying, asking for help from the mother), only the mothers who experience physiological attunement to their child are able to pick up on their children's more subtle cues, thus allowing them to become truly affectively/behaviorally attuned to their children. This explanation thus implies that physiological attunement temporally precedes the occurrence of affective attunement, and that physiological attunement is both necessary and sufficient in order for affective attunement to occur. This idea is illustrated below:

Figure 4.2

*Affective Attunement Resulting from Physiological Attunement*




A caveat to this idea is that there may be a point at which sharing the child's physiological state becomes maladaptive in that it hinders, rather than helps the mother respond more sensitively and appropriately to her child. One could imagine this happening if a child is experiencing a state of distress and his/her mother becomes so distressed as a result that she is unable to help calm or soothe the child. Similarly, one could imagine this being the case if a child who is initially calm becomes physiologically attuned to a dysregulated mother, resulting in both dyad members entering into an affectively dysregulated state.

A third possible explanation is that the two phenomena occur as a parallel process, where the presence or absence of physiological attunement can be considered just a lower-level indicator (physiological attunement) of a higher-level process (affective attunement), or vice versa. In this sense, physiological attunement and affective attunement may be correlated, but the two phenomena do not share any kind of causal relationship. A final explanation of the relationship between physiological attunement and affective attunement is that the associations between these two phenomena that have been observed in prior research are spurious and may be better explained by some third variable, for example heredity (Sethre-Hofstad et al., 2002).

Methodological limitations in the previous research have prevented us from elucidating the true nature of the relationship between physiological and affective attunement. The first of these is that most research on this topic tends to be cross-sectional in nature, making it difficult to determine if the associations between the two types of attunement are meaningful or better accounted for by some other variable. The heavy reliance on correlation in previous studies also means that we have not yet been able to assess for the presence of a causal relationship between the two phenomena. Furthermore, many of these studies only examined *aspects* of affective attunement, rather than the full construct. For example, the Sethre-Hofstad et al. study, which examined attunement between mothers and pre-school age children, defined attunement as the mothers' ability accurately perceive the child's needs, and to respond promptly, contingently, and accurately to the children's cues. Although this represents one of the better attempts at capturing the construct, absent from this definition and measurement of affective attunement is the use of the dyadic interaction by both partners to facilitate child-self regulation. A final limitation of the current research examining the intersection of physiological and affective is attunement is that the reliance on small homogenous samples (e.g., ranging from 64-83) of predominately Caucasian, well-educated parents and non-violence exposed children. Thus, the findings cannot be generalized to other populations, such as high-risk, IPV-exposed samples.

## Chapter 5: The Intersection between IPV, Physiological Attunement, Affective Attunement and Child Outcomes

Very little is known about the association between physiological attunement and affective attunement in normative populations, and even less is known about the relationship between these phenomenon in IPV-exposed populations. However, it is important to understand the intersection between these variables in mothers and children exposed to IPV, given (1) the high rates of externalizing and social-emotional problems observed in IPV-exposed children, (2) the role of attunement in the development of child self-regulation and social-emotional development, (3) the previously identified influence of IPV on mother-child interactional processes, (4) the known effects of IPV on the physiological stress response system in both mothers and children, and (5) the possible associations between attuned physiological stress responses and mother-child affective attunement that have been identified in recent research. To date, only one known study has examined the role of affective attunement in IPV-exposed children's externalizing behavior problems (Johnson & Lieberman, 2007) and only one known study has examined the relationship between physiological attunement and affective attunement in an IPV-exposed sample (Hibel, Granger, Blair & Cox, 2009). These findings are reviewed below.

As reviewed earlier, Johnson and Lieberman (2007) examined whether mothers' attunement to their preschool-age children's feelings of anger and sadness mediated the association between IPV exposure and children's internalizing and externalizing behavior problems. Mothers reported on both their exposure to IPV in the last year, as well as children internalizing and externalizing symptoms using the Child Behavior Checklist. Mothers' attunement to children's negative emotions was coded based on maternal responses to a meta-emotion interview, which evaluated the amount of emphasis the women placed on their children's emotions, the amount of respect communicated for their children's emotions, and their ability to comfort their children when experiencing negative affect. The results of the study found that IPV was not related to mothers' scores of affective attunement, but that mothers' difficulty attuning to their children's experiences of sadness and anger made a unique contribution to children's externalizing behavior problems. When the dyads were categorized dichotomously as attuned or unattuned for anger, nearly half of the children from the unattuned dyads exhibited clinically significant externalizing problems, compared to only 30% of the children from attuned dyads. Similarly, when the dyads were categorized for attunement of sadness, nearly half of the children from the unattuned dyads demonstrated clinically significant internalizing problems, as compared to only 12% of the children from attuned dyads.

Although maternal IPV exposure was not significantly related to affective attunement in this study, there are several methodological limitations that might account for this null finding. First, the sample utilized in this study was relatively small (n = 30), which may have been insufficient power for detecting associations between IPV and attunement. Secondly, the measure used to assess maternal exposure to IPV assessed only events from the past year, and was administered as a structured interview with the interviewer reading the violence items out loud and the mothers responding verbally in turn. Because of this approach, mothers may not have felt comfortable reporting on the full extent of their IPV experiences, possibly limiting the quality of this data. Finally, all of the variables in this study were assessed using maternal report. This is particularly problematic for the measurement of affective attunement, as the meta-emotion interview can only present a picture of how mothers think and talk about their children's emotions, rather than how they actually respond in the moment to negative affect. Without behavioral observations of mother-child interactions it is not possible to know how attuned IPV-exposed mothers actually are to their children's affective experiences.

Hibel and colleagues (2009) conducted the only study to-date examining the association between physiological attunement and affective attunement in a high-risk community sample which included IPV-exposed mother-infant dyads. In this study mothers reported on their IPV-exposure in the last year and the dyads were dichotomously classified as non-exposed or IPV-exposed (based on the endorsement of at least one IPV item). Mother-infant affective attunement was assessed based on observations made during a free-play activity and included the degree of sensitivity, detachment, positive regard, negative regard, and intrusiveness exhibited by the mother during the interaction. These codes were factor analyzed to produce three dimensions of attunement: maternal positive engagement, maternal intrusion, and negative regard. Physiological stress reactivity in both the mothers and infants was assessed using an arm restraint procedure during which the infants were presented with an appealing toy but were physically restrained from playing with it while the mother watched from behind without being able to intervene. Cortisol samples were collected before the task and then 20 and 40 minutes post-task.

The results of this study indicated that while the mothers generally exhibited a decrease in cortisol levels from baseline to peak reactivity and infants generally exhibited an increase in cortisol from baseline to peak, differences emerged when comparing the dyads based on IPV-exposure. More specifically, mothers and infants exposed to IPV demonstrated positively correlated changes in their cortisol levels from pre-task to post-task, while non-exposed dyads did not have correlated cortisol reactivity. Mothers exposed to IPV were also less likely than non-exposed mothers to be behaviorally attuned to their infants during the free-play, as indicated by higher scores of negative regard. Mothers who displayed high levels of negative regard also tended to have more correlated physiological stress responses with their infants. However within-group analysis of the IPV-exposed dyads provided no evidence to suggest that affective attunement buffered against (or that a lack of affective attunement intensified) mother or infant physiological stress reactivity, or the degree of concordance in that stress reactivity.

It is somewhat surprising that affective attunement did not moderate the association between IPV and adrenocortical attunement in this study, given that the rates of coordinated adrenocortical reactivity and affective attunement both differed for the IPV and non-IPV-exposed groups. One reason this might be is that physiological stress reactivity and parenting behavior were measured at temporally different times, with the free-play taking place in the lab before the arm-restraint task was conducted. This is an important methodological limitation because this approach prohibits us from examining how adrenocortical reactivity affects parent-child attunement behaviors (or vice versa) that occur directly during or immediately after the acute stressor is experienced. Furthermore, it is somewhat problematic that maternal parenting behaviors were examined during a free-play paradigm in this study. Though free-play tasks are designed to illicit “natural” interactions between the mother and her child, they are mostly characterized by positive affect for both dyad members. Given that maternal attunement to *negative emotion* is thought to be most influential in children’s development of emotional regulation, it is of critical importance to observe attunement behaviors during instances in which children are likely to experience distress, rather than during enjoyable interactions like a free-play episode.

An additional consideration when interpreting the results of the Hibel et al. study is that because this work was conducted using mother-infant dyads (infants’ mean age was 7 months, with a range in age from 5.0 to 13.4 months), the results cannot be generalized to older children, as research suggests that the HPA-axis functions differently at different stages of development (Kudielka, Hellhammer & Wüst, 2009). For example, newborns do not demonstrate typical daytime diurnal patterns in their cortisol, exhibiting two peaks approximately 12 hours apart in time regardless of time of day (as opposed to one morning peak that is observed in young children, adolescents and adults) (Gunnar & Quevedo, 2007). Furthermore, the continual decrease in cortisol that occurs throughout the day in children and adults is not observed in young children until they cease daytime napping (Gunnar & Quevedo, 2007). This implies that we can draw only limited conclusions regarding HPA-axis functioning when cortisol is measured during infancy.

In addition to the limited generalizability of studies using infant cortisol, there are several additional drawbacks to measuring cortisol in infants as opposed to children or adults. First, research indicates that there are differences in the types of events and situations that can provoke HPA-axis stress responses across the developmental span. For example, early in the first year of life the infant HPA-axis is highly reactive, and cortisol changes can be observed in response to even mild stressors such as the approach of a stranger or a medical exam. However, by the end of the first year of life it becomes increasingly more difficult to invoke such HPA-axis stress responses in infants (Gunnar & Quevedo, 2007). This is in part due to normal developmental changes that are occurring in the HPA-axis during this time, for example more efficient negative feedback in the overall system and decreased sensitivity to ACTH in the adrenal cortex (Gunnar, Brodersen, Krueger & Rigatuso, 1996; Gunnar & Quevedo, 2007).

Second, there are some logistical complications when measuring infant cortisol and physiological reactivity that may be avoided by utilizing child data. For example, large enough saliva samples can be difficult to obtain with young infants and the collection procedure itself can be invasive enough to induce a stress response in infants. Furthermore, in infants younger than one year of age it can be difficult to obtain reliable measures of HPA-axis activity, an issue which does not appear to be fully resolved even when multiple samples are aggregated over time (de Weerth & van Geert, 2002). This is likely due to the fact that infant cortisol measures can be easily influenced by a number of variables including the timing of their sleeping and feeding and small variations in physical health, which may result in high levels of intra-individual variability in day-to-day measurements of cortisol. These findings call into question the reliability of infant cortisol measures, as well as the reliability of any relationships found between infant cortisol measures and other variables of interest (de Weerth & van Geert, 2002). For these reasons there may be some advantage to using childhood cortisol measurements as an indication of overall HPA-axis functioning during the early years of life, as opposed samples collected during infancy.

A final limitation of both the Hibel et al. and Johnson and Lieberman studies is the way in which IPV exposure was defined and measured. For example, both studies only measured the effects of physical and sexual partner violence, but did not include any assessment of psychological aggression in their measurement of IPV. This methodological issue is particularly problematic, given the known impact of psychological aggression on both women's mental health (e.g., Johnson & Leone, 2005; Próspero, 2008) and child's emotional functioning (e.g., Huston et al., 2010; Martinez-Torteya et al., 2009). It is also problematic that both studies only accounted for IPV-exposure occurring in the last year, rather than throughout the child's lifetime, including IPV occurring during the prenatal period. It is essential that we account for the total amount of IPV that children have been exposed to, as research has indicated a dose effect of IPV, where greater cumulative amounts of exposure and more severe exposure are associated with more exaggerated adrenocortical reactivity (e.g., Schechter et al., 2004). Furthermore, it is necessary to account for IPV experienced during pregnancy when considering the full extent of children's exposure to IPV, as we know that the effects of IPV on HPA-axis functioning can begin in the uterine environment even before the child is born (Kaplan, Evans & Monk, 2008; Kapoor, Dunn , Kostaki , Andrews & Matthews, 2006).

In sum, there is some evidence from normative non-violence-exposed samples to suggest that physiological attunement and affective attunement share a significant positive association. There is also some evidence that both physiological and affective attunement may function differently in IPV-exposed and non-violence-exposed mother-child dyads. Finally, there is evidence to suggest that deficits in mother-child attunement, particularly around attunement for negative emotions, contribute significantly to children's behavioral problems. Together these findings suggest that the pathway between IPV exposure and child behavior problems may be explained by the effects of IPV on attunement, at both the physiological and affective levels.

In the current study it is argued that IPV affects physiological and affective attunement in mother-child dyads through complimentary physiological and psychological pathways. At the psychological level IPV can negatively impact the quality of mother-child interactions through its effects on the care-giving system. A woman with a young child is still developing her identity as a mother, and as she opens herself psychologically to take on this role, she also becomes vulnerable to the influence of other significant relationships on her sense of self. The dynamics of power and control in an IPV relationship can significantly undermine a woman's sense of competence as a protector for her child, both in a physical and emotional sense. Feeling ineffective, some IPV-exposed mothers may abdicate the role of care-giver when their children are in distress, either withdrawing from the dyadic interaction or becoming so affectively disorganized they cannot provide an appropriate response to their children. Thus IPV exposed mothers may either simply fail to affectively attune to their children or may misread affective cues and respond in ways that do not serve to help the child feel supported or helped. IPV-exposed children may further contribute to problems with affective attunement, as these children are not likely to experience emotional security within the mother-child dyad. Children who do not experience emotional security within the care-giving relationship may withdraw from dyadic interactions, camouflage their affect, or send disorganized emotional cues, making it difficult for their mothers to decode their emotional state and respond accordingly.

At the physiological level, IPV-exposure is associated with alterations in HPA-axis functioning for mothers and children, such that both are likely to exhibit exaggerated physiological responses to acute stress. Although in non-IPV exposed samples attuned physiological responses might allow mothers to more fully “share” in their children’s emotional states and be more affectively attuned, this may not be true for dyads who exhibit altered stress reactivity. For these dyads, attuned physiological stress responses may actually interfere with successful affective attunement. For example, attuning to a dyadic partner who is experiencing a dysregulated adrenocortical response may only serve to dysregulate (or further dysregulate) the other individual, making it difficult for them to effectively cope with and recover from the stressor. Some IPV-exposed mothers exhibit blunted adrenocortical reactivity instead of exaggerated reactivity. These mothers may be less affectively aroused or engaged, thus they may have a particularly hard time “sharing” in their children’s affective state and be less likely to respond to their children in an affectively attuned manner.

When children experience extreme distress and mother-child attunement does not successfully occur, they are left without anyone to aid them in the regulation of their emotional state. Over time, successive breakdowns in attunement prevent these children from developing effective strategies for managing extreme affective states. Such children are likely to exhibit deficits in self-regulation which manifest in internalizing and externalizing problems. In this way, both physiological and affective attunement in IPV-exposed dyads may contribute to the development of child behavior problems.

# Chapter 6: The Current Study

Children exposed to IPV demonstrate deficits in emotion regulation, as indicated by higher levels of internalizing, externalizing, and social problems when compared to children from non-IPV households. One reason that IPV-exposed children may exhibit such deficits is because IPV can negatively impact mother-child relational processes that are critical to the development of emotion self-regulation skills, namely affective attunement. When affective attunement occurs under normative conditions (e.g., in the absence of IPV), it can help children to recognize and modulate emotions effectively; however in the context of IPV attunement processes may be inhibited or altered. Although there are a number of factors that can influence mother-child affective attunement, IPV represents a significant threat to affective attunement because it is characterized by a chronic pattern of psychological and physical aggression from a trusted relational partner. Because IPV includes both these aspects of threat and betrayal within the context of what is supposed to be a trusting and safe interpersonal relationship, IPV can result in enduring changes in how women perceive themselves and others with whom they share significant relationships. For example, women exposed to IPV may have difficulty accurately interpreting the social and emotional cues of others, and may make interpretations or attributions about others' behaviors that are distorted or inaccurate. This can generalize to women's relationships with their own children, influencing how mothers interpret and respond to their children's affective cues, thus impeding upon their ability to engage in affective attunement with their child.

Further, IPV is known to result in significant and persistent changes to both mothers' and children's physiological (adrenocortical) responses to acute stress. In normative populations, when mother-child physiological stress responses are coordinated, mothers tend to be more attuned to their children's subtle emotional cues, thus allowing them to respond in a more affectively attuned manner. However, it is unclear whether attuned adrenocortical responses confer the same benefit in terms of enhanced mother-child affective attunement for IPV-exposed populations. Previous research on this topic is limited by methodology, including problems with the definition and measurement of affective attunement (e.g., lack of observational assessment), and insufficient assessment of children's IPV exposure (e.g., assessing exposure in the last year rather than lifetime exposure, or neglecting to measure psychological IPV).

An additional limitation in previous studies on this topic is the reliance on variable-centered analytic strategies (e.g., Johnson & Lieberman), and group comparisons based solely on a dichotomous coding for presence or absence of IPV-exposure. This is problematic because individuals' experiences of IPV can vary significantly in nature, and use of such analytic approaches can obscure important individual variations that result from the natural heterogeneity of IPV-exposed populations. The use of both variable-centered and person-centered approaches in research of this nature is critical, as each allows us to draw different conclusions from the data. For example, variable-centered approaches allow us to estimate the strengths of effects attributed to predictor variables of interest (e.g., IPV and attunement) (Bergman, Magnusson & El-Khouri, 2003; Laursen & Hoff, 2006). However, because this approach involves combining subjects into groups, this approach can also have the disadvantage of misrepresenting or obscuring certain relationships that exist between the variables of interest. Conversely, the person-centered makes the assumption that individuals are distinct from one another and as a result, relatively similar subgroups can be identified within samples using predictable patterns occurring across the dependent and independent variables (Bogat, Levendosky, & von Eye, 2005). Based on this, the person-centered approaches allow us to empirically derive within-group patterns of individuals (or subgroups of individuals) that exist within the overall data (von Eye & Bergman, 2003). These patterns or subgroups are identified based on shared attributes, or specific relations among shared attributes. Person-centered approaches are particularly appropriate to use when examining individual differences in patterns of development and associations among particular variables of interest (Bergman et al., 2003; Laursen & Hoff, 2006). Given the heterogeneity of cortisol responses observed in trauma exposed women (e.g., Griffin, Resick & Yehuda, 2005; Inslicht, Marmar, Neylan, Metzler, Hart et al., 2006; Pico-Alfonso, Garcia-Linares, Celda-Navarro, Herbert & Martinez, 2004; Schechter, Zeanah, Myers, Brunelli, Liebowitz et al., 2004; Seedat, Stein, Kennedy & Hauger, 2003), and children (e.g,, Ellis, Essex & Boyce, 2005; Hibel, Granger, Blair & Cox, 2011; Saltzman, Holden & Holahan, 2005), as well as the heterogeneity of women and children's IPBV exposure in the form of severity, chronicity, and timing (e.g. prenatal versus postnatal; Bogat, Levendosky & von Eye, 2005) use of a person-centered analytic approach in the current study will allow for the identification of homogeneous profiles of HPA-axis dysregulation that may each be associated with different patterns of individual risk in terms of IPV-exposure and different patterns of outcomes in terms of attunement and behavior problems (von Eye & Bergman, 2003).

The current study seeks to address five basic science questions regarding the nature of attunement in IPV-exposed mother-child dyads and its contribution to emotion regulation problems in IPV-exposed children:

1. Is physiological dysregulation observed in IPV-exposed mothers and children in response to acute stress (e.g., challenged cortisol levels in response to a standardized lab stress task)?

2. Is affective dysregulation observed in IPV-exposed mothers and children in response to acute stress?
3. How is IPV related to mother-child attunement both at the affective and physiological levels?
4. Is attunement in one system (affective, physiological) meaningfully associated with attunement in the other?
5. Finally, how are physiological and affective attunement (or lack thereof) related to child outcomes in terms of behavioral regulation (e.g., rates of internalizing and externalizing behavior problems)? Additionally, the current study will utilize both variable-centered and person-centered statistical approaches to answer these questions in different ways.

In addition to improving upon previous research by using both variable-centered and person-centered approaches in the current study, the current study can also add to the current body of literature on IPV, attunement and child behavior problems by looking at these variables in pre-school age children (ages 3 – 5 years) and by including teacher report of child behavior problems. In addition to the limitations of examining cortisol in infants mentioned in the previous chapter, it is also important to examine the contribution of attunement to developmental outcomes in pre-school age children because (a) during the pre-school age years, children face specific developmental tasks for which attunement may be particularly important (e.g., navigating interpersonal relationships with peers), and (b) it is during the pre-school age years that child behavior problems begin to show increased stability and become predictive enduring behavioral difficulties which will persist into the late childhood years (Campbell, Shaw, & Gilliom, 2000; Gardner & Shaw,2008). For example, research indicates that 50-60% of children who exhibit disruptive externalizing behavior at ages 3 - 4 years will not outgrow these behaviors and will continue to demonstrate similar externalizing problems during the school-age years and onward (Campbell, Shaw, & Gilliom, 2000). Also prior to age 3 there is such great instability in child behavior due to developmental processes that it is difficult to differentiate between normative behaviors and those which are clinically significant (Gardner & Shaw, 2008). In addition, research shows that children's capacity to use emotional competencies influenced by attunement, such as emotion knowledge, emotion regulation skills, and emotional expression during the preschool years, predicts their long-term social success in during later childhood years (e.g., Denham et al., 2003). Given the importance of the pre-school years in terms of children's subsequent social-emotional development, it is important to understand the ways in which IPV exposure, adrenocortical attunement and affective attunement contribute to children's functioning at this age.

In addition to examining children in their preschool years, this study uses a multi-informant approach (e.g., both teacher report and mother report) to assess child behavior problems. For most, the pre-school age years represent the first time that children spend a significant amount of time in different contexts (e.g., home and school). Children's behaviors may differ across these settings, and multi-informant ratings can help account for these differences (Achenbach, McConaughy & Howell, 1987; Strickland et al., 2012). Additionally, using teacher report in addition to maternal report can help counter the potential impact of reporter bias, for example the potential for mothers under stress or suffering from mental health difficulties to exaggerate the degree of their children's behavior problems (e.g., Najman et al., 2000l Youngstrom et al., 2000).

*6.1 Hypotheses*

*1. IPV and physiological dysregulation.* Mothers and children exposed to greater levels of IPV (psychological, physical and sexual) will exhibit more dysregulated adrenocortical responses to acute stress, as indicated by levels of cortisol in response to a standardized laboratory stress task.

1.a.) It is expected that in children, greater levels of IPV will be associated with more exaggerated adrenocortical stress responses, as indicated by higher baseline cortisol levels prior to the lab stress task and higher levels of cortisol during the peak and recovery periods following the lab task.

1.b.) It is expected that in the mothers, greater levels of IPV will be associated with one of two types of dysregulated adrenocortical stress responses: (1) A majority will demonstrate hypercortisolism, as indicated by higher baseline, peak and recovery cortisol levels in response to the lab stress task, or (2) A subset will exhibit hypocortisolism, as indicated by lower baseline, peak and recovery cortisol levels in response to the lab stress task.

*2. IPV and affective dysregulation.* Mothers and children exposed to greater levels of IPV (both psychological and physical/sexual) will exhibit more dysregulated affective responses to acute stress, as indicated by self-report ratings of emotional distress experienced as a result of the lab stress task.

*3. IPV and Attunement.* It is expected IPV will be differentially associated with physiological attunement and affective attunement:

3.a.) Overall, greater IPV exposure will be associated with greater attuned physiological responses in mothers and their children, but in general these stress responses will indicate attunement for greater levels of cortisol (more exaggerated stress responses).

3.b) It is also expected that there will be a subset of dyads that will be not be attuned for physiological stress reactivity (as it is expected that some mothers will exhibit an attenuated/blunted cortisol response while their children exhibit an exaggerated stress response).

3.c.) It is expected that greater levels of IPV will be associated with less affective attunement during the reunion episode following the lab stress task.

*4. Physiological attunement and affective attunement.*

4.a. ) It is expected that greater physiological attunement will be associated with less affective attunement. In a low-risk context, physiological attunement between a mother and child enhances the mother's ability to read subtle emotion cues in her child, thus enhancing affective attunement. However, in the context of IPV, it is expected that either or both dyad members will become so emotionally aroused that physiological attunement will serve to only further dysregulate both dyad members, thereby interfering with affective attunement

4.b. ) It is also hypothesized that physiological attunement will partially mediate the relationship between IPV and affective attunement (greater physiological attunement will partially explain the association between greater IPV and less affective attunement, for the reason noted above).

*5. IPV, attunement and child outcomes.* IPV and attunement will be related to child outcomes, such that:

5.a.) IPV will directly predict child outcomes, where greater levels of IPV will be related to higher levels of child behavioral problems.

5.b.) Affective attunement will partially mediate the relationship between IPV and child behavior problems.

*6.2 Participants*

Mother-child dyads were recruited from Capital Area Community Services (CACS) Head Start schools all around the greater Lansing community to participate in the "Healthy Moms – Healthy Kids Study". CACS Head Start preschools serve approximately 1,600 children residing in Ingham, Eaton, Clinton, and Shiawasee Counties (CACS Head Start and Early Childhood Programs Annual Report, 2008-2009). Flyers describing briefly describing the study and inclusion/exclusion criteria (see Appendix A) were distributed to all CACS classrooms in the area by the CACS Health Coordinator, as well as directly to parents by CACS Head Start bus drivers and classroom liaisons. In-person recruitment of participants by the study coordinators also occurred at CACS classroom orientations, parent information meetings and family activity nights. Potential participants either phoned the project office, or provided contact information (phone number and/or email) as well as permission to be contacted by study personnel, at which time they completed an intake interview that screened for inclusionary and exclusionary criteria. Prior to completing this screening procedure, exposure to IPV was not discussed during the recruitment process; thus participants could not self-select into the study on the basis of IPV-experiences.

The following inclusionary criteria were utilized to select participants so as to ensure more reliable measurement of the variables of interest:

- Mothers were required to be the biological parent and have legal custody of the child participant.

- Mothers were rquired to speak English as a first language or be reasonably fluent in reading and writing English (e.g., 6th grade level).
- Children were required to be between 3 and 5 years of age.
- Children must have been currently attending a CACS Head Start School in Ingham or Eaton County
- Children were required to have resided with their biological mother for a majority of the time in the last year.

Exclusionary criteria were as follows (so as to ensure for more reliable measurement of salivary cortisol):

- Mothers could not be pregnant at the time of participation.
- Mothers could not be suffering from Cushings disease or Addisons disease (as these types of neuroendocrine disorders are associated with glucocorticoid resistance and show different patterns of HPA-axis functioning from normative samples; e.g., Dai, Buijs & Swaab, 2004; Findling & Raff, 2006).
- Mothers could not be suffering from cancer or receiving cancer treatment (as some cancer treatments can alter levels of cortisol; e.g., Raison, Borisov, Woolwine et al., 2010).
- Children could not have serious birth defects or medical conditions (e.g., cerebral palsy, fragile X syndrome), or significant developmental delays (autism spectrum disorders), as these disorders are also associated with abnormal activation of the HPA-axis (e.g. Hessl, Rivera & Reiss, 2004; Roberts, Clarke, Alcorn et al, 2009).

During the participant recruitment process, 213 women and their children were screened for eligibility for the study. Of these 213 mother-child dyads, 174 were deemed eligible and 39 were deemed ineligible. The most common reasons for ineligibility included: (a) the child was too old ( > 5 years of age), (b) the child was not currently enrolled in Head Start, (c) the mother was pregnant with a due date by which her child would have aged out of the study, (d) the child had a serious medical condition (e.g., seizure disorder) or developmental delay (e.g., autism spectrum disorder), (e) the child was born too premature (e.g., < 37 weeks gestation), (f) the mother was not the biological parent of the child, or (g) the mother did not have legal custody of her child.

Of the 174 mother-child dyads that were eligible to participate in the study, 4 mothers refused participation after being informed of their eligibility and hearing the study description. Of these remaining 170 dyads, 4 mothers could not be reached or did not return phone calls to schedule their in-lab visit; 11 mother-child dyads scheduled an in-lab interview but did not attend and did not reschedule; and 12 mother-child dyads scheduled their in-lab interview and attended but had to terminate the interview early resulting in incomplete or unusable data. The most common reasons for early termination of the in-lab interview included child refusal to provide assent and child illness (e.g., child had a fever over 101, which can affect cortisol levels). Dyads that attended their in-lab interview but had to leave early were given a partial payment of $30 for their participation in the study, and the children received a prize and a snack.

In total, including the 12 dyads whose interviews were terminated early, 156 in-lab interviews were completed. 144 of these visits resulted in complete data; however, one of these dyad's data cannot be used as the research assistant violated protocol during administration of the lab-stress task, thus invalidating the quality of the attunement and cortisol data. The resulting final sample includes 143 dyads with completed in-lab interviews and usable data (See Table 6.1 for a description of sample characteristics).

Table 6.1

*Sample Characteristics (N = 143)*

| Variable | Mean | *SD* | Min. | Max. |
|---|---|---|---|---|
| Mother Age (years) | 29.13 | 4.94 | 19.69 | 43.03 |
| Child Age (years) | 4.25 | 0.66 | 3.04 | 5.61 |
| Family Income (monthly) | $1562.87 | $830.98 | $300.00 | $4950.00 |

| | N | Percent |
|---|---|---|
| Mother Race/Ethnicity | | |
| Caucasian/White | 60 | 42.0 |
| Black/African American | 48 | 33.5 |
| Latina/Hispanic/Chicana | 12 | 8.4 |
| Asian American/Pacific Islander | 5 | 3.5 |
| Native American | 2 | 1.4 |
| Multiracial | 16 | 11.2 |
| Mother Relationship Status | | |
| Single/Never Married | 94 | 65.7 |
| Married | 32 | 22.4 |
| Divorced | 10 | 7.0 |
| Separated | 7 | 4.9 |
| Mother Educational Status | | |
| Some High School | 16 | 11.2 |
| High School Diploma/GED | 24 | 16.8 |
| Some College/Associates Degree | 88 | 61.5 |
| Bachelors Degree | 9 | 6.3 |
| Some Graduate School/Degree | 6 | 4.2 |

Table 6.1 (cont’d)

| | | |
|---|---|---|
| Mother Occupational Status | | |
| Unemployed | 74 | 51.7 |
| Employed Full Time | 32 | 22.4 |
| Employed Part Time | 28 | 19.6 |
| Receiving SSI Disability | 9 | 6.3 |
| Child Gender | | |
| Male | 72 | 50.3 |
| Female | 71 | 49.7 |
| Child Race/Ethnicity | | |
| Caucasian/White | 41 | 28.7 |
| Black/African American | 44 | 30.7 |
| Latino/Hispanic/Chicano | 6 | 4.2 |
| Asian American/Pacific Islander | 4 | 2.8 |
| Native American | 1 | 0.7 |
| Multiracial | 47 | 32.9 |

### *6.3 Procedures*

Upon first contact by project personnel, potential participants were provided with a brief description of the study and were asked to give verbal consent before completing the screening interview (see Appendix B for Intake Screening Interview). Potential participants had the right to refuse to answer any screening questions, as well as to terminate the intake interview without reprecussion. In addition to asking demographic questions and assessing inclusion/exclusion criteria, the screening questionnaire also included several items from the Revised Conflict Tactics Scales (CTS-2; Straus, Hamby, Boney-McCoy, & Sugarman, 1996) to ensure there was an adequate representation of mother-child dyads with a history of IPV-exposure in the sample. Participants were notified of elibility at the conclusion of the screening interview.

Eligible participants were scheduled for an interview at the MSU research office, and were offered two different times, either 3:00 p.m. or 5:30 p.m., in order to maximize the number of participants who could complete interviews on any given day. The interview times were also standardized in this way in order to eliminate differences in cortisol levels due to natural diurnal fluctuations in cortisol.

All interviews were conducted by trained graduate and undergraduate research assistants. At the request of CACS Head Start administration, mothers worked only with highly trained graduate-level research assistants, while the children worked with either trained undergraduate or graduate research assistants. The interviews started with both the mother and the child together in the same room in order to help participants acclimate to the laboratory environment and reduce the likelihood that participants would experience an adrenocortical stress response to being in the laboratory space. During this time, the research assistants went through the consenting procedure with the mother and obtained assent from the child. Mothers were provided with both a written consent form (see Appendix C) and read a verbal description of the study (see Appendix C), as well as were encouraged to ask questions about the study before providing formal written consent. Mothers also provided written consent for their children's participation (Appendix C), as well as for project staff to contact the children's teachers to obtain teacher-report measures to be used in the study (Appendix C). Children were read a brief description of the study and provided verbal assent only after their mothers' consent was obtained.

Following the mother consent and child assent procedures, participants completed a demographic questionnaire and a brief screen for the quality of mother and child saliva (See Appendix D for a copy of all measures), then baseline saliva samples were collected from both the mother and child following the procedures developed by Granger and colleagues (2007). For this, participants were asked to imagine they were chewing their favorite food and then instructed to let the saliva pool in their mouths without swallowing. For mothers, the saliva specimen was deposited through a short drinking straw into a cryogenic storage vial. For children this passive drool technique is difficult, thus they provided saliva by sucking on a swab which was then placed in a cryogenic storage vial, as well. Following collection of the baseline saliva samples, the children were taken into a separate interview room from their mothers for the balance beam task (lab stress task) (Sethre-Hoftsad et al., 2002), which the mothers watched through a two-way mirror. Then mothers were invited into the room with their children for a three minute reunion episode, which was coded for affective attunement by the graduate-level research assistants, who were blind to IPV status and the study hypotheses. Additional saliva samples were taken from both the mother and the child 25 minutes after completion of the balance beam task (peak of cortisol reactivity) and 50 minutes after completion of the task (recovery from cortisol response). In between completion of reunion episode and post-task saliva samples, the mothers completed a number of self and parent-report questionnaires, during which time the child participated in series of fun, interactive lab tasks that were used in a different dissertation study. Following completion of the interview, the mothers were paid $75 for their participation and children received a toy and a snack. All participants also received a referral list that included contact information for local IPV support groups, shelters and resources, as well as other government agencies and programming that are available for resources and aid.

All 143 mothers included in the final sample granted permission for their children's teachers to be contacted for purposes of the study. Following completion of the in-lab assessment, CACS Head Start provided confirmation of each child's most recent/current classroom teacher. Teacher packets were delivered to mailboxes located in the CACS Head Start administrative offices, and for each child included a copy of the mother's signed release form, a consent form to be signed by the teacher (Appendix C), and Child Behavior Checklist - Teacher Report Form (TRF; Achenbach & Rescorla, 2000). Teachers completed these packets and returned all completed materials to the office of the CACS Health Coordinator, were they were retrieved by project personnel. For each completed packet, teachers were compensated with a $10 cash payment.

*6.4 Measures*

6.4.1 *Demographics.* This measure assessed a wide-variety of socio-economic variables (e.g., maternal level of education, family monthly income, type of assistance received from community organizations), information about the mother's past and current romantic partners, and basic demographic information on her child.

6.4.2 *Intimate Partner Violence* was assessed using two measures. Physical violence was assessed with the *Conflict Tactics Scale -2 (CTS-2; Straus, Hamby, Boney-McCoy & Sugarman, 1996).* The CTS-2 is a 39-item self-report measure of the extent to which male romantic partners perpetrated acts of violence against the female reporter. The CTS-2 includes lengthened versions of the three original CTS scales (negotiation, psychological aggression and physical assault), as well as additional scales to assess for sexual abuse/coercion and physical injuries resulting from assaults by the partner. All scales include items that refer to concrete actions or events, rather than views or opinions of abuse. The "negotiation" scale assesses the types of actions partners use to settle conflict, including the degree to which partners express positive regard and respect toward one another. The "psychological violence" scale assesses partners' use of verbal (e.g., threats) and nonverbal actions (e.g., stomping out of the room), while the "physical assault" scale assesses use of physical acts of violence (e.g., hitting, kicking). The "sexual coercion" scale assesses the degree to which partners compel each other to engage in unwanted sexual activity, either verbally or behaviorally. Items are endorsed on a scale of 0 to 6, with a score of 0 indicating that the event did not occur, 1 = event occurred once, 2 = occurred twice, 3 = 3-5 times, 4 = 6-10 times, 5 = 11-20 times, and 6 = more than 20 times. The measure is scored by summing the frequency of items endorsed by their midpoint. Each item also loads onto a scale of minor or severe violence, allowing for severity of violence to be assessed. In the current study, the CTS-2 was administered twice; first to to assess all violence experienced by the mother while pregnant with the child participating in the study (prenatal IPV), and again to assess vioelnce experienced from any partner since her child was born (postnatal IPV).

The CTS-2 was initially validated using an undergraduate college student sample (Straus et al., 1996). The measure demonstrated good internal consistency, with alpha values ranging from .79 for the psychological aggression scale, to .95 for the injury scale. The measure has good construct validity, as indicated by high correlations between the psychological aggression and sexual coercion scales ($r$ = .66), between the physical assault and sexual coercion scales ($r$ = .90) (concurrent validity), and with a scale of social intergration ($r$ = -.29). The measure also demostrated good discriminant validity, as indicated by nonsignificant corrlations between the negotiation scale and the violence scales.

6.4.3 *Psychological abuse* was assessed with the *Psychological Maltreatment of Women Inventory – Short Version (PMWI-S; Tolman, 1989;1995)*. PMWI-S is a 14 item self-report measure assessing two scales of male-perprtrated psychological maltreatment: isolation/domination and verbal/emotional. Examples of items include "my partner used our money or made important financial decisions without talking to me about it" and "my partner blamed me for his problems." Items are endorsed on a scale of 1 to 5, with a score of 1 indicating that the event never occurred, 2 = event occurred rarely, 3 = occurred occasionally, 4 = occurred frequently, and 5 = occurred very frequently. The measure is scored by summing the items together for a total score. Tolman reported a coefficient alpha of .88 for the domination/isolation subscale and .92 for the verbal/emotional subscale. Mothers in the current study completed two versions of the PMW-S; one assessing psychological maltreatment experienced during the prenatal period and one for the postnatal period (defined above).

For all analyses examining IPV as a continuous variables, one sum score was computed to represent all male-perpetrated physical/sexual and psychological violence experienced in the prenatal period; a second sum score was computed to represent all male-perpetrated physical and psychological violence experienced in the postnatal period. Each of these sum scores was derived in the following way: First, a total CTS-2 score was obtained by summing all the scales from the measure (excpet the negotiation scale); this number was then summed with the total PMWI-S score, for each respective time period. Internal consistencies for the individual measures and the summed scores were all in the excellent range (CTS-2 Prenatal $\alpha = .94$; PMWI-S Prenatal $\alpha = .96$; Total Prenatal IPV $\alpha = .97$; CTS-2 Postnatal $\alpha = .95$; PMWI-S Postnatal $\alpha = .96$; Total Postnatal IPV $\alpha = .97$).

6.4.4 *Mother-experienced Life Stressors*: Mothers' exposure to life stressors other than IPV was also assessed in the current study. The measure was used as a potential covariate, as it was hypothesized that exposure to other types of life stressors could influence mothers' adrenocortical functoning and possibly adrenocortical attunement, as well. This variable was later examined for correlations with other variables of interest, with the intention of using mother-experienced life stressors as a covariate in the analyses should it correlate significantly. Mothers' exposure to non-IPV-related life stressors was assesed using the *Life Stressor Checklist - Revised (Wolfe & Kimerling, 1997),* a 15-item self report measure of potentially stressful or traumatic events, for example experiencing a natural disaster, sexual assault (not by a romantic partner), or unexpected death of a loved one. Respondents indicated whether each event on the checklist occurred using a yes/no format. In a large clinical sample, this measure demonstrated good test-retest reliability (percent agreement ranging from 79 to 98; McHugo, Caspi, Kammerer, Mazelis, Jackson, Russell et al., 2005), while in the current study, the LSC-R demonstrated moderate internal consistency ($\alpha = .687$). The number of items positively endoresed was summed to create a total mother-experienced life stressors score for analyses.

6.4.5 *Maternal Mental Health:* Mothers' mental health was measured as a potential covariate in the current study, as it was thought that mental health functioning could have an impact on mother-child affective attunement or mothers' adrenocortical functioning. These scores were later examined for correlations with variables of interest, with the intention of using the maternal mental health scores as covariates in the analyses should they correlate significantly with the main variables of interest. Two different types of maternal mental health problems were measured: depressive symptoms and symptoms of posttraumatic stress disorder. Maternal depressive symptoms were assessed using the *Edinburgh Postnatal Depression Scale (EDPS; Cox, Holden & Sagovsky, 1987)* a 10-item self-report measure of depressive symptoms experienced in the past week. Each item is rated on a scale of 0 to 3, with higher ratings indicating greater depressive symptomotology, and a sum score indicating the total number of depressive symptoms experienced. The EDPS demonstrated good specificity (77%) and sensitivity (85%0, as well as good internal consistency (α = .87) in a clinical sample (Cox et al., 1987). In the current study this scale demonstrated good internal consistency (α = .88), as well.

Maternal posttraumatic stress symptoms were assessed using the *Modified PTSD Symptom Scale – Self Report (MPSS-SR; Falsetti, Resnick, Resick & Kilpatrick, 1993)*. This scale is a 17-item self-report measure of DSM-III-R posttraumatic stress symptoms experienced in the past two weeks. Each item directly corresponds to one of the 17 symptoms of the PTSD diagnostic criteria from the DSM-III-R. For each item, repsondents indicate the frequency with which they have experienced the symptom using a 4-point scale ranging from not at all to 5 or more times per week. For each item positively endorsed, the respondents also rate how much distress they have experienced as a result of the symptom using a 5-point scale ranging from “not at all upsetting” to “extremely upsetting”. In a clinical sample of substance use disorder patients, the MPSS-SR had good sensitivity, correctly identifying 89% of patients diagnosed with PTSD using a structured clinical interview (Falsetti et al., 1993). In the current sudy, respondents completed the MPSS-SR if they endorsed experienceing any prenatal or postnatal IPV, or if they endorsed any item on the Life Stressor Checklist. The 17 items were then summed to create a total PTSD score. In the current study, the MPSS-SR demonstrated excellent internal consistency (α = .922).

6.4.6 *Child Behavior Problems:* The *Child Behavior Checklist – Preschool (CBCL1 ½ - 5; Achenbach & Rescorla, 2000)* is a 99-item parent report measure completed by parents regarding their children’s behavior in the last 2 months. The questionaire assesses seven syndromes indicative of behavioral functioning, including (1) emotionally reactive, (2) anxious/depressed, (3) somatic complaints, (4) withdrawn, (5) sleep problems, (6) attention problems, and (7) aggressive behavior. These subscales load onto two broadband measures of internalizing and externalizing behavior problems, which were used in the current study (Chronbach's alpha of , .84 and .90 respectively). For some post-hoc analyses, the specific subscale score for anxious/depressed problems ($\alpha = .64$) and withdrawn problems ($\alpha = .60$) were also used.

Child behavior problems were also measured using the teacher version of the *Child Behavior Checklist – Teacher Report Form* (TRF; Achenbach & Rescorla, 2000). This is a 99-item measure which produces the same subscales as the parent version with the exception of the sleep problems subscale, as well as broadband scores for internalizing and externalizing. For the current study, only broadband scores from teacher reports were used. In a preschool-age sample of children exposed to IPV, Zerk et al. (2009) reported excellent internal consistency for both the internalizing and externalizing scales (.90 and .91, respectively). In the current study, internal consistency across all items was excellent for both broadband scales (Internalizing $\alpha = .90$; Externalizing $\alpha = .95$).

6.4.7 *Challenged Cortisol and Adrenocortical Attunement:* The *Balance Beam Task (Sethre-Hofstad, Stansbury & Rice, 2002)* is a mild stress task conducted in the lab setting which has been used successfully with children 2 – 4 years of age to induce a measurable cortisol response (see Appendix E for script for beam walking task). In this task, children walked across a balance beam which was approximately 6 inches wide, 30 inches high, and surrounded by soft padding, while their mothers watched from another room. Research assistants remained on either side of the beam during the paradigm to ensure the children's safety. Before walking the beam, the children were instructed to practice on the floor to make sure that they posessed the motor skills required for the task, and to ensure they understood the directions. The children were then instructed to sit and wait for 2 minutes while the research assistants made preparations (e.g., unfolded the mat, wiped the beam clean). This 2-minute delay was included to allow for an anticipatory effect and increase the likelihood of inducing a cortisol response. Following the 2-minute delay, children were helped onto the beam and instructed to perform 4 different tasks: walking forwards, walking backwards, walking sideways, and walking over small cones. These tasks were performed repeatedly until the child was on the beam for a total of 5 minutes. Children who refused to walk the beam or who became too distressed were allowed to either sit on the beam or stand next to the beam with their hand placed on it for the 5 minute duration. Two children from the sample refused to walk on the beam but agreed to sit on the beam for the duration of the task; an additional 2 children refused to either walk or sit on the beam, but stood next to the beam for the duration of the 5-minute period. Following completion of the beam walking, the children were helped off the beam and instructed to wait for their mothers to join them for the reunion period.

6.4.8 *Cortisol enzyme-immunoassay (EIA):* Salivary cortisol was collected from both the mothers and children 3 times throughout the duration of the interview: prior to the beam task (baseline), 25 minutes post-task (peak of cortisol response), and 50 minutes post-task (cortisol recovery). All saliva samples were stored in a locked freezer in order to maintain the integrity of the cortisol until assays could be completed. Cortisol was assayed using a commercially available enzyme immunometric assay specifically designed for use with saliva without modification to the manufacturer's recommended protocol (Salimetrics, 2008). The assay (25 µl test volume) is 510K cleared (US FDA) as a diagnostic measure of adrenal function: range of detection is from 0.003 to 3.0 µg/dl, inter and intra-assay coefficients of variation are less than 10 and 15 %, and the correlation between plasma and saliva is 0.91. The assay is highly specific to cortisol, with less than 0.5% cross-reactivity for other steroids. All assays were completed by the first author of this study and an undergraduate research assistant, with 10% of the samples assayed in duplicate to account for intra-assay variability. Both the inter-assay % coefficient of variability (CV; measure of variability between plates) and the intra-assay % CV (measure of variability between duplicates) were calculated according to the recommendations of Salimetrics (2012) and Schultheiss & Stanton (2009). The inter-assay % CV for the current study (calculated by comparing the values for the high and low control wells of each plate) was 7.92, well within the acceptable range (acceptable is less than 15%). For high controls only the inter-assay % CV was 9.66; for low controls only is was 6.18. The intra-assay % CV (calculated using 95 pairs of cortisol duplicates) was 3.37, also well within the acceptable range (acceptable is less than 10%). When calculating intra-assay % CV by assayer, the values for the first author and undergraduate research assistant were 2.94 and 3.86, respectively.

In the analyses, attunement of adrenocrotical reactivity was examined using both continuous and categorical approaches. For analyses using continuous variables, attunement of adrenocortical reactivity was assessed using four indicators: (1) a difference score computed for mothers' and children's baseline cortisol levels, (2) a difference score computed for mothers' and children's peak cortisol levels (3) a difference score computed for mothers' and children's recovery cortisol level, and (4) the area under curve with respect to ground for the mothers', in order to account for the overall patterns of reactivity observed in the dyads.

6.4.9 *Affective Attunement*. The assessment of affective attunement was conducted in two ways. First, in order to measure the cognitive component of affective attunement – mothers' ability to decode their children's emotional state, also called mentalizing – children's subjective experience of distress from the balance beam task as well as mothers' perception of their children's distress were assessed. Children reported on their experience of distress during the beam task using a 6 point Likert scale (1 = not at all distressing and 6 = exteremely distressing), which was accompanied by the 6 faces of the Wong-Baker FACES Pain Rating Scale (WBFPRS; Wong & Baker, 1988; Wong, Hockenberry-Eaton, Wilson, Winkelstein & Schwartz, 2001). Mothers completed a corresponding 6-point Likert rating scale indicating how stressful or upsetting they thought the beam task was for their children (with 1= not at all stressful and 6 = extremely stressful) (see Appendix F for both scales). Then, concordance of the mothers' and children's stress ratings from the beam task were calculated using a difference score. Mothers also completed a 6-point Likert rating scale to rate their own distress experienced while watching their children completed the beam walking task. This rating was later used to examine whether IPV is related to self-perceived emotional arousal.

The other components of affective attunement (the appropriateness of mothers' repsonding and children's ability to utilize their mothers' repsonses to regulate their affect) was assessed using an in-vivo observational coding scale completed by graduate research assistants during the 3-minute reunion period following the beam task. This is a global coding scale that utilizes a 5-point Likert scale format, where a rating of 1 indicates no evidence of attunement (e.g., the mother does not pay attention or interact with the child, does not act empathically toward the child or attempt to help the child regulate his affective state, and/or the child does not seek out the mother for help with affect regulation) and a rating of 5 indicates high attunement (e.g., the mother is highly empathic and highly facilitative in helping the child to regulate his affect; the child is highly receptive to his mother and is able to use her as an external regulator of emotion) (see Appendix F for full coding scheme). This code was adapted from the "mother-facilitated self-regulation" scale from *The Diagnostic Parent—Child Interaction Coding System (DPICS;* Robinson & Eyberg, 1981). In order to gain more qualitative information about the kinds of attunement strategies mothers used with their children during the reunion task, a checklist of attunement behaviors was also completed during the in-vivo coding. For each dyad, raters indicated whether or not each attunement strategy was used, regardless of whether or not the strategy was successful for helping children regulate their emotions. The types of strategies included use of: (1) Humor (not at the child's expense), (2) Physical comforting (e.g., hugs, kisses, rubbing the child's back, holding child's hand, inviting child to sit on mother's lap), (3) Verbal reassurance/praise (e.g., reassuring the child that he is physically safe and/or praise for the child's behavior), (4) Checking in (e.g., mother shows concern for the well-being of her child, reflects her understanding of her child's emotional state, and/or the mother asks the child to describe his emotional experience), (5) Verbal stress reduction (e.g., the mother uses verbal instructions to help the child regulate himself such as telling the child to take deep breaths), and (6) Distraction (e.g., the mother tries to distract her child from distress, for example by encourage him to play with a toy). A sum score representing the total number of attunement strategies used during the reunion was computed for the analyses. Fifteen videotaped reunion episodes from pilot participants and early interviews were used to establish reliability on this code. After being trained on the coding scheme by the first author, each episode was coded independently by each graduate level research assistant. These independent ratings were then reviewed as a group and conferencing was conducted until all discrepancies in coding were resolved.

## Chapter 7: Results

### *7.1 Missing Data and Imputation*

The first step of analysis was to impute missing data. In the current study, there were several causes for missing data. First, during the lab-based assessment, some mothers missed items on the self-report measures. Second, some mothers and children had difficulty producing sufficient saliva for the cortisol measures (usually due to dehydration or discomfort with the task). Production of saliva typically increased with each successive sample, thus most often participants were missing only a baseline sample. For three participants, although sufficient saliva samples were provided, the ELISA assay procedure yielded unrealistically high or low cortisol values; thus these data were imputed for, as well. Third, several of the teacher packets were not completed in their entirety, or had missed items. Finally, the teachers of five children in the study declined participation, resulting in completely missing TRF data. Despite these data collection issues, however, the overall amount of missing data for the current study was very minimal (less than .05%).

Rather than imputing for all missing data at once, individual measures were imputed separately. This approach allowed for examination of the pattern of missingness at the level of each measure in relation to all other measures with missing data, as well as with key demographic variables. For most individual measures, the percentage of missing data was very small (e.g., CBCL = .064%, CTS-2 Prenatal = .09%; PMWI- S Prenatal = .10%; CTS-2 Postnatal = .02%; PMWI-S Postnatal = .05%, In-vivo attunement code = 0%; Mentalizing = 0%, Attunement strategies = 0%). Other measures were missing somewhat more data, including mother salivary cortisol (1.40%), child salivary cortisol (4.9%) and the TRF (3.56%). For applicable measures, correlations between items loading onto the same subscale were examined and those which correlated most highly as matching variables were selected to use in imputation procedures. For all missing data, imputation was conducted using maximum likelihood procedures. Imputation for most variables was estimated using STATA SE 11(StataCorp., 2009). In order to use highly correlated and theoretically related matching variables, in some instances matching variables with minimal missing data were used (e.g., subscales within the same measure). For these cases, maximum likelihood in LISREL 8.7 (Jöreskog & Sörbom, SSI, Inc., 2004) was used, as STATA SE 11 does not allow for imputation from any variables with missing data. Imputed values were then examined to determine whether they fell within the range of possible values for that specific item or subscale; all imputed values met this criterion.

To determine whether the pattern of missingness within each measure occurred at random, cases were coded dichotomously as having complete data or as missing any data at either the item or subscale level. Next, this pattern of missingness was correlated with all other measures for which data was imputed, as well as with key demographic variables (age of mother, age of child, child gender, mother education, family monthly income, and mother's reported experience of IPV at intake). The pattern of missingness for almost all measures included in the analyses (PMWI-S Prenatal, CTS-2 Prenatal, PMWI-S Postnatal, PMWI-S Postnatal, CBCL, TRF) was not correlated with the pattern of missingness for any other imputed data, or the demographic variables, thus these data are considered missing at random. Within the cortisol data, mothers' baseline, peak and recovery values did not correlate significantly with any other imputed data or demographic variables; however, for two of the women participants all three cortisol values were imputed because these individuals had unreasonably high cortisol values as produced by the ELISA assay procedure. Children's cortisol samples significantly correlated with themselves (baseline with peak, $r = 0.47$, $p = 000$; baseline with recovery, $r = .46$, $p = 000$; peak with recovery, $r = .77$, $p = 000$), but not with any other imputed data or demographic variables. These significant correlations indicate that if a child had difficulty providing one saliva sample, they were likely unable to provide a sample at one or both of the other time points, as well. This finding is consistent with observations made during the lab assessments, where some children simply could not produce sufficient saliva at any time point for various reasons including dry mouth, disliking the feeling of the swab in their mouths, or not understanding how to hold the swab in their mouths correctly. To summarize, all missing data for variables of interest can be considered missing at random, thus the imputed data was used for all analyses. The descriptive statistics post-imputation for all continuous variables used in the analyses can be found in Table 7.1 below.

### *7.2 Data Reduction*

The hypotheses for the current study were tested using data analytic approaches with both continuous and categorical data. For analyses utilizing continuous data, data reduction procedures are described in the above measures section. For analyses utilizing categorical data, data reduction was conducted in the following ways (see Table 7.2 below for a quick summary).

Intimate Partner Violence was categorized into three groups based on a modified version of the coding system developed by the authors of the CTS-2, Straus and colleagues (1996): no violence, minor violence and severe violence. Participants were coded as having experienced severe IPV if they endorsed any of the severe items on either the prenatal or postnatal form of the CTS-2. Participants were coded has having experienced minor IPV if they did not endorse any severe items on either the prenatal or postnatal form of the CTS-2, but had endorsed at least one minor item, with the exception of items number 3 (“My partner insulted or swore at me”), 18 (“My partner shouted or yelled at me”), and 25 (“My partner stomped out of the room or house or yard during a disagreement”) on either form of the CTS-2. These three items, although classified on the CTS-2 as minor violence, are more normative and commonly occurring behaviors during relational conflict (over 90% of the sample endorsed these items on the prenatal and/or postnatal version of the measure), thus if these were the only minor level items endorsed, the participant was placed in the “no violence” category. Finally, participants were placed in the “no violence” category if they did not endorse any minor or severe items on either the prenatal or postnatal form of the CTS-2. This coding resulted in the following group sizes: no violence ($n$ = 22), minor violence ($n$ = 54), severe violence ($n$ = 67).

For cortisol reactivity both mothers and children were categorized into groups based on whether or not they demonstrated reactivity, defined as a 10% or more increase in cortisol from baseline to peak. This value of 10% or more difference between baseline and peak value was selected as it accounts for two times the margin of error that results from the assay procedure, and thus can be considered a conservative estimate of significant change in cortisol between sampling times (Granger, Weisz, McCracken, Ikeda & Douglas, 1996). Those who exhibited 10% or more increase from baseline sampling to peak sampling were classified as reactors ($n$ = 21 for mothers; $n$ = 49 for children), while those who exhibited less than a 10% increase from the two time points were classified as non-reactors ($n$ = 122 for mother; $n$ = 94 for children). Mothers and children were also categorized based on their recovery from the stress paradigm, as well, using the same criteria of 10% decrease. The pattern of cortisol during recovery is important to examine, as previous research has shown that a lack of recovery is characteristic of children with internalizing psychopathology (e.g., Gotlib, Joormann, Hallmayer & Minor, 2008;

Hastings, Ruttle, Serbin, Mills, Stack & Schwartzman, 2011)  Those who demonstrated a 10% or more decrease from peak sampling to recovery sampling were classified as recoverers (*n* = 57 for mothers; *n* = 55 for children), while those who showed less than a 10% change between the two time points where classified as non-recoverers (*n* = 86 for mothers; *n* = 88 for children).  Finally, children were classified based on overall pattern of cortisol response from baseline to peak to recovery.  Those children who demonstrated a 10% or more increase from baseline to peak and then a 10% or more decrease from peak to recovery were classified as reactor-recoverers (*n* = 28).  Those who exhibited a 10% or more increase from baseline to recovery and exhibited either less than a 10% decrease from peak to recovery or an increase in cortisol from peak to recovery were classified as reactor-non-recoverers (*n* = 21), while all others (those who showed no peak from baseline to recovery and those who showed a decline from baseline to recovery) were classified as non-reactors (*n* = 94).

For affective attunement, participants were coded in into to two categories based on the in-vivo attunement code: unattuned (an in-vivo attunement score of 1 or 2; *n* = 31) and attuned (an in-vivo attunement score of 3 or better; *n* = 112).  This categorization was based on the qualitative definitions of the attunement codes, where dyads needed to demonstrate at least one attunement behavior to earn an attunement code of 3; those who were coded as a 2 made unsuccessful attempts to attune, while those coded as a 1 did engage in any attunement behaviors.  Dyads were categorized based on their mentalizing score, as well.  For this categorization, dyads were classified as good mentalizers if the absolute value of their mentalizing score was a 0 or a 1, as these scores indicate either perfect agreement or almost perfect agreement between the mother and child's rating of the stressfulness of the beam task (*n*

= 65), while those with difference scores of 2 or more were categorized as poor mentalizers (*n* = 78).

For child behavior problems, children were categorized based on both mothers' report and teachers' report of behavior problems. First, children's raw scores on both the CBCL and TRF for both the internalizing and externalizing broadband scales were converted into age and gender normed t-scores (Achenbach & Rescorla, 2000). Next, those children with t-scores of 60 or more on *either* of the CBCL or TRF internalizing scales were categorized as negatively adapted for internalizing (*n* = 36), while those with t-scores of less than 60 on both the CBCL and TRF internalizing scales were categorized as positively adapted (*n* = 107). This cut-off of 60 or above was selected because it is the recommended cut-off value for identifying borderline clinically significant problems when using the CBCL measure (Achenbach & Rescorla, 2000). Similarly, those children with t-scores of 60 or more on *either* of the CBCL or TRF externalizing scales were categorized as negatively adapted for externalizing (*n* = 38), while those with t-scores of less than 60 on both the CBCL and TRF externalizing scales were categorized as positively adapted (*n* = 105). Finally, to created more equal group sizes, for some analyses internalizing and externalizing behavior problems were condensed into a single code representing any behavior problems. For this, children were classified as negatively adapted for any behavior problems if they had a t-score of 60 or more for *either* internalizing based on *either* mom or teacher report, or externalizing based on *either* mom or teacher report (*n* = 52); children were classified as positively adapted for any behavior problems if they had t-scores of less than 60 on all four scales (CBCL internalizing, CBCL externalizing, TRF internalizing, TRF externalizing; *n* = 91).

Table 7.1

*Descriptive Statistics for Continuous Variables Post-Imputation, Not Transformed*

| Variable | Mean | *SD* | Min. | Max. |
|---|---|---|---|---|
| Prenatal IPV | 22.72 | 23.62 | 0.00 | 106.00 |
| Postnatal IPV | 23.87 | 25.18 | 0.00 | 103.00 |
| Mother Cortisol: | | | | |
| Baseline | 0.41 | 0.66 | 0.07 | 4.81 |
| Peak | 0.32 | 0.35 | 0.06 | 2.79 |
| Recovery | 0.30 | 0.37 | 0.06 | 3.69 |
| Δ Baseline to Peak | -0.10 | 0.43 | -4.25 | 0.63 |
| Δ Peak to Recovery | -0.02 | 0.15 | -1.05 | 0.90 |
| Area Under Curve (Ground) | 1345.96 | 1704.41 | 92.16 | 13170.69 |
| Child Cortisol: | | | | |
| Baseline | 0.50 | 0.97 | 0.02 | 6.16 |
| Peak | 0.47 | 0.84 | 0.04 | 5.94 |
| Recovery | 0.47 | 0.98 | 0.03 | 10.01 |
| Δ Baseline to Peak | -0.02 | 0.57 | -3.21 | 3.27 |
| Δ Peak to Recovery | -0.01 | 0.47 | -2.41 | 4.07 |
| Mother-Child Cortisol Difference (Absolute Values): | | | | |
| Baseline | 0.36 | 0.71 | 0.00 | 4.81 |
| Peak | 0.30 | 0.62 | 0.00 | 5.45 |
| Recovery | 0.30 | 0.89 | 0.00 | 9.56 |
| Attunement: | | | | |
| # Attunement Strategies Used | 2.90 | 1.30 | 0.00 | 7.00 |
| In-vivo Attunement (Reversed) | 2.46 | 1.16 | 1.00 | 5.00 |
| Mentalizing | 1.89 | 1.40 | 0.00 | 5.00 |
| Child Behavior Problems: | | | | |
| CBCL Internalizing Probs. | 8.52 | 6.48 | 0.00 | 33.00 |
| CBCL Externalizing Probs. | 12.64 | 7.49 | 0.00 | 36.00 |
| CBCL Anxious/Depressed | 2.76 | 2.18 | 0.00 | 10.00 |
| CBCL Withdrawn | 1.48 | 1.72 | 0.00 | 8.00 |
| TRF Internalizing Probs. | 5.25 | 6.12 | 0.00 | 42.00 |
| TRF Externalizing Probs. | 9.11 | 9.86 | 0.00 | 54.00 |

Table 7.2

*Summary of Data Reduction for Categorical Variables*

| Variable | Categories | Coding Strategy | *N* |
|---|---|---|---|
| IPV | None | No items or only items 3, 18, or 25 on pre/postnatal CTS | 22 |
| | Minor | No severe items; any “minor” item on pre/postnatal CTS | 54 |
| | Severe | Any “severe” item on pre/postnatal CTS | 67 |
| Maternal Cortisol | Non-reactor | < 10% change from baseline to peak | 122 |
| | Reactor | ≥ 10% change from baseline to peak | 21 |
| | Non-recoverer | < 10% change from peak to recovery | 86 |
| | Recoverer | ≥ 10% change from peak to recovery | 57 |
| Child Cortisol | Non-reactor | < 10% change from baseline to peak | 94 |
| | Reactor | ≥ 10% change from baseline to peak | 49 |
| | Non-recoverer | < 10% change from peak to recovery | 88 |
| | Recoverer | ≥ 10% change from peak to recovery | 55 |
| Overall Child Cortisol Pattern | Reactor-Recoverer | ≥ 10% change from baseline to peak *and* ≥ 10% change from peak to recovery | 28 |
| | Reactor-Non-recoverer | ≥ 10% change from baseline to peak *and* < 10% change from peak to recovery | 21 |
| | Non-reactor | < 10% change from baseline to peak | 94 |
| Attunement | Attuned | In-vivo attunement score ≥ 3 | 112 |
| | Unattuned | In-vivo attunement score of 1 or 2 | 31 |
| Mentalizing | Good | Mentalizing score of 0 or 1 | 65 |
| | Poor | Mentalizing score ≥ 2 | 78 |
| Internalizing Problems | Positively Adapted | T-score < 60 on both CBCL and TRF internalizing scale | 107 |
| | Negatively Adapted | T-score ≥ 60 on CBCL and/or TRF internalizing scale | 36 |
| Externalizing Problems | Positively Adapted | T-score < 60 on both CBCL and TRF externalizing scale | 105 |
| | Negatively Adapted | T-score ≥ 60 on CBCL and/or TRF externalizing scale | 38 |
| Overall Adaptability | Positively Adapted | T-score < 60 on CBCL & TRF internalizing *and* CBCL & TRF externalizing scales | 91 |
| | Negatively Adapted | T-score ≥ 60 any CBCL or TRF broadband internalizing or externalizing scale | 52 |

*7.3 Hypothesis Testing*

*(1) Is physiological dysregulation observed in IPV exposed mothers and children in response to acute stress?* It was originally hypothesized that mothers and children exposed to greater levels of IPV would exhibit more dysregulated adrenocortical responses to acute stress, as indicated by higher baseline, peak and recovery cortisol levels in response to the lab stress task (hypercortisolism). It was also expected that some mothers exposed to IPV would exhibit a different form of physiological dysregulation, hypocortisolism, as indicated by lower baseline, peak and recovery cortisol levels in response to the lab stress task.

The hypothesis that mothers exposed to greater levels of IPV would exhibit more dysregulated adrenocortical responses (greater cortisol reactivity) to acute stress was tested using hierarchical regression analyses. Because the cortisol data was highly skewed, all three cortisol values (baseline, peak, recovery) for both mothers and children, as well as all four IPV variables (prenatal psychological violence, prenatal physical violence, postnatal psychological violence and postnatal physical violence) were log-transformed prior to conducting the regressions. To illustrate how this transformation corrects for skewedness, Figure 7.1 below shows the distribution for the variable of child baseline cortisol prior to this logarithmic transformation and after.

Figure 7.1 *Distributions for Child Baseline Cortisol Before and After Log-Transformation*

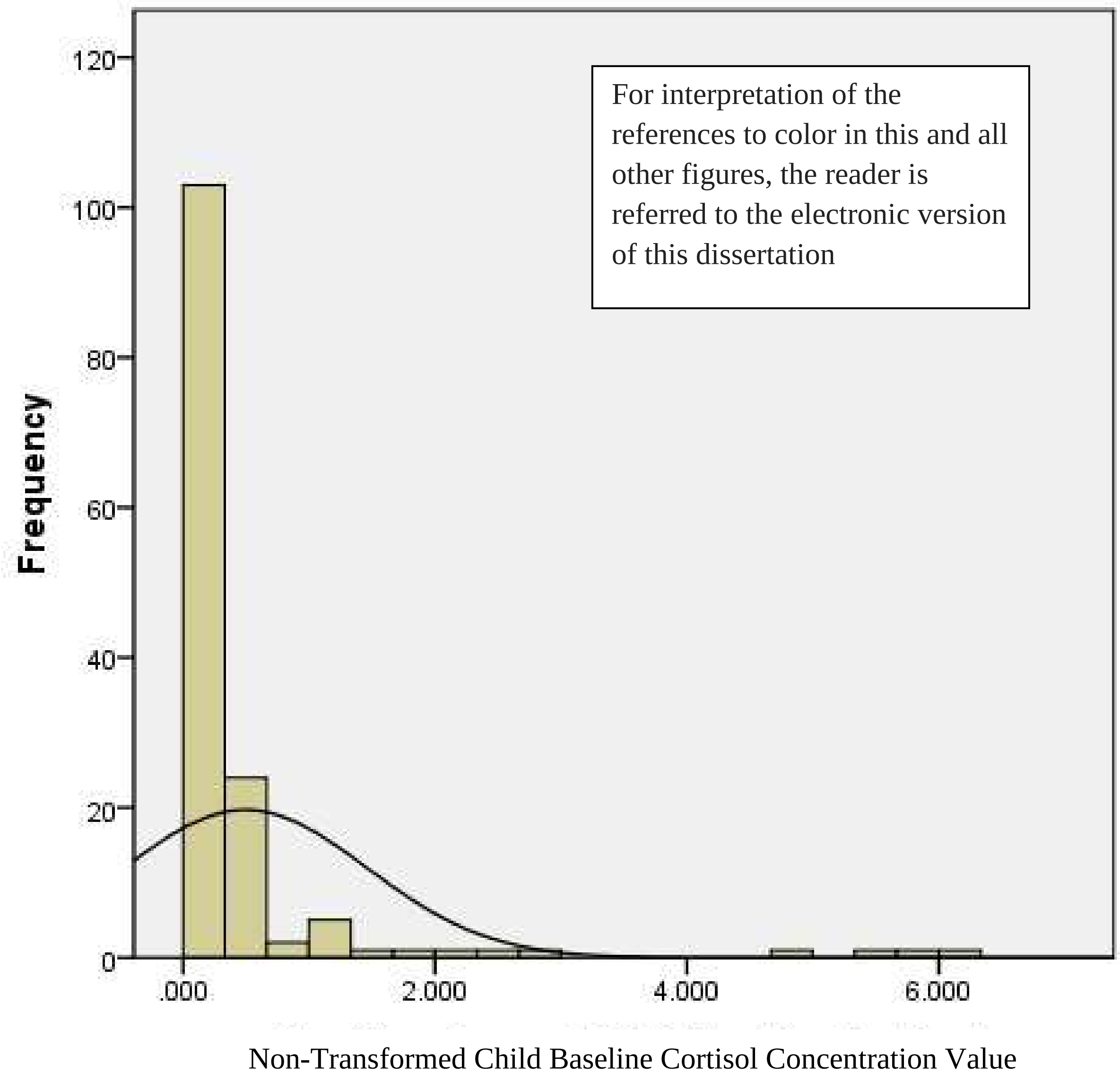
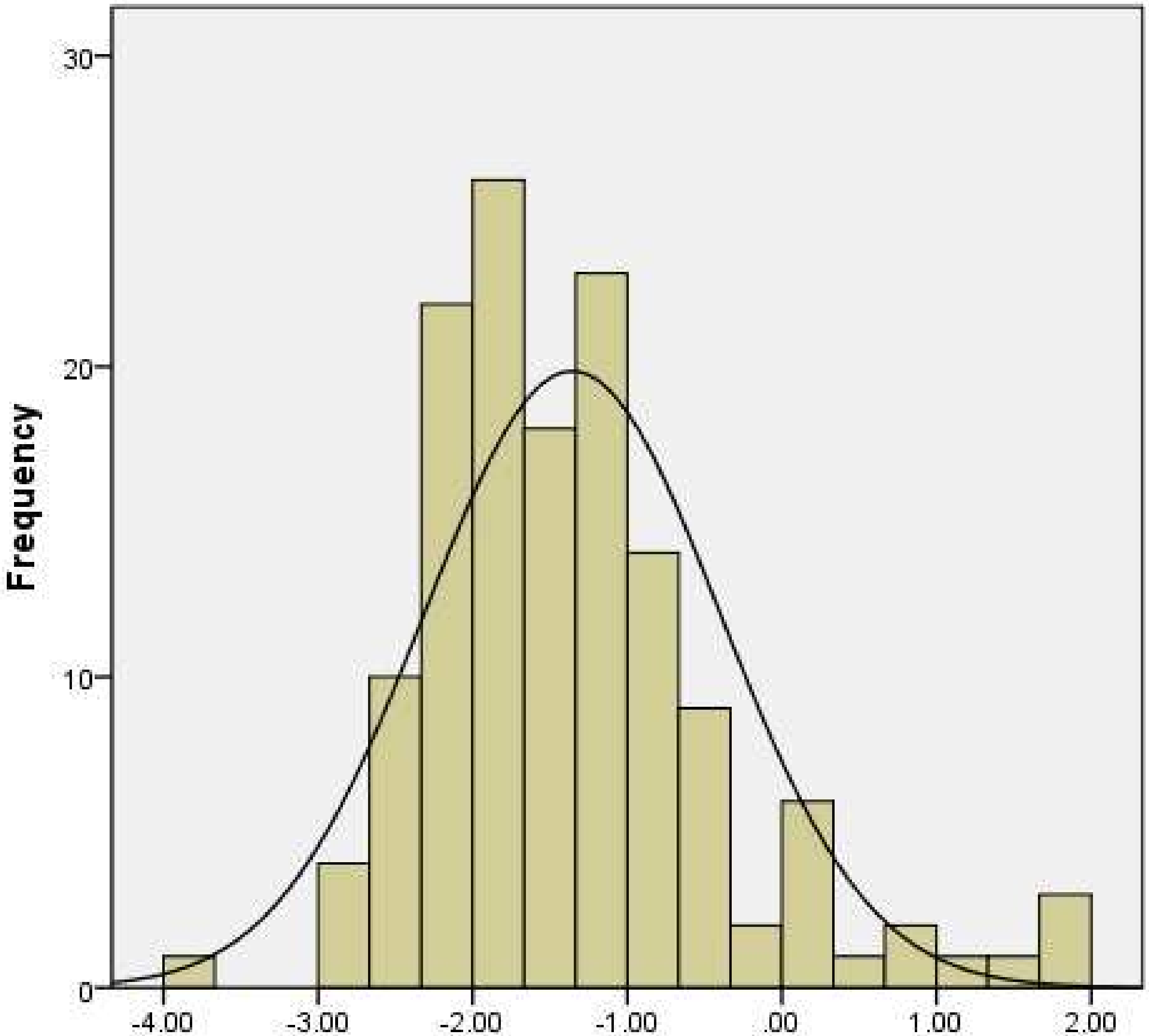


Figure 7.1 (cont'd)



Log-Transformed Child Baseline Cortisol Concentration Value

Next, bivariate correlations between the mothers' rating of anxiety at the start of the lab visit, mothers' rating of anxiety prior to giving the first cortisol sample, number of stressful life experiences experienced by the mother, family income and mothers' baseline, peak and cortisol values were estimated in order to determine if any of these variables should be entered as covariates in the hierarchical regression analyses.   Mothers' ratings of anxiety at the start of the lab visit and anxiety prior to baseline cortisol sampling were significantly correlated with one another ($r$ = .74, $p$ = .000), as well the number of stressful life events ($r$ = .219, $p$ = .009; $r$ = .176, p = .036) but the anxiety ratings, family income and number of stressful life events were not correlated with any of the three mother cortisol values (see Table 7.3 below); thus these variables were not used in the regression analyses.

Table 7.3

*Correlation Matrix for Maternal In-lab Anxiety Ratings and Maternal Cortisol Values*

| Variable | 1 | 2 | 3 | 4 | 5 | 6 | 7 |
|---|---|---|---|---|---|---|---|
| 1. Anxiety Start of Visit | -- | .735** | .219** | -.057 | -.056 | -.029 | -.072 |
| 2. Anxiety Before Baseline | | -- | .167* | -.040 | .025 | .020 | .000 |
| 3. Mom Stressful Life Exp. | | | -- | -.154 | .018 | .024 | .014 |
| 4. Family Income | | | | -- | -.073 | -.036 | -.040 |
| 5. Mom Baseline Sample | | | | | -- | .919* | .894* |
| 6. Mom Peak Sample | | | | | | -- | .953* |
| 7. Mom Recovery Sample | | | | | | | -- |

*Note:* ***p* < .01; **p* <. 05

The first hierarchical regression model was estimated predicting mothers' baseline cortisol values from IPV, with prenatal IPV (sum of prenatal CTS-2 and PMWI-S) entered in block 1, and prenatal and postnatal IPV (sum of postnatal CTS-2 and PMWI-S) entered into block 2. Prenatal and postnatal IPV were entered in separate blocks in order to examine if IPV exposure at each time point exerts unique effects on maternal cortisol levels. In the next two hierarchical regressions the blocks of IPV were entered in the same way as the first regression, however regression 2 predicted to mothers' peak cortisol levels and regression 3 predicted to mothers' recovery cortisol levels. Finally, two additional hierarchical linear regressions were estimated, one predicting to the change between mothers' baseline and peak cortisol values, and the second predicting to the change between mothers' peak and recovery cortisol values. As indicated by Table 7.4, neither prenatal nor postnatal IPV exposure significantly predicted mothers' baseline, peak or recovery cortisol values, or change in cortisol between each sampling period.

Table 7.4

*Predicting Maternal Cortisol from Intimate Partner Violence*

| Model | Predictor | *df* | *F* | *p* | $\Delta R^2$ | *B* | *se b* |
|---|---|---|---|---|---|---|---|
| Predicting to Mother Baseline Cortisol | | | | | | | |
| | Block 1 | 1, 141 | .123 | .726 | .001 | | |
| | Prenatal IPV | | | | | -.040 | .113 |
| | Block 2 | 2, 140 | .068 | .935 | .001 | | |
| | Prenatal IPV | | | | | -.022 | .187 |
| | Postnatal IPV | | | | | -.021 | .185 |
| Predicting to Mother Peak Cortisol | | | | | | | |
| | Block 1 | 1, 141 | .079 | .779 | .001 | | |
| | Prenatal IPV | | | | | -.028 | .101 |
| | Block 2 | 2, 140 | .060 | .941 | .001 | | |
| | Prenatal IPV | | | | | -.001 | .167 |
| | Postnatal IPV | | | | | -.034 | .165 |
| Predicting to Mother Recovery Cortisol | | | | | | | |
| | Block 1 | 1, 141 | .000 | .993 | .000 | | |

Table 7.4 (cont'd)

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | Prenatal IPV | | | | | -.001 | .096 |
| | Block 2 | 2, 140 | .264 | .768 | .004 | | |
| | Prenatal IPV | | | | | .092 | .160 |
| | Postnatal IPV | | | | | -.115 | .157 |
| Predicting to Δ from Baseline – Peak | | | | | | | |
| | Block 1 | 1, 141 | .003 | .957 | .007 | | |
| | Prenatal IPV | | | | | -.004 | .066 |
| | Block 2 | 2, 140 | .282 | .755 | .004 | | |
| | Prenatal IPV | | | | | .061 | .109 |
| | Postnatal IPV | | | | | -.080 | .107 |
| Predicting to Δ from Peak – Recovery | | | | | | | |
| | Block 1 | 1, 141 | 2.457 | .119 | .017 | | |
| | Prenatal IPV | | | | | -.037 | .024 |
| | Block 2 | 2, 140 | 1.293 | .278 | .018 | | |
| | Prenatal IPV | | | | | -.025 | .039 |
| | Postnatal IPV | | | | | -.015 | .039 |

Next, a repeated measures analysis of variance (ANOVA) was conducted in order to examine whether differences in the pattern of cortisol values existed based on the three categories of IPV exposure (no violence, minor violence and severe violence). In this model, the between-subjects factors were the three IPV groups, and the within-subjects factors were the three measures of cortisol: mothers' baseline, mothers' peak, and mothers' recovery. Polynomial contrasts were used to test for linear and quadratic trends in the means of the three cortisol variables.  The repeated measures ANOVA indicated no significant group differences in cortisol based on IPV exposure (Wilks' $\Lambda = .986$, $F(2,140) = .493$, $p = .741$).

Next, in order to test the hypothesis that children exposed to greater levels of IPV would exhibit more dysregulated adrenocortical responses (greater cortisol reactivity) to acute stress, hierarchical regression analyses and repeated measures ANOVA were again conducted. The first hierarchical regression model was estimated predicting children's baseline cortisol values from IPV, with prenatal IPV (sum of prenatal CTS-2 and PMWI-S) entered again in block 1, and prenatal and postnatal IPV (sum of postnatal CTS-2 and PMWI-S) again entered into block 2. In the next four hierarchical regressions, the blocks of IPV were entered in the same way as the first regression, however regression 2 predicted to children's peak cortisol levels, regression 3 predicted to children's recovery cortisol levels, regression 4 predicted to the change between children's baseline to peak cortisol, and regression 5 predicted to the change between children's peak and recovery cortisol values.  As indicated by Table 7.5, neither prenatal nor postnatal IPV exposure significantly predicted children's baseline, peak or recovery cortisol values, or their change in cortisol in between each sampling period.

Table 7.5

*Predicting Child Cortisol from Intimate Partner Violence*

| Model | Predictor | *df* | *F* | *P* | $\Delta R^2$ | *B* | *se b* |
|---|---|---|---|---|---|---|---|
| Predicting to Child Baseline Cortisol | | | | | | | |
| | Block 1 | 1, 141 | .784 | .378 | .006 | | |
| | Prenatal IPV | | | | | .003 | .003 |
| | Block 2 | 2, 140 | .489 | .614 | .007 | | |
| | Prenatal IPV | | | | | .001 | .006 |

Table 7.5 (cont’d)

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | Postnatal IPV | | | | | .002 | .006 |
| Predicting to Child Peak Cortisol | | | | | | | |
| | Block 1 | 1, 141 | 1.322 | .252 | .009 | | |
| | Prenatal IPV | | | | | .004 | .003 |
| | Block 2 | 2, 140 | .670 | .514 | .009 | | |
| | Prenatal IPV | | | | | .003 | .006 |
| | Postnatal IPV | | | | | .001 | .005 |
| Predicting to Child Recovery Cortisol | | | | | | | |
| | Block 1 | 1, 141 | 1.377 | .197 | .012 | | |
| | Prenatal IPV | | | | | .004 | .003 |
| | Block 2 | 2, 140 | .857 | .356 | .015 | | |
| | Prenatal IPV | | | | | .001 | .006 |
| | Postnatal IPV | | | | | .003 | .005 |
| Predicting to Δ from Baseline – Peak | | | | | | | |
| | Block 1 | 1, 141 | .134 | .715 | .001 | | |
| | Prenatal IPV | | | | | -.001 | .002 |
| | Block 2 | 2, 140 | .113 | .893 | .002 | | |
| | Prenatal IPV | | | | | .000 | .003 |
| | Postnatal IPV | | | | | -.001 | .003 |
| Predicting to Δ from Peak – Recovery | | | | | | | |
| | Block 1 | 1, 141 | .978 | .324 | .007 | | |
| | Prenatal IPV | | | | | -.002 | .002 |
| | Block 2 | 2, 140 | .575 | .564 | .008 | | |
| | Prenatal IPV | | | | | -.001 | .003 |
| | Postnatal IPV | | | | | -.001 | .003 |

A repeated measures ANOVA was also conducted in order to examine whether differences in children's pattern of cortisol values existed based on the three categories of IPV exposure (no violence, minor violence and severe violence). In this model, the between-subjects factors were again the three IPV groups, and the within-subjects factors were the three measures of child cortisol: baseline, peak and recovery. Polynomial contrasts were used to test for linear and quadratic trends in the means of the three cortisol variables. The repeated measures ANOVA indicated no significant group differences in child cortisol based on IPV exposure (Wilks' $\Lambda = .993$, $F(2,140) = .250$, $p = .909$).

Next, to test the hypothesis that IPV-exposed mothers and children exhibit patterns of physiological dysregulation, either hypercortisolism or hypocortisolism, predictive configural frequency analyses (PCFA; von Eye, 2002) were conducted. PCFA is a categorical data analysis technique that identifies relationships between specific configurations of predictors and the criterion variable. When configurations occur more often than expected by chance they are identified as types; when configurations occur less often than expected by chance they are identified as antitypes. Procedures for alpha protection reduce the risk of capitalizing on chance. It was particularly important to examine whether different patterns of physiological dysregulation existed in the data using this type of analyses because if such patterns were detected, it might explain the lack of significant findings in the regression and ANOVA analyses.

In the first PCFA, the configurations were composed of IPV, mothers' cortisol reactivity between baseline and peak, and mothers' cortisol reactivity between peak and recovery. There were 3 categories of IPV: 1= no IPV (either prenatal or postnatal), 2 = minor IPV, 3 = severe IPV. For reactivity from baseline to peak there were 2 categories: 1 = non-reactors (less than 10% increase or a decrease from baseline to peak) and 2 = reactors (10% or more increase in cortisol from baseline to peak). For reactivity from peak to recovery there were also 2 categories: 1 = non-decliners (less than 10% decrease or an increase from peak to recovery) and 2 = decliners (10% or more decrease in cortisol from peak from recovery).

The 3 x 2 x 2 cross-classification yielded 12 different configurations. A z-test with the Holland-Copenhaver procedure was used in the search for types and antitypes. The base model was a good fit for the pattern of cell frequencies, *LR* $\chi^2$ (6, *N* = 143) = 8.95, *p* = .18 indicating that the results were accurately explained by the main effects among the variables, thus no types or antitypes were expected to emerge (see Table 7.6 for results).

Table 7.6

*PCFA: Intimate Partner Violence and Maternal Cortisol Reactivity*

| Variables | | | CFA | | | |
|---|---|---|---|---|---|---|
| IPV | Base-Peak | Peak-Rec. | $f_{oijkl}$ | $f_{eijkl}$ | $z_{ijkl}$ | $p_{ijkl}$ |
| None | NR | ND | 12 | 12.31 | -0.09 | .465 |
| None | NR | D | 7 | 6.46 | 0.21 | .416 |
| None | R | ND | 1 | .92 | 0.08 | .468 |
| None | R | D | 2 | 2.30 | -0.20 | .420 |
| Minor | NR | ND | 25 | 30.21 | -0.95 | .172 |
| Minor | NR | D | 21 | 15.86 | 1.29 | .098 |
| Minor | R | ND | 4 | 2.27 | 1.15 | .125 |
| Minor | R | D | 4 | 5.66 | -0.70 | .242 |
| Severe | NR | ND | 43 | 37.48 | 0.90 | .184 |
| Severe | NR | D | 14 | 19.68 | -1.28 | .100 |
| Severe | R | ND | 1 | 2.81 | -1.08 | .140 |
| Severe | R | D | 9 | 7.03 | 0.74 | .228 |

*Note*: *LR* $\chi^2$(6, *N* = 143) = 8.95, *p* = .18; z-test with Holland-Copenhaver procedure; $f_o$ = observed frequency; $f_e$ = expected frequency; NR = Non-reactor; R = Reactor; ND = Non-decliner; D = Decliner.

The second PCFA was modeled in the same way as the first, however in this PCFA children's cortisol reactivity between baseline and peak and children's cortisol reactivity between peak and recovery were used instead of mothers' cortisol reactivity. For this model the base was a good fit for the pattern of cell frequencies [$LR\ \chi^2$ (6, $N$ = 143) = 1.44, $p$ = .96], thus it could not be examined for significant types or antitypes. Please note that for PCFAs such as these where the base model is a good fit for the data and types/antitypes will not emerge, no table will be provided in the manuscript hereafter.

*(2) Is affective dysregulation observed in IPV-exposed mothers and children in response to acute stress?* Similar to the original hypotheses about IPV and adrenocortical dysregulation, it was hypothesized that mothers and children exposed to greater levels of IPV would exhibit more dysregulated affective responses to acute stress, as indicated by higher self-report ratings of emotional distress experienced as a result of the lab stress task. This was tested using hierarchical regression analyses and mothers' and children's self-report ratings of emotional distress experienced during the beam walking stress task. The first of these models was estimated predicting mothers' self-reported ratings of distress during the beam task, with prenatal IPV entered in block 1, and prenatal and postnatal IPV entered into block 2. The second model was estimated similarly, but predicted to the children's rating of their own distress during the beam walking task. In both models, all variables entered were the log-transformed variables to correct for skewedness. As indicated by Table 7.7 below, neither prenatal nor postnatal IPV exposure significantly predicted mothers' or children's ratings of distress as a result of the beam task. One-way ANOVAs were also used to test whether there were differences in mother and child self-rated distress based on the three IPV categories (no IPV, minor IPV, and severe IPV).

Neither the ANOVA for mothers' self-rated distress ($F(2,140) = 1.08$, $p = .343$) nor for the children's self-rated distress ($F(2,140) = 1.48$, $p = .231$) indicated significant group differences.

Table 7.7

*Predicting Self-Reported Distress during Beam Task from Intimate Partner Violence*

| Model | Predictor | *df* | *F* | *p* | $\Delta R^2$ | *b* | *SE b* |
|---|---|---|---|---|---|---|---|
| Predicting to Mother Rated Distress | | | | | | | |
| | Block 1 | 1, 141 | 1.270 | .262 | .002 | | |
| | Prenatal IPV | | | | | .103 | .091 |
| | Block 2 | 2, 140 | 1.703 | .186 | .010 | | |
| | Prenatal IPV | | | | | -.072 | .150 |
| | Postnatal IPV | | | | | .216 | .148 |
| Predicting to Child Rated Distress | | | | | | | |
| | Block 1 | 1, 141 | .013 | .910 | .000 | | |
| | Prenatal IPV | | | | | -.012 | .102 |
| | Block 2 | 2, 140 | .058 | .944 | .001 | | |
| | Prenatal IPV | | | | | .032 | .169 |
| | Postnatal IPV | | | | | -.054 | .167 |

*(3) How is IPV related to mother-child attunement at both the affective and physiological levels?* It was expected that IPV would be differentially associated with physiological attunement and affective attunement such that greater IPV exposure would be associated with greater attuned physiological responses in the dyads, but less affective attunement during the reunion episode following the lab stress-task. Additionally, in order to account for the heterogeneity in

the types of adrenocortical alterations associated with IPV exposure (e.g., hypocortisolism versus hypercortisolism), it was expected that most IPV dyads would be adrenocortically attuned for exaggerated stress responses, while a smaller subset of dyads would be not be attuned for physiological stress reactivity at all (these mothers and children would exhibit different patterns of cortisol reactivity).

In order to examine the associations between IPV and physiological attunement several different structural equation models were estimated using LISREL 8.8 Student Version (Jöreskog & Sörbom, 2006). In structural equation modeling, the minimum number of participants required in order to achieve adequate statistical power for any specified model is to multiply the total number of variables (including both manifest variables and latent constructs) by 10. The number of variables included in each of the following structural equation models ranged from 10 for the smallest model (number of participants needed for adequate power = 100), to 14 for most of the large models (number of participants needed for adequate power = 140). Only one model included 15 variables (number of participants needed for adequate power = 150); this model was not part of the originally proposed analyses but was used for post-hoc analysis. The current study included 143 mother-child dyads; therefore there was sufficient power to reveal any true effects in all but the last of the models, which was slightly underpowered.

All of the following models were initially specified with maximum likelihood (ML) estimation using covariance matrices. For all models the parameters were initially freed for estimation, with the exception of the first model, where the values of some parameters were determined *a priori* in order to replicate a specific model. Five fit indices were selected to evaluate the quality of model fit, with the following values required in order to declare good fit (Hu & Bentler, 1999): the Chi-square test (Chi-square, $p > .05$), the Root Mean Square Error of

Approximation (RMSEA ≤ .06; ≤ .08 = acceptable fit), Comparative Fit Index (CFI ≥ .95), the Goodness-of-Fit Index (GFI ≥ .95), and the Standardized Root Mean-squared Residual (SRMR ≤ .08). Additionally, plausibility checks were conducted to ensure all estimated values were within a reasonable range (e.g., ensuring residual variances were > 0).

The first model estimated was a replication of the structural equation model used by Hibel and colleagues (2009) to examine adrenocortical attunement in an IPV-exposed sample. In this analysis, the intercept and slope/curvature of adrenocortical response was derived from the mothers' baseline, peak and recovery cortisol values, as well as and the children's baseline, peak and recovery cortisol values. The intercepts in this model represent the overall cortisol levels, while slope/curvature represents the change in cortisol across the three time points (baseline, peak and recovery). The slope/curvature latent variables were created by weighting each manifest cortisol variable (baseline, peak and recovery) to correspond to the number of minutes elapsed from the baseline sample. Thus the baseline sample had a weight of 0, the peak sample (which was taken on average 40 minutes after baseline) was weighted .4, and the recovery sample (which was taken on average 25 minutes after peak and 65 minutes after baseline) was weighted .65. Centering the slope/curvature variable on the baseline cortisol measure in this way causes the intercept variable to reflect mean cortisol levels at baseline (Hibel et al., 1999). All of the manifest cortisol variables were then weighted equally onto the intercept latent variable (but values were not predetermined as they were for the slope/curvature latent variable). Overall, this model allows for the examination of mother-child physiological attunement by not only looking at the degree of similarity in cortisol levels in mothers and children, but also the degree of similarity in their patterns and rate of cortisol change in response to the stressful task.

It is of note that in the Hibel model, time of cortisol sampling was also included as a covariate; this was done because cortisol sampling was conducting at different times in the day (and thus various points in participants' diurnal rhythm), introducing variance into the cortisol values. In the current study, all cortisol sampling occurred within a narrow window of time at the end of the diurnal rhythm (between 3:30 and 5:30pm), thus time was not included in the replication of the Hibel model for the current study. Additionally, due to the overall sample size and the very few number of dyads who denied experiencing IPV during both the prenatal and postnatal periods (n = 22), the model could not be estimated to compare physiological attunement in IPV-exposed dyads versus non-exposed dyads. However, this model does examine whether physiological attunement existed in the sample as a whole.

The model, shown in Figure 7.2 below as a path diagram with standardized parameter estimates, demonstrated good model fit, meeting threshold for 4 out of 5 of the *a priori* selected goodness of fit indices (SRMR was significantly higher than the cut-off of .08 recommended by Hu & Bentler, 1999). However, it should be noted that some of the standardized estimates are extremely large (e.g., the loadings of mother peak and recovery indicators onto the mother slope construct), suggesting that there may be reason to distrust this model and that it must be interpreted with caution. t-scores for parameter estimates of the model and all subsequent models are displayed in Appendix G, as well as a complete list of fit indices for each model. See Table 7.8 below for the correlation matrix for variables included in the model. The model indicates that mothers' and children's intercepts, or overall cortisol levels, are significantly correlated, while mothers' and children's slopes/curvatures are not significantly correlated, suggesting that in general the sample did not exhibit an attuned pattern of cortisol reactivity across the three sampling points.

Table 7.8

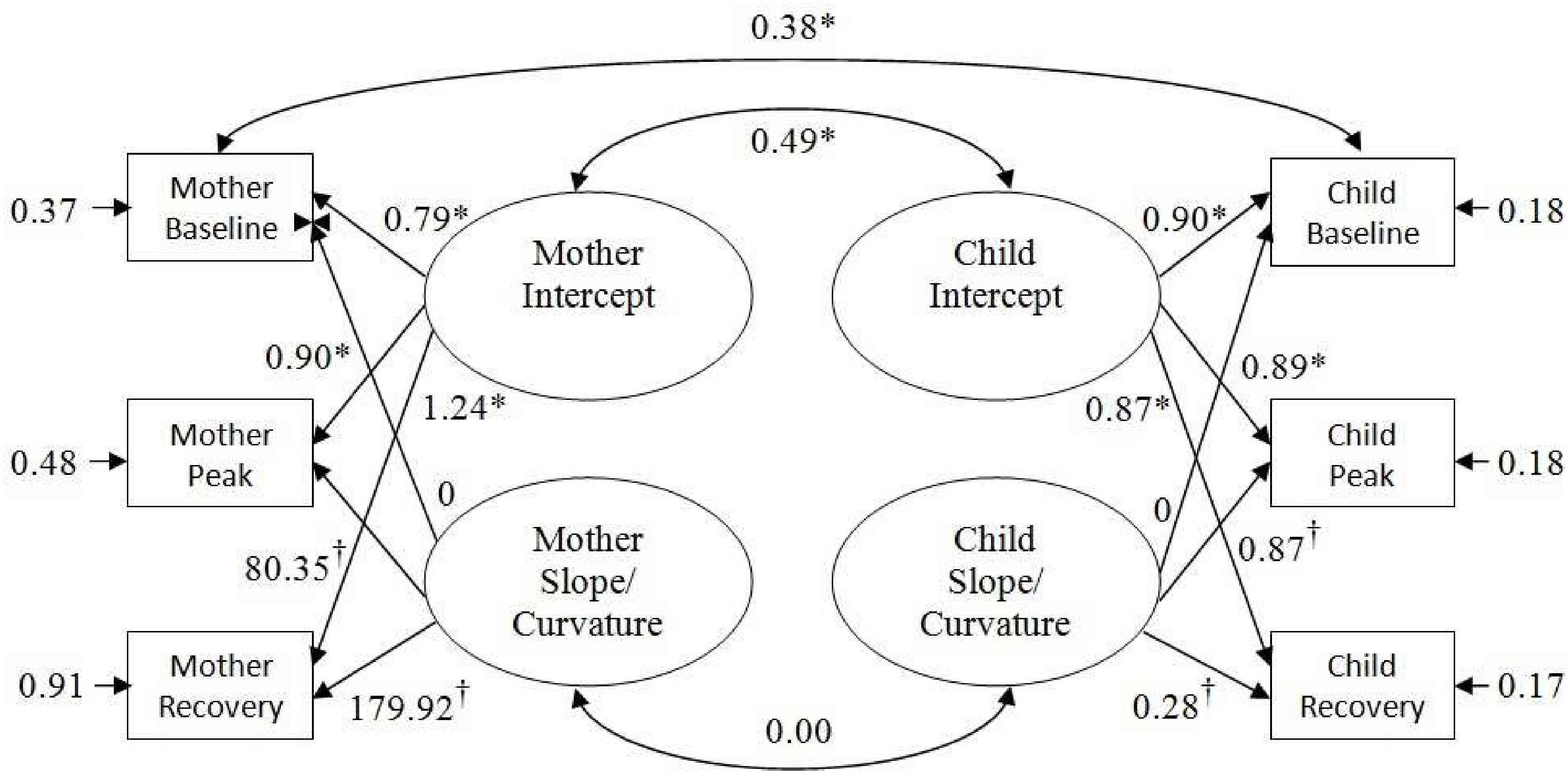
*Correlation Matrix for Structural Equation Model #1*

| Variable | 1 | 2 | 3 | 4 | 5 | 6 |
|---|---|---|---|---|---|---|
| 1. Mother Baseline Cortisol | -- | .821** | .730** | .587** | .693** | .472** |
| 2. Mother Peak Cortisol | | -- | .910** | .379** | .644** | .389** |
| 3. Mother Recovery Cortisol | | | -- | .240** | .595** | .341** |
| 4. Child Baseline Cortisol | | | | -- | .810** | .749** |
| 5. Child Peak Cortisol | | | | | -- | .880** |
| 6. Child Recovery Cortisol | | | | | | -- |

*Note:* $^{**}p < .01$

Figure 7.2

*SEM #1: Attunement of Mother and Child Cortisol Reactivity*




Note: *$p < .05$; †significance test is not conducted; Chi-Square = 9.61 ($df$ = 11), $p$ = 0.566; RMSEA = 0.000 (90% C.I. = 0.00 – 0.08); CFI = 1.00; GFI = 0.98; SRMR = 0.18

The association between IPV and physiological attunement was also examined using PCFA, in order to determine whether there were patterns within the data that could not be detected using a variable centered approach. For the first PCFA, IPV, mothers' baseline to peak cortisol reactivity and children's baseline to peak cortisol reactivity were included in the model. There were 3 categories of IPV: 1= no IPV (either prenatal or postnatal), 2 = minor IPV, 3 = severe IPV. For both mother reactivity and child reactivity from baseline to peak there were 2 categories: 1 = non-reactors (less than 10% increase or a decrease from baseline to peak) and 2 = reactors (10% or more increase in cortisol from baseline to peak). The 3 x 2 x 2 cross-classification yielded 12 different configurations. A z-test with the Holland-Copenhaver procedure was used in the search for types and antitypes. For this model the base model was a good fit for the pattern of cell frequencies [$LR\ \chi^2$ (6, $N$ = 143) = 2.32, $p$ = .89], thus it could not be examined for significant types or antitypes.

A second PCFA was conducted using IPV, mothers' peak to recovery cortisol reactivity, and children's peak to recovery cortisol reactivity. Again, there were three categories of IPV: 1= no IPV (either prenatal or postnatal), 2 = minor IPV, 3 = severe IPV. For both mother reactivity from peak to recovery and child reactivity from peak to recovery there were each 2 categories: 1 = non-decliner (less than 10% decrease, or an increase from peak to recovery), and 2 = decliner (10% or more decrease in cortisol from peak to recovery), yielding 12 different configurations. A z-test with the Holland-Copenhaver procedure was again used in the search for types and antitypes; however the base model was again a good fit for the data [$LR\ \chi^2$ (6, $N$ = 143) = 2.50, $p$ = .87], thus no types or antitypes could be found.

A final PCFA was modeled in order to examine the association between IPV and affective attunement. In this model, IPV, mentalizing, and the in-vivo attunement score were included. The IPV categories were the same three categories listed above. There were 2 categories of attunement: 1 = not attuned, 2 = attuned. There were also 2 categories of mentalizing: 1 = poor mentalizing, 2 = good mentalizing. This yielded 12 configurations. A z-test with Holland-Copenhaver procedure was also used for this model; however the base model was again a good fit for the data [$LR\,\chi^2$ (6, $N$ = 143) = 4.26, $p$ = .64], thus no types or antitypes emerged.

(4) *Is physiological attunement meaningfully associated with affective attunement?* It was originally hypothesized that greater adrenocortical attunement would be associated with less affective attunement. It was also hypothesized that adrenocortical attunement would partially mediate the relationship between IPV and affective attunement. *(5) How are physiological and affective attunement (or lack thereof) related to child outcomes in terms of behavioral regulation (e.g., rates of internalizing and externalizing behavior problems)?* Here it was expected that lower levels of affective attunement would directly predict higher levels of child behavioral problems. It was also hypothesized that greater IPV would predict more child behavior problems and that affective attunement would partially mediate the relationship between IPV and child behavior problems. In order to test the hypotheses for research questions 4 and 5, several different structural equation models were estimated using LISREL 8.8 Student Version (Jöreskog & Sörbom, 2006) and predictive configural frequency analysis was used.

The first full model (Model #2), shown in Figure 7.3 below as a path diagram with standardized parameter estimates, was modeled using four indicators of physiological attunement: the difference between mothers' and children's baseline cortisol values, the difference in peak cortisol values, the difference in recovery cortisol values, as well as the area under the curve with respect to ground for mothers' cortisol. This last indicator was included so the physiological attunement construct would not just reflect the absolute difference between mothers' and children's cortisol values, but also the similarity in their patterns of reactivity over time. Also included in this model were two indicators of affective attunement: the difference between children's distress rating and mothers' ratings of their children's distress (mentalizing score), and the in-vivo attunement code, where smaller values indicate greater affective attunement. Finally, the model included two indicators of child behavior problems: mothers' report of child internalizing problems and mothers' report of child externalizing problems. In the second model that was specified, teacher report of internalizing problems and teacher report of externalizing problems were used as the two indicators for the child behavior problems latent variable. The two models were specified in this way, rather than using internalizing problems as a latent variable with mother report of internalizing and teacher report of internalizing as the indicators, as preliminary analyses indicated that there were no significant correlations between mothers' report and teachers' report of child behavior problems (see Table 7.9 below for a correlation matrix for all variables used for structural equation models #2 and #3).

In order for the model to converge with adequate fit, several modifications were made. First, the model was specified using correlation matrices, rather than covariance matrices to correct for scaling problems with some of the variables included in the model (e.g., area under the curve with respect to ground). Additionally, to correct for negative variances, neither the variance for the affective attunement factor, nor the residual for the difference in peak cortisol were estimated. Finally, the model was estimated using the unweighted least squares (UL) method, as it would not converge using the maximum likelihood method.

After these modifications, the first full structural equation model demonstrated excellent fit, exceeding the thresholds for all 5 of the *a priori* selected goodness of fit indices. Several significant pathways consistent with the original hypotheses emerged, while several did not. Consistent with the original study hypotheses, greater IPV significantly predicted less affective attunement and greater child behavior problems; however contradictory to hypotheses, IPV was not significantly associated with physiological attunement. Also somewhat consistent with hypotheses, physiological attunement shared an inverse relationship with affective attunement, suggesting that as dyads became more physiologically attuned they looked less behaviorally attuned. Contrary to expectations, however, affective attunement was not significantly associated with child behavior problems; thus there was no support for the hypothesis that affective attunement mediates the relationship between IPV and child behavior problems. When this same model was specified with the number of attunement strategies used by the mother added in as an additional indicator of affective attunement, the model retained good fit (Chi-Square = 41.14 ($df$ = 39), $p$ = 0.377; RMSEA = 0.020 (90% C.I. = 0.00 – 0.06); CFI = 1.00; GFI = 0.99; SRMR = 0.042) and the number of attunement strategies significantly loaded onto the affective attunement latent construct ($t$ = -5.81). However, with the inclusion of this indicator, the relationship between IPV and affective attunement was no longer significant (see Appendix G for full model with full fit indices). The other relationships between IPV and child behavior problems and physiological and affective attunement remained significant in this modified version of the model.

The second full model (Model #3, Figure 7.4 below) predicting to child behavior problems as indicated by teacher report of internalizing and externalizing problems, was specified using the same modifications as Model #2. This model also demonstrated excellent model fit, exceeding all of the thresholds for all 5 *a priori* selected fit indices. Similar to the model using mother report of child behavior problems, significant pathways between IPV and affective attunement, and between physiological attunement and affective attunement emerged. However, in this model the direct pathway between IPV and child behavior problems no longer achieved significance, suggesting that teacher reported child behavior problems are not predicted by children's IPV exposure.

Table 7.9

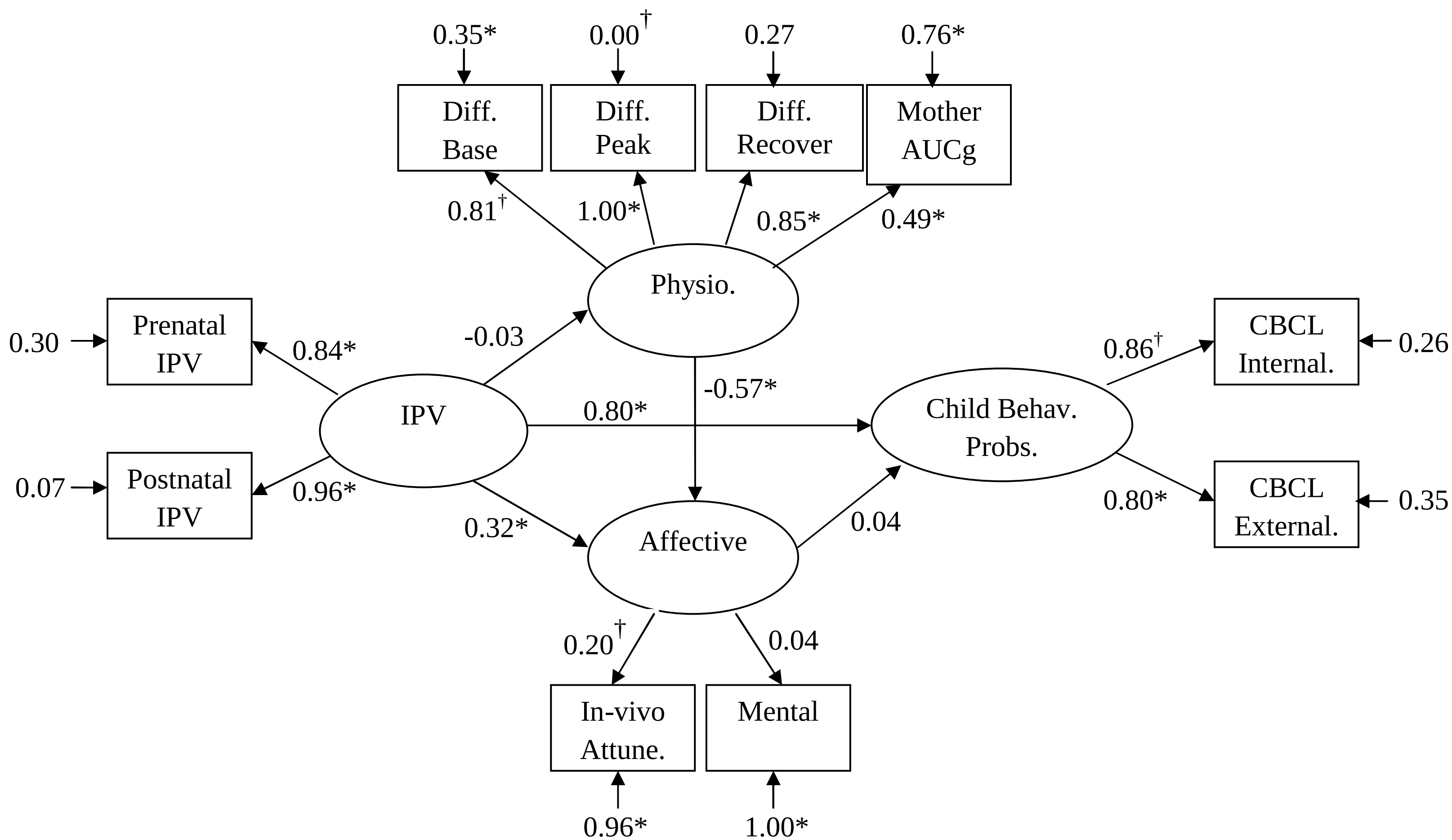
*Correlation Matrix for Structural Equation Models #2 and #3*

| Variable | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. Prenatal IPV | -- | .813** | -.055 | -.021 | .018 | -.055 | .167* | .041 | .289** | .256** | -.074 | -.026 |
| 2. Postnatal IPV | | -- | -.075 | -.066 | -.013 | -.056 | .076 | .179* | .245** | .261** | -.034 | .000 |
| 3. Baseline Cortisol Diff. | | | -- | .632** | .682** | .186* | .102 | -.036 | .005 | .039 | -.087 | -.014 |
| 4. Peak Cortisol Difference | | | | -- | .847** | -.402** | .194* | -.004 | -.040 | -.015 | -.112 | -.057 |
| 5. Recovery Cortisol Diff. | | | | | -- | -.138 | .145 | .029 | -.011 | .027 | -.121 | -.039 |
| 6. Mother AUCg | | | | | | -- | -.047 | .062 | -.027 | .053 | .159 | .198* |
| 7. In-vivo Attunement | | | | | | | -- | -.050 | .101 | .058 | .025 | .069 |
| 8. Mentalizing | | | | | | | | -- | .071 | .018 | .083 | .045 |
| 9. CBCL Internalizing | | | | | | | | | -- | .691** | -.042 | -.041 |
| 10. CBCL Externalizing | | | | | | | | | | -- | .100 | .166* |
| 11. TRF Internalizing | | | | | | | | | | | -- | .658** |
| 12. TRF Externalizing | | | | | | | | | | | | -- |

*Note:* ***p* < .01; **p* < .05

Figure 7.3

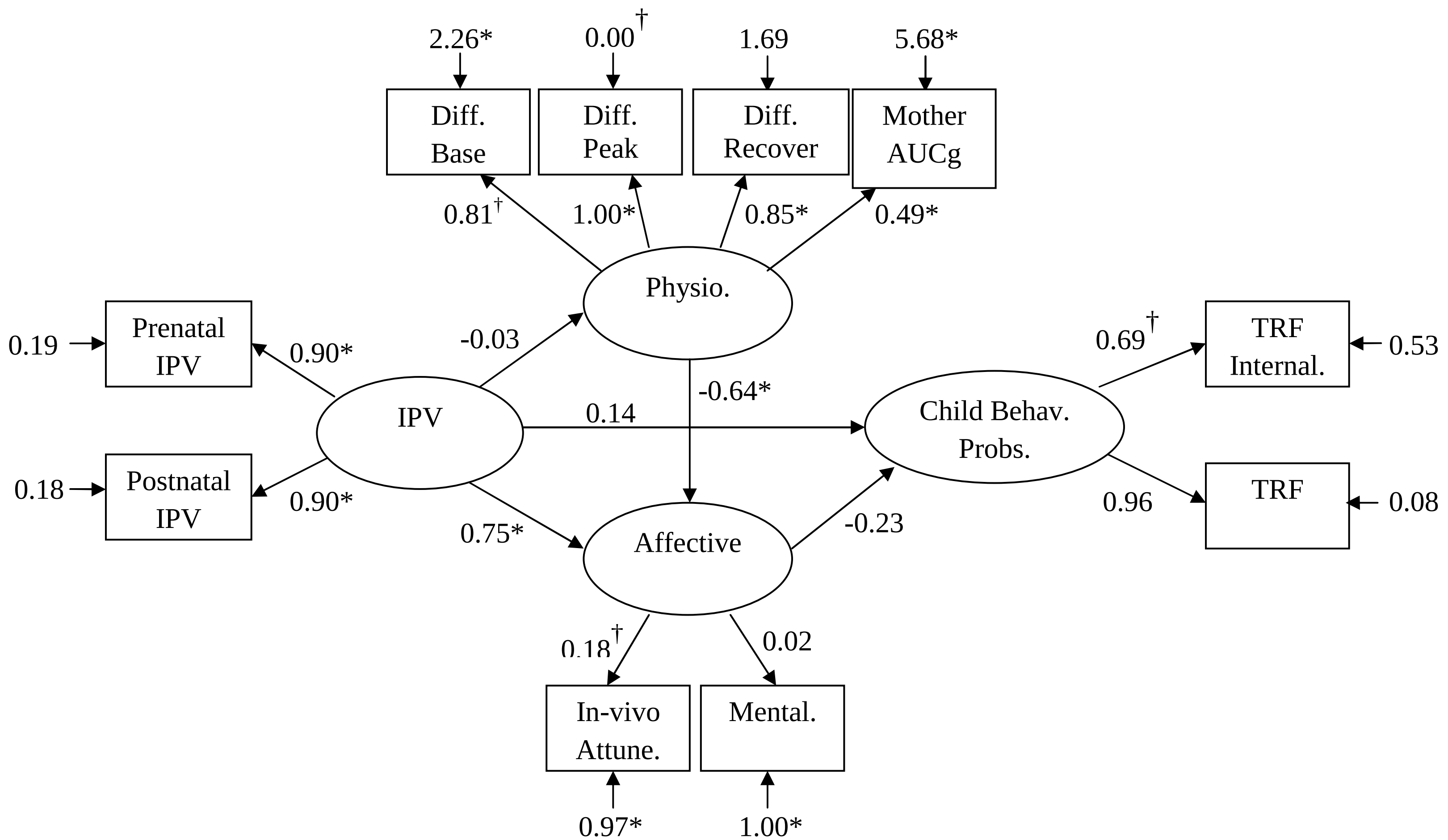
*SEM #2: IPV, Attunement, Maternal Reported Behavior Problems*




Note: **p* < .05; †significance test is not conducted; Chi-Square = 44.35 (*df* = 32), *p* = 0.072; RMSEA = 0.052 (90% C.I. = 0.00 – 0.09); CFI = 1.00; GFI = 0.99; SRMR = 0.043

Figure 7.4

*SEM #3: IPV, Attunement, Teacher Reported Behavior Problems*

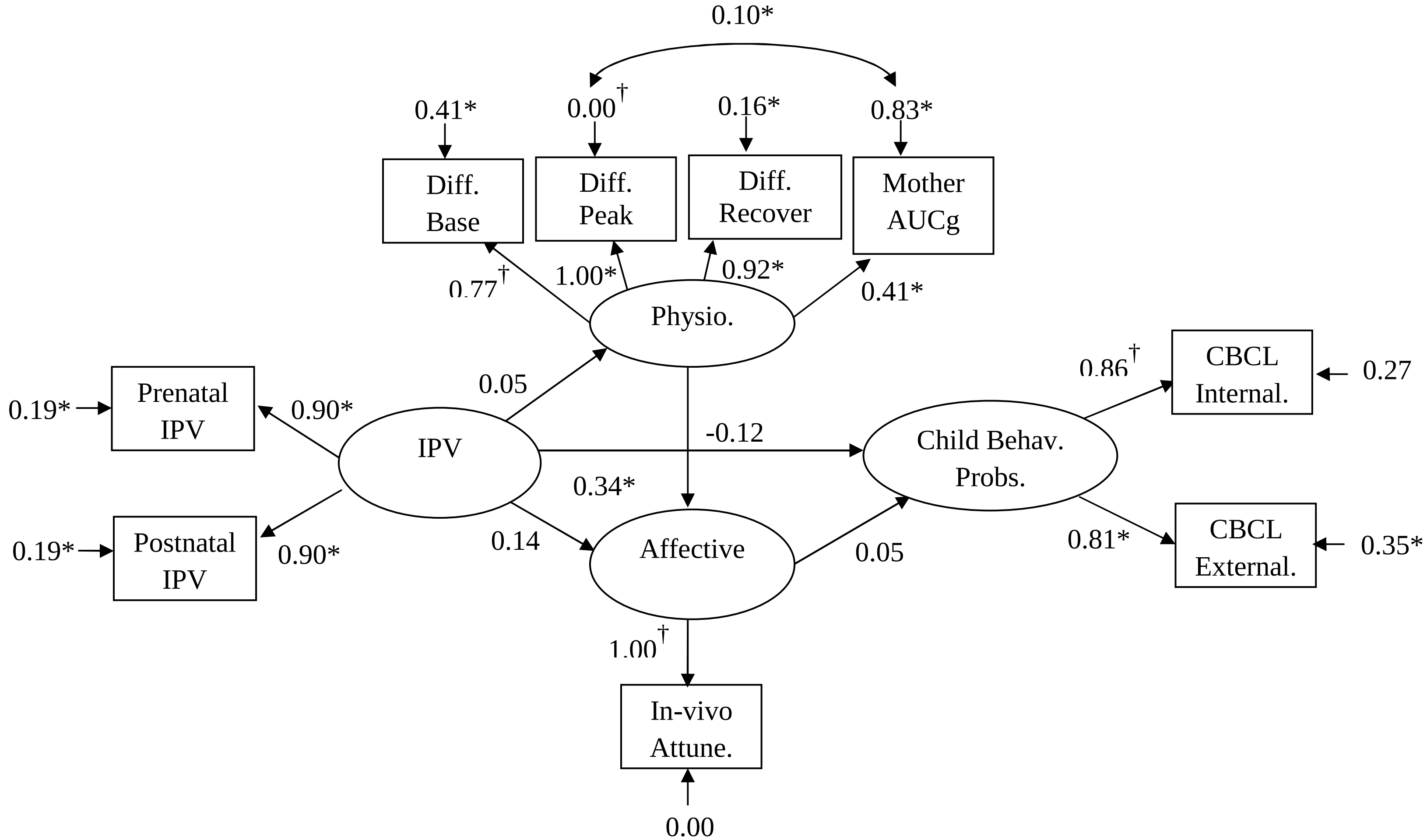



Note: *$p$ < .05; †significance test is not conducted; Chi-Square = 45.89 ($df$ = 33), $p$ = 0.067; RMSEA = 0.052 (90% C.I. = 0.00 – 0.09); CFI = 1.00; GFI = 0.98; SRMR = 0.049

It is of note that in Model #2, when both mentalizing and the in-vivo affective attunement code were both used as indicators of affective attunement together, the mentalizing indicator did not load significantly onto the affective attunement latent construct. Thus, is was hypothesized that these two variables may not be two indicators of a single unified construct, but rather two separate constructs in their own right. Thus, Model #2 was replicated in two modified versions: one using only the in-vivo attunement code as a manifest variable (Model #4, see Figure 7.5 below), and a second using only mentalizing as a manifest variable (Model #5, see Figure 7.6 below). Model #4, using only the in-vivo attunement code, demonstrated good model fit, meeting or exceeding threshold for all 5 *a-priori* selected fit indices. However in this model the only pathway that achieved statistical significant was between IPV and child behavior problems, with IPV predicting greater problem behavior. Model #5, using the mentalizing variable, also demonstrated good model fit, exceeding four out of five of the *a-priori* selected fit indices. However, in this version of the model, none of the pathways between the constructs achieved statistical significance. These findings suggest that although the affective attunement construct as indicated by both mentalizing and the in-vivo attunement score together is not the strongest construct, in the current study, it is more meaningful to consider these variables as two dimensions of a single construct than two separate lone-standing constructs.

Figure 7.5

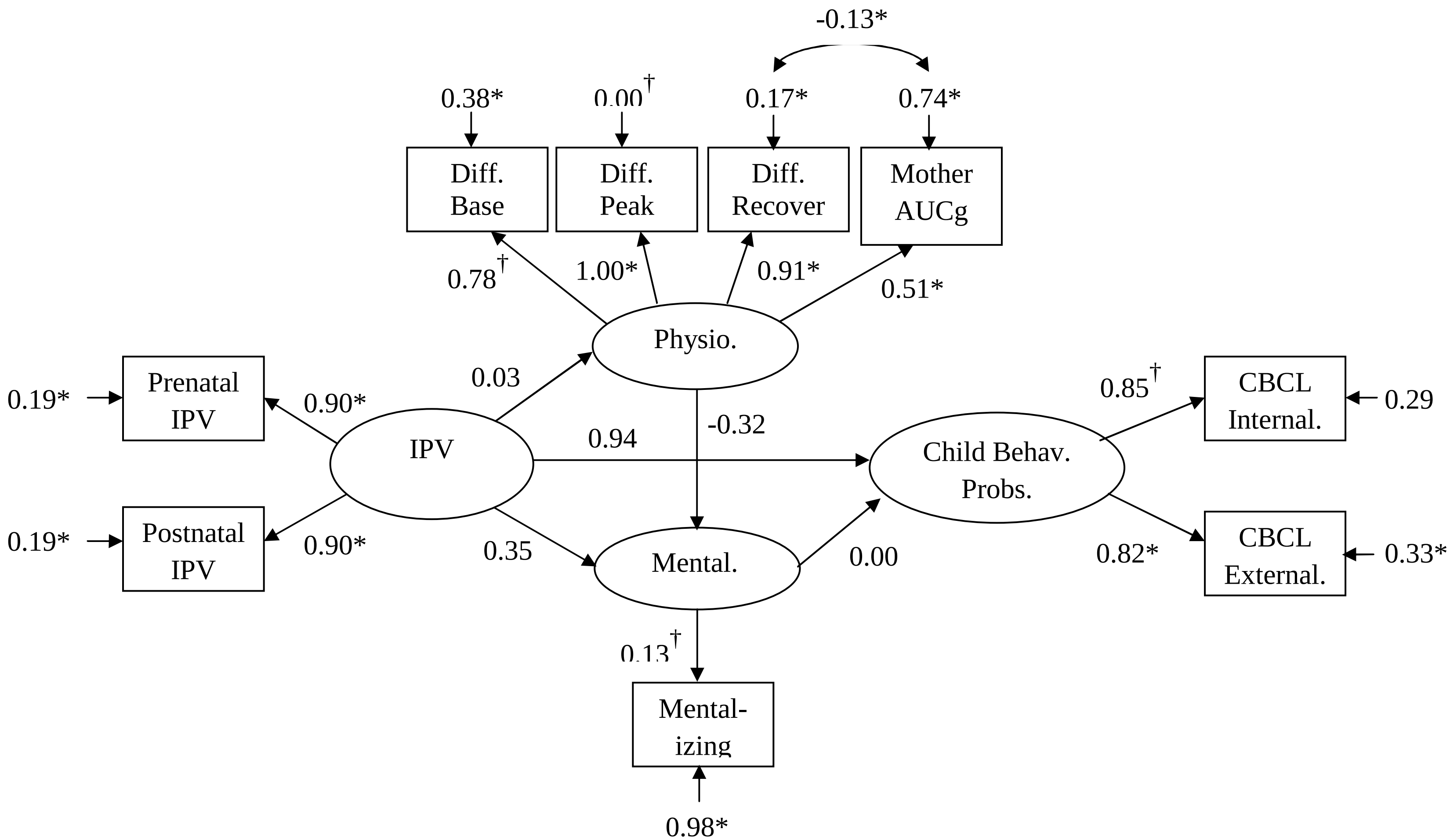
*SEM #4: Full Model Using In-Vivo Attunement Only*




Note: *$p$ < .05; †significance test is not conducted; Chi-Square = 33.46 ($df$ = 24), $p$ = 0.095; RMSEA = 0.053 (90% C.I. = 0.00 – 0.09); CFI = 0.98; GFI = 0.95; SRMR = 0.045

Figure 7.6

*SEM #5: Full Model Using Mentalizing Only*




Note: *$p$ < .05; †significance test is not conducted; Chi-Square = 30.54 ($df$ = 24), $p$ = 0.168; RMSEA = 0.044 (90% C.I. = 0.00 – 0.09); CFI = 0.98; GFI = 0.95; SRMR = 0.040

One additional version of the original Model #2 was also specified, this one predicting to mother reported child depression symptoms only, rather than to all mother reported behavior problems (both internalizing and externalizing). This model (Model #6) was specified based on the theoretical rational that physiological deregulation is often associated specifically with depressive symptomatology in children (Granger, Weisz & Kauneckis, 1994; Guerry & Hastings, 2011), rather than the wide array of behavior problems that are captured by the broadband behavior scales of the CBCL. This model was specified in the same way as Model #2; however rather than predicting to child behavior problems, IPV and affective attunement predicted to a child depression construct as indicated by the anxious/depressed and withdrawn scales of CBCL. In order to achieve convergence, the model was estimated using correlation matrices to correct for scaling problems, and the unweighted least squares method was used. Additionally, to correct for negative variances, neither the variance for the affective attunement factor, nor the residual for the difference in peak cortisol were estimated. Finally, the residuals for pre-natal and post-natal IPV were set equal, and the residuals for anxious depressed symptoms and withdrawn symptoms were set equal. The model, which can be found in Figure 7.7 below with standardized estimates (correlation matrix can be found in Table 7.10 below) had good fit, exceeding all 5 *a-priori* selected fit indices. All of the significant pathways from the original model remained significant in this version of the model, with IPV predicting less affective attunement and more child depressive symptomatology, and greater physiological attunement predicting less affective attunement; the pathways between IPV and physiological attunement and affective attunement and child depressive symptomatology did not achieve significance.

Table 7.10

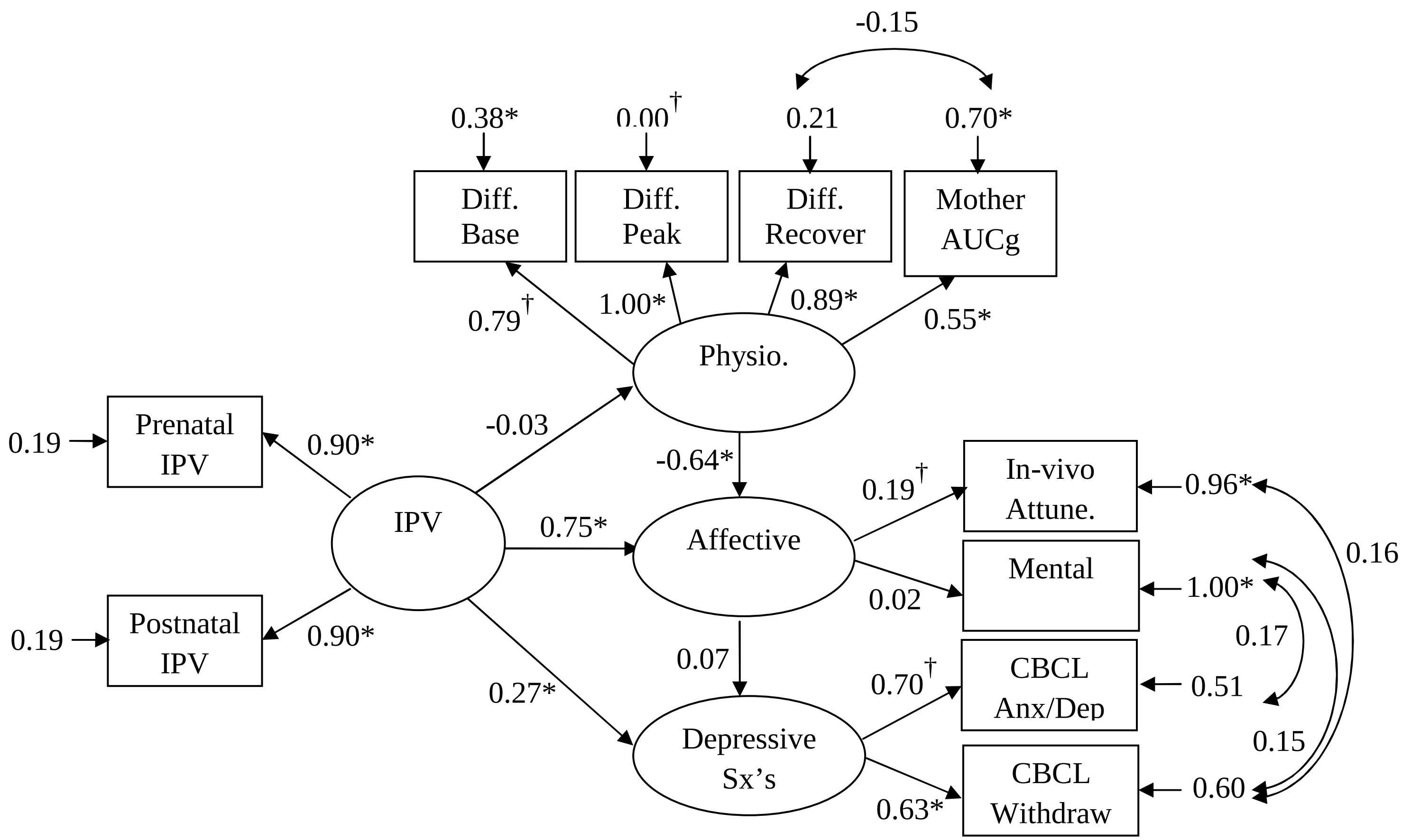
*Correlation Matrix for Structural Equation Model #6*

| Variable | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|---|
| 1. Prenatal IPV | -- | .813** | -.055 | -.021 | .018 | -.055 | .167* | .041 | .300** | .107 |
| 2. Postnatal IPV | | -- | -.075 | -.066 | -.013 | -.056 | .076 | .179* | .273** | .063 |
| 3. Baseline Cortisol Diff. | | | -- | .632** | .682** | .186* | .102 | -.036 | .012 | -.058 |
| 4. Peak Cortisol Difference | | | | -- | .847** | -.402** | .194* | -.004 | .003 | -.093 |
| 5. Recovery Cortisol Diff. | | | | | -- | -.138 | .145 | .029 | -.012 | -.068 |
| 6. Mother AUCg | | | | | | -- | -.047 | .062 | -.129 | -.014 |
| 7. In-vivo Attunement | | | | | | | -- | -.050 | .017 | .184* |
| 8. Mentalizing | | | | | | | | -- | .164 | .159 |
| 9. CBCL Anx/Dep | | | | | | | | | -- | .455** |
| 10. CBCL Withdrawn | | | | | | | | | | -- |

*Note:* ***p* < .01; **p* < .05

Figure 7.7

*SEM #6: IPV, Attunement, Mother Reported Depressive Symptoms*




Note: *$p$ < .05; †significance test is not conducted; Chi-Square = 42.67 ($df$ = 30), $p$ = 0.063; RMSEA = 0.055 (90% C.I. = 0.00 – 0.09); CFI = 1.00; GFI = 0.98; SRMR = 0.052

One final structural equation model was estimated in order to examine physiological attunement based on correlations between mother and child cortisol values, rather than using difference scores (Model #7). In this model, two latent constructs, one representing mother cortisol (as indicated by mothers' baseline, peak and recovery cortisol values) and the other representing child cortisol (as indicated by children's baseline, peak and recovery cortisol values) were included, and allowed to correlate. Due to the limited sample size and in order to have adequate power to run this model, affective attunement and child behavior problems were represented as manifest variables (using the mentalizing variable and mother report of child anxious/depressed symptoms respectively). These variables were selected for both theoretical and practical reasons. First, mentalizing and mother report of anxious/depressed symptoms correlated most strongly with the other variables of interest in the model. Additionally, as mentioned previously, physiological dysregulation is most often associated with depression symptoms in children.

In order to achieve model convergence, several modifications were made to the model. First, the model was specified using correlation matrices, rather than covariance matrices to correct for scaling problems. Additionally, to correct for negative values, the residuals for prenatal IPV and postnatal IPV were set equal to each other, and the residuals for child baseline cortisol, child peak cortisol and child recovery cortisol were set equal to each other. Finally, the model was estimated using the unweighted least squares (UL) method, as it would not converge using the maximum likelihood method. Several of the residuals were freed to correlate, including the residual for child baseline cortisol with the residual for mothers' baseline, peak and recovery cortisol (not shown in Figure 7.8 below so as not to complicate the model image). Even after these changes, the model demonstrated poor fit and is thus not interpretable.

Table 7.11

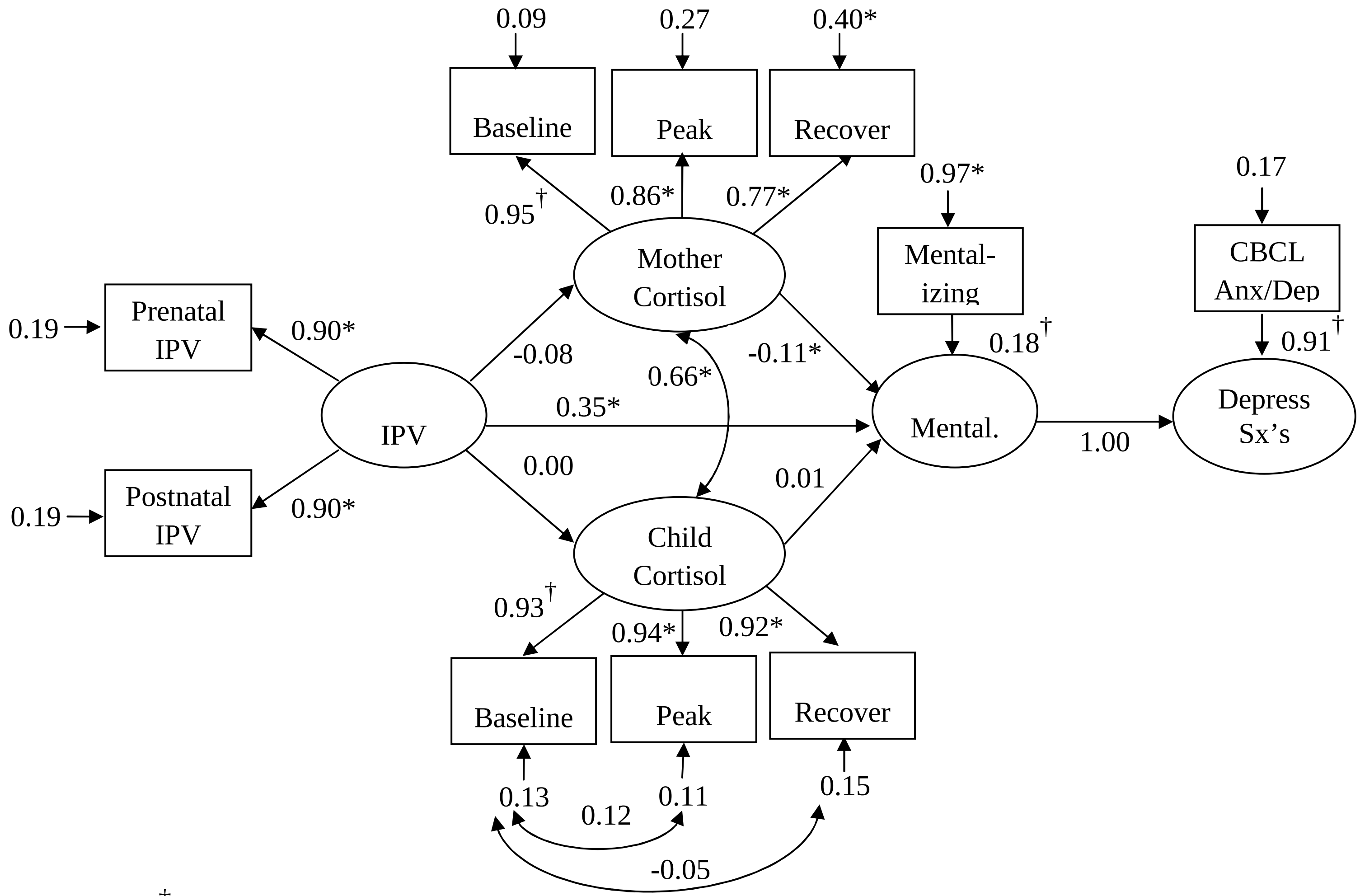
*Correlation Matrix for Structural Equation Model #7*

| Variable | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|---|
| 1. Prenatal IPV | -- | .813** | -.046 | -.056 | -.073 | .013 | - .006 | -.045 | .041 | .300** |
| 2. Postnatal IPV | | -- | -.028 | -.076 | -.095 | .042 | .021 | -.024 | .179* | .273** |
| 3. Mother Baseline Cortisol | | | -- | .821** | .730** | .587** | .693** | .472** | .053 | -.131 |
| 4. Mother Peak Cortisol | | | | -- | .910** | .379** | .644** | .398** | .055 | -.089 |
| 5. Mother Recovery Cortisol | | | | | -- | .240** | .595** | .341** | .064 | -.139 |
| 6. Child Baseline Cort. | | | | | | -- | .810** | .749** | .066 | -.100 |
| 7. Child Peak Cortisol | | | | | | | -- | .880** | .026 | -.040 |
| 8. Child Recovery Cort. | | | | | | | | -- | -.003 | -.041 |
| 9. Mentalizing | | | | | | | | | -- | .164 |
| 10. CBCL Anx/Dep | | | | | | | | | | -- |

*Note:* $**p < .01$; $*p < .05$

Figure 7.8

*SEM #7: Alternative Model of Physiological Attunement*




Note: *$p < .05$; †significance test is not conducted; Chi-Square = 86.39 ($df$ = 26), $p$ = 0.000; RMSEA = 0.128 (90% C.I. = 0.10 – 0.16); CFI = 1.00; GFI = 0.99; SRMR = 0.050

Finally, a number of PCFAs were conducted to examine the associations between IPV, adrenocortical functioning, affective attunement and child outcomes. A list of each of the PCFAs that were completed, including the variables that were included and how they were coded, as well as model fit information can be found in Table 7.12 below. For all of these PCFAs, a z-test with the Holland-Copenhaver procedure was used in the search for types and antitypes. In only three instances, the base model was not a good fit for the data. The first of these, Model #5, examined IPV (3 categories), mentalizing (2 categories), and children's internalizing problems (2 categories), and yielded 12 different configurations. The fit of the base model was $LR\,\chi^2$ (5, $N$ = 143) = 11.67, $p$ = .04; however no significant types or antitypes emerged, as seen in Table 7.13 below. Model #6 examined IPV (3 categories), mentalizing (2 categories), and children's externalizing problems, yielding 12 configurations. The fit of the base model was $LR\,\chi^2$ (5, $N$ = 143) = 11.55, $p$ = .04; however no significant types or antitypes emerged, as seen in Table 7.14 below. The last of these models, Model #8, examined IPV (3 categories), mentalizing (2 categories), and adaptability (2 categories). For this PCFA the base model was again not a good fit for the pattern of cell frequencies [$LR\,\chi^2$ (5, $N$ = 143) = 15.39, $p$ = .01]; however no significant types or antitypes were observed (see Table 7.15 below).

Table 7.12

*List of Predictive Configural Frequency Analyses (PCFA) Conducted*

| Model # | Variables Included & Coding | Model Fit Information |
|---|---|---|
| 1 | IPV: 1 = None, 2 = Minor, 3 = Severe<br>Base to Peak Cortisol: 1 = Not a Reactor, 2 = Reactor<br>In-Vivo Attunement: 1 = Not Attuned, 2 = Attuned | *LR* $\chi^2$ (7, *N* = 143) = 3.92, *p* = .79 |
| 2 | IPV: 1 = None, 2 = Minor, 3 = Severe<br>Base to Peak Cortisol: 1 = Not a Reactor, 2 = Reactor<br>Mentalizing: 1 = Poor Mentalizing,<br>2 = Good Mentalizing | *LR* $\chi^2$ (7, *N* = 143) = 2.92, *p* = .88 |
| 3 | IPV: 1 = None, 2 = Minor, 3 = Severe<br>In-Vivo Attunement: 1 = Not Attuned, 2 = Attuned<br>Internalizing: 1 = Positively Adapted (T- score < 60 on *either* CBCL or TRF)<br>2 = Negatively Adapted (T-score ≥ 60 on *either* CBCL or TRF | *LR* $\chi^2$ (5, *N* = 143) = 4.92, *p* = .43 |
| 4 | IPV: 1 = None, 2 = Minor, 3 = Severe<br>In-Vivo Attunement: 1 = Not Attuned, 2 = Attuned<br>Externalizing: 1 = Positively Adapted (T- score < 60 on *either* CBCL or TRF)<br>2 = Negatively Adapted (T-score ≥ 60 on *either* CBCL or TRF | *LR* $\chi^2$ (5, *N* = 143) = 9.80, *p* = .08 |
| **5** | **IPV: 1 = None, 2 = Minor, 3 = Severe**<br>**Mentalizing: 1 = Poor, 2 = Good**<br>**Internalizing: 1 = Positively Adapted,**<br>**2 = Negatively Adapted** | ***LR* $\chi^2$ (5, *N* = 143) = 11.67, *p* = .04** |
| **6** | **IPV: 1 = None, 2 = Minor, 3 = Severe**<br>**Mentalizing: 1 = Poor, 2 = Good**<br>**Externalizing: 1 = Positively Adapted,**<br>**2 = Negatively Adapted** | ***LR* $\chi^2$ (5, *N* = 143) = 11.55, *p* = .04** |
| 7 | IPV: 1 = None, 2 = Minor, 3 = Severe<br>Attunement: 1 = Not Attuned, 2 = Attuned<br>Adapted: 1 = positively adapted (T-scores < 60 for int. & ext. on CBCL & TRF)<br>2 = negatively adapted (a T-score ≥ 60 for int. and/or ext. on CBCL and/or TRF) | *LR* $\chi^2$ (5, *N* = 143) = 9.62, *p* = .09 |
| **8** | **IPV: 1 = None, 2 = Minor, 3 = Severe**<br>**Mentalizing: 1 = Poor, 2 = Good**<br>**Adapted: 1 = Positively Adapted,**<br>**2 = Negatively Adapted** | ***LR* $\chi^2$ (5, *N* = 143) = 15.39, *p* = .01** |

Table 7.12 (cont'd)

| | | |
|---|---|---|
| 9 | IPV: 1 = None, 2 = Minor, 3 = Severe<br>Cortisol Pattern: 1 = Significant peak, no recovery,<br>2 = Significant peak and recovery,<br>3 = No significant peak<br>Adapted: 1 = Positively Adapted,<br>2 = Negatively Adapted | *LR* $\chi^2$ (8, *N* = 143) = 10.27, *p* = .25 |

Table 7.13

*PCFA #5: IPV, Mentalizing & Child Internalizing Problems*

| Variables | | | CFA | | | |
|---|---|---|---|---|---|---|
| IPV | Mental. | External. | *ƒ*oijkl | *ƒ*eijkl | zijkl | *p*ijkl |
| None | Poor | PA | 7 | 8.98 | -0.66 | .254 |
| None | Poor | NA | 5 | 3.02 | 1.14 | .127 |
| None | Good | PA | 10 | 7.48 | 0.92 | .179 |
| None | Good | NA | 0 | 2.52 | -1.59 | .056 |
| Minor | Poor | PA | 21 | 18.71 | 0.53 | .298 |
| Minor | Poor | NA | 4 | 6.29 | -0.91 | .180 |
| Minor | Good | PA | 24 | 21.70 | 0.49 | .311 |
| Minor | Good | NA | 5 | 7.30 | -0.85 | .197 |
| Severe | Poor | PA | 28 | 30.68 | -0.48 | .314 |
| Severe | Poor | NA | 13 | 10.32 | 0.83 | .202 |
| Severe | Good | PA | 17 | 19.46 | -0.56 | .289 |
| Severe | Good | NA | 9 | 6.55 | 0.96 | .169 |

*Note*: *LR* $\chi^2(5, N = 143) = 11.67$, $p = .039$; z-test with Holland-Copenhaver procedure; *ƒ*o = observed frequency; *ƒ*e = expected frequency; PA = Positively Adapted; NA = Negatively Adapted.

Table 7.14

*PCFA #6: IPV, Mentalizing & Child Externalizing Problems*

| Variables | | | CFA | | | |
|---|---|---|---|---|---|---|
| IPV | Mental. | External. | $f$oijkl | $f$eijkl | zijkl | *p*ijkl |
| None | Poor | PA | 10 | 8.81 | 0.40 | .344 |
| None | Poor | NA | 2 | 3.19 | -0.67 | .253 |
| None | Good | PA | 10 | 7.34 | 0.98 | .163 |
| None | Good | NA | 0 | 2.66 | -1.63 | .052 |
| Minor | Poor | PA | 16 | 18.36 | -0.55 | .291 |
| Minor | Poor | NA | 9 | 6.64 | 0.91 | .180 |
| Minor | Good | PA | 23 | 21.29 | 0.37 | .356 |
| Minor | Good | NA | 6 | 7.71 | -0.61 | .269 |
| Severe | Poor | PA | 31 | 30.12 | 0.16 | .435 |
| Severe | Poor | NA | 10 | 10.90 | -0.27 | .393 |
| Severe | Good | PA | 15 | 19.09 | -0.94 | .175 |
| Severe | Good | NA | 11 | 6.91 | 1.56 | .060 |

*Note*: *LR* $\chi^2(5, N = 143) = 11.55$, $p = .041$; z-test with Holland-Copenhaver procedure; $f$o = observed frequency; $f$e = expected frequency; PA = Positively Adapted; NA = Negatively Adapted.

Table 7.15

*PCFA #8: IPV, Mentalizing & Child Adaptability*

| Variables | | | CFA | | | |
|---|---|---|---|---|---|---|
| IPV | Mental. | Adapt. | $f$oijkl | $f$eijkl | zijkl | *p*ijkl |
| None | Poor | PA | 6 | 7.64 | -0.59 | .277 |
| None | Poor | NA | 6 | 4.36 | 0.78 | .217 |
| None | Good | PA | 10 | 6.36 | 1.44 | .075 |
| None | Good | NA | 0 | 3.64 | -1.91 | .028 |
| Minor | Poor | PA | 15 | 15.91 | -0.23 | .410 |
| Minor | Poor | NA | 10 | 9.09 | 0.30 | .382 |
| Minor | Good | PA | 22 | 18.46 | 0.83 | .205 |
| Minor | Good | NA | 7 | 10.55 | -1.09 | .137 |
| Severe | Poor | PA | 26 | 26.09 | -0.02 | .493 |
| Severe | Poor | NA | 15 | 14.91 | 0.02 | .491 |
| Severe | Good | PA | 12 | 16.55 | -1.11 | .132 |
| Severe | Good | NA | 14 | 9.46 | 1.48 | .070 |

*Note*: *LR* $\chi^2(5, N = 143) = 15.39$, $p = .008$; z-test with Holland-Copenhaver procedure; $f$o = observed frequency; $f$e = expected frequency; PA = Positively Adapted; NA = Negatively Adapted.

# Chapter 8: Discussion

In the current study, both variable-centered and person-centered data analytical approaches were used to examine the relationships among IPV exposure, adrenocortical attunement, affective attunement and children's behavior problems. This is only the second known study to examine the relationship between adrenocortical attunement and affective attunement in an IPV-exposed population, and the first study to examine the contribution of both types of attunement to the development of behavior problems in IPV-exposed children. This study also represents an important contribution to the literature because of several improvements it made upon the measurement of both attunement and IPV. These include examination of the contribution of prenatal IPV exposure to adrenocortical attunement (rather than postnatal IPV exposure alone), measurement of both the cognitive and behavioral aspects of affective attunement, and assessment of affective attunement in the context of child distress, which is the context in which affective attunement is thought to be most important for children's internalization of emotion regulation skills.

Specifically, this study sought to examine five research questions regarding the nature of adrenocortical and affective attunement in IPV-exposed mother-child dyads:

1. Is physiological dysregulation observed in IPV-exposed mothers and children in response to acute stress?
2. Is affective dysregulation observed in IPV-exposed mothers and children in response to acute stress?
3. How is IPV related to mother-child attunement both at the affective and physiological levels?

4. Is attunement in one system (affective, physiological) meaningfully associated with attunement in the other?
5. How are physiological and affective attunement related to child outcomes in terms of behavioral regulation (e.g., rates of internalizing and externalizing behavior problems)?

The following section will provide a brief summary of the findings from the current study as they pertain to each research question, as well as a discussion of the significance of these findings.

*(1) Is physiological dysregulation observed in IPV-exposed mothers and children in response to acute stress?* It was originally hypothesized that mothers and children exposed to greater levels of IPV would exhibit more dysregulated adrenocortical responses to acute stress, as indicated by higher baseline, peak and recovery cortisol levels in response to the lab stress task (hypercortisolism). It was also expected that some mothers exposed to IPV would exhibit a different form of physiological dysregulation, hypocortisolism, as indicated by lower baseline, peak and recovery cortisol levels in response to the lab stress task. In order to test these hypotheses, the relationship between IPV and adrenocortical reactivity for both mothers and children was examined using three different analyses. Overall, the findings from regression analyses, repeated measures ANOVAs and PCFAs used to test these hypotheses suggest there was no evidence of altered adrenocortical functioning as a result of IPV exposure in the current sample.

These null findings are inconsistent with prior research findings regarding cortisol reactivity and IPV-exposure, which has found evidence of altered adrenocortical reactivity in both violence-exposed women and children. For example, Schechter and colleagues (2004) found that IPV-exposed women exhibit lower baseline cortisol and greater peak cortisol reactivity compared to non-exposed women in response to a lab-based stress paradigm. With regard to IPV-exposed children, it was found that those exposed to greater IPV and other home environment stressors exhibit higher baseline and lower peak cortisol reactivity (Ellis, Essex & Boyce, 2005). In another study it was found that over time, children exposed to IPV exhibit increasingly higher peak cortisol levels as compared to non-exposed children (Hibel, Granger, Blair & Cox, 2011).

The discrepancy between these previous findings and the findings of the current study can be understood in several ways. First, it may be the case that IPV exposure is not meaningfully related to changes in HPA-axis functioning, suggesting that the findings from the previous studies may represent spurious associations between IPV exposure and altered adrenocortical reactivity. There is some evidence to support this idea. For example, a recent meta-analyses of adulthood trauma exposure and HPA-axis functioning (including basal cortisol, diurnal cortisol, reactivity to dexamethasone/corticotropin releasing hormone test, and reactivity to a psychological challenge) suggested no significant differences in baseline cortisol levels when comparing groups of non-traumatized individuals, trauma-exposed individuals without PTSD, and trauma-exposed individuals with PTSD (e.g., Klaassens, Giltay, Cuijpers, van Veen & Zitman, 2012). Similarly, Meewisse, Reitsma, de Vries, Gersons and Ollf (2007) found no differences in basal cortisol levels (diurnal or reactivity) when comparing samples of trauma-exposed adults with PTSD and controls without PTSD. Although these studies did not examine IPV specifically, the adulthood traumas in these studies were, like IPV, all chronically occurring (e.g., combat). The authors concluded that the results of the meta-analyses provide evidence that adulthood trauma does not noticeably alter the functioning of the HPA-axis (Klaassens et al., 2012).

Nonetheless, there is a great body of evidence that IPV exposure in both women and children *is* associated with HPA-axis alterations (e.g., Elzinga et al., 2008; Griffin, Resick & Yehuda, 2005; Inslicht, Marmar, Neylan, Metzler, Hart et al., 2006; Pico-Alfonso, Garcia-Linares, Celda-Navarro, Herbert & Martinez, 2004; Seedat et al., 2003). However, in most of this research, the alterations in adrenocortical functioning did not manifest in altered reactivity to acute stress, but rather in altered diurnal cortisol (e.g., Inslicht et al., 2006; Pico-Alfonso, 2004; Seedat et al., 2003). Thus, the lack of association between IPV and adrenocortical functioning in the current study may be a result of the decision to examine cortisol reactivity rather than diurnal cortisol. The distinction between examining diurnal cortisol and cortisol stress reactivity is an important one, as these two measurements provide different types of information. Although it is thought that both types of HPA-axis activity can be affected by exposure to chronic stress, the shape of the diurnal curve (particularly the steepness of the slope from waking to evening cortisol) is thought to be an important marker of risk for certain kinds physical health problems (e.g. fatigue, breast cancer, cardiovascular disease; aches and joint soreness; Conrad, Wilhelm, Roth, Spiegel & Taylor, 2008; Lovell, Moss & Wetherell, 2011; Nicolson, 2008) and mental health problems such as major depressive disorder (e.g., Dedovic, Engert, Duchesne, et al., 2010) and psychosis (e.g., Collip, Nicolson, Lardinois, et al., 2011). Measuring cortisol reactivity, however, can provide information about an individual's level of preparedness to respond to stressful events (baseline cortisol) and their habituation or sensitization to recurring stressful events (peak and recovery) (Nicolson, 2008). Given that the current study was primarily interested in how dyadic processes serve to help children develop self-regulation in the context of stressful events, it was more appropriate to measure cortisol reactivity. However, future research could improve upon the current study by measuring both diurnal cortisol rhythm and cortisol reactivity in the dyads, in order to determine whether IPV is meaningfully related to either form of HPA-axis alteration, and whether physiological attunement can be observed for either type of adrenocortical activity.

Finally, the lack of association between IPV and cortisol reactivity may be a result of the specific procedures used to induce HPA-axis reactivity in the current study. For example, although previous research using the beam-walking paradigm has been shown to induce an adrenocortical stress response in both women and children (Sethre-Hofstad, Stansbury & Rice, 2002), in the current study very few of the mothers exhibited a stress response to the paradigm (e.g., only 14% of women exhibited a significant increase in cortisol from the baseline to peak sampling period). Furthermore, prior to their children completing the beam task, 89% of the women rated themselves as not nervous (a 1 or 2 on the rating scale) and after the task, only 8% of the women reported that watching their child complete the beam walk was very or extremely stressful (5 or 6 on the rating scale). Nearly 15% of the children in the current study reported the beam task as being “a lot hard” or “very hard” (a 5 or 6 on the rating scale), and 34% showed a significant increase in cortisol from baseline to peak sampling. This suggests that while children found the beam walking task to be more stressful than their mothers, the task was not particularly stressful for either member of the dyads. This could be for a number of reasons. Research indicates that in order for an event to be experienced as stressful, it must be novel, unpredictable, and the individual must have low perceived control over the situation (Mason, 1968; Rose, 1984), or have a social-evaluation component (Dickerson & Kemeny, 2004). It is unclear how many of the children had prior exposure to walking on a high beam, as this was not measured in the study; however if this is a task children are generally familiar with, the paradigm may not have evoked a stress response for this reason. For mothers, knowing that they could stop the task at any point and that their child would be caught by research assistant should he/she fall may have decreased the sense of unpredictability and provided an increased sense of control during the task, also buffering against a stress response. Finally, using a stress paradigm that more closely simulated IPV-exposure, or that was more relational in nature, may have been more successful in eliciting cortisol reactivity in the current study.

(2) *Is affective dysregulation observed in IPV-exposed mothers and children in response to acute stress?* Similar to the original hypotheses about IPV and adrenocortical dysregulation, it was hypothesized that mothers and children exposed to greater levels of IPV would exhibit more dysregulated affective responses to acute stress, as indicated by higher self-report ratings of emotional distress experienced as a result of the lab stress task. The findings from hierarchical regression and repeated measures ANOVA indicated that there was no evidence of an association between IPV exposure and affective dysregulation in response to an acute stressor in the current sample. As noted above, one reason for this null finding is that the lab stress task used in this study was not similar in nature to an IPV event. Had the current study utilized a stress task more similar to IPV, more affective dysregulation may have been observed in the IPV-exposed dyads. For example, IPV-exposed infants have been found to show more emotional distress (e.g., freezing, hiding, escape/avoidance behavior, crying) in response to a simulated adult conflict (e.g., telephone argument between an experimenter and the infants' mothers) than non-exposed infants in a previous study of infant conflict sensitivity (DeJonghe, Bogat, Levendosky, von Eye & Davidson, 2005). Additionally, this study relied on self report of distress in order to assess for emotional dysregulation, thus the results may have been impacted by reporter biases such as faking good. Observer ratings of affective facial expressions and

emotional behavior for both the mother and child could be a useful method for eliminating reporter bias in future research.

(3) *How is IPV related to mother-child attunement both at the affective and physiological levels?* It was originally expected that IPV would be differentially associated with physiological attunement and affective attunement such that greater IPV exposure would be associated with greater attuned physiological responses in the dyads, but less affective attunement during the reunion episode following the lab stress-task. Additionally, in order to account for the heterogeneity in the types of adrenocortical alterations associated with IPV exposure (e.g., hypocortisolism versus hypercortisolism), it was expected that most IPV dyads would be adrenocortically attuned for exaggerated stress responses, while a smaller subset of dyads would not be attuned for physiological stress reactivity at all (these mothers and children would exhibit different patterns of cortisol reactivity). The results of this third research question are discussed in three separate subsections below: the first describing the specific relationship found between IPV and physiological attunement based on a model using only cortisol data, the next describing the specific relationship between IPV and physiological attunement using the full structural equation model with all variables of interest, and the last describing the specific relationship between IPV and affective attunement.

(3a) *How is IPV related to mother-child attunement at the physiological level – Results from structural equation modeling with cortisol data only.* In order to address the first hypothesis regarding the nature of IPV and adrenocortical attunement, a structural equation model replicated from a prior study by Hibel and colleagues (2009) was used. This model was able to examine both attunement of cortisol levels and attunement of the pattern of cortisol reactivity for the sample as a whole. The model indicated that mothers' and children's cortisol values were significantly correlated, or attuned, at baseline but not attuned with regard to the overall pattern of cortisol reactivity. This finding suggests that mothers and children are attuned in terms of their anticipatory state, or their state of readiness to respond to an acute stressor; however, once exposed to the stressor, the pattern of physiological responsivity differed for mothers and children.

This finding can be understood in different ways. For example, the lack of attuned response to acute stress found in this study may be reflective of the fact that most mothers did not find the beam task to be stressful while many of their children did. The finding that mothers and children are not attuned for pattern of reactivity may therefore be understood as an artifact of the type of stress paradigm used in the study. This finding may also be understood as a function of mothers' and children's ability to manage physiological arousal during the beam task. For example, even though mothers and children were set to respond similarly to the stress task as indicated by their attuned baseline values, developmental differences may have resulted in the mothers and children responding differently to the task (e.g., mothers' adrenocortical responses may have had more time to become habituated as compared to children; mothers may have more sophisticated cognitive skills to help them perceive the task as less stressful than their children).

The finding that mothers' and children's baseline cortisol levels were significantly correlated can also be understood as a function of mothers and their children being influenced by aspects of the shared environment in which they live together. For example, research from parent-offspring studies indicates that the majority of variance in evening cortisol for both parents and children can be explained by shared family environment rather than by genetic effects (e.g., Bartels et al., 2003; Schreiber et al., 2006; Van Hulle et al., 2012). This is true even after accounting for parental mental health, family socioeconomic status, sex and age (Schreiber at al., 2003). Given the fact that IPV is an aspect of shared family environment, it may be the case that current IPV is contributing to the attuned baseline levels of cortisol observed in this study. However this idea could not be directly assessed using the Hibel structural equation model due to limited sample size and the fact that the current study did not differentiate between current and past IPV exposure. Additionally, other aspects of the shared family environment that could also explain the correlation between mother and child baseline cortisol values, such as community violence, physical safety of the home, etc. were not measured in this study.

(3b) *How is IPV related to attunement at the physiological level – results from the overall structural equation model and PCFA.* In addition to replication of the Hibel model, the relationship between IPV and adrenocortical attunement was also assessed with several other structural equation models using difference scores between mothers' and children's baseline, peak and recovery cortisol values. When defined in this way using structural equation modeling, adrenocortical attunement was a good construct with strong indicator loadings. However, in all of the structural equation models where the construct was defined in this way, adrenocortical attunement was not significantly predicted by IPV, contrary to study hypotheses.

Finally, in predictive configural frequency analysis, IPV exposure was classified into three categories (no IPV exposure versus minor IPV exposure versus severe IPV exposure), and mothers and children were categorized based on whether they exhibited a significant change (of 10% or more) from baseline to peak sampling and from peak to recovery. Consistent with the findings of the structural equation models, the PCFAs did not produce any significant types or antitypes, providing further evidence to suggest there was no association between IPV and adrenocortical attunement in the current study.

Given that IPV was not meaningfully associated with mother or child cortisol values in the earlier hierarchical regressions and repeated measures ANOVAs, the lack of association between IPV and adrenocortical attunement in the full structural equation models was not unexpected. One explanation for the null finding is that the heterogeneity of adrenocortical functioning in the mothers and children may have washed out any meaningful associations between IPV and adrenocortical attunement. However the PCFAs, which can account for this heterogeneity, also failed to show an association between IPV and adrenocortical attunement, suggesting that this result is not an artifact of the statistical approach used. Alternatively, the lack of association between IPV and adrenocortical attunement in this study could also be the result of any of the methodological issues discussed earlier, including the use of cortisol reactivity rather than diurnal cortisol and the nature of the stress paradigm used to elicit a cortisol response in the dyads. Finally, the findings of the current study may simply suggest that IPV does not exert an influence on adrenocortical functioning at either the individual level or the dyadic level.

*(3c) How is IPV related to attunement at the affective level?* Structural equation modeling was also used to examine the association between IPV and affective attunement. The results indicated that greater IPV predicted less affective attunement, consistent with the study hypotheses. This finding is also consistent with previous research. For example, Casanueva and colleagues (2008) found that mothers who were currently experiencing IPV were less emotionally responsive to their children during a home observation compared to mothers who had an IPV history but were no longer exposed. Levendosky et al. (2006) found that mothers exposed to IPV had difficulty responding in a warm and sensitive manner to their infants, and were also more likely to act with hostility or be disengaged from their children than non-exposed

women.  A meta-analytic review by Krishnakumar & Buehler (2000) indicated that higher levels of marital conflict were related to less maternal sensitivity, as well.

One way IPV may affect attunement is by undermining mothers' perceptions of themselves as caregivers resulting in an abdication of the mothering role (Solomon & George, 2008).  The psychological plasticity that occurs as a woman enters motherhood allows for her to create a new schema of the self as a mother, but this same plasticity also makes her susceptible to the influence of other relational experiences during this time, as well.  Because it is a relational experience, IPV can significantly and negatively alter the woman's experience of herself as effective, agentic and worthwhile in relationships.  This undermining of the self may carry over to the woman's sense of self as caregiver for her child.  As a result the woman may disengage from her child, develop distorted interpretations of her child's affective cues, or become emotionally dysregulated by her child's own emotional distress.  Alternatively, children exposed to IPV may contribute to difficulties with attunement as a result of the impact of IPV on their emotional security.   IPV may lead a child to feel that his mother will not be physically or emotionally available to protect him when faced with threat, thus he may camouflage or distort his emotional bids, making it difficult for mothers to engage in attunement effectively (Davies & Wotiach, 2008).  Finally, it is possible that IPV impacts attunement through a "spillover effect", where the negative affect and hostility arising from IPV interactions spill over and come to influence the quality of mother-child interactions (Easterbrooks & Emde, 1988).  However, the current study did not explicitly measure any of these mechanisms, thus this remains an area for further research.  Additionally, in the current study, low ratings of affective attunement could represent either a lack of engagement with the child (e.g., mother's lack of responsivity to the child's bids), or inadequate responding (e.g., the mother's response did not calm the child or even further dysregulated the child), thus it remains unclear whether IPV is more predictive of certain types of lapses in attunement than others.

(4) *Is physiological attunement meaningfully associated with affective attunement?* It was originally hypothesized that greater adrenocortical attunement would be associated with less affective attunement. It was also hypothesized that adrenocortical attunement would partially mediate the relationship between IPV and affective attunement. The reasoning for this was that in a low-risk context, physiological attunement between a mother and child was thought to enhance the mother's ability to detect subtle emotion cues in her child, thus allowing her to react with more behaviorally attuned responses. However, in the context of IPV, it was thought that either the mother or the child, or both, would become so emotionally aroused that physiological attunement would serve only to dysregulate both dyad members, thus interfering with healthy affective attunement. These hypotheses were both tested through structural equation modeling.

The results of the structural equation modeling were in support of the first hypothesis, indicating an inverse relationship where more adrenocortical attunement predicted less affective attunement. However, the structural equation models did not provide any evidence in support of the mediation hypothesis, as there was no significant relationship between IPV and adrenocortical attunement. With regard to the first finding, it is important to note that although the difference in baseline cortisol, difference in peak cortisol, difference in recovery cortisol and area under the curve all loaded as significant indicators in this structural equation model, it is likely that the significant relationship between affective and adrenocortical attunement was driven primarily by the difference in baseline variable, as baseline was the only time mothers and children were significantly attuned for cortisol level (as indicated by the Hibel replication model). Despite this, the finding that adrenocortical attunement inversely predicts affective attunement is important for understanding the nature of attunement in the mother-child dyad, and is consistent with prior work on attunement. For example, some research shows that when a child expresses distress, the quality of maternal responding differs depending on whether the mother experiences empathy for the child's affective state or if she experiences emotional contagion (e.g., sharing of the child's emotional state). For example, work by Milner, Halsey and Fultz (1995) indicates that some mothers are more likely to share in the same affective experience as their infants, such that when their children cried the mothers themselves reported experiencing more distress, sadness and even hostility. These same mothers also showed less capacity for empathy across varying emotional states in their children (happy, quiet, crying), as characterized by poor perspective taking and less empathic concern. Mothers who demonstrated this increased contagion and decreased empathy for their children were found to be at greater risk for maltreating their children. Importantly, the results of this study suggest that 1) shared emotional arousal is not the same as cognitive empathy - the ability to make appropriate appraisals about other's affect and, 2) shared emotional arousal may be predictive of less sensitive responding in mothers.

These findings are important because although the above study defined emotional contagion as the sharing of an emotional state between two individuals, physiological attunement is very similar in nature to emotional contagion. Emotional contagion is considered a multi-level phenomenon, including not only the experience of complementary emotional states but also a coordination of neurophysiological and autonomic nervous system activity (Hatfield, Cacioppo & Rapson, 1994). Similarly, empathic responding in the above study was defined as the capacity for perspective taking and concern, similar to the construct of mentalizing in the current study. Thus, if physiological attunement is similar to emotional contagion and affective attunement is similar to empathic responding, then it is apparent from the results of the Milner et al. study that physiological attunement for distressing emotion may interfere with a mother's capacity to engage in affective attunement.

Furthermore, although this inverse relationship between physiological attunement and affective attunement is inconsistent with other previous studies (e.g., Ruttle, Serbin, Stack, Schwartzman & Shirtcliff, 2011; Sethre-Hofstad et al., 2002; van Bakel et al., 2008) which found that in non-IPV exposed dyads, physiological attunement is positively associated with adrenocortical attunement, the discrepancies may be the result of methodological differences in the measurement of the behavioral attunement variable. For example, Sethre-Hofstad et al. and van Bakel et al. measured maternal sensitivity as an indicator of affective attunement without taking into account the mentalizing aspect of attunement or the child's contribution to affective attunement. Conversely, in the current study affective attunement was measured as a two-dimensional construct (with both cognitive and behavioral components), and took into account both mothers' and children's behavior in the ratings. Additionally, all three previous studies measured affective attunement during separate free-play or teaching tasks that were not stressful in nature and were not temporally linked with the stress task. The current study, however, measured affective attunement during the reunion episode occurring directly after the stress task. This difference in type of paradigm used to assess attunement is of particular importance, given that Ruttle et al. (2011) found different associations between adrenocortical attunement and affective attunement depending on whether dyads faced a low-challenge or high-challenge situation (there was a positive association during the low-challenge task, but not during the high-challenge task). Thus, the different findings regarding the nature of the relationship between the two types of attunement is likely the result of using a high-challenge task in the current study, as opposed to a low-challenge task like a play or teaching paradigm as was used in previous studies. Both methodological choices yield important information. However, given that IPV dyads live in high-risk environments likely characterized by frequent high-challenge situations, it is particularly important to understand the association between adrenocortical and affective attunement using high-challenge paradigms, as did this study.

Additionally, adrenocortical attunement may share a different relationship with attunement when the cognitive component of attunement is taken into account along with behavioral responding, as occurred in this study. Although mentalizing, the cognitive component of affective attunement, may not always predict maternal behavioral responses perfectly (e.g., a mother could accurately interpret their child's affective state and still respond insensitively), it is important to understand the contribution of mentalizing to affective attunement. This is particularly true because there is some evidence suggesting that mentalizing differentially influences the quality of an individual's emotional responding depending on the emotional valence of the situation (Fonagy, Bateman & Luyten, 2012). For example, research shows low stress/threat and high stress/threat situations activate different neural circuitry in the brain, resulting in different types of emotional responding (e.g., Keysers & Gazzola, 2007; Mayes, 2006). Mild to moderately arousing situations, for example, stimulate the prefrontal cortex thus promoting executive functions such as planning, working memory and anticipatory responding (Lieberman, 2007). Under these circumstances, "controlled" mentalizing is most likely occur, allowing the individual to better reflect on his/her own emotions and the emotions of others, and be more purposeful in their emotional responding to others. Highly stressful or emotionally arousing experiences, on the other hand, lead to a shutting down of the prefrontal cortex and activation of the posterior and subcortical areas of the brain, resulting in more automatic and reflexive responding (Lieberman, 2007). When this occurs, controlled mentalizing is compromised, and automatic schemas about others' internal states dictate behavioral responding. By examining mentalizing as a component of affective attunement under the context of high stress (rather than in play contexts), the current study may have captured a different type of emotional phenomenon, thus explaining the inconsistency in findings between this and previous studies.

*(5) How are physiological and affective attunement (or lack thereof) related to child outcomes in terms of behavioral regulation (e.g., rates of internalizing and externalizing behavior problems)?* In this last set of hypotheses, it was expected that lower levels of affective attunement would directly predict higher levels of child behavioral problems. It was also hypothesized that greater IPV would predict more child behavior problems and that affective attunement would partially mediate the relationship between IPV and child behavior problems. Results indicated that greater IPV predicted greater behavior problems when using mothers' report of child behavior; however this relationship did not remain significant when using teachers' report of child behavior. Affective attunement did not mediate the relationship between IPV and mother-reported or teacher-reported child behavior problems.

The finding that IPV directly predicts increased behavior problems in children is consistent with previous research (e.g., Ehrensaft & Cohen, 2012; Malik, 2008; Onyskiw & Hayduk, 2001; Sternberg et al., 2006). However, the lack of association between IPV and child behavior problems when using teacher report is unexpected. This inconsistency may be explained by the fact that in general, mother and teacher report of problem behavior and psychiatric symptoms for preschool-aged children tends to be poorly correlated, a finding which has been documented repeatedly in previous research (e.g., Kinard, 1998; Munkvold, Lundervold, Lie & Manger, 2009; Roskam, Stiévenart, Meunier, Van de Moortele, Kinoo & Nassogne 2010; Strickland, Hopkins & Keenan, 2012; Vitaro, Gagnon & Tremblay, 1991; Youngstrom, Loeber & Stouthamer-Loeber, 2000). Furthermore, this research indicates that similar to the findings of the current study, teachers consistently report fewer internalizing and externalizing problems compared to parents (Youngstrom et al, 2000). For example, Strickland et al. (2012) found that using a structured clinical interview, mothers reported 2.5 more symptoms and clinical diagnoses of conduct disorder and oppositional defiant disorder for a sample of 3-5 year old preschool children than did teachers, independent of child age and gender.

This rater disagreement may be the result of individual level reporter characteristics, such as the reporter's own psychopathology or stress level (Youngstrom et al., 2000), or familiarity with child behavior. For example, mothers in the current study may have less knowledge about and familiarity with child behavior compared to the teachers, thus they may be more likely to over-pathologize developmentally normative behavior. Conversely, the Head Start sample used in this study comes from a high-risk population with many of children exhibiting problem behavior (Snell, Berlin, Voorhees, Stanton-Chapman & Hadden, 2012; Whittaker, Harden, See, Meisch & Westbrook, 2011; Yoshikawa & Knitzer, 1997) thus the teachers in the current study may have become desensitized with regard to what they consider problem behavior, resulting in underreporting of behavior problems. Cross-informant discrepancies may also be the result of children's behavior differing between the school and home contexts (e.g., Achenbach, McConaughy & Howell, 1987; Strickland et al., 2012). For example, the school context may place significantly more structure and behavioral demands on children, helping to externally regulate their behavior within the classroom setting. Conversely, well-developed family dynamics in the household environment may be more likely contribute to child behavioral difficulties (e.g., coercive parent-child exchanges). It is likely that the differences in mother and teacher responding in the current study are a combination of both individual and contextual factors. Nonetheless, the results of the current study have important implications for our understanding of child development in the context of IPV. For example, perhaps for the current sample, the teacher-child relationship or other aspects of the classroom environment are acting as a buffer against the detrimental effects of IPV on children's social-emotional development.

The structural equation model using mother-reported symptoms of child behavior problems indicated that attunement did not mediate the association between IPV and behavior problems. Predictive configural frequency analysis confirmed this null finding. The finding that attunement does not explain the association between IPV and child behavior problems can be interpreted in several ways. First, it may be that behavior problems will arise in the context of IPV despite the quality of mother-attunement because behavior problems are the result of some other mechanism, including the direct effects of IPV on children, or some other mediating variable that was not measured in the current study. For example, there is evidence from previous research that individual level factors pertaining to the child, such as how the child makes appraisals about IPV (e.g., if they engage in self-blame) and temperament (e.g., easy vs. difficult), as well as more distal factors, such as the quality of the neighborhood environment in which the child lives, all affect the degree to which children express behavior problems in the context of IPV (e.g., Fortin, Doucet & Damant, 2011; Martinez-Torteya, Bogat, von Eye & Levendosky, 2009; Minze, McDonald, Rosentraub & Jouriles, 2010; O'Campo, Caughy & Nettles, 2010).

Secondly, affective attunement may buffer against the development of child behavior problems in low-risk populations, but may not be enough to outweigh the direct effects of IPV on children.  For example, research indicates that in non-IPV exposed populations, lack of attunement can be important risk factor in the development of preschool-age children's externalizing problems (e.g., Ensor, Roman, Hart & Hughes, 2012), and increased levels of attunement an can act as a buffer against the development of externalizing behavior (e.g., Healey, Gopin, Grossman, Campbell & Halperin, 2010; Lunkenheimer, Olson, Hollenstein, Sameroff & Winter, 2011).

Thirdly, there may be some other aspect of parenting that better accounts for the relationship between IPV and child behavior problems other than attunement.  For example, research has indicated that other aspects of parenting such as overall warmth, facilitation of problem solving, encouragement of skill use, and use of harsh or inept discipline strategies mediate the relationship between IPV exposure and child internalizing problems (e.g., Gewirtz, DeGarmo & Medhanie, 2011) and externalizing problems (e.g., Huang, Wang & Warrener, 2010) even after accounting for maternal mental health symptoms.  Thus, successful mother-child attunement may be a corollary or byproduct of high levels of warmth in the mother-child relationship; however, it may not be the main aspect of the relationship that confers protection against the effects of IPV.

A final explanation for this null finding is that the pathway between IPV and attunement is significant for other types of outcomes rather than for internalizing and externalizing behavior problems.  For example, while affective attunement is thought to play a role in the development of self-regulation, perhaps it is more relevant for aspects of social-emotional development that are more closely related to the attunement construct itself, such as mentalizing, theory of mind, emotional recognition, and empathy in young children.  For example, research suggests that in low-risk samples, mother-child attunement contributes to the development of prosocial behavior such as sharing and helping behavior in preschool-age children (e.g., Ensor, Spencer & Hughes, 2010; Lindsey, Cremeens & Caldera, 2010). Attunement may be particularly important for the development of prosocial behavior as opposed to problem behaviors during the toddler and early childhood years, as this is the developmental period when children begin to understand how situations are linked to different emotions, as well as the subjectivity of emotional experiences (Harris, 1989; Harris, Johnson, Hutton, Andrews & Cooke, 1989). Research also indicates that children exposed to IPV express greater deficits in these domains of social-emotional functioning (e.g., Fantuzzo & Mohr, 1999; Katz, Hessler, & Annest, 2007). Thus future research should examine the role of the IPV and affective attunement in the development of children's theory of mind, empathy, and use of sharing and helping behavior in addition to internalizing and externalizing problems alone.

In sum, the current study sought to better understand associations between IPV exposure, adrenocortical attunement and affective attunement, and the contribution of such to the development of children's behavior problems. Both person-centered and variable-centered analytic approaches were utilized. The person-centered analysis (PCFAs) did not provide any evidence of adrenocortical dysregulation, adrenocortical attunement or associations between IPV exposure, attunement and child behavior problems. However, because none of the person centered analysis yielded significant results, it is reasonable to make generalizations from the variable-centered analyses alone. The results of the variable-centered analyses also indicated no evidence of altered adrenocortical functioning and only limited evidence of adrenocortical attunement in IPV-dyads. Specifically, IPV-exposed mothers and children did not look attuned for cortisol reactivity, but did exhibit attuned baseline cortisol levels, suggesting that they share similar states of readiness for responding to stress. This shared readiness to detect and respond to stress may be the result of shared environmental influences, including but not limited to current violence exposure. The study also found evidence of an association between IPV and affective attunement, as well as between adrenocortical attunement and affective attunement, but no evidence to suggest that adrenocortical attunement mediates the relationship between IPV and affective attunement. Additionally, although more IPV was found to predict less attunement and greater child behavior problems, there was no evidence to suggest that attunement mediates the relationship between IPV and child behavior problems. This may suggest that IPV and affective attunement may influence other aspects of child functioning, such as the development of theory of mind skills, rather than the development of behavior problems.

### *8.1 Limitations and future research*

As with any research, the current study has its limitations. As mentioned earlier, the current study examined altered adrenocortical functioning in the form of cortisol reactivity, but did not examine diurnal cortisol rhythms. Similarly, adrenocortical attunement was only measured in the form of attunement for reactivity, rather than for attunement of diurnal cortisol. Given that a number of studies have found evidence of altered diurnal cortisol in IPV-exposed women and children (e.g., Inslicht et al., 2006; Pico-Alfonso, 2004; Seedat et al., 2003), future research should examine whether IPV- exposed dyads exhibit coordination in their daily cortisol rhythms, and whether this has any impact on children's behavioral functioning.

Another potential limitation in the current study is the definition of adrenocortical attunement used. Although the current study replicated efforts of measuring adrenocortical attunement that have been used in previous research on this topic, there are different ways to define adrenocortical attunement that could also be considered in future research. For example, the current study measured mother and child cortisol at simultaneous sampling times; however this approach only allows us to see if similar cortisol levels co-occur in the mother and children. This approach does not allow us to test for dependency, or whether one dyad member's cortisol reactivity is influencing the other's. Future efforts to study adrenocortical attunement could utilize a staggered collection of cortisol to examine reciprocal influences of each dyad member on the other. Collecting cortisol in this way could also allow for the use of different statistical modeling, for example use of actor-partner interdependence models, to test for adrenocortical attunement. An additional consideration with regards to the measurement of attunement pertains to the timing of the measurement of adrenocortical attunement and affective attunement. Specifically, because the first cortisol measurement and the stress induction task were both conducted before affective attunement was assessed, the current study could only test for the influence of adrenocortical attunement on affective attunement. The influence of affective attunement on adrenocortical attunement could not be examined in the current study, nor could the current study assess for the presence of a bi-directional, or reciprocal, relationship between the two types of attunement.

The current study attempted to improve upon the methods for measuring affective attunement used in past research by using in-vivo observational coding, and by measuring both the mother's responding and the child's ability to use her responding to self-regulate. The current study also sought to improve upon previous research by taking into account both the cognitive component of attunement (accurately reading the child's emotional state) and the behavioral component of attunement (the sensitivity and appropriateness of the mother's responding). Although these are an important start to more fully capturing the affective attunement construct, future research could benefit from the development of more standardized tools to measure affective attunement. In particular, the mentalizing variable in the current study was calculated by asking mothers to rate their child's affect during the beam task and then calculating the difference between her rating and the child's self-rating of the beam task. Although this is an innovative way to measure this variable, future research could also make use of self-report measures of mentalizing in order to have multiple measurements of the construct.

The current study also improved upon previous research by utilizing both person-centered and variable-centered approaches. However, none of the person-centered analyses yielded significant results. One reason for this is that the current study may have been limited by not including other variables that are known to be related to IPV, adrenocortical dysregulation and child outcomes in the PCFAs. For example, previous research indicates that there are different profiles of adrenocortical dysregulation observed in IPV-exposed populations that can be differentiated by the presence or absence of IPV-related PTSD symptoms (e.g. Griffin et al., 2003; Miller et al., 2007; Schechter et al., 2004). Although maternal PTSD did not emerge as a significant covariate in this study, there may be more nuanced relationships between the variables of interest that may have emerged through the person-centered analyses had maternal PTSD been included in the PCFAs. Additionally, the person-centered analyses may have yielded significant results if other variables such as child age and child PTSD, which was not measured in the current study, were included in the PCFAs. The current study did not have adequate power to conduct PCFAs with these additional variables included; however this would be an important consideration in future research studies examining the associations between IPV, adrenocortical functioning, attunement and child outcomes.

Finally, although the current study utilized a validated stress paradigm from previous research with pre-school aged children, the type of stress paradigm used in future research on adrenocortical and affective attunement in IPV- exposed dyads should be of important consideration. In the current study, only some of the children and even fewer of the mothers responded to the beam walking task with a significant cortisol response. While individual variation in cortisol reactivity is expected, the results of this study suggest that when working with such high-risk samples, a different type of stress task may need to be used to elicit a stress response. Given the various stressors faced by these families, these mother-child dyads may have a higher threshold for perceiving an event as stressful, or for such mothers, seeing their child play on an object high off the ground may be a more commonplace event than it is for mothers from low-risk environments. Furthermore, given that IPV-exposed dyads live in high risk environments where adrenocortical and affective attunement may be particularly important for how children process and respond to witnessing violent incidents, it will be important for future studies to measure these variables using stress paradigms that more closely simulate IPV events.

### *8.2 Clinical and Research Implications*

Despite the above mentioned limitations, the current study yielded results that have important implications for both future research and for clinical practice. For example, the finding that IPV is associated with less affective attunement between mother and child is an important one. This result suggests that IPV does in fact have deleterious effects on mothers' ability to metalize their children's affective states and respond to such emotional states in a sensitive way. Often when young children are distressed they communicate this in overt ways, such as crying or engaging in proximity seeking. However, as children get older, they do not always express every affective experience openly, and cues of distress may become more subtle or even completely camouflaged. This may be especially true for children who experience IPV, as they may not trust in their mothers' ability to respond to their emotional needs sensitively, particularly if she is the perpetrator of violence in the home, or if she has abdicated the role of caregiver in response to violence from a male partner. This finding may suggest that an important point of entry for intervention with mother-child dyads experiencing IPV may be around affective attunement. For example, future clinical interventions may be focused on helping enhance IPV-exposed children's comfort with expression of affective needs, and/or increasing mothers' ability to read and respond to such affect cues. Interventions such as these may not necessarily decrease the likelihood that IPV-exposed children will develop internalizing or externalizing problems; however they may be useful for improving these children's capacity for theory of mind and other important pro-social skills, leading to improved social functioning and greater social support.

Another result from the current study with potential clinical implications is the finding that mothers and children had correlated baseline cortisol levels, suggesting that states of readiness for detecting and responding to threat are shared within the dyad. Although it is not clear why mothers and children are attuned for basal cortisol, previous research suggests that this similarity is likely the result of shared environment influences. Additionally, the finding that increased adrenocortical attunement is detrimental to successful affective attunement has important implications when taken into account with the finding of correlated basal cortisol in the dyads. If mothers and children are indeed experiencing a shared anticipatory response, this may make it difficult for both members of the dyads to use one another for comfort in times of distress or threat. Thus, helping the mother to reduce her anticipatory state through use of self-soothing or cognitive techniques may help reduce her child's state of readiness to react, and also help the mother herself to act as a more effective source of comfort and support when the dyad faces a stressor together.

There are also important research implications from this study. For example, the results of the structural equation modeling suggest that affective attunement is a construct with multiple dimensions, including both cognitive and behavioral aspects. Future research on affective attunement or similar constructs would benefit from measuring both dimensions of the construct and understanding how one influences the other. For example, it will be important to understand whether mentalizing is necessary and/or sufficient for appropriate maternal responding to occur. It will also be important to understand if there are some mothers who may be able to accurately mentalize their children's emotional state but still have difficulty generating a sensitive behavioral response for their children.

The current study also found that children's behavior is different across contexts, for example the home and the school. This suggests that when studying the role of dyadic processes such as attunement in future research, it will be important to clarify whether or not these processes influence children's emotional development across contexts. For example, in the home environment, affective attunement may be an important risk factor for or buffer against the development of child problems; however these influences may not generalize over to other environments outside the home. Additionally, it is yet to be understood if attunement with other care-givers in contexts outside of the home can also act as risk or protective factors in children's development.

In summary, despite its limitations, the current study represents a significant contribution to the literature on the role of dyadic processes in the social-emotional development of IPV-exposed children. Although previous research has examined the relationship between adrenocortical and affective attunement, this is only the second known study to examine the relationship between these two types of attunement in an IPV-exposed population. Furthermore, while the previous studies of attunement in low-risk dyads found that greater adrenocortical attunement enhances affective attunement, the current study suggests that in high-risk populations adrenocortical attunement may not confer this same benefit. Additionally, this is the first known study to examine whether adrenocortical and affective attunement contribute to the development of behavior problems in children growing up in the context of IPV. Although affective attunement did not mediate the association between IPV and children's internalizing and externalizing problems, IPV predicted poorer affective attunement, which may contribute to other aspects of children's emotional develop such empathy and theory of mind skills. Although further research is needed to better understand the complex relationships between IPV, attunement and children's outcomes, the current study represents an early step in elucidating the nature of these relationships. Finally, the current study suggests that intervention strategies for high-risk and IPV-exposed populations should target mothers and children not only at the individual level, but at the dyadic level, as well.

APPENDICES

APPENDIX A – Recruitment Flier

Figure 9.1

*Recruitment Flier*

# The Healthy Moms - Healthy Kids Study!

## Do you have a child who is 3-5 years old?

### PARTICIPATE IN A STUDY ABOUT MOTHER-CHILD RELATIONSHIPS!

We need women to take part in a research interview about stress, parenting, the mother-child relationship, and child development.

- Interview takes place at Michigan State University and will last approximately 90 minutes.
- You must be the biological mother of your child, be between 18 – 36 years old, and you and your child must be in good health.
- You will be paid $75 in cash.
- All information is kept **completely confidential.**

*****************************************************

If you are interested or would like more information,
please call **(517) 432 - 1447**

Faculty Supervisor:
Dr. Alytia Levendosky
Rm. 46 Psychology Bldg

East Lansing, MI 48824

APPENDIX B – Intake Screen Form

# Healthy Moms – Healthy Kids
# Telephone Screening Protocol

"Thank you for your interest in the Healthy Moms – Healthy Kids Study! Before we begin, we need to tell you about the study and your rights as a potential research participant.

This research study is about stress, parenting, the mother-child relationship, and child development. If you are eligible, the information you provide will be used as part of this study. The information you provide during this brief screening, regardless of your eligibility, will be stored in a locked file cabinet in the Healthy Moms - Healthy Kids Study office. If you decide at any point now or in the future that you would like us to destroy and not use your information, we will do so. The possible risks of participating in this study are small and include possible discomfort from discussing sensitive topics. Do you have any questions or concerns about this?"

"Okay, I'd like to find out a little information from you to see if you are eligible to participate in the study. Are you able to answer a few questions right now? (Wait for participant to say yes or no. If no, thank the participant for calling and ask her to call back at a more convenient time.) The questions are about your age, education, income, and racial background as well as your experiences with your romantic partner, including types of conflict that you may have experienced. It should only take about 15 minutes. You can choose not to answer any of the questions or you can choose to end the telephone conversation whenever you want. Even after you answer all the questions, you can still choose to participate or not in the study. Any of these decisions will not affect your relationship with any agencies or Michigan State University. Do you have any questions?"

"If you have any questions as we go along, please ask me. If at any time you have concerns or questions about this study please contact Dr. Alytia Levendosky at (517) 432-1447. If you have questions or concerns about your role and rights as a research participant, would like to obtain information or offer input, or would like to register a complaint about this study you may anonymously contact the Michigan State University's Human Research Protection Program at 517-355-2180."

"Finally, there are no right or wrong answers to these questions. We want women in our study who have lots of different life experiences."

**Demographics & Identifying Information**

"Please spell your first and last name for me"

Name : ________________________________________

Phonetic spelling/pronunciation key:__________________________________

"What is your birth date?" (mm/dd/yyyy) ___________________

"How old are you right now?" Age:_________ → *If younger than 18 or older than 34 years discontinue interview*

Interviewer completes (circle one): **18-21** **22-25** **26-29** **30-34**

"What is **your** racial or ethnic group?" (Read options and circle one)
1 = Native American
2 = Asian American/Pacific Islander
3 = Black, African American
4 = Latino, Hispanic, Chicano
5 = Multiracial (multiracial means having parents of different races)
6 = Caucasian, White

Interviewer completes (circle one): **White** **Non-white**

"Please spell your child's name"

Name: ________________________________

Phonetic spelling/pronunciation: ____________________________

"What is your child's gender"? **Male** **Female**

"What is your child's birth date?" (mm/dd/yyyy) _____________________

"How old is your child right now?"

Age: ____________ → *If younger than 3 years OR older than 5 years, discontinue*

"What is **your child's** racial or ethnic group?" (Read options and circle one)
1 = Native American
2 = Asian American/Pacific Islander
3 = Black, African American
4 = Latino, Hispanic, Chicano
5 = Multiracial (multiracial means having parents of different races)
6 = Caucasian, White

*"Let me go ahead and get some contact information from you, in case we need to get in touch with you or get disconnected from you during the intake screening process."*

**Contact information**

Address:

___________________________________________________________________
Street number and name and apartment #

___________________________________________________________________
City, State, ZIP

Telephone #:______________________ h/c/w, ________________________h/c/w (**Always** get a second telephone number. If the mother does not have a second personal number, ask her for the number of a friend or family member that she would feel comfortable with us contacting to get in touch with her.)

1. Is it OK to contact you on the phone numbers you have provided? **YES** **NO**

If **NO,** is this because you think it may be hard to reach you directly?______
OR because you would rather not to have people in your household know about your participation in our study? ______ (check only one option)

2. When we call, do you have any preferences for any of the following? (read a through d and check all that apply)

_________a) block caller ID when calling
_________b) do not leave a message on answering machine
_________c) only call during the day; indicate specific preferred times:

________________________________________

________________________________________

_________d) only call during the evening; indicate specific preferred times:

________________________________________

________________________________________

_________e) no preference

Do you have an email address? _____________________________________

If **YES,** Is it OK to contact you at the e-mail address you have provided? **YES** **NO**

**INTERVIEWER, please transfer ID number here from previous pages → ID #: ________**

**Date: _________**

**Sensitive Information – TO BE STORED SEPARATELY!**

1. "What Head Start location does your child attend?"

   Head Start School: _______________________________ →*if child does not attend a Head Start school, discontinue the intake*[1]

2. "Is English your first language?" **YES NO**

   If **NO,** "Can you read and understand English?" **YES NO →** *If **NO,** discontinue*

3. "Are you the biological mother of this child?" **YES NO** → *if **NO**, discontinue*

4. "In the past year, have you always lived with your child?" **YES NO** → *if **NO**, discontinue*

5. "Was your child born premature?" **YES NO**
   If **YES**, how many weeks premature? ________ → *If below 37 weeks, discontinue*

6. "Does your child have any of the following:" → *If child has any of these, discontinue*

   Birth defects? Describe: _____________________________________________

   ________________________________________________________________

   Serious medical conditions? Describe: __________________________________

   ________________________________________________________________

   Significant developmental delays? Describe: _____________________________

   ________________________________________________________________

7. "Do you have legal custody of your child?" **YES NO** → *If **NO**, discontinue*

---

[1] Anytime the interviewer discontinues the interview, s/he says "*Thank you for calling us today. Unfortunately you are not eligible for our study. We have specific requirements in terms of (age/health status/relationship status) because we are looking at stress hormones and the effects of experiencing life stress on those hormones. We really appreciate the time you took calling our project and answering our questions! Thank you so much!"*

8. “Are you currently breastfeeding any infants or young children?” **YES NO**

    If **YES,** “if you are eligible to participate, would you be willing to not breastfeed for 2 hours prior to the in person interview?” **YES NO** → *If **NO**, discontinue*

*9.* “Are you currently pregnant?” **YES NO** → *If **YES**, discontinue*

*10.* “Do you currently have any of the following health problems?” (read list) → *If **YES,** discontinue*

| | |
|---|---|
| Cushings | **YES NO** |
| Addisons Disease | **YES NO** |
| Cancer | **YES NO** |

*11.* “Are you currently receiving cancer treatment?” **YES NO** → *If **YES** discontinue*

“Now I’d like to ask you some yes/no questions about your relationships with any romantic partners you may have had during your child’s entire lifetime. Note that this also includes partners who may not be your child’s biological father. Are you by yourself or, if not, are you able to answer these questions at this time?” (Wait for participant to say yes or no. If no, ask the participant to call back at a more convenient time.)

## Domestic Violence Screening

### CTS Postnatal

“No matter how well a couple gets along, there are times when they disagree on major decisions, get annoyed about something the other person does, or just have spats or fights because they’re in a bad mood, they are tired, or for some other reason. They also use many different ways of trying to settle their differences. I’m going to read the same list of some things that you and any of your partners might have done when you had a dispute. Think about each one of the items on the list as I read them to you. At the end, I will ask you if any of these things have happened to you with **ANY** of your romantic partners *since your child’s birth*.”

5. Threatened to hit or throw something at you
6. Threw or smashed or hit or kicked something
7. Threw something at you
8. Pushed, grabbed, or shoved you
9. Slapped you
10. Kicked, bit, or hit you with a fist
11. Hit or tried to hit you with something
12. Beat you up
13. Threatened you with a knife or gun
14. Used a knife or gun on you

Did any of these things happened to you *since your child’s birth?* **YES NO**

If **NO,** did anything else happen that I did not list here *since your child’s birth?*

Describe: ___________________________________

*"Thank you for sharing this information with us. The study manager will go over your information and a scheduler will call you back within the next week or so to let you know if you are eligible for our study."*

"Let me tell you something about the bigger research study. This research study is about stress, parenting, the mother-child relationship, and child cognitive, social and emotional development. One of the stressors we will ask you about is domestic violence. We hope to learn about the strengths that you bring to your situation, your feelings and perceptions of your child, and your child's behaviors both at home and at school. We hope to use this information to help plan better programs for families experiencing domestic violence. If you decide to take part in the study, you and your child will come to the campus of Michigan State University to participate. We will ask you questions about how you have been feeling recently, events that have happened to you including during your pregnancy, your feelings about your child, and the health and behavior of your child. We will also be asking you for 3 saliva samples (drool) from you and your child during the interview. This just involves spitting in a tube. Spitting in the tubes is easy to do, and it is not painful or dangerous. We use the saliva to measure the levels of a stress hormone (cortisol). After we measure the cortisol, we will destroy the samples immediately. We will not run any other biological tests on these samples. One of these saliva samples will be taken after your child engages in a challenging task. For this we will have your child walk across a balance beam. This activity is safe and voluntary. We will also ask your child to engage in a series of fun, interactive assessments to learn how children think. Your child can take a break and visit with you at any time.

The entire interview will take about 2 hours to complete. You will be paid $75 at the end of this interview to thank you for your participation.

"Do you have any questions?"
"Would you like to participate?" **YES** **NO**

*If **YES** → "Let me go ahead and schedule you for an interview. All of our interviews start at either 4 or 6:30 pm. Which day would work best for you?"*

"We offer care for one additional child on some interviews, will you need to bring another child to your interview?" **YES** **NO**

If **YES**: How many children and how old are them? ________________________

"We provide parking during the interview, will you be driving to our project office?"
**YES** **NO**

If **YES:** What is the model and color of the car you will be driving? A research assistant will be waiting for you by the parking lot, and this information will help her recognize you.

__________________________________
__________________________________

There are a few things we need you to avoid before your interview (Read list of things to avoid). We'll provide a snack and water for your child, but he/she will not be able to eat during about the first hour to the interview. Feel free to bring other food/drinks or sippy cup if s/he needs one.

APPENDIX C - Consent Forms

## Healthy Moms – Healthy Kids
## Mother's Consent Form

Thank you for your participation in The Healthy Moms – Healthy Kids Study! We appreciate your time and the effort you made to come in today. This research study is about stress, parenting, the mother-child relationship, and child development. One of the stressors we will ask you about is domestic violence. We hope to learn about the strengths that you bring to your situation, your feelings and views of your child, and your child's behaviors both at home and at school. We hope to use this information to help plan better programs for families experiencing domestic violence. Participation in all or part of this study is *completely* voluntary. You can choose not to answer any questions/questionnaires, or discontinue your participation at any time. If you choose to stop the interview you will still receive full payment for your participation.

<u>What will I do as part of the research study today?</u>

If you decide to take part in the study, you will be asked questions about romantic relationships and conflict you may have had in those relationships, events that have happened to you recently, your feelings about your child, your parenting style, and your child's behaviors at home. We will also be asking your permission to have your child's Head Start teacher fill out 2 brief questionnaires about your child's behavior at school (see the separate form "Mother's Permission for Teacher"). Additionally, we will ask you for 4 saliva samples (drool) from you and 4 from your child during the interview. This involves spitting in a tube—it is very easy to do and it is not painful or dangerous in any way. We will show you how to do this. Three of these saliva samples will be taken after your child does a mild stress task. For this your child will be asked to walk across a balance beam, with soft padding underneath it. You will be able to watch your child during this task but will not be able to help them walk across the beam. A research assistant will be there in case your child needs help with the task. The task is safe as the research assistant will nearby in case your child begins to fall and because there is ample padding underneath the beam. Additionally, the beam is wide enough for most children to walk across without trouble. However, you or your child can stop the task at *any time* if either of you feel uncomfortable.

<u>How long will this interview last today?</u>

The interview lasts approximately 90 minutes. You and your child may take breaks at any time. If you child finishes their portion of the interview early, we have lots of fun games and puzzles for them to play while you complete your interview.

<u>How will you protect my confidentiality?</u>

- <u>Question: Will my information/answers be shared with anyone at Head Start?</u>
- <u>Answer</u>: While Head Start is helping the Healthy Moms – Healthy Kids Study find families to participate in our research, <u>**none**</u> of the information you share during the research interview will be given to any person at Head Start. This includes information you share with us about yourself, your family and your child.

- <u>Question: Who will get to see the results of my child's questionnaires?</u>

- Answer: To protect your child's confidentiality, we will not be sharing their answers from the interview with you, or anyone at Head Start, with 3 exceptions: 1) If your child tells us they are being hurt, 2) If your child tells us they are going to hurt someone else, or 3) If your child tells us they are hurting or plan to hurt themselves. In any of these instances we would let you know.

Also, while we will be asking you for permission to have your child's teacher fill out behavior rating forms, personally identifiable information will not be shared with you, your child's teacher or anyone else at Head Start. Your child's teacher will not be interviewed for the study – they will only be answering questions on a paper-and-pencil questionnaire about your child's behavior at school. When the study is done, we will summarize information from all study participants and will not report information about individuals. This *anonymous summarized* information will be presented to Head Start so that they can provide better services to families.

- Question: How else will you keep my information/answers confidential?
- Answer: All information will be kept strictly confidential to the fullest level according to law. When you signed up for the study during the intake interview, we put your name, child's name, basic demographic information and contact information on a piece of paper along with a unique identification number. This paper is stored under lock and key and is kept separate from all other sensitive information you provided us during the intake screening interview. It will also be kept separate from the information you will provide today. Following the intake interview, this information can only be accessed by the project investigators and research assistants responsible for scheduling interviews. Your name, unique identification number, and contact information is also stored in a digital file that is password protected and stored in a locked office on a computer that is not connected to the internet. Only the project investigators and the research assistants who schedule visits have access to this digital file.
- All of the questionnaires you fill out today will have your identification number on them, so the information you provide today cannot be easily linked with your identity. Your full name will not be on any questionnaires or pieces of paper with your answers to our interview questions. All of the information you provide us today will be kept in locked file cabinets in a locked office at Michigan State University. All saliva samples will have the same identification number and they will be stored in a locked freezer and destroyed after analysis. Your identity will not be revealed in any reports written about this study. We will summarize information from all study participants and will not report information about individuals. The only exception is in the case of child abuse. If you indicate that child abuse is occurring or has occurred in your household, we must make a report to Protective Services. If a report to Protective Services is required, Head Start will *not* be told about this.

Why do you need saliva samples?

We use the saliva to measure levels of a stress hormone in the body called cortisol. After we test the levels of cortisol, we will destroy the samples right away. *We will not run any other tests on these samples.*

Are there any risks or direct benefits of participating for me and my child?

While you may not directly benefit from your participation in this study, your participation will help us to better understanding of the effects of domestic violence on women and young children. You may experience some benefits in telling your history and having someone who can help you find resources.

The possible risks of participating in this study are small and include possible upset or discomfort from discussing topics such as domestic violence. We will provide a list of resources for counseling and other services at the end of the interview.

Can I or my child stop participating if we want?

Yes, absolutely. You can participate in all parts of the study or just some parts. For example, you may want to answer the questions but you may not want your child to do the mild stress task. You have the right to withdraw from this study at any point during the interview with no penalty or negative costs, receiving full payment for your participation. Your decision about whether to participate or not will not affect your relationship with Head Start, Michigan State University, or any other agencies.

Will you videotape any parts of this interview?

We would like to videotape mild stress task that the research assistant will conduct with you and your child. This videotape will be used to make sure that the interviewer is conducting the task properly. The videotape may be coded by trained research assistants for your child's response to the task at the end of the study. The videotape will not have your or your child's name on it; an identification number will be put on it. We will keep this videotape in a locked file cabinet in a locked office at Michigan State University. When we are finished with all interviews and analysis using these data, we will destroy the videotapes (after approximately 24 months).

Videotaping of the mild stress task is *completely* voluntary. If you do not want to be videotaped, you can still do all of the parts of the interview and will not be penalized in any way. You and your child will receive the full payment for this interview, regardless of your decision about videotaping.

Who will have access to the videotape?

Only two groups of people will see the videotapes: the Healthy Moms – Healthy Kids study staff and, in the case of a research audit, the Michigan State University Institutional Review Board staff.

What if I have questions now or later?

If you have any questions now, please feel free to ask us at any time. If at any time you have concerns or questions about this study, such as scientific issues, how to do any part of it, or to report an injury, please contact Dr. Alytia Levendosky at (517) 432-1447.

If you:

- have questions or concerns about your role and rights as a research participant
- would like to obtain information or offer input
- would like to register a complaint about this study

you may anonymously contact the Michigan State University's Human Research Protection Program at 517-355-2180, Fax 517-432-4503, or e-mail irb@msu.edu or regular mail at 202 Olds Hall, MSU, East Lansing, MI 48824.

I have read this form and voluntarily agree that my child and I will participate in this research study.

______________________ ______________________ ______________

Signature of Participant Print Name Date

I have read this form and voluntarily agree to have the mild stress task videotaped for training and reliability purposes.

______________________ ______________________ ______________

Signature of Participant Print Name Date

______________________ ______________________ ______________

Signature of Witness Print Name Date

Alytia Levendosky, Ph.D.
107C Psychology Building
Michigan State University
Department of Psychology
East Lansing, MI 48824

# Healthy Moms - Healthy Kids
# Child Assent Form

*"If it's okay with you today, we will ask you to do some fun activities, like playing games, solving puzzles, and gym activities. We want to learn about kids like you! For some of these things we are going to use a camera to videotape you. Most kids think this stuff is really fun! If you don't want to do any of the activities it is okay. No one will be mad or upset. Just tell me. We will take lots of breaks and if you want to see your mom at any time, you can. When we are finished I have a gift for you so say "thank you" for helping us to learn about kids."*

*Does this sound okay to you – do you want to do the activities today? Yes or no?*

(Interviewer circle one based on child's <u>verbal</u> response): **YES** **NO**

**Healthy Moms – Healthy Kids Study**
**Mother's Permission Form for Teacher**

We would like your permission to have your child's teacher answer some questions about his /her observations of your child's behavior, feelings, and academic performance. Your child's teacher will be told that you are participating in a study of child development by Michigan State University, but nothing else about the study. No information that you give us will be shared with the child's teacher. If you agree to let us contact your child's teacher, please complete the release form below.

-----------------------------------------------------------------------------------------------------

As a parent of, _________________________, I give my permission for my child's teacher,
**Name of Child**

________________________, to answer questions about my child's behavior, feelings,
**Name of Teacher**

and academic performance for a study of child development that I am participating in at Michigan State University.

Parent Name (Print) ____________________________________________

Parent Signature _______________________________________________

Date _____________________

**Healthy Moms – Healthy Kids Study**
**Teacher Consent Form**

Dear Teacher:
The parent or guardian of your student ________________________has provided consented for us to contact you as part of their participation in the Healthy Moms – Healthy Kids Study. This research study is about the child cognitive, social and emotional development. We hope to use the information you share to help plan better strategies and interventions for parents and for teachers who work with young children. We are asking that you complete 2 questionnaires regarding this student's emotions, behavior, and academic performance. The questionnaires should take approximately 20-30 minutes to complete, and you will be paid $10 to thank you for your time. We would like to thank you in advance for your willingness to participate, and for the time and effort you are taking to help us. Your participation in this study is *completely* voluntary. <u>We will provide you with a copy of the release of information from the student's mother</u>.

<u>How will you protect my confidentiality?</u>
All information that you give us will be kept strictly confidential among the project staff. None of the information you provide will be shared with the student, his/her parent, or anyone else at Head Start. Neither your name nor the student's name will appear on any questionnaires. An identification number will be put on them instead. Everything you give us today will be kept in locked file cabinets in a locked office on the campus of Michigan State University. Your identity will not be revealed in any reports written about this study. When the study is completed, we will summarize information from all study participants and will not report information about individuals. This *anonymous summarized* information will be presented to Head Start so that they can provide better services to families.
Your privacy will be protected to the maximum extent allowable by law. The only exception to full confidentiality is in the case of ongoing child abuse or neglect. If you indicate that child abuse or neglect may be occurring in the child's household, we are required to make a report to Child Protective Services.

<u>How will you protect the confidentiality of the student for whom I completing these questionnaires?</u>
As part of their participation in this study, families are ensured confidentiality of all information shared regarding their child. This means that you will not have access to any of the information shared by parents regarding their child during their interview. This also means that the results of the questionnaires that you complete about the child will not be shared with you, the child's family, or anyone else at Head Start.

<u>What if I do not want to answer all or part of the questionnaires?</u>
You have the right to refuse to answer any questions at any point during the interview without penalty or negative consequences. Your decision about whether to participate or not will *not* affect your relationship with Head Start, Michigan State University, or any other agencies or Michigan State University.

<u>What if I have questions now or later?</u>

If at any time you have concerns or questions about this study, such as scientific issues, how to do any part of it, or to report an injury, please contact Dr. Alytia Levendosky at (517) 432-1447.

If you:

- have questions or concerns about your role and rights as a research participant
- would like to obtain information or offer input
- would like to register a complaint about this study

you may anonymously contact the Michigan State University's Human Research Protection Program at 517-355-2180, Fax 517-432-4503, or e-mail irb@msu.edu or regular mail at 202 Olds Hall, MSU, East Lansing, MI 48824.

---

I have read this form and voluntarily agree to participate in this research study.

| _______________________ | _______________________ | _______________ |
|---|---|---|
| Signature of Teacher | Print Name | Date |

| _______________________ | _______________________ | _______________ |
|---|---|---|
| Signature of Research Staff | Print Name | Date |

Alytia Levendosky, Ph.D.
107C Psychology Building
Michigan State University
Department of Psychology
East Lansing, MI 48824

APPENDIX D:
Measures

# Demographics Questionnaire

Subject # _____________
Date of Interview _____________
Name of Interviewer ______________________

1. First name of child: ____________________ **(Interviewer: fill in from assignment sheet prior to interview)**

2. Confirm Child's birthdate: ____/____/_____

3. Are you currently pregnant? **YES** **NO**

4. How many biological children do you currently have? ___________

5. How many people, including yourself, live in your household? _________
   **(If participant is living in a shelter, question refers to household composition before moving into shelter.)**

6. Choose the one that best describes your current marital/relationship status (choose only one):
   (1) single, never married
   (2) married (a) For how long? _______ (in months)
   (3) separated (b) For how long? _______ (in months)
   (4) divorced (c) For how long? _______ (in months)
   (5) widowed (d) For how long? _______ (in months)

7. How would you describe your relationship status with the biological father of your child? (Read all choices and circle one)
   (1) I am still in a relationship with him/her.

   (2) We have been together off and on and are currently together.

   (3)We have been together off and on and are currently <u>not</u> together.

   (4) I have not had a relationship with him/her since the child was born.

   (5) Is deceased.

8. What is your current relationship with the father of your child? (Circle one)

   1 = spouse
   2 = ex-spouse
   3 = partner/fiancé
   4 = ex-partner

5 = friend
6 = acquaintance
7 = stranger
8 = other Please specify: ______________________________

9. Is the child's father involved with the child? **YES** **NO**

10. Does the child's father live with the child? **YES** **NO**

11. Do you currently have custody of your child? **YES** **NO**
**IF NO,** go to question 15

11a. **IF YES**, is it full or joint? **FULL** **JOINT**

11b. **IF JOINT,** who do you share custody with? __________________
(Give name and relationship to child)

11c. **IF JOINT**, how many days per month does your child live with you? ________

12. In the last year, have you always had custody of your child? **YES** **NO**

**IF NO**, please give dates child was not in your custody and indicate who had custody at that time. **(Interviewer: if child is not currently in her custody, circle "present.")**

| Dates | Guardian of child **list role, for example, "foster mother" or "adoptive mother." Give relation to child if applicable; for example, "paternal grandmother," "maternal aunt.")** | How many days per month did/do you see your child? |
|---|---|---|
| ___/___ to ___/___ (present) | | |
| ___/___ to ___/___ (present) | | |
| ___/___ to ___/___ (present) | | |

13. In the past year, have you always lived with your child? **YES** **NO**

→ 13.a **IF NO**, how long did you live apart from your child?

**From**______ **to** ______
mo./yr. mo./yr.

**From**______ **to** ______
mo./yr. mo./yr.

14. What is your religious affiliation, if any? __________________________

15. What is your occupation status? Chose one of the following:

a. Unemployed
b. SSI Disability

c. Full-time employee
d. Part-time employee
e. Contract worker / per diem

16. What is the highest level of education you have completed? (Circle one)

a. Some High School
b. High School Diploma
c. GED
d. Some college, trade school (e.g., Cosmetology, Nursing, Technical/Vocational), or AA degree
e. BA/BS
f. Some grad school or graduate degree

17. Do you currently receive services from . . . ?

| | | |
|---|---|---|
| a. WIC | **YES** | **NO** |
| b. TANF (formerly AFDC) | **YES** | **NO** |
| c. Protective Services | **YES** | **NO** |
| d. Food Stamps | **YES** | **NO** |
| e. Medicaid | **YES** | **NO** |
| f. SSI (Disability) | **YES** | **NO** |
| g. FIA cash assistance/grant | **YES** | **NO** |

h. Any child related programs (e.g., 0-3; Mother-Infant Program; Head Start)? **YES NO**

**Please list:** ________________________________________

18. What is your total family income per month (estimate)?___________
→ **Note: <u>Must</u> be a value over $0 – include <u>all</u> forms of income, including, but not limited to, unemployment, disability, child support, food stamps, Medicaid, etc. PROBE EXTENSIVELY.**

19. Are you currently residing in a shelter for battered women?

(a) **YES** **NO**/888

(b) If **YES,** # days? ________

20. Have you ever stayed in a <u>shelter for battered women</u>?

(a) **YES** **NO**/888

(b) If **YES,** # days? ________

# Health and Quality of Saliva Screen

*"Because we'll be collecting saliva at this visit, I need to get some information about you and your child's current health. So next I'll ask questions about different kinds of things that might affect the hormone levels in your saliva."*

1. Do you have any current dental problems? For example, cuts or sores in mouth, bleeding gums during brushing, or untreated cavities? **YES NO**

2. Have you had your period in the last three months? **YES NO → *<u>If NO</u>*, *<u>SKIP TO #5</u>***

3. Are you currently having your period? **YES NO → *<u>If YES, SKIP TO #5</u>***

4. How many days ago did your period end? _______# of days **(DK=Don't Know)**

5. Are you currently using contraceptives? **YES NO**

    5a. Which kind? _____________________________

6. In the last 2 days (48 hours), have you taken any over-the-counter medicines or prescription medication (other than contraceptives)? **YES NO → *<u>If NO, SKIP TO #8</u>***

7. "Please tell me the following about each prescription medication or over-the-counter medicine you have taken in the last 2 days (48 hours). Please do not include contraceptives":

| Medication Type/Drug Name | What is this medication for? (**all** that apply)<br>1=mental health<br>2=allergy<br>3=inflammation<br>4=pain relief<br>5=other | Do you take this medication everyday or just when needed?<br>1=everyday<br>2=just when needed | How long ago did you last take this medication? *(answer in hours AND minutes)* |
|---|---|---|---|
| A) | | | ______hours AND<br>______minutes |
| B) | | | ______hours AND<br>______minutes |
| C) | | | ______hours AND<br>______minutes |
| D) | | | ______hours AND<br>______minutes |
| E) | | | ______hours AND<br>______minutes |

8. How many cigarettes did you smoke in the last 2 days (48 hours)? ______# of cigarettes

9. How many alcoholic drinks have you had in the last 2 days (48 hours)? ______# of drinks

10. Compared to others your age, would you say your health in the last 2 days (48 hours) was:
1=Excellent
2=Very Good
3=Good
4=Fair
5=Poor

11. Has your child had any immunization shot(s) within the past 3 days (72 hours)? **YES** **NO**
**→ *If NO, SKIP TO #12***

11a. How many days ago did he/she receive the last, that is, the most recent shot?
1 = Today
2 = Yesterday
3 = Day before yesterday
4 = 3 days ago

11b. What time of day did your child receive the last (most recent) immunization?
________________ (0:00-24:00)

12. In the last 2 days (48 hours) did your child take any over-the-counter or prescription medications (including aspirin, Tylenol, Pediacare)? **YES** **NO → *If NO, SKIP TO #13***

13. "Please tell me the following about each prescription medication or over-the-counter medicine that your child has taken in the last 2 days (48 hours)":

| Medication Type/Drug Name | What is this medication for? (**all** that apply)<br>1=mental health<br>2=allergy<br>3=inflammation<br>4=pain relief<br>5=other | Does the child take this medication everyday or just when needed?<br>1=everyday<br>2=just when needed | How long ago did the child last take this medication **(***answer in hours AND minutes)* |
|---|---|---|---|
| A) | | | ______hours AND<br>______minutes |
| B) | | | ______hours AND<br>______minutes |
| C) | | | ______hours AND<br>______minutes |
| D) | | | ______hours AND<br>______minutes |
| E) | | | ______hours AND<br>______minutes |
| F) | | | ______hours AND |

| | | | |
|---|---|---|---|
| | | | ______minutes |
| G) | | | ______hours AND<br>______minutes |

17. Compared to other babies his/her age, would you say his/her health in the last 2 days (48 hours) was:

    1=Excellent
    2=Very Good
    3=Good
    4=Fair
    5=Poor

## Saliva Collection Screening For Mother

"*I also need to get some information about different things such as eating, sleeping, mood, and body temperature that might affect the level of hormones that appear in your saliva. The first thing I'd like to do is take your temperature, and then ask a few questions.*"

INTERVIEWER: OBTAIN TEMPERATURE READING AND OTHER INFORMATION FOR WOMAN AND THEN FOR THE BABY.

1. Woman temperature: _______ ***(IF above 101, obtain second reading to make sure it is accurate. IF > 101 → reschedule )***

2. How many hours ago did you last eat/drink something other than water?
***(Record in hours AND minutes)***
**_______ # of hours AND ______ # of minutes DK=Don't Know**

**3**. How many hours ago did you last lie down to sleep? **(*Record in hours AND minutes)***
**_______ # of hours AND ______ # of minutes DK=Don't Know**

4. How long did you sleep? ***(Record in hours AND minutes)***
**_______ # of hours AND ______ # of minutes**

5. How would you describe this day as compared to others? Was it any more stressful?
**1=Much more**
**2=Slightly more**
**3=No different**
**4=Slightly less**
**5=Much less**

6. How would you describe your mood today? Any different than usual?
**1=Much worse**
**2=Slightly worse**
**3=No different**
**4=Slightly better**
**5=Much better**

## Saliva Collection Screening For Child

*"Next, I need to get your child's temperature and ask a few questions."*

1. Child's RIGHT temple temperature: **________ (If above 101, obtain reading from LEFT temple temperature, to make sure reading is accurate.** ***IF  > 101 → reschedule*****).**

2. How many hours ago did your child last eat/drink something other than water?
***(Record in hours AND minutes)***
**_______ # of hours and ______ # of minutes                    DK=Don't Know**

3. How many hours ago did your child last lie down to sleep? *(Record in hours AND minutes)*
**_______ # of hours and ______ # of minutes                    DK=Don't Know**

4. How long did your child sleep? ***(Record in hours AND minutes)***
**_______ # of hours and ______ # of minutes**

5. How would you describe your child's mood today? Any different than usual?
   - **1=Much worse**
   - **2=Slightly worse**
   - **3=No different**
   - **4=Slightly better**
   - **5=Much better**

## Saliva Sample Record

*"Next we'd like to collect the first saliva from you and your child.  We will collect saliva again 25 minutes after the end of the tasks."*

COLLECTION OF SALIVA SAMPLES 1 & 2

INTERVIEWER: FIRST COLLECT SALIVA FROM THE MOTHER AND THEN FROM THE CHILD.  AFTER YOU COLLECT EACH SAMPLE, RECORD THE NUMBERS FROM THE BAR CODE ON EACH VIAL.

**<u>Mother SALIVA SAMPLE #1</u>:**

| | | |
|---|---|---|
| 1. | Time of day Mother Sample 1 collection completed | ______ pm |
| 2. | Mother saliva sample 1 bar code # | <u>Bar Code for Vial</u><br><br>1) __ __ __-__-__ |
| 3. | INTERVIEWER: Were there any problems with collecting Sample 1 from the mother? | 0=No (SKIP to Child's Sample 1)<br>1=Yes (record notes below) |
| 4. | Note any special circumstances relating to Mother Sample 1 collection and/or reasons for not collecting (e.g., required repeated attempts, woman was too agitated, refused, other). | |

**<u>Child SALIVA SAMPLE #1</u>:**

| | | |
|---|---|---|
| 1. | Time of day Child Sample 1 collection completed | ______ pm |
| 2. | Child saliva sample 1 bar code # | <u>Bar Code for Vial</u><br><br>1) __ __ __ - __ - __ |
| 3. | INTERVIEWER: Were there any problems with collecting Sample 1 from the child? | 0=No (SKIP TO BEAM TASK)<br>1=Yes (record notes below) |
| 4. | Note any special circumstances relating to child Sample 1 collection and/or reasons for not collecting (e.g., required repeated attempts, child was too agitated, refused, other). | |

**BEAM TASK time of completion:** ______pm

**<u>Mother SALIVA SAMPLE #2</u>:**

| | | |
|---|---|---|
| 1. | Time of day Mother Sample 2 collection completed | (Actual)______ pm<br><br>(Expected = beam +5: _______pm) |
| 2. | Mother saliva sample 2 bar code # | <u>Bar Code for Vial</u><br><br>1) __ __ __-__-__ |
| 3. | INTERVIEWER: Were there any problems with collecting Sample 2 from the mother? | 0=No (SKIP to Child's Sample 2)<br>1=Yes (record notes below) |
| 4. | Note any special circumstances relating to Mother Sample 2 collection and/or reasons for not collecting (e.g., required repeated attempts, woman was too agitated, refused, other). | |

**<u>Child SALIVA SAMPLE #2</u>:**

| | | |
|---|---|---|
| 1. | Time of day Child Sample 2 collection completed | (Actual)______ pm<br><br>(Expected = beam +5: _______pm) |
| 2. | Child saliva sample 2 bar code # | <u>Bar Code for Vial</u><br><br>1) __ __ __ - __ - __ |
| 3. | INTERVIEWER: Were there any problems with collecting Sample 2 from the child? | 0=No (SKIP TO NEXT MEASURE)<br>1=Yes (record notes below) |
| 4. | Note any special circumstances relating to child Sample 2 collection and/or reasons for not collecting (e.g., required repeated attempts, child was too agitated, refused, other). | |

## Child Behavior Checklist – 1.5 - 5 (Achenbach & Rescorla, 2000)

Below is a list of items that describe children. For each item that describes the ***now or within the past 2 months***, please circle the ***2*** if the item is ***very true*** or ***often true*** of the child. Circle the ***1*** if the item is ***somewhat or sometimes true*** of the child. If the item is ***not true*** of the child, circle the ***0***. Please answer all items as well as you can, even if some do not seem to apply to the child.

**0 = Not True (as far as you know)**

**1 = Somewhat or Sometimes True**

**2 = Very True or Often True**

| | | | | |
|---|---|---|---|---|
| 1. | Aches or pains (without medical cause; ***do not*** include stomach or headaches) | **0** | **1** | **2** |
| 2. | Acts too young for age | **0** | **1** | **2** |
| 3. | Afraid to try new things | **0** | **1** | **2** |
| 4. | Avoids looking others in the eye | **0** | **1** | **2** |
| 5. | Can't concentrate, can't pay attention for long | **0** | **1** | **2** |
| 6. | Can't sit still, restless or hyperactive | **0** | **1** | **2** |
| 7. | Can't stand having things out of place | **0** | **1** | **2** |
| 8. | Can't stand waiting; wants everything now | **0** | **1** | **2** |
| 9. | Chews on things that aren't edible | **0** | **1** | **2** |
| 10. | Clings to adults or too dependent | **0** | **1** | **2** |
| 11. | Constantly seeks help | **0** | **1** | **2** |
| 12. | Constipated, doesn't move bowels (when not sick) | **0** | **1** | **2** |
| 13. | Cries a lot | **0** | **1** | **2** |
| 14. | Cruel to animals | **0** | **1** | **2** |
| 15. | Defiant | **0** | **1** | **2** |
| 16. | Demands must be met immediately | **0** | **1** | **2** |
| 17. | Destroys his/her own things | **0** | **1** | **2** |
| 18. | Destroys things belonging to his/her family or other children | **0** | **1** | **2** |
| 19. | Diarrhea or loose bowels (when not sick) | **0** | **1** | **2** |
| 20. | Disobedient | **0** | **1** | **2** |
| 21. | Disturbed by any change in routine | **0** | **1** | **2** |
| 22. | Doesn't want to sleep alone | **0** | **1** | **2** |

| | | | |
|---|---|---|---|
| 23. Doesn't answer when people talk to him/her | **0** | **1** | **2** |
| 24. Doesn't eat well | **0** | **1** | **2** |
| 25. Doesn't get along with other children | **0** | **1** | **2** |
| 26. Doesn't know how to have fun; acts like a little adult | **0** | **1** | **2** |
| 27. Doesn't seem to feel guilty after misbehaving | **0** | **1** | **2** |
| 28. Doesn't want to go out of home | **0** | **1** | **2** |
| 29. Easily frustrated | **0** | **1** | **2** |
| 30. Easily jealous | **0** | **1** | **2** |
| 31. Eats or drinks things that are not food – ***don't*** include sweets | **0** | **1** | **2** |
| 32. Fears certain animals, situations, or places | **0** | **1** | **2** |
| 33. Feelings are easily hurt | **0** | **1** | **2** |
| 34. Gets hurt a lot, accident-prone | **0** | **1** | **2** |
| 35. Gets in many fights | **0** | **1** | **2** |
| 36. Gets into everything | **0** | **1** | **2** |
| 37. Gets too upset when separated from parents | **0** | **1** | **2** |
| 38. Has trouble getting to sleep | **0** | **1** | **2** |
| 39. Headaches (without medical cause) | **0** | **1** | **2** |
| 40. Hits others | **0** | **1** | **2** |
| 41. Holds his/her breath | **0** | **1** | **2** |
| 42. Hurts animals or people without meaning to | **0** | **1** | **2** |
| 43. Looks unhappy without good reason | **0** | **1** | **2** |
| 44. Angry moods | **0** | **1** | **2** |
| 45. Nausea, feels sick (without medical cause) | **0** | **1** | **2** |
| 46. Nervous movements or twitching | **0** | **1** | **2** |
| 47. Nervous, highstrung, or tense | **0** | **1** | **2** |
| 48. Nightmares | **0** | **1** | **2** |
| 49. Overeating | **0** | **1** | **2** |
| 50. Overtired | **0** | **1** | **2** |
| 51. Shows panic for no good reason | **0** | **1** | **2** |
| 52. Painful bowel movements (without medical cause) | **0** | **1** | **2** |
| 53. Physically attacks people | **0** | **1** | **2** |
| 54. Picks nose, skin, or other parts of body | **0** | **1** | **2** |
| 55. Plays with own sex parts too much | **0** | **1** | **2** |

| | | | |
|---|---|---|---|
| 56. Poorly coordinated or clumsy | **0** | **1** | **2** |
| 57. Problems with eyes (without medical cause) | **0** | **1** | **2** |
| 58. Punishment doesn't change his/her behavior | **0** | **1** | **2** |
| 59. Quickly shifts from one activity to another | **0** | **1** | **2** |
| 60. Rashes or other skin problems (without medical cause) | **0** | **1** | **2** |
| 61. Refuses to eat | **0** | **1** | **2** |
| 62. Refuses to play active games | **0** | **1** | **2** |
| 63. Repeatedly rocks head or body | **0** | **1** | **2** |
| 64. Resists going to bed at night | **0** | **1** | **2** |
| 65. Resists toilet training | **0** | **1** | **2** |
| 66. Screams a lot | **0** | **1** | **2** |
| 67. Seems unresponsive to affection | **0** | **1** | **2** |
| 68. Self-conscious or easily embarrassed | **0** | **1** | **2** |
| 69. Selfish or won't share | **0** | **1** | **2** |
| 70. Shows little affection toward people | **0** | **1** | **2** |
| 71. Shows little interest in things around him/her | **0** | **1** | **2** |
| 72. Shows too little fear of getting hurt | **0** | **1** | **2** |
| 73. Too shy or timid | **0** | **1** | **2** |
| 74. Sleeps less than most children during the day and/or night | **0** | **1** | **2** |
| 75. Smears or plays with bowel movements | **0** | **1** | **2** |
| 76. Speech problem | **0** | **1** | **2** |
| 77. Stares into space or seems preoccupied | **0** | **1** | **2** |
| 78. Stomachaches or cramps (without medical cause) | **0** | **1** | **2** |
| 79. Rapid shifts between sadness and excitement | **0** | **1** | **2** |
| 80. Strange behavior | **0** | **1** | **2** |
| 81. Stubborn, sullen, or irritable | **0** | **1** | **2** |
| 82. Sudden changes in mood or feelings | **0** | **1** | **2** |
| 83. Sulks a lot | **0** | **1** | **2** |
| 84. Talks or cries out in sleep | **0** | **1** | **2** |
| 85. Temper tantrums or hot temper | **0** | **1** | **2** |
| 86. Too concerned with neatness or cleanliness | **0** | **1** | **2** |
| 87. Too fearful or anxious | **0** | **1** | **2** |
| 88. Uncooperative | **0** | **1** | **2** |

| | | | | |
|---|---|---|---|---|
| 89. | Underactive, slow moving, or lacks energy | **0** | **1** | **2** |
| 90. | Unhappy, sad, or depressed | **0** | **1** | **2** |
| 91. | Unusually loud | **0** | **1** | **2** |
| 92. | Upset by new people or situations | **0** | **1** | **2** |
| 93. | Vomiting, throwing up (without medical cause) | **0** | **1** | **2** |
| 94. | Wakes up often at night | **0** | **1** | **2** |
| 95. | Wanders away | **0** | **1** | **2** |
| 96. | Wants a lot of attention | **0** | **1** | **2** |
| 97. | Whining | **0** | **1** | **2** |
| 98. | Withdrawn, doesn't get involved with others | **0** | **1** | **2** |
| 99. | Worries | **0** | **1** | **2** |
| 100. | Please write in any problems the child has that were not listed above: | **0** | **1** | **2** |

____________________________________________________________

# Life Stressors Checklist – Revised (Wolfe & Kimerling, 1997)

Now we are going to ask you some questions about events in your life that are frightening, upsetting, or stressful to most people. Please think back over your whole life when you answer these questions.

1. Have you ever been in a serious disaster (for example, an earthquake, hurricane, large fire, explosion)?
   **YES NO**

2. Have you ever seen a serious accident (for example, a bad car wreck or an on-the-job accident?
   **YES NO**

3. Have you ever had a very serious accident or accident-related injury (for example, a bad car wreck or an on-the-job accident)?
   **YES NO**

4. Have you ever had a very serious physical or mental illness (for example, cancer, heart attack, serious operation, felt like killing yourself, hospitalized because of nerve problems)?
   **YES NO**

5. Have you ever had an abortion or miscarriage (lost your baby)?
   **YES NO**

6. Have you ever been separated from your child against your will (for example, the loss of custody or visitation or kidnapping)?
   **YES NO**

7. Have you ever been responsible for taking care of someone close to you (not your child) who had a severe physical or mental handicap (for example, cancer, stroke, AIDS, nerve problems, can't hear, see, walk)?
   **YES NO**

8. Has someone close to you died suddenly or unexpectedly (for example, sudden heart attack, murder, or suicide)?
   **YES NO**

9. Has someone close to you died (do NOT include those who died suddenly or unexpectedly)?
   **YES NO**

10. When you were young (before age 16), did you ever see violence between family members (for example, hitting, kicking, slapping, punching)?
    **YES NO**

11. Have you ever seen a robbery, mugging, or attack taking place?
    **YES NO**

12. Have you ever been robbed, mugged, or physically attacked (not sexually) by someone you did not know)?
    **YES NO**

13. Have you ever been bothered or harassed by sexual remarks, jokes, or demands for sexual favors by someone at work or school (for example, a coworker, a boss, a customer, another student, a teacher)?
    **YES NO**

14. Other than the situations described above, conflict or violence with a partner, or violent experiences with a parent during childhood, has anything else happened to you that was really scary, dangerous or violent?
    **YES NO**
    **IF YES,** what was the event?________________________________________

15. Have any of the events mentioned above ever happened to someone close to you so that even though you didn't see it yourself, you were seriously upset by it?
    **YES NO**
    **IF YES,** what was the event?_________________________________________

## Revised Conflicts Tactics Scales (Straus et al., 1996)

No matter how well a couple gets along, there are times when they disagree, get annoyed with the other person, want different things from each other, or just have spats or fights because they are in a bad mood, are tired, or for some other reason. Couples also have many different ways of trying to settle their differences. This is a list of things that might happen when you have differences. Please circle how many times you did each of these things since your child was born, and how many times your partner(s) did them since your child was born. If you or your partner(s) did not do one of these things since your child was born, circle "N/A".

**HOW OFTEN DID THE FOLLOWING HAPPEN SINCE YOUR CHILD WAS BORN:**

**A = ONCE**
**B = TWICE**
**C = 3 – 5 TIMES**
**D = 6 – 10 TIMES**
**E = 11 – 20 TIMES**
**F = MORE THAN 20 TIMES**
**N/A = THIS NEVER HAPPENED**

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 1. I showed my partner(s) I cared even though we disagreed. | A | B | C | D | E | F | N/A |
| 2. My partner(s) showed care for me even though we disagreed. | A | B | C | D | E | F | N/A |
| 3. I explained my side of a disagreement to my partner(s). | A | B | C | D | E | F | N/A |
| 4. My partner(s) explained his/her side of a disagreement to me. | A | B | C | D | E | F | N/A |
| 5. I insulted or swore at my partner(s). | A | B | C | D | E | F | N/A |
| 6. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 7. I threw something at my partner(s) that could hurt. | A | B | C | D | E | F | N/A |
| 8. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 9. I twisted my partner(s) arm or hair. | A | B | C | D | E | F | N/A |
| 10. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 11. I had a sprain, bruise, or small cut because of a fight with my partner(s). | A | B | C | D | E | F | N/A |
| 12. My partner(s) had a sprain, bruise, or small cut because of a fight with me. | A | B | C | D | E | F | N/A |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 13. I showed respect for my partners' feelings about an issue. | A | B | C | D | E | F | N/A |
| 14. My partner(s) showed respect for my feelings about an issue. | A | B | C | D | E | F | N/A |
| 15. I made my partner(s) have sex without a condom. | A | B | C | D | E | F | N/A |
| 16. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 17. I pushed or shoved my partner(s). | A | B | C | D | E | F | N/A |
| 18. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 19. I used force (like hitting, holding down, or using a weapon) to make my partner(s) have oral or anal sex. | A | B | C | D | E | F | N/A |
| 20. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 21. I used a knife or gun on my partner(s). | A | B | C | D | E | F | N/A |
| 22. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 23. I passed out from being hit on the head by my partner(s) in a fight with me. | A | B | C | D | E | F | N/A |
| 24. My partner(s) passed out from being hit on the head in a fight with me. | A | B | C | D | E | F | N/A |
| 25. I called my partner(s) fat or ugly. | A | B | C | D | E | F | N/A |
| 26. My partner(s) called me fat or ugly. | A | B | C | D | E | F | N/A |
| 27. I punched or hit my partner(s) with something that could hurt. | A | B | C | D | E | F | N/A |
| 28. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 29. I destroyed something belonging to my partner(s). | A | B | C | D | E | F | N/A |
| 30. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 31. I went to a doctor because of a fight with my partner(s). | A | B | C | D | E | F | N/A |
| 32. My partner(s) went to a doctor because of a fight with me. | A | B | C | D | E | F | N/A |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 33. I choked my partner(s). | A | B | C | D | E | F | N/A |
| 34. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 35. I shouted or yelled at my partner(s). | A | B | C | D | E | F | N/A |
| 36. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 37. I slammed my partner(s) against a wall. | A | B | C | D | E | F | N/A |
| 38. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 39. I said I was sure we could work out a problem. | A | B | C | D | E | F | N/A |
| 40. My partner(s) were sure we could work it out. | A | B | C | D | E | F | N/A |
| 41. I needed to see a doctor because of a fight with my partner(s), but I didn’t. | A | B | C | D | E | F | N/A |
| 42. My partner(s) needed to see a doctor because of a fight with me, but didn’t. | A | B | C | D | E | F | N/A |
| 43. I beat up my partner(s). | A | B | C | D | E | F | N/A |
| 44. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 45. I grabbed my partner(s). | A | B | C | D | E | F | N/A |
| 46. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 47. I used force (like hitting, holding down, or using a weapon) to make my partner have sex. | A | B | C | D | E | F | N/A |
| 48. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 49. I stomped out of the room or house or yard during a disagreement. | A | B | C | D | E | F | N/A |
| 50. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 51. I insisted on sex when my partner(s) did not want to (but did not use physical force). | A | B | C | D | E | F | N/A |
| 52. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 53. I slapped my partner(s). | A | B | C | D | E | F | N/A |
| 54. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 55. I had a broken bone from a fight with my partner(s). | A | B | C | D | E | F | N/A |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 56. My partner(s) had a broken bone from a fight with me. | A | B | C | D | E | F | N/A |
| 57. I used threats to make my partner(s) have oral or anal sex. | A | B | C | D | E | F | N/A |
| 58. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 59. I suggested a compromise to a disagreement. | A | B | C | D | E | F | N/A |
| 60. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 61. I burned or scalded my partner(s) on purpose. | A | B | C | D | E | F | N/A |
| 62. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 63. I insisted my partner(s) have oral or anal sex (but did not use force). | A | B | C | D | E | F | N/A |
| 64. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 65. I accused my partner(s) of being a lousy lover. | A | B | C | D | E | F | N/A |
| 66. My partner(s) me of this. | A | B | C | D | E | F | N/A |
| 67. I did something to spite my partner(s). | A | B | C | D | E | F | N/A |
| 68. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 69. I threatened to hit or throw something at my partner(s). | A | B | C | D | E | F | N/A |
| 70. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 71. I felt physical pain that still hurt the next day, because of a fight with my partner(s). | A | B | C | D | E | F | N/A |
| 72. My partner(s) felt physical pain the next day, because of a fight we had. | A | B | C | D | E | F | N/A |
| 73. I kicked my partner(s). | A | B | C | D | E | F | N/A |
| 74. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 75. I used threats to make my partner(s) have sex. | A | B | C | D | E | F | N/A |
| 76. My partner(s) did this to me. | A | B | C | D | E | F | N/A |
| 77. I agreed to try a solution to a disagreement my partner(s) suggested. | A | B | C | D | E | F | N/A |

78. My partner(s) tried a solution I suggested. A B C D E F N/A

## Psychological Maltreatment of Women Inventory – Short Version (Tolman, 1995)

The questionnaire asks about actions you and your partner(s) may have experienced in any of your relationships since your child was born. Answer each item as carefully as you can by circling a number next to each statement according to the following scale:

**1 = NEVER**
**2 = RARELY**
**3 = OCCASIONALLY**
**4 = FREQUENTLY**
**5 = VERY FREQUENTLY**

**SINCE YOUR CHILD WAS BORN:**

| | | | | | |
|---|---|---|---|---|---|
| 10a. My partner(s) called me names. | 1 | 2 | 3 | 4 | 5 |
| 10b. I called my partner(s) names. | 1 | 2 | 3 | 4 | 5 |
| 11a. My partner(s) swore at me. | 1 | 2 | 3 | 4 | 5 |
| 11b. I swore at my partner(s). | 1 | 2 | 3 | 4 | 5 |
| 12a. My partner(s) yelled and screamed at me. | 1 | 2 | 3 | 4 | 5 |
| 12b. I screamed and yelled at my partner(s). | 1 | 2 | 3 | 4 | 5 |
| 13a. My partner(s) treated me like an inferior. | 1 | 2 | 3 | 4 | 5 |
| 13b. I treated my partner(s) like an inferior. | 1 | 2 | 3 | 4 | 5 |
| 26a. My partner(s) monitored my time and made me account for my whereabouts | 1 | 2 | 3 | 4 | 5 |
| 26b. I monitored my partner(s) time and made him account for his whereabouts | 1 | 2 | 3 | 4 | 5 |
| 30a. My partner(s) used our money or made important financial decisions without talking to me about it. | 1 | 2 | 3 | 4 | 5 |
| 30b. I used our money or made important financial decisions without talking to my partner(s) about it. | 1 | 2 | 3 | 4 | 5 |
| 32a. My partner(s) was jealous or suspicious of my friends. | 1 | 2 | 3 | 4 | 5 |
| 32b. I was jealous or suspicious of my partner(s) friends. | 1 | 2 | 3 | 4 | 5 |

| | | | | | | |
|---|---|---|---|---|---|---|
| 36a. | My partner(s) accused me of having an affair with another man. | 1 | 2 | 3 | 4 | 5 |
| 36b. | I accused my partner(s) of having an affair with another woman. | 1 | 2 | 3 | 4 | 5 |
| 39a. | My partner(s) interfered in my relationship with other family members. | 1 | 2 | 3 | 4 | 5 |
| 39b. | I interfered in my partner(s)’ relationship with other family members. | 1 | 2 | 3 | 4 | 5 |
| 40a. | My partner(s) tried to keep me from doing things to help myself. | 1 | 2 | 3 | 4 | 5 |
| 40b. | I tried to keep my partner(s) from doing things to help himself. | 1 | 2 | 3 | 4 | 5 |
| 42a. | My partner(s) restricted my use of the telephone. | 1 | 2 | 3 | 4 | 5 |
| 42b. | I restricted my partner(s) use of the telephone. | 1 | 2 | 3 | 4 | 5 |
| 45a. | My partner(s) told me my feelings were irrational or crazy. | 1 | 2 | 3 | 4 | 5 |
| 45a. | I told my partner(s) his feelings were irrational or crazy. | 1 | 2 | 3 | 4 | 5 |
| 46a. | My partner(s) blamed me for his problems. | 1 | 2 | 3 | 4 | 5 |
| 46b. | I blame my partner(s) for my problems. | 1 | 2 | 3 | 4 | 5 |
| 49a. | My partner(s) tried to make me feel crazy. | 1 | 2 | 3 | 4 | 5 |
| 49b. | I try to make my partner(s) feel crazy. | 1 | 2 | 3 | 4 | 5 |

## Edinburgh PDS

**INSTRUCTIONS: Please circle the answer that comes closest to how you have felt _IN THE PAST 7 DAYS_.**

1. I have been able to laugh and see the funny side of things
    - 0 As much as I always could
    - 1 Not quite so much now
    - 2 Definitely not so much now
    - 3 Not at all

2. I have looked forward with enjoyment to things
    - 0 As much as I ever did
    - 1 Rather less than I used to
    - 2 Definitely less than I used to
    - 3 Hardly at all

3. I have blamed myself unnecessarily when things went wrong
    - 3 Yes, most of the time
    - 2 Yes, some of the time
    - 1 Not very often
    - 0 No, never

4. I have been anxious or worried for no good reason
    - 0 No, not at all
    - 1 Hardly ever
    - 2 Yes, sometimes
    - 3 Yes, very often

5. I have felt scared or panicky for no very good reason
    - 3 Yes, quite a lot
    - 2 Yes, sometimes
    - 1 No, not much
    - 0 No, not at all

6. Things have been getting on top of me
    - 3 Yes, most of the time I have not been able to cope at all
    - 2 Yes, sometime I haven't been coping as well as usual
    - 1 No, most of the time I have coped quite well
    - 0 No, I have been coping as well as ever

7. I have been so unhappy that I have had difficulty sleeping
    - 3 Yes, most of the time
    - 2 Yes, sometimes
    - 1 Not very often
    - 0 No, not at all

8. I have felt sad or miserable
  - 3 Yes, most of the time
  - 2 Yes, quite often
  - 1 Not very often
  - 0 No, not at all

9. I have been so unhappy that I have been crying
  - 3 Yes, most of the time
  - 2 Yes, quite often
  - 1 Only occasionally
  - 0 No, never

10. The thought of harming myself has occurred to me
  - 3 Yes, quite often
  - 2 Sometimes
  - 1 Hardly ever
  - 0 Never

**MPTSD-SS**

**NOTE: If no traumatic events are endorsed in the LSC-R, the CTS-2 pregnancy or the CTS-2 postnatal check here _______ and skip to the next measure.**

**INSTRUCTIONS:** The purpose of this scale is to measure the frequency and severity of symptoms associated with traumatic/distressing events, such as aggressive behaviors from your partner or stressful life events. Using the scales listed below, please indicate the frequency of symptoms in the ***past two weeks*** to the left of each item. Then indicate the severity to the right of each item by marking the option that best fits your experience.

**FREQUENCY**
0 Not at all
1 Once per week or less/a little bit/once in a while
2 2 to 4 times per week/somewhat/half the time
3 5 or more times per week/very much/almost always

**SEVERITY**
0 Not at all distressing
1 A little bit distressing
2 Moderately distressing
3 Quite a bit distressing
4 Extremely distressing

**FREQUENCY** **SEVERITY**

| 0-3 | | Not at all | A little bit | Moderately | Quite a bit | Extremely |
|---|---|---|---|---|---|---|
| | 1. Have you had recurrent or intrusive distressing thoughts or recollections about the event(s)? | 0 | 1 | 2 | 3 | 4 |
| | 2. Have you been having recurrent bad dreams or nightmares about the event(s)? | 0 | 1 | 2 | 3 | 4 |
| | 3. Have you had the experience of suddenly reliving the event(s), flashbacks of it, acting or feeling as if it were reoccurring? | 0 | 1 | 2 | 3 | 4 |
| | 4. Have you been intensely EMOTIONALLY upset when reminded of the event(s) (includes anniversary reactions)? | 0 | 1 | 2 | 3 | 4 |
| | 5. Have you been having intense PHYSICAL reactions (e.g., sweaty, heart palpitations) when reminded of the event(s)? | 0 | 1 | 2 | 3 | 4 |
| | 6. Have you persistently been making efforts to avoid thoughts or feelings associated with the event(s)? | 0 | 1 | 2 | 3 | 4 |

| 0-3 | | Not at all | A little bit | Moderately | Quite a bit | Extremely |
|---|---|---|---|---|---|---|
| | 7. Have you been persistently making efforts to avoid activities, situations, or places that remind you of the event(s)? | 0 | 1 | 2 | 3 | 4 |
| | 8. Are there any important aspects of the event(s) that you still cannot recall? | 0 | 1 | 2 | 3 | 4 |
| | 9. Have you markedly lost interest in free time activities since the event(s)? | 0 | 1 | 2 | 3 | 4 |
| | 10. Have you felt detached or cut off from others around you since the event(s)? | 0 | 1 | 2 | 3 | 4 |
| | 11. Have you felt that your ability to experience emotions is less (e.g., unable to have loving feelings, can't cry when sad, feeling numb, etc.)? | 0 | 1 | 2 | 3 | 4 |
| | 12. Have you felt that any future plans or hopes have changed because of the event(s) (e.g., no career, marriage, children, or long life)? | 0 | 1 | 2 | 3 | 4 |
| | 13. Have you been having persistent difficulty falling or staying asleep? | 0 | 1 | 2 | 3 | 4 |
| | 14. Have you been continuously irritable or having outbursts of anger? | 0 | 1 | 2 | 3 | 4 |
| | 15. Have you been having persistent difficulty concentrating? | 0 | 1 | 2 | 3 | 4 |
| | 16. Are you overly alert (e.g., checkin to see who is around you, etc.) since th event(s)? | 0 | 1 | 2 | 3 | 4 |
| | 17. Have you been jumpier, more easil startled, since the event(s)? | 0 | 1 | 2 | 3 | 4 |

APPENDIX E – Balance Beam Task Protocol

## Balance Beam Task Script (Field, 2010)

(Developed from Sethre-Hofstad, Stansbury & Rice, 2002)

<u>CHILD INTERVIEWER</u>: "*Now we are going to play the beam game. For this game, I want to see how well you can walk on this balance beam. You will walk forwards, backwards, sideways and over some cones. You will have to do these things on the beam for a total of 5 minutes. I will remind you of what to do when you are on the beam, and I will tell you when your 5 minutes is over. I need you to do as much of this as you can by yourself, but I will be here to help you if you need, and to make sure that you are safe. It is very important for you to do your best at all these things on the beam. We will be videotaping you on the beam so that we can compare you to other children. Please have a seat right here and I will let you know when it is time for you to get on the beam.*"

- ➔ Interviewer: Have child sit on the floor next to the beam and set the timer for **two minutes.** After the timer goes off, re-set the timer for **five minutes** and then lift the child get on the beam and have them perform the walking tasks in the following order: (1) walk forward, (2) walk backward, (3) walk sideways, (4) walk over cones. Repeat as needed.
    - o The child MUST be on the beam for a total of five minutes; if the child asks to dismount earlier, encourage them to remain on the beam and remind them to try their best. If the child cries they are allowed to discontinue the task early.
    - o If a child cries or refuses to get on the beam to begin with, encourage them to try; if they still refuse, have them sit on the beam (or next to the beam if necessary) for the 5 minute duration
- ➔ After the timer goes off at the 5 minute mark, the mother will enter the room and can respond to the child as needed for a three minute period of time.

<u>MOTHER INTERVIEWER</u>: "*Now we are going to have your child perform the beam task. For this task, your child will be on a balance beam performing a series of walking skills, including walking forward, backward, sideways, and over some cones. He/she will perform these skills for a total of 5 minutes. During this task, _________(child interviewer's name) will be there to provide your child with help if needed, and to make sure that your child is safe. If your child cries during this task, we will discontinue. You will also be able to watch your child perform the skills through this window. You also have the right to stop the task at any point if you feel uncomfortable. Please let me know if this is the case, and I will notify _________(child interviewer's name) to stop the task immediately. I will let you know when the task has ended and at that time we will have you go into the room for a few minutes to comfort your child. Do you have any questions?*"

Interviewer: Have the mother watch her child perform the beam task. When the 5 minute timer goes off, bring the mother to the child interview room and instruct the mother to comfort the child as she normally would. (3 minute reunion episode)

APPENDIX F - Affective Attunement Coding

## MOTHER-CHILD ATTUNEMENT GLOBAL CODE

This scale assesses the extent to which the mother makes an attempt to empathically facilitate the child's behavior at a time when support, assistance, or availability would be helpful to the child. A mother who is highly attuned assists her child in the expression of his/her thoughts or feelings and supports the child's desire to express and control them. A highly attuned mother also engages in conversation, dialogue or behavioral interactions that act in the service of maintaining child's organization. Additionally, this code assesses mothers' ability to provide empathy (e.g., by generally paraphrasing the child's feelings) in ways that facilitate the organization of a child's behavior and his/her coping.

This code also assesses the degree to which the child both seeks out and is able to use the mother's behavior and responsiveness to help regulate his/her own behavior and emotional expression (e.g., their receptiveness to the mother's help).

**Coders please note for codes #3, 4, and 5: If BOTH parts of the code are not met (e.g., the mother's response meets criteria but child's does not), drop the rating to one number below.**

1. No evidence of attuned, facilitative behavior is observed OR mother does not pay attention to child, for example the mother just sits back observing. Mother does not attempt to engage child in any activity or makes a "token gesture" for the benefit of the experimenter, during periods when child could use some support, guidance, etc. Mother does not supply a supportive presence or empathy for the child, when it is clear that the child could use some assistance. The child also may not seek out the mother for assistance in a managing his/her emotion (e.g., child appears emotionally withdrawn).

2. Mother echoes child's comments while sitting back OR has limited involvement with the child. When the child actively seeks out help from the mother their cues may be difficult to understand (e.g., ambivalent, disorganized). The mother may ignore these bids, mistarget or misinterpret child's cues, or make perfunctory attempts at aiding child or acting empathically toward the child. Give the dyad a code of 2 if the mother makes adequate attempts to interact with and engage the child in discussion of his/her emotions or to be empathic, but the child remains disengaged or withdrawn from the mother (e.g., hides face, turns way from the mother, etc.).

3. 1 clear instance of attuned, facilitative behavior by the mother in addition to being available to child; OR 1 clear attempt by the mother to describe what child is feeling and manage those feelings AND a clear **attempt** by the child to utilize the mother for aid in managing his/her emotions (e.g., accepts a hug from the mother and tries to use it help calm down even if he/she doesn't completely settle down).

4. 2 clear instances of genuine attuned, facilitative behavior from the mother in addition to being attentive, where the mother seems emotionally invested - Must be explicit acts which have a genuine and empathic quality of the mother while trying to be helpful in child's management of his/her emotions. The child is receptive of this aid from the mother and is able to use the mother's aid to successfully manage his/her emotions (e.g., accepts a hug from the mother and successfully uses it to calm down).

5. Instances of attuned, facilitative and empathic behavior are frequent (3 or more) or especially salient and characterize much of the mother's way of relating to the child during the reunion episode. The child is very receptive to the mother's help and is able to manage his/her emotional responses well throughout the episode with the aid of the mother.

**<u>Attunement Strategies</u> used by mother: (please check all behaviors observed, even if they are not "successful" for calming the child. Also note any additional behaviors observed if not included on the list)**

- ☐ <u>Humor</u>: The mother uses laughter, humor, joking around that is not at the child's expense

- ☐ <u>Physical comforting</u>**:** The mother uses physical interactions/gestures such as hugging, kissing, rubbing the back of the child, holding the child's hand, inviting/allowing the child to sit on her lap, etc.

- ☐ <u>Verbal reassurance/praise</u>: The mother says things to the child to reassure him/her that he/she is physically safe and ok, and/or the mother praises the child's behavior during the beam task. Examples:
  "You're ok!"
  "You're such a big girl/boy!"
  "You did a great job on the beam!"
  "You listened so well!"

- ☐ <u>Checking in</u>: The mother says things to show concern for the well-being of the child, and/or the mother asks her child to tell her about his/her emotional experience of walking on the beam. Examples:
  "Are you OK?"
  "How are you feeling?"
  "What was that like?"
  "That seemed pretty <u>(fun/scary other emotion word reflecting child's experience)</u>"

- ☐ <u>Verbal stress reduction</u>: The mother uses verbal commands or instructions that are designed to help the child regulate his/herself (may be often be seen in combination with verbal reassurance). Examples":
  "Ok, calm down"
  "Why don't you take a few deep breaths"

- ☐ <u>Distraction</u>: The mother tries to distract her child from their distress, for example by encouraging the child to play with a toy, bringing up a new topic of discussion, etc.

- ☐ <u>Other (please not any other behaviors used by the mother here)</u>:
  ______________________________________________
  ______________________________________________
  ______________________________________________

## Mother-Rated Beam Task  Stress and Perception of Child's Experience

1) Interviewer: "Please rate how stressful it was to watch your child participate in the beam task, where 1 represents not at all stressful and 6 represents extremely stressful:"

| 1 | 2 | 3 | 4 | 5 | 6 |
|---|---|---|---|---|---|
| Not at all stressful | | | | | Extremely stressful |

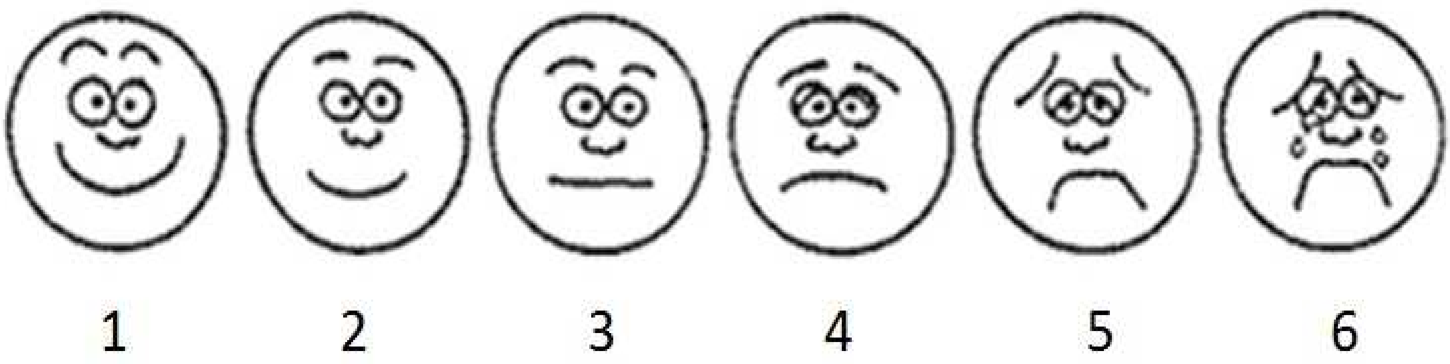
2) "Now, please rate how stressful you think **your child** found the balance beam walking task on the following scale, where 1 represents not at all stressful and 6 represents extremely stressful:"

| 1 | 2 | 3 | 4 | 5 | 6 |
|---|---|---|---|---|---|
| Not at all stressful | | | | | Extremely stressful |

Figure 9.2

*Child Beam Task Rating Scale*

<u>Child Beam Task Rating Scale</u>



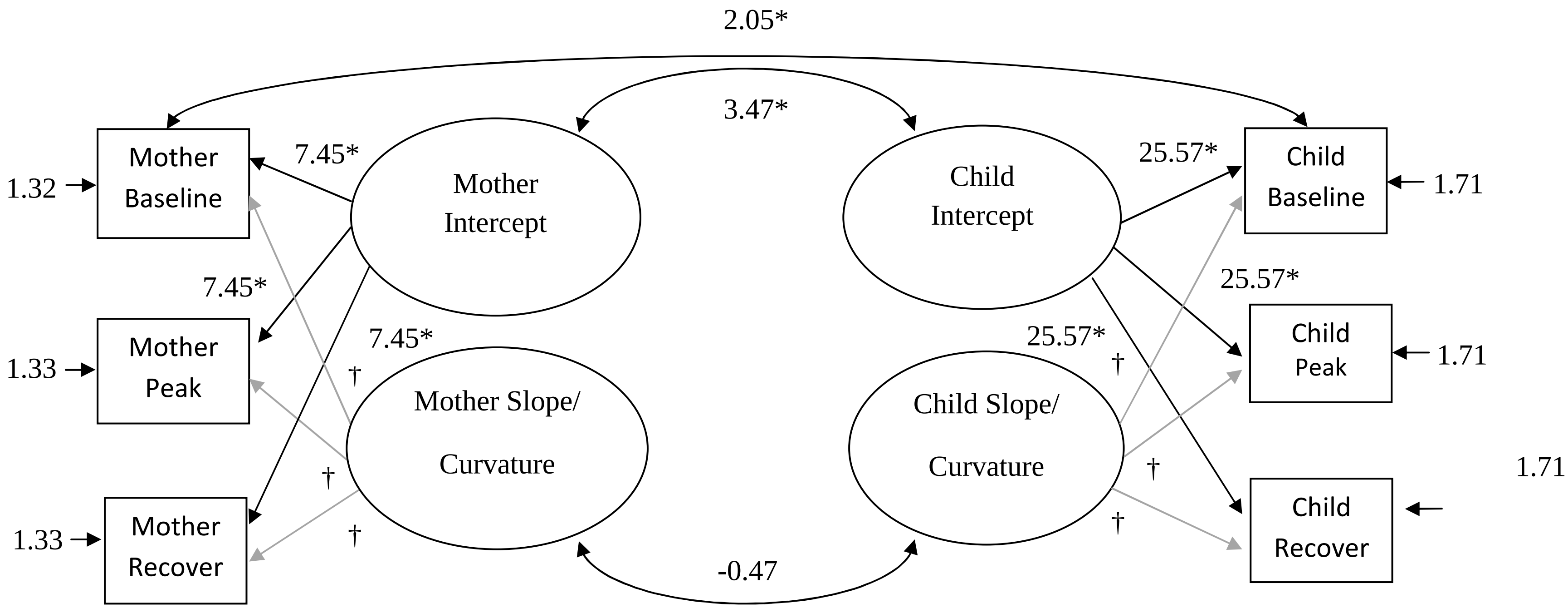

Interviewer read:

"Tell me how walking on the balance beam made you feel, using these faces here. Each face here is for a person who either feels happy because the beam task felt very easy and not scary at all (POINT TO FACE#1), or sad because the beam task felt very hard or very scary (POINT TO FACE #6). See, face 1 is very happy because the beam task did not feel hard at all. Face 2 thought the beam task was just a little bit hard. Face 3 thought it was just a little bit harder. Face 4 thought it was pretty hard. Face 5 thought it was a whole lot hard. And Face 6 thought it was as VERY hard, as hard as you can imagine. Now, point to the face that shows how YOU felt when you walked on the balance beam." (Interviewer circles child's rating)

APPENDIX G:
Structural Equation Models with T-values and LISREL Output

Figure 9.3

*SEM #1: Attunement of Cortisol with T-Values*




Note: *$p$ < .05; [†]significance test is not conducted; Chi-Square = 9.61 ($df$ = 11), $p$ = 0.566; RMSEA = 0.000 (90% C.I. = 0.00 – 0.08); CFI = 1.00; GFI = 0.98; SRMR = 0.18; $R^2$ for Mother Slope and Child Slope = 1.00 (variance was not estimated)

LISREL Output for Structural Equation Model #1 Attunement of Mother and Child Cortisol Reactivity:

Degrees of Freedom = 11
Normal Theory Weighted Least Squares Chi-Square = 9.61 (P = 0.57)
Estimated Non-centrality Parameter (NCP) = 0.0
90 Percent Confidence Interval for NCP = (0.0 ; 9.80)

Minimum Fit Function Value = 0.068
Population Discrepancy Function Value (F0) = 0.0
90 Percent Confidence Interval for F0 = (0.0 ; 0.069)
Root Mean Square Error of Approximation (RMSEA) = 0.0
90 Percent Confidence Interval for RMSEA = (0.0 ; 0.079)
P-Value for Test of Close Fit (RMSEA < 0.05) = 0.80

Expected Cross-Validation Index (ECVI) = 0.22
90 Percent Confidence Interval for ECVI = (0.22 ; 0.29)
ECVI for Saturated Model = 0.30
ECVI for Independence Model = 6.40

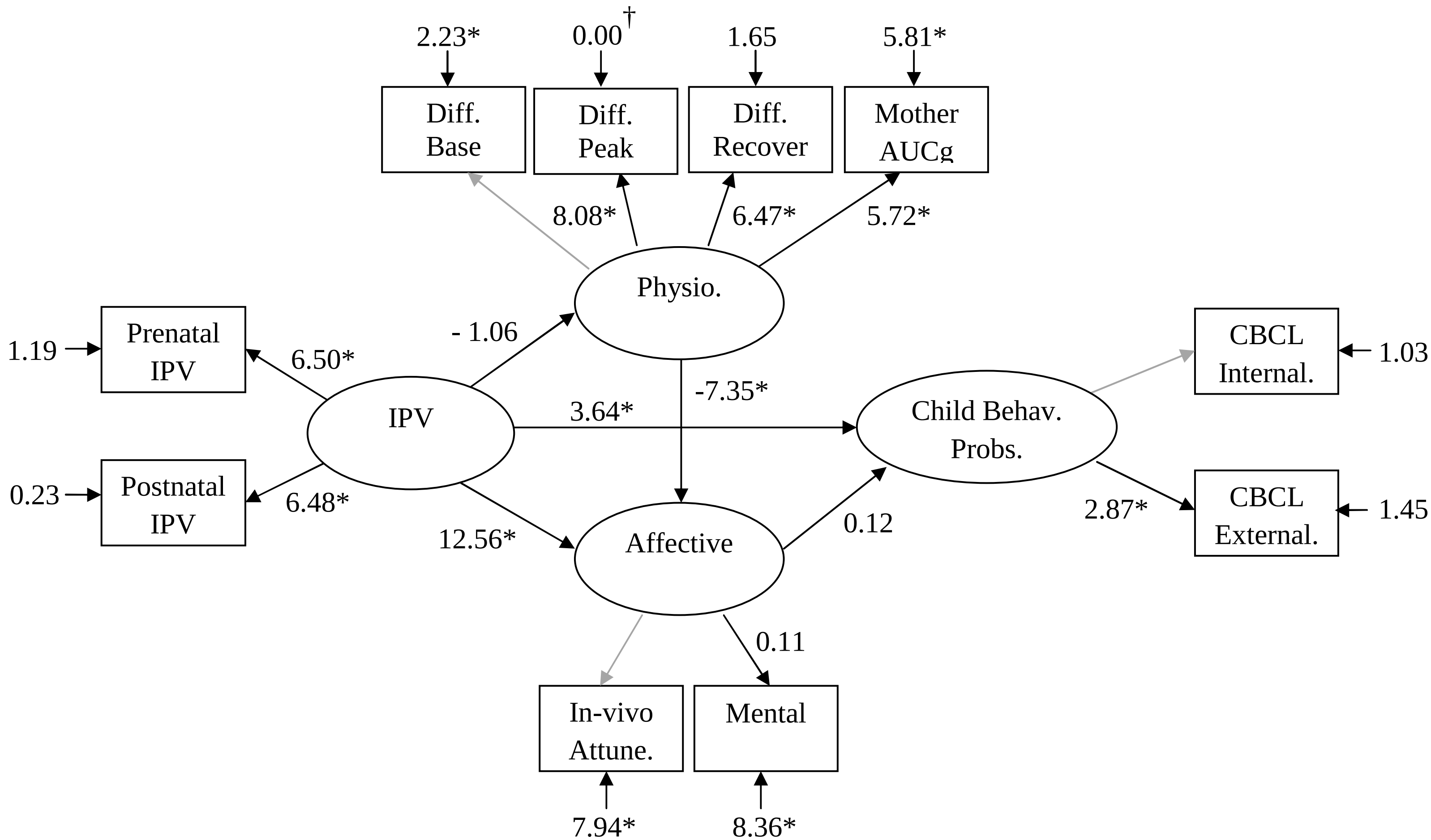
Chi-Square for Independence Model with 15 Degrees of Freedom = 896.36
Independence AIC = 908.36
Model AIC = 29.61
Saturated AIC = 42.00
Independence CAIC = 932.14
Model CAIC = 69.24
Saturated CAIC = 125.22

Normed Fit Index (NFI) = 1.00
Non-Normed Fit Index (NNFI) = 1.02
Parsimony Normed Fit Index (PNFI) = 0.73
Comparative Fit Index (CFI) = 1.00
Incremental Fit Index (IFI) = 1.01
Relative Fit Index (RFI) = 1.00

Root Mean Square Residual (RMR) = 0.070
Standardized RMR = 0.18
Goodness of Fit Index (GFI) = 0.98
Adjusted Goodness of Fit Index (AGFI) = 0.96
Parsimony Goodness of Fit Index (PGFI) = 0.51

Figure 9.4

*SEM #2: IPV, Attunement, Maternal Reported Problems with T-Values*




Note: *$p < .05$; Chi-Square = 44.35 ($df$ = 32), $p$ = 0.072; RMSEA = 0.052 (90% C.I. = 0.00 – 0.09); CFI = 1.00; GFI = 0.99; SRMR = 0.043

LISREL Output for Structural Equation Model #2 Full Model with IPV, Physiological Attunement, Affective Attunement and Maternal Report of Child Behavior Problems:

Degrees of Freedom = 32
Normal Theory Weighted Least Squares Chi-Square = 44.35 (P = 0.072)
Estimated Non-centrality Parameter (NCP) = 12.35
90 Percent Confidence Interval for NCP = (0.0 ; 33.99)

Minimum Fit Function Value = 0.10
Population Discrepancy Function Value (F0) = 0.087
90 Percent Confidence Interval for F0 = (0.0 ; 0.24)
Root Mean Square Error of Approximation (RMSEA) = 0.052
90 Percent Confidence Interval for RMSEA = (0.0 ; 0.086)
P-Value for Test of Close Fit (RMSEA < 0.05) = 0.43

Expected Cross-Validation Index (ECVI) = 0.64
90 Percent Confidence Interval for ECVI = (0.55 ; 0.79)
ECVI for Saturated Model = 0.77
ECVI for Independence Model = 4.24

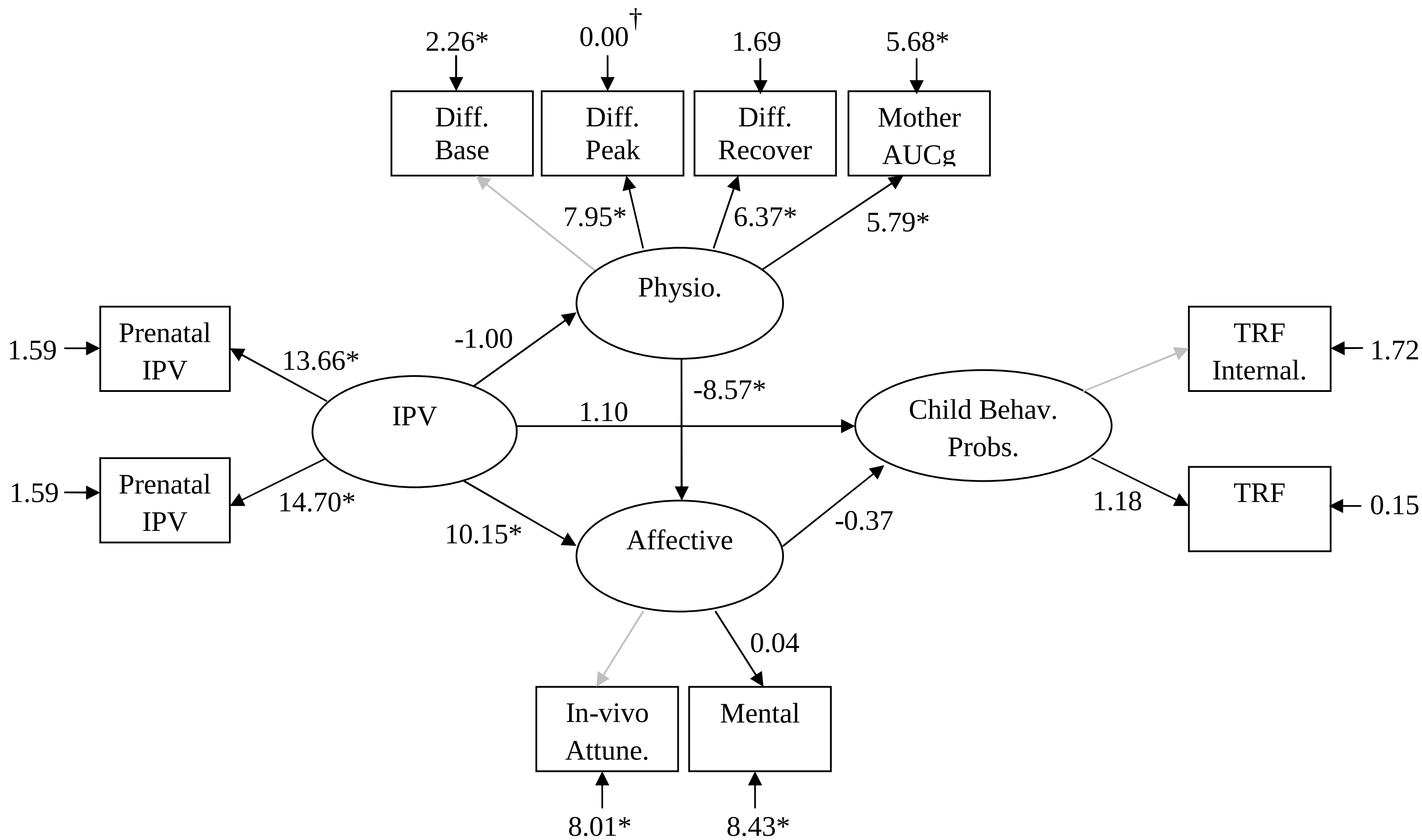
Chi-Square for Independence Model with 45 Degrees of Freedom = 581.81
Independence AIC = 601.81
Model AIC = 90.35
Saturated AIC = 110.00
Independence CAIC = 641.44
Model CAIC = 181.49
Saturated CAIC = 327.96

Normed Fit Index (NFI) = 1.00
Non-Normed Fit Index (NNFI) = 1.08
Parsimony Normed Fit Index (PNFI) = 0.71
Comparative Fit Index (CFI) = 1.00
Incremental Fit Index (IFI) = 1.06
Relative Fit Index (RFI) = 1.00

Root Mean Square Residual (RMR) = 0.043
Standardized RMR = 0.043
Goodness of Fit Index (GFI) = 0.99
Adjusted Goodness of Fit Index (AGFI) = 0.98
Parsimony Goodness of Fit Index (PGFI) = 0.58

Figure 9.5

*SEM #3: IPV, Attunement, Teacher Reported Problems with T-Values*




Note: **p* < .05; Chi-Square = 45.89 (*df* = 33), *p* = 0.067; RMSEA = 0.052 (90% C.I. = 0.00 – 0.09); CFI = 1.00; GFI = 0.98; SRMR = 0.049

LISREL Output for Structural Equation Model #3: Full Model with IPV, Physiological Attunement, Affective Attunement and Teacher Report of Child Behavior Problems:

Degrees of Freedom = 33
Normal Theory Weighted Least Squares Chi-Square = 45.89 (P = 0.067)
Estimated Non-centrality Parameter (NCP) = 12.89
90 Percent Confidence Interval for NCP = (0.0 ; 34.85)

Minimum Fit Function Value = 0.13
Population Discrepancy Function Value (F0) = 0.091
90 Percent Confidence Interval for F0 = (0.0 ; 0.25)
Root Mean Square Error of Approximation (RMSEA) = 0.052
90 Percent Confidence Interval for RMSEA = (0.0 ; 0.086)
P-Value for Test of Close Fit (RMSEA < 0.05) = 0.43

Expected Cross-Validation Index (ECVI) = 0.63
90 Percent Confidence Interval for ECVI = (0.54 ; 0.79)
ECVI for Saturated Model = 0.77
ECVI for Independence Model = 4.02

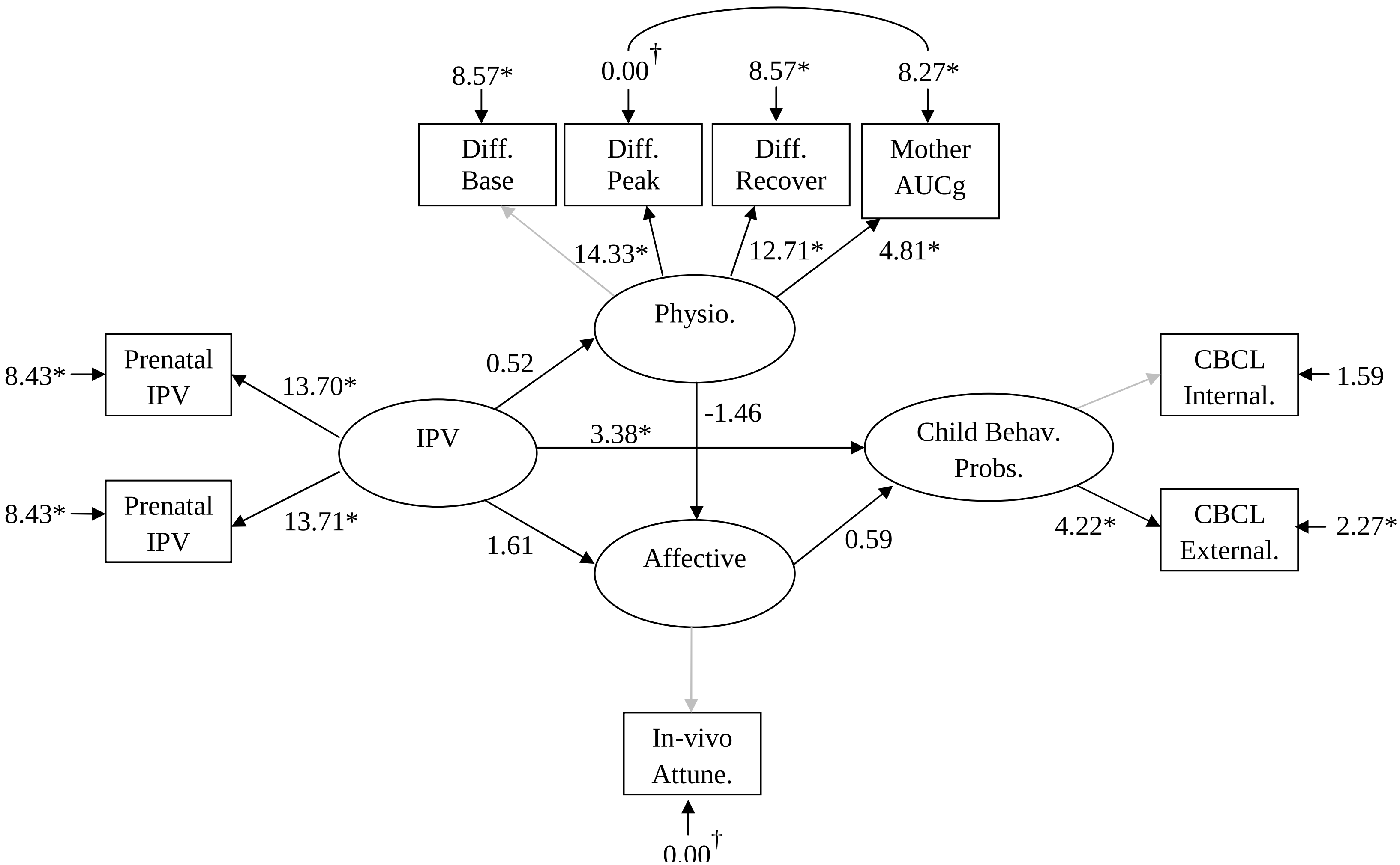
Chi-Square for Independence Model with 45 Degrees of Freedom = 551.02
Independence AIC = 571.02
Model AIC = 89.89
Saturated AIC = 110.00
Independence CAIC = 610.65
Model CAIC = 177.08
Saturated CAIC = 327.96

Normed Fit Index (NFI) = 1.00
Non-Normed Fit Index (NNFI) = 1.09
Parsimony Normed Fit Index (PNFI) = 0.73
Comparative Fit Index (CFI) = 1.00
Incremental Fit Index (IFI) = 1.06
Relative Fit Index (RFI) = 1.00

Root Mean Square Residual (RMR) = 0.049
Standardized RMR = 0.049
Goodness of Fit Index (GFI) = 0.98
Adjusted Goodness of Fit Index (AGFI) = 0.97
Parsimony Goodness of Fit Index (PGFI) = 0.59

Figure 9.6

*SEM #4: Full Model Using In-Vivo Attunement Only with T-Values*




Note: **p* < .05; † significance test is not conducted; Chi-Square = 33.46 (*df* = 24), *p* = 0.095; RMSEA = 0.053 (90% C.I. = 0.00 – 0.09); CFI = 0.98; GFI = 0.95; SRMR = 0.045

LISREL Output for Structural Equation Model #4: Model with IPV, Physiological Attunement, Maternal Report of Child Behavior Problems, and In-Vivo Attunement Only:

Degrees of Freedom = 24
Minimum Fit Function Chi-Square = 34.99 (P = 0.068)
Normal Theory Weighted Least Squares Chi-Square = 33.46 (P = 0.095)
Estimated Non-centrality Parameter (NCP) = 9.46
90 Percent Confidence Interval for NCP = (0.0 ; 28.83)

Minimum Fit Function Value = 0.25
Population Discrepancy Function Value (F0) = 0.067
90 Percent Confidence Interval for F0 = (0.0 ; 0.20)
Root Mean Square Error of Approximation (RMSEA) = 0.053
90 Percent Confidence Interval for RMSEA = (0.0 ; 0.092)
P-Value for Test of Close Fit (RMSEA < 0.05) = 0.42

Expected Cross-Validation Index (ECVI) = 0.53
90 Percent Confidence Interval for ECVI = (0.46 ; 0.67)
ECVI for Saturated Model = 0.63
ECVI for Independence Model = 4.17

Chi-Square for Independence Model with 36 Degrees of Freedom = 574.30
Independence AIC = 592.30
Model AIC = 75.46
Saturated AIC = 90.00
Independence CAIC = 627.96
Model CAIC = 158.68
Saturated CAIC = 268.33

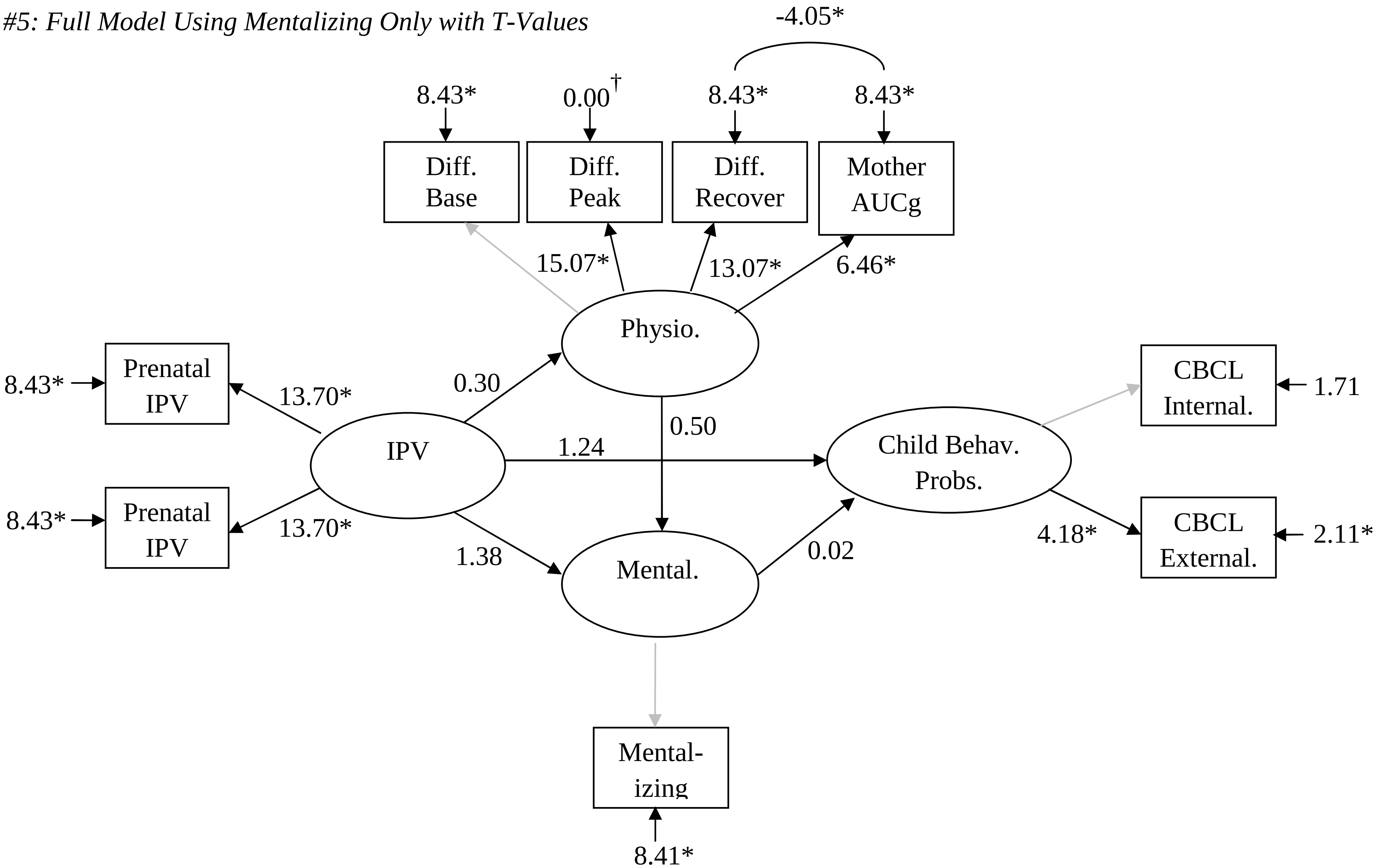
Normed Fit Index (NFI) = 0.94
Non-Normed Fit Index (NNFI) = 0.97
Parsimony Normed Fit Index (PNFI) = 0.63
Comparative Fit Index (CFI) = 0.98
Incremental Fit Index (IFI) = 0.98
Relative Fit Index (RFI) = 0.91

Critical N (CN) = 175.41

Root Mean Square Residual (RMR) = 0.045
Standardized RMR = 0.045
Goodness of Fit Index (GFI) = 0.95
Adjusted Goodness of Fit Index (AGFI) = 0.91
Parsimony Goodness of Fit Index (PGFI) = 0.51

Figure 9.7

*SEM #5: Full Model Using Mentalizing Only with T-Values*




Note: *$p$ < .05; †significance test is not conducted; Chi-Square = 30.54 ($df$ = 24), $p$ = 0.168; RMSEA = 0.044 (90% C.I. = 0.00 – 0.09); CFI = 0.98; GFI = 0.95; SRMR = 0.040

LISREL Output Structural Equation Model #5: Model with IPV, Physiological Attunement, Maternal Report of Child Behavior Problems, and Mentalizing Only:

Degrees of Freedom = 24
Minimum Fit Function Chi-Square = 33.96 (P = 0.085)
Normal Theory Weighted Least Squares Chi-Square = 30.54 (P = 0.17)
Estimated Non-centrality Parameter (NCP) = 6.54
90 Percent Confidence Interval for NCP = (0.0 ; 24.88)

Minimum Fit Function Value = 0.24
Population Discrepancy Function Value (F0) = 0.046
90 Percent Confidence Interval for F0 = (0.0 ; 0.18)
Root Mean Square Error of Approximation (RMSEA) = 0.044
90 Percent Confidence Interval for RMSEA = (0.0 ; 0.085)
P-Value for Test of Close Fit (RMSEA < 0.05) = 0.55

Expected Cross-Validation Index (ECVI) = 0.51
90 Percent Confidence Interval for ECVI = (0.46 ; 0.64)
ECVI for Saturated Model = 0.63
ECVI for Independence Model = 4.12

Chi-Square for Independence Model with 36 Degrees of Freedom = 567.60
Independence AIC = 585.60
Model AIC = 72.54
Saturated AIC = 90.00
Independence CAIC = 621.27
Model CAIC = 155.76
Saturated CAIC = 268.33

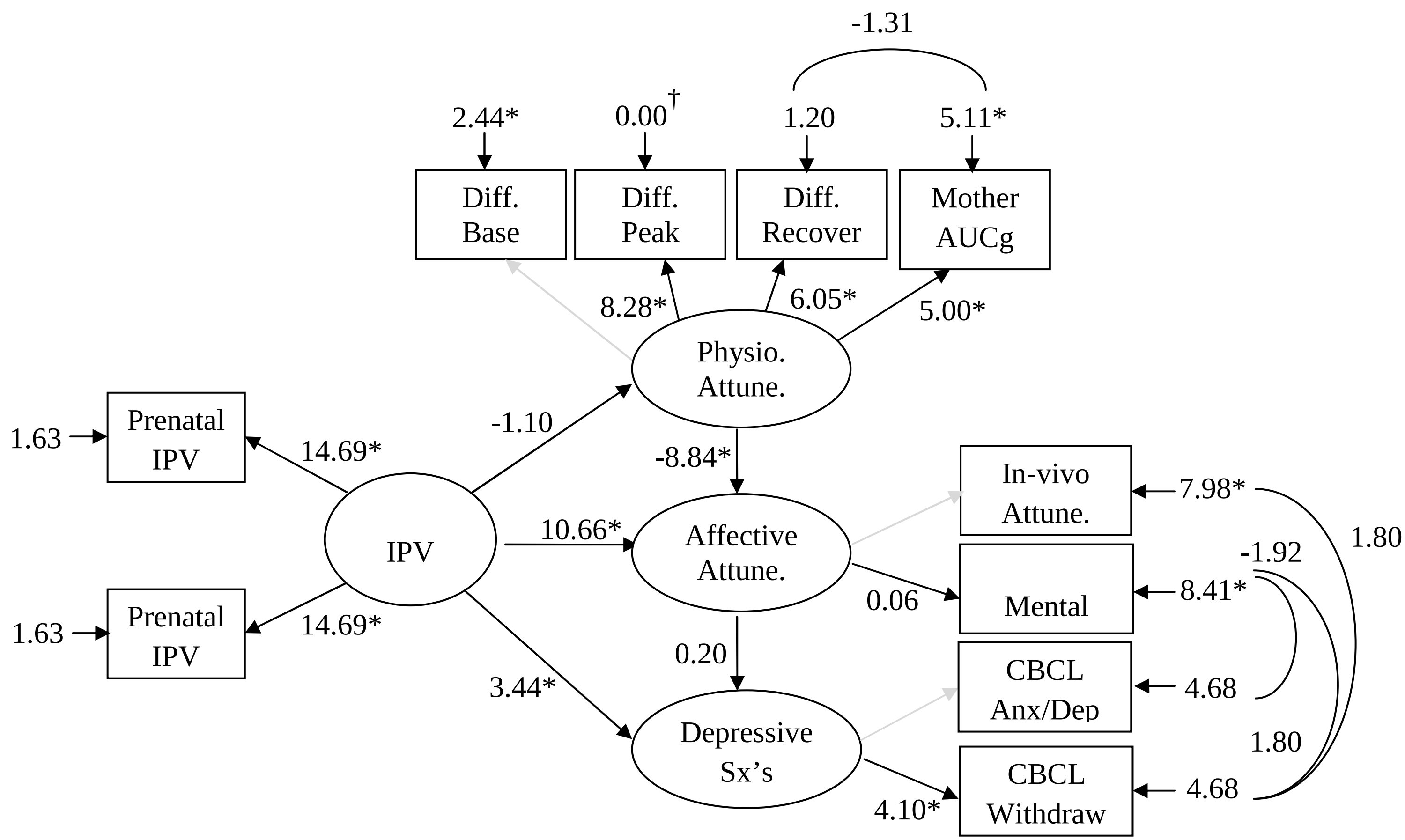
Normed Fit Index (NFI) = 0.94
Non-Normed Fit Index (NNFI) = 0.97
Parsimony Normed Fit Index (PNFI) = 0.63
Comparative Fit Index (CFI) = 0.98
Incremental Fit Index (IFI) = 0.98
Relative Fit Index (RFI) = 0.91

Critical N (CN) = 180.71

Root Mean Square Residual (RMR) = 0.040
Standardized RMR = 0.040
Goodness of Fit Index (GFI) = 0.95
Adjusted Goodness of Fit Index (AGFI) = 0.91
Parsimony Goodness of Fit Index (PGFI) = 0.51

Figure 9.8

*SEM #6: IPV, Attunement, Child Depressive Symptoms with T-Values*




Note: *$p$ < .05; †significance test is not conducted; Chi-Square = 42.67 ($df$ = 30), $p$ = 0.063; RMSEA = 0.055 (90% C.I. = 0.00 – 0.09); CFI = 1.00; GFI = 0.98; SRMR = 0.052. † Error not estimated.

LISREL Output Structural Equation Model #6: Model with IPV, Physiological Attunement, Affective Attunement and Mother Report of Child Depressive Symptoms

Degrees of Freedom = 30
Normal Theory Weighted Least Squares Chi-Square = 42.67 (P = 0.063)
Estimated Non-centrality Parameter (NCP) = 12.67
90 Percent Confidence Interval for NCP = (0.0 ; 34.08)

Minimum Fit Function Value = 0.14
Population Discrepancy Function Value (F0) = 0.089
90 Percent Confidence Interval for F0 = (0.0 ; 0.24)
Root Mean Square Error of Approximation (RMSEA) = 0.055
90 Percent Confidence Interval for RMSEA = (0.0 ; 0.089)
P-Value for Test of Close Fit (RMSEA < 0.05) = 0.39

Expected Cross-Validation Index (ECVI) = 0.65
90 Percent Confidence Interval for ECVI = (0.56 ; 0.80)
ECVI for Saturated Model = 0.77
ECVI for Independence Model = 3.96

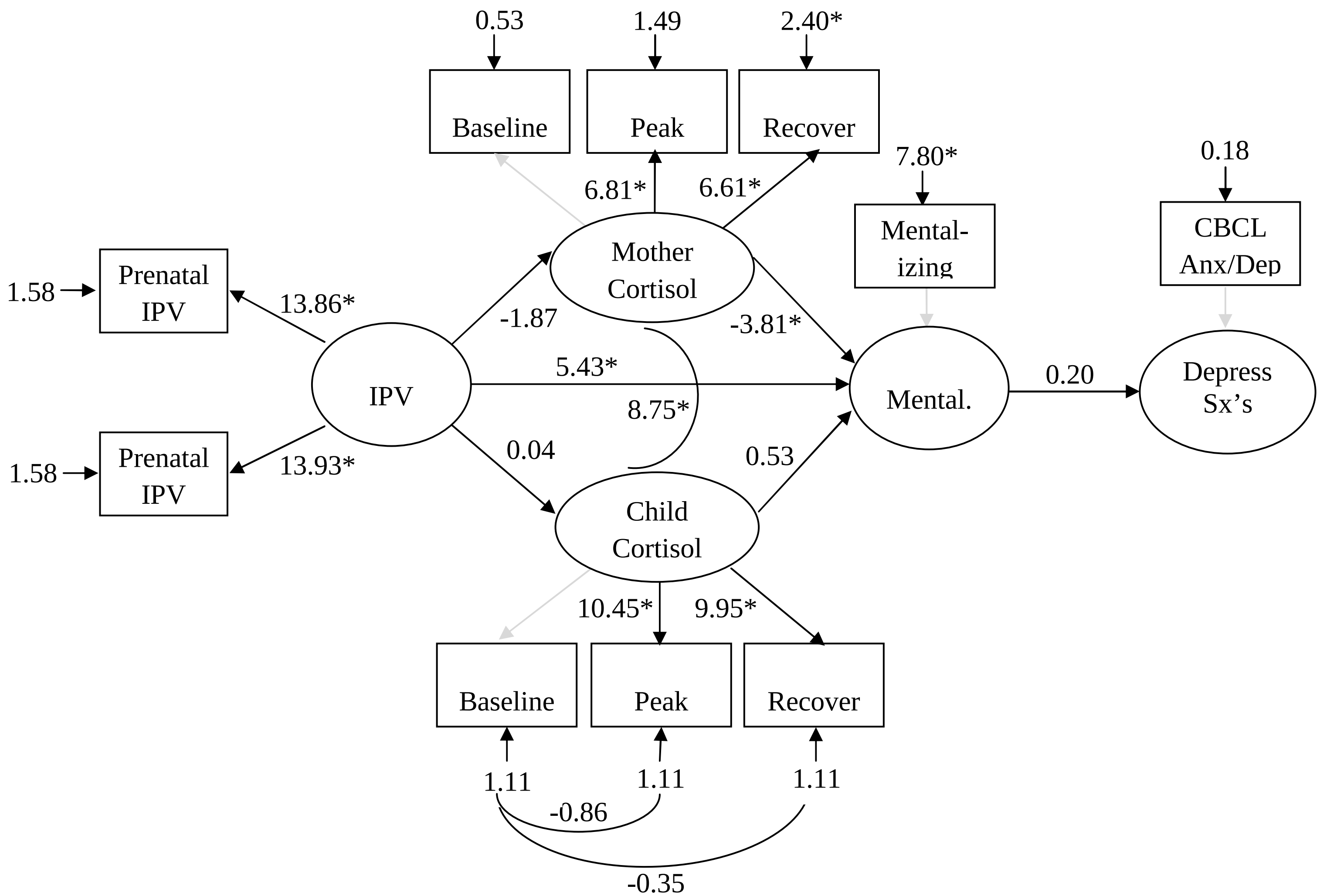
Chi-Square for Independence Model with 45 Degrees of Freedom = 542.77
Independence AIC = 562.77
Model AIC = 92.67
Saturated AIC = 110.00
Independence CAIC = 602.40
Model CAIC = 191.74
Saturated CAIC = 327.96

Normed Fit Index (NFI) = 1.00
Non-Normed Fit Index (NNFI) = 1.09
Parsimony Normed Fit Index (PNFI) = 0.67
Comparative Fit Index (CFI) = 1.00
Incremental Fit Index (IFI) = 1.06
Relative Fit Index (RFI) = 1.00

Root Mean Square Residual (RMR) = 0.052
Standardized RMR = 0.052
Goodness of Fit Index (GFI) = 0.98
Adjusted Goodness of Fit Index (AGFI) = 0.97
Parsimony Goodness of Fit Index (PGFI) = 0.54

Figure 9.9

*SEM #7: Alternative Model of Physiological Attunement*




Note: *$p$ < .05; †significance test is not conducted; Chi-Square = 86.39 ($df$ = 26), $p$ = 0.000; RMSEA = 0.128 (90% C.I. = 0.10 – 0.16); CFI = 1.00; GFI = 0.99; SRMR = 0.050

LISREL Output Structural Equation Model #7: Full Model Using Alternative Model of Physiological Attunement

Degrees of Freedom = 26
Normal Theory Weighted Least Squares Chi-Square = 86.39 (P = 0.00)
Estimated Non-centrality Parameter (NCP) = 60.39
90 Percent Confidence Interval for NCP = (35.93 ; 92.46)

Minimum Fit Function Value = 0.11
Population Discrepancy Function Value (F0) = 0.43
90 Percent Confidence Interval for F0 = (0.25 ; 0.65)
Root Mean Square Error of Approximation (RMSEA) = 0.13
90 Percent Confidence Interval for RMSEA = (0.099 ; 0.16)
P-Value for Test of Close Fit (RMSEA < 0.05) = 0.00

Expected Cross-Validation Index (ECVI) = 1.02
90 Percent Confidence Interval for ECVI = (0.84 ; 1.24)
ECVI for Saturated Model = 0.77
ECVI for Independence Model = 7.44

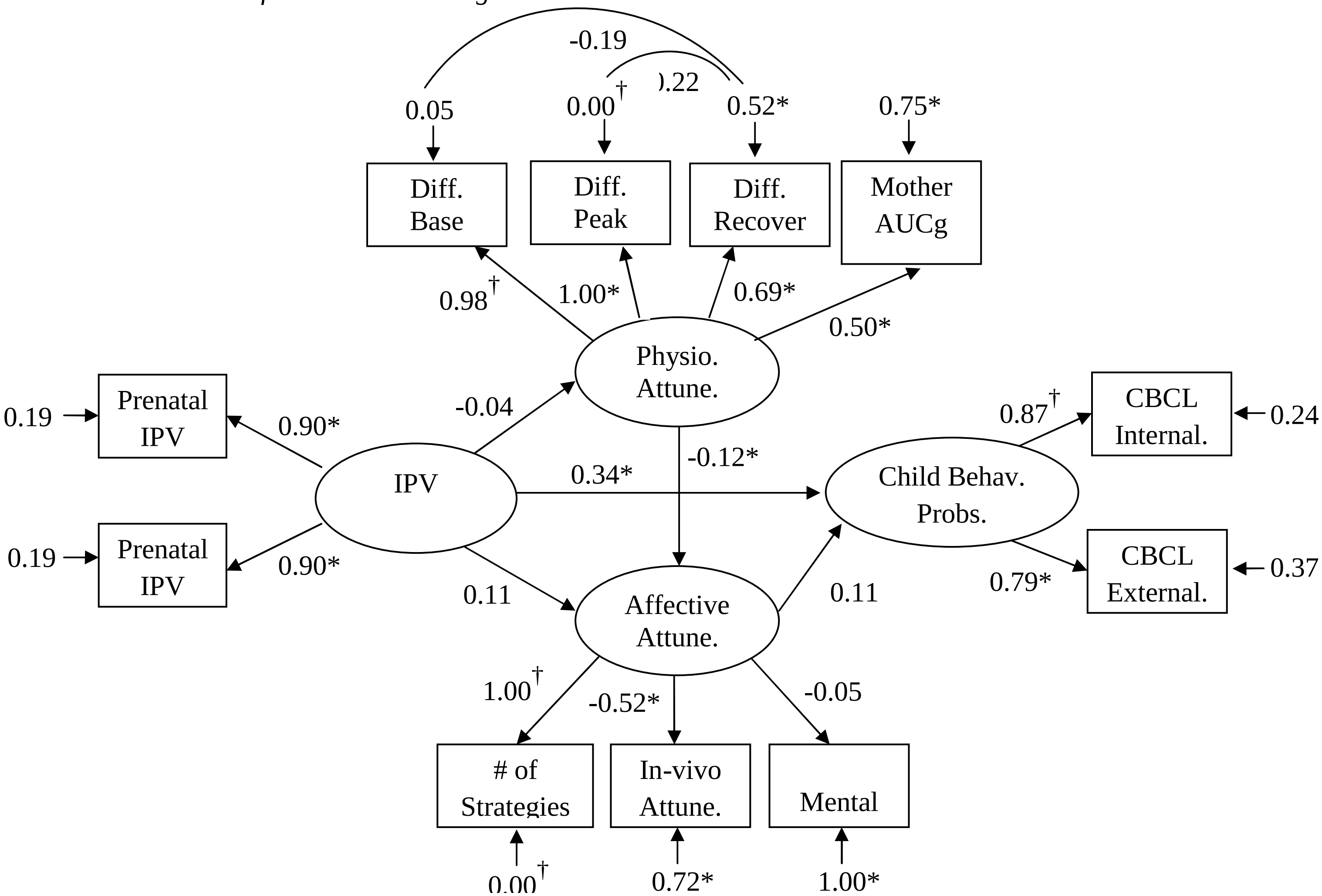
Chi-Square for Independence Model with 45 Degrees of Freedom = 1036.91
Independence AIC = 1056.91
Model AIC = 144.39
Saturated AIC = 110.00
Independence CAIC = 1096.54
Model CAIC = 259.32
Saturated CAIC = 327.96

Normed Fit Index (NFI) = 1.00
Non-Normed Fit Index (NNFI) = 1.05
Parsimony Normed Fit Index (PNFI) = 0.58
Comparative Fit Index (CFI) = 1.00
Incremental Fit Index (IFI) = 1.03
Relative Fit Index (RFI) = 1.00

Root Mean Square Residual (RMR) = 0.049
Standardized RMR = 0.050
Goodness of Fit Index (GFI) = 0.99
Adjusted Goodness of Fit Index (AGFI) = 0.98
Parsimony Goodness of Fit Index (PGFI) = 0.47

Figure 9.10

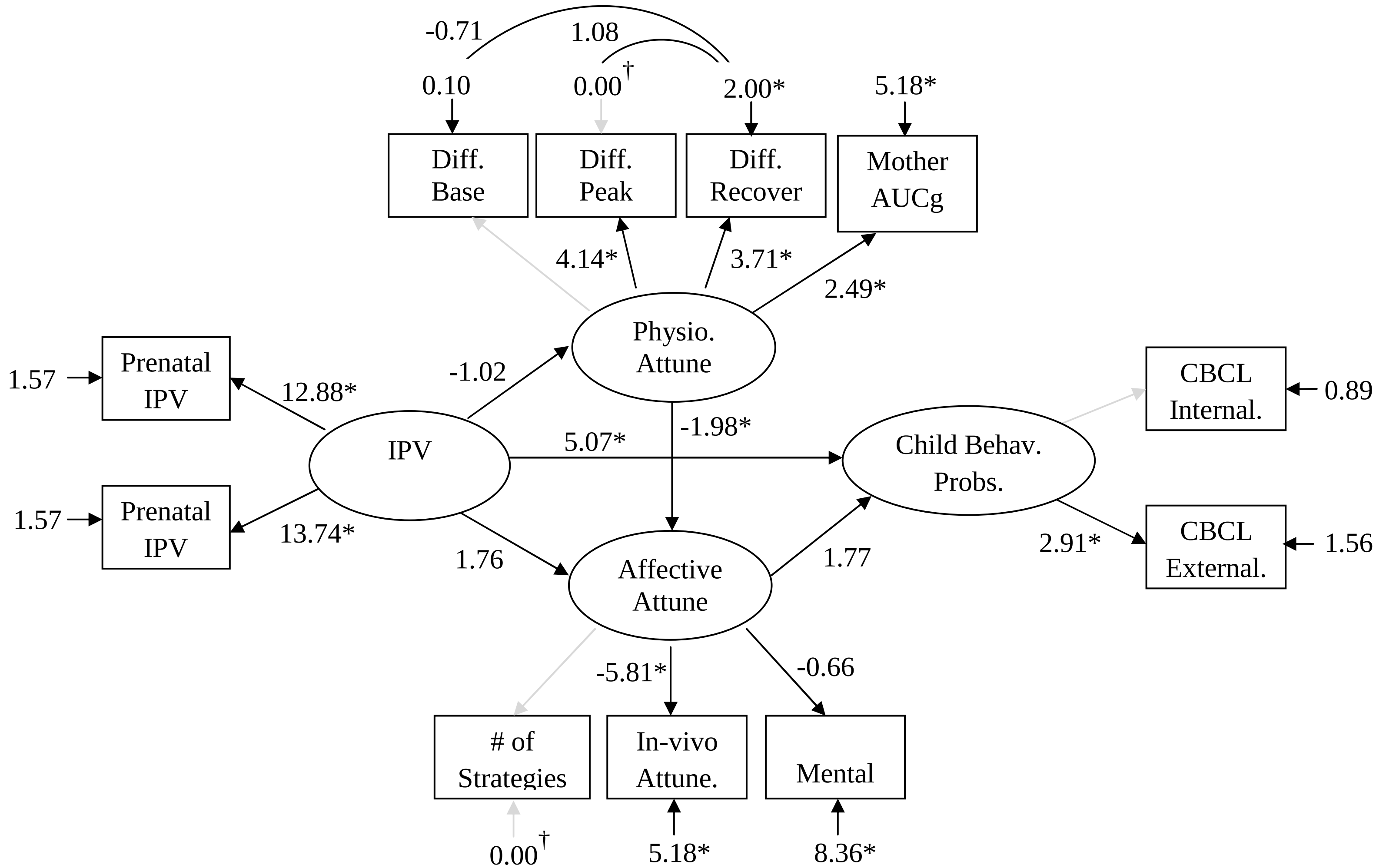
*SEM #8: Full Model with Number of Attunement Strategies*




Note: *$p$ < .05; † significance test is not conducted; Chi-Square = 41.14 ($df$ = 39), $p$ = 0.38; RMSEA = 0.02 (90% C.I. = 0.00 – 0.6); CFI = 1.00; GFI = 0.99; SRMR = 0.042

Figure 9.11

*SEM #8: Full Model with Number of Attunement Strategies, T-values*



Note: *$p$ < .05; Chi-Square = 41.14 ($df$ = 39), $p$ = 0.38; RMSEA = 0.02 (90% C.I. = 0.00 – 0.6); CFI = 1.00; GFI = 0.99; SRMR = 0.042

LISREL Output Structural Equation Model #8: Full Model Including Number of Attunement Strategies

Degrees of Freedom = 39
Normal Theory Weighted Least Squares Chi-Square = 41.14 (P = 0.38)
Estimated Non-centrality Parameter (NCP) = 2.14
90 Percent Confidence Interval for NCP = (0.0 ; 21.73)

Minimum Fit Function Value = 0.12
Population Discrepancy Function Value (F0) = 0.015
90 Percent Confidence Interval for F0 = (0.0 ; 0.15)
Root Mean Square Error of Approximation (RMSEA) = 0.020
90 Percent Confidence Interval for RMSEA = (0.0 ; 0.063)
P-Value for Test of Close Fit (RMSEA < 0.05) = 0.85

Expected Cross-Validation Index (ECVI) = 0.67
90 Percent Confidence Interval for ECVI = (0.65 ; 0.81)
ECVI for Saturated Model = 0.93
ECVI for Independence Model = 4.58

Chi-Square for Independence Model with 55 Degrees of Freedom = 628.13
Independence AIC = 650.13
Model AIC = 95.14
Saturated AIC = 132.00
Independence CAIC = 693.72
Model CAIC = 202.13
Saturated CAIC = 393.55

Normed Fit Index (NFI) = 1.00
Non-Normed Fit Index (NNFI) = 1.10
Parsimony Normed Fit Index (PNFI) = 0.71
Comparative Fit Index (CFI) = 1.00
Incremental Fit Index (IFI) = 1.07
Relative Fit Index (RFI) = 1.00

Root Mean Square Residual (RMR) = 0.042
Standardized RMR = 0.042
Goodness of Fit Index (GFI) = 0.99
Adjusted Goodness of Fit Index (AGFI) = 0.98
Parsimony Goodness of Fit Index (PGFI) = 0.58

# BIBLIOGRAPHY

## BIBLIOGRAPHY


Achenbach, T. M., McConaughy, S. H., & Howell, C. T. (1987). Child/adolescent behavioral and emotional problems: Implications of cross-informant correlations for situational specificity. *Psychology Bulletin, 101*, 213-232.

Achenbach, T. M., & Rescorla, L. A. (2000). *Manual for the ASEBA Preschool Forms & Profiles*. Burlington, VT: University of Vermont, Research Center for Children, Youth, & Families.

Afonso, V. M., Sison, M., Lovic, V., Fleming, A. S. (2007). Medial prefrontal cortex lesions in the female rat affect sexual and maternal behavior and their sequential organization. *Behavioral Neuroscience*, *121*(3), 515-526.

Aguilar, R. J., & Nightingale, N. N. (1994). The impact of specific battering experiences on the self-esteem of abused women. *Journal of Family Violence, 9*(1), 35-45.

Arias, I., & Pape, K. T. (1999). Psychological abuse: Implications for adjustment and commitment to leave violent partners. *Violence and Victims, 14*, 55-67.

Bagner, D. M., Sheinkopf, S. J., Vohr, B. R., & Lester, B. M. (2010). A preliminary study of cortisol reactivity and behavior problems in young children born premature. *Developmental Psychobiology, 52*(6), 574-582.

Banjanin, S., Kapoor, A., & Matthews, S. G. (2004). Prenatal glucocorticoid exposure alters hypothalamic-pituitary-adrenal function and blood pressure in mature male guinea pigs. *Journal of Physiology-London, 558*(1), 305-318.

Bartels, M., Van den Berg, M., Sluyter, F., Boomsma, D. I., and de Geus, E. J. C. (2003). Heritability of cortisol levels: Review and simultaneous analysis of twin studies. *Psychoneuroendocrinology*, *28*, 121 – 137.

Bartels, A., & Zeki, S. (2000). Imaging the neural correlate of love. *European Journal of Neuroscience, 12*, 88-88.

Bartels, A., & Zeki, S. (2004). The neural correlates of maternal and romantic love. *Neuroimage, 21*(3), 1155-1166.

Bergman, L. R., Magnusson, D., & El-Khouri, B. M. (2003). *Studying individual development in an interindividual context: A person-oriented approach.* Vol. 4 of *Paths through life* (D. Magnusson, Series Ed.). Mahwah, NJ: Erlbaum.

Bergman, K., Sarkar, P., Glover, V., & O'Connor, T. G. (2010). Maternal prenatal cortisol and infant cognitive development: Moderation by infant-mother attachment. *Biological Psychiatry, 67*(11), 1026-1032.

Bertram, C. E., & Hanson, M. A. (2002). Prenatal programming of postnatal endocrine responses by glucocorticoids. *Reproduction, 124*(4), 459-467.

Besenski, L. J., Forsyth, S. J., & von Baeyer, C. L. (2007). Screening young children for their ability to use self-report pain scales. *Pediatric Pain Letter, 9*(1), 1-7.

Bevans, K., Cerbone, A. B., & Overstreet, S. (2005). Advances and future directions in the study of children's neurobiological responses to trauma and violence exposure. *Journal of Interpersonal Violence, 20*, 418-425.

Bogat, G. A., Levendosky, A. A., DeJonghe, E., Davidson, W. S., & von Eye, A. (2004). Pathways of suffering: The temporal effects of domestic violence on women's mental health. *Maltrattamento e abuso all'infanzia, 6*(2), 97-112.

Bogat, G. A., Levendosky, A. A., & von Eye, A. (2005). The future of research on intimate partner violence (IPV): person-oriented and variable-oriented perspectives. *American Journal of Community Psychology, 36*, 49-70.

Bonomi, A. E., Anderson, M. L., Rivara, F. P. & Thompson, R. S. (2007). Health outcomes in women with physical and sexual intimate partner violence exposure. *Journal of Womens' Health, 16*(7), 987-997.

Bowlby, J. (1969). *Attachment and loss. Vol. 1: Attachment.* New York: Basic Books.

Brand,S. R., Brennan, P. A., Newport, D. J., Smith, A. K., Weiss, T., & Stowe, Z. N. (2010). The impact of maternal childhood abuse on maternal and infant HPA axis function in the postpartum period. *Psychoneuroendocrinology, 35*(5). 686-693.

Breiding, M. J., Black, M. C., & Ryan, G. W. (2008). Prevalence and risk factors of intimate partner violence in eighteen US states/territories, 2005. *American Journal of Preventative Medicine, 34*(2), 112-118.

Browne, A. (1993). Violence against women by male partners. *American Psychologist,48,* 1077–87.

Browne, A., & Bassuk, S. S. (1997). Intimate violence in the lives of homeless and poor housed women: Prevalence and patterns in an ethnically diverse sample. *Journal of American Orthopsychiatry, 67*(2), 261-278.

Campbell, S. B., Shaw, D. S., & Gilliom, M. (2000). Early externalizing behavior problems: Toddlers and preschoolers at risk for later maladjustment. *Development and Psychopathology, 12*, 467-488.

Carr, L., Iacoboni, M., Dubeau, M.C., Mazziotta, J. C., & Lenzi, G. L. (2003). Neural mechanisms of empathy in humans: A relay from neural systems for imitation to limbic areas. *Proceedings of the National Academy of Sciences of the United States of America, 100*(9), 5497-5502.

Casanueva, C., Martin, S. L., Runyan, D. K., Barth, R. P., & Bradley, R. H. (2008). Quality of maternal parenting among intimate-partner violence victims involved with the child welfare system. *Journal of Family Violence, 23*(6), 413-427.

Cezero, M. A., & Pons, G. (1996). Ecosystem adversity as setting factors in mothers' judgment of child behavior and indiscriminate mothering. *European Journal of Psychological Asssessment, 12*(2), 103-111.

Chan, Y. C., & Yeung, J. W. K. (2009). Children living with violence within the family and its sequel: A meta-analysis from 1995-2006. *Aggression and Violent Behavior, 14*(5), 313-322.

Cicchetti, D., & Walker, E. F. (2001). Editorial: Stress and development: Biological and psychological consequences. *Development and Psychopathology, 13*, 413-418.

Cohen, E., & Knitzer, J. (2002). Children living with domestic violence: The role of early childhood programs. Prepared for the meeting: "Early Childhood, Domestic Violence, and Poverty: Taking the Next Steps to Help Young Children and Their Families." May 2002.

Collip, D., Nicolson, N. A., Lardinois, M., Lataster, T., van Os, J., Myin-Germeys, I., & G.R.O.U.P. ( 2011). Daily cortisol, stress reactivity and psychotic experiences in individuals at about average genetic risk for psychosis. *Psychological Medicine, 41*(11), 2305-2315.

Conrad, A., Wilhelm, F. H., Roth, W. T., Spiegel, D. & Taylor, C. B. (2008). Circadian affective, cardiopulmonary, and cortisol variability in depressed and nonderessed individuals at risk for cardiovascular disease. *Journal of Psychiatric Research, 42*(9), 769-777.

Cox, J. L., Holden, J. M., & Sagovsky, R. (1987). Detection of postnatal depression. Development of the 10-item Edinburgh Postnatal Depression Scale. *The British Journal of Psychiatry, 150*, 782- 786.

Crnic, K. A., Greenberg, M. T., Robinson, N. M., & Ragozin, A. S. (1984). Maternal stress and social support – Effects on the mother-infant relationship from birth to 18 months. *American Journal of Orthopsychiatry, 54*(2), 224-235.

Cummings, E. M., Schermerhorn, A. C., Davies, P. T., Goeke-Morey, M. C., & Cummings, J. S.

(2006). Interparental discord and child adjustment: Prospective investigations of emotional security as an explanatory mechanism. *Child Development, 77*(1), 132-152.

Dai, J. P., Buijs, R., & Swaab, D. (2004). Glucocorticoid hormone (cortisol) affects axonal transport in human cortex neurons but shows resistance in Alzheimer's disease. *British Journal of Pharmacology, 143*(5), 606-610.

Davidson, R. J., Putnam, K. M., & Larson, C. L. (2000). Dysfunction in the neural circuitry of emotion-regulation: A possible prelude to violence. *Science, 289*(5479), 591-594.

Davies, P. T., & Cummings, E. M. (1994). Marital conflict and child adjustment – An emotional security hypothesis. *Psychological Bulletin, 116*(3), 387-411.

Davies, P. T., & Cummings, E. M. (1998). Exploring children's emotional security as a mediator of the link between marital relations and child adjustment. *Child Development, 69*(1), 124-139.

Davies, P. T., & Cummings, E. M. (2002). Study 1: Child responses to interparental conflict – Compaing the relative roles of emotional security and social learning processes. *Family & Relationships*, 41-62.

Davies, P. T., & Forman, E. M. (2002). Children's patterns of preserving emotional security in the interparental subsystem. *Child Development, 73*(6), 1880-1903.

Davies, P. T., Sturge-Apple, M. L., Cicchetti, D., Manning, L. G., & Zale, E. (2009). Children's patterns of emotional reactivity to conflict as explanatory mechanisms in links between interpartner aggression and child physiological functioning. *Journal of Child Psychology and Psychiatry, 50*(11), 1384-1391.

Davies, P. T., & Woitach, M. J. (2008). Children's emotional security in the interparental relationship. *Current Directions in Psychological Science, 17*(4), 269 – 274.

Dayton, C. J., Levendosky, A. A., Davidson, W. S., & Bogat, G. A. (2010). The child as held in The mind of the mother: The influence of prenatal maternal representations on parenting behaviors. *Infant Mental Health Journal, 31*(2), 220-241.

De Bellis, M. D. (2001). Developmental traumatology: The psychobiological development of maltreated children and its implications for research, treatment, and policy. *Development and Psychopathology, 13*, 539-564.

De Bellis M. D., Chrousos, G. P., Dorn, L. D., Burke, L., Helmers, K., Kling, M. A., Trickett, P. K., & Putnam, F.W. (1994). Hypothalamic pituitary adrenal dysregulation in sexually abused girls. *J Clin Endocrinology & Metabolism, 78*, 249–255.

De Bellis, M. D., Baum, A. S., Birmaher, B., Keshavan, M. S., Eccard, C. H., Boring, A. M., Jenkins, F. J., Ryan , N. D. (1999). Developmental traumatology Part I: Biological stress systems. *Biological Psychiatry, 45*, 1259-1270.

Dedovic, K., Engert, V., Duchesne, A., Lue, S. D., Andrews, J., Efanov, S. I., Beaudry, T., & Pruessner, J. C. (2010). Cortisol awakening response and hippocampal volume: vulnerability for major depressive disorder? *Biological Psychiatry, 68*(7), 847-853.

DeJonghe, E. S., Bogat, G. A., Levendosky, A. A., von Eye, A., & Davidson, W. S. (2005). Infant exposure to domestic violence predicts heightened sensitivity to adult verbal conflict. *Infant Mental Health Journal,* 26(3), 268-281.

Denham, S. A., Blair, K. A.., DeMulder, E., Levitas, J., Sawyer, K., Auerbach-Major, S., & Queenan, P. (2003). Preschool emotional competence: Pathway to social competence? *Child Development, 74*(1), 238-256.

De Weerth, C., & van Geert, P. (2002). A longitudinal study of basal cortisol in infants: Intra-individual variability, circadian rhythm and developmental trends. *Infant Behavior and Development, 25*, 375-398.

Dickerson, S. S. & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130*(3), 355-391.

Easterbrooks, M. A., & Emde, R. N. (1988). Marital and parent-child relationships: The role of affect in the family system. In R. Hinde, & J. Stevenson-Hinde (Eds.), *Relationships within families: Mutual influences* (pp. 83-102). Oxford: Oxford University Press.

Eisenberg, N., Spinrad T. L., Eggum, N. D., Silva, K. M., Reiser, M., Hofer, C., Smith, C. L., Gaertner, B. M., Kupfer, A., Popp, T., & Michalik, N. (2010). Relations among maternal socialization, effortful control, and maladjustment in early childhood. *Development and Psychopathology, 22*(3).

Ellis, B. J., Essex, M. J., & Boyce, W. T. (2005). Biological sensitivity to context: II. Empirical explorations of an evolutionary-developmental theory. *Development and Psychopathology, 17*(2), 303-328.

El-Sheikh, M. (2005). The role of emotional responses and physiological reactivity in the marital conflict-child functioning link. *Journal of Child Psychology and Psychiatry, 46*(11), 1191-1199.

El-Sheikh, M., Erath, S. A., Buckhalt, J. A., Granger, D. A., & Mize, J. (2008). Cortisol and childen’s adjustment: The moderating role of sympathetic nervous system activity. *Journal of Abnormal Child Psychology, 36*(4), 601-611.

El-Sheikh, M., Keller, P. S., & Erath, S. A. (2007). Marital conflict and risk for child maladjustment over time: Skin conductance level reactivity as a vulnerability factor. *Journal of Abnormal Child Psychology, 35*(5), 715-727.

El-Sheikh, M., & Whitson, S. A. (2006). Longitudinal relations between marital conflict and child adjustment: Vagal regulation as a protective factor. *Journal of Family Psychology, 20*(1), 30-39.

Elzinga, B. M., Roelofs, K., Tollenaar, M.S., Bakvis, P., van Pelt, J., & Spinhoven, P. (2008). Diminished cortisol responses to psychosocial stress associated with lifetime adverse events – A study among healthy young subjects. *Psychoneuroendocrinology, 33*(2), 227-237.

Elzinga, B. M., Schmahl, C. G., Vermetten, E., van Dyck, R., & Bremner, J. D. (2003). Higher cortisol levels following exposure to traumatic reminders in abuse-related PTSD. *Neuropsychopharmacology, 28*, 1656-1665.

Engfer, A. (1988). The interrelatedness of marriage and the mother-child relationship. In R. A. Hinde & J. Stevenson-Hinde (Eds.), Relationships within families: Mutual influences (pp. 105-118). Oxford, UK: Clarendon.

Ensor, R., Roman, G., Hart, M. J., & Hughes, C. (2012). Mothers' depressive symptoms and low mother-toddler mutuality both predict children's maladjustment. *Infant and Child Development, 21*(1), 52-66.

Ensor, R., Spencer, D., & Hughes, C. (2010). 'You feel sad?' Emotion understanding mediates effects of verbal ability and mother-child mutuality on prosocial behaviors: Findings from 2 years to 4 years. *Social Development, 20*(1), 93-110.

Ehrensaft, M. & Cohen, P. (2012). Contribution of family violence to the intergenerational transmission of externalizing behavior. *Prevention Science, 13*(4), 370-383.

Evans, S. E., Davies, C., & DiLillo, D. (2008). Exposure to domestic violence: A meta-analysis of child and adolescent outcomes. *Aggression and Violent Behavior, 13*(2), 131-140.

Falsetti, S. A., Resnick, H. S., Resick, P. A., & Kilpatrick, D. (1993). The Modified PTSD Symptom Scale: A brief self-report measure of posttraumatic stress disorder. *The Behavioral Therapist, 16*, 161-162.

Fantuzzo, J. W., Fusco, R. A., Mohr, W. K., & Perry, M. A. (2007). Domestic violence and children's presence: A population-based study of law enforcement surveillance of domestic violence. *Journal of Family Violence, 22*(6), 331-340.

Fantuzzo, J. W., & Mohr, W. K. (1999). Prevalence and effects of child exposure to domestic violence. *Future of Children, 9*(3), 21-32.

Feldman, R. (2006). From biological rhythms to social rhythms: Physiological precursors of mother-infant synchrony. *Developmental Psychology, 42*(1), 175-188.

Feldman, R. (2007a). Mother-infant synchrony and the development of moral orientation in childhood and adolescence: Direct and indirect mechanisms of developmental continuity. *American Journal of Orthopsychiatry, 77*(4), 582-597.

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

Feldman, R. (2010). The relational basis of adolescent adjustment: trajectories of mother-child interactive behaviors from infancy to adolescence shape adolescents' adaptation. *Attachment & Human Development, 12*(1-2), 173-192.

Feldman, R., & Greenbaum, C. W. (1997). Affect regulation and synchrony in mother-infant play as precursors to the development of symbolic competence. *Infant Mental Health Journal, 18*(1), 4-23.

Ferreira, A., Dahlöf, L. G., & Hansen, S. (1987). Olfactory mechanisms in the control of Maternal aggression, appetite and fearfulness – Effects of lesions to olfactory receptors, mediodorsal thalamic nucleus, and insular prefrontal cortex. *Behavioral Neuroscience, 101*(5), 709.

Field, L. R., Levendosky, A. A., Bogat, G. A., & von Eye, A. *Domestic violence and affective attunement in the mother-child dyad*. Unpublished manuscript.

Findling, J. W., & Raff, H. (2006). Clinical review: Cushing's syndrome: Important issues in diagnosis and management. *Journal of Endocrinology & Metabolism, 91*(10), 3746-3753.

Finger, B., Hans, S. L., Bernstein, V .J., & Cox, S. M. (2009). Parent relationship quality and infant-mother attachment. *Attachment & Human Development, 11*(3), 285-306.

Fleming, A. S., Miceli, M., & Moretto, D. (1993). Lesions of the medial preoptic area prevent the facilitation of maternal behavior produced by amygdala lesions. Physiology *& Behavior, 31*(4), 503-510.

Fonagy, P., Bateman, A., & Luyten, P. (2012). Overview and introduction. In A. Bateman & P. Fonagy (Eds.),*Handbook of Mentalizing in mental health practice*. (pp. 3-42). Washington, DC: American Psychiatric Press.

Fonzo, G. A., Simmons, A. N., Thorp, S. R., Norman, S. B., Paulus, M. P., & Stein, M. B. (2010). Exaggerated and disconnected insular-amygdalar blood oxygenation level-dependent response to threat-related emotional faces in women with intimate-partner violence posttraumatic stress disorder. *Biological Psychiatry, 68*(5), 433-441.

Fortin, A., Doucet, M., & Damant, D. (2011). Children's appraisals as mediators of the relationship between domestic violence and child adjustment. *Violence and Victims, 26*(3), 377-392.

Fosco, G. M., DeBoard, R. L., & Grych, J. H. (2007). Making sense of family violence – Implications of children's appraisals of interparental aggression for their short- and long-term functioning. *European Psychologist*, *12*(1), 6-16.

Frith, U., & Frith, C. D. (2003). Development and neurophysiology of mentalizing. *Philosophical Transactions of The Royal Society, 358*, 459-473.

Fusco, R. A., & Fantuzzo, J. W. (2009). Domestic violence crimes and children: A population-based investigation of direct sensory exposure and the nature of involvement. *Children and Youth Services Review*, *31*(2), 249-256.

Gardner, F., & Shaw, D. S. (2008). Behavioural problems of infancy and pre-school children. In M. Rutter, D. Bishop, D. Pine, S. Scott, J. Stevenson, E. Taylor, & A. Thapar (Eds.), *Rutter's child and adolescent psychiatry, 5th edition* (pp. 882-894). London: Blackwell Press.

George, C., & Solomon, J. (2008). The caregiving system: A behavioral systems approach to parenting. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (pp. 833-856). New York: The Guilford Press.

Gergely, G. & Watson, J. S. (1999). Early social-emotional development: Contingency perception and the social-biofeedback model. In Philippe Rochat *et al*. (Eds.), *Early social cognition: Understanding others in the first months of life* (pp. 101–136). Englewood Cliffs, NJ: Erlbaum.

Gewirtz,A. H., DeGarmo, D. S., & Medhanie, A. (2011). Effects of mother's parenting practices on child internalizing trajectories following partner violence. *Journal of Family Psychology*, *25*(1), 29-38.

Gianino, A. & Tronick, E. Z. (1992). The mutual regulation model: The infant's self and Interactive regulation and coping and defensive capacities. In T. M. Field, P. M. McCabe & N. Schneiderman (Eds.). *Stress and coping across development.* Englewood Cliffs, NJ: Lawrence Erlbaum Associates.

Goldberg, S., MacKay-Soroka, S., & Rochester, M. (1994). Affect, attachment, and maternal responsiveness. *Infant Behavior & Development*, *17*(3), 335-339.

Gotlib, I. H., Joormann, J., Hallmayer, J., & Minor, K. L. (2008). Biological stress reactivity mediates the relation between genotype and risk for depression. *Biological Psychiatry, 63,* 847-851.

Graham-Bermann, S. A., & Seng, J. (2005). Violence exposure and traumatic stress symptoms as additional predictors of health problems in high-risk children. *Journal of Pediatrics, 146*(3), 349-354.

Granger, D. A., Kivlighan, K. T., Fortunato, C., et al. (2007). Integration of salivary biomarkers into developmental and behaviorally-oriented research: Problems and solutions for collecting specimens. *Physiol Behav, 92*(4), 583-90.

Granger, D. A., Weisz, J. R. & Kauneckis, D. (1994). Neuroendocrine reactivity, internalizing behavior problems, and control-related cognitions in clinic-referred children and adolescents. *Journal of Abnormal Psychology, 103*(2), 267-276.

Granger, D. A., Weisz, J. R., McCracken, J. T., Ikeda, S. C., & Douglas, P. (1996). Reciprocal influences among adrenocortical active
ation, psychosocial processes, and the behavioral adjustment of clinic-referred children. *Child Development, 67*(6), 3250-3262.

Griffin, M. G., Resick, P. A., & Yehuda, R. (2005). Enhanced cortisol suppression following dexamethasone administration in domestic violence survivors. *American Journal of Psychiatry, 162*(6), 1192-1199.

Grych, J. H. & Fincham, F. D. (1990). Marital conflict and children's adjustment: A cognitive-contextual framework. *Psychological Bulletin, 108*(2), 267-290.

Guerry, J. D. & Hastings, P. D. (2011). In search of HPA axis dysregulation in child and adolescent depression. *Clinical Child and Family Psychology Review, 14*(2), 135-160.

Gunnar, M. R., Brodersen, L., Krueger, K., & Rigatuso, J. (1996). Dampening of adrenocortical responses during infancy: Normative changes and individual differences. *Child Development, 67*, 877-889.

Gunnar, M., & Quevedo, K. (2007). The neurobiology of stress and development. *Annual Review of Psychology, 58*, 145-173.

Gunnar, M. R., & Vasquez, D. M. (2001). Low cortisol and a flattening of expected daytime rhythm: Potential indices of risk in human development. *Development and Psychopathology, 13*, 515-538.

Haft, W. L., & Slade, A. (1989). Affect attunement and maternal attachment: A pilot study. *Infant Mental Health Journal, 10*(3), 157-172.

Ham, J., & Tronick, E. (2009). Relational psychophysiology: Lessons from mother-infant physiology research on dyadically expanded states of consciousness. *Psychotherapy Research, 19*(6), 619-632.

Harris, P. L. (1989). Children and emotion: The development of psychological understanding. Cambridge, MA: Basil Blackwell.

Harris, P. L., Johnson, C. N., Hutton, D., Andrews, G., & Cooke, T. (1989). Young children's theory of mind and emotion. *Cognition & Emotion, 3*(4), 379-400.

Hastings, P. D., Ruttle, P., Serbin, L. A., Mills, R. S. L., Stack, D.M., & Schwartzman, A.E. . (2011). Adrenocortical stress reactivity and regulation in preschoolers: Relations with parenting, temperament, and psychopathology. *Developmental Psychobiology, 53*, 694-710.

Hatfield, E., Cacioppo, J., & Rapson, R. L. (1994). Emotional contagion. New York: Cambridge University Press.

Hazen, A. L., Connelly, C. D., Kelleher, K. J., Barth, R. P., & Landsverk, J. A. (2006). Female caregivers' experiences with intimate partner violence and behavior problems in children investigated as victims of maltreatment. *Pediatrics, 117*(1), 99-109.

Healey, D. M., Gopin, C. B., Grossman, B. R., Campbell, S. B., & Halperin, J. M. (2010). Mother-child dyadic synchrony is associated with better functioning in hyperactive/inattentive preschool children. *Journal of Child Psychology and Psychiatry, 51*(9), 1058-1066.

Heim, C., Ehlert, U., & Hellhammer, D. H. (2000). The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders. *Psychoneuroendocrinology, 25,* 1–35.

Hernández-González, M., Navarro-Meza, M., Prieto-Beracoechea, C. A., & Guevara, M. A. (2005). Electrical activity of prefrontal cortex and ventral tegmental area during rat maternal behavior. *Behavioural Processes, 70*(2), 132-143.

Hessl, D., Rivera, S. M., & Reiss, A. L. (2004). The neuroanatomy and neuroendocrinotogy of fragile X syndrome. *Mental Retardation and Developmental Disabilities Research Reviews, 10*(1), 17-2.

Hibel, L. C., Granger, D. A., Blair, C., & Cox, M. J. (2009). Intimate partner violence moderates the association between mother-infant adrenocortical activity across an emotional challenge. *Journal of Family Psychology, 23*(5), 615-625.

Hibel, L. C., Granger, D. A., Blair, C., Cox, M., & The Family Life Project Investigators. (2011). Maternal sensitivity buffers the adrenocortical implications of intimate partner violence exposure during early childhood. *Development and Psychopathology, 23*, 689-701.

Hill, T. D., Nielsen, A. L., & Angel, R. J. (2009). Relationship violence and frequency of intoxication among low-income urban women. *Substance Use & Misuse, 44*(5), 684-701.

Hill, T. D., Schroeder, R. D., Bradley, C., Kaplan, L. M., & Angel, R. J. (2009). The long-term health consequences of relationship violence in adulthood: An examination of low-income women from Boston, Chicago, and San Antonio. *American Journal of Public Health, 99*(9), 1645-1650.

Hodgins, H. S., & Belch, C.(2000). Interparental violence and nonverbal abilities. *Journal of Nonverbal Behavior, 24*(1), 3-24.

Holden, G. W., & Ritchie, K. L. (1991). Linking extreme marital discord, child rearing, and child-behavior problems: Evidence from battered women. *Child Development, 62*(2), 311-327.

Hu, L. T. & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Convential criteria versus new alternatives. *Structural Equation Modeling, 6*(1), 1-55.

Huang, C. C., Wang, L. R., & Warrener, C. (2010). Effects of domestic violence on behavior problems of preschool-aged children: Do maternal mental health and parenting mediate the effects*? Children and Youth Services Review, 32*(10), 1317-1323.

Huston, P. L., Esser-Kahn, A. A., Bogat, G. A., von Eye, A. Levendosky, A. A., & Davidson, W. S. (2010, August). Do psychological and physical IPV affect male and female children's behavior problems? Poster presented at the 118th annual convention of the American Psychological Association, San Diego, CA.

Huth-Bocks, A. C., & Hughes, H. M. (2008). Parenting stress, parenting behavior, and children's adjustment in families experiencing intimate partner violence. *Journal of Family Violence, 23*(4), 243-251.

Huth-Bocks, A. C., Levendosky, A. A., Theran, S. A., & Bogat, G. A. (2004). The impact of domestic violence on mothers' prenatal representations of their infants. *Infant Mental Health Journal, 25*(2), 79-98.

Inslicht, S. S., Marmar, C. R., Neylan, T. C., Metzler, T. J., Hart, S. L., Otte, C., McCaslin, S. E., Larkin, G. L., Hyman, K. B., & Baum, A. (2006). Increased cortisol in women with intimate partner violence-related posttraumatic stress disorder. *Psychoneuroendocrinology, 31*(7), 825-838.

Jaffee, P., Wolfe, D., & Wilson, S. (1990). Children of battered women. Newbury Park, CA: Sage.

Johnson, M. P. (1995). Patriarchal terrorism and common couple violence: Two forms of violence against women. *Journal of Marriage and Fanily, 57*(2), 283-294.

Johnson, M. P. (2005). Domestic violence: Not about gender – or is it? *Journal of Marriage and*

*Family, 67*, 1126-1130.

Johnson, M. P., & Leone, J. M. (2005). The differential effects of intimate terrorism and situational couple violence: Findings from the National Violence Against Women Survey. *Journal of Family Issues, 26*, 322-349.

Johnson, J. K., & Lieberman, A. F. (2007). Variations in behavior problems of preschoolers exposed to domestic violence: The role of mothers' attunement to children's emotional experiences. *Journal of Family Violence, 22*, 297-308.

Jones, A., Godfrey, K. M.,Wood, P., Osmond, C., Goulden, P., & Phillips, D. I. W. (2005). Fetal growth and the adrenocortical response to psychological stress. *Pediatric Research, 58*(5), 1097-1097.

Jonsson, C. O., Clinton, D. N., Fahrman, M., Mazzaglia, G., Novak, S., & S□rhus, K. (2001). How do mothers signal shared feeling-states to their infants? An investigation of affect attunement and imitation during the first year of life. *Scandanavian Journal of Psychology, 42*(4), 377-381.

Jöreskog, K. G. & Sörbom, D. (2004). *LISREL 8.7 for Windows [Computer software]*. Lincolnwood, IL: Scientific Software International, Inc.

Jöreskog, K. G. & Sörbom, D. (2006). *LISREL 8.8 Student Version for Windows*. *[Computer software]*. Lincolnwood, IL: Scientific Software International, Inc.

Kaplan, L. A., Evans, L., & Monk, C. (2008). Effects of mothers' prenatal psychiatric status and postnatal caregiving on infant biobehavioral regulation: Can prenatal programming be modified? *Early Human Development, 84*(4), 249-256.

Kapoor, A., Dunn , E., Kostaki, A., Andrews, M. H., & Matthews, S. G. (2006). Fetal programming of hypothalamo-pituitary-adrenal function: prenatal stress and glucocorticoids. *Journal of Physiology – London, 572*(1), 31-44.

Katz, L. F., Hessler, D. H. & Annest, A. (2007). Domestic violence. Emotional competence, and child adjustment. *Social Development, 16*(3), 513-538.

Katz, L. F., Hunter, E., & Klowden, A. (2008). Intimate partner violence and children's reaction to peer provocation: The moderating role of emotion coaching. *Journal of Family Psychology, 22*(4), 614-621.

Katz, L. F., & Windecker-Nelson, B. (2006). Domestic violence, emotional coaching, and child adjustment. *Journal of Family Psychology, 2*0(1), 56-67.

Kelly, J. B., & Johnson, M. P. (2008). Differentiation among types of intimate partner violence: Research update and implications for interventions. *Family Court Review, 46*(3) 476-499.

Kerig, P. K. (1998). Gender and appraisals as mediators of adjustment in children exposed to interparental violence. *Journal of Family Violence*, *13*(4), 3345-363.

Kernic, M. A., Wolf, M. E., Holt, V. L., McKnight, B., Huebner, C. E., Rivara, F. P. (2003). Behavioral problems among children whose mothers are abused by an intimate partner. *Child Abuse & Neglect*, *27*(11), 1231-1246.

Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M., Nelson, C. B. (1995). Posttraumatic Stress Disorder in the National Comorbidity Survey. *Archives of General Psychiatry*, 52, 1048-1060.

Keysers, C. & Gazzola, V. (2007). Integrating simulation and theory of mind: From self to social cognition. *Trends in Cognitive Sciences*, *11*(5), 194-196.

Kinard, E. M. (1998). Depressive symptoms in maltreated children from mother, teacher, and child perspectives. *Violence and Victims*, *13*(2), 131-147.

Kitzmann, K. M., Gaylord, N. K., Holt, A. R., & Kenny, E. D. (2003). Child witnesses to domestic violence: A meta-analytic review. *Journal of Consulting and Clinical Psychology*, *71*(2). 339-352.

Klaassens, E. R., Giltay, E. J., Cuijpers, P., van Veen, T., & Zitman, F. G. (2012). Adulthood trauma and HPA-axis functioning in healthy subjects and PTSD patients: A meta-analysis. *Psychoneuroendocrinology*, *37*(3), 317-331.

Kochanska, G. (1997). Mutually responsive orientation between mothers and their young children: Implications for early socialization. *Child Development*, *68*, 94-112.

Kochanska, G., Aksan, N., Prisco, T. R., & Adams, E. E. (2008). Mother-child and father-child mutually responsive orientation in the first 2 years and children's outcomes at preschool age: Mechanisms of influence. *Child Development*, *79*(1), 30-44.

Kochanska, G., & Murray, K. T. (2000). Mother-child mutually responsive orientation and conscience development: From toddler to early school age. *Child Development*, *71*(2), 417-431.

Krippendorff, K. (1980). *Content analysis: an introduction to its methodology*. Beverly Hills: Sage Publications.

Krishnakumar, A., & Buehler, C. (2000). Interparental conflict and parenting behaviors: A meta-analytic review. *Family Relations*, *49*(1), 25-44.

Kudielka, B. M., Hellhammer, D. H., & Wüst, S. (2009). Why do we respond so differently?

Reviewing determinants of human salivary cortisol responses to challenge. *Psychoneuroendocrinology, 34*, 2-18.

Laurent, H. K., Stevens, A., & Albow, J. C. (2011). Neural correlates of hypothalamix-pituitary-adrenal regulation of mothers with their infants. *Biological Psychiatry, 70*(9), 826-832.

Laursen, B. P., & Hoff, E. (2006). Person-centered and variable-centered approaches to longitudinal data. *Merrill-Palmer Quarterly, 52*(3), 377-389.

Lee, C. L., & Bates, J. E. (1985). Mother-child interaction at age 2 years and perceived difficult temperament. *Child Development, 56*(5), 1314-1325.

Lenzi, D., Trentini, C., Pantano, P., Macaluso, E., Iacoboni, M., Lenzi, G. L., & Ammaniti, M. (2009). Neural basis of maternal communication and emotional expression processing during infant preverbal stage. Cerebral Cortex, 19(5), 1124-1133.

Leone, J. M., Johnson, M. P., Cohan, C. L., & Lloyd, S. E. (2004). Consequences of male partner violence for low-income minority women. *Journal of Marriage and the Family, 66*(2), 472-490.

Lerner, R. M., & Lerner, J. V. (1983). Temperament-intelligence reciprocities in childhood: A contextual model. In *Origins of intelligence: Infancy and childhood*, Lewis, M. (ed.), (2nd Edn.) New York: Plenum Press.

Levendosky, A. A., Bogat, G. A., & Huth-Bocks, A. C. (2011). The influence of domestic violence on the development of the attachment relationship between mother and young child. *Journal of Psychoanalytic Psychology,* 28, 512-517.

Levendosky, A. A., & Graham-Bermann, A. A. (1998). The moderating effects of parenting stress on children's adjustment in woman-abusing families. *Journal of Interpersonal Violence, 13*(3), 383-397.

Levendosky, A. A., Huth-Bocks, A. C., Shapiro, D. L., & Semel, M. A. (2003). The impact of domestic violence on the maternal-child relationship and preschool-age children's functioning. *Journal of Family Psychology, 17*(3), 275-287.

Levendosky, A. A., Leahy, K. L., Bogat, G. A., Davidson, W. S., & von Eye, A. (2006). Domestic violence, maternal parenting, maternal mental health, and infant externalizing behavior. *Journal of Family Psychology*, 20, 544-552.

Lieberman, D. L. (2007). Social cognitive neuroscience: A review of core processes. *Annual Review of Psychology, 58*, 259-289.

Lieberman, A. F., & Knorr, K. (2007). The impact of trauma: A developmental framework for infancy and early childhood. *Psychiatric Annals, 37*(6), 416-422.

Lindhorst, T. & Oxford, M. (2008). The long-term effects of intimate partner violence on adolescent mothers' depressive symptoms. *Social Science & Medicine, 66*(6), 1322-1333.

Lindsey, E. W., Cremeens, P. R., Colwell, M. J., & Coldera, Y. M. (2009). The structure of parent-child dyadic synchrony in toddlerhood and children's communication competence and self-control. *Social Development, 18*(2), 375-396.

Lindsey, E. W., Cremeens, P. R., & Caldera, Y. M. (2010). Mother-child and father-child mutuality in two contexts: Consequences for young children's peer relationships. *Infant and Child Development, 19*(2), 142-160.

Litrownik, A. J., Newton, R., Hunter, W. M., English, D. & Everson, M. D. (2003). Exposure to family violence in young at-risk children: A longitudinal look at the effects of victimization and witnessed physical and psychological aggression. *Journal of Family Violence, 18*(1), 59-73.

Lorberbaum, J. P., Newman, J. D., Dubno, J. R., Horwitz, A. R., Nahas, Z., Teneback, C. C., Bloomer, C. W., Bohning, D. E., Vincent, D., Johnson, M. R., Emmanuel, M., Brawman-Mintzer, O., Book, S. W., Lydiard, R. B., Ballanger, J. C., & George, M. S. (1999). Feasibility of using fMRI to study mothers responding to infant cries. *Depression and Anxiety, 10*(3), 99-104.

Lorberbaum, J. P., Newman, J. D., Horwitz, A. R., Dubno, J. R., Lydiard, R. B., Hamner, M. B., Bohning, D. E., & George, M. S. (2002). A potential role for thalamocingulate circuitry in human maternal behavior. *Biological Psychiatry, 51*(6), 431-445.

Lovell, B., Moss, M., & Wetherell, M. A. (2011). Perceived stress, common health complaints and diurnal patterns of cortisol secretion in young, otherwise healthy individuals. *Hormones & Behavior*, *60*(3), 301-305.

Lui, L., Li, A. T., & Matthews, S .G. (2001). Maternal glucocorticoid treatment programs HPA regulation in adult offspring: sex-specific effects. *American Journal of Physiologuy-Endocrinology and Metabolism, 280*(5), E729-E739.

Lunkenheimer, E. S., Olson, S. L., Hollenstein, T., Sameroff, A. J., & Winter, C. (2011). Dyadic flexibility and positive affect in parent-child coregulation and the development of child behavior problems. *Development & Psychopathology*, *23*(2), 577-591.

Malik, N. M. (2008). Exposure to domestic and community violence in a nonrisk sample – Associations with child functioning. *Journal of Interpersonal Violence, 23*(4), 490-504.

Marci, C. D., Ham, J., Moran, E., & Orr, S. P. (2007). Physiologic correlates of perceived therapist empathy and social-emotional process during psychotherapy. Journal *of Nervous and Mental Disease, 195*(2), 103-111.

Margolin, G., & Gordis, E. B., (2000). The effects of family and community violence on children. *Annual Review of Psychology, 51*, 445-479.

Margolin, G., & Gordis, E. B., Medina, A. M., & Oliver, P. H. (2003). The co-occurrence of husband-to-wife aggression, family-of-origin aggression, and child abuse potential in a community sample – Implications for parenting. *Journal of Interpersonal Violence, 18*(4), 413-440.

Martinez-Torteya, C., Bogat, G. A., von Eye, A., Davidson, W. S., & Levendosky, A. A. (2009). Women's appraisals of intimate partner violence stressfulness and their relationship to depressive and posttraumatic stress disorder symptoms.*Violence and Victims*, *24*, 707-722.

Martinez-Torteya, C., Huston, P., Bogat, G. A., Levendosky, A. A., Davidson, W. S., & von Eye, A. (2008, November). Psychological IPV predicts women's mental health and children's behavior problems. Poster presented at the 24$^{th}$ annual meeting of the International Society for Traumatic Stress Studies, Chicago, IL.

Mason, J. (1968). A review of psychoendocrine research on the pituitary adrenal cortical system. *Psychosomatic Medicine, 30*, 576-583.

Mayes, L. C. (2006). Arousal regulation, emotional flexibility, medial amygdala function, and the impact of early experience - Comments on the paper of Lewis et al. *Resilience in Children, Annals of the New York Academy of Sciences, 1094*, 178-192.

McCloskey, L. A., & Stuewig, J. (2001). The quality of peer relationships among children exposed to family violence. *Development and Psychopathology, 13*(1), 83-96.

McDonald, R., Jouriles, E. N., Briggs-Gowan, M. J., Rosenfield, D., & Carter, A. S. (2007). Violence toward a family member, angry adult conflict, and child adjustment difficulties: Relations in families with 1-to 3-year-old children. *Journal of Family Psychology, 21*(2), 176-184.

McDonald, R., Jouriles, E. N., Ramisetty-Mikler, S., Caetano, R., & Green, C. E. (2006). Estimating the number of American children living in partner-violent families. *Journal of Family Psychology, 20*(1), 137-142.

McEwen, B. S. (Ed.). (1994). Stress and the nervous system. *Seminars in the Neurosciences, 6*(4), 195–280.

McFarlane, J. M., Groff, J. Y., O'Brien, J. A., & Watson, K. (2003). Behaviors of children who are exposed and not exposed to intimate partner violence: An analysis of 330 black, white, and Hispanic children. *Pediatrics, 112*(3), E202-E207.

McHugo, G. J., Caspi, Y., Kammerer, N., Mazelis, R., Jackson, E. W., Russell, L., Clark, C., Liebschutz, J., & Kimmerling, R. (2005). The assessment of trauma history in women with co-occurring substance abuse and mental disorders and a history of interpersonal violence. *Journal of Behavioral Health Services & Research, 32*, 113-127.

Mechanic, M. B., Weaver, T. L., & Resick, P. A. (2008). Mental health consequences of intimate partner abuse – A multidimensional assessment of four different forms of abuse. *Violence Against Women, 14*(6), 634-654.

Meewisse, M. L., Reitsma, J. B., De Vries, G. J., Gersons, B. P. R., & Olff, M. (2007). Cortisol and post-traumatic stress disorder in adults – Systematic review and meta-analysis. *British Journal of Psychiatry, 191*, 387-392.

Meltzer, H., Doos, L., Vostanis, P., Ford,T., & Goodman, R. (2009). The mental health of children who witness domestic violence. *Child & Family Social Work, 14*(4), 491-501.

Milner, J. S., Halsey, L. B., & Fultz, J. (1995). Empathic responsiveness and affective reactivity to infant stimuli in high- and low-risk for physical child abuse mothers. *Child Abuse & Neglect, 19*(6), 767-780.

Minze, L. C., McDonald, R., Rosentraub, E. L., & Jouriles, E. N. (2010). Making sense of family conflict: Intimate partner violence and preschoolers' externalizing problems. *Journal of Family Psychology, 24*(1), 5-11.

Munkvold, L., Lundervold, A., Lie, S. A., & Manger, T. (2009). Should there be separate parent and teacher-based categories of ODD? Evidence from a general population. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 50*(10), 1264-1272.

Nemeroff, C. B. (2004). Neurobiological consequences of childhood trauma. *Journal of Clinical Psychiatry, 65*(1), 18-28.

Newman, L. K., Harris, M., & Allen, J. (2011). Neurobiological basis of parenting disturbance. *Australian and New Zealand Journal of Psychiatry, 45*, 109-122.

Nicolson, N. A. (2008). Measurement of cortisol. In L.J. Luecken, & L.G. Gallo (Eds.) *Handbook of psychological research methods in health psychology*. Thousand Oaks, CA: SAGE Publications, Inc.

Numan, M., Numan, M. J., & English, J. B. (1993). Excitotoxic amino acid injections into the medial amygdala facilitate maternal behaviorin virgin female rats. *Hormones and Behavior, 27*(1), 56-81.)

O'Campo, P., Caughy, M. O., & Nettles, S. M. (2010). Partner abuse or violence, parenting and neighborhood influences on children's behavioral problems. *Social Science and Medicine, 70*(9), 1404-1415.

O'Leary, K. D. (1999). Psychological abuse: A variable deserving critical attention in domestic violence. *Violence and Victims, 14*(1), 3-23.

Onyskiw, J. E., & Hayduk, L. A. (2001). Processes underlying children's adjustment in families characterized by physical aggression. *Family Relations, 50*(4), 376-385.

Owen, A. E., Thompson, M. P., & Kaslow, N. J. (2006). The mediating role of parenting stress in the relation between intimate partner violence and child adjustment. *Journal of Family Psychology, 20*(3), 505-513.

Owen, A. E., Thompson, M. P., Mitchell, M. D., Kennebrew, S. Y., Paranjape, A., Reddick, T. L., Hargrove, G. L., & Kaslow, N. J. (2008). Perceived social support as a mediator of the link between intimate partner conflict and child adjustment. *Journal of Family Violence, 23*(4), 221-230.

Owen, A. E., Thompson, M. P., Shaffer, A., Jackson, E. B., & Kaslow, N. J. (2009). Family variables that mediate the relation between intimate partner violence (IPV) and child adjustment. *Journal of Family Violence, 24*(7), 433-445.

Papp, L. M., Pendry, P., & Adam, E. K., (2009). Mother-adolescent physiological synchrony in naturalistic settings: Within-family cortisol associations and moderators. *Journal of Family Psychology, 23*(6), 882-894.

Pereira, M. & Morrell, J. I. (2011). Functional mapping of the neural circuitry of rat maternal motivation: Effects of site-specific transient neural inactivation. *Journal of Neuroendocrinology, 23*(11), 1020-1035.

Phillips, D. I. W., & Jones, A. (2006). Fetal programming of autonomic and HPA function: do people who were small babies have enhanced stress responses? *Journal of Physiology – London, 572*(1), 45-50.

Pico-Alfonso, M. A., Garcia-Linares, M. I., Celda-Navarro, N., & Herbert, J., & Martinez, M. (2004). Changes in cortisol and dehydroepiandrosterone in women victims of physical and psychological intimate partner violence. *Biological Psychiatry, 56*(4), 233-240.

Próspero, M. (2008). The effect of coercion on aggression and mental health among reciprocally violent couples. *Journal of Family Violence, 23,* 195-202.

Pryce, C. R., Aubert, Y., Maier, C., Pearce, P. C., & Fuchs, E. (2011). The developmental impact of prenatal stress, prenatal dexamethasone and postnatal social stress on physiology, 273ehavior and neuroanatomy of primate offspring: studies in rhesus macaque and common marmoset. *Psychopharmacology, 214*(1), 33-53.

Raison, C. L., Borisov, A. S., Woolwine, B. J., Massung, B., Vogt, G., & Miller, A. H. (2010). Interferon-alpha effects on diurnal hypothalamic-pituitary-adrenal axis activity: relationship with proinflammatory cytokines and behavior. *Molecular Psychiatry, 15*(5), 535-547.

Reid-Cunningham, A. R. (2009). Parent-child relationship and mother's sexual assault history. *Violence Against Women, 15*(8), 920-932.

Repetti, R. L. (1987). Linkages between work and family roles. *Applied Social Psychology Annual, 7*, 98-127.

Roberts, J. E., Clarke, M. A., Alcorn, K., Carter, J. C., Long, A. C. J., & Kaufmann, W. E. (2009). Autistic behavior in boys with fragile X syndrome: social approach and HPA-axis dysfunction. *Journal of Neurodevelopmental Disorders, 1*(4), 283-291.

Robinson, E. A., & Eyberg, S. M. (1981). The dyadic parent-child interaction coding system: Standardization and validation. *Journal of Consulting & Clinical Psychology, 49*(2), 245-250.

Rodriguez, M, A., Heilemann, M. V., Fielder, E., Ang, A., Nevarez, F., & Mangione, C. M. (2008). Intimate partner violence, depression, and PTSD among pregnant Latina women. *Annals of Family Medicine, 6*(1), 44-52.

Rose, R. M. (1984). Overview of endocrinology of stress. In G.M. Brown, S.H. Koslow, & S. Reichlin (Eds.), *Neuroendocrinology and psychiatric disorder*. New York: Raven Press.

Roskam, I., Stiévenart, M., Meunier, J. C., Van de Moortele, G., Kinoo, P., & Nassogne, M. C. (2010). The reliability of early diagnostic of externalizing behavior in question? Towards a multi-informant and multimethod strategy. *Practiques Psychologiques, 17*(2). 189-200.

Ruttle, P. L., Serbin, L. A., Stack, D. M., Schwartzman, A. E., & Shirtcliff, E. A. (2011). Adrenocortical attunement in mother-child dyads: Importance of situational and behavioral characteristics. *Biological Psychology, 88*(10), 104-111.

Salimetrics. (2008). Saliva collection and handling advice. Salimetrics, LLC.

Salimetrics. (2012). Inter- and intra-assay coefficients of variability. Salimetrics, LLC. Retrieved From http://www.salimetrics.com/documents/spittips/publications/Inter%20and%20 Intra%20Assay%20Coefficients%20of%20Variability.pdf

Saltzman, K. M., Holden, G. W., & Holahan, C. J. (2005). The psychobiology of children exposed to marital violence. *Journal of Clinical Child and Adolescent Psychology*, *34*(1), 129-139.

Schechter, D. S., Zeanah, C. H., Myers, M. M., Brunelli, S. A., Liebowitz, M. R., Marshall, R. D., Coates, S. W., Trabka, K. A., Baca, P., & Hofer, M. A. (2004). Psychobiological dysregulation in violence-exposed mothers: Salivary cortisol of mothers with very young children pre- and post-separation stress. *Bulletin of the Menninger Clinic*, *68*(4), 319-336.

Schechter, D. S., Zygmunt, A.,Coates, S. W., Davies, M., Trabka, K. A., McCaw, J., Kolodji, A., & Robinson, J. L. (2007). Caregiver traumatization adversely impacts young children's mental representations on the MacArthur Story Stem Battery. *Attachment and Human Development*, *9*(3), 187-205.

Schore, A. N. (2002). Dysregulation of the right brain: A fundamental mechanism of traumatic attachment and the psychopathogenesis of posttraumatic stress disorder. *Australian and New Zealand Journal of Psychiatry, 36*(1), 9-30.

Schreiber, J. E., Shirtcliff, E., Van Hulle, C., Lemery-Chalfant, K., Klein, M.H., Kalin, N.H., Essex, M.J., & Goldsmith, H.H. (2006). Enviromental influences on family similarity in afternoon cortisol levels: Twin and parent-offspring designs. *Psychoneuroendocrinology, 31*, 1131-1137.

Schultheiss, O. C., & Stanton, S. J. (2009). Assessment of salivary hormones. In Harmon-Jones, E., Beer, J.S., eds. *Methods in Social Neuroscience*. New York: Guilford Press.

Seckl, J. R. (2004). Prenatal glucocorticoids and long-term programming. *European Journal of Endocrinology, 151*, 49-62.

Seedat, S., Stein, M. B., Kennedy, C. M., & Hauger, R. L. (2003). Plasma cortisol and neuropeptide Y in female victims of intimate partner violence. *Psychoneuroendocrinology, 28*(6), 796-808.

Seedat, S., Videen, J. S., Kennedy, C. M., & Stein, M. B. (2005). Single voxel proton magnetic resonance spectroscopy in women with and without intimate partner violence-related posttraumatic stress disorder. *Psychiatry Research-Neuroimaging*, *139*(3), 249-258.

Sethre-Hofstad, L., Stansbury, K., & Rice, M. A. (2002). Attunement of maternal and child adrenocortical response to child challenge. *Psychoneuroendocrinology, 27*(6), 731-747.

Singer, T., Seymour, B., O'Doherty, Kaube, H., Dolan, R. J., & Frith, C. D. (2004). Empathy for pain involves the affective but not sensory components of pain. Science, 303, 1157-1162.

Shin, L. M, , Rauch, S. L., & Pitman, R. K. (2006). Amygdala, medial prefrontal cortex, and hippocampal function in PTSD. *Annals of the New York Academy of Sciences*, 1071, 67-79.

Skopp, N. A., McDonald, R., Jouriles, E. N., & Rosenfield, D. (2007). Partner aggression and children's externalizing problems: Maternal and partner warmth as protective factors. *Journal of Family Psychology, 21*(3), 459-467.

Slotnick, B. M. (1967). Disturbances of maternal behavior in rat following lesions of cingulate cortex. *Behaviour, 29*, 204.

Slotnick, B. M., & Nigrosh, B. J. (1975). Maternal behavior of mice with cingulate cortical, amygdala, or septal lesions. *Journal of Comparative and Physiological Psychology, 88*(1), 118-127.

Snell, M. E., Berlin, R. A., Voorhees, M. D., Stanton-Chapman, T. L., & Hadden, S. (2012). A survey of preschool staff concerning problem behavior and its prevention in Head Start classrooms. *Positive Behavior Interventions, 14*(2), 98-107.

Sokolowski, M. S., Hans, S. L., & Bernstein, V .J. (2007). Mothers' representations of their infants and parenting behavior: Associations with personal and social-contextual variables in a high-risk sample. *Infant Mental Health Journal, 28*(3), 344-365.

Stansbury, K., & Gunnar, M. R. (1994). Adrenocortical activity and emotional regulation. *Monographs of the Society for Research in Child Development, 59* (2/3), 108- 134.

StataCorp. (2009). *Stata Statistical Software: Release 11*. College Station, TX: StataCorp LP.

Stephens, D. L. (1999). Battered women's views of their children. *Journal of Interpersonal Violence, 14*, 731-746.

Stern, D. N. (1974). Mother and infant at play: the dyadic interaction involving facial, vocal, and gaze behaviors. In M. Lewis & L. A. Rosenblum (Eds.), The effect of the infant on its caregiver. New York: Wiley.

Sternberg, K. J., Baradaran, L. P., Abbott, C. B., Lamb, M. E., & Guterman, E. (2006). Type of violence, age, and gender differences in the effects of family violence on children's behavior problems: A mega-analysis. *Developmental Review, 26*(1), 89-112.

Stinson, J. N., Kavanaugh, T.,Yamada, J., Gill, N., & Stevens, B. (2006). Systematic review of the psychometric properties, interpretability and feasibility of self-report pain intensity measures for use in clinical trials in children and adolescents. *Pain, 125*(1-2), 143-157.

Strathearn, L., Li., J., Fonagy, P., & Montague, P. R. (2008). What's in a smile? Maternal brain responses to infant facial cues. *Pediatrics, 122*(3), 689-689.

Straus, M. A. (1992). Children as witnesses to marital violence: A risk factor for lifelong problems among a nationally representative sample of American men and women. Report of the Twenty-Third Ross Roundtable. Columbus, OH: Ross Laboratories.

Straus, M. A., Hamby, S. L., Boney-McCoy, S., & Sugarman, D. B. (1996). The revised Conflict Tactics Scales (CTS2): Development and preliminary psychometric data. *Journal of Family Issues, 17*(3), 283-316.

Street, A. E., King, L. A., King, D. W., & Riggs, D. S. (2003). The associations among male – Perpetrated partner violence, wives' psychological distress and children's behavior problems: A structural equation modeling analysis. *Journal of Comparative Family Studies, 34*(1), 23.

Strickland, J., Hopkins, J. & Keenan, K. (2012). Mother-teacher agreement on preschoolers' symptoms of ODD and CD: Does context matter? *Journal of Abnormal Child Psychology, 40*(6), 933-943.

Swain, J. E. (2011). The human parental brain: In vivo neuroimaging. *Progress in Neuro-psychopharmacology & Biological Psychiatry, 35*(5), 1242-1254.

Swain,J. E., Konrath, S., Brown, S. L., Finegood, E. D., Akce, L. B., Dayton, C. J., & Ho, S. S. (2012). Parenting and beyond: Common neurocircuits underlying parental and altruistic caregiving. *Parenting: Science and Practice, 12*(2-3), 115-123.

Swain, J. E., Leckman, J. F., Mayes, L. C., Feldman, R., Constable, R. T., & Schultz, R. T. (2003). The neural circuitry of parent-infant attachment in the early postpartum. In *American College of Neuropsychopharmacology,* Peurto Rico.

Swain, J. E., Leckman, J. F., Mayes, L. C., Feldman, R., & Schultz, R. T. (2004). Functional brain imaging and psychology of human parent-infant attachment. *Neuropsychopharmacology, 29*(1), S195.

Swain, J. E., Leckman, J. F., Mayes, L. C., Feldman, R., & Schultz, R. T. (2006). Own baby pictures induce parental brain activations according to psychology, experience and postpartum timing. *Biological Psychiatry, 59*(8), 126S.

Talwar, R., Nitz, K., & Lerner, R. M. (1990). Relations among early adolescent temperament, parent and peer demands, and adjustment: A test of the goodness of fit model. *Journal of Adolescence, 13*, 279-298.

Theran, S. A., Levendosky, A. A., Bogat, G. A., & Huth-Bocks, A. C. (2005). Stability and change in mothers' internal representations of their infants over time. *Attachment & Human Development, 7*(3), 253-268.

Thompson, R. A. (1994). Emotion regulation: A theme in search of definition. *Monographs of the Society for Research in Child Development, 59*(2/3), 25-52.

Tjaden, P., & Thoennes, N. (2000). *Full report of the prevalence, incidence, and consequences of violence against women. Findings from the National Violence Against Women Survey*: U.S. Department of Justice, National Institute of Justice. NCJ 183781.

Tolman, R. M. (1989). The development of a measure of psychological maltreatment of women by their male partners. *Violence and Victims, 4,* 159-177.

Tolman, R. M. (1995). *The validation of the Psychological Maltreatment of Women Inventory*. Paper presented at the 4th International Family Violence Conference, Durham, NH.

Tomlinson, D., von Baeyer, C. L., Stinson, J. N., & Sung, L. (2010). A systematic review of faces scales for the self-report of pain intensity in children. *Pediatrics, 126*(5), E1168-E1198.

Tyrka, A. R., Kelly, M. M., Graberm J. A., DeRose, L., Lee, J. K., Warren, M. P., & Brooks-Gunn, J. (2010). Behavioral adjustment in a community sample of boys: Links with basal and stress-induced salivary cortisol concentrations. *Psychoneuroendocrinology, 35*(8), 1167-1177.

Van Bakel, H. J. A., & Riksen-Walraven, J. M. (2008). Adrenocortical and behavioral attunement in parents with 1-year-old infants. *Developmental Psychobiology*, *50*(2), 196-201.

Van den Bergh, B. R. H., van Calster, B., Smits, T., van Huffel, S., & Lagae, L. (2008). Antenatal maternal anxiety is related to HPA-axis dysregulation and self-reported depressive symptoms in adolescence: A prospective study on the fetal origins of depressed mood. *Neuropsychopharmacology*, *33*(9), 2301-2301.

Van der Kolk, B. A. (2005). Developmental trauma disorder: Towards a rational diagnosis for children with complex trauma histories. *Psychiatric Annals, 35*, 401-408.

Van Hulle, C. A., Shirtcliff, E. A., Lemery-Chalfant, K., & Goldsmith, H. H. (2012). Genetic and environmental influences on individual differences in cortisol level and circadian rhythm in middle childhood. *Hormones and Behavior, 62*(1), 36-42.

Veer, I. M., Oei, N. Y .L., Spinhoven, P., van Buchem, M. A., Elzinga, B. M., & Rombouts, S. A .R .B. (2012). Endogenous cortisol is associated with functional connectivity between the amygdala and medial prefrontal cortex. *Psychoneuroendocrinology*, *37*(7), 1039-1047.

Vermetten, E. & Bremner, J. D. (2002). Circuits and systems in stress. II. Applications to neurobiology and treatment in posttraumatic stress disorder. *Depression and Anxiety, 16*(1), 14-38.

Villarruel, A. M., & Denyes, M. J. (1991). Pain assessment in children: Theoretical and empirical validity. Advances in Nursing Science, 14, 32-41.

Vitaro, F., Gagnon, C., & Tremblay, R. E. (1991). Teachers' and mothers' assessment of children's behaviors from kindergarten to grade two: Stability and change within and across informants. *Journal of Psychopathology and Behavioral Assessment,13*, 325–334.

von Eye, A. (2002). *Configural frequency analysis: Methods, models, and applications*. Mahwah, NJ: Erlbaum.

von Eye, A., & Bergman, L. R. (2003). Research strategies in developmental psychopathology: Dimensional identity and the person-oriented approach. *Development and Psychopathology, Special Issue: Conceptual, methodological, and statistical issues in developmental psychopathology: A special issue in honor of Paul E. Meehl, 15*(3), 553-580.

Waltermaurer, E. M, Ortega, C. A., & McNutt, L. A. (2003). Issues in estimating the prevalence of intimate partner violence: Assessing the impact of abuse status on participation bias. *Journal of Interpersonal Violence, 18*(9), 959-974.

Ware, H. S., Jouriles, E. N., Spiller, L. C., McDonald, R., Swank, P. R., & Norwood, W. D. (2001). Conduct problems among children at battered women's shelters: Prevalence and stability of maternal reports. *Journal of Family Violence, 16*(3), 291-307.

Weinstock, M. (2001). Alterations induced by gestational stress in brain morphology and behavior of the offspring. *Progress in Neuorbiology, 65*(5), 427-451.

Welberg, L. A. M., & Seckl, J.R. (2001). Prenatal stress, glucocorticoids and the programming of the brain. Journal of Neuroendocrinology, 13(2), 113-138.

Weiss, S. J. (2007). Neurobiological alternations associated with traumatic stress. *Perspectives in Psychiatric Care, 43*(3), 114-122.

Whittaker, J. E., Jones Harden, B., See, H. M., Meisch, A. & Westbrook, T. (2011). Family risks and protective factors: Predicting Early Head Start toddlers' social-emotional functioning. *Early Childhood Research Quarterly, 26*(1), 74-86.

Whitaker, R. C., Orzol, S. M., & Kahn, R. S. (2006). Maternal mental health, substance use, and domestic violence in the year after delivery and subsequent behavior problems in children at age 3 years. *Archives of General Psychiatry, 63*(5), 551-560.

Wolfe, D. A., Crooks, C. V., Lee, V., McIntyre-Smith, A., & Jaffe, P. G. (2003). The effects of children's exposure to domestic violence: A meta-analysis and critique. *Clinical Child and Family Psychology Review, 6*(3), 171-187.

Wolfe, J., & Kimerling, R. (1997). Gender issues in the assessment of Posttraumatic Stress Disorder. In: Wilson J, Keane TM, editors. *Assessing psychological trauma and PTSD*. Guilford; New York: 1997. Pp. 192–238.

Wong, D., & Baker, C. (1988). Pain in children: Comparison of assessment scales. *Pediatric Nursing, 14*(1), 9-17.

Wong, D. L., Hockenberry-Eaton, M., Wilson, D., Winkelstein, M. L., & Schwartz, P. (2001). *Wong's essentials of pediatric nursing.* St. Louis: Mosby, Inc.

Yoshikawa, H., & Knitzer, J. (1997). Lessons from the field: Head Start mental health strategies to meet changing needs. New York: National Center for Children in Poverty and the American Orthopsychiatric Association.

Youngstrom, E., Loeber, R., & Stouthamer-Loeber, M. (2000). Patterns and correlates of agreement between parent, teacher, and male adolescent ratings of externalizing and internalizing problems. *Journal of Consulting and Clinical Psychology, 68*(6), 1038-1050.

Zerk, D. M., Mertin, P. G., & Proeve, M. (2009). Domestic violence and maternal reports of young children's functioning. *Journal of Family Violence, 24*(7), 423-432.