

Stress Physiology and Post-traumatic Stress Symptoms in Children Exposed
to Intimate Partner Violence

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Abstract

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Intimate partner violence (IPV) is a prevalent problem in families with many children exposed to the violence each year. IPV has been associated with negative outcomes in children including trauma symptoms. A potential avenue to explore to determine which IPV-exposed children are at greatest risk for trauma symptom development is physiological functioning both at baseline and in response to stress. Previous research has demonstrated that trauma exposed children may display atypical physiological functioning of the hypothalamic-pituitary-adrenal (HPA) axis and sympathetic nervous system (SNS). Additionally, atypical physiological functioning has also been associated with maladjustment in children. However, much of this work has focused on the independent effects of the HPA axis and SNS and results have been inconsistent with some finding increased activation, decreased activation or normative functioning associated with poor child outcomes. Bauer and colleagues (2002) argued that these

physiological systems need to be examined concurrently to understand which children are at highest risk for maladjustment. Additionally, they put forth two competing models, the interactive (asymmetrical activation is associated with increased risk) and additive (symmetrical functioning is associated with increased risk) models, to describe how the HPA axis and SNS may interact. The present study examined whether the inclusion of both systems best predicts trauma symptoms in IPV-exposed children over the individual effects of each system. Furthermore, it investigates which model, the additive or interactive, best describe the interaction of the two physiological systems.

Thirty-five mother-child dyads (children aged 6-12 years) with a history of IPV exposure were included in the study. Children completed a parent-child interaction and mental arithmetic task with saliva samples collected five times over the course of the session. Hierarchical linear regression analyses were conducted to assess whether the interaction between both baseline levels and reactivity levels of cortisol and alpha amylase best predicted child trauma symptoms. Significant interactions were plotted per Aiken and West (1991). Findings supported Bauer et al.'s (2002) suggestion of using a multi-system approach and there was support for both the additive and interactive models. Future directions for research are also discussed.

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Dedication

This is dedicated to all of my boys.

Drew and Jace, I hope that one day you will be proud of me!

To my husband, Dave, thanks for being my biggest cheerleader!

Chapter 1: Introduction

Intimate partner violence (IPV) is a reality for many American families. Physical injury to one or both parties is often only one of the negative consequences associated with IPV. Children may also fall victim to the deleterious effects of IPV. Researchers have attempted to capture the extent of how widespread childhood IPV exposure is. McDonald, Jouriles, Ramisetty-Mikler, Caetano, and Green (2006) suggested around 15.5 million children are exposed each year. Others have found that approximately 6.6% of children are exposed annually, with 17.9% exposed at some point during their lifetime (Hamby, Finkelhor, Turner, & Ormrod, 2011). Although some children appear to navigate this trauma relatively unscathed, others go on to develop serious behavioral and emotional problems including post-traumatic stress symptoms (PTSS; subsyndromal post-traumatic stress disorder) or post-traumatic stress disorder (PTSD; Graham-Bermann, DeVoe, Mattis, Lynch, & Thomas, 2006; Levendosky, Huth-Bocks, Semel, & Shapiro, 2002; Kilpatrick & Willimas, 1997).

Recently, researchers have begun to examine why some IPV-exposed children are more vulnerable to PTSS/PTSD than others (e.g., Graham-Bermann, et al., 2006). This is an important area of inquiry as over half of IPV-exposed children may develop PTSS/PTSD (e.g., Lehmann, 1997). PTSD is also associated with other negative outcomes in children including internalizing and externalizing behavior problems (Graham-Bermann & Levendosky, 1998; Carrion, Weems, Ray, & Reiss, 2002; Seedat, Kammerer, Lockhat, & Stein, 2000; Margolin & Vickerman, 2007; Mertin & Mohr, 2002; McCloskey & Walker, 2000; Graham-Bermann et al., 2006) and problems with academic performance (Thompson & Massat, 2005). Early identification of children who are at highest risk is critical, as it may lead to early intervention and prevention of PTSS/PTSD in this population.

One promising predictor of trauma symptoms in both children and adults is physiological functioning (e.g., Schnurr, Friedman, & Bernardy, 2002; Langeland & Olf, 2008). PTSD has been associated with altered physiological functioning including increased sympathetic activation and both elevated and blunted hypothalamic-pituitary-adrenal (HPA) axis functioning (e.g., Bryant, Salmon, Sinclair, & Davidson, 2007; Pervanidou, Kolaitis, Charitaki, Margeli, Ferentinos, Bakoula, et al., 2007; De Bellis, Baum, Birmaher, Keshavan, Eccard, Boring, et al., 1999). Thus far, research investigating the link between PTSD/PTSS and physiological functioning has focused on the independent predictive effects of either the sympathetic nervous system (SNS) or the HPA axis. However, these two systems are inter-dependent and activity in one system may influence the other (Bauer, Quas, & Boyce, 2002).

There is emerging evidence that assessing multiple systems, and importantly, the interaction between these systems (Bauer et al., 2002; Gordis, Granger, Susman, & Trickett, 2006; El-Sheikh, Erath, Buckhalt, Granger, & Mize, 2008), may increase our ability to predict which children will develop PTSS/PTSD. Bauer et al. (2002) posits two competing models for describing the interaction between the pathways: 1) an additive model in which symmetrical activation (e.g., high SNS and high HPA axis response) indicates increased risk for maladaptive outcomes and 2) the interactive model in which asymmetrical activation (e.g., high SNS and low HPA axis response) indicates increased risk for poor adjustment. In recent studies, child outcomes (e.g., behavior problems, cognitive ability) were best predicted by examining both alpha amylase, a surrogate index of SNS functioning, and cortisol, an index of HPA axis functioning versus examining either of these biomarkers independently (Gordis, et al., 2006; El-Sheikh, et al., 2008; Allwood, Handwerger, Kivlighan, Granger, & Stroud, 2011; Keller, El-Sheikh, Granger, & Buckhalt, 2012; de Vries-Bouw, Jansen, Vermeiren, & Doreleijers, 2012).

Additionally, studies have examined the concurrent effects other combinations of physiological systems such as the HPA axis with the parasympathetic nervous system using respiratory sinus arrhythmia (RSA), a marker of the PNS, as well as other measures of the SNS (e.g., skin conductivity and pre-ejection period) with both the PNS and HPA axis. These studies have also lent support to incorporating a multi-systems approach in understanding children's adjustment (e.g., El-Sheikh, Kouros, Erath, Cummings, Keller, Stanton et al., 2009; El-Sheikh, Arsiwalla, Hinnant, & Erath, 2011; Gordis, Feres, Olezeski, Rabkin, & Trickett, 2010; Quas, Yim, Rush, & Sumaroka, 2012). These findings support the notion that assessing multiple biobehavioral systems offers better prediction of adverse outcomes, which may assist with identifying children at increased risk and lead to more successful, earlier intervention. The current study seeks to address whether examining the dual activation and interaction of these stress response pathways will improve prediction of which children are at risk for PTSS/PTSD as well as understanding which physiological pattern (e.g., symmetrical versus asymmetrical functioning) predicts heightened risk for trauma symptoms in the context of IPV.

Post-traumatic Stress Disorder

Some children who are exposed to IPV go on to develop severe problems including PTSS or PTSD. Per the *Diagnostic and Statistical Manual 4th ed., TR (DSM-IV-TR; American Psychiatric Association, 2000)*, a diagnosis of PTSD is given when an individual meets 6 criteria. The first criteria, Criterion A, is exposure to a traumatic event that leads to or threatens death or harm to an individual or another person and results in feelings of helplessness, horror, or fear. Children may manifest these feelings behaviorally in the form of disorganized or agitated behavior.

The next set of criteria, Criterion B, Criterion C, and Criterion D, represent 3 clusters of symptoms. To meet diagnostic criteria for PTSD an individual must report 1 symptom from the Criterion B cluster, 3 symptoms from the Criterion C cluster, and 2 symptoms from the Criterion D cluster. Criterion B reflects symptoms of re-experiencing the traumatic event. These symptoms include intrusive recollections of the trauma, nightmares or night terrors relating to the trauma, flashbacks to the event, feelings of distress, and heightened physiological reactivity in response to traumatic cues. For children, signs of these symptoms may be observed in their play behavior including repetitive play and acting out of the trauma in their play. Additionally, children's trauma related nightmares or night terrors may shift to dreams filled with more generally frightening content.

Criterion C consists of avoidance and numbing symptoms including avoidance of thoughts, feelings, conversations, activities, places, people, and anything else that is associated with the traumatic event. Additionally, avoidance symptoms may manifest as forgetfulness of the event, lack of interest in formerly pleasurable activities, social disengagement, lack of affect, and feelings that the individual may not live to an old age. In children evidence of symptoms from this cluster may be observed via loss of interest in typically enjoyable activities, disengagement from others, lack of positive affect, and omen formation.

Criterion D includes symptoms of heightened arousal. These symptoms may appear as insomnia, moodiness, trouble with focusing attention, hypervigilance to the environment, and an exaggerated startle response. Children may exhibit this symptom cluster somatically with increased reporting of stomachaches and headaches.

Criterion E and Criterion F center on impaired functioning and the duration of the symptoms. Specifically, Criterion E states that in order for a diagnosis of PTSD, the

aforementioned symptoms must be present for greater than 1 month. Criterion F reflects that the symptoms are severe enough that they impede with the individual's functioning in day-to-day life. This may mean impaired functioning across a variety of domains including academically or socially.

Intimate partner violence and Child PTSS/PTSD

For children exposed to IPV, witnessing threat of physical harm and/or death toward a loved one is a traumatic experience which likely elicits feelings of horror and intense fear. Research investigating PTSS/PTSD in children exposed to IPV has found that anywhere from 3% up to more than half of exposed children will develop PTSD depending measures and criteria used (e.g., Levendosky et al., 2002; Lehmann, 1997). In terms of trauma symptom presentation across development in IPV-exposed youngsters, in general, research has found evidence for trauma symptomatology in infants including heightened arousal, numbing, and fearful or aggressive behaviors, preschool age and school aged children exposed to IPV tend to exhibit re-experiencing and hyperarousal symptoms the most prominently (Bogat, DeJonghe, Levendosky, Davidson, & von Eye, 2006; Levendosky et al., 2002; Graham-Bermann et al., 2006; Graham-Bermann & Levendosky, 1998; Modrowski, Miller, Howell, & Graham-Bermann, 2012). Finally, adolescent trauma symptoms are more uniformly distributed across the symptom clusters, which is in line with adult presentations of PTSD (e.g., Lehman, 1997; Mertin & Mohr, 2002).

The fact that IPV exposure can lead to the aforementioned presentation of symptoms is concerning because symptoms associated with PTSS/PTSD may substantially impact child development including aspects of cognitive, social-emotional, and academic functioning (e.g., Brown, 2005; Thompson & Massat, 2005). However, it is poorly understood why some children

go on to develop these deficits in functioning in response to the trauma whereas others appear to navigate the IPV-exposure without severe effects on their adjustment (Graham-Bermann et al., 2006).

Physiological predictors of PTSS/PTSD in children

To understand the disparity between those who display PTSS/PTSD and those who maintain an adequate level of functioning post IPV-exposure, researchers have identified risk factors for PTSS/PTSD including witnessing the violence, the amount of violence witnessed by the child, history of other traumatic experiences, maternal report of depression, and child's ethnic status (Kilpatrick & Williams, 1997; Graham-Bermann et al., 2006; Graham-Bermann, Castor, Miller, & Howell, 2012). However, in the IPV population, thus far, physiological indicators of risk for PTSS/PTSD have not been identified. Several studies, described below, have examined biomarkers of risk for PTSS/PTSD in children and adolescents who have experienced trauma, including both acute stress such as traumatic injury, as well as stressors of more of a chronic nature like maltreatment.

In studies with children who had experienced a traumatic injury, findings have supported acute physiological measures (i.e., measured at the hospital shortly after the trauma occurred) as predictors of PTSS and PTSD in children. In particular, research provides evidence that cortisol levels, the end-product of the HPA axis, may be predictive of the development of PTSS/PTSD up to 6 months following the traumatic event. Delahanty, Nugent, Christopher, and Walsh (2005) found that increased cortisol in a sample of 8-18 year old children and adolescents who had experienced a traumatic injury predicted their PTSS 6 weeks later. Likewise, Pervanidou et al. (2007a) also found that for children and adolescents who were involved in a motor vehicle accident, increased cortisol measured while at the hospital, predicted PTSD 6 months after the

accident. Finally, Ostrowski, Christopher, van Dulmen, and Delahanty (2007) found that in children and adolescents who experienced a traumatic injury, acute cortisol levels predicted PTSS in all children at 6 weeks and for boys specifically at 7 months.

Other studies, examining indices of SNS functioning, the other major component of the stress response, have also found that elevated SNS functioning measured in the hospital immediately post-stressor also predicted PTSS/PTSD. Kassam-Adams, Garcia-España, Fein, and Winston (2005) found that increased heart rate measured at the hospital predicted PTSS/PTSD 3 months after the traumatic event, in this case a motor vehicle accident, in children and adolescents aged 8-17 years. Bryant et al. (2007) and De Young, Kenardy, and Spence (2007) extended these findings and found that an elevated heart rate measured in the first 24 hours of the hospital visit predicted PTSS/PTSD six months post-trauma in children and adolescents. Likewise, Delahanty et al. (2005) found increased epinephrine (EPI), measured shortly after a motor vehicle accident, predicted trauma symptoms 6 weeks later in children and adolescents.

However, extending these findings to IPV exposed children may be problematic as to date much of the research examining biomarkers as indices of risk has focused on measuring a) acute physiological functioning (i.e., measured at the hospital shortly after the trauma occurred) and b) physiological responses to a traumatic injury of some type. However, IPV-exposure is a different type of stressor and obtaining acute measures of physiology is more challenging in this group compared to a sample comprised of children who have experienced injury accidents. It would be difficult if not impossible to obtain children's acute physiological responses to violence in the case of IPV exposure because researchers are unable to obtain saliva samples immediately post-IPV incident since they typically do not witness the IPV, nor are they present in the immediate aftermath. Additionally, IPV differs from injury-type accidents which are usually

one-time occurrences, or Type I stressors, whereas IPV could be categorized as a Type II stressor because children are often repeatedly and chronically exposed to violence (Terr, 1991; Perkins & Graham-Bermann, 2006).

Although both one-time stressors such as traumatic injury, and stressors that are more of a chronic nature like exposure to IPV (Perkins & Graham-Bermann, 2006), are traumatic experiences for children, these experiences may differ in terms of physiological outcomes for the child. Studies have shown that chronic stress may impact or alter an individual's physiological functioning at baseline or in response to an acute stress. Wolf, Nicholls, & Chen (2008) examined the impact of chronic stress on physiological functioning in healthy and asthmatic children and found that chronic stress was associated with flatter diurnal cortisol patterns in healthy children and in asthmatic children it was associated with lower alpha amylase levels during the day. In terms of response to acute stress, Marin, Martin, Blackwell, Stetler, and Miller (2007) found that women with high levels of chronic stress had elevated cortisol in response to an acute stressor, whereas women who experienced mild levels of chronic stress had lower cortisol in response to acute stress. Luecken, Hagan, Sandler, Tein, Ayers, and Wolchik (2010) examined cortisol reactivity in adolescents who had experienced parental loss as children and compared reactivity between those who had been involved in a parental loss preventive intervention and controls. They found that 6 years post-intervention, the control groups exhibited decreased cortisol reactivity to a stress task as compared to those who participated in the intervention and concluded that the intervention served as a protective mechanism against a suppressed HPA axis response to trauma. Additionally, Matthews, Gump, and Owens (2001) found that adults with high levels of chronic stress had lower blood pressure and catecholamine levels in response to an acute stressor as compared to their counterparts with lower levels of

chronic stress. This provides evidence that children may manifest different symptoms based on the type of stressor because the demands on the body or physiological systems are different for a one-time stress versus repeated stress. Since children's physiological "symptoms" may differ based on whether trauma occurs once or is chronic, taking these differences in stress experiences into account is important for improving our ability to identify which children are most at risk for developing PTSS in the case of a stressor such as IPV.

In addition to studies that have examined the relationship between altered physiology as a predictor of PTSS/D, other studies have focused on making the initial link between trauma and altered physiology; that is, how trauma may impact physiological functioning. These studies have examined physiology in response to either repeated or chronic trauma as well as one-time traumas and have yielded a variety of findings regarding the activation of the SNS and HPA axis in the aftermath of trauma exposure. In terms of HPA axis findings, much of the research to date has focused on the effect of maltreatment, typically a chronic trauma, on the developing HPA axis. Bugental, Martorell, and Barraza (2003) examined the effects of 'harsh' maternal parenting as measured by a composite score of abusive behaviors and nonabusive behaviors (i.e., spanking/slapping) on infants' baseline cortisol and cortisol reactivity. Their findings suggested that while harsh parenting did not significantly relate to basal cortisol levels, there was a relation between harsh parenting and cortisol reactivity with higher levels of harsh parenting predicting increased reactivity.

King, Mandansky, King, Fletcher, and Brewer (2001) investigated basal cortisol levels in 5-7 year-old girls who had recently experienced sexual abuse. Compared to controls, girls who had been sexually abused in the past 2 months exhibited lower basal morning cortisol levels. Interestingly, De Bellis and colleagues (De Bellis et al., 1999) had contrary findings when

examining prepubertal maltreated children with PTSD compared to children with overanxious disorder (OAD) and controls. They observed increased levels of urinary baseline cortisol in maltreated children with PTSD as compared to healthy controls. However, no differences were observed between the maltreated children and children with OAD. Additionally, they found that the intrusive and hyperarousal PTSD symptoms were positively correlated with basal cortisol.

Cicchetti and Rogosch (2001a, 2001b) conducted a pair of studies examining maltreatment status and HPA axis functioning. In the first study they examined how the combination of maltreatment status and behavior problems, specifically internalizing and externalizing problems, were related to basal cortisol levels and diurnal cortisol patterns in school-aged children. They found that maltreated children with internalizing symptomatology had higher morning baseline cortisol levels than non-maltreated children with internalizing behavior problems, maltreated children without internalizing problems, and healthy controls. In addition, maltreated children with internalizing problems also had higher evening baseline cortisol levels than non-maltreated children with internalizing problems.

In terms of maltreated children with externalizing problems, Cicchetti and Rogosch found differences moderated by gender. Maltreated girls with externalizing problems had lower morning basal cortisol than maltreated girls without externalizing problems. Additionally, they found that non-maltreated boys with externalizing problems had the lowest daily cortisol levels. Maltreated children with comorbid internalizing/externalizing problems had flatter diurnal cortisol patterns than in maltreated children who did not have comorbid internalizing/externalizing problems. Overall, their findings seem to suggest that maltreatment status in combination with behavior problems predicts specific patterns of HPA axis functioning. In particular, maltreatment status combined with internalizing seems to be predictive of elevated

morning basal cortisol levels, whereas maltreatment status plus externalizing problems seems to be predictive lower morning basal cortisol levels, but for girls only.

In their second study, Cicchetti and Rogosch again examined maltreatment status and HPA axis functioning, but in this study, they examined whether maltreatment subtype (i.e., sexually abused (SA), physically abused (PA), neglected (N), or emotionally abused (EA) or a combination of groups predicted patterns of cortisol functioning in school-age children. Interestingly, no differences in morning or evening cortisol were observed when comparing overall maltreatment status (any subtype or combination of subtypes of maltreatment) and controls; only when the maltreatment subtypes were taken into account did group differences in basal cortisol between maltreated children and controls emerge. Children who had experienced all types of abuse had the highest morning basal cortisol levels. The groups did not differ in terms of their evening baseline cortisol levels. Children who were PA were most likely to display an atypical diurnal cortisol pattern of low morning cortisol levels combined with elevated evening cortisol levels. Children who were in the N/EA group did not differ from the control children in basal cortisol.

HPA axis functioning in adults who were abused as children has also been investigated and provides evidence for altered functioning in response to trauma. Carpenter, Carvalho, Tyrka, Wier, Mello, Mello et al. (2007) examined baseline cortisol and baseline adrenocorticotrophic hormone (ACTH), another HPA axis marker, as well as cortisol reactivity and ACTH reactivity in adults who have experienced maltreatment as a child. While no differences were found between healthy controls and adults with maltreatment histories in baseline functioning, evidence was found for a blunting of cortisol and ACTH reactivity in response to a laboratory stressor in adults who had been maltreated as children.

Gunnar and colleagues have examined HPA axis functioning in children who have been institutionalized overseas. Concerning baseline levels of cortisol activity, Gunnar, Morison, Chisholm, and Schuder (2001) found that during middle childhood (6-12 years), children who had been institutionalized for 8 months or longer during their first 12 months of life exhibited increased cortisol levels as compared to children who were adopted at 4 months of age or sooner and controls. However, in a later study, Gunnar, Frenn, Wewerka, and Van Ryzin (2009) found differing outcomes when comparing 10 to 12 year olds who had either been adopted after they were 1 year old, adopted before they were 8 months old, and controls. This study examined cortisol reactivity and found that there were no differences between the controls and the late adoptees (i.e., adopted post 12 months of age), but that the early adoptees (i.e., adopted pre 8 months of age) showed evidence of decreased cortisol reactivity in response to a laboratory stressor. It should be noted that the authors argued that findings could be due to children of this age range not showing much cortisol reactivity to a stressor regardless of their background.

Finally, HPA axis functioning has also been examined in children exposed to IPV. Saltzman, Holden, and Holahan (2005) examined both basal cortisol and cortisol reactivity (in response to an interview about the IPV) in 5 – 13 year olds and found that IPV exposure was associated with elevated basal cortisol levels, but not with cortisol reactivity. Sturge-Apple, Davies, Cicchetti, and Manning (2012) examined two year olds exposed to IPV and found that children demonstrated a blunted cortisol response to a laboratory stressor. Likewise, Davies, Sturge-Apple, Cicchetti, Manning, and Zale (2009) studied basal cortisol levels in these two year olds and found heightened morning cortisol levels. The developmental trajectory of IPV-exposed infants HPA axis reactivity was assessed from approximately the age of 7 months, 15 months and 24 months in a study conducted by Hibel, Granger, Blair, Cox, and colleagues (2011). They

found that infants exposed to IPV exhibited increased cortisol reactivity over time (7 months to 24 months) as well as heightened cortisol reactivity levels at 24 months as compared to peers with no IPV exposure who conversely did not exhibit HPA axis reactivity.

Exposure to one-time traumas also affect HPA axis functioning in children even when measured some time from the trauma. Goenjian and colleagues examined the effects of the 1988 Armenian earthquake on physiology in adolescents 5 years or more post-trauma in two studies. In the first study, they investigated adolescents' basal cortisol levels five years post-quake (Goenjian, Yehuda, Pynoos, Steinberg, Tashjian, Yang et al., 1996). They compared basal cortisol levels between 2 groups: a severely traumatized group which lived in a town very close in proximity to the epicenter of the quake and a less traumatized group who lived in a town at a farther distance from the quake. Findings suggested that those living closest to the epicenter (i.e., more traumatized group) had lower morning baseline cortisol levels 5 years post-quake. In their second study, Goenjian and colleagues (Goenjian, Pynoos, Steinberg, Endres, Abraham, Geffner et al., 2003) examined cortisol and ACTH levels pre- and post-exercise in a sample of adolescents 6.5 years post-quake. They found that 6.5 years post-quake, adolescents living closer to the epicenter did not have lower basal cortisol levels pre-exercise, but they did exhibit lower basal ACTH levels.

In a study examining baseline cortisol levels in college students who had been in the path of a hurricane and experienced hurricane damage (high trauma) versus those who resided in a location that had less hurricane damage (low trauma), researchers found a relation between cortisol levels and level of hurricane exposure. Specifically, they found that those who were most heavily impacted by the hurricane (i.e., high trauma) had elevated basal cortisol levels as compared to those farther away (Rotton, Dubitsky, Milov, White, & Clark, 1997).

In a study comparing children who lost a parent in the 9/11 attacks versus those who did not, Pfeffer, Altemus, Heo and Jiang (2007) found that children who lost a parent had elevated basal morning cortisol levels post-9/11 and that these elevated morning levels were consistent for over 2 years although they did decline overall over time. They did not find any differences between the bereaved group and non-bereaved group in terms of basal evening cortisol.

Overall, findings are mixed regarding the relation between HPA axis functioning and trauma. While most studies examining the relationship between trauma exposure and HPA axis functioning seem to find elevated baseline levels of HPA axis activation (e.g., Saltzman et al., 2005; De Bellis et al., 1999; Cicchetti & Rogosch, 2001a; Cicchetti & Rogosch, 2001b; Rotton et al. 1997; Pfeffer et al., 2007; Gunnar et al., 2001; Davies et al., 2009) there are exceptions in which low baseline functioning has been observed (e.g., King et al. 2001, Cicchetti & Rogosch, 2001; Goenjian et al, 1996; Goenjian et al, 2003) and others studies where no relation has been found between trauma and HPA axis activity (e.g., Bugental et al., 2003). Furthermore, HPA axis reactivity has undergone limited research and to date the findings are divergent with two studies finding increased reactivity (Bugental et al., 2003; Hibel et al., 2011) and others finding decreased reactivity (Carpenter et al., 2007; Sturge-Apple et al., 2012; Gunnar et al., 2009) and still others where no differences in HPA axis reactivity was found (e.g., Saltzman et al, 2005; Goenjian et al., 2003).

In terms of SNS functioning, in general, findings are consistent across studies, particularly those which have examined chronic trauma, regarding how trauma and the SNS activation are related, with most studies observing SNS hyperarousal. De Bellis, Lefter, Trickett, and Putnam (1994) studied the effect of sexual abuse on baseline urinary catecholamine levels, in particular NE, EPI, and dopamine (DA) and their metabolites, in a small sample of 8-15 year-

old females. They found a trend for maltreated girls to have higher concentrations of catecholamines than healthy controls over a 24 hour period. Additionally, in another study by De Bellis and colleagues (De Bellis, Baum, et al., 1999), previously mentioned above where maltreated prepubertal children with PTSD were compared to OAD and healthy control children, researchers found that the maltreated children had the highest levels of NE and DA and had higher levels of EPI as compared to the OAD group. Likewise, Gunnar, et al. (2009) found elevated SNS activation in 10 to 12 year olds who had experienced institutionalization for their 1st year of life or greater as compared to controls. In terms of SNS functioning in the context of IPV, Saltzman et al. (2005) found that IPV-exposed children had higher baseline heart rates and higher levels of heart rate reactivity in response to an interview about the violence than controls. Conversely, one study, conducted by Davies and colleagues (2009) found that toddlers exposed to interparental aggression displayed lower baseline SNS functioning.

A different picture emerges for acute trauma, although few studies have examined this relation in children and adolescents. Referring again to the Goenjian et al. (1996) study in which adolescents physiology was assessed 5 years post-earthquake, findings indicated that SNS functioning did not appear to show any long-term effects from trauma exposure. 3-methoxy-4-hydroxyphenylglycol (MHPG), a NE metabolite, was examined in both the high trauma group (resided closer in proximity to the epicenter) and the low trauma group (farther distance from the epicenter) and no differences between groups were observed. Likewise in a study by Pervanidou et al., (2007), no differences were found in catecholamine levels in children immediately following a motor vehicle accident, though by 1 month increased NE was observed in traumatized youth. Jones-Alexander, Blanchard, and Hickling (2005) also found no difference in sympathetic activation as measured by heart rate, skin conductance levels, and blood pressure,

between 8-17 year-old children and adolescents who had experienced a motor vehicle accident (MVA) with PTSD, those who had experienced an MVA but no PTSD and controls in response to a stressor.

Limitations of Research on Trauma and Stress Reactivity

Taken together, these findings on trauma exposure and physiology suggest that trauma may impact physiological functioning in some children. However, how the trauma impacts their physiological functioning (i.e., hypo- or hyperactivation) has been relatively inconsistent across studies for both the HPA axis and the SNS. The divergent pattern of findings related to hypo- or hyperactivation of the stress response pathways are not well understood. Additionally, it is unclear why some studies have identified heightened sympathetic arousal as an indicator of risk for PTSS/PTSD whereas others have not (e.g., Pervanidou et al., 2007; Jones-Alexander et al., 2005) and also why various patterns of HPA axis functioning are observed in association with PTSS/PTSD.

Such divergent findings may reflect a failure to examine the interaction between these two interrelated systems. Research investigating biomarkers of PTSS/PTSD in traumatized children has focused on the independent predictive effects of either SNS or HPA axis activation. This may not provide optimal prediction of PTSS/PTSD as these two stress response pathways are interconnected and interdependent (e.g., Tsigos & Chrousos; 2002 Bauer et al., 2002). By examining the conjoint effects of both pathways together, prediction accuracy may be improved, leading to better early identification techniques and ultimately earlier interventions (Bauer et al., 2002). Recent studies have incorporated the integration of the stress response pathways in predicting child adjustment, and thus far results have been promising. When both pathways have been examined, the interaction between the stress response pathways as measured via cortisol

and alpha amylase have predicted child outcomes, over-and-above the independent effects of the individual pathways (e.g., Gordis et al., 2006; El-Sheikh et al., 2008; Berry et al., 2012). What has not been addressed in the IPV literature, nor in the PTSD/PTSS literature in general, is how the two stress response pathways may work in concert (i.e., symmetrical versus asymmetrical activity) to predict which children will develop PTSS/PTSD.

Furthermore, most research investigating biomarkers of PTSS/PTSD in children exposed to Type II stress has focused on basal levels of biomarkers (e.g., De Bellis et al. 1999). While understanding baseline functioning is valuable, it may also be beneficial to examine the pattern of children's biomarkers in response to a stressor. Examining stress reactivity may be advantageous because many psychopathological states reflect an inability to control arousal. Thus, understanding children's reactivity to a laboratory stressor may be informative of their overall ability to modulate arousal and thereby provide insight in terms of risk status (Bauer et al., 2002).

Stress Response

To address these limitations on research in trauma and stress physiology, it is important to understand the physiology of the stress response and to describe the coordination between the two pathways and how problems may arise when one or both pathways becomes altered due to trauma. When faced with a stressor, the body initiates the stress response in an effort to adapt to the physical or psychological demand; following the stressor, the body returns to homeostasis (Chrousos & Gold, 1992). Two main physiological systems are activated in response to stress: the locus ceruleus-norepinephrine/sympathetic nervous system (LC-NE/SNS) and the hypothalamic-pituitary-adrenal (HPA) axis.

LC-NE/SNS

The LC-NE/SNS is one of two subsystems of the autonomic nervous system (ANS). The SNS controls the body's involuntary activities such as respiration, heart rate, vascular functioning, and gastrointestinal functioning among other functions (Tsigos & Chrousos, 2002; Gunnar & Quevedo, 2007; Gunnar & Davis, 2003). At baseline functioning, the LC-NE/SNS, along with the PNS, work to maintain homeostasis by keeping body systems in balance (Guyton & Hall, 1995). However as the need arises (i.e., encounter a stressor), the baseline level of SNS activation allows the body to quickly adjust either with inhibition or activation of the system (Guyton & Hall). In times of stress, the LC/NE-SNS branch is the initial and more rapid responder. It is comprised of two components or branches, the central component, which includes the LC and NE secreting neurons, and the peripheral components, which includes the SNS and sympathetic adrenal medullary (SAM) system (Stratakis & Chrousos, 1995; Chrousos & Gold, 1992; Tsigos & Chrousos, 2002; Charmandari, Tsigos, & Chrousos, 2005).

The central component of the LC-NE/SNS system (i.e., LC-NE; the locus ceruleus along with other NE secreting neurons) is located in the brainstem, specifically within the medulla and pons (Guyton & Hall, 1995; Tsigos & Chrousos, 2002). When a stressor is perceived either physically or psychologically, the LC/NE is activated and releases NE into the brain resulting in increased alertness, hypervigilance, and arousal. These physiological and arousal changes serve to help the organism address the stressor and increase chances of survival (Chrousos & Gold, 1992; Tsigos & Chrousos, 2002; Gunnar & Quevedo, 2007).

Additionally, the peripheral components of the LC-NE/SNS stress pathway, the SNS and SAM system, are also activated in response to stress. Activation of the SNS results in the firing of sympathetic nerves which innervate various organs and tissues throughout the body. These nerves release NE which serves to stimulate the affected organ and tissues. Sympathetic nerves

also innervate and stimulate the adrenal medulla (part of the SAM system) which in turn produces and releases the catecholamines EPI and NE into the bloodstream (Stratakis & Chrousos, 1995; Sapolsky, 2002; Gunnar & Quevedo, 2007). EPI and NE travel through the bloodstream and along with the sympathetic nerves, stimulate organs and tissues (Aldwin, 1994; Gervitz, 2000; Gunnar & Quevedo, 2007).

The overall effect of these physiological responses is what is commonly referred to as the “fight-or-flight” response (Cannon, 1939). More specifically, sympathetic activation leads to an increase in heart rate, blood pressure, respiration, perspiration, and glucose as well as increased cognitive functioning including hypervigilance, heightened arousal and enhanced attentional functioning (Gunnar & Quevedo 2007; Aldwin, 1994; Gervitz, 2000). These changes allow increased blood supply (i.e., nutrients) to areas of high need for survival including the brain and striated muscles (Aldwin, 1994; Sapolsky, 2000; 2004) and energy is directed away from organs and tissues that are devoted to items of low priority during crisis including growth, digestion, reproduction and other vegetative processes (Aldwin, 1994). Finally, activation of the LC-NE/SNS also stimulates activation of the HPA axis (Charmandari et al., 2005).

Recently, alpha amylase, a digestive enzyme secreted by the saliva glands, has been used in research as a surrogate marker of SNS activity (e.g., Granger, Kivlighan, Blair, El-Sheikh, Mize, Lisonbee et al., 2006). Many studies have shown alpha amylase to be a viable indicator of SNS functioning as it correlates with other measures of SNS functioning as well as demonstrating evidence for increasing in response to stressful situations. While findings have been inconsistent in terms of which indices of SNS functioning that alpha amylase correlates with, Chatterton, Vogelsong, Lu, Ellman, and Hudgens (1996) conducted a study that compared salivary alpha amylase levels to those of serum NE and EPI in response to a rest period, exercise,

and a written examination. They found that salivary alpha amylase activity closely reflected that of NE and that the markers were positively correlated. However, in a review by Nater and Rohleder (2009), the association between NE and alpha amylase was called into question. They highlighted that not all studies have found correlations between NE and alpha amylase (e.g., Nater et al., 2006). Also, the use of alpha amylase as a marker of SNS activation has been criticized by Bosch and colleagues (2011). Among their contentions, they have argued that alpha amylase may not be indicative of SNS functioning partly because salivary glands that excrete alpha amylase are also under control by the PNS. Additionally, they contend that methodological issues may affect salivary alpha amylase levels including not controlling for salivary flow rate, the collection device (sponge versus passive drool) used, and collection practices that are not uniform all may impact alpha amylase output (Bosch et al., 2011). However, a recent study by Thoma, Kirschbaum, Wolf, and Rohleder (2012) provided evidence that alpha amylase is a viable SNS marker as alpha amylase reactivity predicted NE levels in a study with a larger and more diverse sample than previous work examining alpha amylase and SNS relations. Additionally, in terms of salivary flow rate being a confound for alpha amylase levels, Rohleder, Wolf, Maldonado, and Kirschbaum (2006) found that alpha amylase levels after exposure to a stressor (a public speaking and mental arithmetic task) were not associated with salivary flow rate. Thus, while there are limitations and additional research is necessary, there is evidence to support the incorporation of alpha amylase as an SNS marker.

Regarding the reactivity of alpha amylase in response to a stressor, findings have been fairly consistent with most studies finding increases in alpha amylase in response to a stressor. Chatterton, Vogelsong, Lu and Hudgens (1997) found support for alpha amylase reactivity to a stressor by comparing salivary alpha amylase in men who completed a skydiving exercise versus

controls. Specifically, the men who skydived displayed increased alpha amylase levels pre- and post-skydive as compared to controls. In addition to the work by Chatterton and colleagues, Nater and Rohleder (2009) examined numerous studies that included alpha amylase as a biomarker in stress-inducing experiments. They concluded that alpha amylase appeared to be sensitive to stressful situations and showed fairly consistent rises in response to stress across studies.

Alpha amylase has also been used in studies involving children and adolescents. For example, El-Sheikh and colleagues (2008) measured baseline skin conductance levels (SCL), a marker of SNS activation, along with baseline salivary alpha amylase in 8 and 9 year old children. They found evidence for a positive association between SCL (an SNS marker) and salivary alpha amylase ($r = .26, p < .05$). Gordis and colleagues (2006) tested salivary alpha amylase levels in 10 to 14 year olds in response to a stressor (i.e., public speaking task and mental arithmetic task). They found evidence for increased alpha amylase levels in response to the stressor. Likewise, Fortunato, Dribin, Granger, and Buss (2008) found increases in alpha amylase in the majority of 2 year olds who completed a series of challenging tasks.

Alpha amylase has also been shown to be associated with child adjustment. For example, Allwood, et al. (2011) found that increased baseline salivary alpha amylase was associated with heightened levels of anxiety in children and adolescents aged 7-16 years. Alpha amylase has also been linked to externalizing symptoms in elementary-aged children. Keller and El-Sheikh (2009) examined baseline salivary alpha amylase levels and externalizing symptoms in children when they were in the 3rd and 5th grade. They found that alpha amylase levels in 3rd grade predicted externalizing symptoms in children when they reached 5th grade such that for children who exhibited either low or high alpha amylase levels had higher mother reported externalizing

symptoms (i.e., aggression, impulsivity, noncompliance, and delinquency) whereas those who had moderate levels of alpha amylase in 3rd grade had decreased externalizing symptom levels in 5th grade as reported by their mothers. Additionally, reactivity levels of alpha amylase have also been associated with child outcomes. In a group of preschoolers, Spinrad, Eisenberg, Granger, Eggum, Sallquist, Haugen et al. (2009) studied relations between alpha amylase and mother report of effortful control, emotion, and impulsivity. Their findings suggest that for girls, higher alpha amylase reactivity levels predicted higher levels of effortful control as well as decreased levels of anger and impulsivity. Taken together, the research conducted using alpha amylase has offered support for its use as an SNS marker in terms of stress reactivity and in predicting child outcomes.

Hypothalamic-Pituitary-Adrenal Axis:

The HPA axis produces and releases a series of hormones with the primary hormones being corticotropin-releasing hormone (CRH), adrenocorticotrophic hormone (ACTH) and cortisol. Production and release of basal levels of hormones are controlled by the suprachiasmatic nucleus (SCN) of the hypothalamus and follow a circadian pattern, with higher levels of hormones in the morning followed by declining levels throughout the day (Stansbury & Gunnar, 1994; Charmandari et al., 2005). The SCN stimulates the production and release of CRH which in turn dictates the production and release pattern of ACTH and cortisol (Habib, Gold, & Chrousos, 2001; Charmandari et al., 2005). Specifically, CRH travels to the median eminence, a nucleus at the base of the hypothalamus, which in turn is connected to the hypothalamo-hypophysial portal system. This specialized blood system connects to the anterior pituitary and provides a transport pathway for CRH to reach the anterior pituitary gland where it stimulates the production and release of ACTH. When ACTH reaches the adrenal cortex via the bloodstream,

it stimulates the production and release of cortisol (Nicolson, 2007; Charmandari, et al., 2005). Basal levels of cortisol are responsible for many bodily processes. Cortisol drives metabolic processes including gluconeogenesis (i.e., release of glucose into bloodstream) and lipolysis (i.e., release of fats into bloodstream; Tsigos & Chrousos, 2002). Cortisol influences blood pressure levels via its ability to allow catecholamines to act on vascular muscle tissue (Gunnar & Quevedo, 2007; Charmandari et al., 2005). Along those lines, basal cortisol levels play a role in the stress response in that it assists with the fight-or-flight response by increasing tissue sensitivity to NE and EPI (Gunnar & Quevedo, 2007; Sapolsky, Romero, & Munck, 2000).

In response to stress, the HPA axis responds at slower pace. It begins with an increase in the production and release of CRH and arginine vasopressin (AVP) from the parvocellular neurons located in the paraventricular nucleus within the hypothalamus (Charmandari, et al., 2005; Stratakis & Chrousos, 1995). CRH and AVP work together by increasing the other's influence (Chrousos & Gold, 1992; Tsigos & Chrousos, 2002). As is the case during basal functioning, CRH and AVP travel to the median eminence and on to the hypothalamo-hypophysial portal system which connects to the anterior pituitary. CRH and AVP then stimulate the production and release of ACTH from the anterior pituitary. ACTH enters the bloodstream and targets the adrenal cortex resulting in the production and release of cortisol, a steroid hormone (Gunnar & Quevedo, 2007). Cortisol enters the bloodstream and is able to target cells throughout the body (de Kloet, 1991; Aldwin, 1994; Charmandari et al., 2005), with the brain being a major target of cortisol (Gunnar & Quevedo, 2007). Being the slower responder, cortisol levels tend to peak approximately 25 minutes post-stressor as compared to the near immediate response of the LC-NE/SNS and the 10 minute response of the SAM system (Gunnar & Quevedo, 2007).

Like the effects of the LC-NE/SNS and SAM system, the release of cortisol in response to stress initially promotes diversion of energy to areas of high priority by suppressing immunity and appetite, increasing metabolism, and increasing glucose levels in the bloodstream (Aldwin, 1994; Sapolsky, 2000). Additionally, cortisol increases arousal, attention, and focus (Stansbury & Gunnar, 1994). The purpose of these functions is to promote survival of the individual (e.g., Sapolsky, 2004).

Cortisol also functions as a means of terminating the stress response via negative feedback loops (Tsigos & Chrousos, 2002; Charmandari et al., 2005; Stratakis & Chrousos, 1995; Stansbury & Gunnar, 1994). In particular, cortisol acts at the level of the hypothalamus by decreasing the production and release of CRH. Cortisol also acts at the level of the pituitary by inhibiting the production and release of ACTH (Gunnar & Davis, 2003). CRH also self regulates as circulating CRH inhibits the production and release of additional CRH (Tsigos & Chrousos, 2002).

The Interaction Between the Two Pathways:

While the exact nature of the interrelation between the HPA axis and SNS is not entirely clear, physiologically, the HPA axis and LC-NE/SNS are linked at the level of the brain (Bauer et al., 2002; Tsigos & Chrousos, 2002; Charmandari et al., 2005). More specifically, the LC and hypothalamic nuclei where CRH is produced innervate and coactivate each other (Stratakis & Chrousos, 1995; Bauer, et al., 2002). CRH stimulates the firing of LC neurons which releases NE in the brain. In turn, the NE stimulates CRH production and release, thus resulting in the components activating the other (Bauer et al., 2002; Chrousos & Gold, 1992; Charmandari et al., 2005). Once stimulated, CRH production and release and NE release trigger the cascade of events that results in the increase of cortisol and stimulates the SNS into action.

Additionally, research suggests that cortisol and catecholamines, end products of the HPA axis and SNS, respectively, also influence one another in other ways. Sapolsky, Romero, and Munck (2000) examined how basal cortisol effects the functioning of catecholamines. In their review, they concluded that basal cortisol levels play a “permissive” role in terms of the stress response. As mentioned previously, basal cortisol enhances sensitivity of the tissues targeted by catecholamines which allows for catecholamines to fully stimulate the tissue (Gunnar & Quevedo, 2007; Sapolsky et al., 2000). Furthermore, stress-induced levels of cortisol function to terminate the stress response by inhibiting both CRH and ACTH production and release via negative feedback loops as mentioned previously, and also by inhibiting NE release in the brain (Charmandari, et al, 2005; Stratakis & Chrousos, 1995; Chrousos & Gold, 1992).

The discrepant timing of the two stress pathways and the way in which they interact has important functional implications. The stress response evolved to manage acute physical stress, and in the short term, the stress response is an adaptive mechanism that allows for the body to gear up against a potential threat to one’s survival or well-being. The diversion of resources from vegetative functions is not problematic for the body if it occurs briefly and infrequently (Sapolsky, 2000; 2004; Charmandari et al., 2005). However, excessive exposure to catecholamines and cortisol has damaging effects on the body (e.g., Aldwin, 1994; Stratakis & Chrousos, 1995; Charmandari et al., 2005). Both have neurotoxic effects on the brain and as such may impede with brain development and/or alter brain structures (e.g., De Bellis, Keshavan, Clark, Casey, Giedd, Boring et al., 1999). Additionally, the effects of catecholamines and cortisol (e.g., increased heart rate, blood pressure) can be detrimental to the body when there is excessive secretion of both. For example, hypertension is helpful in the short-term in terms of getting nutrients to appropriate muscles quickly thus helping the organism to flee from the source

of stress, but if hypertension is continuous risk for cardiovascular disease may increase (Sapolsky, 2000). Furthermore, ongoing activation of the stress response does not allow for restorative functions like cell repair and detrimental outcomes including issues with growth, reproduction (a consequence of lack of ovulation and decreased testosterone), and decreased immunity (Sapolsky, 2000).

As a result of the interaction between the LC-NE/SNS and HPA axis, and the negative feedback loops present within the HPA axis, the amount of exposure to these neurotoxic compounds is limited in a typical situation. The body is still able to mobilize resources quickly thanks to the LC-NE/SNS and basal levels of cortisol, but after approximately 25 minutes, the HPA axis is able to down-regulate the stress response and bring about homeostasis (Charmandari, et al, 2005; Stratakis & Chrousos, 1995; Chrousos & Gold, 1992).

Since the two pathways influence one another, taking into account the activation of both pathways may provide the most accurate picture of physiological functioning and thereby lead to more accurate prediction of PTSS/PTSD. It follows, that dysregulation of one or both the pathways, could result in suboptimal functioning. For example, if the HPA axis response was insufficient (i.e., low levels of cortisol) SNS activation may be prolonged which is known to be associated with deleterious outcomes (Sapolsky, 2004).

A Multi-system Approach:

Bauer, Quas, and Boyce (2002) have posited that a multi-system approach, incorporating both HPA axis and SNS activation, may be the most optimal method of predicting children's risk status for psychopathology. They propose two models for understanding the interaction between the two pathways: the additive model and the interactive model. The additive model suggests that children who have coinciding underarousal or hyperarousal of the stress response systems

would be most at risk for developing problems. This model implies that the optimal level of functioning for children is when both stress response systems are activated moderately, or when one is highly activated while the other is low in activation. Conversely, the interactive model suggests that symmetrical functioning of the stress response systems is ideal. That is, children would be most at risk when their stress response systems are activated in an asymmetrical fashion. For example, if the HPA axis is highly activated while the SNS has less activation, then the model would predict increased risk for the child. Optimal functioning for the interactive model is defined as “balanced” response from both systems.

Limited research has been conducted comparing the competing models. There is some support for the interactive model being the better predictor of child outcomes. Allwood, Handwerger, Kivlighan, Granger, and Stroud (2011) found that asymmetrical activation of the HPA axis and SNS in children and adolescents predicted increased behavior problems and increased internalizing symptoms. As Bauer et al. (2002) suggest, it seems plausible to contend that the interactive model may be an appropriate model in terms of predicting outcomes from the activation of these two pathways because if the HPA axis is hypoactivated, the SNS response may be overly robust and conversely if the HPA axis is hyperactivated, the SNS response may be too brief, and either activation pattern may result in poor adaptation.

However, there is more empirical support for the additive model being the better predictor of child outcomes as evidenced in work done by Gordis et al. (2006), El-Sheikh et al. (2008), Keller, El-Sheikh, Granger, and Buckhalt (2012), and Berry, Blair, Willoughby, and Granger (2012). In these studies, researchers found that symmetrical activation, either represented by hypoarousal (low HPA axis and low SNS functioning) or hyperarousal (high HPA axis and high SNS functioning) predicted increased maladjustment. Gordis and colleagues

(2006) found that adolescents with low HPA axis and low SNS activation had higher levels of parent-reported aggressive behavior. Similarly, El-Sheikh et al. (2008) found that in 8-9 year old children, those with high baseline HPA axis activity and high baseline SNS activity had the most internalizing and externalizing behavior problems. Likewise, Keller et al. (2012) found that concurrent heightened activity of the SNS and HPA axis predicted lower intellectual ability in 8 and 9 year olds. Additionally, Berry et al. (2012) found that concurrent elevated baseline cortisol and alpha amylase measured in infancy and toddlerhood predicted decreased executive function and academic abilities once children were preschool age.

In terms of PTSS/PTSD, children with trauma symptoms may be those who have hypoarousal of *both* pathways or hyperarousal of *both* pathways. Thus, the additive model would predict that children with symmetrical stress responses, either hypo- or hyper-arousal, would be at increased risk for PTSD/PTSS. However, it is also possible, though there is less empirical support, that an asymmetrical pattern of physiological activity may predict increased trauma symptoms. That is children with high HPA axis activity coupled with low SNS activity or vice versa may have higher trauma symptoms suggesting the interactive model is the best fit for describing the physiological profiles of PTSD/PTSS. These two models have not yet been tested in predicting trauma symptoms in children in the context of IPV.

Because past work examining predictors of PTSS/PTSD has only examined the stress response pathways in isolation, it remains unknown whether the associations that were observed in the aforementioned studies between altered physiological functioning and trauma symptoms were the result of atypical physiology in the particular pathway being studied, or if it was the product of a physiological pattern that was not observed because the other pathway was not assessed. When only examining one of the stress response pathways, it is unclear whether the

other pathway is under- or over- activated. It may be that PTSD/PTSS symptoms are only present in children who show a specific physiological profile (e.g., asymmetrical or symmetrical activation; Yehuda, 2002). Accordingly, I tested whether any links between altered physiological functioning and PTSS/PTSD may be explained by the interaction of the stress response pathways.

Significance of the Present Study:

With the present study I seek to fill gaps in the literature regarding the prediction of PTSS/PTSD symptoms in children exposed to IPV. Research examining predictors of PTSS/PTSD in children has focused on the independent predictive effects of the SNS or the HPA axis. However, there is sufficient evidence to propose a multi-system approach for predicting PTSS/PTSD. Both SNS and HPA axis functioning have been implicated independently in predicting PTSS/PTSD, but this study adds to the literature by incorporating both stress response pathways and more importantly, examining the interaction between these two interrelated systems. Furthermore, the present study examined which physiological pattern, symmetrical activation or asymmetrical activation of the HPA axis and SNS predicts trauma symptoms in children in the context of IPV. Examination of both pathways may lead to more accurate predictions and early identification of which children are more vulnerable for developing PTSS/PTSD. In turn, better prediction and early identification may lead to earlier intervention and prevention of PTSS/PTSD in children exposed to IPV.

Examining PTSS/PTSD in a sample of children exposed to IPV will also help elucidate how children respond physiologically to chronic stress. Much of the work in predicting PTSD/PTSS has examined only acute physiology after a Type I stressor for predicting later PTSS/PTSD (e.g., Pervanidou et al., 2007). This can be problematic when studying physiological

predictors in an IPV context. First, the physiological response to a chronic, Type II stressor may differ from Type I physiological functioning because with a Type I stressor the body is intensely stressed at one point in time (e.g., McTeague & Lang, 2012; De Bellis, 2001). This differs from Type II stressors where physiological systems are repeatedly activated. This may mean that the physiological response in children exposed to Type II stressors such as IPV has a different profile than that seen in Type I traumas. Additionally, due to the nature of IPV (i.e., Type II stressor), it is nearly impossible to attain acute physiology in response to IPV-exposure because researchers tend not to be present when IPV occurs. Given this methodological restraint, physiological functioning must be measured in a different context. In addition to obtaining baseline levels of functioning which past studies have shown to be promising in predicting child outcomes (e.g., El-Sheikh et al., 2008), examining physiological functioning in response to a stressor may also be informative in terms of predicting PTSS/PTSD because many psychological disorders are associated with regulation deficits and understanding children's ability to regulate in response to a stressor may be indicative of their overall ability to modulate arousal (Bauer et al., 2002). This study adds to the literature by examining physiological profiles of children exposed to a Type II stressor and assessing child physiology both at baseline and in response to a stressor.

The current study used alpha amylase and cortisol as physiologic markers of SNS and HPA axis activity, respectively. Alpha amylase follows a diurnal pattern characterized by lowest levels immediately after awakening with increasing levels across the day (Wolf, Nicholls, & Chen, 2008). In terms of the developmental trajectory of alpha amylase, the trajectory appears to coincide with the nutritional changes infants navigate (Granger, Kivlighan, Blair, El-Sheikh, Mize, Lisonbee et al., 2006). As an enzyme, alpha amylase functions to breakdown

carbohydrates and starches, and as Granger et al. (2006) note, the need to breakdown these nutritional components does not usually occur until one reaches approximately 1 year old as an infant's diet is expanded. Thereafter, there is an increase until approximately 5 years at which point it plateaus (O'Donnell & Miller, 1980). In response to a stressor, alpha amylase peaks fairly quickly, as compared to cortisol, with studies showing peak levels of alpha amylase anywhere from 5 to 15 minutes after the start of a challenge (Nater, et al., 2006; Gordis et al., 2006).

Cortisol, used extensively in stress reactivity research with children, was used as a measure of HPA axis functioning (e.g., El-Sheikh, et al., 2008; Gordis, et al., 2006). Cortisol exhibits a different diurnal pattern from alpha amylase in that it is highest post-awakening (peaks approximately 30 minutes upon awakening), followed by a decline throughout the day (Shirtcliff, Allison, Armstrong, Slattery, Kalin, & Essex, 2011). Developmentally, an overall increase in cortisol levels is observed from childhood through the adolescence years (see Gunnar & Vazquez, 2006). Cortisol response time differs from that of alpha amylase in that it is a slower responder to challenge with peak levels achieved at around 25 minutes (Gunnar & Quevedo, 2007).

To assess how HPA axis and SNS functioning predict trauma symptoms in IPV-exposed children, the following aims and hypotheses were examined in the current study (See Figure 1):

Aim 1: Examine the relation between sympathetic nervous system (SNS) functioning and PTSS/PTSD in children exposed to IPV. Predictions related to both hyper- and hypo-reactivity of baseline and reactivity levels were explored in accordance with past research which has shown both heightened and attenuated activation at baseline and heightened levels in response to a stressor (e.g., Allwood et al., 2011; Keller & El-Sheikh, 2009; Spinrad et al.,

2009; See Figure 1a).

H1: Heightened SNS baseline levels and reactivity levels measured via alpha amylase, will predict PTSS/PTSD in IPV-exposed children.

Aim 2: Examine the relation between HPA axis functioning and PTSS/PTSD in children exposed to IPV. Again, predictions related to both hyper- and hypo-reactivity of baseline and reactivity levels were explored in accordance with past research which has shown both heightened and attenuated activation at baseline and in response to a stressor (e.g., Saltzman et al., 2005; King et al., 2001; Bugental et al., 2003; Carpenter et al., 2007; See Figure 1b).

H2: Heightened HPA axis baseline and reactivity levels, measured via cortisol, will predict PTSS/PTSD in IPV-exposed children.

Aim 3: Examine the relations between both stress response pathways and PTSS/PTSD in children exposed to IPV. Interactions between HPA axis and SNS baseline levels and HPA axis and SNS reactivity levels were explored (See Figures 1c and 1d).

H3: The interaction of both stress response pathways will provide better prediction of PTSS/PTSD in children exposed to IPV than the independent effects of each system.

Physiological symmetry versus asymmetry as put forth by the additive and interactive models by Bauer et al. (2002) was tested. Based on the majority of findings (Gordis et al., 2006; El-Sheikh et al., 2008; Keller et al., 2012; and Berry et al., 2012), the following hypothesis is given:

H4: Symmetrical activation of the stress response systems (e.g., high HPA and SNS activity or low HPA and SNS activity,) rather than asymmetrical activation (e.g., high HPA and low SNS activity), will be positively related to PTSS/PTSD in children exposed to IPV.

Chapter II: Methods

The present study was part of a larger NIH-funded intervention study, which examined the efficacy of an emotion-coaching parenting intervention on child outcomes for families exposed to IPV (1-R34-MH076935-01A1). The study was carried out at Seattle-area domestic violence agencies who agreed to partner with the research project as well as at a psychology laboratory affiliated with the University of Washington.

The larger study recruited participants who had a history of IPV but had been out of the violent relationship for at least 6 months and who had children aged 6 to 12 years. Previous studies have shown that IPV can lead to maladjustment in this age group (e.g., Kilpatrick & Williams, 1997; Graham-Bermann & Levendosky, 1998) and in terms of the larger intervention study, Katz and colleagues have seen promise with the protective effects of emotion-coaching in middle childhood (e.g., Katz, Hunter, and Klowden, 2008). Mothers were screened into the study based on their responses to a shortened version of the Conflict Tactics Scales (CTS; Straus, 1979) violence subscale. The violence subscale contains 10 items, with each item asked in regards to the mother's own behavior as well as her partner's behavior. Mothers were asked to report on violent behaviors which occurred during the last year before the violent relationship ended. For the screening, given that it occurred over the phone and that the CTS was administered in addition to other questions, a shortened version was used to expedite the screening process. To determine the presence of past IPV, an affirmative response, to any of the 6 items from the shortened CTS's violence subscale was used. These items included: blocked from leaving the room by the other, pushed/grabbed/shoved by the other, kicked/bit/hit by the other, beat up by the other, threatened with a knife or gun by the other, and use of knife or gun by the other.

Other inclusion criteria included the mother must have a child aged 6 to 12 years, with no developmental delay, and the mother must be the child's biological parent. The requirement for the biological relationship between mother-child was to limit potential confounds that may be associated with including children who were adopted, for example. Likewise, children with developmental delays were excluded due to potential confounds with the disorder and because children needed to be able to communicate with the mother and researchers in order to complete the study. In addition, both mother and child needed to have a command of the English language as all tasks and questionnaires were presented in English. Finally, because the larger study was an intervention study to empirically test the effectiveness of emotion-coaching, a parenting technique, parents were not allowed to be involved in any other parenting programs to verify that any potential changes in child outcomes were due to emotion-coaching not another parenting skill.

In sum, inclusion criteria were: 1) mothers had a history of IPV, 2) mothers have a child aged 6-12 years, 3) mothers must be the biological parent of the child, 4) comfortable use of the English language for both mother and child, 5) the child must not have a developmental delay or a diagnosis of pervasive developmental disorder, and 6) the mother is not enrolled in a parenting program currently. As the purpose of this study was to examine biological predictors of risk for trauma symptoms in all IPV exposed children, the data included in the analyses reflected baseline or pre-intervention measures only; that is, information obtained prior to assignment into either intervention or control group.

Participants

Seventy five mother-child dyads were recruited for the intervention study. Of those, nine dyads dropped out prior to the laboratory session leaving 66. Of the 66 remaining, two

participants refused to provide saliva samples. An additional 11 participants provided either insufficient saliva volume to carry out an assay ($n = 4$) or did not provide saliva at every saliva collection point ($n = 7$), leaving 53 participants. Furthermore, 5 of the remaining participants had saliva samples that fell below the cortisol assay's lower limit of detection. Of the 48 remaining participants, 10 samples were discarded because of insufficient saliva for assay due to quality control measures (i.e., samples needed to be rerun due to poor assay performance) leaving 38. Finally, 3 participants had hormone and/or enzyme levels that were extreme outliers (> 3 standard deviations) and thus were excluded from analyses leaving a final of 35 child participants with usable saliva samples.

Eighteen girls and 17 boys were included in the study. The children's mean age was 9.5 years. The majority of children were white or Caucasian (54.5%), followed by 27.27% who were bi- or multiracial, 12.12% were African American, and 3.03% were American Indian. The mother's mean age was 38.96 years with a range of 29 years to 52 years. The mean education level for the mothers was 1-3 years of college and their average income was in the \$20,000-29,000 range. The average length of time that the mothers experienced abuse was just over 5 years (range 1-156 months) with the average length of time since the abuse ended being just over 2 years (range 4-144 months)¹.

Procedures

All procedures took place at either a domestic violence agency ($n = 27$) or at a psychology laboratory ($n = 8$), affiliated with the University of Washington. Families were compensated \$75 for their participation. Upon arrival to the domestic violence agency or laboratory, families were greeted and introductions to staff were made. Consent and assent were

¹ At the time of the phone screen for inclusion in the study, one participant reported that it had been 4 months since the abuse had stopped. For this participant, assessments were delayed two months in order to meet the minimum 6 month timeframe for the end of the abuse to participate in the study and intervention.

obtained for mothers and children, respectively. Then, children were asked to rinse out their mouths after which the first saliva sample was obtained. Next, children were connected to electrodes to collect heart functioning data. Children then listened to a neutral story on headphones to allow for a second baseline measure of physiology. Following the neutral story, children engaged in a 10-minute discussion with their mother about a recent area of disagreement between them. Directly following the 10-minute discussion, the child completed a 5 minute mental arithmetic task after which three additional saliva samples were collected: one directly following the task, the next 10 minutes post-task, and finally 30 minutes post-task.

Parent-Child Interaction. To determine the topic for the 10-minute discussion, mothers and children completed the Issues Checklist (Robin & Foster, 1989), a brief questionnaire that identified areas of disagreement that had occurred in the past month. Topics included things such as keeping the child's room clean, completion of homework, and fighting with siblings. The research assistant selected two items from the questionnaire, one identified by the mother and one that the child had identified and requested that the participants discuss the topic(s) for 10 minutes. The inclusion of the parent-child interaction was related to the aims of the larger intervention study. The interaction was coded for various parenting behaviors to assess parent's emotion-coaching ability, a central component of the intervention work.

Mental Arithmetic Task. All children were asked to perform mental subtraction and report their answer out loud as quickly as possible to a panel of two research assistants who were seated directly across from them. This task was based off the mental arithmetic portion of the Trier Social Stress Test for Children which has been used in children aged 7 and older (mental arithmetic paradigms have been used in children as young as 5 years; e.g., Doherty-Sneddon, Phelps, & Clark, 2007) and mental arithmetic tasks have demonstrated increases in HPA axis

(i.e., cortisol) and SNS responses (e.g., alpha amylase and heart rate; TSST-C; Buske-Kirschbaum, Jobst, Wustmans, Kirschbaum, Rauh, & Hellhammer, 1997; Gordis, et al., 2006; see Granger, Kivlighan, Blair, El-Sheikh, Mize, & Lisonbee, 2006; Jones-Alexander, Blanchard, & Hickley, 2005). Pilot testing revealed that children responded physiologically (i.e., in terms of cortisol and alpha amylase) to the mental arithmetic task. Children aged 6-7 years were asked to subtract 3 repeatedly beginning with the number 197; children aged 8-10 years, were asked to subtract 7 repeatedly beginning with the number 758; children aged 11-12, were asked to subtract 13 repeatedly beginning with the number 1023. If a mistake was made, the child was asked to start again. This task took approximately 5 to 8 minutes to complete (see Buske-Kirschbaum, et al., 1997). Mental arithmetic tasks have been used in many studies as a stress paradigm (e.g., Jones-Alexander, et al., 2005; Stroud, Foster, Papandonatos, Handwerker, Granger, Kivlighan et al., 2009; Gordis et al., 2006).

Salivary Samples. To assess children's neuroendocrine functioning, saliva samples from the children were collected for measures of cortisol (HPA axis index) and alpha amylase (SNS index). To collect the specimens, two small "sorbettes" (small sponges affixed to a short shaft with an appearance similar to a cotton swab; Salimetrics, State College, PA) were placed under the child's tongue for 2 minutes. Prior to the first saliva collection, children were asked to rinse out their mouths. Approximately 400-600 μ L of saliva was collected via the two sorbettes at each measurement.

Saliva was sampled 5 times. The first saliva sample was collected shortly after the child had arrived at the domestic violence agency or laboratory and had completed consent/assent procedures (Baseline 1). The second sample was collected after the child listened to a neutral story on headphones for 2 minutes (Baseline 2). The third sample was collected immediately

following the mental arithmetic task (Alpha Amylase Reactivity). The fourth sample was collected ten minutes after the end of the mental arithmetic task (Cortisol Reactivity). The fifth sample was collected thirty minutes after the end of the mental arithmetic task (Cortisol and Alpha amylase Recovery). For the present study, only the first 4 saliva samples were used in analyses as the hypotheses centered on baseline levels and reactivity levels of functioning as opposed to recovery levels. All sessions occurred between 10am-6pm and the average time for the first saliva collection was 2:45pm.

Measures

Salivary Assays. Saliva samples were assayed for cortisol and alpha amylase levels at the University of Washington's Biological Anthropology and Biodemography Laboratory. When the procedures were carried out at a local domestic violence agency, samples were stored in a small cooler containing ice packs during the session. At the conclusion of the session, they were then transferred to a freezer in a University of Washington psychology laboratory and stored at approximately -20°C . When procedures occurred at the psychology laboratory, samples were put directly into the -20°C freezer upon collection. Samples were later transferred to the Biological Anthropology and Biodemography Laboratory, also located on the University of Washington campus, and stored at -80°C until time of assay. Prior to the assays, samples were thawed and centrifuged at 2400 rpm for 20 minutes. Given that the study was a multi-year study (5 years), saliva samples were assayed at two different time points, in 2010 and 2012.

Cortisol. Samples were assayed using a competitive enzyme immunoassay for cortisol. The cortisol assay uses a polyclonal rabbit-anti-cortisol-3-carboxymethyloxmine: bovine serum albumin antibody (Munro & Stabenfelt, 1985). Samples were run at a 1:2 dilution. If the sample was above the upper limit of detection, it was rerun at a lower dilution (e.g., neat). Each assay

plate ($N = 12$), contained 3 quality control specimens and were compared against historical in-house control data. All of the control data points fell within the 95% confidence interval range based on the historical assays.

For the first batch of saliva (i.e., 2010), the intra-assay coefficients of variation (CV) was calculated from a total of six plates for the high (1:3 dilution; 693.48 pg/mL), medium (1:8 dilution; 347.48 pg/mL), and low (1:16 dilution; 149.68 pg/mL) controls. The CV for the high controls was 9.07%, the CV for the medium controls was 10.20%, and the CV for the low controls was 10.52%. Regarding the inter-assay CVs, the high control was 7.14%, the medium control was 7.91% and the low control was at 13.24%.

For the second batch of saliva (i.e., 2012), the intra-assay CV was also calculated from a total of 6 plates for high (1:3 dilution; 690.99 pg/mL), medium (1:8 dilution; 259.94 pg/mL), and low (1:16 dilution; 139.38 pg/mL) controls. The CV for the high controls was 4.03%, and for the medium controls was 7.08%, and for the low controls was 17.74%. In terms of the inter-assay CV's for the second batch, the high control was 5.26%, the medium control was 3.68%, and the low control was 7.50%.

Alpha Amylase. Samples were assayed using a kinetic reaction assay kit available through Salimetrics, LLC (State College, PA). Samples were run at a 1:200 dilution unless they fell outside the limit of detection. If the sample was outside the limit of detection, it was rerun at a lower dilution (e.g., 1:50). To assess the validity of each plate ($N = 13$), intra-assay and inter-assay CVs were calculated for a high and low control, both provided by Salimetrics, per plate. For the first batch (i.e., 2010, $n = 8$), the intra-assay CV for the high controls was 3.60% and the inter-assay CV for the high controls was 6%. For the low controls, the intra-assay CV was 8.45% and the inter-assay CV was 11%. For the second batch (i.e., 2012, $n = 5$), the intra-assay

CV for the high controls was 3.94% and the inter-assay CV for the high controls was 10%. For the low controls, the intra-assay CV was 3.42% and the inter-assay CV was 7%.

Cortisol was used as an index of HPA axis functioning and alpha amylase was used as a measure of SNS functioning. Baseline (i.e., resting) and reactivity variables were created for cortisol and alpha amylase and used in the analyses. To address the study hypotheses, as mentioned previously, participant's saliva collected during the first 4 saliva collection time points was used. From the 4 sampling points, two baseline measures and 1 reactivity measure each for cortisol and alpha amylase were used to create the physiology variables.

Two baseline measures of cortisol and alpha amylase were obtained: Baseline 1 which was collected upon arrival to the IPV agency or laboratory (1st sample), and Baseline 2 which was collected directly following the neutral story (2nd sample). Both Cortisol Baseline 1 and Cortisol Baseline 2 were used individually in analyses as measures of HPA axis baseline or resting functioning. Likewise, both Alpha Amylase Baseline 1 and Alpha Amylase Baseline 2 were used as a measure of SNS baseline functioning. In keeping with past research in which salivary samples are collected, two baseline, or pre-stressor, samples were collected because the initial collection (Baseline 1) shortly after arrival to the session site may not reflect a true resting measure as the session is a novel experience for the children (e.g., Gordis et al., 2006). The reason the second baseline is collected following a neutral story on headphones is due to the fact that for the larger intervention study, other physiological measures (i.e., vagal tone) were being assessed in addition to the salivary markers and past research has successfully used listening to a neutral story in the collection of vagal tone (e.g., Gottman, Katz, & Hooven, 1997).

Alpha amylase reactivity variables were created using saliva collected immediately post-stressor (3rd sample). Cortisol reactivity variables were created using the saliva collected 10

minutes post-stressor (4th sample). Reactivity measures of alpha-amylase and cortisol differ in time due to discrepancies in expected response-to-stress times. To create stress reactivity scores for both cortisol and alpha-amylase, residualized change scores were computed by regressing the reactivity scores onto the baseline scores and reflect the difference between a participant's actual score and their predicted score (e.g., Burt & Obradovic, 2013). A residualized change score was computed independently for both baselines resulting in 2 residualized change scores for cortisol and alpha amylase each (i.e., residualized change score using baseline 1 as the independent variable and a residualized change score using baseline 2 as the independent variable).

Intimate Partner Violence. The Conflict Tactics Scales (CTS; Straus, 1979) was completed by the mothers and the violence subscale was used to measure mother report of IPV. The CTS is the most widely used index of IPV and demonstrates good internal consistency (.79-.88), and adequate concurrent and construct validity (Straus, 1990). Participants completed a shortened version of the CTS during the phone screening procedures for inclusion to the study as well as a full version of the CTS prior to the laboratory session. Due to missing data from the full version of the CTS (missing data for 12 of the final 35 participants, or 34.8% of the sample), for the present study, participants' responses to the shortened CTS' violence subscale used during screening procedures were used in analyses as data was available for all participants. The shortened screening version of the CTS contained 6 of the 10 violence subscale items. As mentioned previously, these items included: blocked from leaving the room by the other, pushed/grabbed/shoved by the other, kicked/bit/hit by the other, beat up by the other, threatened with a knife or gun by the other, and use of knife or gun by the other. Mothers reported on the frequency of their own and their partner's violent acts for the last year of the violent relationship. For analyses, a variable reflecting total intimate partner violence was computed which was a

composite score of the mother's own plus her partner's violence. The shortened version of the CTS used in the screening procedures was highly correlated with the full version of the CTS ($r = .67, p < .01$). It also demonstrated adequate internal consistency (.75).

Child Traumatic Stress Symptoms. To assess children's trauma symptoms, 4 questionnaires were used reflecting both child report and mother report.

(1) *Traumatic Stress Symptoms in Children* (TSSIC; Graham-Bermann & Levendosky, 1998) is a 17-item questionnaire that was completed by the mother and used to measure children's symptoms of post-traumatic stress in the past month. The TSSIC has good internal consistency (.80 - .82; Graham-Bermann & Levendosky, 1998; Graham-Bermann, et al., 2006). For analyses, four subscales were used: 1) total PTSD symptoms, 2) intrusive PTSD symptoms, 3) avoidance PTSD symptoms, and 4) arousal PTSD symptoms. Example items include: "repeated acting out or playing out the event(s)", "avoiding activities or play related to the violence or event(s)", and "startles or jumps easily since the violence or event(s)".

(2) *Child PTSD Symptom Scale* (CPSS; Foa, Johnson, Feeney, & Treadwell, 2001) was completed by the child and was used to measure children's symptoms of post-traumatic stress in the past month. Two subscales from the CPSS were used in analyses: 1) total symptom severity and 2) functional impairment. The total symptom severity scale has 17 items including "trying not to think about, talk about, or have feelings about the event", "having upsetting thoughts or images about the event that came into your head when you didn't want them to", and "feeling irritable or having fits of anger". It demonstrates adequate internal consistency (.70-.89), test-retest reliability ($r = .84$), and convergent validity ($r = .80$; Foa, et al., 2001). The functional impairment scale has 7 items tapping whether any of the total symptom severity scale items have impacted the child's daily functioning including activities such as "doing your chores",

relationships with your family”, and “fun and hobby activities”. Internal consistency varies (.35-.89) and test-retest reliability is $r = .70$ (Foa et al., 2001).

(3) *Child Dissociative Checklist* (CDC; Putnam, Helmers, & Trickett, 1993) is a 20 item checklist that was used to determine the child’s level of dissociative symptomatology in the past 12 months. This questionnaire was completed by the mother. It demonstrates high internal consistency (.95), good test-retest reliability ($\rho = .69$, 1 year interval), and good construct validity (Putnam, Helmers, & Trickett, 1993). Total scores for the measure were used in analyses. Example items include: “Child shows rapid regressions in age-level behavior”, “child does not remember or denies traumatic or painful experiences that are known to have occurred”, and “child goes into a daze or trance-like state at times or often appears ‘spaced-out’; teachers may report that he or she ‘daydreams’ frequently in school”.

(4) *Child Behavior Checklist* (CBCL; Achenbach & Edelbrock, 1983) PTSD symptom subscale (Wolfe, Gentile, & Wolfe, 1989) is a 20 item scale completed by the mother used to measure the child’s trauma symptoms in the past 6 months. This subscale demonstrates good internal consistency (.85-.89; Ruggiero & McLeer, 2000; Wolfe et al., 1989) and adequate concurrent validity (Ruggiero & McLeer, 2000). Mothers reported on items including whether the child: “argues a lot”, “can’t concentrate, can’t pay attention for long”, and is “too fearful or anxious”.

In analyses, child trauma symptoms were represented by both the mother’s report and the child’s report of trauma symptomatology resulting in 8 different outcome variables. Specifically, the outcome variables included: 1) the TSSIC total PTSD scale, 2) the TSSIC intrusive PTSD symptoms subscale, 3) the TSSIC avoidance PTSD symptoms subscale, 4) the TSSIC arousal PTSD symptoms subscale, 5) the CBCL PTSD symptom subscale, 6) the CDC total scale, 7) the

CPSS total symptom severity scale, and 8) the CPSS functional impairment scale. Again, the TSSIC, CBCL, and CDC were completed by the mothers and the CPSS scales were completed by the children.

Covariates. Several measures were included to control for potential covariates in analyses. Since child depression and PTSS/PTSD are highly comorbid, a measure of child depression was included (e.g., Ostrowski, et al., 2007). Additionally, given that maternal depression has been shown to affect child functioning, a measure of maternal depression was included (e.g., Hammen, Burge, Burney, & Adrian, 1990). Finally, because child maltreatment and IPV are highly correlated, a measure of maltreatment was also included in the study (e.g., Browne & Hamilton, 1999).

(1) *Child Depression Inventory* (CDI; Kovacs, 1992) is a 27 item questionnaire that was completed by the child and measured child depression symptoms in the past two weeks. It demonstrates high internal consistency (.80-.94) and adequate test-retest reliability (.38-.87; Saylor, Finch, Spirito, & Bennett, 1984). Children must choose which item from a choice of three best explains how they feel. Examples of items include: “I do most things OK; I do most things wrong; I do everything wrong” or “I like being with people; I do not like being with people many times; I do not want to be with people at all”.

(2) *Center for Epidemiologic Studies Depression Scale* (CES-D; Radloff, 1977) is a 20 item questionnaire that was completed by the mothers and measured maternal depressive symptoms. Mothers reported on how they felt for the past week. Items include: “I felt that I was just as good as other people”, “I felt that everything I did was an effort”, and “I had a crying spell”. This measure demonstrates high internal consistency (.85-.90) and an adequate test-retest reliability, ranging from .32-.67, along with good concurrent and construct validity (Radloff,

1977).

(3) *Child Abuse Potential Inventory* (CAP; Milner, 1986) is a 160 item form that was completed by the mothers and measured child abuse and neglect likelihood. The CAP has high internal consistency (.85-.96) and test-retest reliability (.75-.91) and demonstrates evidence for good construct validity as well (Milner, 1986). Examples of items include: “I am often easily upset”, “Children should stay clean”, and “my child has special problems”.

Due to missing data, both the CDI and CES-D were not included as covariates. Additionally, because of the diurnal pattern of cortisol and alpha amylase, an additional covariate was included in analyses, that is, the time of the first saliva sample collection. This was to control for potential effects in physiological measures due to time of day circumstances as opposed to individual differences in functioning.

Chapter III: Results

Prior to hypothesis testing, the data was screened to assess normality. In addition to plotting the data, descriptive statistics were used to determine whether the data had a normal distribution. The CTS scores (measure of IPV) were greatly skewed ($skew = 6.3$) and additionally, had outliers, so a log transformation was used which successfully addressed the issues of skew ($skew = -.2$) and extreme cases. In terms of the physiological variables, for both alpha amylase and cortisol, the Baseline 2 variables (measured after the neutral story) required a log transformation to address outliers. The remaining alpha amylase and cortisol variables (Baseline 1, and residualized change scores) were normally distributed. Of the outcome variables, the CBCL ($skew = 3.02$), TSSIC subscales: total PTSD symptoms ($skew = 2.63$), total intrusive PTSD symptoms ($skew = 4.02$), total avoidance PTSD symptoms ($skew = 2.93$), and total arousal symptoms ($skew = 3.10$), and the CDC ($skew = 4.17$) all had significant issues with skew and outliers. Log transformations were successfully used to address these issues: CBCL ($skew = .39$), total PTSD symptoms ($skew = .32$), total intrusive PTSD symptoms ($skew = 1.93$), total avoidance PTSD symptoms ($skew = 1.22$), total arousal PTSD symptoms ($skew = 1.51$), and CDC ($skew = -.17$). The covariates (child abuse score and time of day of saliva sampling) had normal distributions and thus did not require transformation.

Descriptive Statistics.

Descriptive statistics and correlations for the primary variables are reported in Tables 1 and 2, respectively. For ease of interpretation, means and standard deviations are given in raw form, that is, untransformed, and in the case of the change scores, simple difference scores are used in the table as opposed to the residualized change scores (i.e., Alpha Amylase Reactivity using Baseline 1 = Sample #3 – Sample #1; Alpha Amylase Reactivity using Baseline 2 =

Sample #3 – Sample #2; Cortisol Reactivity using Baseline 1 = Sample #4 – Sample #1; and Cortisol Reactivity using Baseline 2 = Sample #4 – Sample #2)..

IPV. Not surprisingly, given study criteria, all mothers reported some level of IPV. Overall IPV levels were moderately high, with scores on the truncated version of the CTS ranging from 2 – 153 out of a possible range of 0-300, with an average score of just under 30 ($M = 29.94$). When broken down by offender, the mother's responses suggested most of the violence was perpetrated by their partners ($M = 22.76$, range 2-100) as opposed to themselves ($M = 7.18$, range 0-53). Examining total violence (mother's report of her own violence plus her partner's violence), the most frequently reported violent act(s) was pushed/grabbed/shoved the other which was reported by 100% of the mothers. Next was blocking the other from leaving the room (82.4% reported), followed by hit/bit/kicked the other (64.7%). Just over half reported that they or their partner beat up the other (55.9%), with just under half reporting that they or their partner *threatened* with a knife or gun (44.1%) and finally the least reported violent act was the *use* of a knife or gun (8.8%). The mean number of months of abuse was 63.04, with a range of 1 month to 156 months (13 years). The average number of months since the abuse stopped was 27.84 with a range from 4 months to 144 months.

Child Maltreatment. Past research has found that IPV and child maltreatment tend to be correlated (e.g., Browne & Hamilton, 1999), therefore a measure of the likelihood of child maltreatment (CAPI) was included in study protocol. In the present study, IPV and child maltreatment potential indicated a trend toward a positive association ($r = .38$, $p < .10$), consistent with previous findings. Because of this trend between IPV and maltreatment potential, maltreatment status, measured as the raw score on the CAPI, was controlled for in analyses. Overall, the maltreatment potential scores had a mean of 233.35, with a range of 29-450. Slightly

more than half of respondents (52.2%) had scores greater than the 215 cut-off, thus indicating high likelihood toward abuse.

Physiology. Profiles of the children's average physiological response to the laboratory session are presented in Figure 2. Sixty-nine percent of the children had a 10% or greater increase in alpha amylase from collection 1 (initial sample collected upon arrival to the session) to collection 3 (sample collected immediately following the mental arithmetic task). Additionally, a repeated measures analysis of variance (ANOVA) revealed that there was a significant with-in subjects main effect suggesting that the alpha amylase levels differed significantly across time (Wilk's $\lambda = .67$, $F(3,32) = 5.19$, $p = .01$). Post hoc examination using Bonferroni adjusted alphas revealed significant differences between sample 1 (i.e., Baseline 1; taken upon arrival; $M = 29.03$, $SD = 20.96$) and sample 2 (i.e., Baseline 2; taken after listening to a neutral story on headphones; $M = 43.34$, $SD = 30.92$, $p = .01$) and between sample 1 and sample 3 (i.e., alpha amylase reactivity; taken immediately post mental arithmetic; $M = 43.28$, $SD = 32.41$, $p = .03$) with sample 1 having lower alpha amylase levels in both cases. All other post hoc pairwise comparisons were not significant.

In terms of cortisol, 29% of children had a 10% or greater increase from collection 1 (i.e., Baseline 1) to collection 4 (i.e., cortisol reactivity; sample collected 10 minutes post mental arithmetic task). A repeated measures ANOVA indicated a trend toward a with-in subjects main effect suggesting possible differences in cortisol levels across time Wilk's $\lambda = .81$, $F(3,32) = 2.45$, $p = .08$). Post hoc analyses using Bonferroni adjusted alphas showed a trend for differences between sample 1 ($M = 1262.98$, $SD = 804.26$) and sample 4 ($M = 881.20$, $SD = 549.28$, $p = .07$) with sample 1 having higher cortisol levels. All other pairwise comparisons were not significant.

Further comparison of alpha amylase and cortisol levels within each collection time

revealed that the two biomarkers were positively correlated only at collection #2 (post neutral story; $r = .40, p = .02$). Children's individual alpha amylase and cortisol patterns were quite variable as evidenced by large standard deviations for each of the four saliva collections (see Table 1 and Figures 3 and 4). There were no gender differences in cortisol at any collection time. There were gender differences between boys and girls in Baseline 2 alpha amylase ($t = -2.59, p = .01$), with girls having significantly higher levels ($M = 55.52$) than boys ($M = 30.43$). Girls also had significantly higher alpha amylase levels at collection time #3 (i.e., alpha amylase reactivity), immediately post-stressor ($M = 54.03$), than boys ($M = 31.90$), ($t = -2.12, p = .04$), however, there were no significant gender differences in residualized change scores.

The average time of the start of the session/first saliva collection was 2:45pm. There was a negative association between the time of the session and cortisol levels at each of the last two saliva collections. That is, there was a decrease in the level of cortisol for both saliva collections #3 and #4, as time increased (i.e., the later the session started). As previously mentioned, given cortisol's diurnal pattern, this was not surprising, and provided support for the inclusion of the start time of the session as a covariate in analyses.

Trauma Symptoms. Trauma symptoms were reported on by both the mothers and the children. There were no gender differences in reported trauma symptomatology for any of the scales used in analyses. When reporting on themselves via the CPSS, children reported a moderate level of trauma symptoms ($M = 13.12$, range 5-27) and functional impairment ($M = 2.47$, range 0-6). A cut-off score of 11 on the symptom severity scale is considered indicative of PTSD in children (Foa, et al., 2001). The majority of children (64.9%) scored 11 or higher on the scale. All of the children reported some trauma symptoms. In terms of functional impairment, 35.3% reported no functional impairment with the remaining reporting mild to moderately severe

levels of impairment.

Based upon mothers' reporting of trauma symptoms, using the TSSIC specifically, 12.1% (4) of the children would have a PTSD diagnosis (i.e., reported at least 1 intrusive symptom, 3 avoidance symptoms, and 2 arousal symptoms). In terms of total overall symptoms, the average score was 3.5 with a range of 0-14. Examination of the individual symptom clusters revealed that for the intrusive subscale, the average score was .97 (range 0-5). Just over 45% of the mother's reported that their child experienced at least 1 intrusive symptom, the DSM criteria for this cluster. In terms of the avoidance cluster, the average score was 1.3 (range 0-6) with 21.2% of children meeting the avoidance criteria (3 or more avoidance symptoms) and 48.5% of mother's reporting that their child experienced at least 1 avoidance symptom. Regarding arousal symptoms, the average score was 1.12 (range 0-5), with 27.3% of mother's reporting their child experienced 2 or more arousal symptoms, thus meeting DSM criteria for the arousal cluster, with over half of the mothers (51.5%) reporting their child experienced at least 1 arousal symptom.

The mothers also reported on their children's trauma symptoms using the PTSD scale from the CBCL. The average score for children was 7.61, close to the cut-off for PTSD diagnosis, which is a score of 8. Thirty-nine percent of the children had scores of 8 or greater per their mother's report of trauma symptoms. Finally, mother's reported on their children's dissociative symptoms using the CDC. The average score was 4.58 with scores ranging from 0 - 19 with a cut-off of 12 suggesting problematic levels of dissociative symptoms. Over ninety-three percent of children fell below this cut-off with only 2 exceeding it.

Bivariate correlations among the primary variables indicated relationships among many of the trauma symptom variables (i.e., CDC, CPSS, CBCL, and TSSIC; see Table 2). The CDC score was positively correlated with the child abuse score as measured by the CAPI ($r = .48, p =$

.02) suggesting increased maltreatment may indicate increased dissociative symptoms in children. Total violence (IPV) was correlated with both cortisol residualized change scores created using Baseline 1 ($r = .43, p = .01$) and Baseline 2 ($r = .52, p < .01$), but not with any alpha amylase levels. Finally, the alpha amylase residualized change score using Baseline 1 was correlated with the cortisol levels obtained during the 3rd sampling (immediately post-stressor) indicating that as the cortisol levels increased so did the alpha amylase reactivity levels ($r = .37, p = .03$).

Inferential Statistics

To test the four study hypotheses, hierarchical linear regression was used. Regression assumptions regarding multicollinearity, homoscedasticity, and linearity were checked. Not surprisingly, because interaction terms were included in the analyses, issues with multicollinearity were present. However, centering the variables (i.e., subtracting the mean) addressed this issue. Significant interaction terms were plotted using centered variables per Aiken and West (1991). Regression lines were plotted for high (+1 standard deviation above the mean) and low (- 1 standard deviation below the mean) levels of the moderators (i.e., alpha amylase and cortisol) and for the independent variable (i.e., IPV). The slopes of the moderator regression lines were examined for significance.

H1: Heightened SNS baseline levels and reactivity levels measured via alpha amylase, will predict PTSS/PTSD in IPV-exposed children.

To test the first hypothesis, that elevated alpha amylase activity is related to trauma symptoms in IPV-exposed children, hierarchical regressions were performed with the covariates, time of the session and child maltreatment, entered in the first step. IPV was included in Step 2 and the main effect of alpha amylase activity was entered in Step 3. The interaction between

alpha amylase activity and IPV was entered in Step 4 with child trauma symptoms as the outcome variable. First, a regression model was run examining baseline levels of alpha amylase using Baseline 1. None of the regression models run using alpha amylase Baseline 1 were significant for any of the outcome variables (i.e., TSSIC, CDC, CBCL-PTSD, and CPSS). Exploratory analyses were also conducted using the symptom clusters (i.e., intrusive, avoidance, and arousal) as measured by the TSSIC subscales as outcome variables. Again, there were no significant findings when using Baseline 1 levels in the models. However, when alpha amylase Baseline 2 was used in the model, there was a significant interaction effect between alpha amylase baseline 2 and IPV in predicting children's dissociative symptoms (as measured by the CDC), $\Delta R^2 = .14$, $p = .03$, unstandardized $b = 1.09$, $F = 6.04$, $p < .01$ (see Table 3). The interaction was plotted and slopes were tested (see Figure 5). For children with low Baseline 2 alpha amylase levels, as IPV increased, children's dissociative symptoms decreased ($t = -2.61$, $p = .02$). There was no such association between IPV and dissociative symptoms for children with low baseline alpha amylase levels. Exploratory analyses examining the symptom clusters measured by the TSSIC as outcomes were also not significant.

A similar regression was performed examining alpha amylase reactivity. In the first step the covariates, time of session and child maltreatment were entered. Step 2 included IPV, for Step 3 alpha amylase reactivity was entered, and Step 4 was the interaction between alpha amylase reactivity and IPV. The regression model was run with the alpha amylase residualized change score using Baseline 1 and again with the residualized change score using Baseline 2. Again, child trauma symptoms was the outcome variable. Neither of the models were significant for any of the outcome variables including exploratory analyses using the symptom clusters from the TSSIC as the outcome variables.

H2: Heightened HPA axis baseline and reactivity levels, measured via cortisol, will predict PTSS/PTSD in IPV-exposed children.

To test the second hypothesis, that elevated cortisol activity is related to trauma symptoms in IPV-exposed children, hierarchical regressions were performed first using baseline levels of cortisol and then with cortisol reactivity levels. First, examining baseline cortisol levels, for Step 1 the covariates, time of session and child maltreatment were entered. IPV was entered in Step 2. Step 3 included the main effect of baseline cortisol and Step 4 included the interaction between baseline cortisol activity and IPV, with child trauma symptoms as the outcome variable. There were no significant interactions between physiology (using cortisol Baseline 1 or cortisol Baseline 2) and IPV exposure in predicting children's trauma symptoms. Again exploratory analyses were conducted examining the individual symptom clusters as outcomes (via the TSSIC) and there were no significant findings.

Similarly, regression analyses were conducted using cortisol reactivity levels. The covariates, time of session and child maltreatment, were entered in Step 1. For Step 2 IPV was entered. Step 3 included the main effect of cortisol reactivity and Step 4 was the interaction between cortisol reactivity and IPV. Once again, child trauma symptoms were the outcome variable. None of the regression models were significant when using either the Baseline 1 or Baseline 2 residualized change score in predicting trauma symptoms in children exposed to IPV. Exploratory analyses that examined the TSSIC symptom clusters as outcome variables were also not significant.

To test the third and fourth hypotheses, that the interaction of the stress response systems will predict child trauma symptoms over and above the independent effects of the SNS and HPA axis (3rd hypothesis), and an examination of whether symmetrical activation of the two stress

pathways is a better predictor of child trauma symptoms in the context of IPV (4th hypothesis), hierarchical regressions were performed. In Step 1 the aforementioned covariates (child maltreatment and time of session) were entered. IPV was entered in Step 2 as well as the independent effects of cortisol activity and alpha amylase activity. Step 3 included all 2-way interactions (IPV x cortisol activity; IPV x alpha amylase activity; alpha amylase activity x cortisol activity). Finally, Step 4 included the 3-way interaction of IPV x cortisol activity x alpha amylase activity. The 5 measures of child trauma symptoms were used for the dependent variable (*Mother Report*: 1) the TSSIC total PTSD symptoms, 2) the CBCL PTSD subscale, 3) the CDC; *Child Report*: 4) CPSS total symptom severity, and 5) CPSS functional impairment scale). As with hypotheses 1 and 2, exploratory analyses were carried out to examine the symptom clusters as measured by the TSSIC (i.e., TSSIC intrusive PTSD symptoms, TSSIC avoidance PTSD symptoms, and TSSIC arousal PTSD symptoms) as outcome variables to determine if the 3-way interaction was significant and if specific trauma symptoms were associated with differing physiological patterns.

Hypothesis 3: The interaction of the two stress pathways will predict child trauma symptoms in IPV-exposed children over and above the independent effects of each system.

Analyses examining physiological activity were completed first with Baseline 1. Three of the regression models that were run using Baseline 1 alpha amylase and Baseline 1 cortisol had significant interactions. First, when examining total PTSD symptoms as the outcome variable as measured by the TSSIC, there was a significant interaction between physiology and trauma symptoms in children exposed to IPV ($\Delta R^2 = .15$, $p < .05$, unstandardized $b = -.0001$, $F = 2.99$, $p = .05$; See Table 4, Figure 6). Specifically, for children with high Baseline 1 cortisol and high Baseline 1 alpha amylase, as IPV increased, total PTSD symptoms decreased ($t = -3.55$, $p < .01$).

For children with high Baseline 1 cortisol and low Baseline 1 alpha amylase, as IPV increased, there was a trend for increasing PTSD symptoms ($t = 2.15, p = .06$). Additionally, for children with low Baseline 1 cortisol and low Baseline 1 alpha amylase, as IPV increased, total PTSD symptoms increased ($t = 1.51, p = .01$). There was no relationship between low Baseline 1 cortisol and high Baseline 1 alpha amylase and TSSIC total PTSD symptoms.

Next, a significant interaction was found when examining avoidance symptoms as measured by the TSSIC ($\Delta R^2 = .28, p < .01$, unstandardized $b = -.0001, F = 3.57, p = .03$; see Table 5, Figure 7). For children with high Baseline 1 cortisol and high Baseline 1 alpha amylase, as IPV increased, avoidance symptoms decreased ($t = -4.36, p < .01$). For children with high Baseline 1 cortisol and low Baseline 1 alpha amylase, as IPV increased, avoidance symptoms increased ($t = 3.08, p < .05$). Additionally, for children with low Baseline 1 cortisol and high Baseline 1 alpha amylase, as IPV increased, avoidance symptoms increased ($t = 2.48, p < .05$). And for children with low Baseline 1 cortisol and low Baseline 1 alpha amylase, as IPV increased, avoidance symptoms increased ($t = 2.81, p < .05$).

Finally, analyses that examined PTSD symptoms as measured by the CBCL were included as the dependent variable yielded a significant 3-way interaction ($\Delta R^2 = .36, p < .01$, unstandardized $b = -.0001, F = 5.45, p < .01$, see Table 6, Figure 8). For children with high Baseline 1 cortisol and high Baseline 1 alpha amylase, as IPV increased, PTSD symptoms decreased ($t = -3.61, p < .01$). For children with high Baseline 1 cortisol and low Baseline 1 alpha amylase, as IPV increased, PTSD symptoms increased ($t = 3.26, p = .01$). Lastly, for children with low Baseline 1 cortisol and high Baseline 1 alpha amylase, as IPV increased, PTSD symptoms increased ($t = 2.61, p = .03$). No relationship between low Baseline 1 cortisol and low Baseline 1 alpha amylase was observed. Additionally, there were no significant findings

when Baseline 2 levels of alpha amylase and cortisol were used in analyses.

There were no significant interactions between IPV, Baseline 1 cortisol, and Baseline 1 alpha amylase when predicting the TSSIC intrusive or arousal subscales, the CDC, or the CPSS total symptom severity score or functional impairment score. Additionally, when Baseline 2 levels of cortisol and alpha amylase were included in the regression model, none of the outcome variables were significant.

Next, regression analyses were conducted using cortisol and alpha amylase reactivity levels. There was one interaction effect that was a trend, specifically, the interaction between physiological reactivity using Baseline 1 and child dissociative symptoms in children exposed to IPV ($\Delta R^2 = .15$, $p < .05$, unstandardized $b = .71$, $F = 2.67$, $p = .07$, see Table 7, Figure 9). Further graphical probing of the interaction revealed that for children with high cortisol reactivity and low alpha amylase reactivity (when using Baseline 1 to compute the residualized change score) as IPV increased, total dissociative symptoms decreased ($t = -1.84$, $p < .10$). There was no such relationship between physiological reactivity and dissociative symptoms in IPV-exposed children who had high cortisol reactivity and high alpha amylase reactivity, or for those who had low cortisol reactivity paired with either high or low alpha amylase reactivity. All other regression models with physiological reactivity variables using Baseline 1 levels of cortisol and alpha amylase in the analyses were not significant for any other outcome variables. When using the residualized change score created using Baseline 2 for both physiology variables, no significant interactions were found.

H4: Symmetrical activation of the stress response systems (e.g., high HPA and SNS activity or low HPA and SNS activity,) versus asymmetrical activation (e.g., high HPA and low SNS activity), will be positively related to PTSS/PTSD in children exposed to IPV.

The final hypothesis, that symmetrical activation would predict PTSS/PTSD in IPV-exposed children, was partially supported. Examination of the significant findings from Hypothesis 3 showed that concurrent *low* baseline cortisol and alpha amylase predicted an increase in overall trauma symptoms and avoidance symptoms in IPV-exposed children (see Figures 6 and 7). Conversely, concurrent *high* baseline cortisol and alpha amylase levels predicted a decrease in trauma symptoms and avoidance symptoms (see Figures 6, 7, and 8). No findings related to symmetrical reactivity levels predicting trauma symptoms were observed.

Additionally, asymmetrical activation of the stress pathways also predicted trauma outcomes in IPV-exposed children. Asymmetrical baseline activation (i.e., high baseline cortisol and low baseline alpha amylase) predicted increased trauma symptoms and avoidance symptoms in IPV-exposed children (see Figures 6, 7, and 8). Examination of physiological *reactivity* measures suggest that as IPV increases, asymmetrical activation, (i.e., high cortisol reactivity and low alpha amylase reactivity), predicted decreased dissociative symptoms in IPV-exposed children (see Figure 9).

Post hoc analyses.

Post hoc analyses were conducted to see if any potential covariates, specifically, age, gender, or child depression would impact the previously reported significant findings. Due to the small sample size, the additional aforementioned covariates (i.e., age and child gender) were entered separately into the regression model used previously while missing data prevented the use of child depression in regression analyses. Instead, bivariate correlations were performed between child depression and trauma symptoms and child depression and physiological variables. Regarding child depression, correlations revealed that it was not associated any of the trauma outcomes or with any of the physiological variables.

Turning to child age, when examining the TSSIC Total PTSD scale as the outcome variable, Step 1 included age, time of session, and maltreatment status. Step 2 included IPV, Baseline 1 cortisol, and Baseline 1 alpha amylase and Step 3 included all 2-way interactions. Step 4 included the three-way interaction between IPV, Baseline 1 cortisol, and Baseline 1 alpha amylase. While not significant, results indicated a trend in the model ($\Delta R^2 = .15, p = .05$, unstandardized $b = -.0001, F = 2.45, p < .10$). Similarly, when child gender was substituted into the model instead of age, ($\Delta R^2 = .15, p < .05$, unstandardized $b = -.0001, F = 2.47, p < .10$) a trend was observed again.

Next, child age and gender were included in regression analyses predicting the TSSIC avoidance subscale. Again, the findings were similar to the significant findings previously reported. First, when including child age in the equation, Step 1 included child age, time of session, and maltreatment status. Step 2 included IPV, Baseline 1 cortisol, and Baseline 1 alpha amylase. The third step included the 2-way interaction terms and Step 4 was the three-way interaction between IPV, Baseline 1 cortisol, and Baseline 1 alpha amylase. Results again indicated a trend in the model ($\Delta R^2 = .28, p = .01$, unstandardized $b = -.0001, F = 2.90, p = .06$). When child gender was included there were similar findings indicating a trend as well ($\Delta R^2 = .25, p < .01$, unstandardized $b = -.0001, F = 2.94, p = .06$).

Finally, when including first child age and then child gender in regression analyses examining CBCL-PTSD as the outcome variable, findings again reflected the previous significant outcomes. Child age was included with the other covariates (i.e., time of session and maltreatment status) in Step 1. Step 2 included IPV, Baseline 1 cortisol, and Baseline 1 alpha amylase. Step 3 included all two-way interactions, and Step 4 included the three-way interaction between IPV, Baseline 1 cortisol, and Baseline 1 alpha amylase. Again, findings were consistent

with the significant findings previously reported ($\Delta R^2 = .34, p < .01$, unstandardized $b = -.0001$, $F = 6.51, p < .01$). When child gender was entered instead of child age, the findings were similar as well ($\Delta R^2 = .23, p < .01$, unstandardized $b = -.0001, F = 9.19, p < .01$).

Additionally, post hoc analyses, specifically bivariate correlations, were performed to determine if the number of months of IPV exposure and/or months since the IPV exposure discontinued, were correlated with trauma symptoms or physiology. No significant correlations were found between total months of abuse and trauma symptoms. There was a significant correlation between cortisol reactivity using Baseline 1 and total months of abuse, $r = .44, p = .03$. No significant findings between the number of months since the IPV stopped and trauma symptoms or the number of months since the IPV stopped and physiology were observed.

Chapter IV: Discussion

IPV exposure is a reality for millions of children each year (e.g., McDonald et al., 2006). For some, the consequences of this exposure may lead to adjustment difficulties including internalizing and externalizing problems (e.g., Jaffe, Wolfe, Wilson, & Zak, 1986), emotion awareness and regulation deficits (e.g., Katz, Hessler, & Annett, 2007), and difficulties with peers (e.g., McCloskey & Lichter, 2003). The trauma of IPV exposure may also lead to PTSS/PTSD in these children (e.g., Lehman, 1997). However, only a subset of those exposed to intimate partner violence will go on to display trauma symptomatology. The purpose of the present study was to examine how the two branches of the stress response system, the HPA axis and SNS, interact and which physiological patterns predict trauma symptoms in children exposed to a trauma such as IPV.

There was a high level of IPV reported by the mothers which was not unexpected as this was a requirement for eligibility to participate in the study. There was an average of just under 30 acts of total violence in the abusive relationships, and not surprisingly, mothers reported the majority of violence being perpetrated by their partner. While mother's reported approximately 7 incidences of violence perpetrated by themselves on average, their partners averaged around 22 incidences of violence over the last year of the relationship. All mothers experienced incidences of pushing, grabbing, or shoving and over half reported occurrences of hitting, kicking, or biting. A large proportion of the mothers reported the use of weapons in the abusive relationship with just under half indicating that they or their partner threatened the other with a knife or gun. The length of time in abusive relationships was quite variable with mothers indicating abusive relationships that lasted anywhere from 1 month to 13 years, with 5 years being the average. Likewise, there was quite a range in terms of the length of time since the mothers left the abusive

relationship with mothers reporting the abuse stopped anywhere from 4 months to 12 years with the average being just under 30 months.

In terms of children's trauma symptoms, moderate levels of symptoms were reported by the children and their mothers. When children reported on their own trauma symptoms (CPSS), they all indicated experiencing at least one trauma symptom in the past month. Based on their mother's report (TSSIC, CDC, CBCL), there were moderate levels of trauma symptoms for the IPV-exposed children. The children's trauma symptoms were fairly evenly dispersed across the three PTSD clusters of intrusive, avoidance, and arousal symptoms. Arousal symptoms were the most frequently endorsed with over half of mothers indicating that their child had at least one arousal symptom. Roughly half of the mothers endorsed at least one avoidance symptom experienced by their child (48.5%), while fewer mothers reported that their child experienced at least one intrusive symptom (45%). Depending on the measure, for mother report, 12.1 % (4) to 39% (12) of children met criteria for a PTSD diagnosis using the TSSIC and CBCL-PTSD scales, respectively and based on child report, 64.9% had scores above the cut-off for PTSD for the CPSS.

Children's physiological response over the course of the session was partially as expected. Specifically, it was expected that children's baseline cortisol and alpha amylase levels collected pre-stressor would be lower compared to levels post-stressor when cortisol and alpha amylase were expected to peak. However, only a quarter of children showed at least a 10% increase in cortisol from baseline to post-stressor with the highest cortisol levels observed at the first collection (i.e., Baseline 1). Conversely, alpha amylase performed as expected with almost two-thirds of children exhibiting at least a 10% increase in alpha amylase from baseline to post-stressor with the highest levels exhibited immediately post-stressor. While not entirely as

expected, other studies with younger children have found similar patterns of cortisol and alpha amylase response to challenge (Fortunato, et al, 2008).

This unbalanced physiological pattern, with cortisol levels decreasing and alpha amylase levels increasing over the course of the study, may be due to several factors. First, the stressor may not have been stressful enough to evoke a physiological response from the HPA axis. Typically, HPA axis activation is observed when a stressor seems unpredictable or uncontrollable, has an element of social judgment, or in response to negative emotions including fear and frustration, while the SNS is activated in response to physical exertion, a difficult or challenging event, however, as opposed to feeling overwhelmed, the individual feels they are able to cope with the challenge (e.g., Henry, 1992; Dickerson & Kemeny, 2004; see Lovallo & Thomas, 2000). Given that the two stress pathways have different triggers for activation it is possible that the stressor used in the session differentially activated the stress response systems in favor of the SNS. Participants were asked to perform mental arithmetic in front of an audience of two research assistants. Due to the element of public evaluation provided by the mental arithmetic task, it was expected that cortisol would be responsive to this stimuli. However, more children showed an increase in alpha amylase reactivity than cortisol reactivity indicating that perhaps the children felt that while the situation was stressful, it was within their capacity to cope with it. A mental arithmetic paradigm may not evoke feelings of loss of control and mental arithmetic is likely an activity that children may have engaged in at school thereby diminishing feelings of unpredictability, both items important stressor elements for stimulating HPA axis activation.

Secondly, the timing of saliva collection may have not captured the peak of cortisol. While alpha amylase tends to peak approximately 10 minutes after the initiation of the stressor,

cortisol has a slightly delayed response with peak levels being reached around 20-25 minutes post-stressor (Gunnar & Quevedo, 2007; Chrousos & Gold, 1992; Allwood et al., 2011). The total time of the stressor (i.e., mental arithmetic task) during the session was approximately 5-8 minutes. Alpha amylase was collected immediately following the mental arithmetic task and cortisol was collected 10 minutes later. It is possible that one or both of these durations were inaccurate in terms of capturing the peak levels of these biomarkers. Also, the parent-child interaction, the task directly preceding the mental arithmetic task, may have differentially stressed some of the children thereby influencing the stress reactivity timing. The parent-child interaction involved discussing an issue of conflict for 10 minutes. While the participants were afforded some privacy during the discussion (i.e., a room divider separated the participants from the research assistants), they were aware of and could see video cameras recording the interaction which may have been anxiety provoking and/or stress inducing. In their review, Gunnar et al. (2009), found that parent-child conflict discussions typically do not elicit HPA axis reactions in children except for a small subset of anxious children and those with an external locus of control. Therefore, the parent-child interaction may have sparked HPA axis activation prematurely in children with anxious tendencies thereby affecting the timing for collection of reactivity levels of cortisol and alpha amylase.

Finally, the children's divergent physiological profiles to stress may be a product of their trauma exposure. The focus of the present study was to identify physiological predictors of trauma symptomatology. Studies have provided evidence for a link between trauma history and altered physiological functioning (e.g., De Bellis et al., 1999) as well as a link between altered physiological functioning and child maladjustment (e.g., Gordis et al., 2006). A longitudinal study by Shenk, Noll, Putnam, and Trickett (2010) provided evidence for both components, from

trauma to altered physiology, and from altered physiology to adjustment. In the study by Shenk et al., girls with a history of sexual abuse were followed from their preteen years to their early twenties. The authors found that the experience of childhood sexual abuse was associated with altered stress physiology (i.e., asymmetrical vagal tone and cortisol) in late adolescence, and in turn, this altered physiology was associated with depressive symptomatology and antisocial behaviors in these females in their early twenties. Turning to the present study, the hypotheses center on the notion that trauma may cause physiological dysregulation of the HPA axis and SNS in some children which then may lead to heightened trauma symptoms. Therefore, one might not necessarily expect to see a uniform stress reactivity profile because that would suggest that children's stress physiology had not been altered due to trauma exposure (i.e., IPV). The same can be said regarding the activity of alpha amylase and cortisol individually over the course of the session. First, alpha amylase showed a significant increase from Baseline 1 to Baseline 2, but Baseline 2 was not significantly different from the collection #3 (Alpha Amylase Reactivity) levels (there was a significant difference between Baseline 1 and collection #3 also). Similarly, but with levels declining over the course of the session, cortisol only showed a trend for differences between Baseline 1 and collection #4 (Cortisol Reactivity). The presumption of the current study hypotheses was that stress physiology may be altered in children exposed to a trauma such as IPV. Taking into consideration the literature on children with trauma histories and the atypical physiological functioning (e.g., De Bellis, et al., 1999; Pervandiou, 2007; Shenk et al., 2010) this may explain the divergent patterns of cortisol and alpha amylase and their individual patterns of activity in response to the stressor in the IPV-exposed children in the present study, and suggests that their stress physiology functioning may be altered.

Consistent with past research (e.g., Chatterton et al, 1996; Nater, Marca, Florin, Moses,

Langhans, Koller et al., 2006), alpha amylase and cortisol appeared to be measuring two distinct components of the overall stress response in the present study. While the alpha amylase and cortisol samples collected at Baseline 2 (following a neutral story on headphones; pre-stressor) were correlated, the other three samples (Baseline 1, Alpha Amylase Reactivity—immediately following the mental arithmetic task, and Cortisol Reactivity—10 minutes after the conclusion of the mental arithmetic task) were not correlated with one another at their respective collection times. Therefore, this study provides some additional evidence for the inclusion of alpha amylase as a measure of a discrete stress physiology biomarker from HPA axis measures.

Independent effects of cortisol and alpha amylase on children’s trauma symptoms

The first and second hypotheses focused on the independent predictive effects of the SNS and HPA axis on IPV-exposed children’s trauma symptoms. Relations between SNS reactivity and trauma symptoms were examined and contrary to the hypothesis, no significant interactions were found between IPV exposure and alpha amylase reactivity using residualized change scores created with either Baseline 1 or Baseline 2, in predicting trauma symptoms in children. However, when baseline levels of alpha amylase were used in the regression model, a significant finding was revealed. For children with low Baseline 2 alpha amylase (measured after listening to a neutral story on headphones), as IPV increased, children’s dissociative symptoms as measured by the CDC, decreased. Interestingly, in this case, it appears that attenuated baseline SNS levels show more of a protective function as it was associated with less dissociation. This is contrary to Spinrad, Eisenberg, Granger, Eggum, Sallquist, Haugen et al. (2009), who found increased alpha amylase reactivity levels were associated with positive adjustment, specifically increased effortful control for both genders, and with less anger, impulsivity and externalizing

problems for girls. It is interesting that decreased SNS activation was associated with decreased dissociative symptoms specifically. Decreased SNS activation suggests lower levels of arousal and attention (e.g., Chrousos & Gold, 1992) and as such, lower dissociative symptoms seem counterintuitive. Additionally, previous research seems to suggest that lower sympathetic functioning is associated with higher levels of dissociation (e.g., Griffin, Resick, & Mechanic, 1997). Further research is necessary to verify this association.

Past research that has examined alpha amylase and child outcomes have had mixed findings with some studies finding increased alpha amylase levels associated with more trauma symptoms (Thoma, Joksimovic, Kirschbaum, Wolf, & Rohleder, 2012) or increased anxiety (Allwood, Handwerger, Kivlighan, Granger, and Stroud, 2011), some finding decreased alpha amylase reactivity associated with increased behavior problems (De Vries-Bouw, Jansen, Vermeiren, Doreleijers, van de Ven and Popma, 2012), and some finding no associations between alpha amylase levels and internalizing or externalizing symptoms (El-Sheikh et al., 2008; Gordis et al., 2006). Given the breadth of findings related to alpha amylase functioning and child outcomes, firm conclusions about the present study's findings cannot be drawn. There is minimal evidence for heightened SNS activation being associated with more optimal child functioning (e.g., Spinrad et al., 2009). And there is some evidence for a pattern of increased baseline alpha amylase being associated with increased maladjustment (i.e., Thoma et al., 2012; Allwood et al., 2011). The latter being in accordance with studies previously mentioned examining other indices of SNS functioning (e.g., plasma and urinary NE and EPI, and heart rate) and child outcomes which have also found increased sympathetic activation was associated with past trauma and trauma symptomatology (Kassam-Adams, et al., 2005; Bryant et al., 2007; De Young et al., 2007; Delahanty et al., 2005; Pervanidou et al., 2007; De Bellis et al., 1999).

However, similar to the present findings of no relation between alpha amylase reactivity and child trauma symptoms, many of the studies did not find a link between either alpha amylase reactivity and child adjustment (e.g., Gordis et al., 2006; Allwood et al., 2011) or baseline alpha amylase and child adjustment (e.g., De Vries-Bouw et al., 2012; El-Sheikh et al., 2008).

The second hypothesis centered on examining the role of cortisol in predicting trauma symptoms in IPV-exposed children. No significant relations were found between cortisol and trauma symptoms in children exposed to IPV. Past research, as reviewed earlier, has found a variety of outcomes in terms of cortisol reactivity and baseline cortisol and its relation to trauma exposure with trauma exposed youth showing increased, decreased, and no changes in baseline cortisol or cortisol reactivity as compared to control groups or less traumatized participants (e.g., Saltzman et al., 2005; King et al., 2001; Bugental et al., 2003; Goenjian et al., 2003; Carpenter et al., 2007). Studies focusing on physiology predicting trauma symptoms have consistently found that increased baseline cortisol collected in the immediate aftermath of a trauma have predicted later trauma symptoms (e.g., Delahanty et al., 2005; Pevanidou et al., 2007; Ostrowski et al., 2007). It should be cautioned that the current findings may be due to the stressor not eliciting a strong enough HPA axis response or it could be that in this sample there was no relationship between cortisol levels and trauma symptoms. Additionally, past studies that have found increased cortisol levels predicting trauma symptoms were examining Type I, or acute traumas, and due to the timing of the biomarker collection, in past studies the cortisol levels that were collected may have reflected stress-induced levels as they were collected at the hospital shortly after the traumas occurred. Overall, the present study's findings add to the uncertainty in the literature and provide further evidence for the need to incorporate both stress pathways when trying to understand how physiology is affected by trauma exposure and how physiology

predicts trauma symptoms. Inclusion of both pathways may provide clarification of these findings.

Interactions between cortisol and alpha amylase predicting trauma symptoms in IPV-exposed children.

The third hypothesis focused on whether including both stress response pathways, the HPA axis and the SNS, improved the ability to predict trauma symptoms in IPV-exposed children better than the independent predictive effects of each pathway alone. Per Bauer and colleagues (2002) suggestion, alpha amylase and cortisol and their interactions were examined concurrently in IPV exposed children to determine if the inclusion of both stress response pathways and their interactions would predict trauma symptoms in children over and above the independent predictive effects of either the HPA axis or SNS. Evidence was found to support the hypothesis that including both stress response pathways improved the ability to predict children's outcomes.

When incorporating the interaction of cortisol and alpha amylase in the regression analyses, four models were significant in predicting child trauma symptoms. Of the four significant models, 3 of them were when using Baseline 1 measures of cortisol and alpha amylase. That is, Baseline 1 cortisol and Baseline 1 alpha amylase interacted to predict trauma symptoms in IPV-exposed children over and above the independent pathways' effects.

Specifically, the three-way interaction between IPV, Baseline 1 cortisol, and Baseline 1 alpha amylase predicted trauma symptoms as measured by the CBCL PTSD subscale, as well as the TSSIC total PTSD symptoms and TSSIC avoidance symptoms scales. In every case, the three-way interaction predicted trauma symptoms over and above the individual effects of Baseline 1

cortisol and Baseline 1 alpha amylase.

Reactivity levels of cortisol and alpha amylase were not as strong of a predictor of child trauma symptoms in IPV-exposed children. Of the four significant interactions, only one of the interactions was when using reactivity measures of cortisol and alpha amylase, and it was when using the reactivity variable created using Baseline 1 in the residualized change score.

Specifically, cortisol and alpha amylase reactivity in IPV-exposed children predicted dissociative symptoms over and above the independent effects of either the HPA axis or SNS.

It is interesting to consider the lack of significant findings when examining cortisol independently, in particular, as predictor of trauma symptoms versus the significant findings that were revealed when both cortisol and alpha amylase and the interaction between these biomarkers were included in the regression model. Likewise, alpha amylase, when looked at in isolation as a predictor, also did not fare as well with only one significant finding, specifically, predicting dissociative symptoms in IPV-exposed children. However, when the two biomarkers were examined in tandem, there was improved ability for predicting child trauma outcomes. The incorporation of both the SNS and HPA axis resulted in four significant interaction terms as opposed to one (SNS) and none (HPA axis) when examining each physiological predictor alone. This highlights the significance of taking a multiple systems approach when trying to use physiology to understand child outcomes (Bauer et al., 2002).

These findings are in line with previous research which has shown that the interaction of both the HPA axis and SNS improved prediction of child outcomes. Studies by Gordis et al. (2006) and El-Sheikh et al. (2008), Allwood et al. (2011), and Berry, Blair, Willoughby, and Granger (2012) all have found that the incorporation of both the SNS and HPA axis have predicted various child outcomes (e.g., externalizing and academic ability) over and above the

effects of the systems alone. While not all studies have found that interactions explain more variance in outcomes versus the effects of each system alone (e.g., Spinrad et al., 2009; Lisonbee, Pendry, Mize, & Gwynn, 2010), the present study's findings in conjunction with these studies further support the suggestion by Bauer and colleagues (2002) to incorporate multiple systems in research examining physiological indices of risk for child maladjustment.

Additionally, the current study's findings are in line with past research examining other systems and their interactions with the HPA axis and the SNS (i.e., HPA axis x PNS and SNS x PNS) which have shown support for a multi-system approach. Studies have examined how the HPA axis and PNS might work in concert to predict child outcomes such as internalizing symptoms and antisocial behaviors and have demonstrated that the inclusion of multi-systems predicted child outcomes over and above the individual effects of the PNS and HPA axis alone (El-Sheikh, Arsiwalla, Hinnant, & Erath, 2011; Shenk, Noll, Putnam, & Trickett, 2010). Additionally, studies that have examined how the SNS and PNS may interact to predict child functioning have indicated that the multi-system approach is advantageous and predicts externalizing symptoms over and above the independent effects of the two systems (Gordis, Feres, Olezski, Rabkin, & Trickett, 2010; El-Sheikh, Kouros, Erath, Cummings, Keller, & Stanton et al., 2009). In sum, there is evidence for following the suggestion of Bauer and colleagues (2002) in assessing multiple physiological biomarkers of risk for child maladjustment across three different physiological systems. Studies focusing on the HPA axis and SNS, as conducted in the present study, offer support for the multi-system approach. The current study demonstrates how only observing physiology from one system (i.e., the HPA axis) may lead to a spurious conclusion (e.g., no association between the HPA axis functioning and child trauma symptoms) when in reality, a relation may exist, but it is in the context of SNS activity.

Additionally, research focusing on other physiological combinations, that is, on the HPA axis and PNS, and on the SNS and PNS, have provided support for the inclusion of multiple pathways in analyses. Taken together, these studies highlight how the pathways may work in concert to affect child functioning.

Symmetry versus asymmetry in predicting trauma symptoms

The final hypothesis examined whether symmetrical stress response activation (i.e., low cortisol and low alpha amylase activity or high cortisol and high alpha amylase activity) better predicted trauma symptoms in children in the context of IPV as compared to an asymmetrical pattern (e.g., low cortisol coupled with high alpha amylase and vice versa). Bauer et al. (2002) proposed the additive model and the interactive model when describing how the two stress response pathways might interact. The additive model centers on symmetrical activation leading to increased risk. Specifically, if a child exhibits concurrent underarousal of the two systems or concurrent hyperarousal of the two systems, they are hypothesized to have a heightened risk for poor adjustment. Ideal physiological functioning is that of an asymmetrical nature such that the net arousal is that of a medium level achieved through either mid-level arousal of both systems, or by heightened arousal of one system coupled with attenuated arousal of the other system. The interactive model, on the other hand, is based on the presumption that asymmetrical physiological functioning is least adaptive. In this scenario, the two systems are viewed as “complimentary” thus, if one system has increased arousal while the other has decreased arousal, the two systems may become uncoordinated. When the systems are uncoordinated or unbalanced, it may allow for one of the systems to go ‘unchecked’ by the other (e.g., if cortisol levels were low and alpha amylase levels were high, the HPA axis may be unable to down-

regulate the SNS response) and this may lead to higher risk of maladjustment. Optimal functioning occurs when both systems are coordinated and balanced (i.e., both have low arousal, medium arousal, or high arousal; Bauer et al., 2002).

Reactivity Levels and Child Trauma Symptoms: Symmetry versus Asymmetry

In terms of *reactivity* levels of SNS and HPA axis functioning, the hypothesis that symmetrical activation would predict trauma symptoms was not supported that is, symmetrical activation of cortisol and alpha amylase reactivity did not predict child trauma symptoms. Conversely, asymmetrical activation of the HPA axis and SNS reactivity did predict trauma symptoms in IPV-exposed children. A graph of the interaction revealed a seemingly protective effect of asymmetrical activity. Specifically, for children with heightened cortisol reactivity coupled with decreased alpha amylase reactivity, as IPV levels increased, there was a trend for dissociative symptoms to decrease. These findings are consistent with two studies, one examining reactivity levels and the other baseline levels of cortisol and alpha amylase in predicting externalizing symptoms (Gordis et al., 2006; El-Sheikh et al., 2008) Conversely, one study has found that physiological asymmetry predicted increased levels of maladjustment, specifically problem behaviors and anxiety and depression symptoms (Allwood et al., 2011). Thus, while findings are not wholly consistent, the current findings provide additional support for asymmetrical reactivity being associated with better child functioning.

Baseline levels and Child Trauma Symptoms: Symmetry versus Asymmetry

As noted previously, three significant interactions were observed when using baseline levels of cortisol and alpha amylase activation. The 3-way interaction between IPV, baseline cortisol, and baseline alpha amylase predicted trauma symptoms in children as measured by the CBCL PTSD scale, the TSSIC total PTSD scale, and the TSSIC avoidance subscale. Each of

these significant interactions were probed graphically and revealed varying outcomes for child adjustment depending on the nature of the physiological response pattern. First, a closer examination of the three-way interaction between IPV, baseline alpha amylase, and baseline cortisol and its relation to the CBCL-PTSD subscale, revealed that for children with heightened cortisol and heightened alpha amylase, as IPV increases, a decrease was noted in their trauma symptoms as measured by the CBCL-PTSD subscale. In other words, higher baseline levels of cortisol and alpha amylase seemed to be protective in the face of IPV in regards to overall trauma symptoms as evidenced by lower reported trauma symptoms. Additionally, graphical probing of the interaction predicting CBCL-PTSD indicated that for children with higher baseline cortisol coupled with lower levels of baseline alpha amylase, as IPV increases, CBCL trauma symptoms also increase. In this case, unbalanced stress pathways seem to point to increased maladjustment as measured by the CBCL-PTSD scale. Likewise, the inverse was also true, that is, for IPV-exposed children with lower levels of baseline cortisol with concurrent higher levels of baseline alpha amylase, the greater the amount of IPV exposure, the more reported trauma symptoms based on the CBCL trauma subscale. In sum, unbalanced responses from the HPA axis and SNS were indicative of increased trauma symptomatology while concurrent high activation of the stress pathways predicted decreased levels of CBCL PTSD symptoms.

Similar patterns were observed when examining baseline cortisol and baseline alpha amylase in the context of IPV predicting total PTSD symptoms as measured by the TSSIC. IPV-exposed children who demonstrated a balanced physiological response to the stressor, specifically, elevated baseline cortisol along with elevated baseline alpha amylase, as IPV increased, their mothers reported less TSSIC total PTSD symptoms. Again, concurrent high

basal HPA axis and SNS functioning, appeared to be a protective factor against PTSD symptoms in IPV-exposed children. For IPV-exposed children who displayed uncoordinated stress responses, that is, heightened baseline cortisol with lower baseline alpha amylase, there was a trend such that these children had mothers who reported increased total PTSD symptoms on the TSSIC. Finally, for IPV-exposed children who displayed coordinated responses characterized by lower baseline cortisol with lower baseline alpha amylase, their mothers also reported increased levels of PTSD symptoms in the context of IPV.

Once again, concurrent elevated baseline HPA axis and SNS activation seemed to provide a protective effect as it was related to decreased trauma symptoms as measured by the TSSIC total PTSD scale. Likewise, unbalanced stress physiology, specifically high baseline cortisol coupled with low baseline alpha amylase seemed to be indicative of a higher likelihood of poor outcomes in terms of increased trauma symptoms. Evidence was also found for poor adjustment in the face of a balanced low baseline stress physiology response such that low baseline cortisol along with low baseline alpha amylase predicted more trauma symptoms in IPV-exposed children.

Finally, when examining the interaction between baseline alpha amylase and baseline cortisol in children in the context of IPV there were relations between stress physiology and avoidance trauma symptoms, as measured by the TSSIC avoidance subscale. Consistent with earlier outcomes, concurrent high baseline cortisol and high baseline alpha amylase predicted decreased trauma symptoms in IPV-exposed children. Likewise, as was noted earlier, an unbalanced stress response characterized by high baseline cortisol coupled with low baseline alpha amylase predicted increased avoidance symptoms in IPV-exposed children. Additionally, the other uncoordinated pattern, that is, low baseline cortisol coupled with high baseline alpha

amylase, also predicted increased trauma symptoms in IPV-exposed children. Also, consistent with previous findings, concurrent low baseline cortisol and low baseline alpha amylase also predicted increased levels of avoidance symptoms in IPV-exposed children.

Once again, a protective effect was observed when IPV-exposed children demonstrated coordinated heightened baseline cortisol and alpha amylase functioning. Unbalanced stress physiology was once again associated with increased TSSIC avoidance symptoms, such that both combinations (i.e., high baseline cortisol and low baseline alpha amylase or low baseline cortisol and high baseline alpha amylase) were indicative of poor adjustment. Also, concurrent low baseline HPA axis and SNS functioning was predictive of increased avoidance trauma symptoms as noted in earlier findings.

Based on the present findings, there is support for the additive model, or a higher risk when the two systems exhibit a coordinated or symmetrical response when examining *baseline* levels of functioning. However, depending on whether the systems showed hyper- or hypoactivation determined whether there was an increase or decrease in risk for trauma symptoms in children exposed to IPV. Specifically, IPV-exposed children who displayed heightened HPA axis functioning along with heightened SNS functioning showed decreasing levels of both overall trauma symptoms as well as avoidance trauma symptoms. Therefore, symmetrical hyperactivation seemed to function in somewhat of a protective fashion. This is contrary to findings described by El-Sheikh et al. (2008) who found that concurrent high baseline activation of the HPA axis and SNS predicted increased maladjustment, specifically, internalizing symptoms. However, findings related to concurrent low activation were in line with previous research. In the current study, symmetrical low or hypoactivation of baseline levels of the HPA axis and SNS were associated with increased trauma symptoms both overall and for

avoidance symptoms in IPV-exposed children. Keller et al. (2012) similarly found that concurrent underarousal of baseline HPA axis and SNS functioning predicted poor outcomes, in this case, decreased cognitive ability.

In addition, contrary to hypotheses, the present study found evidence for the interactive model, that is, asymmetrical baseline functioning (i.e., uncoordinated baseline HPA axis and SNS functioning) being associated with increased maladjustment in children exposed to IPV when examining baseline levels of physiology. Interestingly, it did not matter whether it was high baseline cortisol with low baseline alpha amylase or low baseline cortisol with high baseline alpha amylase, in general, any combination that was unbalanced predicted increased overall trauma symptoms and increased avoidance trauma symptoms in children who had been exposed to IPV. This finding of asymmetrical functioning predicting increased maladjustment is not consistent with previous research. Past studies examining baseline HPA axis and SNS interactions have instead found that asymmetry of the pathways was associated with better executive functioning and academic achievement (Berry et al., 2012) and lower levels of internalizing and externalizing symptoms (El-Sheikh et al., 2008).

When examining stress physiology *reactivity* levels, a different picture emerged; asymmetrical reactivity was associated with decreased trauma symptoms in IPV-exposed children, suggesting that prediction of child outcomes is dependent upon whether baseline or reactivity levels are examined. Previous findings have been mixed with one study indicating that asymmetrical HPA axis and SNS reactivity was associated with lower levels of parent-reported aggression in 10 -14 year olds (Gordis et al., 2006), while another study suggested asymmetrical reactivity of HPA axis and SNS was associated with decreased positive affect and approach behaviors in toddlers (Fortunato, et al., 2008).

Physiological Patterns Predicting Increased Trauma Symptoms

Low Cortisol/Low Alpha Amylase. The finding that low activation of the SNS and HPA axis was associated with increased maladjustment has been observed in previous research. Many of the studies and reviews that have found an association between low activation and maladjustment have focused on outcomes related to externalizing symptoms and disorders (e.g., Gordis et al., 2006; see Beauchaine, Gatzke-Kopp, and Mead, 2007 and Raine, 2005). However, in the area of PTSD, past research has not uniformly shown that concurrent low activation of the HPA axis and SNS is associated with trauma symptoms. Instead, research has uncovered physiological functioning in PTSD that is quite variable (e.g., Pervanidou, 2008; Delahanty & Nugent, 2006). In response to traumatic events, studies have found increased SNS activation, normal activation (i.e., no differences in SNS functioning between traumatized and non-traumatized children and adolescents), as well as increased, decreased, and normal levels of HPA axis functioning in children and adolescents (e.g., Goenjian et al., 1996; Bryant et al., 2007; Pervanidou et al., 2007; De Bellis et al., 1999).

While studies of adults and children have revealed low cortisol as a physiological marker of PTSS/PTSD as the present study did, in adult studies and in some studies involving children, increased SNS has typically been associated with PTSS/PTSD. Therefore, the fact that low cortisol is one component of the interaction predicting increased trauma symptoms is not surprising, the low SNS aspect however is intriguing. A partial explanation for this may lie with the specific type of trauma symptom that is presented. In a study by Pervanidou et al. (2007) which examined physiological correlates of specific PTSD cluster symptoms, there was evidence for heightened SNS activation being associated with arousal symptoms 1 month post-trauma as opposed to avoidance symptoms. In the present study, when the TSSIC Total PTSD scale was

broken down into subscales, concurrent low activation of the HPA axis and SNS remained significant only for the avoidance subscale. It may be that concurrent low activation predicts avoidance symptoms specifically. This may point to a presentation of a different set of physiological markers dependent upon which symptoms are presented. However, some studies (Delahanty, et al., 2005; De Bellis et al., 1999) have found positive correlations between SNS measures and avoidance symptoms so this remains an area of uncertainty.

Studies examining the startle reflex in participants with PTSD found evidence for attenuated arousal. A blunted somatic response was found in a study by McTeague and Lang (2012) who examined the startle reflex measured by eyeblink magnitude in response to an auditory probe in participants with PTSD. They found that for PTSD sufferers who developed PTSD secondary to a cumulative type of trauma (e.g., maltreatment or IPV exposure) they had differing startle responses compared to those with PTSD subsequent to a one-time traumatic event (e.g., motor vehicle accident). In fact, those with PTSD subsequent to a trauma that was of a more chronic nature displayed a “blunted” startle response compared to those who experienced a single event trauma who displayed a heightened response. Similarly, Medina, Mejia, Schell, Dawson, and Margolin (2001) studied the eyeblink response to an auditory probe in women with histories of Type II stressors (i.e., “corporal punishment and intimate partner aggression”). They found an inverse relationship between eyeblink magnitude and the level of PTSD symptoms. As PTSD symptoms increased, eyeblink magnitude decreased.

The findings from these studies suggest it may be that underarousal of physiological systems is linked to trauma symptoms secondary to a trauma that is typically of an ongoing nature such as IPV (McTeague & Lang, 2012). That is, the type of trauma, either Type I (one time acute trauma) or Type II (repeated, chronic trauma) may influence which physiological

patterns are presented in an individual. It may be that in Type II traumas, those that are of a more chronic nature, there is a physiological response characterized by the down regulation of the HPA axis and SNS. The initial response of the HPA axis may have been to increase the production and release of cortisol. However, given that IPV is typically of a chronic nature (Perkins & Graham-Bermann, 2006; Terr, 1991), over time, the HPA axis may have become down regulated via the negative feedback loops present in the pathway both to the hypothalamus and pituitary gland resulting in overall low baseline cortisol levels (e.g., De Bellis, 2001). It may be that children exposed to IPV are frequently stressed, due to the tense nature of their home life or from assessing the environment for signs of the next altercation between their parents. Since the HPA axis and SNS are interrelated, specifically with neurons that reciprocally innervate the LC and the hypothalamus (Stratakis & Chrousos, 1992; Bauer et al., 2002), down regulation of the HPA axis may result in down regulation of the SNS (e.g., Chrousos & Gold, 1992). Likewise, cortisol activity is also responsible for sensitizing target organs to SNS activation and when cortisol levels are low, this may be reflected in lower SNS arousal (Sapolsky et al, 2000). Additionally, since SNS levels may impact cortisol levels, low SNS levels may help maintain low cortisol levels (Mokuda, Sakamoto, Kawagoe, Ubukata, & Shimizu, 1992).

In addition to concurrent low activation, asymmetrical activation of the stress response pathways also predicted increased trauma symptoms. While this is contrary to the findings of some studies where asymmetrical activation was not associated with maladjustment (e.g., Gordis et al., 2006; El-Sheikh et al., 2008), increased trauma symptoms were observed in IPV exposed children who exhibited both increased cortisol coupled with decreased alpha amylase as well as the inverse, decreased cortisol with concurrent increased alpha amylase. Why asymmetrical functioning was found to be associated with increased PTSS/PTSD may be a result of the two

systems not ‘balancing’ the other out (Bauer et al., 2002).

High Cortisol/Low Alpha Amylase. First, in the case of high cortisol coupled with low alpha amylase predicting increased trauma, as found in all three significant baseline interaction models, it may be that these IPV-exposed children have elevated HPA axis functioning due to the stressful nature of their home life. Many studies examining HPA axis functioning in children exposed to trauma have in fact, found increased cortisol (e.g., Saltzman et al., 2005; De Bellis et al., 1999; Cicchetti & Rogosch, 2001a; Cicchetti & Rogosch, 2001b; Rotton et al., 1997; Pfeffer et al., 2007). It may be that the increased cortisol levels function to suppress the SNS response (e.g., Keller et al., 2012; Berry et al., 2012). As Engert, Vogel, Efanov, Duchesne, Corbo, Ali et al., (2011) noted, animal models have indicated attenuated NE in mice that have been exposed to an inescapable stressor (e.g., Irwin, Ahluwalia, & Anisman, 1986; Anisman & Sklar, 1981), something that typically elicits an HPA axis response indicating that cortisol may be functioning to suppress SNS (see Engert, et al., 2011). As such, in the case of the present study, a child may be left with a physiological profile where cortisol levels are high and alpha amylase levels are low and therefore, according to the interactive model, the two systems may be unable to counterregulate each other (Chrousos & Gold, 1992; Bauer et al., 2002). This uncoordinated response of the two pathways may result in affective regulatory issues such as those seen with trauma symptoms via the neurotoxic effects of elevated cortisol on the brain (e.g., De Bellis, Keshavan, Clark, Casey, Giedd, Boring, Frustaci, et al., 1999).

Other studies have shown increased cortisol to be associated with intrusive and hyperarousal symptoms (De Bellis et al., 1999; Pevanidou et al., 2007). While the present findings did not indicate these specific relationships between cortisol and intrusive and hyperarousal symptoms, the high cortisol/low alpha amylase pattern was related to avoidance

symptoms. There is some evidence that avoidance is linked to decreased SNS functioning. Griffin, Resick, and Mechanic (1997), examined physiology and dissociative symptoms in recent rape victims. They found that those who scored highest on a dissociative scale had lower levels of SNS functioning (i.e., heart rate and SCL) than those who scored low on the dissociative scale. However, contrary to these findings, past research of traumatized children has also shown avoidance symptoms to be associated with increased NE in particular (De Bellis et al., 1999; Pevanidou et al., 2007). Thus, the interaction between high cortisol and low alpha amylase predicting increased total trauma symptoms and avoidance symptoms warrants further investigation. The high cortisol component relating to avoidance symptoms specifically, seems counter to past work (perhaps increased intrusive and arousal symptoms are only observed when high cortisol is coupled with moderate SNS arousal although this was not seen in the present data) and while the low alpha amylase component has been observed in the Griffin et al. study, studies of traumatized children have conversely observed increased SNS markers (i.e., NE) associated with avoidance symptoms (De Bellis et al., 1999; Pevanidou et al., 2007).

Low Cortisol/High Alpha Amylase. Finally, the inverse asymmetrical pattern, that of low cortisol and high alpha amylase, was also observed and associated with increased trauma symptoms as measured by the CBCL-PTSD scale and the TSSIC avoidance scale. This physiological pattern may be due to an HPA axis that has down regulated over the course of time in response to a repetitive stressor such as IPV and as such, it may have left the SNS unchecked leading the two pathways become imbalanced. It has been argued that this particular pattern of physiological imbalance may underlie the maladaptive memory encoding of the traumatic event that has been associated with the re-experiencing and intrusive trauma symptoms that are part of the PTSD symptom clusters (Yehuda, McFarlane, & Shalev, 1998; Delahanty & Nugent, 2006;

Pitman, 1989). Specifically, Delahanty and Nugent (2006) suggest that the underarousal of the HPA axis may be unable to sufficiently suppress the SNS response. Increased SNS activity is associated with increased focus and attention and in the context of a traumatic event this may be disadvantageous in terms of memory consolidation and perhaps, as a result, may lead to increased intrusion symptomatology (Delahanty & Nugent, 2006). Given this, why low cortisol and high alpha amylase predicted increased avoidance symptoms in particular is uncertain. It seems counterintuitive that this physiological pattern of low cortisol and high alpha amylase would predict increased avoidance symptoms as this symptom cluster is indicative of forgetfulness regarding the traumatic experience. However, as noted above, previous research examining the physiological correlates of specific PTSD cluster symptoms in traumatized children have tended to observe increased SNS functioning, NE in particular associated with avoidance symptoms (De Bellis et al., 1999; Pevanidou et al., 2007). Additionally, Goenjian et al., 2003 found a negative correlation between cortisol and avoidance symptoms in youth (6.5 years after the trauma occurred). Therefore in terms of avoidance symptoms specifically, the combination of low cortisol and high alpha amylase appear to be in line with past work.

Physiological Patterns Predicting Decreased Trauma Symptoms

High Cortisol/High Alpha Amylase. In addition to predicting increased trauma symptoms, IPV-exposed children's physiology also exhibited patterns that appeared to be protective against trauma symptomatology. A symmetrical pattern characterized by hyperarousal of both systems was associated with decreased trauma symptoms in these children. In the case of coordinated heightened activation, it may be that the heightened HPA axis response serves to stimulate increased production and release of SNS biomarkers (i.e., NE and EPI) and vice versa (Mokuda et al., 1992; Owens & Nemeroff, 1991). As Engert and colleagues have reviewed, studies have

indicated that increases in CRF, the first hormonal product of the HPA axis, are associated with increases of SNS indices (e.g., Owens & Nemeroff, 1991). Also, given that cortisol acts to sensitize tissues to NE and EPI, this may result in increased activity for both pathways (Sapolsky et al., 2000). The protective nature of this physiological pattern may lie in that the systems balance each other out. Thus, the high SNS response is matched by the cortisol response thereby neither system is going unchecked. In fact, regarding memory consolidation in the context of stress, Delahanty and Nugent (2006) noted that studies have found support for high levels (versus low levels) of cortisol counteracting the memory encoding effects observed in the context of heightened SNS activation mentioned earlier (Borrell, De Kloet, Versteeg, & Bohus, 1983). In this case, it appears that the heightened coactivation of the pathways acts in a protective manner by balancing and containing each others' effects. Likewise, there is evidence that increased SNS levels may function as a protective factor for externalizing problems. Specifically, heightened SNS activation has been identified as a protective biological marker in boys who were otherwise at risk for antisocial behavior (see Raine, 2005 for review). Together, these findings indicate that a heightened physiological response may prove protective against maladjustment including trauma symptoms in IPV-exposed children and for children at risk for antisocial behavior.

In terms of *reactivity* level of SNS activation and HPA axis activation predicting trauma outcomes, a protective effect emerged again. Asymmetrical reactivity was associated with decreased trauma symptoms. Specifically, in the present study, high cortisol reactivity coupled with low alpha amylase reactivity predicted decreased avoidance symptoms in IPV-exposed children. The physiological mechanism underlying this finding may reflect a stress response where increased cortisol reactivity in response to an acute stressor may work to buffer against the

low SNS activity. In times of stress, in the absence of a robust SNS response, the heightened cortisol may work to increase attention and focus and perhaps fend off dissociative symptomology.

Additional Variables Which May Impact Post-trauma Physiology

It is unclear why specific physiological patterns predicted increased maladjustment versus protection. There are likely other variables involved that may moderate these relationships and influence the physiology that is exhibited. Other factors may include variables such as time since the IPV-exposure occurred and other past trauma history (see De Bellis, 2001). For example, the finding that concurrent low activation of the SNS and HPA axis predicted increased trauma symptoms may be mediated by time since trauma. That is, for those children who have had a significant amount of time since their last exposure to IPV, they may exhibit decreased HPA axis functioning. This was observed in a sample of adolescents who experienced an earthquake (Goenjian et al., 1996; 2003). Five years following the earthquake, lower baseline cortisol levels were seen in the youth who were more traumatized (i.e., closer to the epicenter) versus those who were less traumatized (farther away from the epicenter). Similarly, Pfeffer et al., 2007, noted declining cortisol levels over the course of two years in children who were exposed to the 9/11 terror attacks. In the case of SNS activation, no differences were observed 5 years after an earthquake between those who were more traumatized versus those who were less traumatized (Goenjian et al., 1996). Similarly, De Bellis, Chrousos, Dorn, Burke, Helmers, Kling, et al. (1994) found that 7 to 15 year old girls with a history of sexual abuse that had been reported years prior, displayed decreased levels of ACTH in response to exogenous CRH as compared to controls, although their cortisol levels remained normal. As such it may be that the

children with concurrent low physiological pattern may be those who have had more time pass since the IPV relationship ended.

Additionally, IPV may be one of many traumas that these children have endured. In the present study's analyses, child maltreatment was included as a covariate due to evidence that these two traumas frequently co-occur (e.g., Browne & Hamilton, 1999). This is but one example of other potential traumas these children may have faced. In past studies, cumulative trauma exposure has predicted increased PTSD symptoms in youth and adults (e.g., Suliman, Mkabile, Fincham, Ahmed, Stein, & Seedat, 2009; Delahanty & Nugent, 2006; Daviss, Mooney, Racusin, Ford, Fleischer, & McHugo, 2000). Additionally, cumulative trauma has been related to decreased morning baseline cortisol levels and increased afternoon cortisol levels, or in other words, a flatter diurnal pattern in children as compared to examining recent trauma or previous trauma alone (Bevans, Cerbone, & Overstreet, 2008). Previous trauma history was not collected in the present study and therefore was not controlled for. The cumulative effect of trauma on children's physiological functioning may help explain the diverse physiological patterns observed in IPV-exposed children in the present study. Post hoc analyses were conducted to examine potential relationships between the total months of IPV exposure and physiology and trauma symptoms. There were no significant associations between the total months of IPV and trauma symptoms, however, there was a significant positive relationship between cortisol reactivity and total months of abuse, indicating that children whose mothers experienced a higher number of months of abuse had increased cortisol reactivity. Likewise, post hoc correlations between the time since the abuse stopped and physiology and trauma symptoms were also examined. No significant findings were revealed. However, due to sample size limitations, this does not eliminate the possibility that other past trauma history or time since the trauma occurred

affected the children's physiology in the present study. More research with larger samples is warranted to determine whether these factors may play a role in stress physiology.

De Bellis (2001) posited a model describing the psychobiology of trauma (specifically maltreatment) in children, the developmental traumatology model, that incorporates both past trauma and time since trauma influences on children's physiological functioning at baseline and in response to a new stressor. He argues that a physiological mechanism, termed "priming", or hypoactive baseline HPA axis levels with HPA axis hypersensitivity to new stressors, may offer explanation for the range of findings associated with HPA axis functioning in children who have been traumatized (Chrousos & Gold, 1992). He suggests that the divergent findings that are observed in research examining HPA axis functioning in children may be explained by the timing at which the HPA axis measure was collected (i.e., the amount of time that has passed since trauma occurred) and whether or not the participant has a history of past trauma. Specifically, children who experience acute trauma would exhibit increased HPA axis functioning. However, if the stress is chronic and long-term (as is often the case with maltreatment) or occurred years earlier, the child's HPA axis may down regulate resulting in low levels of HPA axis indices. Moreover, if a child with a history of chronic stress or a child who experienced a trauma years earlier then experienced an acute stressor, their HPA axis pathway may respond with hyper-secretion, resulting in high levels of HPA axis indices. This is because the child's system is "primed", that is, there is dysregulation as evidenced by a hypoactive HPA axis, stemming from the chronic or past trauma, and when a current stressor activates the HPA axis pathway heightened activation is observed, a result of the effect of the current stressor activation on the hypoactive HPA axis. Thus, the developmental traumatology model would suggest that in the current study, children with HPA axis underarousal experienced stress

previously in their life history or have a history of chronic stress (i.e., IPV exposure) while those with heightened HPA axis activation experienced a more recent or current stressor. Furthermore, if the child has a past history of trauma, their system may have been “primed” and as such they will hyper-respond to an acute stressor (De Bellis, 2001; Chrousos & Gold, 1992).

Other variables that may influence the physiological patterns observed in the present study include the child’s gender (e.g., Goenjian et al., 1996; Cicchetti & Rogosch, 2001; King et al., 2001), age (e.g., Bugental, et al., 2003; Carpentar et al., 2007), and comorbid psychiatric disorders (e.g., Stratakis & Chrousos, 1995; Cicchetti & Rogosch, 2001). All of these variables may exert an influence on physiological functioning in conjunction with the trauma exposure. In the present study, efforts were made to take into account these additional variables that may affect child physiological functioning. However, due to a small sample size and missing data, all of the aforementioned covariates could not be entered into the analyses simultaneously. However, post hoc analyses suggested that child depression, a potential comorbid psychiatric disorder associated with PTSD and altered physiological functioning, was not associated with the trauma symptom outcome variables or the physiology variables. Additionally, analyses controlling for child gender rendered similar interaction findings as those reported (i.e., findings were significant or showed a trend with and without child gender as a covariate). Regarding child age, as with child gender, post hoc analyses controlling for child age in addition to the previous covariates (i.e., time of session and child maltreatment) yielded similar significant or trending findings. Therefore, in the present sample, it does not appear that these items had an effect on results, but given the small sample size, they cannot be ruled out.

Another factor to take into consideration regarding the diverse findings of the current study centers on the physiology measures. As reported previously, analyses were conducted

using two different measures for the baseline levels of cortisol and alpha amylase. The first baseline measure, Baseline 1, used in analyses was collected from the participants approximately ten minutes after arrival to the session. The second baseline measure, Baseline 2, was collected after the children listened to a neutral story on headphones. The baseline measure that was associated with the significant findings in this study was Baseline 1. This is interesting because cortisol levels were at their peak at Baseline 1 while alpha amylase was at its lowest. It is possible that Baseline 1 was measuring levels of cortisol and alpha amylase that reflected reactivity levels, specifically for cortisol. At first glance, one might expect peak alpha amylase levels because of the time duration from arrival to the session until the first saliva collection was obtained. However, it is possible children may have initiated a stress response on the car ride to the session in anticipation of the unknown. For the children in this study, there was a lot of novelty and perhaps apprehension regarding their participation in the session, something that could trigger the stress response. This may answer why the Baseline 1 measures were associated with trauma symptoms outcomes while Baseline 2 was not.

Limitations

Several limitations of the present study should be noted. First, is the small sample size; findings were based upon a sample of 35 mother-child dyads. This limits the ability to detect effects due to power issues as well as limiting the generalizability of the present findings beyond the sample. While efforts were made to retain 50 mother-child dyads in total for the larger intervention study, including frequent telephone contact with participants, vouchers for travel costs, reminder calls prior to appointments, free childcare at the sessions, and monetary incentives for the participant's time, it was difficult to recruit and retain families. This may be

due in part to the reality of being in a post-IPV relationship. Families living in the aftermath of an IPV relationship may be under a lot of stress. The family may have had to move residences. Mothers may be the sole caretaker and provider for the family which means financial stress and dealing with issues related to childcare. Retention of participants was difficult as well. Participants were asked to attend two pre-intervention sessions for baseline measures (i.e., questionnaires, saliva collection) followed by 10 weeks of parenting sessions. The duration of the study may have been too great a commitment for some. Likewise, missing data was an issue. Not all of the children agreed to provide saliva samples and not all mothers and children completed their questionnaires.

Another limitation is the cross sectional nature of the study; it is unclear whether the altered physiology is a product of the trauma or if the trauma preceded the altered physiology. That is, in the event of a trauma, are some children predisposed for future PTSS/PTSD because of preexisting altered physiological functioning or does the experience of trauma create an altered physiological state which in turn increases risk for the development of PTSS/PTSD? A longitudinal design, like that conducted by Shenk et al., 2012, would explicate which direction the relationship between trauma and physiology goes. Presently, based on Shenk et al.'s findings, as well as findings from studies that have measured physiology immediately post-stressor (typically in a hospital setting; e.g., Delahanty et al., 2005; Pervanidou et al., 2007; Bryant et al., 2007) the evidence seems to indicate that trauma alters physiology which is then followed by increased trauma symptoms, but more research is warranted.

Similarly, the patterns of physiological functioning and its relationship to trauma symptoms in the context of IPV that were observed may not be best described by a linear relationship. Keller et al. (2012) found that interactions between alpha amylase and cortisol

predicting cognitive functioning in children were best depicted by curvilinear models as opposed to linear models. The present study was conducted under the assumption of linear relationships; it is possible that these interactions may be better described in curvilinear terms.

Another limitation centers on the age range of the child participants in the study and the developmental implications on their physiological measures. The age range of the participants for the current study was from 6 to 12 years. During this age range, adrenarche occurs, specifically between ages 6 and 8. Adrenarche results in the increase of DHEA, a hormone secreted from the adrenal glands and may be an indication of pubertal status (Nakamura, Gang, Suzuki, Sasano, & Rainey, 2009). Pubertal status has been linked to cortisol levels in some studies, while other studies have not found a link (see Rotenberg, McGrath, Roy-Gagnon, and Tu, 2012). Rotenberg et al. (2012) examined the effects of various covariates (e.g., sex, age, adrenarche, gonadarche, time of awakening) on cortisol levels and found that adrenarche was associated with cortisol production, however, overall the covariates as a whole accounted for minimal (less than 10%) variance in cortisol levels. In light of the inconsistencies in the literature, adrenarche may or may not have an effect on the current study's findings.

Finally, a further limitation of the study is the issue of eliciting an SNS and HPA axis response to a stressor in children. The findings of the current study are mainly from baseline levels of activation. Of the four significant interaction terms, only one was based on alpha amylase and cortisol reactivity. This may be due to reactivity levels not actually predicting child trauma symptoms in this sample, but it also may be a product of the stressor used in the study and the timing of saliva collections, or due to the hyporeactive nature of children's HPA axis and SNS to stressors. Gunnar, Talge, and Herrera (2009) investigated different stressor paradigms and their ability to elicit an increase in cortisol across development. They found that public

speaking tasks, similar to what was used in the present study, did not always evoke an increased cortisol response. Generally, older children (13 years and up) seemed to demonstrate fairly consistent increases in HPA axis activation, but for younger children findings have been inconsistent. In terms of alpha amylase reactivity, Yim, Granger, and Quas (2010) found that while adults demonstrated increased alpha amylase levels in response to a stressor (i.e., public speaking and mental arithmetic), children did not display increased alpha amylase in response to the stressor. Taken together, the lack of findings based on reactivity measures in the present study may be a product of the failure of the stressor to elicit HPA axis and SNS responses.

Future Directions

Future research examining the interaction between physiological biomarkers and child outcomes would benefit from a longitudinal design. This would elucidate whether individual differences in physiology pre-stressor are risk factors for later maladjustment or, if altered physiological patterns were the consequence of trauma exposure and subsequent PTSS/PTSD development. This information would be valuable for the early identification of children that may be at a higher risk for poor outcomes like PTSD.

Furthermore, the present study should be replicated with a larger sample to determine whether the observed pattern of interactions between the HPA axis and SNS are consistent across studies. Increasing the sample size would also allow the inclusion of additional covariates that have been associated with physiological functioning and trauma symptoms. Inclusion of variables related to participant's trauma exposure such as participants prior trauma history, the length of time since the trauma(s), and the duration of the trauma as well as demographic variables like age and gender, and psychiatric disorder history would allow for deciphering the

diverse physiological patterns observed in the present study and perhaps specify which physiological pattern might be expected given the individual differences reflected by these variables. Along those lines, additional independent variables reflecting other types of traumas such as maltreatment, natural disasters, or traumatic injuries should be tested to see if similar physiological patterns predict trauma symptoms secondary to a trauma other than IPV.

Additionally, future research investigating physiological interactions in IPV-exposed children should incorporate both reactivity and baseline levels in analyses. That is, examine interactions between *baseline* HPA axis functioning and SNS *reactivity* functioning and vice versa. El-Sheikh et al. (2009) studied the effects of PNS and SNS interactions on children's externalizing problems in the context of marital conflict and found interactions between baseline and reactivity measures when examining PNS and SNS interactions. The authors argued that since reactivity levels and baseline levels of PNS and SNS functioning may influence each other, examining their interactions would be a fruitful endeavor. They found support for the inclusion of interactions examining reactivity by baseline levels of functioning with various interactions between baseline and reactivity between the PNS and SNS predicting externalizing symptoms. This too, would be interesting to investigate in relations between the SNS and HPA axis because like the PNS and SNS, the baseline and reactivity levels of each may affect the functioning of the other.

Additionally, it would be interesting to test the interactions between the HPA axis and the PNS as well as the SNS and the PNS to predict trauma outcomes in IPV-exposed children. The PNS, specifically respiratory sinus arrhythmia (RSA), has been associated with child outcomes across a variety of domains including externalizing symptoms (e.g., Katz & Gottman, 1995), internalizing symptoms (Hinnant & El-Sheikh, 2009), peer interactions (Leary & Katz, 2004),

and emotion regulation (Porges, Doussard-Roosevelt, & Maita, 1994). Studies have demonstrated that the interaction between the PNS and HPA axis have predicted child outcomes. El-Sheikh and colleagues (2011) conducted a study that examined the interaction between RSA and cortisol levels in third grade children and its association with child-reported levels of depression and anxiety. They found that the interaction between the HPA axis and PNS predicted children's internalizing symptoms such that asymmetrical activation of the HPA axis and PNS predicted increased depression and anxiety. Shenk et al. (2010) similarly found that asymmetrical functioning of the HPA axis and PNS as measured by cortisol and RSA, respectively, measured during late adolescence also predicted increased depression and antisocial behaviors in adulthood in females with a history of maltreatment. These findings provide support for examining interactions between the HPA axis and PNS to predict outcomes and extending them to incorporate the HPA axis and PNS in studies examining biomarkers of risk for trauma symptoms, specifically in IPV-exposed children, would be informative. In addition, as mentioned previously, El-Sheikh et al. (2009) found evidence for interactions between the PNS and SNS predicting child externalizing symptoms in the context of marital conflict. The examination of the interaction between the PNS and SNS also has not been investigated in IPV-exposed children.

Finally, these findings highlight that trauma symptoms can be predicted in IPV-exposed children using a pain-free non-invasive method, that is, from saliva samples. The implications of this research could assist those involved in child healthcare services as a means for early identification for which children may be in need of services for the prevention of or intervention for PTSS/PTSD. Saliva sampling is a non-invasive tool that could be used in conjunction with parent and child report of traumatic events via interviews or questionnaires to determine who is

at greatest risk for maladjustment. Early prevention or intervention may help ameliorate the consequences of the trauma exposure that is all too common for children.

Conclusion

The present study demonstrated the use of biomarkers for predicting trauma symptoms in IPV-exposed children. Furthermore, it provided support for Bauer et al.'s (2002) multi-system approach when trying to determine risk for maladjustment in children. The present study's findings indicate that the inclusion of the interaction of the SNS and HPA axis predicted IPV-exposed children's trauma symptoms over and above the independent predictive effects of each system. Additionally, there was evidence for both the interactive model and the additive model. Regarding the additive model, concurrent high or low activation predicted trauma symptoms in the context of IPV. However, this was dependent upon whether the activation was characterized by concurrent underarousal or concurrent hyperarousal. In the case of concurrent low activation of the HPA axis and SNS, there were increased trauma symptoms. Interestingly, concurrent high activation of the two systems appeared to be protective as it was associated with decreased trauma symptoms. In terms of the interactive model, baseline asymmetrical activation was associated with increased trauma symptoms, reflected by either increased HPA axis functioning coupled with diminished SNS functioning or vice versa. Conversely, asymmetrical reactivity levels predicted decreased trauma symptoms. These findings add to the literature on physiological functioning in traumatized children and provide support for the inclusion of a multi-system approach.

The implications of this research may be useful in a clinical setting. The ability to assess biomarkers of trauma symptoms in a non-invasive manner in IPV-exposed children may allow

pediatricians and other child health workers a relatively easy way to determine which children have a physiological pattern associated with trauma symptoms and thus identify who might benefit from intervention. Additionally, while further research is necessary, these findings have the potential to extend to traumas outside of IPV exposure including maltreatment, traumatic injury, and natural disasters meaning that those who work with children exposed to trauma in general may have a non-invasive way to assess which children need additional care post-trauma. In addition to being a non-invasive and pain-free method, there is also a cost benefit to examining salivary biomarkers of trauma symptoms. Since not all children exposed to IPV go on to develop trauma symptomatology, using biomarkers associated with trauma symptoms could highlight which children would stand to gain from intervention services and the focus could be on treating those particular children as opposed to all exposed children when they may not all be in need of intervention. Furthermore, understanding the physiology of children who have been exposed trauma may inform the medical field of areas to target for pharmaceutical intervention.

References

- Achenbach, T.M. (1991). *Manual for the Youth Self-Report and 1991 Profile*. Burlington, VT: University of Vermont, Department of Psychiatry.
- Achenbach, T.M., & Edelbrock, C. (1983). *Manual for the Child Behavior Checklist and revised child behavior profile*. Burlington, VT: University of Vermont, Department of Psychiatry.
- Aiken, L.S. & West, S.G. (1991). *Multiple regression: testing and interpreting interactions*. Newbury Park, CA: Sage.
- Anisman, H., & Sklar, L.S. (1981). Social housing conditions influence escape deficits produced by uncontrollable stress: Assessment of the contribution of norepinephrine. *Behavioral and Neural Biology*, 32, 406-427.
- Aldwin, C.M. (1994). *Stress, coping, and development: an integrative perspective*. New York: Guilford Press. (Chap 4).
- Allwood, M.A., Handwerker, K., Kivlighan, K.T., Granger, D.A., & Stroud, L.R. (2011). Direct and moderating links of salivary alpha-amylase and cortisol stress-reactivity to youth behavioral and emotional adjustment. *Biological Psychology*, 88, 57-64.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- Bauer, A.M, Quas, J.A., & Boyce, W.T. (2002). Associations between physiological reactivity and children's behavior: Advantages of a multisystem approach. *Journal of Developmental & Behavioral Pediatrics*, 23, 102-113.
- Badanes, L.S., Watamura, S.E., & Hankin, B.L. (2011). Hypocortisolism as a potential marker of allostatic load in children: Associations with family risk and internalizing disorders. *Development and Psychopathology*, 23, 881-896.
- Beauchaine, T.P., Gatzke-Kopp, L., & Mead, H.K. (2007). Polyvagal theory and developmental psychopathology: Emotion dysregulation and conduct problems from preschool to adolescence. *Biological Psychology*, 74, 174-184.
- Berry, D., Blair, C., Willoughby, M., & Granger, D.A. (2012). Salivary alpha-amylase and cortisol in infancy and toddlerhood: Direct and indirect relations with executive functioning and academic ability in childhood. *Psychoneuroendocrinology*, 37, 1700-1711.
- Bevans, K., Cerbone, A., & Overstreet, S. (2008). Relations between recurrent trauma exposure and recent life stress and salivary cortisol among children. *Development and Psychopathology*, 20, 257-272.

- Bogat, G.A., DeJonghe, E., Levendosky, A.A., Davidson, W.S., & von Eye, A. (2006). Trauma symptoms among infants exposed to intimate partner violence. *Child Abuse & Neglect*, *30*, 109-125.
- Borrell, J., De Kloet, E.R., Versteeg, D.H., & Bohus, B. (1983). Inhibitory avoidance deficit following short-term adrenalectomy in the rat: The role of adrenal catecholamines. *Behavioral and Neural Biology*, *39*, 241-258.
- Bosch, J.A., De Geus, E.J., Veerman, E.C., Hoogstraten, J., Nieuw Amerongen, A.V. (2003). Innate secretory immunity in response to laboratory stressors that evoke distinct patterns of cardiac autonomic activity. *Psychosomatic Medicine*, *65*, 245-258.
- Bosch, J.A., Veerman, E.C., De Geus, E.J., & Proctor, G.B. (2011). Alpha amylase as a reliable and convenient measure of sympathetic activity: Don't start salivating just yet! *Psychoneuroendocrinology*, *36*, 449-453.
- Brown, E.J. (2005). Correlates and treatment of stress disorder in children and adolescents. *Psychiatric Annals*, *35*, 759-765.
- Browne, K.D. & Hamilton, C.E. (1999). Police recognition of the links between spouse abuse and child abuse. *Child Maltreatment*, *4*, 136-147.
- Bryant R.A., Salmon K., Sinclair E., & Davidson P. (2007). Heart rate as a predictor of posttraumatic stress disorder in children. *General Hospital Psychiatry*, *29*, 66-8.
- Bugental, D.B., Martorell, G.A., & Barraza, V. (2003). The hormonal costs of subtle forms of infant maltreatment. *Hormones and Behavior*, *43*, 237-244.
- Buske-Kirschbaum, A., Jobst, S., Wustmans, A., Kirschbaum, C., Rauh, W., & Hellhammer, D. (1997). Attenuated free cortisol response to psychosocial stress in children with atopic dermatitis. *Psychosomatic Medicine*, *59*, 419-426.
- Burt, K.B., & Obradovic, J. (2013). The construct of psychophysiological reactivity: Statistics and psychometric issues. *Developmental Review*, *33*, 29-57.
- Cannon, W.B. (1939). *The Wisdom of the Body*. New York: Norton.
- Carpenter, L.L., Carvalho, J.P., Tyrka, A.R., Wier, L.M., Mello, A.F., Mello, M.F., et al. (2007). Decreased adrenocorticotropic hormone and cortisol responses to stress in healthy adults reporting significant childhood maltreatment. *Biological Psychiatry*, *62*, 1080-1087.
- Carrion, V.G., Weems, C.F., Ray, R., & Reiss, A.L. (2002). Toward an empirical definition of pediatric PTSD: The phenomenology of PTSD symptoms in youth.

- Journal of the American Academy of Child & Adolescent Psychiatry*, 41, 166-173.
- Chatterton, R.T., Vogelsong, K.M., Lu, Y., Ellman, A.B., and Hudgens, G.A. (1996). Salivary alpha-amylase as a measure of endogenous adrenergic activity. *Clinical Physiology*, 16, 433-448.
- Chatterton, R.T., Vogelsong, K.M., Lu, Y., & Hudgens, G.A. (1997). Hormonal responses to psychological stress in men preparing for skydiving. *Journal of Clinical Endocrinology and Metabolism*, 82, 2503-2509.
- Chrousos, G. & Gold, P. (1992). The concepts of stress and stress system disorders. Overview of physical and behavioral homeostasis. *Journal of the American Medical Association*, 267, 1244-52.
- Charmandari, E., Tsigos, C., & Chrousos, G. (2005). Endocrinology of the stress response. *Annual Review of Physiology*, 67, 259-284.
- Cicchetti, D., & Rogosch, F.A. (2001). Diverse patterns of neuroendocrine activity in maltreated children. *Development and Psychopathology*, 13, 677-693.
- Cicchetti D, & Rogosch F.A. (2001). The impact of child maltreatment and psychopathology on neuroendocrine functioning. *Developmental Psychopathology*, 13, 783-804.
- Davies, P.T., Sturge-Apple, M.L., Cicchetti, D., Manning, L., & 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, 1384-1391.
- Daviss, W.B., Mooney, D., Racusin, R., Ford, J.D., Fleischer, A., & McHugo, G.J. (2000). Predicting posttraumatic stress after hospitalization for pediatric injury. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39, 576-583.
- 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., Baum, A.S., Birmaher, B., Keshavan, M.S., Eccard, C.H., Boring, A.M., et al. (1999). Developmental traumatology: Part I. Biological stress systems. *Biological Psychiatry*, 45, 1259-1270.
- De Bellis, M.D., Lefter, L., Trickett, P.K., & Putnam, F.W. (1994). Urinary catecholamine excretion in sexually abused girls. *Journal of the American Academy of Child & Adolescent Psychiatry*, 33, 320-327.
- De Bellis, M.D., Keshavan, M.S., Clark, D.B., Casey, B.J., Giedd, J.N., Boring, A.M., et

- al. (1999). Developmental traumatology Part II: Brain development. *Biological Psychiatry*, 45, 1271-1284.
- De Bellis, M.D., Chrousos, G.P., Dorn, L.D., Burke, L., Helmers, K., Kling, M.A. et al. (1994). Hypothalamic-pituitary-adrenal axis dysregulation in sexually abused girls. *Journal of Clinical Endocrinology and Metabolism*, 78, 249-255.
- de Kloet, E.R. (1991). Brain corticosteroid receptor balance and homeostatic control. *Frontiers in Neuroendocrinology* 12, 95–164.
- Delahanty, D.L. & Nugent, N.R. (2006). Predicting PTSD prospectively based on prior trauma history and immediate biological responses. *Annals New York Academy of Sciences*, 1071, 27-40.
- Delahanty, D.L., Nugent, N.R., Christopher, N.C., & Walsh, M. (2005). Initial urinary epinephrine and cortisol levels predict acute PTSD symptoms in child trauma victims. *Psychoneuroendocrinology*, 30, 121-128.
- deVries-Bouw, Jansen, Vermeiren, Doreleijers, van de Ven, & Popma (2012). Concurrent attenuated reactivity of alpha-amylase and cortisol is related to disruptive behavior in male adolescents. *Hormones and Behavior*, 62, 77-85.
- De Young A.C., Kenardy J.A., & Spence S.H. (2007). Elevated heart rate as a predictor of PTSD six months following accidental pediatric injury. *Journal of Traumatic Stress*, 20, 751-6.
- Dickerson, S.S. & Kemeny, M.E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychology Bulletin*, 130, 355-391.
- Doherty-Sneddon, G., Phelps, F., & Clark, J. (2007). Development of gaze aversion: Qualitative changes over the early school years. *British Journal of Developmental Psychology*, 25, 513-526.
- El-Sheikh, M., Arsiwalla, D.D., Hinnant, B., & Erath, S.A. (2011). Children's internalizing symptoms: The role of interactions between cortisol and respiratory sinus arrhythmia. *Physiology & Behavior*, 103, 225-232.
- El-Sheikh M., Kouros, C.D., Erath, S., Cummings, M.E., Keller, P., Staton, L., et al. (2009). Marital conflict and children's externalizing behavior: Interactions between parasympathetic and sympathetic nervous system activity: I. Introduction. *Monographs of the Society for Research In Child Development*, 74, 1-18.
- El-Sheikh M., Kouros, C.D., Erath, S., Cummings, M.E., Keller, P., Staton, L., et al. (2009). Marital conflict and children's externalizing behavior: Interactions between parasympathetic and sympathetic nervous system activity: II. Interactions Among marital conflict, sympathetic, and parasympathetic nervous systems activity in the

- prediction of children's externalizing problems. *Monographs of the Society for Research In Child Development*, 74, 19-34.
- El-Sheikh M., Kouros, C.D., Erath, S., Cummings, M.E., Keller, P., Staton, L., et al. (2009). Marital conflict and children's externalizing behavior: Interactions between parasympathetic and sympathetic nervous system activity: III. Additional testing of three-way interactions in an independent sample. *Monographs of the Society for Research In Child Development*, 74, 35-43.
- El-Sheikh M., Kouros, C.D., Erath, S., Cummings, M.E., Keller, P., Staton, L., et al. (2009). Marital conflict and children's externalizing behavior: Interactions between parasympathetic and sympathetic nervous system activity: IV. Additional consideration of the role of sympathetic and parasympathetic nervous systems activity in a sample of 6-12-year-olds. *Monographs of the Society for Research In Child Development*, 74, 44-55.
- El-Sheikh M., Kouros, C.D., Erath, S., Cummings, M.E., Keller, P., Staton, L., et al. (2009). Marital conflict and children's externalizing behavior: Interactions between parasympathetic and sympathetic nervous system activity: V. Discussion. *Monographs of the Society for Research In Child Development*, 74, 56-59.
- El-Sheikh, M., Erath, S.A., Buckhalt, J.A., Granger, D.A., & Mize, J. (2008). Cortisol and children's adjustment: The moderating role of sympathetic nervous system activity. *Journal of Abnormal Child Psychology*, 36, 601-611.
- Evans, G.W., Kim, P., Ting, A.H., Teshler, H.B., & Shannis, D. (2007). Cumulative risk, maternal responsiveness, and allostatic load among young adolescents. *Developmental Psychology*, 43, 341-351.
- Foa, E.B., Johnson, K.M., Feeney, N.C., & Treadwell, K.R.H. (2001). The Child PTSD Symptom Scale: A preliminary examination of its psychometric properties. *Journal of Clinical Child Psychology*, 30, 376-384.
- Fortunato, C.K., Dribin, A.E., Granger, D.A., & Buss, K.A. (2008). Salivary alpha-amylase and cortisol in toddlers: Differential relations to affective behavior. *Developmental Psychobiology*, 50, 807-818.
- Gevirtz, R. The physiology of stress. Kenny, Dianna T (Ed); Carlson, John G (Ed); McGuigan, F. Joseph (Ed); Sheppard, John L (Ed). (2000). *Stress and health: Research and clinical applications*. (pp. 53-71). xiii, 467 pp. Amsterdam, Netherlands: Harwood Academic Publishers.
- Goenjian A.K., Pynoos, R.S., Steinberg, A.M., Endres, D., Abraham, K., Geffner, M.E., et al. (2003). Hypothalamic-Pituitary-Adrenal activity among Armenian adolescents with PTSD symptoms. *Journal of Traumatic Stress*, 16, 319-323.

- Goenjian A.K., Yehuda R., Pynoos R.S., Steinberg A.M., Tashjian M., Yang R.K. et al. (1996). Basal cortisol, dexamethasone suppression of cortisol, and MHPG in adolescents after the 1988 earthquake in Armenia. *American Journal of Psychiatry*, *153*, 929-34.
- Gordis, E.B., Feres, N., Olezski, C.L., Rabkin, A.N., & Trickett, P.K. (2010). Skin conductance reactivity and respiratory sinus arrhythmia among maltreated and comparison youth: Relations with aggressive behavior. *Journal of Pediatric Psychology*, *35*, 547-558.
- Gordis, E.B., Granger, D.A., Susman, E.J., & Trickett, P.K. (2006). Asymmetry between salivary cortisol and alpha amylase reactivity to stress: Relation to aggressive behavior in adolescents. *Psychoneuroendocrinology*, *31*, 976-987.
- Gottman, J.M., Katz, L.F., & Hooven, C. (1997). *Meta-emotion: How families communicate emotionally*. Mahwah, NJ: Lawrence Erlbaum.
- Graham-Bermann, S. A., Castor, L. Miller, L. E., & Howell, K. H. (2012). The impact of additional traumatic events to trauma symptoms and PTSD in children exposed to intimate partner violence. *Journal of Traumatic Stress*, *25*, 393-400.
- Graham-Bermann, S.A., De Voe, E.R., Mattis, J.S., Lynch, S., & Thomas, S.A. (2006). Ecological Predictors of Traumatic Stress Symptoms in Caucasian and Ethnic Minority Children Exposed to Intimate Partner Violence. *Violence Against Women*, *12*, 663-692.
- Graham-Bermann, S.A., & Levendosky, A.A. (1998) Traumatic stress symptoms in children of battered women. *Journal of Interpersonal Violence*, *13*, 111-128.
- Graham-Bermann, S. A. & Levendosky, A. A. (1998). The social functioning of preschool-age children whose mothers are emotionally and physically abused. *Journal of Emotional Abuse*, *1*, 59-84.
- Granger, D.A., Kivlighan, K.T., Blair, C., El-Sheikh, M., Mize, J., Lisonbee, J.A. et al. (2006). Integrating the measurement of salivary alpha-amylase into studies of child health, development, and social relationships. *Journal of Social and Personal Relationships*, *23*, 267-290.
- Griffin, M.G., Resick, P.A., & Mechanic, M.B. (1997). Objective assessment of peritraumatic dissociation: Psychophysiological indicators. *American Journal of Psychiatry*, *154*, 1081-1088.
- Gunnar, M.R., & Davis, E.P. (2003). Stress and emotion in early childhood. Lerner, Richard M (Ed); Easterbrooks, M. Ann (Ed); Mistry, Jayanthi (Ed). *Handbook of psychology: Developmental psychology*, Vol. 6. (pp. 113-134). xxi, 666 pp. Hoboken, NJ, US: John Wiley & Sons Inc.

- Gunnar, M. R., Frenn, K., Wewerka, S. S., & Van Ryzin, M. J. (2009). Moderate versus severe early life stress: Associations with stress reactivity and regulation in 10- to 12-year old children. *Psychoneuroendocrinology*, *34*, 62-75.
- Gunnar, M.R., Morison, S.J., Chisholm, K., & Schuder, M. (2001). Salivary cortisol levels in children adopted from Romanian orphanages. *Development and Psychopathology*, *13*, 611-628.
- Gunnar M.R., & Quevedo K.(2007).The neurobiology of stress and development. *Annual Review of Psychology*, *58*, 145-73.
- Gunnar, M.R., Talge, N.M., & Herrera, A. (2009). Stressor paradigms in developmental studies: What does and does not work to produce mean increases in salivary cortisol. *Psychoneuroendocrinology*, *34*, 953-967.
- Gunnar, M.R., & Vazquez, D. (2006). Stress Neurobiology and developmental psychopathology. In D. Cicchetti & D.J. Cohen (Eds.), *Developmental Psychopathology: Developmental Neuroscience* vol. 2 (pp. 533-577). New York: Wiley.
- Guyton, A.C. & Hall, J.E. (1995). *Textbook of Medical Physiology*. WB Saunders Co: Philadelphia, PA
- Habib, K.E., Gold, P.W., & Chrousos, G.P. (2001). Neuroendocrinology of stress. *Endocrinology Metabolism Clinics of North America*, *30*, 695-728.
- Hamby, S., Finkelhor, D., Turner, H., & Ormrod, R. (2011). Children's exposure to intimate partner violence and other family violence (pgs. 1-12). *Juvenile Justice Bulletin—NCJ232272*. Washington, DC: US. Government Printing Office.
- Hammen, C., Burge, D., Burney, E., & Adrian, C. (1990). Longitudinal study of diagnoses in children of women with unipolar and bipolar affective disorder. *Archives of General Psychiatry*, *47*, 1112-1117.
- Henry, J.P. (1992). Biological basis of the stress response. *Integrative Physiological and Behavioral Science*, *27*, 66-83.
- Hibel, L.C., Granger, D.A., Blair, C., & Cox, M.J. (2011). Maternal sensitivity buffers the adrenocortical implications of intimate partner violence exposure during early childhood. *Development and Psychopathology*, *23*, 689-701.
- Hinnant, & El-Sheikh, M. (2009). Children's externalizing and internalizing symptoms over time: The role of individual differences in patterns of RSA responding. *Journal of Abnormal Child Psychology*, *37*, 1049-1061.
- Irwin, J., Ahluwalia, P., & Anisman, Y. (1986). Sensitization of norepinephrine activity

- following acute and chronic footshock. *Brain Research*, 379, 98-103.
- Jaffe, P. G., Wolfe, D. A., Wilson, S., & Zak, L. (1986). Family violence and child adjustment: A comparative analysis of girls' and boys' behavioral symptoms. *American Journal of Psychiatry*, 143, 74-76.
- Jones-Alexander, J., Blanchard, E.B., & Hickling, E.J. (2005). Psychophysiological Assessment of Youthful Motor Vehicle Accident Survivors. *Applied Psychophysiology and Biofeedback*, 30, 115-123.
- Kassam-Adams N., Garcia-España J.F., Fein J.A., & Winston F.K. (2005). Heart rate and posttraumatic stress in injured children. *Archives of General Psychiatry*, 62, 335-40.
- Katz, L.F. & Gottman, J.M. (1995). Vagal tone protects children from marital conflict. *Development and Psychopathology*, 7, 83-92.
- Katz, L.F., Hessler, D., & Annet, A. (2007). Domestic violence, children's emotional competence and peer relations. *Social Development*, 16, 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, 614-621.
- Keller, P.S. & El-Sheikh, M. (2009). Salivary alpha-amylase as a longitudinal predictor of children's externalizing symptoms: Respiratory sinus arrhythmia as a moderator of effects. *Psychoneuroendocrinology*, 34, 633-643.
- Keller, P.S., El-Sheikh, M., Granger, D.A., & Buckhalt, J.A. (2012). Interactions between salivary cortisol and alpha-amylase as predictors of children's cognitive functioning and academic performance. *Physiology & Behavior*, 105, 987-995.
- Kilpatrick, K.L., & Williams, L.M. (1997). Post-traumatic stress disorder in child witnesses to domestic violence. *American Journal of Orthopsychiatry*, 67, 639-644.
- King, J.A., Mandansky, D., King, S., Fletcher, K., & Brewer, J. (2001). Early sexual abuse and low cortisol. *Psychiatry and Clinical Neurosciences*, 55, 71-74.
- Kovacs, M. (1992). *The Children's Depression Inventory*. North Towawanda, NY: Multi-health Systems.
- Langeland W., & Olf, M. (2008). Psychobiology of posttraumatic stress disorder in pediatric injury patients: a review of the literature. *Neuroscience & Biobehavioral Reviews*, 32, 161-74.
- Leary, A. & Katz, L.F. (2004). Family level processes and peer outcomes: The moderating role of vagal tone. *Development & Psychopathology*, 16, 593-608.

- Lehmann, P. (1997). The development of posttraumatic stress disorder (PTSD) in a sample of child witnesses to mother assault. *Journal of Family Violence, 12*, 241-257.
- Levendosky, A.A., Huth-Bocks, A.C., Semel, M.A., & Shapiro, D.L. (2002). Trauma symptoms in preschool-age children exposed to domestic violence. *Journal of Interpersonal Violence, 17*, 150-164.
- Lisonbee, J.A., Pendry, P., Mize, J., & Gwynn, E.P. (2010). Hypothalamic-pituitary-adrenal and sympathetic nervous system activity and children's behavioral regulation. *Mind, Brain, and Education, 4*, 171-181.
- Lovallo, W.R. & Thomas, T.L. (2000). Stress hormones in psychophysiological research: Emotional, behavioral, and cognitive implications. In J.T. Cacioppo, L.G. Tassinary, & G.G. Bernston (Eds.), *Handbook of psychophysiology 2nd ed.* (pp. 342-367). New York, NY, US: Cambridge University Press.
- Luecken, L.J., Hagan, M.J., Sandler, I.N., Tein, J.Y., Ayers, T.S., & Wolchik, S.A. (2010). Cortisol levels six-years after participation in the family bereavement program. *Psychoneuroendocrinology, 35*, 785-789.
- Margolin, G., & Vickerman, K.A. (2007). Posttraumatic stress in children and adolescents exposed to family violence: I. Overview and issues. *Professional Psychology: Research and Practice, 38*, 613-619.
- Marin, T.J., Martin, T.M., Blackwell, E., Stetler, C., & Miller, G.E. (2007). Differentiating the impact of episodic and chronic stressors on hypothalamic-pituitary-adrenocortical axis regulation in young women. *Health Psychology, 26*, 447-455.
- Matthews, K.A., Gump, B.B., & Owens, J.F. (2001). Chronic stress influences cardiovascular and neuroendocrine responses during acute stress and recovery, especially in men. *Health Psychology, 20*, 403-410.
- McCloskey, L.A., & Lichter, E. (2003). Childhood exposure to marital violence and adolescent aggression: Psychological mediators in the cycle of violence. *Journal of Interpersonal Violence, 18*, 1-23.
- McCloskey, L.A., & Walker, M. (2000). Posttraumatic stress in children exposed to family violence and single-event trauma. *Journal of the American Academy of Child & Adolescent Psychiatry, 39*, 108-115.
- 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*, 137-142.
- McTeague, L.M. & Lang, P.J. (2012). The anxiety spectrum and the reflex physiology of

- defense: From circumscribed fear to broad distress. *Depression and Anxiety*, 29, 264-281.
- Medina, A. M., Mejia, V. M., Schell, A. M., Dawson, M. E., & Margolin, G. (2001). Startle reactivity and PTSD symptoms in a community sample of women. *Psychiatry Research*, 101, 157-169.
- Mertin, P., & Mohr, P.B. (2002). Incidence and correlates of posttrauma symptoms in children from backgrounds of domestic violence. *Violence and Victims*, 17, 555-567.
- Milner, J.S. (1986) *The Child Abuse Potential Inventory: Manual* (2nd ed.), Psytec. Inc, DeKalb, IL.
- Modrowski, C., Miller, L. E., Howell, K. H., & Graham-Bermann, S. A. (2012). Consistency of trauma symptoms at home and in therapy for preschool children exposed to intimate partner violence. *Psychological Trauma: Theory, Research, Practice, and Policy*.
- Mokuda, O., Sakamoto, Y., Kawagoe, R., Ubukata, E., & Shimizu, N. (1992). Epinephrine augments cortisol secretion from isolated perfused adrenal glands of guinea pigs. *American Journal of Physiology*, 262, E806-809.
- Munro, C., & Stabenfelt, G. (1985). Development of a cortisol enzyme immunoassay in plasma. *Clinical Chemistry*, 31, 956.
- Nakamura, Y., Gang, H.X., Suzuki, T., Sasano, H., Rainey, W.E. (2009). Adrenal changes associated with adrenarche. *Reviews in Endocrine & Metabolic Disorders*, 10, 19-26.
- Nater, U.M., La Marca, R., Florin, L., Moses, A., Langhans, W., Koller, M.M., et al. (2006). Stress-induced changes in human salivary alpha-amylase activity-associations with adrenergic activity. *Psychoneuroendocrinology*, 31, 49-58.
- Nater, U.M., and Rohleder, N. (2009). Salivary alpha amylase as a non-invasive biomarker for the sympathetic nervous system: Current state of research. *Psychoneuroendocrinology*, 34, 486-496.
- Nater, U.M., Rohleder, N., Scholtz, W., Ehlert, U., & Kirschbaum, C. (2007). Determinants of the diurnal course of salivary alpha-amylase. *Psychoneuroendocrinology*, 32, 392-401.
- Nicolson, N.A. (2007). Measurement of Cortisol. In L.J. Luecken & L.C. Gallo (Eds.), *Handbook of Physiological Research Methods in Health Psychology* (pp. 37-73). Sage Publications, Inc.
- O'Donnell, M.D. & Miller, N.J. (1980). Plasma pancreatic and salivary-type amylase and

- immunoreactive trypsin concentrations: Variations with age and reference ranges for children. *Clinica Chimica Acta*, 104, 265-273.
- Ostrowski S.A., Christopher N.C., van Dulmen M.H., & Delahanty D.L. (2007). Acute child and mother psychophysiological responses and subsequent PTSD symptoms following a child's traumatic event. *Journal of Traumatic Stress*, 20, 677-87.
- Owens, M.J., & Nemeroff, C.B. (1991). Physiology and pharmacology of corticotropin-releasing factor. *Pharmacological Reviews*, 43, 425-473.
- Perkins, S. & Graham-Bermann, S.A. (2006). Contribution of age of first exposure to intimate partner violence on child adjustment. Presentation given at the 22nd Annual Meeting of the International Society for Traumatic Stress Studies, Hollywood, CA, November, 2006
- Pervanidou, P. (2008). Biology of post-traumatic stress disorder in childhood and adolescence. *Journal of Neuroendocrinology*, 20, 632-638.
- Pervanidou, P., Kolaitis, G., Charitaki, S., Margeli, A., Ferentinos, S., Bakoula, C. et al. (2007). Elevated morning serum interleukin (IL)-6 or evening salivary cortisol concentrations predict posttraumatic stress disorder in children and adolescents six months after a motor vehicle accident. *Psychoneuroendocrinology*, 32, 991-999.
- Pfeffer, C.R., Altemus, M., Heo, M., & Jiang, H. (2007). Salivary cortisol and psychopathology in children bereaved by the September 11, 2001 terror attacks. *Biological Psychiatry*, 61, 957-965.
- Pitman, R.K. (1989). Editorial: Post-traumatic stress disorder, hormones, and memory. *Biological Psychiatry*, 26, 221-223.
- Porges, S.W., Doussard-Roosevelt, J.A., & Maita, A.K. (1994). Vagal tone and the physiological regulation of emotion. *Monographs for the Society for Research in Child Development*, 59, 167-186.
- Putnam, F.W., Helmers, K., & Trickett, P.K. (1993). Development, reliability, and validity of a child dissociation scale. *Child Abuse & Neglect*, 17, 731-741.
- Quas, J.A., Yim, I.S., Rush, E., & Sumaroka, M. (2012). Hypothalamic pituitary adrenal axis and sympathetic activation: Joint predictors of memory in children, adolescents, and adults. *Biological Psychology*, 89, 335-341.
- Radloff, L.S. (1977). The CES-D Scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement*, 1, 385-401.
- Raine, A. (2005). The interaction of biological and social measures in the explanation of antisocial and violent behavior. In D.M. Stoff & E.J. Susman (Eds.), *Developmental*

- psychobiology of aggression* (pp. 13-42). New York: Cambridge University Press.
- Robin, A.L., & Foster, S.L. (1980). *Negotiating Parent-Adolescent Conflict: A Behavioral-Family Systems Approach*. New York, NY: Guilford Press
- Rohleder, N., Wolf, J.M., Maldonado, E.F., and Kirschbaum, C. (2006). The psychosocial stress-induced increase in salivary alpha amylase is independent of saliva flow rate. *Psychophysiology*, *43*, 645-652.
- Rotenberg, S., McGrath, J.J., Roy-Gagnon, M.H., Tu, M.T. (2012). Stability of the diurnal cortisol profile in children and adolescents. *Psychoneuroendocrinology*, *37*, 1981-1989.
- Rotton, J., Dubitsky, S.S., Milov, A., White, S/M; et al. (1997). Distress, elevated cortisol, cognitive deficits, and illness following a natural disaster. *Journal of Environmental Psychology*, *17*, 85-98.
- Ruggiero, K.J., & McLeer, S.V. (2000). PTSD scale of the Child Behavior Checklist: Concurrent and discriminant validity with non-clinic-referred sexually abused children. *Journal of Traumatic Stress*, *13*, 287-299.
- Salimetrics, LLC, State College, PA
- 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*, 129-139.
- Sapolsky, R.M. (2002). Endocrinology of the stress-response. Becker, Jill B (Ed); Breedlove, S. Marc (Ed); Crews, David (Ed); McCarthy, Margaret M (Ed). Behavioral endocrinology (2nd ed.). (pp. 409-450). xxx, 776 pp. Cambridge, MA, US: MIT Press.
- Sapolsky R.M. (2000). Stress hormones: good and bad. *Neurobiological Disease*, *7*, 540-542.
- Sapolsky, R.M. (2004). *Why zebras don't get ulcers*. Third Edition. Henry Holy and Co: New York.
- Sapolsky, R.M., Romero, L.M., & Munck, A.U. (2000). How Do Glucocorticoids Influence Stress Responses? Integrating Permissive, Suppressive, Stimulatory, and Preparative Actions. *Endocrine Reviews*, *21*, 55-89.
- Saylor, C.F., Finch, A.J., Spirito, A., & Bennett, B. (1984). The Children's Depression Inventory: A systematic evaluation of psychometric properties. *Journal of Consulting and Clinical Psychology*, *52*, 955-967.

- Schnurr, P.P., Friedman, M.J., & Bernardy, N.C. (2002). Research on posttraumatic stress disorder: Epidemiology, pathophysiology, and assessment. *Journal of Clinical Psychology, 58*, 877-889.
- Seedat, S., Kaminer, D., Lockhat, R., & Stein, D.J. (2000). An overview of post-traumatic stress disorder in children and adolescents. *Primary Care Psychiatry, 6*, 43-48.
- Shenk, C.E., Noll, J.G., Putnam, F.W., & Trickett, P.K. (2010). A prospective examination of the role of childhood sexual abuse and physiological asymmetry in the development of psychopathology. *Child Abuse & Neglect, 34*, 752-761.
- Shirtcliff, E.A., Allison, A.L., Armstrong, J.M., Slattery, M.J., Kalin, N.H., & Essex, M.J. (2012). Longitudinal stability and developmental properties of salivary cortisol levels and circadian rhythms from childhood to adolescence. *Developmental Psychobiology, 54*, 493-502.
- Spinrad, T.L., Eisenberg, N., Granger, D.A., Eggum, N.D., Sallquist, J., Haugen, R.G. et al. (2009). Individual differences in preschoolers' salivary cortisol and alpha amylase reactivity: Relations to temperament and maladjustment. *Hormones and Behavior, 56*, 133-139.
- Stansbury, K., & Gunnar, M.R. (1994). Adrenocortical activity and emotion regulation. *Monographs of the Society for Research in Child Development., 59*, 108-134, 250-283.
- Stratakis C.A., & Chrousos G.P.(1995). Neuroendocrinology and pathophysiology of the stress system. *Annals of the New York Academy of Sciences, 771*, 1-18.
- Straus, M.A. (1979). Measuring Intrafamily Conflict and Violence: The conflict tactics (CT) scales. *Journal of Marriage and the Family, 41*, 75-88.
- Stroud, L.R., Foster, E., Papandonatos, G.D., Handwerker, K., Granger, D.A., Kivlighan, K.T. et al. (2009). Stress response and the adolescent transition: Performance versus peer rejection stressors. *Development and Psychopathology, 21*, 47-68.
- Sturge-Apple, M.L., Davies, P.T., Cicchetti, D., & Manning, L.G. (2012). Interparental violence, maternal emotional unavailability and children's cortisol reactivity to family contexts. *Developmental Psychology, 48*, 237-249.
- Suliman, S., Mkabile, S.G., Fincham, D.S., Ahmed, R., Stein, D.J., & Seedat, S. (2009). Cumulative effect of multiple trauma on symptoms of posttraumatic stress disorder, anxiety, and depression in adolescents. *Comprehensive Psychiatry, 50*, 121-127.
- Terr, L.C. (1991). Childhood traumas: An outline and overview. *American Journal of Psychiatry, 148*, 10-20.

- Thoma, M.V., Joksimovic, L.L., Kirschbaum, C., Wolf, J.M., & Rohleder, N. (2012). Altered salivary alpha-amylase awakening response in Bosnian War refugees with posttraumatic stress disorder. *Psychoneuroendocrinology*, *37*, 810-817.
- Thoma, M.V., Kirschbaum, C., Wolf, J.M., & Rohleder, N. (2012). Acute stress responses in salivary alpha amylase predict increases of plasma norepinephrine. *Biological Psychology*, *91*, 342-348.
- Thompson, T., & Massat, C.R. (2005). Experiences of violence, post-traumatic stress academic achievement and behavior problems of urban African-American children. *Child & Adolescent Social Work Journal*, *22*, 367-393.
- Tsigos, C., & Chrousos, G.P. (2002). Hypothalamic-pituitary-adrenal axis, neuroendocrine factors and stress. *Journal of Psychosomatic Research*, *53*, 865-871.
- Tyrka, A.R., Kelly, M.M., Graber, 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*, 1167-1177.
- Wolf, J.M., Nicholls, E., & Chen, E. (2008). Chronic stress, salivary cortisol, and alpha – amylase in children with asthma and healthy children. *Biological Psychology*, *78*, 20-28.
- Wolfe, V.V., Gentile, C., & Wolfe, D.A. (1989). The impact of sexual abuse on children: A PTSD formulation. *Behavior Therapy*, *20*, 215-228.
- Yehuda, R. (2002). Neuroendocrine alterations in posttraumatic stress disorder. *Primary Psychiatry*, *9*, 30-34.
- Yehuda, R., McFarlane, A.C., & Shalev, A.Y. (1998). Predicting the development of posttraumatic stress disorder from the acute response to a traumatic event. *Biological Psychiatry*, *44*, 1305-1313.
- Yim, I.S., Granger, D.A., & Quas, J.A. (2010). Children's and adults' salivary alpha-amylase responses to a laboratory stressor and to verbal recall of the stressor. *Developmental Psychobiology*, *52*, 598-602.

Table 1.
Descriptive Statistics for Primary Variables.

	Mean (Standard Deviation)
Time of 1 st sample	14:45 (2:11)
Maltreatment	233.35 (122.47)
IPV	29.94 (31.24)
AA Baseline 1	29.03 (20.96)
AA Baseline 2	43.34 (30.92)
AA Baseline 3	43.28 (32.41)
AA Baseline 4	45.83 (58.28)
AA Δ Baseline 1	14.25 (27.47)
AA Δ Baseline 2	-.06 (27.73)
Cortisol Baseline 1	1262.98 (804.26)
Cortisol Baseline 2	1012.77 (647.38)
Cortisol Baseline 3	936.23 (543.34)
Cortisol Baseline 4	881.20 (549.28)
Cortisol Δ Baseline 1	-381.76 (850.76)
Cortisol Δ Baseline 2	-131.57 (558.16)
CBCL PTSD	7.61 (4.18)
CDC Total	4.58 (4.54)
TSSIC Total PTSD	3.52 (4.03)
TSSIC Avoid	1.30 (1.69)
TSSIC Intrusive	.97 (1.40)
TSSIC Arousal	1.12 (1.47)
CPSS Total Sx Severity	13.12 (6.85)
CPSS Functional Impairment	2.47 (2.37)

Note. AA = Alpha Amylase; AA Δ Baseline 1 = AA Reactivity using Baseline 1 (Sample #3 - Sample #1); AA Δ Baseline 2 = AA Reactivity using Baseline 2 (Sample #3 - Sample #2); Cortisol Δ Baseline 1 = Cortisol Reactivity using Baseline 1 (Sample #4 - Sample #1), Cortisol Δ Baseline 2 = Cortisol Reactivity using Baseline 2 (Sample #4 - Sample #1)

Table 2. *Correlations Among Primary Variables.** $p < .05$, ** $p < .01$

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.	21.	22.	23.	
1. Time of 1 st sample	--																							
2. Maltreatment	-.11	--																						
3. IPV	-.02	.38	--																					
4. AA Baseline 1	.02	-.09	-.27	--																				
5. AA Baseline 2	-.14	-.14	-.21	.64**	--																			
6. AA Baseline 3	-.12	-.32	-.19	.54**	.67**	--																		
7. AA Baseline 4	-.28	.15	-.08	.61**	.46*	.64**	--																	
8. AA Δ Baseline 1	-.15	-.28	-.01	-.13	.30	.77**	.29	--																
9. AA Δ Baseline 2	.02	-.21	.01	-.09	-.36*	.45*	.26	.61**	--															
10. Cortisol Baseline 1	-.00	.12	-.19	.04	.12	.10	.18	.09	-.02	--														
11. Cortisol Baseline 2	.01	.11	-.11	.13	.40*	.16	.23	.09	-.28	.66**	--													
12. Cortisol Baseline 3	-.35*	.14	.25	-.22	.08	.19	.00	.39*	.14	.26	.46**	--												
13. Cortisol Baseline 4	-.36*	.20	.37*	-.17	.14	.14	.11	.29	.01	.25	.58**	.87**	--											
14. Cortisol Δ Baseline 1	-.24	.02	.41*	-.14	-.02	-.01	-.10	.10	.02	-.78**	-.25	.32	.41*	--										
15. Cortisol Δ Baseline 2	-.36*	.14	.48**	-.32	-.32	-.05	-.15	.18	.33	-.52**	-.59**	.32	.32	.69**	--									
16. CBCL PTSD	.22	.24	-.14	-.04	-.12	-.28	-.19	-.31	-.21	.08	-.05	-.11	-.10	-.14	-.04	--								
17. CDC Total	.41*	.48*	-.14	-.11	-.20	-.34	-.11	-.32	-.19	.20	.13	-.13	-.13	-.28	-.28	.69**	--							
18. TSSIC Total PTSD	.16	-.11	-.24	.05	-.01	-.14	-.17	-.21	-.18	.05	.08	-.11	-.14	-.14	-.23	.32	.21	--						
19. TSSIC Avoid	.09	-.27	-.26	.20	.07	-.03	-.13	-.19	-.13	.01	.01	-.20	-.21	-.15	-.22	.36*	.23	.87**	--					
20. TSSIC Intrusive	.10	.07	-.15	-.13	-.11	-.24	-.18	-.19	-.19	.02	.12	.00	.03	-.00	-.12	.24	.15	.81**	.56**	--				
21. TSSIC Arousal	.22	-.06	-.20	-.03	-.00	-.08	-.13	-.07	-.10	.17	.11	-.07	-.15	-.26	-.27	.11	.14	.82**	.58**	.52**	--			
22. CPSS Total Sx Severity	-.08	-.28	-.09	-.01	.07	.08	-.15	.10	.02	-.40	.01	.11	.30	.50*	.23	.22	-.08	.51*	.44	.54*	.35	--		
23. CPSS Functional Impairment	-.12	.27	.19	.34	.44	.45	.46	.29	.09	-.22	.21	.43	.38	.39	.08	.07	-.07	.32	.41	.27	.14	.48	--	

Table 3.

Hierarchical Regression of IPV and Baseline 2 Alpha Amylase on Children's CDC Dissociative Symptoms

Outcome	Step	Variable	B	T-Ratio	<u>Increment</u>		<u>Overall</u>	
					R ²	F	R ²	F
CDC	1	Time of Sample	.000	3.82*	.49	8.01*	.49	8.01*
		Abuse Status	.002	4.46*				
	2	IPV	-1.97	-2.75*	.06	2.04	.54	6.35*
	3	Alpha Amylase	-1.67	-2.48*	.00	.10	.55	4.52*
4	IPV x AA	1.09	2.46*	.14	6.04*	.57	6.04*	

Note. AA = alpha amylase, IPV = intimate partner violence.
 + $p < .10$; * $p < .05$

Table 4.

Hierarchical Regression of Baseline Alpha Amylase, Baseline Cortisol, and IPV on Children's TSSIC Total PTSD Symptoms

Outcome	Step	Variable	B	T-Ratio	<u>Increment</u>		<u>Overall</u>	
					R ²	F	R ²	F
TSSIC- Total PTSD	1	Time of Sample	.000	-2.63*	.02	.14	.02	.14
		Abuse Status	.000	-.51				
	2	IPV	-.30	-1.35	.08	.43	.10	.31
		AA	.000	-.14				
		Cortisol	.000	2.55*				
	3	IPV x AA	-.059	-4.22*	.48	4.14*	.58	1.88
		IPV x Cortisol	-.002	-3.56*				
		AA x Cortisol	.000	.51				
	4	IPV x AA x Cortisol	-.0001	-2.37*	.15	5.61*	.49	2.99+

Note. AA = alpha amylase, IPV = intimate partner violence.
+ $p < .10$; * $p < .05$

Table 5.

Hierarchical Regression of Baseline Alpha Amylase, Baseline Cortisol, and IPV on Children's TSSIC Avoidance PTSD Symptoms

Outcome	Step	Variable	B	T-Ratio	<u>Increment</u>		<u>Overall</u>	
					R ²	F	R ²	F
TSSIC-Avoidance	1	Time of Sample	.000	-3.76*	.07	.62	.07	.62
		Abuse Status	-.001	-1.81				
	2	IPV	-.23	-1.39	.08	.44	.15	.49
		AA	.000	-.01				
		Cortisol	.000	3.19*				
	3	IPV x AA	-.05	-4.59*	.33	2.35	.48	1.27
		IPV x Cortisol	-.002	-4.31*				
		AA x Cortisol	.000	.34				
	4	IPV x AA x Cortisol	-.0001	-3.45*	.28	11.93*	.76	3.57*

Note. AA = alpha amylase, IPV = intimate partner violence.
 + $p < .10$; * $p < .05$

Table 6.

Hierarchical Regression of Baseline Alpha Amylase, Baseline Cortisol, and IPV on Children's CBCL-PTSD Symptoms

Outcome	Step	Variable	B	T-Ratio	<u>Increment</u>		<u>Overall</u>	
					R ²	F	R ²	F
CBCL-PTSD	1	Time of Sample	.000	-1.49	.29	3.29+	.29	3.29+
		Abuse Status	.001	3.04*				
	2	IPV	-.051	-.26	.004	.024	.30	1.09
		AA	-.004	-2.36*				
		Cortisol	.000	4.13*				
	3	IPV x AA	-.029	-3.84*	.19	1.24	.49	1.18
		IPV x Cortisol	-.001	-1.99+				
		AA x Cortisol	.000	.007				
	4	IPV x AA x Cortisol	-.0001	-4.56*	.36	20.81*	.69	5.45*

Note. AA = alpha amylase, IPV = intimate partner violence.

+ $p < .10$; * $p < .05$

Table 7.

Hierarchical Regression of Alpha Amylase Reactivity, Cortisol Reactivity, and IPV on Children's CDC Dissociative Symptoms

Outcome	Step	Variable	B	T-Ratio	<u>Increment</u>		<u>Overall</u>	
					R ²	F	R ²	F
CDC	1	Time of Sample	.000	1.28	.49	8.01*	.49	8.01*
		Abuse Status	.002	2.59*				
	2	IPV	-.089	-.36	.06	.63	.55	3.38*
		AA	-.266	-.59				
		Cortisol	-.445	-1.52				
	3	IPV x AA	.133	.40	.01	.05	.55	1.70
		IPV x Cortisol	.314	1.64				
		AA x Cortisol	-1.135	-2.32*				
	4	IPV x AA x Cortisol	.707	2.28*	.15	5.20*	.44	2.66+

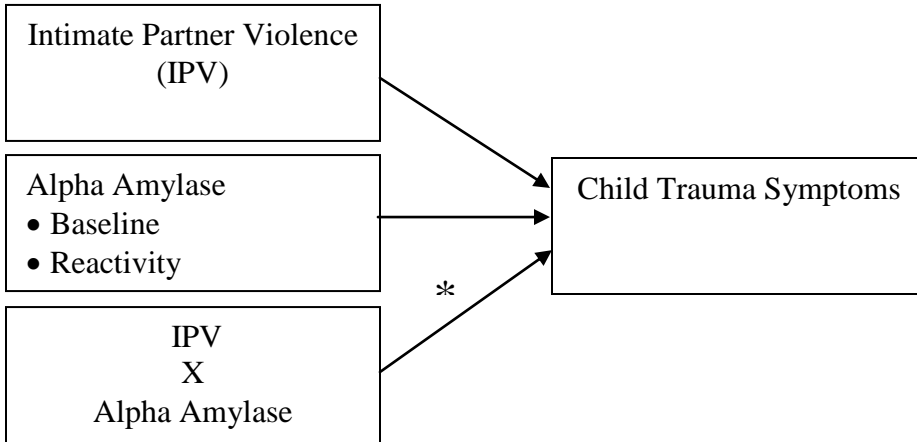
Note. AA = alpha amylase, IPV = intimate partner violence.

+ $p < .10$; * $p < .05$

Figure 1.

Study Models.

1a. Alpha Amylase and IPV Predicting Child Trauma Symptoms



1b. Cortisol and IPV Predicting Child Trauma Symptoms

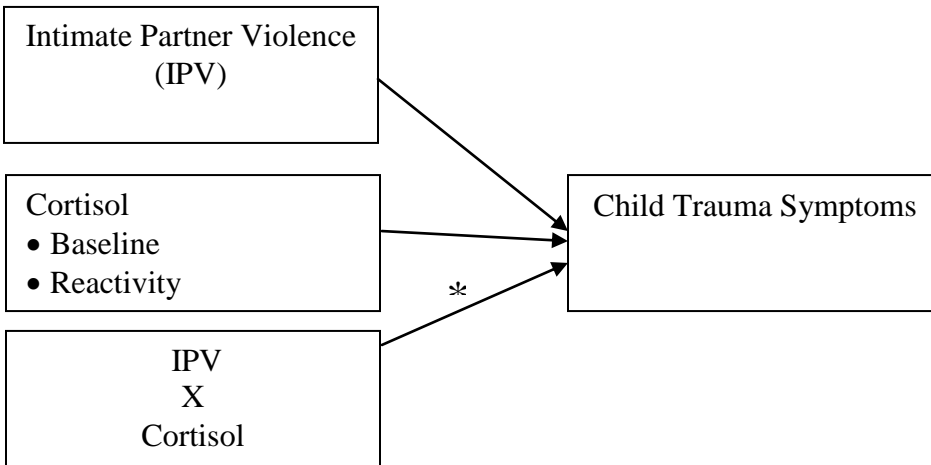
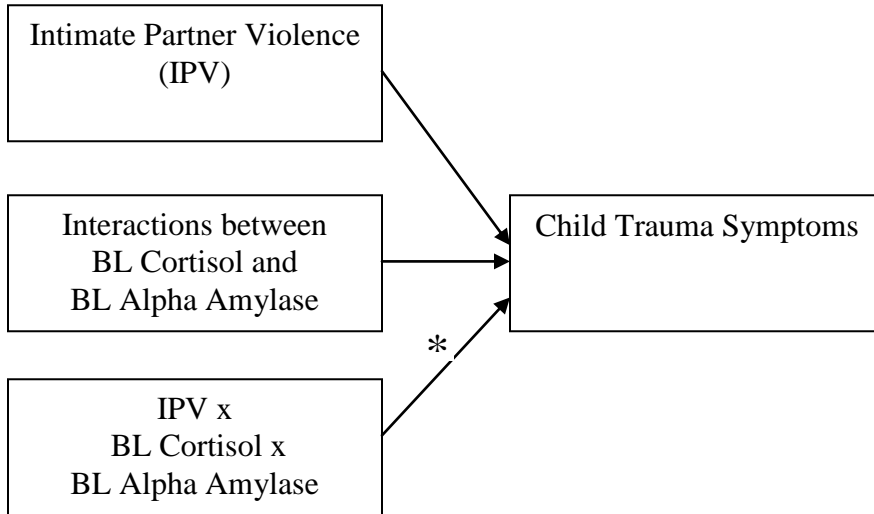


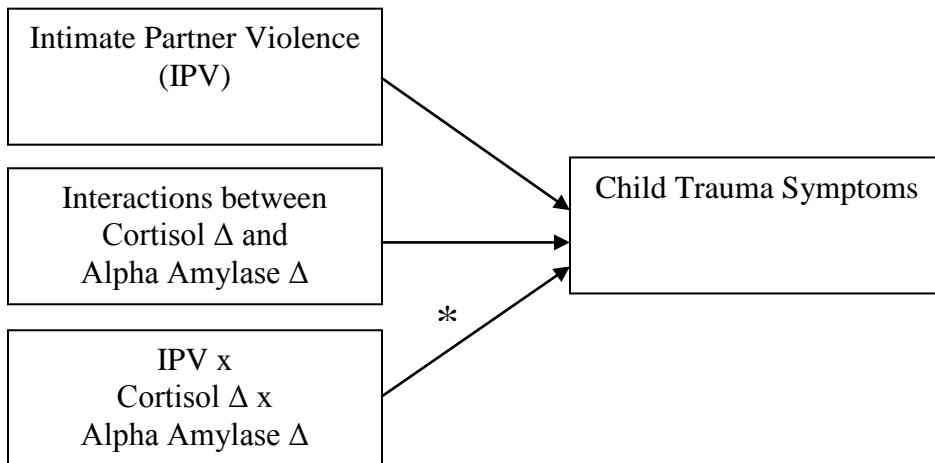
Figure 1 con't.

1c. *Intimate Partner Violence, Baseline Cortisol, and Baseline Alpha Amylase Predicting Child Trauma Symptoms*



Note. BL = Baseline

1d. *Intimate Partner Violence, Cortisol Reactivity and Alpha Amylase Reactivity Predicting Child Trauma Symptoms*



Note. Δ = Reactivity

Figure 2.

Children's Average Alpha Amylase and Cortisol Levels Across Sampling Times.

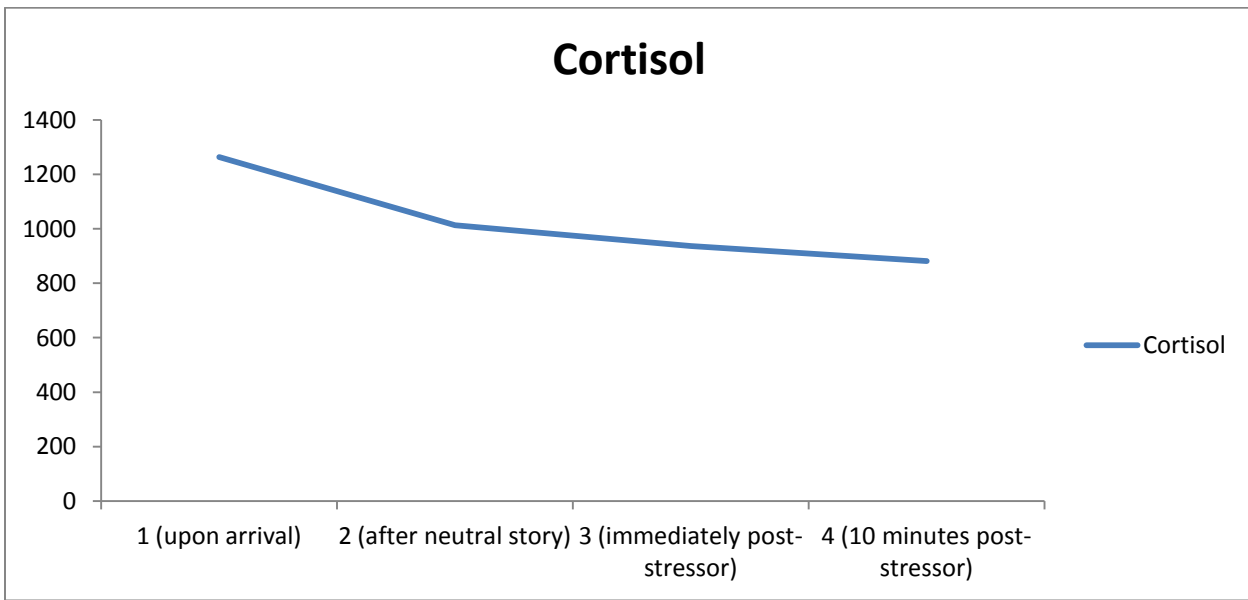
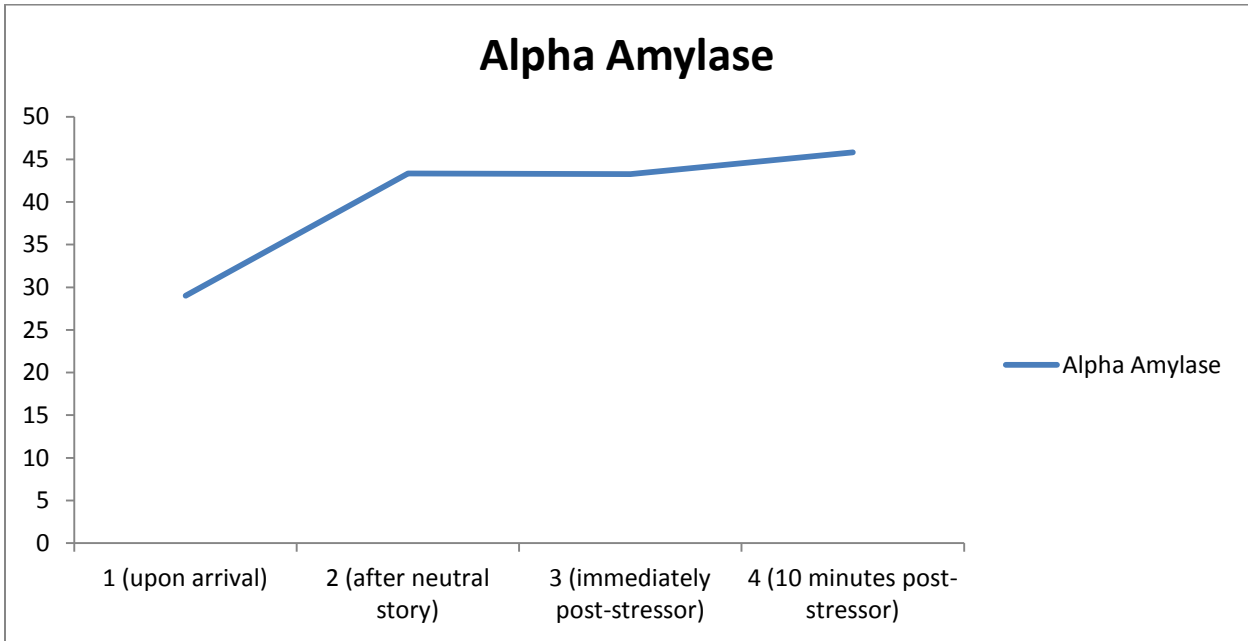


Figure 3.

Children's Individual Alpha Amylase Profiles.

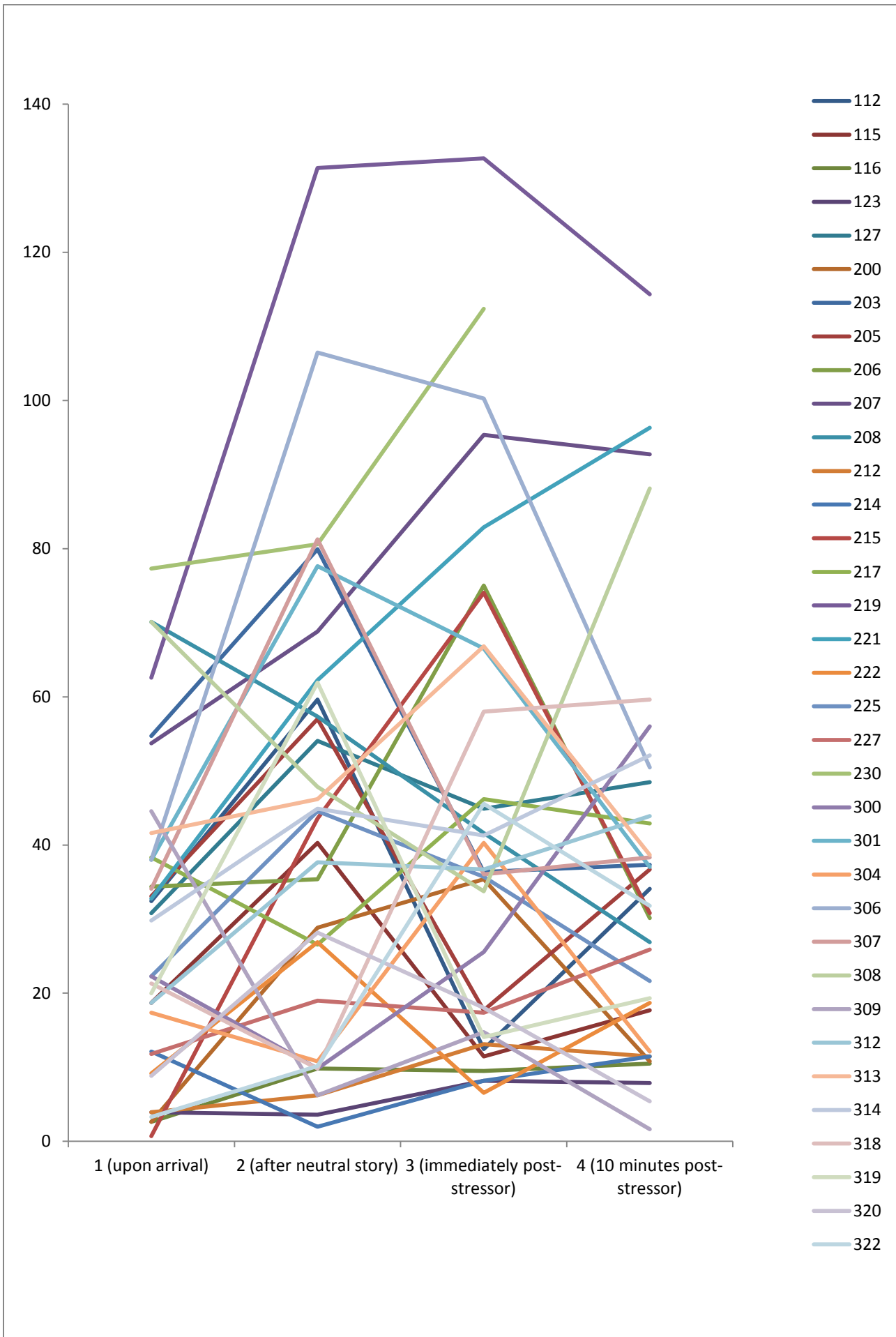


Figure 4.
Children's Individual Cortisol Profiles.

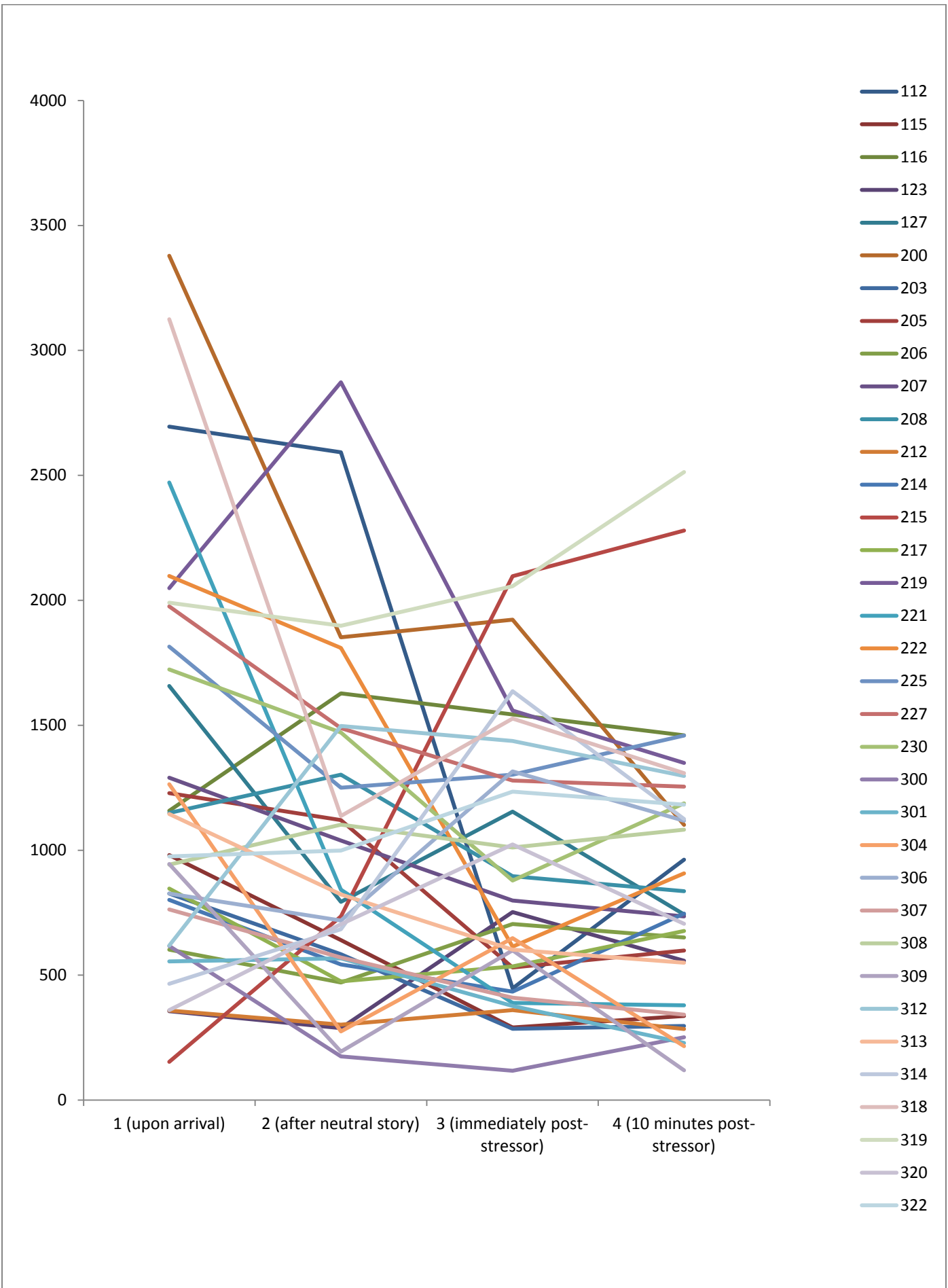


Figure 5.

Interaction between Baseline 2 Alpha Amylase and IPV in Predicting Children's Dissociative Trauma Symptoms

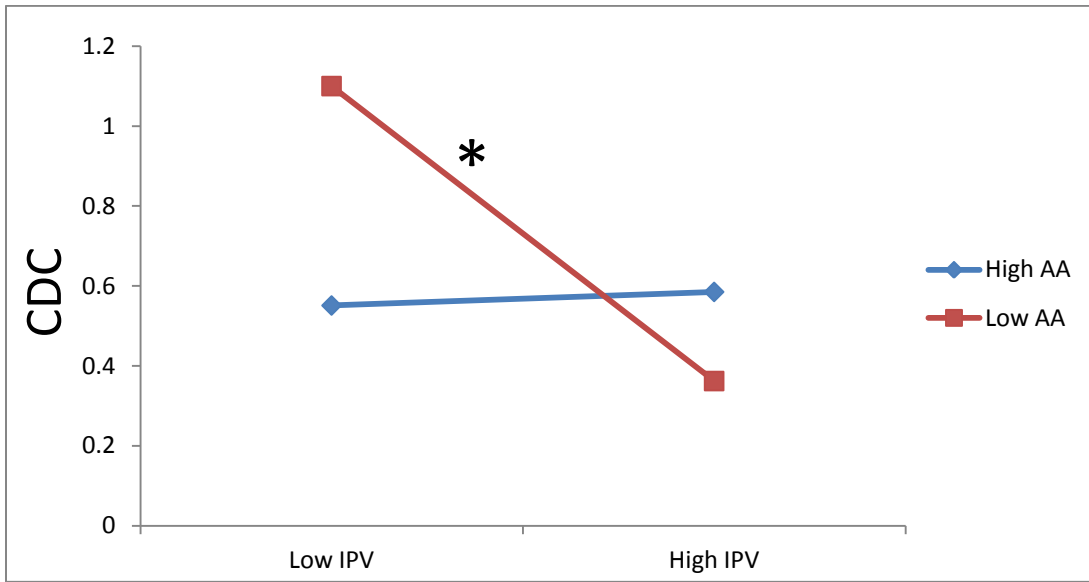


Figure 6.

3-way Interaction Between IPV, Baseline 1 Cortisol, and Baseline 1 Alpha Amylase in Predicting Children's TSSIC Total PTSD Symptoms.

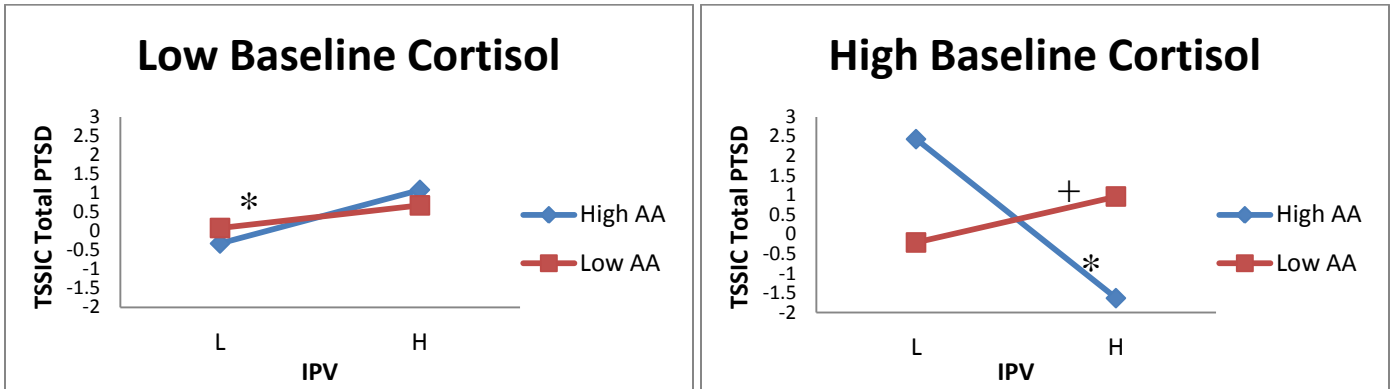


Figure 7.

3-way Interaction Between IPV, Baseline 1 Cortisol, and Baseline 1 Alpha Amylase in Predicting Children's TSSIC Avoidance Symptoms.

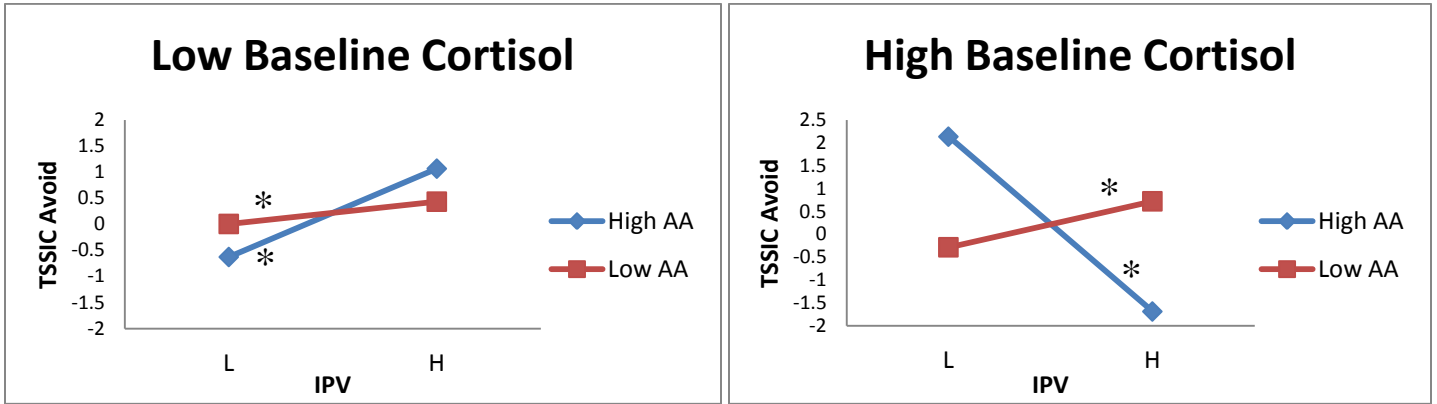


Figure 8.

3-way Interaction Between IPV, Baseline 1 Cortisol, and Baseline 1 Alpha Amylase in Predicting Children's CBCL-PTSD Symptoms.

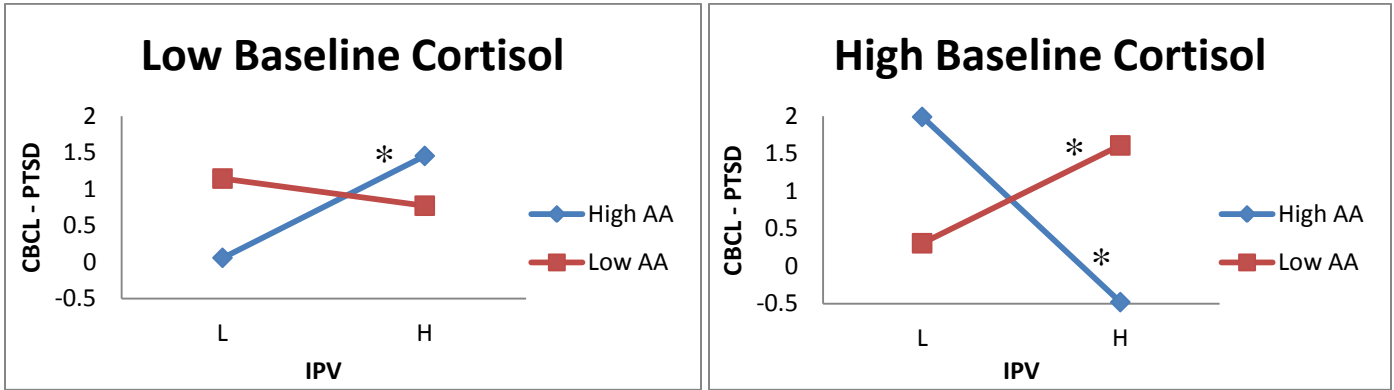
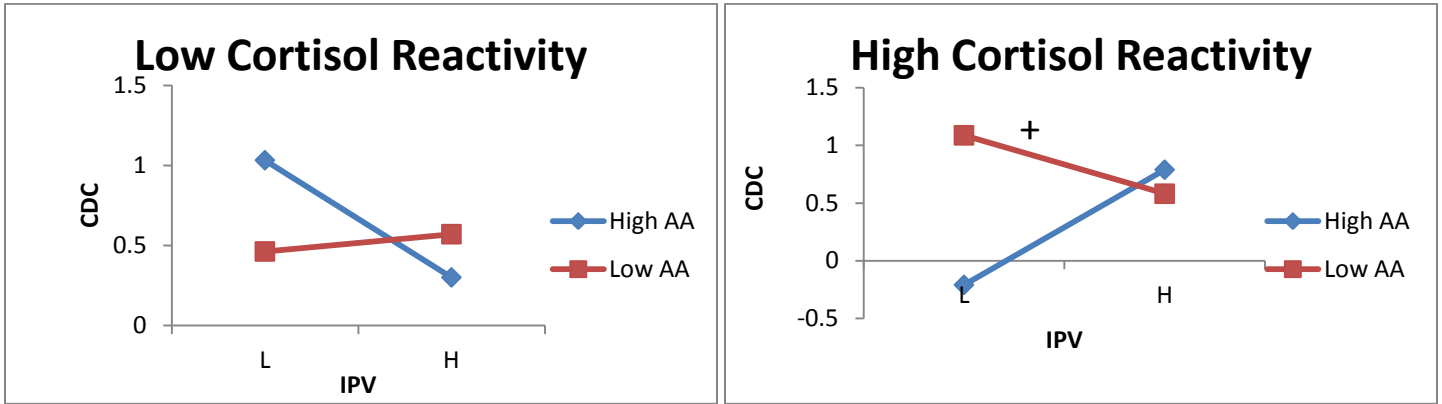


Figure 9.

3-way Interaction Between IPV, Cortisol Reactivity, and Alpha Amylase Reactivity in Predicting Children's Dissociative Trauma Symptoms.



VITA

Tami Rigterink received her Bachelor of Arts degree in Psychology from the University of Michigan and her Master's of Education degree with a focus on risk and prevention from Harvard University. She completed her Doctor of Philosophy at the University of Washington in Developmental Psychology in 2013. Her research interests center on how traumatic events impact children's psychological and physiological functioning.