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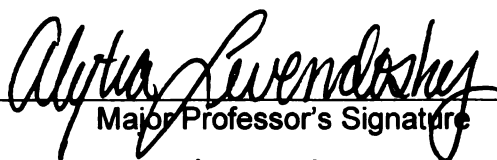
DIFFERENTIAL EFFECTS OF CLINICAL DIAGNOSES AND
TRAUMA CHARACTERISTICS ON CORTISOL OUTCOMES
IN VICTIMS OF INTIMATE PARTNER VIOLENCE

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ARCHANA BASU

has been accepted towards fulfillment
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Ph.D. degree in PSYCHOLOGY



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**DIFFERENTIAL EFFECTS OF CLINICAL DIAGNOSES AND TRAUMA
CHARACTERISTICS ON CORTISOL OUTCOMES IN VICTIMS OF INTIMATE
PARTNER VIOLENCE**

By

Archana Basu

A DISSERTATION

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

DOCTOR OF PHILOSOPHY

Psychology

2010

ABSTRACT

DIFFERENTIAL EFFECTS OF CLINICAL DIAGNOSES AND TRAUMA CHARACTERISTICS ON CORTISOL OUTCOMES IN VICTIMS OF INTIMATE PARTNER VIOLENCE

By

Archana Basu

The current study sought to understand how hypothalamic adrenal axis functioning, as assessed through salivary cortisol, may differ across psychological disorders that are commonly seen in trauma exposed populations. Another study goal was to address the lack of theoretical integration in the literature by examining how various theoretically posited trauma related characteristics (Herman, 1992) might account for variations in cortisol measures.

As intimate partner violence (IPV) tends to be a chronic form of trauma (Griffin, Resick, & Yehuda, 2005; Jones, Gross, & Becker, 2002) and female survivors of IPV have often experienced traumatic events in the early stages of their development as well (Herman, 1992), examining IPV victims can help to assess the role of various trauma related characteristics (i.e. early vulnerability factors such as childhood abuse, trauma chronicity, effects of cumulative types of trauma exposure, and role of social support). Thus, using a sample of adult premenopausal non pregnant female survivors of IPV, this study examined whether specific diagnostic groups [e.g., posttraumatic stress disorder (PTSD), PTSD and Major Depressive Disorder (MDD), and subthreshold symptoms of PTSD/ PTSD and MDD] would differ from each other, and from a control comparison group with no lifetime diagnosis of PTSD/ MDD or exposure to IPV, with respect to

cortisol measures. The study examined basal cortisol concentrations, diurnal rhythm of cortisol, and challenged cortisol measures in the context of a cognitive stress task.

This study did not find support for the effects of the aforementioned diagnostic groups or trauma related characteristics on the cortisol measures assessed in the study. Findings from the current study indicated that trauma exposed women had significantly higher levels of dissociative symptoms, which were inversely related to awakening cortisol concentrations. This finding is consistent with studies in the literature (Simeon, Yehuda, Knutelska, & Schmeidler, 2008) which suggest that various trauma related symptomatology (e.g., PTSD, dissociation) may present with different phenomenological and neurobiological correlates. As a well controlled study, which improved on multiple methodological limitations in the literature, these findings require further examination. Findings suggest that low awakening cortisol concentrations may be a marker for dissociation, which frequently co-occurs with PTSD, which in turn mediates the effects between trauma exposure and later development of PTSD.

DEDICATION

To my parents,
Shobharani Basu and Ashok Kumar Basu,
For loving me unconditionally,
For encouraging me to pursue my passion,
For supporting me against the currents of life,
For inspiring me by example.

ACKNOWLEDGMENTS

First and foremost I would like to acknowledge my dissertation chair, Dr. Alytia A. Levendosky, whose support and guidance were critical in bringing this study to its fruition. It has been my honor and privilege to work with someone as inspiring, dedicated, and loving as her. Alytia's mentorship and compassion have been an invaluable part of my professional and personal development and will continue to influence me well beyond my tenure as a graduate student.

I am deeply indebted to Dr. Joe Lonstein, who has been a role model in selfless mentorship and has influenced me greatly in the time that I worked with him. I thank him for patiently training me in conducting cortisol salivary assays and providing his scholarly mentorship and laboratory equipment in support of my study. This study would not have been possible without his generous support.

I would also like to thank Dr. Kathy E. Stansbury who consulted with me on the methodological and statistical aspects of collecting and analyzing cortisol data. She generously made herself available prior to starting her tenure at Michigan State University and while I was on internship. Finally, I would like to thank Dr. Brent Donnellan for his thoughtful comments on my dissertation proposal and his support of my research.

I would like to acknowledge the extramural and intramural sources of funding for this study - Grants in Aid Award, Society for the Psychological Study of Social Issues, The Graduate School, Michigan State University, and the Department of Psychology, Michigan State University. Additionally, I would like to acknowledge Ms. Stacie Dubay and Ms. Susan Shultz from EVE Inc., who allowed me to recruit participants through

EVE Inc. and use their facilities to complete interviews with women living in their shelter in a safe manner. I am very grateful to the undergraduate and graduate research assistants who assisted in data collection for the study. Also, while there are far too many people than I could possibly acknowledge who encouraged me throughout graduate school and through the course of this study, I would like to acknowledge some who were particularly instrumental in supporting me - my husband, Koushik Ganguly, and my friends, Dr. Janeen A. DeMarte, Dr. Mariam R. Mourad, and Dr. Carolyn J. Dayton. Finally, I would like to thank the women who participated in this study for sharing some of their most difficult personal experiences, whose stories of strength and survival continually inspire my research and clinical endeavors.

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CHAPTER 1

Introduction

Intimate partner violence (IPV: defined here as male physical or sexual violence, and threats of aggression against female partners) is a significant public health problem associated with negative mental health consequences. Battered women are at risk for developing a range of mental health problems, including posttraumatic stress disorder (PTSD), which refers to a constellation of symptoms typically seen in the aftermath of an extreme traumatic stressor (e.g., Cascardi & O'Leary, 1992; Kemp, Green, Hovanitz, & Rawlings, 1995; Kemp, Rawlings, & Green, 1991). Rates of PTSD among battered women vary between 45% and 81% based on sample characteristics, the definition of IPV used, and history of prior interpersonal trauma (Houskamp & Foy, 1991; Kemp et al., 1995; Sharhabani-Arzy, Amir, Kotler, & Liran, 2003; Stein & Kennedy, 2001). In addition, battered women are also at an increased risk for developing major depressive disorder (MDD) and other mood-related difficulties (Cascardi, Langhinrichsen, & Vivian, 1992; Cascardi & O'Leary, 1992; Coker, Watkins, Smith, & Brandt, 2003), a range of anxiety disorders (e.g., generalized anxiety disorder) (Coker et al., 2002; Kemp et al., 1991; Moore, Pepler, Mae, & Michele, 1989; Olson et al., 2003; Wolfe, Zak, Wilson, & Jaffe, 1986), dissociative symptoms (Dorahy, Lewis, & Wolfe, 2007), somatization (Frasier et al., 2004; Lown & Vega, 2001), and substance use related problems (Coker et al., 2002; Doyle, Frank, Saltzman, McMahon, & Fielding, 1999; Frasier et al., 2004; Golding, 1999; Kaysen et al., 2008; Sullivan & Holt, 2008; Weaver & Etzel, 2003).

Mental health consequences of trauma exposure, including IPV, are associated with dysfunctions in the neuroendocrine system including the hypothalamic-pituitary-

adrenal (HPA) axis, which regulates multiple bodily processes including responses to stress. One measure of alterations in the HPA axis is a corticosteroid called cortisol produced by the adrenal gland, which is involved in the stress response. Evidence for alterations in cortisol outcomes in women exposed to a variety of interpersonal and nonpersonal forms of traumas is mixed with studies reporting high (Lemieux & Coe, 1995; Maes et al., 1998; Rasmusson et al., 2001), low (Yehuda, 2002) and comparable levels of basal cortisol (i.e., diurnal or 24-hour cortisol concentrations) (Meewisse, Reitsma, de Vries, Gersons, & Olff, 2007) relative to control comparison groups. Similarly, studies examining challenged cortisol (i.e., changes in cortisol concentrations after exposure to a psychosocial or chemical challenge) that assess HPA axis reactivity also present mixed findings with evidence (Mason et al., 2002) for hyperresponsivity (e.g., Heim et al., 2000; Lindley, Carlson, & Benoit, 2004) and hyporesponsivity levels (e.g., Stein, Yehuda, Koverola, & Hanna, 1997; Yehuda, Golier, Halligan, Meaney, & Bierer, 2004). Finally, studies examining circadian rhythms of cortisol outcomes note alterations at specific times of the day, most consistently in the waking response of individuals with chronic trauma exposure, relative to control comparison groups (e.g., Johnson, Delahanty, & Pinna, 2008).

There are several methodological limitations in the extant literature examining cortisol outcomes in trauma exposed adults, including small sample sizes, the lack of appropriate control for diagnostic comorbidity for disorders that tend to co-occur but are associated with unique alterations in cortisol concentrations such as PTSD and MDD (Yehuda, 2002), appropriate controls for the effects of medications (e.g., contraceptives, anxiolytics), substance use, physical health problems such as autoimmune diseases that

may influence cortisol through their direct effects or the impact of the use of medications, and the timing, frequency, and type of cortisol sampling. In addition, while a handful of studies have assessed whether and how characteristics of trauma such as chronicity, age of trauma onset, severity, and latency of trauma are related to alterations in cortisol concentrations (Griffin et al., 2005; Johnson et al., 2008; Lemieux & Coe, 1995; Rasmusson et al., 2001; Yehuda, 2002; Yehuda, Halligan, & Grossman, 2001), few studies have assessed whether trauma characteristics versus diagnostic outcomes provide a better organizing framework to understand cortisol dysfunction and if there are any incremental advantages in combining these frameworks (Johnson et al., 2008; Miller, Chen, & Zhou, 2007; Pico-Alfonso, Garcia-Linares, Celda-Navarro, Herbert, & Martinez, 2004).

One important issue that has received considerable attention in the literature is the issue of comorbidity. Although PTSD and MDD are both common sequelae of exposure to traumatic events, including IPV, their associated basal cortisol levels are significantly different (Yehuda, Halligan, Golier, Grossman, & Bierer, 2004). For instance, while basal cortisol concentrations in PTSD tend to be low, those diagnosed with MDD tend to have high levels, compared to controls (Yehuda, 2002). However, these disorders are frequently comorbid and some studies suggest that individuals with comorbid diagnoses of PTSD and MDD may represent a subgroup whose neuroendocrine profile is distinct from those with either one of the diagnoses only or other diagnoses like acute stress disorder typically seen in the context of trauma exposure (Stein & Kennedy, 2001; Yehuda, 2002). Some researchers have suggested that these findings could be indicative of etiological differences in subtypes of MDD, and that one of the subtypes of depression

maybe more closely linked to the development of PTSD (e.g., Constans, Lenhoff, & McCarthy, 1997). These findings could also have implications for treatment outcome research in PTSD. For instance, both psychological and pharmacological treatment models can be informed by the development of an integrative and comprehensive model of PTSD that incorporates both neuroendocrine and behavioral components (Friedman, 2002; Rabois, Batten, & Keane, 2002). Similarly, to the extent that trauma characteristics such as chronicity and age of exposure add incrementally to this understanding, a developmental model of the stages of evolution of PTSD, particularly chronic trauma exposure is key (Friedman, 2002). Relatedly, it would also be important to examine whether the neuroendocrine and behavioral components would differ across comorbid conditions, across different stages of chronic PTSD. These findings have important methodological implications as well and a number of studies examining cortisol outcomes in trauma exposed populations fail to use appropriate comparison groups or control for comorbid symptoms resulting in heterogeneous findings of cortisol outcomes in PTSD populations.

Another important issue with the current research on the neurobiology of trauma is the lack of integration of a theoretical framework. Trauma theory provides a useful framework to examine the development of PTSD, particularly in the context of severe and chronic forms of trauma such as IPV (Herman, 1992). The theory posits mechanisms through which specific coping mechanisms adopted in the face of overwhelming stress eventually become dysfunctional and symptomatic of psychopathology. However, attempts to integrate findings from empirically tested hypotheses to inform theoretical understanding of the development of PTSD have been negligible (Miller et al., 2007).

Studies examining cortisol outcomes in adults typically do not examine theoretically derived hypotheses.

However, there is evidence to suggest that theoretically related trauma characteristics might play an important role. For instance, a recent meta-analysis of 107 studies examining cortisol outcomes in the context of different types of chronic stress (assault, abuse, bereavement, unemployment, combat-related war, natural disasters, and caregiving stress) found that a diagnosis of PTSD does not account for the effects of various trauma characteristics (e.g., timing) (Miller et al., 2007). Also, consistent with ideas posited by trauma theory that chronic PTSD has a specific course of development, researchers have suggested that the development of PTSD may involve different stages of evolution, each of which is likely associated with unique cortisol outcomes and neuroendocrine functioning, and behavioral and cognitive profiles (Friedman, 2002). Thus, findings related to neuroendocrine outcomes can inform theoretical development and treatment outcomes research in addition to building a comprehensive model of PTSD.

IPV is one form of trauma that tends to be chronic in nature (Griffin et al., 2005; Jones et al., 2002). In addition, individuals exposed to IPV have often experienced traumatic events in the early stages of their development as well, which indicates the role of early vulnerability factors in the outcomes observed in adulthood (e.g., Heim & Nemeroff, 2001; Seedat, Stein, & Forde, 2005). Thus, examining IPV victims can help to examine the differential impact of early vulnerability factors such as childhood neglect and abuse and later traumatic experiences of trauma such as IPV. Outside the realm of a prospective longitudinal study, such a population provides the opportunity to examine the

differential impact of chronicity and age of trauma exposure. Research examining the effects of chronicity and age of onset of trauma on cortisol outcomes has been limited but indicate that these are important characteristics of trauma exposure that need careful consideration because chronic exposure to trauma is related to lower levels of basal cortisol concentration (Heim et al., 2000; Resnick, Yehuda, Pitman, & Foy, 1995; Santa Ana et al., 2006; Yehuda, 2002; Yehuda, Halligan, & Bierer, 2002) relative to episodic trauma exposures (Lemieux & Coe, 1995; Maes et al., 1998; Rasmusson et al., 2001; Resnick et al., 1995). It is notable that among the studies that have examined the impact of trauma exposure at different developmental stages, the definition of a developmental stage is neither empirically supported nor theoretically driven (e.g., Heim et al., 2000; Santa Ana et al., 2006). Thus, the studies lack specificity about the mechanisms associated with the age of exposure but do suggest differences in neuroendocrine functioning depending on the age of trauma exposure and chronicity or the cumulative effects of trauma exposure over time. Finally, other trauma characteristics that have received limited attention include the relationship of the victim to the perpetrator in interpersonal forms of trauma (e.g., Ketring & Feinauer, 1999; Leahy et al., 2004; Lucenko et al., 2000) and the potential moderating impact of social support (e.g., Regehr, LeBlanc, Jelley, Barath, & Daciuk, 2007).

Despite the possibilities to examine multiple issues related to the development of PTSD in the context of chronic trauma, and the varied cortisol profiles found within adult populations with PTSD, only six studies to date have examined cortisol outcomes in battered women (Griffin et al., 2005; Inslicht et al., 2006; Johnson et al., 2008; Pico-Alfonso et al., 2004; Seedat, Stein, Kennedy, & Hauger, 2003; Young, Toman,

Witkowski, & Kaplan, 2004). Taken together they yield mixed findings with respect to cortisol outcomes and whether risk factors such as IPV or childhood abuse provide a better organizing framework than diagnostic classification.

With respect to studies of challenged cortisol, a number of studies have examined responses in traumatized samples in response to a chemical challenge i.e., a low dose dexamethasone test (DST) but only a handful of studies have examined these outcomes in the context of a psychosocial stressor in adults with trauma exposure (Carpenter et al., 2007; Elzinga et al., 2008; Heim et al., 2000; Santa Ana et al., 2006) and only, one study has examined challenged cortisol outcomes in IPV victims, using a low dose DST (Griffin et al., 2005). Till date no study has examined the effects of a cognitive psychosocial stressor on HPA reactivity in IPV victims. Examining challenged cortisol outcomes in IPV victims can help assess HPA reactivity in individuals exposed to chronic trauma beginning in early developmental stages, compared to those exposed to IPV in adulthood alone. This would help better understand HPA reactivity at different stages of the development of PTSD (Friedman, 2002).

The current study sought to address the aforementioned issues in the literature. First, the study seeks to use diagnoses, typically seen in trauma exposed and IPV populations, as an organizing framework to examine how differences in diagnoses (i.e., PTSD, comorbid PTSD and MDD, subthreshold trauma/ trauma and depression symptoms, and no lifetime exposure to trauma control comparison group) are related to alterations in cortisol measures. Specific cortisol measures that the study assessed include basal (defined as the average of four salivary cortisol samples across the diurnal cycle for the purposes of this study), diurnal trajectories (over four samplings through out the day),

and challenged (defined as changes in cortisol concentrations in the context of a psychosocial stress challenge) cortisol. While studies typically include groups meeting diagnostic criteria only, the current study also seeks to assess whether a diagnostic level of symptom severity is necessary to see alterations in cortisol outcomes i.e. if diagnostic groups (PTSD only and comorbid PTSD and MDD) would differ from a group of battered women with subthreshold symptoms of anxiety and mood disorders. Another area which is largely unexplored in the literature is the role of theoretically relevant variables, if any, in the observed neuroendocrinological differences within trauma exposed populations. Thus, this is one of a handful of studies which seeks to assess how alterations in basal cortisol concentrations, diurnal trajectories of cortisol concentrations, and challenged cortisol measures are accounted for by various trauma related characteristics. Specifically, the study examines how trauma characteristics such as chronicity of trauma exposure (childhood and adulthood versus adulthood exposure only), age of first trauma exposure, cumulative exposure to different types of trauma (IPV, IPV and childhood attachment related abuse, and attachment related abuse and non personal forms of trauma), and social support (general social support and trauma related social support) affect the aforementioned cortisol measures.

To begin with, chapter one, provides a context for the study's population of interest by reviewing the current status of IPV as a public health concern and its consequent mental health sequelae. Chapter two presents a theoretical framework for the study and discusses Trauma theory (Herman, 1992) as a basis to understand longitudinal mechanisms of psychopathology in IPV, particularly the phenomenology of chronic and severe PTSD in IPV. Within this theoretical framework, mechanisms associated with

early trauma exposure (e.g., childhood abuse) that can constitute a diathesis for victimization in adulthood and later psychological disorders are discussed to better understand trauma-related mental health outcomes in adulthood. Chapter three reviews studies examining basal and challenged cortisol outcomes and the diurnal pattern of cortisol in adults exposed to trauma. Studies examining cortisol outcomes in early developmental stages are briefly reviewed to provide a developmental context. Finally, studies with adult female victims of IPV examining cortisol measures are discussed.

Problem of IPV

The U.S. Census estimates that the number of adult women (age 18 or older) experiencing IPV annually is approximately 1.5 million with approximately 4.8 million incidents of IPV perpetrated against women each year in the U.S. (Tjaden & Thoennes, 1998; Tjaden & Thoennes, 2000a). According to two national crime surveys, approximately 20 to 64% of all violence against women is accounted for by IPV (Rennison, 2003; Tjaden & Thoennes, 2000b). Lifetime prevalence estimates for IPV are high. Rates of partner rape range between 8 to 10% (Finkelhor & Yllo, 1987; Russell, 1990; Tjaden & Thoennes, 2000b) and rates of physical assault by partners are estimated at about 22% (Bureau of Justice Statistics, 1998; Tjaden & Thoennes, 2000b). Community-based studies, which also include psychological abuse in their definition of IPV, have produced higher estimates ranging between 18 to 57% (Meisel, Chandler, & Rienzi, 2003; Smith, Thornton, DeVellis, Earp, & Coker, 2002; Waltermaurer, Ortega, & McNutt, 2003). Abused women are at risk for a range of mental health problems (Golding, 1999; Kemp et al., 1995), physical injury, (Rennison, 2003; Tjaden &

Thoennes, 1998; Tjaden & Thoennes, 2000a, 2000b), and even fatal injuries due to an abusive partner (Rennison, 2003), making it a significant public health concern.

Mental health consequences of IPV

Battered women are at high risk for developing PTSD (Ceballo, Ramirez, Castillo, Caballero, & Lozoff, 2004; Coker et al., 2002; Frasier et al., 2004; Houskamp & Foy, 1991; Kemp et al., 1995; Kemp et al., 1991; Lipsky, Field, Caetano, & Larkin, 2005; Mertin & Mohr, 2000; Nixon, Resick, & Nishith, 2004; Noll, Horowitz, Bonanno, Trickett, & Putnam, 2003; Pico-Alfonso et al., 2004; Sharhabani-Arzy et al., 2003). Rates of PTSD among battered women vary between 45% and 81% based on sample characteristics, the definition of IPV, and history of previous interpersonal trauma (Houskamp & Foy, 1991; Kemp et al., 1995; Sharhabani-Arzy et al., 2003; Stein & Kennedy, 2001). In a recent meta-analysis, Golding (1999) calculated the weighted mean prevalence of PTSD among abused women to be 64% across 11 individual studies examining the relationship between IPV and PTSD. Furthermore, studies suggest that frequency and severity of IPV (Cascardi et al., 1992; Houskamp & Foy, 1991; Mertin & Mohr, 2000; Pico-Alfonso et al., 2004) and chronicity of IPV (Bogat, Levendosky, Theran, von Eye, & Davidson, 2003) are related to risk and severity of PTSD.

Another common consequence of IPV is depression (Cascardi et al., 1992; Cascardi & O'Leary, 1992; Christopoulos et al., 1987; Coker et al., 2002; Doyle et al., 1999; Frank & Dingle, 1999; Kemp et al., 1991; Mitchell & Hodson, 1983; Moore et al., 1989; Nixon et al., 2004; Olson et al., 2003; Stein & Kennedy, 2001; Wolfe, Crooks, Lee, McIntyre Smith, & Jaffe, 2003). In a meta-analytic study of 18 studies, Golding (1999) calculated the mean weighted prevalence rate of 48% for MDD. Other mental health

sequelae of IPV include generalized anxiety (Coker et al., 2002; Kemp et al., 1991; Moore et al., 1989; Olson et al., 2003; Wolfe et al., 2003), somatization (e.g., headaches; dizziness; gastrointestinal disturbances, joint pain; numbness, tingling, or pain in the extremities) (Frasier et al., 2004; Lown & Vega, 2001), and dissociation (Dorahy et al., 2007). In addition, research also suggests that battered women are more likely to report suicidal ideation (Coker et al., 2002), and a greater number of suicide attempts relative to non-abused women (Doyle et al., 1999; Frank & Dingle, 1999; Kernic, Wolf, & Holt, 2000; Olson et al., 2003; Roberts, Lawrence, O'Toole, & Raphael, 1997).

It is notable that PTSD and depression are often co-morbid (Golding, 1999). For instance, in one study of women with recent experiences of IPV recruited from IPV agencies and medical clinics, researchers found that PTSD and MDD co-occur more often than would be expected by chance (Stein & Kennedy, 2001). The study also found that 43% of IPV victims with current IPV-related PTSD also suffered from MDD. Similarly in another sample of recent IPV victims, Nixon and colleagues (2004) found that co-morbid PTSD and MDD diagnoses (49%) were more common than a diagnosis of PTSD alone (27%). The study also found that individuals with comorbid diagnoses had more severe PTSD and MDD symptoms relative to individuals with a diagnosis of PTSD alone. Similar outcomes were also reported by Lipsky (2005), who sampled female IPV victims from an urban emergency department to assess the relationship between past year PTSD symptomatology and current depressive symptomatology. The study found that approximately half the sample had symptoms of PTSD. In addition they also had significantly higher mean total depression scores and higher mean scores on a majority of the depression subscales than those without PTSD. Finally, the study found that

depressive symptomatology, among other factors independently predicted PTSD symptoms.

IPV is also associated with personality disorders in the women survivors. For instance, Stanley and Penhale (1999) conducted a pilot study of 13 mothers with severe mental health problems whose children were involved in the child protection system. The study found that 6 of the women had been formally diagnosed with a personality disorder and all 13 women had experienced a history of IPV. Sansone, Reddington, Sky, and Wiederman (2007) also reported a relationship between borderline personality disorder and IPV among a sample of female primary care patients. Sixty four percent of battered women scored in the clinical range on a measure of borderline personality disorder, as compared to only 11.1% of non-abused women. These preliminary findings implicate IPV as an important risk factor for women's mental health problems, particularly PTSD and MDD.

In addition to the role of victimization in mental health outcomes adulthood, it is also important to examine the effects of trauma exposure in preceding developmental periods to better understand the evolution of mental health outcomes observed in adulthood. There is evidence to suggest that trauma exposure in childhood likely constitutes a diathesis for later victimization and hence, the role of early vulnerability factors is also important to consider

CHAPTER 2

Trauma Theory

Development of PTSD from IPV

Trauma theory posits mechanisms for the physiological and psychological sequelae of exposure to traumatic events such as IPV and childhood abuse (Herman, 1992). It explicates the processes underlying early, chronic and severe forms of trauma, and the role of childhood abuse in dysfunctional and violent relationships in adulthood.

The Diagnostic and Statistical Manual of Mental Disorders Text Revision (DSM-IV-TR) operationalizes a traumatic event as a situation in which a person perceives a threat to their life or physical integrity of the self or others. To receive a diagnosis of PTSD, the person must experience intense fear, helplessness, or horror as a result of such a perception (American Psychological Association, 2000). Characteristic responses to trauma exposure involve a complex integration of mental and physical responses. After a traumatic event the system of self-preservation goes into sustained alert, as though the danger might return at any moment. Adaptive changes in arousal, attention, perception, and emotion occur to mobilize the person to fight or to take flight.

Physiological arousal continues unabated and symptoms such as the startle response and irritability to small provocations are typical. Trauma victims have an intense reaction to specific stimuli associated with the traumatic event. They respond to repetitive stimuli associated with, or reminiscent of, the threat as though they are novel and pose a renewed danger. This hyperarousal also affects sleeping patterns. As noted earlier, a victim may also suffer from an array of other anxiety symptoms. Following the

traumatic exposure they do not show a return to normal states of arousal but a state of “relaxed attention” i.e., an elevated baseline of arousal (Herman, 1992, p.36).

Trauma victims also demonstrate intrusive symptoms (e.g., flashbacks). Traumatic experiences become encoded in an abnormal form of memory which spontaneously intrudes into consciousness both as flashbacks during the waking states and also as traumatic nightmares during sleep. Several symptoms of constriction or negative symptoms that suggest the absence of seemingly natural sequelae are also characteristic of trauma victims (e.g., calmness instead of the lack of overwhelming emotions). It has been suggested that during a traumatic event, the overwhelming helplessness can force individuals to ‘escape’ by altering their state of consciousness, referred to as “numbing” (Herman, 1992, p.42). This can be described as an ostensibly paradoxical state of detached calmness, which serves to protect against extreme pain. Similarly subjective feelings of detachment, enhanced perception of imagery, distortion of reality, depersonalization, and derealization are also commonly seen in trauma victims (Herman, 1992). The overwhelming and threatening nature of traumatic events seem to “...recondition the human nervous system” (Herman, 1992, p.36).

Thus, both polarities between heightened perception and numbing symptoms are frequently noted in trauma victims. Negative or constrictive processes strive to keep traumatic memories out of consciousness allowing only a fragment of the memory to surface as an intrusive symptom. This dialectic of opposing states is believed to be the most characteristic feature of PTSD, and as the person vacillates between intense flooding feelings and vacant states of no feeling, the sense of unpredictability and helplessness is heightened, guarding against the integration of the traumatic event.

In addition to the physical symptoms, traumatic experiences can have a lasting impact on the internal representations or schemas of victims. Basic human beliefs, about relationships and the sense of self that is formed and sustained in relation to others are shattered. The victim's fundamental assumptions about safety of the world, positive value of the self and meaningful order of creation are violated (Ebert & Dyck, 2004; Herman, 1992). In sum, due to the overwhelming nature of traumatic threats, there are lasting physiological arousal, memory, emotion, attention, and cognitive changes, particularly in the face of repeated or chronic trauma.

IPV victims are typically exposed to both psychological and physical domination, resulting in significant distortions in the representations of the perpetrator (Herman, 1992). Perpetrators' techniques involve organized efforts directed towards disempowerment and disconnection of the victim by instilling terror and subjugation and destroying the victim's sense of self in relation to others. This may take on the form of threats against others (e.g., friends, relatives, children, parents), who are important to the victims or may harbor or protect the victim. Often loyalty towards the perpetrator requires severing relationships or attachments with others. This typically manifests as relentless accusations of infidelity, demonstrations of jealousy and demands of both physical and symbolic disconnection from others. For instance, token concessions and sacrifices (e.g., burning photographs or letters of loved ones) maybe required.

Fear is typically instilled through unpredictable amounts of violence and enforcement of random petty rules that not only seek to instill fear but also a sense of gratitude for being allowed to live. After experiencing several cycles of reprieve from death, the victim eventually comes to see the perpetrator in the role of a savior. The

physical disconnection increases the victim's dependence and survival on the perpetrator because the more frightened they are the more they cling to their abuser or savior. In doing so victims seek out the humanity in the perpetrator and may eventually come to see the world through the perpetrator's perspective

However, psychological control of a victim is only complete when she has been forced to violate her own moral principles. Violation of principles may involve sexual humiliation and coercion into practices that they find immoral, or disgusting. This might also extend to abuse of the victims' children. Several studies show a significant association between partner abuse and child abuse (Duhamel & Fortin, 2004; Tajima, 2004). This pattern of betrayal may start small but eventually progresses into outrageous physical or sexual abuse (Herman, 1992).

Traumatic events destroy the belief that one can be oneself in relation to others. In some cases survivors may direct their rage and hatred against themselves. The internalization of the blame or fear possibly from the traumatic experience may lead to the development of other psychological disturbances (Olf, Langeland, & Gersons, 2005) and suicidality may become a form of resistance (Herman, 1992).

Chronic trauma, including IPV, can erode the personality structure of a victim. Chronic abuse amplifies hyperarousal symptoms of PTSD, as they are continually hypervigilant, anxious and agitated. Avoidance and constriction also become prominent as the victim goes into sustained survival mode and psychological constriction is critical to such survival. Staggering psychological dysfunction and loss can also result in a state of depression. In such conditions, Hermann (1992) argues that chronic hyperarousal and intrusive symptoms of PTSD may fuse with vegetative symptoms of depression. Thus,

attachment disruptions, the loss of faith and beliefs merge with the isolation and hopelessness of depression. The debased self-image of chronic trauma forms the basis for guilty ruminations in depression and in extreme cases dissociative symptoms as well.

However, there is evidence to suggest that adults exposed to IPV have also experienced significant trauma in earlier stages. For instance, survivors of childhood abuse and neglect are also involved in dysfunctional and/or violent relationships in adulthood (Drapeau & Perry, 2004; Haller & Miles, 2003; Seedat et al., 2005), being raped or sexual harassed (Heim et al., 2002; Russell, 1986). This suggests that a) chronicity of trauma predates victimization in adulthood, b) observed mental health outcomes could be a result of a more longitudinal course of trauma and c) developmental mechanisms across the lifespan need to be examined to better understand outcomes in adulthood.

Early trauma exposure as a vulnerability factor for IPV

Trauma theory not only explicates processes associated with chronic IPV but also posits mechanisms associated with exposure to abuse in childhood that can lead to psychological problems and an increased risk for revictimization in adulthood. According to the theory, abused or neglected children have to adapt to an unsafe and often horrifyingly unpredictable environment. These experiences lead to resourceful, but also destructive, capacities for abnormal states of consciousness, which manifest as an array of perceptual, cognitive, somatic, psychological, and memory-related distortions or difficulties (Herman, 1992; Pollak, Klorman, Thatcher, & Cicchetti, 2001; Pollak, Vardi, Putzer Bechner, & Curtin, 2005). Children growing up in an environment of domination and abuse develop pathological attachments to their abusers, attachments that they strive

to maintain even at the expense of their own well-being (Bowlby, 1982). Adaptation to such a climate of constant danger requires a constant state of alertness and children in an abusive environment develop extraordinary abilities to scan for warning signs.

Consequently, Hermann (1992) posits that the development of psychological disorders is rooted in the array of psychological defenses utilized to cope with such an unpredictable and threatening environment. For instance, some experiences maybe blocked off from conscious awareness and memory, and other experiences maybe minimized or rationalized so that they are not construed as abuse, essentially creating pathways for cognitive distortions and dissociative states (Herman, 1992)

One consequence of these seemingly adaptive defenses is that emotion regulation is severely affected as normative feelings of anger, rage, and hurt have to be contained and compliance has to be maintained. The development of a cohesive and well integrated sense of self or self-representation becomes a challenging task because certain experiences are exaggerated and split off (Bowlby, 1982). Such processes are believed to be the basis for self-blame i.e., a sense of inherent badness to make sense out of the abusive environment that can be used to justify future negative experiences in adulthood as well, and form the basis of a fragmented or dysfunctional personality, consciousness, and memory (Herman, 1992).

The unpredictable and dictatorial parental power of an abusive parent is akin to an adult violent partner's demands for complete control and subjugation. Hermann (Herman, 1992) further argues that IPV in adulthood forces victims to revisit developmental conflicts and relive her earlier struggles of autonomy, initiative, competence, identity, and intimacy. With fundamental problems in trust, autonomy, independence and

intimacy, childhood abuse survivors are driven by fear of abandonment, or exploitation and there is a continual quest for rescue through a powerful savior, who will protect and care for them. The tendency to denigrate oneself, and engage in minimization, rationalization, and idealize those that the victim is attached to, clouds the judgment of the victim, and makes the victim attuned and obedient to the wishes of others, making her vulnerable in the future. It is notable that these processes do not implicate that abuse is actively sought out but rather that the victim's internal representation of intimate relationships is distorted to fear abuse as an unavoidable fate and accepted as the cost of a relationship. Consequently adult survivors of trauma and abuse are at a greater risk for repeating victimization in adult life.

In sum, childhood abuse and neglect has a significant negative impact on the psychological make-up and personality of an individual. These effects can be long lasting due to an increased risk for psychopathology and maladaptive cognitive schema that extend into later developmental stages.

Implications for trauma characteristics

Within this theoretical framework various trauma characteristics are posited to influence psychological outcomes, and are reviewed below.

Age of trauma exposure

One characteristic of trauma exposure that has been theoretically and empirically posited to be an important factor is the age of trauma exposure. Studies with humans (e.g., Swett, Surrey, & Cohen, 1990; Teicher, Glod, Surrey, & Swett, 1993) and animal models (see Heim & Nemeroff, 2001 for a summary of findings) have consistently shown that early exposure to traumatic events can have a lasting negative impact. The effects of

such trauma exposure are believed to be more magnified and/ or enduring, when they occur during the formative years, when physical and neurobiological systems are maturing, and attachment relationships with significant caregivers are still developing (Schorer, 2002a). The critical period for the establishment of an attachment relationship also overlaps with rapid physiological and brain maturation (Schorer, 2002b). Traumatic experiences during such a critical period of development, in the absence of a protective caregiving presence, or worse, if the caregiving system itself is a source of the trauma, is likely to result in significant levels of arousal and inadequate reparative experiences. Given the importance of this developmental period, trauma exposure in early developmental periods can lead to a dysfunctional trajectory of physiological, emotional, and psychological development, (Cicchetti & Curtis, 2006; Heim & Nemeroff, 2001; Pryce et al., 2005).

The most commonly examined traumatic event is childhood sexual abuse (Banyard & Williams, 1996; Bunce, Larsen, & Peterson, 1995; Johnson, Pike, & Chard, 2001). However, studies with other types of abuse and trauma exposure such as the holocaust also report similar findings (Yehuda, Schmeidler, Siever, Binder-Brynes, & Elkin, 1997). In general studies report an inverse relationship between age of childhood sexual abuse and the severity of depression, dissociation (Johnson et al., 2001), and anxiety symptoms (Bunce et al., 1995) and low self-esteem (Bunce et al., 1995) in adulthood. As noted earlier, the rates of revictimization among survivors of childhood abuse victims is high and hence, it is difficult to assess the independent impact of the age at the time of trauma exposure is responsible for adult psychological problems, particularly as it is based on retrospective reports. For instance, Nishith, Mechanic, and

Resick (2000) examined PTSD symptoms as a function of childhood sexual physical abuse versus adult sexual and physical abuse. This study found childhood abuse and adult victimization together, were more predictive of PTSD symptoms rather than childhood abuse alone.

In a study by Banyard and Williams (1996), childhood sexual abuse victims were interviewed prospectively (soon after the experience of abuse after receiving emergency services) and age of trauma experience was found to be an important predictor of mental health outcomes in adulthood. However, when the same participants were re-examined 20 years later in adulthood, and asked for retrospective reports of the same abuse information, these age-related effects were no longer significant. It notable that many in the sample also experienced high rates of revictimization in adulthood. Thus, differences in findings could reflect a methodological confound of retrospective reports as recollections of the details of the abuse maybe influenced by concurrent levels of distress in adulthood (Henry, Moffitt, Caspi, Langley, & Silva, 1994) and/ or the findings could reflect the confounding effects of chronicity/ cumulative experiences of trauma, such that outcomes in adulthood could be a result of the cumulative effects of trauma across the lifespan.

In a retrospective study of Holocaust survivors Yehuda and colleagues (1997) examined the impact of the Holocaust 50 years after the experiences and found that age at the time of Holocaust was inversely related to PTSD symptoms such as psychogenic amnesia, emotional detachment, and hypervigilance. Thus, while age of trauma exposure is an important characteristic for understanding mental health outcomes in adulthood, chronicity of trauma is a common confounding factor due to high rates of revictimization

in adulthood in childhood abuse survivors. In addition, these findings are difficult to examine retrospectively. Finally, experiences of trauma in adulthood are also likely to independently contribute to this pre-existing vulnerability and needs further examination.

Cumulative Effects of Trauma Exposure Based on Relationship to Perpetrator

One aspect of trauma related research that has received considerable evidence with respect to theoretical development but limited empirical examination is the relationship of the perpetrator to the abuse victim. There is limited evidence from empirical studies suggesting that an abuse victim's perceptions of, and reactions to, the traumatic experience is affected by her relationship with the perpetrator (e.g., Ketring & Feinauer, 1999; Leahy, Pretty, & Tenenbaum, 2004; Lucenko, Gold, & Cott, 2000).

According to attachment theory, the relationship between a child and her primary caregiver has important survival implications for the infant (Bowlby, 1982). The quality of the caregiver's responses reinforces certain patterns of behaviors that are learned and internalized because they ensure proximity with the caregiver and consequently survival for the infant. These internalized schemas form an information processing template by incorporating expectations, feelings, and behaviors towards others. In this way, lived experiences with the caregiver help the infant develop a core self of self and others, and are considered important for psychological, affective, and behavioral regulation. Similarly, trauma theory also posits that abuse within the family is comparable to extreme forms of trauma exposure such as war-related torture, because of the horrific transgression that it represents (Golding, 1999; Herman, 1992).

The effect of the relationship of the perpetrator to the victim is most commonly examined in childhood abuse literature. A national survey of child rape found that 48% of

sexual crimes were perpetrated by a trusted non-relative, 41% by relatives, and 11% by strangers (Saunders, Kilpatrick, Hanson, Resnick, & Walker, 1999). A commonly cited reason for these findings is that family members and close non-relatives are most likely to have the opportunity to perpetrate. In cases where family members are perpetrators, the source of abuse is also the source of safety and basic needs, which poses a tremendous psychological challenge for victims.

Studies that have examined the impact of perpetrator relationship to victim, on the psychological outcomes of abuse have found some support for the fact that abuse at the hands of a caregiver or attachment figure leads to more deleterious outcomes relative to perpetration at the hands of a stranger. However, only a limited number of studies have examined these outcomes using heterogeneous methodologies and varying sample characteristics. Most importantly, studies that have failed to find a difference with respect to perpetrator status have not examined the impact of possible protective factors, making it difficult to interpret the lack of differential effects.

For instance, Banyard and Williams (1996) found that the relationship to the perpetrator was a significant predictor of outcomes 20 years later such as emotional distress, sleep problems, dissociative experiences, and overall psychological symptoms. Similarly Leahy and colleagues (2004) reported significantly higher level of PTSD symptoms and dissociative symptoms for survivors of childhood sexual abuse by trusted individuals, guardians/caretakers compared to sexual abuse by other individuals. Perpetration by a father figure has been found to be predictive of high levels of psychological symptoms in adulthood, followed by family members and acquaintances,

and finally strangers (Ketrting & Feinauer, 1999). Findings from these studies suggest that the survivor's relationship with the perpetrator influences adult psychological symptoms.

In contrast, in one study, the effects of perpetrator relationship, caregiving duties, and residential proximity (i.e., if the perpetrator resides with the victim) in adult survivors of childhood sexual abuse were examined in relation to PTSD symptoms (Lucenko et al., 2000). The authors reported that PTSD symptoms were higher in survivors of childhood sexual abuse perpetrated by persons that did not have caretaking responsibilities for the victims. In another study Johnson and colleagues (2001) found that the relationship and level of trust with the perpetrator was not significantly related to PTSD, depression, or dissociation symptom severity. While these studies present contradictory results, it is notable that both studies included samples from a population of treatment seeking victims, which may not be representative of all survivors of childhood sexual abuse. In comparison, studies that found significant differences in psychological symptoms based on relationship to perpetrator were recruited from random surveys.

In addition to the differential impact of perpetrator type on mental health sequelae, there is also evidence to suggest that cumulative trauma exposure has deleterious effects on later psychological outcomes (Briere, Stacey, & Green, 2008; Suliman et al., 2009). For instance, in a large college study of women, Briere and colleagues (2009) found a linear relationship between exposure to different types of traumatic events (cumulative trauma) in childhood and symptom complexity in adulthood. Similar findings were reported by Suliman and colleagues (2009) in a large adolescent sample who had experienced multiple traumatic stressors with respect to PTSD and depressive symptoms.

Despite evidence for the effects of perpetrator relationship to victims and cumulative trauma exposure on the severity of psychological outcomes, no study has examined the impact of cumulative trauma exposure in the context of perpetrator type on cortisol outcomes.

Social Support

An important moderator of trauma related outcomes is social support. Typically social support networks include family members, relatives, friends, and peers (Rosenthal, Feiring, & Taska, 2003). Trauma can erode the sense of trust in others, and Herman (1992) argues that positive social support is a necessary aspect of recovering from traumatic experiences. Furthermore, trauma theory also posits that recovery from a traumatic event is associated with the type of support received in relation to the traumatic experience. Thus, while it is important to examine general levels of social support which has been shown to have a significant moderating influence on the impact of trauma on psychological outcomes (Bal, De Bourdeauhuij, Crombez, & van Oost, 2005; Banyard & Cantor, 2004; Gold et al., 2000; King, King, Foy, & Gudanowski, 1996; Levendosky et al., 2004; Regehr et al., 2007) it is also critical to examine support received in the immediate aftermath of trauma exposure (Davis & Brickman, 1991).

However, the quality of social support is also an important characteristic. For instance, Davis and Brickman (1991) examined whether type of social support moderated psychological adjustment in adult rape victims. This study found that while positive social networks did not influence psychological symptoms, unsupportive social networks had a negative impact on psychological outcome. Similarly, in a study of pregnant women exposed to IPV, Levendosky and colleagues (2004) examined the impact of the

quality of social support on psychological outcomes. This study found that having a high ratio of supporters who had also experienced IPV to supporters who had not experienced IPV predicted impaired quality of support among battered women. It is possible that social support network members suffering from similar problems are unable to provide support to others that can help them rebuild their sense of trust. Thus, findings suggest that not all forms of social support are beneficial for positive health outcomes and it is crucial to examine the level and quality of support received from others, in relation to the traumatic event.

In sum, several theoretically driven moderators of trauma outcomes have been posited to influence the longitudinal mechanisms of psychopathology in the context of trauma. These need to be better integrated in empirical research examining outcomes of traumatic experiences at multiple levels including neurobiological and psycho-emotional functioning to gain a more comprehensive understanding of traumatology and as a next step, to help tailor psychosocial and pharmacological interventions.

CHAPTER 3

Neurobiological model of PTSD: The HPA Axis

The HPA axis is one of the biological systems that has been the focus of trauma-related research. Within this system, cortisol is the main hormone that has been examined. Activation of the HPA axis begins in the locus coeruleus and results in the release of neurotransmitters such as norepinephrine, serotonin, and dopamine. They, in turn, activate the amygdala to stimulate the hypothalamus to release corticotrophin-releasing factor, which stimulates adrenocorticotrophic hormone (ACTH) secretion. ACTH secretion results in elevated cortisol levels. To avoid the detrimental effects of prolonged HPA activation and to inhibit its own release, cortisol acts on pituitary and brain receptors a negative feedback mechanism to return these hormonal levels to a state of equilibrium (Heim & Nemeroff, 2001; Heim et al., 2002). Thus, the axis forms a closed loop negative feedback system, which regulates cortisol levels.

These neuroendocrine changes, or allostatic adjustments, are meant to help short-term adaptations in the face of a stressor. Thus, chronic high levels of stress have been associated with damaging consequence, and the cost of maintaining allostasis i.e., stability or homeostasis through physiological or behavioral change, is referred to as allostatic load. This allostatic load has been shown to have negative effects on various physiological systems such as hormonal imbalance and HPA axis dysregulation (Heim et al., 2000; King, Mandansky, King, Fletcher, & Brewer, 2001; Saltzman, Holden, & Holahan, 2005), neuronal loss in the hippocampus (Bremner et al., 1995; Sapolsky, 1996; Tupler & De Bellis, 2006), electroencephalographic abnormalities (e.g., Anda et al., 2002; Ito et al., 1993; Teicher et al., 1997), skin conductance responsivity, increased

heart rate and blood pressure (e.g., Perry, 1994; Saltzman et al., 2005), and changes in immune functioning (e.g., Altemus, Cloitre, & Dhabhar, 2003). However, despite significant evidence of neurobiological changes and dysfunction, studies examining cortisol outcomes in the context of exposure to traumatic events present a complicated picture.

Chronic activation of the HPA system, typical in the context of repeated exposure to trauma, can have deleterious effects on physiological functions. However, there is considerable variability in findings of cortisol outcomes in adults, with several studies indicating elevated levels of basal cortisol (Lemieux & Coe, 1995; Maes et al., 1998; Rasmusson et al., 2001), and findings from a recent meta-analysis which included 37 studies (PTSD $N=828$; Control $N= 800$) suggesting no differences in basal cortisol levels (Meewisse et al., 2007).

The lack of uniformity in findings is confounded with methodological differences across studies. One key issue in this regard is related to the timing, frequency, and type of cortisol measurement. For instance, studies examining blood or salivary cortisol levels, particularly those using a single sampling of cortisol, do not take into account individual variation in sleep cycles. This is a crucial issue because cortisol levels have a specific diurnal cycle such that there is steady decline in cortisol levels after it peaks approximately 30 minutes post-awakening. Thus, the variation in cortisol outputs is significantly increased even with a difference of several minutes in wake-time.

Another important issue related to diurnal differences in cortisol outcomes is the type of cortisol measurement. While salivary and urinary cortisol levels typically reflect “free” or an ambient fraction of the cortisol hormone, plasma samples indicate total

cortisol. Thus, the type of cortisol measurement and the number of cortisol measurements are both important methodological indicators. For instance in their meta-analytic study Meewisse and colleagues (2007) conducted subgroup analyses assessing the effects of type of cortisol on basal cortisol levels and found that plasma or serum studies showed significantly lower levels of cortisol in PTSD samples rather than control or non-exposed groups (Meewisse et al., 2007). However, it is notable that this study used the “earliest” (with respect to time of day) sample of cortisol available in each study in their analyses thereby using only one data point from each study rather than calculating a combined effect size for each individual study when salivary/ urinary samples were utilized. In fact several researchers argue in favor of utilizing salivary cortisol because it allows an assessment of free or ambient cortisol, which coupled with multiple samplings, provides a more nuanced examination of the trajectory of diurnal cortisol as well as challenged cortisol outcomes. Also, salivary cortisol offers significant advantages over urinary free cortisol with respect to ease of collection, and assays and greater reliability relative to other types of cortisol (Young & Breslau, 2004b).

In addition to the methodological issues associated with timing, frequency, and type of cortisol, other issues such as small sample sizes, adequate control for trauma characteristics while examining the effects of diagnostic status and vice versa are commonly seen in the literature and are reviewed in further detail ahead. However, not all variations are attributable to methodological differences alone and certain commonalities have emerged in the literature. For instance, some researchers have suggested that variations in basal cortisol alterations may represent biological subtypes of PTSD. Repeated activation and hyperarousal typically seen in the face of chronic trauma is

believed to be countered by increased negative feedback to maintain homeostasis and regulate levels of plasma cortisol. This increased negative feedback is believed to be the basis for lower levels of basal cortisol outcomes (Yehuda, 2002). Low cortisol and enhanced negative feedback are understood to represent a biological subtype of PTSD associated with more chronic, or early exposure to, trauma (Miller et al., 2007; Rasmusson et al., 2001; Yehuda, 2002; Yehuda, Halligan, & Grossman, 2001). Other evidence from studies examining the effects of PTSD and MDD comorbidity suggest that another biological subtype of PTSD, associated with higher levels of cortisol, is likely to be comorbid with depressive disorders (Olf, de Vries, Guezelcan, Assies, & Gersons, 2007; Olf, Guezelcan, de Vries, Assies, & Gersons, 2006; Rasmusson et al., 2001; Yehuda, 2002; Young & Breslau, 2004a, 2004b).

Characteristics of trauma such as chronicity, age of trauma exposure, role of protective factors such as social support, and type of trauma exposure have also received some attention in adult populations. For instance, chronicity of trauma has been examined in multiple studies as a predictor of cortisol outcomes in adulthood. As noted earlier researchers have hypothesized that the enhanced negative feedback inhibition likely develops over time in response to the chronic trauma and represents a biologic subtype of PTSD (Yehuda, 2002).

Research in child and adult samples has shown dysfunction in HPA axis. Thus individuals' outcomes in adulthood need to be examined as a function of early childhood exposure to trauma as well as IPV, particularly because there is evidence to suggest that early childhood constitutes a critical period of experience-dependent brain development

and consequently outcomes of early childhood trauma can extend into adulthood (e.g., Anderson, Teicher, Polcari, & Renshaw, 2002).

Review of basal cortisol outcomes in PTSD in children

In general, the limbic system, which plays a critical role in emotion regulation, is most affected by adverse exposure during childhood when these systems are still developing (McCollum, 2006). Although the developing and limited sensory and perceptual systems of young children place limits on the amount of sensory information processing they can occur, children's innate tendencies to adapt, results in increased attendance to the more salient cues in the environment at the expense of other aspects.

For instance, maltreated children's organization of the emotional systems themselves become based upon processing highly salient cues of interpersonal hostility or threat to the self. A number of studies have found support for this form of selective, experienced based learning in maltreated children's perception of emotion and attention regulation (Pollak, 2005; Pollak, Cicchetti, Hornung, & Reed, 2000; Pollak & Kistler, 2002; Pollak et al., 2001; Pollak & Sinha, 2002; Pollak & Tolley-Schell, 2003; Pollak et al., 2005). For instance, maltreated preschoolers have been found to perceive angry faces as highly salient in contrast to other facial displays of emotions (Pollak et al., 2000), display a lower threshold to detect angry facial expressions (Pollak & Sinha, 2002), and have altered abilities to discriminate and categorize angry facial expressions such that they use broader perceptual boundaries for anger perception (Pollak & Kistler, 2002). Similarly, studies also found that maltreated children engage in anticipatory monitoring of the environment for anger related cues (Pollak, 2005). A series of event-related potential studies also found that maltreated children demonstrate differential neural

processing of anger related stimuli, but not for other emotional expressions or non-emotional content when compared to age matched peers (Pollak et al., 2001).

There is also evidence for HPA axis dysfunction in childhood and adolescence in the context of trauma (e.g., Carrion et al., 2002; DeBellis et al., 1999; Goenjian et al., 1996; Hart, Gunnar, & Cicchetti, 1996; Kaufman et al., 1997; King et al., 2001; Lipschitz et al., 2003; Saltzman et al., 2005). For instance, Saltzman and colleagues (2005) examined HPA axis functioning in children, between 5 and 13 years of age, exposed to IPV ($n=21$) in comparison to a clinical group of children ($n=27$) who were receiving mental health services for symptoms of anxiety, depression, and disruptive behavior, but had no documented or self-reported history of IPV in the family. Salivary cortisol was assessed pre- and post-interview to assess basal functioning, and the response to interview which included an assessment of their IPV exposure and trauma symptoms. With respect to HPA axis functioning, IPV exposure was associated with higher levels of basal salivary cortisol. The mean basal cortisol levels were 62% higher in IPV exposed children, and they continued to display higher levels of cortisol post-interview as well. This study did not assess the timing or chronicity of IPV exposure, nor did it control for the high comorbidity of child abuse (direct physical violence from a caregiver) in the children exposed to IPV (48%). While this is likely to be representative of what children from households with IPV generally experience, this makes it difficult to examine the unique effects of IPV exposure and child abuse. Similarly, others studies have examined the effects of trauma or depression, due to various types of abuse and neglect, on HPA functioning, without assessing the contribution of specific types of traumatic events (Carrion et al., 2002; Kaufman et al., 1997; Lipschitz et al., 2003). For instance, Carrion

and colleagues (2002) compared 51 trauma-exposed children (30 boys and 21 girls) to 31 age and gender matched healthy controls (age range 7-14 years) on their diurnal salivary cortisol measured 4 times a day over the course of 3 days. Their study found significantly elevated cortisol levels in the trauma exposed group compared to the control group. Nearly 51% of the children in trauma-exposed sample had experienced multiple traumatic events.

In another study Kaufman and colleagues (1997) assessed HPA axis functioning in children with a history of abuse using a corticotropin-releasing hormone challenge intravenously. The sample consisted of depressed abused ($n=13$), depressed nonabused ($n=13$) and healthy control children ($n=13$). The depressed abused children were further segregated as those who continued to experience ongoing adversities (e.g., emotional abuse, poverty, IPV exposure) and those that were living in fairly stable home environments. The authors found significantly elevated levels of cortisol levels in the group of abused depressed children who were also experiencing concurrent adversity. The other subgroups displayed similar levels of cortisol levels. While this finding is consistent with Hart and colleagues (1996), who reported greater afternoon salivary cortisol levels in depressed maltreated children when compared to depressed nonmaltreated children, it is in contrast to DeBellis and colleagues (1999) study with sexually abused girls who were also administered a CRH challenge. However, in the latter study the girls were living in fairly stable homes. In a later study DeBellis and colleagues (1999) compared cortisol levels in children exposed to multiple form of trauma, who were diagnosed with PTSD ($n=15$), with age and gender matched children with other anxiety-disorders only ($n=10$) and healthy nonabused controls ($n=24$), and

found significantly increased cortisol levels in the PTSD group compared to all other comparison groups.

In studies examining cortisol levels in adolescents (Goenjian et al., 1996; Lipschitz et al., 2003) the findings have been mixed. Goenjian (1996) reported lower basal cortisol levels in adolescents ($n=37$) in two Armenian cities, 5 years after they had experienced an earthquake. In contrast Lipschitz and colleagues (2003) examined cortisol levels in adolescents with current PTSD ($n=20$), controls with trauma-exposure and no PTSD ($n=9$) and healthy controls ($n=19$). In response to a chemical challenges using DST all groups performed similarly with respect to cortisol outcomes. However, all subjects who had PTSD and comorbid diagnoses of major depression had significantly higher levels of basal salivary cortisol compared to the other adolescents. In a sample of children and adolescents (ages 8-18) Delahanty and colleagues (2005) examined cortisol levels in 56 boys and 26 girls. The sample was recruited from hospital trauma centers and most of the participants were involved in a vehicular accident. This found that higher levels of cortisol were associated with greater PTSD symptoms.

Review of basal cortisol outcomes in PTSD in adults

While cortisol outcomes in children have implicated dysfunction in the HPA axis, they have typically indicated higher levels of cortisol output. In contrast, findings from adult studies, which also indicate HPA axis dysfunction, have typically shown lower levels of basal cortisol relative to healthy controls. Thus, researchers have posited that in the face of chronic exposure, unlike exposure limited to childhood, the HPA axis develops an enhanced negative feedback inhibition (Yehuda, 2002). Typically studies with adults that have examined recently traumatized subjects have reported elevated

cortisol levels, (Lemieux & Coe, 1995; Maes et al., 1998; Rasmusson et al., 2001).

Relatedly, studies taking into account the effects of prior victimization provide further support for this hypothesis. For instance, in a study of female victims of rape, Resnick and colleagues (1995) reported lower levels of cortisol in women who also had a prior history of assault. Only women without a prior history of traumatization had higher levels of cortisol. Women with lower cortisol levels and prior history of assault also had a higher probability of developing PTSD, implicating the role of early vulnerability factors and/or the impact of cumulative trauma exposure.

Evidence that prior history of trauma and lower protective factors affects cortisol outcomes also comes from a study of police recruits enrolled in basic constable training program by Regehr and colleagues (2007). Study participants ($n = 84$; 71.4% male) were exposed to a stressful simulation of a policing procedure. Cortisol outcomes were related to subjective reports of psychological stress which in turn was predicted by prior history of trauma and reduced social support. The study found no direct relationship between cortisol levels and prior history of trauma or trauma symptoms, suggesting a mediated model of alterations for cortisol outcomes such that a vulnerability to psychological stress, induced through a history of traumatization, is heightened or reactivated in later situations of acute stress resulting in altered cortisol outcomes. This is the only study examining the impact of social support on cortisol outcomes.

The development of an enhanced negative feedback inhibition in response to chronic trauma also suggests that other characteristics of trauma such as severity and potentially the age of trauma exposure could also have an impact on cortisol outcomes. With respect to severity of symptoms, findings suggest an inverse relationship between

cortisol levels and severity of trauma consistent with the enhanced negative feedback inhibition mechanism (Boscarino, 1996; Yehuda, Boissoneau, Lowy, & Giller, 1995). For instance Boscarino (1996) found lower basal cortisol in a large epidemiologic sample of over 2000 Vietnam veterans with PTSD compared to those without PTSD. Cortisol levels were significantly lower in combat veterans with very high exposure compared with those with low or no exposure. Similar findings were also reported in a smaller sample of Vietnam veterans with respect to combat severity and morning cortisol outcomes (Yehuda et al., 1995). In sum, victims exposed to chronic forms of trauma, particularly a combination of childhood and adult victimization are more likely to evidence lower levels of basal cortisol while those experiencing relatively recent traumatic events only are more likely to demonstrate elevated levels of basal cortisol.

Relatedly, studies also suggest that severity of PTSD symptoms, which are likely to be positively correlated with severity of trauma exposure, is also inversely related to cortisol levels. For instance, Olff and colleagues (2006) reported evidence of this inverse relationship in a sample of chronic PTSD patients exposed to civilian trauma (e.g., abuse, loss of a loved one, vehicular accident). Similarly studies examining whether cortisol dysfunction is related to trauma exposure or the presence of diagnostic levels of PTSD, also indicative of symptom severity, one study suggests that diagnostic levels of PTSD, indicating severity of symptoms, is important. Wessa and colleagues (2006) examined diurnal cortisol patterns in three groups, trauma exposed with PTSD ($n = 29$), without PTSD ($n = 19$) and non exposed controls ($n = 15$). While the three groups did not differ with respect to the waking response, PTSD subjects showed significantly lower levels of cortisol for the peak response compared to trauma exposed and healthy subjects. This

pattern remained stable throughout the day. Although the two trauma-exposed groups did not differ with respect to the type of trauma they experienced or the time span since trauma onset, it is unclear whether the groups were similar with respect to duration of trauma exposure, suggesting a confounding effect of chronicity as well as a possible lifetime diagnosis of PTSD.

With respect to chronicity and age of traumatic events researchers have reported that lower cortisol levels are associated with an early traumatic history (Carpenter et al., 2007; Rasmussen et al., 2001; Santa Ana et al., 2006; Yehuda, Halligan, & Grossman, 2001). For instance Yehuda and colleagues (2001) reported an inverse relationship between childhood emotional abuse and cortisol levels among adult children of holocaust survivors. Also, Santa Ana and colleagues (2006) assessed differences in cortisol functioning in individuals with childhood ($n = 25$) versus adult exposure to trauma ($n = 33$). This study found that participants with a history of childhood trauma (conceptualized as prior to age 18) had lower levels of cortisol at baseline compared to individuals with a history of victimization in adulthood (age of trauma exposure after 18). In a sample of adults without current psychopathology Carpenter and colleagues (2007) compared subjects with a history of moderate to severe childhood maltreatment ($n = 23$) to those without such a history ($n = 27$) and found lower levels of baseline cortisol in the group with a childhood history of abuse. However, neither of the studies examined whether the age of trauma exposure coincided with a critical period of biological maturation of the limbic system or dyadic emotion regulation as suggested by empirical evidence (McCollum, 2006) and theoretical implications (Schoore, 2002b). While retrospective report poses a tremendous methodological challenge particularly given the pathoplastic

effects of concurrent stressors (Henry et al., 1994), this issue needs further examination to expand theoretical definitions of critical stages of development.

Relatedly, some researchers have reported a lack of significant differences across trauma type (Young et al., 2004) however, others have noted that the type of trauma exposure (e.g., interpersonal/ attachment-related versus combat related) could be an important issue (Meewisse et al., 2007). This issue relates to perpetrator relationship discussed before, which has been shown to have a differential impact on psychological outcomes. In their meta-analysis Meewisse and colleagues (2007) conducted subgroup analyses to examine the effects of type of trauma on basal cortisol. Four types of traumatic events were coded - war veterans, victims of sexual or physical abuse, refugees, and various trauma. Findings indicated lower cortisol levels in victims with victims of sexual or physical abuse but no differences were found for other types of trauma when compared with healthy controls. However, as the authors note, their findings could be confounded with gender effects because the lack of adequate information regarding the break up between gender and each trauma type from each study did not allow the authors to control for the effects of gender. Similarly, the authors also note that these findings could also reflect the effects of chronicity or age of onset of trauma as sexual or physical abuse typically begin early in the developmental lifespan and tend to be chronic relative to other types of trauma such as combat war, but the lack of adequate information did not allow for statistical control of these issues (Meewisse et al., 2007).

Finally, with respect to the issue of comorbidity between PTSD and depressive disorders and biological subtype of PTSD, only a handful of studies have examined the effects of such diagnostic comorbidity on cortisol outcomes (Olf et al., 2006; Yehuda,

Golier et al., 2004; Yehuda, Halligan et al., 2004; Yehuda et al., 1990; Yehuda, Teicher, Trestman, Levengood, & Siever, 1996; Young & Breslau, 2004b; Young et al., 2004). Among them, two studies, which included samples exposed to civilian trauma (e.g., abuse, loss of a loved one, vehicular accident) (Olf et al., 2006) and a sample of Vietnam combat veterans (Yehuda et al., 1990) reported no significant effect of depression on cortisol outcomes with comorbid PTSD. In contrast, some other studies reported elevated basal cortisol as a result of an interaction effect between PTSD comorbid depression (e.g., Yehuda, Halligan, & Bierer, 2002; Young & Breslau, 2004b; Young et al., 2004). For instance, in a large community epidemiologic study of 516 individuals Young and Breslau (2004b) found that individuals with a lifetime PTSD diagnosis, exhibited elevated salivary cortisol, but only when they had a comorbid diagnosis of lifetime MDD. None of the other conditions i.e., exposure to trauma, lifetime diagnosis of PTSD or MDD alone were associated with alterations in basal cortisol. Similar findings were also reported by Yehuda, Halligan, and Bierer, (2001) in a sample of Holocaust survivors. The preponderance of evidence suggests that the PTSD and MDD comorbid condition may represent a specific subtype with a neuroendocrine profile distinct from a diagnosis of PTSD alone.

In addition to findings about basal cortisol levels in PTSD, an examination of the diurnal pattern of cortisol also provides information about specific aspects of HPA axis dysfunction in PTSD and their relationship to diagnostic classification and trauma characteristics.

Diurnal Rhythm of Cortisol

Cortisol facilitates the alert state upon awakening in animals and humans. It has a natural circadian rhythm in humans, with high levels in the morning followed by a steady decline to low levels at night. In general studies have found that individuals with PTSD tend to have significantly lower cortisol levels at several points during the circadian rhythm particularly in the early mornings and late evening (de Kloet et al., 2007; Rohleder, Joksimovic, Wolf, & Kirschbaum, 2004; Wessa et al., 2006; Yehuda, 2002). However, studies have provided mixed evidence about whether the blunted cortisol is seen in the awakening response (de Kloet et al., 2007; Rohleder et al., 2004; Yehuda et al., 1996) or in the hour after awakening (Wessa et al., 2006). Findings have been replicated across different types of trauma including war veterans (de Kloet et al., 2007; Yehuda et al., 1996) and refugees (Rohleder et al., 2004) In addition, at least one study reported similar findings for individuals diagnosed with PTSD as well as trauma exposed veterans without PTSD (de Kloet et al., 2007).

There is limited evidence to suggest that these outcomes are likely related to trauma characteristics such as latency of exposure and chronicity of the trauma. For instance, it is notable that the trauma exposures in each of the aforementioned studies represent multiple incidents of trauma exposure with a considerable latency period between exposure and cortisol assessment. However, in a study of a shelter population of IPV victims Johnson and colleagues (Johnson et al., 2008) examined the impact of PTSD severity and abuse chronicity on the cortisol awakening response in a sample of 52 battered women. Findings suggested that IPV-related PTSD and abuse chronicity have different, opposite, effects on waking salivary cortisol in battered women. While PTSD severity was associated with significantly greater cortisol output in the first hour after

awakening, chronic abuse was associated with lower total cortisol output in the first hour after awakening. While the association between chronic abuse and lower cortisol output after awakening is consistent with other studies and the enhanced negative feedback mechanism, the association between IPV related PTSD severity and cortisol in the first hour after awakening could be understood as reactivity of the HPA axis in response to more recent IPV experiences i.e., since this was a shelter population for whom the latency of IPV exposure was relatively short and the experience of being in the shelter itself implied safety concerns that the association with cortisol could reflect reactivity to more recent and/ or current stressors, which is consistent with findings from studies examining more recent forms of trauma exposure (Lemieux & Coe, 1995; Maes et al., 1998; Rasmussen et al., 2001; Resnick et al., 1995) and also with findings suggesting greater cortisol reactivity to day to day stressors in PTSD (Yehuda, 2002).

Research examining the diurnal pattern of cortisol also suggests that one key feature of PTSD is a pattern of greater cortisol reactivity. For instance, Yehuda and colleagues (1996) conducted a comprehensive circadian rhythm analysis through plasma cortisol sampling every 30 minutes over a 24 hour period under controlled laboratory conditions, with three groups of patients, combat veterans with PTSD, veterans and civilians with MDD, and non psychiatric control subjects. Individuals with PTSD displayed a more dynamic range of cortisol concentrations such that the lower the trough (lowest point in the circadian rhythm) among PTSD individuals the greater the time spent at the zenith (peak response), the lower the mean levels of basal cortisol, even though there was no significant difference in mean levels of peak responses between the PTSD and comparison control group. This pattern was found to be distinct from MDD

individuals who had higher levels of basal cortisol and an elevated trough. Similar findings have also been reported by Thaller et al. (1999) and Hoffman et al. (1989), who compared peak and trough responses, which also indicated great variability between these points relative to control comparisons.

Researchers suggest that these findings of an increased range of cortisol in PTSD indicate that the HPA axis may be hyperresponsive to stress related cues, including day to day environmental challenges. Relatedly studies assessing challenged cortisol to investigate HPA reactivity also yield mixed evidence, but a preponderance of evidence indicates support for cortisol hypersuppression in individuals with PTSD.

Challenged Cortisol

Several studies have examined HPA axis reactivity by examining challenged cortisol either in the face of a psychosocial stressor (e.g., Bremner et al., 2003; Carpenter et al., 2007; Elzinga et al., 2008; Heim et al., 2000; Santa Ana et al., 2006; Simeon, Knutelska, Yehuda et al., 2007) or more commonly through a chemical challenge such as the DST (e.g., Atmaca, Kuloglu, Tezcan, Onal, & Ustundag, 2002; Griffin et al., 2005; Newport, Heim, Bonsall, Miller, & Nemeroff, 2004; Stein et al., 1997; Yehuda, Golier et al., 2004; Yehuda, Halligan et al., 2004; Yehuda, Halligan, Grossman, Golier, & Wong, 2002). However, only a handful of studies have used psychosocial or cognitive stressors to assess challenged cortisol in adults with trauma exposure. Three of these studies examined challenged cortisol across various diagnostic groups in the context of childhood trauma (Bremner et al., 2003; Heim et al., 2000; Santa Ana et al., 2006) and two studies have examined such outcomes in preclinical samples of individuals without current psychiatric diagnosis (Carpenter et al., 2007; Elzinga et al., 2007). One other study

has examined different diagnostic groups using a combination of a psychosocial stressor and DST primarily focusing on the effects of dissociative disorders/ symptoms (Simeon, Knutelska, Yehuda et al., 2007)

Methodological limitations discussed in the context of basal cortisol studies also extend to studies with challenged cortisol with a lack of controls for comorbid symptoms, small sample sizes, and the lack of appropriate comparison groups. It should also be noted that most studies examined populations with early and/or chronic forms of trauma. Taken together these findings generally indicate a blunted response to stressors.

For instance, Heim and colleagues (2000) examined cortisol outcomes in women after sexual and physical abuse in childhood (defined as prior to their first menses). Forty-nine women were recruited into 4 groups: no history of childhood abuse or psychiatric disorder ($n=12$), those diagnosed with MDD with a childhood history of abuse ($n=13$), those without a diagnosis of MDD with a childhood history of abuse ($n=14$), and those with a current diagnosis of MDD without a childhood history of abuse ($n=10$). Using the Trier Social Situations Test (TSST) as a psychosocial stressor, the study reported significant group differences such that women with a history of childhood abuse and depression exhibited higher cortisol responses than all other groups. The four groups also differed with respect to cortisol concentrations such that abused women with current depression had increased levels of cortisol compared to all other groups. This study did not control for symptoms of anxiety and PTSD, and women with a history of childhood abuse had current comorbid symptoms, thus these findings should be interpreted with caution, as they do not reflect the unique or independent effects of depressive symptoms alone. In another study, which examined the effect of PTSD, in a

mixed sample of men and women, as a result of trauma in childhood (defined as prior to age 18) ($n=25$) were compared with individuals with PTSD, as a result of trauma in adulthood (defined as after age 18) ($n=33$), and healthy controls ($n=31$), on a cold-water immersion task (Santa Ana et al., 2006). Subjects with childhood trauma had lower cortisol at baseline and all post-task measurements. They also did not show decline in cortisol over a 2-hour post stress period as seen in individuals with trauma exposure in adulthood and controls.

In another study by Carpenter and colleagues (2007) also used the TSST as a psychosocial stressor, to assess the effects of significant childhood maltreatment in a nonclinical sample of healthy adults (defined as individuals without a current psychiatric diagnosis). Two groups of participants included a group with moderated to severe childhood maltreatment ($n=23$) and another with subjects without such a history ($n=27$). The study found that compared with the group without the childhood history of maltreatment, the maltreated group showed significantly lower baseline to peak change responses, and no differences were found with respect to baseline cortisol concentrations. These findings suggest blunted HPA axis responsivity to a psychosocial stressor, even though subjective ratings of anxiety induction in both groups were equivalent. Since participants in this study did not meet criteria for PTSD or MDD or other psychiatric diagnoses, it is possible that the findings represent enduring outcomes in the face of childhood maltreatment. Alternately, the findings may represent functioning in a population of maltreated individuals with subthreshold but not diagnostic levels of psychopathology. In the third study which examined cortisol outcomes in the context of childhood abuse and PTSD, Bremner and colleagues (2003) reported no differences

between the PTSD ($n = 23$) and healthy control groups ($n = 18$), in a mixed sample of men and women. Although the PTSD had several comorbid diagnoses of mood- and anxiety-related disorders the study did not control for the effects of concurrent diagnoses or possible effects of daily stressors. Also subjects were included if they had experienced child abuse, defined as abuse before the age of 18 such as rape, molestation physical assault etc., thereby including a wide time line of exposure to traumatic events.

Finally, Elzinga and colleagues (2008) assessed cortisol in a college population of healthy women and men (i.e., without current psychopathology) but with a history of high ($n=33$) to low ($n=47$) childhood abuse using the TSST. Consistent with prior studies, results indicated a blunted cortisol response among individuals with a history of abuse compared to those with low or no such history. The groups did not differ with respect to subjective ratings of anxiety of the task and baseline cortisol. However, it is notable that these findings were primarily driven by the men in the sample, and women did not demonstrate the blunted responses. As the authors note these findings in women could be confounded by the effects of contraceptives or menstrual cycle, which was not controlled for in this study.

Several studies have examined HPA reactivity and challenged cortisol using a chemical challenge (e.g., Atmaca et al., 2002; Griffin et al., 2005; Lindley et al., 2004; Newport et al., 2004; Stein et al., 1997; Yehuda, Golier et al., 2004; Yehuda, Halligan et al., 2004; Yehuda, Halligan, Grossman et al., 2002). Despite some inconsistencies in findings, a majority of these studies are consistent with findings from studies employing psychosocial stressors suggesting cortisol hypersuppression. For instance, Stein and colleagues (1997) examined plasma cortisol in 19 women with severe childhood sexual

abuse (which included various diagnoses and comorbid conditions that are common sequelae of childhood trauma such as MDD, dissociation disorders, and PTSD) and 21 control women, in a low dose DST and reported that the abused group had significantly enhanced suppression of plasma cortisol. Similar findings were also reported by other researchers (Kellner, Yehuda, Arlt, & Wiedemann, 2002; Yehuda, Golier et al., 2004) in samples of combat war veterans and Holocaust survivors. These findings of challenged cortisol in PTSD populations are significantly different from evidence for cortisol non suppression in populations with MDD, suggesting different neuroendocrinological profiles across diagnoses.

Finally, one study examined the effects of TSST and DST in a mixed sample of men and women with dissociative disorders, PTSD, and healthy controls (Simeon, Knutelska, Yehuda et al., 2007). Although findings from this study suggested no differences between the three groups, with respect to cortisol reactivity to the TSST, the group underwent the TSST while the HPA axis was still under the influence of the DST, making it difficult to interpret the findings.

In sum, findings from studies examining HPA axis sensitivity have produced somewhat mixed evidence, as a result of multiple methodological issues, and the role of frequently co-occurring mental health conditions in trauma exposed populations, which are still being explored. However, a majority of the findings thus far, indicate evidence for cortisol hyper suppression in trauma exposed populations.

Studies examining IPV and cortisol outcomes

With respect to cortisol outcomes in adult IPV victims, only six studies have examined cortisol outcomes in IPV victims (Griffin et al., 2005; Inslicht et al., 2006;

Johnson et al., 2008; Pico-Alfonso et al., 2004; Seedat et al., 2003; Young et al., 2004). Seedat and colleagues (2003) examined the basal cortisol levels in female IPV victims. The sample included 10 subjects with a current diagnosis of PTSD, 12 subjects without a current or lifetime diagnosis of PTSD, and 16 control subjects. The study found IPV to be the primary distinguishing factor across groups, such that individuals exposed to IPV demonstrated significantly lower levels of basal cortisol compared to controls, regardless of a PTSD diagnosis. However, it is notable that the IPV exposed group also differed significantly with respect to a childhood history of abuse/ trauma from the control group and the analyses did not control for the effects of childhood trauma suggesting that the observed outcomes could be a result of more chronic exposure to trauma, starting early in the developmental lifespan.

In contrast, in a later study Pico-Alfonso and colleagues (2004) reported elevated cortisol outcomes after controlling for age, smoking, pharmacologic treatment, and lifetime history of victimization. The study compared salivary cortisol outcomes between women exposed to physical ($n=70$) or psychological ($n=46$) violence and nonabused control women ($n=46$). Findings suggested that IPV victims had more severe symptoms of depression, anxiety, incidence of PTSD and higher levels of evening cortisol compared with control women, regardless of the type of abuse. IPV was found to be the main predictor of changes in hormonal levels and diagnostic status did not have a mediating effect on the impact of IPV on hormonal levels. However, this study did not examine the effects of PTSD comorbid with MDD, and it is unclear how many individuals had comorbid diagnoses, which has been associated with elevated level of cortisol (e.g., Young & Breslau, 2004a; Young & Breslau, 2004b). In addition, this sample included

women who were currently in abusive relationships unlike the sample in Seedat et al.'s (2003) study, and hence these findings could reflect the effects of concurrent stressors.

In a different study, Griffin and colleagues (2005) compared plasma cortisol of female IPV survivors with PTSD ($n = 15$), with PTSD and depression ($n=27$), those without PTSD or depression diagnoses ($n=8$), and a matched group of nontraumatized participants ($n=14$). IPV survivors with PTSD, regardless of comorbid depression, had significantly lower baseline morning cortisol compared to healthy controls and trauma survivors without diagnoses. Additionally, this is the only study to date that has examined challenged cortisol outcomes in IPV victims. The study found that IPV survivors with only a PTSD diagnosis showed significantly greater cortisol suppression to a low-dose DST compared to the healthy controls and the comorbid group, consistent with the enhanced negative feedback inhibition in PTSD.

In another study, Inslicht and colleagues (2006) examined salivary cortisol outcomes in female IPV victims with lifetime (current or remitted) PTSD ($n=29$) and in women who were exposed to IPV but never developed PTSD ($n=20$). This study found that women with lifetime PTSD had significantly higher cortisol levels across the day compared to abuse-exposed participants without PTSD, after controlling for age, depression, severity, and latency of abuse. However, the study did not control for concurrent stressors nor did it include a control comparison group of non abused women.

In a study of low income women Young and colleagues (2004) found no differences between women who were recently assaulted versus those that IPV. They also reported no effects on cortisol secretion for IPV if the last exposure occurred more than 1 year prior to cortisol assessment. However the sample of women who had experienced

IPV was very small subset ($n=17$) of a larger study. Thus, it is possible that the small sample size restricted detection of any significant group differences. Finally, Johnson and colleagues (2008) examined the waking response in IPV victims and found that PTSD severity and abuse chronicity have different, opposite, effects on waking salivary cortisol in battered women. While chronicity was associated with low awakening cortisol, severity of trauma was associated with increased cortisol concentrations.

In sum, only a handful of studies have examined cortisol outcomes in battered women. Taken together the findings are relatively mixed and present various methodological differences and shortcomings. Notably most of the studies have not examined the role of any theoretically informed trauma characteristics on cortisol outcomes except for the role of trauma chronicity, which has typically been limited to the experiences of IPV alone, despite evidence for the presence of other types of abuse exposure in early developmental stages in such a population.

CHAPTER 4

Rationale and Hypotheses

This study proposes to examine basal (defined as the average of the four salivary cortisol samples across the diurnal cycle), diurnal rhythm (i.e., trajectory of cortisol concentrations as assessed at four time points through the day) and challenged (defined as changes in cortisol concentrations in the context of a cognitive stress task) cortisol measures in a sample of pre-menopausal non pregnant adult women. The goal of the study is to examine these cortisol measures as a function of diagnostic classifications (PTSD only, PTSD and MDD comorbid, and subthreshold symptoms) and trauma characteristics (e.g., chronicity, earliest age of trauma exposure, cumulative trauma experiences based on types of trauma exposure, and social support). Thus, hypotheses are based on two organizing frameworks – (1) diagnostic classifications and (2) trauma related characteristics.

Specific Hypotheses

A. Basal Cortisol

Hypotheses based on diagnostic classification:

Women were grouped into (1) PTSD only, (2) PTSD and MDD, (3) Subthreshold symptoms, and (4) Control

1. Women with a lifetime PTSD diagnosis, without comorbid MDD, will have lower levels of basal cortisol compared to women in the control group.
2. Women with a lifetime PTSD diagnosis, without comorbid MDD will have lower levels of basal cortisol compared to the trauma exposed women with subthreshold symptoms.

3. Women with comorbid diagnoses of lifetime PTSD and lifetime MDD will have higher levels of basal cortisol compared to women in the control group.
4. Women with diagnoses of lifetime PTSD and comorbid lifetime MDD will have higher levels of basal cortisol compared to the trauma exposed women with subthreshold symptoms.

Hypotheses based on trauma characteristics:

Groupings for trauma characteristics are proposed to be based on naturally occurring variation found in the sample and did not form the basis for recruitment into the study.

The following group differences are hypothesized based on chronicity:

Women were grouped to distinguish 1) women with childhood abuse and IPV, 2) women with IPV only but no history of childhood abuse, and 3) control comparison group.

5. Women with a childhood history of trauma and IPV will have lower levels of basal cortisol compared to women in the control group.
6. Women without a childhood history of trauma and only a history of IPV in adulthood will have higher levels of basal cortisol compared to women in the control group.

The following is hypothesized based on age of trauma exposure:

Only for women with childhood abuse and IPV.

7. Age of trauma exposure will be positively related to basal cortisol such that earlier trauma exposure will be related to lower levels of basal cortisol.

The following is hypothesized based on cumulative trauma exposure and relationship to perpetrator:

Only for women with childhood abuse and IPV.

8. Childhood trauma perpetrated by primary caregivers and unrelated but close individuals will result in more significant dysfunction as seen in lower levels of basal cortisol compared to unrelated perpetrators.

The following is hypothesized based on social support:

9. Social support will moderate the impact of trauma on cortisol outcomes such that women exposed to lifetime trauma without social support will have lower cortisol outcomes relative to women with social support from their network.

B. Diurnal Rhythm of Cortisol

Hypotheses based on diagnostic classification:

Women were grouped into (1) PTSD only, (2) PTSD and MDD, (3) Subthreshold symptoms, and (4) Control

1. Women with a lifetime diagnosis of PTSD will have significantly lower levels of cortisol at all four time points through day compared to women in the control group.
2. Women with comorbid diagnoses of lifetime PTSD and MDD will have higher levels of cortisol at all time points through day compared to women in the control group.
3. Women with comorbid diagnoses of lifetime PTSD and MDD will have significantly higher levels of cortisol at all time points through day compared to women in the PTSD only group.
4. Women with a lifetime diagnosis of PTSD will show a greater range of cortisol responses as assessed through the difference between the peak to trough cortisol assessments.

5. No specific hypotheses are made with respect to the women with subthreshold symptoms. Exploratory analyses will examine trajectories of diurnal cortisol outcomes in this group.

Hypotheses based on trauma characteristics:

The following group differences are hypothesized based on chronicity:

Women were grouped to distinguish 1) women with childhood abuse and IPV, 2) women with IPV only but no history of childhood abuse, and 3) control comparison group.

6. Women with a childhood history of trauma and IPV will have significantly lower levels of cortisol at all four time points through the day compared to women in the control group.
7. Women without a childhood history of trauma and only a history of IPV in adulthood will have higher levels of cortisol at all four time points through the day compared to women in the control group.

The following is hypothesized based on age of trauma exposure:

Only for women with childhood abuse and IPV.

8. Early trauma exposure will be positively related to the awakening cortisol response as well as cortisol at later points in the day.

The following is hypothesized based on cumulative trauma exposure and relationship to perpetrator:

Only for women with childhood abuse and IPV

9. Childhood trauma perpetrated by primary caregivers and unrelated but close individuals will result in more significant dysfunction as seen in lower levels of basal cortisol compared to unrelated perpetrators.

The following is hypothesized based on social support:

10. Social support will moderate the impact of trauma on cortisol outcomes such that women exposed to lifetime trauma without social support will have lower cortisol outcomes at each time point relative to women with social support from their network.

C. Challenged cortisol

Hypotheses based on diagnostic classification:

Women will be grouped into (1) PTSD only, (2) PTSD and MDD, (3) Subthreshold symptoms, and (4) Control

1. Women with a lifetime diagnosis of PTSD will have lower levels of cortisol in response to a stress manipulation relative to the control group.
2. Exploratory analyses will examine challenged cortisol outcomes in women with comorbid lifetime diagnoses of PTSD and MDD relative to the control group.
3. Exploratory analyses will examine challenged cortisol outcomes in women with subthreshold symptoms.

Hypotheses based on trauma characteristics:

The following group differences are hypothesized based on chronicity:

Women will be grouped to distinguish 1) women with childhood abuse and IPV, 2) women with IPV only but no history of childhood abuse, and 3) control comparison group.

4. Exploratory analyses will examine how trauma chronicity is related to challenged cortisol outcomes.

The following is hypothesized based on age of trauma exposure:

Only for women with childhood abuse and IPV.

5. Exploratory analyses will examine if and how age of trauma exposure is related to challenged cortisol outcomes.

The following is hypothesized based on cumulative trauma exposure and relationship to perpetrator:

Only for women with childhood abuse and IPV

6. Exploratory analyses will examine if and how perpetrator relationship is related to challenged cortisol outcomes.

The following is hypothesized based on social support:

7. Exploratory analyses will examine if and how social support moderates challenged cortisol outcomes.

CHAPTER 5

Methods

Participants

The sample consists of 92 women including 12 women for a control comparison group, who had no lifetime exposure to IPV or other interpersonal forms of trauma or lifetime diagnosis of mood disorders or PTSD. Additionally, 14 participants meet lifetime criteria for PTSD only, 43 participants meet lifetime criteria for PTSD and MDD, and 19 women were classified having subthreshold PTSD or PTSD and MDD symptoms, based on the DSM-IV-TR criteria (American Psychological Association, 2000). Four women met full criteria for MDD only and were removed from analyses. See Table 1.

Procedures

Participants from diverse ethnic/cultural and socioeconomic backgrounds were recruited from the greater Lansing area. Specifically, 52% of the sample identified themselves as being Caucasian, 25% African American, 8% Hispanic, 7% Asian, 6% Multiracial and 2% Biracial. Additionally, 25% of the sample identified as having a High School or GED degree, 51% reported completing “some college”/ trade school/ Associates degree, 13% reported completing a bachelor’s degree, and 11% reported completing “some graduate school”/ a graduate degree. The mean age of the sample was 27 years (range: 18.7 years – 41.9 years) and the mean monthly income was \$2332.31 (range: \$00.00 - \$12,500). See Tables 2, 3, and 4 for demographic information about the sample organized by diagnostic group status.

Participants were recruited by informing key community stakeholders (e.g., Personal Protection Order Office, Work First, Salvation Army, Ballentine’s Stepping

Stones, Cristo Rey, Faith Based agencies and churches) via letters, flyers, and formal appointments with directors of programs. Flyers were also posted in various community locations such as grocery stores, laundromats, hospital emergency rooms (e.g., Sparrow and Ingham Hospital Emergency Rooms), and other community health facilities (e.g., Women's Center of Greater Lansing, Women's Personal Growth and Therapy Center, Clinton Eaton Ingham Community Mental Health Counseling Center, Insight Recovery Center). To over sample for IPV victims, particularly women with more severe IPV exposure, recruitment efforts were focused on a local domestic violence shelter – End Violent Encounters Inc. (EVE). In addition to posting flyers at EVE, recruitment announcements were also made at their support group meetings. Approximately 28 % of the sample reported living in a domestic violence shelter at some point in their life ($n = 25$), including 11% ($n=10$) who reported living in the shelter during the study. Only 7% ($n = 6$) of the sample reported being in an abusive relationship during the study.

Interested participants who contacted the MSU project office underwent a 15 minute phone screen to assess for multiple inclusion and exclusion criteria. To be included all women had to be (1) English-speaking, (2) between 18 to 41 years old, (3) not pregnant, (4) not lactating or breast feeding, (5) no diagnosis of neuroendocrine disorders (e.g., Dai, Buijs, & Swaab, 2004) or endocrine disorders (Cushings, Addisons Diseases), cancer, or those currently receiving cancer therapy (see Findling & Raff, 2006; Golden et al., 2007), (6) free of any lifetime diagnoses of a psychotic disorder or cognitive impairments, (7) could not meet criteria for substance use dependence or abuse in the past three months, (8) been involved in a heterosexual romantic relationship for at least 6 weeks in the past 2 years, and (9) have a minimum grade 6 education to ensure

that informed consent could be obtained. Additional criteria for IPV exposed women included (1) the last IPV experience should have occurred within the past two years and (2) the women had to endorse minimum two symptoms of PTSD or PTSD and MDD criteria on the phone screen. The study was designed to recruit groups based on diagnostic groups. Hypotheses and analyses based on trauma characteristics were based on naturally occurring variation found in this population, thus recruitment did not screen for trauma characteristics.

Trained undergraduate assistants conducted the initial intake over phone, which began with a standardized introduction to the study, followed by a screen for the aforementioned inclusion and exclusion criteria. See Appendix C for the screening procedure. Additionally, the Alcohol Use Disorders Identification Test (AUDIT) (Babor, de la Fuente, Saunders, Grant, & World Health Organization, 1992) measure was used to screen for alcohol use and dependence. A score of 8 or higher indicates the presence of alcohol dependence. Thus, to meet criteria for participation in the study the women would have to score 7 or lower on the AUDIT. Women who reported drug use for non medical reasons more than 1-2 times and / or indicated impairment in daily functioning more than 1-2 times in the past 3 months were deemed ineligible for the study.

Eligible participants were scheduled for an interview at the MSU research office. However, residents of EVE were offered the option of completing the interview at EVE itself. This was done to facilitate recruitment and participation of a shelter population who represent a high risk population of interest with limited resources and differ from community populations experiencing IPV (e.g., Levendosky et al., 2004). All participants

were scheduled at 05.30 pm in the evening to standardize time of cortisol sampling during the interview process.

Trained graduate and undergraduate research assistants conducted interviews. All graduate students were advanced graduate students who had completed their Masters degree in clinical psychology and were trained in the administration of Structured Clinical Interview for DSM-IV Axis I & II Disorders (SCID) (First, Spitzer, Gibbon, & Williams, 1996a) through a workshop in the department of psychology at Michigan State University. Additionally, all graduate assistants were observed during two mock administrations of the SCID to assess for reliability. Graduate research assistants also served as evaluative observers for the psychosocial stress task with participants and were trained on the requisite protocol and observed during two mock interviews.

Undergraduate assistants were trained in administering all remaining measures by having them observe a mock interview, followed by an administration of the interview to the principal investigator, and finally the principal investigator observing the assistant administer the interview to a mock participant. To ensure fidelity to the interview protocol, undergraduate assistants were observed twice in vivo during administration of interviews with participants – the first time within the first three interviews and on a second occasion, after approximately six interviews.

All participants began by completing a consent form approved by the Institutional Review Board (IRB), informing them of their rights as participants, and the steps taken by the project to ensure their confidentiality (e.g., identifying information will not be stored with their data and will be kept in a secure location in accordance with IRB rules). Participants were informed about the payment they would receive for their time spent in

the interview. A total of \$30 was paid to the first 55 participants; \$20 upon completion of the interview and \$10 upon obtaining the three saliva samples from the take home kit. Subsequently, \$40 was paid to the remaining participants; \$25 upon completion of the interview and \$15 upon obtaining the three saliva samples from the take home kit. Payment was increased to facilitate recruitment of the required sample size within the available time line, following IRB approval.

Following the requisite consent forms participants completed a brief screener about any foods and drinks they consumed, and how many cigarettes they had smoked in the past few hours and a demographic questionnaire. See Appendix C. Next, participants were given a five minute rest period following which they gave their first saliva sample (i.e., baseline saliva sample). Next, the cognitive stress protocol [i.e., an adapted version of the Trier Social Stress Test (TSST) (Kirschbaum, Pirke, & Hellhammer, 1993)] was administered to induce moderate psychosocial stress under laboratory conditions (see Appendix C for cognitive stress task protocol details), to provide estimates of HPA reactivity. The TSST has been used in a number of studies assessing the effects of psychosocial stress and has been found to effective in eliciting a stress response (e.g., Elzinga et al., 2008; Simeon, Knutelska, Yehuda et al., 2007).

Upon completion of the cognitive stress challenge task, three saliva samples were collected – immediately after the stress task, and 10- and 25-minutes post completion of the stress task. The interviewer noted the exact time of each saliva sampling. Saliva was collected from participants following procedures developed by Granger and colleagues (2007). See Appendix C for protocol for saliva collection. During the post task period participants were asked to rate their perceived stressfulness of the cognitive challenge

task and were debriefed about the cognitive challenge. See Appendix C for the cognitive challenge and debriefing protocol.

Next, the graduate assistant conducted a structured clinical interview to assess for current and lifetime PTSD and MDD (SCID-I) and borderline personality disorder (SCID-II). After this, the undergraduate assistants helped participants complete questionnaires related to domestic violence, childhood history of trauma, daily stressors, PTSD, anxiety, depression, dissociative symptoms, and social support. In addition participants completed two brief checklists 1) to assess for the presence of any autoimmune disorders and to get information about related treatments/ medication use (see Appendix C) and 2) to assess their smoking habits and substance use in the last 48 hours (see Appendix C). Finally, interviewers provided participants with take home kit and explained the protocol of obtaining saliva samples for the following 48 hours (one sample at 9 pm the following night, a second sample immediately after awakening, and a third sample within 45 minutes of awakening sample). Assistants also made plans to collect samples from the participants, which could be any location of the participant's choosing.

Cortisol enzyme-immunoassay (EIA)

Cortisol were assayed using a commercially available enzyme immunometric assay specifically designed for use with saliva without modification to the manufacturer's recommended protocol (Salimetrics, 2008). The assay (25 µl test volume) is 510K cleared (US FDA) as a diagnostic measure of adrenal function: range of detection is from 0.003 to 3.0 µg/dl. The assay is highly specific to cortisol, with less than 0.5% cross-reactivity for other steroids. All assays were performed in duplicate by the principal

investigator, with assistance from two undergraduate assistants, all of whom were trained and supervised by Dr. Lonstein. All assay plates had standard curve correlations of 0.90 or higher. The mean inter assay variation was 12.8 per cent and intra assay variability was 7.75 per cent.

Measures

Demographics. Participants completed a demographic questionnaire to identify information about their age, race/ethnicity, education level, and family income. See Appendix C for a copy of the measure.

Alcohol Use Disorder Identification Test (AUDIT, Babor & Grant, 1989). This measure was developed as a brief multi-cultural screening tool for the early identification of at-risk problem drinkers (Babor et al., 1992). It was created by a working group of the World Health Organization by choosing questions that discriminated high risk drinkers in a six nation study. It contains a series of ten questions that include three questions on use, four dependence questions, and three items about problems. The 10 questions are designed to be administered as an interview or as pencil and paper questionnaire. A cut-off score of 11 or more generally indicates dependence while a score of 8 or higher indicates high risk for abuse.

Severity of Violence Against Women Scales (SVAWS, Marshall, 1992). The SVAWS is a 46-item instrument designed to assess IPV, defined as adult male violence and threats of violence against female romantic partners. The questionnaire is composed of nine subscales: symbolic violence; mild, moderate, and serious threats of physical violence; minor, mild, moderate, and serious actual physical violence; and sexual violence. Example items include, "Hit or kicked a wall, door, or furniture," "Threatened

to hurt you,” “Pushed or shoved you,” “Beat you up,” and “Physically forced you to have sex.” Ratings are on a 4-point scale with response choices ranging from “Never” to “Many times.” The SVAWS has good psychometric properties. Marshall (1992) reported coefficient alphas for the nine subscales ranging from .89 for symbolic violence to .96 for both mild and serious physical violence. In the current study the reliability coefficient was 0.98. In order to exclude mild violence (e.g., “Shook a finger at you”), and examine the effects of physical and sexual violence, only item numbers 14 and higher were used to create a sum score as an indicator of the severity of IPV. A dichotomous score was created based on the sum score to indicate the presence (i.e., any score greater than 0) or absence (i.e., sum score equal to 0) of IPV.

Childhood Trauma Questionnaire – Short Form (CTQ, Bernstein et al., 2003) The CTQ is a 28-item, self-report measure for retrospectively assessing multiple types of abuse and neglect experienced during childhood and adolescence. The questionnaire has five clinical scales each consisting of five items: physical, sexual, and emotional abuse, and physical and emotional neglect. The instrument also includes a 3-item minimization/denial scale to detect under-reporting of maltreatment. Example questions include, “People in my family hit me so hard it left me with bruises or marks,” “Someone tried to touch me in a sexual way,” and “People in my family called me things like ‘stupid,’ ‘lazy,’ or ‘ugly.’” Items are rated on a 5-point Likert scale with response options ranging from “Never” to “Very Often True.” The CTQ is a reliable and valid measure of childhood trauma across a variety of individuals who differ in terms of age, sex, ethnicity, socioeconomic status, psychopathology, and life experiences. Test-retest reliabilities range from .79 for the physical neglect scale to .86 for the entire measure in a

sample of adult substance abusers (Bernstein & Fink, 1998) Bernstein and Fink (1998) reported internal consistency reliability coefficients ranging from a median of .66 for physical neglect to a median of .92 for sexual abuse across a variety of clinical and non-clinical samples. In the current study the reliability coefficient was 0.86 for the physical abuse scale and 0.93 for the sexual abuse scale. A sum score was calculated using endorsements of physical and sexual abuse items. Also, a dichotomous score was created to indicate the presence (i.e., any score greater than 0) or absence (i.e., sum score equal to 0) of childhood abuse or neglect.

Posttraumatic Diagnostic Scale (PDS, Foa, 1995). This 38-item self-report scale assesses all symptoms from all 6 criteria sets of PTSD based on the DSM-IV. The PDS begins with a list of 13-items that screen for the experience of a traumatic event including sexual victimization during childhood or adulthood, accident/fire, torture, and life threatening illness. If more than one event is endorsed participants are asked to identify the traumatic event bothered her the most. Participants who did not endorse any items in this section do not complete the subsequent sections of the PDS.

To assess for feeling of danger for DSM-IV-TR criterion A, participants were asked to answer 6-items related to each traumatic event (e.g., thinking that her life or someone else's life was in danger and feeling helpless or terrified) using a yes-no format.

Characteristics of Traumatic Events: To assess for severity of the traumatic event, participants were also asked to report severity of injuries, how traumatic the event was at the time for each event endorsed. Each item was measured on a 4-point Likert scale ranging from "Not at all" to "A lot." Items were be summed for the worst traumatic event to produce a traumatic event severity score. Participants were asked to report the age at

which each endorsed traumatic event occurred. The youngest age at the time of the traumatic event that the participant identifies as being the most distressing was utilized to assess earliest age of trauma exposure.

In the case of interpersonal traumatic events, participants were asked to report their relationship to the perpetrator, which included 3 options - family member (e.g. father, brother, step-father), family friend or neighbor, stranger, except item 3 in which participants could also endorse spouse/ partner as a perpetrator. In addition, due to high rates of revictimization participants were asked how often she has experienced each event. Two categorical variables were created to indicate the amount of cumulative trauma exposure based on relationship to perpetrator. First, a categorical variable was created using two different categories of attachment related trauma – 1) intimate partner violence and 2) familial childhood abuse and intimate partner violence and 3) multiple types of trauma exposure including attachment related trauma (intimate partner violence or intimate partner violence and familial childhood abuse) and non attachment related forms of trauma exposure (e.g., house fire, accident, tornado). A second categorical variable was created as follows - 1) attachment related (intimate partner violence or intimate partner violence and familial childhood abuse) and 2) non attachment related forms of trauma exposure.

The PDS is reported to have high test-retest reliability for total symptom severity ($\alpha=.82$) and for each of the symptoms clusters (α range= .77-.85). It was also found to have high convergent validity with the PTSD portion of the SCID, $\kappa=.65$ (Foa, 1995). In the current study the reliability coefficient was 0.98.

Dissociative Experiences Scale (DES, Carlson & Putnam, 1993). The DES is a 28-item self-report measure that helps identify dissociative psychopathology. It inquires about dissociative experiences in the daily lives of subjects. Example items include, “Some people have the experience of finding themselves in a place and having no idea how they got there”, “Some people find that they have no memory for some important events in their lives (for example, a wedding or graduation)”, and “Some people find that they become so involved in a fantasy or daydream that it feels as though it were really happening to them”. The scale has been used in clinical and non clinical adult populations including samples of individuals diagnosed with PTSD. The measure has been reported to have high reliability and validity coefficients. For instance Rosenthal and Rosnow (1991) reported test retest reliability coefficient of .84 and Frischolz and colleagues (1990) reported a coefficient of .96. The scale has also been shown to have good construct and criterion validity (Carlson & Putnam, 1993). In the current study the reliability coefficient was 0.94. The measure uses a scale from 0 to 100 and a total score is calculated by averaging scores across all 28 items. The total score will be utilized as a measure of the extent of dissociation.

Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I, First et al., 1996a). The SCID-I is a semi-structured interview for diagnosing most major DSM-IV Axis I psychiatric diagnoses. It is one the most widely measures in research studies of psychopathology. This measure is well suited for both inpatient psychiatric patients as well as non-clinical populations, including female IPV victims (e.g., Griffin et al., 2005; Inslicht et al., 2006). The SCID-I contains specific modules corresponding to specific DSM-IV diagnoses that can be used independently of one another, depending on the

purpose of the study. For the current study the anxiety and mood-disorder modules were used to assess for DSM-IV diagnoses and symptoms. High test-retest and inter rater reliability and validity coefficients have been reported for both anxiety and mood-disorder modules (e.g., Shea, Zlotnick, & Weisberg, 1999; Zannarini et al., 2000). In the current study the reliability coefficient was 0.96 for current MDD, 0.95 for past MDD, and 0.96 for PTSD. A total symptom count for PTSD and MDD as well as a dichotomous score indicating the presence or absence of both PTSD and MDD were calculated. Next a categorical variable was created to identify the presence of PTSD, PTSD and comorbid MDD, individuals with subthreshold symptoms, and a control comparison group with no symptoms. It is notable that four individuals were classified as meeting criteria for MDD only. Due to the small sample size of the group, these individuals were removed from further analyses.

Structured Clinical Interview for DSM-IV Axis II Disorders (SCID-II, First, Spitzer, Gibbon, & Williams, 1996b) The SCID-II is a widely used semi-structured interview for making DSM-IV Axis II (Personality Disorder) diagnoses. Akin to the SCID-I, the SCID-II also contains specific modules corresponding to specific DSM-IV Axis II diagnoses that can be used independently of one another. For the current study the borderline personality disorder module will be used. High inter rater reliability ($k = 0.91$) and validity coefficients have been reported for the borderline personality disorder module (e.g., Maffei et al., 1997). In the current study the reliability coefficient was 0.94. A dichotomous score indicating the presence or absence of borderline personality disorder was calculated.

Daily Hassles and Uplifts Scale (DHUS, DeLongis, Folkman, & Lazarus, 1988).

The DHUS is a 53-item self measure that assesses the impact of events and relationships that affect individuals on a daily basis. Participants are asked to rate each item (e.g., friends, relatives, work load, church or community organizations, cooking, health) with respect to how much of a hassle or irritant each of them and to what extent they were an uplift in their life. This measure has been widely used and shown to have high reliability ($\alpha = 0.94$) (e.g., Day, Therrien, & Carroll, 2005). In the current study the reliability coefficient was 0.94. For this study item 4 “Your spouse” will be changed to “Your partner”. A sum score was calculated for the hassles to assess daily stressors.

Crisis Support Scale (CSS, Joseph, Andrews, Williams, & Yule, 1992). This 7-item measure assesses perceptions of received social support. Respondents are asked to rate their perceptions of 1) general current social support in past 4-6 weeks and 2) social support immediately following the traumatic event that bothered them the most. A 7-point Likert scale ranging from “Never” to “Always” is utilized to assess the reception of social support after the experience of a traumatic event. Examples of the items in the CSS are “Someone willing to listen,” “Sympathy and support from others,” and “Feeling let down.” In a review of 11 studies that used the CSS, Elklit and colleagues (2001) reported good internal consistency, discriminant validity, and a mean alpha of .74. In the current study the reliability coefficient was 0.91 for current social support scale and 0.81 for the trauma related social support scale. Items were summed to create two social support scores: social support at the time of the traumatic event and current social support.

CHAPTER 6

Results

Missing Data

Data were checked for missing values, expected range, and the presence of outlier values. With respect to cortisol data, missing data (i.e. nine participants who did not return the take home kit which equals 27 samples, i.e. 4% of total cortisol samples) and undetectable levels of cortisol values were substituted on a case by case basis, using mean values for the diagnostic group under which the participant was classified and the specific time of sampling that was missing (41 samples across 14 cases, i.e. 6.7% of total cortisol samples were mean substituted). Regarding outlier values above the mean (defined as three standard deviations above the mean of each diagnostic group) or salivary cortisol values beyond the normative human range, only one saliva sample was found to be an outlier. This data point was not substituted using mean substitution and was removed from pertinent analyses using listwise deletion. See Table 5 for descriptive statistics for cortisol values pre and post mean substitution.

Data transformation

Cortisol data were checked to determine whether the distribution of variables meets the assumptions of statistical tests. As is typical for cortisol data, cortisol distributions were found to be positively skewed. Hence, log transformed cortisol values using logarithmic transformations (to the base 10) were used for all analyses.

Hypotheses Testing

Prior to specific hypothesis testing, the shelter population was compared with IPV exposed women who completed the interview at MSU with respect to demographics (i.e.,

age, ethnicity, income, and education). The groups did not differ significantly with respect to these factors and were combined for all analyses. In the next step, diagnostic groups were compared using univariate Analysis of Variance (ANOVA) or chi-square tests to assess for differences in demographics (i.e., age, ethnicity, income, and education) and other factors known to affect cortisol concentrations that could confound findings (i.e., use of medication and contraception, dissociative symptoms, daily stressors, borderline personality diagnosis, time of saliva sampling, food and alcohol consumption, or cigarette smoking immediately prior to saliva sampling). Dissociative symptoms were found to significantly differ across diagnostic groups ($F_{3,84} = 5.40, p < .01$). See Table 6. Post-hoc analyses were conducted using the Games-Howell test to account for differences in sample size and variance of the groups, which indicated that the control group endorsed significantly fewer dissociative symptoms relative to all other trauma-exposed diagnostic groups (i.e. PTSD, PTSD and MDD, subthreshold groups). Also, the subthreshold groups endorsed significantly fewer dissociative symptoms than the PTSD and MDD comorbid group. Thus, dissociative symptoms were included as a covariate in all analyses that examined differences across all four diagnostic groups but was not included in analyses which only examined differences between trauma-exposed diagnostic groups.

Finally, due to the relatively high inter assay variation, which appeared to be driven by three assay plates, all analyses were also conducted using a subset of the data ($n=74$), after removing data from those three plates. Findings were consistent with data from the complete data set, and hence, findings from the complete data are presented.

Hypothesis for basal cortisol based on diagnostic classification

The first hypothesis examined differences across diagnostic groups with respect to basal cortisol concentrations. Analysis of Covariance (ANCOVA) indicated that there were no significant differences across diagnostic groups ($F_{3,83} = .379, n.s.$). However, dissociative symptoms were a significant predictor of basal cortisol values ($F_{1,83} = 4.683, p < .05$). See Table 8 and Figure 1 for mean levels of basal cortisol concentrations across groups. To further examine whether posttraumatic symptoms and depressive symptoms account for variation in basal cortisol concentrations, post hoc analyses using continuous data (i.e., sum scores of post traumatic stress symptoms and depressive symptoms) were entered in a step wise regression model to predict basal cortisol concentrations. In the final step dissociative symptoms was included in the model. The final model which included dissociative symptoms, indicated a trend towards significance ($p = .07$) and consistent with prior categorical analyses, only dissociative symptoms was a significant predictor of basal cortisol concentrations ($p < .05$).

Hypothesis for basal cortisol based on trauma characteristics: chronicity

The next hypothesis sought to examine whether differences in chronicity of trauma exposure accounted for variations in basal cortisol concentrations. A categorical chronicity variable was created to indicate whether individuals had not experienced trauma ($n=12$), versus trauma in adulthood only (i.e., intimate partner violence) ($n=21$), versus trauma in childhood and adulthood (childhood physical and/or sexual abuse and intimate partner violence) ($n=55$). Univariate F test indicated that there were no significant differences across chronicity groups ($F_{2,85} = .295, n.s.$). Based on prior findings related to the effects of dissociative symptoms on basal cortisol concentrations post hoc analyses were conducted to examine differences in dissociative symptoms

across chronicity groups. Univariate F test indicated that the three chronicity groups differed significantly. Games Howell post hoc test indicated that the group with no trauma exposure had significantly lower dissociation symptoms. However, groups with trauma exposure regardless of whether they experienced trauma in adulthood or more chronic trauma (i.e., both childhood and adulthood) did not differ from each other. Thus, as a next step, ANCOVA with chronicity groups as a predictor, and dissociative symptoms and age of participants as covariates [age was included as a covariate because it could be a confounding factor for abuse chronicity and is significantly correlated to chronicity ($p < .05$)]. Chronicity ($F_{2,83} = .532, n.s.$) and age were not significant predictors for basal cortisol. However, dissociative symptoms ($F_{1,83} = 4.648, p < .05$) predicted basal cortisol concentrations beyond chronicity and participant age.

Hypothesis for basal cortisol based on trauma characteristics: earliest age of trauma exposure

To assess whether the earliest age of trauma exposure affects basal cortisol concentrations, age of earliest trauma exposure was entered into a regression model with dissociative symptoms, which was found to be significantly correlated with earliest age of trauma exposure ($r = -0.213, p = .05$). Dissociative symptoms ($t = -2.173, \beta = -.246, p < .05$), but not earliest age of trauma exposure, was a significant predictor of basal cortisol concentrations.

Hypothesis for basal cortisol based on trauma characteristics: cumulative trauma exposure based on relationship to perpetrator

To examine whether relationship to perpetrator affects basal cortisol concentrations, two types of categorical variables used. First, a categorical variable

examining whether two different categories of attachment related trauma – 1) intimate partner violence ($n= 11$) and 2) familial childhood abuse and intimate partner violence ($n= 7$), and 3) multiple types of trauma exposure including attachment related trauma (intimate partner violence or intimate partner violence and familial childhood abuse) and non attachment related forms of trauma exposure (e.g., house fire, accident, tornado) ($n= 58$) was created. A second categorical variable which examined 1) attachment related (intimate partner violence or intimate partner violence and familial childhood abuse) ($n= 18$) and 2) non attachment related forms of trauma exposure ($n= 58$) was created. Univariate F tests did not indicate significant findings for either type of classifications for basal cortisol concentrations.

Hypothesis for basal cortisol based on trauma characteristics: social support

The effects of two types of social support on basal cortisol concentrations were assessed – 1) general levels of perceived social support and 2) perceived social support at the time of the event identified by the participant as being the most traumatic. Bivariate correlations indicated that neither type of social support is significantly correlated with basal cortisol levels. Univariate F tests indicated that neither type of social support is a significant predictor of basal cortisol.

Hypothesis for diurnal rhythm of cortisol based on diagnostic groups

Repeated measures ANCOVA were used to examine the effect of diagnostic groups, with dissociation as a covariate, on the diurnal rhythm of cortisol. The model indicated a significant effect for dissociative symptoms only ($F_{1,82} = 3.878, p=.05$) with a significant linear trend ($p <.01$). However, as noted earlier, this finding appears to be driven by the association between dissociation and awakening cortisol as dissociative

symptoms are not associated with cortisol concentrations at any other time in the day. See Table 9 for the between subjects effects across diagnostic groups and Figure two.

Additionally, there was a significant quadratic trend for time of day ($p < .001$). Graphical analyses indicates that all four groups showed a linear increase in cortisol concentrations from night to wake up to the 45 minutes post awakening sample and a decline in cortisol concentrations towards the evening as assessed through the pre cognitive stress task baseline sample.

Additional post hoc analyses were also conducted to examine whether diagnostic groups may predict cortisol variations at circumscribed times during the day. For instance there is evidence to suggest that trauma exposed groups, particularly those that have experienced chronic trauma exposure, may not show the typical increase in cortisol concentrations within the first hour of the day, relative to healthy control comparison groups (Johnson et al., 2008). To further assess this issue, difference scores were calculated by subtracting the 45-minutes post wake up cortisol concentration values from the wake up cortisol concentration values. Next, these difference scores were used as outcome variables in univariate F tests with diagnostic groups as predictors. This method has been used to assess change in cortisol concentrations in the literature (Simeon, Knutelska, Smith, Baker, & Hollander, 2007). Similar to prior findings the results did not indicate significant differences in changes in cortisol concentrations in the first hour of awakening between diagnostic groups. However, dissociative symptoms were a significant predictor of changes in cortisol concentrations within the first hour of waking up ($p = .05$). The relationship between dissociative symptoms and changes in cortisol concentrations is likely due to its significant correlation with the waking up sample ($r = -$

0.257, $p < .05$), as dissociative symptoms are not correlated with the 45 minutes post wake up sample or the difference scores.

Hypothesis for diurnal rhythm of cortisol based on trauma characteristics: chronicity

Repeated measures ANCOVA were used to examine the effect of chronicity, with dissociation as a covariate, on the diurnal rhythm of cortisol. The model indicated a trend towards a significant effect for dissociative symptoms ($F_{1,83} = 3.334$, $p = .07$) with a significant linear trend ($p < .01$), and no significant effects for chronicity. Post hoc analyses using difference scores were consistent with these findings, indicating significant effects for dissociative symptoms ($F_{1,83} = 3.849$, $p = .05$) but not chronicity ($F_{2,83} = .717$, *n.s.*).

Hypothesis for diurnal rhythm of cortisol based on trauma characteristics: earliest age of trauma exposure

Repeated measures *F* test was used to examine the effect of earliest age of trauma exposure. Consistent with analyses for basal cortisol, dissociation was included in the model. Analyses indicated a significant effect for dissociative symptoms ($F_{1,72} = 3.914$, $p = .05$) with a significant linear trend ($p < .01$), but no significant effects for earliest age of trauma exposure. Post hoc regression analyses using difference scores as the outcome variables suggested consistent findings, with a significant effect for dissociative symptoms ($p < .05$) but not earliest age of trauma exposure.

Hypothesis for diurnal rhythm of cortisol based on trauma characteristics: cumulative trauma exposure based on relationship to perpetrator

Repeated measures ANOVA, with previously described categorical variables was used to assess the effects of attachment- and non-attachment related trauma experiences

on the diurnal rhythm of cortisol. However, F tests were not significant for either predictor. Univariate analyses using difference scores as outcomes also did not indicate significant effects for perpetrator type on changes in cortisol concentrations in the first of hour of waking up.

Hypothesis for diurnal rhythm of cortisol based on trauma characteristics: social support

Bivariate correlations indicated that neither 1) general levels of perceived social support nor 2) perceived social support at the time of the most traumatic event is significantly correlated with cortisol concentrations at any point in the day. Thus, F tests indicated that neither type of social support is a significant predictor of the diurnal rhythm of cortisol. These findings were consistent with regression analyses using difference scores as outcome variables and social support as predictor variables.

Hypothesis for challenged cortisol based on diagnostic classification

Repeated measures ANCOVA was used to examine the effect of diagnostic groups, with dissociation as a covariate, on challenged cortisol. The model indicated a trend towards mean differences across diagnostic groups ($p=.07$). However, no other trends were found to be significant over time. See Table 10 for the between subjects effects across diagnostic groups and Figure three. To further assess changes in cortisol concentrations as a result of the cognitive stress task two difference scores were calculated – 1) difference between baseline cortisol concentrations and the saliva sampling immediately post task and 2) difference between the saliva sampling immediately post task and the final post task cortisol sampling (i.e., 25 minutes post task). These times were chosen based on the rationale that individuals would show the largest increase in cortisol concentrations between baseline and immediately post task.

Subsequent to the cortisol sampling post task individuals were debriefed about the stress task, and typically control subjects would show a decline in cortisol concentrations thereafter, demonstrating the greatest changes in cortisol concentrations from peak response post task to the final sampling 25 minutes post task (Simeon, Knutelska, Smith et al., 2007). These two difference scores were used as outcome variables in univariate F tests with diagnostic groups as predictors. Similar to prior findings the results did not indicate significant differences in changes in cortisol concentrations related to different diagnostic groups. Dissociative symptoms were included as a covariate and were also non significant.

Hypothesis for challenged cortisol based on trauma characteristics: chronicity

Repeated measures ANCOVA was used to examine the effect of chronicity groups, with dissociation as a covariate, on challenged cortisol. However, neither chronicity nor dissociative symptoms were significant predictors of mean differences or trends in cortisol concentrations over time. These findings were consistent with results from univariate F tests using the two difference scores as outcome variables, chronicity groups as predictors, and dissociative symptoms as a covariate.

Hypothesis for challenged cortisol based on trauma characteristics: earliest age of trauma exposure

Repeated measures analyses using earliest age of trauma exposure and dissociative symptoms as predictors, indicated that neither factor was a significant predictor of mean differences or trends in cortisol concentrations over time. These findings were consistent with results from regression analyses using both difference scores as outcome variables.

Hypothesis for challenged cortisol based on trauma characteristics: cumulative trauma exposure based on relationship to perpetrator

Repeated measures ANOVA was used to examine the effect of relationship to perpetrator on challenged cortisol. However, F tests were not significant for either predictor. Univariate analyses using difference scores as outcomes also did not indicate significant effects for perpetrator type on changes in cortisol concentrations related to the cognitive stress task.

Hypothesis for challenged cortisol based on trauma characteristics: social support

Repeated measures analyses using general levels of social support and dissociative symptoms as predictors, indicated that neither factor significantly predicted mean differences in cortisol concentrations, or trends in changes in cortisol concentrations over time. These findings were consistent with results from regression analyses using both difference scores as outcome variables. Similar results were found for perceived social support at the time of most traumatic event identified by the participant.

CHAPTER 7

Discussion

The goal of the current study was to examine whether and how typical mental health disorders seen in the context of IPV and chronic trauma exposure, and various trauma related characteristics, are associated with salivary cortisol measures, as an indicator of HPA axis dysfunction. Controlling for multiple methodological confounds typically seen in the literature, the study found that diagnostic groups were not differentially related to any cortisol measures. An examination of other common trauma sequelae including dissociative symptoms and borderline personality diagnosis, suggested that dissociative symptoms were the only significant predictor of cortisol concentrations. Specifically low levels of awakening cortisol concentrations were associated with high levels of dissociative symptoms, suggesting that psychological factors other than the commonly explored PTSD and mood disorders are important for future examination.

Basal and diurnal cortisol findings

Findings from the current study did not support the primary hypotheses of differences in basal cortisol concentrations across various diagnostic groups PTSD, PTSD and MDD, subthreshold trauma/ trauma and depressive symptoms relative to the control comparison group. These findings are inconsistent with the majority of the extant literature, though partially consistent with a meta-analytic study, which did not find differences between PTSD and control subjects with respect to basal cortisol concentrations (Meewisse et al., 2007). As noted earlier, this meta-analytic study used only one data point, i.e. the earliest available cortisol measure (with respect to time of

day), from each study rather than calculating a combined effect size for each study. This methodology could have influenced study findings. Alternately, the lack of significant differences between PTSD and control comparison groups, could suggest that differences in basal cortisol concentrations typically noted in the literature are accounted for by other frequently co-occurring trauma related factors (e.g., dissociation).

Findings in the current study also did not support hypotheses regarding differences in basal cortisol concentrations related to differences in trauma related characteristics such as chronicity of trauma exposure (i.e., adulthood versus childhood and adulthood trauma), differences in the types of cumulative trauma experienced (i.e., interpersonal versus interpersonal and non-personal forms of trauma), age of earliest trauma exposure, and the amount of general social support and/ or trauma-related social support available to women.

Although the study's primary hypotheses were not supported, dissociative symptoms were consistently found to predict basal cortisol concentrations. This association was specific to the relationship between awakening cortisol and dissociative symptoms. Women with trauma exposure differed from their control counterparts and reported significantly higher levels of dissociative symptoms, which was significantly associated with lower levels of awakening cortisol. These findings are partially consistent with the limited literature examining the relationship between dissociative symptoms and cortisol levels in adults with a history of trauma exposure (Neylan et al., 2005; Simeon, Knutelska, Smith et al., 2007; Simeon et al., 2008). For instance, Neylan and colleagues (2005) examined the relationship between PTSD and dissociative symptoms and salivary cortisol concentrations, in 30 active duty police officers and found that dissociative

symptoms at the time of trauma exposure were inversely related to cortisol at awakening. However, after accounting for the effects of age, PTSD symptoms and peritraumatic distress (panic and related negative reactions) only PTSD symptoms were found to significantly predict cortisol at awakening.

In a different study, Simeon and colleagues (2008) examined dissociation and posttraumatic stress and cortisol in adults approximately nine months after being exposed to the World Trade Center attack on 9/11. Twenty one highly exposed adults (1/3 rd of whom met full PTSD criteria) were compared with 10 healthy controls without major trauma exposure. The two groups did not differ on cortisol measures however dissociative symptoms were significantly associated with lower levels of morning plasma cortisol concentrations. The study findings also suggested that dissociation and posttraumatic stress may differ with respect to both phenomenological and biological correlates. Specifically the study reported that dissociation at the time of assessment was associated with childhood trauma while posttraumatic stress was not. Similarly other factors such as exposure magnitude, peritraumatic distress, and early posttraumatic stress, were also associated with posttraumatic stress but not with dissociation at the time of assessment. The authors suggest that this may reflect two different processes i.e. while factors associated with posttraumatic stress tap into arousal dissociation represents “..shut-down processes...” (Simeon et al., 2008, p.327).

The conceptualization posited by Simeon and colleagues (2008) is consistent with the phenomenological understanding of dissociative features. As per trauma theory, Hermann (1992) suggested that the development of psychological disorders is rooted in the psychological defenses used to cope with an unpredictable and threatening

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environment. Thus, experiences that are not integrated into conscious awareness and memory lay the cornerstone for cognitive distortions and dissociative states. As a result, dissociative symptoms are commonly seen in the face of trauma exposure (Herman, 1992; van der Hart, Nijenhuis, & Steele, 2006). Though ostensibly adaptive in the short term, this results in a disruption of functions of consciousness, memory, identity or perception, and also appears to be associated with specific neuroendocrinological dysfunction, which maybe different from other co-occurring trauma related conditions such as PTSD and MDD.

There is evidence to suggest that dissociative symptoms predict autonomic hyporesponsivity as assessed through diminished heart rate and galvanic skin responses (Griffin, Resick, & Mechanic, 1997; Sierra et al., 2002). In behavioral terms dissociative symptoms, which present as mind-blanking, lapses in memory and attention, symptoms of depersonalization and derealization etc., are viewed as coping mechanisms to mentally and emotionally disconnect one self from traumatic stressors (Carlson & Putnam, 1993; van der Hart et al., 2006). Thus it is likely that major stress systems such as the HPA axis and the noradrenergic axes may function differently in the presence of dissociation. As these systems play a key role in attention and arousal, the presence of posttraumatic stress symptoms in the absence of dissociation maybe associated with hyperarousal and intrusive symptomatology, but in the presence of dissociative symptoms, maybe indicative of autonomic blunting and reflected through low cortisol levels. This may in turn explain dissociative symptoms as being characterized by ‘shutting down’ behaviors, as a means for women to turn their attention away from very overwhelming traumatic experiences.

The current study's findings are also reminiscent of studies showing low basal (Yehuda, 2002) and awakening cortisol in populations with chronic trauma (Delahanty et al., 2005). However, it has been suggested that low cortisol may not be associated with the diagnosis of PTSD per se but may predate and be a risk factor for the development of PTSD, marking greater HPA axis vulnerability to traumatic events in relation to early life trauma (King et al., 2001; Young & Breslau, 2004b). Thus it is possible that the current study's findings reflect that low awakening cortisol is a risk factor associated specifically with dissociative symptoms which in turn could mediate the effects of trauma exposure on PTSD.

Findings in the literature also suggest that factors such as peritraumatic dissociation (i.e. dissociation at the time of trauma exposure) may be relevant to examine. While peritraumatic dissociation has been associated with PTSD (e.g., Nishi et al., 2010) and dissociation at later stages (Bremner & Brett, 1997; Simeon, Greenberg, Nelson, Schmeidler, & Hollander, 2005), a growing body of literature shows that the contribution of peritraumatic dissociation to later PTSD declines after accounting for baseline PTSD severity (Marshall & Schell, 2002) or accounting for other factors such as peritraumatic distress (i.e., fear of death, loss control, etc.) (Gershuny, Cloitre, & Otto, 2003). In the current study, the trauma-exposed groups had significantly higher dissociative symptoms than the control group and diagnostic groups were not predictive of basal cortisol. Thus, it is possible that other factors such as peritraumatic dissociation and peritraumatic distress which were not evaluated in the current study, may better account for the findings and observed relationships. For instance, the presence of peritraumatic dissociation at the time of a traumatic event may be a more important factor rather than

the development of PTSD at a later stage, and its effects may be mediated by later dissociative symptoms.

Another factor that may have influenced the lack of findings in the current study is the assessment of specific psychological constructs. For instance, the measure of dissociative symptoms asks participants to report how often they engage in specific behaviors or experience certain events that represent dissociative symptoms. While reports on the measure of dissociation is more likely to assess current or recent behaviors/ experiences, diagnoses and posttraumatic stress symptoms in the current study were assessed based on the event identified by the participant as being most traumatic, which could have occurred at any time in their life. It is possible that a current or more proximal diagnosis of PTSD i.e. related to more recent events only (e.g., IPV specific PTSD) may be better predictive of cortisol concentrations. Some support for this idea comes from findings from Simeon and colleagues' (2005) study, which found that the effects of dissociation following a recent traumatic event on cortisol was distinguishable from posttraumatic stress related to childhood trauma history. However, this issue needs further examination due to the limited number of studies examining the effects of dissociative symptoms, and the significant differences in sample characteristics and types of trauma examined, between the current study and the aforementioned studies examining the role of dissociation on cortisol concentrations.

Finally, another possibility for assessing whether there are different subtypes of neuroendocrine profiles in trauma exposed women which is associated with unique psychological factors as predictors could be to examine whether there are distinct clusters of cortisol profiles and examine which psychological factors or trauma related

characteristics are associated with these cortisol profiles. Although this methodology has not been used in the literature examining cortisol measures in trauma exposed women, studies examining behavioral measures have found it effective in identifying unique predictors for differing behavioral clusters (Tremblay et al., 2004).

It is notable that the current study improves on a number of methodological issues in the literature by accounting for diagnostic and symptomatic comorbidity, use of medication and contraception, substance abuse/ dependence, and physical health problems that have been shown to influence cortisol measures. After accounting for these factors, this study did not find support for the impact of factors typically noted in the literature (e.g., diagnostic classification, chronicity). Dissociation was the only robust predictor of awakening cortisol, which is consistent with the limited number of studies examining the association between dissociation and cortisol measures. As dissociation and other trauma sequelae (e.g., PTSD, MDD) are frequently comorbid and most studies have not examined the effects of dissociation on cortisol concentrations, findings from the current study suggest a need for further examination of the differential correlates of typical trauma sequelae.

Challenged cortisol

With respect to challenged cortisol measures, the study did not find evidence of group differences in changes in cortisol concentrations across diagnostic groups or related to differences in trauma related characteristics such as chronicity of trauma exposure (i.e., adulthood versus childhood and adulthood trauma), differences in the types of cumulative trauma experienced (i.e., interpersonal versus interpersonal and non-

personal forms of trauma), age of earliest trauma exposure, and the amount of general social support and/ or trauma-related social support available to women.

These findings are inconsistent with extant literature which has demonstrated differences in the stress response system in adults exposed to traumatic events relative to control comparison groups (e.g., Bremner et al., 2003; Carpenter et al., 2007; Elzinga et al., 2008; Heim et al., 2000; Santa Ana et al., 2006; Simeon, Knutelska, Yehuda et al., 2007). There were no significant differences in mean levels of cortisol concentrations across diagnostic groups or differences in changes in cortisol concentrations pre- and post-paradigm. However, due to a trend in these findings, it is possible that unequal and small sample sizes across diagnostic groups may have influenced the lack of significant findings. This is also supported by the fact that the mean ratings of perceived stress due to the cognitive stress task was in the moderate to high range across all groups. See Table 7. Alternately, it is possible that despite the participants self perceived stress ratings, the stress paradigm was not effective in mobilizing the stress response system. The current study used an adapted version of the TSST paradigm. While the original paradigm has been shown to be effective in mobilizing the stress response system and has shown differences in rates of changes in cortisol concentrations relative to diagnostic status (Carpenter et al., 2007; Elzinga et al., 2008; Heim et al., 2000; Simeon, Knutelska, Yehuda et al., 2007), this is the only study to use the adapted protocol. Differences in the current study's paradigm, particularly the mock recording using an audiotape rather than a videotape, as in the original paradigm, may have affected these findings. It is possible that the stress perceived as a result of having to give a speech, and be interviewed, while being videotaped is likely to be more anxiety provoking than being audio recorded only.

Limitations

The use of retrospective reporting within a cross sectional design precludes the possibility of a comprehensive and accurate assessment of lifetime exposure to traumatic events. Also, sample sizes varied across diagnostic groups, with very small cell sizes in some groups, which may have made it difficult to detect group differences, particularly with respect to challenged cortisol measures.

Findings in the current study are partially consistent with the limited literature examining the association between dissociative symptoms and cortisol measures. Findings from one of these studies suggests that other factors such as peritraumatic dissociation and peritraumatic distress may be important mediators in predicting cortisol concentrations. These factors were not assessed in the current study and suggest an important area for future examination. Another, methodological issue that needs further examination is whether diagnoses and assessment of posttraumatic stress symptoms based on the event identified by the participant as being most traumatic or more proximal diagnosis of PTSD i.e. related to more recent events only (e.g., IPV specific PTSD) is a better predictor of cortisol concentrations.

The lack of significant findings related to challenged cortisol measures maybe attributable to the use of an adapted version of the original TSST. It is likely that using the original TSST, which has consistently been shown to be effective in mobilizing the stress response system, may have yielded different results. Additionally, the use of chemical stressor such as dexamethasone test may also provide a more effective measure of the stress response system.

Finally, factors related to cortisol sampling could also have affected the lack of findings. For instance, the study relied on self report of participants regarding time of cortisol collection. The use of MEMS cap provides a more accurate and objective means of measuring participants' compliance to the protocol of collecting saliva samples at specific times. Additionally, the study had no way of ensuring participants' fidelity to instructions of storing saliva samples in their freezer at home prior to pick up. Another limitation of the current study was the use of only one sampling for each time of day. Repeated sampling of salivary cortisol at the same time of day, across multiple days, would have provided a more accurate measure of cortisol levels.

Conclusion

The goal of the current study was to examine differences in basal, diurnal, and challenged cortisol measures in a sample of premenopausal non pregnant adult women, who differed with respect to diagnostic status (PTSD, PTSD and MDD, subthreshold symptoms) and trauma characteristics (chronicity of trauma, differences in cumulative trauma types, age of first trauma exposure, and social support) relative to control comparison counterparts.

The study did not find evidence in support of the major hypotheses for the differential effects of diagnostic groups and trauma characteristics, after controlling for typical methodological limitations. Results indicated a relationship between dissociative symptoms and awakening cortisol such that trauma exposed women, regardless of their diagnostic status had higher levels of dissociative symptoms relative to women in the control group, which was inversely related to cortisol levels at awakening. Findings suggest that HPA axis functioning may be different in the presence of dissociative

symptoms and may differ from HPA axis functioning in the presence of PTSD when dissociative symptoms are not present. Studies examining the relationship between dissociation and its biological correlates are very limited but have shown similar results. These findings are consistent with the phenomenological presentation of dissociative symptoms as 'shutting down' mechanisms, which likely serve to protect trauma survivors from the overwhelming stress associated with traumatic experiences. Future studies should further examine whether and how stress systems such as the HPA axis function differently in the presence of PTSD when other comorbid symptomatology such as depression and dissociation are present.

APPENDICES

APPENDIX A

Table 1.

Classification of sample based on diagnostic groups

Diagnosis	<i>n</i>
Control	12
PTSD only	14
PTSD and MDD	43
Subthreshold symptoms	19
MDD only	4
Total	92

Table 2.

Age and Monthly Income Across Diagnostic Groups

Diagnosis	Age		Monthly Income	
	Mean	S.D (Range)	Mean	S.D (Range)
PTSD only	28.50	7.21 (18.70 - 41.00)	1338.43	907.48 (300.00 - 3000.00)
PTSD and MDD	28.19	6.44 (18.76 - 41.50)	1984.91	2219.32 (160.00 - 12000.00)
Subthreshold	25.84	7.78 (19.55 - 40.44)	3115.12	3819.81 (00.00 - 12000.00)
Control	22.78	2.13 (18.99 - 26.28)	3510.00	3501.55 (400.00 - 12500.00)

No significant differences in age or monthly income across diagnostic groups

Table 3.

Education information across diagnostic groups (percentage of total)

Education Control	PTSD	PTSD + MDD	Subthreshold	
	(n) %	(n) %	(n) %	(n) %
High school/ GED	(4) 4.50	(15) 17.00	(2) 2.30	(1) 1.1
Some college/ Trade School/				
Associates Degree	(5) 5.70	(22) 25.00	(13) 14.80	(5) 5.70
College	(2) 2.30	(3) 3.40	(3) 3.40	(3) 3.40
Graduate	(3) 3.40	(3) 3.40	(1) 1.10	(3) 3.40

No significant differences in education levels across diagnostic groups

Table 4.

Ethnicity information across diagnostic groups (percentage of total)

Ethnicity Control	PTSD	PTSD + MDD	Subthreshold	
	(n) %	(n) %	(n) %	(n) %
African American	(7) 8.00	(8) 9.10	(5) 5.70	(1) 1.10
Caucasian	(6) 6.80	(20) 22.70	(12) 13.60	(8) 9.10
Hispanic	(0) 0.00	(6) 6.80	(1) 1.10	(1) 1.10
Other	(1) 1.10	(9) 10.20	(1) 1.10	(2) 2.30

No significant differences in ethnicity across diagnostic groups

Table 5.

Descriptives for Cortisol Values Pre and Post Mean Substitution by Diagnostic Group

	Pre mean substitution	Post mean substitution
	Mean (S.D)	Mean (S.D)
PTSD		
Baseline	.35 (.11)	.35 (.09)
Immediately post task	.34 (.10)	.34 (.01)
10 minutes post task	.33 (.12)	.33 (.11)
25 minutes post task	.31 (.10)	.31 (.09)
Night	.30 (.13)	.30 (.13)
Awakening	.32 (.12)	.32 (.12)
45 minutes post awakening	.41 (.12)	.41 (.12)

Table 5 continued.

Descriptives for Cortisol Values Pre and Post Mean Substitution by Diagnostic Group

	Pre mean substitution	Post mean substitution
	Mean (S.D)	Mean (S.D)
PTSD + MDD		
Baseline	.27 (.13)	.27 (.11)
Immediately post task	.28 (.10)	.28 (.09)
10 minutes post task	.28 (.11)	.28(.10)
25 minutes post task	.28 (.12)	.28 (.11)
Night	.27 (.13)	.27 (.11)
Awakening	.37 (.20)	.37 (.17)
45 minutes post awakening	.46 (.32)	.46 (.27)

Table 5 continued.

Descriptives for Cortisol Values Pre and Post Mean Substitution by Diagnostic Group

	Pre mean substitution	Post mean substitution
	Mean (S.D)	Mean (S.D)
Subthreshold		
Baseline	.27 (.11)	.27 (.11)
Immediately post task	.26 (.11)	.26 (.11)
10 minutes post task	.26 (.11)	.26(.12)
25 minutes post task	.26 (.12)	.25 (.12)
Night	.26 (.13)	.25 (.16)
Awakening	.39 (.17)	.39 (.16)
45 minutes post awakening	.46 (.32)	.48 (.27)

Table 5 continued.

Descriptives for Cortisol Values Pre and Post Mean Substitution by Diagnostic Group

	Pre mean substitution	Post mean substitution
	Mean (S.D)	Mean (S.D)
Control		
Baseline	.23 (.19)	.23 (.19)
Immediately post task	.20 (.13)	.20 (.13)
10 minutes post task	.21 (.13)	.21(.13)
25 minutes post task	.21 (.14)	.21 (.14)
Night	.33 (.42)	.33 (.38)
Awakening	.37 (.22)	.37 (.21)
45 minutes post awakening	.47 (.25)	.47 (.23)

Table 6

Dissociative Symptoms and Daily Hassles Diagnostic Groups

Diagnosis	Dissociative Symptoms		Daily Hassles	
	Mean	S.D	Mean	S.D
PTSD only	583.57	(452.41)	56.29	(23.89)
PTSD and MDD	531.21	(443.13)	56.46	(22.62)
Subthreshold	317.79	(173.67)	49.00	(18.59)
Control	112.50	(59.72)	40.50	(9.98)

No significant differences across groups with respect to daily hassles. All trauma exposed groups significantly differ from control group with respect to dissociative symptoms.

Table 7

Self Perceived Stress Ratings for Cognitive Stress Task across Diagnostic Groups

Diagnosis	Stress Ratings	
	Mean	S.D
PTSD only	58.46	25.44
PTSD and MDD	74.05	17.26
Subthreshold	70.53	17.79
Control	58.33	16.42

Table 8.

*Between Subjects Effects for Basal Cortisol Concentrations Across Diagnostic Groups
(ANCOVA)*

Source	df	F	<i>p</i>	η^2	Power
Diagnosis	3	.38	.77	.01	.12
Dissociation	1	4.68	.03	.05	.57

Table 9.
Repeated Measures ANCOVA for Diurnal Cortisol Concentrations Across Diagnostic Groups

Source	df	F	<i>p</i>	η^2	Power
Between subjects					
Diagnosis	3	1.39	.25	.05	.36
Dissociation	1	3.88	.05	.05	.50
Within subjects					
Diurnal					
Linear	1	.54	.47	.01	.11
Quadratic	1	19.38	.00	.19	.99
Cubic	1	1.68	.20	.02	.25
Diurnal * Diagnosis					
Linear	3	1.12	.35	.04	.29
Quadratic	3	1.26	.30	.04	.33
Cubic	3	.45	.72	.02	.14
Diurnal * Dissociation					
Linear	1	7.68	.01	.09	.78
Quadratic	1	.27	.60	.00	.08
Cubic	1	1.36	.25	.02	.21

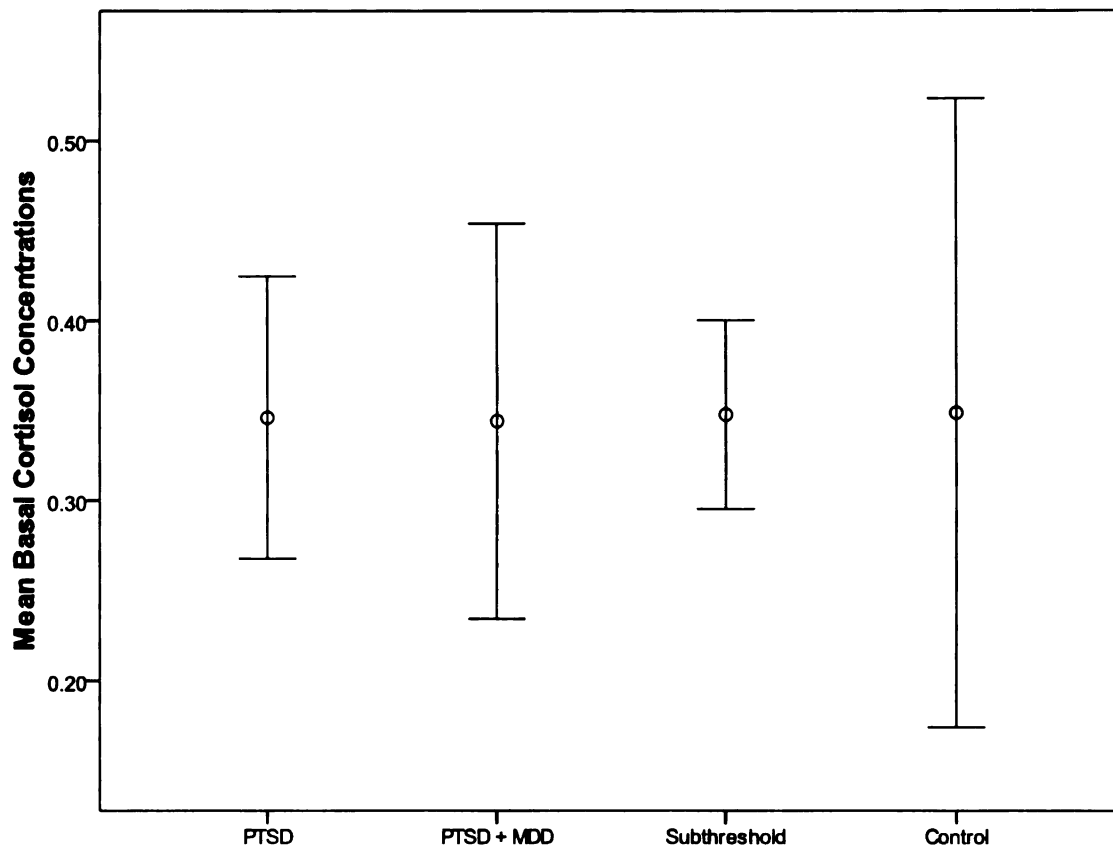
Table 10.

Repeated Measures ANCOVA for Challenged Cortisol Concentrations Across Diagnostic Groups

Source	df	F	<i>p</i>	η^2	Power
Between subjects					
Diagnosis	3	2.36	.07	.07	.57
Dissociation	1	.01	.93	.00	.05
Within subjects					
Challenged					
Linear	1	.02	.88	.00	.05
Quadratic	1	.02	.88	.00	.05
Cubic	1	.50	.48	.01	.11
Challenged * Diagnosis					
Linear	3	1.01	.36	.04	.28
Quadratic	3	.13	.95	.01	.07
Cubic	3	.88	.46	.03	.23
Challenged * Dissociation					
Linear	1	.47	.50	.01	.10
Quadratic	1	.03	.85	.00	.05
Cubic	1	1.20	.28	.01	.19

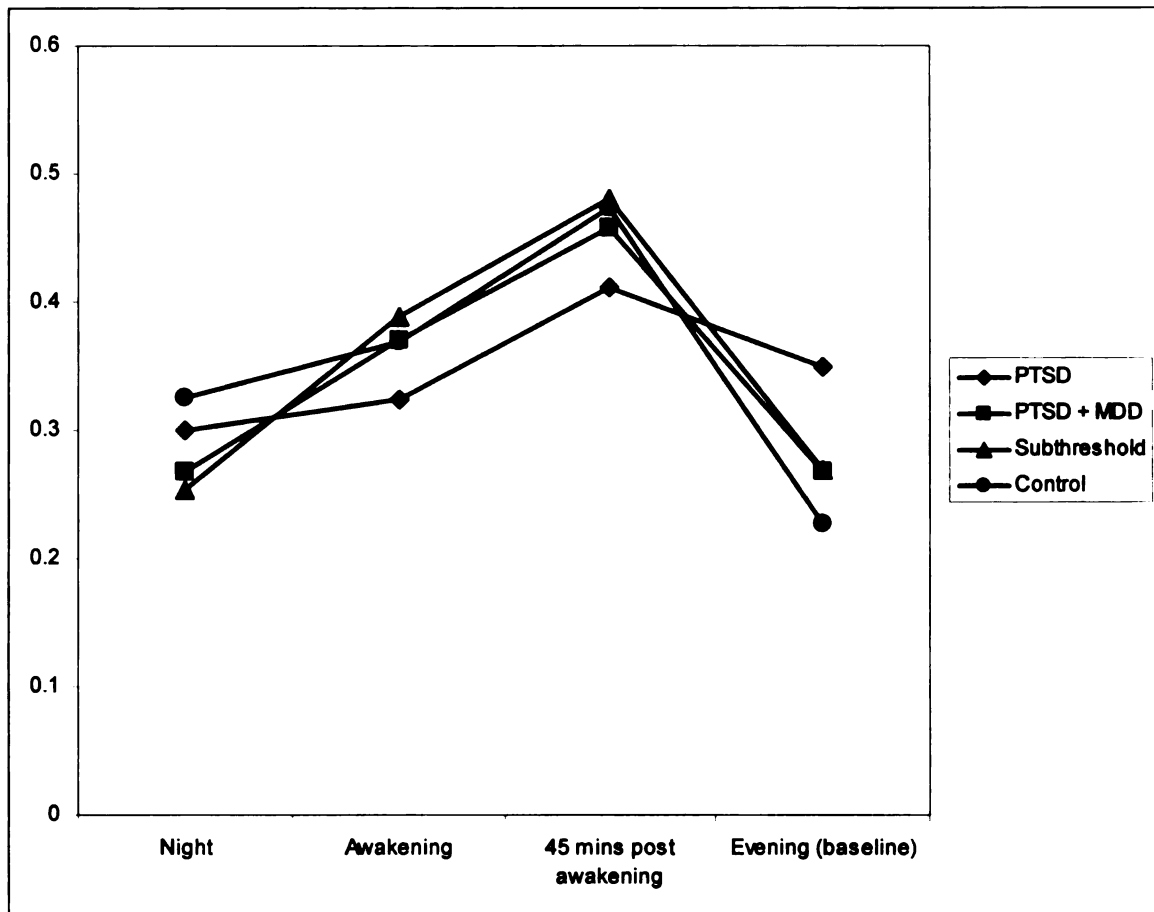
APPENDIX B

Figure 1.
Basal Cortisol Concentrations Across Diagnostic Groups



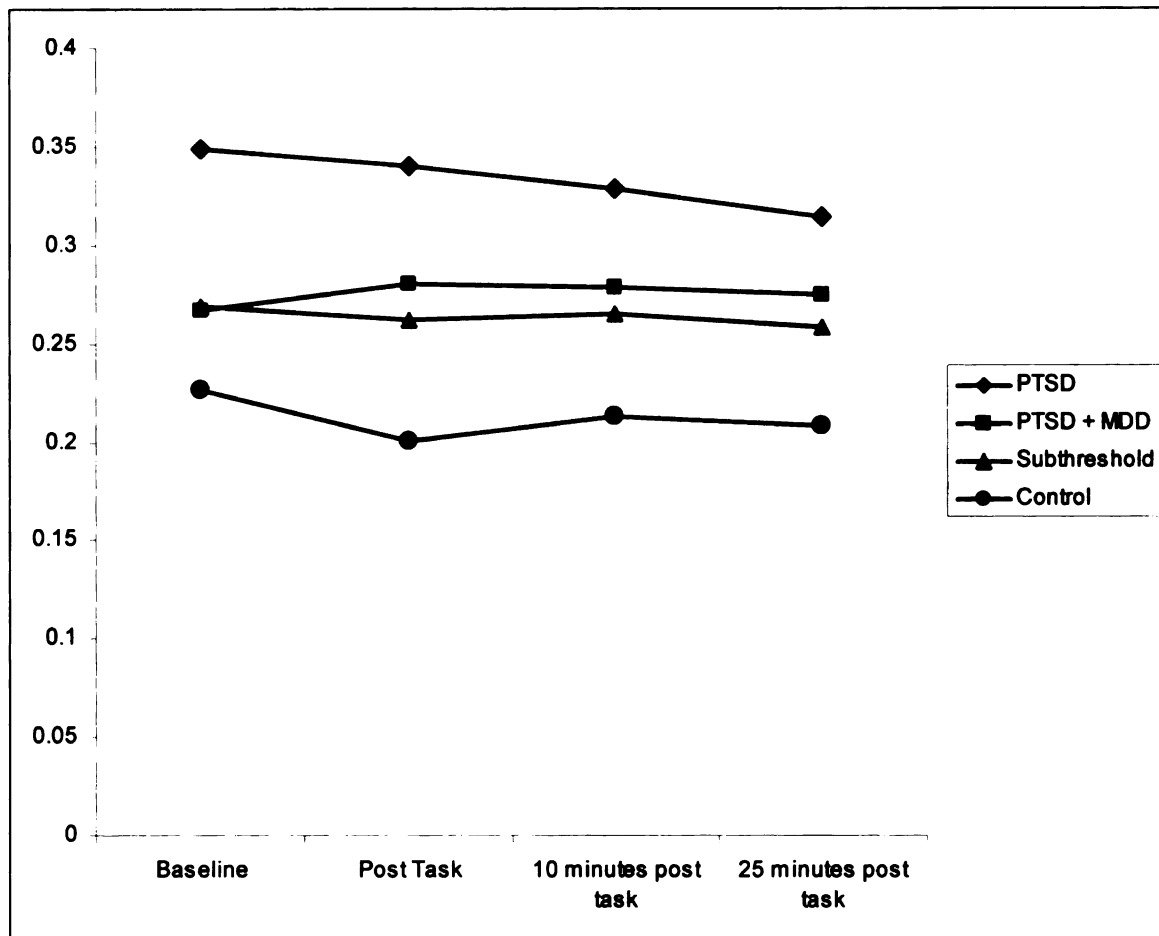
**** Error bars represent +/- 1 Standard deviation ** PTSD= Posttraumatic Stress Disorder, MDD = Major Depressive Disorder, Subthreshold = Subthreshold symptoms of PTSD or PTSD and MDD, Control = Control comparison group with no lifetime exposure to intimate partner violence/ interpersonal forms of trauma, or PTSD/ mood disorder diagnoses.***

Figure 2.
Diurnal Cortisol Concentrations Across Diagnoses



PTSD = Posttraumatic Stress Disorder, MDD = Major Depressive Disorder, Subthreshold = Subthreshold symptoms of PTSD or PTSD and MDD, Control = Control comparison group with no lifetime exposure to intimate partner violence/ interpersonal forms of trauma, or PTSD/ mood disorder diagnoses.

Figure 3.
Challenged Cortisol Concentrations Across Diagnoses



PTSD = Posttraumatic Stress Disorder, MDD = Major Depressive Disorder, Subthreshold = Subthreshold symptoms of PTSD or PTSD and MDD, Control = Control comparison group with no lifetime exposure to intimate partner violence/ interpersonal forms of trauma, or PTSD/ mood disorder diagnoses.

APPENDIX C

Phone Screen

Standardized protocol to introduce study: “Thank you for your interest in the Women’s Health Study. Let me tell you a little bit about our study so you know what the study is about. I also need to ask you some background questions and some health-related questions to make sure that you are eligible for our study. This will take about 10 minutes, is this a good time for you to talk?”

Following the standardized protocol to introduce the study, the following questions will be asked to screen participants for the study:

1. What is your date of birth <MM/ DD/ YYYY>? ____/ ____/ _____
2. What is the highest level of education you have completed?
 - I. Grade: 1 2 3 4 5 6 7 8 9 10 11 12 13=GED
 - II. Some college
 - III. College (B.A/ B Sc)
 - IV. Trade School (Cosmetology, Nursing, Technical/Vocational)
 - V. Associates degree
 - VI. Some graduate school
 - VII. Graduate School
 - VIII. Other _____ (if less than grade 6 → Not Eligible)
3. Are you currently pregnant? Y → Not Eligible
4. Are you currently breast feeding? Y N
 - I. If yes → Not Eligible
 - If No → Continue with screen

Y N

↓ ↓

I. If yes -- when did you get married/ start living together <MM/ YYYY>

____ / ____

↓

II. If no -- in the past two years have you been involved in a romantic relationship with a man, for 6 weeks or longer (i.e. 1.5 months)?

Y N → Not Eligible

6. Has your partner ever been violent towards you? →

**If No skip to
Q. no. 8**

I. If yes, has your partner been violent towards you in the past 2 years?

If No

→

Not eligible

7. In the past month have you experienced any of the following:

- 111

IV. Do you have a strong reaction when you are faced with reminders of some of those incidents? Y N

Note examples _____

V. Do you try to avoid thinking/ feeling/ talking about those experiences?

Y N

VI. Do you try to avoid activities/ places/ people that remind you about those experiences? Y N

VII. Are there large parts of those experiences that you cannot remember?

Y N

VIII. Do you feel that you have lost interest in activities that you used to enjoy before these incidents with your partner?

Y N

IX. Do you feel detached/ disconnected/ removed from others since these incidents? Y N

X. Do you feel unable to experience certain emotions since these experiences (e.g., loving feelings/ angry feelings/ sadness/ crying)

Y N

XI. Do you feel like you may not do certain things in life, like have a career or some other goal you may have previously wanted to do?

Y N

XII. Do you have difficulty falling asleep, or difficulty staying asleep?

Y N

XIII. Do you feel irritable or have outbursts of anger?

Y N

XIV. Do you experience difficulty concentrating?

Y N

XV. Do you feel like you are on edge / vigilant/ worrying about things?

Y N

XVI. Do you have a strong reaction to small things? That you get easily scared or
upset?

Y N

XVII. Do you feel depressed or sad most of the day nearly everyday?

Y N

XVIII. Has there been significant changes in your weight since these incidents
(weight loss or weight gain)?

Y N

XIX. Do you find that you sleep too little or too much?

Y N

XX. Do you feel fatigue and loss of energy a lot of the time?

Y N

XXI. Do you find it physically difficult to do daily chores and errands (like your
body or arms/ legs heavy and you have to drag yourself to do anything)?

Y N

XXII. Do you find it hard to relax and find yourself physically restless a lot?

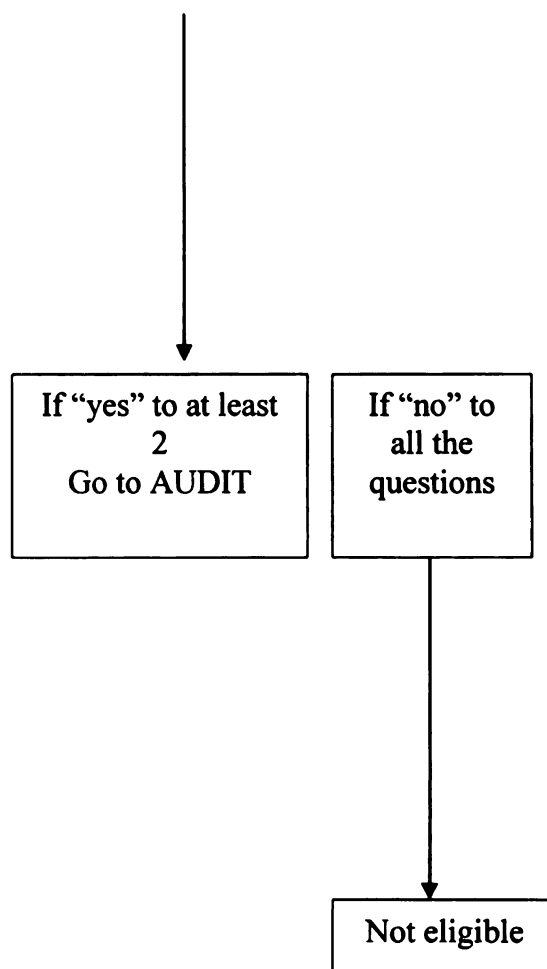
Y N

XXIII. Do you have negative feelings about your self like feelings of
worthlessness?

Y N

XXIV. Do you feel guilty or blame yourself a lot?

Y N



8. Have you ever had any psychological problems (e.g., depression, anxiety problems etc.)? If yes, note following details

i. Describe exact nature of the problem _____

ii. When did you first notice these problems? _____

iii. Were you ever formally diagnosed with any disorder? If yes, by whom
(psychologist/ psychiatrist/primary care provider/ counselor etc.)?

iv. Are you still having similar difficulties? _____

9. Have you ever been exposed to a major traumatic event (e.g., been in danger of being hurt or killed or witnessed a loved in danger of being hurt or killed)?

Questions to screen for other drug use related difficulties

1. Have you ever used any drugs for non medical purposes? (Use the following as prompts/ rule outs)

- | | | |
|--|---|---|
| 1a. Cannabis (marijuana, pot, grass, hash, etc.) | Y | N |
| 1b. Cocaine (coke, crack, etc.) | Y | N |
| 1c. Amphetamine type stimulants (speed, diet pills, ecstasy, etc.) | Y | N |
| 1d. Inhalants (nitrous, glue, petrol, paint thinner, etc.) | Y | N |
| 1e. Sedatives or Sleeping Pills (Valium, Serepax, Rohypnol, etc.) | Y | N |
| 1f. Hallucinogens (LSD, acid, mushrooms, PCP, Special K, etc.) | Y | N |
| 1g. Opioids (heroin, morphine, methadone, codeine, etc.) | Y | N |
| 1h. Any other drugs for non medication purposes?_____ | Y | N |

2. In the past 3 months how often have you used (for each endorsement from above)?

	Never	1 -3 times per year	Monthly 1 – 2 times/ month	Weekly 1-3 times/week	Daily/ Almost daily 4 – 7 times/ week
Cannabis					
Cocaine					
Stimulants					
Inhalants					
Sedatives					
Hallucinogens					
Opioids					
Other					

3. During the past 3 months, how often has your use of (fill from above) led to social, legal or financial problems or problems in daily functioning or work (e.g. running errands, attending school, taking care of children, and reaching work on time?)
- a. Never
 - b. 1 -2 times in the past 3 months
 - c. At least once a month
 - d. Weekly basis
 - e. Daily
4. In the past 3 months, have you received counseling/ rehabilitation services, or any other help/ treatment for using (fill from above)? Note details of multiple treatments, if applicable.
- 4a. When did you start treatment _____ ?
 - 4b. For how long did you receive treatment _____ ?
 - 4c. What kind of treatment did you receive _____ ?

Questions to screen for psychotic disorders

1. Have you ever seen things that other people could not see?
- a. If yes, describe _____
 - b. How often does it happen?
2. Have you ever hear voices, noises or sounds that others could not?
- a. If yes, describe _____
 - b. How often does it happen?
3. Have you smelled or tasted something strange that you could not explain and others could not smell/ taste?
- a. If yes, describe _____

b. How often does it happen?

Questions to screen for neurological/ endocrine/ medical disorders

1. Have you ever been diagnosed with any of the following disorders?

1a. Alzheimer's Disease	Y	N
1b. Aphasia	Y	N
1c. Bell's Palsy	Y	N
1d. Stroke	Y	N
1e. Encephalitis	Y	N
1f. Epilepsy	Y	N
1g. Huntington's Disease	Y	N
1h. Parkinson's Disease	Y	N
1i. Muscular Dystrophy or Sclerosis	Y	N
1j. Cushing's Disease	Y	N
1k. Addisons Disease	Y	N
1l. Cancer	Y	N
1m. Any cognitive impairments	Y	N

Have you ever been diagnosed with any other major medical problem _____?

If yes, note the name, and if undergoing any treatment _____

Questions to screen for medication usage

1. What medications (prescription or over the counter), including hormonal birth control, are you currently taking?
 - a. If none, have you taken any medications (prescription or over the counter) or birth control, in the past 3 months?

Name	Dosage	Since when (note duration)?
------	--------	-----------------------------

Questions for to establish menstrual cycle

1. Do you have a regular menstrual cycle? Y N
 - 1a. If no, please describe your typical menstrual cycle in the past 3 months (how often did they occur and for how long each time did your periods last)?

2. Have you skipped any periods in the past 3 months? Y N
3. Have you skipped period in the past month? Y N
4. What is the first date of your last periods? <MM/DD/YYYY> ____ / ____ / ____

5. How many days did your periods last in the past month?
6. How many days do your periods typically last?

“Our study is interested in understanding how women’s experiences with their partners affect their psychological and physical health, and how they cope with these experiences. Our study involves one face to face interview at the MSU campus, which typically takes between 1.5 – 2 hours. During the interview we will ask you about your experiences with your partner, and some questions about your moods and behaviors. We are also interested in your daily life experiences such as taking care of the kids, going to work etc. and any help or support you get from other people such as your friends/ family or anyone else. Some of these questions will be in interview form and for some of them you can fill up the questionnaires on your own. A part of the interview also involves performing some tasks like writing a paragraph on a topic. Finally, during the interview we will collect some saliva samples and also give you a kit to take home and collect your saliva at specific times in the day. Will you be able to take saliva samples at home safely? [For women who indicate currently living with their partner add: “Do you have any concerns about having problems with your partner if you take saliva samples at home?”]

You will be paid a total of \$40 for participating in our study. You will receive \$25 at the completion of the interview and \$15 when we receive the saliva that you collect at home. We can collect the take home kit from you, from any location of your choice or you can return it to our MSU project office. Do you have any questions so far?”

Cognitive Stress Challenge

After initial rapport formation, participants will complete the demographic questionnaire with the interviewer to allow them time to acclimatize to the research setting. Next, participants will be given a 5 minute resting period after which they will give their first, baseline, saliva sample. The resting period prior to the stress task is necessary to avoid potential activation of the HPA axis, which could confound with later activation during the cognitive stress challenge {Kirschbaum, 1993 #291}.

Next, the interviewer (referred to as the experimenter from hereon) will inform the participants about the task they are required to work on and introduce it as follows “I will now ask you to perform a task. You have to deliver a speech for a job application, for which you will have five minutes to prepare. A person trained in behavioral observation will be joining us and she will take notes about the way you give your speech and the things you say. You should imagine that you applied for a job position and you are being invited by that company or place to interview. Your speech should be five minutes long. This speech will be voice recorded and we will time you. You can make notes while you are preparing but you cannot use the notes once you start speaking. Make sure that your speech is believable because the observer will ask you additional questions. After this task I will ask you to do one more task and I will tell you about that after you deliver your speech”. The experimenter then asks whether the participant if she has any questions regarding the protocol and will answer any such questions.

Once the participant is ready to give the speech, an advanced graduate student (referred to as the observer from hereon) will enter the room set up a mock audio taping device (the performance will not be recorded) and “switch on” the recording. The

experimenter will use a stop watch to time the participant. The observer will begin by asking the participant their name and then ask them, "What qualifies you in particular for this position?"

After five minutes, the experimenter will introduce the next task as follows, "Let's move on to the next task. Now we want you to solve a calculation task. Please count aloud backwards from 2083 to zero in 13-step sequences. Please calculate as quickly and correctly as possible. If you miscalculate, we will point out your mistake and you have to start all over again from 2083. Do you have any questions? Please start."

Prime numbers will be used as subtractors for this task, because these make it more difficult {Kirschbaum, 1993 #291}. Protocol sheets with all intermediate numbers will be available to the experimenter and the observer so that they will not have to calculate it themselves. If the participant miscalculates, the observer will respond with the standard phrase "Error. 2083." until the participants reaches 0 or makes 4 mistakes having started over each time. The experimenter will note the number of errors and the number that the subject eventually reached as a performance measure.

Debriefing

Next, a detailed debriefing will be conducted with the participants, which will include an explanation why the stress protocol had to be included for the current research question. In addition, participants will be asked to rate the stressfulness of the paradigm on a scale of 0 – 100. Participants will be told that the wording of the instructions and tasks were chosen to assess how different people respond to anxiety-provoking tasks and the task themselves were not meant to be evaluative of the participants in any way. They will also be informed that the task had not been tape recorded. The debriefing text will be

as follows, “The reason we asked you to perform these tasks is because they are anxiety provoking for most individuals and one of the things we are trying to study is how people respond in anxiety provoking situations. The entire set up of having an observer and the way the instructions were given was designed to make it realistic and believable for you, so as to create the anxiety and see how people usually react in such situations. We did not tape record this interview nor were you being evaluated or tested on your performance of either of those tasks. Your performance on any of those tasks has no bearing for this interview. We really appreciate your giving us your best effort.”

Protocol for collecting saliva samples

Participants were asked to imagine chewing their favorite food, moving their jaws as if chewing while letting saliva collect under their tongue. Then, the participants were asked to gently pass the saliva through a short plastic straw into a cryogenic vial. The samples were frozen and later centrifuged for assays in Dr. Lonstein's lab. At the end of the interview each participant was given a kit with written instructions, 3 color coded and labeled tubes and straws, and checklists for bedtime, awakening and 45 minutes post-awakening. The pre-cognitive stress sampling was used to establish baseline for the stress task and also used as the evening sample for basal cortisol. The specimen collection procedure was shown to each participant and instruction about completing the accompanying form was discussed (time of sample, what she ate, drank, when she brushed her teeth or smoked in the 30 minutes prior to sampling). Participants were contacted via telephone for reminders for the awakening and post-awakening sample (45 minutes later), if the participants provided their consent to do so. Participants were asked to refrigerate the samples until they returned it to the research office or a research assistant collected them at a time determined in conjunction with the participant.

Demographic Questionnaire

1. First Name _____
2. Last Name _____
3. How many biological children do you currently have? _____
4. What is your Date of Birth <MM/DD/YYYY> ____ / ____ / ____
5. What is your race/ ethnicity (choose only one)?
 - 5.a. European American/ Caucasian
 - 5.b. African American
 - 5.c. Hispanic (not white)
 - 5.d. Asian
 - 5.e. Native American
 - 5.f. Biracial
 - 5.g. Multiracial
 - 5.h. Other (please describe) _____
6. Choose one that best describes your current relationship status (choose only one):
 - 6.a. single
 - 6.b. never married but in a relationship
 - 6.b.i. are you living with your partner? **YES** **NO**
 - if YES, when did you start living together <MM/YYYY>
_____ / _____
 - 6.c. married
 - 6.c.i. when did you get married <MM/YYYY> _____ / _____
 - 6.d. separated
 - 6.d.i. when did you separate <MM/YYYY> _____ / _____
 - 6.e. divorced
 - 6.e.i. when did you divorce <MM/YYYY> _____ / _____
 - 6.f. widowed
 - 6.f.i. when were you widowed <MM/YYYY> _____ / _____
7. What is your religious affiliation (if any)? _____
8. Are you currently employed/ have you been employed in the past 3 months? **YES** **NO**

- 8.a. If yes, what is/was your occupation? (*Please be specific. For example, bookkeeper, cashier, computer programmer*)
- 8.b. Are/ were you working
- 8.b.i. full time
- 8.b.ii. part time
9. What is the highest level of education you have completed (choose only one)?
- 9.a. Grade: 1 2 3 4 5 6 7 8 9 10 11 12 13=GED
- 9.b. Some college
- 9.c. Trade School (Cosmetology, Nursing, Technical/Vocational)
- 9.d. AA degree
- 9.e. BA/BS
- 9.f. Some grad school
- 9.g. Graduate degree
- 9.g.i. Masters
- 9.g.ii. Ph.D.
- 9.g.iii. Law
- 9.g.iv. Medical Degree
10. Have you been in school during the last year? **YES NO**
- If YES, please describe what you are currently doing in school.
-
-
11. What is your total family income **per month** (please include financial aid/ loans/ disability/ unemployment etc. and give your best estimate)? _____
12. Do you currently receive services from any of the following:
- | | | | |
|-------|---|------------|-----------|
| 12.a. | Women, Infants and Children (WIC) | YES | NO |
| 12.b. | Temporary Assistance for Needy Families (TANF) | YES | NO |
| 12.c. | Protective Services | YES | NO |
| 12.d. | Food Stamps | YES | NO |
| 12.e. | Medicaid | YES | NO |
| 12.f. | Social Security Disability (SSI) | YES | NO |
| 12.g. | Family Independence Agency (FIA) | YES | NO |
| 12.h. | Any child related programs (e.g., 0-3; Mother-Infant Program; Head Start) | YES | NO |
| 12.i. | Any other sources of support/ services? _____ | YES | NO |

Brief Screen prior to baseline sampling

1. Please note the time you finished your last meal: _____ AM/ PM
2. If you have eaten anything since then, please note what it was and when you ate it

3. Did you drink anything other than water in the past 2 hours? Y N
 - a. If YES, please tell us what you drank (including alcoholic and non alcoholic) _____
 - b. How many glasses/ cans did you drink _____
 - c. What time did you have the last drink _____
4. Have you smoked in the past 1 hour Y N
 - a. How many cigarettes have you smoked _____

Aautoimmune diseases

Have you ever been diagnosed with any of the following?

- _____ Lupus
- _____ Graves' Disease
- _____ Hyperthyroidism
- _____ Celiac Disease
- _____ Gluten Intolerance
- _____ Irritable Bowel Syndrome
- _____ Hashimoto's Hypothyroidism
- _____ Polycystic Ovary Syndrome (PCOS)
- _____ Addison's Disease
- _____ Scleroderma (Skin thickening/ Skin ulcers on the fingers)
- _____ Sjogren's Syndrome
- _____ Multiple Sclerosis
- _____ Insulin-Dependent Type 1 Diabetes
- _____ Fibromyalgia
- _____ Chronic Fatigue Syndrome
- _____ Rheumatoid Arthritis

If yes, to any of the above note the following individually for each disease:

Diagnosis

When were you diagnosed? <MM/ YYYY> ____/____/____

Did you undergo any treatment? Y N

If yes, what medications are/ were you taking?

Name _____

Dosage/ Frequency _____

When did you start the medication? <MM/YYYY> ____/____

Substance use

1. Do you smoke? Y N
 - a. If yes, how often do you smoke?
 - i. Very occasionally 1-2 times per month
 - ii. 2-3 times a week
 - iii. 1-3 cigarettes per day
 - iv. 4-10 cigarettes per day
 - v. 1 pack per day
 - vi. More than 1 pack per day
 - vii. Other (please explain) _____
 - b. Have you smoked in the past 48 hours? Y N
 - i. If yes, please answer the following:
 1. how many cigarettes did you smoke _____
 2. when was the last cigarette you smoked:
Date:
Time:
 - c. Are you currently in the process of trying to quit smoking? Y N
 - i. If yes, are you using any nicotine patches/ gum/ medication?
Y N
 - ii. Please describe what nicotine patches/ gum/ medication you are using _____
2. Did you consume any drinks containing alcohol in the past 48 hours? Y N
3. If yes, please answer the following:
 - a. how many drinks have you had in the past 48 hours?
 - b. when was the last drink you had
Date:
Time:

REFERENCES

REFERENCES

- Altemus, M., Cloitre, M., & Dhabhar, F. S. (2003). Enhanced cellular immune response in women with PTSD related to childhood abuse. *The American journal of psychiatry*, 160(9), 1705-1707.
- American Psychological Association. (2000). *Diagnostic and Statistical Manual of Mental Disorders- Text Revision* (4 ed.). Washington D.C.: American Psychological Association.
- Anda, R. F., Whitfield, C. L., Felitti, V. J., Chapman, D., Edwards, V. J., Dube, S. R., et al. (2002). Adverse childhood experiences, alcoholic parents, and later risk of alcoholism and depression. *Journal of Psychiatric Services*, 1001-1009.
- Anderson, C. M., Teicher, M. H., Polcari, A., & Renshaw, P. F. (2002). Abnormal T2 relaxation time in the cerebellar vermis of adults sexually abused in childhood: potential role of the vermis in stress-enhanced risk for drug abuse. *Psychoneuroendocrinology*, 27(1-2), 231-244.
- Atmaca, M., Kuloglu, M., Tezcan, E., Onal, S., & Ustundag, B. (2002). Neopterin levels and dexamethasone suppression test in posttraumatic stress disorder. *Eur Arch Psychiatry Clin Neurosci*, 252(4), 161-165.
- Babor, T. F., de la Fuente, J. R., Saunders, J., Grant, M., & World Health Organization. (1992). Alcohol Use Disorders Identification Test. http://whqlibdoc.who.int/hq/2001/WHO_MSD_MSB_01.6a.pdf
- Bal, S., De Bourdeauhui, I., Crombez, G., & van Oost, P. (2005). Predictors of trauma symptomatology in sexually abused adolescents: A 6-month follow-up study. *Journal of Interpersonal Violence*, 20, 1390-1405.
- Banyard, V. L., & Cantor, E. N. (2004). Adjustment to college among trauma survivors: An exploratory study of resilience. *Journal of College Student Development*, 45, 207-221.
- Banyard, V. L., & Williams, L. M. (1996). Characteristics of child sexual abuse as correlates of women's adjustment: A prospective study. *Journal of Marriage & the Family*, 58(4), 853-865.
- Bernstein, D. P., & Fink, L. (1998). *Childhood Trauma Questionnaire manual*. San Antonio, TX: The Psychological Corporation.
- Bernstein, D. P., Stein, J. A., Newcomb, M. D., Walker, E., Pogge, D., & Ahluvalia, T. (2003). Development and validation of a brief screening version of the Childhood Trauma Questionnaire. *Child Abuse and Neglect*, 27(2), 169-190.

- Bogat, G., Levendosky, A. A., Theran, S., von Eye, A., & Davidson, W. S. (2003). Predicting the psychosocial effects of interpersonal partner violence (IPV): How much does a woman's history of IPV matter? *Journal of Interpersonal Violence*, 18(11), 1271-1291.
- Boscarino, J. A. (1996). Posttraumatic stress disorder, exposure to combat, and lower plasma cortisol among Vietnam veterans: findings and clinical implications. *J Consult Clin Psychol*, 64(1), 191-201.
- Bowlby, J. (1982). *A secure base: Clinical applications of attachment theory*. London: Routledge.
- Bremner, J. D., & Brett, E. (1997). Trauma-related dissociative states and long-term psychopathology in posttraumatic stress disorder. *Journal of Traumatic Stress*, 10, 37-49.
- Bremner, J. D., Randall, P., Scott, T. M., Capelli, S., Delaney, R., McCarthy, G., et al. (1995). Deficits in short-term memory in adult survivors of childhood abuse. *Psychiatry research*, 59(1-2), 97-107.
- Bremner, J. D., Vythilingam, M., Vermetten, E., Adil, J., Khan, S., Nazeer, A., et al. (2003). Cortisol response to a cognitive stress challenge in posttraumatic stress disorder (PTSD) related to childhood abuse. *Psychoneuroendocrinology*, 28, 733-750.
- Briere, J., Stacey, K., & Green, B. L. (2008). Accumulated childhood trauma and symptom complexity. *Journal of Traumatic Stress*, 21(12), 223-226.
- Bunce, S. C., Larsen, R. J., & Peterson, C. (1995). Life after trauma: Personality and daily life experiences of traumatized people. *Journal of Personality and Social Psychology*, 63, 165-168.
- Bureau of Justice Statistics. (1998). *Bureau of Justice Statistics sourcebook of the criminal justice statistics--1997*: U.S. Department of Justice. NCJ171147.
- Carlson, E. B., & Putnam, F. (1993). An update on the dissociative experiences scale. *Dissociation*, 6(1), 16-27.
- Carpenter, L. L., Carvalho, J. P., Tyrka, A. R., Wier, L. M., Mello, A. F., Mello, M. F., et al. (2007). Decreased adrenocorticotrophic hormone and cortisol responses to stress in healthy adults reporting significant childhood maltreatment. *Biol Psychiatry*, 62(10), 1080-1087.
- Carrion, V. G., Weems, C. F., Ray, R. D., Glaser, B., Hessel, D., & Reiss, A. L. (2002). Diurnal salivary cortisol in pediatric posttraumatic stress disorder. *Biol Psychiatry*, 51(7), 575-582.

- Cascardi, M., Langhinrichsen, J., & Vivian, D. (1992). Marital aggression: Impact, injury, and health correlates for husbands and wives. *Archives of Internal Medicine*, 152, 1178-1184.
- Cascardi, M., & O'Leary, K. D. (1992). Depressive symptomatology, self-esteem, and self-blame in battered women. *Journal of Family Violence*. Vol 7(4), Dec 1992, pp. 249-259, 7(4), 249-259.
- Ceballo, R., Ramirez, C., Castillo, M., Caballero, G. A., & Lozoff, B. (2004). Domestic violence and women's mental health in Chile. *Psychology of Women Quarterly*, 28(4), 298-308.
- Christopoulos, C., Cohn, D. A., Shaw, D. S., Joyce, S., Sullivan-Hanson, J., & Kraft, S. (1987). Children of abused women: I. Adjustment at time of shelter residence. *Journal of Marriage and the Family*, 49(3), 611-619.
- Cicchetti, D., & Curtis, W. J. (2006). The developing brain and neural plasticity: Implications for the normality psychopathology & Resilience. In D. Cicchetti (Ed.), *Developmental psychopathology: Developmental neuroscience* (Vol. 2, pp. 1-64). New Jersey: John Wiley & Sons Inc.
- Coker, A. L., Smith, P. H., Thompson, M. P., McKeown, R. E., Bethea, L., & Davis, K. E. (2002). Social support protects against the negative effects of partner violence on mental health. *Journal of Women's Health and Gender Based Medicine*, 11(5), 465-476.
- Coker, A. L., Watkins, K. W., Smith, P. H., & Brandt, H. M. (2003). Social support reduces the impact of partner violence on health: Application of structural equation models. *Preventive Medicine: An International Journal Devoted to Practice and Theory*, 37(3), 259-267.
- Constans, J. I., Lenhoff, K., & McCarthy, M. (1997). Depression subtyping in PTSD patients. *Annals of Clinical Psychiatry*, 9(4), 235-240.
- Dai, J., Buijs, R., & Swaab, D. (2004). Glucocorticoid hormone (cortisol) affects axonal transport in human cortex neurons but shows resistance in Alzheimer's disease. *British Journal of Pharmacology*, 143(5), 606-610.
- Davis, R. C., & Brickman, E. (1991). Supportive and unsupportive responses of others to rape victims: Effects on concurrent victim adjustment. *American Journal of Community Psychology*, 19, 443-451.
- Day, A. L., Therrien, D. L., & Carroll, S. A. (2005). Predicting psychological health: Assessing the incremental validity of emotional intelligence beyond personality, type a behaviour, and daily hassles. *European Journal of Personality*, 19(6), 519-536.

- de Kloet, C. S., Vermetten, E., Heijnen, C. J., Geuze, E., Lentjes, E. G., & Westenberg, H. G. (2007). Enhanced cortisol suppression in response to dexamethasone administration in traumatized veterans with and without posttraumatic stress disorder. *Psychoneuroendocrinology*, 32(3), 215-226.
- DeBellis, M. D., Baum, A. S., Birmaher, B., Keshavan, M. S., Eccard, C. H., Boring, A. M., et al. (1999). A.E. Bennett Research Award: Developmental Traumatology. Part I: Biological stress systems. *Biological Psychiatry*, 45, 1259-1270.
- 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(2), 121-128.
- DeLongis, A., Folkman, S., & Lazarus, R. S. (1988). The impact of daily stress on health and mood: Psychological and social resources as mediators. *Journal of Personality and Social Psychology*, 54(3), 486-495.
- Dorahy, M. J., Lewis, C. A., & Wolfe, F. A. M. (2007). Psychological distress associated with domestic violence in Northern Ireland. *Current Psychology*, 25(4), 295-305.
- Doyle, J. P., Frank, E., Saltzman, L. E., McMahon, P. M., & Fielding, B. D. (1999). Domestic violence and sexual abuse in women physicians: Associated medical, psychiatric, and professional difficulties. *Journal of Women's Health and Gender Based Medicine*, 8(7), 955-965.
- Drapeau, M., & Perry, J. C. (2004). Childhood trauma and adult interpersonal functioning: A study using the Core Conflictual Relationship Theme Method (CCRT). *Child Abuse & Neglect*, 28(10), 1049-1066.
- Duhamel, M., & Fortin, A. (2004). Justification of violence towards children with mothers who are victims of domestic violence. *Canadian Journal of Community Mental Health*, 23(1), 47-63.
- Ebert, A., & Dyck, M. J. (2004). The experience of mental death: The core feature of complex posttraumatic stress disorder. *Clinical Psychology Review*, 24, 617-635.
- Elklit, A., Pedersen, S. S., & Jind, L. (2001). The crisis support scale: Psychometric qualities and further validation. *Personality and Individual Differences*, 31, 1291-1302.
- Elzinga, B. M., Roelofs, K., Tollenaar, M. S., Bakvis, P., van Pelt, J., & Spinhoven, P. (2008). Diminished cortisol responses to psychosocial stress associated with lifetime adverse events a study among healthy young subjects. *Psychoneuroendocrinology*, 33(2), 227-237.
- Findling, J. W., & Raff, H. (2006). Cushing's Syndrome: important issues in diagnosis and management. *Journal of clinical endocrinology and metabolism*, 91(10), 3746-3753.

- Finkelhor, D., & Yllo, K. (1987). *License to rape: Sexual abuse of wives*. New York: Free Press.
- First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1996a). *Structured Clinical Interview for DSMIV Axis I Disorders* (Research Version, Patient Edition SCID-I/P ed.). New York: New York State Psychiatric Institute.
- First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1996b). *Structured Clinical Interview for DSMIV Axis 2 Disorders* (Research Version, Patient Edition SCID-I/P ed.). New York: New York State Psychiatric Institute.
- Foa, E. B. (1995). *Posttraumatic Stress Diagnostic Scale*. Minneapolis, MN: Pearson, Inc.
- Frank, E., & Dingle, A. D. (1999). Self-reported depression and suicide attempts among U.S. women physicians. *American Journal of Psychiatry*, 156(2), 1887-1894.
- Frasier, P. Y., Belton, L., Hooten, E., Campbell, M. K., DeVellis, B., Benedict, S., et al. (2004). Disaster down east: Using participatory action research to explore intimate partner violence in eastern North Carolina. *Health Education and Behavior*, 31(4), 69s-84s.
- Friedman, M. J. (2002). Future pharmacotherapy for post-traumatic stress disorder: prevention and treatment. *Psychiatric clinics of North America*, 25(2), 427-441.
- Frischholz, E. J., Braun, B. G., Sachs, R. G., Hopkins, L., Schaeffer, D. M., Lewis, J., et al. (1990). The Dissociative Experiences Scale: Further replication and validation. *Dissociation*, 3(3), 151-153.
- Gershuny, B. S., Cloitre, M., & Otto, M. W. (2003). Peritraumatic dissociation and PTSD severity: do event-related fears about death and control mediate their relation? *Behavior Research and Therapy*, 41, 157-166.
- 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. *The American journal of psychiatry*, 929-934.
- Gold, P. B., Engdahl, B. E., Eberly, R. E., Blake, R. J., Page, W. F., & Frueh, B. C. (2000). Trauma exposure, resilience, social support, and PTSD construct validity among former prisoners of war. *Social Psychiatry and Psychiatric Epidemiology*, 35, 36-42.
- Golden, W. M., Weber, K. B., Hernandez, T. L., Sherman, S. I., Woodmansee, W. W., & Haugen, B. R. (2007). Single-dose rexinoid rapidly and specifically suppresses serum thyrotropin in normal subjects. *Journal of clinical endocrinology and metabolism*, 92(1), 124-130.

- Golding, J. M. (1999). Intimate partner violence as a risk factor for mental disorders: a meta-analysis. *Journal of Family Violence*, 14, 99-132.
- Granger, D. A., Kivlighan, K. T., El-Sheikh, M., Gordis, E. B., & Stroud, L. R. (2007). Salivary alpha-amylase in biobehavioral research - Recent developments and applications. *Annals of the New York Academy of Sciences*, 1098, 122-144.
- Griffin, M. G., Resick, P. A., & Mechanic, M. B. (1997). Objective assessment of peritraumatic dissociation: Psychophysiological indicators. *The American Journal of Psychiatry*, 154(8), 1081-1088.
- Griffin, M. G., Resick, P. A., & Yehuda, R. (2005). Enhanced cortisol suppression following dexamethasone administration in domestic violence survivors. *American journal of psychiatry*, 162(6), 1192-1199.
- Haller, D. L., & Miles, D. R. (2003). Victimization and perpetration among perinatal substance abusers. *Journal of Interpersonal Violence*, 18(7), 760-780.
- Hart, J., Gunnar, M., & Cicchetti, D. (1996). Altered neuroendocrine activity in maltreated children related to symptoms of depression. *Development and Psychopathology*, 8(1), 201-214.
- Heim, C., & Nemeroff, C. B. (2001). The role of childhood trauma in the neurobiology of mood and anxiety disorders: Preclinical and clinical studies. *Biological Psychiatry*, 49(12), 1023-1039.
- Heim, C., Newport, D. J., Heit, S., Graham, Y. P., Wilcox, M., Bonsall, R., et al. (2000). Pituitary-adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. *Journal of the American Medical Association*, 284(5), 592-597.
- Heim, C., Newport, D. J., Wagner, D., Wilcox, M. M., Miller, A. H., & Nemeroff, C. B. (2002). The role of early adverse experience and adulthood stress in the prediction of neuroendocrine stress reactivity in women: A multiple regression analysis. *Depression and Anxiety*, 15(3), 117-125.
- Henry, B., Moffitt, T. E., Caspi, A., Langley, J., & Silva, P. A. (1994). On the "remembrance of things past": A longitudinal evaluation of the retrospective method. *Psychological Assessment*, 6(2), 92-101.
- Herman, J. L. (1992). *Trauma and recovery*. New York: Basic Books, Inc.
- Hoffman, L., Burges Watson, P., Wilson, G., & Montgomery, J. (1989). Low plasma beta-endorphin in post-traumatic stress disorder. *Aust N Z J Psychiatry*, 23(2), 269-273.

- Houskamp, B. M., & Foy, D. W. (1991). The assessment of posttraumatic stress disorder in battered women. *Journal of Interpersonal Violence*, 6(3), 367-375.
- Inslicht, S. S., Marmar, C. R., Neylan, T. C., Metzler, T. J., Hart, S. L., Otte, C., et al. (2006). Increased cortisol in women with intimate partner violence-related posttraumatic stress disorder. *Psychoneuroendocrinology*, 31(7), 825-838.
- Ito, Y., Teicher, M. H., Glod, C. A., Harper, D., Magnus, E., & Gelbard, H. A. (1993). Increased prevalence of electrophysiological abnormalities in children with psychological, physical, and sexual abuse. *The Journal of neuropsychiatry and clinical neurosciences*, 401-408.
- Johnson, D. M., Delahanty, D. L., & Pinna, K. (2008). The cortisol awakening response as a function of PTSD severity and abuse chronicity in sheltered battered women. *Journal of Anxiety Disorders*, 22(5), 793-800.
- Johnson, D. M., Pike, J. L., & Chard, K. M. (2001). Factors predicting PTSD, depression, and dissociative severity in female treatment-seeking childhood sexual abuse survivors. *Child Abuse and Neglect*, 25(179-198).
- Jones, L. P., Gross, E., & Becker, I. (2002). The Characteristics of Domestic Violence Victims in a Child Protective Service Caseload. *Families in Society*, 83(4), 405-415.
- Joseph, S., Andrews, B., Williams, R., & Yule, W. (1992). Crisis support and psychiatric symptomatology in adult survivors of the Jupiter cruise ship disaster. *British Journal of Clinical Psychology*, 31, 63-73.
- Kaufman, J., Birmaher, B., Perel, J., Dahl, R. E., Moreci, P., Nelson, B., et al. (1997). The corticotropin-releasing hormone challenge in depressed abused, depressed nonabused, and normal control children. *Biological psychiatry*, 42(8), 669-679.
- Kaysen, D., Pantalone, D. W., Chawla, N., Lindgren, K. P., Clum, G. A., Lee, C., et al. (2008). Posttraumatic stress disorder, alcohol use, and physical health concerns. *Behavioral Medicine*, 31(2), 115-125.
- Kellner, M., Yehuda, R., Arlt, J., & Wiedemann, K. (2002). Longitudinal course of salivary cortisol in post-traumatic stress disorder. *Acta psychiatrica Scandinavica*, 105(2), 153-155; discussion 155-156.
- Kemp, A., Green, B. L., Hovanitz, C., & Rawlings, E. I. (1995). Incidence and correlates of posttraumatic stress disorder in battered women: Shelter and community samples. *Journal of Interpersonal Violence*, 10(1), 43-55.
- Kemp, A., Rawlings, E. I., & Green, B. L. (1991). Post-traumatic stress disorder (PTSD) in battered women: A shelter sample. *Journal of Traumatic Stress*, 4(1), 137-148.

- Kernic, M. A., Wolf, M. E., & Holt, V. L. (2000). Rates and relative risk of hospital admission among women in violent intimate partner relationships. *American Journal of Public Health, 90*(9), 1416-1420.
- Ketring, S. A., & Feinauer, L. L. (1999). Perpetrator-victim relationship: Long-term effects of sexual abuse for men and women. *American Journal of Family Therapy, 27*.
- King, D. W., King, L. K., Foy, D. W., & Gudanowski, D. M. (1996). Prewar factors in combat-related posttraumatic stress disorder: Structural equation modeling with a national sample of female and male Vietnam veterans. *Journal of Consulting and Clinical Psychology, 64*, 520-531.
- King, J. A., Mandansky, D., King, S., Fletcher, K. E., & Brewer, J. (2001). Early sexual abuse and low cortisol. *Psychiatry and clinical neurosciences, 55*(1), 71-74.
- Kirschbaum, C., Pirke, K. M., & Hellhammer, D. H. (1993). The 'Trier Social Stress Test'- a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology, 28*, 76-81.
- Leahy, T., Pretty, G., & Tenenbaum, G. (2004). Perpetrator methodology as a predictor of traumatic symptomatology in adult survivors of childhood sexual abuse. *Journal of Interpersonal Violence, 19*, 521-540.
- Lemieux, A. M., & Coe, C. L. (1995). Abuse-related posttraumatic stress disorder: evidence for chronic neuroendocrine activation in women. *Psychosomatic Medicine, 57*(2), 105-115.
- Levendosky, A. A., Bogat, G. A., Theran, S. A., Trotter, J. S., von Eye, A., & Davidson, W. S., II. (2004). The social networks of women experiencing domestic violence. *American of Community Psychology, 34*(12), 95-109.
- Lindley, S. E., Carlson, E. B., & Benoit, M. (2004). Basal and dexamethasone suppressed salivary cortisol concentrations in a community sample of patients with posttraumatic stress disorder. *Biological Psychiatry, 55*, 940-945.
- Lipschitz, D. S., Rasmusson, A. M., Yehuda, R., Wang, S., Anyan, W., Gueoguieva, R., et al. (2003). Salivary cortisol responses to dexamethasone in adolescents with posttraumatic stress disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 42*(11), 1310-1317.
- Lipsky, S., Field, C. A., Caetano, R., & Larkin, G. L. (2005). Posttraumatic stress disorder symptomatology and comorbid depressive symptoms among abused women referred from emergency department care. *Violence and Victims, 20*(6), 645-659.

- Lown, E., & Vega, W. A. (2001). Intimate partner violence and health: Self-assessed health, chronic health, and somatic symptoms among Mexican American women. *Psychosomatic Medicine*, 63(3), 352-360.
- Lucenko, B. A., Gold, S. N., & Cott, M. A. (2000). Relationship to perpetrator and posttraumatic symptomatology among sexual abuse survivors. *Journal of Family Violence*, 15, 169-179.
- Maes, M., Lin, A., Bonaccorso, S., van Hunsel, F., Van Gastel, A., Delmeire, L., et al. (1998). Increased 24-hour urinary cortisol excretion in patients with post-traumatic stress disorder and patients with major depression, but not in patients with fibromyalgia. *Acta psychiatrica Scandinavica*, 98(4), 328-335.
- Maffei, C., ., Fossati, A., Agostoni, I., Barraco, A., Bagnato, M., Deborah, D., et al. (1997). Interrater reliability and internal consistency of the structured clinical interview for DSM-IV axis II personality disorders (SCID-II) version 2.0. . *Journal of Personality Disorders*, 11(3), 279-284.
- Marshall, G. N., & Schell, T. L. (2002). Reappraising the link between dissociation and PTSD symptom severity: evidence from a longitudinal study of community violence survivors. *Journal of Abnormal Psychology*, 111, 626-636.
- Marshall, L. (1992). Development of the severity of violence against women scales. *Journal of Family Violence*, 7, 103-121.
- Mason, J. W., Wang, S., Yehuda, R., Lubin, H., Johnson, D., Bremner, J. D., et al. (2002). Marked lability in urinary cortisol levels in subgroups of combat veterans with posttraumatic stress disorder during an intensive exposure treatment program. *Psychosomatic Medicine*, 64(2), 238-246.
- McCollum, D. (2006). Child maltreatment and brain development. *Minnesota medicine*, 89(3), 48-50.
- Meewisse, M. L., Reitsma, J. B., de Vries, G. J., Gersons, B. P., & Olf, M. (2007). Cortisol and post-traumatic stress disorder in adults: systematic review and meta-analysis. *British journal of psychiatry*, 191, 387-392.
- Meisel, J., Chandler, D., & Rienzi, B. M. (2003). Domestic violence prevalence and effects on employment in two California TANF populations. *Violence Against Women*, 9(10), 1191-1212.
- Mertin, P., & Mohr, P. B. (2000). Incidence and correlates of posttraumatic stress disorder in Australian victims of domestic violence. *Journal of Family Violence*, 15(4), 411-422.

- Miller, G. E., Chen, E., & Zhou, E. S. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological Bulletin*, 133(1), 25-45.
- Mitchell, R. E., & Hodson, C. A. (1983). Coping with domestic violence: Social support and psychological health among battered women. *American Journal of Community Psychology*, 11, 629-654.
- Moore, T. E., Pepler, D., Mae, R., & Michele, K. (Eds.). (1989). *Effects of family violence on children: New directions for research and intervention*. Hillsdale, NJ: Lawrence Erlbaum Associates.
- Newport, D. J., Heim, C., Bonsall, R., Miller, A. H., & Nemeroff, C. B. (2004). Pituitary-adrenal responses to standard and low-dose dexamethasone suppression tests in adult survivors of child abuse. *Biol Psychiatry*, 55(1), 10-20.
- Neylan, T. C., Brunet, A., Pole, N., Best, S. R., Metzler, T. J., Yehuda, R., et al. (2005). PTSD symptoms predict waking salivary cortisol levels in police officers. *Psychoneuroendocrinology*, 30(4), 373-381.
- Nishi, D., Matsuoka, Y., Yonemoto, N., Noguchi, H., Kim, Y., & Kanba, S. (2010). Peritraumatic Distress Inventory as a predictor of post-traumatic stress disorder after a severe motor vehicle accident. *Psychiatry and Clinical Neurosciences*, 64(2), 149-156.
- Nishith, P., Mechanic, M. B., & Resick, P. A. (2000). Prior interpersonal trauma: The contribution to current PTSD symptoms in female rape victims. *Journal of Abnormal Psychology*, 109(1), 20-25.
- Nixon, R. D. V., Resick, P. A., & Nishith, P. (2004). An exploration of comorbid depression among female victims of intimate partner violence with posttraumatic stress disorder. *Journal of Affective Disorders*, 82(2), 315-320.
- Noll, J. G., Horowitz, L. A., Bonanno, G. A., Trickett, P. K., & Putnam, F. W. (2003). Revictimization and self-harm in females who experienced childhood sexual abuse: Results from a prospective study. *Journal of Interpersonal Violence*, 18(12), 1452-1471.
- Olf, M., de Vries, G.-J., Guezalcan, Y., Assies, J., & Gersons, B. P. R. (2007). Changes in cortisol and DHEA plasma levels after psychotherapy for PTSD. *Psychoneuroendocrinology*, 32(6), 619-626.
- Olf, M., Guezalcan, Y., de Vries, G.-J., Assies, J., & Gersons, B. P. R. (2006). HPA- and HPT-axis alterations in chronic posttraumatic stress disorder. *Psychoneuroendocrinology*, 31(10), 1220-1230.

- Olf, M., Langeland, W., & Gersons, B. P. R. (2005). Effects of appraisal and coping on the neuroendocrine response to extreme stress. *Neuroscience & Biobehavioral Reviews*, 29(3), 457-467.
- Olson, B. D., Curtis, C. E., Jason, L. A., Ferrari, J. R., Horin, E. V., & Davis, M. I. (2003). Physical and sexual trauma, psychiatric symptoms, and sense of community among women in recovery: Toward a new model of shelter aftercare. *Journal of Prevention and Intervention in the Community*, 26(1), 67-80.
- Perry, B. D. (1994). Neurobiological sequelae of childhood trauma: PTSD in children. In M. Murburg (Ed.), *Catecholamine Function in Posttraumatic Stress Disorder: Emerging Concepts* (pp. 173-189). Washington, DC: American Psychiatric Press.
- Pico-Alfonso, M. A., Garcia-Linares, M. I., Celda-Navarro, N., Herbert, J., & Martinez, M. (2004). Changes in cortisol and dehydroepiandrosterone in women victims of physical and psychological intimate partner violence. *Biological psychiatry*, 56(4), 233-240.
- Pollak, S. D. (2005). Early adversity and mechanisms of plasticity: integrating affective neuroscience with developmental approaches to psychopathology. *Dev Psychopathol*, 17(3), 735-752.
- Pollak, S. D., Cicchetti, D., Hornung, K., & Reed, A. (2000). Recognizing emotion in faces: developmental effects of child abuse and neglect. *Dev Psychol*, 36(5), 679-688.
- Pollak, S. D., & Kistler, D. J. (2002). Early experience is associated with the development of categorical representations for facial expressions of emotion. *Proc Natl Acad Sci U S A*, 99(13), 9072-9076.
- Pollak, S. D., Klorman, R., Thatcher, J. E., & Cicchetti, D. (2001). P3b reflects maltreated children's reactions to facial displays of emotion. *Psychophysiology*, 38(2), 267-274.
- Pollak, S. D., & Sinha, P. (2002). Effects of early experience on children's recognition of facial displays of emotion. *Dev Psychol*, 38(5), 784-791.
- Pollak, S. D., & Tolley-Schell, S. A. (2003). Selective attention to facial emotion in physically abused children. *J Abnorm Psychol*, 112(3), 323-338.
- Pollak, S. D., Vardi, S., Putzer Bechner, A. M., & Curtin, J. J. (2005). Physically abused children's regulation of attention in response to hostility. *Child Dev*, 76(5), 968-977.
- Pryce, C. R., Rüedi-Bettschen, D., Dettling, A. C., Weston, A., Russig, H., Ferger, B., et al. (2005). Long-term effects of early-life environmental manipulations in rodents

- and primates: Potential animal models in depression research. *Neurosci Biobehav Rev*, 29(4-5), 649-674.
- Rabois, D., Batten, S. V., & Keane, T. M. (2002). Implications of biological findings for psychological treatments of post-traumatic stress disorder. *Psychiatric clinics of North America*, 25(2), 443-462, viii.
- Rasmusson, A. M., Lipschitz, D. S., Wang, S., Hu, S., Vojvoda, D., Bremner, J. D., et al. (2001). Increased pituitary and adrenal reactivity in premenopausal women with posttraumatic stress disorder. *Biological psychiatry*, 50(12), 965-977.
- Regehr, C., LeBlanc, V., Jelley, R. B., Barath, I., & Daciuk, J. (2007). Previous trauma exposure and PTSD symptoms as predictors of subjective and biological response to stress. *Canadian journal of psychiatry*, 52(10), 675-683.
- Rennison, C. M. (2003). *Intimate partner violence, 1993-2001*: U.S. Department of Justice. NCJ 197838: Bureau of Justice Statistics.
- Resnick, H. S., Yehuda, R., Pitman, R. K., & Foy, D. W. (1995). Effect of previous trauma on acute plasma cortisol level following rape. *American Journal of Psychiatry*, 152(11), 1675-1677.
- Roberts, G. L., Lawrence, J. M., O'Toole, B. I., & Raphael, B. (1997). Domestic violence in the emergency department: I. Two case-control studies of victims. *General Hospital Psychiatry*, 19(1), 5-11.
- Rohleder, N., Joksimovic, L., Wolf, J. M., & Kirschbaum, C. (2004). Hypocortisolism and increased glucocorticoid sensitivity of pro-Inflammatory cytokine production in Bosnian war refugees with posttraumatic stress disorder. *Biological psychiatry*, 55(7), 745-751.
- Rosenthal, R., & Rosnow, R. L. (1991). *Essentials of behavioral research: Methods and data analysis*. New York: McGraw-Hill.
- Rosenthal, S., Feiring, C., & Taska, L. (2003). Emotional support and adjustment over a year's time following sexual abuse discovery. *Child Abuse and Neglect*, 27, 641-661.
- Russell, D. E. H. (1986). *The secret trauma*. New York: Basic Books.
- Russell, D. E. H. (1990). *Rape in marriage*. Bloomington, IN, US: Indiana University Press.
- Salimetrics, L. (2008). EXPANDED RANGE High Sensitivity SALIVARY CORTISOL ENZYME IMMUNOASSAY KIT. from http://www.salimetrics.com/Portals/0/pdfs/ER_Cort_Research_Kit_Insert.pdf

- Saltzman, K. M., Holden, G. W., & Holahan, C. J. (2005). The psychobiology of children exposed to marital violence. *Journal of Clinical Child and Adolescent Psychology*, 34(1), 129-139.
- Sansone, R. A., Reddington, A., Sky, K., & Wiederman, M. W. (2007). Borderline personality symptomatology and history of domestic violence among women in an internal medicine setting. *Violence and Victims*, 22(1), 120-126.
- Santa Ana, E. J., Saladin, M. E., Back, S. E., Waldrop, A. E., Spratt, E. G., McRae, A. L., et al. (2006). PTSD and the HPA axis: Differences in response to the cold pressor task among individuals with child vs. adult trauma. *Psychoneuroendocrinology*, 31(4), 501-509.
- Sapolsky, R. M. (1996). Why stress is bad for your brain. *Science*, 273, 749-750.
- Saunders, B. E., Kilpatrick, D. G., Hanson, R. F., Resnick, H. S., & Walker, M. E. (1999). Prevalence, case characteristics, and long-term psychological correlates of child rape among women: A national survey. *Child Maltreatment*, 4, 187-200.
- Schore, A. (2002a). Advances in neuropsychanalysis, attachment theory, and trauma research: Implications for self psychology. *Psychoanalytic Inquiry*, 22(3), 433-484.
- Schore, A. (2002b). Dysregulation of the right brain: a fundamental mechanism of traumatic attachment and the psychopathogenesis of posttraumatic stress disorder. *Australian and New Zealand Journal of Psychiatry*, 36(1), 9-30.
- Seedat, S., Stein, M. B., & Forde, D. R. (2005). Association Between Physical Partner Violence, Posttraumatic Stress, Childhood Trauma, and Suicide Attempts in a Community Sample of Women. *Violence and Victims*, 20(1), 87-98.
- Seedat, S., Stein, M. B., Kennedy, C. M., & Hauger, R. L. (2003). Plasma cortisol and neuropeptide Y in female victims of intimate partner violence. *Psychoneuroendocrinology*, 28(6), 796-808.
- Sharhabani-Arzy, R., Amir, M., Kotler, M., & Liran, R. (2003). The toll of domestic violence: PTSD among battered women in an Israeli sample. *Journal of Interpersonal Violence*, 18(11), 1335-1346.
- Shea, M. T., Zlotnick, C., & Weisberg, R. B. (1999). Commonality and specificity of personality disorder profiles in subjects with trauma histories. *Journal of Personality Disorders*, 13(3), 199-210.
- Sierra, M., Senior, C., Dalton, J., McDonough, M., Bond, A., Phillips, M. L., et al. (2002). Autonomic response in depersonalization disorder. *Archives of General Psychiatry*, 59(9), 833-838.

- Simeon, D., Greenberg, J., Nelson, D., Schmeidler, J., & Hollander, E. (2005). Dissociation and posttraumatic stress one year after the World Trade Center disaster. *Journal of clinical psychiatry*, 66, 231-237.
- Simeon, D., Knutelska, M., Smith, L., Baker, B. R., & Hollander, E. (2007). A preliminary study of cortisol and norepinephrine reactivity to psychosocial stress in borderline personality disorder with high and low dissociation. *Psychiatry research*, 149(1-3), 177-184.
- Simeon, D., Knutelska, M., Yehuda, R., Putnam, F., Schmeidler, J., & Smith, L. M. (2007). Hypothalamic-pituitary-adrenal axis function in dissociative disorders, post-traumatic stress disorder, and healthy volunteers. *Biological psychiatry*, 61(8), 966-973.
- Simeon, D., Yehuda, R., Knutelska, M., & Schmeidler, J. (2008). Dissociation versus posttraumatic stress: cortisol and physiological correlates in adults highly exposed to the World Trade Center attack on 9/11. *Psychiatry Res*, 161(3), 325-329.
- Smith, P. H., Thornton, G. E., DeVellis, R., Earp, J., & Coker, A. L. (2002). A population-based study of the prevalence and distinctiveness of battering, physical assault, and sexual assault in intimate relationships. *Violence Against Women*, 8(10), 1208-1232.
- Stanley, N., & Penhale, B. (1999). The mental health problems of mothers experiencing the child protection system: Identifying needs and appropriate responses. *Child Abuse Review*, 8(1), 34-45.
- Stein, M. B., & Kennedy, C. (2001). Major depressive and post-traumatic stress disorder comorbidity in female victims of intimate partner violence. *Journal of Affective Disorders*, 66(2-3), 133-148.
- Stein, M. B., Yehuda, R., Koverola, C., & Hanna, C. (1997). Enhanced dexamethasone suppression of plasma cortisol in adult women traumatized by childhood sexual abuse. *Biol Psychiatry*, 42(8), 680-686.
- Suliman, S., Mkabile, S. G., Fincham, D. S., Rashid, A., 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(2), 121-127.
- Sullivan, T. P., & Holt, L. J. (2008). PTSD symptom clusters are differentially related to substance use among community women exposed to intimate partner violence. *Journal of Traumatic Stress*, 21(2), 173-180.
- Tajima, E. A. (2004). Correlates of the Co-Occurrence of Wife Abuse and Child Abuse Among a Representative Sample. *Journal of Family Violence*, 19(6), 399-410.

- Teicher, M. H., Ito, Y., Glod, C. A., Andersen, S. L., Dumont, N., & Ackerman, E. (1997). Preliminary evidence for abnormal cortical development in physically and sexually abused children using EEG coherence and MRI. *Annals of the New York Academy of Sciences*, 821, 160-175.
- Thaller, V., Vrkljan, M., Hotujac, L., & Thakore, J. (1999). The potential role of hypocortisolism in the pathophysiology of PTSD and psoriasis. *Collegium antropologicum*, 23(2), 611-619.
- Tjaden, P., & Thoennes, N. (1998). *Prevalence, incidence, and consequences of violence against women: Findings from the National Violence Against Women Survey*. U.S. Department of Justice, National Institute of Justice, Centers for Disease Control and Prevention. NCJ 172837.
- Tjaden, P., & Thoennes, N. (2000a). *Extent, nature, and consequences of intimate partner violence: Findings from the National Violence Against Women Survey*. U.S. Department of Justice, National Institute of Justice. NCJ 181867.
- Tjaden, P., & Thoennes, N. (2000b). *Full report of the prevalence, incidence, and consequences of violence against women. Findings from the National Violence Against Women Survey*. U.S. Department of Justice, National Institute of Justice. NCJ 183781.
- Tremblay, R. E., Nagin, D. S., Seguin, J. R., Zoccolillo, M., Zelazo, P. D., Boivin, M., et al. (2004). Physical Aggression During Early Childhood: trajectories and Predictors. *Pediatrics*, 114, 43-50.
- Tupler, L. A., & De Bellis, M. D. (2006). Segmented hippocampal volume in children and adolescents with posttraumatic stress disorder. *Biological psychiatry*, 59(6), 523-529.
- van der Hart, O., Nijenhuis, E. R. S., & Steele, K. (2006). *The haunted self: Structural dissociation and the treatment of chronic traumatization*. New York: W W Norton & Company.
- Waltermauer, E. M., Ortega, C. A., & McNutt, L. A. (2003). Issues in estimating the prevalence of intimate partner violence. Assessing the impact of abuse status on participation bias. *Journal of Interpersonal Violence*, 18(9), 959-974.
- Weaver, T. L., & Etzel, J. C. (2003). Smoking patterns, symptoms of PTSD and depression: Preliminary findings from a sample of severely battered women. *Addictive Behaviors*, 28(9), 1665-1679.
- Wessa, M., Rohleder, N., Kirschbaum, C., & Flor, H. (2006). Altered cortisol awakening response in posttraumatic stress disorder. *Psychoneuroendocrinology*, 31(2), 209-215.

- Wolfe, D. A., Crooks, C. V., Lee, V., McIntyre Smith, A., & Jaffe, P. G. (2003). The effects of children's exposure to domestic violence: A meta-analysis and critique. *Clinical Child and Family Psychology Review*, 6(3), 171-187.
- Wolfe, D. A., Zak, L., Wilson, S. K., & Jaffe, P. (1986). Child witnesses to violence between parents: Critical issues in behavioral and social adjustment. *Journal of Abnormal Child Psychology*, 14(1), 95-104.
- Yehuda, R. (2002). Current status of cortisol findings in post-traumatic stress disorder. *Psychiatric clinics of North America*, 25(2), 341-368, vii.
- Yehuda, R., Boisoineau, D., Lowy, M. T., & Giller, E. L., Jr. (1995). Dose-response changes in plasma cortisol and lymphocyte glucocorticoid receptors following dexamethasone administration in combat veterans with and without posttraumatic stress disorder. *Arch Gen Psychiatry*, 52(7), 583-593.
- Yehuda, R., Golier, J. A., Halligan, S. L., Meaney, M., & Bierer, L. M. (2004). ACTH response to dexamethasone in PTSD. *American journal of psychiatry*, 161(8), 1397-1403.
- Yehuda, R., Halligan, S. L., & Bierer, L. M. (2001). Relationship of parental trauma exposure and PTSD to PTSD, depressive and anxiety disorders in offspring. *J Psychiatr Res*, 35(5), 261-270.
- Yehuda, R., Halligan, S. L., & Bierer, L. M. (2002). Cortisol levels in adult offspring of Holocaust survivors: relation to PTSD symptom severity in the parent and child. *Psychoneuroendocrinology*, 27(1-2), 171-180.
- Yehuda, R., Halligan, S. L., Golier, J. A., Grossman, R., & Bierer, L. M. (2004). Effects of trauma exposure on the cortisol response to dexamethasone administration in PTSD and major depressive disorder. *Psychoneuroendocrinology*, 29(3), 389-404.
- Yehuda, R., Halligan, S. L., & Grossman, R. (2001). Childhood trauma and risk for PTSD: Relationship to intergenerational effects of trauma, parental PTSD, and cortisol excretion. *Development and Psychopathology. Special Issue: Stress and development: Biological and psychological consequences*, 13(3), 733-753.
- Yehuda, R., Halligan, S. L., Grossman, R., Golier, J. A., & Wong, C. (2002). The cortisol and glucocorticoid receptor response to low dose dexamethasone administration in aging combat veterans and Holocaust survivors with and without posttraumatic stress disorder. *Biological Psychiatry*, 52(5), 393-403.
- Yehuda, R., Schneidler, J., Siever, L. J., Binder-Brynes, K., & Elkin, A. (1997). Individual differences in posttraumatic stress disorder symptom profiles in Holocaust survivors in concentration camps or in hiding. *Journal of Traumatic Stress*, 10, 453-463.

- Yehuda, R., Southwick, S. M., Nussbaum, G., Wahby, V., Giller, E. L., Jr., & Mason, J. W. (1990). Low urinary cortisol excretion in patients with posttraumatic stress disorder. *J Nerv Ment Dis*, 178(6), 366-369.
- Yehuda, R., Teicher, M. H., Trestman, R. L., Levengood, R. A., & Siever, L. J. (1996). Cortisol regulation in posttraumatic stress disorder and major depression: a chronobiological analysis. *Biological psychiatry*, 40(2), 79-88.
- Young, E. A., & Breslau, N. (2004a). Cortisol and catecholamines in posttraumatic stress disorder: an epidemiologic community study. *Archives of general psychiatry*, 61(4), 394-401.
- Young, E. A., & Breslau, N. (2004b). Saliva cortisol in posttraumatic stress disorder: a community epidemiologic study. *Biological psychiatry*, 56(3), 205-209.
- Young, E. A., Toman, R., Witkowski, K., & Kaplan, G. (2004). Salivary cortisol and posttraumatic stress disorder in a low-income community sample of women. *Biological Psychiatry*, 55, 621-626.
- Zanarini, M. C., Skodol, A. E., Bender, D., Dolan, R., Sanislow, C., Schaefer, E., et al. (2000). The Collaborative Longitudinal Personality Disorders Study: reliability of axis I and II diagnoses. *Journal of Personality Disorders*, 12(4), 291-299.

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