9-2010

An Examination of Hypothalamic-Pituitary-Adrenal Axis Reactivity as a Partial Mediator of the Relation Between Trauma and Self-injurious Behavior

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AN EXAMINATION OF HYPOTHALAMIC-PITUITARY-ADRENAL AXIS REACTIVITY AS A PARTIAL MEDIATOR OF THE RELATION BETWEEN TRAUMA AND SELF-INJURIOUS BEHAVIOR

A Dissertation Presented

by

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

DOCTOR OF PHILOSOPHY

September 2010

Clinical Psychology

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ACKNOWLEDGMENTS

I wish to express my unending gratitude to my advisor, Sally Powers, for her thoughtful guidance and enthusiastic support, not only through the process of working on this dissertation project, but throughout graduate school. She has been a wonderful support and role model over these past six years.

I also wish to thank my committee members for their helpful comments and suggestions on all stages of this project. Thanks to Aline Sayer for her guidance on statistical and methodological issues, both in this project and on others. Thanks also to Jerrold Meyer and Christine King for their feedback and support. Thank you all for helping me to become a better researcher.

My friends, classmates, and family have also provided the encouragement and support that helped keep me going during this process. Special thanks to Mark Manning, who offered both statistical and moral support. Finally, thanks to the 400 individuals who participated in the study, who patiently answered hundreds of questions, spit into tubes, and were observed arguing with their partners. I have learned so much from you.

ABSTRACT

AN EXAMINATION OF HYPOTHALAMIC-PITUITARY-ADRENAL AXIS REACTIVITY AS A PARTIAL MEDIATOR OF THE RELATION BETWEEN TRAUMA AND SELF-INJURIOUS BEHAVIOR

SEPTEMBER 2010

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Past work has linked self-injurious behavior (SIB) to a history of traumatic experiences and to problems regulating affect. While this affect dysregulation is conceptualized as occurring at a biological (as well as a behavioral) level, relatively little is known about the biological mechanisms involved. The current study explored whether reactivity of the hypothalamic-pituitary-adrenal (HPA) axis to an interpersonal stressor mediated the relation between trauma and SIB in a sample of 178 18-21 year-old heterosexual dating couples. As predicted, both trauma experience and symptoms positively predicted SIB. While the mediating model was not supported, SIB was associated with an HPA axis response marked by heightened reactivity to interpersonal stress within the context of lower cortisol levels. Trauma symptoms and experience interacted with adult attachment security to predict HPA axis response in different ways for men and women, a compelling set of findings suggesting the importance of contextual factors in the study of trauma and HPA axis function. Future directions for the study of trauma, HPA axis reactivity, and SIB are discussed.

TABLE OF CONTENTS

Page

LIST OF TABLES

Table Page

LIST OF FIGURES

Figure Page

CHAPTER 1

INTRODUCTION

Self-injurious behavior (SIB) is a disturbing and potentially dangerous behavior that is seen with some frequency not only in clinical populations, but also among the general population, particularly among adolescents (e.g., DiClemente, Ponton, & Hartley, 1991; Whitlock, Eckenrode, & Silverman, 2006). Several etiological and functional theories of SIB suggest that difficult life experiences in childhood (including abuse, neglect, and parental separation and loss) interfere with the development of adaptive affect regulation strategies, leaving individuals vulnerable to maladaptive regulation strategies such as SIB (e.g., Yates, 2004). While this affect dysregulation is conceptualized as occurring at a biological (as well as a behavioral) level, relatively little is known about the biological mechanisms involved with SIB and SIB researchers have emphasized the importance of future work understanding the biochemistry of self-injury (e.g., Klonksy, 2007). Despite its major role in regulating the body's response to stress and arousal, the hypothalamic-pituitary-adrenal (HPA) system has received little attention in the study of SIB. The current study aims to better understand the links between trauma and SIB. Specifically, this study evaluates a proposed model of SIB behavior in which HPA axis reactivity in response to a stressor partially mediates the relation between trauma and SIB.

Definition of SIB

Broadly defined, self-injurious behavior refers to a range of deliberate behaviors that involve the direct destruction or alteration of one's body without conscious suicidal intent (Favazza, 1998). These behaviors fall along a continuum ranging from relatively

benign and socially acceptable forms, such as ear piercing and tattooing, to severely harmful and uniformly socially unacceptable forms, such as eye enucleation and autocastration (Walsh & Rosen, 1988). Several categorization schemes have been proposed to better understand and classify these behaviors. Given the variety of terms and definitions used in the literature on SIB, a brief overview of two well-regarded classification schemes is presented to clarify how SIB is conceptualized in this study.

Walsh and Rosen (1988) propose four types of behaviors involving "the alteration of physical appearance and body configuration" (p. 6) that are generated from combinations of three dimensions: 1) severity of bodily damage, 2) psychological state during self-injury, and 3) social acceptability. According to this typology, neither Type I (including small body piercings and tattoos) nor Type II self-injury (including more radical piercings, large tattoos, and ritual scarring) is considered pathological because these behaviors are not deviant along all three dimensions. Types III and IV, however, are considered to be pathological behaviors. Type III self-injury includes behaviors such as skin-cutting and self-inflicted skin-burning. These behaviors involve mild to moderate bodily damage, are performed while in a state of psychological crisis, and are generally unacceptable in all social groups, with the possible exception of a few peers who also engage in the behavior. Type IV self-injury involves behaviors such as autocastration, eye enculeation, and limb amputation that involve severe bodily harm, are performed in a psychotic state, and are uniformly socially unacceptable.

Favazza's system (1996, 1998) distinguishes self-injurious behaviors that are culturally sanctioned from those that are socially deviant. He classifies deviant forms of self-injurious behavior into three categories, Major, Stereotypic, and

Superficial/Moderate, based on the degree of tissue damage, rate, and pattern of the behavior. Major self-injury is similar to Walsh and Rosen's Type IV and includes extreme behaviors that are most commonly associated with psychosis or acute intoxication. These acts involve severe tissue damage, are infrequent, and tend to occur suddenly. Stereotypic self-injury includes behaviors such as head banging and self-biting that are typically monotonous, repetitive, and rhythmic and are most commonly seen among institutionalized mentally retarded individuals and individuals with autism, psychosis, and some genetic disorders. Superficial/moderate self-injury is similar to Walsh and Rosen's Type III behaviors. These acts include skin cutting, burning, and wound excoriation.

The present study focuses on Type III or Superficial/Moderate self-injury. A variety of terms have been used to refer to this phenomenon, the most common of which include SIB, deliberate self-injury, non-suicidal self-injury, and self-mutilation. In reviewing the relevant literature, I use the term SIB regardless of the term used by the original researchers. Unless otherwise noted, the phenomena discussed fit within the Type III and Superficial/Moderate SIB classifications.

Prevalence

This type of SIB is of great clinical significance because of its prevalence among individuals with psychiatric diagnoses as well as the general population. Prevalence estimates vary widely, in part because the incidence of SIB is difficult to measure due to underreporting or concealment by individuals engaging in these behaviors. Additionally, definitions of SIB used in research vary widely, with some being overinclusive (e.g., including drug overdose and apparent suicide attempts), and others being underinclusive

(e.g., studying only wrist cutting). Despite the flaws in estimating the prevalence of SIB, most studies do report relatively high rates among clinical populations. Briere and Gil (1998) found that 20-21% of their clinical sample (including both outpatients and inpatients) reported at least occasional self-injury. Studies of adolescent clinical samples report prevalence rates as high as 61% (DiClemente et al., 1991).

While SIB has perhaps been most commonly associated with borderline personality disorder, it is also associated with a number of other Axis I and Axis II disorders. In their sample of self-injurers, Briere and Gil (1998) reported that the most common psychiatric disorders reported were post-traumatic stress disorder (PTSD) (73%), unspecified dissociative disorders (40%), borderline personality disorder (37%), and dissociative identity disorder (29%). SIB is also associated with depression, substance abuse, eating disorders, schizophrenia, anxiety disorders, and other personality disorders (Suyemoto, 1998). It is important to note, however, that the estimates of comorbidity between SIB and Axis I and II conditions may be overestimated because most studies of SIB rely on clinical (often inpatient) and incarcerated samples.

General population studies suggest that SIB also occurs in the non-clinical population and is not exclusively a symptom of a specific form of psychopathology. Estimates in the general adult population vary from .75% (Favazza & Conterio, 1988) to 4% (Briere & Gil, 1998). Similar to work in clinical samples, prevalence rates are highest in non-clinical adolescent and young adult samples, with prevalence estimates ranging from 13% to 38% (Gratz, Conrad, & Roemer, 2002; Muehlenkamp & Gutierrez, 2004; Ross & Heath, 2002). In a large study of almost 3000 adolescents (Whitlock et al., 2006), 17% of participants reported having engaged in SIB at some point in their lives

and the 12-month prevalence was 7.3%. Rates of help-seeking were also quite low, with 40% of self-injurers reporting that no one was aware of the behavior and only 21% reporting having disclosed SIB to a mental health professional. Thus, SIB is a problem among both clinical and non-clinical populations, particularly among adolescents. Further, because of the secrecy and shame associated with the behavior, SIB may go unrecognized and untreated, especially in the general population.

Proposed Functions of SIB

Several theories have been offered to explain the functions served by SIB (see Klonsky, 2007 and Suyemoto, 1998 for extensive reviews). These theories are influenced by various theoretical orientations (psychoanalytic, psychodynamic/object relations, learning theory, interpersonal, and so on) and lay primary emphasis, respectively, on motivations such as sexual and aggressive conflicts, expression and regulation of emotion, modeling, reinforcement, and the creation of boundaries between self and other. Whether or not it is the main focus of the functional theory, the idea that SIB serves to regulate affect or reduce tension is involved in most of these theories. Further, models that place primary emphasis on affect regulation and tension-reduction have garnered the most empirical support (Suyemoto, 1998). These models posit that a primary function of SIB is the alleviation of intolerable emotion or tension that the individual experiences as overwhelming. The emotions or internal experiences involved may include tension, anxiety, loneliness, depression, emptiness, anger, dissociation, and intrusive flashbacks (Briere & Gil, 1998).

Empirical support for affect regulation and tension-reduction theories comes from numerous studies in which self-injurers report their reasons for engaging in SIB. Many

of the reasons reported involve attempts to manage or escape from uncomfortable emotional and psychological states. In a sample of female inpatients, the most common reason given for SIB was "to end intolerable tension" in the context of overwhelming affect (Herpertz, 1995, p. 61). Similarly, 98% of the adolescent inpatient self-injurers in Nixon, Cloutier, and Aggarwal's study (2002) provided affect-related reasons for engaging in SIB (examples included coping with depression, relieving unbearable tension, coping with anxiety or fear, expressing anger, dealing with unbearable pain, and ending dissociative experiences). Studies conducted by DiClemente et al. (1991) and Favazza and Conterio (1989) found that individuals engaging in SIB reported similar motives. Klonksy (2007) reviewed 11 studies in which individuals reported their reasons for engaging in SIB. In both clinical and non-clinical samples of adults and adolescents, affect-regulation reasons were endorsed by a majority of participants. Primate studies identify anxiety (as induced by a pharmacological agent) as a trigger for self-biting behavior in some monkeys, also implicating distress-reduction in the phenomenology of SIB (Major et al., 2009).

Further support for affect regulation models comes from descriptions of the phenomenology of SIB behavior. The accounts of SIB provided in the research literature are remarkably consistent (see Haines, Williams, Brain, & Wilson, 1995; Herpertz, 1995; Winchel & Stanley, 1991 for examples and Klonksy, 2007 for a review). Prior to the self-injurious act, the individual experiences a negative, dysphoric affective state that is often brought about by some kind of interpersonal stressor, such as real or perceived abandonment or rejection (Haines et al., 1995). Dysphoric affect increases and evolves into a feeling of mounting tension, often accompanied by feelings of emptiness and

numbness. The act of SIB is typically followed by an immediate reduction in dysphoric affect (e.g., reduced anger and anxiety, increased calm).

Evidence from both human and primate studies suggests that SIB is temporarily effective in reducing tension and negative affect. Nixon et al. (2002), for instance, found that 93% of self-injurers in their sample experienced relief following an act of self-injury. Kemperman, Russ, and Shearin (1997) reported that all 38 of their self-injuring female sample reported some form of mood enhancement following acts of self-injury (e.g., decrease in negative affect, increase in positive affect, and decrease in dissociation). Haines et al. (1995) offer physiological evidence of the tension-reducing properties of SIB. Incarcerated men with a history of SIB showed decreased psychophysiological arousal on several measures after exposure to an imaginal script involving self-injury. A similar decrease was not seen in the prison or non-prison controls without a history of SIB. Similar results were found using this methodology in a non-incarcerated sample (Brain, Haines, & Williams, 1998). Studies of primates directly assessed physiological arousal before, during, and after incidents of SIB. In a small sample of rhesus monkeys who engaged in self-biting, heart rate increased just prior to acts of self-biting and then decreased to baseline levels within 30-60 seconds of the biting incident (Marinus, Chase, Rasmussen, Jorgensen, & Novak, 1999). While this finding emerges from only one small sample of rhesus monkeys, and thus the generalizability to human samples is unclear, this work contributes to the evidence for the tension-reducing properties of SIB.

SIB and Childhood Trauma and Stress

Consistent with the notion that SIB is an attempt to regulate negative and overwhelming affect, work exploring the risk factors associated with SIB offers some

explanation as to why individuals who self-injure may have particular difficulty with affect regulation. Much of the literature studying the risk factors for SIB has focused on traumatic childhood experiences, including sexual and physical abuse, neglect, and parental separation and loss. Further, these types of childhood experiences have been linked to the development of affect dysregulation (Gunnar, 2000; Schore, 2002), suggesting a possible mediating path from childhood trauma to SIB.

In the work on the childhood origins of SIB, one of the most robust and consistent findings is the association between childhood sexual abuse and later SIB, an association found in both clinical and non-clinical samples (e.g., Briere & Gil, 1998; Darche, 1990; Gladstone et al., 2004; Gratz, et al., 2002; Langbehn & Pfohl, 1993; van der Kolk, Perry, & Herman, 1991; Zlotnick et al., 1996). For instance, in an inpatient female sample, Zlotnick et al. (1996) found that 79% of women who engaged in SIB also reported a history of childhood sexual abuse. Moreover, several studies find that childhood sexual abuse has a unique relationship to later SIB even when other distressing childhood experiences (such as separation, loss, physical abuse, and neglect) are controlled (Boudewyn & Liem, 1995; Gratz et al., 2002; see Zweig-Frank, Paris, & Guzder, 1994ab for notable exceptions).

While childhood sexual abuse typically shows the strongest association with later SIB, other difficult childhood experiences are also related to SIB. Several studies, for instance, reveal an association between childhood physical abuse and later SIB (Carroll, Schaffer, Spensley, & Ambramowitz, 1980; Langhbehn & Pfohl, 1993; van der Kolk et al., 1991), although others find no association (e.g., Gladstone et al., 2004; Zweig-Frank et al., 1994ab). Work exploring gender differences offers some suggestion that the link

between childhood physical abuse and later SIB may be stronger for men (Gratz et al., 2002).

Childhood neglect has been related to SIB, although evidence for this association is also mixed. In support of the link between childhood neglect and adult SIB, Dubo, Zannarini, and Lewis (1997) found that emotional abuse was a stronger predictor of SIB than either childhood sexual or physical abuse; physical neglect, however, did not predict SIB. van der Kolk et al. (1991) found a positive association between childhood neglect and SIB in a sample of individuals with a personality disorder or bipolar II illness and Martin and Waite (1994)[1] found a similar association between SIB and emotional neglect in a non-clinical adolescent sample. In a college sample, Gratz et al. (2002) found that parental emotional neglect predicted SIB among women, but not men. Other work, however, has found no relation between childhood neglect and adult SIB (e.g., Zweig-Frank et al., 1994ab).

The relatively small number of studies investigating the association between SIB and childhood separation and loss offers some evidence to suggest an association with parental separation. van der Kolk et al. (1991), for instance, found that separation from parents was associated with cutting, but not other forms of SIB. Gratz et al. (2002) found that childhood separation was the most significant predictor of SIB among males, but did not predict SIB in females.

Work with primates also suggests associations between early adversity and SIB. Classic work done by Harlow and Harlow (1962) found that the single best predictor of SIB and other stereotypic behaviors is an impoverished early rearing environment. Monkeys reared in isolation for the first six months of life developed “isolation

syndrome," a pervasive pattern of abnormal behaviors. Further, many of these monkeys, especially males, went on to develop SIB in adolescence (Suomi, Harlow, & Kimball, 1971). More recent studies of rhesus macaques have linked the development of SIB to early and chronic exposure to stressful events (Novak, 2003). SIB is associated with a number of stressors in early life, including individual (as opposed to social) housing at an early age, longer time in individual housing, rearing by peer surrogates (as opposed to mothers), and greater exposure to stressful medical procedures such as blood draws (Jorgensen, Kinsey, & Novak, 1998; Lutz, Chase, & Novak, 2000; Lutz, Davis, Ruggiero, & Suomi, 2007; Lutz, Well, and Novak, 2003).

Thus, in both human and primate samples, several traumatic or stressful life experiences contribute to the risk of engaging in SIB behavior in adolescence and adulthood. Moreover, these childhood variables have also been linked to the development of affect dysregulation. In his work with infants, Schore (2002) argues that relational traumas (such as abuse and neglect by a caregiver) have the potential to both actively dysregulate the infant and deny the infant of strategies needed to regulate the distressed state. Further, he contends that relational traumas significantly alter major stress-regulating neurochemicals, specifically those central to the HPA axis. Both Schore (2002) and Gunnar (2000) argue that a variety of early adverse conditions (ranging from parental insensitivity to neglect and abuse) affect the development of the child's stress-related physiological systems (namely the HPA axis), leaving the child vulnerable to the development of psychopathological behaviors and conditions later in life. SIB may be one such pathological behavior. Thus, it seems plausible that childhood traumatic experience may lead to dysregulated affect, leaving the child vulnerable to developing

maladaptive affective coping mechanisms such as SIB. Further, at a biological level, it seems reasonable to view HPA axis function as a marker of affect dysregulation given the association between HPA function and subjective distress (Miller, Chen, & Zhou, 2007) and the commonly found associations between dysregulated HPA axis function and depression and anxiety disorders (e.g., Gold, Goodwin & Chrousos, 1988; Young, Abelson, & Cameron, 2004), both of which entail a breakdown of the capacity to regulate negative emotion. Further, several studies of both humans and animals have found that individual differences in reactivity to stress correspond to behavioral and self-report measures of emotion regulation (Kirschbaum et al., 1995; Nachmias, Gunnar, Mangelsdorf, Paritz, & Buss, 1996; Suomi, 1991). For instance, individuals with exaggerated HPA axis responses to stress (e.g., heightened and prolonged cortisol increases) demonstrate deficient coping strategies and report more negative affect (see Scarpa & Raine, 1997 for a review; Stansbury & Gunnar, 1994).

Even in the presence of childhood trauma, development of dsyregulated affect and later maladaptive coping strategies is not inevitable; identifying and understanding protective factors is critically important. Secure attachment serves as one such protective factor in buffering against the potentially destructive effects of childhood trauma, separation, and loss (e.g., Finkelhor & Brown, 1984; Hayashi & Strickland, 1998; Heinzer, 1995; Hetherington, 1989), and in one study was found to be the most important predictor of long-term outcomes for children exposed to severe stressors (McFarlane, 1988).

Consideration of the role and function of the attachment system helps us to better understand its protective functions and possible links to affect regulation and SIB. A

primary role of attachment relationships in early childhood is the modulation of the child's physiological arousal (van der Kolk, 1996a). In infancy, the young child has few resources for dealing with arousal and stress. Parental (often maternal) sensitivity, including appropriate soothing and stimulation, acts as an external organizer for the infant's biological and behavioral regulation until the child is capable of regulating arousal (Spangler, Schieche, Ilg, Maier, & Ackermann, 1994). Insensitive caregiving can interfere with the development of the infant's ability to self-regulate at both a behavioral and biological level. At a biological level, HPA axis function has been implicated specifically. For instance, Spangler et al. (1994) found that infants of mothers who exhibited highly insensitive behaviors during play interactions showed both behavioral indications of negative emotion and heightened HPA axis activity that were not seen in infants of more sensitive mothers. Presumably, sensitive caregiving behaviors helped infants effectively manage and reduce the arousal prompted by the play task. Infants whose mothers did not provide sensitive care were not able to regulate their arousal and showed behavioral signs of distress that interfered with play. In the absence of adequately sensitive caregiving, young children may not develop the behavioral and biological capacities needed to regulate affect and arousal.

A handful of studies has explored links between attachment style and SIB in adolescents and young adults, although the pattern of associations is not entirely straightforward. Gratz et al. (2002) found that insecure paternal (but not maternal) attachment significantly predicted SIB among women, although attachment did not predict SIB in men. Rulf Fountain (2001) found a link between SIB and poor attachment to mothers, but not fathers. In a study by van der Kolk et al. (1991), insecure attachment

style, inferred from reports of parental separation, environmental chaos, and neglect, was associated with SIB. Further, their findings suggested that while childhood trauma variables contributed to the initiation of SIB behaviors, the lack of secure attachments to caregivers maintained SIB behavior over the course of a four-year follow-up. Given this conclusion, it seems reasonable to suggest that secure attachment plays a protective role, buffering against the potentially dysregulating effects of childhood trauma. This moderating role of secure attachment deserves further study.

In sum, consistent with the above-reviewed work suggesting that a primary function of SIB is emotion regulation, the negative childhood experiences frequently associated with SIB are known to disrupt the growing child's ability to regulate affect at both the behavioral and the biological levels. Left more vulnerable to experiencing overwhelming affect, the individual with a history of trauma is at risk for turning to potentially maladaptive strategies for coping with stress, including SIB. Thus, affect dysregulation may play a mediating role in explaining the relationship between trauma (perhaps specifically childhood trauma) and SIB behavior. While many SIB researchers assume that the interference with affect and arousal modulation occurs at a biological level, most do not specify what biological systems or processes might be involved. Yates (2004), for instance, mentions only possible "neurobiological reorganization" (p. 74) and emphasizes the need for empirical exploration of the potential processes mediating the association between childhood maltreatment and SIB. Given the major role of the HPA axis in regulating the body's response to stress and arousal, HPA axis dysregulation is one plausible mechanism and may mediate the relation between traumatic experience and SIB. To date, very little work has explored possible links between the HPA axis and SIB.

Nonetheless, a good deal of work provides strong evidence for associations between trauma, PTSD, and HPA axis functioning. Before reviewing this work, I present a brief overview of the HPA system.

HPA Axis Functioning

The HPA axis is one of the neurochemical systems that mobilizes the body's energy in order to deal with a stressor (van der Kolk, 1996b). When the HPA axis is activated by a stressor, the hypothalamus releases corticotropin-releasing hormone (CRH), which then stimulates the anterior pituitary to secrete adrenocorticotropin hormone (ACTH). Secretion of ACTH then prompts the adrenal cortex to release cortisol into the bloodstream. Glucocorticoids (cortisol and related steroid hormones) operate via a negative feedback loop to regulate further hormone release, suppressing HPA axis activity and restoring basal cortisol levels. Specifically, elevated levels of cortisol suppress the output of CRH and ACTH by acting on glucocorticoid receptors in the hippocampus, hypothalamus, and pituitary. Through this feedback loop, the glucocorticoids suppress physiological reactions to the stressor that, while adaptive in the short-term, would result in long-term damage to the body if chronically activated (Miller et al., 2007). In humans, cortisol has received the most research attention because it is the major hormonal product of the HPA system, exerts regulatory influences on the system, and is a recognized indicator of HPA axis functioning and reactivity (Stansbury & Gunnar, 1994).

PTSD, Trauma, and the HPA Axis

A large amount of work focuses on HPA axis function in individuals with PTSD. While there are conflicting findings in this area and the connections between trauma and

the HPA system are far from completely understood, this work provides strong evidence linking both trauma symptoms/PTSD and traumatic experience to altered HPA axis function (for extensive reviews, see de Kloet et al., 2006; Yehuda, 1997, 2002).

Given that stress is typically associated with high cortisol levels, researchers initially expected to find elevated basal cortisol levels among individuals with a diagnosis of PTSD (Yehuda, 1997). Contrary to this hypothesis, most studies examining mean urinary cortisol find lower levels in individuals with PTSD relative to both healthy and psychiatric controls. This association has been found in several different PTSD patient populations, including combat veterans (Boscarino, 1996; Mason, Giller, Kosten, Ostroff, & Podd, 1986; Yehuda, Southwick, Nussbaum et al., 1990), Holocaust survivors (Yehuda, Kahana, et al., 1995), and mothers of child cancer survivors (Glover & Poland, 2002). Despite some discrepant findings indicating either no cortisol differences (e.g., Baker et al., 1999) or elevated cortisol in PTSD patients (e.g., Pitman & Orr, 1990), most studies find an association between PTSD and low basal cortisol levels (Yehuda, 2002).

PTSD has also been associated with increased concentrations of glucocorticoid receptors. Several studies have found that veterans with PTSD have a larger number of lymphocyte glucocorticoid receptors relative to both healthy controls and combat veterans without PTSD (e.g., Yehuda, Lowy, Southwick, Shaffer, & Giller, 1991; Yehuda, Resnick, Kahana, & Giller, 1993). Further, the number of glucocorticoid receptors is strongly positively correlated with PTSD symptoms, suggesting that alterations in the HPA axis might be related to PTSD symptomatology (Yehuda, Lowy, et al., 1991). Other work has also found that adults with trauma histories (both those who

have PTSD and those who do not) show increased responsiveness to glucocorticoids (Yehuda, Golier, Yang, & Tischler, 2004).

The dexamethasone suppression test (DST), a measure of HPA negative feedback (i.e., the system's ability to turn off the stress response), also reveals HPA axis alterations in PTSD patients. Dexamethasone is a synthetic glucocorticoid that mimics cortisol's effects. In response to administration of dexamethasone, the HPA axis suppresses cortisol production. An exaggerated cortisol suppression response to low-dose dexamethasone has consistently been found in PTSD related to combat exposure (Yehuda, Southwick, et al., 1993; Yehuda, Boisoneau, Lowy, & Giller, 1995), domestic violence (Griffin, Resick, & Yehuda, 2005), and a variety of combat and civilian traumas (Yehuda, Halligan, Golier, Grossman, & Bierer, 2004).

While no theory of altered HPA axis function in PTSD exists that can easily account for all findings of HPA axis irregularities associated with PTSD, perhaps the most prominent theory is that offered by Yehuda (1997, 2002; see also de Kloet et al., 2006) involving enhanced negative feedback sensitivity. She suggests that the experience of traumatic events may result in chronic increases in the release of CRF (and subsequently cortisol). Over time, these high levels of CRF alter the responsiveness and number of glucocorticoid receptors in the pituitary. These receptors may become hypersensitive, resulting in enhanced binding of cortisol, which then results in an enhanced negative feedback signal. In other words, the HPA axis becomes increasingly efficient in shutting down the stress response, which may be an adaptive response to minimize the potential damage done to body tissues by a chronically activated stress response. The increased responsiveness of the glucocorticoid receptors then results in

attenuated basal cortisol levels. This hypothesized sequence results in what Yehuda refers to as a "sensitized" HPA axis. This sensitized system is marked both by low basal levels of cortisol and by enhanced reactivity to stress. In the context of low background activity (i.e., low basal cortisol), the HPA axis has a greater ability to pick up on and respond to environmental stress cues.

Yehuda argues that the sensitized HPA axis associated with PTSD is consistent with the hypervigilance, increased startle response, and physiological arousal in response to trauma reminders that characterize the disorder. Individuals with PTSD might be expected to respond to an acute stressor with a more marked cortisol stress response (de Kloet et al., 2006; Yehuda, Giller, Southwick, Lowy, & Mason, 1991). The enhanced sensitivity of the negative feedback system should allow for both a faster reaction to stress and a faster physiological recovery from stress to baseline cortisol levels. (See review by de Kloet et al., 2006 for alternative hypothesized mechanisms for altered HPA axis function in PTSD.)

Much of the existing work confounds trauma and trauma-related psychopathology, making it unclear whether the above-described pattern of HPA axis dysregulation is specific to PTSD or to the experience of trauma and chronic life stress. For instance, Stein, Yehuda, Koverola, and Hanna (1997) found that women who had a history of childhood sexual abuse showed both enhanced cortisol suppression in response to the DST challenge and increased density of glucocorticoid receptors relative to non-abused controls. Because the majority (69%) of abuse survivors also had PTSD, the authors could not determine whether HPA axis dysregulation was related to childhood sexual abuse or to abuse plus related psychopathology.

Some work suggests that the above-described pattern of HPA axis dysregulation is specific to PTSD. For instance, Griffin et al. (2005) compared baseline cortisol levels between two groups of female domestic violence survivors, those with PTSD and those without PTSD. Survivors with PTSD had lower baseline cortisol levels than survivors without PTSD. Further, baseline cortisol levels did not differ between survivors without PTSD and nontraumatized controls. Similarly, Yehuda, Kahana, et al. (1995) compared 24-hour urinary cortisol excretion among Holocaust survivors with PTSD, Holocaust survivors without PTSD, and a control group not exposed to the Holocaust. They found that Holocaust survivors with PTSD showed lower mean 24-hour urinary cortisol secretion than either the Holocaust-exposed non-PTSD group or the non-traumatized controls. They concluded that low cortisol levels were associated with clinically significant PTSD symptoms rather than exposure to trauma.

A growing body of literature, however, suggests that the experience of chronic stress and trauma (and not the presence of trauma-related symptoms per se) is associated with altered HPA axis function. In a sample of police officers and firefighters, Witteveen et al. (2010) found that basal cortisol was not associated with the presence of PTSD; rather, lower basal cortisol was found in individuals who had experienced more negative life events (especially events that threatened their life and social/occupational functioning). A meta-analysis of studies exploring basal cortisol in individuals with PTSD found that PTSD was associated with low cortisol only when compared to controls with no trauma history; basal cortisol levels did not differ between people with trauma histories with and without PTSD (Meewisse, Reirsma, de Vries, Gersons, & Olff, 2007). Similarly, Flory et al. (2009) found that the association between low baseline cortisol

levels and a history of childhood physical abuse was not explained by the presence of PTSD. Moreover, in one study of combat veterans (reported in Yehuda, Resnick, et al., 1993), veterans without PTSD had more glucocorticoid receptors than normal controls (but fewer than veterans with PTSD), indicating that HPA axis dysregulation can be seen among trauma-exposed individuals who do not develop PTSD.

Further, animal models suggest that the sensitization of the HPA feedback system may develop as an adaptation to chronic stress experienced early in life (see Yehuda, Giller, et al., 1991). Studies of rats show that early exposure to stress results in permanently increased glucocorticoid receptor density, which in turn allows for stronger glucocorticoid negative feedback when the HPA system is activated in response to a stressor. Heightened negative feedback ultimately allows for faster recovery from stress, protecting the animal from the harmful consequences of chronically elevated levels of glucocorticoids. Potentially consistent with this model, Yehuda suggests that sensitization of the HPA system does not appear to be a consequence of PTSD, but rather is a pre-existing risk factor for the development of the condition (Yehuda, 2002). Further, she suggests that low cortisol levels may occur in individuals who experienced adversity early in life; childhood emotional abuse and parent psychopathology were implicated in one study (Yehuda, Halligan, & Grossman, 2001). Alteration of HPA axis function may interact with later trauma, contributing to the development of PTSD. Although this suggestion is largely speculative, it lends support to the notion that early and/or chronic stress can lead to altered HPA axis activity.

Given that the literature is unclear as to whether altered HPA axis function is associated with the experience of trauma per se or whether altered HPA axis function is

uniquely associated with PTSD (or more generally the presence of trauma-related symptoms), the present study examined links between HPA axis reactivity, SIB and measures of both trauma experience and trauma-related symptoms.

HPA Axis Functioning and SIB in Humans

While several studies have explored the links between trauma and HPA axis activity, to my knowledge, only two unpublished studies have explored possible connections between HPA axis function and SIB in the non-mentally retarded population. Rulf Fountain (2001) did not find differences in baseline cortisol between college students who did and did not engage in SIB. While she attempted to assess hypothesized differences in HPA axis reactivity to a stressor task, reactivity was not successfully measured due to methodological issues. In preliminary analyses conducted with a sub-sample of participants used in the current study, McArdle (2004) found associations between SIB and HPA axis reactivity to an interpersonally stressful task, some of which were consistent with the pattern of hypersensitive HPA axis reactivity seen in people with trauma histories and PTSD. For instance, the more recently a woman engaged in SIB, the earlier she reached peak cortisol levels. Further, women who had high levels of trauma symptoms reached peak cortisol earlier the higher their SIB severity/frequency score.

To the best of my knowledge, these two unpublished studies are the only studies examining SIB and HPA axis functioning in people without mental retardation (MR). Three studies have explored this association in the MR population, with contradictory results. While one study found no difference in cortisol levels between mentally retarded adults with and without SIB (Sandman, Barron, Chicz-DeMet, & DeMet, 1990), another found a trend toward lower cortisol levels in mentally retarded adults who engaged in

SIB (Verhoeven et al., 1999), and the third found a trend toward higher cortisol levels (Symons, Sutton, Walker, & Bodfish, 2003). The relevance of this work to the non-MR population is unclear, as the intent, functions, and associated developmental and psychosocial correlates of SIB in these groups are believed to differ (Favazza & Rosenthal, 1993). At this time, it is unknown whether the same biological systems are involved in SIB in both populations. Nonetheless, the association between HPA axis function and SIB has become a recent area of interest in both the MR and non-MR populations.

HPA Axis Functioning and SIB in Primates

Support for a relation between SIB and altered HPA axis function also comes from studies finding HPA axis differences in primates who engage in SIB (typically defined as self-biting) (although, see Davenport, Lutz, Tiefenbacher, Novak, & Meyer, 2008, who found no differences between monkeys who engaged in SIB and controls in HPA axis function following relocation stress). In one study comparing male rhesus monkeys with and without SIB histories, monkeys with a history of SIB had lower mean plasma cortisol levels than control monkeys (Tiefenbacher, Novak, Jorgensen, & Meyer, 2000). Further, the frequency of self-biting was negatively correlated with morning cortisol levels. The authors originally interpreted these data as suggesting that self-injuring monkeys had lower levels of circulating cortisol, reflective of a persistent dysregulation of the HPA axis. Later work by this group, however, suggested that, in fact, the monkeys with SIB histories did not have lower basal levels of cortisol, but were showing lower levels of cortisol following the stress of the blood draw procedure (Tiefenbacher et al., 2004). In this later work, Tiefenbacher et al. collected urinary, as

opposed to blood, cortisol samples to eliminate the stress induced by a blood draw. Mean cortisol levels did not differ between monkeys with and without SIB histories and basal cortisol was not related to the frequency or recency of SIB. This study also assessed the sensitivity of the HPA negative feedback loop using both the DST and a combined dexamethasone/ACTH challenge. High frequency self-biters showed attenuated cortisol suppression in response to the DST relative to low frequency biters and a trend towards an attenuated cortisol response to ACTH stimulation. The authors suggested that these findings might be indicative of desensitization of glucocorticoid negative feedback, which stands in contrast to the work linking trauma and PTSD to enhanced negative feedback in humans. At the same time, in a more recent study, this group reported some support for an association between self-wounding in monkeys and enhanced HPA negative feedback (Chen et al., 2010). While there are some conflicting findings, both the primate work and the human work suggest that SIB is associated with alterations in the HPA axis' reactivity to stress.

The Present Study

Work on the functions of SIB emphasizes its tension-reducing and affect-regulating properties and suggests that individuals engaging in SIB have difficulty regulating their emotions in response to stress. Further, several of the risk factors associated with SIB (e.g., childhood abuse, neglect, and loss) have been shown to impair an individual's ability to regulate affect and arousal at both a behavioral and biological level. It seems reasonable to suggest that, at the biological level, this affect dysregulation may manifest as dysregulation of the HPA axis, one of the body's major stress response systems. If individuals have difficulty regulating their emotional response to stress, they

may then be vulnerable to developing maladaptive affective coping strategies, such as SIB. In the current study, I test this mediating model of SIB which is depicted in Figure 1.

This model was evaluated in a non-clinical sample of 18-21 year old men and women. This population is of particular interest for several reasons. First, SIB has been relatively understudied in the general population, despite emerging evidence that SIB is not restricted to clinical populations and is surprisingly prevalent among adolescents and young adults in the general population (e.g., Gratz et al., 2002; Whitlock et al., 2006). Moreover, childhood trauma and maltreatment are not uncommon among the general population; Scher, Forde, McQuaid, and Stein (2004), for instance, report prevalence rates of 30% for women and over 40% for men in an adult community sample. Further, studying behaviors in a non-patient sample reduces the impact of many of the confounds prevalent in work with patient samples, such as the use of psychotropic medications and psychological treatments.

Hypotheses

The present study evaluated three main hypotheses:

Hypothesis 1

Consistent with a large body of work linking various childhood traumatic stressors to later SIB, I hypothesized that both a history of exposure to traumatic events and the presence of trauma-related symptoms would positively predict SIB.

Hypothesis 2

Based on work showing that traumatic experience can lead to dysregulation of the HPA axis and the predominantly theoretical work linking affect dsyregulation (both

behavioral and biological) to SIB, I predicted that the relations between trauma experience and trauma symptoms and SIB would be at least partially mediated by HPA axis reactivity, as measured by participants' HPA axis response to an interpersonal stress task.

The interpersonal stress task involved dating couples attempting to resolve a source of recent disagreement during a 15-minute videotaped discussion task. Participants' responses to an interpersonal stressor such as this seem especially relevant to the study of SIB, given the good deal of work suggesting that acts of SIB are often precipitated by actual or perceived interpersonal problems or conflicts, including arguments with significant others, separation, and perceived rejection (e.g., Herpertz, 1995; Ghaziuddin, Tsai, Naylor, & Ghaziuddin, 1992; Suyemoto, 1998). Thus, a conflict discussion with a romantic partner seems to be the type of event that might be perceived as especially stressful for an individual engaging in SIB.

The precise nature of the mediating role of HPA axis reactivity is unclear and I consider it to be an exploratory question. In the current study, I examined multiple aspects of HPA axis functioning, including cortisol level during the discussion task, the rate of change during the discussion, and curvature (the shape of the entire trajectory of cortisol change from entry to the lab, anticipation of the stressor, during the stressor, and through recovery). Yehuda's model (1997, 2002) of the relation between PTSD and HPA axis function would suggest that individuals with trauma histories might have a sensitized HPA system that is maximally responsive to stress cues from the environment. Such individuals might be expected to respond to an acute stressor with a steeper rise and fall in cortisol stress response, with the enhanced sensitivity of the negative feedback

system allowing for a faster physiological recovery. This pattern of HPA axis reactivity may occur within the context of overall lower levels of cortisol, given the consistent links in past literature between PTSD/trauma experience and lower basal cortisol.

Hypothesis 3

Based on the work suggesting that secure attachment bonds can protect individuals from the destructive long-term effects of trauma, I expected that adult attachment style would moderate the relation between HPA axis reactivity and both trauma experience and trauma symptoms. I expected that secure attachment within current romantic relationships would serve as a protective buffer, such that individuals with a secure attachment style would not show the expected association between trauma and HPA axis dysregulation.

Most of the work on the protective functions of secure attachment relationships has focused on attachment to caregivers in infancy and early childhood. In the present study, I explored whether secure attachment in current adult romantic relationships served a similar function. Adult attachment theory would suggest that, through their provision of safe haven caregiving, secure adult attachment relationships will promote reduced stress and anxiety, improved coping, and increased feelings of safety and security, contributing to psychological and physical well-being (Feeney & Collins, 2004), a claim that has some empirical support (e.g., Feeney & Kirkpatrick, 1996). Further, Diamond and Hicks (2005) posit that secure adult attachment may serve as a protective buffer between negative life events and altered HPA axis function. They suggest that by facilitating positive emotions and buffering against negative appraisals of life stressors,

secure and supportive adult attachment relationships may attenuate the deleterious effects of negative and stressful events on HPA axis function.

Exploratory Questions

Gender

There are several reasons to explore whether gender moderates the above-presented hypotheses. First, gender differences in SIB have been understudied because until recently, SIB was viewed as a largely female phenomenon (Suyemoto, 1998) and the majority of studies of SIB used female samples (e.g., Favazza & Conterio, 1989). We now know, however, that a subset of men does engage in SIB, with some work even suggesting that prevalence rates may be similar for men and women (e.g., Gratz et al., 2002; Muehlenkamp & Gutierrez, 2004). Despite comparable prevalence rates, there is evidence of gender differences in the etiological factors involved in SIB. For instance, Gratz and colleagues found that while childhood sexual abuse, emotional neglect, and insecure attachment predicted SIB in women, physical separation from fathers predicted SIB in men (Gratz, 2006; Gratz et al., 2002; Gratz & Chapman, 2007). Further, preliminary work with a sub-sample of the data to be used in the current study suggested that the functions of SIB may differ by gender, with women providing more affect regulation reasons for engaging in SIB behaviors (McArdle, 2004). Finally, given findings that women tend to have stronger HPA axis responses to interpersonal stress than men (e.g., Kiecolt-Glaser et al., 1996), gender needs to be taken into account in these analyses.

Trauma Experience and Trauma Symptoms

As mentioned previously, the empirical work linking altered HPA axis function to trauma has not clarified whether it is the experience of trauma per se that relates to altered HPA axis function or the development of trauma-related symptoms (i.e., PTSD symptoms) that relates to altered HPA axis function. The current study explores the above-described questions through evaluating two series of models, one in which a measure of trauma experience was related to HPA axis reactivity and SIB and the other in which a measure of trauma-related symptoms was related to HPA axis reactivity and SIB. While no direct statistical comparisons are made between these two series of models, comment is made about whether similar relations with HPA axis reactivity and SIB are observed for trauma experience and trauma symptoms.

CHAPTER 2

METHOD

Participants

Participants for this study were 356 older adolescents (ages 18-21, mean age = 19.23 years, SD = .788) who were part of a larger study exploring a biopsychosocial model of adolescent depression. For reasons related to the goals of the larger study, all participants were in opposite-sex dating relationships and participated with their partners. The length of the relationship ranged between 2 months to greater than 3 years, with a mean duration of 15.20 months and a standard deviation of 11.00. The sample was representative of older adolescents in the western Massachusetts community from which participants were recruited, and participants reported their ethnic identities as non-Hispanic European American (87.5%), Latino/Latina (3.9%), African American (1.4%), Asian American/Pacific Islander (4.7%), Native American (.3%), or other (2.2%). Participants were recruited from the western Massachusetts area through flyers, posters, and presentations in University of Massachusetts undergraduate courses. Each participant received $60, and those who were University of Massachusetts undergraduates also received extra credit points for their participation.

Procedure

The data for this study were taken from the initial session of a larger longitudinal study of adolescent dating relationships. During an initial telephone screening interview, participants were invited to the lab with their romantic partner to participate in a study of their behaviors and physiological reactions in response to a conflict negotiation task with their partner. They were instructed to abstain from drinking alcohol, using illegal drugs,

or visiting the dentist within a 24 hour period prior to the study. They were also asked not to exercise, eat, drink (with the exception of water), smoke cigarettes, or brush their teeth within two hours of the study to reduce any contaminants in their saliva that might affect the accuracy of the cortisol measurements. Participants rinsed their mouths thoroughly with water 10 minutes before giving the first saliva sample to minimize contaminants. Because cortisol levels follow a circadian rhythm, participants were invited into the lab at 4:00 pm, the time of day when cortisol levels are most stable (Kirschbaum & Helhammer, 1989).

Upon arrival at the lab, participants completed informed consent forms and an Admission Questionnaire containing questions about variables that could potentially affect HPA axis functioning, such as the number of hours slept the night before, prescription medication or vitamins taken, phase of the menstrual cycle, and the possibility of pregnancy. The questionnaire also inquired about adherence to the pre-study instructions (e.g., drinking alcohol, smoking cigarettes, etc.). Because acute illness could affect HPA axis functioning, participants were also given an oral thermometer and reported their temperature. If participants had an elevated temperature or felt ill, or if they had violated any of the pre-study instructions, the study appointment was rescheduled.

Following completion of the Admission Questionnaire, the first salivary cortisol sample (the entry sample) was collected using a passive drool procedure recommended by Salimetrics, LLC (State College, Pennsylvania). Participants passively drooled down a straw into a small plastic vial with their heads tilted forward until the required amount of saliva had been collected. Participants were then read a detailed description of the

conflict discussion task and each member of the couple identified recent sources of disagreement. Fifteen minutes later, the second saliva sample was collected as a measure of pre-discussion anticipatory anxiety.

A research assistant then randomly selected one of the topics of disagreement to serve as the subject of the conflict discussion task. The couple was brought into a room equipped with three wall-mounted digital video cameras and a couch. The couples were given the selected discussion topic and were instructed to spend 15 minutes discussing the issue in an attempt to resolve the conflict. The participants were aware that their discussion would be videotaped.

Following completion of the conflict discussion task, couples were taken to another room to fill out a series of questionnaires. Five additional saliva samples were collected at regular intervals as participants completed the questionnaires; these samples provided measures of cortisol reactivity during the conflict task and recovery from the task.

Measures

Saliva Collection Procedures

To measure participants' HPA axis reactivity before, during, and after the conflict discussion task, seven salivary cortisol samples were collected over the course of 1 hour and 35 minutes. After cortisol is secreted from the adrenal gland, it takes 15 to 20 minutes to enter the saliva; thus, each saliva sample actually measures participants' cortisol reactions from 15 to 20 minutes earlier (Stansbury & Gunnar, 1994). For example, the first saliva sample was taken 10 minutes into the data collection sample; thus, it actually represents participants' cortisol levels 5-10 minutes prior to entering the

lab. The second sample was collected 15 minutes after participants were read a vivid description of the conflict discussion task. Thus, the second sample measures participants' cortisol levels in response to the anticipation of this interpersonally stressful event. The third saliva sample was collected 10 minutes following completion of the task; this sample measures participants' cortisol levels during the conflict discussion task. The remaining five saliva samples were taken 20, 30, 45, and 60 minutes following the conflict discussion task and reflect recovery from the task. In sum, collection of these seven saliva samples allows for assessment of the trajectories of the participants' stress responses from 5-10 minutes before entering the lab, through their anticipation of the conflict discussion, during the conflict discussion, and throughout a recovery period of approximately 40 minutes following the discussion.

Immediately after each saliva sample was collected, the vial was sealed and placed in frozen storage (-20° C) until shipped on dry ice to Salimetrics, LLC for analysis of cortisol levels. All samples were divided into two vials and separately assayed for salivary cortisol with the use of a highly sensitive enzyme immunoassay (Salimetrics, State College, Pennsylvania). Thus, each cortisol sample had two values, resulting in a total of 14 values for the seven samples. The test used 25 μL of saliva (for singlet determinations), and it had a lower limit of sensitivity of .003 μg/dl, a range of sensitivity from .003 to 1.2 μg/dl, and average intra- and interassay coefficients of 4.13% and 8.89% variation, respectively. Method accuracy, determined by spike recovery, was 105%, and linearity, determined by serial dilution, was 95%. Since blood contamination can falsely elevate salivary analyte levels, samples were tested for blood contamination by Salimetrics before being assayed for cortisol.

Admission Questionnaire

This self-report questionnaire was specifically designed for the larger project to collect information relevant to HPA axis function. Participants were asked about current health status, medications, recent use of alcohol or illegal drugs, recent food intake, exercise, tooth brushing, dental work, and amount of sleep the evening prior to the study. Women were also asked about oral contraceptive use, pregnancy, and phase of menstrual cycle. See Appendix A for a copy of this measure.

Self-Injurious Behavior Questionnaire (SIB-Q)

The frequency and recency of SIB behaviors were measured using a revised version of the SIB-Q (McArdle, 2004; Rulf Fountain, 2001). This questionnaire asked the participant how many times and how recently he or she engaged in eight specific types of self-injury that would be classified as Type III SIB, specifically: bruising, hitting, hair-pulling, scratching, biting, poisoning, burning, and cutting. Participants reported the frequency and recency of these behaviors using a 7-point Likert scale. Participants were also asked to report their reasons for engaging in these behaviors. See Appendix B for a copy of this measure.

Based on the methodology of Rulf-Fountain (2001) and McArdle (2004), a weighted-continuous measure of SIB was calculated that took into consideration both the severity of the form of the behavior and the frequency of the behavior. SIB behaviors were classified as Mild, Moderate, or Severe and were given severity weightings of 1, 2, and 3, respectively. The classification of SIB behaviors as mild, moderate, or severe followed those suggested by Rulf Fountain (2001) and McArdle (2004). Mild SIB behaviors included self-bruising, self-hitting, and hair-pulling. Moderate SIB behaviors

included self-scratching and self-biting. Severe SIB behaviors were self-poisoning, self-burning, and self-cutting. For each category of self-injury, the severity weighting was multiplied by the frequency of the behavior. The product of severity weight x frequency was then summed across all categories of SIB endorsed by the participant (i.e. $frequency_1*severity_1 + frequency_2*severity_2 + frequency_3*severity_3\ldots$). For example, if a participant reported having cut herself "between 6-10 times" (severity of 3 and frequency of 3) and having bitten herself "between 2-5 times" (severity of 2 and frequency of 2), the weighted score would be (3*3) + (2*2) = 13.

Adjustments were made to some participants' SIB scores due to inconsistencies in their reports. For instance, some participants, when asked about the recency of a particular type of SIB, indicated that they had engaged in that SIB behavior at some point, but reported a frequency of "never." In those cases (affecting 11 participants), the frequency score was recoded as "one time in my life." Similarly, on the basis of their recency response, some participants indicated that they had engaged in a particular type of SIB in the past, however, they did not report the frequency. In those cases (affecting 8 participants), the frequency was assumed to be "one time in my life." Finally, in some cases, participants endorsed a category of SIB, but on a later item asking them about specific situations or feelings that prompted SIB (item #14), provided a response suggesting that they were reporting events that did not really fit into the category they had endorsed. For example, one participant reported engaging in behavior that produced bruising and later explained that he had punched a wall when his sports team lost. In cases like these, the participant's response was changed to indicate no SIB in the relevant category. This affected 13 participants.

Some participants had missing data on the SIB-Q. Participants who had missing data on four or more categories of SIB (5 participants) were dropped from the study. For participants with some missing data on three or fewer categories of SIB (20 participants), their SIB composite score was calculated based on the data that they did report.

Once the SIB composite scores were calculated, the descriptive statistics were reviewed. As expected, the scores were strongly right-skewed and positively kurtotic. Because of concern about the non-normal distribution of the data, the square root of the SIB composite score was used in all further analyses. The square root transformed data were closer to normally distributed than the untransformed data.

Trauma Experiences Questionnaire (TEQ)

The TEQ (Vrana & Lauterbach, 1994) was used to assess participants' exposure to traumatic events throughout the lifepsan. Participants were asked whether they had experienced a wide range of types of traumatic events (e.g., serious accidents, natural disasters, violent crime, childhood physical or sexual abuse, unwanted sexual experiences, etc.). If participants indicated that they had experienced a given type of trauma, they then reported the number of events experienced and their age at the time, as well as a series of four questions that assessed the intensity of the traumatic event: degree of injury incurred, perceived life threat at the time, and perception of how traumatic the event was at the time and currently. Responses to the TEQ can be analyzed individually according to trauma type or a total traumatic exposure score can be calculated by summing the number of events reported. A trauma intensity score can be calculated by summing the four questions regarding trauma severity. In past research, the TEQ has demonstrated acceptable test-retest reliability (r = .91 for the number of events reported;

and *r* ranging from .72 to 1.0 for the occurrence of specific events; Lauterbach & Vrana, 1992 cited in Vrana & Lauterbach, 1994). See Appendix C for a copy of this measure.

In the current study, a measure of traumatic experience was calculated that took into account both the frequency and severity of the traumatic events experienced. For each type of traumatic event, the reported frequency of trauma (ranging on a scale from 0 to 3) was multiplied by the average of the four questions assessing severity.[2] The product of frequency x intensity was then summed across all types of trauma reported by the participant to generate a total traumatic experience score. Summing across trauma types is justified based on the work on multiple risk factors suggesting that it is not the presence of any particular risk factor, but rather the number of risk factors in a child's life that predicts later psychopathology (e.g., Rutter, 1979; Seifer, Sameroff, Baldwin, & Baldwin, 1992).

A few additional notes are needed to explain the calculation of the TEQ composite score. One trauma category was omitted from the TEQ composite score: having experienced a natural disaster. This category was omitted because a very high number of participants endorsed this category and, when given the opportunity to explain the event, reported that they had experienced one or several hurricanes, although very few appeared to have been directly affected by these events. Thus, it appeared that people were over-endorsing this category, so it was dropped for the purposes of analysis. Additionally, for most types of trauma, participants reported the number of traumatic experiences of a given type that they had experienced (one, two, or three or more). The authors of this measure conceptualized frequency somewhat differently for three categories of trauma: childhood physical/sexual abuse, adult unwanted sexual experience,

and adult relationship abuse. These categories were conceptualized to be more chronic types of abuse that likely had occurred on multiple occasions. Instead of asking participants to report the number of events in these categories, the measure asked participants to report the ages at onset and offset of abuse. When calculating the TEQ composite score, the participants were given a frequency score of three, assuming that these types of abuse occurred three or more times. Finally, at the end of the TEQ, participants who had not endorsed any trauma on the measure were asked to report on the most traumatic event that had happened to them. These responses were included in the TEQ composite score.

The TEQ asked participants whether they endorsed the same traumatic event in more than one category. When participants indicated that they had reported the same event more than once, adjustments were made to the TEQ composite score. For instance, if a participant reported witnessing a car accident in two different categories, the average intensity was taken from the eight items assessing intensity (the four intensity items in the first category and the four intensity items in the second category). This average intensity was then multiplied by the number of events (in this case one). These adjustments were made to prevent against inflation of trauma scores.

Once the TEQ composite scores were calculated, the descriptive statistics were reviewed. The scores were strongly right-skewed and positively kurtotic. Because of concern about the non-normal distribution of the data, the square root of the TEQ composite score was used in all further analyses. The square root transformed data were closer to normally distributed than the untransformed data.

Trauma Symptom Checklist-40 (TSC-40)

The TSC-40 (Briere & Runtz, 1989) is a 40-item, self-report measure that assesses the prevalence of symptoms that are likely to have arisen from adult or childhood trauma experiences. The checklist consists of 6 subscales including: Anxiety, Depression, Dissociation, Sexual Abuse Trauma Index, Sexual Problems, and Sleep Disturbance. A total scale score can be calculated and has been found to be more reliable than any of the subscales (alpha between .89 and .91) (Briere & Runtz, 1989). See Appendix D for a copy of this measure.

Some participants had missing data on this measure. Fourteen participants had one missing item on a subscale; in these cases, the participants' mean score was inserted for the missing item.

Confirmatory factor analytic work conducted as part of a previous project with this dataset yielded a "trauma factor" that distinguished trauma-related symptoms from other symptoms of depression and anxiety (Powers, 2009). This trauma factor was derived from scores on the following TSC-40 subscales: dissociation, anxiety, depression, and sleep problems. Participants' trauma factor scores were used in the current analyses as the measure of participants' trauma-related symptomatology and have the advantage of factoring out symptoms of depression and anxiety. Further, given that factor scores are based on the correlations between the items that make up the factor (i.e., they sum the items using a series of weights to reflect the comparative contribution of each item to the composite), the factor score is a more reliable composite score than the raw total score.

Experiences in Close Relationships Scale (ECR)

The ECR is a 36-item self-report measure used to assess attachment in romantic relationships (Brennan, Clark, & Shaver, 1998). The scale measures two dimensions of attachment: Avoidance and Anxiety. The Avoidance subscale assesses avoidance of intimacy and dependence on one's romantic partner. The Anxiety subscale measures individuals' anxiety about rejection and abandonment. Items are rated on a 7-point Likert scale, ranging from 1 (Disagree strongly) to 7 (Agree strongly). See Appendix E for a copy of this measure.

Thirty-four participants had between one and three missing items on a subscale; in these cases, the participants' mean score for the subscale was inserted for the missing items. In the current sample, subscales had acceptable reliability (Cronbach's alpha: Avoidance .86, Anxiety .90).

Analytic Strategy

The following description explains the series of models run to explore each hypothesis. As I am interested in the relations of trauma experience to SIB and cortisol as well as the relations of trauma symptoms to SIB and cortisol, each model described below was run two ways. Each model was run with TEQ (the traumatic experience measure) as the main independent variable and then each model was run separately with trauma factor score (the measure of trauma symptoms) as the main independent variable. For simplicity's sake, the following explanation of each model refers to "trauma variable" as the main independent variable.

Mediation Analyses

The hypothesized mediation model was evaluated with a series of path analyses using LISREL Version 8.80 (Jöreskog & Sörbom, 2006). First, a path model was fit to evaluate whether the trauma variable predicted SIB as hypothesized. As the sample consisted of men and women who were in dating relationships, the data were interdependent. This interdependence between dating partners was modeled statistically following the recommendations made by Kenny, Kashy, and Cook (2006) for path analysis with dyadic data. Specifically, this model was specified separately for men and women within the same model; to capture the interdependence, paths were specified between the men's trauma variable and the women's trauma variable and the residual variances of men's SIB and women's SIB were correlated. See Figure 2 for a graphic depiction of this model. All of the path analysis models described below were run in this manner in order to statistically account for the interdependence of the data.

Assuming the expected relation between the trauma variable and SIB was found, a second path model was fit to evaluate whether HPA axis reactivity mediated this association (see Figure 3)[3]. If mediation was present, the coefficients for the indirect (or mediator) effects would be statistically significant. An advantage of a path analysis is the ability to simultaneously define multiple mediators, and the presentation of a statistic to discern whether the indirect effect is significant. The indirect effects are estimated as the product of the direct effects that comprise them. For instance, the indirect effect of the trauma variable on SIB through cortisol level is estimated as the product (a x d) of the coefficient for the path from trauma to cortisol level (a) and the coefficient for the path from cortisol level to SIB (d). In the mediation model, three indirect effects were tested:

the indirect effects of the trauma variable on SIB through cortisol level during the conflict discussion, rate of change during the discussion, and curvature (shape of the entire trajectory of cortisol change from entry to the lab, anticipation of the stressor, during the stressor, and during recovery). In this mediation model, the interdependence of the data was modeled statistically as explained above. It should be noted that Figure 3 is a conceptual model depicting how mediation was assessed and does not depict the full model that was tested in which the paths for men and women were estimated separately and the interdependence of the dyadic data was modeled.

Moderating Effects of Adult Attachment Security

The hypothesized moderating effects of adult attachment security on the relations between the trauma variable and the three components of the cortisol trajectory were tested by creating interaction terms between the trauma variable and both adult attachment variables (attachment avoidance and anxiety). In this model, the following additions were made to the above-described mediation model: the three cortisol variables were predicted from the trauma variable, attachment anxiety, attachment avoidance, and each attachment variable's interaction with the trauma variable. Finding that either or both trauma x attachment interactions significantly predicted components of the cortisol trajectory would provide support for the hypothesis that adult attachment security moderated relations between trauma and HPA axis reactivity. See Figure 4 for a conceptual diagram displaying how moderation by attachment was assessed. It should be noted that Figure 4 does not depict the full model that was tested in which the paths for men and women were estimated separately and the interdependence of the dyadic data was modeled.

Moderating Effects of Gender

In order to assess whether gender moderated any of the above-proposed relations, the following basic approach was taken: two sets of models were run and compared, one in which the paths described above were estimated freely and one in which the paths of interest were constrained to be equal between men and women. As a simple example using the mediation model, the paths from the men's trauma variable to men's SIB and from the women's trauma variable to women's SIB were estimated freely. In order to test whether the relation between the trauma variable and SIB differed between men and women, a second model was run in which these two paths were constrained to be equal for men and women. Then, a chi-square comparison test was run in order to determine whether estimating the paths freely improved model fit. If the chi-square test reached significance, this would suggest that the freely estimated model was a better fit to the data indicating that gender moderated that relation. If, on the other hand, the chi-square test did not reach statistical significance, this would suggest that the freely estimated model was not a better fit to the data and that the constrained model should be retained, providing evidence that gender did not moderate the relation. In the results section, the specific details of which paths were constrained will be explained, however, the above-described strategy presents the basic approach to how gender differences were assessed.

Estimation of HPA Axis Reactivity

In order to plot the temporal trajectories of participants' HPA stress response, I used growth modeling using Hierarchical Linear Modeling (HLM) Version 6 (Raudenbush, Bryk, Cheong, Congdon, & du Toit, 2004). HLM addresses the challenges inherent in the analysis of dependent data from couples and from repeated measurements

of cortisol levels in response to the conflict discussion task. In this dataset, women's cortisol responses and men's cortisol responses were nested within the couple. Information about the association between the scores in the couple and among repeated measures was used to compute a more precise standard error in testing regression coefficients. A further advantage of this technique is that it adjusts the cortisol responses for measurement error, thereby providing true cortisol responses for each person and enabling a more precise estimation of effects.

The growth models generated in HLM yielded three variables that capture the curvilinear trajectory[4] of the cortisol stress response: the intercept, the linear component, and the quadratic component. The data were centered so that the intercept represented predicted cortisol levels at a timepoint in the middle of the conflict discussion. Hence, the intercept will be referred to as "cortisol level." The linear component is the linear rate of change in cortisol level at any given timepoint from entry through recovery. In other words, the linear component indicates how fast the cortisol level is changing at a particular timepoint and will now be referred to as "rate of change during the discussion." The quadratic component captures the shape or curvature of the cortisol trajectory from entry into the study, the discussion task, and through the recovery period (and will now be referred to as "curvature").

Several variables that were not of primary interest, but that potentially affected HPA axis functioning, were assessed in the Admission Questionnaire (e.g., allergy medications, oral contraceptives, psychotropic medications, antibiotics) or by laboratory assay (e.g., blood contamination of the saliva). Those variables that were found to be

significantly associated with cortisol reactivity in this sample were statistically controlled when the cortisol trajectories were modeled.

HLM estimates the cortisol trajectories separately for men and women, generating an equation estimating the women's trajectory and another equation estimating the men's trajectory. The coefficients for the level, rate of change, and curvature for each individual participant were obtained from the residual file. These coefficients were used as the three measures of HPA axis reactivity in the path analyses to test whether they mediated the hypothesized relation between trauma and SIB.

CHAPTER 3

RESULTS

Descriptive Characteristics of the Sample

Before addressing the primary research questions, I examined descriptive statistics for the independent and dependent variables. See Table 1 for descriptive statistics by gender for the outcome and predictor variables. Paired samples t-tests compared the means on these variables between men and women. As can be seen in Table 1, men had higher scores on attachment avoidance and men had a faster average rate of cortisol change during the discussion (i.e., the average coefficient for the cortisol linear term was more negative for men than women). There were no gender differences found for any of the other variables (the SIB composite, TEQ composite, trauma factor score, cortisol level, cortisol curvature, or attachment anxiety). See Table 2 for a table of the correlations between these variables.

SIB-Q Descriptive Statistics

Out of the total analysis sample, 45.2% of participants endorsed at least one act of SIB. Somewhat more men than women reported SIB, with 52.2% of the men and 42.24% of the women reporting SIB. The difference in percentages of men and women engaging in SIB reached significance at the .10 level ($z = 1.37$, $p = .09$). As described in detail above, participants reported how frequently and how recently they engaged in eight types of self-injurious behavior. Descriptive information on the frequency and recency of each category of SIB can be found in Table 3. As seen in this table, the most commonly reported SIB category was hitting (21% of the sample), followed by scratching (18%),

bruising (18%), biting (17%), hair pulling (11%), burning (8%), cutting (5%), and eating toxic/dangerous substances (3%).

TEQ Descriptive Statistics

Out of the total analysis sample, 67.1% of participants reported having experienced at least one traumatic event. The percentages of men and women who reported at least one traumatic event were quite similar, with 68.6% of the men and 65.7% of the women in the sample reporting at least one event. The difference between the men's and women's percentages was not statistically significant ($z = .33$, $p = .37$). Participants who did not endorse any traumatic events when asked about specific types of trauma were asked to report the most traumatic event that had happened to them (see items 347-352 on the TEQ). When these responses were taken into account, 90.3% of participants reported at least one traumatic event. Again, the percentage of men (90.9%) and the percentage of women (89.7%) reporting at least one traumatic event did not differ ($z = .12$, $p = .45$).

As described in detail above, the TEQ inquires about several different categories of trauma. For each category, the participants reported whether or not they experienced that type of trauma, how many times such a trauma occurred, and at what age. They also answered four questions intended to measure the intensity of the traumatic experience. Table 4 presents descriptive information on the frequency, average intensity, and average TEQ composite for each of the trauma categories. Descriptive information for the abuse categories (childhood abuse, adult unwanted sexual experience, and adult relationship abuse) was reported separately in Table 5 because the questions asked about these categories were somewhat different. Specifically, instead of reporting the number of

times these forms of abuse occurred, participants reported the ages when the abuse began and ended. Thus, Table 5 presents descriptive information about the frequencies for age of onset and offset, as well as the average intensity and average TEQ composite for each of these three categories.

TSC-40 Descriptive Statistics

In order to provide information about the prevalence of trauma-related symptomatology in the sample, descriptive statistics for the total score and six subscales of the TSC-40 are presented in Table 6. For the path analyses, recall that factor scores were used as the measure of trauma-related symptomatology. The trauma factor scores were derived from scores on the following TSC-40 subscales: dissociation, anxiety, depression, and sleep problems.

HLM Analyses

Multilevel modeling was used to plot the temporal trajectory of cortisol reactivity. Because cortisol values were positively skewed, natural log transformed values were used as the outcomes in developing the growth model. The multilevel modeling approach used specified two linked models (i.e., the Level 1 and Level 2 models).

In the Level 1 model, partners' cortisol trajectories were modeled with intercept, linear slope, and quadratic terms to reflect the curvilinear pattern of rising and falling cortisol associated with reactivity to and recovery from the stressor. This quadratic model was selected based on both theoretical considerations – HPA axis response to a discrete stressor should involve cortisol levels rising to a peak and then declining back to baseline – and statistical evidence from prior analyses with this dataset (Powers et al., 2006). The Level 1 model was represented by the following equation:

$$Y_{ij} = \beta_{1j}(\text{Female}) + \beta_{2j}(\text{Female Linear}) + \beta_{3j}(\text{Female Quadratic}) +$$

$$\beta_{4j}(\text{Male}) + \beta_{5j}(\text{Male Linear}) + \beta_{6j}\ (\text{Male Quadratic}) + r$$

Y_{ij} is the log cortisol level for individual *i* in couple *j*, with $j = 1, \ldots, n$ couples. The variables "female" and "male" were dummy variables coded 0 or 1 to indicate to which partner the cortisol level belongs. Consequently, for women, β_{1j} is the model intercept, the predicted value of cortisol when the origin of time is zero. Time was rescaled in this model so that the intercept would represent the conflict discussion timepoint. Thus, β_{1j} represents the predicted value of women's cortisol during the discussion. β_{2j} is the women's rate of change during the discussion, and β_{3j} is the curvature of the women's cortisol trajectory over the entire period of assessment (i.e., the quadratic component). β_{4j}, β_{5j}, and β_{6j} represent the same parameters for the men's trajectory. Finally, *r* is the error, which is assumed to have a mean of zero and a constant variance s^2.

The Level 2 model predicted men's and women's intercept, linear term, and quadratic term from control variables found to be significantly related to cortisol in prior analyses done with this sample (Powers, Laurent, & Granger, 2010). The following control variables were significantly related to cortisol and were included in the growth models: for women, being sick predicted the rate of change (i.e., the linear term); for men, taking allergy medication predicted the rate of change, and the number of hours slept the night before saliva collection predicted cortisol level (i.e., the intercept); for both men and women, blood contamination predicted the cortisol level. The Level 2 model was represented by the following equations:

$$\beta_{1j} = \gamma_{10} + \gamma_{11}(\text{Female Blood Contamination}) + u_{1j}$$

$$\beta_{2j} = \gamma_{20} + \gamma_{21}(\text{Female Illness}) + u_{2j}$$

$$\beta_{3j} = \gamma_{30} + u_{3j}$$

$$\beta_{4j} = \gamma_{40} + \gamma_{41}(\text{Male Blood Contamination}) + \gamma_{42}(\text{Male Hours Slept})\ u_{4j}$$

$$\beta_{5j} = \gamma_{50} + \gamma_{51}(\text{Male Allergy Medication}) + u_{5j}$$

$$\beta_{6j} = \gamma_{60} + u_{6j}$$

After running this two-level model, the following coefficients were obtained at Level 1: women's cortisol level ($\beta_1 = -1.66$, $t(197) = -38.57$, $p < .001$), women's rate of change ($\beta_2 = -.11$, $t(197) = -5.68$, $p < .001$), women's curvature ($\beta_3 = -.19$, $t(198) = -6.75$, $p < .001$), men's cortisol level ($\beta_4 = -1.65$, $t(196) = -38.66$, $p < .001$), men's rate of change ($\beta_5 = -.29$, $t(197) = -12.70$, $p < .001$), and men's curvature ($\beta_6 = -.21$, $t(198) = -6.12$, $p < .001$). A graph of the average cortisol trajectory for women can be seen in Figure 5 and a graph of the average cortisol trajectory for men can be seen in Figure 6.

Path Analyses

Analyses with TEQ Score (Trauma Experience) as the Main Predictor

Direct Effects of TEQ Score on SIB Score (Model 1)

To evaluate the hypothesis that a history of traumatic experience would positively predict SIB, a path analysis was conducted in which men's TEQ score predicted men's SIB score and women's TEQ score predicted women's SIB score. As predicted, the paths from TEQ to SIB were positive and statistically significant for both men and women (see Figure 7). The standardized coefficients for these paths reveal that the effect of women's TEQ on women's SIB score was medium in size (.26), while the effect of men's TEQ on men's SIB score was small in size (.16). Also of note, the correlation

between the residual variance of men's SIB score and women's SIB score was significant, which demonstrates the importance of modeling the interdependence of the dyadic data.

Mediation by the Cortisol Trajectory (Model 2)

Given that the expected relations between TEQ score and SIB score were found, the planned path analyses were conducted to evaluate whether the cortisol trajectory mediated the relation between trauma experience and SIB. See Figure 8 for path coefficients. The hypothesis that the cortisol trajectory would mediate the relation between TEQ and SIB was not supported for either men or women. Neither the indirect effect of men's TEQ score on men's SIB score nor the indirect effect of women's TEQ score on women's SIB score reached significance. (See Table 7 for indirect, direct, and total effects on SIB.) Further, neither men's nor women's TEQ score predicted any of the cortisol variables.

There was evidence that the men's cortisol trajectory did predict men's SIB score. Specifically, men's cortisol level negatively predicted men's SIB score; thus, for men, lower cortisol levels during the conflict task were associated with higher SIB scores. In addition, the men's cortisol curvature negatively predicted SIB score; thus, men who had steeper cortisol trajectories had higher SIB scores. Overall, the women's cortisol trajectory did not predict women's SIB score, although there was a marginally significant negative relation between the women's cortisol curvature and women's SIB score that was similar to the pattern seen in men; thus, women who had steeper cortisol trajectories tended to have higher SIB scores, although this effect did not reach significance at the .05 level.

Gender Moderation (Models 2a-2c)

In order to assess whether gender moderated the relations in this mediation model, a series of models was run in which some paths were constrained to be equal between men and women. First, in Model 2a, the paths from the cortisol variables to SIB score as well as the path from TEQ score to SIB score were constrained to be equal for men and women. Then, in Model 2b, just the path from men's TEQ score to men's SIB score was constrained to be equal to the path from women's TEQ score to women's SIB score. Finally, in Model 2c, the paths from each of the three cortisol variables to SIB score were constrained to be equal for men and women.[5] A series of model comparison tests was conducted comparing each of these three models to the baseline model (Model 2, the fully unconstrained mediation model) in order to determine which model best fit the data. That basic logic behind these comparison tests was explained in the Analytic Strategy section. See Table 8 for the model comparison tests.

This series of model comparisons found support for Model 2a, the model in which both the paths from TEQ score to SIB score and the paths from the cortisol variables to SIB score were constrained to be equal. The fully unconstrained model was not a better fit to the data than any of the three constrained models described above, suggesting that gender did not moderate the relations between the cortisol trajectory and SIB score or the relations between TEQ score and SIB score. Thus, Model 2a, in which the cortisol-SIB score and TEQ score-SIB score paths were constrained to be equal for men and women, received the most support. (See Figure 9 for path coefficients.) In this model, TEQ score positively predicted SIB score. This association was not mediated by the cortisol trajectory for men or women as the indirect paths from men's TEQ to men's SIB and

women's TEQ to women's SIB were not significant (see Table 7). The cortisol trajectory was associated with SIB score. Specifically, cortisol level negatively predicted SIB score, as did the cortisol curvature. Thus, participants whose cortisol was less elevated during the conflict discussion and those who had steeper cortisol trajectories had higher SIB scores.

Adult Attachment as a Moderator

In order to assess whether adult attachment moderated the relation between TEQ score and the cortisol trajectory, participants' scores on attachment avoidance and attachment anxiety, as well as interactions between TEQ score and attachment avoidance and TEQ score and attachment anxiety were added as predictors of the three cortisol variables. Of note, these variables were added to the previously described model (Model 2a) in which the paths from the three cortisol variables to SIB score and the paths from TEQ score to SIB score were constrained to be equal between men and women. This model revealed that while attachment anxiety did moderate associations between TEQ score and the cortisol trajectory, attachment avoidance did not. Further, attachment avoidance had no direct effects on the cortisol trajectory. Consequently, both the main effects of attachment avoidance on the cortisol trajectory and the attachment avoidance x TEQ score interactions were dropped from the model. A model comparison test suggested that keeping the paths from attachment avoidance to the cortisol trajectory and the paths from the attachment avoidance x TEQ interaction to the cortisol trajectory did not improve model fit ($\Delta\chi^2 = 21.56$, $\Delta\ df = 20$, $p > .36$), offering empirical support for the removal of these paths.

The revised model (referred to as Model 3) can be seen in Figure 10 and the coefficients for this model can be seen in Table 9. In this model, women's attachment anxiety moderated the association between women's TEQ score and women's cortisol rate of change during the discussion. Men's attachment anxiety did not moderate any associations between TEQ score and the cortisol trajectory.

In addition to this interaction effect, there were direct effects of attachment anxiety on the cortisol trajectory for men and women. Specifically, attachment anxiety positively predicted cortisol level for both men and women; participants with higher attachment anxiety scores had higher levels of cortisol during the conflict discussion task. Attachment anxiety also positively predicted the cortisol rate of change during the discussion for women, but not for men. Thus, cortisol levels dropped more slowly for women with higher levels of attachment anxiety.[6] Finally, attachment anxiety negatively predicted the curvature of the cortisol trajectory for men, but not for women. Thus, men with higher levels of attachment anxiety had steeper cortisol trajectories. Consistent with previous models, cortisol level and curvature negatively predicted SIB score. Further, there was still no evidence that the cortisol trajectory mediated the relations between TEQ score and SIB score for either men or women (see Table 7 for indirect effects).

Gender Moderation (Models 3a – 3c)

In order to determine whether gender moderated any of the relations in this model, a series of models was run in which various paths were constrained to be equal for men and women. In Model 3a, the direct paths from attachment anxiety to the cortisol trajectory as well as the paths from the attachment anxiety x TEQ interaction to the cortisol trajectory were set to be equal between men and women. To better understand

possible moderation by gender, Model 3b constrained just the paths from the attachment anxiety x TEQ interaction to the cortisol trajectory and Model 3c constrained just the direct paths from attachment anxiety to the cortisol trajectory. See Table 8 for results of model comparison tests.

Each of these models was compared to the baseline model (Model 3 in which none of the attachment paths was constrained). The baseline model was not a better fit to the data than either Model 3a (all of the attachment anxiety paths constrained) or Model 3c (main effects of attachment anxiety constrained). The baseline model was a better fit than Model 3b (paths from the attachment anxiety x TEQ score interaction constrained) at the .07 level. This series of model comparisons suggests that gender did not moderate the main effects of attachment anxiety on the cortisol trajectory. However, gender did moderate the interaction between TEQ score and attachment anxiety in predicting the cortisol trajectory. Thus, Model 3c, in which the main effects of attachment anxiety on the cortisol trajectory were constrained, but the interaction effects were not, had the most support. In this model, women's attachment anxiety moderated the relation between women's TEQ score and women's cortisol rate of change; men's attachment anxiety, however, did not moderate any of the associations between TEQ score and the cortisol trajectory.

In order to understand the nature of these interactions, the association between women's TEQ and the cortisol rate of change during the discussion was plotted at high and low levels of women's anxiety and at high and low TEQ scores (high scores being one standard deviation above the mean and low scores being one standard deviation below the mean). Figure 12 shows that the association between women's trauma

experience and their cortisol rate of change depended on level of attachment anxiety. When attachment anxiety was high, the rate of change became more negative (i.e., cortisol was declining more quickly) as TEQ increased. In contrast, when attachment anxiety was low, there appeared to be the opposite effect; the rate of change became less negative (i.e., cortisol was declining more slowly) as TEQ increased. [7]

In addition to this interaction effect, there were direct effects of attachment anxiety on the cortisol trajectory for men and women. (See Figure 11 for path diagram and Table 9 for path coefficients.) Specifically, attachment anxiety positively predicted cortisol level and rate of change during the discussion. Thus, participants with higher levels of attachment anxiety tended to have higher cortisol levels during the conflict discussion task and their cortisol levels fell more slowly during the discussion. Attachment anxiety also negatively predicted the cortisol trajectory's curvature. Thus, individuals with higher levels of attachment anxiety had steeper cortisol trajectories. Consistent with previous models, cortisol level and cortisol curvature negatively predicted SIB score. Further, there was still no evidence that the cortisol trajectory mediated the relations between TEQ score and SIB score for either men or women (see Table 7 for indirect effects). [8]

Analyses with Trauma Factor Score (Trauma Symptoms) as the Main Predictor

Direct Effects of Trauma Factor Score on SIB Score (Model 4)

To evaluate the hypothesis that trauma-related symptoms would positively predict SIB, a path analysis was run in which men's trauma factor score predicted men's SIB score and women's trauma factor score predicted women's SIB score. As expected, the paths from trauma factor score to SIB score were positive and statistically significant for

both men and women (see Figure 13). The standardized coefficients for these paths reveal that the effect of women's trauma factor score on women's SIB score was large in size (.44), while the effect of men's trauma factor on men's SIB score was medium in size (.27). These effect sizes are larger in size than those seen in the comparable TEQ model.

Mediation by the Cortisol Trajectory (Model 5)

Given that the expected relations between trauma factor score and SIB score were found, the planned path analysis was run to evaluate whether the cortisol trajectory mediated the relation between trauma symptoms and SIB (see Figure 14 for path diagram and coefficients). Similar to the findings of the TEQ model, the hypothesis that the cortisol trajectory would mediate the relation between the trauma factor score and SIB score was not supported for either men or women. Neither the indirect effect of men's trauma factor score on men's SIB score nor the indirect effect of women's trauma factor score on women's SIB score reached significance (see Table 10 for indirect, direct, and total effects on SIB). Further, neither men's nor women's trauma factor score predicted any of the cortisol variables.

There was evidence that the men's cortisol trajectory did predict SIB score. Specifically, men's cortisol level negatively predicted men's SIB score and the men's cortisol curvature also negatively predicted the SIB score. Thus, men whose cortisol was less elevated during the conflict discussion and whose cortisol trajectories were steeper tended to have higher SIB scores. No aspects of the women's cortisol trajectory predicted women's SIB score.

Gender Moderation (Models 5a-5c)

In order to assess whether gender moderated the relations in this mediation model, a series of models was run in which some paths were constrained to be equal between men and women. The series of models is the same as that described above for the TEQ models. In Model 5a, the paths from the cortisol variables to SIB and the paths from trauma factor score to SIB were constrained to be equal for men and women. In Model 5b, the paths from trauma factor score to SIB score were constrained to be equal for men and women. In Model 5c, the paths from the cortisol variables to SIB score were constrained to be equal. Each of these models was compared to the baseline model (Model 5, the fully unconstrained mediation model) in which all paths were freely estimated (see Table 11 for model comparison tests).

This series of model comparisons found support for Model 5c, the model in which the paths from the cortisol variables to SIB score were constrained to be equal (see Figure 15). The fully unconstrained model was a better fit to the data than the model in which the paths from cortisol to SIB and trauma factor score to SIB were constrained. The additional model comparisons reveal which associations were moderated by gender. The unconstrained model was not a better fit to the data than the model in which the direct paths from the cortisol trajectory to SIB score were constrained, suggesting that gender did not moderate these associations. The unconstrained model was, however, a better fit than the model in which the paths from trauma factor score to SIB score were constrained, suggesting that gender did moderate this relation. Thus, Model 5c (see Figure 15) received the most support. In this model, the paths from the three cortisol variables to SIB score were constrained to be equal between men and women, while the

paths from trauma factor to SIB score were freely estimated. Comparison of the standardized coefficients finds that the effect of trauma factor score on SIB score is stronger for women (.43) than for men (.28); thus the nature of the association between trauma factor score and SIB was similar for men and women, but the magnitude differed.

In addition to the positive association between trauma factor score and SIB score, this model also shows a negative association between cortisol level and SIB score and between cortisol curvature and SIB score. (See Figure 15 for path coefficients.) Thus, similar to the TEQ models, those participants who had less elevated cortisol levels during the conflict discussion and steeper cortisol trajectories had significantly higher SIB scores. As in the unconstrained model, the relation between trauma factor score and SIB score was not mediated by the cortisol trajectory for either men or women (see Table 10 for indirect, direct, and total effects on SIB).

Adult Attachment as a Moderator

In order to assess whether adult attachment moderated the relation between trauma factor score and the cortisol variables, participants' scores on attachment avoidance and attachment anxiety, as well as interactions between trauma factor score and attachment avoidance and trauma factor score and attachment anxiety were added as predictors of the cortisol trajectory. Of note, these variables were added into the previously described model (Model 5c) in which the paths from the three cortisol variables to SIB score were constrained to be equal between men and women. This model revealed that while attachment avoidance did moderate associations between trauma factor score and the cortisol trajectory, attachment anxiety did not. Consequently, the attachment anxiety x trauma factor interactions were dropped from the model. A

model comparison test between these models suggested that keeping the paths from the attachment anxiety x trauma factor interaction to the cortisol trajectory did not improve model fit ($\Delta\chi^2 = 13.51$, $\Delta\ df = 10$, $p > .19$), providing empirical support for the removal of these paths.

The revised model (referred to as Model 6) can be seen in Figure 16 and Table 12. In this model, men's attachment avoidance moderated the associations between men's trauma factor score and men's cortisol level and the association between men's trauma factor score and men's cortisol rate of change (although this latter effect was marginal, reaching significance at the .10 level).

In addition to these interaction effects, there were direct effects of attachment anxiety on the cortisol trajectory for men and women. Specifically, attachment anxiety positively predicted cortisol level and the rate of change during the discussion for both men and women (although this latter effect was marginally significant for men). Thus, individuals with higher levels of attachment anxiety had higher levels of cortisol during the discussion that fell more slowly. Attachment anxiety also negatively predicted the curvature of the men's cortisol trajectory; men with higher levels of attachment anxiety had steeper trajectories. Attachment avoidance did not have any direct effects on the cortisol trajectory for either men or women. Consistent with previous models, cortisol level and cortisol curvature negatively predicted men's SIB score. Also consistent with previous models, there was no evidence that the cortisol trajectory mediated the relations between trauma factor score and SIB score for either men or women (see Table 10 for indirect effects).

Gender Moderation (Models 6a – 6c)

In order to determine whether gender moderated any of the relations in this model, a series of models was run in which various paths were constrained to be equal for men and women. In Model 6a, the direct paths from attachment anxiety and attachment avoidance to the cortisol trajectory as well as the paths from the attachment avoidance x trauma factor interaction to the cortisol trajectory were constrained to be equal between men and women. To better understand possible moderation by gender, Model 6b constrained just the paths from the attachment avoidance x trauma factor interaction to the cortisol trajectory and Model 6c constrained just the direct paths from attachment anxiety and avoidance to the cortisol trajectory. See Table 11 for results of model comparison tests.

Each of these models was compared to the baseline model (Model 6 in which none of the attachment paths was constrained). The baseline model was not a better fit to the data than either Model 6a (all of the attachment paths constrained) or Model 6c (main effects of the attachment variables constrained). The baseline model was a better fit than Model 6b (paths from the attachment avoidance x trauma factor score interaction constrained) at the .06 level. This series of model comparisons suggests that gender did not moderate the main effects of attachment avoidance and anxiety on the cortisol trajectory. However, gender did moderate the interaction between trauma factor score and attachment avoidance in predicting the cortisol trajectory.[9] Thus, Model 6c, in which the main effects of attachment anxiety and avoidance were constrained, but the interaction effects were freely estimated, received the most support. In this model (see Figure 17 for path diagram and Table 12 for coefficients), men's attachment avoidance

moderated the relation between men's trauma factor score and both men's cortisol level and cortisol rate of change during the discussion; women's attachment avoidance, however, did not moderate any of the associations between trauma factor score and the cortisol trajectory.

In order to understand the nature of these interactions, the association between men's trauma factor score and these aspects of the cortisol trajectory were plotted at high and low levels of men's avoidance and at high and low trauma factor scores (high scores being one standard deviation above the mean and low scores being one standard deviation below the mean). Figure 18 shows how the association between men's trauma symptoms and men's cortisol level during the conflict task depended on level of attachment avoidance. When avoidance was high, the men's cortisol level during the discussion decreased (was less elevated) as trauma factor score increased. In contrast, when avoidance was low, there appeared to be the opposite effect; the men's cortisol level during the discussion increased as trauma factor score increased.

Figure 19 shows how the association between men's trauma factor score and men's rate of cortisol change during the discussion depended on the level of attachment avoidance. When attachment avoidance was high, the rate of change became less negative (i.e., cortisol was declining less rapidly) as trauma factor score increased. In contrast, when attachment avoidance was low, there appeared to be the opposite effect; the rate of recovery became more negative (i.e.,cortisol was declining more quickly) as trauma factor score increased.

In addition to these interaction effects, there were direct effects of attachment anxiety on the cortisol trajectory for men and women. Specifically, attachment anxiety

positively predicted cortisol level and cortisol rate of change during the discussion. Attachment anxiety negatively predicted the cortisol trajectory's curvature at a marginal level of significance ($p < .10$). Thus, participants with higher levels of attachment anxiety had higher levels of cortisol during the conflict task, less rapid decline in cortisol levels, and steeper curvature. Attachment avoidance did not have any direct effects on the cortisol trajectory for either men or women. Consistent with previous models, cortisol level and cortisol curvature negatively predicted SIB score. Further, there was still no evidence that the cortisol trajectory mediated the relations between trauma factor score and SIB score for either men or women (see Table 10 for indirect effects). [10]

CHAPTER 4

DISCUSSION

Expanding the current understanding of SIB is critical given that it is a potentially dangerous behavior that is surprisingly prevalent not only in clinical samples, but also in the general population, especially among adolescents and young adults. The present study extends previous work suggesting that problems with affect regulation are implicated in SIB. Specifically, the findings of the present study indicate that individuals who engage in higher levels of SIB show a distinct pattern of HPA axis reactivity to an interpersonal stressor. The current study also further extends prior work linking trauma and SIB, finding associations between SIB and both trauma experience and symptoms in a non-patient sample. Although the mediating model, by which HPA axis reactivity was proposed to mediate the relation between trauma and SIB, was not supported, the study's findings add to the existing literature linking traumatic experience and trauma-related psychopathology to alterations in the HPA stress response. Specifically, the findings suggest that the next steps in exploring links between trauma and the HPA response will need to take a more complex view of these relations. For instance, the results demonstrate that trauma symptoms and trauma experience, while having some similar relations to HPA axis reactivity, also have unique associations. Further, the current study shows that links between trauma and HPA axis reactivity are moderated by both gender and attachment style. I elaborate upon each of these points in the following sections.

SIB and HPA Axis Reactivity

A large body of theoretical and empirical work with both humans and primates suggests that a primary function of SIB is regulation of affect and/or tension reduction

(e.g., see reviews by Klonksy, 2007 and Suyemoto, 1998). While many SIB researchers assume that individuals who engage in SIB have problems with affect and arousal modulation at a biological level, relatively little is known about what biological systems or processes might be involved. To the best of my knowledge, the current study is one of the first to report an association between SIB and HPA axis function in people without mental retardation. In this study, individuals engaging in higher levels of SIB displayed a more reactive HPA axis response to an interpersonal stress task in that their cortisol trajectories showed steeper curvature (e.g., steeper cortisol rise and fall). This steeper and more acute response occurred within the context of lower levels of cortisol than those seen in individuals with less reported SIB. This pattern of HPA axis response suggests that individuals with higher levels of SIB may be more sensitive to the interpersonal stress associated with discussing a conflict with a romantic partner. This appears consistent with prior work suggesting that acts of SIB are often precipitated by interpersonal problems or conflicts (e.g., Herpertz, 1995; Ghaziuddin, et al., 1992; Suyemoto, 1998), although future studies are needed to explore whether the pattern of HPA axis response observed in this study is unique to interpersonal stress as opposed to more general stress.

While the current study cannot definitively speak to why this pattern of HPA axis reactivity is associated with SIB, one plausible explanation is that individuals who have more acute stress responses are left vulnerable to developing maladaptive strategies for coping with their distress, one of which may be SIB. This explanation is conceptually consistent with primate work showing reductions in physiological indicators of stress following acts of self-injury (Marinus et al., 1999) and human work showing reductions

in physiological arousal and negative affect among individuals with SIB histories following exposure to imaginal self-injury scripts (Brain et al., 1998; Haines et al., 1995). This work suggests that SIB is an effective (if ultimately maladaptive) strategy for coping with intense physiological stress. The current study suggests that individuals who engage in SIB, in fact, have a more sensitive physiological stress response to interpersonal conflict.

Of note, this pattern of HPA axis reactivity bears marked similarity to that associated with PTSD and trauma reported in much of the literature. Yehuda (e.g., 1997, 2002) suggests that because of their exposure to trauma and stress, the HPA axis response is downregulated to protect the body from the potentially harmful effects of a chronically activated stress response. This downregulation results in lower levels of cortisol and in a more sensitized HPA system that is maximally responsive to stress cues and quite efficient in shutting down the stress response (via enhanced negative feedback). While the HPA axis response that was linked to SIB in the current study was not, in fact, directly associated with either trauma symptoms or trauma experience, the nature of the HPA axis profile seems quite similar to that associated with PTSD and trauma in the literature. Possible reasons as to why trauma as assessed in the current study was not directly linked to HPA axis reactivity are discussed below.

Trauma and HPA Axis Reactivity

While the hypothesized relations between trauma experience and SIB and between trauma symptoms and SIB were found, these relations were not mediated by the HPA axis response. Further, the expected main effects of trauma symptoms and experience on the cortisol trajectory were not found. The mediation hypothesis posited

that trauma experience (or symptomatic response to trauma experience) has a dysregulating effect on the HPA stress response. In turn, this altered HPA stress response then leaves individuals vulnerable to developing maladaptive coping strategies including SIB. While evidence for mediation was not found in the present study, it is possible that this proposed mediation might be found under certain circumstances. For instance, Schore (2002) suggests that relational traumas (for instance, abuse and/or neglect by a caregiver) may have particularly dysregulating effects on the HPA axis. Thus, it may be that the association between relational traumas (including abuse and neglect by a caregiver, and potentially also including adult relationship abuse) and SIB is mediated by HPA axis reactivity. The current study used a broad measure of trauma that included both relational and non-relational events (e.g., car accidents, fires, etc.). It may be that a mediating relation does not hold when trauma is conceptualized so broadly. Further, relatively few individuals in the sample reported childhood physical and sexual abuse, which may be the forms of trauma that have a particularly dysregulating effect on the HPA system's reactivity to interpersonal conflict.

Similarly, a few recent studies suggest that different types of trauma may be associated with different HPA axis profiles. Meewisse et al.'s (2007) meta-analysis of studies looking at basal cortisol levels in individuals with PTSD found that only individuals who had experienced physical or sexual abuse had lower basal cortisol than non-traumatized controls. Flory et al. (2009) explored relations between basal cortisol levels and five types of childhood trauma (sexual abuse, physical abuse, emotional abuse, physical neglect, and emotional neglect). Only physical abuse and physical neglect were related to basal cortisol. Further, while physical abuse was associated with lower basal

cortisol, physical neglect was associated with higher basal cortisol. The authors suggested that different types of trauma may have distinct effects on the HPA system. By using a broad trauma variable that collapses across multiple types of trauma, it is possible that distinct effects of different types of trauma on the HPA axis response may be washed out.

In addition to the type of trauma experienced, age and/or developmental period at the time of the trauma may also influence the effects that trauma has on the HPA system. Much of the existing work exploring associations between PTSD and HPA axis function focuses on adult traumas (e.g., combat exposure, adult sexual assault, domestic violence, etc.), suggesting that experiencing trauma in adulthood is associated with functioning of the HPA system. Nonetheless, other work provides reason to suggest that traumatic events experienced in childhood, and perhaps especially in early childhood, may have particularly important effects on HPA axis function. Work by Schore (2002), for instance, suggests that the first two years of life are critical to the development of the infant's biobehavioral response to stress and arousal. Schore (2002) and Gunnar (2000) suggest that adverse conditions in early life negatively affect the development of the child's HPA axis, leaving the child vulnerable to developing psychopathological conditions later in life. Further, much of the work on the development of SIB specifically implicates the role of trauma and life stress occurring in childhood versus adulthood (e.g., van der Kolk, 1996a; van der Kolk & Fisler, 1994; Yates, 2004). The present study did not explore whether age at the time of trauma moderated relations between trauma experience and trauma symptoms and HPA axis response. Further, participants reported very few traumatic experiences occurring before the age of three, which would limit the

ability to detect potential associations between such early traumas and HPA axis reactivity. Thus, future work looking at associations between traumatic experience, the HPA axis response, and SIB could explore the possible influence of the age/developmental period during which trauma occurred.

An additional consideration in exploring whether the HPA axis stress response mediates relations between trauma and SIB is the context in which stress occurs. In the present study, HPA axis reactivity to an interpersonal conflict task was assessed. This type of interpersonally stressful event may be especially relevant for individuals who engage in SIB given findings that SIB often follows perceived or actual interpersonal rejections or conflict. This close conceptual connection, however, may not be present between HPA axis response to an interpersonal stressor and the general measures of trauma experience and symptoms used. It is plausible that individuals who have experienced particular types of trauma show altered HPA axis response only in reaction to particular types of stressors (e.g., relational trauma may be more closely associated with response to interpersonal stress, while non-relational trauma may be more closely associated with response to a more general stressor or to a stressor with implications for personal safety). Thus, because of the interpersonal nature of the stress task used, the study task may have provided a better opportunity to see connections between cortisol and SIB than between trauma and cortisol. Future work could explore whether trauma is associated with the HPA axis response to different types of stress tasks.

Moderating Effects of Adult Attachment

While the expected main effects of trauma experience and symptoms on the cortisol trajectory were not found, the trauma variables did predict the cortisol trajectory

in interaction with adult attachment. These findings offer general support for the exploratory hypothesis that adult attachment would moderate associations between trauma and HPA axis response. For women, adult attachment anxiety moderated the relation between trauma experience and HPA axis reactivity. At high levels of attachment anxiety, women's trauma experience negatively predicted the cortisol rate of change during the discussion. In other words, when attachment anxiety was high, cortisol was falling faster during the discussion for women with more trauma experience. This finding is at least partially consistent with the pattern of HPA axis activity described by Yehuda (e.g., 2002) among people with PTSD and trauma histories (i.e., more acute reactivity in the context of overall lower levels of cortisol). While the overall level of cortisol was not lower for women with high attachment anxiety and trauma experience, these women did show a more responsive stress response in that cortisol levels were coming down more rapidly. These findings provide partial support for my hypothesis that secure adult attachment would play a protective role, buffering the effects of trauma on HPA axis response. For women, the expected associations between trauma symptoms and the cortisol trajectory were only seen when attachment anxiety was high.

For men, a different pattern was observed. Avoidant attachment moderated the relation of trauma symptoms to both cortisol level and cortisol rate of change during the discussion. These findings are partially consistent with the pattern of HPA axis activity seen among individuals with PTSD and trauma histories as described by Yehuda (i.e., more acute reactivity in the context of overall lower levels of cortisol). Consistent with the past work, the present findings show that when men's attachment avoidance was high, trauma symptoms were associated with lower levels of cortisol. However, the

positive association between trauma symptoms and the cortisol rate of change during the discussion (i.e., slower cortisol decline) is inconsistent with the expected HPA axis response. It may be that, although men with trauma symptoms have usually developed HPA systems that react and recover quickly to stressors, men with trauma symptoms who are avoidantly attached and cannot escape a forced conflict task are challenged beyond what their systems can normally deal with, resulting in slower decline in cortisol during the conflict task. Perhaps, then, avoidant attachment under conditions of a non-escape conflict situation challenges the stress system more than non-attachment related stressors. At this point, this explanation is speculative and would need to be tested by studies comparing HPA axis reactivity under different stress conditions.

Main Effects of Adult Attachment on HPA Axis Reactivity

While not of primary interest in this study, there were also main effects of attachment anxiety on the cortisol trajectory. Within both the trauma experience and trauma symptoms models, attachment anxiety positively predicted cortisol level and the rate of change at the discussion and negatively predicted cortisol curvature (although this effect was marginal in the trauma symptoms model). Thus, attachment anxiety was associated with an HPA axis response in which cortisol levels were generally higher, cortisol was falling more slowly at the discussion, and the curvature of the trajectory was steeper. These findings suggest that individuals high in attachment anxiety were quite reactive to the conflict task (as indicated by the steepness of the cortisol trajectory) and their cortisol was declining at a slower rate during the discussion. Perhaps anxiously attached individuals find the conflict discussion to be particularly threatening, and,

consequently experience a higher level of physiological stress than more securely attached individuals.

Previous work (Powers et al., 2006) with a sub-sample of the current data also found that insecurely attached individuals showed patterns of greater physiological stress reactivity to the interpersonal conflict task. Of note, however, the pattern of associations was somewhat different from that seen in the present analyses. While Powers et al. found that attachment avoidance was related to women's HPA axis response and attachment anxiety was related to men's HPA axis response, the current analyses suggest that, attachment anxiety, but not avoidance, predicted HPA axis reactivity to the interpersonal stress task in similar ways for men and women. While a few methodological differences exist that may contribute to the discrepancy in findings (e.g., the previous analyses used only a sub-sample, the data were analyzed in somewhat different ways), the different pattern of associations between attachment and cortisol may also reflect the inclusion of trauma measures in the present analyses. It may well be that the presence of trauma influences associations between attachment and HPA axis reactivity (which is also seen in the above-described interactions between attachment and trauma).

Gender

The present study also explored whether gender moderated any of the hypothesized relations between trauma, HPA axis response, attachment, and SIB. This question was largely exploratory and there were few theoretical reasons to predict specific gender differences. In fact, most of the relations were similar for men and women (specifically, the relations between the cortisol trajectory and SIB, between trauma experience and SIB, and the main effects of attachment anxiety and avoidance on

the cortisol trajectory). Nonetheless, a few noteworthy gender differences were observed. While trauma symptoms positively predicted SIB for both men and women, this association was stronger for women. While a clear explanation of this finding is not readily apparent, past research on SIB suggests a few possibilities. Work by Gratz et al., (2002), for instance, suggests that the types of childhood traumatic events that are associated with the development of SIB may differ between men and women. Thus, it is possible that, in the current sample, participants may have experienced more of those events that are associated with SIB in women. With the exception of Gratz's work, few studies have explored gender differences in the risk factors for SIB. While the current study does not present clear findings as to gender differences, the results do suggest that continued work on differential relations between trauma and SIB for men and women may be fruitful.

There were also gender differences in how attachment moderated the relations between trauma and the cortisol trajectory. As described above, attachment anxiety moderated relations between trauma experience and the cortisol trajectory for women, while attachment avoidance moderated the relations between trauma symptoms and the cortisol trajectory for men. While the precise explanation of this finding remains unclear, this pattern of gender differences provides support to the argument made by Pietromonaco, Greenwood, and Felman Barrett (2004) that gender needs to be considered when studying the influences of attachment style in the context of relationship conflict.

Trauma Experience and Trauma Symptoms

The present study also suggests that, when exploring associations between trauma, HPA axis function, attachment, and SIB, the distinction between trauma

symptoms and trauma experience is important. While both trauma symptoms and trauma experience predicted SIB, this association was stronger for trauma symptoms. This might suggest that the predictive difference between trauma symptoms and experience is one of degree; that is, individuals with trauma histories will show increased risk of SIB, but individuals with trauma histories who are also experiencing trauma-related symptoms are at greater risk. Other findings from this study, however, suggest that the distinction between trauma symptoms and experience may not simply be additive. For instance, trauma symptoms and trauma experience interacted differently with attachment to predict the cortisol trajectory (symptoms interacted with men's avoidance and experience interacted with women's anxiety), suggesting a more qualitative difference in the effects of trauma symptoms and experience.

Limitations

While the present study does have a number of strengths and contributes to the current understanding of links between stress physiology and both trauma and SIB, the study's limitations should be kept in mind when interpreting the results. This study relied on self-report measures of trauma experience, trauma symptoms, and SIB. Given that SIB is often associated with shame and secrecy, it is possible that participants may have under-reported the extent of their self-injury. As mentioned previously, reliance on a self-report measure of traumatic experience likely reduced my ability to measure traumatic events that occurred at very young ages, during a period of time that individuals could not remember. An additional limitation of the present study was its cross-sectional nature. The proposed links between trauma and HPA axis function and between HPA axis function and SIB implied a chronological sequence (i.e., traumatic

experience brings about HPA axis dysregulation, which then increases vulnerability for the development of SIB behaviors at some later time point). The study did find that HPA axis reactivity predicted SIB; however, this study cannot establish temporal precedence as all variables were measured at the same time point.

Further, the associations between the HPA axis response and SIB are correlational in nature. While theoretical reasons suggest that a highly reactive HPA system might contribute to vulnerability for the development of SIB, the possibility that engaging in SIB affects HPA axis reactivity cannot be ruled out. For instance, it is possible that SIB coping is, in itself traumatic, and thus leads to a downregulated, but more sensitive HPA axis reaction. The possibility that a third variable (perhaps genetic factors or temperament/personality traits) leads to both downregulated, sensitive HPA axis function and to SIB should also be considered. Prospective longitudinal work is needed to address these issues.

Another potential limitation of the current study was the use of a non-clinical sample. Given that several of the variables of interest (SIB, trauma symptoms, and rates of abuse and other forms of trauma) are lower in the general population than in clinical populations, this study may not have been able to detect effects that may be present in clinical samples. For instance, the rates of relational traumas (such as parental physical and sexual abuse) were quite low in this sample and these types of relational traumas may, in fact, be those most likely to be associated with HPA axis reactivity. Certainly, future work with clinical samples exploring associations between trauma, the HPA axis response, and SIB is indicated. At the same time, the use of a non-clinical sample is an important strength of this study. Researchers are increasingly recognizing that SIB is not

infrequent among adolescents in the general population, making exploration of a college sample particularly relevant. Further, that this study found important relations between SIB and both trauma symptoms/experience and between SIB and HPA axis reactivity contributes to our understanding of factors contributing to SIB in the general population.

Clinical Implications

In light of emerging evidence that SIB is a surprisingly common problem, not only in clinical populations, but also among adolescents and young adults in the general population, research on the correlates of SIB as well as risk and protective factors is of great clinical relevance. In particular, understanding the psychophysiological processes involved in self-injury expands the current understanding of the behavior's functions and offers some potential insights for intervention. The most commonly proposed functional theory of SIB holds that individuals who engage in SIB experience intense and intolerable arousal and that SIB functions to reduce this distress. Much of this work is based on the self-reported reasons individuals give to explain their self-injury. The current study provides objective evidence that individuals who engage in SIB do have highly sensitive physiological reactions to interpersonal stress.

Better understanding this link between physiological reactivity to stress and SIB may also inform clinical interventions for SIB, suggesting targets for intervention. For instance, this study's findings that self-injurers have highly sensitive reactions to stress suggest that clinical interventions for SIB will need to focus on building distress tolerance and stress-reduction skills, perhaps especially in the context of interpersonal conflict. Further, these findings provide additional support for existing treatment approaches that focus on these skills, such as dialectical behavior therapy (e.g., Miller,

Rathus, & Linehan, 2007). Further, a fuller understanding of SIB may help clinicians and others respond to and work with self-injurers in more productive ways. SIB tends to be a behavior that is met with misunderstanding and clinical work highlights the negative counter-transference reactions it evokes (e.g., Rayner, Allen & Johnson, 2005). Equipped with the understanding that self-injurers have a highly sensitive physiological response to stress (which may have roots in adverse and traumatic experience), clinicians and family members may gain better insight into SIB and respond in more supportive and validating ways.

Conclusions

This study demonstrates that individuals in a non-patient sample who engage in SIB have a particularly reactive HPA stress response to interpersonal conflict. This work is one of the first studies in a non-MR human sample to report links between SIB and the HPA axis. This association with HPA axis function adds to the existing work on the functions of SIB by highlighting a potential biological mechanism through which problems in affect regulation are associated with SIB. In addition, this study further extends the existing work exploring the influence of trauma on the HPA system. This study, in conjunction with other recent work, provides evidence that the study of trauma's effects on the HPA axis needs to move in more complex directions. Specifically, the findings demonstrate the importance of exploring ways in which attachment and gender interact with trauma to influence HPA axis response. Further, they underscore the necessity of understanding the potentially distinct relations between HPA axis reactivity and trauma experience and trauma-related psychopathology.

Footnotes

[1] Of note, the definition of SIB used in this study could be considered overinclusive as it included suicide attempts.

[2] For one category of trauma, receiving news of the mutilation, serious injury, or violent or unexpected death of someone close, the intensity score was based on the average of three questions, because participants were not asked whether they themselves had been injured.

[3]As will be described in further detail below, HPA axis reactivity is not represented by a single variable. Rather, it is represented by three variables that I refer to as cortisol level, rate of change during the discussion, and curvature.

[4]Previous work with this sample has indicated that a curvilinear model best fit the cortisol trajectory, which tends to rise in anticipation of the conflict discussion before recovering to normal values following the discussion (Powers, Pietromonaco, Gunlicks, & Sayer, 2006).

[5]The paths between TEQ score and the cortisol variables were not constrained in any of these models because these paths did not reach significance for men or women in the unconstrained mediation model.

[6] On average, the cortisol rate of change was negative. Prior analyses with this sample found, that in the average trajectory, cortisol peaked at anticipation of the conflict and declined through the discussion and the recovery window. Consequently, I interpret the positive relation between attachment anxiety and rate of change as indicating that as anxiety rises, cortisol is falling more slowly during the discussion (i.e., the rate of change is less negative).

[7]Because the model comparison between the baseline model and Model 3b was significant at a marginal (.07) level, I ran additional analyses that provided further evidence that gender did moderate the relation between attachment anxiety and TEQ score. I ran the model separately, once for women alone and then for men alone. In the women's model, the attachment anxiety x TEQ interaction predicted cortisol rate of change ($\beta = -.029$, $p < .001$). In the men's model, this interaction term did not predict any component of the cortisol trajectory.

[8]Model fit statistics for this model were examined and suggested that this final model did not adequately fit the data ($\chi^2(56) = 192.75$, $p < .001$; RMSEA = .12 (90% CI = .10, .14); NNFI = -.08; CFI = .33; SRMR = .10). The complexity of the model and the complexity of the phenomena assessed (i.e., there are several other predictors of SIB and HPA axis reactivity in addition to those studied here) may help to explain the relatively poor model fit. More importantly, however, these models address pointed questions about relations between trauma, HPA axis reactivity, and SIB. Further, the series of model comparison tests reported in Table 8 indicate that this final model is a better fit to than data than previous models.

[9]Because the model comparison between the baseline model and Model 6b was significant at a marginal (.06) level, I ran additional analyses that provided further evidence that gender moderated the effects of the attachment avoidance x trauma factor score interaction on the cortisol trajectory. I ran the model separately, once for men alone and then for women alone. In the men's model, the attachment avoidance x trauma factor interaction predicted men's cortisol level ($\beta = -.076$, $p < .05$) and there was a trend toward the attachment avoidance x trauma factor interaction predicting the men's rate of change ($\beta = .028$, $p < .06$). In the women's model, this interaction term did not predict any component of the cortisol trajectory.

[10]As seen in the final TEQ model, model fit indices also suggest that this model does not adequately fit the data ($\chi^2(68) = 223.27$, $p < .001$; RMSEA = .11 (90% CI = .10, .13; NNFI = .08; CFI = .48; SRMR = .09). Nonetheless, the model comparison tests indicate that this final model is a better fit than previous models.

Table 1

Descriptive Statistics for Independent and Dependent Variables

	Men			Women		
Variable	*M*	*SD*	Range	*M*	*SD*	Range
SIB						
Raw SIB Composite Score	3.62	5.88	0.00 - 43.00	3.42	6.52	0.00 - 32.00
Square Root of SIB Composite[b]	1.26	1.43	0.00 - 6.56	1.04	1.53	0.00 - 5.66
TEQ						
Raw TEQ Composite Score	6.96	7.20	0.00 - 37.00	7.58	9.90	0.00 - 80.75
Square Root of TEQ Composite[b]	2.29	1.31	0.00 - 6.08	2.34	1.46	0.00 - 8.99
Trauma Factor Score[b]	0.05	2.14	-3.65 - 10.30	-0.02	1.70	-3.52 - 5.75
Cortisol						
Cortisol Intercept[b]	-1.63	0.62	-3.41 - 0.29	-1.66	0.62	-3.34 - 0.26
Cortisol Linear Term[a]	-0.28	.30	-1.03- 0.81	-0.12	0.26	-0.81 - 0.80
Cortisol Quadratic Term[b]	-0.23	.40	-1.24 - 1.95	-0.19	0.32	-1.03 - 1.27
ECR Subscales						
Attachment Anxiety Score[b]	3.55	1.02	1.00 - 6.35	3.71	1.01	1.31 - 6.80
Attachment Avoidance Score[a]	2.65	0.78	1.33 - 5.39	2.46	0.77	1.33 - 5.06

Note. The sample size for the TEQ composite and raw scores is 175 men and 175 women. The sample size for all other variables is 178 men and 178 women.
[a]The difference between the means for men and women on these variables was significant at the .05 level. [b]The difference between the means for men and women on these variables was not significant ($p > .10$).

Table 2

Correlations between Dependent and Independent Variables, by Gender

Variables	1	2	3	4	5	6	7	8
1. Square Root TEQ Composite	--	.299 ***	.076	-.117	.008	.235 ***	.254 ***	.261 ***
2. Trauma Factor Score	.128	--	.021	-.066	-.064	.302 ***	.182 **	.446 ***
3. Cortisol Intercept	.074	.017	--	.339 ***	-.553 ***	.189 **	.034	.054
4. Cortisol Linear Term	-.010	.043	-.112	--	-.205 ***	.106	-.114	.015
5. Cortisol Quadratic Term	-.044	-.005	-.470 ***	.026	--	-.069	-.034	-.093
6. Attachment Anxiety	.083	.324 ***	.253 ***	.118	-.152 **	--	.168 **	.235 ***
7. Attachment Avoidance	.028	.191 **	.044	.019	-.039	.083	--	.082
8. Square Root SIB Composite	.180 **	.282 ***	-.118	-.028	-.087	.032	.136	--

Note. Correlations below the diagonal are the correlations among the men's variables and correlations above the diagonal are the correlations among the women's variables. With the exception of correlations with the TEQ composite, the above correlations were calculated using the sample of 178 couples used in the trauma factor analyses. The correlations with the TEQ composite were calculated from the sample of 175 couples used in the TEQ analyses. The correlations between the TEQ composite and the trauma factor score were calculated from 173 couples.

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

Table 3

Frequency and Recency of SIB, by Category

	Bruising		Hitting		Hair Pulling		Scratching		Biting		Eating		Burning		Cutting	
Frequency	Count	Percent	Count	Percent	Count	Percent	Count	Percent	Count	Percent	Count	Percent	Count	Percent	Count	Percent
Never	291	82.20%	281	79.15%	314	89.46%	288	82.05%	295	83.10%	345	97.18%	323	91.76%	336	94.65%
Once	13	3.67%	24	6.76%	5	1.42%	15	4.27%	16	4.51%	8	2.25%	12	3.41%	7	1.97%
2-5 times	32	9.04%	31	8.73%	19	5.41%	27	7.69%	33	9.30%	2	0.56%	15	4.26%	9	2.53%
6-10 times	5	1.41%	9	2.53%	5	1.42%	9	2.56%	4	1.13%	0	0%	1	0.28%	0	0%
11-20 times	5	1.41%	4	1.13%	3	0.85%	8	2.28%	4	1.13%	0	0%	1	0.28%	1	0.28%
Over 20 times	8	2.26%	6	1.69%	5	1.42%	4	1.14%	3	0.85%	0	0%	0	0%	2	0.01%
At least one act	63	17.80%	74	20.85%	37	10.54%	63	17.95%	60	16.90%	10	2.82%	29	8.24%	19	5.35%
Recency																
Never	291	82.20%	284	80.00%	314	89.46%	289	82.34%	295	83.10%	346	97.46%	323	91.76%	336	94.65%
Past Week	2	0.56%	4	1.13%	9	2.56%	2	0.57%	1	0.28%	2	0.56%	1	0.28%	1	0.28%
Past Month	7	1.98%	6	1.69%	2	0.57%	3	0.85%	1	0.28%	0	0%	2	0.57%	0	0%
Past 6 Months	11	3.11%	10	2.82%	5	1.42%	5	1.42%	12	3.38%	0	0%	4	1.14%	0	0%
Past Year	11	3.11%	14	3.94%	6	1.71%	7	1.99%	13	3.66%	1	0.28%	5	1.42%	2	0.56%
1-5 Years Ago	24	6.78%	28	7.89%	11	3.13%	29	8.26%	20	5.63%	3	0.85%	13	3.69%	8	2.25%
>5 Years Ago	8	2.26%	9	2.53%	4	1.14%	16	4.56%	13	3.66%	3	0.85%	4	1.14%	8	2.25%
At least one act	63	17.80%	71	20.00%	37	10.54%	62	0.1766	60	16.96%	9	2.53%	29	8.24%	19	5.35%

[a] This variable represents the number of individuals who reported at least one act of SIB in each category. [b] This variable represents the number of individuals who reported engaging in SIB in each category on the basis of their responses to the recency question.

Table 4

Descriptive Statistics from the TEQ: Frequency, Average Intensity, and TEQ Composite Scores, by Category

	Accident		Violent Crime		Witness		Personal Danger		Received News		Other Trauma		Can't Tell		Other Event	
Variable	Count	Percent	Count	Percent	Count	Percent	Count	Percent	Count	Percent	Count	Percent	Count	Percent	Count	Percent
Frequency																
0 Times	251	71.71%	296	84.57%	322	92.00%	277	79.14%	235	67.14%	326	93.41%	334	95.43%	233	69.97%
1 Time	53	15.14%	42	12.00%	21	6.00%	58	16.57%	76	21.71%	19	5.44%	8	2.29%	70	21.02%
2 Times	33	9.43%	4	1.14%	2	0.57%	8	2.29%	26	7.43%	1	0.29%	2	0.57%	16	4.80%
3 or More Times	13	3.71%	8	2.29%	5	1.43%	7	2.00%	13	3.71%	3	0.86%	6	1.71%	14	4.20%
At least one event	99	28.29%	54	15.43%	28	8.00%	73	20.86%	115	32.86%	23	6.59%	16	4.57%	100	30.03%
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Average Intensity*	2.44	1.17	3.07	1.26	2.09	0.86	3.60	1.17	3.18	0.95	2.70	0.69	3.08	1.07	2.67	0.99
Raw TEQ Composite*	3.89	3.01	4.45	3.59	2.96	2.55	4.70	2.88	4.71	2.87	3.41	2.38	6.13	3.83	3.76	2.27

Note. These descriptive statistics are calculated from the 350 participants who had complete TEQ data.
*Both the Average Intensity and the Raw TEQ Composite variables represent the averages for people who endorsed that category of trauma and do not represent the average for the entire sample.

Table 5

Descriptive Statistics for the TEQ Abuse Categories: Duration, Average Intensity, and TEQ Composite Scores

	Childhood Abuse		Adult Unwanted Sexual Experience		Adult Relationship Abuse	
Variable	Count	Percent	Count	Percent	Count	Percent
Age at **Onset** of Abuse						
No Trauma Endorsed	320	91.43%	337	96.29%	327	93.43%
Age 3 or Younger	5	1.43%	0	0%	0	0%
Age 4-6	10	2.86%	0	0%	0	0%
Age 7-10	7	2.00%	0	0%	0	0%
Age 11-13	2	0.57%	0	0%	1	0.29%
Age 14-16	6	1.71%	6	1.71%	9	2.57%
Age 17-19	0	0%	6	1.71%	13	3.71%
Age 20-22	0	0%	1	0.29%	0	0%
Older than age 22	0	0%	0	0%	0	0%
At least one event	30	8.57%	13	3.71%	23	6.57%
Age at **Offset** of Abuse						
No Trauma Endorsed	320	91.43%	337	96.29%	327	93.43%
Age 3 or Younger	1	0.29%	0	0%	0	0%
Age 4-6	3	0.86%	0	0%	0	0%
Age 7-10	8	2.29%	0	0%	0	0%
Age 11-13	8	2.29%	0	0%	0	0%
Age 14-16	8	2.29%	6	1.71%	6	1.71%
Age 17-19	2	0.57%	6	1.71%	17	4.86%
Age 20-22	0	0%	1	0.29%	0	0%
Older than age 22	0	0%	0	0%	0	0%
	M	*SD*	*M*	*SD*	*M*	*SD*
Average Intensity*	3.00	1.10	2.77	1.43	2.79	1.09
Raw TEQ Composite*	8.98	3.35	8.31	4.29	8.38	3.26

Note. The descriptive statistics are calculated from the 350 participants who had complete TEQ data.
* Both the Average Intensity and the Raw TEQ Composite variables represent the averages for people who endorsed that category of trauma and do not represent the average for the entire sample.

Table 6

TSC-40 Descriptive Statistics

Subscale	*M*	*SD*	Range
Dissociation	3.72	2.72	0.00 - 16.00
Anxiety	4.52	3.15	0.00 - 16.00
Depression	5.88	3.37	0.00 - 19.00
Sleep	6.02	3.39	0.00 - 18.00
Post-Sexual Abuse Trauma	2.79	2.31	0.00 - 12.00
Sexual Problems	3.64	2.94	0.00 - 16.00
Total Score	23.77	11.73	0.00 - 76.00

N = 356

Table 7

Indirect, Direct, and Total Effects on SIB Score in the TEQ Analyses

	Indirect	Direct	Total
Model 2 (Baseline Mediation Model)			
Men's TEQ	-0.009	0.186 **	0.177 **
Women's TEQ	-0.015	0.284 ***	0.270 ***
Model 2a (Constrained Mediation Model)			
Men's TEQ	-0.004	0.236 ***	0.232 ***
Women's TEQ	-0.013	0.236 ***	0.224 ***
Model 3 (Baseline Attachment Model)			
Men's TEQ	-0.003	0.236 ***	0.234 ***
Women's TEQ	-0.011	0.236 ***	0.226 ***
Men's Attachment Anxiety	-0.017		-0.017
Women's Attachment Anxiety	-0.023		-0.023
Men's Attachment Anxiety x TEQ	-0.010		-0.010
Women's Attachment Anxiety x TEQ	0.005		0.005
Model 3c (Constrained Attachment Model)			
Men's TEQ	-0.002	0.236 ***	0.234 ***
Women's TEQ	-0.011	0.236 ***	0.226 ***
Men's Attachment Anxiety	-0.022		-0.022
Women's Attachment Anxiety	-0.022		-0.022
Men's Attachment Anxiety x TEQ	-0.010		-0.010
Women's Attachment Anxiety x TEQ	0.005		0.005

* $p < .10$, ** $p < .05$, *** $p < .01$

Table 8

Model Comparisons for the TEQ Analyses

	MFF $\chi 2$	*df*	$\Delta\chi 2$	Δdf	*p*-value	Reject H_0?
Model 2 (Baseline)	171.84	29				
Model 2a	175.06	33	3.22	4	0.52	no
Model 2b	172.65	30	0.81	1	0.37	no
Model 2c	174.15	32	2.31	3	0.51	no
Model 3 (Baseline)	191.15	53				
Model 3a	199.48	59	8.33	6	0.42	no
Model 3b	198.09	56	6.94	3	0.07	yes
Model 3c	192.75	56	1.60	3	0.39	no

Note. MFF $\chi 2$ = Minimum Fit Function Chi-Square. Within each group of models, the baseline model in which relevant paths were not constrained is compared to each of the constrained models that follows. The null hypothesis states that the baseline model does not fit as well as the constrained model.

Table 9

Coefficients for the Direct Effects in the TEQ Attachment Moderation Models

Path	Men's Coefficient	Women's Coefficient
Model 3 - Baseline Attachment Model		
TEQ-SIB	0.236 ***	0.236 ***
TEQ-Cortisol Level	0.022	0.025
TEQ-Cortisol Rate of Change	-0.005	-0.017
TEQ-Cortisol Curvature	-0.009	0.003
Attachment Anxiety-Cortisol Level	0.171 ***	0.127 ***
Attachment Anxiety-Cortisol Rate of Change	0.032	0.042 **
Attachment Anxiety-Cortisol Curvature	-0.065 **	-0.025
Attachment Anxiety x TEQ-Cortisol Level	0.053	-0.037
Attachment Anxiety x TEQ-Cortisol Rate of Change	0.003	-0.029 **
Attachment Anxiety x TEQ-Cortisol Curvature	-0.013	0.009
Cortisol Level-SIB	-0.318 **	-0.318 **
Cortisol Rate of Change-SIB	0.077	0.077
Cortisol Curvature-SIB	-0.543 ***	-0.543 ***
Model 3c - Constrained Attachment Model		
TEQ-SIB	0.236 ***	0.236 ***
TEQ-Cortisol Level	0.024	0.021
TEQ-Cortisol Rate of Change	-0.005	-0.016
TEQ-Cortisol Curvature	-0.010	0.005
Attachment Anxiety-Cortisol Level	0.150 ***	0.150 ***
Attachment Anxiety-Cortisol Rate of Change	0.038 **	0.038 **
Attachment Anxiety-Cortisol Curvature	-0.041 **	-0.041 **
Attachment Anxiety x TEQ-Cortisol Level	0.052	-0.036
Attachment Anxiety x TEQ-Cortisol Rate of Change	0.004	-0.029 **
Attachment Anxiety x TEQ-Cortisol Curvature	-0.011	0.008
Cortisol Level-SIB	-0.318 **	-0.318 **
Cortisol Rate of Change-SIB	0.077	0.077
Cortisol Curvature-SIB	-0.543 ***	-0.543 ***

* $p < .10$, ** $p < .05$, *** $p < .01$

Table 10

Indirect, Direct, and Total Effects on SIB Score in the Trauma Factor Analyses

	Indirect	Direct		Total	
Model 5 - Baseline Mediation Model					
Men's Trauma Factor Score	-0.003	0.187	***	0.183	***
Women's Trauma Factor Score	0.002	0.394	***	0.396	***
Model 5c - Constrained Mediation Model					
Men's Trauma Factor Score	-0.001	0.184	***	0.183	***
Women's Trauma Factor Score	0.004	0.393	***	0.396	***
Model 6 - Baseline Attachment Model					
Men's Trauma Factor Score	0.001	0.184	***	0.185	***
Women's Trauma Factor Score	0.008	0.393	***	0.400	***
Men's Attachment Anxiety	-0.010			-0.010	
Women's Attachment Anxiety	-0.023			-0.023	
Men's Attachment Avoidance	-0.002			-0.002	
Women's Attachment Avoidance	0.001			0.001	
Men's Attachment Avoidance x Trauma Factor	0.011			0.011	
Women's Attachment Avoidance x Trauma Factor	-0.001			-0.001	
Model 6c - Constrained Attachment Model					
Men's Trauma Factor Score	0.002	0.184	***	0.186	***
Women's Trauma Factor Score	0.007	0.393	***	0.400	***
Men's Attachment Anxiety	-0.019			-0.019	
Women's Attachment Anxiety	-0.019			-0.019	
Men's Attachment Avoidance	-0.002			-0.002	
Women's Attachment Avoidance	-0.002			-0.002	
Men's Attachment Avoidance x Trauma Factor	0.010			0.010	
Women's Attachment Avoidance x Trauma Factor	-0.001			-0.001	

* $p < .10$, ** $p < .05$, *** $p < .01$

Table 11

Model Comparisons for Trauma Factor Analyses

	MFF $\chi 2$	*df*	$\Delta\chi 2$	Δdf	*p*-value	Reject H_0?
Model 5 (Baseline)	181.73	29				
Model 5a	193.31	33	11.58	4	0.02	yes
Model 5b	189.14	30	7.41	1	0.01	yes
Model 5c	185.87	32	4.14	3	0.25	no
Model 6 (Baseline)	220.25	62				
Model 6a	230.85	71	10.60	9	0.30	no
Model 6b	227.63	65	7.38	3	0.06	yes
Model 6c	223.28	68	3.03	6	0.81	no

Note. MFF $\chi 2$ = Minimum Fit Function Chi-Square. Within each group of models, the baseline constrained model was compared to each of the constrained models that follow. The null hypothesis states that the baseline model does not fit better than the constrained model.

Table 12

Coefficients for the Direct Effects in the Trauma Factor Attachment Models

Path	Men's Coefficient	Women's Coefficient
Model 6 - Baseline Trauma Factor Attachment Model		
Trauma Factor-SIB	0.184 ***	0.393 ***
Trauma Factor-Cortisol Level	-0.021	-0.015
Trauma Factor-Cortisol Rate of Change	-0.001	-0.014
Trauma Factor-Cortisol Curvature	0.010	-0.008
Attachment Anxiety-Cortisol Level	0.156 ***	0.123 **
Attachment Anxiety-Cortisol Rate of Change	0.040 *	0.041 **
Attachment Anxiety-Cortisol Curvature	-0.063 **	-0.017
Attachment Avoidance-Cortisol Level	0.055	0.006
Attachment Avoidance-Cortisol Rate of Change	-0.006	-0.042
Attachment Avoidance-Cortisol Curvature	-0.026	-0.007
Attachment Avoidance x Trauma Factor-Cortisol Level	-0.076 **	<.001
Attachment Avoidance x Trauma Factor-Cortisol Rate of Change	0.028 *	-0.017
Attachment Avoidance x Trauma Factor-Cortisol Curvature	0.021	0.001
Cortisol Level-SIB	-0.266 **	-0.266 **
Cortisol Rate of Change-SIB	0.026	0.026
Cortisol Curvature-SIB	-0.488 **	-0.488 **
Model 6c - Constrained Trauma Factor Attachment Model		
Trauma Factor-SIB	0.184 ***	0.393 ***
Trauma Factor-Cortisol Level	-0.017	-0.020
Trauma Factor-Cortisol Rate of Change	0.001	-0.015
Trauma Factor-Cortisol Curvature	0.005	-0.005
Attachment Anxiety-Cortisol Level	0.139 ***	0.139 ***
Attachment Anxiety-Cortisol Rate of Change	0.040 **	0.040 **
Attachment Anxiety-Cortisol Curvature	-0.034 *	-0.034 *
Attachment Avoidance-Cortisol Level	0.030	0.030
Attachment Avoidance-Cortisol Rate of Change	-0.027	-0.027
Attachment Avoidance-Cortisol Curvature	-0.013	-0.013
Attachment Avoidance x Trauma Factor-Cortisol Level	-0.075 **	-0.001
Attachment Avoidance x Trauma Factor-Cortisol Rate of Change	0.030 **	-0.017
Attachment Avoidance x Trauma Factor-Cortisol Curvature	0.021	0.002
Cortisol Level-SIB	-0.266 **	-0.266 **
Cortisol Rate of Change-SIB	0.026	0.026
Cortisol Curvature-SIB	-0.488 **	-0.488 **

* p <.10, ** p <.05, *** p <.01

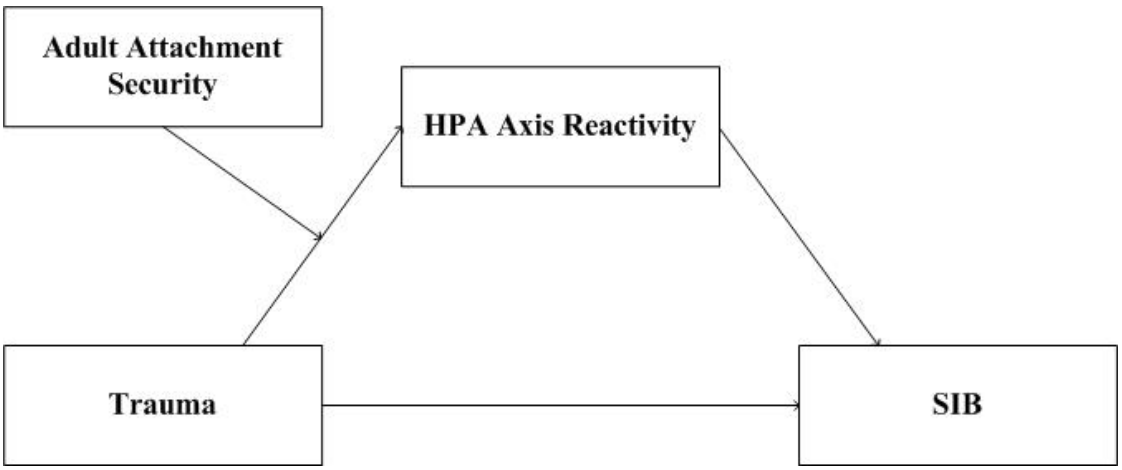

Figure 1. Conceptual diagram of the proposed mediation model with moderation by attachment.

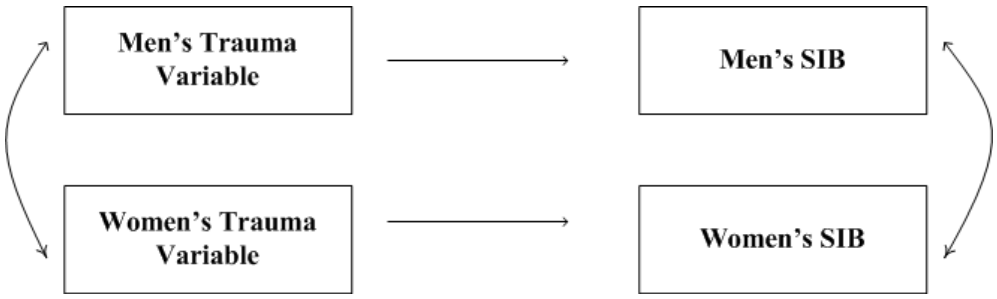

Figure 2. Path analysis evaluating the direct effects of the trauma variable on SIB accounting for the interdependence of the dyadic data.

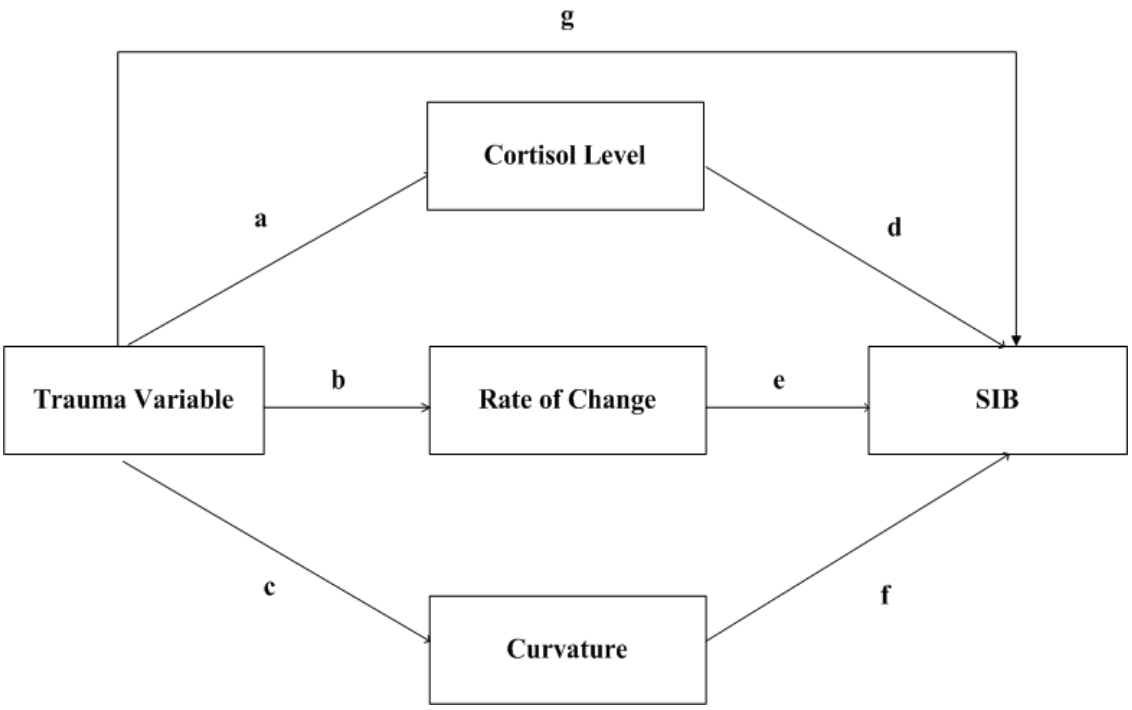

Figure 3. Conceptual model of the path analysis testing the direct and indirect (mediating) effects of the trauma variable on SIB.

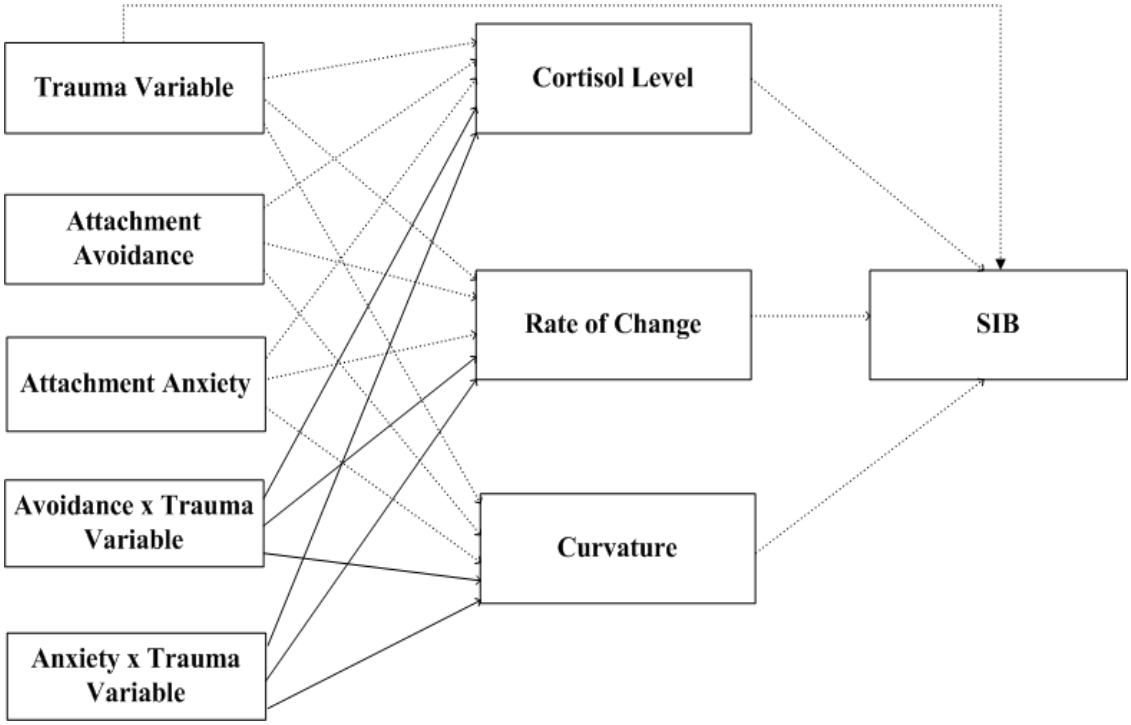

Figure 4. Conceptual diagram displaying how the attachment moderation hypotheses were evaluated. The solid lines show the paths that are directly relevant to the attachment moderation hypothesis. The dotted lines show the others paths that were modeled that are not directly relevant to the attachment moderation hypotheses.

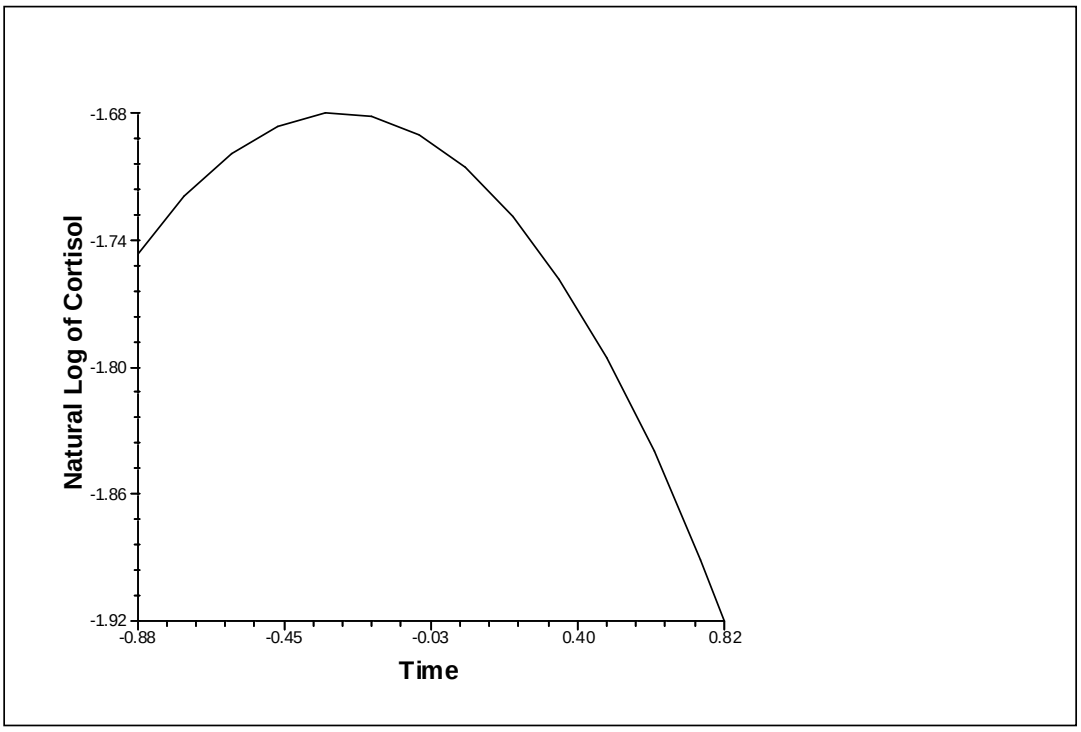

Figure 5. Graph of the women's average cortisol trajectory. Note that the discussion is occurring between -.10 and .25.

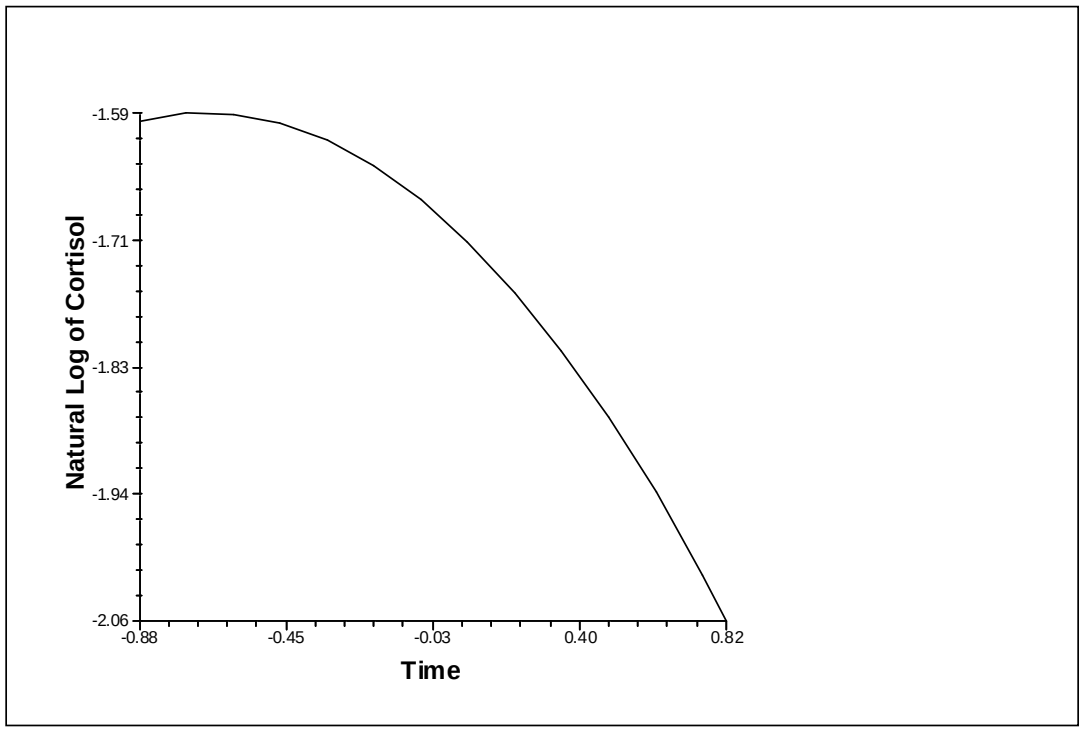

Figure 6. Graph of the men's average cortisol trajectory. Note that the discussion is occurring between -.10 and .25.

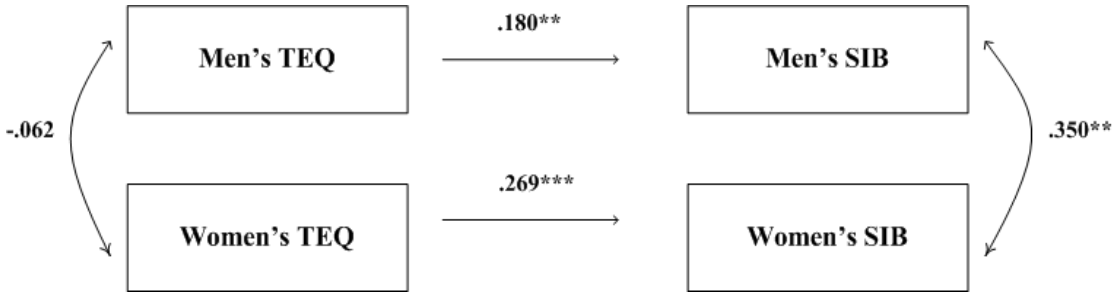

Figure 7. Model 1 - Direct effects of TEQ on SIB.
* p <.10, ** p <.05, *** p <.01

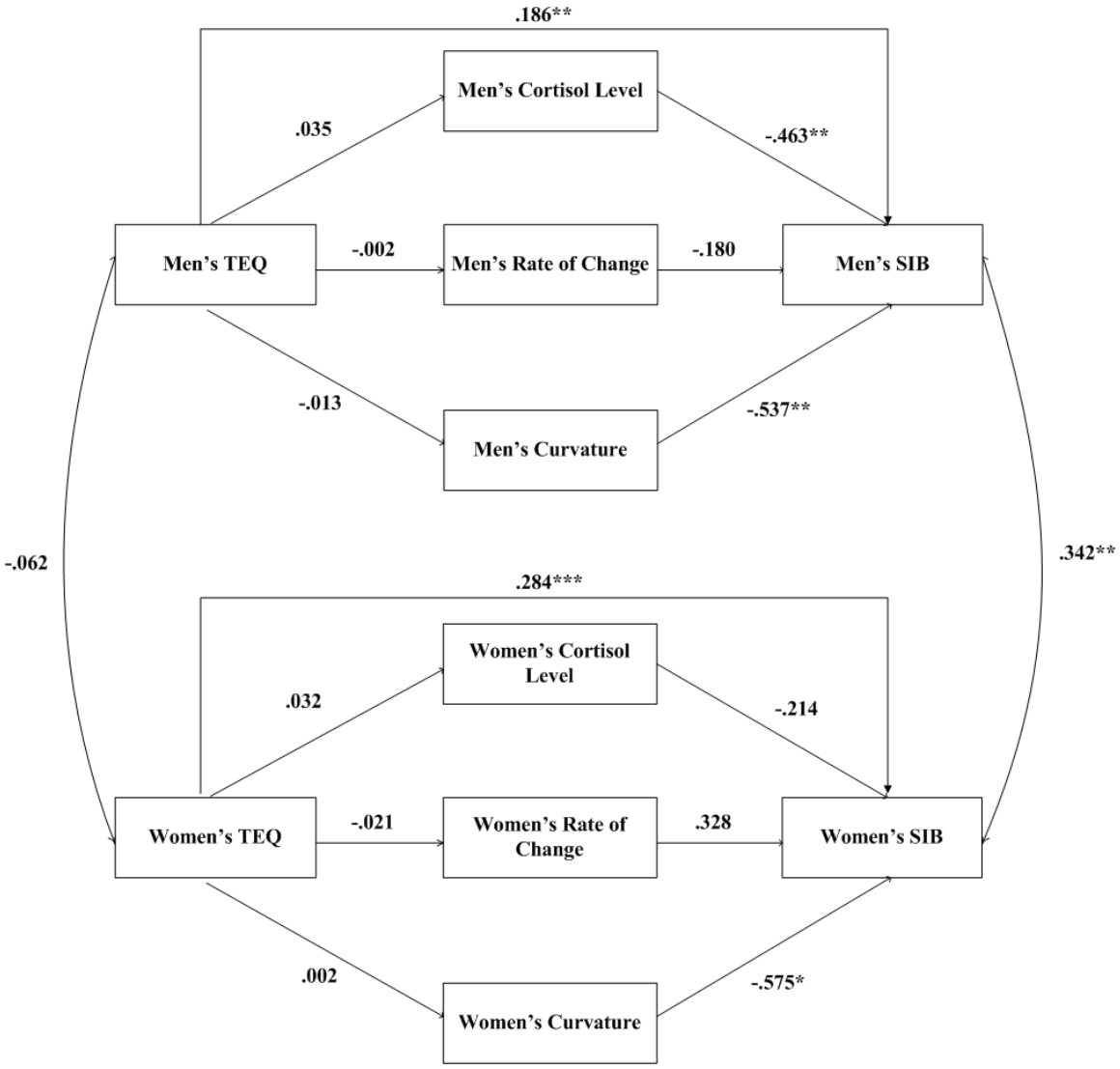

Figure 8. Model 2 – Baseline mediation model with TEQ as the main predictor.
* p <.10, ** p <.05, *** p <.01

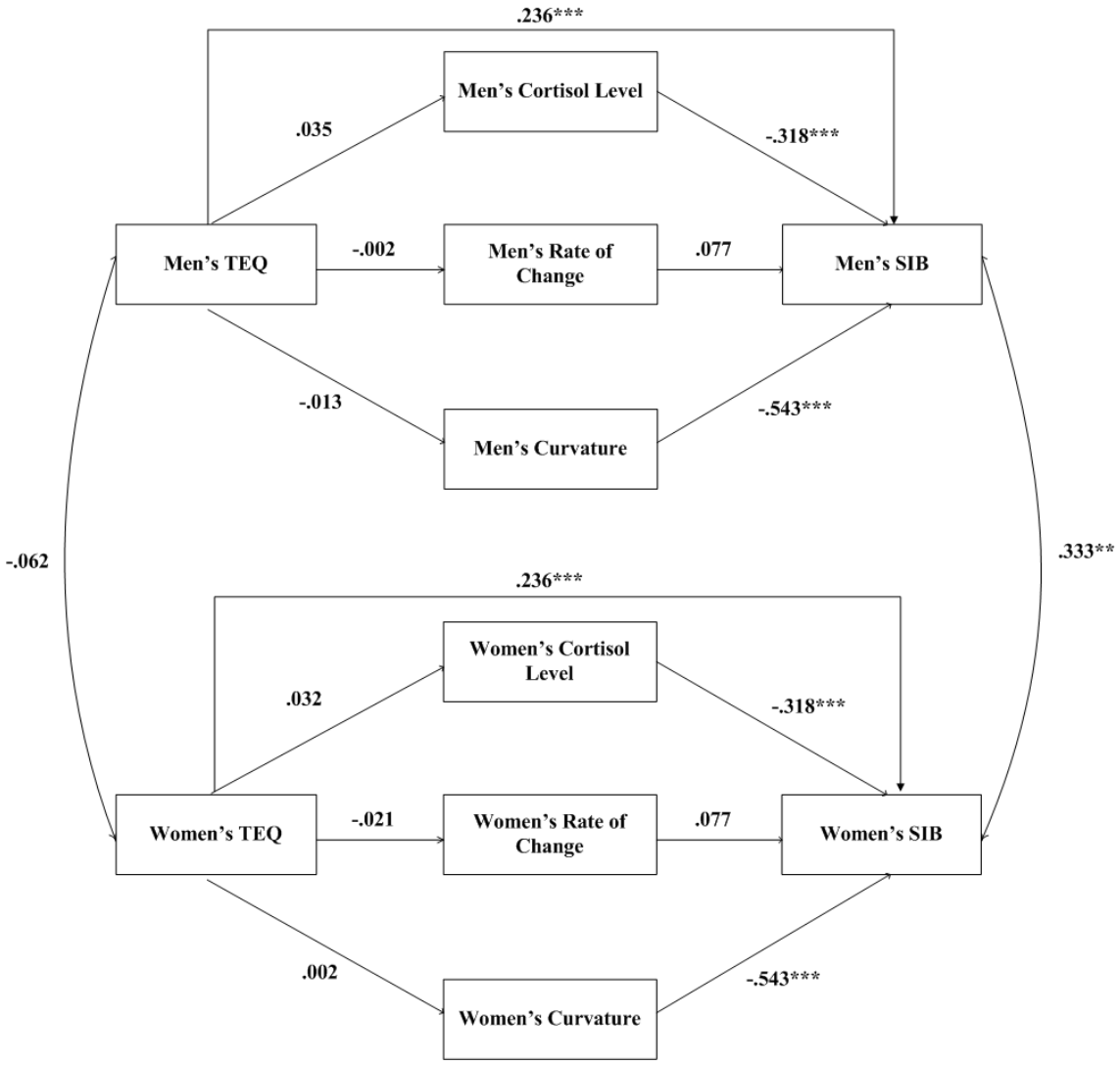

Figure 9. Model 2a – Constrained mediation model with TEQ as the main predictor. In this model, the paths from TEQ-SIB and Cortisol Trajectory-SIB were constrained to be equal for men and women.
* p <.10, ** p <.05, *** p <.01

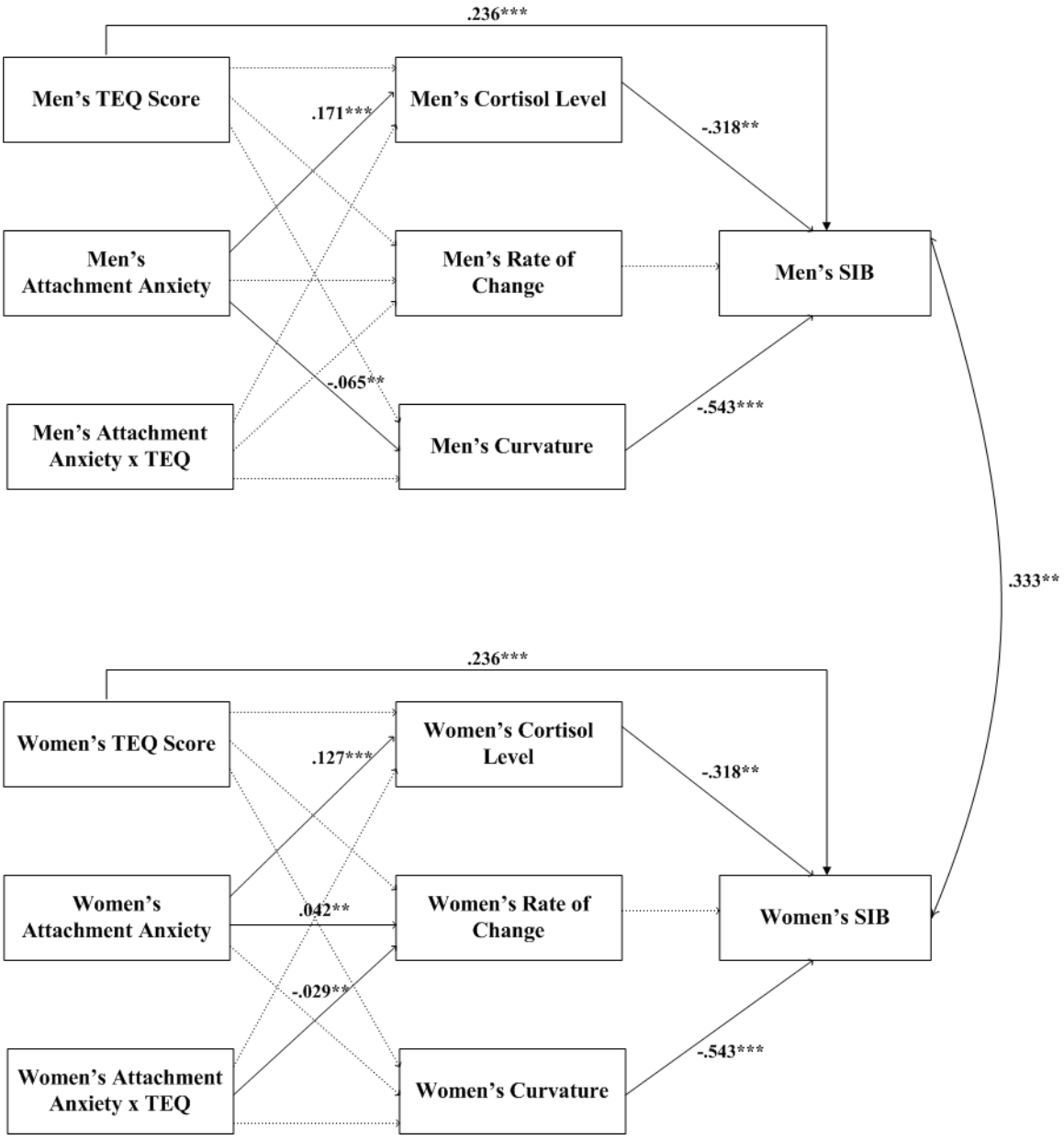

Figure 10. Model 3 - Baseline attachment moderation model. The main effects and interaction effects of attachment anxiety were freely estimated for men and women. The paths from TEQ-SIB and from Cortisol Trajectory-SIB were constrained to be equal for men and women. Only the coefficients for the paths that reached statistical significance at $p < .10$ were included. Paths depicted in dashed lines represent paths that were modeled, but that did not reach statistical significance. Although not depicted, paths were specified correlating all of the exogenous variables with each of the other exogenous variables.
* $p < .10$, ** $p < .05$, *** $p < .01$

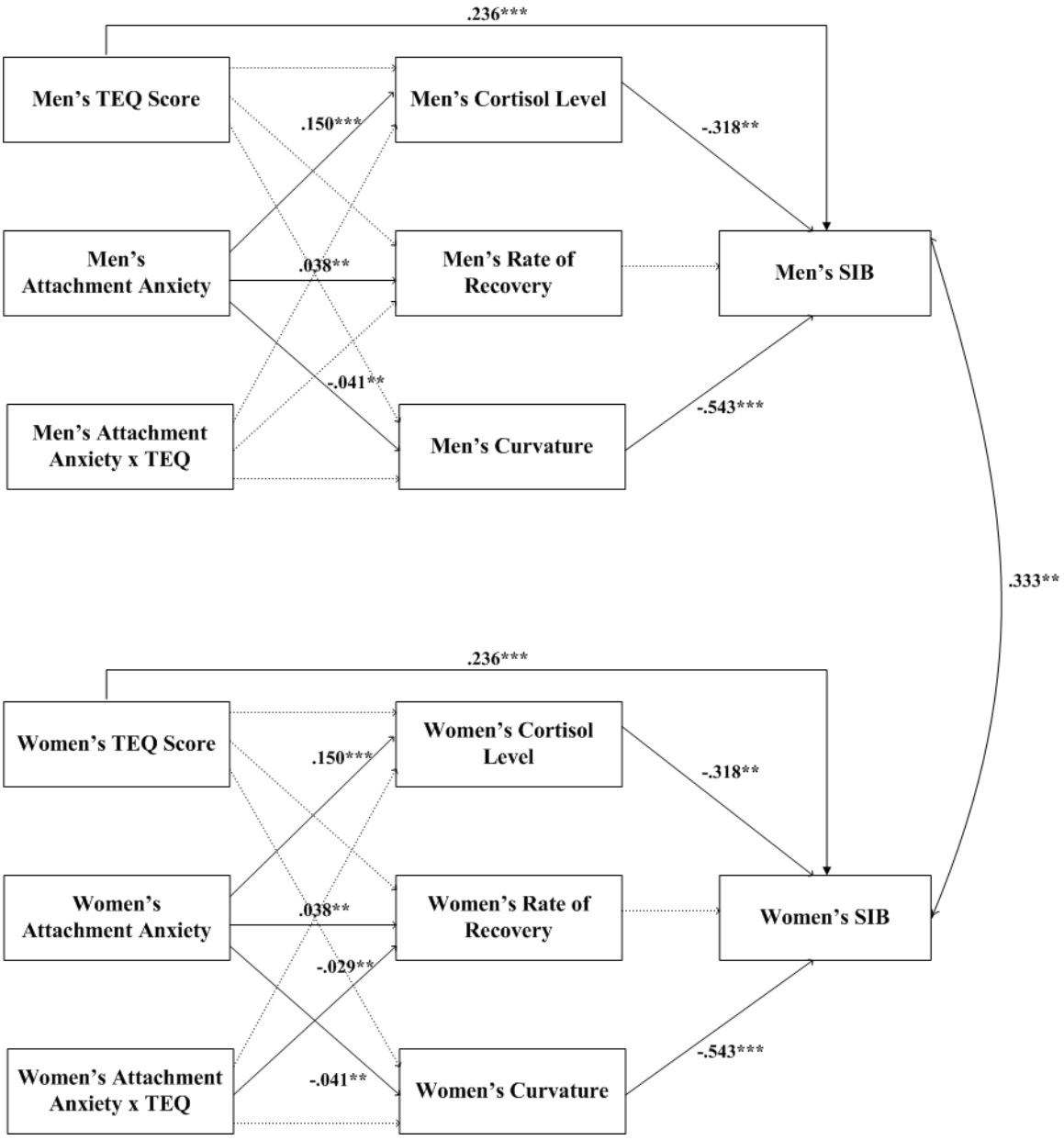

Figure 11. Model 3c – Constrained attachment moderation model with TEQ as the main predictor. The main effects of attachment anxiety were constrained to be equal between men and women. The paths from TEQ-SIB and from Cortisol Trajectory-SIB were constrained to be equal for men and women. Only the coefficients for the paths that reached statistical significance at $p < .10$ were included. Paths depicted in dashed lines represent paths that were modeled, but that did not reach statistical significance. Although not depicted, paths were specified correlating all of the exogenous variables with each of the other exogenous variables.
* $p < .10$, ** $p < .05$, *** $p < .01$

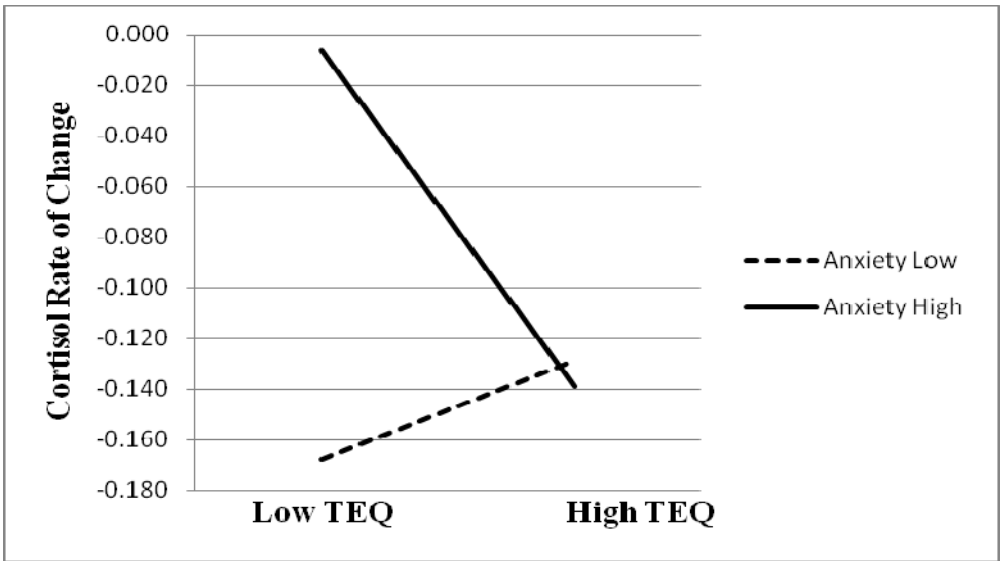

Figure 12. Association of women's TEQ score and cortisol rate of change plotted at two levels of attachment anxiety. Note that "low" levels of attachment anxiety and TEQ score refer to values 1 *SD* below the mean and "high" levels of attachment anxiety and TEQ score refer to values 1 *SD* above the mean of each variable.

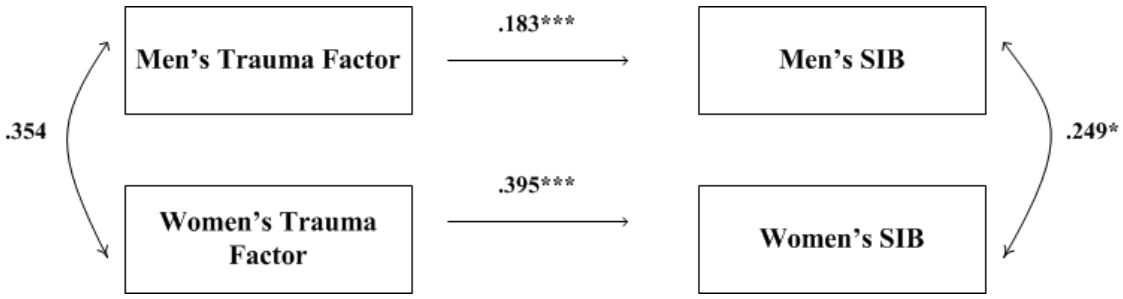

Figure 13: Model 4 - Direct effects of trauma factor score on SIB.
* p <.10, ** p <.05, *** p <.01

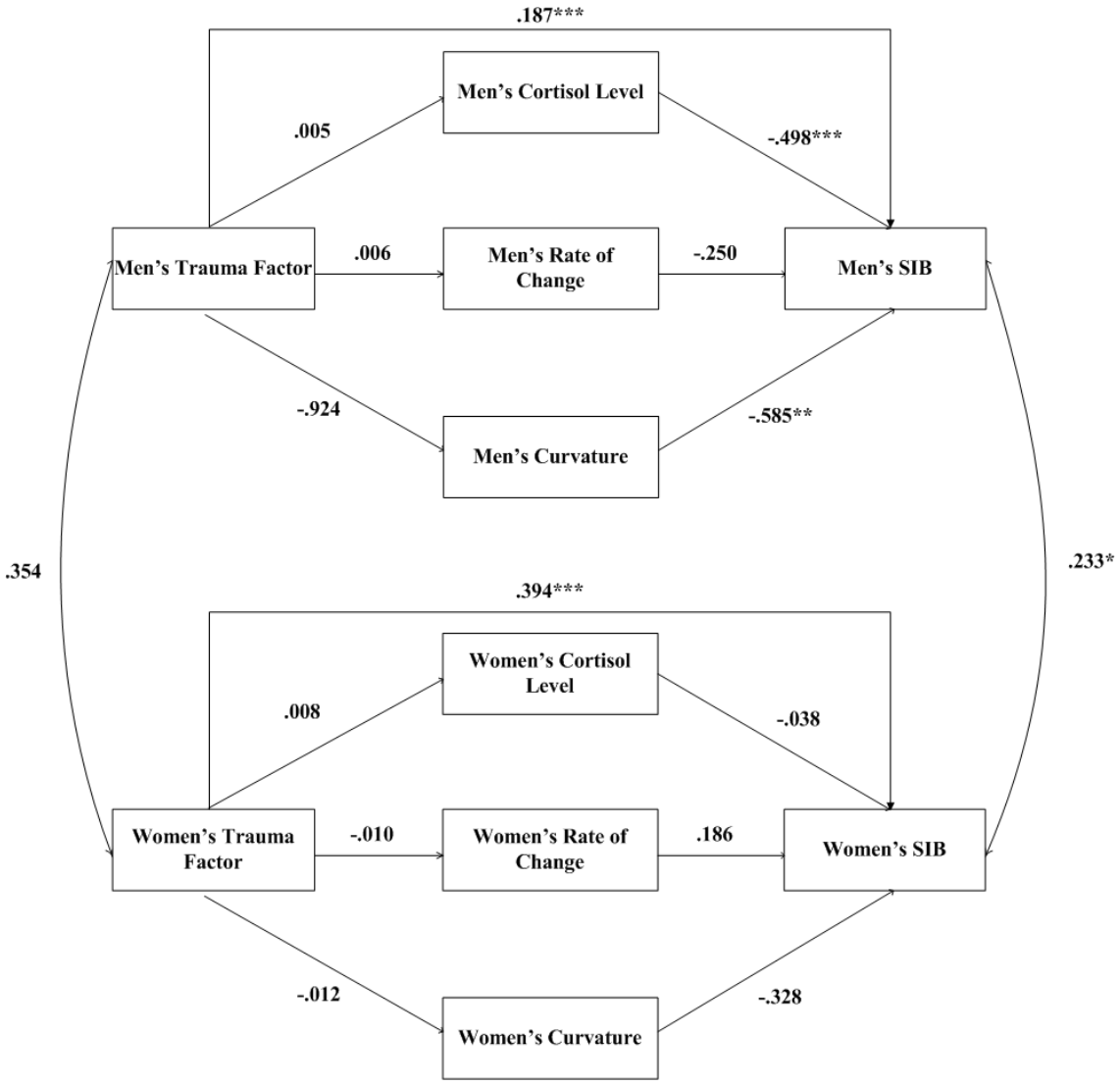

Figure 14. Model 5 – Baseline mediation model with trauma factor score as the main predictor.
* p <.10, ** p <.05, *** p <.01

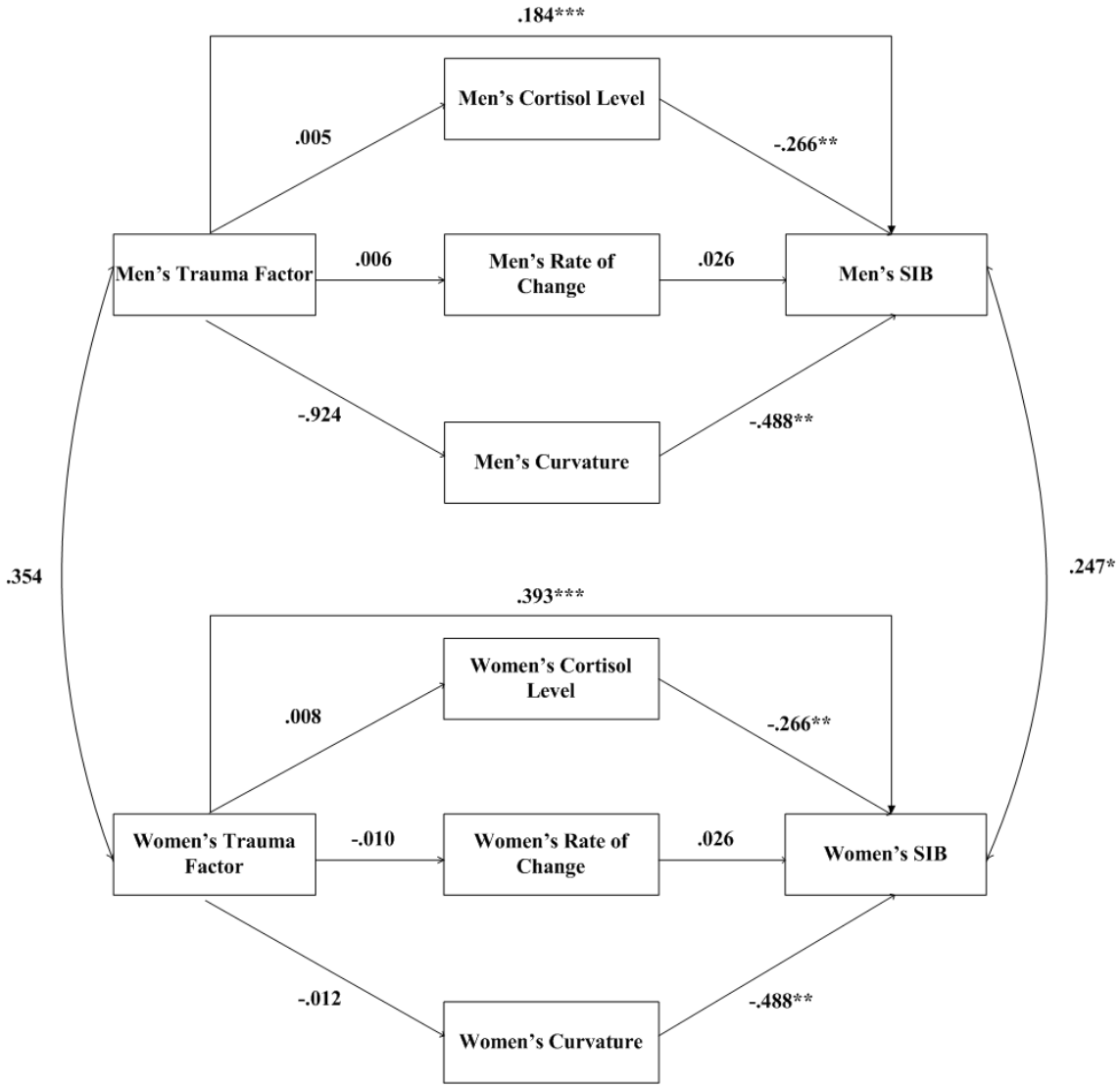

Figure 15. Model 5c – Constrained mediation model with trauma factor score as the main predictor. In this model, the paths from Cortisol Trajectory-SIB were constrained to be equal for men and women.
* p <.10, ** p <.05, *** p <.01

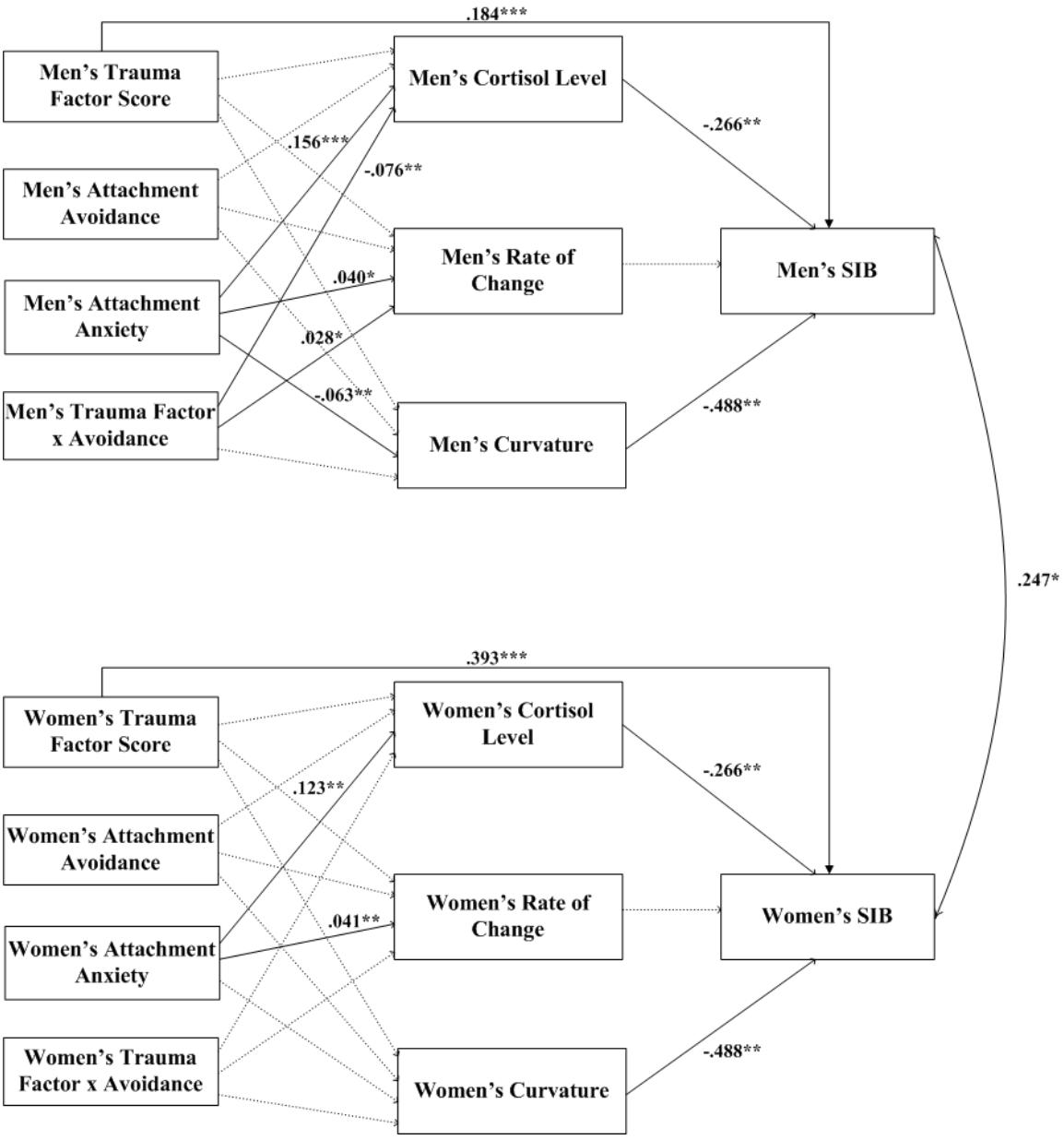

Figure 16. Model 6 - Baseline attachment moderation model with trauma factor score as the main predictor. In this model, all of the attachment main and interaction effects were freely estimated for men and women. The paths from Cortisol Trajectory-SIB were constrained to be equal for men and women. Only the coefficients for the paths that reached statistical significance at $p < .10$ were included. Paths depicted in dashed lines represent paths that were modeled, but that did not reach statistical significance. Although not depicted, paths were specified correlating all of the exogenous variables with each of the other exogenous variables.
* $p < .10$, ** $p < .05$, *** $p < .01$

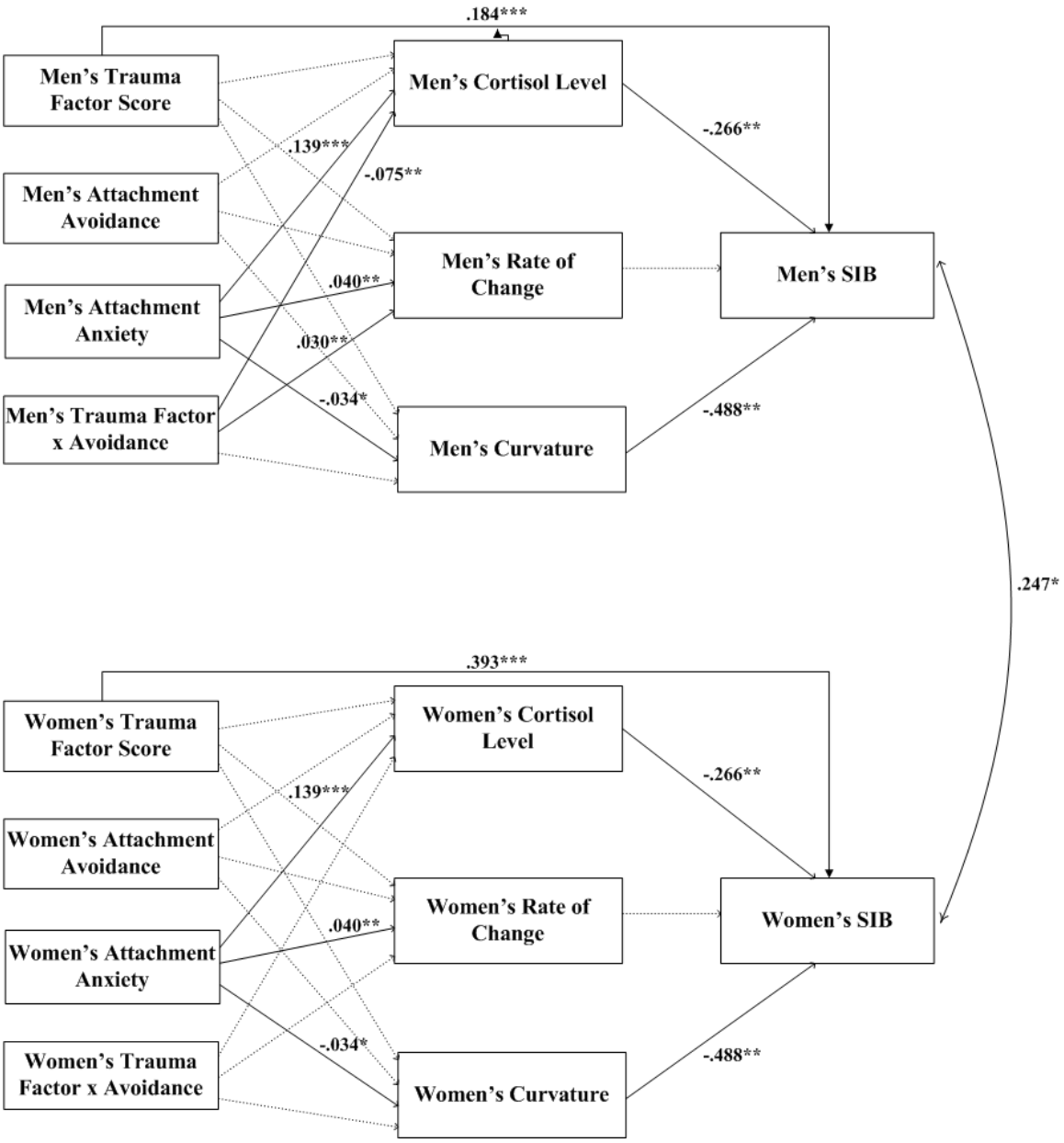

Figure 17: Model 6c – Constrained attachment moderation model with trauma factor score as the main predictor. The main effects of attachment avoidance and anxiety were constrained to be equal for men and women. The paths from Cortisol Trajectory-SIB were also constrained to be equal for men and women. Only the coefficients for the paths that reached statistical significance at $p < .10$ were included. Paths depicted in dashed lines represent paths that were modeled, but that did not reach statistical significance. Although not depicted, paths were specified correlating all of the exogenous variables with each of the other exogenous variables.
* $p < .10$, ** $p < .05$, *** $p < .01$

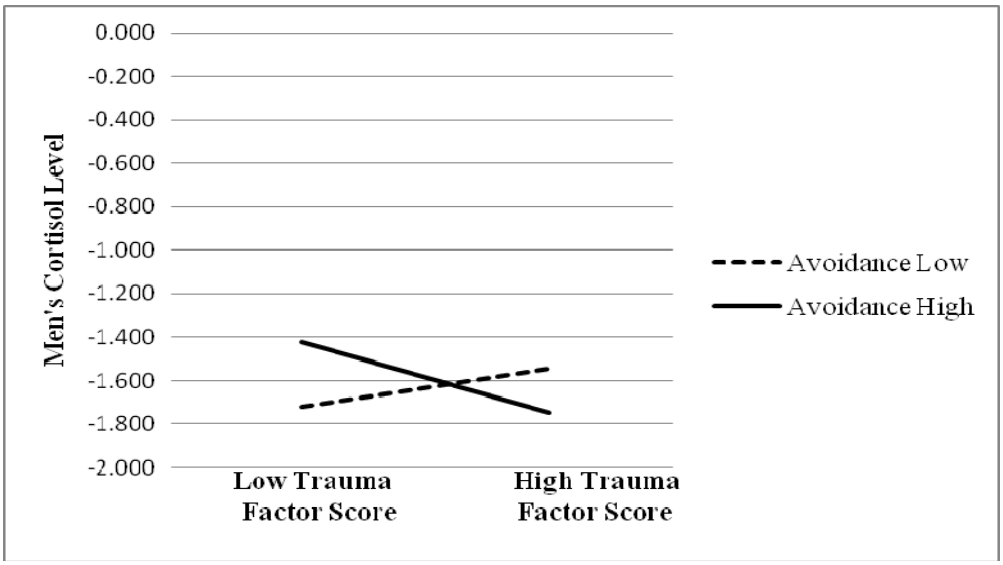

Figure 18. Association of men's trauma factor score and cortisol level plotted at two levels of attachment avoidance. Note that "low" levels of attachment avoidance and trauma factor score refer to values 1 *SD* below the mean and "high" levels refer to values 1 *SD* above the mean of each variable.

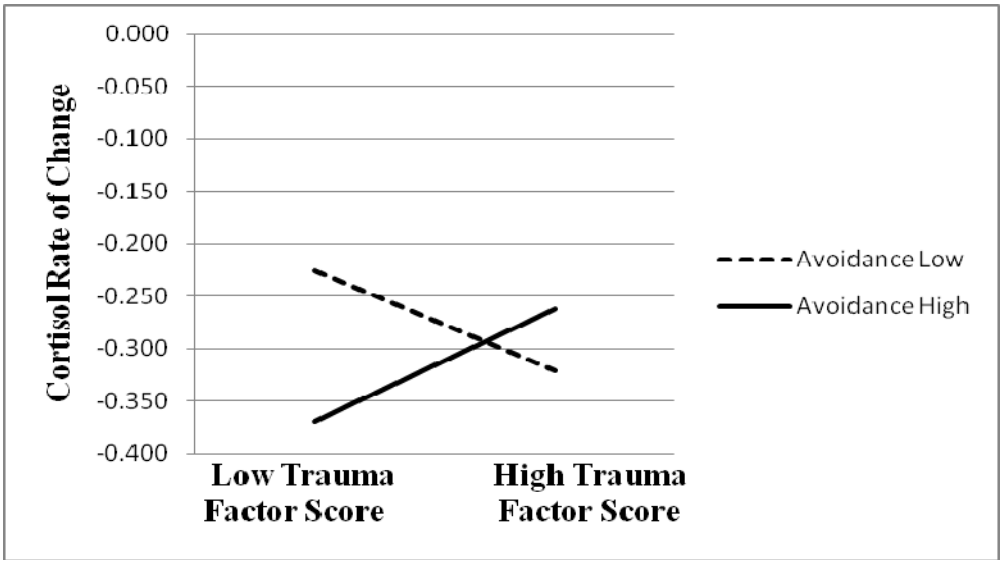

Figure 19. Association of men's trauma factor score and cortisol rate of change plotted at two levels of attachment avoidance. Note that "low" levels of attachment avoidance and trauma factor score refer to values 1 *SD* below the mean and "high" levels refer to values 1 *SD* above the mean of each variable.

APPENDIX A

ADMISSION QUESTIONNAIRE

Please answer the following questions about yourself. Please be honest. There are no right or wrong answers. Your information will be kept completely anonymous and confidential. Please circle ALL that apply.

A. What medications did you take today?

				dose (mgs.)
1)	Antibiotics	yes	no	
2)	The pill	yes	no	
3)	Aspirin	yes	no	
4)	Advil/Tylenol	yes	no	
5)	Cold medicine	yes	no	
6)	Allergy medicine	yes	no	
7)	Asthma medicine	yes	no	
8)	Norpramin/Pertofrane (Desipramine)	yes	no	
9)	Adapin/Sinequan (Doxepin)	yes	no	
10)	Anafranil (Chloripramine)	yes	no	
11)	Tofranil (Imipramine)			
12)	Aventyl/Pamelor (Nortriptyline)	yes	no	
13)	Triptil/Vivactil (Protriptyline)	yes	no	
14)	Surmontil (Trimipramine)	yes	no	
15)	Manerix (Moclobemide)	yes	no	
16)	Nardil (Pheneizine)	yes	no	
17)	Parnate (Tranylcypromine)	yes	no	
18)	Prozac (Fluvoxetine)	yes	no	
19)	Luvox (Fluvoxamine)	yes	no	
20)	Pazil (Paroxetine)	yes	no	
21)	Zoloft (Sertraline)	yes	no	
22)	Asendin (Amoxapine)	yes	no	
23)	Wellbutrin (Bupropion)	yes	no	
24)	Ludiomil (Maprotiline)	yes	no	
25)	Remeron (Mirtazapine)	yes	no	
26)	Serzone (Nefazodone)	yes	no	
27)	Desyrel (Trazodone)	yes	no	

What medications did you take today?

				dose (mgs.)
28)	Effexor (Venlafaxine)	yes	no	
29)	Tegretol (Carbamazepine)	yes	no	
30)	Depakene/Depakote/Epival (Phenytion, Primidone, Valproic Acid	yes	no	
31)	Eskalith/Lithane/Lithobid (Lithium)	yes	no	
32)	Inapsine (Droperidol)	yes	no	
33)	Haldol (Haloperidol)	yes	no	
34)	Loxapac/Loxitane (Loxapine)	yes	no	
35)	Moban (Molindone)	yes	no	
36)	Imap (Fluspirilene)	yes	no	
37)	Orap (Pimozide)	yes	no	
38)	Largactil/Thorazine (Chlorpromazine)	yes	no	
39)	Moditen/Permitil/Prolixin (Fluphenazine)	yes	no	
40)	Serentil (Mesoridazine)	yes	no	
41)	Nozinan (Methotrimeprazine)	yes	no	
42)	Neuleptil (Percyazine)	yes	no	
43)	Trilafon (Perphenazine)	yes	no	
44)	Piportil L4 (Pipotiazine)	yes	no	
45)	Compazine/Stemetil (Prochlorperazine)	yes	no	
46)	Sparine (Promazine)	yes	no	
47)	Majeptil (Thioproperazine)	yes	no	
48)	Mellaril (Thioridazine)	yes	no	
49)	Stelazine (Trifluoperazine)	yes	no	
50)	Vesprin (Triflupromazine)	yes	no	
51)	Fluanxol (Flupenthixol)	yes	no	
52)	Navane (Thiothixene)	yes	no	
53)	Clopixol (Zuclopenthixol)	yes	no	
54)	Clozaril (Clozapine)	yes	no	
55)	Zyprexa (Olanzapine)	yes	no	
56)	Risperdal (Risperidone)	yes	no	
57)	OTHER	yes	no	

2) Have you been taking any of the above medication on a daily basis for the past two weeks, but did not take today? yes no

a. If yes, what medication? _____

3) Did you smoke any cigarettes today? yes no
   a. If yes, how long ago did you have your last cigarette? ____mins

4) Did you brush your teeth in the last three hours? yes no

5) When you brushed your teeth today, did your gums bleed? yes no

6) In the past 24 hours, have you had dental work? yes no

7) In the past 24 hours, have you experienced any injury to your mouth, such as burning your mouth or tongue, cutting your mouth or lip, having a sore tooth, any irritation of blisters on your mouth or lips? yes no

8) In the past 24 hours, have you drunk any alcohol? yes no

9) In the past 30 minutes, have you had any dairy products? yes no

10) Did you use any other drugs (marijuana, cocaine, etc.) today? yes no

11) When did you eat or drink last? _____am/pm

12) Did you drink alcohol or take any non-prescription drugs last night? yes no
   a. If yes, please describe _____

13) Record temperature _____

14) (For women only) Use calendar to fill out DAY AND LENGTH OF LAST PERIORD
   IF CURRENTLY, WHEN BEGAN _____

15) What time did you fall asleep last night? _____

16) What time did you wake up today? _____

17) How many hours of sleep have you gotten in the past 24 hours?

APPENDIX B

SELF INJURIOUS BEHAVIOR QUESTIONNAIRE (SIB-Q)

Sometimes people engage in behaviors that are harmful to their bodies. These behaviors are sometimes accidental, and sometimes intentional. Please answer these questions with respect to intentional behavior. Please indicate *when* was the last time you engaged in such behavior, as well as the frequency with which the behavior has occurred over your lifetime. If "never" please choose this option.

1. Have you ever engaged in any behavior that was *deliberately* harmful to your body? (i.e. you harmed yourself *on purpose*.)

Most recent Time:
a) Never
b) in the past week
c) in the past month
d) in past 6 months
e) in the past year
f) over 1 year ago (within 5 years)
g) over 5 years ago

Frequency:
a) None
b) one time in my life
c) between 2-5 times in my life
d) between 6-10 times in my life
e) between 11-20 times in my life
f) over 20 times in my life

2. Have you ever intentionally engaged in behavior that produced bruising?

Most recent Time:
a) Never
b) in the past week
c) in the past month
d) in past 6 months
e) in the past year
f) over 1 year ago (within 5 years)
g) over 5 years ago

Frequency:
a) None
b) one time in my life
c) between 2-5 times in my life
d) between 6-10 times in my life
e) between 11-20 times in my life
f) over 20 times in my life

3. Have you ever deliberately hit yourself?

Most recent Time:
a) Never
b) in the past week
c) in the past month
d) in past 6 months
e) in the past year
f) over 1 year ago (within 5 years)
g) over 5 years ago

Frequency:
a) None
b) one time in my life
c) between 2-5 times in my life
d) between 6-10 times in my life
e) between 11-20 times in my life
f) over 20 times in my life

4. Have you ever intentionally pulled out your hair or eyelashes?

Most recent Time:
a) Never
b) in the past week
c) in the past month
d) in past 6 months
e) in the past year
f) over 1 year ago (within 5 years)
g) over 5 years ago

Frequency:
a) None
b) one time in my life
c) between 2-5 times in my life
d) between 6-10 times in my life
e) between 11-20 times in my life
f) over 20 times in my life

5. Have you ever purposely scratched yourself with fingernails or other objects hard enough to leave marks or cause bleeding?

Most recent Time:
a) Never
b) in the past week
c) in the past month
d) in past 6 months
e) in the past year
f) over 1 year ago (within 5 years)
g) over 5 years ago

Frequency:
a) None
b) one time in my life
c) between 2-5 times in my life
d) between 6-10 times in my life
e) between 11-20 times in my life
f) over 20 times in my life

6. Have you ever deliberately bit yourself hard enough to leave marks?

Most recent Time:
a) Never
b) in the past week
c) in the past month
d) in past 6 months
e) in the past year
f) over 1 year ago (within 5 years)
g) over 5 years ago

Frequency:
a) None
b) one time in my life
c) between 2-5 times in my life
d) between 6-10 times in my life
e) between 11-20 times in my life
f) over 20 times in my life

7. Have you ever purposely eaten toxic substances or sharp objects?

Most recent Time:
a) Never
b) in the past week
c) in the past month
d) in past 6 months
e) in the past year
f) over 1 year ago (within 5 years)
g) over 5 years ago

Frequency:
a) None
b) one time in my life
c) between 2-5 times in my life
d) between 6-10 times in my life
e) between 11-20 times in my life
f) over 20 times in my life

8. Have you ever intentionally burned yourself with a lit cigarette, match, or other?

Most recent Time:
a) Never
b) in the past week
c) in the past month
d) in past 6 months
e) in the past year
f) over 1 year ago (within 5 years)
g) over 5 years ago

Frequency:
a) None
b) one time in my life
c) between 2-5 times in my life
d) between 6-10 times in my life
e) between 11-20 times in my life
f) over 20 times in my life

9. Have you ever purposely cut or gouged yourself with a razor blade, broken glass, or other?

Most recent Time:
a) Never
b) in the past week
c) in the past month
d) in past 6 months
e) in the past year
f) over 1 year ago (within 5 years)
g) over 5 years ago

Frequency:
a) None
b) one time in my life
c) between 2-5 times in my life
d) between 6-10 times in my life
e) between 11-20 times in my life
f) over 20 times in my life

10. Are there other self-injurious behaviors that you have engaged in that were not listed?

YES NO

11. If you responded "yes" to question #10, when and with what frequency did you engaged in the self-injurious behavior?

Most recent Time:
a) Never
b) in the past week
c) in the past month
d) in past 6 months
e) in the past year
f) over 1 year ago (within 5 years)
g) over 5 years ago

Frequency:
a) None
b) one time in my life
c) between 2-5 times in my life
d) between 6-10 times in my life
e) between 11-20 times in my life
f) over 20 times in my life

12. If applicable, how often do you hide your bruises, wounds, or scars from others?
a. never
b. about 25% of the time
c. about 50% of the time
d. about 75% of the time
e. always

13. If applicable, how often do you exhibit your bruises, wounds, or scars to others?
    a. never
    b. about 25% of the time
    c. about 50% of the time
    d. about 75% of the time
    e. always

14. If applicable: Are there specific occasions, situations, or feelings that cause you to want or need to engage in self-injurious behaviors?

15. If applicable: To the right is a list of potential reasons for engaging in self-injurious behaviors. Please choose up to four reasons that currently apply to you or applied to you while you were engaging in self-injurious behaviors.

    a. for the excitement/rush
    b. to distract from painful feelings or thoughts
    c. to deal with physical instead of mental pain
    d. to distract from memories
    e. to show the pain inside
    f. to see blood
    g. to get a reaction from others
    h. to express anger or frustration at others
    i. to punish myself
    j. to express anger or frustration at myself
    k. to bring myself back to reality
    l. to reduce tension or anxiety
    m. to escape from reality
    n. to deal with feelings of loneliness
    o. to gain control over my body
    p. to re-enact events from the past
    q. suicide attempt
    r. instead of suicide
    s. to get help or care
    t. because my friends do it
    u. I don't know why

16. Are there reasons why you engage in self-injurious behaviors that were not listed? If so, please briefly describe two of them.

APPENDIX C

TRAUMA EXPERIENCES QUESTIONNAIRE (TEQ)

This questionnaire is comprised of a variety of traumatic events which you may have experienced. For each of the following "numbered" questions, indicate whether or not you have experienced the event. **If you have experienced one of the events**, indicate "YES" and complete the items in the box immediately following it that ask for more details. **If you have not experienced the event**, indicate "NO" and go to the next "numbered" item after the box. 1 = YES 2 = NO

250. Have you been in or witnessed a serious industrial, farm, or car accident, or a large fire or explosion? 1 = YES 2 = NO

IF you answered No to #250, please skip to NEXT PAGE

251. How many times? 1 = once 2 = twice 3 = three or more

252. How old were you at the time(s)?

1 = age 3 or younger
2 = age 4-6
3 = age 7-10
4 = age 11 -13
5 = age 14-16
6 = age 17-19
7 = age 20-22
8 = older than 22

253. Were you injured?

Not at all						Severely
1	2	3	4	5	6	7

254. Did you feel your life was threatened?

Not at all						Severely
1	2	3	4	5	6	7

255. How traumatic was this event for you at the time?

Not at all						Severely
1	2	3	4	5	6	7

256. How traumatic is this event for you now?

Not at all						Severely
1	2	3	4	5	6	7

257. What was the event? _____

258. Have you been in a natural disaster such as a tornado, hurricane, flood, or major earthquake? 1 = YES 2 = NO

If you answered No to #258, please skip to NEXT PAGE

259. How many times? 1 = once 2 = twice 3 = three or more

260. How old were you at the time(s)?

1 = age 3 or younger
2 = age 4-6
3 = age 7-10
4 = age 11-13
5 = age 14-16
6 = age 17-19
7 = age 20-22
8 = older than 22

261. Were you injured?

Not at all						Severely
1	2	3	4	5	6	7

262. Did you feel your life was threatened?

Not at all						Severely
1	2	3	4	5	6	7

263. How traumatic was this event for you at the time?

Not at all						Severely
1	2	3	4	5	6	7

264. How traumatic <u>is</u> this event for you now?

Not at all						Severely
1	2	3	4	5	6	7

265. What was the event? _____

266. Have you been a victim of a violent crime such as rape, robbery, or assault?
1 = YES 2 = NO

If you answered No to #266, please skip to NEXT PAGE

267. How many times? 1 = once 2 = twice 3 = three or more

268. How old were you at the time(s)?

1 = age 3 or younger
2 = age 4-6
3 = age 7-10
4 = age 11 -13
5 = age 14-16
6 = age 17-19
7 = age 20-22
8 = older than 22

269. Were you injured?

Not at all						Severely
1	2	3	4	5	6	7

270. Did you feel your life was threatened?

Not at all						Severely
1	2	3	4	5	6	7

271. How traumatic was this event for you at the time?

Not at all						Severely
1	2	3	4	5	6	7

272. How traumatic <u>is</u> this event for you now?

Not at all						Severely
1	2	3	4	5	6	7

273. What was the event? _____

274. As a child, were the victim of either physical or sexual abuse?
1 = YES 2 = NO

If you answered No to #274, please skip to NEXT PAGE

275. How old were you when it began?___
1 = age 3 or younger
2 = age 4-6
3 = age 7-10
4 = age 11 -13
5 = age 14-16
6 = age 17-19
7 = age 20-22
8 = older than 22

276. How old were you when it ended? ___
1 = age 3 or younger
2 = age 4-6
3 = age 7-10
4 = age 11 -13
5 = age 14-16
6 = age 17-19
7 = age 20-22
8 = older than 22

277. Were you injured?

Not at all						Severely
1	2	3	4	5	6	7

278. Did you feel your life was threatened?

Not at all						Severely
1	2	3	4	5	6	7

279. How traumatic was this event for you at the time?

Not at all						Severely
1	2	3	4	5	6	7

280. How traumatic is this event for you now?

Not at all						Severely
1	2	3	4	5	6	7

281. Was the assailant male or female? 1 = Male 2 = Female

****Indicate all categories that describe the experience with either 1 = YES or 2 = NO**

282. physical abuse 1 = YES 2 = NO

283. sexual penetration of the mouth, anus, or vagina 1 = YES 2 = NO

284. no sexual penetration, but the assailant attempted to force you to complete such an act 1 = YES 2 = NO

285. some other form of sexual contact (e.g., touch your sexual organs or were forced to touch assailant's sexual organs) 1 = YES 2 = NO

286. no sexual contact occurred, however, the assailant attempted to touch your sexual organs, or tried to make you touch his/her sexual organs 1 = YES 2 = NO

287. As an adult, have you had any unwanted sexual experiences that involved the threat or use of force? 1 = YES 2 = NO

If you answered No to #287, please skip to NEXT PAGE

288. How old were you when it began? ____

1 = age 3 or younger
2 = age 4-6
3 = age 7-10
4 = age 11 -13
5 = age 14-16
6 = age 17-19
7 = age 20-22
8 = older than 22

289. How old were you when it ended? ___

1 = age 3 or younger
2 = age 4-6
3 = age 7-10
4 = age 11 -13
5 = age 14-16
6 = age 17-19
7 = age 20-22
8 = older than 22

290. Were you injured?

Not at all						Severely
1	2	3	4	5	6	7

291. Did you feel your life was threatened?

Not at all						Severely
1	2	3	4	5	6	7

292. How traumatic was this event for you at the time?

Not at all						Severely
1	2	3	4	5	6	7

293. How traumatic is this event for you now?

Not at all						Severely
1	2	3	4	5	6	7

294. Was the assailant male or female? 1 = Male 2 = Female

****Indicate all categories that describe the experience with either 1 = YES or 2 = NO**

295. physical abuse 1 = YES 2 = NO

296. sexual penetration of the mouth, anus, or vagina 1 = YES 2 = NO

297. no sexual penetration, but the assailant attempted to force you to complete such an act 1 = YES 2 = NO

298. some other form of sexual contact (e.g., touch your sexual organs or were forced to touch assailant's sexual organs) 1 = YES 2 = NO

299. no sexual contact occurred, however, the assailant attempted to touch your sexual organs, or tried to make you touch his/her sexual organs

1 = YES 2 = NO

300. As an adult, have you ever been in a relationship in which you were abused either physically or otherwise? 1 = YES 2 = NO

If you answered NO to #300, please skip to NEXT PAGE.

301. How old were you when it began? ____

1 = age 3 or younger
2 = age 4-6
3 = age 7-10
4 = age 11 -13
5 = age 14-16
6 = age 17-19
7 = age 20-22
8 = older than 22

302. How old were you when it ended? ___

1 = age 3 or younger
2 = age 4-6
3 = age 7-10
4 = age 11 -13
5 = age 14-16
6 = age 17-19
7 = age 20-22
8 = older than 22

303. Were you injured?

Not at all						Severely
1	2	3	4	5	6	7

304. Did you feel your life was threatened?

Not at all						Severely
1	2	3	4	5	6	7

305. How traumatic was this event for you at the time?

Not at all						Severely
1	2	3	4	5	6	7

306. How traumatic is this event for you now?

Not at all						Severely
1	2	3	4	5	6	7

307. Have you witnessed someone being mutilated, seriously injured, or violently killed? 1 = YES 2 = NO

If you answered no to #307, please skip to NEXT PAGE.

308. How many times? 1 = once 2 = twice 3 = three or more

309. How old were you at the time(s)?
1 = age 3 or younger
2 = age 4-6
3 = age 7-10
4 = age 11 -13
5 = age 14-16
6 = age 17-19
7 = age 20-22
8 = older than 22

310. Were you injured?

Not at all						Severely
1	2	3	4	5	6	7

311. Did you feel your life was threatened?

Not at all						Severely
1	2	3	4	5	6	7

312. How traumatic <u>was</u> this event for you at the time?

Not at all						Severely
1	2	3	4	5	6	7

313. How traumatic <u>is</u> this event for you now?

Not at all						Severely
1	2	3	4	5	6	7

314. Have you ever been in serious danger of losing your life or of being seriously injured?

1 = YES 2 = NO

If you answered no to #314, please skip to NEXT PAGE.

315. How many times? 1 = once 2 = twice 3 = three or more

316. How old were you at the time(s)?

1 = age 3 or younger
2 = age 4-6
3 = age 7-10
4 = age 11 -13
5 = age 14-16
6 = age 17-19
7 = age 20-22
8 = older than 22

317. Were you injured?

Not at all						Severely
1	2	3	4	5	6	7

318. Did you feel your life was threatened?

Not at all						Severely
1	2	3	4	5	6	7

319. How traumatic <u>was</u> this event for you at the time?

Not at all						Severely
1	2	3	4	5	6	7

320. How traumatic <u>is</u> this event for you now?

Not at all						Severely
1	2	3	4	5	6	7

321. What was the event? _____

322. Have you received news of the mutilation, serious injury, or violent or unexpected death of someone close to you? 1 = YES 2 = NO

If you answered no to #322, please skip to NEXT PAGE.

323. How many times? 1 = once 2 = twice 3 = three or more

324. How old were you at the time(s)?
1 = age 3 or younger
2 = age 4-6
3 = age 7-10
4 = age 11 -13
5 = age 14-16
6 = age 17-19
7 = age 20-22
8 = older than 22

325. What relation was this person to you? _____

326. Did you feel your life was threatened?

Not at all						Severely
1	2	3	4	5	6	7

327. How traumatic <u>was</u> this event for you at the time?

Not at all						Severely
1	2	3	4	5	6	7

328. How traumatic <u>is</u> this event for you now?

Not at all						Severely
1	2	3	4	5	6	7

329. Have you ever had any other very traumatic event like these?
1 = YES 2 = NO

If you answered no to #329, please skip to NEXT PAGE.

330. How many times? 1 = once 2 = twice 3 = three or more

331. How old were you at the time(s)?
1 = age 3 or younger
2 = age 4-6
3 = age 7-10
4 = age 11 -13
5 = age 14-16
6 = age 17-19
7 = age 20-22
8 = older than 22

332. Were you injured?

Not at all						Severely
1	2	3	4	5	6	7

333. Did you feel your life was threatened?

Not at all						Severely
1	2	3	4	5	6	7

334. How traumatic was this event for you at the time?

Not at all						Severely
1	2	3	4	5	6	7

335. How traumatic is this event for you now?

Not at all						Severely
1	2	3	4	5	6	7

336. What was the event? _____

337. Have you had any experiences like these that you feel you can't tell about? (NOTE: you don't have to describe the event.)

1 = YES 2 = NO

If you answered NO to #337, please GO TO QUESTIONS AT END OF PAGE

338. How many times? 1 = once 2 = twice 3 = three or more

339. How old were you at the time(s)?

1 = age 3 or younger
2 = age 4-6
3 = age 7-10
4 = age 11 -13
5 = age 14-16
6 = age 17-19
7 = age 20-22
8 = older than 22

340. Were you injured?

Not at all						Severely
1	2	3	4	5	6	7

341. Did you feel your life was threatened?

Not at all						Severely
1	2	3	4	5	6	7

342. How traumatic <u>was</u> this event for you at the time?

Not at all						Severely
1	2	3	4	5	6	7

343. How traumatic <u>is</u> this event for you now?

Not at all						Severely
1	2	3	4	5	6	7

344. **<u>If you answered "YES" to one or more of the questions above</u>,** which was the <u>MOST</u> traumatic thing to have happened to you? Fill in the number of the question (e.g., #442 for natural disaster): _____

345. Did you answer YES to more than one question above while thinking about the same event?

YES___ NO__

346. If yes, which items refer to the same event?_____

****

<u>If you answered "NO" to all questions</u>, describe briefly the most traumatic thing to happen to you: (Otherwise, **skip and answer #353).**

_____

---

347. How many times? once__ twice__ three or more__

348. How old were you at the time(s)?

1 = age 3 or younger
2 = age 4-6
3 = age 7-10
4 = age 11 -13
5 = age 14-16
6 = age 17-19
7 = age 20-22
8 = older than 22

349. Were you injured?

Not at all						Severely
1	2	3	4	5	6	7

350. Did you feel your life was threatened?

Not at all						Severely
1	2	3	4	5	6	7

351. How traumatic <u>was</u> this event for you at the time?

Not at all						Severely
1	2	3	4	5	6	7

352. How traumatic <u>is</u> this event for you now?

Not at all						Severely
1	2	3	4	5	6	7

APPENDIX D

TRAUMA SYMPTOM CHECKLIST-40 (TSC-40)

How often have you experienced each of the following in the **last two months?**

	Never			Often
1) Headaches	0	1	2	3
2) Insomnia (trouble getting to sleep)	0	1	2	3
3) Weight loss (without dieting)	0	1	2	3
4) Stomach problems	0	1	2	3
5) Sexual problems	0	1	2	3
6) Feeling isolated from others	0	1	2	3
7) "Flashbacks" (sudden, vivid, distracting memories)	0	1	2	3
8) Restless sleep	0	1	2	3
9) Low sex drive	0	1	2	3
10) Anxiety attacks	0	1	2	3
11) Sexual overactivity	0	1	2	3
12) Loneliness	0	1	2	3
13) Nightmares	0	1	2	3
14) "Spacing out" (going away in your mind)	0	1	2	3
15) Sadness	0	1	2	3
16) Dizziness	0	1	2	3
17) Not feeling satisfied with your sex life	0	1	2	3
18) Trouble controlling your temper	0	1	2	3

	Never			Often
19) Waking up early in the morning and can't get back to sleep	0	1	2	3
20) Uncontrollable crying	0	1	2	3
21) Fear of men	0	1	2	3
22) Not feeling rested in the morning	0	1	2	3
23) Having sex that you didn't enjoy	0	1	2	3
24) Trouble getting along with others	0	1	2	3
25) Memory problems	0	1	2	3
26) Desire to physically hurt yourself	0	1	2	3
27) Fear of women	0	1	2	3
28) Waking up in the middle of the night	0	1	2	3
29) Bad thoughts or feelings during sex	0	1	2	3
30) Passing out	0	1	2	3
31) Feeling that things are "unreal"	0	1	2	3
32) Unnecessary or over-frequent washing	0	1	2	3
33) Feelings of inferiority	0	1	2	3
34) Feeling tense all the time	0	1	2	3
35) Being confused about your sexual feelings	0	1	2	3
36) Desire to physically hurt others	0	1	2	3
37) Feelings of guilt	0	1	2	3
38) Feelings that you are not always in your body	0	1	2	3
39) Having trouble breathing	0	1	2	3
40) Sexual feelings when you shouldn't have them	0	1	2	3

APPENDIX E

EXPERIENCES IN CLOSE RELATIONSHIPS SCALE

The following statements concern how you feel in romantic relationships. We are interested in how you generally experience relationships, not just in what is happening in a current relationship. Respond to each statement by indicating how much you agree or disagree with it. Write the number in the space provided, using the following rating scale:

1	2	3	4	5	6	7
Disagree Strongly	**…….**	**…….**	**Neutral / Mixed**	**…….**	**…….**	**Agree Strongly**

____ **1** I prefer not to show a partner how I feel deep down

____ **2** I worry about being abandoned

____ **3** I am very uncomfortable being close to romantic partners

____ **4** I worry a lot about my relationships

____ **5** Just when my partner starts to get close to me I find myself pulling away

____ **6** I worry that romantic partners won't care about me as much as I care about them

____ **7** I get uncomfortable when a romantic partner wants to be very close

____ **8** I worry a fair amount about losing my partner

____ **9** I don't feel comfortable opening up to romantic partners

____ **10** I often wish that my partner's feelings for me were as strong as my feelings for him/her

____ **11** I want to get close to my partner, but I keep pulling back

____ **12** I often want to merge completely with romantic partners, and this sometimes scares them away

____ **13** I am nervous when partners get too close to me

____ **14** I worry about being alone

____ **15** I feel comfortable sharing my private thoughts and feelings with my partner

____ **16** I My desire to be very close sometimes scares people away

____ **17** I try to avoid getting too close to my partner

____ **18** I need a lot of reassurance that I am loved by my partner

____ **19** I find it relatively easy to get close to my partner

____ **20** Sometimes I feel that I force my partners to show more feeling, more commitment

____ **21** I find it difficult to allow myself to depend on romantic partners

____ **22** I do not often worry about being abandoned

____ **23** I prefer not to be too close to romantic partners

____ **24** If I can't get my partner to show interest in me, I get upset or angry

____ **25** I tell my partner just about everything

____ **26** I find that my partner(s) don't want to get as close as I would like

____ **27** I usually discuss my problems and concerns with my partner

____ **28** When I'm not involved in a relationship, I feel somewhat anxious and insecure

____ **29** I feel comfortable depending on romantic partners

____ **30** I get frustrated when my partner is not around as much as I would like

____ **31** I don't mind asking romantic partners for comfort, advice, or help

____ **32** I get frustrated if romantic partners are not available when I need them

____ **33** It helps to turn to my romantic partner in times of need

____ **34** When romantic partners disapprove of me, I feel really bad about myself

____ **35** I turn to my partner for many things, including comfort and reassurance

____ **36** I resent it when my partner spends time away from me

REFERENCE LIST

Baker, D. G., West, S. A., Nicholson, W. E., Ekhator, N. N., Kasckow, J. W., Hill, K. K., et al. (1999). Serial CSF corticotropin-releasing hormone levels and adrenocortiocal activity in combat veterans with posttraumatic stress disorder. *American Journal of Psychiatry, 156,* 585-588.

Boscarino, J. A. (1996). Posttraumatic stress disorder, exposure to combat, and lower plasma cortisol among Vietnam veterans: Findings and clinical implications. *Journal of Consulting and Clinical Psychology, 64*(1), 191-201.

Boudewyn, A. C., & Liem, J. H. (1995). Childhood sexual abuse as a precursor to depression and self-destructive behavior in adulthood. *Journal of Traumatic Stress, 8*(30), 445-459.

Brain, K. L., Haines, J., & Williams, C. L. (1998). The psychophysiology of self-mutilation: Evidence of tension reduction. *Archives of Suicide Research, 4,* 227−242.

Brennan, K. A., Clark, C. L., & Shaver, P. R. (1998). Self-report measurement of adult attachment: An integrative overview. In J. A. Simpson & W. S. Rholes (Eds.), *Attachment theory and close relationships* (pp. 46-76). New York: Guilford Press.

Briere, J., & Gil, E. (1998). Self-mutilation in clinical and general population samples: Prevalence, correlates, and functions. *American Journal of Orthopsychiatry, 68*(4), 609-620.

Briere, J. & Runtz, M. (1989). The trauma symptom checklist (TSC-40): Early data on a new scale. *Journal of Interpersonal Violence, 4,* 151-163.

Carroll, J., Schaffer, C., Spensley, J., & Ambramowitz, S. I. (1980). Family experiences of self-mutilating patients. *American Journal of Psychiatry, 137*(7), 852-853.

Chen, G. L., Novak, M. A., Meyer, J. S., Kelly, B. J., Vallender, E. J., & Miller, G. M. (2010). *TPH2* 5′- and 3′- regulatory polymorphisms are differentially associated with HPA axis function and self-injurious behavior in rhesus monkeys. *Genes, Brain, and Behavior,* 1-13.

Darche, M. A. (1990). Psychological factors differentiating self-mutilating and non-self-mutilating adolescent inpatient females. *The Psychiatric Hospital, 21*(1), 31-35.

de Kloet, C. S., Vermetten, E., Geuze, E., Kavelaars, A., Heijnen, C. J., & Westenberg, H. G. M. (2006). Assessment of HPA-axis function in posttraumatic stress disorder: Pharmacological and non-pharmacological challenge tests, a review. *Journal of Psychiatric Research, 40,* 550-567.

Davenport, M. D., Lutz, C. K., Tiefenbacher, S., Novak, M. A., & Meyer, J. S. (2008). A rhesus monkey model of self-injury: Effects of relocation stress on behavior and neuroendocrine function. *Biological Psychiatry, 63,* 990-996.

Diamond, L. M., Hicks, A. M. (2005). Attachment style, current relationship security, and negative emotions: The mediating role of physiological regulation. *Journal of Social and Personal Relationships, 22*(4), 499-518.

DiClemente, R. J., Ponton, L. E., & Hartley, D. (1991). Prevalence and correlates of cutting behavior: Risk for HIV transmission. *Journal of the American Academy of Child and Adolescent Psychiatry, 30*(5), 735-739.

Dubo, E. D., Zannarini, M. C., & Lewis, R. E. (1997). Childhood antecedents of self-destructiveness in borderline personality disorder. *Canadian Journal of Psychiatry, 42,* 63-69.

Favazza, A. R. (1996). *Bodies under siege.* Baltimore: The Johns Hopkins University Press.

Favazza, A. R. (1998). The coming of age of self-mutilation. *Journal of Nervous and Mental Disease, 186*(5), 259-268.

Favazza, A. R., & Conterio, K. (1988). The plight of chronic self-mutilators. *Community Mental Health Journal, 24*(1), 22-30.

Favazza, A. R., & Conterio, K. (1989). Female habitual self-mutilators. *Acta Psychiatrica Scandinavica, 79*(3), 283-289.

Favazza, A. R., & Rosenthal, R. J. (1993). Diagnostic issues in self-mutilation *Hospital and Community Psychiatry, 44*(2), 134-140.

Feeney, B. C., & Collins, N. L. (2003). Motivations for caregiving in adult intimate relationships: Influences on caregiving behavior and relationship functioning. *Personality and Social Psychology Bulletin, 29,* 950-968.

Feeney, B. C., & Kirkpatrick, L. A. (1996). The effects of adult attachment and presence of romantic partners on physiological responses to stress. *Journal of Personality and Social Psychology, 70,* 255-270.

Finkelhor, D., & Browne, A. (1984). The traumatic impact of child sexual abuse: A conceptualization. *American Journal of Orthopsychiatry, 55,* 530-541.

Flory, J. D., Yehuda, R., Grossman, R., New, A. S., Mitropoulou, V., & Siever, L. J. (2009). Childhood trauma and basal cortisol in people with personality disorders. *Comprehensive Psychiatry, 50,* 34-37.

Ghaziuddin, M., Tsai, L., Naylor, M., & Ghaziuddin, N. (1992). Mood disorder in a group of self-cutting adolescents. *Acta Paedopsychiatric, 55,* 103-105.

Gladstone, G. L., Parker, G. B., Mitchell, P. B., Malhi, G. S., Wilhelm, K., & Austin, M. (2004). Implications of childhood trauma for depressed women: An analysis of pathways from childhood sexual abuse to deliberate self-harm and revictimization. *American Journal of Psychiatry, 161*(8), 1417-1425.

Glover, D. D., & Poland, R. E. (2002). Urinary cortisol and catecholamines in mothers of child cancer survivors with and without PTSD. *Psychoneuroendocrinology, 27*(7), 805-819.

Gold, P. W., Goodwin, F. K., & Chrousos, G. P. (1988). Clinical and biochemical manifestations of depression. *New England Journal of Medicine, 319,* 413-420.

Gratz, K. L. (2006). Risk factors for deliberate self-harm among female college students: The role and interaction of childhood maltreatment, emotional inexpressivity, and affect intensity/reactivity. *American Journal of Orthopsychiatry, 76*(2), 238-250.

Gratz, K. L., & Chapman, A. L. (2007). The role of emotional responding and childhood maltreatment in the development and maintenance of deliberate self-harm among male undergraduates. *Psychology of Men and Masculinity, 8*(1), 1-14.

Gratz, K. L., Conrad, S. D., & Roemer, L. (2002). Risk factors for deliberate self-harm among college students. *American Journal of Orthopsychiatry,72*(1), 128-140.

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

Gunnar, M. R. (2000). Early adversity and the development of stress reactivity and regulation. In C. A. Nelson (Ed.), *The Minnesota Symposia on Child Psychology: Vol. 31. The Effects of Early Adversity on Neurobehavioral Development* (pp. 163-200). Mahwah, NJ: Lawrence Erlbaum Associates.

Haines, J., Williams, C. L., Brain, K. L., & Wilson, G. V. (1995). The psychophysiology of self-mutilation. *Journal of Abnormal Psychology, 104*(3), 471-489.

Harlow, H., & Harlow, M. (1962). The effect of rearing conditions on behavior. *Bulletin of the Menninger Clinic, 26,* 213-224.

Hayashi, G. M., & Strickland, B. R. (1998). Long-term effects of parental divorce on love relationships: Divorce as attachment disruption. *Journal of Social and Personal Relationships, 15,* 23-38.

Heinzer, M. M. (1995). Loss of a parent in childhood: Attachment and coping in a model of adolescent resilience. *Holistic Nursing Practice, 9,* 27-37.

Herpertz, S. (1995). Self-injurious behavior: Psychopathological and nosological characteristics in subtypes of self-injurers. *Acta Psychiatrica Scandinavica, 91*, 57-68.

Hetherington, E. M. (1989). Coping with family transitions: Winners, losers, and survivors. *Child Development, 60,* 1-14.

Jöreskog, K. & Sörbom, D. (2006). LISREL (Version 8.80) [Computer software]. Lincolnwood, IL: Scientific Software International.

Jorgensen, M. J., Kinsey, J. H., & Novak, M. A. (1998). Risk factors for self-injurious behavior in captive rhesus monkeys (*Macaca mulatta*). *American Journal of Primatology, 57,* 39-40.

Kemperman, I., Russ, M. J., & Shearin, E. (1997). Self-injurious behavior and mood regulation in borderline patients. *Journal of Personality Disorders, 11,* 146-157.

Kenny, D. A., Kashy, D. A. & Cook, W. L. (2006). *Dyadic data analysis*. New York, NY: Guilford Press.

Kiecolt-Glaser, J. K., Netwon, T., Cacioppo, J. T., MacCallum, R. C., Glaser, R., & Malarkey, W. B. (1996). Marital conflict and endocrine function: Are men really more physiologically affected than women? *Journal of Consulting and Clinical Psychology, 64*(2), 324-332.

Kirschbaum, C., & Helhammer, D. H. (1989). Salivary cortisol in psychobiological research: An overview. *Neuropsychobiology, 23*(3), 150-169.

Kirschbaum, C., Prussner, J. C., Stone, A. A., Federenko, I., Gaab, J. Lintz, D., et al. (1995). Persistent high cortisol responses to repeated psychological stress in a subpopulation of healthy men. *Psychosomatic Medicine, 57,* 468-474.

Klonsky, E. D. (2007). The functions of deliberate self-injury: A review of the evidence. *Clinical Psychology Review, 27,* 226-239.

Langbehn, D. R., & Pfohl, B. (1993). Clinical correlates of self-mutilation among psychiatric inpatients. *Annals of Clinical Psychiatry, 5,* 45-51.

Lutz, C. K., Chase, W. K., & Novak, M. A. (2000). Abnormal behavior in singly housed *Macaca mulatta*: Prevalence and risk factors. *American Journal of Primatology, 51,* 71.

Lutz, C. K., Davis, E. B., Ruggiero, A. M, & Suomi, S. (2007). Early predictors of self-biting in socially housed rhesus macaques (*Macaca mulatta*). *American Journal of Primatology, 69,* 584-590.

Lutz, C. K., Well, A., & Novak, M. (2003). Stereotypic and self-injurious behavior in rhesus macaques: A survey and retrospective analysis of environment and early experience. *American Journal of Primatology, 60,* 1-15.

Major, C. A., Kelly, B. J., Novak, M. A., Davenport, M. D., Stonemetz, K. M., & Meyer, J. S. (2009). The anxiogenic drug FG7142 increases self-injurious behavior in male rhesus monkeys (*Macaca mulatta). Life Sciences, 85,* 753-758.

Marinus, L. M., Chase, W. K., Rasmussen, K. L., Jorgensen, M. J., & Novak, M. A. (1999). Reaction of rhesus monkeys with self-injurious behavior to heart rate testing: Is biting a coping strategy? [Abstract]. *American Journal of Primatology, 49,* 79.

Martin, G., & Waite, S. (1994). Parental bonding and vulnerability to adolescent suicide. *Acta Psychiatrica Scandinavica, 89,* 246-254.

Mason, J. W., Giller, E. L., Kosten, T. R., Ostroff, R. B., & Podd, L. (1986). Urinary free-cortisol levels in posttraumatic stress disorder patients. *Journal of Nervous and Mental Disease, 174,* 145-59.

McArdle, E. T. (2004). HPA-axis reactivity to interpersonal stress in young adults who self-injure. (Doctoral Dissertation, University of Massachusetts Amherst, 2002). *Dissertation Abstracts International, 64*(10-B), 5226.

McFarlane, A. C. (1988). Recent life-events and psychiatric disorder in children: The interaction with preceding extreme adversity. *Journal of Clinical Psychiatry, 29*(5), 677-690.

Meewisse, M., Reitsma, J. B., De Vries, G., Gersons, B. P. R., & Olff, M. (2007). Cortisol and post-traumatic stress disorder in adults: Systematic review and meta-analysis. *British Journal of Psychiatry, 19,* 387-392.

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.

Miller, A. L., Rathus, J. H., & Linehan, M. M. (2007). *Dialectical behavior therapy with suicidal adolescents.* New York, NY: Guilford Press.

Muehlenkamp, J. J., & Gutierrez, P. M. (2004). An investigation of differences between self-injurious behavior and suicide attempts in a sample of adolescents. *Suicide and Life-Threatening Behavior, 34*(1), 12-23.

Nachmias, M., Gunnar, M., Mangelsdorf, S., Paritz, R. H., & Buss, K. (1996). Behavioral inhibition and stress reactivity: The moderating role of attachment security. *Child Development, 67,* 508-522.

Nixon, M. K., Cloutier, P. F., & Aggarwal, S. (2002). Affect regulation and addictive aspects of repetitive self-injury in hospitalized adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry, 41*(11), 1333-1341.

Novak, M. A. (2003). Self-injurious behavior in rhesus monkeys: New insights into its etiology, physiology, and treatment. *American Journal of Primatology, 59,* 3-19.

Pietromonaco, P. R., Greenwood, D., & Feldman Barrett, L. (2004). Conflict in adult close relationships: An attachment perspective. In W. S. Rholes & J. A. Simpson (Eds.), *Adult attachment: Theory, research, and clinical implications* (pp. 267-299). New York, NY: Guilford Press.

Pitman, R. K., & Orr, S. P. (1990). Twenty-four hour urinary cortisol and catecholamine excretion in combat-related posttraumatic stress disorder. *Biological Psychiatry, 27,* 245-247.

Powers, S. (2009, April). *Hormones and lover's quarrels: How stress translates into depression*. Distinguished Faculty Lecture, University of Massachusetts at Amherst.

Powers, S. I., Laurent, H. K., & Granger, D. (2010). *Individual coordination and dyadic attunement in HPA and SNS reactions to relationship conflict in dating couples.* Unpublished manuscript.

Powers. S. I., Pietromonaco, P. R., Gunlicks, M.m & Sayer, A. (2006). Dating couples' attachment styles and patterns of cortisol reactivity and recovery in response to a relationship conflict. *Journal of Personality and Social Psychology, 90*(4), 613-628.

Raudenbush, S., Bryk, A., Cheong, Y. F., Congdon, R, & du Toit, M. (2004). *HLM6: Hierarchical linear and nonlinear modeling.* Lincolnwood, IL: Scientific Software International, Inc.

Rayner, G., Allen, S., & Johnson, M. (2005). Countertransference and self-injury: A cognitive-behavioural cycle. *Journal of Advanced Nursing, 50*(1), 12-19.

Ross, S., & Heath, N. (2002). A study of the frequency of self-mutilation in a community sample of adolescents. *Journal of Youth and Adolescence, 31*(1), 67-77.

Rulf Fountain, A. (2001). Self-injurious behavior in university undergraduate students. (Doctoral Dissertation, University of Massachusetts Amherst, 2001). *Dissertation Abstracts International, 62*(1-B), 596.

Rutter, M. (1979). Protective factors in children's responses to stress and disadvantage. In M. W. Kent & J. E. Rolf (Eds.), *Primary prevention of psychopathology: Vol. 3. Social competence in children* (pp. 49–74). Hanover, NH: University Press of New England.

Sandman, C. A., Barron, J. L, Chicz-DeMet, A., & DeMet, E. M. (1990). Plasma β-endorphin levels in patients with self-injurious behavior and stereotypy. *American Journal on Mental Retardation, 95*(1)*,* 84-92.

Scarpa, A., & Raine, A. (1997). Psychophysiology of anger and violent behavior. *Psychiatric Clinics of North America, 20,* 375-394.

Scher, C. D., Forde, D. R., McQuaid, J. R., & Stein, M. B. (2004). Prevalence and demographic correlates of childhood maltreatment in an adult community sample. *Child Abuse and Neglect, 28,* 167-180.

Schore, A. N. (2002). Dysregulation of the right brain: A fundamental mechanism of traumatic attachment and the psychopathogenesis of posttraumatic stress disorder. *Australian and New Zealand Journal of Psychiatry, 36,* 9-30.

Seifer, R., Sameroff, A. J., Baldwin, C. P., & Baldwin, A. (1992). Child and family factors that ameliorate risk between 4 and 13 years of age. *Journal of the American Academy of Child & Adolescent Psychiatry, 31,* 893–903.

Spangler, G., Schieche, M., Ilg, U., Maier, U., & Ackermann, C. (1994). Maternal sensitivity as an external organizer for biobehavioral regulation in infancy, *Developmental Psychobiology, 27*(7), 425-437.

Stansbury, K., & Gunnar, M. R. (1994). Adrenocortical activity and emotion regulation. *Monographs of the Society for Research in Child Development, 59*(2-3), 250-283.

Stein, M. B., Yehuda, R., Koverola, C., & Hanna, C. (1997). Enhanced dexamethasone suppression of plasma cortisol in adult women traumatized by childhood sexual abuse. *Biological Psychiatry, 42,* 680-686.

Suomi, S. J. (1991). Up-tight and laid-back monkeys: Individual differences in response to social challenges. In S. Baruth, W. Hall, & R. Dooling (eds.), *Plasticity of development* (pp. 27-56). Cambridge, MA: MIT Press.

Suomi, S., Harlow, H., & Kimball, S. (1971). Behavioral effects of prolonged partial social isolation in the rhesus monkey. *Psychological Reports, 29,* 1171-1177.

Suyemoto, K. L. (1998). The functions of self-mutilation. *Clinical Psychology Review, 18,* 531-554.

Symons, F. J., Sutton, K. A., Walker, C., & Bodfish, J. W. (2003). Altered diurnal pattern of salivary substance P in adults with developmental disabilities and chronic self-injury. *American Journal on Mental Retardation, 108*(1), 13-18.

Tiefenbacher, S., Novak, M. A., Jorgensen, M. J., & Meyer, J. S. (2000). Physiological correlates of self-injurious behavior in captive, socially-reared rhesus monkeys. *Psychoneuroendocrinology, 25,* 799-817.

Tiefenbacher, S., Novak, M. A., Marinus, L. M., Chase, W. K., Miller, J. A., & Meyer, J. S. (2004). Altered hypothalamic-pituitary-adrenal function in rhesus monkeys (Macaca mulatta) with self-injurious behavior. *Psychoneuroendocrinology, 29,* 501-515.

van der Kolk, B. A. (1996a). The body keeps the score: Approaches to the psychobiology of posttraumatic stress disorder. In B. A. van der Kolk, A. C. McFarlane, & L. Weisaeth (Eds.), *Traumatic stress: The effects of overwhelming experience on mind, body, and society* (pp. 214-241). New York, NY: Guildford Press.

van der Kolk, B. A. (1996b). The complexity of adaptation to trauma: Self-regulation, stimulus discrimination, and characterological development. In B. A. van der Kolk, A. C. McFarlane, & L. Weisaeth (Eds.), *Traumatic stress:The effects of overwhelming experience on mind, body, and society* (pp. 182-213). New York, NY: Guildford Press.

van der Kolk, B. A., & Fisler, R. E. (1994). Childhood abuse and neglect and loss of self-regulation. *Bulletin of the Menninger Clinic, 58,* 145-168.

van der Kolk, B. A., Perry, J. C., & Herman, J. L. (1991). The childhood origins of self-destructive behavior. *American Journal of Psychiatry, 148,* 1665-1671.

Verhoeven, W. M. A., Tuinier, S., van den Berg, Y., W., Coppus, A. M. W., Fekkes, D., Pepplinkhuizen, L., et al. (1999). Stress and self-injurious behavior: Hormonal and serotonergic parameters in mentally retarded subjects. *Pharmacopsychiatry, 32,* 13-20.

Vrana, S., & Lauterbach, D. (1994). Prevalence of traumatic events and post-traumatic psychological symptoms in a nonclinical sample of college students. *Journal of Traumatic Stress, 7,* 289-302.

Walsh, B. W., & Rosen, P. M. (1988). *Self-mutilation: Theory, research, and treatment.* New York, NY: Guilford Press.

Whitlock, J., Eckenrode, J., & Silverman, D. (2006). Self-injurious behaviors in a college population. *Pediatrics, 117*(6), 1939-1948.

Winchel, R. M., & Stanley, M. (1991). Self-injurious behavior: A review of the behavior and biology of self-mutilation. *American Journal of Psychiatry, 148*(3), 306-315.

Witteveen, A.B., Huizink, A. C., Slottje, P., Bramsen, I., Smid, T., & van der Ploeg, H. M. (2010). Associations of cortisol with posttraumatic stress symptoms and negative life events: A study of police officers and firefighters. Psychoneuroendocrinology (2010), doi:10.1016/j.psyneuen.2009.12.013

Yates, T. M. (2004). The developmental psychopathology of self-injurious behavior: Compensatory regulation in posttraumatic adaptation. *Clinical Psychology Review, 24,* 35-74.

Yehuda, R. (1997). Sensitization of the hypothalamic-pituitary-adrenal axis in posttraumatic stress disorder. In R. Yehuda & A. C. McFarlane (Eds.), *Psychobiology of posttraumatic stress disorder* (pp. 57-75). New York: New York Academy of Sciences.

Yehuda, R. (2002). Current status of cortisol findings in post-traumatic stress disorder. *Psychiatric Clinics of North America, 25,* 341-368.

Yehuda, R., Boisoneau, D., Lowy, M. T., & Giller, E. L. (1995). Dose-response changes in plasma cortisol and lymphocyte glucocortocoid receptors following dexamethasone administration in combat veterans with and without posttraumatic stress disorder. *Archives of General Psychiatry, 52,* 583-593.

Yehuda, R., Giller, E. L., Southwick, S. M., Lowy, M. T., & Mason, J. W. (1991). Hypothalamic-pituitary-adrenal dysfunction in posttraumatic stress disorder. *Biological Psychiatry, 30,* 1031-1048.

Yehuda, R., Golier, J. A., Yang, R., & Tischler, L. (2004). Enhanced sensitivity to glucocorticoids in peripheral mononuclear leukocytes in posttraumatic stress disorder. *Biological Psychiatry, 55,* 1110-1116.

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, 3,* 389-404.

Yehuda, R., Halligan, S. L., & Grossman, R. (2001). Childhood trauma and risk for PTSD: Relationship to intergenerational transmission of trauma, parental PTSD and cortisol excretion. *Developmental Psychopathology, 27,* 733-53.

Yehuda, R., Kahana, B., Binder-Brynes, Southwick, S. M., Mason, J. W. & Giller, E. L. (1995). Low urinary cortisol excretion in Holocaust survivors with posttraumatic stress disorder. *American Journal of Psychiatry, 152*(7), 982-986.

Yehuda, R., Lowy, M. T., Southwick, S. M., Shaffer, S., & Giller, E. L. (1991). Lymphocyte glucocortiocid receptor number in posttraumatic stress disorder. *American Journal of Psychiatry, 148,* 499-504.

Yehuda, R., Resnick, H., Kahana, B., & Giller, E. L. (1993). Long-lasting hormonal alterations to extreme stress in humans: Normative or maladaptive? *Psychosomatic Medicine, 53*(3), 287-297.

Yehuda, R., Southwick, S. M., Krystal, J. H., Bremner, D., Charney, D. S., & Mason, J. W. (1993). Enhanced suppression of cortisol following dexamethasone administration in posttraumatic stress disorder. *American Journal of Psychiatry, 150*(1), 83-86.

Yehuda, R., Southwick, S. M., Nussbaum, G., Wahby, V. S., Giller, E. L., & Mason, J. W. (1990). Low urinary cortisol excretion in patients with posttraumatic stress disorder. *Journal of Nervous and Mental Disease, 178*(6), 366-369.

Young, E. A., Abelson, J. L., & Cameron, O. G. (2004). Effect of comorbid anxiety disorders on the hypothalamic-pituitary-adrenal axis response to a social stressor in major depression. *Biological Psychiatry, 56,* 113-120.

Zlotnick, C., Shea, T., Pearlstein, T., Simpson, E., Costello, E., & Begin, A. (1996). The relationship between dissociative symptoms, alexithymia, impulsivity, sexual abuse, and self-mutilation. *Comprehensive Psychiatry, 37*(1), 12-16.

Zweig-Frank, H., Paris, J., & Guzder, J. (1994a). Psychological risk factors and self-mutilation in male patients with BPD. *Canadian Journal of Psychiatry, 39*(5), 266-268.

Zweig-Frank, H., Paris, J., & Guzder, J. (1994b). Psychological risk factors for dissociation and self-mutilation in female patients with borderline personality disorder. *Canadian Journal of Psychiatry, 39*(5), 259-264.