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Relationship of adolescent baseline cortisol and maternal support on adulthood depression in
female victims of childhood sexual abuse

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ABSTRACT

Childhood maltreatment is an issue of major public health concern linked to adverse developmental outcomes throughout childhood, adolescence and adulthood. Recent studies and literature emphasize the need to investigate the biobehavioral risk factors of psychopathology amongst populations where maltreatment, specifically childhood sexual abuse, has occurred. Previous research has found that victims of chronic stress and trauma present with an attenuated stress response to compensate for prolonged hypothalamic-pituitary-adrenal axis activation. A prolonged stress response is associated with adult depression, a condition also associated with early life adversity, while positive social support is known to buffer against future psychopathology. This study focuses specifically on the relationship between childhood sexual abuse, baseline cortisol levels, maternal social support, and depressive symptoms in adulthood.

The data used comes from an ongoing prospective, longitudinal study of women who have undergone substantiated childhood sexual abuse ($n=64$) and a comparison group ($n=86$). Sexual abuse was determined at the initial visit (Time 1). Baseline cortisol was measured 7 years after the initial visit during adolescence (Time 4) as well as the participants' perceived maternal support. Depressive symptoms were assessed 6 years later during adulthood (Time 6). While controlling for race and age, a multiple linear regression showed that there were no significant interactions between childhood sexual abuse, baseline cortisol, maternal social support, and depressive symptoms in adulthood. These results show that there are discrepancies amongst research and future studies must continue to focus on the biobehavioral risks that victims of maltreatment face which may negatively influence their long-term health.

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Chapter 1 Introduction

HPA Axis

In the presence of a stressor, the body fights to maintain homeostasis. One way the body does this is through activation of the hypothalamic-pituitary-adrenal axis (HPA axis). Upon HPA axis activation, neurons in the hypothalamus stimulate the release of corticotropin-releasing hormone (CRH) (Smith & Vale, 2006). CRH transports to the pituitary gland, stimulating the release of the adrenocorticotropic hormone (ACTH). ACTH then targets the adrenal cortex where glucocorticoids are synthesized and secreted from the zona fasciculata. The glucocorticoid cortisol serves as the main molecule of the entire human stress response. This steroid hormone acts to regulate human bodily processes such as metabolism, the immune system, the cardiovascular system, and behaviors (Smith & Vale, 2006). Cortisol binds to two receptors: the mineralocorticoid receptor and the glucocorticoid receptor (type-I and type-II receptors, respectively). However, cortisol is more prone to bind to the mineralocorticoid receptors, which maintain low, normal levels of cortisol in the blood throughout the circadian rhythm.

Cortisol also acts to modulate the HPA axis through a negative feedback loop. In times of high stress when cortisol levels are elevated in the body, cortisol then binds to the lower affinity glucocorticoid receptor (Stephens & Wand, 2012). Once activated, these glucocorticoid receptors stop the stress response from continuing by exerting negative feedback on the hippocampus, pituitary, and the hypothalamus (Swaab, Bao, & Lucassen, 2005). Essential to survival, cortisol induces gluconeogenesis and modifies metabolism to meet bodily needs during stress. When

bound to glucocorticoid receptors, cortisol affects inflammation, cardiovascular function, blood pressure, arousal, learning and memory (Stephens & Wand, 2012). Due to the wide array of effects cortisol has on multiple body systems, the stress response must be tightly regulated to ensure normal body functioning occurs.

Attenuation Hypothesis

A person's genetics, childhood environment, and current stress all impact HPA axis activity and cortisol production in adulthood (Stephens & Wand, 2012). In a healthy individual, cortisol displays a diurnal rhythm where cortisol rises after awakening, peaking after 30-40 minutes and then declines throughout the day reaching its lowest point in the afternoon (Trickett, Noll, Susman, Shenk, & Putnam, 2010). Though crucial to keeping a healthy stress response system, hypoexcretion of cortisol during early childhood can cause problematic health issues, such as cognitive deficiencies and neuropsychiatric and metabolic disorders (Stephens & Wand, 2012). In a normal stress response, cortisol rises within minutes in response to a discrete stressor. Post-stressor, cortisol returns to baseline levels fairly quickly, reflective of cortisol's half-life in blood and saliva which is approximately one hour (Nicolson, 2008). When stress becomes chronic and exposure to cortisol is prolonged, wear and tear of the body occurs. The body becomes excessively vulnerable to the catabolic characteristics of glucocorticoids causing deleterious effects on cardiovascular and immune functioning as well as on brain structures (Raison & Miller, 2003; R.M. Sapolsky, Romero, & Munck, 2000). A widespread system of cortisol receptors consists in the hippocampus, amygdala, and prefrontal cortex. These receptors act as transcription factors and regulate gene expression, and excess cortisol can alter their

response. (Stephens & Wand, 2012). This can lead to a loss in cognitive and affective abilities, alterations in anxiety levels, as well as an inhibitions of parasympathetic bodily processes, such as sleep, sexual activity, and endocrine processes responsible for growth (Lupien, McEwen, Gunnar, & Heim, 2009; Swaab et al., 2005). The set point of stress response varies within individuals. If the set point is too low, the stress response may fail to activate during a threatening situation. Conversely, if the set point is too high, the stress response may fail to terminate rapidly post-stressor (Susman, 2006). Failure to terminate rapidly exposes an individual to unnecessary wear and tear and thus more vulnerability to disease.

Under immense and chronic stress, the HPA axis adapts to protect the body from the negative effects of overexposure to cortisol by adapting and secreting less cortisol. Hyposecretion of cortisol prevents the destructive effects that prolonged cortisol exposure can have on the hippocampus and frontal cortex and cardiovascular and immune function. The attenuation hypothesis (Susman, 2006) predicts that early and severe prolonged stress initially increases the stress response, ultimately leading to the suppression of the stress response as the body works to combat the excess of cortisol. Throughout development, it is crucial that young children maintain bodily equilibrium to nurture their growth. Studies have shown that children attenuate emotions and biological arousal as they go through unpredictable and adverse environments (Susman, 2006). Therefore, an attenuated stress response originally protects the body from the deleterious effects of cortisol when stressors fail to cease and impair normal body functioning.

Due to the knowledge of cortisol's effect on the body, it is important to understand how the HPA axis responds among individuals who undergo severe or chronic stress. Victims of childhood sexual abuse undergo a complex burden of stress at a very young age. Proposed

models have discussed that this unique type of maltreatment inflicts traumatic sexualization, betrayal, powerlessness, and stigmatization upon the victim leading to an array of short and long-term health consequences (Finkelhor & Browne, 1985). The attenuation hypothesis has been supported in past studies where developmental trajectories of baseline cortisol were higher during childhood and lower during adulthood among victims (Stein, Yehuda, Koverola, & Hanna, 1997; Trickett et al., 2010). Hypo and hyperactive HPA axis activation has been documented in victims of maltreatment suggesting that potentially the proximity to the abuse can cause the stress response to vary (Trickett et al., 2010). This suggests that at different time points during development, baseline cortisol may vary in victims of maltreatment, suggesting individuals may be at risk for certain health outcomes dependent on the developmental stage where hypo or hyperactivity occurs.

Cortisol, Depression, and Sexual Abuse

Many studies in the past and present focus on the extent to which victims of trauma and maltreatment face psychiatric disorders. The general notion tends to be that childhood sexual abuse contributes to one's vulnerability to suffer with depressive episodes and depression (Fergusson, Horwood, & Lynskey, 1996). Previous studies have found that women diagnosed with lifetime depression were two times more likely to be victims of intrafamilial childhood sexual abuse compared to women who were not depressed. Additionally, victims of childhood sexual abuse were more susceptible to have at least one depressive episode in their lifetime (Weiss, Longhurst, & Mazure, 1999). One particular study found that 100% of women in the sample who experienced sexual abuse with forced penetration developed depression (Bifulco,

Brown, & Adler, 1991). This indicates a clear connection between childhood sexual abuse and depression.

While the trends clearly exist there is not a fully understood mechanism describing the process. Using both animal and human studies, researchers have theorized, hypothesized and executed experiments looking into what aspect of early lifetime stress manifests itself within the body into adulthood depression. The dysregulation of the HPA axis seen in victims who have suffered chronic stress has been implicated as a factor leading to later depression.

Major depressive disorder is typically marked by a hyperactive stress response, indicating an increased release of CRH and cortisol (Weiss et al., 1999). Stressful episodes trigger depression as well and elevate salivary and urinary cortisol levels (Swaab et al., 2005). However, there remain contradictions to this research. In a study of inpatients in a psychotherapy program, the depressed patients had a significantly lower rise in cortisol after awakening and a study among elderly depressed patients showed hyposecretion in urinary diurnal cortisol (Huber, Issa, Schik, & Wolf, 2006; Oldehinkel et al., 2001). Lower cortisol responses to a dexamethasone/corticotrophin releasing hormone test, retrospectively, have also predicted suicide attempts among depressed inpatients as well as avoidant reactions to social threat under stress (Carpenter et al., 2007). Women with a history of child abuse exhibited different responses to ovine corticotrophin-releasing factor (oCRF) and exogenous corticotrophin-releasing factor (CRF) dependent on whether they were currently depressed or not (Heim & Nemeroff, 2001). Abused women without depression had normal cortisol responses to oCRF while the abused women with depression showed blunted ACTH responses to exogenous CRF.

In a recent study by Shenk et al. (2010), in victims of sexual abuse, a blunted cortisol area under the curve (AUC) response paired with an asymmetrical physiological response of

vagal withdrawal in late adolescence predicted higher depressive symptoms in young adulthood. (Shenk, Noll, Putnam, & Trickett, 2010). Therefore, in these cases, the protective effect of attenuated cortisol also holds possible risk to later psychopathology.

Overall, without an experimental design it cannot be determined whether or not the hypersecretion of cortisol is the main mechanism of developing depression. Only approximately 25% of subjects with major depressive disorder show increased basal plasma cortisol levels and other studies failed to find hyperactivity in the HPA axis as a cause for depression (Swaab et al., 2005). More research needs to be completed in order to better understand the relationship between the role of the HPA axis, depression and sexual abuse.

Impact of Social Support

Social support is the perception that an individual is cared for, loved, guided and valued by the people or network around them (Cobb, 1976). Social support can come in the form of psychological, physical and financial help whether it be from peers, teachers, siblings or parents. It is widely acknowledged that social support seems to play a significant role in maintaining both physical and psychological health. Research confirms that high quality positive support can increase resilience to stress, protect the body from developing trauma-related psychopathology, as well as reduce morbidity and mortality (Southwick, Vythilingam, & Charney, 2005). In physiological terms, social support has been shown to be associated with reduced salivary cortisol levels as well as lower cardiovascular reaction when acute stressors are present (Ozbay et al., 2007). In terms of health, the quality of social support, or lack thereof, holds many implications toward an individual's health.

Two models describe how social support may work to promote well-being. The Main Effect Model (Cohen & Wills, 1985) hypothesizes that social support is an independent process that brings benefits to overall health by providing positive affect. Additionally, the recipient of this support gains a sense of predictability and stability in life as well as a recognition of self-worth that ultimately shape coping strategies, behaviors, and overall health as a person goes through the stressors of life. The Buffering Effect Model (Cohen & Wills, 1985) suggests social support is only beneficial to the health of an individual when stress is present. When a stressor is present, social support can then intervene by influencing the individual's perception or cognitive evaluation of the stressor or by reducing the behavioral and psychological reaction toward the stressor (Tremblay, Hébert, & Piché, 1999). In terms of the Buffering Effect Model, individuals under high or chronic stress must receive a greater amount of social support in order to obtain its benefit (Tremblay, Hébert, & Piché, 1999). Essentially, social support can provide active coping mechanisms as an individual evaluates, responds, and works to resolve a new or threatening circumstance.

Social support is of major public health significance with an effect on life expectancy as strong as the effects of obesity, cigarette smoking, hypertension, and physical activity (Robert M. Sapolsky, 2004). While there is evidence that positive social support leads to beneficial health outcomes, a lack of social support poses a serious risk for development of a wide array of somatic, emotional and psychiatric disorders, such as depression (Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003). Poor social support in men and women is associated with not only the onset, but also the relapse of depression (Paykel, 1994). The effects of poor social support are not only short-term but long-term as well. For example, lack of quality parental care during childhood increases the risk of that child facing depression in adulthood (Andrews, Brown, &

Creasey, 1990). Quality of social support is a variable of interest in terms of determining the risk of not only an individual's physiological health, but also psychosocial health.

The association between adult depressive disorders and poor social support suggest that the quality of personal relationships may mediate or moderate the association between childhood adversity and health outcomes depending on the intensity of the adversity and the quality and quantity of the social support (Hill, Pickles, & Burnside, 2001). Through retrospective studies in which adults describe their childhood, links have been made connecting lack of sufficient parental care and sexual abuse in childhood to psychiatric disorders post-childhood (Andrews et al., 1990). Childhood sexual abuse combined with poor parental care predicted DSM-IV major depression in women, occurring within the later ages of 21-30 (Hill et al., 2001).

In many cases, the mother plays a large role in providing (or failing to provide) social support to her child. So far the general consensus in research among victims of childhood abuse supports that the non-offending mother of the victim is the most significant social influence on a child's safety, recovery, and later mental health (Pintello & Zuravin, 2001). Amongst girls who were sexually abused, those who perceived their mothers to be supportive demonstrated less behavioral difficulties and greater capacities in social and academic domains (Tremblay et al., 1999). Maternal behaviors can also trigger epigenetic changes of the glucocorticoid receptor gene promotor in the hippocampus as seen in preclinical studies (Weaver et al., 2004). The DNA methylation and chromatin structure changes that occur lead to a modification in the HPA axis that may underlie the mechanism of how a mother's behaviors and social support physiologically lower risk for stress-related disorders such as depression (Kaufman et al., 2006).

Childhood sexual abuse can lead to a division between the mother and child due to the offender, who, in many cases, is a family member (Lovett, 1995). Continued abuse after the

mother's discovery of the situation can lead to a distant and rejecting relationship as the mother has blatantly failed to protect her child (Lovett, 1995). Victims have been seen to recant disclosure in response to perceived low levels of their mother's support, setting them behind in their recovery progress and causing the internalization of the trauma (Pintello & Zuravin, 2001). Therefore, it is essential that maternal support remains a key factor in a child's resilience and recovery.

Overview of Study

The current study investigated the association between baseline cortisol in adolescence, quality of maternal social support in adolescence and depressive symptoms in adulthood among females. The data comes from an ongoing prospective study of women. One portion of the sample of women have undergone substantiated childhood sexual abuse ($n=64$) and the other portion of the sample served as a control group, matched to the experimental group in terms of age, race, SES, district of residence, family composition, and nonsexual traumas, only differing in regards to they did not experience child sexual abuse ($n=86$). In the longitudinal design of this study, the women underwent six different assessments from childhood to young adulthood (Time 1 through Time 6). Sexual abuse was determined at the initial visit (Time 1) by referral of the Childhood Protective Services. The three aims of the present study were to determine: 1) if baseline cortisol in levels in adolescence predict depressive symptoms in adulthood and 2) if perceived maternal social support and a history of childhood sexual abuse predict depressive symptoms in adulthood (see [Figure 1](#)). The study hypothesized that childhood sexual abuse would be predictive of lower levels of baseline cortisol leading to increased depressive

symptoms in young adulthood, with poor perceived social support of the mother increasing the risk of developing depression.

Chapter 2 Methods

Sample Characteristics

The data collected comes from an ongoing, longitudinal, prospective study of sexually abused women and a matched-control group that measured biopsychosocial markers related to development. The average age of the abuse onset was 7.5 years and the average duration lasted about 2 years. 70% of females suffered vaginal and/or anal penetration and 60% of perpetrators were the biological father or a father figure (stepfather, mother's live-in boyfriend). 52% of the sample suffered physical abuse and sexual abuse violence.

The longitudinal design of this study was cross-sequential consisting of six time point assessments each representing a different cross-section of ages. As the study progressed, after the initial assessment at Time 1, 13 females in the comparison group revealed some form of childhood sexual abuse. These participants were dropped from the comparison group and added to a "noncriterion abuse" group and were not included in the group analyses.

Trained clinical interviewers carried out each 3-4 hour session and the assessments focused on biological, psychosocial, and social domains of the females, comprehending their full growth and development. History of childhood sexual abuse was determined at the first assessment (Time 1). For the purposes of this particular study, data was taken from Time 4 and Time 6. Out of the sample, 146 participants were seen at both Time 4 and Time 6. However, 17 participants were not seen at Time 4, only Time 6. T-tests were used to see if there were any significant differences between these groups in age, race, group (abuse or comparison), baseline cortisol, perceived maternal social support, and depressive symptoms. There was no significant

difference between the groups ($p > .05$) and therefore, these results prove that it is very unlikely this missing data altered results.

In terms of SES, all families ranged from low to middle class. Amongst the women in the study, 49% were Caucasian, 46% African-American, 4% Hispanic, and 1% Asian-American. At Time 4, 66 women were in the sexually abused group and 84 women were in the control group. At time 6, 68 women were in the sexually abused group and 84 women were in the control group. At the initial assessment Time 1, the mean age of the females was 11.11 with a standard deviation of 3.02, ranging in age from 5.91-16.89. At the time 4 assessment, the mean age of the sample was 18.05 years with a standard deviation of 3.41, with ages ranging from 10.63 to 25.91. At the time 6 assessment, the mean age of the sample was 24.42 with a standard deviation of 3.19 with ages ranging from 18.12-32.14 (See [Table 1](#). for demographics).

Sample Recruitment and Retention

The Child Protective Service (CPS) of the greater Washington, DC, metropolitan area referred the sexually abused females ($n=84$). To be eligible for the study the females had to: a) be 6-16 years old; b) participate within 6 months of disclosure of abuse; c) have undergone substantiated sexual abuse including genital contact and/or penetration; c) been perpetrated by a family member; d) have a nonabusing caregiver of the female participate (typically the mother) who could provide information on the environment, family functioning, demographics, and information regarding their own personal developmental history.

Advertisements in newspapers and posters in welfare, daycare, and community facilities in the neighborhoods of the abused participants were used to recruit the comparison sample

(n=82). Comparison families contacted personnel in the study. To be eligible to participate the females of this sample had to have no prior contact with protective service agencies and similar demographics to a same-aged abuse participant. These females were not aware of the study involving sexually abused females and believed the study to be only about “female growth and development.” Caregivers were told at the end of the first interview that this study pertained to sexual abuse and a few cases were dropped because of some history of childhood sexual abuse. 70% of the comparison females resided in the same zip code district of the abused females. In terms of race, ethnicity, age, SES and family constellation (one or two parent families) and experience of nonsexual traumas, the abused and comparison did not differ statistically (Trickett, Noll, & Putnam, 2011).

The University institutional review board provided approval for the study, and a Federal Certificate of Confidentiality was obtained. The nonabusing caregivers provided consent for subjects under 18. Subjects over the age of 18 signed for themselves. Subjects between 6-17 years old also provided assent for themselves.

From Time 2 to Time 6 retention rates have ranged from 84% to 88% with no significant difference by group. Over 96% of the sample was retained, as well as reassessed, at Time 4, 5 and 6. The study design employed several strategies to retain the sample over the course of this longitudinal study. To retain the participants in this longitudinal study, several strategies were used including home visits to those with interrupted or unlisted telephone service, monetary incentives for returning phone calls, locating techniques, additional contact persons for the subject, and mailings, such as newsletters, birthday, holiday and congratulation cards. Additionally, the study communicated the importance of the longitudinal study to participants and worked to instill a sense of pride for adding vital data through their participation. Every

participant in the study received monetary compensation for completing assessments at a rate set by the NIH Healthy Volunteer Office Monetary. For more information, see Trickett et al., 2011.

Procedure

Typical assessments for each time wave began in the morning between 8:30 to 9:30 am to control for diurnal fluctuations in hormones. Upon arrival, the informed consent/assent process took place. A protocol lasting approximately 4 hours then took place which included a collection of saliva samples as well as cognitive, behavioral, and health assessments. For this study's purposes, only a subset of measures were used in analyses.

Measures

Sexual Abuse Status

The Child Protective Services' records of the greater Washington, DC, metropolitan area provided data on the sexual abuse status of each participant in the study. Each female within the abuse group at the onset of the study was between 6-16 years old, had disclosed the abuse within the past 6 months, and had undergone substantiated intrafamilial sexual abuse, including genital contact and/or penetration. The median age of sexual abuse onset was 7.8 years and the median duration was 24 months. The comparison group was coded as 0, while the abuse group was coded as 1. As the study progressed, if a participant within the comparison group revealed prior childhood sexual abuse that was disclosed at Time 1, they were moved into a separate group, coded as 3.

Cortisol

The baseline cortisol used in this study comes from the Time 4 assessment when the participants were in adolescence. This assessment began at approximately 8:30 am and once consent/assent was obtained, an initial saliva sample was taken. After a resting period of approximately twenty minutes where the participants were instructed to relax and quietly fill out demographic questionnaire forms, a second saliva sample was taken. This period allowed the subjects to calm down and minimize potential stress effects attributable to participating in this study such as hectic mornings preparing for and anticipating the visit, and/or the stress associated with traveling to or locating the area where the assessments took place. The second saliva sample represents the participant's baseline cortisol. Samples were stored at -70°C until they were sent to Salimetrics Laboratories in State College, PA, where they were assayed in duplicate using a highly sensitive enzyme immunoassay. The test used 25ml of saliva per determination, has a lower limit sensitivity of .003 $\mu\text{g}/\text{dl}$, standard curve range from .007-1.8 $\mu\text{g}/\text{dl}$, and the intra- and inter-assay coefficients of variation averages are 5.10% and 8.20% respectively. Baseline cortisol concentrations from the saliva sample are represented in $\mu\text{g}/\text{dl}$.

Depression

The Beck Depression Inventory-II, abbreviated as the BDI-II (A. Beck, Steer, Ball, & Ranieri, 1996) was administered to evaluate mood functioning 2 weeks before the Time 6 assessment. The BDI-II measure consists of 21 items assessing symptoms of major depressive disorder based off the Diagnostic and Statistical Manual for Mental Disorders (DSM-IV). Each of the 21 items consists of four self-evaluative statements scored 0-3, with 0 indicating no

depressive symptom and 3 representing a strong depressive symptom. Responses are summed and scores range from 0 to 63 with higher scores associated with greater depression severity (Whisman, Perez, & Ramel, 2000). A score of 10-18 signifies mild to moderate depression, 19-29 signifies moderate to severe depression, and 30-63 signifies severe depression (A. T. Beck, Steer, & Carbin, 1988). This measure is widely used and has excellent internal consistency ($\alpha = .93$) and good concurrent validity ($r=.71$) among other measures of depression (A. Beck et al., 1996). After reverse-scoring negatively worded items, all of the items answered by each of the participants in the time 6 assessment were totaled to determine each individual's depression symptom score.

Perceived Maternal Support

In order to investigate participants' perceived maternal support at Time 4, the study administered the Inventory of Parent and Peer Attachment, abbreviated as the IPPA (Armsden & Greenberg, 1987). This measure has been formulated from the attachment theory of psychoanalyst John Bowlby. Bowlby theorized that secure attachment leads to an unconscious assurance of a trustworthy and helpful figure for support which also instills a belief that oneself is worthy of love and care. The IPPA specifically assesses the psychological security one feels their parents and peers provide. Degree of mutual trust, quality of communication, and extent of anger and alienation are the three broad dimensions used to measure perceived support (Armsden & Greenberg, 1987). For the purpose of this study, only the Mother Attachment section of the revised version of the IPPA was used. Twenty-five items comprise the Mother Attachment section and it has good internal reliability ($\alpha = .87$). After reverse-scoring the negatively worded

items, these items were summed to give a score of perceived maternal support (Armsden & Greenberg, 1987). Higher scores represent a more secure attachment between the female in the study and her mother.

Data Analysis Plan

Descriptive statistics were computed for continuous variable and frequencies were computed for categorical values. See [Table 1](#). Measures that were reversed coded, were reverse scored before summation and use in analyses. For descriptive purposes, independent samples t-tests were run to compare means for the abused and comparison group for the following: race, age, baseline cortisol, and maternal social support at Time 4, and age and depressive symptoms at Time 6. When Levene's Test for Equality was significant, adjusted degrees of freedom were used. A Pearson correlation analysis was then performed to examine the associations between all continuous variables.

A multiple linear regression was run to test whether childhood sexual abuse, baseline cortisol at Time 4, and mother's social support at Time 4 predicted depression at Time 6, determined through BDI scores. Race and age were controlled for in this interaction.

Chapter 3 Results

As seen in [Table 1](#), sexually abused and comparison participants were similar among demographic variables as well as in the measures of interest. There were no significant differences between the sexual abuse group and the control group in mean baseline cortisol concentration at Time 4 (0.42, 0.32 μ g/dl, -1.15(79.78)), maternal social support at Time 4 (93.16, 89.07, -1.02(138)), age at Time 6 (24.85, 24.19 years, -1.27(150)), and depressive symptoms at Time 6 (10.98, 8.88, -1.42(112.75)).

The Pearson correlation analyses (see [Table 2](#)) only showed significance between age at Time 6 and mother's social support ($r = .178$, $p = .041$) and between age at Time 6 and race ($r = -.168$, $p = .039$). Baseline cortisol at Time 4 and depression at Time 6 were trending towards significance ($r = -.160$, $p = .075$). There were no other significant differences. Even though potential covariates, age and race, did not significantly correlate with depression, they were still included within the model.

Multiple linear regression analyses were performed to identify the impact of childhood sexual abuse, baseline cortisol and perceived maternal social support at Time 4 on symptoms of depression at Time 6. Nonsignificant interactions were trimmed to produce the final model (see [Table 3](#)). The model was not significant ($p > .05$) and the predictors explained 3.4% of the variance in depression symptoms at Time 6. Symptoms of depression at Time 6 was not predicted by childhood sexual abuse ($\beta = 5.14$, $p > .05$), baseline cortisol at time 4 ($\beta = -2.29$, $p > .05$), and perceived social support of the mother at time 4 ($\beta = -.032$, $p > .05$).

Chapter 4 Discussion

Summation

Childhood sexual abuse is a major public health concern linked to adverse developmental outcomes throughout childhood, adolescence and adulthood. Previous studies have shown that females who experience childhood sexual abuse differ across many biopsychosocial domains when compared to similar females who have not experienced sexual abuse. Studies have shown that sexually abused females may exhibit cognitive deficits, dissociative symptoms, maladaptive sexual development, higher rates of obesity, more major illnesses and healthcare utilization, posttraumatic stress disorder, and self-mutilation (Trickett et al., 2011). Sexually abused females have also been shown to have attenuated cortisol levels and higher risks of developing depression (Bifulco et al., 1991; Trickett et al., 2010). The aim of this study was to determine the biobehavioral risks of developing depression among female victims of childhood sexual abuse. The study sought to test the hypothesis that childhood sexual abuse would be predictive of lower levels of baseline cortisol in adolescence leading to increased depressive symptoms in young adulthood, with poor perceived social support of the mother increasing the risk of developing depression.

To test this model, data from a longitudinal, prospective study of females were used. Approximately half of the sample experienced substantiated, familial sexual abuse while the other half served as a comparison group, statistically similar to the sexually abused women in terms of race, ethnicity, age, SES, and family constellation (one or two parent families) and experience of nonsexual traumas. This design allowed for predictions about childhood sexual

abuse effects on baseline cortisol and perceived mother's social support in adolescence and the outcome of adulthood depression.

After running a Pearson correlation analyses and multiple linear regression between baseline cortisol, perceived support of the mother, and the outcome of depression, the results of the interactions proved to be overall insignificant. These findings indicate that depressive symptoms at Time 6 were not predicted by childhood sexual abuse, baseline cortisol at Time 4, and perceived maternal at Time 4.

Childhood sexual abuse brings a complex and unique burden of stress. Unlike other forms of abuse, childhood sexual abuse is specifically seen to cause traumatic sexualization, betrayal (especially in the case of intrafamilial abuse), and stigmatization upon the victim leading to numerous negative short and long-term consequences (Finkelhor & Browne, 1985). Specific studies have shown that 100% of women who suffered forced penetration developed depression and women who suffered with lifetime depression were two times more likely to have experience intrafamilial sexual abuse (Bifulco et al., 1991; Weiss et al., 1999). In light of the chronic trauma and stress imposed on an individual, the attenuation hypothesis (Susman, 2006) predicts that to adapt and adjust to the influx of cortisol from prolonged activation of the HPA axis, the body reacts by reducing cortisol levels. Higher cortisol levels in childhood and lower cortisol levels in adulthood have been seen in victims of childhood sexual abuse (Stein et al., 1997; Trickett et al., 2010). Though the HPA axis attenuates to modulate the deleterious effects of cortisol, lower cortisol has been associated with depression (Carpenter et al., 2007; Huber et al., 2006; Shenk et al., 2010). Strong and positively perceived social support has been seen to increase resilience to stress, reduce the risk of developing trauma-related psychopathology, and lower salivary cortisol (Ozbay et al., 2007; Southwick et al., 2005). Conversely, poor social support has been associated

with depression and relapse of depression (Heinrichs et al., 2003; Paykel, 1994). When paired with childhood sexual abuse, poor parental care predicted depression in women within the age range of 21 to 30 (Hill et al., 2001). Childhood sexual abuse has also been seen to divide and create a hostile mother-daughter relationship (Lovett, 1995). Many studies confirm the significant influence mothers have on their children's later mental health and positive mother support has been seen to lower depression in children (Kaufman et al., 2006; Pintello & Zuravin, 2001).

What was found within this study contrasts with past published literature linking childhood sexual abuse to adulthood depression (Bifulco et al., 1991; Fergusson et al., 1996; Weiss et al., 1999) and attenuated levels of cortisol (Stein et al., 1997; Trickett et al., 2010). Additionally, the findings of this study contrast with literature linking positive perceived social support to less depression (Ozbay et al., 2007; Southwick et al., 2005) and negative perceived social support to greater risk of depression or relapse (Andrews et al., 1990; Heinrichs et al., 2003; Hill et al., 2001; Paykel, 1994). Although the results of this study were deemed insignificant, this does not mean that childhood sexual abuse is an issue lacking serious risks to victims or unworthy of greater public health attention.

There are undoubtedly hazards and risks associated with childhood sexual abuse and though this study failed to find significant correlations, insights can be gathered and further questions for future research can be raised. Though known to be associated with abuse, conflict, and trauma, depression is still a highly complex and misunderstood disease. It is extremely difficult to assign specific factors as to why one is depressed and another is not. Sexual abuse often co-occurs with other forms of abuse and therefore, it is difficult to rule out whether

outcomes are due to other types of abuse, how the abuses interact with each other, or sexual abuse, specifically.

This study may highlight that timing is crucial when determining the outcome of depression. Studies have shown that support during early adolescence remains much more imperative than when compared to later adolescence (Stice, Ragan, & Randall, 2004). In terms of this study, support was measured when the mean age of the sample was 18. It is possible that these females' outcomes (depression) may not have been as dependent on their mother's support as they have grown to be more independent individuals possibly receiving more crucial support from friends, significant others, etc. More studies at various time points through childhood and adolescent should measure social support of the mother to determine at which point it holds the most value in determining future psychopathology.

In terms of the HPA axis, hypo and hyperactivity of the stress response can vary depending on proximity to the maltreatment or the developmental stage of the lifespan (Trickett et al., 2010). Though the baseline cortisol of the sexually abused female was not attenuated in adolescence, this cannot speak for all time points of their life. It is possible we chose the wrong time point to search for attenuation. Therefore, it is crucial that not just one measurement of cortisol is studied, but rather all of them, and how these trajectories over development interact with each other to produce certain health outcomes. Additionally, measuring baseline cortisol is not the only way to measure the activity of the HPA axis. Cortisol levels can remain normal while other probes may indicate regulatory abnormalities such as increased CRH and ACTH followed with decreased adrenal sensitivity (Nicolson, 2008). In order to understand the overall functioning of the HPA axis in victims of abuse, multiple measures must be taken into account, not just cortisol levels.

It is clear that the attenuation provides short-term positive health outcomes as prolonged exposure to cortisol causes deleterious effects on brain structures such as the hippocampus and frontal cortex as well as cardiovascular and immunological functioning (Raison & Miller, 2003; R.M. Sapolsky et al., 2000). Perhaps attenuation holds more positive long-term risks than negative. One particular study by (Heim, Newport, Bonsall, Miller, & Nemeroff, 2001) found that women abused in childhood who did not have adult depression released less cortisol in response to a ACTH₁₋₂₄ stimulation test (250 µg) when compared to the currently depressed abused women in the study. Future studies should expand on this idea and investigate attenuation in terms of a resilience factor rather than a risk for psychopathology. More research should be done comparing the groups of people who attenuate cortisol levels in times of high stress and trauma with those who do not to determine the long-term effects of the originally protective effect.

Since the results did not match this literature, it is imperative to analyze the study: the strengths, the weaknesses, and the implications it has for the future of research concerning the biobehavioral processes and health outcomes concerning victims of childhood sexual abuse.

Strengths of the Study

This study offers valuable insight as it offers a research design that is longitudinal and prospective. Many studies and evidence gained in childhood sexual abuse research in the past has relied on retrospective self-reports from adults as they recall their childhood, adulthood and experiences. The design of this study allows for assertions of stronger casual inferences on the forthcoming effects of childhood sexual abuse and the progression of female development as

confounds can be controlled. The statistical similarities between the experimental and control group at the beginning of the study (gender, age, race, SES, family constellation, geographic residency) allow stronger conclusions to be drawn in terms of the distinct effects of sexual abuse. Overall, combined with the comprehensive conceptual framework, in depth biopsychosocial assessments, and relatively small attrition rates within the two decades of the study, the design offers excellent data to connect childhood sexual abuse to developmental and health outcomes.

Weaknesses of the Study

The study does have some limitations that compromise the generalizability of the findings. The sample size of the study is relatively small. Among the sexually abused experimental group only the most severely abused were included, limiting the size of this sample. The sample cannot also generalize the effects of sexual abuse onto males, as the sample is only comprised as females, although the effects for males and females are likely considerable. Additionally, every female in the abused group has undergone at least one intervention as the abused group was recruited by the Child Protective Service. Therefore, there is no information through this study about the population of people who undergo childhood sexual abuse and never disclose or report the abuse. This sample may have undergone the most severe abuse. Although informed the measures, such as the IPPA, were confidential before they were completed participants may have feared their mother would see their answers, construing data about true perceived social support of the mother.

Research on Sexual Abuse/Research Efforts

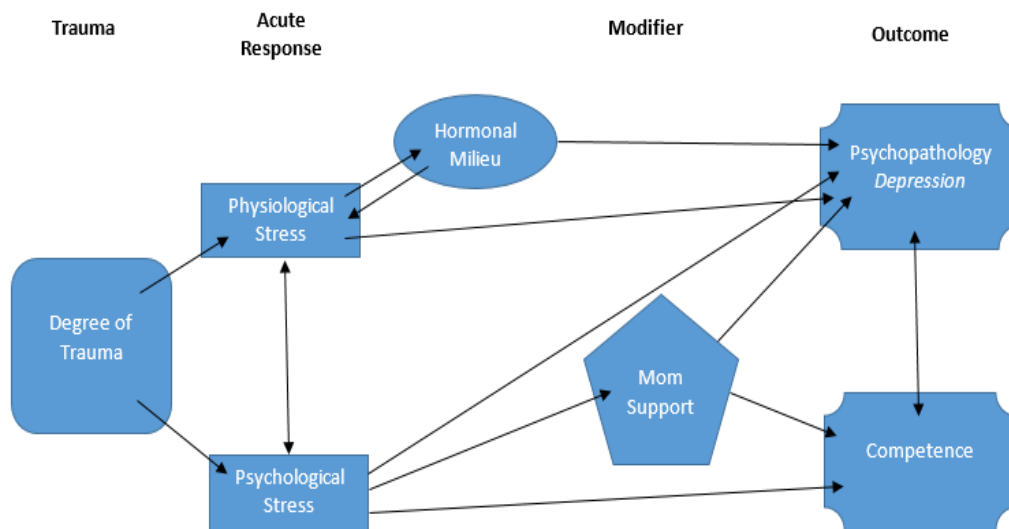
Though the annual number of sexually abused children in the United States is extremely difficult to estimate, the National Child Abuse and Neglect Data System reported a total of 69,184 cases in 2008, encompassing 9% of the total number of validated maltreatment cases (Services, 2008). However, these are only the cases that have been reported and the numbers of sexually abused children may be substantially greater. Though sexual abuse varies drastically throughout each state, it is a prominent issue affecting millions of children in terms of their well-being, short and long-term health, which in turn affects their children's lives, as well as their impact on the community, society, and the country. Childhood sexual abuse is a public health issue that cannot be ignored and must receive the necessary attention it deserves.

The National Center for Injury Prevention and Control of the Centers for Disease Control and Prevention discuss vital organizations that are needed in terms of policy and prevention for childhood sexual abuse. The CDC highlights the importance of creating youth-serving organizations where sexual abuse is not only addressed but also prevented by creating a safe place with clear goals and processes to implement prevention policies and practices which can be implemented in a prevention plan (Saul & Audage, 2007). Additionally, grassroots prevention programs such as Darkness to Light aim to reduce incidence and raise public awareness of the prevalence and consequences of childhood sexual abuse with hopes to empower the public to stop this issue highlighting that childhood sexual abuse is an adult issue ("End Childhood Sexual Abuse," 2013). The five steps this organization urges the public to do are learn the facts about childhood sexual abuse, minimize the opportunity, openly discuss topics of safety and boundaries with children, highlight to the public in order to have for protecting children against abuse are learn the facts, minimize opportunity, recognize the signs, and react responsively.

The National Coalition to Prevent Child Sexual Abuse and Exploitation highlights the need to think about long-term solutions to childhood sexual abuse rather than short-terms by increasing research and public awareness, while diminishing risks for abusive environments, enhancing policies and encouraging collaboration between multidisciplinary and grassroots organizations (The National Coalition To Prevent Child Sexual Abuse and Exploitation, 2012). In terms of public health principles, it is more costly to prevent health risks rather than treat and intervene subsequently. However, with the widespread issue, that is child abuse, prevention and intervention strategies both hold immense significance at this point and time. Child sexual abuse is not only a short-term problem for children but also a long-term health issue, affecting the child, the child's family, the community, and the overall health of the country. Research must continue to discover the mechanisms as to why children of abuse are at risk and what can be done to not only help them and their health but to decrease and end the issue of childhood sexual abuse.

Chapter 5 Figures

Figure 1 Conceptual Model



Redrawn from (Trickett et al., 2011).

Chapter 6 Tables

Table 1. Demographic Information and Variable Means

	Sexually Abused Mean (SD) or <i>n</i>	Comparison Mean (SD) or <i>n</i>	<i>t</i> (<i>df</i>)
Time 4 (N=150)	66	84	
Age	18.53(3.57)	17.76(3.34)	-1.37(148)
Race			
Caucasian	48	46	
African-American	27	46	
Hispanic	3	2	
Asian	1	0	
Baseline Cortisol	0.42(0.69)	0.32(0.26)	-1.15(79.78)
Maternal Social Support	93.16(23.16)	89.07(23.98)	-1.02(138)
Time 6 (N=152)	68	84	
Age	24.85(3.36)	24.19(3.05)	-1.27(150)
Depressive Symptoms	10.98(10.00)	8.88(7.09)	-1.42(112.75)

Table 2 Pearson Correlations

	1.	2.	3.	4.	5.
Depressive Symptoms (T6)					
Group	.122 (.147)				
Baseline Cortisol (T4) (ug/dl)	-.160 (.075)	.103 (.214)			
Maternal Social Support (T4)	-.086 (0.345)	.086 (.311)	.164 (.054)		
Age (T6) (years)	.045 (.597)	.103 (.206)	.178* (.041)	-.046 (.608)	
Race	.069 (.417)	.065 (.396)	-.002 (.982)	-.034 (.692)	-.168* (.039)

Note. * = $p < .05$

Table 3. Multiple Linear Regression of Adolescent Cortisol and Maternal Support Predicting Young Adulthood Depressive Symptoms

Variable	B	SE	β	F	df	p	R²
Intercept	5.139	10.589				.628	
Baseline Cortisol (T4) (ug/dl)	-2.289	1.965	-.116			.247	
Maternal Social Support	-.032	.038	-.080			.412	
Age (T6) (years)	.177	.280	.065			.528	
Race	1.076	1.455	.073			.461	
Group	1.651	1.710	.092			.336	
Full Model				.781	5, 110	.566	0.034

Note. * = $p < .05$

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