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Cortisol and testosterone in association with history of sexual abuse within incarcerated adolescent youth

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Cortisol and testosterone in association with history of sexual abuse within incarcerated adolescent youth

by

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The student author, whose presentation of the scholarship herein was approved by the program of study committee, is solely responsible for the content of this thesis. The Graduate College will ensure this thesis is globally accessible and will not permit alterations after a degree is conferred.

Iowa State University

Ames, Iowa

2017

DEDICATION

To my sister Zoya, who remains a remarkable role model in all of my professional endeavors. To my parents, without their support and encouragement I would not have pursued mastery of anything.

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ABSTRACT

Adolescence is a challenging developmental transition in the lives of young adults, yet despite these challenges youth must normatively transition through adolescence and take on adult roles and responsibilities. Research on the topic of adolescence has addressed many of the biological, psychological, and social changes that occur during this time and this evidence converges on the notion that this normative transition can be a period of vulnerabilities or opportunities. Exposure to many forms of early adversity can exacerbate the biopsychosocial challenges of adolescence, with emerging research illustrating that child abuse and maltreatment exert particularly deleterious consequences on adolescent physiology and development. What is under-studied is whether specific forms of maltreatment in early life have immediate and long lasting effects on physiological health and wellbeing of developing youth. The present study examined cortisol and testosterone, two hormones that play important roles in adolescence and pubertal development. In addition to being acutely responsive to context, the two hormones follow a diurnal rhythm, which is highly associated with health outcomes. The present study examined effects of sexual abuse on diurnal testosterone and cortisol of incarcerated adolescent girls and boys (N=101, 49.5% female, 53% Non-White), ages 13-18 (M=16.17). The present study examined whether sexual abuse had a unique effect on cortisol and testosterone diurnal slopes for males and females. Gender was explored as a potential moderator of the relationship between sexual abuse and hormones. Race, BMI, and puberty were controlled for in examining the effects of sexual abuse on waking hormones and diurnal slope. Sexually abused youth had steeper cortisol slopes (p<.005). Girls had flatter cortisol slopes (B=.088, p<.05) and lower waking testosterone (p<.001). No significant effect of sexual abuse on testosterone was found after

controlling for gender. Examining cortisol and testosterone rhythms as a function of sexual abuse can inform research and practice of the potentially adaptive response to this form of trauma in a highly vulnerable sample. Finally, information gathered from this study can open doors for improvement of intervention and prevention strategies, particularly for at-risk youth growing up in unfavorable environments.

CHAPTER ONE: INTRODUCTION

Adolescence is often described as a crucial and perhaps most turbulent period in development. During this time, biological maturation and formation of adult roles and responsibilities take place. Although not all adolescents will experience setbacks during this developmental stage, the adolescents who do encounter disruptors at this time can experience negative developmental and health outcomes (Steinberg, 2014). Problems such as behavior and emotion regulation difficulties contribute to the 200% increase in morbidity and mortality among adolescents (Dahl, 2004). In fact, many of the negative outcomes such as depression, alcohol and substance abuse, sexual health problems, eating disorders, and others, which contribute to the increase in disability and death occur during this sensitive period of development (Steinberg, 2014). The changes that occur at this time range from formation of new social groups and responsibilities, to maturation of brain regions implicated in emotion and reward (Steinberg, 2014; Andersen, 2016). The stress response and reproductive systems undergo maturation during this time as well. These significant environmental and biological changes increase vulnerability to psychopathology (Andersen, 2016; Gunnar, and Quevedo, 2007), among other negative outcomes such as obesity (Dietz, 1994). The malleability of different biological mechanisms that undergo maturation during this time suggests the importance of understanding the role of environment in biological formatting and shaping of developmental trajectories, especially for predicting health outcomes. The environment can greatly affect the timing, duration, and trajectory of critical development in adolescence, but also significantly shape future health outcomes.

In order to understand the adaptive and maladaptive outcomes as a result of these disturbances, it is important to examine adolescent experiences through a biopsychosocial lens.

Consequently, this study focuses on incarcerated adolescent youth's history of sexual abuse, as a specific form of early adversity and predictor of alterations in diurnal profiles of two hormones, cortisol and testosterone (see Figure 1.). The present study contributes to extant literature on at-risk youth in one particularly novel way. Namely, the sample is derived from *incarcerated* youth, which is not a commonly investigated population in the scientific literature, particularly in examining outcomes of early adversity. This sample is unique as it portrays the portion of youth who find the transition into adulthood especially challenging and are likely at-risk and considered highly vulnerable to negative health outcomes. Little is known about physiological outcomes of early adversity in incarcerated adolescent girls, although there have been more studies on incarcerated boys (see Gostisha, Vitacco, Dismukes, Breiman, Merz, and Shirtcliff, 2014). However, the scarcity of research is especially true for examinations of sexual abuse in incarcerated boys and girls. The present study aims to disentangle some of the factors that have influenced their developmental trajectories, specifically focusing on sexual abuse as the main predictor of altered cortisol and testosterone diurnal profiles. Examining these health outcomes (i.e., diurnal hormones) as a function of a specific form of abuse, while considering gender and pubertal maturation and other potential control variables, provides insight into the disturbances and vulnerabilities of adolescents whose developmental trajectories have been altered by prior trauma. The present examination can further demonstrate how the cortisol and testosterone mechanisms have adapted or recovered from sexual violence.
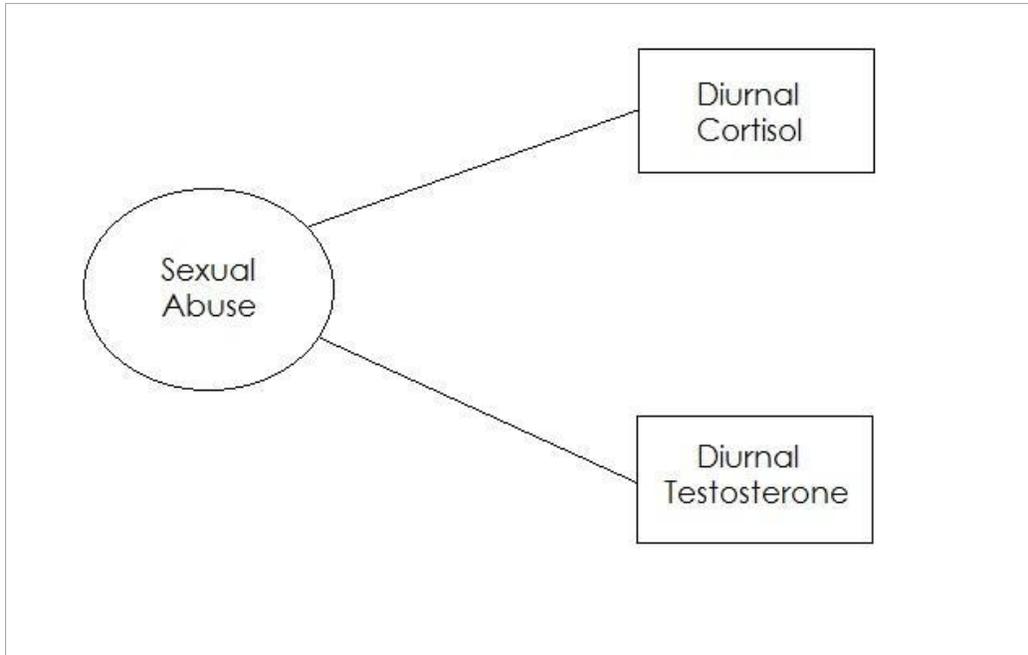
Figure 1. Model for present study.

Girls and boys experience adolescence differently, not only in timing and tempo of pubertal development, but also in the negative outcomes that result in disruption of their developmental trajectories. Boys are more likely to exhibit conduct disorder, and develop attention-deficit hyperactivity disorder (ADHD), whereas girls are more likely to develop depression, eating disorders, distorted body image, and anxiety (Zahn-Waxler, et al., 2008; Compas, Orosan, and Grant, 1993). This can occur for different reasons, such as boys and girls experiencing different environmentally-driven risk factors, having a different threshold for developing serious problems as an outcome, also having different biological processes like gene expression that could vary between the sexes (Zahn-Waxler, et al., 2008).

Because of the great interplay of biology and environment, one of the most heavily researched causes of disruption in development is early adversity. Early adversity can be in forms of poverty, abuse, and neglect, which can greatly impact development in a number of ways, from gene to mental health, and behavior (Thompson, and Haskins, 2014; Fairchild, and

Passamonti, 2017; McLaughlin, Lane, and Bush, 2016). Extant research highlights the negative impact adversity has on different markers of development (e.g., neglect on neurobiology; Humphreys, King, and Gotlib, 2017). However, the findings vary when considering the severity and type of adversity, particularly in the case of abuse (e.g., emotional, physical, sexual abuse; Wind, and Silvern, 1992). Adolescence is a crucial period for development, close to if not as sensitive as the infant years in terms of susceptibility, therefore it is highly vulnerable to disruptions that can change its trajectory and tempo of development. For example, adolescent years are a time of high prevalence of sexual assault and abuse, with estimates ranging between approximately 12 to 33% for girls, and 5 to 8% for boys before the age of 18 (Finkelhor, Shattuck, Turner, and Hamby, 2014). This type of abuse can trigger early onset of puberty in girls, which has been associated with fertility and menstrual issues as well as reproductive system cancers (Trickett, Noll, and Putnam, 2011). Boys who have been sexually abused are at risk for psychological distress, substance abuse, and sexual dysfunction (Holmes, and Slap, 1998).

The changes across different mechanisms are crucial, because maturation across each of the mechanisms is assumed to provide developing individuals with skills necessary for independence, and set them up for successful transition into adulthood (Steinberg, 2014).

The present study tackles one particularly harmful form of early adversity - sexual abuse – and investigates the effect it has on female and male development. Developmental and health outcomes are reflected in diurnal profiles of cortisol and testosterone, which are described in detail in Chapter Two. The present study aimed to determine whether there is an association between incarcerated youth's history of sexual abuse and their hormone profiles. Extant research has confirmed that early adversity greatly alters physiology, particularly the stress response

mechanism (Gunnar, 2000; Zahn-Waxler, et al., 2008), and that it can accelerate or delay pubertal development (Belsky, Ruttle, Boyce, Armstrong, and Essex, 2015; Koss, Mliner, Donzella, and Gunnar, 2016). This study has the potential to significantly contribute to the existing literature on incarcerated youth, especially by providing more information about the influence of sexual abuse on cortisol and testosterone profiles. Such information can be crucial in forming trauma-informed prevention or treatment programs in order to help at-risk youth thrive.

CHAPTER TWO: THEORETICAL FRAMEWORK

There are several frameworks that inform the present study, beginning with the life course theory, which is appropriate given its focus on developmental trajectories. Additionally, the life course theory recognizes unique and impactful events as significant disruptors of the normative trajectory. On the other hand, the biopsychosocial model allows for the examination of the biological, psychological, and social factors that can impact development. It is considered comprehensive and recognizes the varying roles of environmental, biological, and psychological factors in shaping development. Finally, developmental psychopathology is a discipline that emphasizes the importance of examining both adaptive and maladaptive, or typical and atypical development, specifically considering the biopsychosocial aspects of development. These three frameworks are suitable for the present investigation considering the characteristics of the sample, and the uniqueness of the experience of sexual abuse.

Life Course Theory

Life course theory (Elder, 1998) can be employed in studies of development for its focus on transitions and trajectories. Specifically, the theory posits that certain life events can reframe or reorganize an individual's life trajectory, which could result in disruption or change of the developmental course. Elder (1998) describes the transformative events as unique trauma, specifically focusing his discussion on grand, environmentally-driven events such as war and economic fluctuations. Considering these examples of life-changing events, they appear to have commonalities with traumatic experiences considered in the present study. For example, war is considered an extended form of trauma and can greatly impact physiological functioning over the course of development and well into adulthood (Rohleder, Joksimovic, Wolf, and Kirschbaum, 2004). Although life course theory typically focuses on transitions through multiple

stages across the lifespan and is often used to frame longitudinal studies. The focus on specific

events that alter the normative trajectory of development is relevant to the present study.

Biopsychosocial Theory

The biopsychosocial model originated as a philosophy and guide for clinical care. It

allowed for an understanding of disease and illness through multiple levels of organization and

experience (Borrell-Carrio, Suchman, and Epstein, 2004). George Engel offered the

biopsychosocial model as an alternative to the biomedical model, which was prevailing in the

mid- 20th century (Engel, 1977). He rooted for a more complex understanding of clinical

phenomena, and stressed that the sociocultural factors should not be ignored in clinical practice.

George Engel's contribution to science was the change of the way clinicians and scientists

understand the individual and address their specific needs through a multidimensional

investigation (i.e., biopsychosocial). The biopsychosocial model of development is often used in

the study of adolescence. Specifically, the biopsychosocial investigation of adolescent conduct

problems examines biological dispositions and social contexts which place children at risk

through experiences with social groups like parents, peers, and schools (Dodge, and Pettit, 2003).

A similar approach has been used in the study of risk-taking behaviors during adolescence

(Irwin, and Millstein 1986). A closely-related field of developmental psychopathology

investigates development by examining the interplay of biopsychosocial factors, with the

assumption that atypical developmental pathways can inform typical development, and vice

versa (Cicchetti, and Toth, 2009). Developmental psychopathology is described as an

interdisciplinary approach to examining biological, psychological, and social aspects of normal

and abnormal development across the lifespan (Cicchetti, and Toth, 2009). This perspective

focuses on both adaptive and maladaptive developmental processes. It assumes that, in order to

comprehensively examine and understand psychopathology, the organization and disorganization of systems should be viewed together. Developmental psychopathology also emphasizes individual developmental pathways of adaptation following adversity or long-term trauma, therefore importance is placed on understanding the pathways of individuals who have diverged from normative trajectories and adapted (Cicchetti, and Rogosch, 2002). Sroufe and Rutter (1984) referred to this approach as the study of the origins and patterns of individual behavioral maladaptation, which also focuses on processes underlying the change and consistency in those patterns. Because the study of developmental psychopathology is often used in psychology and psychiatry, a large portion of this perspective are evidence-informed prevention and intervention. The idea is to incorporate the biobehavioral research into intervention strategies and distinguish processes which can potentially alter the developmental trajectories through prevention and intervention (Cicchetti, and Toth, 2009). In this study, the deviations from normative development, as indexed by altered cortisol and/or testosterone diurnal rhythms as a function of sexual abuse, can inform us of the developmental processes of adaptation or maladaptation following such trauma in an at-risk youth sample.

The Mechanisms

One particular way environmental stressors can shape development is by influencing biological mechanisms. Specifically, hormones that are implicated in the stress response and pubertal maturation are highly susceptible to environmental stressors. This vulnerability increases during adolescence due to an increase in organization and maturation of the brain, physiology, and behavior. Cortisol and testosterone are described below as biomarkers that are often examined as being responsive to the environment, as well as for their implication in pubertal maturation.

Hypothalamic-pituitary adrenal (HPA) axis and cortisol

The stress response system undergoes maturation during adolescence and is vulnerable to environmental stressors. Hormones released as part of the stress response (i.e., glucocorticoids, GCCs), such as the steroid cortisol, are produced by a cascade of signaling events in the hypothalamic-pituitary-adrenal (HPA) axis. When a stressful stimulus is perceived, the hypothalamus secretes corticotrophin-releasing hormone (CRH), which triggers the pituitary to release adrenocorticotropic-releasing hormone (ACTH). ACTH then travels to stimulate the adrenal glands to release cortisol into the periphery (see Figure 2.). Cortisol is one of the most commonly examined biomarkers in regard to stress physiology. Namely, the release of cortisol is deemed an adaptive response to context and the environment. The term "stress" was coined by Hans Selye, who is often referred to as the founder of modern stress research. Selye (1950) described the stress response as a process of detecting the stressor, reacting and resisting (i.e., coping), and finally exhaustion of the resources (see Sapolsky, Romero, and Munck, 2000). This response involves activation of the sympathetic nervous system (SNS), and the release of glucocorticoids from the adrenal cortex, which help provide energy for the response.

The general role of glucocorticoids is to regulate glucose concentrations in the periphery (Sapolsky, et al., 2000), and to ensure there is sufficient immediate energy for the system to respond to stressful stimuli. Cortisol is also in charge of restoring balance within the body (i.e., homeostasis), via its negative feedback loop, which signals the brain to discontinue the cascade and recover from the response (blue pathway in Figure 2.). The process of cortisol release is relatively fast, so the mechanism has the ability to release cortisol within minutes to hours in a given context. Indeed, the stressor can be physically and/or psychologically threatening, or novel and unpredictable to elicit a cortisol response (Dickerson and Kemeny, 2004; Sapolsky, et al.,

2000). The HPA mechanism is meant to provide energy to respond as adequately as possible, and later return to "normal". This property of the HPA is evolutionary, as Sapolsky (2004) illustrated the mechanism: being chased by a lion will increase glucocorticoid secretion to allow a gazelle to flee the predator by providing required resources, and subsiding once the threat is gone.
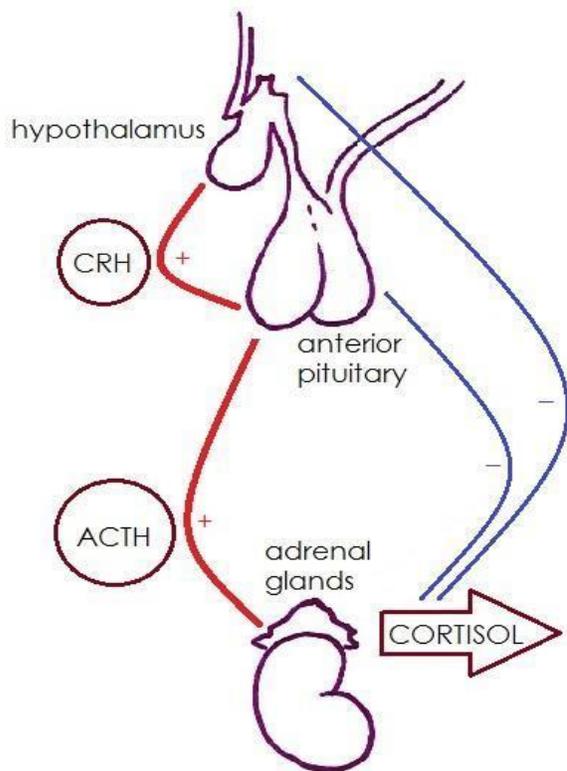
Figure 2. The Hypothalamic-Pituitary Adrenal (HPA) Axis

A sufficiently resource-charged response will allow successful escape from the predator (Sapolsky, et al., 2000). In humans, this applies to acute laboratory stressors such as the Trier Social Stress Test, Montreal Imaging Stress Task, and cold-pressor task, all of which are specifically designed to elicit an acute cortisol response and observe the recovery of the system

within a laboratory setting (Dickerson, and Kemeny, 2004). Other social contexts like public speaking and competition are often used as a form of social evaluative threat to psychological resources (e.g., self-esteem and image, etc.) and social status (Gruenewald, Kemeny, and Aziz, 2006). In the case of exercise, which requires energy for performance, salivary cortisol responds differently depending on duration and intensity of exercise (Jacks, et al. 2002; Kirschbaum, and Hellhammer, 1994).

In response to childhood trauma, however, the response can be different, especially if the experience is long-term and if coping resources are exhausted by the experience (Trickett, Noll, Susman, Shenk, and Putnam, 2010). This is where complexity arises: in the case of early adversity and childhood trauma, many children will acutely respond to adversity as expected when the trauma is novel (i.e., increase in cortisol), but their HPA can fail to recover because the stress is long term (Susman, 2006). The disruption of the negative feedback signal can result in excessive secretion of cortisol, leading to disarray of the mechanisms which appropriately respond to stress and restore balance. Over time, the excess of cortisol and "malfunction" of this signaling can lead to hypocortisolism, which is indicative of chronic stress (Badanes, Watamura, and Hankin, 2011). For example, war experiences are related to hypocortisolism, similarly to exposure to physical abuse (Heim, et al., 2000; Carpenter et al., 2011). Importantly, sexual abuse is often considered a long-term form of trauma and is associated with hypocortisolism as well (Trickett, et al., 2010). Chronic stress is important in the present study when considering the long-term aspect of sexual abuse trauma (Trickett, Kurtz, and Noll, 2005). For this reason, the stress response is almost expected to exhaust itself when exposed to long-term production of cortisol without signaling of the negative feedback, and therefore lead to hypocortisolism. This can result in two different outcomes: first, the systems that have been exhausted by prior

experiences are often unable to mount a response to acute stressors, potentially because they have adapted to stressful environments (e.g., maltreatment). Second, since cortisol *release* is not dependent on the environment, in other words, it is responsive to stress but produced throughout the day regardless of the context, for inflammation and tissue repair among other things (e.g., Girod and Brotman, 2004), its diurnal rhythm can become altered by long-term exposure. This is noticeable particularly in the way cortisol decreases toward the end of day (i.e., flatter or steeper slopes, Miller, et al., 2007) or in the cortisol awakening response (Wust, Wolf, Hellhammer, Federenko, Schommer, and Kirschbaum, 2000). Excessive secretion of cortisol can lead to dysregulation of the stress response mechanism, and has been associated with psychopathology (Burke, Fernald, Gertler, & Adler, 2005). Because cortisol secretion rises with respect to pubertal stage (Kiess, et al., 1995) and is often related to vulnerability to psychopathology when the system is disrupted (McEwen, 2004), girls are especially prone to depression during adolescence (Gunnar, Wewerka, Frenn, Long, Griggs, 2009). Cortisol plays a significant role in coping with day-to-day stressors as well as life-threatening situations. This biomarker contributes to maintaining health and balance within the organism and is highly vulnerable to long term stressors particularly during sensitive periods such as adolescence. The stressful experiences that occur during a time when the brain, body, and behavior are undergoing maturation can significantly shape proximal and future health outcomes.

Hypothalamic-pituitary gonadal (HPG) axis and testosterone

The present study examines salivary testosterone as a biomarker of pubertal development in an incarcerated adolescent sample. In addition to being a driver of sexual and brain maturation, testosterone has been linked to undesirable behavior in at-risk adolescents (Vermeersch, T'Sjoen, Kaufman, and Vincke, 2008). Testosterone is an androgen produced by

the brain, gonads, and adrenal gland. It is a product of the hypothalamic-pituitary-gonadal (HPG) axis. The HPG axis (see Figure 3.) begins in the hypothalamus, similarly to the HPA axis. The hypothalamus releases gonadotropin-releasing hormone (GnRH), which triggers the anterior pituitary to release follicle-stimulating hormone (FSH) and luteinizing hormone (LH). FSH and LH are important players in fertility and the menstrual cycle. The gonads then release testosterone to target cells. Although testosterone is typically assumed a masculine hormone, strictly produced by men, it is produced in smaller amounts by the ovaries and adrenal glands of women, as well (Angold, Costello, Erkanli, and Worthman, 1999). This biomarker demonstrates a diurnal rhythm similarly to cortisol, with highest levels in the morning, decreasing toward mid-day, and lowest in the late evening for both women and men (Ankarberg, and Norjavaara, 1999; Dabbs, 1990). In adolescent girls, testosterone varies based on the developmental stage. For example, girls in early stages of puberty have significantly lower testosterone compared to mid- and late stage pubertal girls (Ankarberg, and Norjavaara, 1999). Estradiol is the primary estrogen responsible for breast development, stimulating ovulation and menstruation, fat distribution and bone adjustment in female growth spurt (Shirtcliff, Dahl, and Pollak, 2009). However, testosterone is also a significant biomarker for girls, as it has been associated with problem or delinquent behavior (Granger, Shirtcliff, Zahn-Waxler, Usher, Klimes-Dougan, and Hastings, 2003), poor family constellations, presence of fathers, as well as depression (Booth, Johnson, Granger, Crouter, and McHale, 2003; Angold, et al., 1999). In adolescent boys, testosterone is responsible for pubertal development, but also has been associated with delinquency and violent behavior (Dabbs, Jurkovic, Frady, 1991). Specifically, high testosterone levels are related to more violent crimes of late-adolescent boys (Dabbs, et al., 1991).
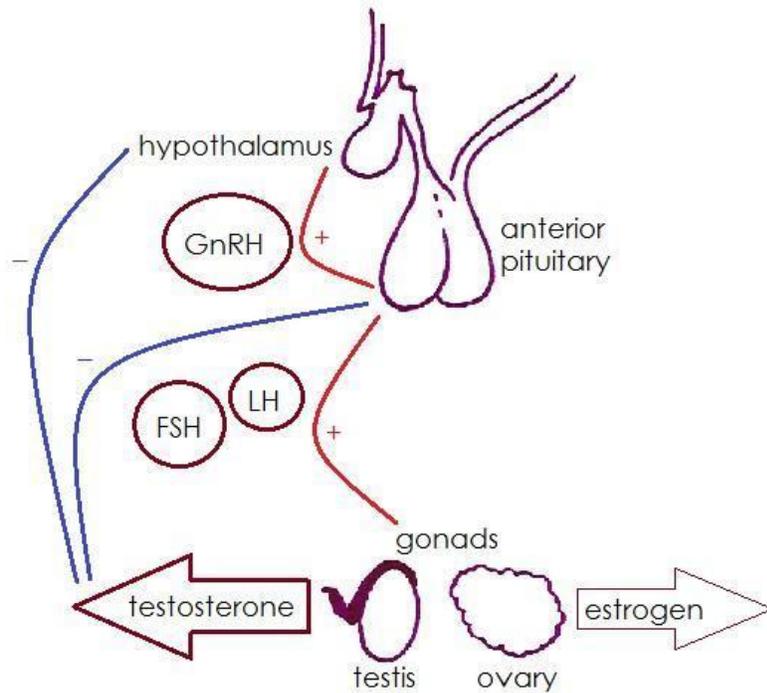
Figure 3. The Hypothalamic-Pituitary Gonadal (HPG) Axis.

Not much is known about the impact of early adversity on the testosterone profile. The more commonly investigated relationship is between testosterone and risk taking, delinquent behavior, antisocial behavior (Susman, et al., 2017; Dabbs, et al., 1991), as well as conduct disorder, which have been associated with higher levels of testosterone in girls (Pajer, Tabbah, Gardner, Rubin, and Czambel, Wang, 2006). The extant literature is not clear on the acute response and regulation of sex hormones particularly during adolescence, much less in developing girls. The common association of testosterone is with behavioral outcomes such as aggressive behavior (Vermeersch, T'Sjoen, Kaufman, and Vincke, 2008), responses to competition and challenge in young and adult populations (Schaal, Tremblay, Soussignan, and Susman, 1996). Although research on testosterone in adolescent female populations is scarce, this biomarker remains an important hormone related to reproductive maturity specifically as it is

known to rise during puberty (Ankarberg, and Norjavaara, 1999). Testosterone has also been associated with problem behavior in girls, especially during adolescence (Granger, et al., 2003) and for this reason is a biomarker worth examining given the context and characteristics of this sample (i.e., incarcerated).

Testosterone and cortisol were chosen for the important roles these biomarkers have in physiological functioning as well as development, particularly during adolescence and pubertal maturation. Testosterone rises with respect to pubertal stage, and is associated with problem behavior in adolescent girls and boys (Granger, et al., 2003; Dabbs, et al., 1991). While cortisol also rises with respect to pubertal stage (Kiess, et al., 1995), its mechanism is highly responsive to long-term trauma.

The diurnal rhythm

Cortisol and testosterone mechanisms are highly influenced by prior developmental experiences. Their variation across the day can be shaped by short-term and long-term experiences. Cortisol and testosterone follow a circadian rhythm regulated by the hypothalamus (Chan, and Debono, 2010; Dismukes, et al., 2015). Without any disruptions to the mechanisms, concentrations of these hormones are low at night, sometimes hardly detectable, and slowly build up to peak following awakening in the morning. During the day, cortisol and testosterone steadily decrease toward the evening (Chan, and Debono, 2010; Dabbs, 1990). Prior research on the adolescent circadian rhythm indicates that a stable cortisol rhythm is established during adolescence. Specifically, cortisol appears to be high during childhood, but "dips" for approximately two years before puberty (Shirtcliff, et al., 2012). This change in cortisol rhythm is associated with chronological age and preparation for puberty. It can also be associated with maturation of social and biological mechanisms and thus vulnerability of the adolescent

transition. In adolescent girls, the rhythm differs based on stage of pubertal development; pre-menacheal girls have lower post awakening cortisol, compared to girls who are post-menarcheal, or in mid to late puberty (see Figure 4.; Oskis, et al., 2009).
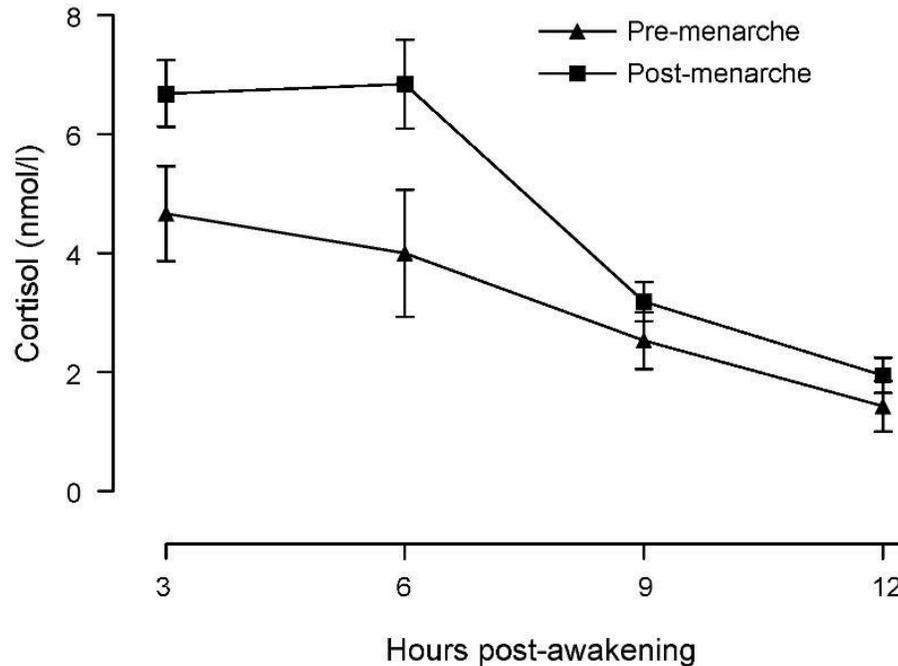
Figure 4. Salivary cortisol concentrations in pre-menarcheal and post-menarcheal girls (Oskis, et al., 2009)

This rhythm is established sometime in childhood (Price, Close, and Fielding, 1983). Although infants do not necessarily exhibit cortisol responsivity to acute stressors, however this can be dependent on caregiving. The stress response is formed in the early years of life so factors such as sensitive and responsive care, and attachment style can have an impact on a child's stress response mechanism (Gunnar, and Quevedo, 2007). Secure attachment, as well as sensitive and responsive caregivers can serve as buffer from stressors early in life.

There appears to be a difference in rhythms between developing girls and boys, with girls having higher cortisol, steeper slopes, and more curvature to their rhythm compared to boys (Shirtcliff, et al., 2012). Girls' circadian rhythms become flatter as they advance through

puberty. Such differences can be attributed to timing of pubertal development, as girls often undergo puberty before boys. Notably, adolescent delinquent boys have an altered cortisol diurnal rhythm (Popma, Doreleijers, Jansen, Van Goozen, Van Engeland, and Vermeiren, 2007). Diurnal cortisol also varies with respect to BMI, in some cases higher BMI is associated with higher cortisol throughout the day (Oskis, et al., 2009), whereas in other studies higher BMI has been associated with lower morning cortisol, and flatter slopes in girls (Shirtcliff, et al., 2012).

Altered cortisol rhythms demonstrated through changes in cortisol awakening response (CAR), slope, or heightened overall day or evening cortisol can be indicative of exposure to long lasting adverse experiences (Koss, et al., 2016; Koss, Hostinar, Donzella, and Gunnar, 2014), such as abuse (Trickett, Noll, Susman, Shenk, and Putnam, 2010). This effect further extends when considering the negative impact stressful experiences such as child sexual abuse can have on future health as described in more detail in Chapter Three. Higher levels of testosterone are typically associated with aggression, poor family relationships, delinquency (Susman, Peckins, Bowes, and Dorn, 2017), and antisocial behavior (Susman, et al., 2017; Archer, 2004) which are potentially important factors in the present study. Extant research assumes acceleration in pubertal development for girls with history of child sexual abuse (Noll, et al., 2017), however, evidence is inconclusive for boys, and the maturational timing can vary depending on the types and severity of abuse (Wind, and Silvern, 1992). Cortisol secretion changes in adolescence compared to childhood, as the HPA axis becomes more established (Keiss, et al., 1995), while testosterone production typically rises during puberty and along with other hormones awakens pubertal development (Matchock, Dorn, and Susman, 2007). Alterations in diurnal levels of cortisol and testosterone can be indicative of disruptions in development, causing significant deterioration of health in males and females.

CHAPTER THREE: LITERATURE REVIEW

The following review of the literature will begin with an overview of the importance of studying adolescence as one of the most challenging transitions in the lives of developing youth. Changes in the brain and physiology are described to illustrate the complexity of this crucial period in the lifespan, and demonstrate the malleability of biobehavioral mechanisms during adolescence. Following the overview of some of the changes that occur during period, I cover the literature on early adversity and the great immediate and extended impact it can have on physiology. This chapter will provide background information on each of the components of the model (refer back to Figure 1.). Finally, I will conclude the literature review by describing the research specifically focusing on the effect of early adversity on biomarkers such as cortisol and testosterone. I will stress the importance of examining the effect of sexual abuse on girls and boys who have recently undergone pubertal maturation by examining the way their hormones change and adapt following sexual trauma. The present examination will follow the overarching assumption of life course theory, that unique events alter the developmental trajectory of individuals, while also considering the importance of examining outcomes through a biopsychosocial lens. The narrative will include underlying assumptions of developmental psychopathology, considering the characteristics of the present sample.

Importance of Studying Adolescence

The sample used in the present study consists of incarcerated adolescent girls and boys, which provides great opportunities for investigating the potentially harmful environmental influences that have altered developmental trajectories. This sample is especially suitable for examining the effects of harsh environments on developmental processes. Across developmental periods, adolescence is viewed as a particularly demanding developmental stage for many youth.

The environment and biology interact to make this period challenging. This stage begins with puberty and reactivation of hormones which contribute to pubertal maturation (e.g., adrenal hormones such as cortisol, and sex hormones such as testosterone), which is why it is often described as a period of "raging hormones." Many biological, psychological, and social changes occur for developing individuals. Dahl (2004) describes adolescence as a time of resilience, decision-making, and reasoning improvement. Because adolescence begins with biological changes that jump-start puberty and is expected to end with attainment of adult roles and responsibilities, it is important to investigate this transition by examining the different environmental factors and biological outcomes.

Although most adolescents will transition through adolescence into adulthood without any lasting negative effects, there is a significant number of those who do not (Dahl, 2004). In such cases, the developmental and health outcomes become very complex depending on the timing, length, and type of adverse experiences. This is particularly important when considering the consequences of experiencing child sexual abuse on health and well-being (Noll, et al., 2017; Trickett, and Putnam, 1993). Girls, for example, can be impacted in a variety of ways such as experiencing early onset of puberty and developing depression (Angold, et al., 1999; Negriff, and Susman, 2011), and boys are likely to develop substance abuse problems (Holmes, and Slap, 1998). Adolescence is a developmental stage of heightened risk for developing problematic patterns such as internalizing and externalizing disorders (Steinberg, 2014). Internalizing disorders are particularly prominent in girls, although can exhibit in boys as well during this transition. Externalizing disorders such as aggression, hostility, callousness, and destruction of property are also common in adolescence (Dahl, 2004). Additionally, there is a possibility of developing problematic patterns such as depressive symptoms, which could persist into or recur

in adulthood (Pine, Cohen, Cohen, and Brook, 1999; Steiger, Allemand, Robins, and Fend, 2014). The stress of pubertal changing often brings about a heightened vulnerability for developing depression in many adolescent girls (Harkness, Bruce, and Lumley, 2006). This vulnerability increases even more if girls have been sexually abused (Trickett, Kurtz, and Noll, 2005). Many individuals who do not overcome adversity often remain in adverse environments or the consequences of the stressors recur in their adulthood. For example, such individuals may experience revictimization (Trickett, Noll, and Putnam 2011), become obese (McCarthy-Jones, and McCarthy Jones, 2014), or suffer from cardiovascular disease (Shonkoff, et al., 2011). More specifically, the experiences in adolescence will shape physiological, behavioral, social, and psychological outcomes in adulthood (Steinberg, 2014; Dahl, 2004).

Amid the psychosocial and physiological changes that occur in adolescence, the brain undergoes significant restructuring. The changes that occur in the brain during this time are complex. More specifically, there are changes that occur in the brain that offset the hormonal cascade to begin puberty, following which are changes that are a consequence of the pubertal maturation from increase in pubertal hormone levels, and finally, there are changes which are independent of puberty. The three different mechanisms can create internal asynchrony within the brain (Dahl, 2004). Importantly, early reproductive maturity or onset of developing of pubertal characteristics does not necessarily correspond with brain maturation (Dahl, 2004). This notion is often forgotten when attributing risky, sensation-seeking behavior to adolescents and their "raging hormones." Adolescents (and college-age youth) take more risks such as binge drinking, crime-related risks, and car crashes compared to children and adults (Steinberg, 2007). The long-standing assumption was that adolescents are irrational and unaware or not concerned about harms and consequences of the risky behavior. However, the adolescents' logical

reasoning is comparable to adults. Adolescents are able to estimate the risk and vulnerability to it just as easily as adults do (Steinberg, 2007). Preventive programs seem to be lacking in promoting positive behavior and raising awareness about consequences of risk taking (e.g., unprotected sex, substance abuse, etc.) for adolescents. Although adolescents have fairly well developed logical reasoning by the age 15, there seems to be a delay in psychosocial maturity. The psychosocial immaturity potentially undermines what could otherwise be adult-level decision making. Specifically, the psychosocial capacities such as impulse control, delay of gratification, resistance to peer influence and pressure, and emotion regulation are undeveloped and thus undermine the otherwise competent decision making (Steinberg, 2007). These are only several different mechanisms that are susceptible to environmental interference and deviation from the normative developmental trajectory. This information will be important when considering the fact that the sample is of incarcerated adolescent youth. Early adverse experiences can have significant effects on maturation, depending on the timing, duration, and intensity of experiences.

Puberty

Pubertal maturation is a set of dynamic processes of transitioning from pre-reproductive to reproductive stages of the life course. Although an important part of pubertal maturation, development of secondary sexual characteristics and attainment of reproductive maturity are not the sole milestones achieved during this time. Pubertal maturation is a rather tumultuous biological process, which brings about cognitive and physical changes that will be carried into adulthood.

Pubertal development is often measured through self-reported surveys or visual inspection of secondary sexual characteristics for clinical and research purposes. Most

commonly, visual assessment is used to determine stages of maturation according to Marshall and Tanner (1969, 1970). So-called "Tanner staging" involves visual inspection of the secondary sex characteristics, such as breasts and pubic hair growth, and genital development, as well as assessment of age at which the specific stage of development has been reached (Marshall, and Tanner, 1969). Adolescents are categorized into one of five Tanner stages based on how much development they have experienced. This type of examination is often done by clinicians or highly trained researchers. Conversely, the Pubertal Development Scale (PDS; Petersen, Crockett, Richards, and Boxer, 1988) is a self-report survey which asks adolescents if they have noticed specific changes to their body (e.g., height, hair growth, skin changes, etc.). The female version assesses breast development and time of menarche (i.e., first menstruation). The male version inquires about facial hair and deepening of the voice. The final score (1-4) determines stage of development (Petersen, et al. 1988). This type of assessment of pubertal maturation avoids discomforts of visual inspection, however may encounter issues with honesty in reporting. The PDS score can be converted to a 5-point scale corresponding to Tanner stages (Shirtcliff, Dahl, and Pollak, 2009).

In addition to assessment of secondary sexual characteristics, pubertal development is reflected in markers of physiological functioning. Namely, one of the first indicators of pubertal maturation is adrenarche, which involves increase in production of adrenal hormones such as dehydroepiandrosterone (DHEA), which contributes to skin changes and growth of pubic and axillary hair (Dahl, 2004; Shirtcliff, Dahl, and Pollak, 2009). Additionally, production of certain hormones such as estrogens and testosterone noticeably increases to stimulate and accommodate ongoing maturation. This increase in sex hormones is called gonadarche, and accounts for development of secondary sexual characteristics and brain reorganization (Dahl, 2004; Sisk, and

Zehr, 2005). The hormones that are abundant during this time account for reproductive maturation, but also for other physiological and behavioral changes during this time.

Potential disruption in successfully advancing through developmental stages is determined based on the timing and tempo of puberty. Specifically, if certain developmental milestones occur in discordance with age, maladaptation is thought to occur. Early puberty creates a discrepancy between physical and cognitive maturity, which inadequately prepares youth for navigating transition to adulthood (Noll, et al., 2017). Specifically, early puberty has been associated with behavioral and mental health problems, such as delinquency, depression, and early sexual debut (Noll, et al., 2017, Kaltiala-Heino, Marttunen, Rantanen, and Rimpelä, 2003). Early pubertal maturation brings about several notable risk factors in the lives of developing young women. For example, prolonged exposure to estrogens (due to early onset) is associated with fertility and menstrual problems in the future, as well as reproductive cancers such as ovarian and breast cancer. On the other hand, boys who undergo puberty earlier are likely to report more physical illness and symptoms, higher level of depression compared to on-time maturing boys, higher rate of tobacco use, and more emotional reliance on others (Graber et al., 1997).

Early life stress can lead to early adrenarche, as well as heightened HPA activity and thus excessive production of cortisol. Gonadal steroids can have modulating effects on the HPA axis, thus influencing recovery of cortisol release (i.e., negative feedback loop). As mentioned previously, adolescence is considered a stressful life stage in and of itself, therefore additional stressors can greatly interfere with the course of development. Notably, such interference can influence timing and tempo of pubertal development, which can in turn corrupt future health.

Early Adversity

Early life adversity refers to different life events which are considered stressful and have a negative effect on many aspects of health and wellbeing. Research has shown great impact early adversity can have down to the genetic level (e.g., Daskalakis, et al., 2013), that can also be represented in other markers of health like hormone levels, and mental health factors. More specifically, adverse experiences which occur during sensitive developmental periods can interfere by a form of "biological embedding" while prolonged adversities can impact health by accumulating over time (i.e., cumulative damage; Maccari, Krugers, Morley-Fletcher, Szyf, and Brunton, 2014). Different stressful life experiences such as child abuse and maltreatment have been attributed to changes in telomere length, altered HPA axis functioning, development of depression, chronic pain and fatigue, eating disorders (Monteleone, et al., 2017; Gunnar, and Quevedo, 2007), irritable bowel, as well anxiety and PTSD (Springer, Sheridan, Kuo, and Carnes, 2003). For these reasons, early adversity is considered one of the main contributing factors to overall developmental disruption that can also lead to health deterioration. Adverse experiences during early development can make long-lasting changes to biopsychosocial mechanisms. Therefore, examining biological and adaptive responses to adversity across different time points in development can inform intervention and prevention strategies for at-risk youth.

Sexual abuse

Child sexual abuse (CSA) is a complex adverse experience which can greatly contribute to dysregulation of psychological and biological systems. CSA varies in severity across reports in National surveys and the scientific literature, thus some of the associated factors may depend on type, duration, and intensity of the sexual abuse, as well as time since trauma occurred

(Putnam, 2003; Weems and Carrion, 2007). Due to the complexity of this experience, the definition of sexual abuse varies in reports, resulting in potentially underrepresented statistics on prevalence. According to the National Center for Victims of Crime (NCVC, 2017), 1 in 5 girls and 1 in 20 boys become a victim of child abuse, and the most vulnerable period appears to be between the ages of 7 and 13 years. In the United States, 16% of youth between the ages of 14 and 17 are sexually victimized in a one-year period, while 28% are sexually victimized over the course of their lifetime (Crimes Against Children Research Center, 2017). Some of the clinical outcomes of child sexual abuse include sexual dysfunction, depression (Bonomi, Cannon, Anderson, Rivara, and Thompson, 2008), and low self-esteem (Finkelhor, and Browne, 1985).

Generally, girls are at a higher risk (2-3 times) of becoming victims of sexual abuse (Putnam, 2003), the abuse is more often in a family context, and can occur at any time during early development- it can begin in infancy, prior to puberty, and occur over an extended period of time (Trickett, et al., 2005). Conversely, boys who experience sexual abuse are more likely perpetrated by males outside of their immediate family context (Holmes, and Slap, 1998). The information on health outcomes of male victims is scarce and outdated, likely due to underreporting. Trickett, Noll, and Putnam (2011) reported a host of biopsychosocial outcomes associated with sexual abuse in their intergenerational examination of the impact of sexual abuse on female development. Among those were outcomes closely related to the present examination (i.e., development and hormone outcomes) such as early onset of puberty, depression and DSM diagnoses, altered HPA axis responses, maladaptive sexual development, early motherhood, and revictimization (Trickett, Noll, and Putnam, 2011).

There are two aspects of sexual abuse which are important to consider: firstly, this is often a repeated trauma, although it could be a single-occurrence experience (Trickett, et al.,

2005). Secondly, the impact of sexual abuse will depend on proximity of trauma, in other words, physiological outcomes may vary depending on time since trauma was experienced. For example, there can be a difference in PTSD symptoms and cortisol levels in people reporting distant or recent traumas (Weems, and Carrion, 2007). Research on sexual abuse survivors follows two traditional methods based on proximity of trauma. Cross-sectional designs are used in research on children and adolescents soon after the trauma has been identified. This type of research can be helpful in capturing change across development following the trauma. However, such studies often disregard age or developmental stage as potential confounds. Specifically, the intensity or duration of abuse may differ depending on age of the victim (Trickett, et al., 2005). The second commonly used design is the retrospective form of investigating sexual abuse. Retrospective designs are valuable for determining effects in adulthood, however, these types of investigations often face confounding effects of memory (Trickett, et al., 2005). The commonly noted drawback of retrospective reports is the potential for poor memory of the experience, which can impact wholesome report collection. Because of the different ways it can be examined, the sexual abuse literature has yielded mixed findings on the impact this sort of trauma has on physiological health. However, it is certain that time since trauma, and time of assessment is crucial for investigating the impact sexual abuse has on physiology and mental health. Interestingly, depending on age of reporters, what is considered and reported as trauma by children and adolescents can vary, with adolescents reporting more severe traumas (Taylor, and Weems, 2009). Considering the age of the sample used in the present study, assessments were conducted relatively soon following the trauma, as the sample consists of adolescent reporters. Close proximity of trauma is expected to yield results indicative of the earlier stage (i.e., proximal) of impact of sexual abuse on hormones and development.

Several decades ago, Finkelhor and Browne (1985) provided a framework for understanding problems resulting from history of sexual abuse from a clinical lens, by introducing four broad traumagenic dynamics. The four traumagenic dynamics specify how and why child sexual abuse is traumatizing to individuals, and how it impacts their psychology and development. First, *traumatic sexualization* refers to shaping of the child's sexuality via inappropriate and dysfunctional strategies of manipulation. The child learns through the sexual abuse experience and the perpetrator about sexual behavior and sexual morality, which can then result in future inappropriate sexual behaviors, confusion regarding associations to sexual activities and emotion, as well as misconceptions about the sexual self (Finkelhor, and Browne, 1985). The second dynamic is *betrayal*, which refers to the feeling that occurs when the child realizes that the perpetrator who they depend on is causing them harm. This is particularly harmful if the perpetrator is a family member who the child greatly depends on. Betrayal can also come from a significant person who the child trusts and is supposed to be protecting them from harm. This is often a family member who may not believe or disregards reports of abuse for whatever reason (Finkelhor, and Browne, 1985). Indeed, the most commonly reported perpetrators are family members, close relatives, or acquaintances (Negriff, Schneiderman, Smith, Schreyer, and Trickett, 2014). Maternal support is crucial in children's adjustment following disclosure of sexual abuse, in terms of lowering potential for developing internalizing and externalizing disorders, depression, or PTSD (Zajac, Ralston, and Smith, 2015). Incidentally, parental discord, domestic violence, divorce, and not living with both parents are predictors of child sexual abuse (NCVC, 2017). The third dynamic is *powerlessness* or disempowerment, which refers to the process of rendering the victim powerless by continually disregarding the child's desires, sense of efficacy, and will (Finkelhor, and Browne, 1985). This includes repeated

invasion of the child's body and space against their will, which can be exacerbated by coercion and manipulation. Additionally, powerlessness includes attempts of disclosure which are not taken seriously, as well as any situation in which a child feels trapped, that may or may not involve force or threat. Powerlessness does not have to stem from the abuser, particularly in case of attempts of disclosure. The final traumagenic dynamic is *stigmatization*, which refers to the guilt, shame, and negative connotation that are communicated to the child through the abuse, and are later fused into their self-image (Finkelhor, and Browne, 1985). Shame and guilt are conveyed by the perpetrator and pressure of secrecy, or inferred from family, community, and societal assumptions about sexual abuse. The environment can play a significant role in imposing negative characteristics of the victims such as referring to them as "spoiled goods." Each of the four dynamics are associated with specific observed outcomes of CSA. Traumatic sexualization can lead to confusion about sexual identity, inappropriate sexualization or physical abuse of own children (i.e., intergenerational), confusion about sexual norms and standards, as well as using sex to obtain and give affection (Finkelhor, and Browne, 1985). Stigmatization leads to feeling of isolation from society, potential involvement in drug and alcohol abuse, as well as criminal activity and prostitution. Betrayal is associated with extreme dependency and trust insecurity, particularly in younger victims, as well as impaired judgement. Hostility and anger can be a response to betrayal, and aggression is a common response especially in adolescents. Antisocial behavior and delinquency are also associated with history of victimization (Gold, Sinclair, and Balge, 1999). Powerlessness can result in anxiety and fear, nightmares, somatic complaints, clinging behavior, and hypervigilance (Finkelhor, and Browne, 1985).

Finally, individuals with history of sexual abuse are more likely to experience revictimization in adolescence and adulthood (Barnes, Noll, Putnam, and Trickett, 2009; Krahé

et al., 1999). In fact, Trickett, Noll, and Putnam (2011) suggest that individuals with history of sexual abuse are about twice as likely to experience sexual or physical revictimization during later adolescence or early adulthood. There are factors that could predict resilience in sexually abused adolescents. Williams and Nelson-Gardell (2012) found that school engagement, social support provided by the caregiver, caregiver education and socioeconomic status (SES), as well as hope and expectancy are important in supporting adolescents and fostering resilience following sexual abuse.

The present study marks sexual abuse as the unique transformative experience for the developmental trajectory. It is not typically the case for events such as sexual abuse to be considered modifiers of a trajectory within the life course perspective. However, there is evidence indicating that experiencing sexual abuse in early life can significantly impact girls' development over time. The present investigation aims to determine whether this is true for male adolescents as well, by examining the effect of sexual abuse on hormones in both sexes. Importantly, Trickett and Putnam (1993) underline three major ways in which sexual abuse can impact the pubertal development and transition into adulthood for girls. First, the changes girls undergo as part of pubertal maturation, specifically sexual maturation, can become overwhelmingly stressful when paired with sexual abuse. This is particularly important when considering the notorious vulnerability to psychopathology (e.g., depression) during adolescence, which can be enhanced by sexual abuse. Second, a crucial component of a successful transition is support provided by family and peers (Trickett, and Putnam, 1993). Since victims often know the perpetrator, this can greatly impact their social support network, and thus how girls manage the undergoing changes. This is particularly effective when the perpetrator is closely tied to the girl, such as a father figure or other family member, which can drive a wedge between the victim

and the rest of the support providers (e.g., mother) and thus rid the girl of social support during an already stressful period of development (Trickett, and Putnam, 1993). Finally, sexual abuse can trigger early onset of puberty. Experience of sexual abuse can interfere with the signaling of hormones implicated in pubertal maturation. The available research has found that the timing and duration of puberty will depend on the type of childhood abuse. Specifically, child abuse can delay or jumpstart pubertal maturation (Zabin, Emerson, and Rowland, 2005). However, sexual abuse has been shown to jumpstart pubertal maturation specifically in girls (Belsky, et al., 2015; Zabin, et al., 2005). Early maturation is thought to be an evolutionary response to sexual abuse, preparing the girls to reach reproductive maturity in order to escape the harmful environment (Noll, et al., 2017). It is assumed that sexual abuse speeds up the pubertal process as a form of adapting to the negative environment, and quickly attaining resources for independence and reproductive success (Noll, et al., 2017). Early pubertal timing has been associated with early sexual debut, depression, and delinquency in girls (Negriff, and Susman, 2011). Sexual abuse can highly impact biological mechanisms by triggering early onset of puberty, however its effects on the male developmental trajectory have yet to be determined.

Markers of Exposure and Development

As stated previously, the two biomarkers chosen for the present study have very important roles in adolescent development and overall physiological functioning across the lifespan. Cortisol is highly responsive to context and environment, but excessive secretion can also have great future health ramifications. Changes in the cortisol production are manifested through altered diurnal rhythms of the hormone, as well as responses to acute stressors (e.g., laboratory, exercise, etc.). Because cortisol is so sensitive to environmental influences and can fluctuate over the course of minutes, the length of exposure can alter the mechanism. Such an

alteration can be caused by prolonged exposure to adverse experiences or trauma, such as sexual abuse.

The alteration in diurnal cortisol becomes complex when considering proximity of trauma (Weems, and Carrion, 2007). Trauma can alter HPA regulation and result in steeper declines during the day, and higher cortisol in the evening, specifically in youth with PTS symptoms (Weems, and Carrion, 2008). Cortisol levels may be elevated for a period of time following CSA trauma, but will be blunted in adulthood (Trickett, et al., 2011). Retrospective investigation of CSA shows hypocortisolism in adults who reported having experienced sexual abuse in childhood. This change in cortisol secretion potentially serves an adaptive role, preventing the system from overflowing with cortisol, and thus having detrimental effects on health (Trickett, et al., 2011). Capturing the change in cortisol profiles soon after trauma has occurred would provide insight into how the mechanism adapts. Additionally, the sample in the present study can yield novel or unexpected results, considering their current environment (i.e., incarceration), history of sexual abuse, and developmental stage.

Changes in cortisol profiles that are closely related to sexual abuse are associated with mental and physical health outcomes. More specifically, history of child sexual abuse can fuel onset of depression (Bonomi, Cannon, Anderson, Rivara, Thompson, 2008) or substance abuse (Putnam, 2003), disrupt the HPA axis and result in prolonged secretion of cortisol, suggesting chronic stress exposure (Trickett et al 2010), and lead to anxiety and post-traumatic-stress (PTS, Trickett, et al., 2005). Factors such as socioeconomic status (SES) and family constellation add to the risk of child sexual abuse (Putnam, 2003), and are associated with hypocortisolism, as well as development of psychopathology (McCarthy-Jones and McCarthy-Jones, 2014). Similarly, factors such as early adversity, callous unemotional traits, and consequent development of

antisocial behavior are associated with low levels of cortisol as well (Hawes, Brennan, and Dadds, 2009).

The existing literature on potential disturbance of testosterone, one of the main androgens that spikes during puberty, has mixed findings and requires further investigation, particularly in populations that have been exposed to sexual abuse. Prior research has predominantly focused on testosterone in maturing adolescent boys, as testosterone levels are higher in males, however this hormone rises during pubertal maturation in girls as well (Susman, et al., 2017). Low levels of testosterone have been associated with adolescent girls' poor relationships with mothers and consequent risky behavior, as well as poor relationships with their fathers and depressive symptoms (Booth, 2003). Additionally, steep decline in testosterone across the day has been associated with disruptive behavior problems in girls (Granger, et al., 2003). The handful of studies on testosterone in girls point to intriguing associations between testosterone profiles and behavioral outcomes. However, there is still a lot to be examined specifically in adolescents who have experienced early adversity. Although testosterone is significantly higher in boys, girls' testosterone can provide insight into their physiological functioning following sexual trauma, especially considering the role of testosterone in reproductive maturation and problem behavior for both sexes.

In male populations, high testosterone has been associated with violent crimes and aggression (Dabbs, et al., 1991). More specifically, much of the literature on male adolescents examines the relationships between pubertal maturation and violence (Felson, and Haynie, 2002) with evidence suggesting the important role of social factors in the maladaptive outcomes of risk-taking and delinquency as a result of succumbing to peer pressure (Felson, and Haynie, 2002). Additionally, pre-teen boys that are perceived as more dominant have higher testosterone

compared to their less socially dominant counterparts (Schaal, Tremblay, Soussignan, and Susman, 1996). In this case, 13 year-olds with history of physical aggression have lower testosterone compared to boys who have no history of physical aggression (Schaal, et al., 1996). This evidence may suggest the important role of pubertal timing in examining the link between testosterone and aggressive behavior.

Looking at specific biomarkers in adolescence is crucial for examining how unique events alter the normative developmental trajectory. Among many biomarkers implicated in the changes that occur during adolescence, cortisol and testosterone are especially important. Although the two biomarkers are important in all stages of the lifespan, they are closely related to the period of adolescence and some of the factors examined in the present study. Examining the cortisol mechanism in adolescence is relevant because it is particularly vulnerable to changes caused by the environment that can greatly impact mental and physical health in the future. Although girls' testosterone studies are few, it is difficult to deny the importance of studying this biomarker of puberty, sexual functioning, and problem behavior in adolescent girls who are incarcerated. Especially when considering the associations testosterone has been found to have with behavior problems and poor familial relationships in girls (Booth, 2003; Granger, et al., 2003). Similarly, the association between risk-taking, aggressive and delinquent behavior and testosterone has been established in boys, particularly within the sensitive period of adolescence (Dabbs, et al., 1991, Reynolds, et al., 2007). It is clear that adverse environmental factors have a grave impact on the normative trajectory of adolescent development, particularly though changes that are reflected in biomarkers. Taken together, cortisol and testosterone can provide insight into the physiologic response to relatively proximal sexual abuse trauma in developing youth.

Hypotheses

The present study tested whether history of sexual abuse has an effect on cortisol and testosterone profiles of incarcerated adolescent boys and girls. Gender is considered as a potential moderator of the effect, while pubertal stage, BMI, and race were tested as control variables. This study tested the following hypotheses.

1. **Diurnal cortisol will be associated with sexual abuse.** The literature indicates that female survivors of sexual abuse have attenuated cortisol levels. Considering the context of data collection, and the effect sexual abuse trauma can have on HPA axis functioning, it is expected that sexual abuse will be a significant predictor of variation in diurnal cortisol. Research examining HPA functioning in adolescent boys as a function of sexual abuse is scarce.

2. **Diurnal testosterone will be associated with sexual abuse.** Prior research indicates that there is an association between testosterone levels and problem behavior and delinquency in adolescence. Considering the characteristics of the present sample, it is expected that the sample will exhibit high testosterone levels. Because of the role testosterone plays in reproductive maturation and social status, testosterone is expected to be associated with sexual abuse.

3. **Gender will moderate links between sexual abuse and hormone levels.** Prior research indicates that there are gender differences in hormone levels and diurnal patterns. Additionally, there have been studies indicating different outcomes of sexual abuse based on gender. Therefore gender is considered as a potential moderator of the relationship between sexual abuse and hormones.

CHAPTER FOUR: METHODS

Sample

The present study used secondary data from a study of incarcerated adolescent youth. This dataset consists of youth (*N*=101, 49.5% girls), ages 13-18 years (M=16.17), 34% White and 54% of other ethnicity, from multiple correctional facilities across the United States. While boys' data was collected from one institution, the girls' data came from four different institutions.

Procedure

The University of New Orleans IRB approved the study (see Appendix for approval letter). Parental and individual assent were obtained for all participants prior to participation in the study. Data collection was conducted following a 1-2 week period of acclimation following admission to the facility to minimize effects of the incarcerated setting. Collection occurred over the course of three days, on two of which saliva samples were collected along with the Daily Diary, and on the third, the Life Stress Interview (LSI) was conducted and other self-report measures were administered.

Measures

Childhood Trauma Questionnaire (CTQ)

The Childhood Trauma Questionnaire (CTQ, Bernstein, and Fink, 1998) is a 28-item inventory designed to measure childhood and adolescent abuse. It can be scored on several different subscales of abuse or neglect: emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect, as well as denial of abuse or minimization. The participants are asked to provide a score from 1 to 5 based on the frequency of occurrence, where 1 is "Never

True" and score of 5 is "Very Often True." This measure can be used for assessing trauma exposure in adolescents ages 12 and above. The Sexual Abuse subscale contains five items, for which scoring ranges between 5 ("Never True") to 25 ("Very Often True") and include items such as "I believe that I was sexually abused", "Someone molested me", and "Someone threatened to hurt me or tell lies about me unless I did something sexual with them." The reliability of the CTQ in the present study was excellent (α=.97) and reliability of sexual abuse subscale was good (α=.79). Sexual abuse scores for boys ranged from 0 (no abuse) to 14 (M=1.26, SD=3.19) and for girls between 0 (no abuse) and 20 (M=5.84, SD=7.12). Out of 101 youth in this sample, 38 (37.6%) reported sexual abuse, with 12 (23%) out of 51 boys and 26 (52%) out of 50 girls reporting sexual abuse.

Pubertal Development Scale

The Pubertal Development Scale (PDS, Petersen et al., 1988) is a non-invasive measure of pubertal maturation in girls and boys. It consists of items used to determine growth spurt, presence of body hair, breasts and menstruation in girls, and facial hair and deepening of voice in boys. The female version also inquires about date of or age at first menstruation. In addition to assessing weight and height, the scale asks if the respondent thinks their development is earlier or later compared to same-age peers. The PDS can be translated into five-point scale corresponding to Tanner stages (Shirtcliff, Dahl, and Pollak, 2009). The Pubertal Development Scale was converted to Tanner stages using SPSS. Average stage for girls was M=4.41 (SD=.64), and for boys M=3.61 (SD=.80) in this sample.

Body-Mass Index (BMI)

Body mass index is a measure of body fat based on height and weight. A high BMI can be an indicator of obesity, which may lead to health problems. The BMI variable was centered at the 50th percentile to specifically examine individuals that would be considered overweight. In this sample, 37% of boys and 36% girls were at or above 50% percentile.

The Daily Diary

The Daily Diary is a questionnaire that corresponds each saliva collection or day of collection (Shirtcliff, Granger, Booth, and Johnson, 2005). It measures time of awakening, time of sample collection, medication use, exercise, moods, and daily hassles or uplifts. This measure provided time since wakening information used to create the time-varying variable corresponding to diurnal slope.

Saliva collection

Ten total saliva samples were collected from each participant on two separate days, one week apart. Five saliva samples were collected on each day during the following time points: upon waking, 30 minutes following waking, at 11:30 am, 5:30 pm, and at bedtime. The specific times were selected to account for the diurnal rhythmicity of biomarkers the saliva samples were later assayed for. The participating youth were on a strict waking, bedtime, and mealtime schedule which allowed for restricted collection times. Additionally, the two days of sample collection were separated by one week considering menstrual cyclicity.

Enzyme immunoassays

Cortisol and testosterone enzyme immunoassays (EIA) were performed by trained laboratory technicians in a Biosafety Level II laboratory at the University of New Orleans. Saliva samples were assayed in duplicate using well-established highly sensitive enzyme immunoassay (EIA) kits (Salimetrics, State College, PA) for salivary cortisol and testosterone. Saliva samples, standards, and unknowns were pipetted into wells of a 96-well microwell plate. The wells are coated with monoclonal antibodies for cortisol, and polyclonal antibodies to testosterone. Following incubation, unbound components are washed away in a plate washer. Bound cortisol or testosterone is then covered with tetramethylbenzidine (TMB) solution and reaction is stopped by sulfuric acid following 30 minutes of development. Optical density is read using a microwell plate reader at 450nm (Biotek™, Winooski, VT) and processed using computer software. The standards are known concentrations used to create a standard curve, which allows extrapolation the amount of cortisol or testosterone in the unknown samples (i.e., saliva samples) by comparing optical density to the standard curve. This is done for each plate, independently generating concentrations for each well, after which two wells that correspond to the same sample are averaged. In the case of significant differences between the duplicate well values, sample was re-assayed on another microwell plate.

Data analysis

Data were cleaned using SPSS v24.0 by centering the CTQ sexual abuse scale at score of 5, as this score indicates five "Never" responses to the subscale questions, therefore report of no abuse. Furthermore, cortisol and testosterone were log transformed, as this is common when hormone data is not normally distributed. The PDS scores were translated to Tanner stages using the Shirtcliff et al. (2009) method. BMI was centered at the 50th percentile in order to examine

the scores on the higher end of the scale. Hierarchical Linear Modeling (HLM; Raudenbush, Bryk, Congdon, 2004) was the primary analysis tool for testing the hypotheses in the present study. HLM allows for examination of within person factors (at level 1) and between-person factors (at level 2) through multilevel modeling which corrects for the inherent dependency in the data for having saliva samples nested within an individual. Level 1 has each sample of cortisol and testosterone, respectively in separate models, as the outcome with up to 10 observations on each individual. Time-varying predictors of the hormone were included at level 1 including time since waking and awakening response. Once the level 1 equation was validated, level 1 predictors were examined as the level 2 outcomes of interest using a slopes-as-outcomes approach (e.g., Shirtcliff, et al., 2012). By adding the time since waking variable, we are able to examine the changes in hormone levels as time progresses, therefore inferring the fluctuations in diurnal slope. Level 2 variables are specific to the individual, but can predict time-varying factors with a cross-level interaction. Sexual abuse was included as a predictor of the waking hormone level (captured with the intercept), the diurnal slope (captured with time-since-waking), and the CAR (captured with the sample 2 dummy variable). Gender was considered as a moderator of these relationships. In addition, BMI, race, and puberty were tested as control variables. Table 1 below demonstrates the base model used to test hypotheses in this study.

Table 1. HLM base model used to test hypotheses using log-transformed cortisol as example outcome in Level 1.

Level 1:

$$LNCORT_{ij} = \beta_{0j} + \beta_{1j}*(CAR_{ij}) + \beta_{2j}*(TSW_{ij}) + r_{ij}$$

Level 2:

$\beta_0 = \gamma_{00} + \gamma_{01}(SA) + \gamma_{02}(covariate) + U_0$	This equation examines if SA predicts the hormone levels upon awakening (β_0)
$\beta_{1TSW} = \gamma_{10} + \gamma_{11}(SA) + \gamma_{12}(covariate) + U_1$	This equation examines if SA predicts the hormone slope or diurnal rhythm (β_1)
$B_{2CAR} = \gamma_{20} + \gamma_{21}(SA) + \gamma_{22}(covariate) + U_2$	This equation examines if SA predicts the hormone awakening response (β_2)

CHAPTER FIVE: RESULTS

The data were analyzed in HLM in order to model the nested structure of the hormone data which has multiple samples (level 1) per individual (level 2).A base model was first constructed in which time-varying variables predicted each respective hormone at level 1 (refer back to Table 1). In Level 1 cortisol was predicted by time varying variables: the intercept, time since waking (TSW), and cortisol awakening response (CAR). The intercept is time zero, which corresponds to hormone levels at waking, TSW corresponds to the fraction of time that has passed since waking and each of the samples is matched to time of collection, resulting in a variable for diurnal slope. Finally, CAR is a dummy code corresponding to the second sample, collected approximately 30 minutes following waking (0= not CAR, 1=CAR). Testosterone was also predicted by waking cortisol, awakening response, and diurnal slope (i.e., time since waking). These predictors become outcomes of interest in level 2, following the slopes-as-outcomes approach. Table 2 below provides descriptive statistics by gender for the main variables examined in the following analyses.

Table 2. Descriptive statistics by gender.

	Boys (n=51)	Girls (n=50)	Total (N=101)
Age	14-18 (M=16.21)	13-18 (M=16.14)	13-18 (M=16.17)
Race (% Non-White)	62.7%	44%	54%
Sexual Abuse	12 (23%)	26 (52%)	38 (37.6%)
Puberty (stage 4-5)	21 (41%)	30 (60%)	51 (50.5%)
BMI (above 50%)	19 (37%)	18 (36%)	37 (36.6%)

Hypothesis 1: Effect of sexual abuse on cortisol

The null or blank model was constructed to determine SD values and calculate ICCs. This model simply had Log transformed cortisol as an outcome, and Log transformed testosterone as an outcome in separately tested models. For cortisol the between-individuals ICC was 26%, and 73% within individual variation.

To test the first hypothesis of whether sexual abuse (SA) was a significant predictor of waking hormone level, the diurnal slope, and the CAR for cortisol, SA was added as a predictor of each in the respective level-2 equations. SA had a significant main effect on the diurnal slope (i.e., time since waking), indicating a greater decrease in cortisol level across the day with each unit increase in sexual abuse (B=-.003, $p< .015$). SA did not have a significant effect on CAR (B=-.006, $p=.528$), and did not have a significant main effect on waking cortisol (B=.011, $p=.196$).

Next, I examined whether SA continued to have a main effect on diurnal slope and waking cortisol after controlling for gender. Gender had a significant main effect on the diurnal slope (B=.112, $p< .001$), such that girls had flatter cortisol slopes compared to boys. Gender was also a significant predictor of waking cortisol (B=.447, $p<.001$), such that girls had higher waking cortisol compared to boys. Gender was not a significant predictor of CAR (B=-.102, $p=.39$). After controlling for gender, SA continued to have a significant effect on diurnal slope (B=-.006, $p< .001$) such that SA was associated with steeper cortisol diurnal declines. SA main effects remained non-significant for waking cortisol and CAR. Because testing of the base model did not produce significant results for CAR, future models no longer loaded new covariates as predictors of CAR.

In prior research, sexual and physical abuse and other types of maltreatment have been reported to be highly correlated, raising possible concerns with multicollinearity. In this study,

SA was highly correlated with physical abuse (r= .602, p<.001), with physical neglect (r=.534, p<.001), with emotional neglect (r=.535, p<.001), and with emotional abuse (r=.533, p<.001). These associations suggest moderate collinearity, so in order to test if sexual abuse has a unique effect on the hormones, the SA subscale was modeled alone, in the same model as the physical abuse (PA) subscale, and then in the same model as a composite CTQ score with the exception of SA (i.e., physical abuse, emotional abuse, and physical and emotional neglect subscales).

Adding the CTQ composite of four subscales to the base model did not change effects of SA on diurnal cortisol (B=-.005, p< .05) or waking cortisol (*p= ns*). Gender remained a trend-level predictor of the diurnal slope and waking cortisol (*p*=.077).

Adding physical abuse to the base model did not change the effect of SA on diurnal cortisol, as SA remained a significant predictor (B=-.005, p <.05). However, addition of PA changed significance of gender effects on waking cortisol (B=.356, p=.047).

In order to disentangle the effects of sexual abuse from different types of abuse, the SA subscale was compared to the other four CTQ subscales. This was done in two ways: First, the composite of four subscales was loaded as a predictor of cortisol into the base model along with SA, additionally, since sexual abuse and physical abuse were most highly correlated, physical abuse was loaded as a predictor along with sexual abuse in a separate model. Second, given the emphasis on SA and the possible overlap of SA with other forms of abuse, an unstandardized residual variable was created by regressing physical abuse on sexual abuse. A similar residual variable was created by regressing the CTQ composite of the other four subscales on sexual abuse. These residual variables were controlled for in respective models (i.e., physical and sexual abuse, sexual abuse and composite).

The next model included sexual abuse, gender, and the residual PA score. Gender remained a significant predictor of diurnal slope (B=.092, p=.002), SA remained a significant predictor of the diurnal slope (B=-.005, p=.006), and the residual PA score did not have a significant effect on diurnal slope (B=.000, p=.855). The residual PA score also did not have a significant effect on waking cortisol (B=-.000, p=.987), the effect of gender on waking cortisol decreased to trend-level significance (B=.339, p=.060), and SA did not have a significant effect on waking cortisol (B=-.003, p=.827). This suggests that SA has an effect on cortisol's diurnal slope that is independent of PA, but PA does not add predictive value to cortisol's diurnal slope beyond SA.

The next model included sexual abuse, gender, and the residual computed by regressing the CTQ four- subscale composite score on sexual abuse. The residual did not have a significant effect on diurnal slope (B=-.000, p=.608) or waking cortisol (B=.000, p=.860). Gender remained a significant predictor of the diurnal slope, and reached trend-level significance in effect on waking cortisol (B=.330, p=.077). Sexual abuse was not a significant predictor of waking cortisol, but remained a significant predictor of the diurnal slope (B=-.004, p<.05), again suggesting that SA has a unique effect on cortisol's diurnal rhythm which is distinct from CTQ.

Table 3. HLM Analysis Results of Sexual Abuse, Physical Abuse, and CTQ Predicting Cortisol.

	Waking Cortisol		Diurnal Cortisol	
	B	p	B	p
Intercept	3.543	<.001	-.099	<.001
Gender	.356	.047[*]	.093	.002[*]
Sexual Abuse	.000	.990	-.005	.016[*]
Physical Abuse	-.008	.646	-.000	.984
CTQ Subscales	.000	.918	-.000	.597

* $p < .05$

The next step was to test if important covariates explain away the effects of SA beginning with race (0= White, 1=Other-Ethnicity) as a potentially significant covariate to consider. Race appeared to be have a significant effect on waking cortisol (B=-.264, p=.01), such that White adolescents had higher waking cortisol. Race was not a significant predictor of the diurnal slope (B=.014, p=.350). SA was not a significant predictor of waking cortisol (B=-.000, p=.617) but remained a significant predictor of diurnal slope (B=-.005, p=.004) after accounting for race. Finally, gender was not a significant predictor of waking cortisol (B=.307, p=.093) but remained a significant predictor of diurnal cortisol (B=.098, p<.001).

The next tested covariate was Body Mass Index (BMI). This covariate was included, considering that body fat can alter cortisol metabolism and its diurnal feedback to the brain. The model showed BMI as a significant predictor of waking cortisol (B=.007, p=.038), while gender did not have a significant effect on waking cortisol (B=-.100, p=.29). BMI did not have a significant effect on diurnal slope (B=-.000, p=.584), but the effect of gender remained significant (B=.101, p<.001). The effect of SA on diurnal slope remained (B=-.005, p=.002) after accounting for BMI.

In sum, SA had a significant effect on the diurnal slope after controlling for gender, race, BMI, and other types of abuse. SA predicted steeper cortisol slopes, but had no significant effect on CAR and waking cortisol. Girls had flatter slopes and higher waking cortisol, but there were no gender differences in CAR. These findings are consistent with the first hypothesis, which expected to find altered diurnal slope as a function of sexual abuse.

Hypothesis 2: Effect of sexual abuse on testosterone

The testosterone base model included testosterone predicted by waking hormone levels, the diurnal slope, and potential testosterone-awakening response (TAR) at level-1. The three

time-varying variables then became outcomes of interest at level 2 and were predicted by SA. Gender was later added to the base model, considering differences in testosterone levels between boys and girls. For testosterone, the between-individuals ICC was 45% and 54% for within individual variation.

To test the second hypothesis of whether sexual abuse (SA) was a significant predictor of testosterone, SA was added as a predictor of each in the respective level-2 equations. The base model indicated that SA had a significant effect on waking testosterone (B=-.028, p=.019) such that youth who had greater sexual abuse had lower waking testosterone levels. SA was not a significant predictor of TAR (B=.002, p= .476), or the diurnal rhythm (B=.000, p=.281).

The next step was to determine gender differences. Gender was a significant predictor of testosterone at waking (B=-.695, p< .001), and of diurnal testosterone (B=.021, p=.005) such that girls had lower waking testosterone and flatter diurnal slope. After gender was controlled for, SA effect on waking testosterone became nonsignificant (p=.766). Because there were no significant effects on TAR, the following covariates were not loaded as predictors of TAR at level 2.

Next, the same covariates that were considered in testing SA effects on cortisol were tested for testosterone. First, BMI was added to the base model, along with gender. BMI had a significant effect on diurnal testosterone (B=.0003, p= .048) such that higher BMI was associated with flatter testosterone rhythm. SA and Gender were not significant predictors of diurnal slope or waking testosterone in this model. Upon addition of Race as a control variable, gender remained a significant predictor of waking testosterone (B=-.600, p<.001), but gender was no longer a significant predictor of diurnal slope (p=.149). Race and SA were not significant predictors of waking testosterone or diurnal slope in this model.

Next, SA effects were examined with effects of other types of abuse, similarly to the cortisol strategy. In order to test whether there are unique effects of SA, physical abuse and CTQ composite scores were added individually to the base model. Addition of physical abuse as predictor of waking and diurnal testosterone did not change effect of sexual abuse, as both PA and SA were not significant. Gender remained a significant predictor of waking testosterone, such that girls had lower waking testosterone (B=-.581, $p<.001$). Addition of the PA residual variable created by regressing physical abuse on sexual abuse did not change the null effect of sexual abuse on testosterone. Interestingly, gender effect on testosterone diurnal slope became marginally significant (B=.014, $p=.067$). Addition of CTQ composite did not change SA effect on testosterone at waking or diurnal slope. Gender was no longer a significant predictor of diurnal slope at the $p<.05$ level (B=.013, $p=.071$). Gender remained a significant predictor of waking testosterone (B=-.592, $p<.001$). In testing the effects of the CTQ composite and its corresponding residual variable, the effect of SA, CTQ, and residual did not change. SA was not a significant predictor of diurnal slope. Gender was not a significant predictor of diurnal slope ($p=.085$). The CTQ composite and residual did not have a significant effect on waking testosterone or diurnal slope in this model.

In sum, SA had a significant main effect on waking testosterone, such that greater amount of SA was associated with lower waking testosterone. When controlling for gender, SA was no longer a significant predictor of waking testosterone. Girls had significantly flatter testosterone slopes and lower waking cortisol. There were no differences in testosterone when controlling for race, and high BMI was associated with flatter testosterone slopes. These findings were not consistent with the second hypothesis, as SA did not have a significant effect on testosterone.

Hypothesis 3: Examining gender as a potential moderator

To test the final hypothesis, a gender by SA interaction term was created then added as predictor to the base model for each hormone. The purpose of this was to determine if there are gender differences in the effect of SA on hormones (see Figure 5.). The first model included the interaction term in addition to gender and SA as predictors of waking cortisol and diurnal slope. The main effects of SA (B=-.004, p=.006) and gender (B=-.055, p=.002) on diurnal cortisol remained significant. The interaction between SA and gender had a trend-level (B=.002, p=.084) effect on diurnal slope indicating a potential overlap between gender and SA effects. These results indicated that boys had steeper cortisol slopes overall (B=-.055, p=.002), that increase in SA resulted in steeper slopes overall (B=-.004, p=.006) and that sexually abused boys had flatter slopes compared to girls, although this effect was only significant at trend-level (p=.084). The effect of gender on waking cortisol remained as in prior results, with boys having lower waking cortisol (B=-.237, p=.01). SA and interaction term did not have significant main effects on waking cortisol.

To test gender as a potential moderator in SA effects on testosterone, the SA x gender interaction was added to the testosterone base model. In this model SA, gender, and the interaction term did not have significant effects on diurnal slope. (ps>.12). SA and the interaction term did not have significant effects on waking testosterone (ps>.83), however gender had a significant main effect on waking testosterone (B=.287, p<.001), such that boys had higher testosterone compared to girls.

Finally, puberty was added as a potentially significant covariate to the base model, considering gender differences in development. In testing the effect of puberty on cortisol, SA remained a significant predictor of diurnal slope (B=.-005, p=.02), gender had a trend-level significant effect

on waking cortisol (B=-.172, *p*=.07) and a significant effect on diurnal slope (B=-.049, *p*<.001), such that boys had steeper cortisol slopes compared to girls. Puberty did not have a significant effect on waking or diurnal cortisol.

Cortisol in High and Low SA Males and Females
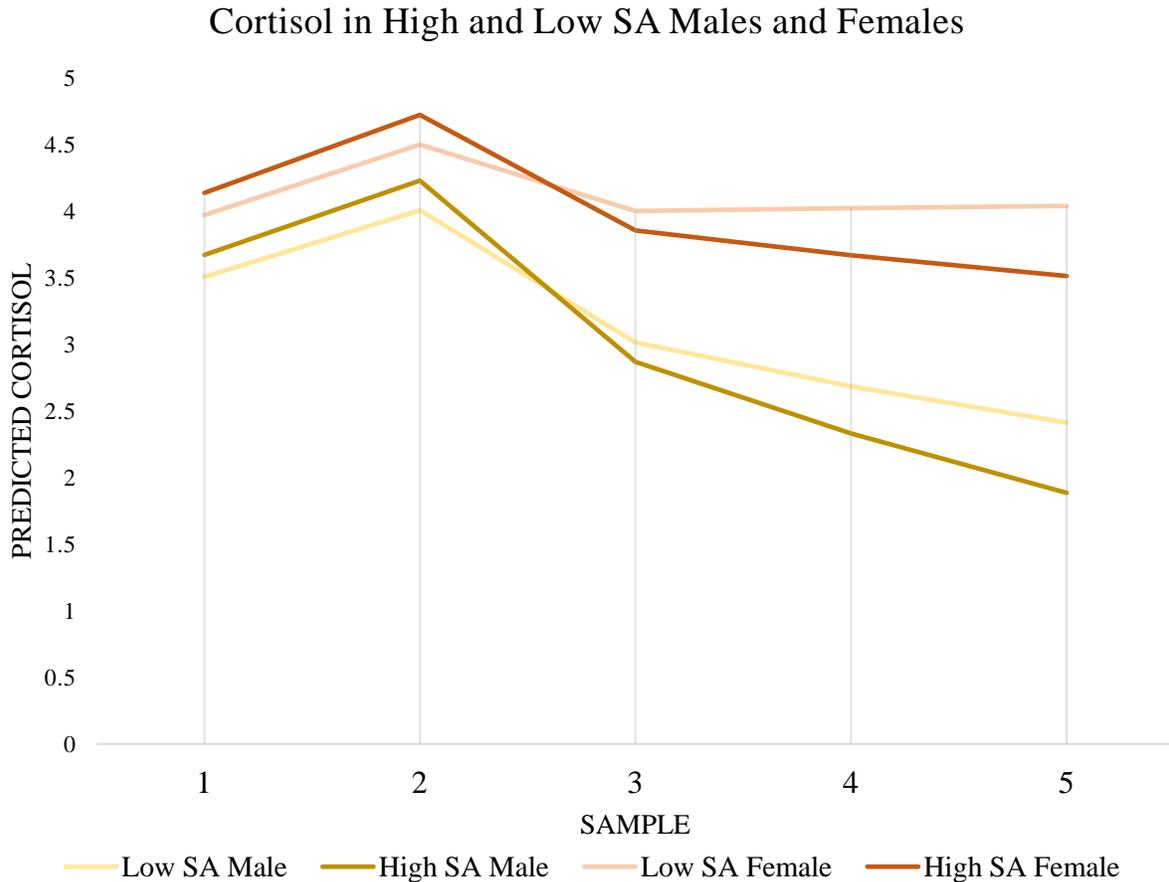
Figure 5. Cortisol in male and female adolescents with high or low sexual abuse.

In testing whether puberty has an effect on testosterone, puberty was added as a predictor to the testosterone base model. SA did not have a significant main effect on waking testosterone or diurnal slope, and puberty was not a significant predictor of waking testosterone. Gender remained a non-significant predictor of diurnal slope, and a significant predictor of waking testosterone, such that boys had higher waking testosterone (B=.290, *p*<.001). Puberty was not a

significant predictor of waking testosterone, but main effect on testosterone diurnal slope was significant at trend level (B=.007, *p=*.08).

In sum, testing the final hypothesis confirmed the effect of gender and SA on cortisol slope, with girls having higher waking levels and flatter slopes, and greater SA predicting steeper cortisol slope. There was no effect of puberty on waking cortisol or diurnal slope. Testing the effects of gender by SA interaction indicated that sexually abused boys had flatter cortisol slopes compared to girls, although this effect was only significant at trend-level. Addition of the interaction created a potential overlap between gender and SA effects on diurnal cortisol slope. Girls had lower waking testosterone and flatter testosterone slopes, while puberty had a trend-level effect on slope indicating flatter slopes with increase in maturation.

CHAPTER SIX: DISCUSSION

The present study examined the effect of sexual abuse on diurnal rhythms of cortisol and testosterone, by testing its effects on waking hormone levels, awakening response, and diurnal slope. Gender was a moderator of interest, while potential control variables included race, BMI, and puberty. Unique effect of sexual abuse was examined by testing the effects of other types of abuse. Sexually abused youth had steeper cortisol slopes, but no significant differences in CAR and waking cortisol. Girls had flatter cortisol slopes, and higher waking cortisol. In testing differences between sexual abuse and other types of abuse, the effect of sexual abuse on diurnal slope remained significant, indicating a unique effect of sexual abuse on diurnal cortisol slope. These findings were not explained away by obesity, race, or puberty. The interaction between sexual abuse and gender was significant at trend-level in predicting diurnal cortisol and changed the effect of sexual abuse, indicating that sexual abuse effects may differ by gender. Sexual abuse had a significant effect on waking testosterone overall; however, when controlling for gender, sexual abuse no longer had a significant main effect on waking testosterone. Boys had higher waking testosterone compared to girls. Puberty and gender did not have an effect on diurnal cortisol or diurnal testosterone, but puberty was associated with flatter testosterone slopes at trend-level. These findings did not change when controlling for race and BMI.

The purpose of this study was to determine if sexual abuse had a significant effect on cortisol and testosterone. Although sexual abuse did not alter the testosterone slope, it did have a significant effect on cortisol's rhythm. In this study, sexual abuse was associated with steeper cortisol slopes. Steeper slopes indicate that sexually abused youth are recovering fairly normally throughout the day, and are likely not finding their current environment stressful. Prior research has found that cortisol responds to child sexual abuse trauma as expected (i.e., increase in

cortisol secretion) for a time, but this changes in adulthood. Trickett and colleagues (2010) found that cortisol activity is significantly higher closely following sexual abuse and that slopes are flatter for sexually abused females compared to controls. Trickett and colleagues' (2010) study observed the attenuation of cortisol across development at six time points between the ages of 6 and 30 years. In our study, the HPA appeared to recover in sexually abused youth, indicating that their environment restores or does not interfere with their negative feedback loop. It is possible that they are finding their current environment stable and safe compared to harmful environments experienced prior to incarceration.

Extant research has also found that proximity of the experience is important in assessing the change in HPA regulation. Weems and Carrion (2007) found that proximity of trauma deserves special consideration when evaluating cortisol rhythms as a function of trauma and PTS (post-traumatic stress) symptoms, specifically noting that higher evening cortisol is positively associated with more proximal trauma and PTS symptoms. Unfortunately, the present study did not examine how much time has passed since the trauma nor did we examine mental health factors in association with sexual abuse. Nevertheless, the discrepancy in diurnal rhythm findings compared to prior research could point to a potential overlap of mental health or adaptation in how sexual abuse effects physiological functioning. Prior research has found that sexually abused females meet more criteria for DSM diagnoses compared to non-abused females (Trickett, et al., 2011) and PTS symptomology is common in reporters of child abuse (Weems, and Carrion, 2007; 2008). In addition to examining mental health factors as a function of sexual abuse, future studies should consider behavioral correlates as well. Prior research has found that youth with less extreme internalizing symptoms have steeper diurnal cortisol, while medication can also produce a steep slope (Ruttle, Shirtcliff, Serbin, Fisher, Stack, and Schwartzman, 2011).

It is also important to consider the current environment of the sample examined in the present study. Although we did not test the perceived stress or hormone outcomes as a function of incarceration, it is reasonable to assume that HPA functioning is different from prior research due to the incarceration (Dismukes, et al., 2015). Such an environment can be a refuge from the harmful environment the youth come from, but can also be incredibly stressful for youth who have experienced less harmful or different backgrounds (Gostisha, et al., 2014). Incidentally, several female adolescents in this study specifically noted that they committed murder in order to escape the harmful environment. In the case of incarceration serving as a safe, stable space for youth who come from extremely harmful backgrounds, the diurnal slope may indeed be following a typical pattern with high levels in the morning and low levels in the evening, but it is steeper because it has been shaped by chronic stress exposure. In other words, the HPA is functioning fairly normally because the system has been primed by prior environments to expect stressors throughout the day. When the stressors are not encountered, the HPA axis recovery is hurried. Youth who do not come from as severe backgrounds may find the incarceration stressful (refer back to Figure 5. for visual example; but see Gostisha, et al., 2014) and therefore have flatter slopes, or in other words, higher levels of evening cortisol. It is possible that there are components of the incarceration context that are causing the low abuse group distress.

In this study we found that girls had flatter slopes and higher waking cortisol compared to boys. Although there were no significant differences in CAR, the sex differences found in this study are consistent with the literature since diurnal cortisol is assumed to mature during adolescence and become flatter as youth develop, particularly girls (Shirtcliff, Allison, Armstrong, Kalin, and Essex, 2012). We also examined differences in cortisol while controlling for race, and found that White adolescents had higher waking cortisol, which is consistent with

prior research. One study found that African-American youth have lower waking cortisol compared to Hispanic and White adolescents (DeSantis , Adam, Doane, Mineka, Zinbarg, and Craske, 2007). It is beyond the scope of this study to further examine racial differences in cortisol levels by examining individual time points in the way prior studies have (i.e., morning versus bedtime levels, DeSantis, et al., 2007). Importantly, in testing the effect of sexual abuse on diurnal cortisol, sexual abuse was compared to physical abuse and other types of abuse within the measure, resulting in a robust effect of sexual abuse, and no significant effects of other types of abuse on cortisol. This is an exciting finding, considering the common issue of collinearity of different types of abuse, particularly physical and sexual abuse (Ackerman, et al., 1998), which was the case in this study as well. It is possible that these findings support the assumption that sexual abuse is truly unique compared to other types of childhood abuse.

In testing the effects of sexual abuse on testosterone, we found that greater amount of abuse resulted in lower waking testosterone. However, this effect was no longer significant when controlling for gender. It is very likely that the effect on the gonadal axis is different, especially considering that male and female testosterone is secreted from different organs and in different amounts, particularly during adolescence and in response to stress. This effect may present differently in larger samples of sexually abused youth. In this study, girls had lower waking testosterone compared to boys, while boys had steeper slopes compared to girls. This is not consistent with the literature, as prior studies have found that girls had steeper slopes compared to boys, although this steep decline can be associated with pubertal development (Granger, et al., 2003). This study did not find significant sex differences in testosterone diurnal slope, potentially due to the characteristics of the sample that have been associated with high levels of testosterone in both boys and girls. More specifically, delinquency has been associated with high testosterone

in both sexes (Trickett, et al., 2005; Felson, and Haynie, 2002). Sexually abused youth are also more likely to exhibit aggression, delinquent behaviors, and school problems (Kendall-Tackett, et al.,1993; Holmes, and Slap, 1998). It is possible that the testosterone mechanism responds to sexual abuse differently in boys and girls, however a larger sample of victimized youth is needed to test this.

In testing the final hypothesis of whether gender moderated the effect of sexual abuse on hormones, the effects of sexual abuse on testosterone were explained away by gender. Gender differences in testosterone were confirmed, with girls having flatter slopes and lower waking cortisol compared to boys, which is consistent with prior research. Puberty did not explain away the effect of sexual abuse on diurnal cortisol, and was only significant at trend-level for diurnal testosterone. Notably, the gender by sexual abuse interaction was significant at trend level, indicating there may be a different effect of sexual abuse depending on gender, with sexually abused boys having flatter cortisol slopes. This intriguing interaction between gender and sexual abuse should be examined further in future research. Especially considering the gender differences in health outcomes that are associated with. Sexually abused boys and girls are more prone to emotional and behavioral problems, as well as suicidal thoughts and suicide attempts (Garnefski, and Arneds, 1997). These consequences are more prevalent in boys compared to girls especially in terms of aggressive and criminal behavior, drug and alcohol abuse, as well as suicidal behavior and truancy (Garnefski, and Arends, 1997). Determining the gender differences in physiological regulation as a function of a specific type of abuse requires a larger sample size.

Implications of these findings

In this study the strong effect was that of sexual abuse on cortisol slope. This robust finding can shed light on the way the stress response system adapts following sexual abuse

trauma, but also serve as a preliminary result regarding HPA axis functioning within the context of incarceration. This study raises a special concern, which is the importance of examining the factors of unique environments, such as incarceration, in greater depth. If the environment is deemed stressful or perhaps comforting, its impact on biological mechanisms has the potential to inform intervention strategies. The findings in this study point to an intriguing aspect of the incarceration context, which is that it can be both helpful and stressful to different youth, depending on their backgrounds. Similar to the assumption that different types of abuse, severity, and intensity have varied effects on health, these findings show that individual-specific factors should be examined to determine how developing systems are shaped by changing environments. In other words, incarceration may be helpful for the recovery of the biological systems for the youth who come from especially harmful environments because it provides a stable and secure atmosphere to those who are not used to having one. It is possible that the youth who were sexually abused in this sample are recovering fairly normally toward the end of the day because their HPA axis is not anticipating any stressors. On the other hand, those who find the incarceration stressful may lack certain resilience or adaptive mechanisms that can be determined through further examination of the severity of adverse experiences, mental health symptomology, genetic factors, and coping resources available in facilities. Finding differences between the two groups can provide more information on the ways biological and psychological mechanisms adapt during this sensitive period of development, as well as how these adapted mechanisms respond to non-traditional environments such as incarceration.

These findings are also relevant for intervention strategies, especially considering that incarcerated youth are already undergoing a form of intervention by *being* incarcerated. The present study can be helpful in differentiating the youth who benefit from this unique

environment, compared to those who do not. These findings suggest that a safe, stable environment could be key to stress response regulation if made available during a sensitive time like adolescence. Recognizing this type of trauma within incarcerated youth can aid in actively resisting and preventing revictimization in the future through sexuality education, therapy, and provision of a safe space where maltreated youth can thrive and recover from prior adversities. The facilities that provide safety and stability to maltreated youth would promote resilience through a trauma-informed approach, and therefore taking advantage of the malleability of biological systems in adolescence. Providing care at this time can help modify the alterations in developmental trajectories caused by early adversity, and consequently improve future health outcomes.

Limitations

Although the present study has its strengths including access to a unique and vulnerable sample, as well as strict times of collection and number of samples per person, it also suffers from several notable limitations. First, the sample size is likely an issue in examining effects of interactions, limiting the ability to test the effects of sexual abuse by gender. Additionally, the sample included a small number of individuals (37.6%) who reported sexual abuse, which could be restricting the findings. This study did not examine associations between sexual abuse and mental health symptomology. It is recommended that future studies examine larger samples of sexually abused youth, specifically examining mental health outcomes (e.g., depression and PTS symptoms) in addition to physiological regulation as a function of sexual abuse. Finally, the present study did not have the ability to compare incarcerated youth to non-incarcerated youth, therefore neglecting to test the potential benefits of incarceration.

Conclusions

This study sheds light on the relationship between sexual abuse and cortisol regulation in incarcerated adolescents. The robust finding of the relationship between sexual abuse and steeper cortisol slopes can clarify the way the stress response system adapts following sexual trauma in the context of incarceration. Incarceration is important to consider, as youth from particularly harmful environments (i.e., those who reported sexual abuse) may be experiencing stability and safety, opposed to stress of being incarcerated. Due to the vulnerabilities of developing systems in adolescence, as well as the association between sexual abuse and mental health in boys and girls, it is recommended that future studies consider mental health in association with sexual abuse in youth populations. Investigating the adaptive responses of the stress and gonadal axes is valuable in uncovering the characteristics of new environments that victimized youth can benefit from. The intriguing association between gender and sexual abuse should be examined further in a larger sample of victimized youth. The null effect of sexual abuse on testosterone can be attributed to different mechanisms between the sexes that are responsible for production of this hormone. The present study demonstrates that sexual abuse has a unique effect on the stress response system, but consideration of the context has great potential for informing intervention strategies.

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APPENDIX: IRB APPROVAL LETTER

*University Committee for the Protection
of Human Subjects in Research
University of New Orleans*

Campus Correspondence

Principal Investigator: Birdie Shirtcliff

Date: February 12, 2010

Protocol Title: "Biological Markers of Aggression in Adolescent Female
                                 Offenders"

IRB#:   12Dec09

Your proposal was reviewed by the full IRB. The group voted to approve your proposal pending that you adequately address several issues. Your response to those issues has been received and you have adequately addressed all of the issues raised by the committee. However, the committee still has the following suggestions:

- The committee suggests making consent form clear on whether information will be shared. "Portions of the study may be shared with clinical staff if it aids in your treatment programming." Should charge "maybe" to "will be" for more clarification.
- The consent letter was found to be lacking important information by the committee, such as, a clear amount of time the questionnaire and interview will take.

Please submit a copy of consent form. Upon approval of these clarifications, you will be sent a letter stating that your project is now in compliance with UNO and Federal regulations. Your response to the modifications requested will not be reviewed by the full committee

Please remember that approval is only valid for one year from the approval date. Any changes to the procedures or protocols must be reviewed and approved by the IRB prior to implementation. Use the IRB number listed on this letter in all future correspondence regarding this proposal.

If an adverse, unforeseen event occurs (e.g., physical, social, or emotional harm), you are required to inform the IRB as soon as possible after the event.

Best of luck with your project!
Sincerely,

Robert Laird, Ph.D., Chair
Committee for the Protection of Human Subjects in Research