PERSONALITY, HORMONES, INTELLIGENCE, AND MATURITY: PSYCHOPATHY
AND HPA/HPG BALANCE AMONG ADOLESCENT OFFENDERS.

by

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ABSTRACT

The interplay of hormonal balance, intellectual ability, and psychosocial maturity has not yet been investigated in adolescents with psychopathic traits. We recruited 58 adolescent offenders and collected self-report measures of psychosocial maturity (The Risk-Sophistication-Treatment-Inventory-Self-Report; RSTI-SR) and conducted an IQ test (Kaufman-Brief Intelligence Test-Second Edition), as well as an interview to estimate their level of psychopathic characteristics (Psychopathy Checklist-Youth Version; PCL-YV). We also collected a sample of salivary cortisol and testosterone at the beginning of the session to assess youth’s resting concentrations of hormones and two samples of cortisol and testosterone at 20 minutes and 40 minutes following a social stress induction to assess their immediate reaction to stress, as well as their ability to recover from stress. The primary findings from this study were that intelligence moderated the relationships between the antisocial facet and each of baseline cortisol, cortisol reactivity, and the ratio of baseline testosterone to cortisol reactivity. Thus, in this study, when the role of intelligence was considered hormonal secretion was more closely related to antisocial traits than more traditional psychopathic traits. These and other findings are discussed in further detail.
DEDICATION

This thesis is dedicated to all of my friends and family for providing me with the necessary emotional support, patience, and encouragement to help me complete this manuscript. In particular, I would like to thank my parents Kent and Susan Harrison and my sister Carley Harrison for their endless love and support from afar. I would also like to thank my boyfriend Jarrette Davis for always demonstrating strength and patience and providing unwavering moral support. Finally, I would like to show appreciation to my good friends Abby Clark, Cameron Powe, Emily MacDougall, and Hannah Price for letting me bounce ideas off of them when I was at a loss and inspiring me with their hard work and perseverance.
LIST OF ABBREVIATIONS AND SYMBOLS

\( \alpha \) Cronbach’s Alpha: an index of internal consistency

AIC Akaike criteria for fitting covariate structures into analysis of variance

\( \text{AUC}_G \) Area under the curve with respect to ground

\( \text{AUC}_I \) Area under the curve with respect to increase

\( \beta \) Standardized beta coefficient: a measure of how strongly each predictor variable influences the outcome variable.

CU Callous and unemotional traits

CRH Corticotrophin-releasing hormone

\( F \) Fisher’s \( F \) ratio: a ration of two variances

FSIQ Full scale intelligence quotient

GLM General linear model

HPA Hypothalamic-pituitary-adrenal

HPG Hypothalamic-pituitary-gonadal

ICC Interclass correlation: a measure of reliability

ICU Inventory of Callous and Unemotional Traits

IQ Intelligence quotient

K-BIT 2 Kaufman Brief Intelligence Test-Second Edition

\( M \) Mean: the sum of a set of measurements divided by the number of measurements in the set

OMPFC Orbitofrontal and medial regions of the prefrontal cortex
\[ p \] Probability associated with the occurrence under the null hypothesis of a value as extreme as or more extreme than the observed value

PANAS The Positive and Negative Affect Scale

PCL-YV Psychopathy Checklist-Youth Version

PFC Prefrontal cortex

PIQ Performance intelligence quotient

\( r \) Pearson product-moment correlation

RSTI-SR The Risk-Sophistication-Treatment-Inventory-Self-Report

SD Standard deviation: the square root of variance

SRO Self Report of Offending

STAT Sternberg’s Triarchic Abilities Test

STICSA The State–Trait Inventory for Cognitive and Somatic Anxiety

\( t \) Computed value of \( t \) test

TSST Trier Social Stress Test

VIQ Verbal intelligence quotient

\(<\) Less than

\(=\) Equal to
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CHAPTER 1
INTRODUCTION

Individuals with psychopathic traits can have a costly impact on society, due to associations between psychopathy and increased risk for violence (Swogger, Walsh, Homaifar, Caine, & Conner, 2012), criminal involvement (Hare, & Neumann, 2009), and poor treatment response (Ogloff, Wong, & Greenwood, 1990). For these reasons, individuals with psychopathic traits have been studied extensively to uncover how the syndrome develops and to determine the factors that set individuals with psychopathic traits apart from the rest of society. Through these investigations, researchers have found associations between psychopathy and many functional abnormalities including decreased stress reactivity (O'Leary, Loney, Eckel, 2007) and poor performance on cognitive tasks (Ishikawa, Raine, Lenz, Bihrlle, & Lacasse, 2001). Similarly, intellectual ability has been investigated to determine whether these functional abnormalities relate to intelligence (e.g., DeLisi, Vaughn, Beaver, Write, 2010; Spironelli, Segrè, Stegagano, & Angrilli, 2014). Alternatively, increased intellectual ability among those with psychopathic traits may allow them to manipulate and control others (e.g., Cleckley, 1941, 1950, 1955, 1988).

The study of psychopathic traits among children and adolescents may be especially beneficial in uncovering the etiology of psychopathy, given that youth allow for the examination of these traits as they develop. More recently, researchers have investigated the role that psychosocial maturity plays in adolescent psychopathy and offending, in order to divulge the developmental trajectory of psychopathy (e.g., Leistico & Salekin, 2003). Despite the recent
attention on adolescent psychopathy, the interplay of hormonal balance, intellectual ability, and psychosocial maturity has not yet been investigated in adolescents with psychopathic traits.

There is some debate within the field of psychopathy research regarding the most valid way to conceptualize the syndrome. Perhaps the most notable conceptualization of psychopathy was articulated by Cleckley (1941/1988) who theorized that psychopathy consisted of a cluster of traits including superficial charm, intelligence, manipulation, impulsive behavior, and deficient affective experiences. Subsequently, Karpman (1941) purported that psychopathy had two distinct subtypes; a) those who are charming, cold, and calculated manipulators (primary psychopathy) and b) those who are characterized by an impulsive and irresponsible lifestyle wrought with antisocial activity (secondary psychopathy). More recently there has been support for clustering psychopathy into models that emphasize three or four facets. Within the three-facet model, antisocial behavior is removed from the model so that psychopathy consists of an arrogant and deceitful interpersonal style, deficient affective experience, and impulsive and irresponsible lifestyle (Cooke & Michie, 2001). Other researchers (e.g., Vitacco, Neumann, & Jackson, 2005) believe that antisocial behavior is an important aspect of psychopathy and follow a four-facet model of psychopathy, in which the syndrome is conceptualized as consisting of an interpersonal facet (e.g., superficial charm), affective facet (e.g., shallow affect), lifestyle facet (e.g., impulsivity), and antisocial facet (e.g., early behavior problems). Controversy remains regarding which conceptualization fits the construct of psychopathy best, but there appears to be some general consensus that psychopathy is underpinned by at least two broad factors and three finer grained facets (interpersonal, affective, and impulsive-irresponsible).

One important theory pointing to the etiology of psychopathic traits is the triple balance model of emotion (Van Honk & Schutter, 2006). This theory integrates causal models of
psychopathy that emphasize the importance of low fear responses, as well as punishment insensitivity, and reward seeking tendencies (referred to as motivational imbalance). These models also delineate that psychopathic individuals show deficits in the function of particular brain regions. Specifically, individuals with psychopathic traits have deficits in their amygdala and orbitofrontal and medial regions of the prefrontal cortex (OMPFC) that impede their ability to use emotional signals from the environment to guide their behavior. Van Honk and Schutter (2006) contributed to these theories by incorporating an understanding of the mutually inhibitory neuroendocrine function of the Hypothalamic-Pituitary-Adrenal Axis (HPA) and the Hypothalamic-Pituitary-Gonadal Axis (HPG).

Cortisol is a glucocorticoid messenger from the HPA axis that acts on the amygdala to assist in gene expression of corticotrophin-releasing hormone (CRH), the hormone that ultimately enhances one’s perception of fear (Rosen & Schulkin, 1998). This expression of CRH often invokes behaviors of withdrawal (Schulkin, 2003) and may allow for sensitivity to punishment (Van Honk, Schutter, Hermans, & Putman, 2003). For this reason, cortisol is often used as an index to measure reactivity to stress (e.g., Glenn, Raine, Schug, Gao, & Granger, 2011; O’Leary, Loney, Eckel, 2007; O’Leary, Taylor, Eckel, 2010). In contrast, testosterone, which produces reductions in fear responses and has rewarding properties (Boissy & Boussiou, 1994), is often involved in instrumental aggression (Van Honk, Schutter, Hermans, Putman, Tuiten, & Koppeschaar, 2004) and reward sensitivity (Carr, Fibirger, & Phillips. 1989). Thus, these steroid hormones work in concert to act on the amygdala and its adjoining neural networks to express genes that facilitate reactions to fearful situations. These reactions are often either behavioral activation leading to approach (via testosterone) or behavioral inhibition leading to withdrawal (via cortisol; Meisel & Sachs, 1994; Wood, 1996). Interestingly, these hormones
communicate by suppressing the others’ function. Specifically, cortisol can reduce the affect that testosterone has on tissues (Johnson, Kamilaris, Chrousos, & Gold, 1992) and testosterone can diminish cortisol’s communication with the hypothalamus (Viau, 2002). The relationship between the HPA and HPG axes explains the low fear and motivational imbalance among individuals with psychopathic traits as being due to reduced productivity of the HPA, and thus low levels of cortisol, in conjunction with enhanced activity of the HPG, producing greater levels of testosterone.

Van Honk and Schutter (2006) hypothesized that the combination of high testosterone and low cortisol found among those with psychopathic traits would be more pronounced for those with primary psychopathic traits (which emphasize interpersonal and affective features) than secondary psychopathic traits. Further, these authors explained that this unique balance of the neuroendocrine axes may contribute to reduced communication within and between cortices and sub-cortices, which inhibit communication regarding emotional content in the environment. Thus, the three balances that they proposed to influence psychopathic traits were: (1) the subcortical balance, (2) the subcortical-cortical balance, and (3) the cortical balance. The subcortical balance is the balance of the HPA axis via cortisol and the HPG axis via testosterone in reaction to reward or punishment, which acts as an emotional homeostasis. The subcortical-cortical balance consists of the communication between subcortical regions, such as the amygdala, and cortical regions, such as the OMPFC, and affects socioemotional functioning. The cortical balance relates to the symmetry between the right PFC and the left PFC, the cortical regions that have been implicated in withdrawal behavior and approach behavior, respectively. Moreover, coupling of these cortical regions can be enhanced by cortisol and reduced by testosterone (Schutter & Van Honk, 2004, 2005).
As an adjunct to this theory, Terburg and colleagues (2009), further emphasized the role of testosterone and cortisol in the triple balance theory of emotion. The researchers explained that, firstly, high doses of testosterone can heighten attention toward aggressive stimuli by acting on the amygdala. Secondly, the researchers contended that angry facial expressions are processed in the amygdala, hypothalamus, and brainstem before being projected to the orbitofrontal cortex. Finally, these authors argued that communication between these regions is important for the functions of perceiving emotions and changing one’s response to a feared stimulus (e.g., approaching something that invokes fear). Cortisol and testosterone facilitate this communication by regulating the amount of communication (i.e., more for cortisol, less for testosterone), recognition of threatening emotions (i.e., better recognition for cortisol and less accurate recognition for testosterone), and approach decisions (i.e., more cognitively informed approach decisions for cortisol and less inhibition of approach behaviors for testosterone).

In the same vein as the triple balance model of emotion, the neurodevelopmental hypothesis of psychopathy development (Gao, Glenn, Schug, Yang, & Raine, 2009) outlined that people who score high on psychopathic measures differ on many aspects of brain and neuroendocrine function from an early age, including lower levels of cortisol and higher levels of testosterone. They proposed that the lower levels of cortisol among individuals with psychopathic traits contribute to their lack of anxiety regarding criminal activity. Moreover, testosterone has been related to increases in impulsivity and aggression. Gao and colleagues further hypothesized that lower than average levels of cortisol and higher than average levels of testosterone in childhood may impede children’s ability to develop socially by reducing their responsivity to stressors and consequently reducing fear of negative consequences. These mechanisms may increase their risk for future aggression, antisocial behavior, and poor empathy.
development (Shirtcliff, Vitacco, Graf, Gostisha, Merz, & Zaun-Waxler, 2009; Yang, Raine, Colletti, Toga, & Narr, 2010).

These theories have also been supported by research investigating brain structures among those with psychopathic traits. Brain imaging studies have found that people scoring high on psychopathy with extensive criminal histories tend to have lower brain mass in their prefrontal cortex and amygdala (Yang et al., 2010; Yang, Raine, Lencz, Bihrlle, LaCasse, & Colletti, 2005), as well as reduced brain activity in the amygdala when making decisions about whether to punish another person (Osumi, Nakao, Kasuya, Shinoda, Yamada, & Ohira, 2012). These findings are enlightening given the extensive mass of glucocorticoid receptors within the prefrontal cortex and amygdala (McEwen, De Kloet, & Rostene, 1986).

The triple balance model of emotion is also highly supported by the data on cortisol and testosterone levels within psychopathic populations. Among adult samples, researchers have found correlations between psychopathic traits and both daily cortisol fluctuations and stress reactivity within the community. Daily levels of cortisol among undergraduates were negatively associated with factor one (interpersonal-affective), but contrary to the triple balance model, positively associated with factor two (lifestyle-antisocial) and these findings were only applicable to female participants (Vaillancourt & Sunderani, 2011). Cortisol has also been implicated in punishment insensitivity and reward dependency among undergraduate students; baseline cortisol levels shared an inverse relationship with choosing risky decks and money loss during the IOWA gambling task (Van Honk et al., 2003).

Along with the daily fluctuations, cortisol reactivity has also evidenced an inverse relationship to psychopathic traits among adults. Specifically, despite the general finding that cortisol elevates in response to stress, cortisol reactivity in response to a social stressor
demonstrated an inverse relationship to psychopathic traits among undergraduate males, such that those with higher psychopathy scores exhibited what appeared to be an inhibited cortisol increase in reaction to stress when compared to non-psychopathic controls, this finding was not supported with females (O'Leary et al., 2007). Interestingly, when the researchers excluded females with low progesterone from the analysis, a relationship between cortisol stress reactivity and psychopathic traits among females was revealed, but in the opposite direction of males. Females with high psychopathy lacked the decreased cortisol response characteristic of female controls. This natural decrease in cortisol in response to stress among controls was theorized to contribute to “tend or befriend” behaviors of females in the face of stress, leading to increased cooperation rather than competition (O'Leary et al., 2010). One recent study investigated cortisol stress reactivity in relation to psychopathic traits in a young adult detained sample. They found that among those who demonstrated a decreased cortisol response to a social stressor, primary psychopathic traits, especially the affective traits, were related to a steeper decline (Johnson, Mikolajewski, Shirtcliff, Eckel, & Taylor, 2015).

In following with the triple balance model of emotion, the inverse interaction between cortisol and testosterone has also been supported by research. Among male offenders, those with primary psychopathic traits (interpersonal-affective traits) had significantly higher plasma testosterone levels, which were also related to lower post-stressor (vein cannulation) cortisol levels (Dolan, Anderson, & Deakin, 2001). Within the community, this interaction was more complex. Among adults recruited from temporary employment agencies there was no significant association between psychopathic traits and baseline cortisol or testosterone. There was however a significant relationship between psychopathy and the ratio of baseline testosterone to cortisol reactivity, within the predicted direction. When the role of specific psychopathic factors was
investigated, only factor two (lifestyle-antisocial) from the traditional two-factor model shared a significant relationship with the above testosterone/cortisol ratio (Glenn, 2011; Glenn et al., 2011).

Studies on children and adolescents provide further support for the triple balance model and the neurodevelopmental hypothesis by replicating many of the findings found among adults. This being said, not many studies investigated the HPA and HPG balance among children and adolescents with psychopathic or callous and unemotional traits (CU\(^1\)). Among those studies, one longitudinal study of clinic-referred males found that daily cortisol levels were inversely related to primary (interpersonal-affective), but not secondary psychopathy (lifestyle-antisocial; Burke, Loeber, Lahey, 2007). Further, within a sample of incarcerated adolescent males researchers found that a combination of CU traits and a history of abuse was related to lower waking levels of cortisol as well as a steeper decline in cortisol throughout the day (Gostisha et al., 2014). Moreover, in comparison to externalizing behaviors, such as hyperactivity and impulsivity, CU traits were a more robust predictor of reduced daily cortisol production among preadolescent and adolescent boys diagnosed with early onset conduct disorder (von Polier et al., 2013). Similarly, in comparison to youth with conduct problems and controls, youth with CU traits and a combination of CU traits and conduct problems had lower daily levels of cortisol. Although group differences for testosterone were also investigated, no differences were detected in this study (Loney, Butler, Lima, Counts, & Eckel, 2006).

Interestingly, Johnson and colleagues (2014) found that the interpersonal facet was specifically related to coupling of cortisol and testosterone levels throughout the day among male adolescent offenders. This was a novel finding, suggesting that this facet may contribute to an

\(^1\) CU traits are a cluster of traits similar to the affective facet of psychopathy, thus representing one of four facets of psychopathy
increase in communication between the HPA and HPG axes. Researchers also found that boys diagnosed with ADHD who scored high in CU traits showed significantly lower cortisol stress reactivity than those with low CU traits (Stadler et al., 2011). Another longitudinal study, of youth followed from three months old to fifteen years old, demonstrated a negative relationship between daily levels of cortisol and each of proactive and reactive aggression, global aggression, and poor impulse control, which map onto items in the lifestyle and antisocial facets of psychopathy. This finding was only applicable to males in the study and neither gender demonstrated a relationship between cortisol levels and CU traits (Poustka et al., 2010).

Numerous other studies have emphasized the importance of the contribution of the HPA on regulation of inhibition and antisocial behavior. Those with a combination of CU traits and hypoactive HPA systems may develop antisocial behavior even without unstable environmental factors (Hawes, Brennan, & Dadds, 2009). The reverse of this relationship also holds true, preadolescent boys with ADHD and comorbid anxiety, a condition believed to be incompatible with Cleckley’s (1988) conception of psychopathy, demonstrated significantly higher cortisol reactivity after having blood drawn (Hastings, Fortier, Utendale, Simard, & Robaey, 2009).

Despite empirical support for the connection between psychopathic traits and the balance of the HPA and HPG axes, the causal direction of this relationship is still unknown. Many of the traits that are expressed by an irregular balance of these axes, including impulsivity and inattention, are likely to lead to impaired functioning, negative reactions from others, increased stressors, and consequently affect one’s ability to regulate stress (Buitelaar, 2013). There is some support for theories that emphasize the influence of the environment on the HPA and HPG balance. For example, male adolescents with greater recent exposure to violence demonstrated significantly lower cortisol reactivity to social stress than controls (Peckins, Dockray, &
Echenrode, 2012). Moreover, in contrast to recent-onset depression, which is generally associated with an increase in the cortisol response, chronic depression was associated with a blunted cortisol response (Booij, Bouma, De jonge, Ormel, & Oldehinkel, 2013). Among criminal offenders, those with psychopathic traits demonstrated an association between daily levels of cortisol and histories of physical abuse, as well as emotional and physical neglect. Psychopathic offenders in the sample also demonstrated the signature pattern of lower diurnal cortisol (Cima, Smeets, & Jelicic, 2008). Therefore, chronic environmental stress may serve to shift the balance within the HPA. Interestingly, differences in cortisol reactivity can be found from an early age. At 15 months, infants, who were later assessed as having a combination of conduct problems and CU traits, demonstrated significantly higher cortisol reactivity in response to a scary mask task than infants with future conduct problems alone or the comparison group (Mills-Koonce et al., 2014). This finding could be evidence for a critical period before the decline of stress response system among those with CU traits. Given the inhibitory function of the HPA axis on the HPG axis, the HPG system could be affected as well. However, none of these studies investigated testosterone in order to better understand this potential shift.

In summary, findings within various populations of both children and adults suggest that males with psychopathic traits often demonstrate lower than average daily levels of cortisol (e.g., Burke et al., 2007; Gostisha et al., 2014; Loney et al., 2006; Vaillancourt & Sunderani, 2011; von Polier et al., 2013). Moreover, within the general male population cortisol increases in response to stress, but males with psychopathic traits demonstrate a blunted cortisol response to stress (e.g., Dolan et al., 2001; O’Leary et al., 2007; Stadler et al., 2011). Findings for females are less consistent, but findings from O’Leary and colleagues (2010) suggest that females without psychopathic traits demonstrate a decrease in cortisol in response to stress, whereas
those with psychopathic traits do not demonstrate a cortisol response. In regards to testosterone, adults with psychopathic traits demonstrated higher than average levels of daily testosterone (Dolan et al., 2001) and a relationship between the interpersonal facet and the ratio of baseline testosterone to cortisol reactivity (Glenn et al., 2011). These findings have not yet been demonstrated in children or adolescents (e.g., Loney et al., 2006), however Johnson and colleagues (2014) found that youth scoring high on the interpersonal facet had greater coupling of cortisol and testosterone than those with lower scores. For each of these relationships, when specific facets of psychopathy were investigated, no consistent findings emerged. Some studies found relationships between hormone levels and the primary factor (interpersonal/affective; Burke et al., 2007; Dolan et al., 2001; Johnson et al., 2014). Whereas Glenn and colleagues (2011) found more robust associations with the secondary factors (interpersonal/antisocial; Glenn et al., 2011) and Vaillancourt and colleagues (2011) found associations with both factors.

Hormonal stress reactivity has been measured in a number of ways, and many researchers use multiple analyses to view the impact of a stressor on hormonal reactivity. Among studies investigating the relationship between cortisol reactivity and psychopathic traits, three main analyses were used. The two most common methods among these studies were using an ANOVA to view the changes in cortisol across time points (e.g., O’Leary et al., 2007; O’Leary et al., 2010; Stadler et al., 2011) and subtracting the post-stressor sample from the baseline sample to calculate a number that represented the change in cortisol from baseline to post-stressor (Dolan et al., 2001; Mills-Koonce et al., 2014; Milner, 1977). Another potential method is to calculate the area under the curve, which is a calculation of the volume of cortisol across time points, including baseline, post-stressor, and often one more sample of either pre- or post-stressor hormone (Glenn et al., 2011). Area under the curve can be calculated in one of two
ways. The first way is with respect to ground (AUC\(_G\)), which includes all levels of cortisol. The second way is with respect to increase (AUC\(_I\)), which only includes changes in cortisol over time (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003).

Recent studies investigating hormonal reactivity for purposes not relating to psychopathy have used the same three methods. Among these studies the most common method was subtracting baseline levels from post-stressor levels (Edwards & Casto, 2015; Jaffee et al., 2015; Martinez-Torteya et al., 2015; Turan et al., 2015), followed by ANOVAs to view differences across time point (Hostinar, Johnson, & Gunnar, 2015; Luecken et al., 2015; Smith & Jordan, 2015), and area under the curve (AUC\(_I\) only, Cărnuţă Crişan, Vulturar, Opre, & Miu, 2015; AUC\(_G\) and AUC\(_I\), Hankin, Badanes, Smolen, & Young, 2015). Using area under the curve appears to be the least common method, but serves the benefit of allowing for calculation of hormone levels across more than two time points (unlike the method of subtracting baseline measures from post-stressor measures) and controlling for the amount of time between time intervals. In addition, area under the curve provides a single number that can be used in the calculation of ratios between cortisol and testosterone. Ratios allow for a representation of the intertwined relationship between cortisol and testosterone, which is important to measure given theory regarding the feedback relationship between these hormones (e.g., Terburg et al., 2009; VanHonk & Schutter, 2006) and recent findings that psychopathy is related to increased coupling between the two hormones (Glenn et al., 2011; Johnson et al., 2014). In conclusion, there are three common methods of measuring cortisol reactivity and each of these methods yield different outputs and offer differing conceptualizations of stress reactivity. In order to provide a point of comparison across studies, it may be beneficial to use multiple methods. Moreover, given that the relationship between psychopathy and stress reactivity and has not been investigated in a
detained adolescent sample, a precedent has not yet been set on how to measure stress reactivity among this population.

**Stress Inductions Among Adolescent Offenders**

In order to study HPA and HPG reactions to stress, a stress induction task must be identified that will have an effect on cortisol and testosterone. There is not a large body of research on effective stress inductions for use with offender samples, let alone detained youth. Among adults, male inmates within a treatment study were exposed to a speech task in which they were asked to prepare a ten-minute speech for the parole board requesting early release. Change in cortisol from baseline to post-stressor was related to various outcome measures, such as treatment gains and completion of therapy (Fishbein et al., 2009). Within the adolescent literature a few studies have used stress inductions with adolescent offenders, but none of them were detained. For youth meeting criteria for early and late onset Conduct Disorder, the Prisoner’s Dilemma task was used to induce stress by exposing participants to a feigned opponent that was uncooperative and antagonistic. Participants also completed a frustrating computer task. Those exposed to both of the stress inductions had significantly higher cortisol reactions than those who were not exposed to a stress induction (Fairchild et al., 2008). In another study of adolescent males with disruptive behavior disorder, participants were asked to conduct a five-minute speech in front of (what they were told was) a jury of three psychologists. Cortisol increased significantly following the stress task (de Vries-Bouw et al., 2012). The same finding was observed among adolescents who were part of a delinquency diversion program who were asked to conduct the same task as de Vries-Bouw and colleagues (Popma et al., 2007). Taken together, it appears that social stressors involving presentations have a general effect on cortisol reactivity within offender samples. These findings suggest that stressors with an element
of social evaluation or marginalization will allow for cortisol to increase despite the general levels of stress that offenders already experience in detention.

**Psychopathy, Intelligence, and Hormonal Balance**

One important aspect of functioning that may be affected by brain and neuroendocrine (HPA and HPG) irregularities is intelligence, given that many aspects of intellectual functioning are housed within the prefrontal cortex (dorsolateral PFC, Barbey, Colom, & Grafman, 2013; Barbey, Colom, Paul, & Grafman, 2014; lateral PFC, Cole, Yarkoni, Repovš, Anticevic, & Braver, 2012), limbic system (hippocampal volume, Amat et al., 2008) and the their connecting white matter fiber tracts (amygdala with orbitofrontal and frontopolar regions, Barbey et al., 2014). The intertwining relationships between intelligence, psychopathy, and hormonal balance are complex. For example, high stress reactivity can acutely impair cognitive abilities, especially on memory tasks (Robinson, Leach, Owen-Lynch, & Siinram-Lea, 2013). This being said, reduced reactivity has been associated with lower cognitive ability (Ginty, Phillips, Roseboom, Carroll, & Derooij, 2012). Low verbal intelligence is also considered a risk factor for antisocial behavior (Leech, Day, Richardson, & Goldschmidt, 2003; Vermeiren, De Clippele, Schwab-Stone, Ruchkin, & Deboutte, 2002).

Intelligence also appears to be influenced by cortisol and testosterone. Hormones may influence learning, intelligence, and memory in adolescents (Arain et al., 2013). Among five-year-old boys, testosterone levels were positively correlated with fluid intelligence (Azurmendi et al., 2005). In adulthood, very low and very high levels of testosterone were related to low IQ, this finding held true for both men (Kutlu, Ekerbicer, Ari, Uyanik, Zeren, & Tan, 2001) and women (Tan & Tan, 1998). Somewhat different findings were found among pre-pubertal children. When intellectually gifted and intellectually non-gifted pre-pubertal children were
compared, those who were intellectually gifted had significantly lower daily testosterone levels and that finding held for both sexes (Ostatníková et al., 2000). When this comparison was conducted on intellectually delayed, average, and gifted pre-adolescents, those with average intelligence had significantly higher levels of testosterone in comparison with both intellectually gifted and intellectually delayed males. Within this sample, intelligence was not related to testosterone levels among females (Ostatníková et al., 2007). Moreover, among boys at age eight testosterone and fluid intelligence were unrelated, but by age ten fluid intelligence and testosterone gained a positive relationship. Interestingly this relationship reversed to a negative relationship by age twelve and the authors suggested that this finding might be an artifact of the dynamic and protracted maturation of the adolescent brain that may fluctuate throughout development. They also found that the relationship between testosterone levels and intelligence was stronger when testosterone levels were greater than 14 pg/ml, thus potentially explaining the null result among eight year olds (Shangguan, & Shi, 2009).

Cortisol has also been found to relate to intelligence. Higher levels of daily cortisol among older adults were related to poor performance on measures of executive functioning, processing speed, and visual-spatial memory (Franz et al., 2011). Others have found reduced cognitive abilities in relation to increases in cortisol levels as a consequence of prolonged stress (Blair et al., 2011; Sandström et al., 2011). Alternatively, some studies have found that lower levels of daily cortisol, or low variability in cortisol throughout the day (Osterberg, Karlson, & Hansen, 2009) were related to lower cognitive abilities such as visual-spatial memory and processing speed (Kennedy et al., 2014). The authors hypothesized that the discrepant findings were due to a possible U-shaped relationship between cortisol and cognitive functioning, whereby both very low and very high levels of cortisol are related to poor cognitive outcomes.
This hypothesis is similar to the previous finding that very low and very high levels of testosterone were related to low intelligence scores (Kutlu et al., 2001; Tan & Tan, 1998).

There are two general ways in which psychopathy has been theorized to relate to intelligence. One such theory is that psychopathic traits (e.g., impulsivity or irresponsibility) represent functional deficits within the brain due to left hemispheric dysfunction (Nijman, Merckelbach, & Cima, 2009). Another theory was introduced by Cleckley (1988), who stated that individuals with psychopathic traits often have superior intellectual ability and superficial charm. According to Cleckley, these traits help psychopathic individuals attract and manipulate their victims and, in some cases, avoid detection by the criminal justice system. The literature on intelligence and psychopathy has thus far been mixed. Only two studies found absolutely no association between intelligence and psychopathy, one within a detained, mixed-gender adolescent sample (Allen, Briskman, Humayun, Dadds & Scott, 2013) and one within a detained adult male sample (Flores-Mendoza, Alvergena, Herrero, Adad, 2008). The remaining studies found an association between psychopathy and intelligence, but in opposing directions.

In line with the view that psychopathy is due to brain impairment, some researchers have found that psychopathy and intelligence share an inverse relationship; as psychopathy scores go up, intelligence scores go down (clinically deferred male juveniles, Burke et al., 2007; adult civil psychiatric patients, DeLisi, Vaughn, Beaver, & Write, 2010; violent adult male offenders, Heinzen, Kohler, Godt, Geiger, & Huchzermeier, 2011; community sample, Neumann & Hare, 2006; female forensic inpatients, Spironelli, Segrè, Stegagano, & Angrilli, 2014). Specifically, low intelligence in combination with psychopathic traits was associated with poorer impulse control (Heilbrun, 1982), irresponsibility, as well as lacking empathy, goals, and remorse (DeLisi et al., 2010). Further, this relationship may be especially pronounced among European
Americans in comparison to African Americans. In particular, lower intelligence among European American males with psychopathic traits was related to more extensive histories of violent behavior. In contrast, among African American males, psychopathy alone predicted violence (based on a sample of adult males in county jail, Walsh, Swogger, & Kosson, 2004). Cognitive studies have also found that people scoring high on psychopathy measures tended to perform worse on cognitive tests such as the Wisconsin Card Sorting Task (Ishikawa et al., 2001).

In line with Cleckley’s prototypical view of psychopathy, some studies found that higher levels of psychopathy were linked with higher levels of intelligence. Among adult males with a history of violent crimes, those with a combination of high intelligence and psychopathic traits were more likely to commit their first violent offense earlier and display worse behavior while detained compared to those without psychopathic traits (Johansson & Kerr, 2005). Similarly, among serious juvenile offenders, higher intelligence has been associated with elevated levels of self-reported offending. In addition, those with both higher levels of psychopathy and more extensive criminal histories tended to be relatively more intelligent than those with lower psychopathic traits and less offending (Hampton, Drabick, & Steinberg, 2014). In contrast, undergraduates who were high on both psychopathy and intelligence, especially verbal intelligence (VIQ), self-reported less criminal behaviors (Wall, Sellbom, & Goodwin, 2013).

All of the previously presented studies investigated either full scale intelligence quotient (FSIQ) or VIQ. When performance IQ (PIQ) was investigated among psychiatrically disordered juvenile offenders in a maximum-security hospital, both Psychopathy Checklist Youth-Version (PCL-YV) total and factor one scores correlated positively with PIQ and the discrepancy between PIQ and VIQ. This finding held true even after controlling for FSIQ, education, broken
homes, and substance dependency (Nijman et al., 2009). The researchers interpreted the PIQ over VIQ pattern as suggestive of a left-hemispheric dysfunction among psychopathic participants.

When both the three-facet and the four-facet latent variable view of psychopathy were analyzed using confirmatory factor analysis, the affective, lifestyle, and antisocial behavior facets (within the four facet model) negatively predicted VIQ. In contrast, the interpersonal facet positively predicted VIQ. These models accounted for 36% of the variance in IQ. The negative relationship between IQ with lifestyle and antisocial facets may suggest a cognitive deficit related to difficulty in regulating behavior (Vitacco et al., 2005). Further, the finding that the interpersonal facet had a positive relationship with IQ is in line with Cleckley’s (1988) conceptualization that psychopathic traits in combination with intelligence allow individuals to more easily manipulate and take advantage of others. This result was replicated using FSIQ within a sample of detained male inmates, the only difference was that the antisocial facet had a positive relationship with FSIQ; psychopathy facets accounted for 25% of the variance in IQ (Vitacco et al., 2008).

When the type of intelligence was investigated against the three-facet model, the results were similar. Interpersonal style consistently correlated positively, with both VIQ and a comprehensive view of intelligence that incorporated creativity (Sternberg’s Triarchic Abilities Test; STAT). In contrast, the lifestyle facet only correlated with STAT intelligence, in a positive direction (Salekin, Neumann, Leistico, & Zalot, 2004). Taken together, when intelligence was predicted by specific facets of psychopathy, the most consistent finding was that the interpersonal facet was related positively with intelligence (which is in line with Cleckley’s conception of psychopathy) and the lifestyle facet was related negatively to intelligence, but the
direction of this relationship depended on the type of intelligence that was investigated. Therefore, intellectual abilities may assist in both allowing those with interpersonal traits to manipulate, control, and coerce their victims, while those who lack intellectual abilities are not able to benefit from the inhibitory action of intelligence on impulsive and antisocial behavior. Not many studies have attempted to reconcile psychopathy, intelligence, and hormones together, but one study found that having both high CU traits and verbal ability was related to increased violent delinquency, and these individuals also displayed reduced stress reactivity, measured through skin conductance (Muñoz, Frick, Kimonis, & Aucoin, 2008).

Further, given Van Honk and Schutter’s (2006) prediction that the specific HPA/HPG balances implicated in the etiology of psychopathy are more pronounced among primary psychopaths, the HPA/HPG balance may act as the drive to manipulate and use others, while intelligence may assist in propagating interpersonal traits by adding the tools to successfully do so. Whether or not intelligence is linked to psychopathy, these aforementioned findings make examining intelligence as a moderator of specific biological and non-biological outcomes an important endeavor.

**Psychopathy, Maturity, and Hormonal Balance**

Intelligence and maturity occur predominantly in the same regions of the brain, including the PFC and limbic areas (Court, 2013). In addition, measures of intelligence and psychosocial maturity are often correlated (Colwell, Cruise, Guy, McCoy, Fernandez, & Ross, 2005; Galambos, MacDonald, Naphtali, Cohen, & de Frias, 2005; Mantzicopoulos, & Oh-Hwang, 1998). Given their similarities, like intelligence, maturity may be a protective factor against developing psychopathic traits earlier in life (Pan, 2009) or it may, too, moderate the expression of psychopathy.
Maturity has been conceptualized in many different ways, including maturity based on chronological age, psychosocial maturity, and physical development or puberty. For the purposes of this paper, psychosocial maturity will be the focus, as that is what was measured via the Risk Sophistication Treatment Inventory (RSTI-SR). Psychosocial maturity refers to one's abilities to demonstrate (1) responsibility: autonomy, self-reliance, and a clear identity; (2) temperance: not giving in to impulses, avoiding extreme decision making, and requesting assistance from others when appropriate; and (3) perspective taking: understanding the complexity of a given situation and evaluating decisions within a broader context (Steinberg & Cauffman, 1996). Through prototypical analysis, researchers have determined that psychosocial maturity, especially for the purposes of the juvenile justice system, is best represented by three basic developmental skill sets: (1) autonomy, (2) cognitive capacities, and (3) emotion regulation (Salekin, Rogers, & Ustad, 2001; Salekin, Yff, Neumann, Leistico, & Zalot, 2002).

The process of maturation is highly dependent on hormones. Hormones, especially testosterone, relate to physical maturity during adolescence through their action on the brain and throughout the body, and consequently psychosocial maturity. Through adolescence the brain undergoes a constant state of development and maturation (Peper, van den Heuvel, Mandl, Pol, & Van Honk, 2011), which coincides with an insurgence of sex hormones, including estrogen, progesterone, and testosterone (Sisk & Foster, 2004). Sisk and colleagues (2004) postulated that at the start of puberty the brain activates the HPG to release the gonadotropin-releasing hormone, which increases steroid hormone production. These steroid hormones feedback to the brain and modulate secretion of gonadotropin releasing hormones, thus organizing, and activating neural structures implicated in adolescent behavior, specifically reproductive behavior. Testosterone levels increase across pubertal stages for both sexes (Matchock, Dorn, & Susman, 2007) and
increase steadily with age in males until at least age 17 (Khairullah et al., 2014). An increase in white matter throughout the brain coincides with endogenous levels of testosterone, but this finding was only consistent for males (Peper et al., 2011). Myelination also increases within the frontal lobe, an area important for cognitive processes and intelligence. During adolescence, this surge in myelin synthesis occurs more for males than females (Giedd et al., 1999), but females often advance through the pubertal stages sooner than males (Gunnar, Wewerka, Frenn, Long, & Griggs, 2009). Cortisol reactivity in response to a social stressor has also been shown to increase with pubertal stage, after controlling for age (Gunnar et al., 2009).

The introduction of increased estrogen and testosterone during puberty is also implicated in adolescents’ emotional volatility and impulsivity (Arain et al., 2013). Moreover, one major change that occurs in the brain is maturation of the limbic system, implicated in self-control, decision-making, emotion regulation, and risk taking behaviors (Arain et al., 2013). These behaviors are similar to Steinberg and Cauffman’s (1996) criteria of responsibility, temperance, and perspective criteria of psychosocial maturity, as well as the prototypical psychosocial conceptualization of autonomy, cognitive skills, and emotion regulation.

Studying maturity as it relates to adolescent psychopathy is also important in order to uncover the etiology of the syndrome. Adolescence is a time of gaining and solidifying maturity and independence, thus adolescents cannot be assumed to have established unwavering maturity. Further, because adolescents have not yet fully developed, their criminal trajectory might also change over time and those who are currently committing crimes may stop engaging in criminal activity once they reach adulthood (e.g., Moffitt, 1993). Moreover, psychopathic traits are often presented at juvenile hearings due to the similarity between psychopathic traits and the criteria that often need to be presented in waiver hearings, such as of dangerousness, treatment
amenability, and developmental maturity. Psychopathic traits are often presented for such purposes as evaluating or justifying transfer of adolescents to adult court (Penney & Moretti, 2005).

Among the few investigations of psychopathy in relation to developmental maturity, measures of maturity have consistently correlated negatively with measures of psychopathy (e.g., Sociomoral Reflection Measure-Adapted Version, Hornsveld, Kraaimaat, Zwets, 2012; Risk, Sophistication-Maturity, Treatment Inventory, Leistico & Salekin, 2003; Psychosocial Maturity Inventory, Future Outlook Inventory, Resistance to Peer Pressure Inventory, and Weinberger Adjustment Inventory, Skeem & Cauffman 2003). More specifically, psychopathic traits have been found to have an inverse relationship with juvenile’s amenability to treatment (Leistico & Salekin, 2003), responsibility, social perspective taking, time-perspective, and impulse control (Skeem & Cauffman 2003). Moreover, using the three-facet model, the interpersonal facet has been associated with impulse control; the affective facet was associated with time perspective, social perspective, and impulse control; and, the lifestyle facet was associated with time perspective and impulse control, all with an inverse relationship. Furthermore, studies have used maturity as a measure of divergent validity for psychopathy, given that some aspects of psychopathy, such as lack of remorse and irresponsibility, may be extreme manifestations of common immaturity within adolescent development (e.g., lacking social perspective taking or impulse control, Skeem & Cauffman 2003). However, it should be noted that many psychosocial maturity scales include prosocial items, which inflate the strength of the inverse correlations with psychopathy facets. Still, there are examples of “mature” psychopathic individuals both in the theoretical literature and in the scientific literature (e.g., Babiak, & Hare, 2006; Cleckley, 1988). For example, it is plausible to imagine psychopathic individuals who are socially skilled, can
regulate their emotions, and can plan ahead with ease. Much of this contradictory information suggests that developmental maturity might moderate the expression of psychopathy and highlights the importance of examining maturity as a potential moderator of psychopathic expression in adolescents. These complicated associations and equivocal findings on the relations between psychopathy, intelligence, maturity, and hormones, which were largely dependent on age and gender, highlight the need for research that carefully documents the potential moderating effect of key variables.

The Current Study

Understanding the neurobiological underpinnings of psychopathic traits can assist in understanding the etiology of psychopathy and determining those who are at risk of developing psychopathic traits (Shirtcliff et al., 2009). Both of these benefits of understanding neurobiology can help us develop interventions, measure efficacy of interventions for this group, and help us understand what attributes of psychopathy may not benefit from therapy due to intractable aspects of their biology. The current study was designed to help researchers and clinicians better understand the balance of HPA and HPG functioning among adolescents with psychopathic traits. This study was also designed to further elucidate whether these relations are moderated by intelligence and maturity. In order to examine these relations, baseline, post-stressor, and post-stress recovery samples of salivary cortisol and testosterone were compared. The current study is similar to past research on this topic in that a) this study examines the relationship between psychopathy and baseline levels of cortisol and testosterone as well as cortisol reactivity, and b) the study also examines how psychopathic traits relate to intelligence. This study differs from past research in that a) it examines how hormonal reactivity relates to psychopathy within an incarcerated sample of juveniles, b) it examines testosterone reactivity rather than baseline
testosterone and cortisol reactivity alone to alleviate the potential problem of testosterone reactivity to social stressors (e.g., competition; Edwards & Casto, 2015), and c) the study examines the impact that intelligence and maturity have on the relation between psychopathy and hormonal balance.

The present study addressed five specific hypotheses. First, based on the triple balance model of emotion (Terburg et al., 2009; Van Honk & Schutter, 2006) and previous research relating to the hormonal make-up of psychopathic traits (e.g., Glenn et al., 2011), it was hypothesized that the ratio of baseline testosterone to cortisol reactivity (using area under the curve) would predict psychopathic traits. More specifically, it was expected that as psychopathic traits increased so would baseline testosterone, but cortisol levels in reaction to stress would be low. Second, following Van Honk and Schutter’s (2006) hypothesis that the ratio of cortisol to testosterone would be more pronounced among those with primary psychopathic traits, it was further hypothesized that this relationship would be stronger for the interpersonal (facet 1) and affective (facet 2) facets of psychopathy, than lifestyle (facet 3), or antisocial (facet 4) facets. Third, considering confirmatory factor analysis data on how intelligence relates to the three and four facet model of psychopathy (e.g., Vitacco et al., 2005; Vitacco et al., 2008; Salekin et al., 2004) and Cleckley’s (1988) conceptualization of intelligence as being a hallmark of psychopathic traits, it was expected that intelligence (as measured by FSIQ) would hold a small positive relationship with the interpersonal facet (facet 1) of psychopathy and a negative relationship with the lifestyle facet (facets 3) using the four-facet model. Fourth, it was hypothesized that maturity would behave much like intelligence, with a positive relationship with the interpersonal facet and a negative relationship with the lifestyle facet of psychopathy. Finally, given previous findings on the relationship between stress reactivity, cognitive abilities,
and CU traits (Muñoz et al., 2008), as well as the impact of hormones, especially testosterone, on maturing the brain (Sisk & Foster, 2004), it was expected that intelligence and maturity would moderate the strength of the relationship between psychopathic traits and the ratio of baseline testosterone to cortisol reactivity, specifically within the interpersonal facet.
CHAPTER 2

METHODOLOGY

Participants

All youth (male and female) that attended visitation at the local detention center were approached for the study. In total, 116 youth/visitor groups were approached to participate in the study, 73 (63%) of which agreed to participate. The most common reasons for not agreeing to participate were simply not wanting to (35%) or not having a guardian present to consent (28%). Of these, twelve (16%) left the detention center and were no longer interested or available to complete the study in the community and three (4%) completed the first session, but one moved, one was sent off to a treatment program, and one was no longer reachable. The final sample comprised 58 participants (50% of those approached). The sample was predominantly male (78%) and African American (64%), followed by European American (33%), and mixed ethnicity (3%). Ages in this sample ranged from 12 to 18 with a mean age of 15.83 ($SD = 1.33$).

Apparatus

Assay of Cortisol and Testosterone Levels

Salivettes were used to collect and store the salivary cortisol and testosterone samples. Salivettes are a three-piece apparatus, with a container, a lid and a cotton swab. A research assistant (RA) asked participants to chew on the cotton swab for two minutes per time point. The RA then requested that the participant enclose the cotton swab in the Salivette container without touching it with their fingers, to prevent contamination. The Salivettes were transported from the
data collection site in a cooler, then stored in a -80 freezer until all participants were collected in order to analyze all of the samples at once.

**Materials**

**Social Stress Induction Task**

Given the impact of oral presentations on cortisol reactivity (e.g., de Vries-Bouw et al., 2012; Fishbein et al., 2009; Popma et al., 2007), an adaption of the Trier Social Stress Test (TSST, Kirschbaum, Pirke, & Hellhammer, 1993) protocol was used for this study. The TSST has been adapted for use as a stress induction in child and adolescent studies with success. Kudielka, and colleagues (2004) adapted the task by not providing children with preparation time for the presentation. Bouma, and colleagues (2009) adapted the task by requesting that participants present generally about themselves and their lives in front of a camera. For the purposes of this study, the TSST was adapted to be more relevant to detained participants by adding the criteria that participants discuss themselves in relation to their criminal behavior (a factor that was common to all of our participants). To better suit the young sample, we also reduced the time duration and burden on participants.

Nine different confederates, four female and five male, were used throughout data collection to increase the generalizability of the findings. The RA began the task by introducing participants to one of the nine confederates and delivering the following instructions:

“In the next task, you will have three minutes to prepare a three minute presentation for Ms./Mr. Smith (the confederate). Imagine that Ms./Mr. Smith is conducting a release review for you and she/he is responsible for deciding whether you should receive a reduced sentence. Prepare a presentation that explains the aspects about yourself and your current situation that make you a good candidate for early release.”
The RA further explained that the presentation would not actually affect their sentence, in order to stay within ethical limits of keeping the participant informed. The confederate then provided the participant with a pencil and paper and provided three minutes to prepare. Following preparation time, the confederate asked the participant to begin the presentation. The confederates also interjected once during the presentation to ask the participant to explain how they thought their crime had impacted their family. If the participant finished the presentation before three minutes had elapsed, the confederate responded with the standardized response “your time is not up, please continue.” If the participant discontinued a second time, the confederate remained silent until the three minutes had passed. After three minutes, the participant was asked to sequentially subtract 7 from 100 as quickly and accurately as possible. This task took two minutes to complete and every time that participants made an error they were asked to restart from 100.

Measures

Psychopathic Traits

In order to measure levels of psychopathy, the Psychopathy Checklist-Youth Version (PCL-YV; Forth, Kosson, & Hare, 2003) was administered. The PCL-YV is a 20-item scale with three coding options for each item (0, 1, or 2; 2 meeting full criteria for the item) that yields a total score of 40. This interview has been used extensively in research measuring psychopathic traits in adolescents. Across numerous investigations the measure has established various forms of reliability, including internal consistency (ranging from .85-.94) and inter-rater reliability. The best inter-scorer reliability was found within institutional settings (.93), followed by probation (.90). The PCL-YV has also demonstrated moderate to large associations with other established
measures of psychopathy including the Antisocial Process Screening Device and Conduct Disorder within the DSM-IV-TR (Forth et al., 2003).

For this study, three independent raters coded the PCL-YVs by using information from the PCL-YV interview. Three collateral sources were also used to code the PCL-YVs. The Inventory of Callous-Unemotional Traits-Youth Version (ICU, Frick, 2004), was used as a self-report of CU traits. In addition, the Interpersonal Callous Emotion-Parent Report (ICE-P, Salekin, unpublished) was used to gain information from guardians on participants’ behaviors and traits related to psychopathy. Further, the Self-Report of Offending inventory (SRO; Huizinga et al., 1991) was used to gain a comprehensive self-report of their offense history. The three independent coders consisted of the first author, who coded the majority of the interviews (n=37; 64%), and two RA’s who coded twelve (21%, designated as Coder One) and nine (15%, designated as Coder Two) interviews each. To ensure reliability across coders, inter-rater reliability was calculated for fourteen randomly selected files (seven files per RA) using a two-way mixed, consistency-based, single-measures interclass correlation (ICC; McGraw & Wong, 1996) in comparison to ratings completed by the first author. For total PCL-YV scores, the resulting ICCs were within the excellent range (Cicchetti, 1994) for both RAs, ranging from $ICC = .96$ for Coder Two to $ICC = .97$ for Coder One. The coders differed slightly on each of the four facets of psychopathy, but the ICCs remained within the excellent range for each of the Interpersonal Facet (Coder One: $ICC = .76$; Coder Two: $ICC = .91$), the Affective Facet (Coder One: $ICC = .87$; Coder Two: $ICC = .77$), the Lifestyle Facet (Coder One: $ICC = .87$; Coder Two: $ICC = .93$), and the Antisocial Facet (Coder One: $ICC = .97$; Coder Two: $ICC = .94$). The PCL-YV demonstrated excellent internal consistency for this sample, Cronbach’s alpha of .86, and was significantly correlated with the ICU, demonstrating good external validity, $r = .37, p = .01$. 

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Collateral sources of psychopathy. The Interpersonal Callous Emotion-Parent Report (ICE-P; newly developed by Salekin and colleagues) was provided to guardians as a parent report of psychopathic traits. It contains 36 items regarding interpersonal callousness and lack of emotionality among adolescents. The questionnaire takes approximately ten minutes to complete and the items are scored on a 3-point Likert scale with the options: no (0), some (1), or yes (2). Given that this is a new measure, the psychometric properties of the measure have not yet been investigated.

The Inventory of Callous-Unemotional Traits-Youth Self-Report (ICU-YSR; Frick, 2004) was provided to participants as a self-report of CU traits. It consists of 24 self-report items that can be clustered into three subscales: callous, uncaring, and unemotional traits. The questionnaire takes approximately ten minutes to complete and the items are scored on a 4-point Likert scale from 0 (not at all true) to 3 (definitely true). Support has been found for the reliability and validity of the ICU (Essau, Sasagawa, & Frick, 2006; Fanti, Frick, & Georgiou, 2009; Roose, Bijttbier, Decoene, Claes, & Frick, 2010).

To gain an understanding of the participants’ full criminal repertoire, including unreported criminal offending, our research group adapted the Self-Report of Offending inventory (SRO; Huizinga et al., 1991) into an interview to probe for a more in depth understanding of the severity, frequency, and type of participants’ offense history. The SRO has 24 items that assess a variety of different types of offenses (violent, property, money fraud etc.) and queries participants about how many times they have committed specific crimes (e.g., “taken something from another person by force, using a weapon?”) with pre-set intervals of frequency (e.g., one time, two times, five or more times etc.). Items 12 (“Have you ever been paid by someone for sex”) and 14 (“Have you ever killed someone”) were excluded because of the
sensitive nature of these questions and low base rates of these criminal behaviors among youth. Within the interview, the RA asked participants if they had ever engaged in the specific criminal behaviors. The RA then gained a more precise measure of frequency by asking the number of times that the youth had engaged in each specific criminal act in their lifetime, as well as within the past month. Finally, RAs asked participants what specific measures they had taken to avoid detection by the criminal justice system and whether they were successful in hiding their crime(s). This was done in order to aid in scoring specific items on the PCL-YV, including manipulation for personal gain, impression management tactics, and a comprehensive history of serious criminal involvement, and criminal versatility.

Despite general skepticism that youth will be honest about their history of offending, a review of self-report of offending studies found test-retest reliability estimates ranging from .85-.99 (Huizinga & Elliott, 1986). Given that participants are unlikely to remember fabricated answers from a previous test, good test-retest reliability suggests that participants, for the most part, seem to be honest with researchers about their offense history. Further, only one study has examined the discrepancy between results obtained from self-report versus interview questions regarding offending behavior (e.g., Horney & Marshall, 1992) and they found that there was no difference between the amounts of offending reported for either format.

**Intelligence**

Intelligence was indexed using the Kaufman Brief Intelligence Test-2nd version (K-BIT 2, Kaufman & Kaufman, 2004). The K-BIT-2 is a test used to measure both verbal and non-verbal ability of people between the ages of 4 and 90 and takes approximately 20 minutes to administer. The instrument uses verbal knowledge, riddles, and visual images to examine one’s ability to comprehend and respond to questions. It was standardized using a sample of 2120 participants in
a nationally representative sample. The KBIT-2 has good convergent validity and has been compared against other brief instruments (Bowers, & Pantle, 1998) as well as more comprehensive intellectual batteries (WISC-III, Canivez, Neitzel, & Martin, 2005; Stanford Binet; Prewett, & McCaffery, 1993), with reliable results. When compared to the more established intelligence screener of the Shipley Institute for Living Scale, the original K-BIT produced comparable IQ scores. In addition, the K-BIT appeared to be better suited to forensic participants and below average readers (Bowers, & Pantle, 1998). Thus, it was an appropriate instrument for our detained sample.

**Maturity**

The Risk-Sophistication-Treatment-Inventory-Self-Report (RSTI-SR; based off the RSTI; Salekin, 2004) was used as a measure of psychosocial maturity. This measure includes 74 items assessing youths’ personality factors, antisocial behaviors, attitudes about legal difficulties, and environmental influences. The RSTI-SR is composed of three subscales measuring items specific to risk, sophistication-maturity, and treatment amenability. The RSTI-SR has not been extensively studied, but the RSTI from which it is based has established convergent validity by demonstrating significant relationships between each cluster and important defining factors. More specifically, risk was related to younger age of criminal onset, violent crimes, and both violent and non-violent symptoms of Conduct Disorder (Leistico & Salekin, 2003; Spice, Viljoen, Gretton, & Roesch, 2013). Further, the measure has demonstrated good to excellent internal consistency within each cluster (risk: \( \alpha = .77 \), sophistication-maturity: \( \alpha = .69 \), treatment amenability: \( \alpha = .87 \); Spice et al., 2013). In this sample, the sophistication maturity scale demonstrated excellent internal consistency reliability, \( \alpha = .84 \).
Self-Report of Distress

Given that the effectiveness of the TSST for cortisol stress induction is unknown among detained adolescents, we also collected self-report measures of distress in correspondence with the baseline, post-stressor, and post-stress recovery hormonal samples, to explore whether the participants found the task subjectively distressing. The Positive and Negative Affect Scale (PANAS; Watson, Clark, & Tellgen, 1988) was provided to participants as a measure of distress. The PANAS is a brief questionnaire that requires participants to endorse any emotions that they are currently experiencing from a list of emotions. The scale contains six versions based on which time is specified in the instructions: moment, today, past few days, week, past few weeks, year, and general. Given our interest in participants’ acute emotional state, only the moment version of the scale was used. The moment version of the PANAS has good internal consistency for both the positive affect (PA; $\alpha=.89$) and negative affect (NA; $\alpha=.85$) factors and excellent convergent and discriminant validity when compared to established mood descriptors (PA, positive mood descriptors, $r=.95$ and negative mood descriptors, $r=-.02$; NA, positive mood descriptors, $r=-.15$ and negative mood descriptors, $r=.91$). The PANAS also has standard means for positive ($M=29.7$) and negative scales ($M=14.8$). This allows for comparisons across samples. The PANAS was used in the present study to gauge differences among a range of emotions between baseline, post-stressor, and post-stress recovery, rather than focusing solely on negative affect states. The internal consistency reliability for the positive ($\alpha=.83$) and negative scales ($\alpha=.79$) were within the excellent range for this sample. Given that the PANAS was provided to participants three times during the procedures, test-retest reliability was tested for the positive and negative affect scales using a two-way mixed, consistency-based, single-measures
ICC. The ICC analysis resulted in ICC statistics within the good to excellent range for the negative (ICC = .70) and positive affect scales (ICC = .84), respectively.

In conjunction with the PANAS, the State–Trait Inventory for Cognitive and Somatic Anxiety (STICSA; Ree, MacLeod, French, and Locke, 2000) was also used as a self-report of stress. The STICSA is a 21-item inventory used to assess participant’s physical and psychological symptoms of stress and anxiety. Participants were asked to indicate how much they agreed with a statement that described their current state (e.g., “I can’t get some thought out of my mind.” or “I feel trembly and shaky.”). They were provided with a 4-point Likert scale ranging from 1-“not at all” to 4-“very much so.” The STICSA has good internal consistency (α= .92) and is strongly correlated with two other measures of anxiety suggesting good convergent validity (State–Trait Anxiety Inventory (STAI), r=.61, p = .01; Depression Anxiety Stress Scales (DASS), r = .67, p = .01; Gros, Antony, Simms, & McCabe, 2007). Given our interest in examining current stress states rather than traits within participants we only used the state version of the inventory. Items 5 (“I feel like I’m missing out on things because I can’t make up my mind soon enough.”) and 16 (“I keep busy to avoid uncomfortable thoughts.”) were excluded, as these statements did not directly address the participants’ current state. For this sample, the internal consistency reliability was within the excellent range, Cronbach’s alpha was .89. Like the PANAS, this measure was provided to participants in the second session in correspondence with the baseline, post-stressor, and post-stress recovery hormonal samples, thus a two-way mixed, consistency-based, single-measures ICC analysis was conducted to gauge test-retest reliability. The ICC analysis demonstrated good test-retest reliability, ICC = .75.
Procedure

Following consent and assent from the guardian and the youth, a research assistant (RA) provided the guardian with a measure of psychopathy (ICE; Salekin, unpublished) to gain collateral information in order to code the PCL-YV. An RA then contacted the youth or the facility to arrange a time to meet with them for the first of two sessions. For the first session, participants started by filling out questionnaires regarding psychosocial-maturity (RSTI-SR, Salekin 2004), CU traits (ICU, Frick, 2004) and offense history (SRO, Huizinga et al., 1991). RAs also administered the PCL-YV (Forth et al., 2003).

Following the first session, the RA arranged a time to meet with the participant. This second session was scheduled at a time in which the youth had neither eaten within the past hour or was regularly scheduled to eat. This was done in order to account for diurnal fluctuations of cortisol related to food anticipation and consumption and ensure that samples were not contaminated with residual food particles following consumption (Legler, Brandenberger, Hetter, Simeoni, & Reindhart, 1982). Given that the detention center is a highly controlled environment with regularly scheduled meals and prohibitions on caffeinated beverages and cigarettes, there was no need to ask participants to refrain from any substances and the second session was scheduled based on their meal schedule. However, those youth in the community were asked to abstain from consuming any food, drink, or substances an hour prior to the session. To further control for diurnal fluctuations of cortisol all participants began the procedures between the times 2 pm and 6 pm, as cortisol peaks in the morning and usually settles in the afternoon (Khan-Dawood, Choe, & Dawood, 1984; Yehuda et al., 2003). The RA began the second session by having participants complete the PANAS and STICSA to establish the participants’ baseline self-report of stress. The RA then collected a baseline measure of salivary
cortisol and testosterone using the Salivettes. This was followed by an IQ test (K-BIT 2, Kaufman, & Kaufman, 2004). Following the administration of the IQ test, RAs introduced the participants to a confederate and begin the adapted TSST, which took eight minutes to complete. The RA then provided participants with the self-report of stress reactivity measures again and started a movie. The same movie was played for all participants so that participants were occupied while the RA waited twenty minutes for the cortisol stress reaction to be measurable in saliva and to control for what participants did during this time. When twenty minutes had passed, the RA collected the post-stressor cortisol and testosterone sample. Participants were given an additional twenty minutes to relax (while still watching the movie), followed by self-report of stress reactivity and a final collection of salivary cortisol and testosterone to obtain a measure of post-stress recovery. The entire task took approximately four hours over two session and adolescents were provided with $20 for their participation in the study.

**Extraction and Analysis of Cortisol and Testosterone Levels**

Our lab collaborated with an affiliate within the Biology Department at the University of Alabama to properly assay the separate baseline, post-stressor, and post-stress recovery cortisol and testosterone. The first step in processing the Salivettes was to transport them on ice to a centrifuge within the Anthropology Department on campus. We allowed approximately thirty minutes for the samples to thaw and spun 36 samples at a time. The centrifuge liberated approximately 1 ml of saliva from each cotton swap within the Salivettes. Thus, 1 ml was pipetted from each participant or the maximum amount of saliva available, for those with less than 1 ml. Amounts less than 1 ml were controlled for when calculating pg/ml of hormone. The samples were then combined with 2 ml each of distilled water in 18 x 150 mm tubes. Cortisol and testosterone were extracted from the water/saliva solution using C18 solid phase extraction
Columns were primed using two consecutive washes with 2 ml of 100% MeOH followed by two consecutive washes with 2 ml of distilled water. In order to extract the hormones from possible contaminants in the saliva, the 3 ml samples were pulled through the columns using the vacuum manifold, leaving the hormone bound to the columns. Two consecutive washes of distilled water were pulled through the columns a final time to ensure removal of bound salts. Hormone was eluted from the columns into 12 × 75 mm borosilicate vials using two consecutive 2 ml washes of 100% MeOH. The 4 ml of eluted solvent was capped and stored in a -20 degree freezer overnight, and then evaporated using an evaporating manifold connected to a nitrogen tank with low pressure (7 bar). This method left a pellet of hormone dried on the bottom of the vial. The pellet was then resuspended in 20 μl of 100% EtOH and 280 μl of enzyme immunoassay buffer (EIA). 20 μl of the final solution was removed from each vial to create the pooled sample, and stored at -20 °C overnight until radioimmunoassays could be conducted.

Testosterone and cortisol radioimmunoassays were conducted using pre-coated enzyme immunoassay 480 strip well plates (testosterone: Mouse Anti-Rabbit IgG; cortisol: Goat Anti-Mouse IgG) purchased from Cayman Chemicals (Ann Arbor, MI). Each sample was run in duplicate using five separate assay plates per hormone. All cortisol and testosterone values are reported as pg/ml. The intra-assay coefficients of variation for the cortisol were all below 18% with an average of 12.6% and the inter-assay coefficient of variance was 16.2%. None of the intra-assay coefficients of variation for testosterone exceeded 19% with an average of 10.7% and the inter-assay coefficient of variance for testosterone was 13.7%.
Before running individual samples, serial dilutions from 1:1 to 1:32 were conducted from the pooled sample to ensure use of ideal dilutions to detect testosterone and cortisol levels. The curve generated from the serial dilutions of testosterone from our sample was parallel to the standard curve created from the EIA kit testosterone standards, thus when comparing slopes, $t(12) = -.11, p = .91$. Similarly, the curve generated from the serial dilutions of cortisol was parallel to the standard curve created from standards supplied with the cortisol EIA kit, thus when comparing slopes, $t(12) = -.03, p = .98$. We also used the serial dilutions to determine which concentrations were within the linear phase of the standard curve. Based on the serial dilution curve that was produced, we determined that 1:4 best matched the level of detectable hormones within the pooled sample. Thus, all samples were diluted with an additional 840 μl of EIA buffer yielding a total volume of 1120 μl per sample. The remaining 2025 μl pool was also diluted to a volume of 6075 μl.

One score of post-stress recovery was uninterpretable because it yielded an amount that was higher than the maximum binding for the tray. All scores for testosterone and cortisol were log10-transformed to adjust for skewness. From these transformations, one outlier (greater than 3 standard deviations from the mean) was removed from the baseline cortisol scores. This resulted in the removal of one participant’s baseline cortisol score from the analyses and consequently the removal of all said participant’s cortisol composites that required baseline cortisol for calculation (i.e. cortisol AUC$_G$, cortisol AUC$_I$, change in hormone from baseline to post-stressor, change in cortisol from baseline to post-stress recovery, and all ratios).

Both cortisol and testosterone reactivity were assessed by calculating the area under the curve with respect to ground (AUC$_G$) for each saliva sample time point (baseline, post-stressor,
and post-stress recovery) across participants using the formula below in order to derive a single score denoting hormone stress reactivity (Pruessner et al., 2003):

$$\sum_{i=1}^{k-1} [t_i^* (m_i + m_{i+1})/2]$$

Within this formula, \( k \) denotes the number of samples, \( t_i \) is the interval between sample \( i \) and sample \( i + 1 \) and \( m_i \) is the level of the hormone for sample \( i \).

Area under the curve with respect to increase (AUC\( _I \)) was also calculated for cortisol and testosterone, in order to emphasize changes in hormone levels across time points. AUC\( _I \) is calculated by subtracting the product of the baseline score multiplied by the sum of the time distances, denoted by the formula below (Pruessner et al., 2003):

$$\left( \sum_{i=1}^{k-1} [t_i^* (m_i + m_{i+1})/2] \right) - \left( m_1 \sum_{i=1}^{n-1} t_i \right)$$

To examine possible relationships between psychopathy and ratios of testosterone and cortisol, each cortisol and testosterone measure was first converted to standard t-scores. Then, as outlined in Glenn and colleagues (2011), the ratios of baseline testosterone/baseline cortisol and baseline testosterone/cortisol AUC\( _G \) were calculated. In order to explore other possible relationships, baseline cortisol/baseline testosterone, baseline testosterone/cortisol AUC\( _L \) ratios, baseline cortisol/testosterone AUC\( _G \), and baseline cortisol/testosterone AUC\( _I \) were also calculated.

**Statistical Analyses**

First, descriptive analyses were conducted to determine means, standard deviations, skewness, and kurtosis for all study variables. In addition, Pearson Product Moment correlations were generated, to determine the relations between all study variables. These correlational analyses were also run to establish any covariates that would influence the impact of the
predictors on the outcome variables. More specifically, age and sex, as well as the time of day that the saliva samples were collected were tested as correlations with hormones, given that cortisol and testosterone levels may vary based on age (Carpenter, Tyrka, Ross, Khoury, Anderson, & Price, 2009), sex (Glenn et al., 2011; Loney et al., 2006), and diurnal fluctuations based on time of day (Legler et al., 1982).

Next, to further test the study hypotheses, hierarchical regressions were run to determine the impact of hormones, intelligence, and maturity variables on psychopathy variables. Specifically, age and sex were run as covariates in step one. Individual hormone composites, including baseline and reactivity measures ($AUC_G$ and $AUC_I$) of cortisol and testosterone, as well as the ratio of baseline testosterone to cortisol reactivity ($AUC_G$ and $AUC_I$), were contained in step two. Either intelligence or maturity variables were contained in step three, including K-BIT-2 total scaled score and the RSTI sophistication-maturity scale. Within the final step interactions between hormone and intelligence/maturity variables were entered as predictors. Separate hierarchical regressions were conducted for each facet of psychopathy.

In addition to the primary analysis to investigate the proposed hypotheses, exploratory analyses were conducted to further investigate the relationships between psychopathy and the hormonal variables in this sample. Previous research has demonstrated the influence of psychopathic traits on hormone levels across time points (e.g., O’Leary et al., 2010). Thus, using SAS Version 9.2, separate mixed, within-subjects repeated measures analyses of variance and covariance were used to investigate the effects of psychopathy on hormone variation across time point (baseline, post-stressor, and post-stress recovery of testosterone and cortisol), while controlling for age, sex, and time point. Age and sex, were entered as between-subjects fixed factors, time was treated as a fixed within-subjects repeated-measure, psychopathy scores (total
score, interpersonal facet, affective facet, lifestyle facet, or antisocial facet) were entered as covariates, and participants were treated as random variables. Covariance models were fitted using Akaike (AIC) criteria. The best fitting covariate structure for cortisol (AIC = 153.6) and testosterone (AIC = 95.4) across time point was Variance Components. Thus, each mixed model was modeled using the Variance Components covariate structure.

General linear models (GLMs) were conducted to examine main effects of the hormone composites on psychopathy total score and individual facets, controlling for age and sex. The specific hormone composites of interest included baseline hormone, hormone reactivity (AUC\textsubscript{G} and AUC\textsubscript{I}), change in hormone from baseline to post-stressor (Log10 transformed post-stressor sample minus Log10 transformed baseline sample), change from post-stressor to post-stress recovery (Log10 transformed post-stress recovery sample minus Log10 transformed post-stressor sample), change from baseline to post-stress recovery (Log10 transformed post-stress recovery sample minus Log10 transformed baseline), as well as the previously discussed ratios (baseline testosterone/baseline cortisol, baseline cortisol/baseline testosterone, baseline testosterone/cortisol AUC\textsubscript{G}, baseline cortisol/testosterone AUC\textsubscript{G}, and baseline testosterone/cortisol AUC\textsubscript{I}, baseline cortisol/testosterone AUC\textsubscript{I}). Transformed hormonal levels were used to ensure that values were standardized before being compared. This helped to eliminate any artifacts due to the time at which the samples were collected and ensure that all values were within the same scale. Any models that included significant relationships among covariates (i.e. age and sex were related to the hormone composite) were run as regressions without the addition of age and sex. Those that only had relationships between age and the hormone composite were run as GLMs with standardized residual values (from the regression of the hormone composite with age) in place of age.
Given the proposed feedback relationship between cortisol and testosterone and findings suggesting that psychopathy scores may predict coupling of these hormones (Johnson et al., 2014), as well as hormone levels in conjunction with stressful life circumstances (Gostisha et al., 2014), general linear models were also used to examine main effects of psychopathy total score and individual facets on the hormone composites, controlling for age and sex. Each of the composites that were included in the GLMs with hormones on psychopathy were also included.
CHAPTER 3
RESULTS

Descriptive Statistics

Descriptive statistics for psychopathy, cortisol, testosterone, intelligence, and maturity scores, as well as self-report of stress are provided in Table 3.1. Across the sample, PCL-YV total scores ranged from one to thirty-five ($M=15.22; SD=8.17$), with ten (17.24%) participants scoring greater than or equal to a total score of twenty-five. This score is relatively low in comparison with the majority of mixed-gender detained adolescent samples (e.g., $M = 20.50; SD = 7.39$; Tsang et al., 2015; $M = 22.74; SD = 5.48$; Fink, Tant, Tremba, & Kiehl, 2012), but comparable to a female sample of detained youth in Alabama ($M = 16.37; SD = 8.00$; Schrum, & Salekin, 2006) and a male sample of detained youth in the Dutch provinces of Limburg and Brabant ($M = 13.84; SD = 6.48$, Feilhauer, Cima, & Arntz, 2012). Untransformed cortisol means varied from 544.31 ($SD = 451.83$) at baseline to 632.99 ($SD = 641.79$) at post-stressor, and 507.08 ($SD = 558.37$) at post-stress recovery. In addition, untransformed testosterone means varied from 70.29 ($SD = 71.81$) at baseline to 55.51 ($SD = 33.22$) at post-stressor, and 58.61 ($SD = 42.12$) at post-stress recovery. Cortisol and testosterone values were within normal limits in line with previous research studying comparable juvenile populations (e.g., cortisol: Fairchild et al., 2008; Popma et al., 2007; von Polier et al., 2013; testosterone: Denson, Ronay, von Hippel, & Schira, 2013; Loney et al., 2006). Intelligence and maturity had mean scores of 86.19 ($SD = 15.12$) and 40.09 ($SD = 8.86$), respectively.
### Table 3.1: Descriptive statistics

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<tr>
<td></td>
<td>M</td>
<td>N</td>
<td>P</td>
</tr>
<tr>
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<tr>
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<tr>
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<td></td>
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<tr>
<td>SD</td>
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<td>(9.93)</td>
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<table>
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<tr>
<td><strong>Total</strong></td>
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<tr>
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<tr>
<td>M</td>
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<td>SD</td>
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<td>.33</td>
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P1, PANAS Baseline Positive Scale; N1, PANAS Baseline Negative Scale; P2, PANAS Post-Stressor Positive Scale; N2, PANAS Post-Stressor Negative Scale; P3, PANAS Post-Stress Recovery Positive Scale; N3, PANAS Post-Stress Recovery Negative Scale; S1, STICS Baseline; S2, STICS Post-Stressor; S3, STICS Post-Stress Recovery; P, Psychopathy Total; F1, Interpersonal Facet; F2, Affective Facet; F3, Lifestyle Facet; F4, Antisocial Facet; B, Maturity Scale; M1, Autonomy; M2, Cognitive Capacities; M3, Emotion Regulation; I, IQ total; VIQ, Verbal IQ; VNIQ, Non-Verbal IQ; B, Baseline Sample; REC, Post-Stress Recovery Sample; *After log10 transformation; **After log10 transformation and removal of outliers.
Each of the PANAS positive and negative scale total scores and the STICSA total scores were log-10 transformed to adjust for skewness. After log transforming these measures, the post-stressor and post-stress recovery PANAS negative affect scales and STICSA total scores remained skewed. Thus, four outliers were removed from the post-stressor and post-stress recovery PANAS negative scale and post-stress STICSA, and five from the post-stress recovery STICSA.

**Checks For Potential Confounds**

**Confederates**

Results showed that the confederate that the youth was assigned for the TSST was unrelated to changes in testosterone levels, $r = -.02, p = .87$, or cortisol levels, $r = .02, p = .88$, from baseline to post stressor. Further, the sex of the confederate assigned was not significantly related to differences between baseline and post-stressor testosterone, $r = -.09, p = .50$ or cortisol, $r = -.05, p = .73$.

**Medication**

To ensure that participants were not taking medication that would interfere with their cortisol or testosterone levels, we asked each participant about their current medications. Only 22% ($n = 13$) of the sample were taking medication at the time of the second session. Of those who were taking medication, 38% ($n = 5$) were taking multiple medications, with stimulants for the treatment of ADHD ($n = 6, 46\%$), antidepressants ($n = 6, 46\%$), and mood stabilizers ($n = 6, 46\%$) being the most common medications taken by participants, either alone or in combination with other medications. None of the participants reported taking medications with well-documented effects on cortisol or testosterone production, such as beta-blockers, glucocorticoids,
or estrogens (Handa et al., 1994). When analyzed, none of baseline (cortisol: $F(1, 54) = .24, p = .63$; testosterone: $F(1, 55) = 1.61, p = .21$), post-stressor (cortisol: $F(1, 55) = .45, p = .51$; testosterone: $F(1, 55) = 1.32, p = .26$), or post-stress recovery (cortisol: $F(1, 55) = .45, p = .50$; testosterone: $F(1, 54) = 1.68, p = .20$) concentrations differed depending on whether the youth was taking medication or not. Nor were antidepressants, ADHD stimulants, or mood stabilizers significantly related to levels of baseline (ADHD: cortisol, $F(1, 54) = .01, p = .99$, testosterone, $F(1, 54) = .51, p = .48$; antidepressants: cortisol, $F(1, 54) = .24, p = .63$, testosterone, $F(1, 54) = .02, p = .90$; mood stabilizers: cortisol, $F(1, 54) = 1.30, p = .26$, testosterone, $F(1, 54) = .85, p = .36$), post-stressor (ADHD: cortisol, $F(1, 54) = .70, p = .41$, testosterone, $F(1, 54) = 2.34, p = .13$; antidepressants: cortisol, $F(1, 54) = 1.06, p = .31$, testosterone, $F(1, 54) = .06, p = .81$; mood stabilizers: cortisol, $F(1, 54) = 2.10, p = .15$, testosterone, $F(1, 54) = 1.43, p = .24$), or post-stress recovery cortisol or testosterone (ADHD: cortisol, $F(1, 54) = .92, p = .34$, testosterone, $F(1, 54) = .04, p = .84$; antidepressants: cortisol, $F(1, 54) = 2.04, p = .16$, testosterone, $F(1, 54) = 1.13, p = .29$; mood stabilizers: cortisol, $F(1, 54) = .54, p = .46$, testosterone, $F(1, 54) = .14, p = .71$).

**Check for Potential Covariates**

Given the developmental stage of our participants, it is not surprising that age demonstrated a positive relationship with both cortisol and testosterone. Specifically, age was related to both the post-stressor, $r = .39, p = .01$, and post-stress recovery, $r = .46, p = .01$, testosterone samples, as well as each of the cortisol samples, baseline $r = .38, p = .01$, post-stressor $r = .29, p = .03$, and post-stress recovery $r = .27, p = .04$.

Sex differed across each of the testosterone samples, baseline $t(56) = 2.65, p = .01$, post-stress $t(56) = 3.50, p = .01$, and post-stress recovery $t(56) = 2.30, p = .03$, but only the post-
stressor sample of cortisol, \( t(56) = 2.75, p = .01 \). When sex was related to the hormonal samples, males had higher scores.

None of the times at which baseline (\( M \) time= 4:23 pm; cortisol: \( r = -.10, p = .65 \), testosterone: \( r = -.10, p = .47 \)), post-stressor (\( M \) time= 5:18 pm; cortisol: \( r = -.21, p = .12 \), testosterone: \( r = .13, p = .35 \)), or post-stress recovery (\( M \) time= 5:52 pm; cortisol: \( r = -.23, p = .10 \), testosterone: \( r = .13, p = .36 \)) samples were collected were related to any of the cortisol or testosterone scores. None of the testosterone time points differed significantly, \( F(2,55) = .28, p = .76 \) (baseline to post-stressor: mean difference = 14.78; post-stressor to post-stress recovery: mean difference = 2.29; baseline to post-stress recovery mean difference = 12.48). In contrast, cortisol differed significantly from post-stressor to post-stress recovery (\( M \) decrease = 125.91), but not baseline to post-stressor (-14.25) or post-stress recovery (140.15), \( F(2,55) = 13.05, p = .01 \). See Table 3.1 for descriptive statistics for cortisol and testosterone values by time point and sex. Correlations among psychopathy variables and hormone composites can be found in Table 3.2 and 3.3.
Table 3.2: Correlations among psychopathy, hormones, intelligence, and maturity

| Measure                              | 1   | 2      | 3      | 4      | 5    | 6      | 7      | 8      | 9      | 10    | 11     | 12     | 13     | 14     | 15     | 16     | 17     | 18     | 19     | 20     | 21     | 22     |
|--------------------------------------|-----|--------|--------|--------|------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|--------|
| 1. Psychopathy                      | -   | .83**  | .72**  | .84**  | .78**| .15    | .01    | .24    | -.14   | -.12   | .01    | -.21   | .14    | .34**  | .26    | .31*   | -.06   | .11    | -.28*  | -.09   | .14    | -.13   |
| 2. Facet 1                          | -   | .47**  | .66**  | .53**  | .30* | .18    | .36**  | -.04   | .27*   | -.02   | -.19   | .17    | .39**  | .33*   | .35**  | .17    | .27*   | -.07   | .12    | .24    | -.16   |
| 3. Facet 2                          | -   | .52**  | .39*   | .01    | -.02 | .01    | -.11   | .06    | -.02   | .03    | .07    | .19    | .17    | .17    | -.11   | -.03   | -.21   | -.03   | -.06   | -.03   | -.06   | -.03   |
| 4. Facet 3                          | -   | .54**  | .21    | -.17   | .28* | -.16   | .08    | -.08   | -.26   | .14    | .32*   | .28*   | .27*   | -.13   | .08    | -.31*  | -.22   | .19    | .01    |        |        |
| 5. Facet 4                          | -   | -.11   | -.17   | .03    | -.09 | -.11   | .14    | -.23   | -.03   | .22    | .14    | .22    | -.17   | .01    | -.32*  | -.17   | -.02   | -.13   |        |        |        |        |
| 6. Baseline Cortisol                | -   | .45**  | .76**  | .58**  | .42**| -.32*  | -.28*  | .70**  | .03    | .08    | -.02   | .18    | .21    | .12    | .02    | .38**  | .11    |        |        |        |        |        |
| 7. Baseline Testosterone            | -   | .42**  | .15    | .91**  | .31  | .53**  | .66**  | .11    | .07    | .13    | .12    | .09    | .11    | .21    | -.33*  |        |        |        |        |        |        |        |
| 8. Cortisol AUC<sub>e</sub>         | -   | -.12   | .47**  | -.12   | .50**| .45**  | .19    | .24    | .11    | .21    | .27*   | .06    | .08    | .41**  | .21    |        |        |        |        |        |        |        |
| 9. Cortisol AUC<sub>f</sub>         | -   | -.13   | .23    | -.04   | .79**| .01    | .02    | -.02   | .13    | .08    | .05    | .20    | -.11   | .24    |        |        |        |        |        |        |        |        |
| 10. Testosterone AUC<sub>e</sub>    | -   | .07    | .41**  | -.29*  | .23  | .19    | .26    | .14    | .11    | .11    | .32*   | -.37** |        |        |        |        |        |        |        |        |        |        |
| 11. Testosterone AUC<sub>f</sub>    | -   | -.12   | .45**  | .08    | .06  | .08    | -.09   | -.13   | -.03   | .01    | -.06   | -.12   |        |        |        |        |        |        |        |        |        |        |
| 12. Ratio Baseline T/C AUC<sub>e</sub> | -   | .23    | -.06   | -.13  | .04  | -.11   | .18    | -.05   | -.01   | .05    | -.21   | -.15   |        |        |        |        |        |        |        |        |        |        |
| 13. Ratio Baseline T/C AUC<sub>f</sub> | -   | .09    | .07    | .11   | -.01 | .02    | -.01   | .05    | .24    | -.11   |        |        |        |        |        |        |        |        |        |        |        |        |
| 14. IQ total                        | -   | .88**  | .91**  | .01   | .14  | -.12   | -.16   | -.03   | .07    |        |        |        |        |        |        |        |        |        |        |        |        |        |
| 15. Verbal IQ                       | -   | .61**  | .08    | .18   | .05  | -.06   | -.11   | .03    |        |        |        |        |        |        |        |        |        |        |        |        |        |        |        |
| 16. Non-Verbal IQ                   | -   | -.05   | .11    | -.12  | -.21 | .06    | .08    |        |        |        |        |        |        |        |        |        |        |        |        |        |        |        |        |
| 17. RSTI Maturity                   | -   | .86**  | .66**  | .78**  | .36**| .15    |        |        |        |        |        |        |        |        |        |        |        |        |        |        |        |        |
| 18. Autonomy                        | -   | .27*   | .50**  | .31*  | .06  |        |        |        |        |        |        |        |        |        |        |        |        |        |        |        |        |        |

*p < .05; **p < .01
Table 3.3: Correlations among psychopathy and hormone composites

| Measure | 1     | 2     | 3     | 4     | 5     | 6     | 7     | 8     | 9     | 10    | 11    | 12    | 13    | 14    | 15    | 16    | 17    | 18    | 19    | 20    | 21    | 22    | 23    | 24    | 25    | 26    | 27    | 28    | 29    |
|---------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|-------|
| P       | .83** | .72** | .84** | .78** | .15   | .08   | .05   | .01   | .21   | .04   | .24   | -.14  | .12   | .01   | -.21  | .14   | -.03  | .11   | -.11  | .45** | .01   | -.05  | -.02  | .19   | -.15  | -.09  | .14   | -.13  |     |
| F1      | -.47** | .66** | .53** | .3   | .19   | .12   | .18   | .32   | .13   | .36** | .04   | .27** | .02   | -.19  | .17   | .02   | .12   | -.13  | .08   | -.06  | .05   | -.03  | .08   | -.16  | .09   | .24   | .16   |     |
| F2      | -.52** | .39** | .01   | -.11  | -.12  | .02   | .11   | .08   | .01   | -.11  | .06   | -.02  | .03   | .07   | -.05  | .11   | .02   | .04   | .06   | -.04  | .08   | -.12  | .02   | .13   | -.06  | .03   |     |
| F3      | -.54** | .21   | .09   | .11   | -.01  | .17   | .03   | .28** | -.16  | .08   | -.08  | -.26  | .14   | .06   | .24   | .19   | .12   | -.04  | .05   | -.04  | .17   | -.14  | .05   | .19   | .01   |     |
| F4      | .21   | .09   | -.08  | -.01  | .01   | -.14  | .03   | -.09  | -.11  | .14   | -.23  | -.03  | -.04  | -.17  | -.08  | .12   | -.13  | .16   | .04   | .21   | .14   | .09   | -.02  | .13   |     |
| C1      | -.50** | .52** | .45** | .35** | .22   | .76** | -.58** | .42** | -.32** | -.28** | .70** | .47** | .70** | .51** | .47** | .51** | .06   | .48** | -.21  | -.15  | .38** | .38** | .11   |     |
| C2      | .89** | .34** | .54** | .17   | .92** | .18   | .37** | -.03  | .50** | .22   | .04   | .24   | -.14  | .02   | .49** | .16   | .38** | .11   | -.33** | -.25  | .39** | -.35** |     |
| C3      | .24   | .47** | .08   | .85** | .18   | .28** | -.02  | .53** | .15   | .24   | -.28** | .15   | .34** | .31** | .50** | .14   | .36** | .22   | .27** | .25   |     |
| T1      | .56** | .67** | .42** | -.15  | .91** | -.31  | .53** | .66** | .44** | .52** | .52** | .52** | -.11  | -.18  | -.21  | .66** | .15   | .64** | .21   | .33** |     |
| T2      | .53** | .56** | .01   | .79** | .36** | -.03  | .36** | -.46** | .07   | .19   | -.25  | .17   | -.12  | .11   | .26** | -.45** | -.19  | .39** | -.42** |     |
| T3      | .30** | .05   | .76** | .11   | .41** | .45** | .54** | .14   | .47** | .44** | .03   | .18   | -.06  | -.33** | .52** | .14   | .46** | -.30** |     |
| CAUCa   | -.12  | .47** | .12   | .50** | .45** | .18   | .44** | -.31** | .21   | .15   | -.08  | .11   | .01   | -.25  | .41** | .21   |     |
| Tauc    | -.13  | .23   | -.04  | .79** | .40** | .48** | .34** | -.39** | .83** | .02   | .79** | .18   | .02   | .18   | -.11  | .24   |     |
| TAUCc   | -.07  | .41** | .45** | .57** | .25   | .46** | .45** | -.07   | .15   | -.15  | .38** | .01   | .43** | .32** | .37** |     |
| TAUc    | -.12  | .29** | -.34  | -.84** | .09   | -.03  | .28** | .01   | .28** | .83** | -.23  | .50** | .06   | .12   |     |
| TAUCe   | .23   | .57** | .02   | .80** | -.70** | -.21  | -.11  | -.26  | .65** | .47** | .31** | .21   | -.15  |     |
| T/AUCc  | .04   | .60** | .03   | .01   | .54** | .13   | .57** | .45** | .11   | .44** | .24   | .11   |     |
| CTAUCc  | .39** | .87** | .87** | .03   | .21   | .03** | .12   | .45** | -.12  | .03   | .47** |     |
| C/TAUCc | -.30** | .23   | .48** | -.01  | .47** | -.66** | .09   | .53** | .25   | .10   |     |
| T/CT    | -.93** | .37** | .25   | .43** | .35** | .21   | -.13  | .41** |     |
| C/CT    | -.45** | .25   | .32** | .38** | .24   | .25   | .06   | .45** |     |
| C1-C2   | -.26  | .87** | .28** | .08   | .22   | -.03  | .44** |     |
| C2-C3   | -.26  | .11   | .09   | .05   | .02   | .18   |     |
| C3-C4   | -.33  | .12   | .25   | .04   | .34** |     |
| T1-T2   | -.57** | .58** | .11   | .01   |     |
| T2-T3   | -.34** | .06   | .06   |     |
| T3-T4   | -.19   | .12   |     |
| Age     | .07   |     |

Psychopathy: Total; F1, Interpersonal Facet; F2, Affective Facet; F3, Lifestyle Facet; F4, Antisocial Facet; T1, Baseline Testosterone; T2, Post-Stress Testosterone; T3, Post-Stress Recovery Testosterone; C1, Baseline Cortisol; C2, Post-stress Cortisol; C3, Post-Stress Recovery Cortisol; T AUCG, Testosterone Reactivity with Respect to Ground; T AUCI, Testosterone Reactivity with Respect to Increase; C AUCG, Cortisol Reactivity with Respect to Ground; C AUCI, Cortisol Reactivity with Respect to Increase; T/C AUCG, Ratio of Baseline Testosterone to Cortisol Reactivity with Respect to Ground; T/C AUCI, Ratio of Baseline Testosterone to Cortisol Reactivity with Respect to Increase; C/T AUCG, Ratio of Baseline Cortisol to Testosterone Reactivity with Respect to Ground; C/T AUCI, Ratio of Baseline Cortisol to Testosterone Reactivity with Respect to Increase; T; C, Ratio of Baseline Testosterone to Baseline Cortisol; CT, Ratio of Baseline Cortisol to Baseline Testosterone; T1-T2, Change in Testosterone from Baseline to Post-Stress; T2-T3, Change in Testosterone from Baseline to Post-Stress Recovery; T3-T4, Change in Testosterone from Post-Stress to Post-Stress Recovery; T1-T3, Change in Testosterone from Baseline to Post-Stress Recovery; C1-C2, Change in Cortisol from Baseline to Post-Stress; C2-C3, Change in Cortisol from Baseline to Post-Stress Recovery; C1-C3, Change in Cortisol from Baseline to Post-Stress Recovery

*p<.05; **p<.01
Correlational Analyses

Correlational analyses were conducted to examine the relations between the primary study variables. Specifically, the study examined the relations between psychopathy, intelligence, maturity, and hormones. Baseline cortisol and baseline testosterone were positively correlated, $r = .45, p = .01$, as is typical of most samples (e.g., Loney et al., 2006). The correlation between psychopathy and intelligence demonstrated a significant positive relationship; $r = .34, p = .01$. Non-verbal IQ was also significantly correlated with psychopathy, $r = .31, p = .02$, and verbal IQ approached significance, $r = .26, p = .05$. Maturity was not significantly related to psychopathy, $r = -.06, p = .65$, and nor were the emotion regulation, $r = -.09, p = .53$, or autonomy RSTI subscales, $r = .11, p = .42$. This being said, the cognitive capacities RSTI subscale demonstrated a significant negative correlation with psychopathy, $r = -.28, p = .03$. The correlation coefficients for psychopathy and baseline cortisol and testosterone were, $r = .15, p = .28$ and $r = .00, p = .99$, respectively. Similarly, the correlation coefficients for psychopathy and cortisol reactivity ($AUC_G$), $r = .24, p = .07$, testosterone reactivity ($AUC_G$), $r = .12, p = .39$, and the ratio of baseline testosterone to cortisol reactivity ($AUC_G$), $r = -.21, p = .12$, were not significant. Facet level correlations are also located in Table 3.2 and 3.3. Notable in this regard is that the interpersonal facet demonstrated significant positive correlations with baseline cortisol, $r = .30, p = .02$, cortisol $AUC_G$, $r = .36, p = .01$, and testosterone $AUC_G$, $r = .27, p = .04$. Further, the interpersonal facet held a positive relationship with IQ total, $r = .39, p = .01$, verbal IQ, $r = .33, p = .01$, non-verbal IQ, $r = .35, p = .01$, and the autonomy subscale of the RSTI, $r = .27, p = .04$. In addition, the lifestyle facet held significant positive relationships with each of IQ total, $r = .32, p = .01$, verbal IQ, $r = .28, p = .04$, and non-verbal IQ, $r = .27, p = .04$ and a significant negative relationship with the cognitive capacities subscale of the RSTI, $r =$
.31, p = .02. Like the lifestyle facet, the antisocial facet also demonstrated a significant negative relationship with cognitive capacities, r = -.32, p = .02.

Correlational analyses were also run for each of the PANAS positive, PANAS negative, and STICSA measures with their corresponding cortisol and testosterone sample (e.g., a correlational analysis with baseline STICSA and baseline cortisol) to examine whether participants subjective experiences matched their hormonal levels. The self-report measures did not correspond well with hormonal levels. The PANAS positive affect scale was not correlated with baseline cortisol, r = -.04, p = .77 or testosterone, r = .14, p = .32, post-stressor cortisol, r = -.05, p = .70 or testosterone, r = -.18, p = .18, nor post-stress recovery, cortisol, r = -.05, p = .74 or testosterone, r = -.06, p = .68. Similarly, the PANAS negative affect scale was not correlated with baseline cortisol, r = -.01, p = .96 or testosterone, r = -.18, p = .18, post-stressor cortisol, r = .06, p = .69, nor post-stress recovery cortisol, r = .18, p = .20, or testosterone, r = -.12, p = .40. The PANAS negative scale was however significantly associated with post-stressor testosterone, r = -.32, p = .02. The STICSA measures were not correlated with any of the cortisol (baseline: r = .01, p = .98, post-stressor: r = -.02, p = .88, and post-stress recovery: r = -.10, p = .47) or testosterone samples either (baseline: r = -.09, p = .52, post-stressor: r = -.21, p = .14, and post-stress recovery: r = .04, p = .78).

**Regression Analyses**

Separate hierarchical regressions were run predicting total psychopathy and each of the individual facets (interpersonal, affective, lifestyle, and antisocial) with age and sex as covariates, and hormone variables (baseline cortisol, cortisol reactivity with respect to ground and increase, baseline testosterone, testosterone reactivity with respect to ground and increase, and the ratios of baseline testosterone/cortisol reactivity with respect to ground and increase), IQ
total (K-BIT 2 total scaled score), and the interaction term (hormone variable x IQ total) as predictors.

The hierarchical regression with the interaction term baseline cortisol x IQ total predicting psychopathy total score revealed an interaction that approached significance, $\beta (51) = 2.38, p = .09$. The same was found for the interpersonal facet, $\beta (51) = 2.38, p = .06$. The interaction term was not significant for either the affective facet, $\beta (51) = -.44, p = .78$, nor the lifestyle facet, $\beta (51) = 1.77, p = .22$. However, a significant interaction (baseline cortisol x IQ total) was revealed to predict the antisocial facet, $\beta (51) = 3.77, p = .01$, contributing 11% to the variability of the facet (see Table 3.4). Visual inspection of the interaction revealed that higher IQ influenced a positive relationship between baseline cortisol and levels of the antisocial facet, whereas lower IQ influenced an inverse relationship between baseline levels of cortisol and the antisocial facet (see Figure 3.1). There were no significant main effects for baseline cortisol on any of the psychopathy variables.

**Table 3.4: Standardized beta coefficients and adjusted $r^2$ for interactions between hormone composites and IQ total scaled score on the antisocial facet**

<table>
<thead>
<tr>
<th>Interactions</th>
<th>Standardized Coefficients</th>
<th>$\beta$</th>
<th>Adj $R^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline Cortisol x IQ</td>
<td></td>
<td>3.77**</td>
<td>.11</td>
</tr>
<tr>
<td>Cortisol AUC$_i$ x IQ</td>
<td></td>
<td>-13.01*</td>
<td>.08</td>
</tr>
<tr>
<td>(Baseline Testosterone/Cortisol AUC$_i$) x IQ</td>
<td></td>
<td>1.89*</td>
<td>.07</td>
</tr>
</tbody>
</table>

*p < .05; ** p < .01
Figure 3.1: Interaction between baseline cortisol and IQ total on the antisocial facet

The hierarchical regression with cortisol reactivity using $AUC_G$, IQ total, and the interaction term (cortisol $AUC_G \times$ IQ total) as predictors of psychopathy total score did not reveal a significant interaction, $\beta (51) = .84, p = .68$. The interaction term (cortisol $AUC_G \times$ IQ total) did not reveal a significant interaction for any of the psychopathy facets either. The interaction between cortisol reactivity using $AUC_I$ and IQ total was not significantly predictive of psychopathy total score, $\beta (51) = -8.31, p = .14$, but significantly predicted the antisocial facet, $\beta (51) = -13.01, p = .03$, contributing 8% to the variability in this facet (see Table 3.4). In contrast to the interaction between baseline cortisol and IQ, higher IQ was associated with an inverse relationship between cortisol reactivity and the antisocial facet, but lower IQ, was associated
with a positive relationship between cortisol reactivity and the antisocial facet (see Figure 3.2). There were no significant main effects for cortisol reactivity on any of the psychopathy variables.

Figure 3.2: Interaction between cortisol reactivity (AUC) and IQ total on the antisocial facet

No significant interaction was revealed for the interaction term of baseline testosterone by IQ total predicting psychopathy, $\beta (52) = .72, p = .56$. Further, no significant interactions were found for any of the psychopathy facets. When the interaction terms for testosterone reactivity (both with respect to ground and increase) and IQ total were regressed on psychopathy, neither were significant, $\beta (52) = 1.02, p = .31$ and $\beta (52) = 1.88, p = .89$, respectively. Moreover,
none of the psychopathy facets were predicted by either interaction term, nor were there any significant main effects for any testosterone variables on any of the psychopathy variables.

There were no significant main effects for either ratio of baseline testosterone/cortisol reactivity on any of the psychopathy variables. Further, there was no significant interaction effect for the IQ total with the ratio of baseline testosterone/cortisol reactivity with respect to ground on psychopathy total score, \( \beta (51) = .14, p = .91 \) or any of the facets. Nor was there a significant interaction effect for the IQ total with the ratio of baseline testosterone/cortisol reactivity with respect to increase on psychopathy total score, \( \beta (50) = 1.04, p = .20 \). This interaction did however significantly predict the antisocial facet, \( \beta (50) = 1.89, p = .03 \), contributing 7% to the variability in scores on the facet. Visual inspection of the interaction revealed that higher IQ was related to a positive relationship between the ratio and the antisocial facet. In contrast, lower IQ was related to a negative relationship between the ratio and the antisocial facet (see Figure 3.3).

Despite this significant finding, the direction of the ratio is unclear. For example, antisocial behavior may be related to a combination of greater testosterone and lower cortisol reactivity or lower testosterone and greater cortisol reactivity. When correlations between the ratio and baseline testosterone and cortisol AUC\(_I\) were investigated, baseline testosterone and the ratio were positively correlated, \( r = .66, p = .01 \), and cortisol AUC\(_I\) and the ratio were negatively correlated, \( r = -.80, p = .01 \) (See Table 3.3). The directions of the correlation coefficients suggest that as the ratio increases so does testosterone, but cortisol AUC\(_I\) decreases. Thus, it is likely that this ratio represents higher baseline testosterone and lower cortisol AUC\(_I\).
Separate hierarchical regressions were also run predicting total psychopathy and each of the individual facets with age and sex as covariates, and hormone variables, maturity (RSTI sophistication-maturity scale score), and the interaction term (hormone variable x maturity) as predictors. There were no significant main effects for any of the hormone composites on any of the psychopathy variables. Moreover, there were no significant interaction effects that predicted psychopathy using maturity and either baseline cortisol, $\beta (51) = .05, p = .98$, cortisol reactivity using $\text{AUC}_G, \beta (51) = -2.06, p = .25$, cortisol reactivity using $\text{AUC}_I, \beta (50) = -1.90 p = .74$, baseline testosterone, $\beta (52) = -.32, p = .78$, testosterone reactivity using $\text{AUC}_G, \beta (51) = -.96 p = .
.45, testosterone reactivity using AUC_I, \( \beta (51) = 2.36 p = .90 \), or the ratio of baseline testosterone/cortisol reactivity using AUC_G, \( \beta (51) = -.14 p = .86 \), or AUC_I, \( \beta (50) = -.14 p = .86 \). In addition, none of the facets were significantly related to any of the interaction terms, but the interaction between cortisol reactivity using AUC_G and maturity approached significance for the antisocial facet, \( \beta (51) = -3.33 p = .06 \).

**Distress Across Time Points: Repeated Measures**

To investigate whether self-report of distress differed across time points, a repeated measures analysis was conducted with the PANAS positive and negative affect scales and the STICSA total score. The PANAS positive affect scale significantly decreased from baseline \( (M = 27.97; SD = 8.81) \) to post-stressor \( (M = 25.53; SD = 9.93) \) and was still significantly lower than baseline at post-stress recovery \( (M = 26.12; SD = 8.79) \), \( F (2, 55) = 7.45, p = .01 \). Interestingly, the PANAS negative affect scale, significantly decreased from baseline \( (M = 15.62; SD = 5.82) \) to post-stress recovery \( (M = 14.79; SD = 6.40) \), but not baseline to post-stressor \( (M = 15.24; SD = 6.70) \), \( F (2, 49) = 3.29, p = .05 \). The STICSA total, significantly decreased from baseline \( (M = 26.80, SD = 8.37) \) to post-stressor \( (STICSA: M = 26.57, SD = 10.44) \), post-stressor to post-stress recovery \( (M = 24.74, SD = 8.49) \), as well as baseline to post-stress recovery, \( F (2, 45) = 8.48, p = .01 \).

**Categorical Data Analyses**

Given the potential for age and sex differences regarding the study hypotheses, the data were also analyzed categorically using GLM. Because it is believed that adolescence may differ from childhood in meaningful ways, this was thought to be an important demarcation for the analyses. Gender was analyzed dichotomously to further explore these potentially important group differences. When significant interactions were found involving age, age was
dichotomized into two groups to separate young from older adolescents (age 12-15, \(n = 19\) and age 16-18, \(n = 39\)) and the significant facet was dichotomized using a median split to present the data graphically and visually inspect the relationship.

**Mixed, within-subjects repeated measures, age, sex, and psychopathy on cortisol**

Within the mixed model, with psychopathy total on cortisol across time points, psychopathy was not predictive of cortisol, \(F(1, 158) = .22, p = .64\), but there was a main effect for age, \(F(1, 158) = 4.35, p = .04\), partial eta squared = .02, and an interaction effect for sex and time, \(F(1, 158) = 3.79, p = .02\), partial eta squared = .04. Regarding the sex by time interaction, inspection of mean scores indicated that males had higher levels of cortisol at each time point. These differences approached significance, for post-stressor, males \(M = 719.21, SD = 699.89\); females \(M = 334.52, SD = 189.83\), \(F(1, 158) = 3.60, p = .06\), and post-stress recovery, males \(M = 550.39, SD = 589.86\); females \(M = 357.16, SD = 416.88\), \(F(1, 158) = 3.36, p = .07\).

Similarly, the interpersonal facet was not significantly related to cortisol over time, \(F(1, 158) = .03, p = .85\), but with this facet in the model, a main effect of age, \(F(1, 158) = 12.15, p = .01\), partial eta squared = .06, emerged, as well as interactions between time by sex, \(F(1, 158) = 4.06, p = .02\), eta squared = .04, and sex by age, \(F(1, 158) = 4.34, p = .04\), partial eta squared = .03. With the interpersonal facet in the model, sexes differed significantly within the post-stressor, \(F(1, 158) = 4.95, p = .03\), and post-stress recovery cortisol samples, \(F(1, 158) = 4.62, p = .03\).

With the affective facet in the model, there were significant interaction effects for time by sex, \(F(1, 158) = 3.68, p = .03\), partial eta squared = .02, and age by affective facet, \(F(1, 158) = 4.88, p = .03\), partial eta squared = .02. The post hoc analyses did not reveal any significant differences among sex from any of the time points. Figure 3.4 displays the interaction between
the affective facet and age. More specifically, younger participants had lower levels of cortisol than older participants at baseline, but younger participants with high scores on the affective facet dropped in cortisol at the post-stressor time point, whereas most other groups increased in cortisol or stayed the same.

**Figure 3.4: Change in cortisol over time by levels of the affective facet and age**

![Graph showing cortisol levels across time by levels of the affective facet and age]

Similar to the model for the interpersonal facet, the lifestyle facet was not significantly related to cortisol levels across time points, $F(1, 158) = 1.28, p = .26$, but there was a significant interaction of time and sex, $F(1, 158) = 3.61, p = .03$, partial eta squared = .04. Within this model, sexes differed significantly within the post-stressor, $F(1, 158) = 4.54, p = .03$, and post-stress recovery cortisol samples, $F(1, 158) = 4.24, p = .04$.

The final model followed much the same pattern, the antisocial facet was not significantly related to cortisol values, $F(1, 158) = 3.31, p = .07$, there was a significant main effect for age, $F(1, 158) = 15.16, p = .01$, partial eta squared = .07, and there was a significant interaction for time by sex, $F(1, 158) = 3.51, p = .03$, partial eta squared = .03. Moreover a main
effect for sex, $F(1, 158) = 3.62, p = .06$ and an interaction for age by the antisocial facet approached significance, $F(1, 158) = 3.63, p = .06$.

**Mixed, within-subjects repeated measures, age, sex, and psychopathy on testosterone**

The mixed model with psychopathy total on testosterone across time points demonstrated a significant interaction effect for age and sex, $F(1, 158) = 4.20, p = .04$, eta squared = .02. There was no significant main effect for psychopathy total, $F(1, 158) = 2.78, p = .10$. The post hoc contrasts revealed significant differences between males and females across each time point. For each time point males were significantly higher, than females, (baseline: males $M = 80.15$, $SD = 78.48$, females $M = 36.16$, $SD = 16.88$; post-stressor: males $M = 61.68$, $SD = 33.94$, females $M = 34.15$, $SD = 19.53$; post-stress recovery: males $M = 63.86$, $SD = 45.04$, females $M = 38.91$, $SD = 19.54$).

With the interpersonal facet in the model, there was a significant interaction effect for age and the interpersonal facet, $F(1, 158) = 11.44, p = .01$, eta squared = .05. Sexes differed significantly across time points, baseline, $F(1, 158) = 4.78, p = .03$, post-stress, $F(1, 158) = 4.76, p = .03$, and post-stress recovery, $F(1, 158) = 4.44, p = .04$. Figure 3.5 displays an interesting trend with age in which that younger participants that were high on interpersonal traits had similar testosterone levels to older participants.
The affective facet was also significantly related to testosterone values across time points. There were significant interaction effects for age and sex, $F(1, 158) = 4.61, p = .03$, eta squared $= .02$, and age and the affective facet, $F(1, 158) = 10.73, p = .01$, eta squared $= .04$. Sex differences were significant across each time point, baseline: $F(1, 158) = 5.36, p = .02$; post-stressor, $F(1, 158) = 5.37, p = .02$; and post-stress recovery, $F(1, 158) = 5.02, p = .03$. Similar Figure 3.6 demonstrates that both younger and older participants that were high on affective traits changed only slightly in testosterone levels across time points. In contrast young participants with low levels of affective traits dropped from post-stressor to post-stress recovery and older participants with low levels of affective traits increased from post-stressor to post-stress recovery.
Within the model including the lifestyle facet, the lifestyle facet was not significantly related to testosterone over time, $F(1, 158) = .45, p = .50$. The only main effects that emerged were for age, $F(1, 158) = 8.42, p = .01$, eta squared = .04 and sex, $F(1, 158) = 4.67, p = .03$, eta squared = .02. Sex differed significantly across each time point, baseline, $F(1, 158) = 4.76, p = .03$; post-stressor, $F(1, 158) = 4.79, p = .03$; and post-stress recovery, $F(1, 158) = 4.38, p = .04$.

The model including the antisocial facet behaved very much like the lifestyle facet, the antisocial facet was not significantly related to testosterone, $F(1, 158) = .13, p = .71$, but there were main effects for age, $F(1, 158) = 10.71, p = .01$, eta squared = .04 and sex, $F(1, 158) = 4.57, p = .03$, eta squared = .02, on testosterone over time. The sexes differed significantly on testosterone values across time point, baseline, $F(1, 158) = 4.69, p = .03$; post-stressor, $F(1, 158) = 4.63, p = .03$; and post-stress recovery, $F(1, 158) = 4.31, p = .04$. 
General linear models of age, sex, and composite hormones on psychopathy variables

Each of the testosterone AUC$_G$, change in cortisol from baseline to post-stressor, change in cortisol from baseline to post-stress recovery, and the ratio of baseline testosterone/baseline cortisol and the ratio of baseline cortisol/baseline testosterone, were significantly correlated with sex (see Table 3.3), thus due to multicollinearity issues these analyses were run as regressions and sex was not included as a covariate. Testosterone AUC$_G$ and cortisol AUC$_G$ were significantly related to age, thus standardized residuals for the regression of age on these hormone composites were included in the model instead of age. Psychopathy total, the affective facet, and the antisocial facet were not significantly predicted by any hormone composites.

The interpersonal facet was significantly predicted by cortisol AUC$_I$, $F(1, 49) = 4.29, p = .04$, partial eta squared = .07, there was also a main effect for age, $F(1, 49) = 3.74, p = .06$, and an interaction effect for age and cortisol AUC$_I$, $F(1, 49) = 3.77, p = .06$, that approached significance. This facet was also predicted by an interaction between sex with change in cortisol from post-stressor to post-stress recovery, $F(1, 51) = 4.20, p = .05$, partial eta squared = .07. Figure 3.7 displays that females demonstrated a negative relationship between the interpersonal facet and change in cortisol, in comparison to males that appeared to have a positive relationship. Further, the interpersonal facet was significantly predicted by testosterone AUC$_G$, $F(1, 54) = 4.48, p = .04$, partial eta squared = .08.
The affective facet was significantly predicted by cortisol AUC$_G$, $F (1, 50) = 4.22, p = .08$, partial eta squared = .08, sex, $F (1, 50) = 5.01, p = .03$, partial eta squared = .09, and an interaction between cortisol AUC$_G$ and sex, $F (1, 50) = 5.12, p = .03$, partial eta squared = .09. Figure 3.8 demonstrates that females had a negative relationship between cortisol reactivity and the scores on the interpersonal facet, whereas males did not demonstrate a distinct relationship between these variables.
Figure 3.8: The relationship between the affective facet and cortisol reactivity ($AUC_G$) by sex

The lifestyle facet was significantly predicted by the same interaction as the interpersonal facet: between sex with change in cortisol from post-stressor to post-stress recovery, $F (1, 51) = 5.59, p = .02$, partial eta squared $= .10$. Further, the same finding emerged when the lifestyle facet was plotted with change in cortisol from post-stressor to post-stress recovery by sex, in that females demonstrated a negative relationship between the lifestyle facet and change in cortisol, in comparison to males that appeared to have little to no relationship. (see Figure 3.9).
General linear models of age, sex, psychopathy scores, on composite hormone variables

When the models were reversed, with hormone composite variables as outcomes and psychopathy variables as predictors, many more significant relationships arose. Using a general linear model to explore the effects of psychopathy total, age, and sex, on change in cortisol from post-stressor to post-stress recovery, there was a significant interaction effect of sex and psychopathy total score on changes in cortisol from post-stressor to post-stress recovery, $F(1, 51) = 5.95, p = .02$, partial eta squared = .10. The same finding arose when the interpersonal facet replaced psychopathy total, $F(1, 51) = 7.12, p = .01$, partial eta squared = .11. These two
relations were plotted in Figures 3.10 and 3.11 and displayed much the same interaction, in which females demonstrated a negative relationship between the hormone composite and the psychopathy variable.

**Figure 3.10:** *The relationship between the PCL-YV total and change in cortisol from post-stressor to post-stress recovery by sex*
An interaction between the interpersonal facet and sex, $F(1, 50) = 4.12, p = .05$, partial eta squared = .03 also significantly predicted the change in cortisol from baseline to post-stress recovery. To investigate how sexes differed with scores on the interpersonal facet, the relationship was plotted by sex. Females demonstrated a negative relationship between the change in cortisol from baseline to post-stress recovery, whereas males did not appear to demonstrate any relationship (see Figure 3.12).
When psychopathy facets replaced the psychopathy total score within the AUC<sub>G</sub> testosterone model, the model including the interpersonal facet demonstrated a significant interaction with the interpersonal facet and age, \( F(1, 50) = 4.71, p = .03 \), partial eta squared = .06. Visual inspection of Figure 3.13 suggests that younger participants displayed a positive relationship between testosterone AUC<sub>G</sub> and the interpersonal facet, whereas older youth did not display a significant relationship between these two variables. The affective facet approached significance, \( F(1, 50) = 3.35, p = .07 \), and so did an interaction between the affective facet and age, \( F(1, 50) = 3.33, p = .07 \). Within this model, the only significant effect was a main effect for
age, $F(1, 50) = 6.74, p = .01$, partial eta squared = .02. Neither the lifestyle, $F(1, 50) = .60, p = .44$, nor the antisocial facet, $F(1, 50) = .02, p = .89$, added anything significant to the model.

**Figure 3.13: The relationship between the interpersonal facet and testosterone reactivity (AUC$_C$) by age**

There was a significant interaction effect for the affective facet and age, $F(1, 50) = 7.73, p = .01$, partial eta squared = .11, on the baseline testosterone/baseline cortisol ratio. The same was true for the ratio of baseline cortisol/baseline testosterone, which had an interaction effect between the affective facet and age, $F(1, 50) = 5.03, p = .03$, partial eta squared = .07. There was also a significant interaction effect for the affective facet and age, $F(1, 50) = 9.58, p = .01$,.
partial eta squared = .15, on the baseline testosterone/cortisol reactivity (AUCG) ratio. Similarly, when the ratio was reversed baseline cortisol with testosterone reactivity using AUCG, there was an interaction effect for the affective facet and age, $F(1, 49) = 7.79, p = .01$. Figures 3.14, 3.15, 3.16, and 3.17 display minute differences between older and younger participants, such that younger participants demonstrated a slightly more positive relationship between the affective facet and the ratios of baseline testosterone/baseline cortisol and baseline testosterone/cortisol reactivity (AUCG), but a slightly more negative relationship for the ratios of baseline cortisol/baseline testosterone and baseline cortisol/baseline testosterone reactivity (AUCG). None of the other psychopathy scores were significant: total psychopathy $F(1, 49) = 1.09, p = .30$, interpersonal $F(1, 49) = 1.02, p = .32$, lifestyle $F(1, 49) = .21, p = .65$, and antisocial facets, $F(1, 49) = .46, p = .50$. 
Figure 3.14: The relationship between the affective facet and the ratio of baseline testosterone/baseline cortisol by age
Figure 3.15: The relationship between the affective facet and the ratio of baseline cortisol/baseline testosterone by age
Figure 3.16: The relationship between the affective facet and the ratio of baseline testosterone/baseline cortisol reactivity (AUCG) by age
The only effect that emerged for the lifestyle facet was a significant interaction for sex and lifestyle, $F(1, 51) = 9.51, p = .01$, partial eta squared = .14 predicting change in cortisol from post-stressor to post-stress recovery. Figure 3.18 demonstrates that females held a negative relationship with the hormone composite and the lifestyle facet, whereas males had a slightly positive relationship. There was no impact of any interactions containing the antisocial facet.
Figure 3.18: The relationship between the lifestyle facet and change in cortisol from post-stressor to post-stress recovery by sex
CHAPTER 4
DISCUSSION

Psychology and biology have a complex relationship, in which their functions are often entangled and interacting. Thus, it is important to study them together to understand how psychological phenomena may develop and elucidate the underlying processes involved. Neuroendocrine functions are an especially critical area of investigation due to the wide-ranging effects of hormones throughout the body and brain. One of the primary aims of the present study was to understand how cortisol and testosterone interact to affect psychopathic traits among detained adolescents. This study was also developed to expand on the field’s understanding of how intelligence relates to psychopathy among youth. Further, this was one of the first (e.g., Leistco & Salekin, 2003) investigations of the relationship between psychosocial maturity and psychopathy among detained youth. Finally, this study examined the relations among psychopathy, intelligence, and maturity and how these variables might impact the interaction between psychopathy and hormonal production. The study had 5 primary hypotheses predicting that a) the ratio of baseline testosterone to cortisol reactivity would predict psychopathic traits, b) this relationship would be stronger for the interpersonal and affective facets of psychopathy, than the other two facets, in addition, both c) intelligence and d) maturity would demonstrate a positive relationship with the interpersonal facet and a negative relationship with the lifestyle facet, and finally e) intelligence and maturity would moderate the strength of the relationship between psychopathy and the ratio of baseline testosterone to cortisol reactivity.
Among our adolescent offender sample, the ratio of baseline testosterone to cortisol reactivity did not predict psychopathic traits when psychopathy was measured as a single construct (i.e. PCL-YV total score). Within the extant literature on hormonal reactivity and psychopathic traits, only one study (Glenn et al., 2011) has investigated the ratio of baseline testosterone to cortisol reactivity and found that this ratio was significantly related to psychopathy total score and factor two scores. We were unable to replicate this finding relating psychopathy to the interplay of baseline testosterone and cortisol reactivity within our youth sample. Also contrary to prediction, the ratio was associated with the antisocial facet, but only when this relationship was moderated by intelligence.

Some interesting findings emerged for both intelligence and maturity. Partially in line with the prediction, intelligence was related to both the interpersonal facet and the lifestyle facet, but unexpectedly both of these relationships were in the positive direction. A positive relationship between the lifestyle facet and intelligence suggests that those with higher scores on this facet also had higher IQ scores. Though surprising, this finding is similar to that found by Salekin and colleagues (2004). It may be that this relationship reflects the use of intelligence to artfully rationalize oneself through irresponsible and impulsive behavior, by creating excuses for said behavior (Gino, & Ariely, 2012; Tsang, 2002). Moreover, one of the items contained within the lifestyle facet is parasitic orientation, a tendency toward exploiting others, thus it may require some intellectual prowess to finesse others into engaging in exploitive relationships.

It should be noted that the mean total IQ score for this sample was significantly lower than the general population at 86.19 in comparison to the average scaled score of 100. This score was likely influenced by the Low Average verbal IQ score (scaled score = 83.12), given that the non-verbal score approached that of the general population (scaled score = 93.10). Although odd
in comparison to the general population, this is not a new finding. Within a community adolescent sample, those who had previous contact with the England Youth Offending Services had much lower verbal IQ ($M = 81.12$) than non-verbal IQ ($M = 90.80$, Allen et al., 2013). Another sample obtained low means (81.94) on the Peabody Picture Vocabulary Test-3rd Edition, which is meant to measure verbal ability, suggesting that detained youth may have more issues with verbal comprehension than the population in which these tests were averaged.

Unlike intelligence, sophistication maturity as measured by the RSTI-SR, was not related to psychopathy, but individual aspects of maturity such autonomy and cognitive capacities were related to specific psychopathy variables. Autonomy, a measure of ones ability to act independently and hold strong values, was positively correlated with the interpersonal facet. Further, the cognitive capacities subscale predicted psychopathy total score and the interpersonal facet. Within the RSTI-SR, cognitive capacities are conceptualized as one’s ability to make thoughtful and informed decisions, while controlling salient impulses and delaying gratification when the situation requires. The subscale contains questions such as, “I focus on what I will win rather than what I will lose,” and “I weigh the costs and benefits before acting.” This finding corresponds with the finding that intelligence is predictive of psychopathy, given that cognitive capacities not only require a degree of intelligence, but wisdom to use intelligence to inform decisions.

Interestingly, cognitive capacities in this study held a negative relationship with the interpersonal facet, suggesting that those with interpersonal traits, such as impression management and manipulation for personal gain were, less likely to have mature cognitive capacities. Thus, within this study having more general intellectual abilities may have increased one’s likelihood of engaging in behaviors such as pathological lying and manipulating others
(facet 1), as well as engaging in risky behaviors and exploiting others (facet 3), but having greater cognitive capacities may have buffered these behaviors by assisting youth in using their intelligent in less antisocial ways.

When psychopathy was measured as a single construct, intelligence did not moderate the relationship between psychopathy and any hormone variables. However, three significant interactions emerged predicting the antisocial facet. Intelligence moderated the relationship between the antisocial facet and each of baseline cortisol, cortisol reactivity measured by AUC_t, and the ratio of baseline testosterone/cortisol reactivity (using AUC_t). Intelligence moderated each of these relationships in very different ways. Among those with higher intelligence, as levels of baseline cortisol increased so did levels of the antisocial facet, but increases in cortisol reactivity were related to reduced scores on the antisocial facet. Moreover, increases in the ratio (suggesting greater difference between baseline testosterone and cortisol reactivity) coincided with increases in the antisocial facet among those with higher intelligence. The opposite was true for those with lower levels of intelligence, as baseline cortisol levels increased, scores on the antisocial facet decreased, but increases in cortisol reactivity were related to increases in scores on the antisocial facet. Further, like baseline cortisol, increases in the ratio related to decreases in scores on the antisocial facet for those with lower intelligence.

Past research has also found a relationship between hormones and antisocial behavior (e.g., Poustka et al., 2010) and hormones and intelligence (e.g., Blair et al., 2011; Franz et al., 2011; Kennedy et al., 2014), but this study was one of the first (e.g., Muñoz et al., 2008) to reconcile each of these three variables together. In order to understand these complex findings, it is important to understand what the antisocial facet consists of. The antisocial facet is composed of five items including, poor anger control, early behavior problems, serious criminal behavior,
serious violations of conditional release, and criminal versatility. Thus, it is can generally be reduced to a measure of aggression and criminal involvement and perhaps a proxy for Conduct Disorder. Given that there is no way to determine causation from the current study design, we can speculate about these cross-sectional findings in multiple ways. For example, those with high intelligence that are more involved in aggression and crime, may have higher levels of stress (suggested by higher cortisol levels) because they are more aware of the consequences of their actions. In contrast, a combination of higher intelligence and higher circulating levels of cortisol may allow some individuals to plan out their crimes and be more adept at covering them up and thus engage in more crime because it is reinforcing. This conceptualization is similar to what Cleckley (1988) deemed to be “successful psychopathy”. Successful psychopaths are thought to be those with psychopathic traits and either less criminal involvement or less detection by the law. Cognitive comparisons of individuals with successful psychopathic traits, and those with unsuccessful psychopathic traits, have found that those with successful traits perform better on cognitive tests such as the Wisconsin Card Sorting Task (see Ishikawa et al., 2001) than both unsuccessful psychopaths and non-psychopathic comparison groups. Furthermore, in some studies, individuals with successful psychopathic traits have higher stress reactivity, measured through skin conductance. This higher reactivity may be implicated in these individuals’ ability to process risky situations and make better decisions either leading to less criminal behavior or a better ability to mask their crimes (Gao & Raine, 2010). Another way to conceptualize this finding is that higher intelligence may allow those with psychopathic traits the ability to rationalize their aggressive and criminal behavior (Gino, & Ariely, 2012; Tsang, 2002) to the extent that they do not attend to their physiology. Whereas those with lower levels of intelligence
would be less able to cognitively over-ride signals from their stress system and thus would engage in less aggression and crime.

Despite the finding that those with higher intelligence engaged in more crime and aggression when they had higher levels of circulating cortisol (or vice versa; those with higher circulating cortisol engage in more antisocial acts), those who engaged in less crime displayed higher cortisol reactivity in relation to stress (or vice versa). This finding appears to be more intuitive than the last; intelligence may allow these individuals to foresee the consequences of their actions by attending to their body’s natural reaction to engaging in antisocial acts. Thus, they would engage in less of these behaviors. This being said, the finding that those with higher intelligence demonstrated an inverse relationship between cortisol reactivity and antisocial behavior may also offer an explanation for why those with higher intelligence and higher circulating cortisol also engaged in more antisocial acts. Those with higher circulating cortisol may have met their threshold for cortisol production and therefore they may not have had room to increase their hormone levels. In biological terms, these individuals may have experienced homeostatic overload, a condition in which the body attempts to reconcile increases in stress, beyond those experienced in daily environments, by increasing glucocorticoid production. This response cannot be maintained without causing damage to the individual (Romero, Dickens, & Cyr, 2009). Therefore, those with already high levels of circulating cortisol may be less likely to increase the production of cortisol in response to acute stress, which may result in an inability to respond to stressful circumstances and thus lead to more involvement in crime. In contrast, those with the ability to produce a cortisol/physiological stress response are able to attend to and make decisions based on their physiological response. Consequently, they may be less involved in risky behaviors that could result in negative consequences, such as aggression and crime.
Finally, those with higher intelligence also demonstrated an increase in antisocial behavior in relation to a greater discrepancy between baseline levels of testosterone and cortisol reactivity. Moreover, the positive correlation between the ratio and baseline testosterone and negative correlation between the ratio and cortisol reactivity suggests that the ratio represented higher baseline testosterone and lower cortisol reactivity. Thus, it is likely that those with higher intelligence and a more pronounced ratio of higher circulating testosterone and lower cortisol reactivity demonstrated greater involvement in antisocial behavior. It may be that a combination of higher intelligence and this specific hormonal balance allows such individuals to commit more antisocial acts either because they are more cunning and able to get away with the crimes because of their intelligence, or better able to justify their actions and thus commit more antisocial acts with less remorse or some combination of the two. These findings warrant further research to investigate how intelligence may impact the relationship between cortisol and antisocial traits.

The exploratory analyses in this study were also enlightening. In general, cortisol demonstrated more significant change from post-stressor to post-stress recovery, unlike most stress reactivity studies that demonstrate a significant pulse of cortisol approximately twenty minutes following a stressor and a slower decline in cortisol twenty minutes after the pulse, among non-psychopathic participants (e.g., Stadler et al., 2011; O’Leary et al., 2007). Interestingly, the self-report measures of distress for this study corroborate a greater impact from post-stressor to post-stress recovery than baseline to post-stressor. In addition, there was next to no relationship between the self-report measures of distress and the hormone levels. One would think that negative emotions or feelings following the TSST might correspond with hormonal reactions, but that was not the case. It is possible that these findings suggest that the TSST was
not be sufficiently stress inductive for this population and may have constituted more of a minor annoyance.

The mixed-model repeated measures analyses revealed relationships between hormonal changes across time and primary psychopathy. The affective facet was predictive of cortisol across time point (baseline through post-stress recovery) and the interpersonal and affective facets were predictive of testosterone across time point. Each of these main effects was also accompanied by interactions between the significant facet and age of participant. Younger participants with high affective and interpersonal traits were the most salient in these analyses. Youth aged 12-15 with high levels of interpersonal facet traits behaved more like older participants than their low interpersonal counterparts. This group also demonstrated noticeable drops in cortisol twenty minutes post-stressor, which were not pronounced within the other groups. Thus, the combination of young age and higher factor one traits appeared to influence more hormonally reactivity when these youth were faced with a stressor.

In addition, five findings emerged from the exploratory GLMs investigating hormonal influences on psychopathy, when controlling for age and sex. These relations were further explored in a categorical fashion because age and sex may well affect the nature of the relations between psychopathy and hormones. The interpersonal facet was inversely predicted by cortisol reactivity, specifically the area under the curve with respect to increase, suggesting that those with lower cortisol increases after a stressor tended to have lower scores on the interpersonal facet. Moreover, the interpersonal facet was positively predicted by testosterone reactivity with respect to ground, thus those scoring high on the interpersonal facet generally produced more testosterone across time point. Additionally, the interpersonal and lifestyle facets were both predicted by an interaction between sex and a change in cortisol from post-stressor to post-stress
recovery. Finally, cortisol reactivity (with respect to ground) interacted with sex to predict the affective facet. Further inspection of these interactions revealed that males generally did not seem to differ in their cortisol levels depending on their levels of psychopathy, but females with higher scores on these facets demonstrated a drop in cortisol from post-stressor to post-stress recovery. This finding is in contrast to that of O’Leary and colleagues (2010) who found that females in the comparison group showed a drop in cortisol from baseline to post-stressor, but females with psychopathic traits had a blunted cortisol reaction.

Interestingly, when the analyses were reversed many more significant relationships surfaced that demonstrated psychopathy’s possible impact on hormone composites. Among cortisol variables, interactions between sex and psychopathy total, interpersonal, and lifestyle scores predicted change in cortisol from post-stressor to post-stress recovery. Note that those last two interactions were a mirror image of those found when the same hormone composite acted as a predictor for psychopathy. Moreover, the interaction between sex and the interpersonal facet predicted the change in cortisol from baseline to post-stress recovery as well. Males and females behaved in the same way with change from baseline to post-stress recovery as they did with change in post-stressor to post-stress recovery. However, females with higher interpersonal facet scores exhibited a large drop in cortisol, while males remained relatively stable.

For the testosterone variables, the interpersonal facet, and an interaction between the interpersonal facet and age predicted testosterone reactivity (AUC\(_G\), thus including all levels of testosterone). Closer inspection of the interaction between age and the interpersonal facet revealed that younger participants increased in testosterone reactivity with higher scores on the interpersonal facet, whereas older participants did not show a visible effect. This finding may suggest that, given their stage of puberty, younger participants with higher interpersonal traits
were experiencing spikes in testosterone that were uncharacteristic for their age, whereas older participants had higher testosterone levels to begin with. In addition, the affective facet predicted an increase in testosterone from post-stressor to post-stress recovery. The present study was the first to look at how psychopathy relates to testosterone reactivity to stressful/challenging stimuli. This being said, this was not the first study to find that testosterone is stress-responsive (e.g., Eatough, Shirtcliff, Hanson, & Pollak, 2009; Marceau, Dorn, & Susman, 2012).

Some of the ratios were also predicted by the affective facet and corresponding interactions. The two ratios of baseline measures, baseline cortisol/baseline testosterone and baseline testosterone/baseline cortisol, were predicted by the affective facet and an interaction with age. The same was true for the ratios of baseline testosterone/cortisol reactivity (area under the curve with respect to ground) and baseline cortisol/testosterone reactivity (area under the curve with respect to ground). Graphical representations of these relationships suggested that differences across the age groups were only slight.

In line with Von Honk and Schutter’s (2006) prediction that the interaction of psychopathy and the balance between the HPA and HPG would be more pronounced within primary psychopathy, most of the relationships between psychopathy and hormones involved the interpersonal or affective facets, with the exception of the lifestyle facet which was predicted by an increase in cortisol from post-stressor to post-stress recovery. Most notably, the affective facet was implicated in predictive relationships with each of the ratios, save for those that included reactivity with respect to increase. These ratios represent the intertwined relationship between cortisol and testosterone and thus most closely represent the emotional balance that Van Honk and Schutter (2006) described. As the authors stated, the unique balance of cortisol and testosterone among those with psychopathic traits may contribute to flattened affect, a reward
driven motivational imbalance, and poor communication between cortical and subcortical regions implicated in socio-emotional communication with the environment. All but the motivational balance are represented by traits within the affective facet such as, lacking remorse, shallow affect, and callous lack of empathy.

This study adds to a growing body of literature suggesting that the relationship between psychopathy and the HPA and HPG axes may be complex, and psychopathy may interact with these systems to impact hormonal production. It is of interest, that psychopathy was implicated in more relations predicting hormone composites than the reverse (i.e. hormone composites predicting psychopathy variables). Although on one level, this might be considered counterintuitive, this finding does not violate resounding theories regarding psychopathy development (e.g., the triple balance model or the neurodevelopmental hypothesis) because they emphasize a feedback relationship between the HPA/HPG and psychopathic traits. This being said, due to multicollinearity issues, the analyses predicting psychopathy from hormone variables may have missed some true relationships.

Other researchers have found similar relationships. Recently, Gostisha and colleagues (2014) found that scores on the affective scale interacted with levels of recent life stress to impact daily cortisol production among incarcerated males. Specifically, higher affective traits in combination with more recent stress predicted a steeper cortisol awakening response. Moreover, males with callous traits and a history of abuse had reduced waking cortisol and a steeper decrease in cortisol throughout the day. Von Polier and colleagues (2013) also found that CU traits, but not antisocial behavior, were especially predictive of reduced diurnal cortisol levels among youth with early onset Conduct Disorder. Indeed, despite considerable support for the connection between the balance of the HPA and HPG axes and psychopathic traits, the causal
direction of this relationship is yet to be determined. Thus, it is reasonable to believe that hormonal functions affect some aspects of psychopathy and, in turn, psychopathy predicts some aspects of those hormonal functions. For example, having higher levels of testosterone and lower levels of cortisol may lead one to be sensation seeking, but being sensation seeking can result in negative responses in the environment that result in habituation to stress over time.

Taken together, testosterone, cortisol, and psychopathy interacted in many interesting ways within this study, suggesting a possible role for hormonal interactions within psychopathy development and/or maintenance. Understanding these relationships better may guide our field in terms of identifying those at risk of developing psychopathic traits, creating more effective treatments, and even possible pharmacological interventions. Of course, much more research on this topic must be conducted before these implications will be possible and fully realized.

These findings should be considered in light of a few limitations. Firstly, youth without guardians present were not allowed to participate in the study due to IRB requirements of parental consent. In which case, the sample may have been subject to a selection bias, as those without parents/guardians present for visitation may have differed on important aspects related to the study, for example, lack of supervision and support from parents. In addition, the sample size may have impacted the power of the study to detect relationships. Another limitation of the study was that participants were not provided with a standardized resting period before taking the baseline saliva sample, which some studies have provided (e.g., Glenn et al., 2011; O’Leary et al., 2010), thus the baseline measure cannot be considered a true baseline. This may explain why there were not many findings suggesting differences between baseline and post-stressor samples. Further, the detention center is a stressful place for many youth, thus some participants may have met their threshold of stressful experiences, given the constant stress of being away from their
families in a restrictive, and corrective legal environment. Therefore, they may not have had room to increase their hormone levels.

Furthermore, due to a time pressure to administer the sessions before the participants were released from the detention center, the female subjects were not tested at designated points in their menstrual cycle. O’Leary and colleagues (2010) found that the relationship between cortisol and psychopathy can be masked by low levels of progesterone, thus menstrual phase may impact results. Moreover, the sample contained an uneven distribution of males and females, so differences based on sex may have been skewed. This being said, this distribution mirrored that of the gender composition within the detention center and too many studies exclude females from analyses of detained samples for this same reason. Each of these issues may have created an opportunity for a type II error or an inability to detect true relationships within the population from the sample.

The implications of this study suggest a couple avenues of future investigation. Firstly, for studies of hormonal reactivity among detained youth to be effective, a stressor must be identified for this population. There are many restraints on what kind of tasks can be used within these facilities. Youth detention and custody facilities often have high security and restrictions on internet, thus, online games, and tasks requiring extensive equipment are unlikely to be permitted. Furthermore, these facilities are legally liable and responsible for the safety of their detainees and visitors and therefore they are often weary of stress inducing tasks that might invoke rage or violence toward researchers, staff, or other students. Finally, incarcerated youth constitute two protected populations: namely, children and prisoners. With these considerations, it will be a necessary endeavor to further identify potential stress inductions for this population that raise levels of stress but also fit within the parameters of research within detention facilities.
Another important avenue of research is further incorporating females into analyses of hormonal reactivity and psychopathic traits. In general, very few studies of adolescent offenders include females, given their lower base rates within these settings. For this reason some have said that female offenders are neglected by social science researchers and often misunderstood within the juvenile justice system (Hoyt, & Scherer, 1998). Moreover, females are increasingly becoming involved with the juvenile justice system due to simple assault charges, status offenses, and violations of conditional release (Chesney-Lind, & Randall, 2013). Further, in 2009, females accounted for 30% of all juvenile crime and 18% of violent crime (Puzzanchera, & Adams, 2011). Needless to say females constitute an important portion of the adolescent offender population. From this study, and others (e.g., O’Leary et al., 2010; Vaillancourt, & Sunderani, 2011), we also know that females may differ in their hormonal reaction to stress in general and may display different interactions between stress and psychopathic traits.

There were many findings suggesting that testosterone and cortisol may have a complex and interacting relationship with psychopathy. Specifically, interactions between the affective facet and age were predictive of various combinations of ratios of the two hormones (e.g., baseline testosterone/baseline cortisol and baseline testosterone/cortisol reactivity (with respect to ground). Moreover, the antisocial facet was predicted by an interaction between intelligence and the ratio of baseline testosterone/cortisol reactivity (with respect to increase). Future directions should investigate further how cortisol and testosterone relate to each other throughout the day, and in relation to acute, and chronic stress. Moreover, many studies have investigated the neurobiological underpinnings of psychopathy in relation to stress reactivity and hormone production, but very few have examined how these youth are cognitively or behaviorally impacted by stress and hormonal production. Future studies should not only collect hormonal
induces of stress reactivity, but behavioral as well. Such studies could help in elucidating a
direction for the relationship between psychopathy and hormones by demonstrating how
psychopathic traits may interact with hormones to manifest behaviorally in the moment.

**Conclusion**

In summary, this study was designed to clarify how hormonal functioning, intelligence,
and maturity relate to psychopathy among detained adolescents and to investigate how these
variables may influence each other. The resulting relationships provided further support for the
hypothesis that cortisol and testosterone interact to influence psychopathic traits. These findings
also corroborate recent findings implying that psychopathic traits may in turn influence hormonal
functioning, especially traits within primary psychopathy relating to interpersonal and affective
functioning. Moreover, the present findings expanded on the current conceptualization of how
psychopathy relates to intelligence. In particular, among this sample intelligence was positively
associated with psychopathic traits relating to impairments of interpersonal functioning and
lifestyle. Finally, a novel finding was produced suggesting that intelligence may interact with
cortisol and testosterone production to influence antisocial behavior, but this finding was not
evident for more traditional psychopathic traits. These findings warrant future research to
elucidate the complex interplay of intelligence and hormonal functioning and to further examine
how these variables impact the development of antisocial behavior in general and psychopathic
personality traits in particular.
REFERENCES


APPENDIX

IRB Approval

September 27, 2013

Randy Salekin, PhD
Department of Psychology
College of Arts and Sciences
Box 870348

Re: IRB# 13-009
“The Influence of Personality Traits and Hormones on Criminal Involvement”

Dear Dr. Salekin:

The University of Alabama IRB has received the revisions requested by the full board on 8/16/13. The board has reviewed the revisions and your protocol is now approved for a one-year period. Please be advised that your protocol will expire one year from the date of approval, 8/16/13.

If your research will continue beyond this date, complete the IRB Renewal Application by the 15th of the month prior to project expiration. If you need to modify the study, please submit the Modification of An Approved Protocol Form. Changes in this study cannot be initiated without IRB approval, except when necessary to eliminate apparent immediate hazards to participants. When the study closes, please complete the Request for Study Closure Form.

Should you need to submit any further correspondence regarding this proposal, please include the assigned IRB application number. Please use reproductions of the IRB approved stamped consent/assent forms to obtain consent from your participants.

Good luck with your research.

Sincerely,

[Signature]