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The Association between Affective Psychopathic Traits, Time Incarcerated, and Cortisol Response to Psychosocial Stress

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Abstract

Previous research has demonstrated psychopathic personality traits are significantly predictive of blunted cortisol reactivity to a performance-based stressor task (Trier Social Stress Test; TSST) in college students. However, the relationship between cortisol reactivity and psychopathy has not been explored in high risk samples such as incarcerated populations. Further, the role of imprisonment in relation to cortisol stress reactivity has not been previously explored, but could have practical and conceptual consequences in regards to rehabilitation and biological sensitivity to context, respectively. The current study tested the hypotheses that both psychopathic personality traits and amount of time incarcerated are related to cortisol blunting in response to stress among incarcerated young adults. A sample of 49 young adult male offenders was recruited to complete the TSST. Salivary hormone samples were taken just prior to and 20 min post-stressor, and participants were interviewed with the Psychopathy Checklist – Youth Version. Variables quantifying the amount of time at the present facility prior to the date of testing and number of commitments in juvenile facilities were also collected. Correlational analyses indicated that only number of incarcerations was related to blunted cortisol. Hierarchical linear modeling revealed that time incarcerated and number of commitments were related to a blunted cortisol response among responders and declining cortisol reactivity among nonresponders, respectively. Controlling for time incarcerated, psychopathic traits were significantly related to cortisol decline in response to the stressor among nonresponders, but were not related to blunted cortisol among responders. Results of this project highlight the potential biological effects of prolonged and repeated incarcerations, and extend our understanding about the relationship between psychopathic traits and cortisol reactivity in an incarcerated sample.

Introduction

Psychopathy represents a heterogeneous personality style that can vary considerably in symptom presentation (Brinkley et al., 2004). Psychopathy is also a unique classification, in that it not only affects the individual with the diagnosis, but also places others at risk for physical harm, with related mental and physical health concerns and costs. A substantial number of affected individuals experience comorbid substance use and other personality disorders and are generally considered to have poor response to treatment (Harris & Rice, 2006; Taylor & Lang, 2006). The array of psychological symptoms and poor treatment response associated with psychopathic traits make understanding the underlying aspects contributing to this phenotype a serious public health concern.

One underlying aspect that has been hypothesized to be related to psychopathic traits is aberrant or blunted stress reactivity (Liken, 1995; Patrick et al., 1993). Stress reactivity is a risk factor involved in fear conditioning (e.g., response to distress/punishment cues) and the socialization of conscience. Extremes of stress reactivity disrupt the careful processing of social feedback cues and promote failed socialization efforts and subsequent behavioral dysregulation (Lykken, 1957, 1995). Biological indicators of stress reactivity are of particular importance because they might have long term effects (i.e., contribute to both initiation and maintenance of psychopathology) and offer promise for early identification of risk factors that could help advance better prevention and intervention.

One such biological indicator of stress reactivity comes from the hypothalamic-pituitary-adrenal (HPA) axis. The HPA axis is a stress reactivity network that connects the central nervous and endocrine system (Kudielka & Kirschbaum, 2005). Activation of the HPA axis is triggered by novel/threatening social stimuli and results in the release of the end product cortisol from the adrenal gland (Fries et al., 2009). Cortisol's response profile is relatively slow and, consequently, cortisol reactivity patterns have long lasting repercussions on brain and behavioral functioning as well as immediate implications for brain activation patterns by changing membrane excitability (Lupien et al., 2006; Sapolsky et al., 2000). The HPA axis interacts extensively with limbic and paralimbic neurocircuitry such as the amygdala, insula, and anterior cingulate cortex, causing some to rename this axis the LHPA axis (Limbic HPA axis; Gunnar & Vazquez, 2001; Shirtcliff et al., 2009). There are numerous neural extensions connecting the limbic system to the hypothalamus, which is the structure responsible for triggering the cascade that releases cortisol into the blood stream to target organs (Risold et al., 1997). Given this limbic-HPA link, studies that examine acute HPA reactivity to an acute challenge are sorely needed, especially in relation to the development of antisocial behavior, as physiological under- reactivity could be due to inherent personality traits (i.e., antisocial traits) or could represent habituation to prior stressful life events (Phillips et al., 2013), such as frequent and/or long periods of incarceration.

One phenotype that is prevalent in prison populations is psychopathy, a phenotype marked by incapacity to experience empathy and guilt (Cleckley, 1976) and therefore represents the aspects of antisocial personality that would be most affected by HPA deficiencies. It has been suggested that psychopathic personality traits (lack of empathy, guilt, and interpersonal callousness) are the underlying cause of persistent and severe forms of antisocial behavior

like ASPD (Hare et al., 1991; Walters, 2003). Researchers have begun investigating basal or diurnal HPA functioning in incarcerated individuals (Brewer-Smyth & Burgess, 2008; Gostisha et al., 2014; Johnson et al., 2014), but no studies have investigated HPA reactivity in an incarcerated setting. This gap in the literature is surprising, given that reactivity to threat or emotion-laden stimuli is theorized to be a distinguishable feature of psychopathy (Benning et al., 2005; Drislane et al., 2013; Pastor et al., 2003).

One of the most prominent and well validated stress induction measures used in prior hormone research is the Trier Social Stress Test (TSST; Gaab et al., 2003; Kirschbaum et al., 1993). The TSST has been shown to induce a significant increase in cortisol levels and heart rate in college samples (Gaab et al., 2003; Kirschbaum et al., 1993) but has never been utilized in incarcerated samples in prior studies. Previous studies utilizing the TSST have demonstrated a lack of stress-induced increases in cortisol among college students high in psychopathic traits regardless of level of antisocial behavior (O'Leary et al., 2007; O'Leary et al., 2010). The results of the O'Leary et al. studies imply that hypo-reactive HPA axis may be a neurobiological indicator of psychopathic traits; however this effect has not been shown in individuals who are at the highest likelihood to exhibit the phenotype of psychopathy, such as incarcerated samples.

Aside from psychopathic traits, there are a variety of stressful experiences that could be linked to blunted HPA reactivity in a secure correction facility. Individuals entering incarceration for the first time may experience elevated levels of stress during incarceration (Brown & Ireland, 2005). However, recent research has also shown that adults who experienced early adverse life events during development demonstrate blunted cardiovascular and cortisol response to laboratory stress (Lovallo, 2013). It has been hypothesized that prolonged stress “gets under the skin” and tends to result in a down-regulated biological stress system, leading to what is termed “burnout” of the HPA axis (Heim et al., 2000; Miller et al., 2007). In support of this theory, a recent study found that greater life stress exposure was related to hypoarousal of the HPA axis (Gostisha et al., 2014). The mechanism for this hypocortisolism has been speculated to be related to reduced adrenal cortical secretion or reactivity, or enhanced negative feedback inhibition of the HPA axis (Heim et al., 2000). The reduced impact of cortisol on target cells could potentially be related to increased cortisol binding (resulting in less “free” and bioavailable cortisol) or reduced sensitivity of target cells for cortisol (Heim et al., 2000). The hypocortisolism phenomenon has never been assessed before in incarcerated samples, despite the fact that burnout of the HPA axis could have profound effects on rehabilitation by causing less responsiveness to treatment efforts. Further, research from foster children indicates that number of placements is associated with blunted diurnal cortisol (Fisher et al., 2011) and that time since a traumatic experience elapsed is an important moderator of cortisol functioning (Weems & Carrion, 2007; Miller et al., 2007). However, number of placements in incarcerated facilities and amount of time incarcerated has never before been examined in relation to cortisol reactivity. The experience of incarceration may have similar effects on the HPA axis as foster placements or other adverse life events, but could also have a unique impact on HPA reactivity and is thus highly important to investigate. Therefore, both amount of time incarcerated prior to testing date and number of times incarcerated appear to be important variables that could lead to blunting of HPA axis response. Along these same

lines, repeated substance abuse can cause recruitment of stress-related neurocircuitry brain stress systems including the HPA axis, and is related to a similar down-regulation of the HPA axis over time (Koob & Le Moal, 2008; Lovallo, 2006). Antisocial behavior has been associated with substance abuse (e.g., Taylor & Lang, 2006), and therefore assessment of previous substance use is necessary in order to parse out the effects of personality traits (i.e., psychopathy) and stressful experiences (i.e., incarceration).

In conclusion, the current study is novel in many respects, including its use of a well-known stress test (i.e., TSST) in an incarcerated sample to test whether psychopathic traits are associated with blunted cortisol reaction to psychosocial stress. This hypothesis was based on prior research demonstrating that Factor 1 psychopathic traits are predictive of blunted cortisol reactivity in college students, but this effect has never before been tested in an incarcerated sample of males who are more likely to demonstrate the phenotype of psychopathy. The effects of the amount of time incarcerated prior to the experiment and the number of times incarcerated were also evaluated in order to better understand whether these stressful experiences lead to hypocortisol response to stress. Finally, presence of substance use disorders was also assessed as a potential covariate in analyses.

Materials and Methods

Participants

A total of 49 (mean age = 18.41, $SD = .31$) young adult male offenders aged 18 and older were recruited for the current project. Participants resided at a high-risk residential facility for male offenders who were convicted of crimes prior to the age of 18, but who turned 18 while at this facility. Offenders were referred for serious felony crimes, and many had been referred from other facilities of lower security (low or moderate) due to poor behavior or additional crimes committed in those facilities. Offenders were sentenced for at least one year, with time added for poor behavior if necessary. However, all participants were tested within their first year at the facility. It should be noted that at the time of the experiment, the facility was under heightened stress due to speculations that the facility might be shut down, and it was closed shortly after the completion of the experiment. This may have increased the stress felt among the participants of the study. All 18 year-olds at this facility were asked to participate in the study, of which only 2 refused due to factors unrelated to the project (i.e., being released in a few days from the facility). The ethnicity of the sample was African American (63.3%), White (34.7%), and other (2%).

Procedure

The study was conducted in two sessions run in the afternoon over two days. Potential participants were recruited by project personnel who explained the general procedures of the study to the participant and obtained consent. Participants were told to abstain from excessive exercise, eating, and consuming caffeinated beverages 1 h prior to the first session, as these activities can impact cortisol production (Takai et al., 2004).

Participants started the first session between 3 and 4 PM when cortisol levels are most stable (Kudielka et al., 2004). Participants were escorted into an office within the participant's

residential cottage to conduct the first session. This was a room that they were familiar with, as it was in their cottage, and thus eliminated the risk of inducing cortisol reactivity due to a novel environment like a laboratory. Lab personnel administered a brief adherence questionnaire to identify whether the participant had exercised, eaten, or consumed caffeine within the past hour. If the participant had engaged in any of these activities, they were rescheduled for a different day and asked to refrain from these activities prior to the next session. Participants next relaxed for 10 minutes and then provided their first saliva sample (see methods below). Then the participant began the TSST (see methods). Twenty minutes after the TSST ended, the participant was asked to provide a second saliva sample. Participants were then rewarded with a food treat of their choice for participation in the study.

The second session occurred on a separate day after the TSST, as the interviews probe for sensitive information that could produce a stress response. Participants' interviews were audio recorded in order to estimate inter-rater reliability.

Measures

Psychosocial Stress Test—Before the TSST started, participants were instructed to relax for 10 min. Next, the TSST (Kirschbaum et al., 1993) began with a 10 min preparation period followed by a 5 min mock job interview/public speaking task in front of two college student confederates and the experimenter, who were described as being trained in behavioral observation. A video camera was present and participants were told that they were being filmed and that the film would be reviewed by an expert behavioral analyst (though no filming actually occurred). After the mock job interview, a 5 min calculation task was completed with feedback from the confederates.

Stress Response—Two saliva samples were collected during the first study session to assess change in cortisol concentration. Participants deposited 4 mL of saliva directly into a vial to assess baseline cortisol concentration after the 10 min relaxation phase and immediately before the 10 min preparation phase for the TSST. Participants provided another 4 mL of saliva 20 min after the TSST was completed.

Institutional Records Review—The official institutional file for each participant was used to code offending history in order to complete the Psychopathy Checklist-Youth Version (PCL-YV; see interviews below). Additionally, some adolescents entering incarceration could experience high levels of stress during the initial weeks of incarceration, which could affect HPA functioning (Brown & Ireland, 2005). Thus, amount of time incarcerated at the current facility prior to the date of the experiment and number incarcerations were calculated and loaded onto the base model for cortisol to assess for potential covariation (see results below).

Interviews

Psychopathy Checklist-Youth Version (PCL-YV)—The PCL-YV (Forth et al., 2003) measures psychopathic traits in individuals 18 and younger and consists of a semi-structured interview and criminal file review. The items from the PCL-YV measure two factors: Factor

1 measures lack of empathy/guilt, superficial charm and manipulation and yields a continuous score which served as the dependent variable for the proposed project. Factor 2 measures antisocial behavior such as early behavioral problems and impulsivity. These factors can be further delineated into four facets: Interpersonal facet (impression management, grandiose sense of self, pathological lying, manipulation), Affective facet (lack of remorse, shallow affect, callous lack of empathy, failure to accept responsibility), Lifestyle facet (stimulation seeking, lacks goals, impulsivity and irresponsibility), and Antisocial facet (poor anger control, early behavior problems, juvenile delinquency, revocation of conditional release, criminal versatility). Primary analyses utilized the two factors in analyses, whereas the secondary analyses utilized the facets. An extensive literature has documented the reliability and validity of the PCL-YV (Forth et al., 2003). A random sample of interviews (approximately 12% of the sample) were independently coded from the audio tape by a graduate research assistant who was blind to the initial coding. Inter-rater reliability (Intra-class correlation, ICC) on total symptom count was .88.

Mini International Neuropsychiatric Interview (MINI)—Presence of substance abuse and other psychopathology was determined by administering the entire MINI (Sheehan et al., 1998), which is a widely used structured psychiatric interview. The MINI has good inter-rater and test-retest reliability and excellent convergent validity with other well-known structured interviews (see Sheehan et al., 1998). Reliability for diagnoses in the current sample was good (kappas were all 1.0, with the exception of marijuana abuse at .57), and the reliability correlation (ICC) on symptom counts ranged from .85 - 1.0, with the exception of psychotic symptoms, $r = .63$.

Self-report Measure

Perceived Stress—This was assessed both times a saliva sample was taken by asking participants to rate their perceived stress level (“Please rate your current level of stress using the following scale”) on a 7-point Likert rating scale ranging from 1 (No Stress) to 7 (Maximum Stress). This is a common procedure in the greater stress induction literature to track cognitive appraisal of stress stimuli (e.g., Nater et al., 2005; Nejtcek, 2002). Additionally, observers (i.e., experimenter and confederates) independently rated the amount of stress they observed from the participant during the TSST, using the same 7-point Likert rating scale. These observer ratings were later averaged and used as another validity check for the TSST.

Analyses

Pearson correlations were calculated among measures as an initial check on hypothesized associations and to assess the need for inclusion of proposed control variables in the analyses. Follow up analyses were conducted using Hierarchical Linear Modeling (HLM v6.05; Raudenbush et al., 2004) to account for the inherent nesting of the two cortisol samples collected within the individual (level-1) across 49 individuals (level-2). Hierarchical Linear Modeling (HLM) is advantageous as it allows for the simultaneous modeling of hormone levels and the dynamic changes in cortisol functioning. Analyses were conducted with the two cortisol samples as the outcome of interest (Y_{CORT}). At level-1, cortisol

intercept was estimated which represented the highest cortisol level regardless of whether an individual evinced increased or decreased cortisol response to the stressor, as well as two additional variables that best captured the change in cortisol in response to the stressor. Descriptive statistics indicated that some individuals demonstrated an increase in cortisol in response to the stressor, whereas others demonstrated a decrease in cortisol, consistent with previous research (e.g., Kirschbaum et al., 1993; Schommer, et al., 2003). The separation of cortisol responders and nonresponders into different groups has demonstrated useful in prior research (Pruessner et al., 2010). A major advantage of HLM is that these different trajectories can be captured within the same model. The best fitting model that accurately characterized cortisol functioning across the sample included a variable estimating the slope for individuals that evinced an increase in cortisol reactivity ($\beta_{1\text{Responders}}$) and a variable capturing the slope for individuals that evinced a decrease in cortisol reactivity ($\beta_{2\text{Nonresponders}}$). The “responder” and “nonresponder” designations were between-subjects variables that separated individuals who showed a rise or positive response in cortisol reactivity to the stressor, and individuals who declined in response to the stressor. For ease of interpretation, the dummy code was set to have positive values indicating greater reactivity, captured by a dummy code that increased in value (0, 1), or to indicate a sharp drop in cortisol in nonreactors, captured by a dummy code that decreased in value, (1, 0). Once these predictors were modelled, the intercept (β_0) could be interpreted as peak cortisol levels observed for that individual. In accordance with Snijders and Bosker (1994, 2002), and following the model presented in Hrushka et al., (2005), we used R^2 estimates to provide estimates of effect sizes from the HLM models of interest. However, it should be noted that Hrushka et al., (2005) caution that R^2 estimates are not to be viewed as a universal estimate due to some important caveats with using this metric. One important caveat to using R^2 to estimate the size of an effect is that lack of heterogeneity within the sample can greatly reduce the true effect size (Hrushka et al., 2005). Given the limited heterogeneity within our sample (all were incarcerated, antisocial youth), this caveat should be heeded for interpretation. Also, R^2 estimates lump together estimates of between and within person variance (Hrushka et al., 2005); thus, when interpreting overall changes in the model using the R^2 , it is worth noting that fluctuations in this metric are common due to changes in variance at the within-subjects level.

Once a level-1 or within-individual equation is established, level-1 predictors can become outcomes-of-interest at level-2. Cross-level interactions capture how individual difference factors impact level-1 associations, specifically cortisol functioning. Analyses focused on time incarcerated, number of incarcerations, and psychopathy as an individual difference factor capable of impacting cortisol functioning. Below is an example of the base model.

Level-1 Model

$$Y_{\text{CORT}} = \beta_0 + \beta_{1\text{Responders}} + \beta_{2\text{Nonresponders}}$$

Level-2 Model

$$\begin{aligned}\beta_0 &= \gamma_{00} + \gamma_{01}(\text{Psychopathy factor/facet}) + U_{0(\text{Residual variance and error term})} \\ \beta_{1\text{Responders}} &= \gamma_{10} + \gamma_{11}(\text{Psychopathy factor/facet}) \\ \beta_{2\text{Nonresponders}} &= \gamma_{20} + \gamma_{21}(\text{Psychopathy factor/facet})\end{aligned}$$

Results

Age and ethnicity are often used as covariates as they are usually significantly related to cortisol functioning (e.g., Hajat et al., 2010; Seeman et al., 2001). However, most participants were age 18 at time of testing (mean = 18, $SD = .30$) so age had a highly restricted range; consequently, age was not a significant predictor of cortisol response to stress in the current study ($B = .03$, $p = .86$). Additionally, ethnicity was unrelated to cortisol stress response ($X^2 = 85.86$, $p = .21$). All other descriptive information and bivariate correlations between control variables and variables of interest can be found in Table 1. As can be seen in Table 1, the hypothesized relationships between psychopathy facets and cortisol reactivity were not significant. Additionally, time imprisoned at the facility was not significantly related to cortisol response to stress was not significant; however, number of prior commitments was significantly related to blunted cortisol.

Hierarchical Linear Modeling was used to further investigate the lack of significant findings. The following variables were measured for potential covariation in analyses: time imprisoned at the current facility, number of incarcerations, and presence of current substance use disorder. The amount of time incarcerated ranged from 64 - 341 days ($m = 190.60$, $SD = 65.22$), and the amount of incarcerations ranged from 1 – 5 incarcerations ($m = 1.90$, $SD = 1.02$). As shown in Table 2, both time variables (time incarcerated and number of commitments) were examined in the above model first to assess their impact on cortisol functioning. Table 2 provides the beta weights for each parameter, and it is evident that time incarcerated at the current facility was significantly related to peak cortisol levels ($B = .004$, $p = .021$) and blunted cortisol responsivity among the responders ($B = -.004$, $p = .007$), but did not affect the nonresponders ($B = .001$, $p = \text{n.s.}$; $R^2 = 3.2\%$). Thus, 3.2% of the variance can be attributed to time spent incarcerated at our facility. On the other hand, number of commitments was not significantly related to cortisol levels and did not affect the responders' cortisol reactivity, but was significantly related to steeper decline for nonresponders ($B = -.293$, $p = .039$; $R^2 = 1.6\%$). Since time imprisoned at the current facility prior to testing affected more parameters than number of commitments (two of three parameters in the model were affected), and explained more variance than number of placements, it was included as a predictor in all subsequent analyses loaded onto the two parameters that were affected (intercept and “responders” parameters). Additionally, given the high stress at the facility at the time of testing, it was necessary to control for the amount of time they had been in that environment prior to participating in the experiment. Presence of a current substance use disorder was not significantly related to cortisol stress response, and was thus left out as a covariate in analyses. Hence, there were two level two predictors per model (time incarcerated as a covariate and the construct of interest) resulting in a ratio of 24 participants per variable. Therefore, the sample size was large enough to eliminate the concern for biased parameter estimates and their standard errors (Bell et al., 2010).

The observed range of pre-stress ($M = .17 \mu\text{g/dL}$, $SD = .12$; range = .01-.60) and post-stress cortisol levels ($M = .21 \mu\text{g/dL}$, $SD = .18$; range = .01-.97) were within normal parameters for the age range sampled (e.g., O'Leary et al., 2007; 2010). A repeated measures ANOVA confirmed an increase in self-reported and observer reported perceived stress ratings for the whole sample ($F = 16.45$, $p < .001$; $F = 28.59$, $p < .001$, respectively). Thus, it appears that the TSST was effective in inducing self-reported and observable stress reaction, even within an incarcerated sample. However, self-reported and observer reported perceived stress ratings were not correlated with cortisol response to stress, as can be seen in Table 1.

The two-level HLM showed some within-person variability in cortisol levels (19%, of total variability), as well as substantial variability in cortisol between individuals (81%, of the total variability was due to between-person effects; $X^2 = 380.304$, $p < .001$), with intercept ($B = 1.922$, $p < .001$), responder dummy code ($B = .958$, $p < .001$), and nonresponder dummy code ($B = .295$, $p = .014$) significantly predicting cortisol responsivity to the stressor.

Factors (Factor 1 and Factor 2) and facets (Affective, Interpersonal, Antisocial, and Lifestyle) of psychopathic traits as assessed by the PCL-YV were distinctly related to aspects of cortisol response to the TSST. As Table 2 indicates, Factor 1 was not significantly related to peak cortisol levels or the cortisol responsivity among responders. Factor 1 did, however, significantly predict a steeper cortisol decline in response to the TSST (nonresponders; $B = -.035$, $p = .02$; $R^2 = 2.2\%$). In other words, 2.2% of between-individual variance not accounted for by time of day can be attributed to psychopathy scores. Among individuals who evinced decreased cortisol in response to the TSST, those with high Factor 1 psychopathic traits decreased faster and had a steeper slope. Factor 2 was unrelated to any aspect of cortisol responsivity. Total psychopathy scores from the PCL-YV were unrelated to peak cortisol levels or the cortisol responsivity among responders, but significantly predicted a steeper cortisol decline in response to the TSST (nonresponders; $B = -.025$, $p = .007$; $R^2 = 1\%$).

To understand further how Factor 1 psychopathy was related to cortisol nonresponse, the specific facets of the PCL-YV Factor 1 (Affective and Interpersonal) were examined as predictors of cortisol response and nonresponse to the TSST. The Affective facet was uniquely related to a significantly steeper decline in cortisol in response to the TSST within nonresponders ($B = -.081$, $p = .02$), but not within responders ($R^2 = 3.1\%$). The interaction between affective psychopathy, time incarcerated, and group status (responders vs. nonresponders) is depicted in Figure 1. The Interpersonal facet was unrelated to cortisol responsivity to the TSST. The two facets from Factor 2 (Lifestyle and Antisocial facets) remained unrelated to cortisol reactivity.

Discussion

The goal of the current project was to clarify how cortisol reactivity to an acute laboratory stressor relates to psychopathic traits in an incarcerated sample. Previous research investigating the association between psychopathic personality traits and cortisol production has found that men with elevated affective/interpersonal psychopathic personality traits show

blunted cortisol reactivity to stress (O'Leary et al., 2007, 2010). The current study aimed to replicate and extend the O'Leary et al. (2007, 2010) findings by utilizing a severely antisocial group of individuals to test the hypothesis that affective psychopathic traits predicted blunted cortisol response to stress.

Contrary to prediction, initial correlations did not support the hypothesized relationships between psychopathic personality traits and cortisol response to stress in this incarcerated sample. Only number of previous incarcerations was related to blunted cortisol response to stress on a covariate level. There could be many reasons why initial correlations were not significant – for example, the confound of time spent at a stressful imprisoned situation could be interfering with the ability to parse out psychopathy effects, or it could be that simple correlations are not powerful enough statistically to detect effects in this small of a sample. Further inspection of the data revealed that about half of the sample evinced an increase in cortisol, whereas the other half decreased in response to the stressor. Thus, HLM was used to decipher whether distinct trajectories could be parsed from the overall sample, and whether the variables of interest were significantly related to cortisol slope.

HLM analyses revealed that Factor 1 psychopathic traits were related to a faster decline in cortisol stress response among individuals who evinced a decreased cortisol response. Individuals with high levels of affective psychopathic symptoms in particular evinced a steep decline in cortisol in response to a social stress test. Previous research (O'Leary et al., 2007, 2010) has found a blunted cortisol response in individuals with high Factor 1 psychopathy scores, suggesting callous symptoms may be linked with HPA functioning; however, these studies did not distinctly model the response trajectories of responders and nonresponders to the TSST, and were not conducted among a severely antisocial sample. In the prior O'Leary et al. studies, participants were in one group in the analyses – thus, it could be that the blunting effects association with psychopathic traits were actually due to nonresponding among some in that subgroup. Neurologically, the amygdala has been shown activated in response to emotional or stressful stimuli, and it is notable that individuals with high psychopathic traits tend to display reduced amygdala activation to fear related stimuli, causing some to hypothesize that amygdala hyporesponsivity is a hallmark feature of psychopathy (Blair, 2007). As noted earlier, limbic structures, including the amygdala, are integrally related to the onset of an HPA response to stress; thus, these brain areas are implicated in blunted or negative response to stress. The steep decline in cortisol evinced in response to the social stressor could thus be interpreted to mean that affectively callous psychopaths acclimate quickly and effectively to stressful situations, and may not pick up on cues in their environment (e.g., cues of distress), or learn from and internalize information in their environment (e.g., the effects of hurting someone else).

However, as depicted in Figure 1, there were two trajectories for individuals high in psychopathic traits. One of the groups evinced a healthy increase in cortisol without significant blunting in response to the stressor. This finding is contrary to the previous O'Leary et al. (2007, 2010) studies and raises the question as to whether there exists a subgroup of psychopaths that indeed have an intact and functional stress response. Interestingly, a recent paper by Gostisha and colleagues (2014) published a similar finding in regards to an intact cortisol diurnal rhythm among incarcerated psychopathic adolescents.

Gostisha et al. hypothesized that a U-shaped curve exists in association cortisol functioning, such that incarcerated individuals with psychopathic traits and chronic stress may have an HPA system that is either blunted or over-responsive, depending on individual difference variables. They further speculated that there is something about the context of incarceration that uniquely suits adolescents that are high on callousness and evince high stress (Gostisha et al., 2014). Whereas a blunted HPA axis would cause an individual to become hardened and less responsive to environmental stimuli, an intact HPA system could theoretically be related to increased “openness” to salient social cues. Such a physiological profile would greatly aid an individual with psychopathic tendencies to respond to social threats quickly and efficiently, for better or for worse.

It was also notable that amount of time imprisoned and number of commitments both appear to be related to a blunting or decline in cortisol responsivity to stress. Specifically, on a correlational basis, the number of commitments to correctional facilities was related to a blunted cortisol response to stress, and HLM analyses revealed a steep decline in cortisol reactivity among those that responded negatively to the task (nonresponders). Amount of time spent in the secure facility prior to the testing date was also related to higher cortisol prior to stress, and a blunted cortisol stress response among responders. Although not tested in the current study, one other source of cortisol blunting could be frequency of committing crimes, which is likely confounded in the number of placements these adolescents had been placed in. The stress response system is highly plastic (Boyce & Ellis, 2005) and these patterns of responding could represent what could be considered “adaptive calibration” of the overall stress response circuit to unpredictable and potentially dangerous situations (Ellis et al., 2012). While a declining cortisol response could be considered an “adaptive” change in the short term, in the long term consistently changing environments and being exposed to unpredictable environments (e.g., prison) likely hinders learning and social sensitivity to feedback (Del Giudice et al., 2012). Indeed, cortisol production facilitates memory and learning, especially emotion-related memory, given its strong connections with limbic structures such as the hippocampus (Roozendaal, 2000, 2002). One main goal of incarceration is rehabilitation, but low cortisol or blunted cortisol reactivity in the context of incarceration may actually work against this goal by impeding learning capacity due to constant environmental stress. It could also exacerbate antisocial behavior further by desensitizing prisoners to valuable information about dangers and threats in the environment, thus leading to higher rates of risk taking.

The mechanisms underlying a “less than zero” or negatively sloped HPA response to a stressor has been documented and described, but not well explained despite the increasing interest in “blunted” HPA response to stress (e.g., Phillips et al., 2013). We speculate that there may be several different mechanisms through which the HPA axis shows extreme nonresponse to stress, focusing below on one potential mechanism. Phillips et al. (2013) recently pointed to bases for blunting in cardiovascular and HPA responding, and one hypothesis that is relevant to the current results was that elements of the stress response can be “titrated into the activity of some other physiological response system.” Phillips et al (2013) used a blunted cardiovascular response to stress as an example of this titration of energy, but the same principle could be at play in regards to a “less than zero” cortisol response to a social stressor as we observed here. Interestingly, there is precedent for a shift

from HPA-driven responding to Hypothalamic-Pituitary-Gonadal (HPG)-driven responding to particular stressors or challenges in the animal literature, especially among strains of mammals known to be more dominant, territorial, and aggressive (e.g., Wobber et al., 2009). It has been hypothesized that the divergent responding patterns is related to differential perception of the stressors, with some likely to perceive a social event or challenge as a “stressor,” and respond with a cortisol response, whereas others perceive the same event as a “dominance threat” and express a testosterone response instead (Wobber et al., 2009). Along these same lines, cortisol response to the TSST did not correlate with self and observer report of stress. While there could be many reasons why there is a divergence between the biological and reported experience of the stress, including blunted or “burned out” biological response despite the subjective feelings of stress, it could be that among these severely antisocial individuals, there was a testosterone response rather than cortisol response. However, this is just a speculation, as testosterone was not collected as part of this study.

Consistent with this mechanism, the results of the current paper complement another recent paper (Johnson et al., 2014) that found significant decoupling between diurnal cortisol and testosterone in association with high callousness among a severely antisocial adolescent sample, such that individuals with elevated callousness typically had low cortisol at the moments in which their testosterone was elevated. Another study conducted using the same sample of incarcerated youth as Johnson et al. (2014) also found that incarcerated youth with high Factor 1 scores and child abuse histories had the most severely blunted HPA diurnal rhythms (Gostisha et al., 2014). These articles fit with the idea that elevated callousness is linked with heightened activity from the HPG-axis instead of the HPA-axis both while resting and during stressful events. The combination of high testosterone and low cortisol could be a particularly hazardous combination (Glenn et al., 2011) as high testosterone has been associated with aggressive behavior and low cortisol and stress has been associated with insensitivity to signals of distress or pain in others (Shirtcliff et al., 2009). Given that testosterone was not measured in the current project, the reasons for a decrease in cortisol can only be speculated; however, future research should extend these methodologies to include both the HPA and HPG-axes.

There are some limitations that should be considered when interpreting results of the current project. First, the data from the current study were unable to parse out causation between variables. Additionally, other variables of interest were not addressed in the current project, such as length of sentence and severity of previous antisocial behavior, both of which could also be related to blunted cortisol response. Perhaps a longitudinal study would be able to address causation between psychopathy and altered cortisol response to stress. Also, further research about whether there is a causal effect between amounts of time incarcerated, number of placements, and blunted cortisol reactivity would be useful in understanding effectiveness of rehabilitation efforts in incarcerated settings and may lead to new innovation when it comes to treatment of incarcerated adolescents. Second, the sample size was smaller than optimal, which could have increased Type II error by hindering our ability to detect significant effects, and also could have caused us to find significant effects that otherwise would have not been detected due to the idiosyncrasies of the sample (Type I error). Further, there are potential issues that can arise with the use of HLM with dyadic data (Singer & Willet, 2003), though these issues did not appear to impact the findings of the study. Finally,

it remains unclear how diurnal cortisol and cortisol reaction to stress operate together in relation psychopathy and whether they differ in individuals who are incarcerated. It will be important for future research to characterize both aspects of HPA activity in relation to psychopathy and effects of incarceration in order to gain a better understanding of how different components of HPA axis functioning are inter-related and associated with these conditions.

Conclusion

In conclusion, a burgeoning field has highlighted neurobiological mechanisms, including hormone functioning, to be specifically related to aspects of antisociality including psychopathic traits. The current study is novel in its emphasis on testing specific aspects of antisocial traits among incarcerated males with severe antisocial behavior. This has allowed for a broader perspective in understanding the role of specific traits of psychopathy as they relate to the biological underpinnings of this phenotype. Results add to research supporting the link between the affective dimension of psychopathy to decreased cortisol response to stress among prisoners, but further delineate another group of individuals high on psychopathy that exhibited an intact stress response. . Future studies should continue to research stress response among incarcerated individuals, especially hormonal mechanisms underlying antisocial phenotypes to better explain how they are related to severe and persistent antisocial behavior.

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Figure 1.

Cortisol concentrations predicted before the onset of the TSST and 20 min post-TSST based on HLM modeling at two standard deviations above and below the mean on time imprisoned and affective psychopathic traits as assessed by the PCL-YV.

Table 1

Variable	1	2	3	4	5	6	7	8	9	10	11	12
1. Cortisol change (.15, .18)	---		5									
2. Baseline Cortisol (.17, .14)	0.16	---	5									
3. Peak Cortisol (.31, .24)	0.84	0.68	---									
4. Age (18.01, .30)	-0.02	0.01	-0.01	---								
5. Time Imprisoned (192.92, 67.17)	0.06	0.27	0.2	0.11	---							
6. No. Commitments (1.88, 1.02)	-0.33*	-0.06	-0.28	0.09	0.01	---						
7. Presence of SUD (55% met full criteria)	0.02	0.05	0.04	-0.08	-0.28+	0.1	---					
8. PCL-YV F1 Score (7.67, 3.77)	0.03	0.04	0.05	-0.07	0.18	-0.01	-0.2	---				
9. PCL-YV F2 score (11.89, 3.00)	0.1	0.05	-0.1	-0.12	-0.24	0.09	0.22	0.35*	---			
10. PCL-YV tota score (23.07, 6.82) I	-0.02	0.04	0.04	-0.17	-0.01	0.06	-0.03	0.86***	0.74***	---		
11. Self-Reported Stress Change (.96, 1.64)	-0.07	0.02	-0.05	-0.17	-0.08	-0.2	0.04	0.06	0.01	0.03	---	
12. Observer-Reported Stress Change (3.86, 1.67)	-0.07	0.06	-0.02	-0.17	-0.36*	-0.11	0.38**	-0.15	0.07	-0.02	0.02	---

+p<.10;

* p<.05;

** p<.01,

*** p<.001

Table 2
HLM model coefficients

	Cortisol Responsivity		
	Intercept ^a	Responders Slope	Nonresponders Slope
Time Incarcerated	.004 *	-.004 **	.001
Number of Commitments	-.081	-.081	-.293 *
MINI Substance Abuse	.332	-.125	-.178
PCL-YV Factor 1	-.013	.012	-.035 *
PCL-YV Factor 2	.059	--	-.049
PCL-YV Total Score	-.001	.010	-.025 **
PCL-YV Affective Facet	.024	.014	-.081 *
PCL-YV Interpersonal Facet	.016	--	.040
PCL-YV Lifestyle Facet	.124 ⁺	--	-.090
PCL-YV Antisocial Facet	.029	.008	-.045

⁺ p<.10;

* p<.05;

** p<.01.

-- Fixed Note:

^a Intercept - pre-stress cortisol level

MINI - Mini International Neuropsychiatric Interview

PCL-YV – Psychopathy Checklist - Youth Version