

PSYCHOPATHY, HEART RATE, AND SKIN CONDUCTANCE
IN ADOLESCENT OFFENDERS

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ABSTRACT

Recently, researchers have advocated for a greater focus on measuring neurobiological underpinnings of serious behavior problems such as psychopathy. In particular, psychopathy has garnered significant attention for its relationship with physiological processes. Using a sample of adolescent offenders, this study explored the relationship between scores on the Psychopathy Checklist-Youth Version (PCL:YV; Forth, Kosson, & Hare, 2003) and levels of heart rate (HR), Respiratory Sinus Arrhythmia (RSA), and skin conductance (SC) using a white noise stimulus and countdown task. Correlational results indicated a positive, significant association between baseline skin conductance level (SCL) and the PCL:YV Interpersonal Facet and PCL:YV total score. MANCOVA analyses indicated that skin conductance response (SCR) responders during the anticipatory (pre-noise) and reactivity (post-noise) periods had significantly higher PCL:YV Lifestyle/Behavioral (Factor 2) and Lifestyle (Facet 3) scores than those with no SCRs. There were no significant associations between PCL:YV scores and HR activity across baseline, anticipatory, and reactivity periods. Study results suggest some differences in the biological functioning of adolescent offenders with psychopathic traits, but the small sample used in this study may have contributed to insignificant associations. Future directions for research are discussed.

DEDICATION

For my grandparents, Ron and Rita, *virtute et opera*; Luke and Lorraine, *buaidh no bas*.

LIST OF ABBREVIATIONS AND SYMBOLS

β	Beta: Regression coefficient, the average amount by which the dependent variable increases with unit increases in the independent variable; the slope of a line
d_+	Pooled mean effect size estimate
df	Degrees of Freedom: Number of values in a final calculation that are free to vary (i.e., number of independent observations minus the number of estimated population parameters)
F	F statistic: Value calculated by the ratio of two sample variances
ICC	Interclass Correlation Coefficient: Measure of reliability of measurements or ratings
M	Mean: The sum of a set of values divided by the number of values in the set
n	Sample size of a group
N	Sample size of a population
N_{fs}	Fail-safe N: Estimation of the number of studies confirming the null hypothesis that would be needed to reverse a conclusion that a significant relationship exists
p	Probability associated with the occurrence under the null hypothesis of a value as extreme as or more extreme than the observed value
Q_B	Homogeneity test
r	Pearson product-moment correlation
R^2	R-squared: Coefficient of determination, a measure of effect size
SD	Standard Deviation: Value of variation from the mean
SE	Standard Error: Measure of the statistical accuracy of an estimate
t	T statistic: Value determining whether sample means differ
μ	Micro
χ^2	Chi-square test of significance of model fit

<	Less than
>	More than
=	Equal to
+/-	Plus or minus
%	Percentage

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1. INTRODUCTION

Although the definition of psychopathy has evolved since Hervey Cleckley's first description of the condition in 1941, it remains a constellation of traits that has the potential to negatively affect both the individual and society at large. Cleckley's initial definition of psychopathy, later elaborated on by Robert Hare (1991), has included the presence of interpersonal, affective, and lifestyle or behavioral features as well as potentially antisocial characteristics (Hare, 2003). Interpersonal traits have included such features as superficial charm, grandiosity, manipulation and deceit, while the affective component includes traits such as shallow affect, lack of remorse, lack of empathy, and impersonal relationships. Lifestyle or behavioral features have included such characteristics as lacks goals, inability to accept responsibility for one's actions, and failure to follow a specific life plan. Antisocial characteristics have included explicit antisocial acts and misbehaviors such as misconduct at work and criminal versatility. Thus, according to Cleckley (1976), psychopathy appears to be a multidimensional concept with both personality and behavioral features.

Similar to Hervey Cleckley, Robert Hare's (2003) model for psychopathy includes both personality and behavioral traits. Hare's model is based on his attempts to more systematically index psychopathy which resulted in the development of the Psychopathy Checklist and its revised version (PCL-R; Hare, 1991, 2003). The PCL is composed of two factors, where Factor 1 (F1) represents interpersonal and affective traits of psychopathy and Factor 2 (F2) represents the lifestyle and behavioral characteristics of psychopathy. In recent years, this model has been

further parsed into three (interpersonal, affective, and lifestyle) and four (interpersonal, affective, lifestyle, and antisocial) facet models (Cooke & Michie, 2001; Hare & Neumann, 2006; 2010; Patrick, Fowles, & Krueger, 2009).

Child and Adolescent Psychopathy

Over two decades ago the psychopathy concept was downwardly extended to youth. Research examining child psychopathy has shown that child psychopathy is associated with fearlessness, risk taking, a specific temperamental style and neurocognitive profile. In addition, child psychopathy was shown to be associated with childhood conduct problems (Dadds et al., 2006; Frick et al., 2003; Pardini, Lochman, & Powell, 2007), persistent delinquency (Pardini, Obradovic´ & Loeber, 2006), future recidivism (Boccaccini et al., 2007), and development of antisocial personality disorder in adulthood (Lynam et al. 2007; Pardini & Loeber, 2008). A primary characteristic highlighted in Cleckley's (1941) description of psychopathy was a deficit in general emotional reactivity, and since his early description, a prominent view has been that psychopathic individuals have problems with emotional processing (Blair, 1995; Blair, Jones, Clark, & Smith, 1997; Cleckley, 1976; Eysenck, 1964; Fowles, 1988; Gray, 1987; Lykken, 1957, 1995; Mealey, 1995; Patrick, 1994; Pichot, 1978; Trasler, 1978, 1973). The downward extension of the psychopathy construct to children and adolescents has since revealed that children and adolescents with psychopathic traits demonstrate a similar number of emotional deficits to that which has been observed in adults with psychopathy (Frick, Lilienfeld, Ellis, Loney, & Silverthorn, 1999; Frick et al., 2003; Lynam, Charnigo, Moffitt, Raine, Loeber, & Stouthamer-Loeber, 2009; Obradovic, Pardini, Long, & Loeber, 2007). Specifically, the research has shown that children and adolescents express reduced responding to distressing stimuli (Kimonis, Frick,

Fazekas, & Loney, 2006), deficits in sadness and fear recognition processing (Blair, Colledge, Murray, & Mitchell, 2001), and difficulties with moral and emotional processing (Blair, 1997).

Physiology and Psychopathy

Recently, researchers have advocated for a greater focus on measuring neurobiological underpinnings of serious behavior problems and personality conditions such as psychopathy. Since James and Cannon's seminal debates, physiological responses have long been viewed as tied to emotion, with increases reflecting general emotional arousal, specific emotions, or both (reviewed in Lang, 1994). Given the defining emotional difficulties associated with psychopathy, it is not surprising that psychopathy has garnered significant attention for its relationship with physiological processes, albeit with a major focus on adult populations (see review by Arnett 1997; Gao, Glenn, Schug, Yang, & Raine, 2009; Hare 1978, 1982; Ogloff & Wong, 1990; Patrick, Hicks, Nichol, & Krueger, 2007; Verona, Patrick, Curtin, Bradley, & Lang, 2004).

Within the psychopathy literature, a common measurement of physiological processes is electrodermal activity (EDA) and heart rate (HR). EDA includes measures of skin conductance level (SCL) and skin conductance responses (SCR). SCL is the most common measure of tonic level of electrical conductivity of skin, while SCR is the faster, phasic change in electrical conductivity. Changes in SCL appear to reflect changes in autonomic arousal (Braithwaite, Watson, Jones, & Rowe, 2013), and for electrodermal measures in the emotion and physiology literature, SCL has been highlighted as the most frequently reported response variable, followed by SCR rate and SCR amplitude (Kreibeg, 2010). Similarly, HR has been cited in the research area of emotion and physiological responding as the most frequently reported cardiovascular variable and, overall, the most commonly reported autonomic variable (Kreibeg, 2010).

Skin conductance and heart rate are coordinated by the autonomic nervous system

(ANS), which contains two components: the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). The heart is subject to influence from both the SNS and PNS, in that chronotropic (i.e., rate-related) cardiac effects are controlled primarily by the PNS, whereas inotropic effects such as contractile force and stroke volume are controlled primarily by the SNS. Increased heart rate can be due to increased activation of the SNS, decreased activation of the PNS, or a combination of both. In contrast, the SNS exclusively controls excitation of the palmer eccrine sweat glands (in turn causing increased conductivity in the skin's surface).

Measuring Physiological Processes

In order to evaluate heart rate and skin conductance in psychopathic populations, researchers have traditionally used fear-related tasks and stimuli designed to elicit an autonomic response. These tasks have included aversive conditioning (Flor, Birbaumer, Hermann, Ziegler, & Patrick, 2002; Lykken, 1957), anticipated threat (Hare, 1982; Ogloff & Wong, 1990), startle reflex and visual threat primes (Herpertz et al., 2005; Levenston, Patrick, Bradley, & Lang, 2000), passive avoidance learning (Lykken, 1957; Newman & Kosson, 1986), and response reversal (Mitchell, Colledge, Leonard, & Blair, 2002; Newman, Patterson, & Kosson, 1987). Overall, these and other types of tasks have revealed that increased stimulation-seeking, fearlessness, disinhibition, and associated psychopathic, antisocial, criminal, and violent behavior consistently appear to be underpinned by low autonomic arousal (Fishbein et al., 1989; Fowles, 1993; Fowles, Kochanska, & Murray, 2000; Gatzke, Raine, Loeber, Stouthamer-Loeber, & Steinhauer, 2002; Lahey, Hart, Pliszka, & Applegate, 1993; Raine, 1993; Scarpa, Raine, Venables, & Mednick, 1997; Shannon, Beauchaine, Brenner, Neuhaus, & Gatzke-Kopp, 2007; Patrick, 1994; Volavka, 1995).

One of the most commonly used paradigms for evaluating autonomic responses (e.g., HR

and skin conductance) to aversive stimuli (e.g. a white noise burst) is the countdown task (Hare & Craigen, 1974). This task can include either signaled trials or both signaled and un-signaled trials. The task has typically been used to examine autonomic activity during several phases: i) a baseline or resting period which is the period before the aversive stimulus (referred to as the “anticipation” period) and ii) the period after the aversive stimulus has been presented (often referred to as the “reactivity” period). Using the countdown paradigm and other tasks, several stable associations have been found between psychopathy and i) low resting heart rate (Raine et al., 2014; Wilson & Scarpa, 2011), ii) normal and even exaggerated heart rate reactions during anticipation of an aversive stimulus but decreased skin conductance responses (Hare & Craigen, 1974; Hare, Frazelle, & Cox, 1978; Ogloff & Wong, 1990), and iii) autonomic hyporeactivity in response to aversive stimuli (Gao & Raine, 2010; Lorber, 2004; Mednick, 1977; Patrick, 1994; Raine, 1993).

Theoretical Basis for Autonomic Hypoarousal

Theoretical explanations for the association between decreased autonomic functioning and psychopathic behavior in children and adolescents can be postulated from two camps that have primarily focused on antisocial and aggressive behavior. The first, “fearlessness theory” presumes that low arousal during the resting period of physiological tasks represents low fear and anxiety (Raine, 1993). Decreased fear is then thought predispose a child or adolescent for future antisocial and criminal behavior, based on the belief that “fearlessness” would allow for easier commitment of such acts. On the other hand, a lack of anxiety in childhood would likely contribute to poor learning and socialization due to the inability to feel anxious about punishment and consequence (Raine, 2002).

Some research supports the fearlessness theory, as it does appear as though autonomic

hypoarousal contributes to a fearless temperament in infancy and childhood (Fowles, Kochanska, & Murray, 2000; Kagan, 1994; Scarpa, Raine, Venables, & Mednick, 1997). Further evidence stems from research examining skin conductance, including two studies demonstrating that 3-year-olds with poor fear conditioning were more likely to be aggressive at 8 years of age (Gao, Raine, Venables, Dawson & Mednick, 2009) and more likely to be classified as criminal offenders by age 23 (Gao, Raine, Venables, Dawson & Mednick, 2010). Poor electrodermal fear conditioning has also been found in adolescent offenders (Syngelaki et al., 2013), while another study has indicated that individuals characterized at age 3 as less fearful and inhibited were more likely to have higher psychopathy scores at age 28 (Glenn, Raine, Venables, & Mednick, 2007).

The second major theory prescribes that autonomic underarousal originates from the need for under aroused individuals to seek out stimulation and excitement in order to increase their arousal levels to more pleasant or average levels (Eysenck, 1977; Quay, 1965; Raine, 1993; Raine, Reynolds, Venables, Mednick, & Farrington, 1998). According to this assertion, antisocial behavior is considered a product of the individual's desire to engage in stimulating activities. Supporting the stimulation-seeking theory are results denoting that preschool boys who chose to watch videotapes depicting intense anger had lower heart rates than controls, and also that low resting heart rate was associated with externalizing problems (El-Sheikh, Ballard, & Cummings, 1994). Resting heart rate at age 3 years has also been associated with stimulation-seeking behavior, in addition to aggressive behavior at 11 years (Raine et al., 1997, 1998).

Taking these two theories into account, Raine (2002) has suggested that the fearlessness and stimulation-seeking theories should be combined. Raine and colleagues (1998) point to evidence that both fearlessness and stimulation-seeking at age 3 has predicted aggression at 11 years old and it may be that low arousal produces both temperaments. However, an issue within

this area of research is that although fearlessness and stimulation-seeking have been characterized by some as a defining feature of psychopathy (e.g., Lykken, 1957; Hare, 1998), there are no studies known to this investigator examining the role of fearlessness and stimulation in autonomic underarousal as related to psychopathic behavior (see: Glenn et al., 2007). In general, most research investigating autonomic underarousal in children and adolescents has focused on antisocial and aggressive behavior. Thus, it is difficult to assume that either fearlessness or stimulation-seeking (or both) tendencies contribute to the autonomic hypoarousal found in children and adolescents with psychopathic behaviors. Given that psychopathy research has primarily used heart rate and skin conductance as measurements of autonomic arousal, the following sections will highlight those few studies examining heart rate, skin conductance, and child/adolescent psychopathic traits in hopes of shedding light on those particular areas in need of further investigation.

Psychopathy and Physiology in Children/Adolescents

Historically, the bulk of research examining autonomic deficiencies in serious child and adolescent behavior has focused on behavior often associated with psychopathy, such as aggression and antisocial/disruptive behavior (e.g., Beauchaine, Katkin, Strassberg & Snarr, 2001; Beauchaine, Hong, & Marsh, 2008; Lorber, 2004; Raine, Venables & Mednick, 1997). For example, one early study by Raine, Venables, and Williams (1990) found significantly lower heart rate and skin conductance activity in adolescents who were convicted of crimes during a 9-year follow up. Another longitudinal study showed that low resting skin conductance level as measured in childhood was the best predictor of poor outcome and serious antisocial behavior in adolescence, even within a group of children diagnosed with a disruptive behavior disorder (Van Bokhoven, Matthys, van Goozen, & van Engeland, 2005).

At present, there are few psychopathy-specific studies looking at atypical electrodermal and cardiovascular patterns in children and adolescents (see Table 1 for a summary). The sole study examining low resting heart rate and psychopathic traits in children and adolescents found that this pattern was associated with total psychopathy and impulsive features of psychopathy in a community sample of children and adolescents (Raine et al., 2014). Raine and colleagues indicated that their results provide further evidence for low resting heart rate as a “potential biomarker” for psychopathic traits in children (Raine et al., 2014, p. 290).

Although only one study has examined HR in children, it has been far more common for physiological research to examine the link between skin conductance and child psychopathy. For instance, one community study involving 9- to 10-year-old twins and triplets found a link between SCR hyporeactivity and higher psychopathy scores (Isen et al., 2010). In this study, electrodermal hyporeactivity (as produced by an orienting paradigm task) was associated only with the interpersonal facet of psychopathy in boys, indicating that it is a marker of manipulation and deceitfulness (Isen et al., 2010).

Using the same, large community sample, Wang and colleagues (2012) also examined psychopathic traits in 9- to 10-year-old twins and triplets using a countdown task. Results indicated that both larger heart rate acceleration and fewer non-specific skin conductance responses (NS-SCR) were significantly associated with psychopathic traits during anticipation of signaled white-noise bursts (Wang et al., 2012). However, larger HR acceleration was specific to callousness-disinhibition while reduced NS-SCR was only associated with the manipulative-deceitfulness (Wang et al., 2012). Furthermore, the negative association between the manipulative- deceitfulness factor and NS-SCR was only found in boys but not in girls (Wang et al., 2012). The authors concluded that autonomic deficits might predispose at-risk children to

later psychopathic traits.

In terms of research with adolescents, early evidence suggested that psychopathy-prone adolescents have lower SCR amplitude to an initial tone presented in orienting paradigms (Borkovec, 1970; Siddle, Nicol, & Foggitt, 1973). Later research found that 13-year-old males with psychopathic tendencies (as measured by total scores on the Psychopathy Screening Device (PSD; PCL-R; Hare, 1991) showed less skin conductance responding to distress cues and threatening stimuli (Blair, 1999), although this was an institutionalized sample with severe emotional and behavioral difficulties. Yet the boys with psychopathic traits also showed greater SCRs in response to angry faces than distress cues.

To date, it appears that only one study examined the anticipatory SCR in adolescents with psychopathic traits using the countdown paradigm. Specifically, Fung et al. (2005) reported that 16-year-old boys with high scores on the Child Psychopathy Scale (Lynam, 1997) demonstrated reduced anticipatory skin conductance and lower electrodermal responsivity to white-noise bursts. However, electrodermal hyporesponsivity was not a product of psychopathy per se but could be attributed to differences in antisocial behavior between adolescent groups. In other words, individuals who engaged in delinquent behavior were characterized by reduced responsivity, regardless of psychopathy status. Fung et al. (2005) also exclusively sampled male adolescents at risk for antisocial outcomes. Furthermore, although these adolescent findings are consistent with the adult psychopathy literature, results need to be replicated using adolescent offender samples. A summary of the studies on HR and SCR are presented in Table 1.

Table 1

Summary of Studies Investigating Psychopathy and Autonomic Processes in Youth

Study	Sample	Task	Psychopathy Scale	HR Results	SC Results
Blair, 1999	42 male children. Boarding school for emotional difficulties and a mainstream school	International Affective Picture System (IAPS): distress cues, threatening, neutral	Psychopathy Screening Device (PSD; PCL-R; Hare, 1991); teacher report	X	< SCR responsiveness to distress cues ($F(1, 46) = 3.57, p < 0.05$); > SCR responsiveness to angry face than distress cues ($F(1, 15) = 4.70, p < 0.05$)
Fung et al., 2005	335 16-year-old males. Community.	5 trial countdown	Child Psychopathy Scale (CPS; Lynam, 1997); mother report	X	SCL did not differ between psychopathy and control ($t(127) = 1.249, p = .214$). >SCR non-responsiveness to signaled anticipatory ($\chi^2(1, N = 130) = 6.044, p = .014$) >SCR non-responsiveness to signaled responsivity ($\chi^2(1, N = 130) = 4.333, p = .037$) >SCR non-responsiveness to unsignaled responsivity ($\chi^2(1, N = 130) = 8.613, p = .003$)
Isen et al., 2010	605 sets of 9-10 year-old male and female twins. Community.	3 trial orienting	CPS (Lynam, 1997); caregiver report	X	< SCR magnitude to unsignaled and nonaversive auditory stimuli in boys (b range = $-.37^*$ to $-.98^{**}$) The Manipulative/Deceitful factor predicted mean SCR magnitude after controlling for externalizing problems ($b = -0.39, t(381) = 2.90, p = .01$).
Raine et al., 2014	334 11-17-year old male and female schoolchildren.	Resting heart rate was assessed twice	APSD (Frick & Hare, 2001); caregiver report	< resting HR ($\beta = 0.06, SE = 0.02, t = 2.68, p = .01$)	X

Wang et al., 2012	843 9-10-year-old male and female twins. Community.	5 trial countdown	CPS (Lynam, 1997); caregiver report	> signaled anticipatory HR acceleration ($\beta = 0.70, SE = 0.32, t = 2.68, p = .05$); (+) with callousness-disinhibition ($\beta = 0.52, SE = 0.25, p < .05$)	< signaled anticipatory non-specific SCR ($\beta = -5.77, SE = 2.10, p < .01$); (-) with manipulative-deceitfulness in boys ($\beta = -3.69, SE = 1.76, p < .05$)
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Limitations of Existing Evidence

Aggression, Antisocial/Disruptive Behavior, and Psychopathy

Although most research appears to concur that decreased autonomic arousal is associated with child/adolescent psychopathy and psychopathy-related behavior like aggression and disruptive behavior, there is some conflicting evidence. For example, two studies have failed to demonstrate a relationship between heart rate and levels of aggression or antisocial behavior in children/ adolescents (Fowles & Furuseth, 1994; Raine & Venables, 1984a). Another study (Beauchaine et al., 2008) found that aggressive girls with conduct disorder exhibited a pattern of *increased* skin conductance levels higher levels early in baseline period, which gradually decreased. This pattern was opposite to that found in the age-matched control group. In addition, Raine and Venables (1984b) found that although skin conductance orienting response (SCOR) frequencies were negatively correlated with antisocial behavior in adolescent males, there was no association between SCOR amplitude and antisocial behavior. Further, although one study confirmed an association between lower baseline heart rate and skin conductance and aggression, the results indicated that aggression did not differentiate between conduct disordered youth and controls (Zahn & Kruesi, 1993). However, this study found that the disruptive boys (those diagnosed with Conduct Disorder (CD)/Oppositional Defiant Disorder (ODD) and/or Attention Deficit Hyperactivity Disorder (ADHD)) did have lower ANS reactivity (Zahn & Kruesi,

1993). Additionally, van Goozen et al. (1998) found that children with CD/ODD had lower baseline levels of HR and SC but higher HR levels during provocation and frustration as compared with healthy control children.

With regard to research specifically examining psychopathy, one recent study involving individuals in the community found that increased autonomic arousal and electrodermal orientating measured at age 3 were associated with higher psychopathy scores at age 28 (Glenn et al., 2007). The authors hypothesized that high arousal and orienting may reflect increased attentional processing or good prefrontal functioning (Glenn et al., 2007). These cognitive abilities may be markers of a “successful psychopath” who has avoided getting caught and convicted and who is able to appear smooth, engaging, and manipulative. Another study found that psychopathy-prone adolescent boys did not significantly differ from control participants on average SCL (Fung et al., 2005). Children with psychopathic tendencies have also been shown to demonstrate hyporesponsive SCR to distress cues relative to controls, but significantly greater SCRs in response to angry faces than distress cues (Blair, 1999.)

To date, there has only been one meta-analysis looking at the autonomic patterns involved in psychopathy, aggression, and antisocial/disruptive behavior (CD) across different age groups (Lorber, 2004). Results reflected no association between resting HR and psychopathy/ sociopathy in adults: the mean effect size of the association across 17 studies was 0.06, with a 95% confidence interval that included zero (-0.08, 0.21). There were not enough studies to look at this association in children and adolescents. Low resting HR was associated with conduct problems and aggression in children and adolescents, with a relatively large mean aggregate effect size of -0.33, with a 95% confidence interval that excluded zero (-0.23, -0.43).

There was a small association found between task HR (in response to a stimulus) and

psychopathy/sociopathy in adults, with a mean aggregate effect size of -0.16, with a 95% confidence interval that included zero (-0.41, 0.09). Task HR, however, was not associated with aggression (mean effect size = -0.02, with a 95% confidence interval that included zero (-0.15, 0.11) or conduct problems in youth (mean effect size = -0.04, with a 95% confidence interval that included zero (-0.21, 0.14). Positive associations between conduct problems and HR reactivity (change from baseline to task) were found only for negative stimuli (as opposed to non-negative stimuli) ($N_{fs} = 7$). Stimuli were coded negative if the stimuli used were designed to be anger provoking, very loud (e.g., tones > 90 dB), painful (e.g., electric shock), or otherwise aversive (e.g., pictures of mutilated body parts). Nonnegative stimuli comprised all other stimuli (e.g., orienting tones, slides of affectively neutral images, etc.). Although a positive association between conduct problems and HR reactivity was found across child and adolescent studies at a mean aggregate effect size of 0.20, the confidence interval for adolescents included zero and the lower bound of the confidence interval for child studies hovered just above zero (0.002). Thus, HR reactivity is positively associated with conduct problems in children.

Further results from Lorber's (2004) meta-analysis indicated that there was a significant relationship between low resting SC and psychopathy/sociopathy in adults, and the effect size was relatively large at -0.30 ($N_{fs} = 36$), with a 95% confidence interval that excluded zero (-0.15, -0.46). Relationships between low resting SC and psychopathy/sociopathy and aggression in children and adolescents could not be determined due to the failure to find three or more studies with this age category. There was small, negative relationship found between low resting SC and conduct disorders in youth, with a mean aggregate effect size of -0.15, with a 95% confidence interval that included zero (0.00, -0.28). There was substantial heterogeneity in effect sizes ($p < .01$), with effect sizes ranging from -1.02 to 0.89.

Although yielding a significant and large mean effect in adults ($M = 0.43$; $N_{fs} = 69$), the association between lower task EDA (in response to a stimulus) and psychopathy/ sociopathy yielded an insignificant and smaller effect in adolescents ($M = 0.07$; $Q_B(1) = 12.24, p < .01$). Effect sizes were marginally heterogeneous only in adolescents (d_+ range: 0.06 to 0.62, $p < .10$). EDA reactivity was significantly, negatively related to psychopathy/ sociopathy in adults ($M = -0.31$; $N_{fs} = 29$), with a 95% confidence interval that excluded zero (-0.48, -0.13). Data was unavailable to examine this relationship in children and adolescents, nor was there data available to investigate EDA reactivity and conduct disorders in youth.

Taking all the results into consideration, Lorber (2004) suggested that the association of conduct problems with HR, combined with a lack of any HR findings for psychopathy/ sociopathy, creates support for distinguishing between the psychopathology underlying conduct problems in childhood/adolescents and adult psychopathy/sociopathy. That is, while it appears as though autonomic activity is distinctive between aggression and psychopathy/sociopathy in adults, there is little evidence of different autonomic activity between those children with conduct problems and those children with aggression. Children with conduct disorder and those with aggression had similar resting HR patterns to that of adults with aggression and similar resting EDA patterns as psychopathic/sociopathic adults.

The Parasympathetic Nervous System (PNS)

Gaps in the literature also include a lack of research examining the role played by the parasympathetic nervous system (PNS) in psychopathic behavior. While skin conductance and heart rate are products of sympathetic influences, the heart is also subject to influence from the PNS via vagal tone. Vagal tone is a biological process involving the activity of the vagus nerve and afferent feedback and communications among various levels of the nervous system including

the cortex and amygdala (Porges, Doussard-Roosevelt, & Maiti, 1994). This tenth cranial nerve provides inhibitory input to the heart via the PNS and helps regulate metabolic output in response to environmental events. The vagus nerve generally inhibits heart activity, such that increased vagal tone slows heart rate and is associated with the ability to self-regulate, while decreased vagal tone speeds heart rate and is associated with poor self-regulation and behavioral inflexibility independent of sympathetic activity (Appelhans & Luecken, 2006; Hastings et al., 2008; Lewis, Lamm, Segalowitz, Stieben, & Zelazo, 2006; Ochsner & Gross, 2008; Porges, 2007a; Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996; Thayer & Lane, 2000).

Autonomic hyporeactivity as caused by the vagus nerve has been reliably assessed using resting levels of Respiratory Sinus Arrhythmia (RSA), which is the regular variability of heart rate caused by the influence of the vagus nerve on the sinoatrial node (Akselrod, Gordon, Snidman, Shannon, & Cohen, 1985; Beauchaine, 2001). RSA is mediated predominantly by fluctuations of vagal nerve traffic, therefore providing an acceptable, non-invasive measurement of vagal tone (Grossman & Taylor, 2007; Yasuma & Hayano, 2004). A review of physiology and emotional responding research suggested that studies examining ANS and emotional responding have “long been impeded by the exclusive use of ‘convenience measures’, such as HR and electrodermal activity” (Kreibeg, 2010, p. 409). The review recommended that given the dual activation of the heart by the SNS and PNS, HR is not informative of the respective branch’s influence upon cardiac functioning, and measures such as RSA should be preferred (Kreibeg, 2010).

Thus far, measurements of RSA in psychopathy and physiology studies have not been common. However, Raine and Venables (1984) first suggested early on that the low heart rate recorded in antisocial individuals may be a function of increased vagal tone and reflect a passive

coping response to mildly stressful situations (i.e., so-called ‘resting states’ prior to some other experimental manipulation). This suggestion reflects the traditional favoring of data demonstrating autonomic hyporeactivity in individuals with psychopathic traits. Reflecting upon Lorber’s (2004) meta-analytic results that aggression was associated with low resting HR and high HR reactivity, it is important to note that he also discussed the idea that aggression may be tied to vagal functioning. Moreover, Lorber (2004) suggested that this functioning might specifically take place in the nucleus ambiguus of the medulla, an area associated with social affective behavior (Beauchaine, 2001; Porges, 1995, 2001).

Given the dual influence of the parasympathetic and sympathetic nervous system on resting heart rate, a lower resting (baseline) heart rate could thus be a consequence of either decreased sympathetic modulation to the heart or increased cardiac vagal modulation. In addition, non-reciprocal activity in the SNS and PNS represents functional deficiencies that place individuals at risk for emotional dysregulation (Beauchaine, 2001). Although there has been no research with RSA and psychopathic traits in youth, existing evidence has linked measures of vagal tone (such as RSA) to psychopathy-related behavior. Indeed, low levels of resting (baseline) RSA and stress-induced RSA levels have traditionally been linked to child/adolescent psychological disorders and behavior problems such as aggression, depression, anxiety, self-injury, and disruptive behavior (Beauchaine, 2001; Beauchaine et al., 2007; Beauchaine et al., 2008; Beauchaine, Gatzke-Kopp, & Mead, 2007; Gordis, Feres, Olezeski, Rabkin, & Trickett, 2010; Pine et al., 1996; Shannon et al., 2007; Silk, Steinberg, & Morris, 2003; Thayer & Lane, 2000).

One study conducted by Beauchaine and colleagues (2007) also found developmental differences in RSA activity and disruptive behavior. The initial results indicated that

preschoolers, middle school children and adolescents either with aggressive ODD and CD or at risk for CD all demonstrated decreased SNS-linked cardiac activity (HR) at baseline. However, decreased (PNS-linked) baseline RSA and RSA reactivity was found for middle school and adolescent males with aggressive ODD/CD, but not for preschool children at risk for conduct problems. The authors concluded that decreased RSA in older children with aggressive ODD/CD appear to develop sometime between the preschool and the middle school years. Beauchaine and colleagues (2007) further proposed a biosocial developmental model for conduct problems, whereas impulsive children develop effective vagal modulation of emotion if they experience an environment promoting emotion regulation skills such as arousal de-escalation, positive reinforcement of prosocial behaviors, and consequences for aggressive behavior. These children thus resist developing CD. On the other hand, impulsive children raised within an emotionally-dysregulated environment, where arousal and aggression is negatively reinforced, may develop poor vagal modulation of emotion, and subsequently, CD and delinquency (Beauchaine et al., 2007).

To date, there appears to be only one study examining adolescent antisocial behavior and both sympathetic and parasympathetic activity. However, the results of this study were contrary to expectations of decreased SNS and increased PNS activity. Using measurements of heart rate variability, Mezzacappa and colleagues (1997) indicated that antisocial 15-year-old boys were characterized by low resting heart rates (SNS), but demonstrated *reduced* vagal functioning (PNS). Given that reduced vagal control is usually reflective of increased heart rate, the authors note that sympathetic influences would have to substantially decrease in order for heart rate to decrease, as was found in the study. Mezzacappa and co-investigators (1997) further indicate that this indirectly supports the theory that conduct disorders are associated with reduced

noradrenergic function. However, this study did not focus on psychopathy specifically, and as can be seen from the literature reviewed above the findings related to ANS activity and child/adolescent psychopathy are mixed.

Current Study

Given the need for increased research examining neurobiological correlates of adult psychopathy it is also important to determine if those same correlates can be found in adolescents. Previous adult research appears to have established a relationship between heart rate, skin conductance, and psychopathy (e.g., Arnett, 1997; Gao et al., 2009) but there is a dearth of evidence highlighting this relationship in adolescent offenders. Given that psychopathic traits are traditionally more prevalent in custodial settings a larger effect may be found by examining this psychological/physiological relationship in adolescents who have been detained for criminal charges. Researchers have also yet to examine both sympathetic (heart rate and skin conductance) and parasympathetic (vagal tone) nervous system associations with psychopathy in adolescent offenders, and it may be that using a criminally antisocial sample will allow for a narrower focus on different psychopathic facets related to autonomic processes such as interpersonal, affective, and lifestyle (specifically, stimulation-seeking) traits. Low anxiety and fearfulness (LAF) have also been identified as important facets to consider in examinations of psychopathy and physiology. However, a recent confirmatory analysis and structural equation modeling has indicated that LAF is not separate from the other four aspects of the PCL-R (Neumann, Hare, & Johansson, 2013). Seeing as LAF has been reported as remaining subsumed under the general PCL psychopathy construct, it will not be separately investigated in this current study (Neumann et al., 2013).

The literature further indicates conflicting information regarding the relationship between

the autonomic nervous system and psychopathy in children and adolescents (Beauchaine et al., 2007; Glenn et al., 2007; Mezzacappa et al., 1997), thus necessitating further clarification using an adolescent offender sample, rather than a traditional focus on community members. This dissertation project explores the under-investigated relationship between heart rate, vagal tone (as indexed by RSA), skin conductance, and psychopathy in adolescent offenders where the concentration of psychopathic traits are thought to be higher using a white noise and countdown paradigm.

Hypotheses

Hypothesis one. Based on Raine's conclusion (1993; 2002; et al., 2014) that low resting heart rate is the best-replicate biological correlate of antisocial behavior in children and adolescents, it is expected that decreased baseline heart rate and RSA patterns will be associated with, and predict, higher psychopathy scores. Given the association between attenuated baseline electrodermal activity and adolescent disruptive behaviors (e.g., Lorber, 2004; van Goozen et al., 1998; van Goozen et al., 2005) decreased baseline SCL should also be positively associated with, and predict, psychopathic behavior.

Hypothesis two. In line with results from adult and childhood studies (e.g., Dindo & Fowles, 2011; Fung et al., 2005; Ogloff & Wong, 1990; Wang et al., 2012), it is expected that increased HR scores and decreased SCR in anticipation of the white-noise stimuli during the signaled trials will be associated with, and predict, higher psychopathy scores.

Hypothesis three. Lower HR scores and decreased SCR in response to the white-noise stimuli (reactivity) during the signaled trials are expected to be positively associated with, and predict, psychopathy scores. This prediction reflects similar patterns found in prior studies with adults, children, and adolescents (e.g., Blair, 1999; Fung et al., 2005; Gao & Raine, 2010; Isen et

al., 2010).

Hypothesis four. Given that autonomic hyporeactivity in response to aversive stimuli has been suggested to specifically reflect the interpersonal (Fowles, 1993; Patrick, 1994) facet of psychopathy, hypotheses 1 through 3 are expected to have greater associations with the Interpersonal- Affective factor of the two-factor psychopathy model, and the Interpersonal facet of the four-factor psychopathy model. All analyses controlled for aggression in light of the extensive evidence linking ANS processes to aggressive behaviors (e.g., Beauchaine, Katkin, Strassberg & Snarr, 2001; Beauchaine, Hong, & Marsh, 2008; Lorber, 2005; Raine, Venables & Mednick, 1997).

2. METHODOLOGY

Participants

Participants were adolescent offenders recruited from the Tuscaloosa County Juvenile Detention Center and Juvenile Court, as part of a study examining multi-method assessments of the Risk-Sophistication-Treatment Inventory Abbreviated (RSTI-A; Salekin, 2011) and a brief intervention. The adolescent sample consists of 56 males aged 13-18 years old ($M = 15.91$). The participants' race was primarily Black ($N = 46$; 82.1%), followed by White ($N = 9$; 16.1%) and Bi-racial ($N = 1$; 1.8%). Typical charges for this sample include, but are not limited to robbery, burglary, domestic violence, possession of drugs, disorderly conduct, truancy, criminal mischief, and auto theft. These charges represent offenses that either led to the youths' incarceration or required the youths' appearance in court.

Procedure

IRB approval was obtained before the study was initiated. Two graduate students, including this author, trained eight undergraduate research assistants (RAs) to aid with recruiting participants. Of these undergraduate RAs, four senior students also received extensive training from the graduate students in administering the psychological and physiological measures. Before testing began, informed consent was obtained from parents and assent from youth who participated in the study. Participants were informed that they were free to discontinue testing for any reason and at any time during the session. They were also informed that their participation withdrawal would in no way affect their placement at the detention center or their legal situation.

Those who agreed to participate were recruited from the Tuscaloosa County Juvenile Detention Center and completed testing procedures in a private, confidential room in the facility. Those youth recruited from the Tuscaloosa County Juvenile Court participated in the testing session either in a quiet, private space within their home or in a private area at a nearby library, depending on the youths' preferences. Youth completed all measures in one testing session, taking breaks between tasks when necessary. Youth who indicated difficulty with reading were read items aloud. The average room temperature across all testing sessions was 71.9 degrees Fahrenheit and the average relative humidity was 57.0 %. This was indexed with a hand held combination temperature/ humidity gauge.

Measures

Physiological Data Recorder

A BioLog™ recorder was administered to measure heart rate and skin conductance. Researchers have extensively used this ambulatory data logger to investigate issues in children such as generalized anxiety disorders, motion sickness, and aggression (Hubbard et al., 2002; Muth & Elkins, 2007; Rotha et al., 2008). Before the recording began, participants were notified they would be hooked up to a machine to view and measure their heart rate and skin response, and that the machine would not hurt. Two skin conductance electrodes (silver/ silver chloride) were filled with Biogel (UFI; Morro Bay, CA) cream, an electrolyte contact medium, and attached to the index and middle finger of the participants' non-dominant hand using adhesive tape. Three heart electrodes were also placed on the inside of the participants' left, inner knee, just above the right collarbone, and on the right side of the neck. A white noise countdown paradigm was presented, modeled after a similar task used by Fung and colleagues (Fung et al., 2005), and Iacono and colleagues (Iacono, 1998; Taylor, Carlson, Iacono, Lykken, & McGue,

1999). The paradigm consists of four trials; each trial recorded HR, RSA, and SCL patterns during the 3-minute baseline period, and HR and SCR patterns during a 12-s anticipatory period, and a 20-s reactivity period.

In order to collect baseline HR, RSA, and SC information, participants were instructed to remain still for three minutes while fixating on a black dot against a white background displayed on a computer monitor positioned 1 m in front of them. Physiological recording was then paused while participants were administered several personality questionnaires. Following the completing of these measures, participants were again instructed to observe the computer monitor while wearing headphones. They were also given the following instructions:

In this situation you will see numbers counting down on the computer screen from 12 to 0. One number will appear every second. When you see the number 0, you will hear a loud noise for 1 second (s). Sometimes this loud noise will come on without any warning, however. There is nothing you need to do in this task apart from keeping your head and body as still as you can. Do you have any questions?

Heart rate and skin conductance recording then resumed with a 12-s resting period where the participants were instructed to observe the computer monitor while still wearing headphones. In Trials 1 and 3 (signaled trials), the anticipatory period included a numeric countdown running from 12 to 0. The countdown was displayed in the center of the screen at the rate of one number per second. When 0 appeared, a 1-s burst of 90-dB white noise with a 50-ms rise and fall time was heard through the headphones. The reactivity period encompassed the 20 seconds immediately following the noise burst. On Trials 2 and 4 (un-signaled trials), the countdown period did not include a visually displayed numeric countdown. The inter-trial interval was approximately 45 seconds, and participants were not made aware of the number of trials or the

alternating nature of the trials. The computer-generated white noise burst was consistently kept at 90-dB across all trials and participations. DSP Mobile, an iPhone, evaluated the white noise burst for intensity using dB Volume® app found to accurately evaluate noise conditions under 95-dB (Keene et al., 2013).

Scoring of physiological data recorder. For the purpose of this study, HR mean, RSA mean, and SCL mean were calculated in the baseline condition. HR mean and SCR frequency and amplitude were calculated within two periods across four trials: (a) Trial 1, signaled anticipatory and signaled reactivity, (b) Trial 2, un-signalized anticipatory and un-signalized reactivity, (c) Trial 3, signaled anticipatory and signaled reactivity, and (d) Trial 4, un-signalized anticipatory and un-signalized reactivity.

HR and RSA were derived from techniques in the manual *Inter-Beat-Interval Editing for Heart Period Variability Analysis: An Integrated Training Program with Standards for Student Reliability Assessment* (Porges, 2007b). This manual was designed for use alongside the CardioEdit and CardioBatch computer programs developed by Dr. Stephen Porges, director of the Brain and Body Center at the University of Illinois. Porges' vagal tone method of calculating RSA is empirically supported (Denver, Reed, & Porges, 2007; Grossman, VanBeek, & Wientjes, 1990; Porges, 2007a).

The first procedure involved cleaning interbeat interval data collected using the Biolog. As per procedures outlined in the manual, each participant's heart rate data was hand edited using the CardioEdit program in order to remove any unwanted artifacts. Artifacts are errors in the interbeat interval data that are likely due to the digitizing process of the data or to physiological anomalies. After data cleaning, the mean heart rate level was calculated using the mean lengths of the interbeat intervals for each baseline, anticipatory, and reactivity period. For

the baseline period, RSA was also extracted from one of the predominant rhythms exhibited in the data via computations of the participant's heart period series using the CardioBatch computer software. Mean RSA was quantified for sequential 30 s epochs, and HR data was quantified in inter-beat intervals (IBIs).

SCL baseline data was provided by the Biolog recorder computer software output. Similar to the interbeat interval data, the first step involved examining SCL data for any unwanted artifacts. Any unusual patterns were hand-edited conservatively. Each SCL pattern within a baseline period was then averaged across the 3-minute period (e.g., Dindo & Fowles, 2011).

SCR elicited during the anticipatory and reactivity periods was extracted using Ledalab, a Matlab-based software that uses "Discrete Deconvolution Analysis" (DDA) and "Continuous Deconvolution Analysis" (CDA) to examine skin conductance data (see Benedek & Kaernbach, 2010a and Benedek & Kaernbach, 2010b for technical details). Skin conductance data obtained from the Biolog recorder computer software output was formatted accordingly prior to Ledalab analyses. SC data was sampled at a frequency of 10 hertz (Hz). A rise in SCL was judged a response if it occurred within a window of 1-4 s after stimulus onset. A skin conductance response (SCR) was defined as an increase in conductivity exceeding 0.05 microsiemens (μS 10–6 s) in amplitude and SCR data were provided by the Ledalab output. SCRs were sampled within 4 s prior to (anticipatory period) and after (reactivity period) the white noise. In consideration of recommendations from Dawson, Schell, and Filion (2001), both SCR frequency and amplitude data was examined.

Psychopathy Checklist-Youth Version (PCL:YV; Forth, Kosson, & Hare, 2003)

Modeled after the Psychopathy Checklist-Revised (PCL-R; Hare, 2003), the PCL:YV is a

20-item interview based assessment of psychopathic traits in youth 12-18 years of age. Each item is scored on a 3-point Likert scale from 0 (no) to 2 (yes) based on the degree to which behavior matches item descriptions. The PCL:YV was coded from interview questions and file information. Two- and four-factor models include interpersonal, affective, and lifestyle facets, while the four-factor model also includes an antisocial facet. Both the PCL-R and the PCL:YV have studies to show high internal consistency indices (e.g., Forth et al., 2003; Gretton, Hare, & Catchpole, 2004; Salekin et al., 2004). The PCL:YV has also been found to have high inter-rater reliability of .90 or above (e.g.; Forth et al., 2003; Salekin et al., 2004).

Peer Conflict Scale (PCS; Marsee & Frick, 2007)

The PCS is a 40-item self-report measure of reactive (20 items) and proactive (20 items) aggression. The reactive aggression subscale contains 10 reactive overt items and 10 reactive relational items, while the proactive aggression subscale includes 10 proactive overt items and 10 proactive relational items. Items are scored on a 4-point Likert scale from 0 (not at all true) to 3 (definitely true). There is empirical support for the distinction between the reactive and proactive, and relational and overt PCS scales as well as the relational and overt scales (Barry, Grafeman, Adler, & Pickard, 2007; Marsee & Frick, 2007; Marsee et al., 2011; Munoz, Frick, Kimonis, & Aucoin, 2008). Confirmatory factor analyses indicate an appropriate 4-factor model fit for both males and females and across high school, detained, and residential samples (Marsee et al., 2011). Internal consistency estimates have also been deemed satisfactory for the 4 factors (Marsee et al., 2011).

3. DATA ANALYTIC PLAN

The first step of analyses included running descriptive statistics of the PCL:YV, PCS, and physiological measures of psychopathy in the total sample. Analyses were also performed to ensure no violations of normality, linearity, multicollinearity, and homoscedasticity. The reliability of the PCL:YV and PCS were calculated using Cronbach's alpha (α). Inter-rater reliability of PCL:YV ratings were calculated using intraclass correlation coefficients (ICC) with a two-way random effects model with consistency agreement (McGraw & Wong, 1996).

In consideration of the ties between externalizing behavior (particularly, aggression and antisocial behavior) and autonomic arousal (e.g., Gordis et al., 2010; Raine, 2002), and research indicating that age can co-vary between ANS processes (e.g., Lorber, 2004), correlational analyses were conducted to determine whether reactive and proactive aggression and age were related to psychopathy and the physiological measures.

Next, correlation analyses were performed to examine the relationship between total facet/factor scores of the PCL:YV and the following variables: HR, RSA, and SCL baseline scores, HR and SCR anticipatory scores, and HR and SCR reactivity scores. A ratio of the PCL:YV F1 and F2 scores and HR/RSA was also used in the analysis, seeing as non-reciprocal activity in the SNS and PNS is indicative of pathology (Beauchaine, 2001). HR/SCR anticipatory and reactivity variables were correlated with PCL:YV scores across the three signaled trials and

two un-signalized white noise trials. Partial correlations were run in order to control for PCS Reactive and Proactive Aggression scores. Partial correlations using just the PCL:YV total scores were also run in order to control for PCS Reactive, PCS Proactive, and PCL:YV Antisocial facet 4 scores. Given that Fung and colleagues (2005) and Raine and Venables (1984) used SCR frequency to dichotomize participants into “responders” and “non-responders,” additional analyses were run. Participants were categorized as non-responders if they did not exhibit an SCR in any trial (i.e., total number of SCRs = 0) ($n = 26$; 46.4%), whereas participants were labeled as “responders” if they demonstrated at least one SCR in at least one trial (i.e., total number of SCRS > 1) ($n = 30$; 53.6%). A one-way multivariate analysis of covariance (MANCOVA) was conducted to compare PCL:YV scores between responders and non-responders, controlling for PCS Reactive and Proactive subscale scores. A one-way univariate analysis of covariance (ANCOVA) was also conducted to only compare PCL:YV total scores between responders and non-responders, controlling for PCS Reactive and Proactive subscale scores and PCL:YV Antisocial facet 4 scores.

Regression models were used to further evaluate whether psychopathy scores on the PCL:YV can be predicted by HR, RSA, and SCL baseline scores; RSA and SCR anticipatory scores; and RSA and SCR reactivity scores in each of the three signaled and two un-signalized trials. Both the three-factor and four-factor models of the PCL:YV were included in all analyses. For analyses involving all PCL:YV facets and factors, PCS Reactive and Proactive Aggression scores were entered on the first block, and the physiological data were entered simultaneously on the second block. For analyses only involving the PCL:YV total score, PCS Reactive, PCS Proactive, and PCL:YV Antisocial facet 4 scores were entered on the first block, and the physiological data were entered simultaneously on the second block

4. RESULTS

Preliminary Analyses

For descriptive statistics of the PCL:YV, the PCS, and physiological measures, please refer to Table 2. The internal consistency of the PCL:YV and PCS was deemed adequate and excellent, respectively, using Cronbach's alpha (PCL:YV $\alpha = 0.69$, PCS $\alpha = 0.93$). The interrater reliability analysis revealed ICCs between 0.71 and 0.81 (see Table 3), which is within the range of good to excellent (Cicchetti & Sparrow, 1981; Fleis, 1981). Evaluation of the normality assumption using Kolmogorov-Smirnov testing revealed non-significant results for HR, RSA, and SCL data, suggesting that the null hypothesis for a normal distribution of these variables can be rejected. However, the Kolmogorov-Smirnov statistic for SCR amplitude and frequency values, and PCL:YV and PCS total scores, revealed significant results, indicating that the null for a normal distribution of these variables cannot be rejected.

Tests of skewness and kurtosis revealed that the distribution of both SCR values and PCS total scores was positively skewed and leptokurtic, with data clustering at the low end of values and scores. The distribution of PCL:YV total scores was negatively skewed with negative kurtosis values, indicating a clustering of scores at the high end with too many scores in the extremes. Given that it is common for SCR amplitude and frequency values to be positively skewed and leptokurtic it is generally recommended that SCR values be transformed using log or square root transformations (Boucsein, 2012; Braithwaite, Watson, Jones, & Rowe, 2013; Dawson, Schell, & Fillion, 2001). Given that both corrections produce roughly comparable results (Dawson et al. 2001), both log transformations and square root transformations were used to correct the distribution of SCR

frequency and amplitude values. Results revealed that neither of these transformations was sufficient to normalize the distribution of SCR values.

Correlational analyses to determine the relationship between aggression, age, and the psychopathy/physiological scores indicated that age was not significantly related to any variable. However, PCS proactive and reactive aggression scores were related to several PCL: YV factors and facets (see). Thus, aggressive and antisocial behavior was controlled for in all subsequent analyses using scores from the PCS and the PCL: YV Antisocial facet 4.

Table 2
Study Variable Descriptive Statistics, Including Total and Subscale Scores

Scale	Mean	SD	N	Skewness	Kurtosis
RSA Baseline	7.34	1.05	56	-.82	1.50
HR Baseline	69.58	10.53	56	.49	.27
HR Trial 1 Anticipatory	68.17	10.01	56	.38	.96
HR Trial 1 Reactivity	68.60	10.73	56	.27	.16
HR Trial 2 Anticipatory	68.17	9.83	56	.41	.90
HR Trial 2 Reactivity	70.02	11.34	56	.42	.09
HR Trial 3 Anticipatory	68.80	11.42	56	.68	1.01
HR Trial 3 Reactivity	68.91	10.72	56	.58	.71
HR Trial 4 Anticipatory	68.94	10.48	56	.41	.56
HR Trial 4 Reactivity	69.73	10.61	56	.39	-.104
SCL Baseline	4.99	3.60	56	.44	-1.04
SCR Trial 1 Anticipatory Frequency	.23	.43	56	1.30	-.31
SCR Trial 1 Reactivity Frequency	.20	.40	56	1.57	.48
SCR Trial 2 Anticipatory Frequency	.14	.40	56	2.93	8.68
SCR Trial 2 Reactivity Frequency	.09	.34	56	4.22	18.82

SCR Trial 3 Anticipatory Frequency	.09	.29	56	2.96	7.01
SCR Trial 3 Reactivity Frequency	.12	.38	56	3.27	11.05
SCR Trial 4 Anticipatory Frequency	.12	.33	56	2.33	3.56
SCR Trial 4 Reactivity Frequency	.14	.40	56	2.93	8.68
SCR Trial 1 Anticipatory Amplitude	.08	.22	56	3.20	9.30
SCR Trial 1 Reactivity Amplitude	.06	.21	56	4.76	23.98
SCR Trial 2 Anticipatory Amplitude	.08	.47	56	7.22	53.17
SCR Trial 2 Reactivity Amplitude	.02	.14	56	7.35	54.62
SCR Trial 3 Anticipatory Amplitude	.02	.13	56	7.31	54.10
SCR Trial 3 Reactivity Amplitude	.01	.05	56	5.86	37.32
SCR Trial 4 Anticipatory Amplitude	.02	.10	56	6.00	38.93
SCR Trial 4 Reactivity Amplitude	.01	.07	56	6.81	48.71
PCL:YV	20.86	5.05	56	-.14	-.64
Interpersonal	3.40	1.45	56	-.14	-.53
Affective	4.04	1.43	56	-.30	.46
Lifestyle	5.64	1.57	56	-.38	.15
Antisocial	5.02	2.09	56	-.16	-.87
Interpersonal-Affective	8.41	2.51	56	-.16	-.46
Lifestyle-Behavioral	9.82	2.49	56	-.09	-.41
PCS	20.86	5.05	56	2.24	7.77
Reactive	12.14	9.80	56	1.29	2.20
Reactive-Overt	9.46	7.27	56	.69	-.46
Reactive-Relational	2.68	3.82	56	2.86	11.34
Proactive	5.14	7.23	56	3.22	14.51
Proactive-Overt	3.25	4.42	56	2.25	6.45
Proactive-Relational	1.89	3.30	56	3.88	20.14

Note: SD = standard deviation, N = sample size

RSA Baseline= Respiratory Sinus Arrhythmia Baseline Period, HR= Heart Rate Level Baseline Period, HR Trial 1 Anticipatory= Heart Rate Trial 1 Anticipatory Period, HR Trial 1 Reactivity= Heart Rate Trial 1 Anticipatory Period, HR Trial 2 Anticipatory= Heart Rate Trial 2 Anticipatory Period, HR Trial 2 Reactivity= Heart Rate Trial 2 Anticipatory Period, HR Trial 3 Anticipatory= Heart Rate Trial 3 Anticipatory Period, HR Trial 3 Reactivity= Heart Rate Trial 3 Anticipatory Period, HR Trial 4 Anticipatory= Heart Rate Trial 4 Anticipatory Period, HR Trial 4 Reactivity= Heart Rate Trial 4 Anticipatory Period, SCL Baseline= Skin Conductance Baseline Level, SCR Trial 1 Anticipatory Frequency= Skin Conductance Response Frequency Trial 1 Anticipatory Period, SCR Trial 1 Reactivity Frequency= Skin Conductance Response Frequency Trial 1 Reactivity Period, SCR Trial 2 Anticipatory Frequency= Skin Conductance Response Frequency Trial 2 Anticipatory Period, SCR Trial 2 Reactivity Frequency= Skin Conductance Response Frequency Trial 2 Reactivity Period, SCR Trial 3 Anticipatory Frequency= Skin Conductance Response Frequency Trial 3 Anticipatory Period, SCR Trial 3 Reactivity Frequency= Skin Conductance Response Frequency Trial 3 Reactivity Period, SCR Trial 4 Anticipatory Frequency= Skin Conductance Response Frequency Trial 4 Anticipatory Period, SCR Trial 4 Reactivity Frequency= Skin Conductance Response Frequency Trial 4 Reactivity Period, SCR Trial 1 Anticipatory Amplitude= Skin Conductance Response Amplitude Trial 1 Anticipatory Period, SCR Trial 1 Reactivity Amplitude= Skin Conductance Response Amplitude Trial 1 Reactivity Period, SCR Trial 2 Anticipatory Amplitude= Skin Conductance Response Amplitude Trial 2 Anticipatory Period, SCR Trial 2 Reactivity Amplitude= Skin Conductance Response Amplitude Trial 2 Reactivity Period, SCR Trial 3 Anticipatory Amplitude= Skin Conductance Response Amplitude Trial 3 Anticipatory Period, SCR Trial 3 Reactivity Amplitude= Skin Conductance Response Amplitude Trial 3 Reactivity Period, SCR Trial 4 Anticipatory Amplitude= Skin Conductance Response Amplitude Trial 4 Anticipatory Period, SCR Trial 4 Reactivity Amplitude= Skin Conductance Response Amplitude Trial 4 Reactivity Period, PCL: YV= Psychopathy Check-List: Youth Version Total Score, Interpersonal= Psychopathy Check-List: Youth Version Interpersonal Facet, Affective= Psychopathy Check-List: Youth Version Affective Facet, Lifestyle= Psychopathy Check-List: Youth Version Lifestyle Facet, Antisocial= Psychopathy Check-List: Youth Version Antisocial Facet, Interpersonal-Affective= Psychopathy Check-List: Youth Version Interpersonal-Affective Factor, Lifestyle-Behavioral= Psychopathy Check-List: Youth Version Lifestyle-Behavioral Factor, PCS= Peer Conflict Scale Total Score, Reactive= Peer Conflict Scale Reactive Subscale, Reactive-Overt= Peer Conflict Scale Reactive-Overt Subscale, Reactive-Relational= Peer Conflict Scale Reactive-Relational Subscale, Proactive= Peer Conflict Scale Proactive Subscale, Proactive-Overt= Peer Conflict Scale Proactive-Overt Subscale, Proactive-Relational= Peer Conflict Scale Proactive-Relational Subscale.

Table 3
Inter-rater Reliability of the PCL: YV Ratings

PCL: YV	ICC (% agreement)
Facet 1	0.74 (74%)
Facet 2	0.71 (71%)
Facet 3	0.81 (81%)
Facet 4	0.78 (78%)
Factor 1	0.73 (73%)
Factor 2	0.81 (81%)
Total Score	0.78 (78%)

Note: n = 10

Correlation Analyses

Evidence suggests that low baseline HR, RSA, and SCL patterns are associated with disruptive and aggressive behavior (Lorber, 2004; Raine, 2014; van Goozen et al., 1998; van Goozen et al., 2005), and Pearson product-moment partial correlations were first used to examine the relationship between psychopathy and baseline HR, RSA, and SCL, controlling for aggression and antisocial behaviour (see Table 5). Results indicated that only SCL baseline period was significantly related to a PCL:YV facet, specifically, the Interpersonal facet ($r = .27$, $p = .05$) and to the PCL:YV total score ($r = .27$, $p = .05$) (please see Table 5).

Based on adult and childhood studies, it was anticipated that increased HR scores and decreased SCR during signaled anticipatory periods, and attenuated HR and SCR activity during

signaled reactivity periods would be associated with increased psychopathy scores (e.g., Blair, 1999; Dindo & Fowles, 2011; Fung et al., 2005; Gao & Raine, 2010; Isen et al., 2010; Ogloff & Wong, 1990; Wang et al., 2012). Results examining HR mean during the anticipatory and reactivity periods revealed no significant associations (see Table 5). Given that log and square root transformations of SCR frequency and amplitude values remained insufficient to normalize the distribution of SCR values, a Spearman's rank-order correlation was used to evaluate the association between SCR frequency and amplitude values and PCL: YV facets and factors. As seen in Table 6, no significant Spearman's rho coefficients were found after the log transformation.

Table 4

Bivariate Pearson Product-Moment Correlations Between the PCL: YV and PCS, Including Total and Subscale Scores

Scale	PCL : YV	F1	F2	F3	F4	F-1	F-2	PCS	RE	R-O	R-R	PA	P-O	P-R
PCL: YV	--													
F1	.70* *	--												
F2	.31*	.15	--											
F3	.67* *	.39* *	-.03	--										
F4	.65* *	.27*	-.21	.37* *	--									
F-1	.68* *	.76* *	.72* *	.23	.04	--								
F-2	.78* *	.37* *	-.07	.81* *	.80* *	.18	--							
PCS	.33*	.23	.11	.14	.23	.24	.28*	--						
RE	.33*	.26*	.08	.15	.25	.23	.29*	.96* *	--					
R-O	.38* *	.29* *	.06	.16	.34* *	.22	.36* *	.86* *	.94* *	--				
R-R	.14	.12	.09	.07	-.01	.16	.07	.82* *	.77* *	.51* *	--			
PA	.29*	.15	.14	.12	.17	.22	.22	.92* *	.78* *	.64* *	.77* *	--		
P-O	.39* *	.22	.15	.18	.24	.27*	.30*	.91* *	.80* *	.72* *	.67* *	.95* *	--	
P-R	.12	.04	.10	.02	.05	.12	.08	.80* *	.64* *	.44* *	.80* *	.92* *	.75* *	--

Note: ** Indicates correlation is significant at the .01 level; * Indicates correlation is significant at the .05 level

PCL: YV= Psychopathy Check-List: Youth Version Total Score, F1= Psychopathy Check-List: Youth Version Interpersonal Facet, F2= Psychopathy Check-List: Youth Version Affective Facet, F3= Psychopathy Check-List: Youth Version Lifestyle Facet, F4= Psychopathy Check-List: Youth Version Antisocial Facet, F-1= Psychopathy Check-List: Youth Version Interpersonal-Affective Factor, F-2= Psychopathy Check-

List: Youth Version Lifestyle-Behavioral Factor, PCS= Peer Conflict Scale Total Score, RE= Peer Conflict Scale Reactive Subscale, R-O= Peer Conflict Scale Reactive-Overt Subscale, R-P= Peer Conflict Scale Reactive-Relational Subscale, PA= Peer Conflict Scale Proactive Subscale, P-O= Peer Conflict Scale Proactive-Overt Subscale, P-R= Peer Conflict Scale Proactive-Relational Subscale.

Table 5

Partial Pearson Product Moment Correlations Between the PCL: YV, HR, RSA, and SCL data, Including Total and Subscale Scores, Controlling for PCS Reactive and Proactive Subscale Scores and PCL:YV Facet 4 Scores

Scale	PCL: YV Total Score	F1	F2	F3	F3	F-1	F-2	F-1/ F-2 Ratio
Skin Conductance Level Baseline	.21 (.27*)	.27*	.11	.15	-.01	.20	.14	.11
Respiratory Sinus Arrhythmia Baseline	-.05 (.08)	.09	.19	.02	-.18	.14	-.13	.19
Heart Rate Baseline	-.01 (-.03)	-.07	-.09	.07	.03	-.10	.09	-.11
RSA/HR Ratio	.02 (.10)	.13	.21	-.05	-.10	.20	-.13	.22
Heart Rate Trial 1 Anticipatory	.03 (-.01)	-.01	-.11	.18	.06	-.07	.19	-.18
Heart Rate Trial 1 Reactivity	.02 (-.00)	-.01	-.05	.09	.03	-.03	.12	-.09
Heart Rate Trial 2 Anticipatory	-.06 (-.13)	-.15	-.17	.10	.06	-.22	.13	-.21
Heart Rate Trial 2 Reactivity	0 (-.07)	-.07	-.11	.06	.08	-.11	.10	-.14
Heart Rate Trial 3 Anticipatory	.06 (-.02)	-.01	-.11	.09	.12	-.05	.16	-.16
Heart Rate Trial 3 Reactivity	0 (-.08)	-.03	-.20	.15	.10	-.14	.18	-.22
Heart Rate Trial 4 Anticipatory	-.02 (-.09)	-.01	-.25	.14	.09	-.15	.16	-.23
Heart Rate Trial 4 Reactivity	.04 (.00)	.02	-.16	.15	.06	-.06	.17	-.16
Average HR Trial 1-4 Anticipatory	.00 (-1.06)	-.05	-.16	.13	.08	-.12	.16	--

Average HR Trial 1-4 Reactivity	.02 (-.04)	-.02	-.13	.11	.07	-.09	.57	--
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Note: ** Indicates correlation is significant at the .01 level; * Indicates correlation is significant at the .05 level

The values in parentheses represent the PCL: YV total scores controlled for using scores from the PCS and the PCL: YV Antisocial facet 4. PCL: YV Total Score= Psychopathy Check-List: Youth Version Total Score, F1= Psychopathy Check-List: Youth Version Interpersonal Facet, F2= Psychopathy Check-List: Youth Version Affective Facet, F3= Psychopathy Check-List: Youth Version Lifestyle Facet, F4= Psychopathy Check-List: Youth Version Antisocial Facet, F-1= Psychopathy Check-List: Youth Version Interpersonal-Affective Factor, F-2= Psychopathy Check-List: Youth Version Lifestyle-Behavioral Factor, Respiratory Sinus Arrhythmia Baseline = Respiratory Sinus Arrhythmia Baseline Period, Heart Rate Baseline = Heart Rate Level Baseline Period, Heart Rate Trial 1 Anticipatory= Heart Rate Trial 1 Anticipatory Period, Heart Rate Trial 1 Reactivity = Heart Rate Trial 1 Reactivity Period, Heart Rate Trial 2 Anticipatory= Heart Rate Trial 2 Anticipatory Period, Heart Rate Trial 2 Reactivity = Heart Rate Trial 2 Reactivity Period, Heart Rate Trial 3 Anticipatory = Heart Rate Trial 3 Anticipatory Period, Heart Rate Trial 3 Reactivity= Heart Rate Trial 3 Reactivity Period, Heart Rate Trial 4 Anticipatory= Heart Rate Trial 4 Anticipatory Period, Heart Rate Trial 4 Anticipatory = Heart Rate Trial 4 Anticipatory Period, Average HR Trial 1-4 Anticipatory = Average Heart Rate Trial 1-4 Anticipatory Period, Average HR Trial 1-4 Reactivity = Average Heart Rate Trial 1-4 Reactivity Period, SCL= Skin Conductance Baseline Level.

Table 6

Bivariate Spearman's Rank Order Correlations Between the PCL: YV and SCR Data Including Total and Subscale Scores

Scale	PCL: YV Total	F1	F2	F3	F4	F-1	F-2
SCR Trial 1 Anticipatory Frequency	.02 (.02)	.11 (.10)	-.01 (.05)	.18 (.18)	-.01 (-.01)	.03 (.03)	.12 (.13)
SCR Trial 1 Reactivity Frequency	.12 (.10)	.12 (.13)	-.06 (-.04)	.17 (.17)	.07 (.06)	-.02 (-.01)	.13 (.15)
SCR Trial 2 Anticipatory Frequency	.12 (-.10)	.12 (.19)	-.06 (-.10)	.17 (.08)	.07 (-.05)	-.02 (.03)	.13 (.04)
SCR Trial 2 Reactivity Frequency	.12 (.09)	.02 (.01)	.04 (.03)	-.04 (-.01)	.07 (.10)	.05 (.02)	.08 (.13)
SCR Trial 3 Anticipatory Frequency	-.17 (-.19)	.01 (.01)	-.22 (-.19)	.02 (.03)	-.08 (-.09)	-.15 (-.13)	-.05 (-.03)
SCR Trial 3 Reactivity Frequency	.09 (-.01)	.04 (-.02)	.22 (.19)	.19 (.14)	-.09 (-.14)	.08 (.04)	.10 (.04)
SCR Trial 4 Anticipatory Frequency	.13 (.15)	.19 (.17)	-.17 (-.12)	.17 (.19)	.15 (.13)	.02 (.02)	.20 (.23)
SCR Trial 4 Reactivity Frequency	.15 (.15)	.20 (.19)	-.10 (-.10)	.16 (.11)	.13 (.15)	.06 (.05)	.15 (.12)
Average SCR Frequency Trial 1-4 Anticipatory	.03 (-.12)	.11 (.12)	-.12 (-.09)	.39 (.48)	.03 (-.01)	-.03 (-.01)	0.1 (.09)
Average SCR Frequency Trial 1-4 Reactivity	0.12 (.33)	0.10 (.10)	0.03 (.02)	0.12 (.41)	.05 (.04)	0.04 (0.03)	0.12 (0.11)

SCR Trial 1 Anticipatory Amplitude	.03 (.11)	.14 (.16)	-.04 (-.12)	.18 (.16)	.03 (.16)	.02 (-.05)	.13 (.18)
SCR Trial 1 Reactivity Amplitude	.13 (.00)	.14 (.11)	-.06 (-.15)	.17 (.03)	.08 (-.06)	.00 (-.05)	.14 (.01)
SCR Trial 2 Anticipatory Amplitude	.13 (.18)	.14 (.16)	-.06 (.25)	.17 (.12)	.07 (.09)	.00 (.17)	.14 (.15)
SCR Trial 2 Reactivity Amplitude	.13 (.08)	.03 (.07)	.05 (.01)	-.04 (-.13)	.08 (.08)	.06 (.10)	.07 (-.02)
SCR Trial 3 Anticipatory Amplitude	-.17 (.02)	.01 (.06)	-.23 (-.13)	.03 (.04)	-.06 (.19)	-.15 (-.09)	-.04 (.13)
SCR Trial 3 Reactivity Amplitude	.09 (.12)	.04 (.09)	.22 (.26)	.20 (.14)	-.10 (.01)	.08 (.12)	.11 (.12)
SCR Trial 4 Anticipatory Amplitude	.14 (.27*)	.20 (.21)	-.16 (-.01)	.17 (.25)	.16 (.13)	.03 (.16)	.21 (.28*)
SCR Trial 4 Reactivity Amplitude	.15 (.10)	.20 (.11)	-.09 (-.10)	.17 (-.01)	.13 (.15)	.06 (.01)	.15 (.05)
Average SCR Amplitude Trial 1-4 Anticipatory	.03 (0.15)	.12 (.15)	-.12 (.00)	.17 (.14)	.05 (.14)	.06 (.07)	.11 (.19)
Average SCR Amplitude Trial 1-4 Reactivity	3.34 (.08)	.10 (.10)	.03 (.01)	.13 (.00)	.05 (.18)	.05 (.05)	.12 (.04)

Note: ** Indicates correlation is significant at the .01 level; * Indicates correlation is significant at the .05 level.

The values in parentheses represent the SCR frequency and amplitude values prior to the log or square root transformation

PCL: YV Total Score= Psychopathy Check-List: Youth Version Total Score, F1= Psychopathy Check-List: Youth Version Interpersonal Facet, F2 = Psychopathy Check-List: Youth Version Affective Facet, F3 = Psychopathy Check-List: Youth Version Lifestyle Facet, F4 = Psychopathy Check-List: Youth Version Antisocial Facet, F-1 = Psychopathy Check-List: Youth Version Interpersonal-Affective Factor, F-2 = Psychopathy Check-List: Youth Version Lifestyle-Behavioral Factor, SCR Trial 1 Anticipatory Frequency = Skin Conductance Response Frequency Trial 1 Anticipatory Period, SCR Trial 1 Reactivity Frequency = Skin Conductance Response Frequency Trial 1 Reactivity Period, SCR Trial 2 Anticipatory Frequency = Skin Conductance Response Frequency Trial 2 Anticipatory Period, SCR Trial 2 Reactivity Frequency= Skin Conductance Response Frequency Trial 2 Reactivity Period, SCR Trial 3 Anticipatory Frequency = Skin Conductance Response Frequency Trial 3 Anticipatory Period, SCR Trial 3 Reactivity Frequency = Skin Conductance Response Frequency Trial 3 Reactivity Period, SCR Trial 4 Anticipatory Frequency = Skin Conductance Response Frequency Trial 4 Anticipatory Period, SCR Trial 4 Reactivity Frequency = Skin Conductance Response Frequency Trial 4 Reactivity Period, Average SCR Trial 1-4 Frequency Anticipatory = Average Skin Conductance Response Trial 1-4 Frequency Anticipatory Period, Average SCR Trial 1-4 Frequency Reactivity = Average Skin Conductance Response Trial 1-

4 Frequency Reactivity Period, SCR Trial 1 Anticipatory Amplitude= Skin Conductance Response Amplitude Trial 1 Anticipatory Period, SCR 1 Reactivity Amplitude = Skin Conductance Response Amplitude Trial 1 Reactivity Period, SCR 2 Anticipatory Amplitude = Skin Conductance Response Amplitude Trial 2 Anticipatory Period, SCR 2 Reactivity Amplitude = Skin Conductance Response Amplitude Trial 2 Reactivity Period, SCR 3 Anticipatory Amplitude = Skin Conductance Response Amplitude Trial 3 Anticipatory Period, SCR 3 Reactivity Amplitude= Skin Conductance Response Amplitude Trial 3 Reactivity Period, SCR 4 Anticipatory Amplitude = Skin Conductance Response Amplitude Trial 4 Anticipatory Period, SCR 4 Reactivity Amplitude = Skin Conductance Response Amplitude Trial 4 Reactivity Period, Average SCR Trial 1-4 Amplitude Anticipatory = Average Skin Conductance Response Trial 1-4 Amplitude Anticipatory Period, Average SCR Trial 1-4 Amplitude Reactivity = Average Skin Conductance Response Trial 1-4 Amplitude Reactivity Period.

MANCOVA/ANCOVA

A one-way multivariate analysis of covariance (MANCOVA) was conducted to test the hypothesis that there would be one or more mean differences between SCR frequency (responders versus non-responders) and PCL:YV facet and factor scores, controlling for aggression using PCS scores. Preliminary assumption testing indicated that the PCL:YV Lifestyle facet and PCL:YV Lifestyle-Behavioral factor violated the assumption of equal variance, and so an alpha of .01 was used to determine significance of these variables in the univariate F-test. There was a statistically significant difference between SCR responders and non-responders on the combined dependent variables, Wilks' Lambda = .76, $F(5, 47) = 2.93$, $p = .02$; partial eta squared = .238. When the results for the dependent variables were considered separately, using a Bonferroni adjusted alpha level of 0.01, the only differences to reach statistical significance were the Lifestyle facet, $F(1, 4) = 6.70$, $p = .01$; partial eta squared = .116, and F2, $F(1, 4) = 8.48$, $p = .01$; partial eta squared = .143. An inspection of the mean scores indicated that SCR responders were rated as having slightly higher scores on the Lifestyle facet ($M = 6.12$, $SD = 1.19$) and Lifestyle-Behavioral factor ($M = 10.20$, $SD = 2.31$) than non-responders ($M = 5.19$, $SD = 1.83$, and $M = 9.38$, $SD = 2.65$, respectively).

A one-way univariate analysis of covariance (ANCOVA) was also conducted to test the hypothesis that there would be one or more mean differences between SCR frequency (responders versus non-responders) and the PCL:YV total score, controlling for aggression using

PCS scores and the PCL:YV Antisocial facet 4. The effect of SCR frequency on PCL:YV total scores after controlling for aggression was not significant, $F(1, 51) = 1.32, p = .26$.

Regression Analyses

A hierarchical regression was used to assess the ability of the PCL:YV Interpersonal facet to predict SCL baseline scores, after controlling for the influence of aggression. The PCS Proactive and Reactive Total aggression subscales were entered at Step 1, explaining 1.2% of the variance, but not significantly so. After entry of the Interpersonal facet at Step 2, the total variance explained by the model as a whole was 8.6%, $F(3, 52) = 1.64, p = .19$. In the final model, only the Interpersonal facet was statistically significant, $\beta = .28, p = .05$. A second hierarchical regression was run to assess the ability of the PCL:YV total score to predict SCL baseline scores, controlling for PCS and PCL:YV Antisocial facet 4 scores. The PCS Proactive and Reactive Total aggression subscales and PCL:YV Antisocial facet 4 scores were entered at Step 1, explaining 1.2% of the variance, but not significantly so. After entry of the PCL:YV total score at Step 2, the total variance explained by the model as a whole was 8.5%, $F(4, 51) = 1.18, p = .33$. In the final model, only the PCL:YV total score was statistically significant, $\beta = .27, p = .05$.

5. DISCUSSION

Recent shifts in research of psychopathy have led to further interest in child psychopathy and underlying physiological processes. In addition to conflicting results within the literature, the empirical focus on adults, community populations and the sympathetic nervous system led this study to investigate both the sympathetic and parasympathetic processes underlying psychopathic traits in adolescent offenders. The results of this study add to increasing evidence of the difficulty in teasing out specific physiological processes underlying a single emotional-behavioral trait.

In light of evidence suggesting that low resting heart rate, RSA, and SCL is associated with psychopathic traits and aggressive/disruptive behavior, this study's first hypothesis proposed that PCL:YV scores would be negatively associated with and predict baseline HR, RSA, and SCL patterns. Contrary to this hypothesis, baseline SCL was positively associated with the PCL:YV Interpersonal facet and the PCL:YV total score. The association with the Interpersonal facet was in line with the fourth hypothesis, which posited that all physiological scores would have the strongest association with the Interpersonal facet and Interpersonal-Affective factor of the PCL:YV.

The lack of relationship found between baseline HR/ RSA and psychopathy suggests that HR/RSA may not be a biological marker for psychopathy in adolescents. However, the pattern of insignificant results appeared to demonstrate that baseline HR had a negative relationship with the Interpersonal and Affective PCL:YV facets ($r = -0.07$ and $r = -.09$, respectively), and a positive relationship with the Lifestyle and Behavioral facets ($r = 0.07$ and $r = .03$, respectively).

These small correlations mirror the relatively small correlation found by Raine (2014) between resting HR and APSD total ($r = -0.15$), although they are much smaller in the current study. Seeing as RSA baseline values had a non-significant positive correlation with F1 ($r = .14$) and a negative correlation with F2 ($r = -.13$), it appears as though there may be different cardiac sympathetic and parasympathetic processes underlying distinct psychopathic characteristics.

Alternatively, HR/RSA may depend upon some moderator and thus may be a function of a third variable not addressed in this study. Lorber's (2014) meta-analysis also did not find an association between resting HR and psychopathy/ sociopathy in adults (mean effect size was small). In adolescents, the association between resting HR and conduct disorder was significant and moderate in size.

A significant relation in this study was the relationship between baseline SCL and the PCL:YV Interpersonal facet and PCL:YV total score. This finding is similar to that found by Beauchaine and colleagues (2008), in which aggressive girls with conduct disorder had increased baseline SCL that gradually decreased and also similar to Glenn and colleagues (2007), who found that children at 3-years-old with increased baseline SCL had higher psychopathy scores at 28. Glenn and colleagues (2007) posited that the increased baseline SC suggests higher levels of attentional processing or prefrontal processing that may serve to mark a "successful psychopath." It may be that the link between increased ANS functioning and higher psychopathic traits such as manipulation, deception, and charm represents adaptive functioning in the face of a stressful, arousing environment.

The second hypothesis of this study anticipated a negative relationship between PCL:YV scores and SCR values (amplitude and frequency) within the anticipatory period of signaled trials, and a positive relationship between PCL:YV scores and HR activity within this period.

The second hypothesis of this study anticipated a negative relationship between PCL:YV scores and SCR values (amplitude and frequency) within the anticipatory period of signaled trials, and a positive relationship between PCL:YV scores and HR activity within this period. Similarly, the third hypothesis expected a negative relationship between PCL:YV scores and SCR/ HR activity within the reactivity period of the signaled trials. Correlational analyses did not reveal these relations and nor were any significant SCR/HR and PCL:YV scores noted. These results are similar to that found by Raine and Venables (1984b) who neglected to find a relationship between skin conductance orienting response frequencies and antisocial behavior in adolescent males. However, MANCOVA analyses indicated that SCR responders during the anticipatory and reactivity periods had higher Lifestyle facet and Lifestyle-Behavioral factor scores than those with no SCRs. This was contrary to hypotheses 2, 3, and 4, which proposed that a negative relationship between SCR anticipatory and reactivity would be more strongly associated with the Interpersonal facet. Yet Blair (1999) also found a positive relationship between psychopathic tendencies and increased SCR in response to threatening stimuli. The author did not investigate whether this relationship was more strongly associated with F1 or F2 characteristics.

Relatedly, even with a total sample size of 95 studies, Lorber's meta-analysis (2014) found variable associations between HR reactivity patterns and psychopathy/sociopathy/conduct problems. Indeed, no association between HR reactivity and psychopathy/sociopathy was found in adults, and there was only a marginally significant heterogeneity in effect sizes. For children and adolescents, greater HR reactivity was associated with conduct problems but maintained a small effect size. With regard to task HR, there was little association with psychopathy/sociopathy in adults (mean effect size = -0.16), and no relationship with conduct problems in children or adolescents (mean effect size = -0.04).

However, there appears to be a pattern to the relations between HR anticipatory/reactivity and the psychopathy factors in this study, in that HR activity had a negative relationship with the Interpersonal and Affective PCL:YV factor (F2) (aligning with this study's fourth hypothesis), and a positive relationship with the Lifestyle and Behavioral facets across anticipatory and reactivity values. The average correlation for HR anticipatory and the Interpersonal/Affective factor (F1) was $r = -.12$ and the average correlation for the Lifestyle/Behavioral factor (F2) was $r = .16$, while the average correlation for HR reactivity and F1 was $r = -.09$, and the average correlation for F2 was $r = .57$. Although none of the associations were significant, it may be that with a larger sample size, these associations may have met the threshold for statistical significance. If this pattern were to hold with a larger sample, it would suggest that hypoaroused cardiac processes in individuals with psychopathic traits is distinctly linked with interpersonal and affective deficits, while hyperaroused cardiac processes may underlie lifestyle and behavioral deficits. On the one hand, hypoaroused cardiac processes underlying interpersonal and affective psychopathic traits aligns with the fearlessness and stimulation-seeking theories of underarousal. On the other hand, Wang and colleagues (2012) have suggested that increased HR (particularly, accelerated HR during the anticipatory period) may be a biological marker of both reduced emotional regulation and behavioral disinhibition. Thus, it may be that extreme states on either end of the spectrum of fearlessness and stimulation seeking may contribute to psychopathic behavior in different ways (Wang et al., 2012). Furthermore, there was no clear pattern with respect to correlational analyses of SCR frequency and amplitude values, which calls into question the differential contributions of sympathetic nervous system processes.

Conclusion

Overall, study results hint to some differences in the biological functioning of those with psychopathic traits although it is premature to conclude that physiological research with offending adults with psychopathic traits can be extended downward to adolescent offenders. In consideration of this study's significant baseline SCL association, the ability to manipulate, deceive, and charm people may be a product of greater, pre-existing prefrontal functioning, which subsequently sets the individual up with the ability to adapt to a stressful environment where success requires increased arousal and attention. In addition, those individuals with at least one SCR in anticipation of or in reaction to the white noise stimulus received slightly higher Lifestyle facet and F2 scores than those with no SCRs. It appears as though increased ANS functioning may also have some drawbacks in an arousing environment, in that increased anticipation of and reaction to stressful events can be characterized by impulsivity and irresponsibility.

This possible interpretation fits within the biosocial model put forth by Beauchaine and colleagues (2007), who proposed that children develop effective vagal modulation of emotion within environments that promote emotion regulation skills such as arousal de-escalation, positive reinforcement of prosocial behaviors, and consequences for aggressive behavior. Within the context of the "fearlessness" theory, increased baseline arousal may represent increased fear and anxiety. It may be that individuals experiencing fear and anxiety in a demanding environment (e.g., courtroom or detention center) draw more heavily upon their interpersonally psychopathic traits such as charm, manipulation, or deception in order to increase control of the situation. This could be particularly necessary for success if an individual is demonstrating increased impulsivity and irresponsibility as part of their increased arousal. Similarly, in

consideration of the “stimulation-seeking” theory, autonomic hyperarousal may originate from the stimulating circumstances of a courtroom or detention setting, and interpersonal processes are one way an individual seeks to bring arousal levels down to more average levels and control their levels of impulsivity and irresponsibility. Working within the framework of the aforementioned theories of autonomic dysfunction reveals that increased arousal can serve an adaptive purpose for adolescents in an environment that places demands for emotional regulation and rapid learning from consequences.

Given that hypotheses 1-4 were not fully supported, the insignificant results may alternatively signify that the HR, RSA, SCL, and SCR variables are generally unrelated to psychopathy. Indeed, previous research with children and adolescents has produced conflicting results about finding significant, attenuated associations between physiological processes such as heart rate and skin conductance and psychopathic traits such as aggression and antisocial behavior (Beauchaine et al., 2007; 2008; Fowles & Furuseth, 1994; Glenn et al., 2007; Lorber, 2004; Raine & Venables, 1984a, 1984b; Zahn & Kruesi, 1993). An additional explanation may be that the influence of clinically significant factors beyond those specifically addressed overshadowed any existing influence of the factors examined in this study, thus resulting in a rejection of the other hypotheses. For example, physiological processes have been linked to depression, anxiety, self-injury, ADHD, and other disruptive disorders like conduct disorder and oppositional defiant disorder (e.g., Beauchaine et al., 2007, Shannon et al., 2007; van Goozen et al., 1998; Zahn & Kruesi, 1993), so perhaps controlling for these factors may have allowed for significant associations to be identified.

Limitations

The current study had a number of limitations. First, this study had a relatively small ($N = 56$) and homogeneous sample that may have resulted in some non-normality in the data. Moreover, current biopsychological research appears to use large samples that range from 334 (Raine et al., 2014) to 843 (Wang et al., 2012), and 1,795 (Raine et al., 1997) to 2,129 (Isen et al., 2010). However, significant findings in other studies have had moderate to small effect sizes for both adult and youth populations (Lorber, 2004), particularly in comparison to the large effect sizes produced by this study. Additionally, given that the sample was entirely male, our results may not extend to female children and adolescents. Third, the use of a countdown paradigm and 90-dB white-noise stimuli may not have elicited a large enough startle response to produce a SCR. Given the positively skewed distribution of SCR values, and the relatively high rate of non-responding, it may be that the 90-dB white noise was not intense enough. Fung and colleagues (2005) also found a high rate of SCR non-responding, so it may be that the countdown task is unlikely to produce a highly strong response. Lastly, a limited range of scores and the shape of the SCR and PCL:YV total score distributions may have affected the study findings.

Future Directions and Final Remarks

The current study offered a number of interesting findings that may provide hints to the differential functioning between those with elevated versus non-elevated psychopathic traits. The results also suggest several future research directions. First, researchers should continue examining psychophysiological results with large samples as well as diverse samples. Second, using an aversive stimulus such as the countdown task denotes reliance on the fearlessness theory to explain the physiological results, in that significantly attenuated ANS results implies

that antisocial behavior stems from a lack of fear (e.g., Fowles et al., 2000; Syngelaki et al., 2013). Alternatively, using the stimulation-seeking theory as a guideline may warrant different methods of measurement that could augment previously established significant findings. Other tasks and stimuli then might be designed to test this theory. Finally, future research may also consider measuring other variables such as heart rate variability (HRV) or SCR magnitude across varying periods of time and numbers of trials. To our knowledge, this study is the only psychopathy-centered project to have examined RSA in addition to the more commonly used HR and SCR in adolescent offenders, and it appears necessary that RSA continue to be included in study analyses.

Evidence from this study and other literature demonstrates that the identification of neurobiological referents of psychopathy in adolescent offenders remains open to investigation. In addition to psychology, the increase in neurobiological research continues across a plethora of academic areas and does not appear likely to slow down in the near future. Recent research funding trends also continue to encourage analyses that include neurobiological correlates of behavior. As technological advancement expands the ability of researchers to examine the brain, focus will likely narrow to the smaller, micro-level processes and systems of the brain. Overall, broad interpretations about the physiological processes underlying adolescent psychopathy may be insufficient and can only apply to the group level and under specific conditions. A greater breadth of research on ANS activity could influence the development of intervention and treatment protocols for children and adolescents with serious antisocial behavior, perhaps even developing the ability of measurements to detect or diagnosis chronic or persistent cases.

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APPENDIX

Office for Research
Institutional Review Board for the
Protection of Human Subjects

THE UNIVERSITY OF
ALABAMA
R E S E A R C H

February 8, 2016

Randall T. Salekin, Ph.D.
Professor
Department of Psychology
College of Arts & Sciences
The University of Alabama
Box 870348

Re: IRB # 12-007-R4 "Multi-method Assessment of RSTI-A and a Brief Intervention"

Dear Dr. Salekin:

The University of Alabama Institutional Review Board has granted approval for your renewal application.

Your renewal application has been given expedited approval according to 45 CFR part 46. Approval has been given under expedited review category 8 as outlined below:

(8) Continuing review of research previously approved by the convened IRB as follows:
(a) where (i) the research is permanently closed to the enrollment of new subjects; (ii) all subjects have completed all research-related interventions; and (iii) the research remains active only for long-term follow-up of subjects; or
(b) where no subjects have been enrolled and no additional risks have been identified; or
(c) where the remaining research activities are limited to data analysis.

Your application will expire on February 7, 2017. If your research will continue beyond this date, complete the relevant portions of the IRB Renewal Application. If you wish to modify the application, complete 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, complete the appropriate portions of the IRB Study Closure Form.

Should you need to submit any further correspondence regarding this proposal, please include the above application number.

Good luck with your research.

Sincerely,



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