Summer 7-9-2019

Identifying error-related, response inhibition, and structural integrity deficits associated with youth with elevated psychopathic traits and predictive of future recidivism

James M. Maurer
University of New Mexico

www.manaraa.com

J. Michael Maurer

Candidate

Psychology

Department

This dissertation is approved, and it is acceptable in quality and form for publication:

Approved by the Dissertation Committee:

Kent A. Kiehl, Ph.D.   , Chairperson

Vincent P. Clark, Ph.D.

Eric D. Claus, Ph.D.

Brandi C. Fink, Ph.D.

Jeremy Hogeveen, Ph.D.

**IDENTIFYING ERROR-RELATED, RESPONSE INHIBITION
AND STRUCTURAL INTEGRITY DEFICITS ASSOCIATED
WITH YOUTH WITH ELEVATED PSYCHOPATHIC TRAITS
& PREDICTIVE OF FUTURE RECIDIVISM**

by

J. MICHAEL MAURER

M.S., University of New Mexico, 2016
B.A., University of California, Davis, 2011
A.A., Fullerton College, 2009

DISSERTATION

Submitted in Partial Fulfillment of the
Requirements for the Degree of

Doctor of Philosophy

Psychology

The University of New Mexico
Albuquerque, New Mexico

July, 2019

ACKNOWLEDGMENTS

Identifying error-related processing, response inhibition, and structural integrity deficits associated with youth with elevated psychopathic traits and predictive of future recidivism

by

J. Michael Maurer

A.A., Fullerton College, 2009

B.A., University of California, Davis, 2011

M.S., University of New Mexico, 2016

Ph.D., University of New Mexico, 2019

Abstract

Juvenile delinquency poses a severe cost to society. Adolescent offenders who are more likely to recidivate, including youth scoring higher on measures of psychopathic traits, and non-psychopathic life-course persistent offenders, are characterized by cognitive control deficits which may increase their propensity towards future recidivism. In this dissertation, we found that youth scoring high on psychopathic traits exhibited increased functional connectivity during response inhibition and reduced functional connectivity during error-related processing, and reduced structural integrity in the inferior fronto-occipital fasciculus and uncinate fasciculus, compared to youth scoring low on psychopathic traits. In addition, we found error-related processing deficits and reduced structural integrity of the corticospinal tract were predictive of future recidivism in samples of incarcerated adolescent male offenders. The results of this dissertation help extend previous research by identifying novel deficits characteristic of youth with

elevated psychopathic traits and predictive of future recidivism in potential life-course

persistent antisocial adolescent offenders.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

INTRODUCTION

Background and Significance

Psychopathy is a multifaceted personality disorder commonly associated with interpersonal, affective, and behavioral dysfunction (Hare, 2003). Psychopaths are habitually described by their overall dearth of moral emotions and a lifestyle characterized by heightened impulsivity and irresponsibility; this antisocial lifestyle severely increases the likelihood of a number of poor outcomes for psychopaths, particularly an increased risk of future recidivism (Hemphill, Hare, & Wong, 1998). While only 0.5 – 1% of the general population meets the established diagnostic criteria for psychopathy, the base rate increases tremendously in incarcerated settings, whereby 15 – 25% of incarcerated offenders meet the criteria for psychopathy (Hare, 2003). Though comprising a minority of the general population, psychopaths are responsible for a disproportionate amount of total criminal offenses, creating an estimated financial burden of $450 billion annually (Kiehl & Hoffman, 2011). Additionally, psychopaths are four times more likely to recidivate in the twelve months following institutional release, particularly by engaging in violent criminal activity, compared to non-psychopathic offenders (Rice & Harris, 1997). Due to these aforementioned reasons, researchers have attempted to characterize both the personality traits and neurobiological deficits characteristic of individuals with heightened psychopathic traits.

History of Psychopathy

The criterion for psychopathy was initially influenced by early physicians' concepts of related conditions. Most notably, Philippe Pinel, a French physician, coined the term "*manie sans delire*" or "*insanity without delirium*" to describe individuals who

engaged in severe antisocial behavior but could not be characterized by any of the traditional symptomatology commonly associated with individuals experiencing psychosis (Pinel, 1800). The Italian criminologist Cesare Lombroso later developed the concept of a "*born criminal*", suggesting that certain individuals in society may engage in antisocial behavior due to underlying neurobiological deficits established at birth (Lombroso, 1911). The term *"psychopathy"* itself was first used by the German psychiatrist Julius Koch, but not in the modern sense of the definition. Instead, Koch developed the concept of "*personality disorders*", referring to them as "*psychopathic inferiorities*" (J. L. A. Koch, 1889). The first use of the term psychopathy in the modern sense of the definition was used by the German psychiatrist Emil Kraepelin in his classic psychiatry textbook "*Lehrbuch der Psychiatrie*". Here, Kraepelin included an entire section devoted to individuals who engaged in manipulative and antisocial behavior and exhibited a strong propensity towards impulsivity and aggression (Bleuler, 1975).

The modern study of psychopathy was first pioneered by the American psychiatrist Hervey Cleckley. In his prominent book titled "*The Mask of Sanity*", Cleckley described his experience working in psychiatric hospitals where he encountered and interacted with many individuals with elevated psychopathic traits (Cleckley, 1941). Cleckley's work remains quite significant and has largely influenced the current conception of a psychopath. In fact, Cleckley's work directly motivated the modern scoring criteria for psychopathy, outlined by Robert Hare's Psychopathy Checklist (PCL). Hare operationalized and used the characteristics described by Cleckley's work to create the PCL (Hare, 1980) and the subsequent Hare Psychopathy Checklist – Revised (PCL-R) (Hare, 1991, 2003).

Assessing Psychopathic Traits

The PCL-R has been established as the gold standard for the assessment of psychopathic traits across a variety of different samples, including participants recruited from incarcerated and forensic settings and psychiatric hospitals, and also participants meeting criteria for substance use disorders (SUDs) (Hare, 1996, 2003; McDermott et al., 2000). The PCL-R contains twenty items to assess psychopathic traits, including (1) glibness and superficial charm, (2) a grandiose sense of self-worth, (3) a need for stimulation and proneness to boredom, (4) pathological lying, (5) conning and manipulative behavior, (6) a lack of remorse or guilt, (7) shallow affect, (8) callousness and/or a lack of empathy, (9) a parasitic lifestyle, (10) poor behavioral controls, (11) promiscuous sexual behavior, (12) early behavioral problems, (13) a lack of realistic, long-term goals, (14) impulsivity, (15) irresponsibility, (16) a failure to accept responsibility for their actions, (17) many short-term marital relationships, (18) juvenile delinquency, (19) revocation of conditional release, and (20) criminal versatility (Hare, 2003). Scoring of the PCL-R relies on a semi-structure interview and an extensive file review to provide collateral information to score participants on the twenty items comprising the PCL-R. Each item of the PCL-R can be scored based on the following criteria: a score of zero reflects that the *item does not apply to the individual*, a score of one indicates that the *item applies somewhat to the individual*, and a score of two means the *item definitely applies to the individual* (Hare, 2003). PCL-R total scores can then potentially range from 0 to 40, with a score 30 or above reflecting an individual who meets the established diagnostic criteria for psychopathy (Hare, 2003). The mean PCL-R total score in incarcerated settings is a 22 (Hare, 2003), whereas mean PCL-R total scores

are typically quite low in community samples, with individuals typically scoring under a 3 (Neumann & Hare, 2008).

Factor analysis of the PCL-R reveals two underlying factors of psychopathic traits (Harpur, Hare, & Hakstian, 1989). Factor 1 of the PCL-R is comprised of interpersonal and affective traits (i.e., PCL-R items 1, 2, 4, 5, 6, 7, 8, and 16), whereas Factor 2 of the PCL-R includes lifestyle and antisocial/developmental traits (i.e., PCL-R items 3, 9, 10, 13, 14, 15, 18, 19, and 20) (Harpur et al., 1989). PCL-R items 11 and 17 (promiscuous sexual behavior and many short-term marital relationships, respectively) do not load onto either of the two factors of the PCL-R (Harpur et al., 1989). Additionally, a four-facet model of psychopathic traits has been developed, with Facet 1 of the PCL-R reflecting interpersonal items (i.e., PCL-R items 1, 2, 4, and 5), Facet 2 of the PCL-R including affective traits (i.e., PCL-R items 6, 7, 8, and 16), Facet 3 of the PCL-R comprising lifestyle traits (i.e., PCL-R items 3, 9, 13, 14, and 15), and Facet 4 of the PCL-R including antisocial/developmental traits (i.e., PCL-R items 10, 12, 18, 19, and 20) (Hare & Neumann, 2006). Consistent with the two-factor model of the PCL-R, PCL-R items 11 and 17 do not load onto any of the four facets (Hare & Neumann, 2006).

While most researchers refer to the two-factor or four-facet model of psychopathic traits, other researchers have developed a three-factor model of psychopathic traits, which deliberately excludes antisocial items (i.e., items comprising PCL-R Facet 4) (Cooke & Michie, 2001). Using confirmatory factor analysis, the three-factor model identifies the three following factors: (1) arrogant and deceitful interpersonal lifestyle, (2) deficient affective experience, and (3) impulsive and irresponsible behavior (Cooke & Michie, 2001). This three-factor model was developed,

as some researchers remain apprehensive over the inclusion of items reflecting antisocial behavior within the superordinate construct of psychopathy, believing criminal and antisocial behavior to be a consequence, rather than a foundation, of psychopathic traits (Cooke & Michie, 2001; Skeem & Cooke, 2010). However, this three-factor model of psychopathic traits has been criticized due to severe statistical issues, including being over-factored, where ten different facets (which underlie three superordinate factors) explain only 13 items of the PCL-R, and flawed parameters, including negative variance (Hare & Neumann, 2010). Due to the limitations observed with the three-factor model, researchers have typically included PCL-R total score, scores from the two-factor model, or scores from the four-facet model in analyses performed.

Also, important to note, some of the traits characteristic of psychopathy (e.g., PCL-R Factor 2 traits) are quite similar to those used to diagnose individuals on the Diagnostic and Statistical Manual of Mental Disorders (DSM-V) Axis II disorder "*Antisocial Personality Disorder (APD)*" (American Psychiatric Association, 2013). While psychopathy and APD are two conditions that do share many similarities, these are separate constructs. The assessment of APD relies strictly on the measurement of antisocial behavior while ignoring the affective and interpersonal dysfunction central to the diagnostic criteria of psychopathy. Additionally, it is exceedingly rare for incarcerated individuals in maximum-security correctional facilities to fail to meet criteria for APD; 80 – 90% of such individuals will meet criteria for APD compared to only 15 – 25% meeting criteria for psychopathy (Hart & Hare, 1989).

Neurocognitive Deficits Associated with Psychopathy

Individuals who meet the diagnostic criteria for psychopathy exhibit a number of neurocognitive deficits. Researchers have proposed various theories to potentially explain neurocognitive deficits associated with psychopathic individuals. For example, David Lykken proposed the "*low fear hypothesis*" which suggests that individuals who meet criteria for psychopathy exhibit a subdued fear response, increasing the likelihood of these individuals to participate in dangerous and risky behavior (Lykken, 1995). Supporting the low fear hypothesis, previous studies have shown that individuals scoring higher on psychopathy display weak electrodermal responses in anticipation of aversive events (Hare, 1978), poor passive avoidance learning (Newman & Kosson, 1986), and a lack of startle potentiation (Patrick, 1994). However, a recent meta-analysis suggests that psychopathic individuals exhibit deficits in threat detection and responsivity rather than experiencing a reduced subjective experience of the emotion of fear (Hoppenbrouwers, Bulten, & Brazil, 2016). Furthermore, a number of published reports have shown that individuals scoring high on psychopathy can in fact exhibit normal fear conditioning and emotional processing when specifically directed towards the stimuli of interest (Anderson & Stanford, 2012; Anderson et al., 2017; Baskin-Sommers, Curtin, Li, & Newman, 2012; Baskin-Sommers, Curtin, & Newman, 2011, 2013; Carolan, Jaspers-Fayer, Asmaro, Douglas, & Liotti, 2014; C. L. Larson et al., 2013; Newman, Curtin, Bertsch, & Baskin-Sommers, 2010). These studies support the "*response modulation hypothesis*" proposed by Joseph Newman. This theory suggests that individuals scoring high on psychopathy may exhibit fear conditioning or emotional processing deficits due to failures in appropriately processing information that is peripheral to ongoing, goal-directed behavior, including affective, inhibitory, or other potentially important stimuli (Newman

& Lorenz, 2003). Thus, when their attention is properly devoted to processing fear- or emotion-related stimuli, psychopathic individuals are able to exhibit proper functioning. Related to this theory, the "*attentional bottleneck hypothesis*" suggests that psychopathic individuals exhibit difficulty reallocating their attention, instead becoming hyper-focused on a single aspect of experimental paradigms at the price of subsequent cognitive functioning (Newman & Baskin-Sommers, 2012).

Additional models have been proposed to illuminate the neurobiological deficits commonly associated with psychopathic individuals. An early theory proposed by James Blair suggested amygdala and ventromedial prefrontal cortex (vmPFC) dysfunction relating to affective deficits characteristic of psychopathic individuals (Blair, 2003, 2007). Blair's perspective suggests that psychopathic individuals exhibit deficits in stimulus-reinforcement learning and decision-making (Blair, 2007). For example, amygdala dysfunction can result in impairments in an individual's ability to learn to avoid associations connected with the distress of others and a reduced vmPFC response suggests deficits in prediction error signaling (Blair, 2007). Additionally, the personality trait changes that accompany vmPFC damage have been labeled "*pseudopsychopathy*" (Blumer & Benson, 1975) and "*acquired sociopathic personality*" (Damasio, 1994), including a lack of empathy, irresponsibility, and deficits within decision-making processes, which bear strong similarities to traits characteristic of psychopathy. Psychopaths and vmPFC lesion patients perform similarly on measures of moral judgment (Koenigs, Kruepke, Zeier, & Newman, 2011). Finally, individuals with psychopathic traits have also exhibited reduced gray matter volume in the vmPFC (Boccardi et al., 2011) and amygdala (Boccardi et al., 2011; Ermer, Cope, Nyalakanti,

Calhoun, & Kiehl, 2012; Yang, Raine, Colletti, Toga, & Narr, 2010), lending credence to amygdala and vmPFC dysfunction proposed by Blair.

Kent Kiehl later expanded upon Blair's early conception of neurobiological deficits commonly associated with psychopathic individuals with the "*paralimbic hypothesis*" (Kiehl, 2006). In addition to the amygdala and vmPFC dysfunction proposed by Blair, Kiehl suggested that psychopaths exhibit dysfunction in a number of paralimbic and surrounding brain regions, including the anterior superior temporal gyrus, anterior cingulate cortex (ACC), posterior cingulate cortex (PCC), insula, and parahippocampal gyrus (Kiehl, 2006). Supporting this hypothesis, (1) lesions to the ACC are associated with emotional unconcern and perseveration deficits (Mesulam, 2000), and difficulties in affective face processing (Hornak et al., 2003), error-processing (Swick & Turken, 2002), and response inhibition (Tekin & Cummings, 2002), all deficits which are characteristic of psychopathy (Blair, Jones, Clark, & Smith, 1997; Brazil et al., 2009; Kiehl, Smith, Hare, & Liddle, 2000; Kosson, Suchy, Mayer, & Libby, 2002; Maurer, Steele, Edwards, et al., 2016; Newman, 1998; Steele, Maurer, Bernat, Calhoun, & Kiehl, 2016), (2) the PCC is associated with affective word processing (Maddock & Buonocore, 1997), which has been shown to be dysfunctional in psychopathic offenders (Kiehl, Hare, McDonald, & Brink, 1999; Williamson, Harpur, & Hare, 1991), (3) the right anterior superior temporal gyrus is associated with the processing of abstract words (Kiehl, Liddle, et al., 1999; Kiehl et al., 2004), which has also been shown to be deficient in psychopathic individuals (Kiehl et al., 2004), and (4) the parahippocampal gyrus is involved in affective processing (Fusar-Poli et al., 2009), and psychopathic individuals have exhibited reduced hemodynamic activity in the parahippocampal gyrus during processing

of affective information (Muller et al., 2003). Furthermore, neuroimaging studies have been performed supporting Kiehl's hypothesis of paralimbic dysfunction in psychopathic individuals, observing reduced gray matter in the insula (Cope et al., 2012; de Oliveira-Souza et al., 2008; Gregory et al., 2012; Ly et al., 2012; Walters, Ermer, Knight, & Kiehl, 2015), parahippocampal gyrus (Boccardi et al., 2011; Ermer et al., 2012; Tiihonen et al., 2008; Walters et al., 2015), ACC (Baskin-Sommers, Neumann, Cope, & Kiehl, 2016; Boccardi et al., 2011; Cope et al., 2012; Ly et al., 2012; Yang et al., 2010), and PCC (Boccardi et al., 2011; de Oliveira-Souza et al., 2008; Ermer et al., 2012; Tiihonen et al., 2008; Yang et al., 2010).

Finally, a recent theoretical approach has been developed, titled the "*impaired integration model*" (R. K. Hamilton, Hiatt Racer, & Newman, 2015), which suggests psychopathic individuals experience specific impairment in switching between large-scale brain networks, including the default mode network and salience network, which contain regions implicated in paralimbic dysfunction, including the ACC, PCC, insula, and amygdala. Recent neuroimaging investigations have supported the impaired integration model. For example, psychopathic individuals exhibit abnormalities in shifting between the default mode network (i.e., a task-negative network) and task-positive networks, by failing to deactivate regions of the default mode network during a task-positive Go/NoGo task (Freeman et al., 2015). In addition, those scoring higher on psychopathy have exhibited aberrant functional connectivity between regions of the default mode network and salience network (Philippi et al., 2015) and reduced hemodynamic activity in regions of the default mode network and salience network during an auditory oddball experimental paradigm (Anderson, Maurer, Steele, & Kiehl,

2018). Individuals with elevated psychopathic traits have also been associated with ineffective switching between resting-state dominant networks and those involved in externally focused attention (Sheng, Gheytanchi, & Aziz-Zadeh, 2010).

Understanding the Construct of Psychopathy in Youth Samples

Despite recent interest in understanding the neurobiological underpinnings of psychopathy, treatment interventions have often proven ineffective for this disconcerting population (Rice & Harris, 1997). Compared to adults, younger samples may prove more amenable to treatment intervention approaches, as personality traits are still in nascent stages of development. For example, youth meeting criteria for externalizing disorders have exhibited improvement in self-regulation, operationally defined as increasing amplitude of the N2 and P300 event-related potentials (ERPs) through cognitive-behavioral therapy (Woltering, Granic, Lamm, & Lewis, 2011). Furthermore, despite the seemingly ineffective nature of treatment intervention approaches for adult psychopathic offenders, optimism exists that juveniles scoring high on measures of psychopathy can be treated successfully based on studies incorporating an intensive decompression approach (M. F. Caldwell, 2011; M. F. Caldwell, McCormick, Umstead, & Van Rybroek, 2007). This approach has been shown to reduce both recidivism rates and both interpersonal (Facet 1) and affective (Facet 2) psychopathic traits in severely at-risk juvenile samples after involvement in the intensive decompression approach (M. F. Caldwell, 2011). With the advancement of effective treatment intervention approaches for youth scoring high on measures of psychopathy, researchers have attempted to identify abnormalities early in development that are consistent with adult psychopathic offenders, in order to potentially identify youth who may benefit most from treatment.

Due to the pejorative label of psychopathy, combined with the poor outcome measures typically associated with adult psychopathic offenders, including poor treatment outcomes and increased recidivism rates (Hemphill et al., 1998; Rice & Harris, 1997), researchers do not refer to youth scoring high on measures of psychopathy as 'psychopaths'. Rather, researchers have referred to such youth as scoring high on "*callous-unemotional (CU)*" traits, reflecting Factor 1 psychopathic traits, or youth scoring high on psychopathic traits, reflecting individuals scoring high on both Factor 1 and Factor 2 psychopathic traits, to reduce the stigma associated with the term psychopathy and to additionally provide optimism that these extremely at-risk youth can be treated successfully.

In order to assess psychopathic traits in youth and adolescent samples, a downward extension of the PCL-R was developed, referred to as the Hare Psychopathy Checklist: Youth Version (PCL:YV) (Forth, Hart, & Hare, 1990; Forth, Kosson, & Hare, 2003). The twenty items comprising the PCL-R were modified for age appropriateness to establish the twenty items of the PCL:YV, which include the following: (1) impression management, (2) a grandiose sense of self-worth, (3) stimulation seeking, (4) pathological lying, (5) manipulation for personal gain, (6) a lack of remorse, (7) shallow affect, (8) callousness and/or a lack of empathy, (9) a parasitic lifestyle, (10) poor anger controls, (11) impersonal sexual behavior, (12) early behavioral problems, (13) lacking goals, (14) impulsivity, (15) irresponsibility, (16) a failure to accept responsibility for their actions, (17) unstable interpersonal relationships, (18) serious criminal behavior, (19) serious violations of conditional release, and (20) criminal versatility (Forth et al., 2003). Scoring of the PCL:YV is incredibly similar to that of the PCL-R and results in

the same factor and facet structure previously reported in adult samples (Neumann, Kosson, Forth, & Hare, 2006).

In addition to the PCL-R, researchers have established assessments to measure psychopathic traits using parent- or caregiver-report. However, psychopathic traits are often better assessed using self-report compared to parent- or caregiver-report (Jolliffe et al., 2003; Kamphaus & Frick, 2002) as incarcerated youth tend to have low levels of adult supervision and typically come from families where parents and/or caregivers have not had enough recent contact with the adolescent to provide current ratings of their true characteristics (Fink, Tant, Tremba, & Kiehl, 2012; Loney, Frick, Clements, Ellis, & Kerlin, 2003). Therefore, there often is an increased discrepancy between parent/caregiver-report and self-report in incarcerated youth (De Los Reyes & Kazdin, 2005).

Expanding upon self-report measures of psychopathic traits in adolescent samples, it appears that the PCL:YV and self-report measures of adolescent psychopathic traits should not be used interchangeably. For example, self-report measures of adolescent psychopathic traits have shown poor classification agreement across measures (Cauffman, Kimonis, Dmitrieva, & Monahan, 2009; Fink et al., 2012; Lee, Vincent, Hart, & Corrado, 2003; Skeem & Cauffman, 2003). Self-report measures of adolescent psychopathic traits have also shown to be poorer predictors of delinquency and antisocial indices compared to the PCL:YV (Boccaccini et al., 2007; Cauffman et al., 2009; Douglas, Epstein, & Poythress, 2008; Fink et al., 2012; Sharp & Kine, 2008; Spain, Douglas, Poythress, & Epstein, 2004). Finally, some studies have been performed showing dissimilar executive functioning (Baskin-Sommers et al., 2015), functional

neuroimaging (Harenski, Harenski, & Kiehl, 2014), and error-related processing (Maurer et al., 2018) deficits when incorporating the PCL:YV and self-report measures of adolescent psychopathic traits within the same sample. These results suggest that self-report measures of adolescent psychopathic traits do not provide a compatible assessment of psychopathic traits as measured via the PCL:YV (Fink et al., 2012).

Psychopathic traits, at least in low to moderate levels, typically reduce naturally for a large majority of youth (Frick, 2009; Lee, Klaver, Hart, Moretti, & Douglas, 2009; Lynam, Caspi, Moffitt, Loeber, & Stouthamer-Loeber, 2007). Some psychopathic traits, including impulsivity, irresponsibility, and stimulation seeking are often seen as quite normative in typical adolescent development (Seagrave & Grisso, 2002). However, for a subsample of youth scoring high on psychopathic traits, the disorder appears to remain stable throughout development (Blonigen, Hicks, Krueger, Patrick, & Iacono, 2006; Frick et al., 2003; Hemphälä, Kosson, Westerman, & Hodgins, 2015; Lynam, 1997; Lynam et al., 2007; Neumann, Wampler, Taylor, Blonigen, & Iacono, 2011; Obradovic, Pardini, Long, Loeber, & 2007, 2007). Much of the research investigating psychopathic traits in youth and adolescent samples have focused on CU traits (i.e., PCL:YV Factor 1 traits), which are distinguished by a blatant disregard for others, a lack of empathy, and affective deficits (Frick, 2009). Higher levels of CU traits typically help distinguish youth on a life-course persistent trajectory towards severe antisocial behavior (Frick, 2009). CU traits were added to the DSM-V as a specifier for the diagnosis of conduct disorder (CD) (American Psychiatric Association, 2013), with the DSM-V using the term "*with limited prosocial emotions*". Although CU traits in youth reflect the interpersonal and affective dysfunction (i.e., PCL:YV Factor 1 traits) characteristic of adult psychopathic offenders,

others have viewed lifestyle (i.e., PCL:YV Facet 3) psychopathic traits as the most important predictor for identifying youth on a life-course persistent trajectory towards severe antisocial behavior (Lynam, 1997). These traits are typically highly associated with delinquency and antisocial indices (Frick, Bodin, & Barry, 2000; Lynam, 1997). Similar to adult psychopathic offenders, it appears to be the conglomeration of interpersonal, affective, lifestyle, and antisocial/developmental traits that help distinguish youth scoring high on measures of psychopathic traits from other externalizing disorders, including youth who meet criteria for CD or Oppositional Defiant Disorder (ODD) (Neumann et al., 2011). Youth who meet criteria for these latter externalizing disorders are more likely to meet the criteria for APD as adults as opposed to psychopathy (Frick & Nigg, 2011; Simonoff et al., 2004).

Compared to youth strictly meeting criteria for externalizing disorders, including CD and ODD, youth with elevated psychopathic traits exhibit a number of comparable neurocognitive deficits as adult psychopathic offenders, including reduced sensitivity to punishment cues (Vitale et al., 2005), passive avoidance learning (Finger et al., 2008), and perspective-taking deficits (Cheng, Hung, & Decety, 2012). In addition, youth scoring high on measures of psychopathy exhibit dysfunctional processing in the amygdala, including reduced responsiveness to fearful facial expressions (Jones, Laurens, Herba, Barker, & Viding, 2009; Marsh et al., 2008; Viding et al., 2012; White et al., 2012). Youth meeting criteria for CD typically exhibit the opposite pattern, with increased hemodynamic activity in the amygdala when processing fearful facial expressions (Herpertz et al., 2008; Passamonti et al., 2010). This is consistent with the idea that externalizing disorders are related to emotional dysfunction and threat

sensitivity differences, compared to the reduced empathic response typically associated with youth scoring high on measures of psychopathy (Frick, 2012).

Structural neuroimaging studies reveal brain abnormalities in youth with elevated psychopathic traits that are consistent with adult psychopathic offenders. For example, youth scoring high on measures of psychopathy exhibit reduced gray matter volume and concentration in the amygdala (Aghajani, Klapwijk, et al., 2016; B. M. Caldwell et al., 2019; Cohn et al., 2016; Walters & Kiehl, 2015), ACC (B. M. Caldwell et al., 2019; Sebastian et al., 2016), caudate (B. M. Caldwell et al., 2019), fusiform gyrus (Fairchild et al., 2011), hippocampus (Cope, Ermer, Nyalakanti, Calhoun, & Kiehl, 2014; Wallace et al., 2014), insula (B. M. Caldwell et al., 2019; Cohn et al., 2016; Raschle et al., 2018), middle frontal gyrus (Sebastian et al., 2016), parahippocampal gyrus (Cope et al., 2014; Ermer, Cope, Nyalakanti, Calhoun, & Kiehl, 2013), PCC (B. M. Caldwell et al., 2019; Ermer et al., 2013), putamen (B. M. Caldwell et al., 2019), orbitofrontal cortex (OFC) (B. M. Caldwell et al., 2019; Cope et al., 2014; Ermer et al., 2013; Sebastian et al., 2016), and temporal pole (B. M. Caldwell et al., 2019; Cope et al., 2014; Ermer et al., 2013; Steele, Rao, Calhoun, & Kiehl, 2017; Wallace et al., 2014). However, other published studies have found that youth with elevated psychopathic traits exhibited *increased* gray matter volume in several regions, including the ACC (B. M. Caldwell et al., 2019; De Brito et al., 2009), caudate (Fairchild et al., 2011), medial prefrontal cortex (mPFC) (Ermer et al., 2013), insula (B. M. Caldwell et al., 2019), nucleus accumbens (NAcc) (Fairchild et al., 2011), OFC (B. M. Caldwell et al., 2019; De Brito et al., 2009), and temporal pole (De Brito et al., 2009). Disparate results between studies may be due to differences related to the specific assessment used to measure psychopathic traits (B. M.

Caldwell et al., 2019) or due to important differences related to structural MRI data analysis, including failing to control for head size in analyses performed (De Brito et al., 2009).

With functional magnetic resonance imaging (fMRI), studies have reported a number of abnormalities in youth scoring high on measures of psychopathy. Compared to youth scoring lower on measures of psychopathic traits, youth scoring high on measures of psychopathic traits have exhibited the following deficits: (1) reduced hemodynamic activity in the OFC and caudate during stimulus-reinforcement exposure (Finger et al., 2011), (2) reduced hemodynamic activity in the OFC when processing rewards (Finger et al., 2011), (3) reduced hemodynamic activity in the amygdala during reward feedback (Cohn et al., 2015), (4) increased hemodynamic activity in the vmPFC and caudate during reversal learning (Finger et al., 2008), (5) reduced hemodynamic activity in the amygdala during the processing of fearful face stimuli (Jones et al., 2009; Lozier, Cardinale, VanMeter, & Marsh, 2014; Marsh et al., 2008; Viding et al., 2012; White et al., 2012), (6) reduced hemodynamic activity in the amygdala, hippocampus, insula, caudate, globus pallidus, and ACC during drug cue reactivity (Vincent, Cope, King, Nyalakanti, & Kiehl, 2017), (7) reduced hemodynamic activity in the amygdala when processing unpleasant pictures (Harenski et al., 2014), (8) reduced hemodynamic activity in the ACC (Lockwood et al., 2013; Marsh et al., 2013), amygdala (Marsh et al., 2013), putamen (Marsh et al., 2013), and insula (Lockwood et al., 2013; Marsh et al., 2013) when viewing others in pain, (9) reduced hemodynamic activity in the insula when assessing harm intention (Michalska, Zeffiro, & Decety, 2016), (10) reduced hemodynamic activity in the insula when deciding whether an action was legal or illegal (Marsh et al., 2011),

(11) reduced hemodynamic activity in the amygdala during affective theory of mind processing (Sebastian et al., 2012), (12) increased hemodynamic activity in the insula, ACC, and caudate when punishing unfair offers (White, Brislin, Meffert, Sinclair, & Blair, 2013), and (13) reduced hemodynamic activity in subregions of the basal ganglia, including the caudate, NAcc, globus pallidus, substantia nigra, and subthalamic nucleus during error-related processing (Maurer et al., in press).

Additionally, a few studies have been performed investigating white matter (WM) integrity in youth with elevated psychopathic traits. One study performed found that youth scoring high on psychopathic traits exhibited reduced fractional anisotropy (FA) in the right uncinate fasciculus (UF) (Breeden, Cardinale, Lozier, VanMeter, & Marsh, 2015), consistent with adult psychopathic offenders (Craig et al., 2009; Hoppenbrouwers et al., 2013; Motzkin, Newman, Kiehl, & Koenigs, 2011; Sobhani, Baker, Martins, Tuvblad, & Aziz-Zadeh, 2015; Sundram et al., 2012; Vermeij, Kempes, Cima, Mars, & Brazil, 2018; Wolf et al., 2015). However, other studies performed found that youth scoring higher on measures of psychopathic traits exhibited increased FA in the right UF (Pape et al., 2015; Sarkar et al., 2013). Youth with elevated psychopathic traits have also exhibited increased FA values in a number of WM tracts extending beyond the right UF, including the anterior thalamic radiation, corticospinal tract, forceps minor, inferior fronto-occipital fasciculus, and corpus callosum (Pape et al., 2015).

Goals of Current Dissertation

In this dissertation, we sought to expand upon previous studies identifying neurocognitive deficits associated with youth with elevated psychopathic traits. Most of these previous studies performed have investigated affective dysfunction in youth with

elevated psychopathic traits. To date, relatively few studies have been performed identifying cognitive control deficits in youth with elevated psychopathic traits. Such youth have been characterized by reduced amplitude of the error-related positivity (Pe) ERP component (Maurer, Steele, Cope, et al., 2016) and reduced hemodynamic activity in subregions of the basal ganglia during error-related processing (Maurer et al., in press). In this dissertation, we sought to expand upon previous research in the following ways: First, in addition to error-related processing deficits, we investigated potential response inhibition deficits in youth with elevated psychopathic traits, and second, we investigated cognitive control processes through the use of Independent Component Analysis (ICA) to identify potential functional connectivity deficits in youth with elevated psychopathic traits.

In this dissertation, we also hoped to expand upon the limited number of studies that have been performed identifying abnormalities in WM tracts in youth with elevated psychopathic traits (Breeden et al., 2015; Pape et al., 2015; Sarkar et al., 2013). Disparate results between previous studies (e.g., reduced FA in the right UF (Breeden et al., 2015) and increased FA in the right UF (Pape et al., 2015; Sarkar et al., 2013)) may be related to the specific operational definition of adolescent psychopathy used in the study. For example, Sarkar et al. (2013) considered youth with a PCL:YV total score 20 or above to be part of the psychopathic group, which is 10 points lower than the traditional cut-off score used (Forth et al., 2003). As such, some studies may actually be measuring youth meeting criteria for externalizing disorders, including CD and ODD, populations that typically exhibit increased FA values in the UF (Sarkar et al., 2013; Zhang et al., 2014) instead of youth characterized by elevated psychopathic traits. However, increased FA in

the UF may also reflect a developmental abnormality in youth with elevated psychopathic traits, including pruning failure or decreases in axonal diameter, packing density, or branching (Beaulieu, 2002). Thus, we sought to investigate whether youth with elevated psychopathic traits were associated with increased or reduced FA values in the right UF and additional WM tracts.

Finally, in this dissertation, we hoped to expand upon a burgeoning line of research identifying neurobiological variables predictive of future recidivism. Youth scoring higher on the PCL:YV are typically associated with an increased recidivism rate (Corrado, Vincent, Hart, & Cohen, 2004; Vincent, Odgers, McCormick, & Corrado, 2008), but in addition, variables including age at release (Baglivio et al., 2016; Mallett, Fukushima, Stoddard Dare, & Quinn, 2012; Parsons Winokur, Smith, Bontrager, & Blankenship, 2008), substance use severity (Colins et al., 2011; Hoeve, McReynolds, Wasserman, & McMillan, 2013; Myner, Santman, & Cappelletty, 1998), intelligence (Vermeiren, Schwab-Stone, Ruchkin, De Clippele, & Deboutte, 2002), and impulsivity (Miner, 2002) are strong predictors of future recidivism in adolescent samples. However, these measures of risk assessment, personality traits, and neuropsychological measures are only serving as proxy measures for the direct measurement of the brain's inhibitory and cognitive control system. More direct measures of brain activity and structural integrity may lend incremental utility to the prediction of future recidivism. To date, three studies have been performed in incarcerated adult offenders, identifying neurobiological variables predictive of recidivism, including reduced hemodynamic activity in the ACC during error-related processing (Aharoni et al., 2013; Steele et al., 2015), increased amplitude of the Pe ERP component during error-related processing (Steele et al., 2015),

and a brain age model that predicted chronological age based on sMRI data (Kiehl et al., 2018). However, to date, no study has attempted to predict future recidivism outcomes using neurobiological variables in a high-risk sample of incarcerated adolescent offenders. We sought to replicate an existing study whereby increased Pe amplitude was predictive of recidivism in adult offenders (Steele et al., 2015) and also whether structural integrity abnormalities via diffusion tensor imaging (DTI) were predictive of future recidivism in a sample of adolescent offenders.

Study 1: Identifying functional connectivity deficits during error-related processing and response inhibition in youth with elevated psychopathic traits

INTRODUCTION

Relatively little research has been performed investigating cognitive control deficits, including response inhibition and error-related processing deficits, in youth scoring high on measures of psychopathy. Brain regions critical to successful response inhibition and error-related processing, including the ACC and subregions of the basal ganglia have been shown to be dysfunctional in youth with elevated psychopathic traits (Lockwood et al., 2013; Marsh et al., 2013; Maurer et al., in press). However, a number of other regions implicated in successful cognitive control have been infrequently studied in relation to youth with elevated psychopathic traits. Brain regions critical to successful response inhibition include the lateral and ventrolateral prefrontal cortex (Braver, Barch, Gray, Molfese, & Snyder, 2001; Horn, Dolan, Elliott, Deakin, & Woodruff, 2003; Liddle, Kiehl, & Smith, 2000), inferior frontal gyrus (Aron, Robbins, & Poldrack, 2004; Garavan, Ross, Murphy, Roche, & Stein, 2002; Mathalon, Whitfield, & Ford, 2003; Steele et al., 2013), inferior parietal lobule (Braver et al., 2001; Garavan et al., 2002; Liddle et al., 2000; Mathalon et al., 2003; Menon, Adleman, White, Glover, & Reiss, 2001; Steele et al., 2013; Wager et al., 2005), pre-supplementary motor area (Mostofsky et al., 2003), ACC (Garavan et al., 2002; Kiehl, Liddle, & Hopfinger, 2000; Liddle et al., 2000; Steele et al., 2013), cuneus (Garavan, Ross, Kaufman, & Stein, 2003; Liddle et al., 2000; Tian & Yao, 2008), and the thalamus and subregions of the basal ganglia (Aron et al., 2007; Garavan et al., 2002; Mathalon et al., 2003; Menon et al., 2001; Steele et al., 2013; Wager et al., 2005). Important to note, the brain regions responsible for error-

related processing often overlap with those involved in successful response inhibition (Braver et al., 2001; Garavan et al., 2002; Horn et al., 2003; Kiehl, Liddle, et al., 2000; Liddle et al., 2000; Menon et al., 2001; Steele, Claus, et al., 2014).

To date, very few studies have been performed identifying response inhibition deficits in youth scoring high on measures of psychopathy. The N2 ERP component is thought to measure inhibitory processing, as the amplitude of the N2 is larger in response to "NoGo" stimuli than to "Go" stimuli (Folstein & Van Petten, 2008). There is also support for the notion that the N2 reflects response conflict, as there is a competition between NoGo and Go responses (Nieuwenhuis, Yeung, Van Den Wildenberg, & Ridderinkhof, 2003). In relation to youth with elevated psychopathic traits, one study found that higher scores on a self-report measure of adolescent psychopathic traits (the Antisocial Process Screening Device (Frick & Hare, 2001)) were positively correlated with amplitude of the N2 ERP component during response inhibition on a Go/NoGo task (Sumich, Sarkar, Hermens, Kelesidi, et al., 2012), which is consistent with one previously published report with adult psychopathic offenders (Munro et al., 2007), but inconsistent with another previously published report with adult psychopathic offenders (Kiehl, Smith, et al., 2000). Increased N2 amplitude during response inhibition in youth with elevated psychopathic traits may reflect a potential compensatory mechanism to correctly perform the Go/NoGo task, as the amplitude of the N2 typically declines between childhood and adolescence, reflecting improvements in cognitive control (Sumich, Sarkar, Hermens, Ibrahimovic, et al., 2012).

Similar to response inhibition processes, very little research has been performed investigating error-related processing deficits in youth with elevated psychopathic traits.

One study found that youth scoring high on the PCL:YV exhibited comparable amplitude of the error-related negativity (ERN/Ne) and reduced amplitude of the Pe compared to youth scoring low on the PCL:YV (Maurer, Steele, Cope, et al., 2016). While the ERN/Ne is thought to reflect initial, automatic error-detection and action-monitoring processes (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Coles, Meyer, & Donchin, 1990), the Pe is involved in later, more elaborate error-processing stages, including assessing the motivational (Ullsperger, Harsay, Wessel, & Ridderinkhof, 2010) or affective (Overbeek, Nieuwenhuis, & Ridderinkhof, 2005) significance or incorrect responses. This report suggests that youth scoring high on the PCL:YV do not exhibit deficits in the initial identification of an error, but exhibit deficits in processing the motivational or affective significance of error-related information (Maurer, Steele, Cope, et al., 2016). These results are consistent with those previously reported in adult psychopathic offenders (Brazil et al., 2009; Maurer, Steele, Edwards, et al., 2016; Steele, Maurer, et al., 2016). Like the increased N2 observed in youth with elevated psychopathic traits (Sumich, Sarkar, Hermens, Kelesidi, et al., 2012), the reduced amplitude of the Pe in youth with elevated psychopathic traits suggests a potential developmental abnormality. Compared to the amplitude of the ERN/Ne, which increases in amplitude throughout development, reflecting improved efficiency in error-detection and action-monitoring processes, the amplitude of the Pe is invariant throughout development, showing comparable amplitude between youth and adult samples (Davies, Segalowitz, & Gawin, 2004; Ladouceur, Dahl, & Carter, 2007; Santesso, Segalowitz, & Schmidt, 2006).

A recent fMRI study has also investigated error-related processing deficits in youth with elevated psychopathic traits. In this report, PCL:YV scores were negatively related to hemodynamic activity during error-processing in subregions of the basal ganglia, including the caudate, NAcc, globus pallidus, substantia nigra, and subthalamic nucleus (Maurer et al., in press). Dysfunction occurring within these subregions of the basal ganglia has important implications for error-related processing in general. Subregions of the basal ganglia have been shown to play an important, though indirect role in error-related processing, as they help monitor and steadily predict the result of ongoing events (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Mathalon et al., 2003), determining whether the end result of events will be favorable or not (Holroyd & Coles, 2002). When encountering events that deviate from expectancies, including the commission of errors, the basal ganglia relays information to regions of the salience network, including the dorsal ACC (Ham, Leff, de Boissezon, Joffe, & Sharp, 2013; Kennerley, Walton, Behrens, Buckley, & Rushworth, 2006) via the thalamus. The ACC then signals the need for increased cognitive control to regions involved in the executive control network, including lateral prefrontal structures (Egner, 2009). The results obtained in this fMRI study suggest that error-related processing deficits previously measured with ERPs (Maurer, Steele, Cope, et al., 2016) may be due in part to underlying dysfunction occurring downstream in subregions of the basal ganglia (Maurer et al., in press), as the Pe is believed to arise from the ACC (Edwards, Calhoun, & Kiehl, 2012; van Veen & Carter, 2002). However, to date, investigating functional connectivity with brain regions critical for cognitive control processes, including response inhibition and

error-related processing, has never been investigated in relation to youth with elevated psychopathic traits.

Functional connectivity investigates the correlations between neural activity between spatially distinct brain regions (Friston, 1994). Group Independent Component Analysis (ICA) can be applied to fMRI data to identify spatially distinct and temporally coherent components of brain activity (Calhoun, Adali, McGinty, et al., 2001). ICA can provide a measure of both functional connectivity and task-relatedness, allowing for the identification of brain networks that are comprised of multiple brain regions, as well seeing which networks may be implicated in various forms of psychopathology (Erhardt et al., 2011). ICA is a data-driven, multivariate approach and eliminates the arbitrary choice of *a priori* seed regions. As such, ICA may more sensitive and be able to detect subtle differences between subjects compared to traditional seed-based approaches (W. Koch et al., 2010).

Relatively few studies have investigated functional connectivity in relation to youth with elevated psychopathic traits. Aghajani and colleagues (2016) found that youth scoring high on psychopathic traits exhibited reduced functional connectivity between the basolateral amygdala and a cluster including the ACC, mPFC, PCC, and striatal regions and increased functional connectivity between the centromedial amygdala with a cluster including the vmPFC and OFC during a resting-state fMRI task (Aghajani, Klapwijk, et al., 2016). In another study, Aghajani and colleagues (2016) found that youth scoring high on psychopathic traits exhibited reduced functional connectivity between the centromedial amygdala and a cluster including the dorsal and ventral ACC and DLPFC and increased functional connectivity between the basolateral amygdala and a cluster

containing the posterolateral PFC, DLPFC, VLPFC, and rostromedial PFC (Aghajani, Colins, et al., 2016). Marsh et al. (2008) reported that CU symptom severity was associated with reduced functional connectivity between the amygdala and vmPFC during a fear perception task (Marsh et al., 2008). Important to note, many of the aforementioned functional connectivity studies incorporated resting-state fMRI tasks, rather than examining specific experimental paradigms, including those investigating various forms of cognitive control. Some have questioned the value of resting-state functional connectivity, suggesting that observations made during resting-state conditions cannot be applied to other cognitive processes (Callard & Marguiles, 2014; Morcom & Fletcher, 2007). In addition, these resting-state functional connectivity studies relied on seed-based functional connectivity analyses, which is associated with their own set of limitations. For example, studies typically use the same ROI as the seed region for all subjects, which may be highly variable between subjects (Sohn et al., 2015). Seed-based analyses also require an *a priori* selection of a voxel or cluster, which may bias connectivity towards specific, smaller, or overlapping sub-systems, rather than larger, distinct networks (Buckner, Andrews-Hanna, & Schacter, 2008). Thus, it may be beneficial to investigate functional connectivity during specific experimental paradigms, including those measuring cognitive control processes, using a data-driven, multivariate approach, such as ICA.

In Study 1, we sought to expand upon previous research which has identified response inhibition (Sumich, Sarkar, Hermens, Kelesidi, et al., 2012) and error-related processing deficits (Maurer, Steele, Cope, et al., 2016; Maurer et al., in press) in youth with elevated psychopathic traits by examining such processes using ICA functional

connectivity analyses. We specifically hypothesized that compared to youth scoring low on the PCL:YV, youth with elevated psychopathic traits would exhibit increased functional connectivity during response inhibition and reduced functional connectivity during error-related processing, consistent with previously performed studies incorporating ERPs and fMRI (Maurer, Steele, Cope, et al., 2016; Maurer et al., in press; Sumich, Sarkar, Hermens, Kelesidi, et al., 2012).

METHOD

Participants

Participants in Study 1 included $n = 327$ incarcerated male adolescent offenders recruited from juvenile correctional facilities in the state of New Mexico and Wisconsin. Participants were excluded from analyses for meeting the following criteria: occurrence of traumatic brain injury (TBI) accompanied with a significant loss of consciousness ($n = 9$) or an estimated intelligence quotient (IQ) less than 70 ($n = 15$). Additionally, participants were excluded for meeting diagnostic criteria for anxiety disorders, as these disorders have been shown to influence hemodynamic activity during error-related (Fitzgerald et al., 2005) and response inhibition (Carrion, Garrett, Menon, Weems, & Reiss, 2008) processing. Participants were excluded for meeting criteria for generalized anxiety disorder (GAD) ($n = 3$) and post-traumatic stress disorder (PTSD) ($n = 22$). Additionally, participants were excluded for meeting criteria for psychosis ($n = 1$) and gender dysphoria ($n = 1$). Two additional participants were excluded for completing only one run of the Go/NoGo experimental paradigm. This resulted in a final sample of $n = 274$ incarcerated male adolescent offenders, ranging from 13 to 20 years of age ($M = 17.25$ years, $SD = 1.22$ years) at the time of MRI data collection. The sample was

predominantly right-handed (7% reported being left-hand dominant). Participants largely self-identified as Hispanic/Latino (55%), with the remaining self-identifying as Black or African American (21%), White (15%), American Indian or Alaskan Native (7%), or Native Hawaiian or other Pacific Islander (1%). One percent of the sample chose not to disclose their race or ethnicity.

Initial contact was made with potential study participants and informed consent was obtained. Individuals 18 years of age or older provided written informed consent and individuals younger than 18 years of age provided written informed assent in conjunction with parent/guardian consent. Participants were informed of their right to terminate participation at any point, the lack of institutional benefits, and that their participation would not affect their facility status or parole. Participants received remuneration at the hourly labor wage of the facility. All research protocols were approved by the Ethical and Independent Review Services (E&I), the Office for Human Research Protections (OHRP), and the juvenile detention center where data collection occurred.

Assessments

Psychopathic traits were assessed using the Hare PCL:YV (Forth et al., 2003). The mean PCL:YV total score for this sample was 24.71 (SD = 6.20) (ranging from 2 to 38). The Cronbach's alpha for the PCL:YV (all items) was .80 in the current sample, reflecting good internal consistency. Sixty-seven participants scored high on the PCL:YV (i.e., a PCL:YV total score 30 or above, M total score = 32.34, SD = 2.11) and n = 67 scored low on the PCL:YV (i.e., a PCL:YV total score 20 or below, M total score = 16.46, SD = 3.45) and these groups were used in subsequent two-sample t-test analyses described below. In addition to investigating psychopathic traits, additional assessments

were also administered to assess intelligence quotient (IQ), psychopathology, and TBI. Full-scale IQ was estimated using the Vocabulary and Matrix Reasoning sub-tests of the Wechsler Adult Intelligence Scale – 3rd Edition (WAIS-III) (Wechsler, 1997) for participants sixteen years of age or older and from the Wechsler Intelligence Scale for Children – 4th Edition (WISC-IV) (Wechsler, 2003) for participants younger than sixteen years of age ($M = 90.62$, $SD = 11.55$). Psychopathology was assessed using the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS) (Kaufman, Birmaher, & Brent, 1997). Additionally, history of TBI was evaluated using a post-head injury symptoms questionnaire (King, Crawford, Wenden, Moss, & Wade, 1995).

Go/NoGo Experimental Paradigm

Participants performed a response inhibition Go/NoGo fMRI experimental paradigm (Kiehl, Liddle, et al., 2000) consisting of two experimental runs, each comprising 245 visual stimuli. The stimuli were presented to participants using the computer-controlled visual and auditory software package, Presentation (www.neurobs.com). Each stimulus appeared for 250 milliseconds (ms) in white text within a continuously displayed rectangular fixation box. Participants were instructed to respond as quickly and accurately as possible with their right index finger via a button box every time the target ("Go") stimulus (a white letter "X") appeared and to withhold responding when the distracter ("NoGo") stimulus (a white letter "K") appeared. Targets appeared with higher frequency (84% of trials, 412 total trials, with 206 on each run) than distracters (16% of trials, 78 total trials, with 39 on each run) to establish a strong stimulus-response mapping on Go trials. Two NoGo stimuli were never presented sequentially. The stimuli were approximately 3 x 5 visual degrees against a black

background. The inter-stimulus interval was jittered (1 – 3 seconds stimulus onset asynchrony [SOA]), averaging 1.5 seconds. The SOA between Go stimuli was varied pseudo-randomly between 1000, 2000, and 3000 ms, subject to the constraint that three Go stimuli were presented within each six-second period. The NoGo stimuli were interspersed among the Go stimuli in a pseudo-random manner subject to two constraints: the minimum SOA between Go and NoGo stimuli was 1000 ms and the SOA between successive NoGo stimuli was in the range of eight to fourteen seconds. Behaviorally, "Hits" were defined as successful behavior responses to the Go stimuli, "Correct Rejects (CR's)" were defined as successfully inhibiting a behavioral response to NoGo stimuli, and "False Alarms (FA's)" were defined as incorrect behavioral responses to NoGo stimuli. Prior to fMRI recording, each participant performed a block of ten practice trials to ensure task instructions were clearly understood.

Imaging Parameters and Preprocessing

Images were collected using the Mind Research Network's mobile Siemens 1.5 Tesla (T) Avanto stationed at the juvenile correctional facilities where participants were housed, with advanced SQ gradients (max slew rate 200 T/m/s 346 T/m/s vector summation, rise time 200 microseconds (μs)) equipped with a twelve-element head coil. The EPI gradient-echo pulse sequence (TR/TE 2000/39 ms, flip angle 75°, FOV 24 x 24 cm, 64 x 64 matrix, 3.4 x 3.4 mm in plane resolution, 5 mm slice thickness, 30 slices) effectively covers the entire brain (150 mm) in 2000 ms. Head motion was limited using padding and restraint, and was evaluated using INRIalign, a mutual information algorithm unbiased by local signal change (Freire & Mangin, 2001; Freire, Roche, & Mangin, 2002). No participants were excluded due to excessive motion, defined as

movement greater than two SD's away from the mean (i.e., greater than 2 mm translation or 1.5° rotation).

Functional images were reconstructed offline at 16-bit resolution and manually reoriented to approximately the anterior commissure/posterior commissure (AC/PC) plane. Functional images were spatially normalized to the Montreal Neurological Institute (MNI) template via a parameter affine transformation using smooth basis functions to account for nonlinear differences and spatially smoothed 8 mm full-width at half maximum) in the Statistical Parametric Mapping 12 (SPM12) software package (http://www.fil.ion.ucl.ac.uk/spm/software/spm12/) (Calhoun et al., 2017). Response types (CR's, FA's, and Hits) were modeled as separate events. Event-related responses were modeled using a synthetic response function composed of two gamma functions. The first gamma function modeled the hemodynamic response using a peak latency of six seconds. A term proportional to the derivative of this gamma function was used to model the small "overshoot" of the hemodynamic response on recovery. A latency variation amplitude-correction method was used to provide a more accurate estimate of the hemodynamic response for each condition that controlled for differences between slices, timing, and variation across regions in the latency of the hemodynamic response (Calhoun, Stevens, Pearlson, & Kiehl, 2004).

Independent Component Analysis

Functional connectivity during the Go/NoGo experimental paradigm was assessed using group ICA (Calhoun & Adali, 2012; Calhoun, Adali, Pearlson, & Pekar, 2001a). ICA is an application of blind source separation that decomposes an fMRI dataset into maximally spatially independent components and their associated time courses. ICA was

performed using the Group ICA of fMRI (GIFT) toolbox version 4.01a

(http://mialab.mrn.org/software/gift). Following SPM12 pre-processing, a group ICA was

performed on the pre-processed fMRI data (Calhoun, Adali, et al., 2001a). The methods

prescribed by these processes were organized in batch scripts and performed via the

group ICA of fMRI (GIFT) MATLAB toolbox version 3.0b

(http://mialab.mrn.org/software/gift/index.html). fMRI time series data for all participants

were first compressed through principal component analysis (PCA). A two-stage PCA

data reduction step was implemented: at the single-subject level, 150 principal

components (PC's) were extracted, followed by a group PCA step using 75 components

(Erhardt et al., 2011). We chose to investigate 75 ICA components as it has been

suggested that a higher number of components (i.e., 70 to 100) consistently and stably

estimates the data (Abou-Elseoud et al., 2010; Allen et al., 2011; Steele et al., 2017;

Ystad, Eichele, Lundervold, & Lundervold, 2010). The data reduction was followed by a

group spatial ICA, performed on the participant's aggregate data, resulting in the final

estimation of our independent components (Calhoun, Adali, Pearlson, & Pekar, 2001b).

The algorithm used in this process was the infomax algorithm, which attempts to

minimize the mutual information of network outputs (Bellemann et al., 1995).

From the group spatial ICA, we reconstructed spatial maps and their

corresponding ICA time courses that represented both the spatial and temporal

characteristics of each component, subject, and session (Erhardt et al., 2011). These maps

and time courses were then subjected to a second-level analysis to determine whether the

IC's were task-related as well to determine which components reflected non-artifact

networks.

We averaged the spatial maps produced during the spatial ICA across the two sessions. The spatial maps were converted to *z*-score maps and then entered into a second-level one-sample *t*-test to identify voxels that contributed significantly to a given component for the group (Calhoun, Adali, et al., 2001a). Next, these components were analyzed statistically and compared to group-specific thresholds to observe trends in modulation among the subjects.

We performed a temporal sorting of the ICA time courses using an SPM12 design matrix corresponding to the Go/NoGo stimuli. Temporal sorting is a method by which we can compare the model's time course with the ICA time course. Using a multiple linear regression sorting criteria, the concatenated ICA time courses were fit to the model time course. This resulted in a set of beta weights for each regressor associated with a particular subject and IC. The value of the resulting beta weight indicates the degree to which the component was modulated by the task. We also calculated the event-related averages of the time courses for all components. Each plot of the event-related average depicts the level of task-related functional activity for that particular component over the course of the experimental period.

Statistical Analyses

For each IC in this study, we performed a number of statistical analyses on the beta weights resulting from the ICA. These analyses included calculating the mean and standard deviation of the beta weights and one- and two-sample *t*-tests on the beta weights. The mean and standard deviation for task modulation of the hemodynamic response were calculated for the entire set of subjects and for the score-related subject subgroups. Next, one- and two-sample *t*-tests were performed on the beta weights

obtained for each component to identify significant differences in modulation within and among the various participant groups. The one-sample *t*-test (one degree of freedom) provides information on the degree and direction of the task modulation for all participants within particular brain regions, whereas the two-sample *t*-test (two degrees of freedom) compares the differences in modulation between the low-scoring PCL:YV group (*n* = 67) and the high scoring PCL:YV group (*n* = 67). The two-sample *t*-test allows the opportunity to compare differences in the degree to which certain brain regions exhibited a hemodynamic response to the task.

RESULTS

Behavioral Results

Response times (RTs) and frequency for Hits and FA's were analyzed. As expected, participants responded faster to NoGo stimuli (*M* = 403 ms, *SD* = 81 ms) than Go stimuli (*M* = 448 ms, *SD* = 118 ms), *t*(273) = 21.82, *p* < .001. Participants made significantly more errors to NoGo stimuli (*M* = 26.68, *SD* = 11.69) compared to Go stimuli (*M* = 13.49, *SD* = 26.43), *t*(273) = 7.87, *p* < .001. A main effect for post-error slowing (PES) was observed (Rabbit, 1981) (*M* = 42 ms, *SD* = 109 ms), *t*(273) = 6.45, *p* < .001. PES was defined as the difference in RT for Go stimuli preceded by a correct response to NoGo stimuli versus an incorrect response to NoGo stimuli. Thus, incorrect responses to NoGo stimuli should result in a subsequent behavioral slowing to Go stimuli compared to correct responses to NoGo stimuli (Rabbit, 1981). Indeed, participants responded more slowly after error trials (*M* = 457 ms, *SD* = 89 ms) compared to correct trials (*M* = 414 ms, *SD* = 67 ms) in the current report.

ICA Results

We performed a full statistical analysis on the group ICA results. Seventy-five ICs were estimated through group ICA. We removed components that were related to motion artifacts or correlated with spatial maps for white matter, cerebral spinal fluid, or the ventricular system (Stevens, Kiehl, Pearlson, & Calhoun, 2007). Next, we tested the remaining components for differences in task modulation between $n = 67$ low- and $n = 67$ high-scoring participants on the PCL:YV. Three components met the criteria for statistically significant associations with PCL:YV scores (i.e., corrected for Family-Wise Error Rate (FWE)), which included Components #24, 66, and 69. Youth scoring high on the PCL:YV exhibited increased functional connectivity with brain regions incorporated within Component 24, including the precuneus, superior parietal lobule, postcentral gyrus, paracentral gyrus, and cingulate gyrus compared to youth scoring low on the PCL:YV (see Figure 1). Additionally, youth scoring high on the PCL:YV exhibited increased functional connectivity with regions included in Component 66, including the ACC, medial frontal gyrus, superior frontal gyrus, middle frontal gyrus, and subcallosal gyrus compared to youth scoring low on the PCL:YV (see Figure 2). On the other hand, youth scoring high on the PCL:YV exhibited reduced functional connectivity with regions included in Component 69, including the insula, precentral gyrus, postcentral gyrus, superior temporal gyrus, inferior parietal lobule, and lentiform nucleus compared to youth scoring low on the PCL:YV (see Figure 3 and Table 1 for the list of MNI coordinates with descriptions of the associated brain regions for the chosen components).

DISCUSSION

Youth with elevated psychopathic traits have been previously characterized by a number of dysfunctional neurocognitive processes during a variety of experimental

paradigms. These tasks have primarily emphasized affective and emotional processing deficits, suggesting youth with elevated psychopathic traits exhibit reduced hemodynamic activity during fearful face processing (Jones et al., 2009; Lozier et al., 2014; Marsh et al., 2008; Viding et al., 2012; White et al., 2012), processing of harm intention (Michalska, Zeffiro, & Decety, 2016), affective theory of mind processing (Sebastian et al., 2012), or processing of unpleasant pictures (Harenski et al., 2014). Youth scoring high on measures of psychopathic traits have also been previously associated with reduced hemodynamic activity when viewing others in pain (Lockwood et al., 2013; Marsh et al., 2013) and increased hemodynamic activity when punishing unfair offers (White et al., 2013). In addition to affective and emotional processing deficits, youth with elevated psychopathic traits have also been characterized by reduced hemodynamic activity during stimulus-reward exposure (Finger et al., 2011), processing reward stimuli (Cohn et al., 2015; Finger et al., 2011), drug cue reactivity (Vincent et al., 2017), and when judging whether an action was legal or illegal (Marsh et al., 2011), and increased hemodynamic activity during reversal learning (Finger et al., 2008).

Surprisingly, relatively little research has been performed relating cognitive control deficits to youth with elevated psychopathic traits. Such youth have been previously characterized by response inhibition deficits, indexed by increased amplitude of the N2 ERP component (Sumich, Sarkar, Hermens, Kelesidi, et al., 2012). Furthermore, youth scoring high on the PCL:YV have previously exhibited error-related processing deficits, by exhibiting reduced amplitude of the Pe ERP component (Maurer, Steele, Cope, et al., 2016) and reduced hemodynamic activity in subregions of the basal ganglia, including the caudate, NAcc, globus pallidus, substantia nigra, and subthalamic

nucleus (Maurer et al., in press). No study to date has attempted to investigate functional connectivity deficits during cognitive control processes in youth with elevated psychopathic traits. Existing studies incorporating functional connectivity analyses have typically incorporated resting-state fMRI tasks and analyzed fMRI data using seed-based functional connectivity analyses. Here, we expanded upon previous research by investigating functional connectivity deficits during cognitive control via a data-driven, multivariate approach in ICA functional connectivity analyses.

In the current report, two IC's (i.e., IC 24 and 66) corresponded to response inhibition processes. Compared to youth scoring low on the PCL:YV, youth scoring high on the PCL:YV exhibited increased functional connectivity with brain regions incorporated within Components 24 and 66. Component 24 was comprised of the precuneus, superior parietal lobule, postcentral gyrus, paracentral lobule, and cingulate gyrus and Component 66 was comprised of the ACC, superior, medial, and middle portions of the frontal gyrus, and subcallosal gyrus. These regions are typically recruited when attempting to inhibit a prepotent behavioral response, including the ACC (Garavan et al., 2002; Kiehl, Liddle, et al., 2000; Liddle et al., 2000; Steele et al., 2013), cingulate gyrus (Horn et al., 2003; Stevens et al., 2007), superior frontal gyrus (Picton et al., 2006; Simmonds, Pekar, & Mostofsky, 2008; Steele et al., 2013; Stevens et al., 2007; Swick, Ashley, & Turken, 2008), medial frontal gyrus (Picton et al., 2006; Roth et al., 2007), middle frontal gyrus (Steele et al., 2013; Stevens et al., 2007; Swick et al., 2008), superior parietal lobule (Booth et al., 2003; Stevens et al., 2007) precuneus (Fu et al., 2008; Mobbs et al., 2007; Roth et al., 2007; Steele et al., 2013), postcentral gyrus (Ayalon, Ancoli-Israel, & Drummond, 2009; Roth et al., 2007), paracentral lobule (Aron

et al., 2007; Aron & Poldrack, 2006; Chambers, Garavan, & Bellgrove, 2009), and subcallosal gyrus (Bannbers et al., 2013; López-Caneda et al., 2012). Our results are consistent with the lone ERP study associating response inhibition dysfunction in youth with elevated psychopathic traits (Sumich, Sarkar, Hermens, Kelesidi, et al., 2012). Specifically, increased functional connectivity may reflect a potential compensatory mechanism (Hawelleck, Hipp, Lewis, Corbetta, & Engel, 2011; Iraji et al., 2015; Wolf et al., 2009) whereby youth scoring high on the PCL:YV recruit additional brain regions compared to youth scoring low on the PCL:YV when attempting to inhibit a prepotent response.

In addition to exhibiting increased functional connectivity during response inhibition, youth scoring high on the PCL:YV also exhibited reduced functional connectivity with brain regions in Component 69, which included the insula, precentral gyrus, superior temporal gyrus, inferior parietal lobule, and lentiform nucleus. These results help support previous studies showing error-related dysfunction in youth with elevated psychopathic traits, as some have considered the insula to contribute to the amplitude of the Pe (Schroder, Moran, Moser, & Altmann, 2012; Ullsperger et al., 2010), and reduced hemodynamic activity in the lentiform nucleus during error-related processing has been previously observed in youth with elevated psychopathic traits (Maurer et al., in press). In addition, the insula (Hester, Fassbender, & Garavan, 2004; Klein et al., 2007; Magno, Foxe, Molholm, Robertson, & Garavan, 2006; Menon et al., 2001; Steele, Claus, et al., 2014), superior temporal gyrus (Braet et al., 2011; Hester, Foxe, Molholm, Shpaner, & Garavan, 2005; Mathalon et al., 2003; Steele, Claus, et al., 2014), inferior parietal lobule (Braet et al., 2011; Hester et al., 2005; Mathalon et al.,

2003; Steele, Claus, et al., 2014; Wittfoth, Küstermann, Fahle, & Herrmann, 2008), and lentiform nucleus (Galléa, Bernhardina de Graaf, Pailhous, & Bonnard, 2008; Hester, Nestor, & Garavan, 2009; Mathalon et al., 2003; Maurer et al., in press) have all shown to contribute to successful error-related processing. By exhibiting reduced functional connectivity, this suggests that youth with elevated psychopathic traits are not recruiting brain regions to the same extent as youth scoring low on the PCL:YV during error-related processing. Deficits related to error-related processing can have serious implications for youth with elevated psychopathic traits. Specifically, such dysfunction may result in a reduced capacity to evaluate their mistakes, resulting in an increased propensity to engage in severe antisocial behavior, incarceration, recidivism, and substance use proclivity (Edens, Campbell, & Weir, 2007; Gregory et al., 2012).

Limitations

There are limitations to note associated with the current report which limits the generalizability of our results. First, the best evidence of the continuity of psychopathic traits from adolescence to adulthood comes from longitudinal evidence showing moderate stability of psychopathic traits from age 13 to 23 (Lynam et al., 2007). As such, there exists the possibility that youth in the current study may not grow up to meet the established diagnostic criteria for psychopathy as adults. Longitudinal research is desperately needed to measure whether functional connectivity deficits observed in youth samples can serve as a potential biomarker for the development of adult psychopathy. Second, it is not clear whether our current results extend to adolescent girls with elevated psychopathic traits. Gender differences are apparent in regards to error-related processing (Larson, South, & Clayson, 2011), and there exists the possibility that adolescent females

with elevated psychopathic traits exhibit different functional connectivity patterns compared to adolescent males with elevated psychopathic traits. Finally, we recruited participants from juvenile correctional facilities. Compared to youth recruited from community samples, incarcerated youth differ on several variables, most notably, substance use severity, general intelligence, and anxiety (Foley, 2001; Wasserman, McReynolds, Lucas, Fisher, & Santos, 2002), all of which have an impact on cognitive control measures. Therefore, it remains to be seen whether our results extrapolate to participants from community samples scoring high on measures of psychopathy.

Conclusions

In sum, youth with elevated psychopathic traits, as assessed via the PCL:YV, exhibited increased functional connectivity within regions incorporated within Component 24, including the precuneus, superior parietal lobule, postcentral gyrus, paracentral gyrus, and cingulate gyrus and increased functional connectivity with regions in Component 66, including the ACC, medial frontal gyrus, superior frontal gyrus, middle frontal gyrus, and subcallosal gyrus compared to youth scoring low on the PCL:YV during response inhibition. In addition, youth scoring high on the PCL:YV exhibited reduced functional connectivity with regions included in Component 69, including the insula, precentral gyrus, postcentral gyrus, superior temporal gyrus, inferior parietal lobule, and lentiform nucleus compared to youth scoring low on the PCL:YV during error-related processing. These results are consistent with previous studies in which youth with elevated psychopathic traits exhibited increased N2 amplitude during response inhibition (Sumich, Sarkar, Hermens, Kelesidi, et al., 2012) and reduced Pe amplitude (Maurer, Steele, Cope, et al., 2016) and reduced hemodynamic activity within subregions

of the basal ganglia (Maurer et al., in press) during error-related processing. This is the first evidence to suggest youth with elevated psychopathic traits exhibit functional connectivity deficits during cognitive control.

Study 2: Interpersonal and affective psychopathic traits related to reduced white matter integrity in incarcerated adolescent offenders

INTRODUCTION

Many studies to date have associated youth with elevated psychopathic traits with reduced amygdala responsiveness to fear-relevant stimuli (Jones et al., 2009; Lozier et al., 2014; Marsh et al., 2008; Viding et al., 2012; White et al., 2012). The amygdala is a structure within the temporal lobe that is typically involved in coding the affective significance of stimuli, and information processed by the amygdala is typically fed forward to the vmPFC to help guide decision-making (Cardinal, Parkinson, Hall, & Everitt, 2002). In addition to deficits in processing fear-relevant stimuli, dysfunction has commonly been reported in youth with elevated psychopathic traits in both the amygdala (Cohn et al., 2015; Jones et al., 2009; Lozier et al., 2014; Marsh et al., 2013; Marsh et al., 2008; Sebastian et al., 2012; Viding et al., 2012; Vincent et al., 2017; White et al., 2012) and vmPFC (Finger et al., 2008) during a variety of experimental paradigms. Additionally, youth with elevated psychopathic traits have been characterized by reduced functional connectivity between the amygdala and vmPFC (Aghajani, Klapwijk, et al., 2016; Marsh et al., 2008).

These latter functional connectivity studies suggest that WM tracts that connect temporal and prefrontal regions may be implicated in youth with elevated psychopathic traits. WM is comprised of myelinated axons, which help increase electrical resistance. Axons are organized into fiber bundles, and when grouped together, the myelin sheaths appear white, hence, why they are referred to as WM. Long WM fiber bundles are called '*fasciculi*' or '*tracts*'. The integrity of WM tracts can be investigated using diffusion

tensor imaging (DTI), a quantitative MRI technique that allows for the measurement of water movement within tissue microstructure (Le Bihan, Turner, Douek, & Patronas, 1992). One of the most commonly derived measures that can inform researchers about the cellular microstructure of WM tracts is referred to as "*fractional anisotropy (FA)*". FA is a measure of anisotropic water diffusion and reflects the degree or directionality of cellular structures within fiber tracts, and therefore, structural integrity. In pure isotropy, FA values would be zero, and with increasing anisotropy, the value increases towards one. Light axonal packing would leave more intercellular water, resulting in less restriction of diffusion, thus, lowering FA. On the other hand, a high degree of myelination would cause axons to be more tightly packed together, increasing FA values.

WM steadily increases throughout childhood and adolescence (Giedd et al., 1999), with FA values typically increasing throughout development, reflecting increased myelination (Beaulieu, 2002) and more effective long-range transmission of information across distributed brain regions (Mesulam, 2000). FA appears to be influenced by several factors, including myelination, axonal diameter, packing density, and branching (Beaulieu, 2002) and increased FA indicates more mature WM bundles (Lebel, Walker, Leemans, Phillips, & Beaulieu, 2008). Thus, degradation of WM structural integrity is associated with a reduction in FA (Mori & Zhang, 2006) and reduced FA has been routinely associated with cognitive deficits in both healthy and clinical populations (Mettenburg, Benzinger, Shimony, Snyder, & Sheline, 2012; Quinque et al., 2012; Turken et al., 2008; Wozniak et al., 2007).

The uncinate fasciculus (UF) may be a WM tract of particular importance for youth with elevated psychopathic traits, as it is the primary pathway connecting

structures of the temporal lobe, including the amygdala, to prefrontal structures, namely the vmPFC (Petrides & Pandya, 2007). Reduced FA in the right UF has been shown to play a critical role in emotional empathy in general (Oishi et al., 2015). In addition, a highly replicated finding in adult psychopathic offenders is reduced FA in the right UF (Craig et al., 2009; Hoppenbrouwers et al., 2013; Motzkin et al., 2011; Sobhani et al., 2015; Sundram et al., 2012; Vermeij et al., 2018; Wolf et al., 2015). Additionally, reduced FA in the right UF has been observed in adult females scoring higher on psychopathy (Lindner et al., 2017). Reduced FA in the right UF appears to be driven by interpersonal dysfunction, as higher PCL-R Factor 1 and Facet 1 scores have been associated with reduced FA in the right UF (Wolf et al., 2015). As FA is an indirect measure of WM integrity, many factors may contribute to reduced FA values in the right UF in adult psychopathic offenders, including fiber diameter, fiber density, or myelination (Winston, 2012).

Extending beyond the right UF, several studies have been performed observing a negative relationship between psychopathy scores and FA values in additional WM tracts in adults, including the inferior fronto-occipital fasciculus (Hoppenbrouwers et al., 2013; Sundram et al., 2012; Vermeij et al., 2018), bilateral anterior thalamic radiation (Hoppenbrouwers et al., 2013), dorsal cingulum (Sethi et al., 2015; Vermeij et al., 2018), genu of the corpus callosum (Sundram et al., 2012), internal capsule (Sundram et al., 2012), anterior corona radiata (Sundram et al., 2012), inferior longitudinal fasciculus (Vermeij et al., 2018), and fornix (Vermeij et al., 2018).

Unlike studies performed with adult psychopathic offenders, the relationship between adolescent psychopathic traits and WM structural integrity remains largely

inconsistent. One study found that youth scoring high on psychopathic traits exhibited reduced FA in the right UF (Breeden et al., 2015), consistent with adult psychopathic offenders (Craig et al., 2009; Hoppenbrouwers et al., 2013; Motzkin et al., 2011; Sobhani et al., 2015; Sundram et al., 2012; Vermeij et al., 2018; Wolf et al., 2015). However, two other studies have found that youth scoring high on psychopathic traits exhibit *increased* FA values in the right UF (Pape et al., 2015; Sarkar et al., 2013). Dissimilar results obtained between studies may be related to the operational definition of adolescent psychopathy used by the study. For example, Sarkar et al. (2013) considered youth who had a PCL:YV total score of 20 or above to be included as part of the psychopathic group, which is ten points lower than the traditional cut-off score used (Forth et al., 2003). As such, this study may actually be assessing the relationship between FA values and youth meeting criteria for externalizing disorders rather than adolescent psychopathy. Youth meeting criteria for externalizing disorders, including CD and ODD, typically exhibit increased FA values in the right UF (Sarkar et al., 2013; Zhang et al., 2014). Extending beyond the right UF, Pape et al. (2015) observed a positive relationship between adolescent psychopathy scores and FA values in numerous WM tracts, including the anterior thalamic radiation, corticospinal tract, forceps minor, inferior fronto-occipital fasciculus, and corpus callosum (Pape et al., 2015).

In Study 2, we sought to expand upon previous studies by investigating the relationship between WM structural integrity in the right UF and PCL:YV scores in a high-risk sample of incarcerated male offenders. We specifically hypothesized that a significant negative relationship would emerge between PCL:YV scores and FA values in the right UF, consistent with both youth (Breeden et al., 2015) and adults scoring high on

measures of psychopathy (Craig et al., 2009; Hoppenbrouwers et al., 2013; Motzkin et al., 2011; Sobhani et al., 2015; Sundram et al., 2012; Vermeij et al., 2018; Wolf et al., 2015). In addition, we explored the relationship between FA values and PCL:YV scores in additional WM tracts, including the anterior thalamic radiation, corticospinal tract, cingulum, forceps major, forceps minor, inferior longitudinal fasciculus, and superior longitudinal fasciculus to see if adolescent psychopathic traits were associated with WM structural integrity in any other WM tracts.

METHOD

Participants

Participants in Study 2 included $n = 314$ incarcerated male adolescent offenders recruited from juvenile correctional facilities in the states of New Mexico and Wisconsin. Participants were excluded from analyses for meeting the following criteria: occurrence of a TBI accompanied with a significant loss of consciousness ($n = 17$), due to the tremendous role TBIs play in WM structural integrity (Yuan et al., 2007), or an estimated IQ less than 70 ($n = 11$). Additionally, participants were excluded for meeting diagnostic criteria for psychosis ($n = 5$), not satisfying quality control criteria ($n = 9$), or being over the age of 18 ($n = 18$) so that age-related confounds did not influence the current results. This resulted in a final sample of $n = 254$ incarcerated male adolescent offenders, ranging from 14 to 18 years of age ($M = 17.04$ years, $SD = 1.08$ years) at the time of MRI data collection. The sample was predominantly right-handed (10% reported being left-hand dominant). Participants largely self-identified as Hispanic/Latino (48%), with the remaining self-identifying as Black or African American (29%), White (16%), American

Indian or Alaskan Native (5%), or Native Hawaiian or other Pacific Islander (1%). One

percent of the sample chose not to disclose their race or ethnicity.

Initial contact was made with potential study participants and informed consent

was obtained. Individuals provided written informed assent in conjunction with

parent/guardian consent. Participants were informed of their right to terminate

participation at any point, the lack of institutional benefits, and that their participation

would not affect their facility or parole status. Participants received remuneration at the

hourly labor wage of the facility. All research protocols were approved by E&I, OHRP,

and the juvenile detention centers where data collection occurred.

Assessments

Psychopathic traits were assessed using the Hare PCL:YV (Forth et al., 2003).

The mean PCL:YV total score in this sample was 25.45 (*SD* = 5.92) (ranging from 8 to

38). The Cronbach's alpha for the PCL:YV (all items) was .80 in the current sample,

reflecting good internal consistency. In addition to PCL:YV total scores, we incorporated

the use of a two-factor model of psychopathic traits, with Factor 1 comprising

interpersonal and affective traits and Factor 2 consisting of lifestyle and

antisocial/developmental traits (Neumann et al., 2006). The mean PCL:YV Factor 1 score

was 8.01 (*SD* = 3.32) and the mean Factor 2 score was 15.13 (*SD* = 2.81) in the current

sample. PCL:YV Factor 1 and 2 scores were significantly positively correlated (*r* = .52, *p*

< .001), consistent with previous reports (Mailloux, Forth, & Kroner, 1997). We also

examined a four-facet model of psychopathic traits, with four latent facets representing

the underlying dimensions of the adolescent expression of psychopathy: interpersonal

(Facet 1), affective (Facet 2), lifestyle (Facet 3), and antisocial/developmental (Facet 4),

respectively (Neumann et al., 2006). We investigated both factor and facet scores of the PCL:YV to reveal more specific directional and stable effects than those with PCL:YV total scores alone. For example, factor and facets of the PCL-R are associated with dimensional traits that have often revealed discrete (sometimes opposing) relationships with neurobiological measures (Anderson, Steele, Maurer, Bernat, & Kiehl, 2015; Juárez, Kiehl, & Calhoun, 2013; Maurer, Steele, Edwards, et al., 2016; Philippi et al., 2015; Steele, Maurer, et al., 2016). In addition, a negative relationship has been previously observed between Factor 1 and Facet 1 scores on the PCL-R and FA in the right UF (Wolf et al., 2015).

Additional Assessments

In addition to investigating psychopathic traits, additional assessments were also administered to assess IQ, substance dependence, psychopathology, and TBI. Full-scale IQ was estimated using the WAIS-III (Wechsler, 1997) for participants sixteen years of age or older and from the WISC-IV (Wechsler, 2003) for participants younger than sixteen years of age ($M = 90.78$, $SD = 10.83$). Both psychopathology and substance dependence were assessed using the K-SADS (Kaufman et al., 1997). Number of substance dependencies were calculated by summing the total number of substances (both alcohol and drug, including cannabis, stimulants, sedatives/hypnotics/anxiolytics, cocaine, opioids, phencyclidine [PCP], hallucinogens, solvent/inhalants, and other substances) for which participants met lifetime dependence diagnoses ($M = 1.68$, $SD = 1.60$). Additionally, history of TBI was evaluated using a post-head injury symptoms questionnaire (King et al., 1995).

MRI Data Acquisition

Diffusion-weighted echo-planar MRI was acquired at the juvenile correctional facilities where data collection occurred using the Mind Research Network's Siemens 1.5T Avanto Mobile MRI System equipped with a 12-element head coil. Diffusion sensitizing gradients were applied along 30 non-collinear directions (b value = 800 s/mm^2). Five interleaved nondiffusion weighted (b value = 0 s/mm^2) volumes were collected during each run to enable corrections for motion and eddy current distortions. Images were collected with the following parameters: repetition time (TR) = 9200 ms, echo time (TE) = 84 ms, field of view = 256 x 256 mm, matrix size = 128 x 128, slice thickness = 2 mm, no gap, voxel size = 2 x 2 x 2 mm^3, 70 slices. The sequence was repeated twice, and the data were combined to improve the signal-to-noise ratio. Head motion was limited using padding and restraint.

DTI Analyses

All images were visually inspected for quality, leaving a final sample of n = 254 participants. Brain extraction from the b0 image to remove non-brain tissue, correction of eddy current-induced possible distortions of diffusion-weighted images, and construction of Fractional Anisotropy (FA) maps were performed using the FMRIB's Diffusion Toolbox (FDT) in FMRIB Software Library (FSL) version 4.1 (www.fmrib.ox.ac.uk/fsl/). Tract Based Spatial Statistics (TBSS) workflow (Smith et al., 2006) of FSL was used to non-linearly warp the individual FA maps to the 1.0 x 1.0 x 1.0 mm^3 FA template in the MNI space. To minimize the partial volume effect, non-linearly warped FA images were averaged to construct the WM skeleton image. Subsequently, ROI averaged values were extracted from each participant from the intersection of the skeleton image and the Johns Hopkins University (JHU) WM atlas ROIs (Mori, Wakana,

Nagae-Poetscher, & van Zijl, 2005). ROIs from the JHU atlas included the following: 1) left anterior thalamic radiation, (2) right anterior thalamic radiation, (3) left corticospinal tract, (4) right corticospinal tract, (5) left cingulum (i.e., cingulate gyrus), (6) right cingulum (i.e., cingulate gyrus), (7) left cingulum (i.e., hippocampus), (8) right cingulum (i.e., hippocampus), (9) forceps major, (10) forceps minor, (11) left inferior fronto-occipital fasciculus, (12) right inferior fronto-occipital fasciculus, (13) left inferior longitudinal fasciculus, (14) right inferior longitudinal fasciculus, (15) left superior longitudinal fasciculus, (16) right superior longitudinal fasciculus, (17) left uncinate fasciculus, (18) right uncinate fasciculus, (19) temporal portion of the left superior longitudinal fasciculus, and (20) temporal portion of the right superior longitudinal fasciculus.

RESULTS

Correlational Results

In the current report, number of substance dependencies were significantly positively correlated with PCL:YV Factor 2 scores ($r = .14$, $p = .026$) and significantly negatively correlated with PCL:YV Facet 2 scores ($r = -.13$, $p = .039$). IQ scores were significantly negatively correlated with PCL:YV Factor 2 scores ($r = -.13$, $p = .040$), Facet 2 scores ($r = -.14$, $p = .030$), and Facet 3 scores ($r = -.14$, $p = .029$). Participant's age during the time of MRI collection was significantly negatively correlated with PCL:YV total scores ($r = .23$, $p < .001$), Factor 1 scores ($r = .23$, $< .001$), Factor 2 scores ($r = .18$, $p = .003$), Facet 1 scores ($r = -.20$, $p = .002$), Facet 2 scores ($r = -.19$, $p = .002$), and Facet 3 scores ($r = -.20$, $p = .001$). IQ scores were significantly positively correlated with number of substance dependencies ($r = .30$, $p < .001$) and participant's age during

MRI data collection was significantly positively correlated with number of substance dependencies ($r = .23$, $p < .001$). IQ scores and participant's age during MRI data collection were moderately positively correlated ($r = .12$, $p = .064$). PCL:YV scores and covariate measures were not significantly correlated with average FA values.

In addition, FA values in the right corticospinal tract were significantly positively correlated with PCL:YV Facet 1 scores ($r = .12$, $p = .049$), FA values in the right cingulum (i.e., cingulate gyrus) were positively correlated with PCL:YV Facet 1 scores ($r = .13$, $p = .042$), and FA values in the temporal portion of the right superior longitudinal fasciculus were significantly positively correlated with PCL:YV Facet 1 scores ($r = .12$, $p = .048$). No other significant correlations emerged between PCL:YV scores and FA values in any of the WM ROIs. However, IQ scores showed a number of significant positive correlations with FA values in the WM ROIs, including the left anterior thalamic radiation ($r = .19$, $p = .002$), the right anterior thalamic radiation ($r = .15$, $p = .015$), the left corticospinal tract ($r = .16$, $p = .009$), the left cingulum (i.e., hippocampus) ($r = .17$, $p = .006$), the right cingulum (i.e., hippocampus) ($r = .20$, $p = .002$), the left inferior fronto-occipital fasciculus ($r = .14$, $p = .029$), the right inferior fronto-occipital fasciculus ($r = .13$, $p = .034$), the left inferior longitudinal fasciculus ($r = .14$, $p = .030$), the right inferior longitudinal fasciculus ($r = .19$, $p = .002$), the left superior longitudinal fasciculus ($r = .16$, $p = .010$), the right superior longitudinal fasciculus ($r = 13$, $p = .038$), the right uncinate fasciculus ($r = .15$, $p = .016$), and temporal portion of the right superior longitudinal fasciculus ($r = .14$, $p = .024$). Finally, participant's age during MRI data collection was also significantly positively correlated with FA values in the WM ROIs, including the left anterior thalamic radiation ($r = .19$, $p = .003$), the right anterior

thalamic radiation (r = .24, p < .001), the left corticospinal tract (r = .13, p = .043), the right cingulum (i.e., hippocampus) (r = .17, p = .005), forceps major (r = .14, p = .030), forceps minor (r = .17, p = .006), the left inferior fronto-occipital fasciculus (r = .14, p = .026), the right inferior fronto-occipital fasciculus (r = .18, p = .005), the left inferior longitudinal fasciculus (r = .13, p = .033), the right inferior longitudinal fasciculus (r = .19, p = .002), the left superior longitudinal fasciculus (r = .18, p = .005), the left uncinate fasciculus (r = .14, p = .025), and the left superior longitudinal fasciculus (r = .21, p = .001). Finally, number of substance dependencies were significantly positively correlated with FA values in the right uncinate fasciculus (r = .15, p = .021).

Group-Based Analyses

Using independent samples *t*-tests, youth scoring high on the PCL:YV (n = 70 with a PCL-R total score 30 or above; M PCL:YV total score = 32.50, SD = 2.22) and youth scoring low on the PCL:YV (n = 67 with a PCL:YV total scoring 20 or below; M PCL:YV total score = 17.85, SD = 2.94) did not significantly differ with respect to IQ, t(135) = 0.75, p = .453, or number of substance dependencies, t(135) = -0.69, p = .489. However, groups did significantly differ with respect to age at MRI data collection, with youth scoring high on the PCL:YV being younger (M age = 16.90 years, SD = 1.10 years) compared to youth scoring low on the PCL:YV (M age = 17.39 years, SD = 0.92 years), t(135) = 2.82, p = .006.

Additionally, groups did not significantly differ with respect to FA values in the left anterior thalamic radiation, t(135) = -0.56, p = .575, right anterior thalamic radiation, t(135) = -0.48, p = .631, left corticospinal tract, t(135) = -0.78, p = .439, right corticospinal tract, t(135) = -1.00, p = .318, left cingulum (i.e., cingulate gyrus), t(135) =

-0.39, *p* = .700, right cingulum (i.e., cingulate gyrus), *t*(135) = -0.59, *p* = .554, left

cingulum (i.e., hippocampus), *t*(135) = -0.23, *p* = .815, right cingulum (i.e.,

hippocampus), *t*(135) = -0.86, *p* = .390, forceps major, *t*(135) = -0.10, *p* = .923, forceps

minor, *t*(135) = -0.44, *p* = .658, left inferior fronto-occipital fasciculus, *t*(135) = -0.68, *p* .

499, right inferior fronto-occipital fasciculus, *t*(135) = -0.25, *p* = .802, left inferior

longitudinal fasciculus, *t*(135) = -0.23, *p* = .821, right inferior longitudinal fasciculus,

t(135) = 0.36, *p* = .721, left superior longitudinal fasciculus, *t*(135) = -0.42, *p* = .673,

right superior longitudinal fasciculus, *t*(135) = -0.24, *p* = .814, left uncinate fasciculus,

t(135) = -0.70, *p* = .484, right uncinate fasciculus, *t*(135) = -0.21, *p* = .836, the temporal

portion of the left superior longitudinal fasciculus, *t*(135) = 0.11, *p* = .915, or temporal

portion of the right superior longitudinal fasciculus, *t*(135) = -0.58, *p* = .562. Finally,

groups did not significantly differ with respect to average FA values, *t*(135) = -0.84, *p* = .

401.

Multiple Regression Analyses

Multiple regression analyses were performed to assess the amount of variance in

twenty WM ROIs explained by PCL:YV total, factor, or facet scores or covariate

measures using our entire sample. Each of the multiple regression analyses performed

had FA values from a WM ROI as the dependent measure, and either PCL:YV total,

factor, or facet scores and four covariate measures (participant's age at MRI data

collection, IQ, number of substance dependencies, and average FA) as simultaneous

predictor variables in separate analyses performed. Although PCL:YV factor and facet

scores were significantly correlated with covariate measures (see correlational analyses

above), the variance inflation factor in multiple regression analyses performed was typically under 1.3, suggesting no problems in collinearity (Menard, 1995).

Multiple Regression Analyses with PCL:YV total scores

In the first set of multiple regression analyses performed, PCL:YV total scores were entered along with covariate measures (i.e., participant's age at MRI data collection, IQ, number of substance dependencies, and average FA) to see if any variables entered into analyses were significant predictors of FA values in any of the *a priori* WM tracts investigated. PCL:YV total scores emerged as a moderate predictor of reduced FA in the right inferior longitudinal fasciculus ($\beta = -.07$, $p = .052$), but PCL:YV total scores were never a significant predictor of FA values in any of the *a priori* WM tracts investigated.

Multiple Regression Analyses with PCL:YV factor scores

In the second set of multiple regression analyses performed, PCL:YV factor scores (i.e., both PCL:YV Factor 1 and Factor 2 scores) were entered along with covariate measures (i.e., participant's age at MRI data collection, IQ, number of substance dependencies, and average FA) to see if any variables entered into analyses were significant predictors of FA values in any of the *a priori* WM tracts investigated.

PCL:YV Factor 1 scores emerged as a significant predictor of reduced FA in the right inferior longitudinal fasciculus ($\beta = -.09$, $p = .027$) and the right uncinate fasciculus ($\beta = -.13$, $p = .014$). In addition, PCL:YV Factor 1 scores emerged as a moderate predictor of reduced FA in the left cingulum (i.e., cingulate gyrus) ($\beta = -.08$, $p = .052$) and the left uncinate fasciculus ($\beta = -.08$, $p = .082$), whereas PCL:YV Factor 2 scores emerged as a moderate predictor of increased FA in the forceps minor ($\beta = -.08$, $p = .054$). No other significant PCL:YV-related effects emerged in the second set of multiple

regression analyses performed. Regarding covariate measures, participant's age at MRI data collection and IQ scores typically emerged as significant predictors of increased FA in *a priori* WM tracts investigated (see Table 2).

Multiple Regression Analyses with PCL:YV facet scores

In the third set of multiple regression analyses performed, PCL:YV facet scores (i.e., PCL:YV Facet 1, 2, 3, and 4 scores) were entered along with covariate measures (i.e., participant's age at MRI data collection, IQ, number of substance dependencies, and average FA) to see if any variables entered into analyses were significant predictors of FA values in any of the *a priori* WM tracts investigated.

PCL:YV Facet 1 scores emerged as a significant predictor of reduced FA in the right uncinate fasciculus (β = -.10, *p* = .046), whereas PCL:YV Facet 2 scores emerged as a significant predictor of reduced FA in both the left inferior fronto-occipital fasciculus (β = -.08, *p* = .047) and right inferior fronto-occipital fasciculus (β = -.06, *p* = .044), and the right inferior longitudinal fasciculus (β = -.11, *p* = .010). Alternatively, PCL:YV Facet 3 scores emerged as a significant predictor of increased FA in the left uncinate fasciculus (β = .09, *p* = .040).

In addition, PCL:YV Facet 2 scores emerged as a moderate predictor of reduced FA in the left anterior thalamic radiation (β = -.08, *p* = .061), PCL:YV Facet 3 scores emerged as a moderate predictor of increased FA in the forceps minor (β = .07, *p* = .079) right superior longitudinal fasciculus (β = -.06, *p* = .061), and uncinate fasciculus (β = .10, *p* = .056). Regarding covariate measures, participant's age at MRI data collection and IQ scores typically emerged as significant predictors of increased FA in *a priori* WM tracts investigated (see Table 3).

DISCUSSION

It has been suggested that reduced responsiveness in the amygdala to affective stimuli, combined with inadequate signaling of reinforcement expectancies between the vmPFC and amygdala, underlie the social and behavioral deficits associated with youth with elevated psychopathic traits (Blair, 2007). Dysfunction has commonly been reported in youth with elevated psychopathic traits in both the amygdala (Cohn et al., 2015; Jones et al., 2009; Lozier et al., 2014; Marsh et al., 2013; Marsh et al., 2008; Sebastian et al., 2012; Viding et al., 2012; Vincent et al., 2017; White et al., 2012) and vmPFC (Finger et al., 2008), and also reduced functional connectivity between the amygdala and vmPFC (Aghajani, Klapwijk, et al., 2016; Marsh et al., 2008).

These latter findings inspired researchers to investigate the structural connectivity of the UF, the primary pathway connecting regions of the anterior lobe, including the amygdala, to prefrontal structures, including the vmPFC (Petrides & Pandya, 2007). Adult psychopathic individuals have routinely exhibited reduced FA values in the right UF (Craig et al., 2009; Hoppenbrouwers et al., 2013; Lindner et al., 2017; Motzkin et al., 2011; Sobhani et al., 2015; Sundram et al., 2012; Vermeij et al., 2018; Wolf et al., 2015). However, this consistent negative relationship between structural integrity of the right UF has often not been replicated in youth samples. For example, one study found that youth scoring high on psychopathic traits did exhibit reduced FA in the right UF (Breeden et al., 2015), consistent with previous studies with adult psychopathic individuals. On the other hand, other studies have found that youth scoring high on psychopathy exhibit increased FA in the right UF (Pape et al., 2015; Sarkar et al., 2013). These different results between studies may have to do with differences related to the operational definition of adolescent

psychopathy (Sarkar et al., 2013) or they may be related to different developmental trajectories of WM development in youth with elevated psychopathic traits. Thus, we sought to investigate the relationship between WM structural integrity and adolescent psychopathy scores in a sample of *n* = 254 incarcerated male adolescent offenders in the current study.

Consistent with a previously published study with youth with elevated psychopathic traits (Breeden et al., 2015) and previous studies performed with adult psychopathic offenders (Craig et al., 2009; Hoppenbrouwers et al., 2013; Lindner et al., 2017; Motzkin et al., 2011; Sobhani et al., 2015; Sundram et al., 2012; Vermeij et al., 2018; Wolf et al., 2015), we found that psychopathy scores were significantly negatively related to FA in the uncinate fasciculus in a sample of incarcerated male adolescent offenders. Specifically, the negative relationship was driven by PCL:YV Factor 1 and Facet 1 scores, consistent with a previously published study performed with adult psychopathic offenders indicating interpersonal dysfunction was associated with reduced FA in the uncinate fasciculus (Wolf et al., 2015). Reduced FA in the uncinate fasciculus has been previously associated with deficits in emotional empathy (Oishi et al., 2015) and may relate to the reduced functional connectivity previously observed between the amygdala and vmPFC in youth with elevated psychopathic traits (Aghajani, Klapwijk, et al., 2016; Marsh et al., 2008). Specifically, Marsh et al. (2008) found that youth with elevated psychopathic traits exhibited reduced functional connectivity between the amygdala and vmPFC when processing fearful facial expressions (Marsh et al., 2008). Youth with elevated psychopathic traits have previously been characterized by reduced hemodynamic activity in the amygdala when processing fearful facial expressions (Jones

et al., 2009; Lozier et al., 2014; Marsh et al., 2008; Viding et al., 2012; White et al., 2012). The amygdala codes the affective significance of stimuli and feeds this information forward to the vmPFC to guide decision-making (Cardinal et al., 2002). Thus, youth with elevated psychopathic traits may not view fearful facial expressions as affectively significant, combined with reduced FA in the uncinate fasciculus, exhibit dysfunctional connectivity between the amygdala and vmPFC.

Two other previous studies performed found increased FA in the right UF in youth with elevated psychopathic traits (Pape et al., 2015; Sarkar et al., 2013). The disparate results obtained in these two previous report and our current report and those obtained by (Breeden et al., 2015) may simply be due to the operational definition of adolescent psychopathy used in studies performed. Studies that found increased FA in the right UF in youth with elevated psychopathic traits may actually be measuring youth meeting criteria for externalizing disorders, including CD and ODD. In fact, increased FA in the right UF has been previously observed in youth meeting criteria for externalizing disorders (Sarkar et al., 2013; Zhang et al., 2014). In addition, we found that PCL:YV Facet 3 scores, including stimulation seeking, impulsivity, and irresponsibility, traits characteristic of externalizing disorders, were associated with increased FA in the left UF in the current report.

Extending beyond the right uncinate fasciculus, we also found that youth with elevated psychopathic traits exhibited reduced FA in additional WM tracts, including the right inferior longitudinal fasciculus and bilateral inferior fronto-occipital fasciculus. Adult psychopathic offenders have also been characterized by reduced FA in the inferior longitudinal fasciculus (Vermeij et al., 2018) and inferior fronto-occipital fasciculus

(Hoppenbrouwers et al., 2013; Sundram et al., 2012; Vermeij et al., 2018). In addition, PCL:YV Facet 2 scores were negatively related to FA in the inferior longitudinal fasciculus and inferior fronto-occipital fasciculus, consistent with a previous study with adult psychopathic offenders (Vermeij et al., 2018).

In the current report, we found that both PCL:YV Factor 1 and Facet 2 scores were negatively related to FA in the right inferior longitudinal fasciculus (ILF) and PCL:YV Facet 2 scores were negatively related to FA in both the left and right inferior fronto-occipital fasciculus (IFOF). The ILF and IFOF are in close relationship to each other, and even are involved in similar functional roles, specifically for the perception of faces and facial emotions. Patients with prosopagnosia experience difficulty in face recognition, and are associated with reduced structural integrity of both the ILF and IFOF (Thomas et al., 2009). In addition, more extensive damage associated with the ILF and IFOF have been associated with an increased inability to properly identify facial emotions (Genova et al., 2015; Philippi, Mehta, Grabowski, Adolphs, & Rudrauf, 2009), specifically negative emotions, including fear, disgust, anger, and sadness (Crespi et al., 2014). Youth with elevated psychopathic traits have been characterized by deficits in accurately identifying facial expressions, including sadness (Blair & Coles, 2000; Blair, Colledge, Murray, & Mitchell, 2001) and fear (Blair & Coles, 2000; Blair et al., 2001). Blair's *Violence Inhibition Mechanism model* (Blair, 1995) suggests that certain emotions, including fear and sadness, may serve as a distress cue that typically helps stop the continuation of aggressive behavior. Youth and adults with elevated psychopathic traits exhibit decreased sensitivity to these distress cues and are associated with heightened aggression, which may be associated with ILF and IFOF dysfunction.

We also found that IQ scores were associated with increased FA values in a collection of WM tracts in the current report. This positive relationship between FA and IQ is thought to reflect more efficient information transfer in those characterized by higher intelligence (Chiang et al., 2009; Deary et al., 2006; Yu et al., 2008). Similarly, we found that participant's age at MRI data collection was also positively associated with FA values in a variety of WM tracts. FA values have been shown to increase in WM tracts between childhood and adolescence (Ashtari et al., 2007; Barnea-Goraly et al., 2005; Klingberg, Vaidya, Gabrieli, Moseley, & Hedehus, 1999; Schmithorst, Wilke, Dardzinski, & Holland, 2002), which has been suggested to reflect proper synaptic pruning and ongoing axonal myelination occurring throughout adolescence, leading to more efficient information transfer in older adolescents (Sowell et al., 2004).

Limitations

Limitations in the current study should be noted which may reduce the generalizability of our findings. First, similar to study 1, there exists the possibility that youth in the current study may not grow up to meet the established diagnostic criteria for psychopathy. Longitudinal research is desperately needed to measure whether WM microstructure abnormalities in youth samples can serve as a potential biomarker for the development of adult psychopathy, especially since the overwhelming majority of DTI studies performed with adult psychopathic offenders are associated with reduced FA values in WM tracts (Craig et al., 2009; Hoppenbrouwers et al., 2013; Lindner et al., 2017; Motzkin et al., 2011; Sobhani et al., 2015; Sundram et al., 2012; Vermeij et al., 2018; Wolf et al., 2015). Second, as our analyses remained largely exploratory, we performed a significant number of statistical tests without multiple comparison

correction. Analyses in our study remained largely exploratory, as we attempted to identify which WM tracts were implicated in adolescent psychopathy, while also attempting to identify hemisphere-specific abnormalities, as previous studies with adult samples have localized WM structural integrity deficits to the right UF (Craig et al., 2009; Hoppenbrouwers et al., 2013; Motzkin et al., 2011; Sobhani et al., 2015; Sundram et al., 2012; Vermeij et al., 2018; Wolf et al., 2015). We wanted to examine whether other hemisphere-specific structural integrity deficits would appear in other WM tracts in youth with elevated psychopathic traits. Some have suggested that adjustment for multiple comparisons is less critical in exploratory studies (Althouse, 2016). Still, even if we performed fewer statistical tests, such as combining bilateral ROIs to a single, unilateral ROI, and exploring only those WM tracts previously implicated in previous studies, it remains to be seen whether our results extrapolate to other samples. Finally, we only investigated FA, and not measures of radial diffusivity (RD) or axial diffusivity (AD), which are additional measures of WM microstructure (Winklewski et al., 2018). However, RD and AD produce biased results due to their estimation method, which incorporates a sorting procedure that does not take into account all three eigenvalues of the diffusion tensor, and is highly susceptible to noise, and can result in inflated AD values and reduced RD values (Basser & Pierpaoli, 2011; Pierpaoli & Basser, 1996). FA is not susceptible to the same biases due to differences in the estimation, including incorporating all three eigenvalues and is believed to be a more reliable measure of WM microstructure (Basser & Pierpaoli, 2011; Pierpaoli & Basser, 1996). Still, it remains to be seen whether our results were specifically related to FA, or if AD and RD deficits also would have been observed in this sample.

Conclusions

Consistent with hypotheses, in the current report, higher PCL:YV Factor 1 and Facet 1 scores were associated with reduced FA in the right UF, consistent with a previous study performed with youth with elevated psychopathic traits (Breeden et al., 2015) and studies performed with adult psychopathic offenders (Craig et al., 2009; Hoppenbrouwers et al., 2013; Motzkin et al., 2011; Sobhani et al., 2015; Sundram et al., 2012; Vermeij et al., 2018; Wolf et al., 2015). In addition, higher Factor 1 and Facet 2 scores were associated with reduced FA in the right inferior longitudinal fasciculus and bilateral inferior fronto-occipital fasciculus, consistent with adult psychopathic offenders (Hoppenbrouwers et al., 2013; Sundram et al., 2012; Vermeij et al., 2018). Dysfunction within these WM tracts could potentially relate to some of the characteristic deficits associated with youth with elevated psychopathic traits, including empathy-related deficits, and dysfunction in processing negative emotions, including sadness and fear.

Study 3: Error-related processing deficits predictive of recidivism in a sample of

incarcerated male adolescent offenders

INTRODUCTION

Juvenile delinquency poses a serious problem to society. Every year, two million youth are arrested and 60,000 are detained within the United States, making justice-involved youth a high-risk and vulnerable population (Sickmund, Sladky, Kang, & Puzzanchera, 2011). Although the rate of youth incarceration has been steadily declining since 1995, it still remains that the United States incarcerates a higher proportion of youth than any other developed country (Annie E. Casey Foundation, 2013). The vast majority of youth are arrested for low-level, non-violent crimes, including alcohol and marijuana use (Puzzanchera, 2014). Twenty-two percent of juveniles are arrested for higher-level, non-violent crimes, including theft, whereas only 5% of juveniles are arrested for violent crimes, including murder, rape, and aggravated assault (Puzzanchera, 2014). The average juvenile is incarcerated for around three to four months (Sickmund & Puzzanchera, 2014), resulting in an average cost of $21,000 - $28,000 per juvenile for United States taxpayers. In addition, one report by economists estimated that the indirect costs of juvenile incarceration to United States taxpayers to range from $8 - $21 billion per year when considering the cost of recidivism, later involvement in the criminal justice system, unemployment, the loss of future earnings, and additional health care costs (Justice Policy Institute, 2015).

Moffitt (1993) distinguished between two distinct patterns of juvenile delinquency: "*adolescent-limited*" and "*life-course persistent*" offenders (Moffitt, 1993). Adolescent-limited offenders describe the vast majority of juvenile delinquents; that is,

these individuals engage in criminal behavior during adolescence, but cease engaging in criminal activity after the transition from adolescence to adulthood. This is consistent with the *age-crime curve* (Farrington, 1986), which suggests that engagement in criminal activity tends to increase in late childhood and peak during the teenage years (i.e., around 15 – 19 years old) and then steadily declines in the early twenties. Around 40 – 60% of juvenile delinquents cease engaging in criminal activity by early adulthood (Farrington, 2003; Piquero, 2001). However, a small number of juveniles begin to engage in antisocial behavior during childhood and continue engaging in criminal activity after the transition to adulthood, becoming life-course persistent offenders (Moffitt, 1993). Juveniles who continue to engage in criminal activity as adults are typically characterized as individuals who started offending at a very early age, typically before the age of twelve (Loeber & Farrington, 2011). In addition, crimes committed as a youth including drug dealing, possession of weapons, and marijuana use are associated with a higher likelihood of persistence of criminal activity into adulthood (Rosenfeld, Helene, & Finn-Aage, 2012). For life-course persistent offenders, the transition from adolescence to adulthood is characterized by increasing severity of offenses and a growth in lethal violence (Farrington, 2003; Piquero, 2001).

Depending on the specific study and the exact operational definition of recidivism used, as many as 40 – 70% of adolescents recidivate within one year after institutional release (Gordon, Diehl, & Anderson, 2012; Grunwald, Lockwood, Harris, & Mennis, 2010). Criminal recidivism for both adolescent-limited and life-course persistent offenders are associated with both static and dynamic risk factors. Static risk factors are those that are inherent to the individual and cannot be altered through clinical

interventions. For example, static risk factors include male sex, low intelligence (Vermeiren et al., 2002), engaging in criminal behavior and having a first conviction at an earlier age, and increased severity of juvenile delinquency (Cottle, Lee, & Heilbrun, 2001; Loeber & Farrington, 1998; Loeber, Farrington, Stouthamer-Loeber, & Raskin White, 2008; Marczyk, Heilbrun, Lander, & Dematteo, 2003; Myner et al., 1998). Dynamic risk factors include conduct disorder symptomatology (Kotler & McMahon, 2005; van Dam, Janssens, & De Bruyn, 2004), anxiety and depression symptomatology (Cottle et al., 2001), attention-deficit/hyperactivity disorder (ADHD) symptomatology (Gordon et al., 2012), substance use severity (Ford, 2005; Hawkins et al., 2000; Myner et al., 1998; Schubert, Mulvey, & Glasheen, 2011), psychopathic traits (Das, 2008; Marczyk et al., 2003; Walters, 2003), and interaction with peers who engage in criminal activity (Cottle et al., 2001; Pardini, Obradovic, & Loeber, 2006).

Researchers have long believed that deficits in impulse control may related to the engagement of delinquent behavior (Barratt & Patton, 1983; Eysenck & Eysenck, 1976; Robbins & Bryan, 2004). Indeed, studies have shown that deficits associated with neuropsychological functioning are commonly associated with antisocial behavior (Loeber, 1990; Moffitt, 1990). Moffitt (1993) suggested one key differentiation between adolescent-limited and life-course persistent adolescent offenders may relate to neuropsychological impairments occurring during childhood in life-course persistent antisocial offenders. These deficits, including those related to self-control, may relate to the continuing engagement of antisocial behavior in adulthood (Moffitt, 1993).

There is strong evidence that impulsivity, or the inability to regulate self-control, is an important predictor of the engagement of delinquent behavior (Farrington, Loeber,

& Van Kammen, 1990; Gottfredson & Hirschi, 1990; Vitacco, Neumann, Robertson, & Durrant, 2002; Vitacco & Rogers, 2001). Gottfredson & Hirschi (1990)'s '*General Theory of Crime*' posits that impulsive youth lack proper self-control, which makes them more likely to engage in criminal activity, as it provides them with immediate gratification of desires and are more likely to surrender to the influence of peers who also engage in criminal behavior (Gottfredson & Hirschi, 1990). While adolescents tend to have less self-restraint compared to adults, this tends to decline linearly with age, suggesting that adolescents may "age out" of impulsive behaviors (Cauffman & Steinberg, 2000). However, this linear relationship may exist for adolescent-limited offenders only. Life-course persistent antisocial offenders do not appear to show the same age-related reduction of impulsivity (Leverso, Bielby, & Hoelter, 2015).

Adult risk assessment tools for future violent offenses tend to focus on static risk factors, placing less emphasis on dynamic risk factors (Borum, 2000). Individual risk factors may be less stable and accurate in adolescents compared to adults due to ongoing psychosocial development (Borum, 2000). Due to the enormous changes that occur during adolescence, including social, pubertal, emotional, and cognitive changes, predictors of recidivism in adolescent samples must be sensitive to developmental context (Hoge, 1999). It may be beneficial to identify potential neurobiological variables predictive of recidivism, as these can serve as a more direct measurement of the brain's inhibitory and cognitive control system compared to risk assessments, personality tests, and neuropsychological tests, which only serve as proxy measures. Additionally, it may be beneficial to identify neurobiological variables predictive of recidivism where the developmental trajectory of such variables is well established. One such neurobiological

variable may be the amplitude of the error-related positivity (Pe) ERP component. Compared to the amplitude of the ERN/Ne, which increases in amplitude throughout development, reflecting improved efficiency in error detection and action-monitoring processes, the amplitude of the Pe is rather invariant throughout development, showing comparable amplitude between youth and adult samples (Davies et al., 2004; Ladouceur et al., 2007; Santesso et al., 2006). The ERN/Ne and Pe appear to reflect distinct stages of error-related processing. The ERN/Ne reflects initial, automatic error-detection and action-monitoring processes (Falkenstein et al., 1991; Gehring et al., 1990), whereas the Pe is involved in later, more elaborate error-processing stages including assessing the motivational (Ullsperger et al., 2010) or affective (Overbeek et al., 2005) significance of incorrect responses. Neural generators of the ERN/Ne and Pe have been localized to the ACC (Edwards et al., 2012; van Veen & Carter, 2002). A previous study with $n = 45$ incarcerated adult offenders found that increased amplitude of the Pe was associated with a greater risk of future recidivism, above other measures included in analyses, including PCL-R factor scores, substance use severity, and participant's age when released from the correctional facility (Steele et al., 2015). The authors suggested that offenders who exhibited increased Pe amplitude may be at greatest risk of future recidivism as they are not categorizing response errors as events to be minimized in the future. In addition, the amplitude of the Pe has been shown to be malleable through mindfulness meditation (Larson, Steffen, & Primosch, 2013). Thus, it may be beneficial to investigate whether the amplitude of the Pe similarly predicts future recidivism in incarcerated adolescent samples.

In the current report, we investigated whether the amplitude of the Pe ERP component predicted future recidivism in a sample of $n = 104$ incarcerated male adolescent offenders. We specifically hypothesized that offenders who eventually were rearrested would be characterized by reduced Pe amplitude compared to those who were not rearrested. We hypothesized the increased Pe amplitude predictive of recidivism previously observed in incarcerated adult offenders (Steele et al., 2015) may reflect a potential compensatory mechanism, attempting to overcome post-error related deficits experienced as adolescents. In addition to the use of traditional time-domain ERP analyses, we also examined a more detailed parcellation of ERP responses by decomposing overlapping variance between and within ERP components using principal component analysis (PCA) (Dien, Khoe, & Mangun, 2007). This approach has been incorporated in a number of reports from our laboratory (Anderson et al., 2015; Fink et al., 2016; Maurer, Steele, Cope, et al., 2016; Maurer, Steele, Edwards, et al., 2016; Maurer et al., 2018; Steele et al., 2015; Steele, Fink, et al., 2014; Steele, Maurer, et al., 2016), demonstrating that PCA of ERP components provides a more sensitive and predictive measure compared to traditional time-domain ERP analyses. In the current report, four principal components were extracted, one reflecting mean ERN/Ne amplitude, and the remaining three subcomponents reflecting early, middle, and late subcomponents underlying the mean amplitude of the Pe. The three separate subcomponents of the Pe appear to reflect distinctive cognitive processes and are associated with unique patterns of hemodynamic activity in subregions of the ACC (Edwards et al., 2012). In particular, the early Pe subcomponent has been previously associated with both caudal and rostral ACC (cACC and rACC, respectively)

hemodynamic activity, the middle Pe subcomponent has been associated with cACC hemodynamic activity, and the late Pe subcomponent has been associated with rACC hemodynamic activity (Edwards et al., 2012). We specifically hypothesized that the PCA subcomponents which corresponded to the time-domain ERN/Ne and Pe ERP components would show more significant relationships with rearrest outcomes compared to traditional time-domain ERP analyses.

METHOD

Participants

Participants were recruited from a maximum-security juvenile correctional facility in the state of New Mexico who participated in a larger overall study (Southwest Advanced Neuroimaging Cohort – Youth (SWANC-Y)) and who had performed a response inhibition Go/NoGo task while incarcerated and had reliable follow-up information post-release. The final sample consisted of $n = 104$ incarcerated male offenders, ranging from 13 to 20 years of age ($M = 17.64$ years, $SD = 1.08$ years) at the time of electroencephalography (EEG) data collection. Exclusionary criteria for Study 3 included the following: (1) previous history of TBI accompanied with a significant loss of consciousness, (2) significant movement during data collection, (3) reasons related to behavioral performance (i.e., poor behavioral performance or making less than four errors), as reliability analyses suggest that the ERN/Ne and Pe can be quantified in as few as four to six trials (Olvet & Hajcak, 2009; Pontifex et al., 2010; Steele, Anderson, et al., 2016), and (4) meeting criteria for mood disorders or anxiety disorders, due to the important role these disorders play both the ERN/Ne (Chiu & Deldin, 2007; Olvet & Hajcak, 2008) and Pe (Bridwell, Steele, Maurer, Kiehl, & Calhoun, 2015). None of these

n = 104 participants met any of the exclusionary criteria and were thus retained in final analyses. The sample was predominantly right-handed, with 9% reporting left-hand dominance. Participants largely self-identified as Hispanic/Latino (73%), with the remaining self-identifying as American Indian or Alaskan Native (10%), White (8%), or Black or African American (7%). Two percent of the sample chose not to disclose their race or ethnicity.

Initial contact was made with potential study participants and informed consent was obtained. Individuals 18 years of age or older provided written informed consent and individuals younger than 18 years of age provided written informed assent in conjunction with parent/guardian consent. Participants were informed of their right to terminate participation at any point, the lack of institutional benefits, and that their participation would not affect their facility status or parole. Participants received remuneration at the hourly labor wage of the facility. All research protocols were approved by the Ethical and Independent Review Services (E&I), the Office for Human Research Protections (OHRP), and the juvenile detention center where data collection occurred.

Assessments

Data from several potential risk factors were obtained to examine the incremental utility of the ERP measures. These measures were similar to those previously included in neuroprediction models of recidivism (Aharoni et al., 2013; Steele et al., 2015), modified for age appropriateness. These measures included psychopathic traits, number of substance dependencies, and age at release.

Psychopathic traits were assessed using the Hare PCL:YV (Forth et al., 2003). The mean PCL:YV total score for the sample was 23.49 (SD = 6.38) (ranging from 2 to

35). The Cronbach's alpha for the PCL:YV (all items) was .84 in the current sample, reflecting good internal consistency. In addition to PCL:YV total scores, we incorporated the use of a two-factor model of psychopathic traits, with Factor 1 comprising interpersonal and affective traits, and Factor 2 consisting of lifestyle and antisocial/developmental traits (Neumann et al., 2006). The mean PCL:YV Factor 1 score was 6.68 (SD = 3.12) and the mean Factor 2 score was 14.66 (SD = 3.38) in the current sample. PCL:YV Factor 1 and 2 scores were significantly positively correlated (r = .56, p < .001), consistent with previous reports (Mailloux et al., 1997). In analyses performed, we included the PCL:YV factor interaction term (i.e., PCL:YV Factor 1 multiplied by PCL:YV Factor 2) to reduce multicollinearity in analyses performed (Corrado et al., 2004; Skeem & Mulvey, 2001) (M = 103.87, SD = 60.68).

Substance dependence and psychopathology were assessed using the K-SADS (Kaufman et al., 1997). Number of substance dependencies were calculated by summing the total number of substances (both alcohol and drug, including cannabis, stimulants, sedatives/hypnotics/anxiolytics, cocaine, opioids, phencyclidine [PCP], hallucinogens, solvent/inhalants, and other substances) for which participants met lifetime dependence diagnoses (M = 2.13, SD = 1.58). In addition to previously being used as covariate measures in previous reports, the covariates chosen have also been shown to be significant predictors of recidivism in youth samples, including PCL:YV scores (Corrado et al., 2004; Vincent et al., 2008), age at release (Baglivio et al., 2016; Mallett et al., 2012; Parsons Winokur et al., 2008), and substance use severity (Colins et al., 2011; Hoeve et al., 2013; Myner et al., 1998).

Assessments were also administered to assess IQ and TBI for exclusionary purposes. Full-scale IQ was estimated using the WAIS-III (Wechsler, 1997) for participants sixteen years of age or older and from the WISC-IV (Wechsler, 2003) for participants younger than sixteen years of age ($M = 92.86$, $SD = 10.33$). History of TBI was evaluated using a post-head injury symptoms questionnaire (King et al., 1995).

Stimuli and Task

EEG data were collected in a small room separate from the general population housing. After placement of electrodes, participants were seated in a comfortable chair 60 cm away from a computer monitor on which the task stimuli were presented and were instructed to refrain from excessive blinking and movement during data collection. Participants then performed a response inhibition Go/NoGo task (Kiehl, Liddle, et al., 2000). Stimuli were presented to participants using the Neurobehavioral Systems Inc. visual software package, Presentation (www.neurobs.com). Each stimulus appeared for 250 ms in white text within a continuously displayed rectangular fixation box against a black background. Participants were instructed to respond as quickly and accurately as possible with their right index finger every time the target Go stimulus (a white "X") appeared and to withhold a response whenever the distracter NoGo stimulus (a white "K") appeared. Targets appeared at a higher frequency (84% of trials, 412 total trials, with 206 on each run) than distracters (16% of trials, 78 total trials, with 39 on each run) to establish a strong stimulus-response mapping on Go trials. NoGo stimuli were never presented sequentially. The inter-stimulus interval was pseudo-randomly jittered (1 – 3 second stimulus onset asynchrony [SOA] averaging 1.5 seconds). The SOA between Go stimuli varied with the constraint that three Go stimuli were presented within each six-

second period. The NoGo stimuli were interspersed among the Go stimuli in a pseudo-random manner subject to two constraints: the minimum SOA between Go and NoGo stimuli was 1000 ms and the SOA between successive NoGo stimuli was in the range of eight to fourteen seconds. Hits were defined as successful responses to Go stimuli, whereas FA's were defined as incorrect responses to NoGo stimuli. Prior to recording, each participant performed a block of ten practice trials to ensure task instructions were clearly understood.

EEG Recordings

EEG data were collected using two computers and a 64-channel BioSemi amplifier. The first computer used Presentation software to deliver the stimuli, accept responses, and send digital triggers to the EEG acquisition computer when a stimulus or response occurred. The second computer acquired EEG data using BioSemi software and amplifiers. All signals with BioSemi software were low-pass filtered using a fifth-order sinc filter with a half-power cutoff of 204.8 Hz, then digitized to 1024 Hz during data collection. EEG activity was recorded using sintered Ag-AgCl active electrodes placed in accordance with the 10-20 International System (Jasper, 1958). The participant's nose was used as a reference site. Six electrodes were placed on the participant's face above, below, and lateral to the canthus of each eye to measure electrooculogram. All offsets were kept below 10 kΩ.

Analytic Strategy

Preprocessing included down-sampling to 512 Hz, bad channel detection and replacement, epoching, eye-blink removal, and low-pass filtering to 15 Hz. Bad channels were identified as having activity four standard deviations away from the mean of their

surrounding electrodes. ERP epochs were defined relative to the response, from 1000 ms pre- to 2000 ms post-response. The ICA utility in EEGlab software (Delorme & Makeig, 2004) was used to derive eye-blink components; then, using a template matching algorithm (Jung, Makeig, Westerfield, Courschesne, & Sejnowski, 2000), blink components were identified and removed from the data.

Classic time-domain ERP response-locked ERP components relative to FA's were extracted: the ERN/Ne, the mean amplitude of the negative deflection occurring 0 – 100 ms, and the Pe, the mean amplitude of the positive deflection, occurring 94 – 500 ms. A subset of nine electrodes representing maximal time-domain component activation were selected for the ERN/Ne [AF3, AFz, AF4, F3, Fz, F4, FC3, FCz, and FC4] and the Pe [C3, Cz, C4, CP3, CPz, CP4, P3, Pz, and P4] and used in subsequent analyses. Response-locked ERP components were baseline corrected with a -200 to -50 ms window relative to FA's. Within each trial, individual electrodes with activity ±100 µV were omitted from analyses. Applying these criteria, 17.64% of response-locked trials were excluded from analyses. An additional data reduction method, PCA, was also performed on the response-locked data (Chapman & McCarry, 1995). Temporal PCA with varimax rotation was performed on the covariance matrix from all scalp electrodes to define a four-component response-locked solution for FA trials accounting for 92.32% of the variance.

In order to evaluate which time-domain ERP components the individual principal components best represented, multiple regression analyses were performed. The time-domain ERP component (i.e., either ERN/Ne or Pe mean amplitude) was entered as the dependent measure and the four principal components were entered as predictor variables. The time-domain ERN/Ne was best predicted by the first principal component

(i.e., PC1) ($\beta = 0.90$, $p < .001$), whereas the time-domain Pe was best predicted by the second principal component (i.e., PC2) ($\beta = 0.29$, $p < .001$), the third principal component (i.e., PC3) ($\beta = 0.12$, $p = .005$), and the fourth principal component ($\beta = 0.64$, $p < .001$). Temporally, PC2 reflects a late subcomponent underlying the Pe, PC3 reflects an early subcomponent underlying the Pe, and PC4 reflects a middle subcomponent underlying the Pe. These three subcomponents underlying the Pe appear to reflect distinct cognitive processes and are associated with unique patterns of hemodynamic activity in subregions of the ACC (Edwards et al., 2012). In particular, the early subcomponent underlying the Pe has been associated with both caudal and rostral ACC hemodynamic activity, the middle subcomponent underlying the Pe has been associated with caudal ACC hemodynamic activity, and the late subcomponent underlying the Pe has been associated with rostral ACC hemodynamic activity (Edwards et al., 2012).

Rearrest Data

Rearrest data, including arrest date and charge, were obtained from the New Mexico Courts Case Lookup website (https://caselookup.nmcourts.gov/caselookup/app). Release dates were obtained from the maximum-security juvenile correctional facility in the state of New Mexico where data collection occurred and the rearrest date of the first offense after release was obtained from the New Mexico Courts Case Lookup website. As of 6/1/2019, $n = 87$ participants (83.7% of the sample) had been re-arrested compared to $n = 17$ participants (16.3% of the sample) who had not been re-arrested.

The outcome variable for Cox regression analyses (described below) was the number of months between release from incarceration and the subject's rearrest date. Offense type was classified into one of 27 common felony categories. In line with

previous literature (Corrado et al., 2004), offenses were classified as violent or non-violent offenses and minor parole and probation violations were not included. Due to the overwhelming majority of participants engaging in violent offenses, we performed analyses to predict time to rearrest of any crime, rather than specifically predicting time to rearrest for violent and non-violent crimes separately. The mean time to rearrest was 24.93 months (SD = 22.96 months), ranging from 1 month to 106 months, and the mean age at release was 18.23 years (SD = 1.24).

RESULTS

Behavioral Results

RTs and frequency for Hits and FA's were analyzed. As expected, participants responded faster to NoGo stimuli (M = 383 ms, SD = 48 ms) than Go stimuli (M = 420 ms, SD = 54 ms), $t(103)$ = 8.13, p < .001. Participants made significantly more errors to NoGo stimuli (M = 24.55, SD = 12.37) compared to Go stimuli (M = 14.46, SD = 24.50), $t(103)$ = 3.76, p < .001. A main effect for PES (Rabbit, 1981) (M = 23 ms, SD = 72 ms), $t(103)$ = 3.33, p = .001, was observed. PES was defined as the difference in RT for Go stimuli preceded by a correct response to NoGo stimuli versus an incorrect response to NoGo stimuli. Thus, incorrect responses to NoGo stimuli should result in a subsequent behavioral slowing to Go stimuli compared to correct responses to NoGo stimuli (Rabbit, 1981). Indeed, participants responded more slowly after error trials (M = 381 ms, SD = 82 ms) compared to correct trials (M = 357 ms, SD = 34 ms) in the current report.

Correlational Analyses

In the current study, number of substance dependencies were significantly positively correlated with PCL:YV total scores (r = .38, p < .001) and the PCL:YV factor

interaction term (r = .36, p < .001). No other significant correlations emerged between PCL:YV scores and covariate measures.

Additionally, PC4 mean amplitude, reflecting a middle subcomponent underlying the Pe, was significantly negatively correlated with PCL:YV total scores (r = -.20, p = .039) consistent with a previously published report (Maurer, Steele, Cope, et al., 2016). No other significant correlations emerged between the PCL:YV factor interaction term, release age, and number of substance dependencies with the time-domain or PCA measures of the ERN/Ne or Pe.

The time-domain ERN/Ne and Pe were significantly positively correlated (r = .22, p = .022). Furthermore, PC1 (reflecting the time-domain ERN/Ne) was significantly positively correlated with the time-domain ERN/Ne (r = .89, p < .001), whereas PC2, PC3, and PC4 (the three subcomponents underlying the time-domain Pe) were significantly positively correlated with the time domain Pe (r = .82, p < .001, r = .72, p < .001, and r = .94, p < .001, respectively). PES was not significantly correlated with any of the predictor variables. In addition, none of the variables included in analyses were significantly correlated with RTs or error rates.

Finally, PC3 mean amplitude was significantly negatively correlated with time to rearrest (r = -.24, p = .027). No other significant correlations emerged between time to rearrest in months and the remaining electrophysiological measures.

Group-Based Analyses

Using independent samples t-tests, participants who were rearrested (n = 87) and those who were not rearrested (n = 17) did not significantly differ with respect to PCL:YV total scores, t(101) = 1.82, p = .072, or PCL:YV factor interaction scores, t(102)

= 1.77, *p* = .079, number of substance dependencies, *t*(102) = 0.38, *p* = .702 or release age, *t*(102) = -1.32, *p* = .191.

In addition, groups did not significantly differ with respect to the mean amplitude of the ERN/Ne, *t*(102) = -0.34, *p* = .733, Pe, *t*(102) = 0.19, *p* = .851, PC1, reflecting the time-domain ERN/Ne, *t*(102) = -0.08, *p* = .939, PC2, reflecting a late subcomponent underlying the Pe, *t*(102) = -0.52, *p* = .607, PC3, reflecting an early subcomponent underlying the Pe, *t*(102) = 0.37, *p* = .710, or PC4, reflecting a middle subcomponent underlying the Pe, *t*(102) = -0.20, *p* = .843.

Data Analysis

Two analytical approaches were used to prospectively predict rearrest. First, logistic regression analyses were performed to identify variables useful in predicting who would or would not be rearrested. A binary outcome variable of rearrested or not rearrested was used as the dependent measure. Second, Cox proportional hazard regressions were used to predict time to rearrest. Cox regression tasks 'time at risk' into account by using the time to rearrest as the outcome variable. Time to rearrest was calculated as the number of months between the participant's release date and rearrest date. The reliability of the Cox regressions was assessed by using bootstrapping with 9,999 iterations.

Logistic Regression Analyses

Logistic regression analyses were performed to identify variables useful in predicting who would or would not be rearrested. The binary recidivism category (i.e., recidivism versus no recidivism) was entered as the dependent measure. In Model 1, covariate measures alone were used to predict recidivism. In Model 2, time-domain

measures of the ERN/Ne and Pe were entered along with covariate measures to predict recidivism, and in Model 3, PCA measures reflecting the time-domain ERN/Ne and Pe were entered along with covariate measures to predict recidivism.

In Model 1, covariate measures entered alone did not produce an overall significant model of rearrest, $X^2 = 5.508$, $p = .138$, and classified individuals with an overall accuracy of 83.7%, those who were rearrested with an accuracy of 100%, and those who were not rearrested with an accuracy of 0%. Additionally, none of the variables entered into analyses were significant predictors of rearrest.

In Model 2, the time-domain ERN/Ne and Pe entered along with covariate measures did not result in an overall significant model of rearrest, $X^2 = 5.798$, $p = .326$, and classified individuals with an overall accuracy of 84.6%, those who were rearrested with an overall accuracy of 100%, and those who were not rearrested with an accuracy of 5.9%. None of the variables entered into analyses were significant predictors of rearrest; the PCL:YV factor interaction term was a moderate predictor of recidivism ($r = .069$).

In Model 3, the PCA measures reflecting the time-domain ERN/Ne and Pe (i.e., PC1, PC2, PC3, and PC4) were entered along with covariate measures did not result in an overall significant model, $X^2 = 6.447$, $p = .489$, and classified individuals with an overall accuracy of 84.6%, those who were rearrested with an accuracy of 100%, and those who were not rearrested with an accuracy of 5.9%. None of the variables entered into analyses were significant predictors of rearrest; the PCL:YV factor interaction term was a moderate predictor of recidivism ($p = .085$).

Cox Proportional Hazard Regression Analyses

As the number of participants who were rearrested during the follow-up period was nearly five times greater than those who were not rearrested (i.e., $n = 87$ compared to $n = 17$), we performed additional Cox proportional hazard regression analyses to see if any aforementioned variables were significant predictors of time to rearrest in a subsample of the $n = 87$ participants who were re-arrested. Predictor variables in the Cox regression analyses were the same as those mentioned in logistic regression analyses.

First, a set of Cox regressions were computed to identify electrophysiological variables useful in predicting time to rearrest. In total, two Cox regression models were performed, one with the time-domain ERN/Ne and Pe entered as predictor variables and another model where the four PCA measures reflecting the time-domain ERN/Ne and Pe were entered into analyses to see if any variables uniquely predicted time to rearrest. These analyses were performed, as the PCA subcomponents were significantly positively correlated with one another. For example, mean amplitude of PC3 and PC4 were significantly positively correlated ($r = .72$, $p < .001$). It is necessary to check collinearity before establishing Cox models, otherwise unstable estimates of coefficients and wide confidence intervals may appear (Ziegel & Allison, 1997).

Model 1 was an overall insignificant predictor of rearrest ($X^2 = 2.588$, $p = .274$), and neither the time-domain measure of the ERN/Ne ($p = .407$) or Pe ($p = .131$) were significant predictors of recidivism.

Model 2 was a significant predictor of rearrest ($X^2 = 13.200$, $p = .010$), and the mean amplitude of PC3, an early subcomponent of the time-domain Pe, emerged as a significant predictor of rearrest ($p = .006$). The other three PCA subcomponents did not emerge as significant predictors of recidivism. As PC3 emerged as a unique predictor of

time to rearrest, this variable was chosen to be included in the final set of Cox proportional hazard regression analyses performed.

Survival Analysis

Cox proportional-hazard regressions were then used to examine (1) the zero-order effects of PC3 amplitude on means to rearrest for any crime, and (2) the shared and unique influence of PC3 amplitude and other potential risk factors on months to rearrest for any crime. The reliability of the beta coefficients was evaluated by resampling each Cox distribution in a bootstrapping sequence with 9,999 iterations.

Cox Model 1 examined the zero-order effect on PC3 mean amplitude on months to rearrest before entering other covariates into the model. The overall model was statistically significant in this case ($X^2 = 6.921$, $p = .009$) and PC3 mean amplitude was a significant predictor of time to rearrest ($p = .009$). For every unit increase in PC3 amplitude, the probability of rearrest increased 2.00 times (see Figure 4 and Table 4).

Cox Model 2 examined the shared and unique influence of PC3 amplitude and other potential risk factors on months to rearrest. The overall model was statistically significant in this case ($X^2 = 12.459$, $p = .014$), with PC3 mean amplitude ($p = .003$) and release age ($p = .025$) emerging as significant predictors of time to rearrest. For every unit increase in PC3 amplitude, the probability of rearrest increased 2.17 times. For every unit increase in release age, the probability of rearrest increased 1.22 times (see Table 4).

DISCUSSION

Around 40 – 70% of juvenile delinquents will recidivate within one year after institutional release (Gordon et al., 2012; Grunwald et al., 2010). Researchers have identified both static and dynamic risk factors predictive of future recidivism in

adolescent offenders, including male sex, low intelligence (Vermeiren et al., 2002), an earlier age of onset of engaging in criminal behavior, an increased severity of juvenile delinquency (Cottle et al., 2001; Loeber & Farrington, 1998; Loeber et al., 2008; Marczyk et al., 2003; Myner et al., 1998), conduct disorder symptomatology (Kotler & McMahon, 2005; van Dam et al., 2004), anxiety and depression symptomatology (Cottle et al., 2001), ADHD symptomatology (Gordon et al., 2012), substance use severity (Ford, 2005; Hawkins et al., 2000; Myner et al., 1998; Schubert et al., 2011), psychopathic traits (Das, 2008; Marczyk et al., 2003; Walters, 2003), and interaction with peers engaging in criminal activity (Cottle et al., 2001; Pardini et al., 2006). However, compared to adults, individual risk factors may be less stable and accurate in adolescents due to ongoing psychosocial development (Borum, 2000). As such, it would be beneficial to identify potential neurobiological variables with known developmental trajectories (Hoge, 1999); variables deviating from typical adolescent neurodevelopment may help identify neurobiological variables to target with treatment intervention to reduce the high recidivism rate in adolescent offenders. One such neurobiological variable may be the amplitude of the Pe ERP component, as it has an established and stable neurodevelopmental trajectory (Davies et al., 2004; Ladouceur et al., 2007; Santesso et al., 2006), and it has previously emerged as a significant predictor of future recidivism in incarcerated adult offenders (Steele et al., 2015).

In the current report, increased amplitude of the Pe, measured via PCA, was significantly associated with time to rearrest, which was contrary to our original hypothesis. We had originally hypothesized that reduced Pe amplitude would be significantly associated with time to rearrest in adolescent offenders. The increased Pe

predictive of recidivism in adult offenders might reflect a potential compensatory mechanism, attempting to overcome post-error processing deficits experienced in adolescence (Steele et al., 2015). While contrary to hypotheses, the results in the current report are intriguing given the neurodevelopmental trajectory of the amplitude of the Pe. Compared to the amplitude of the ERN/Ne, which increases in amplitude throughout development, reflecting improved efficiency in error-detection and action-monitoring processes, the developmental trajectory of the Pe is rather invariant, exhibiting similar amplitude between youth and adult samples (Davies et al., 2004; Ladouceur et al., 2007; Santesso et al., 2006). Increased Pe amplitude may thus reflect a potential biomarker for the identification of life-course persistent antisocial offenders.

Moffitt (1993) had previously suggested that adolescent-limited and life-course persistent antisocial offenders may be differentiated by neuropsychological impairments that occurred during childhood in life-course persistent antisocial offenders (Moffitt, 1993). Supporting this notion, poor performance on neuropsychological tests associated with executive control has been previously associated with persistent delinquency (Ogilvie, Stewart, Chan, & Shum, 2011). This, combined with results obtained from previous neuroimaging studies in adult samples (Aharoni et al., 2013; Kiehl et al., 2018; Steele et al., 2015) and results obtained in the current report, suggest deficits within executive control are predictive of recidivism. Error-related processing is an important function of executive control and associated with a neural network comprising the ACC, anterior insula, and other regions of the prefrontal cortex that are well connected to the thalamus and basal ganglia (Posner, Rothbart, Sheese, & Tang, 2007). Source localization attempts (Dehaene, Posner, & Tucker, 1994; Hermann, Rommler, Ehlis, Heidrich, &

Fallgatter, 2004) and fMRI (Edwards et al., 2012; van Veen & Carter, 2002) studies

converge on the ACC as the generator for the Pe ERP component. Additionally, PC3,

reflecting an early subcomponent of the Pe, emerged as the strongest predictor of time to

rearrest in the current report. The early subcomponent of the Pe has been previously

associated with both cACC and rACC hemodynamic activity (Edwards et al., 2012).

Reduced hemodynamic activity in the cACC has also been shown to predict future

recidivism in incarcerated adult offenders (Aharoni et al., 2013; Steele et al., 2015). Thus,

increased Pe amplitude, combined with previous results obtained via neuroimaging

approaches, suggests that the caudal ACC and potentially rostral ACC are dysfunctional

in youth with an increased propensity towards recidivism. In addition, while the Pe has

shown invariant development throughout adolescence, there is evidence that the Pe

amplitude is malleable, increasing in amplitude through mindfulness meditation training

(M. J. Larson et al., 2013). Thus, this developmental anomaly of increased Pe amplitude

in youth with an increased risk of recidivism may be able to stabilize in amplitude

through specialized treatment intervention approaches, including mindfulness.

Mindfulness-based interventions are feasible in juvenile correctional facilities

(Himelstein, Hastings, Shaprio, & Heery, 2012) and have been previously associated with

reduced recidivism rates (Bleick & Abrams, 1987).

In the current report, we also observed that age at release was a significant

predictor of future recidivism. However, there typically exists a negative relationship

between age at release and future recidivism, with older youth being less likely to

recidivate compared to younger youth (Baglivio et al., 2016; Mallett et al., 2012; Parsons

Winokur et al., 2008). In the current study, we found that older age at release was

associated with a quicker time to rearrest. As the age at release in our sample was restricted (i.e., ranging from 14 to 21 years old at the time of release), this suggests that perhaps the older individuals in this sample could be best described as life-course persistent antisocial offenders. Indeed, when forcing a binary split in terms of age of release in the current sample, nearly 80% of the offenders in the older age of release group were already re-incarcerated by 24 months; for those younger in age, 80% had been re-incarcerated by 45 months. For life-course persistent offenders, the transition from adolescence to adulthood is characterized by increasing severity of offenses and an increase in lethal violence (Farrington, 2003; Piquero, 2001), and as such, once released, the older participants in the study performed more serious crimes, and we re-arrested at a quicker rate compared to youth who were younger. Additionally, participants in the younger age group were typically under 18 years of age, and thus when released from the correctional facility, went back to live with their families and/or caregivers, which likely served as a protective factor.

Limitations

There are some limitations to note in the current report which may reduce the generalizability of our current results. First, we recruited participants from a maximum-security juvenile correctional facility, and it is not clear whether the results obtained in our current report extend to lower risk samples of incarcerated juvenile offenders. Specifically, our sample had a very small base rate of those who were not re-arrested (16.3%). Thus, in our current report, we cannot speak of potential protective variables that are associated with reduced recidivism rates. Even in our current sample, we reported non-significant independent sample *t*-test and logistic regression analyses, likely due to

the substantial sample size difference between youth who were rearrested and those who were not rearrested.

Second, it is not clear whether our results extend to incarcerated juvenile female offenders. Important to note, gender differences exist for both the ERN/Ne and Pe amplitude, whereby males exhibit greater amplitude of the ERN/Ne and Pe compared to females (Larson et al., 2011). Thus, it's entirely possible that increased Pe amplitude does not similarly predict recidivism in incarcerated juvenile female offenders. In addition, it has been suggested that different variables may be predictive of recidivism for male and female juvenile offenders, with more variables being significant predictors of recidivism for male juvenile offenders compared to female juvenile offenders (Minor, Wells, & Angel, 2008; Schwalbe, Fraser, Day, & Cooley, 2006; Trulson, Marquart, Mullings, & Caeti, 2005). Thus, it is imperative for future research to see whether similar or dissimilar neurobiological variables emerge as significant predictors of recidivism for male and female juvenile offenders.

Finally, while we included important variables in analyses known to predict juvenile recidivism, this was by no means an exhaustive list of variables. For example, socioeconomic status (Cottle et al., 2001; Onifade, Petersen, Bynum, & Davidson, 2011), IQ scores (Cottle et al., 2001; Ganzer & Sarason, 1973; Haynes & Bensch, 1981), and impulsivity (Miner, 2002) have been previously been shown to be predictors of future recidivism in youth samples. In addition, we did not replicate an effect routinely observed in previous papers showing that scores on the PCL:YV predict future recidivism (Asscher et al., 2011; Corrado et al., 2004; Vincent et al., 2008). This may because our current study incorporated the use of the PCL:YV factor interaction term, rather than individual

PCL:YV factor scores in order to reduce multicollinearity effects, or may also potentially reflect a ceiling effect, as youth in our high-risk sample all tended to score very high on psychopathic traits.

Conclusions

The results obtained in the current report extend previous studies identifying increased Pe amplitude in adult offenders (Steele et al., 2015) and age at release in youth offenders (Baglivio et al., 2016; Mallett et al., 2012; Parsons Winokur et al., 2008) as significant predictors of time to rearrest. Given the developmental trajectory of the Pe amplitude (Davies et al., 2004; Ladouceur et al., 2007; Santesso et al., 2006), our results suggest that increased amplitude of the Pe may be a potential biomarker for the identification of life-course persistent adolescent offenders. The amplitude of the Pe has been shown to be malleable, increasing in amplitude through mindfulness meditation intervention training (M. J. Larson et al., 2013). Thus, this developmental anomaly of increased Pe amplitude in youth with an increased risk of recidivism may be able to stabilize in amplitude through specialized treatment intervention approaches, including mindfulness.

Study 4: Reduced structural integrity of the corticospinal tract predictive of recidivism in a sample of incarcerated male adolescent offenders

INTRODUCTION

While previous studies attempting to identify neurobiological deficits predictive of future recidivism have incorporated ERPs (Steele et al., 2015), and both functional (Aharoni et al., 2013; Steele et al., 2015) and structural MRI (Kiehl et al., 2018), no study has incorporated the use of DTI to predict recidivism. This may be an especially important consideration for youth samples, given the developmental trajectory of WM. Compared to gray matter, which follows an inverted U-shaped curve that peaks during adolescence and decreases into early adulthood, WM increases steadily throughout childhood and adolescence (Giedd et al., 1999). FA values increase in value in WM tracts throughout development, reflecting increased myelination (Beaulieu, 2002), and reduced FA values have been routinely associated with cognitive deficits in both healthy and clinical populations (Mettenburg et al., 2012; Quinque et al., 2012; Turken et al., 2008; Wozniak et al., 2007), as performance on complex cognitive tasks requires the coordinated activity of distributed brain regions. Thus, reduced FA suggests reduced long-range transmission of information across distributed brain regions (Mesulam, 2000).

Stated previously, it may be especially important to identify neurobiological variables predictive of future recidivism in adolescent samples with a known developmental course (Hoge, 1999). Results that deviate significantly from typical adolescent neurodevelopment may help identify neurobiological variables to potentially target with treatment intervention approaches to potentially reduce the high recidivism rate in adolescent offenders. One WM tract that may be potentially related to an increased

propensity for future recidivism is the corticospinal tract. Like the Pe ERP component, the typical development of the corticospinal tract is well-known, as FA values in the corticospinal tract reach adult myelination levels very early in development, making it one of the first WM tracts to fully develop (Kochunov et al., 2012), showing partial myelination even at birth (Flechsig, 1901). FA values in the corticospinal tract tend to peak around two years of age and remain relatively unchanged after this point throughout the lifespan (Gao et al., 2009).

The corticospinal tract contains fibers running from the primary motor, premotor, supplementary motor, somatosensory, parietal, and cingulate cortex to the spinal cord, and plays a significant role in the control of complex, voluntary movements (Rizzolatti & Luppino, 2001). This tract may be a particularly important WM tract to target in relation to recidivism propensity, as it plays a significant role in impulsivity, a key characteristic associated with life-course persistent antisocial offenders (Leverso et al., 2015). In healthy control participants, the corticospinal tract plays an important role in modulating impulsivity (Brown, Manuck, Flory, & Hariri, 2006; Farr, Hu, Zhang, & Li, 2012), with higher FA in the right corticospinal tract associated with less impulsive behavior on a delay discounting task in healthy adolescents (Olson et al., 2009). Additionally, lower FA values have been observed in the corticospinal tract in populations characterized by heightened impulsivity, including those meeting criteria for methamphetamine use disorder (Uhlmann et al., 2016) and ADHD (Chuang, Wu, Huang, Weng, & Yang, 2013; L. S. Hamilton et al., 2008).

There is strong evidence that impulsivity is an important predictor in future delinquent behavior (Farrington et al., 1990; Gottfredson & Hirschi, 1990; Vitacco et al.,

2002; Vitacco & Rogers, 2001). Additionally, adolescents who continue to engage in criminal activity as adults do not appear to "age out" of impulsivity (Leverso et al., 2015). Due to the strong link between future criminal activity and impulsivity, combined with the aforementioned negative relationship between the structural integrity of the corticospinal tract and impulsivity, we specifically hypothesized that reduced structural integrity of the corticospinal tract would be associated with a quicker time to rearrest in a sample of $n = 110$ incarcerated male adolescent offenders. In addition, we explored whether the structural integrity of the anterior thalamic radiation, cingulum (i.e., cingulate gyrus and hippocampus), forceps major, forceps minor, inferior fronto-occipital fasciculus, inferior longitudinal fasciculus, superior longitudinal fasciculus, arcuate fasciculus, and uncinate fasciculus was also predictive of future recidivism. As we did not have any strong *a priori* hypotheses as to whether or not these WM tracts would be significantly related to future recidivism, these latter analyses remained exploratory.

METHOD

Participants

Participants were recruited from a maximum-security juvenile correctional facility in the state of New Mexico who participated in a larger overall study (SWANC-Y) and who had participated in DTI collection while incarcerated and had reliable follow-up information post-release. Exclusionary criteria for this study included: occurrence of a TBI accompanied with a significant loss of consciousness, due to the tremendous role TBIs play in WM structural integrity (Yuan et al., 2007), an estimated IQ less than 70, meeting diagnostic criteria for psychosis, or not satisfying quality control criteria. None of the participants met any of the exclusionary criteria, resulting in a final sample of $n =$

110 incarcerated male adolescent offenders, ranging from 14 to 19 years of age ($M =$ 17.56 years, $SD = 1.09$ years) at the time of MRI data collection. The sample was predominantly right-handed (10% reported being left-hand dominant). Participants largely self-identified as Hispanic/Latino (79%), with the remaining self-identifying as American Indian or Alaskan Native (8%), White (7%), or Black or African American (3%). Three percent of the sample chose not to disclose their race or ethnicity.

Initial contact was made with potential study participants and informed consent was obtained. Individuals 18 years of age or older provided written informed consent and individuals younger than 18 years of age provided written informed assent in conjunction with parent/guardian consent. Participants were informed of their right to terminate participation at any point, the lack of institutional benefits, and that their participation would not affect their facility or parole status. Participants received remuneration at the hourly labor wage of the facility. All research protocols were approved by E&I, OHRP, and the juvenile detention centers where data collection occurred.

Assessments

Data from several potential risk factors were obtained to examine the incremental utility of the DTI measures. These measures were similar to those previously included in neuroprediction models of recidivism (Aharoni et al., 2013; Steele et al., 2015), modified for age appropriateness, and were the same as those included in Study 3. These measures included psychopathic traits, number of substance dependencies, and age at release.

Psychopathic traits were assessed using the Hare PCL:YV (Forth et al., 2003). The mean PCL:YV total score for the sample was 24.89 ($SD = 5.93$) (ranging from 8 to 35). The Cronbach's alpha for the PCL:YV (all items) was .83 in the current sample,

reflecting good internal consistency. In addition to PCL:YV total scores, we incorporated

the use of a two-factor model of psychopathic traits, with Factor 1 comprising

interpersonal and affective traits, and Factor 2 consisting of lifestyle and

antisocial/developmental traits (Neumann et al., 2006). The mean PCL:YV Factor 1 score

was 7.30 (SD = 3.08) and the mean Factor 2 score was 15.28 (SD = 3.05) in the current

sample. PCL:YV Factor 1 and 2 scores were significantly positively correlated (r = .59, p

< .001), consistent with previous reports (Mailloux et al., 1997). In analyses performed,

we included the PCL:YV factor interaction term (i.e., PCL:YV Factor 1 multiplied by

PCL:YV Factor 2) to reduce multicollinearity in analyses performed (Corrado et al.,

2004; Skeem & Mulvey, 2001) (M = 117.03, SD = 61.48).

    Substance dependence and psychopathology were assessed using the K-SADS

(Kaufman et al., 1997). Number of substance dependencies were calculated by summing

the total number of substances (both alcohol and drug, including cannabis, stimulants,

sedatives/hypnotics/anxiolytics, cocaine, opioids, phencyclidine [PCP], hallucinogens,

solvent/inhalants, and other substances) for which participants met lifetime dependence

diagnoses (M = 2.30, SD = 1.62). In addition to previously being used as covariate

measures in previous reports, these covariate measures have also been shown to be

significant predictors of recidivism in youth samples, including PCL:YV scores (Corrado

et al., 2004; Vincent et al., 2008), age at release (Baglivio et al., 2016; Mallett et al.,

2012; Parsons Winokur et al., 2008), and substance use severity (Colins et al., 2011;

Hoeve et al., 2013; Myner et al., 1998).

    Assessments were also administered to assess IQ and TBI for exclusionary

purposes. Full-scale IQ was estimated using the Vocabulary and Matrix Reasoning sub-

tests of the Wechsler Adult Intelligence Scale – 3rd Edition (WAIS-III) (Wechsler, 1997) for participants sixteen years of age or older and from the Wechsler Intelligence Scale for Children – 4th Edition (WISC-IV) (Wechsler, 2003) for participants younger than sixteen years of age ($M = 93.48$, $SD = 11.85$). History of TBI was evaluated using a post-head injury symptoms questionnaire (King et al., 1995).

MRI Data Acquisition

Diffusion-weighted echo-planar MRI was acquired at the juvenile correctional facilities where data collection occurred using the Mind Research Network's Siemens 1.5T Avanto Mobile MRI System equipped with a 12-element head coil. Diffusion sensitizing gradients were applied along 30 non-collinear directions (b value = 800 s/mm^2). Five interleaved nondiffusion weighted (b value = 0 s/mm^2) volumes were collected during each run to enable corrections for motion and eddy current distortions. Images were collected with the following parameters: repetition time (TR) = 9200 ms, echo time (TE) = 84 ms, field of view = 256 x 256 mm, matrix size = 128 x 128, slice thickness = 2 mm, no gap, voxel size = 2 x 2 x 2 mm^3, 70 slices. The sequence was repeated twice, and the data were combined to improve the signal-to-noise ratio. Head motion was limited using padding and restraint.

DTI Analyses

All images were visually inspected for quality, leaving a final sample of $n = 254$ participants. Brain extraction from the b0 image to remove non-brain tissue, correction of eddy current-induced possible distortions of diffusion-weighted images, and construction of Fractional Anisotropy (FA) maps were performed using the FMRIB's Diffusion Toolbox (FDT) in FMRIB Software Library (FSL) version 4.1

(www.fmrib.ox.ac.uk/fsl/). Tract Based Spatial Statistics (TBSS) workflow (Smith et al., 2006) of FSL was used to non-linearly warp the individual FA maps to the 1.0 x 1.0 x 1.0 mm^3 FA template in the MNI space. To minimize the partial volume effect, non-linearly warped FA images were averaged to construct the WM skeleton image. Subsequently, ROI averaged values were extracted from each participant from the intersection of the skeleton image and the Johns Hopkins University (JHU) WM atlas ROIs (Mori et al., 2005). ROIs from the JHU atlas included the following: 1) left anterior thalamic radiation, (2) right anterior thalamic radiation, (3) left corticospinal tract, (4) right corticospinal tract, (5) left cingulum (i.e., cingulate gyrus), (6) right cingulum (i.e., cingulate gyrus), (7) left cingulum (i.e., hippocampus), (8) right cingulum (i.e., hippocampus), (9) forceps major, (10) forceps minor, (11) left inferior fronto-occipital fasciculus, (12) right inferior fronto-occipital fasciculus, (13) left inferior longitudinal fasciculus, (14) right inferior longitudinal fasciculus, (15) left superior longitudinal fasciculus, (16) right superior longitudinal fasciculus, (17) left uncinate fasciculus, (18) right uncinate fasciculus, (19) temporal portion of the left superior longitudinal fasciculus, and (20) temporal portion of the right superior longitudinal fasciculus.

Rearrest Data

Rearrest data, including arrest date and charge, were obtained from the New Mexico Courts Case Lookup website (https://caselookup.nmcourts.gov/caselookup/app). Release dates were obtained from the maximum-security juvenile correctional facility in the state of New Mexico where data collection occurred, and rearrest dates were obtained from the New Mexico Courts Case Lookup website. All participants included in Study 4 were eventually rearrested after their release date. The outcome variable for Cox

regression analyses (described below) was the number of months between release from incarceration and the subject's rearrest date. Offense type was classified into one of 27 common felony categories. In line with previous literature (Corrado et al., 2004), offenses were classified as violent or non-violent offenses and minor parole and probation violations were not included. Due to the overwhelming majority of participants engaging in violent offenses, we performed analyses to predict time to rearrest of any crime, rather than specifically predicting time to rearrest for violent and non-violent crimes separately. The mean time to rearrest was 25.66 months ($SD = 20.14$ months), ranging from 6 months to 101 months, and the mean age at release was 18.18 years ($SD = 1.21$).

RESULTS

Correlational Results

In the current report, number of substance dependencies were significantly correlated with the PCL:YV factor interaction term ($r = .30$, $p = .002$) but were not significantly correlated with age at release ($r = .15$, $p = .126$). Age at release was not significantly correlated with the PCL:YV factor interaction term ($r = -.07$, $p = .449$).

For the *a priori* WM tract of interest, the corticospinal tract, FA values in the left corticospinal tract were not significantly correlated with the PCL:YV factor interaction term ($r = .04$, $p = .690$), number of substance dependencies ($r = -.17$, $p = .073$), or age at release ($r = .08$, $p = .410$). Similarly, FA values in the right corticospinal tract were not significantly correlated with the PCL:YV factor interaction term ($r = -.06$, $p = .528$), number of substance dependencies ($r = -.17$, $p = .082$), or age at release ($r = .02$, $p = .826$). For the additional WM tracts investigated, FA values in the temporal portion of the superior longitudinal fasciculus (i.e., the arcuate fasciculus) were significantly negatively

correlated with number of substance dependencies ($r = .20$, $p = .038$). No other significant correlations emerged between FA values in any of the WM ROIs or covariate measures.

Finally, FA values in the right corticospinal tract were significantly positively correlated with time to rearrest ($r = .21$, $p = .026$). No other significant correlations emerged between time to rearrest in months and FA values in any of the remaining WM ROIs.

Cox Proportional Hazard Regression Analyses

We performed Cox proportional hazard regression analyses to see if any aforementioned variables were significant predictors of time to rearrest. Predictor variables in the Cox regression analyses included FA values from the WM tracts, the PCL:YV factor interaction term, age at release, and number of substance dependencies.

Cox proportional-hazard regression was used to examine (1) the zero-order effects of FA values within the right corticospinal tract to months to rearrest for any crime, and (2) the shared and unique influence of FA values in the right corticospinal tract and other potential risk factors on months to rearrest for any crime. The reliability of the beta coefficients was evaluated by resampling each Cox distribution in a bootstrapping sequence with 9,999 iterations.

Cox Model 1 examined the zero-order effect on FA values within the right corticospinal tract to months to rearrest for any crime before entering other covariates into the model. The overall model was statistically significant in this case ($X^2 = 5.986$, $p = .014$), FA values in the right corticospinal tract were a significant predictor of time to rearrest ($p = .011$). The exp(B) value here was 0.000034, suggesting a very large

difference between groups (i.e., when performing a binary split on FA values in the right corticospinal tract), and the coefficients value (B) was negative, suggesting that individuals exhibiting reduced FA values in the right corticospinal tract were associated with a quicker time to rearrest (see Table 5).

Cox Model 2 examined the shared and unique influence of FA values within the right corticospinal tract and other potential risk factors on months to rearrest. The overall model was statistically significant in this case (X^2 = 16.619, p = .002), with FA values in the right corticospinal tract (p = .013) and release age (p = .005) emerging as a significant predictor of time to rearrest. The exp(B) value was 0.000069 for FA values in the right corticospinal tract, suggesting a very large difference between groups (i.e., when performing a binary split on FA values in the right corticospinal tract), and the coefficients value (B) was negative, suggesting that individuals exhibiting reduced FA values in the right corticospinal tract were associated with a quicker time to rearrest (see Figure 5 and Table 5). In addition, age at release was associated with faster time to rearrest; for every unit increase in increase in related age, the probability of rearrest increased 1.07 times (see Table 5).

We also performed additional exploratory analyses, to see if FA values in nineteen additional WM tracts, including the L/R anterior thalamic radiation, left corticospinal tract, L/R cingulum (i.e., cingulate gyrus, L/R cingulum (i.e., hippocampus), forceps major, forceps minor, L/R inferior fronto-occipital fasciculus, L/R inferior longitudinal fasciculus, L/R superior longitudinal fasciculus, L/R uncinate fasciculus, and L/R arcuate fasciculus. However, there were not any significant associations between FA values in any of the aforementioned ROIs and time to rearrest.

DISCUSSION

Consistent with hypotheses, reduced FA in the right corticospinal tract was associated with a faster time to rearrest in a sample of $n = 110$ incarcerated male adolescent offenders. The corticospinal tract plays an important role in the control of complex, voluntary movement (Rizzolatti & Luppino, 2001) and helps regulate impulsivity (Brown et al., 2006; Farr et al., 2012). For example, higher FA has been associated with less impulsive behavior on a delay discounting task (Olson et al., 2009) and lower FA values have been observed in the corticospinal tract in populations characterized by heightened impulsivity, including those meeting criteria for methamphetamine use disorder (Uhlmann et al., 2016) and ADHD (Chuang et al., 2013; L. S. Hamilton et al., 2008).

Reduced FA values in the corticospinal tract suggest structural integrity deficits, including light axonal packing, leaving more intercellular water, and less restriction of diffusion, thus, lowering FA values. In addition, reduced FA suggests that long-range transmission of information across distributed brain regions is implicated in incarcerated youth with a faster proclivity towards recidivism (Mesulam, 2000). The *corona radiata* contains the corticospinal tract, and increased WM microstructural integrity in the corona radiata has been previously associated with better cognitive control (Chaddock-Heyman et al., 2013; Seghete, Herting, & Nagel, 2013). Additionally, better executive control has been previously associated with reduced impulsivity (Romer et al., 2009). Thus, reduced FA in the corticospinal tract, and by extension, the corona radiata, may play a role with both executive function deficits and the impulsive nature characteristic of incarcerated youth at greater risk of recidivism.

Increased FA values in the right corticospinal tract have been previously associated with less impulsive performance and reduced delay discounting in healthy adolescents (Olson et al., 2009). High rates of delay discounting are typically found in participants who are willing to forgo greater rewards available and show a preference for smaller rewards that are available immediately (Matta, Gonçalves, & Bizarro, 2012). In Gottfredson & Hirschi (1990)'s '*General Theory of Crime*', the authors posit that impulsive youth lack self-control, making them more likely to engage in criminal activity, as it provides them with immediate gratification of their desires. These youth may be more likely to interact with other peers engaging in criminal behavior in order to achieve immediate gratification of their desires as well (Gottfredson & Hirschi, 1990). Interacting with peers who engage in criminal behavior has been shown to be a strong predictor of future recidivism (Cottle et al., 2001; Pardini et al., 2006).

Reduced FA in the right corticospinal tract may serve as a potential biomarker for identifying individuals at greater risk of recidivism. FA values in the corticospinal tract reach full maturity very early in development, around two years of age and remain unchanged throughout development (Gao et al., 2009). Although this study did not have a control group of participants who were not rearrested, reduced FA values in the right corticospinal tract were associated with a faster time to rearrest compared to those who exhibited increased FA, and may help identify those who are at greatest risk of engaging in future criminal activity. As higher FA has been previously associated with reduced levels of impulsivity (Olson et al., 2009), youth who exhibit reduced structural integrity of the right corticospinal tract may engage in impulsive criminal activity to provide immediate gratification of their desires, without fully considering the consequences of

their behavior. In order to help reduce the high recidivism rate observed in incarcerated adolescent offenders, treatment intervention approaches may be developed to help improve structural integrity in the corticospinal tract. For example, individuals who participate in mindfulness meditation (Wheeler, Arnkoff, & Glass, 2017) and other forms of meditation (Luders, Clark, Narr, & Toga, 2011) exhibit increased FA in the corticospinal tract compared to control participants. As forms of meditation have been successfully implemented in juvenile correctional facilities before (Himelstein et al., 2012), this could provide an innocuous intervention approach attempting to reduce recidivism in incarcerated adolescent offenders.

In the current report, we also observed that age at release was a significant predictor of future recidivism. However, there typically exists a negative relationship between age at release and future recidivism, with older youth being less likely to recidivate compared to younger youth (Baglivio et al., 2016; Mallett et al., 2012; Parsons Winokur et al., 2008). In the current study, we found that age at release was associated with a quicker time to rearrest. As the age at release in our sample was restricted (i.e., ranging from 14 to 21 years old at the time), this suggests that perhaps the older individuals in this sample could be best described as life-course persistent offenders. Indeed, when forcing a binary split in terms of age of release in the current sample, nearly 80% of the offenders in the older age of release group were re-incarcerated by 36 months. For life-course persistent offenders, the transition from adolescence to adulthood is characterized by increasing severity of offenses and an increase in lethal violence (Farrington, 2003; Piquero, 2001).

Limitations

Consistent with our third study, there are some limitations to note in the current report which may reduce the generalizability of our current results. First, we recruited participants from a maximum-security juvenile correctional facility, and it is not clear whether the results obtained in our current report extend to lower risk samples of incarcerated juvenile offenders. Specifically, we were not able to investigate whether increased FA values in the right corticospinal tract or other WM tracts were associated with individuals who were not rearrested. Thus, in our current report, we cannot speak of potential protective variables that reduce recidivism rates.

Second, it is not clear whether our results extend to incarcerated juvenile female offenders. Important to note, males and females exhibit microstructure differences, whereby males exhibit increased FA values in the thalamus, corpus callosum, cingulum (Menzler et al., 2011) and superior longitudinal fasciculus (Kanaan et al., 2012) compared to females. Additionally, females have exhibited increased FA values in the corticospinal tract compared to males (Schmithorst, Holland, & Dardzinski, 2008). Thus, it is entirely possible that reduced FA values in the right corticospinal tract do not similarly predict recidivism in incarcerated juvenile female offenders. In addition, it has been suggested that different variables may be predictive of recidivism for male and female juvenile offenders, with more variables being significant predictors of recidivism for male juvenile offenders compared to female juvenile offenders (Minor et al., 2008; Schwalbe et al., 2006; Trulson et al., 2005). Thus, it is imperative for future research to see whether similar or dissimilar neurobiological variables emerge as significant predictors of recidivism for male and female juvenile offenders.

Finally, while we included important variables in analyses known to predict juvenile recidivism, this was by no means an exhaustive list of variables. For example, socioeconomic status (Cottle et al., 2001; Onifade et al., 2011), IQ scores (Cottle et al., 2001; Ganzer & Sarason, 1973; Haynes & Bensch, 1981), and impulsivity (Miner, 2002) have been previously been shown to be predictors of future recidivism in youth samples. In addition, we did not replicate an effect routinely observed in previous papers showing that scores on the PCL:YV predict future recidivism (Asscher et al., 2011; Corrado et al., 2004; Vincent et al., 2008). This may because our current study incorporated the use of the PCL:YV factor interaction term, rather than individual PCL:YV factor scores in order to reduce multicollinearity effects, or may also potentially reflect a ceiling effect, as youth in our high-risk sample all tended to score very high on psychopathic traits.

Conclusions

To our knowledge, this was the first study to investigate whether WM structural integrity obtained via DTI was useful in predicting time to recidivism in a sample of incarcerated adolescent offenders. In the current study, reduced FA in the right corticospinal tract was associated with faster time to rearrest. Reduced FA in the right corticospinal tract suggests long-range transmission of information across distributed brain regions is implicated in time to rearrest (Mesulam, 2000) and may relate to the impulsive nature characteristic of youth who are more likely to engage in criminal activity (Farrington et al., 1990; Gottfredson & Hirschi, 1990; Vitacco et al., 2002; Vitacco & Rogers, 2001). As the corticospinal tract is one of the first to reach maturity, showing comparable FA values between two-year-old children and adults (Gao et al.,

2009), reduced FA in the right corticospinal tract may reflect a potential biomarker for the development of life-course persistent antisocial offenders.

GENERAL DISCUSSION

In this dissertation, the following four analyses were performed: (1) a group ICA analysis to identify response inhibition and error-related processing functional connectivity deficits associated with youth scoring high on the PCL:YV, (2) a DTI analysis to identify whether youth with elevated psychopathic traits were characterized by reduced structural integrity in WM tracts, especially the uncinate fasciculus, (3) prediction analyses to identify whether mean amplitude of the Pe ERP component was associated with recidivism, and (4) prediction analyses to identify whether structural integrity of the corticospinal tract and other WM tracts were predictive of recidivism. The following results were obtained in this dissertation. First, in Study 1, we found that youth scoring high on the PCL:YV were associated with functional connectivity deficits. Compared to youth scoring low on the PCL:YV, youth scoring high on the PCL:YV exhibited increased functional connectivity with brain regions involved in successful response inhibition and reduced functional connectivity with brain regions incorporated in standard error-related processing. In the second study, we found that higher PCL:YV scores (especially Factor 1, Facet 1, and Facet 2 scores, reflecting interpersonal and affective psychopathic traits, respectively) were associated reduced structural integrity of the uncinate fasciculus, inferior longitudinal fasciculus, and inferior fronto-occipital fasciculus. In Study 3, we found that increased amplitude of the error-related positivity (Pe) ERP component was associated with a faster time to recidivism in a sample of incarcerated adolescent offenders. Finally, in Study 4, we found that reduced structural integrity of the right corticospinal tract was associated with a faster time to recidivism in a sample of incarcerated adolescent offenders.

In Study 1, we added to the growing body of literature identifying structural and functional deficits associated with youth scoring high on measures of psychopathy. Previous neuroimaging studies have primarily focused on affective and emotional processing deficits in youth with elevated psychopathic traits, observing reduced hemodynamic activity in the amygdala during processing of fearful face stimuli (Jones et al., 2009; Lozier et al., 2014; Marsh et al., 2008; Viding et al., 2012; White et al., 2012), reduced hemodynamic activity in the ACC (Lockwood et al., 2013; Marsh et al., 2013), amygdala (Marsh et al., 2013), putamen (Marsh et al., 2013), and insula (Lockwood et al., 2013; Marsh et al., 2013) when viewing others in pain, and reduced hemodynamic activity in the amygdala during affective theory of mind processing (Sebastian et al., 2012). However, relatively little research has been performed identifying cognitive control deficits in youth with elevated psychopathic traits. To date, youth scoring high on measures of psychopathic traits have exhibited increased N2 ERP amplitude during response inhibition task (Sumich, Sarkar, Hermens, Kelesidi, et al., 2012) and reduced Pe ERP amplitude (Maurer, Steele, Cope, et al., 2016) and hemodynamic activity in subregions of the basal ganglia, including the caudate, NAcc, globus pallidus, substantia nigra, and subthalamic nucleus (Maurer et al., in press) during error-related processing. We sought to expand the limited number of studies performed identifying cognitive control deficits in youth with elevated psychopathic traits by performing ICA functional connectivity analyses.

Compared to youth scoring low on the PCL:YV, we found that youth scoring high on the PCL:YV exhibited increased functional connectivity with brain regions involved in response inhibition, including the precuneus, superior parietal lobule, postcentral

gyrus, paracentral lobule, and cingulate gyrus (Component 24) and the ACC, superior, medial, and middle portions of the frontal gyrus, and subcallosal gyrus (Component 66). In addition, we found that youth scoring high on the PCL:YV were characterized by reduced functional connectivity with brain regions involved in error-related processing, including the insula, precentral gyrus, superior temporal gyrus, inferior parietal lobule, and lentiform nucleus (Component 69). Our results help support previous research identifying compensatory mechanisms during response inhibition (Sumich, Sarkar, Hermens, Kelesidi, et al., 2012) and less efficient processing of error-related information (Maurer, Steele, Cope, et al., 2016; Maurer et al., in press) in youth with elevated psychopathic traits. Results obtained in Study 1 are the first to suggest that youth with elevated psychopathic traits exhibit functional connectivity deficits during cognitive control.

In Study 2, we investigated whether PCL:YV scores were associated with structural integrity deficits. Previous studies with adult psychopathic offenders have routinely identified a negative relationship between psychopathy scores and FA in the right uncinate fasciculus, the main WM tract that connects anterior temporal lobe regions, including the amygdala, to prefrontal structures, including the vmPFC(Craig et al., 2009; Hoppenbrouwers et al., 2013; Lindner et al., 2017; Motzkin et al., 2011; Sobhani et al., 2015; Sundram et al., 2012; Vermeij et al., 2018; Wolf et al., 2015). Reduced FA in the right UF has been reported in youth scoring high on psychopathic traits (Breeden et al., 2015), but this effect has not been replicated in two other studies (Pape et al., 2015; Sarkar et al., 2013), instead showing increased FA in the right UF in youth scoring high on psychopathic traits. One of these latter studies identified additional WM tracts where

youth with elevated psychopathic traits showed increased FA, including the anterior thalamic radiation, corticospinal tract, forceps minor, inferior fronto-occipital fasciculus, and corpus callosum (Pape et al., 2015). In Study 2, we sought to expand upon existing DTI studies identifying structural integrity deficits associated with youth with elevated psychopathic traits by examining the relationship between FA (a measure of structural integrity) in *a priori* WM tracts and PCL:YV scores (total, factor, and facet scores).

Consistent with previous studies performed with both youth and adults with elevated psychopathic traits, we found that psychopathy scores were significantly negatively related to FA in the uncinate fasciculus in a sample of incarcerated male adolescent offenders. Specifically, this negative relationship was driven by PCL:YV Factor 1 and Facet 1 scores, consistent with a previously published study with adult psychopathic offenders identifying interpersonal dysfunction associated with reduced FA in the uncinate fasciculus (Wolf et al., 2015). The results obtained in Study 2 help support a growing body of evidence suggesting dysfunctional connectivity between the amygdala and vmPFC in youth with elevated psychopathic traits (Aghajani, Klapwijk, et al., 2016; Marsh et al., 2008), and may help relate to deficits typically associated with youth with elevated psychopathic traits, including deficits within emotional empathy and processing of fearful face stimuli (Jones et al., 2009; Lozier et al., 2014; Marsh et al., 2008; Viding et al., 2012; White et al., 2012).

Additionally, consistent with previous studies performed with adult psychopathic offenders (Hoppenbrouwers et al., 2013; Sundram et al., 2012; Vermeij et al., 2018), we found that psychopathy scores were negatively related to FA in the right inferior longitudinal fasciculus and bilateral inferior fronto-occipital fasciculus. Specifically, this

negative relationship was driven by PCL:YV Factor 1 and Facet 2 scores, consistent with a previous study with adult psychopathic offenders (Vermeij et al., 2018). Dysfunction occurring within the inferior longitudinal fasciculus and inferior fronto-occipital fasciculus could additionally relate to the affective dysfunction commonly associated with youth with elevated psychopathic traits, including accurately identifying negative emotions including sadness (Blair & Coles, 2000; Blair et al., 2001) and fear (Blair & Coles, 2000; Blair et al., 2001).

In Study 3, we investigated whether error-related processing deficits were associated with future recidivism in a sample of incarcerated male adolescent offenders. Two patterns of juvenile delinquency have been distinguished, including adolescent-limited and life-course persistent antisocial offenders (Moffitt, 1993). The vast majority of adolescent offenders will stop engaging in criminal activity when becoming adults. However, adolescent offenders who continue to engage in criminal activity in adulthood are characterized by an earlier initiation into antisocial behavior (Loeber & Farrington, 2011), heightened impulsivity (Leverso et al., 2015), and neuropsychological deficits (Moffitt, 1993). However, these measures only serve as proxy measures to the brain's inhibitory and cognitive control system, and neurobiological variables may serve as strong predictors of recidivism. In addition, these variables may serve as stronger predictors of future recidivism, as the developmental trajectories of these variables are often well-established (Hoge, 1999). As such, we investigated whether the Pe ERP component was a significant predictor of future recidivism in a sample of incarcerated male adolescent offenders. Not only is the developmental trajectory of the Pe amplitude known (Davies et al., 2004; Ladouceur et al., 2007; Santesso et al., 2006), a previous

study in incarcerated adult offenders found that increased amplitude of the Pe was associated with future recidivism (Steele et al., 2015).

Contrary to our original hypotheses, but consistent with a previously published study (Steele et al., 2015), increased amplitude of the Pe was associated with quicker time to rearrest in a sample of incarcerated adolescent offenders. As the development of the Pe amplitude is rather invariant throughout development, increased amplitude of the Pe may serve as a potential biomarker for the identification of life-course persistent antisocial behavior. Source localization attempts (Dehaene et al., 1994; Hermann et al., 2004) and fMRI (Edwards et al., 2012; van Veen & Carter, 2002) studies converge on the ACC as the generator for the Pe ERP component. Additionally, the early subcomponent underlying the Pe has been previously associated with cACC and rACC hemodynamic activity (Edwards et al., 2012). These suggest that the ACC may be implicated in predicting future recidivism in youth with elevated psychopathic traits, consistent with incarcerated adult offenders (Aharoni et al., 2013; Steele et al., 2015). The amplitude of the Pe has been shown to be malleable, increasing in amplitude through mindfulness meditation intervention training (M. J. Larson et al., 2013). Thus, this developmental anomaly of increased Pe amplitude in youth with an increased risk of recidivism may be able to stabilize in amplitude through specialized treatment intervention approaches, including mindfulness.

Finally, in Study 4, we investigated whether the structural integrity of the right corticospinal tract was associated with quicker time to rearrest. Like the Pe amplitude, the developmental trajectory of structural integrity in the corticospinal tract is well established, as FA values in the corticospinal tract reach adult myelination levels very

early in development, showing comparable FA values between two-year-old children and adults (Gao et al., 2009). The corticospinal tract also plays a significant role in regulating impulsivity, as higher FA in the right corticospinal tract has been associated with less impulsive behavior on a delay discounting task (Olson et al., 2009), and lower FA has been observed in the corticospinal tract in populations characterized by heightened impulsivity, including those meeting criteria for methamphetamine dependence (Uhlmann et al., 2016) and ADHD (Chuang et al., 2013; L. S. Hamilton et al., 2008). Impulsivity plays an important role in predicting future delinquent behavior (Farrington et al., 1990; Gottfredson & Hirschi, 1990; Vitacco et al., 2002; Vitacco & Rogers, 2001), and life-course persistent antisocial offenders do not appear to "age out" of impulsivity (Leverso et al., 2015).

Consistent with hypotheses, reduced structural integrity in the right corticospinal tract was associated with a faster time to recidivism in a sample of $n = 110$ incarcerated adolescent offenders. Reduced FA values suggest long-range transmission of information across distributed brain regions is implicated in incarcerated youth with a faster proclivity towards recidivism (Mesulam, 2000). Incarcerated youth exhibiting reduced FA in the right corticospinal tract are likely associated with self-control deficits, engaging in criminal activity, as it provides them with the immediate gratification of their desires. Reduced FA within the corticospinal tract may also serve as a potential biomarker for identifying individuals at greatest risk of recidivism, understanding the developmental trajectory of this WM tract (Gao et al., 2009).

References

Abou-Elseoud, A., Starck, T., Remes, J., Nikkinen, J., Tervonen, O., & Kiviniemi, V. (2010). The effect of model order selection in group PICA. *Human Brain Mapping, 31*(8), 1207-1216.

Aghajani, M., Colins, O. F., Klapwijk, E. T., Veer, I. M., Andershed, H., Popma, A., . . . Vermeiren, R. R. J. M. (2016). Dissociable relations between amygdala subregional networks and psychopathy trait dimensions in conduct-disordered juvenile offenders. *Human Brain Mapping, 37*, 4017-4033.

Aghajani, M., Klapwijk, E. T., Van der Wee, N. J., Veer, I. M., Rombouts, S. A. R. B., Boon, A. E., . . . Colins, O. F. (2016). Disorganized amygdala networks in conduct-disordered juvenile offenders with callous-unemotional traits. *Biological Psychiatry, 82*, 283-293.

Aharoni, E., Vincent, G. M., Harenski, C. L., Calhoun, V. D., Sinnott-Armstrong, W., Gazzaniga, M. S., & Kiehl, K. A. (2013). Neuroprediction of future rearrest. *Proceedings of the National Academy of Sciences, 110*, 6223-6228.

Allen, E. A., Erhardt, E. B., Damaraju, E., Gruner, W., Segall, J. M., Silva, R. F., . . . Calhoun, V. D. (2011). A baseline for the multivariate comparison of resting-state networks. *Frontiers in Systems Neuroscience, 5*(2), 1-23.

Althouse, A. D. (2016). Adjust for multiple comparisons? It's not that simple. *The Annals of Thoracic Surgery, 101*(5), 1644-1645.

American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders (5th Ed.)*. Arlington, VA: American Psychiatric Publishing.

Anderson, N. E., Maurer, J. M., Steele, V. R., & Kiehl, K. A. (2018). Psychopathic traits associated with abnormal hemodynamic activity in salience and default mode networks during auditory oddball task. *Cognitive, Affective, & Behavioral Neuroscience, 18*(3), 564-580.

Anderson, N. E., & Stanford, M. S. (2012). Demonstrating emotional processing differences in psychopathy using affective ERP modulation. *Psychophysiology, 49*(6), 792-806.

Anderson, N. E., Steele, V. R., Maurer, J. M., Bernat, E. M., & Kiehl, K. A. (2015). Psychopathy, attention, and oddball target detection: New insights from PCL-R Facet scores. *Psychophysiology, 52*(9), 1194-1204.

Anderson, N. E., Steele, V. R., Maurer, J. M., Rao, V., Koenigs, M. R., Decety, J., . . . Kiehl, K. A. (2017). Differentiating emotional processing and attention in

psychopathy with functional neuroimaging. *Cognitive, Affective, & Behavioral Neuroscience, 17*(3), 491-515.

Annie E. Casey Foundation (2013). *Reducing youth incarceration in the United States*. Baltimore, MD.

Aron, A. R., Durston, S., Eagle, D. M., Logan, G. D., Stinear, C. M., & Stuphorn, V. (2007). Converging evidence for a fronto-basal-ganglia network for inhibitory control of action and cognition. *The Journal of Neuroscience, 27*(44), 11860-11864.

Aron, A. R., & Poldrack, R. A. (2006). Cortical and subcortical contributions to Stop signal response inhibition: Role of the subthalamic nucleus. *Journal of Neuroscience, 26*, 2424-2433.

Aron, A. R., Robbins, T. W., & Poldrack, R. A. (2004). Inhibition and the right inferior frontal cortex. *Trends in Cognitive Sciences, 8*, 170-177.

Ashtari, M., Cervellione, K. L., Hasan, K. M., Wu, J., McIlree, C., Kester, H., . . . Kumra, S. (2007). White matter development during late adolescence in healthy males: A cross-sectional diffusion tensor imaging study. *NeuroImage, 35*(2), 501-510.

Asscher, J. J., van Vugt, E. S., Stams, G. J. J. M., Deković, M., Eichelsheim, V. I., & Yousfi, S. (2011). The relationship between juvenile psychopathic traits, delinquency, and (violent) recidivism: A meta-analysis. *The Journal of Child Psychology and Psychiatry, 52*(11), 1134-1143.

Ayalon, L., Ancoli-Israel, S., & Drummond, S. P. (2009). Altered brain activation during response inhibition in obstructive sleep apnea. *Journal of Sleep Research, 18*(2), 204-208.

Baglivio, M. T., Wolff, K. T., Piquero, A. R., Bilchik, S., Jackowski, K., Greenwald, M. A., & Epps, N. (2016). Maltreatment, child welfare, and recidivism in a sample of deep-end crossover youth. *Journal of Youth and Adolescence, 45*, 625-654.

Bannbers, E., Gingnell, M., Engman, J., Morell, A., Sylvén, S., Skalkidou, A., . . . Poromaa, I. S. (2013). Prefrontal activity during response inhibition decreases over time in the postpartum period. *Behavioural Brain Research, 241*, 132-138.

Barnea-Goraly, N., Menon, V., Eckert, M., Tamm, L., Bammer, R., Karchemskiy, A., . . . Reiss, A. L. (2005). White matter development during childhood and adolescence: A cross-sectional diffusion tensor imaging study. *Cerebral Cortex, 15*(12), 1848-1854.

Barratt, E. S., & Patton, J. H. (1983). *Impulsivity: Cognitive, behavioral, and psychophysiological correlates*. In M. Zuckerman (Ed.), Biological Bases of

Sensation-Seeking, Impulsivity, and Anxiety (pp. 77-121). Hillsdale, NJ: Lawrence Erlbaum Associates.

Baskin-Sommers, A. R., Brazil, I. A., Ryan, J., Kohlenberg, N. J., Neumann, C. S., & Newman, J. P. (2015). Mapping the association of global executive functioning onto diverse measures of psychopathic traits. *Personality Disorders: Theory, Research, and Treatment, 6*(4), 336-346.

Baskin-Sommers, A. R., Curtin, J. J., Li, W., & Newman, J. P. (2012). Psychopathy-related differences in selective attention are captured by an early event-related potential. *Personality Disorders: Theory, Research, and Treatment, 3*(4), 370-378.

Baskin-Sommers, A. R., Curtin, J. J., & Newman, J. P. (2011). Specifying the attentional selection that moderates the fearlessness of psychopathic offenders. *Psychological Science, 22*(2), 226-234.

Baskin-Sommers, A. R., Curtin, J. J., & Newman, J. P. (2013). Emotion-modulation startle in psychopathy: Clarifying familiar effects. *Journal of Abnormal Psychology, 122*(2), 458-468.

Baskin-Sommers, A. R., Neumann, C. S., Cope, L. M., & Kiehl, K. A. (2016). Latent-variable modeling of brain gray-matter volume and psychopathy in incarcerated offenders. *Journal of Abnormal Child Psychology, 125*(6), 811-817.

Basser, P. J., & Pierpaoli, C. (2011). Microstructural and physiological features of tissues elucidated by quantitative-diffusion-tensor MRI. *Journal of Magnetic Resonance Imaging, 213*(2), 560-570.

Beaulieu, C. (2002). The basis of anisotropic water diffusion in the nervous system - A technical review. *NMR in Biomedicine, 15*, 435-455.

Bellemann, M. E., Spitzer, M., Brix, G., Kammer, T., Loose, R., Schwartz, A., & Guckel, F. (1995). Neurofunctional MRI imaging of higher cognitive performance of the human brain. *Radiology, 35*, 272-282.

Blair, R. J. R. (1995). A cognitive developmental approach to morality: Investigating the psychopath. *Cognition, 52*, 1-29.

Blair, R. J. R. (2003). Neurobiological basis of psychopathy. *The British Journal of Psychiatry, 182*(1), 5-7.

Blair, R. J. R. (2007). The amygdala and ventromedial prefrontal cortex in morality and psychopathy. *Trends in Cognitive Sciences, 11*(9), 387-392.

Blair, R. J. R., & Coles, M. (2000). Expression recognition and behavioural problems in early adolescence. *Cognitive Development, 15*, 421-434.

Blair, R. J. R., Colledge, E., Murray, L., & Mitchell, D. G. (2001). A selective impairment in the processing of sad and fearful expressions in children with psychopathic tendencies. *Journal of Abnormal Child Psychology, 29*, 491-498.

Blair, R. J. R., Jones, L., Clark, F., & Smith, M. (1997). The psychopathic individual: A lack of responsiveness to distress cues? *Psychophysiology, 342*, 192-198.

Bleick, C. R., & Abrams, A. I. (1987). The transcendental meditation program and criminal recidivism in California. *Journal of Criminal Justice, 15*(3), 211-230.

Bleuler, E. (1975). *Lehrbuch der Psychiatrie*. Berlin-Heidelberg-New York.

Blonigen, D. M., Hicks, B. M., Krueger, R. F., Patrick, C. J., & Iacono, W. G. (2006). Continuity and change in psychopathic traits as measured via normal-range personality: A longitudinal-biometric study. *Journal of Abnormal Psychology, 115*, 85-95.

Blumer, D., & Benson, D. F. (1975). *Personality changes with frontal lobe lesions*. In: Benson, D. F., Blumer, D. editors. Psychiatric Aspects of Neurological Disease. Grune and Stratton; New York. pp. 151-170.

Boccaccini, M. T., Epstein, M., Poythress, N. G., Douglas, K. S., Campbell, J., Gardner, G., & Falkenbach, D. M. (2007). Self-report measures of child and adolescent psychopathy as predictors of offending in four samples of justice-involved youth. *Assessment, 14*(4), 361-374.

Boccardi, M., Frisoni, G. B., Hare, R. D., Cavedo, E., Najt, P., Pievani, M., . . . Tiihonen, J. (2011). Cortex and amygdala morphology in psychopathy. *Psychiatry Research, 193*(2), 85-92.

Booth, J. R., Burman, D. D., Meyer, J. R., Lei, Z., Trommer, B. L., Davenport, N. D., . . . Mesulam, M. M. (2003). Neural development of selective attention and response inhibition. *NeuroImage, 20*(2), 737-751.

Borum, R. (2000). Assessing violence risk among youth. *Journal of Clinical Psychology, 56*(10), 1263-1288.

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review, 108*(3), 624-652.

Braet, W., Johnson, K. A., Tobin, C. T., Acheson, R., McDonnell, C. M., Hawi, Z., . . . Garavan, H. (2011). fMRI activation during response inhibition and error processing: The role of the DAT1 gene in typically developing adolescents and those diagnosed with ADHD. *Neuropsychologia, 49*(7), 1641-1650.

Braver, T. S., Barch, D. M., Gray, J. R., Molfese, D. L., & Snyder, A. (2001). Anterior cingulate cortex and response conflict: Effects of frequency, inhibition, and errors. *Cerebral Cortex, 11*, 825-836.

Brazil, I. A., de Bruijn, E. R., Bulten, B. H., von Borries, A. K., van Lankveld, J. J., Buitelaar, J. K., & Verkes, R. J. (2009). Early and late components of error monitoring in violent offenders with psychopathy. *Biological Psychiatry, 65*(2), 137-143.

Breeden, A. L., Cardinale, E. M., Lozier, L. M., VanMeter, J. W., & Marsh, A. A. (2015). Callous-unemotional traits drive reduced white-matter integrity in youths with conduct problems. *Psychological Medicine, 45*, 3033-3046.

Bridwell, D. A., Steele, V. R., Maurer, J. M., Kiehl, K. A., & Calhoun, V. D. (2015). The relationship between somatic and cognitive-affective depression symptoms and error-related ERPs. *Journal of Affective Disorders, 172*, 89-95.

Brown, S. M., Manuck, S. B., Flory, J. D., & Hariri, A. (2006). Neural basis of individual differences in impulsivity: Contributions of corticolimbic circuits for behavioral arousal and control. *Emotion, 6*, 239-245.

Buckner, R. L., Andrews-Hanna, J. R., & Schacter, D. L. (2008). The brain's default network: Anatomy, function, and relevance to disease. *Annals of the New York Academy of Sciences, 1124*, 1-38.

Caldwell, B. M., Anderson, N. E., Harenski, K. A., Sitney, M. H., Caldwell, M. F., Van Rybroek, G. J., & Kiehl, K. A. (2019). The structural brain correlates of callous-unemotional traits in incarcerated male adolescents. *NeuroImage: Clinical, 22*, 101703.

Caldwell, M. F. (2011). Treatment-related changes in behavioral outcomes of psychopathy facets in adolescent offenders. *Law and Human Behavior, 35*(4), 275-187.

Caldwell, M. F., McCormick, D. J., Umstead, D., & Van Rybroek, G. J. (2007). Evidence of treatment progress and therapeutic outcomes among adolescents with psychopathic features. *Criminal Justice and Behavior, 34*(5), 573-587.

Calhoun, V. D., & Adali, T. (2012). Multisubject independent component analysis of fMRI: A decade of intrinsic networks, default mode, and neurodiagnostic discovery. *IEEE Reviews in Biomedial Engineering, 5*, 60-73.

Calhoun, V. D., Adali, T., McGinty, V. B., Pekar, J. J., Watson, T. D., & Pearlson, G. D. (2001). fMRI activation in a visual-perception task: Network of areas detected

using the General Linear Model and Independent Components Analysis. *NeuroImage, 14*(5), 1080-1088.

Calhoun, V. D., Adali, T., Pearlson, G. D., & Pekar, J. J. (2001a). A method for making group inferences from functional MRI data using independent component analysis. *Human Brain Mapping, 14*(3), 140-151.

Calhoun, V. D., Adali, T., Pearlson, G. D., & Pekar, J. J. (2001b). A method for making group inferences using independent component analysis of functional MRI data: Exploring the visual system. *NeuroImage, 13*(6), 88.

Calhoun, V. D., Stevens, M., Pearlson, G. D., & Kiehl, K. A. (2004). fMRI analysis with the general linear model: Removal of latency-induced amplitude bias by incorporation of hemodynamic derivative terms. *NeuroImage, 22*, 252-257.

Calhoun, V. D., Wager, T. D., Krishnan, A., Rosch, K. S., Seymour, K. E., Nebel, M. B., . . . Kiehl, K. A. (2017). The impact of T1 versus EPI spatial normalization templates for fMRI data analysis. *Human Brain Mapping, 38*(11), 5331-5342.

Callard, F., & Marguiles, D. S. (2014). What we talk about when we talk about the default mode network. *Frontiers in Human Neuroscience, 8*, 1-5.

Cardinal, R. N., Parkinson, J. A., Hall, J., & Everitt, B. J. (2002). Emotion and motivation: The role of the amygdala, ventral striatum, and prefrontal cortex. *Neuroscience & Biobehavioral Reviews, 26*(3), 321-352.

Carolan, P. L., Jaspers-Fayer, F., Asmaro, D. T., Douglas, K. S., & Liotti, M. (2014). Electrophysiology of blunted emotional bias in psychopathic personality. *Psychophysiology, 51*(1), 36-41.

Carrion, V. G., Garrett, A., Menon, V., Weems, C. F., & Reiss, A. L. (2008). Posttraumatic stress symptoms and brain function during a response-inhibition task: An fMRI study in youth. *Depression and Anxiety, 25*(6), 514-526.

Cauffman, E., Kimonis, E. R., Dmitrieva, J., & Monahan, K. C. (2009). A multimethod assessment of juvenile psychopathy: Comparing the predictive utility of the PCL:YV, YPI, and NEO PRI. *Psychological Assessment, 21*(4), 528-542.

Cauffman, E., & Steinberg, L. (2000). (Im)maturity of judgment in adolescence: Why adolescents may be less culpable than adults. *Behavioral Sciences & The Law, 18*, 741-760.

Chaddock-Heyman, L., Erickson, K. I., Voss, M. W., Powers, J. P., Knecht, A. M., Pontifex, M. B., . . . Kramer, A. F. (2013). White matter microstructure is associated with cognitive control in children. *Biological Psychiatry, 94*(1), 109-115.

Chambers, C. D., Garavan, H., & Bellgrove, M. A. (2009). Insights into the neural basis of response inhibition from cognitive and clinical neuroscience. *Neuroscience & Biobehavioral Reviews, 33*, 631-646.

Chapman, R. M., & McCarry, J. W. (1995). EP component identification and measurement by principal component analysis. *Brain and Cognition, 27*, 288-310.

Cheng, Y., Hung, A. Y., & Decety, J. (2012). Dissociation between affective sharing and emotion understanding in juvenile psychopaths. *Development and Psychopathology, 24*(2), 623-636.

Chiang, M. C., Barysheva, M., Shattuck, D. W., Lee, A. D., Madsen, S. K., Avedissian, C., . . . Thompson, P. M. (2009). Genetics of brain fiber architecture and intellectual performance. *Journal of Neuroscience, 29*(7), 12-24.

Chiu, P. H., & Deldin, P. J. (2007). Neural evidence for enhanced error detection in major depressive disorder. *The American Journal of Psychiatry, 164*(4), 608-616.

Chuang, T.-C., Wu, M.-T., Huang, S.-P., Weng, M.-J., & Yang, P. (2013). Diffusion tensor imaging study of white matter fiber tracts in adolescent attention-deficit/hyperactivity disorder. *Psychiatry Research: Neuroimaging, 211*(2), 186-187.

Cleckley, H. (1941). *The Mask of Sanity*. St. Louis, Mosby.

Cohn, M. D., Veltman, D. J., Pape, L. E., van Lith, K., Vermeiren, R. R. J. M., van den Brink, W., . . . Popma, A. (2015). Incentive processing in persistent disruptive behavior and psychopathic traits: A functional magnetic resonance imaging study in adolescents. *Biological Psychiatry, 78*, 615-624.

Cohn, M. D., Viding, E., McCrory, E. J., Pape, L. E., van den Brink, W., Doreleijers, T. A. H., . . . Popma, A. (2016). Regional grey matter volume and concentration in at-risk adolescents: Untangling associations with callous-unemotional traits and conduct disorder symptoms. *Psychiatry Research: Neuroimaging, 254*, 180-187.

Colins, O., Vermeiren, R., Vahl, P., Markus, M., Broekaert, E., & Doreleijers, T. (2011). Psychiatric disorder in detained male adolescence as a risk factor for serious recidivism. *The Canadian Journal of Psychiatry, 56*(1), 44-49.

Cooke, D. J., & Michie, C. (2001). Refining the construct of psychopathy: Towards a hierarchical model. *Psychological Assessment, 13*(2), 171-188.

Cope, L. M., Ermer, E., Nyalakanti, P. K., Calhoun, V. D., & Kiehl, K. A. (2014). Paralimbic gray matter reductions in incarcerated adolescent females with psychopathic traits. *Journal of Abnormal Child Psychology, 42*, 659-668.

Cope, L. M., Shane, M. S., Segall, J. M., Nyalakanti, P. K., Stevens, M. C., Pearlson, G. D., . . . Kiehl, K. A. (2012). Examining the effect of psychopathic traits on gray matter volume in a community substance abuse sample. *Psychiatry Research, 204*(2-3), 91-100.

Corrado, R. R., Vincent, G. M., Hart, S. D., & Cohen, I. M. (2004). Predictive validity of the Psychopathy Checklist: Youth Version for general and violent recidivism. *Behavioral Sciences & The Law, 22*(1), 5-22.

Cottle, C. C., Lee, R. J., & Heilbrun, K. (2001). The prediction of criminal recidivism in juveniles: A meta-analysis. *Criminal Justice and Behavior, 28*(3), 367-394.

Craig, M. C., Catani, M., Deeley, Q., Latham, R., Daly, E., Kanaan, R., . . . Murphy, D. G. M. (2009). Altered connections on the road to psychopathy. *Molecular Psychiatry, 14*, 946-953.

Crespi, C., Cerami, C., Dodich, A., Canessa, N., Arpone, M., Iannaccone, S., . . . Cappa, S. F. (2014). Microstructural white matter correlates of emotion recognition impairment in Amyotrophic Lateral Sclerosis. *Cortex, 53*, 1-8.

Damasio, A. R. (1994). *Decartes' Error: Error, Reason, and the Human Brain*. Grosset/Putnam; New York.

Das, J. (2008). P*sychopathic traits in Dutch adolescent offender and community samples*. Wageningen, Netherlands: Posen & Looyen.

Davies, P. L., Segalowitz, S. J., & Gawin, W. J. (2004). Development of response monitoring in 7- to 25-year olds. *Developmental Neuropsychology, 25*(3), 355-376.

De Brito, S. A., Mechelli, A., Wilke, M., Laurens, K. R., Jones, A. P., Barker, G. J., . . . Viding, E. (2009). Size matters: Increased grey matter in boys with conduct problems and callous-unemotional traits. *Brain, 132*(Pt 4), 843-852.

De Los Reyes, A., & Kazdin, A. E. (2005). Informant discrepancies in the assessment of childhood psychopathology: A critical review, theoretical framework and recommendations for further study. *Psychological Bulletin, 131*, 483-509.

de Oliveira-Souza, R., Hare, R. D., Bramati, I. E., Garrido, G. J., Ignácio, F. A., Tovar-Moll, F., & Moll, J. (2008). Psychopathy as a disorder of the moral brain: Fronto-temporo-limbic grey matter reductions demonstrated by voxel-based morphometry. *NeuroImage, 40*(3), 1202-1213.

Deary, I. J., Bastin, M. E., Pattie, A., Clayden, J. D., Whalley, L. J., Starr, J. M., & Wardlaw, J. M. (2006). White matter integrity and cognition in childhood and old age. *Neurology, 66*(4), 505-512.

Dehaene, S., Posner, M. I., & Tucker, D. M. (1994). Localization of a neural system for error detection and compensation. *Psychological Science, 5*(5), 303-305.

Delorme, A., & Makeig, S. (2004). EEGlab: An open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *Journal of Neuroscience Methods, 134*(1), 9-21.

Dien, J., Khoe, W., & Mangun, G. R. (2007). Evaluation of PCA and ICA of simulated ERPs: Promax vs. Infomax rotations. *Human Brain Mapping, 28*(8), 742-763.

Douglas, K. S., Epstein, M. E., & Poythress, N. G. (2008). Criminal recidivism among juvenile offenders: Testing the incremental and predictive validity of three measures of psychopathic features. *Law and Human Behavior, 32*, 423-438.

Edens, J. F., Campbell, J. S., & Weir, J. M. (2007). Youth psychopathy and criminal recidivism: A meta-analysis of the psychopathy checklist measures. *Law and Human Behavior, 31*(1), 53-75.

Edwards, B. G., Calhoun, V. D., & Kiehl, K. A. (2012). Joint ICA of ERP and fMRI during error-monitoring. *NeuroImage, 59*(2), 1896-1903.

Egner, T. (2009). Prefrontal cortex and cognitive control: Motivating functional hierarchies. *Nature Neuroscience, 12*, 821-822.

Erhardt, E. B., Rachakonda, S., Bedrick, E. J., Allen, E. A., Adali, T., & Calhoun, V. D. (2011). Comparison of multi-subject ICA methods for analysis of fMRI data. *Human Brain Mapping, 32*, 2075-2095.

Ermer, E., Cope, L. M., Nyalakanti, P., Calhoun, V. D., & Kiehl, K. A. (2012). Aberrant paralimbic gray matter in criminal psychopathy. *Journal of Abnormal Psychology, 121*(3), 649-658.

Ermer, E., Cope, L. M., Nyalakanti, P., Calhoun, V. D., & Kiehl, K. A. (2013). Aberrant paralimbic gray matter in incarcerated male adolescents with psychopathic traits. *Journal of the American Academy of Child and Adolescent Psychiatry, 52*(1), 94-103.

Eysenck, H. J., & Eysenck, S. B. G. (1976). *Psychoticism as a dimension of personality*. Hodder and Stoughton, London.

Fairchild, G., Passamonti, L., Hurford, G., Hagan, C. C., von dem Hagen, E. A. H., van Goozen, S. H. M., . . . Calder, A. J. (2011). Brain structure abnormalities in early-

onset and adolescent-onset conduct disorder. *American Journal of Psychiatry, 168*, 624-633.

Falkenstein, M., Hohnsbein, J., Hoormann, J., & Blanke, L. (1991). Effects of crossmodal divided attention on late ERP components: II: Error processing in choice reaction tasks. *Electroencephalography and Clinical Neurophysiology, 78*(6), 447-455.

Farr, O. M., Hu, S., Zhang, S., & Li, C. S. (2012). Decreased saliency processing as a neural measure of Barratt impulsivity in healthy adults. *NeuroImage, 63*, 1070-1077.

Farrington, D. P. (1986). *"Age and Crime" in Crime and Justice: An Annual Review of Research, vol. 7*, Chicago, IL.

Farrington, D. P. (2003). *Key results from the first forty years of the Cambridge Study in Delinquent Development*. in Taking Stock of Delinquency: An Overview of Findings from Contemporary Longitudinal Studies, Longitudinal Research in the Social and Behavioral Sciences: An Interdisciplinary Series, New York: Kluwer-Plenum, pp. 137-183.

Farrington, D. P., Loeber, R., & Van Kammen, W. B. (1990). *Long-term criminal outcomes of hyperactivity-impulsivity-attention deficit and conduct problems in childhood*. In Straight and Devious Pathways from Childhood to Adulthood (pp. 62- 81). Cambridge, England: Cambridge University Press.

Finger, E. C., Marsh, A. A., Blair, K. S., Reid, M. E., Sims, C., Ng, P., . . . Blair, R. J. (2011). Disrupted reinforcement signaling in the orbitofrontal cortex and caudate in youths with conduct disorder or oppositional defiant disorder and a high level of psychopathic traits. *American Journal of Psychiatry, 168*(2), 152-162.

Finger, E. C., Marsh, A. A., Mitchell, D. G., Reid, M. E., Sims, C., Budhani, S., . . . Blair, J. R. (2008). Abnormal ventromedial prefrontal cortex function in children with psychopathic traits during reversal learning. *Archives in General Psychiatry, 65*(5), 586-594.

Fink, B. C., Steele, V. R., Maurer, M. J., Fede, S. J., Calhoun, V. D., & Kiehl, K. A. (2016). Brain potentials predict substance abuse treatment completion in a prison sample. *Brain and Behavior, 6*(8), e00501.

Fink, B. C., Tant, A. S., Tremba, K., & Kiehl, K. A. (2012). Assessment of psychopathic traits in an incarcerated adolescent sample: A methodological comparison. *Journal of Abnormal Child Psychology, 40*(6), 971-986.

Fitzgerald, K. D., Welsh, R. C., Gehring, W. J., Abelson, J. L., Himle, J. A., Liberzon, I., & Taylor, S. F. (2005). Error-related hyperactivity of the anterior cingulate cortex in obsessive-compulsive disorder. *Biological Psychiatry, 57*(3), 287-294.

Flechsig, P. (1901). Developmental (myelogenetic) localisation of the cerebral cortex in the human. *Lancet, 158*, 1027-1030.

Foley, R. M. (2001). Academic characteristics of incarcerated youth and correctional educational programs: A literature review. *Journal of Emotional and Behavioral Disorders, 9*(4), 248-259.

Folstein, J. R., & Van Petten, C. (2008). Influence of cognitive control and mismatch on the N2 component of the ERP: A review. *Psychophysiology, 45*, 152-170.

Ford, J. A. (2005). Substance use, the social bond and delinquency. *Sociological Inquiry, 75*, 109-127.

Forth, A. E., Hart, S. D., & Hare, R. D. (1990). Assessment of psychopathy in male young offenders. *Psychological Assessment, 2*(3), 342-344.

Forth, A. E., Kosson, D. S., & Hare, R. D. (2003). *The Psychopathy Checklist: Youth Version*. Toronto, ON, Canada: Multi-Health Systems.

Freeman, S. M., Clewett, D. V., Bennett, C. M., Kiehl, K. A., Gazzaniga, M. S., & Miller, M. B. (2015). The posteromedial region of the default mode network shows attenuated task-induced deactivation in pychopathic prisoners. *Neuropsychology, 29*(3), 493-500.

Freire, L., & Mangin, J. F. (2001). Motion correction algorithms may create spurious brain activations in the absence of subject motion. *NeuroImage, 14*, 709-722.

Freire, L., Roche, A., & Mangin, J. F. (2002). What is the best similarity measure for motion correction in fMRI time series? *IEEE Transactions on Medical Imaging, 21*, 470-484.

Frick, P. J. (2009). Extending the construct of psychopathy to youth: Implications for understanding, diagnosing, and treatment antisocial children and adolescents. *Canadian Journal of Psychiatry, 54*(12), 803-812.

Frick, P. J. (2012). Developmental pathways to conduct disorder: Implications for future directions in research, assessment, and treatment. *Journal of Clinical Child and Adolescent Psychology, 41*(3), 378-389.

Frick, P. J., Bodin, S. D., & Barry, C. T. (2000). Psychopathic traits and conduct problems in community and clinic-referred samples of children: Further development of the Psychopathy Screening Device. *Psychological Assessment, 12*, 382-393.

Frick, P. J., Cornell, A. H., Bodin, S. D., Dane, H. E., Barry, C. T., & Loney, B. R. (2003). Callous-unemotional traits and developmental pathways to severe conduct problems. *Developmental Psychology, 39*(2), 246-260.

Frick, P. J., & Hare, R. D. (2001). *The Antisocial Process Screening Device (APSD).* Toronto, Ontario, Canada: Multi Health Systems.

Frick, P. J., & Nigg, J. T. (2011). Current issues in the diagnosis of Attention Deficit Hyperactivity Disorder, Oppositional Defiant Disorder, and Conduct Disorder. *Clinical Psychology, 8*, 77-107.

Friston, K. (1994). Functional and effective connectivity in neuroimaging: A synthesis. *Human Brain Mapping, 2*, 56-78.

Fu, L.-P., Bi, G.-H., Zou, Z.-T., Wang, Y., Ye, E.-M., Ma, L., . . . Yang, Z. (2008). Impaired response inhibition function in abstinent heroin dependents: An fMRI study. *Neuroscience Letters, 438*(3), 322-326.

Fusar-Poli, P., Placentino, A., Carletti, F., Allen, P., Landi, P., Abbamonte, M., . . . Politi, P. L. (2009). Laterality effect on emotional faces processing: ALE meta-analysis of evidence. *Neuroscience Letters, 452*(3), 262-267.

Galléa, C., Bernhardina de Graaf, J., Pailhous, J., & Bonnard, M. (2008). Error processing during online motor control depends on the response accuracy. *Behavioural Brain Research, 193*(1), 117-125.

Ganzer, V. J., & Sarason, I. G. (1973). Variables associated with recidivism among juvenile delinquents. *Journal of Consulting and Clinical Psychology, 40*(1), 1-5.

Gao, W., Lin, W., Chen, Y., Gerig, G., Smith, J. K., Jewells, V., & Gimore, J. H. (2009). Temporal and spatial development of axonal maturation and myelination of white matter in the developing brain. *American Journal of Radiology, 30*(2), 290-296.

Garavan, H., Ross, T. J., Kaufman, J., & Stein, E. A. (2003). A midline dissociation between error-processing and response-conflict monitoring. *NeuroImage, 20*, 1132-1139.

Garavan, H., Ross, T. J., Murphy, K., Roche, R. A. P., & Stein, E. A. (2002). Dissociable executive functions in the dynamic control of behavior: Inhibition, error detection, and correction. *NeuroImage, 17*, 1820-1829.

Gehring, W. J., Coles, M. G. H., Meyer, D. E., & Donchin, E. (1990). The error-related negativity: An event-related brain potential accompanying errors. *Psychophysiology, 27*, S34.

Genova, H. M., Rajagopalan, V., Chiaravalloti, N., Binder, A., Deluca, J., & Lengenfelder, J. (2015). Facial affect recognition linked to damage in specific white matter tracts in traumatic brain injury. *Social Neuroscience, 10*, 27-34.

Giedd, J. N., Blumenthal, J., Jeffries, N. O., Castellanos, F. X., Liu, H., Zijdenbos, A., & Rapoport, J. L. (1999). Brain development during childhood and adolescence: A longitudinal MRI study. *Nature Neuroscience, 2*(10), 861-863.

Gordon, J. A., Diehl, R. L., & Anderson, L. (2012). Does ADHD matter? Examining attention deficit and hyperactivity disorder on the likelihood of recidivism among detained youth. *Journal of Offender Rehabilitation, 51*(8), 497-518.

Gottfredson, M. R., & Hirschi, T. (1990). *A general theory of crime*. Stanford, CA: Stanford University Press.

Gregory, S., Ffytche, D., Simmons, A., Kumari, V., Howard, M., Hodgins, S., & Blackwood, N. (2012). The antisocial brain: Psychopathy matters. *Archives in General Psychiatry, 69*(9), 962-972.

Grunwald, H. E., Lockwood, B., Harris, P. W., & Mennis, J. (2010). Influences of neighborhood context, individual history and parenting behavior on recidivism among juvenile offenders. *Journal of Youth and Adolescence, 39*(9), 1067-1079.

Ham, T., Leff, A., de Boissezon, X., Joffe, A., & Sharp, D. J. (2013). Cognitive control and the salience network: An investigation of error processing and effective connectivity. *Journal of Neuroscience, 33*(16), 7091-7098.

Hamilton, L. S., Levitt, J. G., O'Neill, J., Alger, J. R., Luders, E., Phillips, O. R., . . . Narr, K. L. (2008). Reduced white matter integrity in attention-deficit hyperactivity disorder. *NeuroReport, 19*(17), 1705-1708.

Hamilton, R. K., Hiatt Racer, K., & Newman, J. P. (2015). Impaired integration in psychopathy: A unified theory of psychopathic dysfunction. *Psychological Review, 122*(4), 770-791.

Hare, R. D. (1978). Psychopathy and electrodermal responses to nonsignal stimulation. *Biological Psychology, 6*(4), 237-246.

Hare, R. D. (1980). A research scale for the assessment of psychopathy in criminal populations. *Personality and Individual Differences, 1*(2), 111-119.

Hare, R. D. (1991). *The Hare Psychopathy Checklist - Revised Manual*. Multi-Health Systems: Toronto.

Hare, R. D. (1996). Psychopathy: A clinical construct whose time has come. *Criminal Justice and Behavior, 23*(1), 25-54.

Hare, R. D. (2003). *Manual for the Hare Psychopathy Checklist - Revised (2nd Ed)*. Toronto, Canada: Multi-Health Systems.

Hare, R. D., & Neumann, C. S. (2006). *The PCL-R assessment of psychopathy: Development, structural properties, and new directions*. In C. J. Patrick (Ed.), Handbook of Psychopathy (pp. 58-88). New York: Guilford Press.

Hare, R. D., & Neumann, C. S. (2010). The role of antisociality in the psychopathy construct: Comment on Skeem and Cooke (2010). *Psychological Assessment, 22*(2), 446-454.

Harenski, C. L., Harenski, K. A., & Kiehl, K. A. (2014). Neural processing of moral violations among incarcerated adolescents with psychopathic traits. *Developmental Cognitive Neuroscience, 10*, 181-189.

Harpur, T. J., Hare, R. D., & Hakstian, A. R. (1989). Two-factor conceptualization of psychopathy: Construct validity and assessment implications. *Psychological Assessment, 1*(1), 6-17.

Hart, S. D., & Hare, R. D. (1989). Discriminant validity of the Psychopathy Checklist in a forensic psychiatric population. *Psychological Assessment, 1*(3), 211-219.

Hawelleck, D. J., Hipp, J. F., Lewis, C. M., Corbetta, M., & Engel, A. K. (2011). Increased functional connectivity indicates the severity of cognitive impairment in multiple sclerosis. *Proceedings of the National Academy of Sciences, 108*(47), 19066-19071.

Hawkins, J. D., Herrenkohl, T. I., Farrington, D. P., Brewer, D., Catalano, R. F., Harachi, T. W., & Cothern, L. (2000). *Predictors of youth violence*. In Juvenile Justice Bulletin. Washington, DC: Office of Juvenile Justice and Delinquency Prevention, US Department of Justice.

Haynes, J. P., & Bensch, M. (1981). The P > V sign on the WISC-R and recidivism in delinquents. *Journal of Consulting and Clinical Psychology, 49*(3), 480-481.

Hemphälä, M., Kosson, D. S., Westerman, J., & Hodgins, S. (2015). Stability and predictors of psychopathic traits from mid-adolescence through early adulthood. *Scandinavian Journal of Psychology, 56*(6), 649-658.

Hemphill, J. F., Hare, R. D., & Wong, S. (1998). Psychopathy and recidivism: A review. *Legal and Criminological Psychology, 3*(1), 139-170.

Hermann, M. J., Rommler, J., Ehlis, A. C., Heidrich, A., & Fallgatter, A. J. (2004). Source localization (LORETA) of the error-related negativity (ERN/Ne) and positivity (Pe). *Cognitive Brain Research, 20*(2), 294-299.

Herpertz, S. C., Huebner, T., Marx, I., Vloet, T. D., Fink, G. R., Stoecker, T., . . . Herpertz-Dahlmann, B. (2008). Emotional processing in male adolescents with child-onset conduct disorder. *The Journal of Child Psychology and Psychiatry, 49*(7), 781-791.

Hester, R., Fassbender, C., & Garavan, H. (2004). Individual differences in error processing: A review and reanalysis of three event-related fMRI studies using the go/nogo task. *Cerebral Cortex, 14*(9), 986-994.

Hester, R., Foxe, J. J., Molholm, S., Shpaner, M., & Garavan, H. (2005). Neural mechanisms involved in error processing: A comparison of errors made with and without awareness. *NeuroImage, 27*, 602-608.

Hester, R., Nestor, L., & Garavan, H. (2009). Impaired error awareness and anterior cingulate cortex hypoactivity in chronic cannabis users. *Neuropsychopharmacology, 34*, 2450-2458.

Himelstein, S., Hastings, A., Shaprio, S., & Heery, M. (2012). Mindfulness training for self-regulation and stress with incarcerated youth: A pilot study. *Probation Journal, 59*(2), 151-165.

Hoeve, M., McReynolds, L. S., Wasserman, G. A., & McMillan, C. (2013). The influence of mental health disorders on severity of reoffending in juveniles. *Criminal Justice and Behavior, 30*(3), 289-301.

Hoge, R. D. (1999). An expanded role for psychological assessments in juvenile justice systems. *Criminal Justice and Behavior, 26*, 251-266.

Holroyd, C. B., & Coles, M. G. H. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review, 109*(4), 679-709.

Hoppenbrouwers, S. S., Bulten, B. H., & Brazil, I. A. (2016). Parsing fear: A reassessment of the evidence for fear deficits in psychopathy. *Psychological Bulletin, 142*(6), 573-600.

Hoppenbrouwers, S. S., Nazeri, A., de Jesus, D. R., Stirpe, T., Felsky, D., Schutter, D. J. L. G., . . . Voineskos, A. N. (2013). White matter deficits in psychopathic offenders and correlation with factor structure. *PLoS ONE, 8*(8), e72375.

Horn, N. H., Dolan, M., Elliott, R., Deakin, J. F. W., & Woodruff, P. W. R. (2003). Response inhibition and impulsivity: An fMRI study. *Neuropsychologia, 41*(14), 1959-1966.

Hornak, J., Bramham, J., Rolls, E. T., Morris, R. G., O'Doherty, J., Bullock, P. R., & Polkey, C. E. (2003). Changes in emotion after circumscribed surgical lesions of the orbitofrontal and cingulate cortices. *Brain, 126*, 1691-1712.

Iraji, A., Benson, R. R., Welch, R. D., O'Neill, B. J., Woodard, J. L., Imran Ayaz, S., . . . Kou, Z. (2015). Resting state functional connectivity in mild traumatic brain injury at the acute stage: Independent Component and seed-based analyses. *Journal of Neurotrauma, 32*(14), 1031–1045.

Jasper, H. H. (1958). The ten-twenty electrode system of the International Federation. *Electroencephalography and Clinical Neurophysiology, 10*, 371-375.

Jolliffe, D., Farrington, D. P., Hawkins, J. D., Catalano, R. F., Hill, K. G., & Kosterman, R. (2003). Predictive, concurrent, prospective and retrospective validity of self-reported delinquency. *Criminal Behaviour and Mental Health, 13*, 179-197.

Jones, A. P., Laurens, K. R., Herba, C. M., Barker, G. J., & Viding, E. (2009). Amygdala hypoactivity to fearful faces in boys with conduct problems and callous-unemotional traits. *American Journal of Psychiatry, 166*, 95-102.

Juárez, M., Kiehl, K. A., & Calhoun, V. D. (2013). Intrinsic limbic and paralimbic networks are associated with criminal psychopathy. *Human Brain Mapping, 34*(8), 1921-1930.

Jung, T. P., Makeig, S., Westerfield, M., Courschesne, E., & Sejnowski, T. J. (2000). Removal of eye activity artifacts from visual event-related potentials in normal and clinical subjects. *Clinical Neurophysiology, 111*(10), 1745-1758.

Justice Policy Institute. (2015). *Sticker shock: Calculating the full price tag for youth incarceration*. Available at: http://www.justicepolicy.org/uploads/justicepolicy/documents/sticker_shock_final_v2.pdf

Kamphaus, R. W., & Frick, P. J. (2002). *Clinical assessment of child and adolescent personality and behavior (2nd ed.)*. Boston: Allyn and Bacon.

Kanaan, R. A., Allin, M., Picchioni, M., Barker, G. J., Daly, E., Shergill, S. S., . . . McGuire, P. K. (2012). Gender differences in white matter microstructure. *PLoS ONE, 7*(6), e38272.

Kaufman, J., Birmaher, B., & Brent, D. (1997). Schedule for Affective Disorders and Schizophrenia for School-Aged Children: Present and Lifetime Version (K-SADS-PL): Initial reliability and validity data. *Journal of the American Academy of Child and Adolescent Psychiatry, 37*(7), 980-988.

Kennerley, S. W., Walton, M. E., Behrens, T. E. J., Buckley, M. J., & Rushworth, M. F. S. (2006). Optimal decision making and the anterior cingulate cortex. *Nature Neuroscience, 9*, 940-947.

Kiehl, K. A. (2006). A cognitive neuroscience perspective on psychopathy: Evidence for paralimbic system dysfunction. *Psychiatry Research, 142*(2-3), 107-128.

Kiehl, K. A., Anderson, N. E., Aharoni, E., Maurer, J. M., Harenski, K. A., Rao, V., . . . Steele, V. R. (2018). Age of gray matters: Neuroprediction of recidivism. *NeuroImage: Clinical, 19*, 813-823.

Kiehl, K. A., Hare, R. D., McDonald, J. J., & Brink, J. (1999). Semantic and affective processing in psychopaths: An event-related potential study. *Psychophysiology, 36*, 765-774.

Kiehl, K. A., & Hoffman, M. B. (2011). The criminal psychopath: History, neuroscience, treatment, and economics. *Jurimetrics, 51*, 355-397.

Kiehl, K. A., Liddle, P. F., & Hopfinger, J. B. (2000). Error processing and the rostral anterior cingulate: An event-related fMRI study. *Psychophysiology, 37*(2), 216-223.

Kiehl, K. A., Liddle, P. F., Smith, A. M., Mendrek, A., Forster, B. B., & Hare, R. D. (1999). Neural pathways involved in the processing of concrete and abstract words. *Human Brain Mapping, 7*, 225-233.

Kiehl, K. A., Smith, A. M., Hare, R. D., & Liddle, P. F. (2000). An event-related potential investigation of response inhibition in schizophrenia and psychopathy. *Biological Psychiatry, 483*, 210-221.

Kiehl, K. A., Smith, A. M., Mendrek, A., Forster, B. B., Hare, R. D., & Liddle, P. F. (2004). Temporal lobe abnormalities in semantic processing by criminal psychopaths as revealed by functional magnetic resonance imaging. *Psychiatry Research: Neuroimaging, 130*, 27-42.

King, N. S., Crawford, S., Wenden, F. J., Moss, N. E. G., & Wade, D. T. (1995). The Rivermead Post Concussion Symptoms Questionnaire: A measure of symptoms commonly experienced after head injury and its reliability. *Journal of Neurology, 242*(9), 587-592.

Klein, T. A., Endrass, T., Kathmann, N., Neumann, J., Von Cramon, D. Y., & Ullsperger, M. (2007). Neural correlates of error awareness. *NeuroImage, 34*, 1774-1781.

Klingberg, T., Vaidya, C. J., Gabrieli, J. D., Moseley, M. E., & Hedehus, M. (1999). Myelination and organization of the frontal white matter in children: A Diffusion Tensor MRI study. *NeuroReport, 10*, 2817-2821.

Koch, J. L. A. (1889). *Kurzgefaßter Leitfaden der Psychiatrie. Mit Rücksichtnahme auf die Bedürfnisse der Studirenden, der practischen Aerzte und der Gerichtsärzte*. 2nd Edition, Ravensburg: Dom.

Koch, W., Teipel, S., Mueller, S., Buerger, K., Bokde, A. L., Hampel, H., . . . Meindl, T. (2010). Effects of aging on default mode network activity in resting state fMRI: Does the method of analysis matter? *NeuroImage, 51*(1), 280-287.

Kochunov, P., Williamson, D. E., Lancaster, J., Fox, P., Cornell, J., Blangero, J., & Glahn, D. C. (2012). Fractional anisotropy of water diffusion in cerebral white matter across the lifespan. *Neurobiology of Aging, 33*(1), 9-20.

Koenigs, M., Kruepke, M., Zeier, J., & Newman, J. P. (2011). Utilitarian moral judgment in psychopathy. *Social Cognitive and Affective Neuroscience, 7*(6), 708-714.

Kosson, D. S., Suchy, Y., Mayer, A. R., & Libby, J. (2002). Facial affect recognition in criminal psychopaths. *Emotion, 24*, 398-411.

Kotler, J. S., & McMahon, R. J. (2005). Child psychopathy: Theories, measurement, and relations with the development and persistence of conduct problems. *Clinical Child and Family Psychology Review, 8*, 291-325.

Ladouceur, C. D., Dahl, R. E., & Carter, C. S. (2007). Development of action monitoring through adolescence into adulthood: ERP and source localization. *Developmental Science, 10*(6), 874-891.

Larson, C. L., Baskin-Sommers, A. R., Stout, D. M., Balderston, N. L., Curtin, J. J., Schultz, D. H., . . . Newman, J. P. (2013). The interplay of attention and emotion: Top-down attention modulates amygdala activation in psychopathy. *Cognitive, Affective, & Behavioral Neuroscience, 13*(4), 757-770.

Larson, M. J., South, M., & Clayson, P. E. (2011). Sex differences in error-related performance monitoring. *NeuroReport, 22*(44-48), 44.

Larson, M. J., Steffen, P. R., & Primosch, M. (2013). The impact of a brief mindfulness meditation intervention on cognitive control and error-related performance monitoring. *Frontiers in Human Neuroscience, 7*(308), 1-12.

Le Bihan, D., Turner, R., Douek, P., & Patronas, N. (1992). Diffuson MR imaging: Clinical applications. *American Journal of Roentgenology, 159*(3), 591-599.

Lebel, C., Walker, L., Leemans, A., Phillips, L., & Beaulieu, C. (2008). Microstructural maturation of the human brain from childhood to adulthood. *NeuroImage, 40*(3), 1044-1055.

Lee, Z., Klaver, J. R., Hart, S. D., Moretti, M. M., & Douglas, K. S. (2009). Short-term stability of psychopathic traits in adolescent offenders. *Journal of Clinical Child and Adolescent Psychology, 38*(5), 595-605.

Lee, Z., Vincent, G. M., Hart, S. D., & Corrado, R. R. (2003). The validity of the Antisocial Process Screening Device as a self-report measure of psychopathy in adolescent offenders. *Behavioral Sciences & The Law, 21*(6), 771-786.

Leverso, J., Bielby, W., & Hoelter, L. F. (2015). Back on the streets: Maturation and risk factors for recidivism among serious juvenile offenders. *Journal of Adolescence, 41*, 67-75.

Liddle, P. F., Kiehl, K. A., & Smith, A. M. (2000). Event-related fMRI study of response inhibition. *Human Brain Mapping, 12*(2), 100-109.

Lindner, P., Budhiraja, M., Westerman, J., Savic, I., Jokinen, J., Tiihonen, J., & Hodgins, S. (2017). White matter correlates of psychopathic traits in a female community sample. *Social Cognitive and Affective Neuroscience*, 1500-1510.

Lockwood, P. L., Sebastian, C. L., McCrory, E. J., Hyde, Z. H., Gu, X., De Brito, S. A., & Viding, E. (2013). Association of callous traits with reduced neural response to others' pain in children with conduct problems. *Current Biology, 23*(10), 901-905.

Loeber, R. (1990). Development and risk factors of juvenile antisocial behavior and delinquency. *Clinical Psychology Review, 10*(1), 1-41.

Loeber, R., & Farrington, D. P. (1998). *Serious and violent juvenile offenders: Risk factors and successful interventions*. Thousand Oaks, CA: Sage.

Loeber, R., & Farrington, D. P. (2011). *Young homicide offenders and victims: Risk factors, prediction, and prevention from childhood*. New York: Springer.

Loeber, R., Farrington, D. P., Stouthamer-Loeber, M., & Raskin White, H. (2008). *Violence and serious theft: Development and prediction from childhood to adulthood*. New York: Taylor & Francis.

Lombroso, C. (1911). *Crime: It's causes and remedies*. London: William-Heinemann.

Loney, B. R., Frick, P. J., Clements, C. B., Ellis, M. L., & Kerlin, K. (2003). Callous-unemotional traits, impulsivity, and emotional processing in adolescents with antisocial behavior problems. *Journal of Clinical Child and Adolescent Psychology, 32*, 66-80.

López-Caneda, E., Cadaveira, F., Crego, A., Gómez-Suárez, A., Corral, M., Parada, M., . . . Rodríguez Holguín, S. (2012). Hyperactivation of right inferior frontal

cortex in young binge drinkers during response inhibition: A follow-up study. *Addiction, 107*(10), 1796-1808.

Lozier, L. M., Cardinale, E. M., VanMeter, J. W., & Marsh, A. A. (2014). Mediation of the relationship between callous-unemotional traits and proactive aggression by amygdala response to fear among children with conduct problems. *JAMA Psychiatry, 71*(6), 627-636.

Luders, E., Clark, K., Narr, K. L., & Toga, A. W. (2011). Enhanced brain connectivity in long-term meditation practitioners. *NeuroImage, 57*(4), 1308-1316.

Ly, M., Motzkin, J. C., Philippi, C. L., Kirk, G. R., Newman, J. P., Kiehl, K. A., & Koenigs, M. (2012). Cortical thinning in psychopathy. *American Journal of Psychiatry, 169*(7), 743-749.

Lykken, D. T. (1995). *The antisocial personalities*. Hillsdale, NJ: Erlbaum.

Lynam, D. R. (1997). Pursuing the psychopath: Capturing the fledgling psychopath in a nomological net. *Journal of Abnormal Psychology, 106*, 425-438.

Lynam, D. R., Caspi, A., Moffitt, T. E., Loeber, R., & Stouthamer-Loeber, M. (2007). Longitudinal evidence that psychopathy scores in early adolescence predict adult psychopathy. *Journal of Abnormal Psychology, 116*, 155-165.

Maddock, R. J., & Buonocore, M. H. (1997). Activation of left posterior cingulate gyrus by the auditory presentation of threat-related words: An fMRI study. *Psychiatry Research: Neuroimaging, 75*, 1-14.

Magno, E., Foxe, J. J., Molholm, S., Robertson, I. H., & Garavan, H. (2006). The anterior cingulate and error avoidance. *The Journal of Neuroscience, 26*(18), 4769-4773.

Mailloux, D. L., Forth, A. E., & Kroner, D. G. (1997). Psychopathy and substance use in adolescent male offenders. *Psychological Reports, 81*(2), 529-530.

Mallett, C. A., Fukushima, M., Stoddard Dare, P., & Quinn, L. (2012). Factors related to recidivism for youthful offenders. *Criminal Justice Studies, 26*(1), 84-98.

Marczyk, G. R., Heilbrun, K., Lander, T., & Dematteo, D. (2003). Predicting juvenile recidivism with the PCL:YV, MAYSI, and YLS/CMI. *International Journal of Forensic Mental Health, 2*(1), 7-18.

Marsh, A. A., Finger, E. C., Fowler, K. A., Adalio, C. J., Jurkowitz, I. T., Schechter, J. C., . . . Blair, R. J. (2013). Empathic responsiveness in amygdala and anterior cingulate cortex in youths with psychopathic traits. *Journal of Child Psychology and Psychiatry, 54*(8), 900-910.

Marsh, A. A., Finger, E. C., Fowler, K. A., Jurkowitz, I. T. N., Schechter, J. C., Yu, H. H., . . . Blair, R. J. R. (2011). Reduced amygdala-orbitofrontal connectivity during moral judgments in youths with disruptive behavior disorders and psychopathic traits. *Psychiatry Research: Neuroimaging, 194*, 279-286.

Marsh, A. A., Finger, E. C., Mitchell, D. G. V., Reid, M. E., Sims, C., Kosson, D. S., . . . Blair, R. J. R. (2008). Reduced amygdala response to fearful expressions in children and adolescents with callous-unemotional traits and disruptive behavior disorders. *American Journal of Psychiatry, 165*, 712-720.

Mathalon, D. H., Whitfield, S. L., & Ford, J. M. (2003). Anatomy of an error: ERP and fMRI. *Biological Psychology, 64*(1-2), 119-141.

Matta, A. D., Gonçalves, F. L., & Bizarro, L. (2012). Delay discounting: Concepts and measures. *Psychology & Neuroscience, 5*(2), 135-146.

Maurer, J. M., Steele, V. R., Cope, L. M., Vincent, G. M., Stephen, J. M., Calhoun, V. D., & Kiehl, K. A. (2016). Dysfunctional error-related processing in incarcerated youth with elevated psychopathic traits. *Developmental Cognitive Neuroscience, 19*, 70-77.

Maurer, J. M., Steele, V. R., Edwards, B. G., Bernat, E. M., Calhoun, V. D., & Kiehl, K. A. (2016). Dysfunctional error-related processing in female psychopathy. *Social Cognitive and Affective Neuroscience, 11*(7), 1059-1068.

Maurer, J. M., Steele, V. R., Fink, B. C., Vincent, G. M., Calhoun, V. D., & Kiehl, K. A. (2018). Investigating error-related processing in incarcerated adolescents with self-report psychopathy measures. *Biological Psychology, 132*, 96-105.

Maurer, J. M., Steele, V. R., Vincent, G. M., Rao, V., Calhoun, V. D., & Kiehl, K. A. (in press). Adolescent psychopathic traits negatively relate to hemodynamic activity within the basal ganglia during error-related processing. *Journal of Abnormal Child Psychology*.

McDermott, P. A., Alterman, A. I., Cacciola, J. S., Rutherford, M. J., Newman, J. P., & Mullholland, E. M. (2000). Generality of Psychopathy Checklist - Revised factors over prisoners and substance-dependent patients. *Journal of Consulting and Clinical Psychology, 68*(1), 181-186.

Menard, S. (1995). *Applied logistic regression analysis: Sage University Series on quantitative applications in the social sciences*. Thousand Oaks, CA: Sage.

Menon, V., Adleman, N. E., White, C. D., Glover, G. H., & Reiss, A. L. (2001). Error-related brain activation during a Go/NoGo response inhibition task. *Human Brain Mapping, 12*, 131-143.

Menzler, K., Belke, M., Wehrmann, E., Krakow, K., Lengler, U., Jansen, A., . . . Knake, S. (2011). Men and women are different: Diffusion tensor imaging reveals sexual dimorphism in the microstructure of the thalamus, corpus callosum and cingulum. *NeuroImage, 54*(4), 2557-2562.

Mesulam, M. M. (2000). *Principles of Behavioral and Cognitive Neurology (2nd Ed.).* Oxford University Press, New York.

Mettenburg, J. M., Benzinger, T. L., Shimony, J. S., Snyder, A. Z., & Sheline, Y. I. (2012). Diminished performance on neuropsychological testing in late life depression is correlated with microstructural white matter abnormalities. *NeuroImage, 60*(4), 2182-2190.

Michalska, K. J., Zeffiro, T. A., & Decety, J. (2016). Brain response to viewing others being harmed in children with conduct disorder symptoms. *Journal of Child Psychology and Psychiatry, 57*(4), 510-519.

Miner, M. H. (2002). Factors associated with recidivism in juveniles: An analysis of serious juvenile sex offenders. *Journal of Research in Crime and Delinquency, 39*(4), 421-436.

Minor, K. I., Wells, J. B., & Angel, E. (2008). Recidivism among juvenile offenders following release from residential placements: Multivariate predictors and gender differences. *Journal of Offender Rehabilitation, 46*(3-4), 171-188.

Mobbs, D., Eckert, M. A., Mills, D., Korenberg, J., Bellugi, U., Galaburda, A. M., & Reiss, A. L. (2007). Frontostriatal dysfunction during response inhibition in Williams Syndrome. *Biological Psychiatry, 63*(2), 256-261.

Moffitt, T. E. (1990). Juvenile delinquency and attention deficit disorder: Boys' developmental trajectories from age 3 to age 15. *Child Development, 61*(3), 893-910.

Moffitt, T. E. (1993). 'Life-course persistent' and 'adolescent-limited' antisocial behavior: A developmental taxonomy. *Psychological Review, 100*, 674-701.

Morcom, A. M., & Fletcher, P. C. (2007). Does the brain have a baseline? Why we should be resisting a rest. *NeuroImage, 37*(4), 1073-1082.

Mori, S., Wakana, S., Nagae-Poetscher, L., & van Zijl, P. C. M. (2005). *MRI Atlas of Human White Matter*. Elsevier, Amsterdam, The Netherlands.

Mori, S., & Zhang, J. (2006). Principles of diffusion tensor imaging and its applications to basic neuroscience research. *Neuron, 51*, 527-539.

Mostofsky, S. H., Schafer, J. G., Abrams, M. T., Goldberg, M. C., Flower, A. A., Boyce, A., . . . Pekar, J. J. (2003). fMRI evidence that the neural basis of response inhibition is task-dependent. *Brain Research. Cognitive Brain Research, 17*(2), 419-430.

Motzkin, J. C., Newman, J. P., Kiehl, K. A., & Koenigs, M. (2011). Reduced prefrontal connectivity in psychopathy. *The Journal of Neuroscience, 31*(48), 17348-17357.

Muller, J. L., Sommer, M., Wagner, V., Lange, K., Taschler, H., Roder, C. H., . . . Hajcak, G. (2003). Abnormalities in emotion processing within cortical and subcortical regions in criminal psychopaths: Evidence from a functional magnetic resonance imaging study using pictures with emotional content. *Biological Psychiatry, 542*, 152-162.

Munro, G. E., Dywan, J., Harris, G. T., McKee, S., Unsal, A., & Segalowitz, S. J. (2007). Response inhibition in psychopathy: The frontal N2 and P3. *Neuroscience Letters, 418*(2), 149-153.

Myner, J., Santman, J., & Cappelletty, G. G. P., B. F. (1998). Variables related to recidivism among juvenile offenders. *International Journal of Offender Therapy and Comparative Criminology, 42*(1), 65-80.

Neumann, C. S., & Hare, R. D. (2008). Psychopathic traits in a large community sample: Links to violence, alcohol use, and intelligence. *Journal of Consulting and Clinical Psychology, 76*(5), 893-899.

Neumann, C. S., Kosson, D. S., Forth, A. E., & Hare, R. D. (2006). Factor structure of the Hare Psychopathy Checklist: Youth Version (PCL:YV) in incarcerated adolescents. *Psychological Assessment, 18*(2), 142-154.

Neumann, C. S., Wampler, M., Taylor, J., Blonigen, D. M., & Iacono, W. G. (2011). Stability and invariance of psychopathic traits from late adolescence to young adulthood. *Journal of Research in Personality, 45*(2), 145-152.

Newman, J. P. (1998). *Psychopathic behavior: An information processing perspective*. Psychopathy: Theory, Research, and Implications for Society. Kluwer Academic Publishers, Dordrecht, The Netherlands. pp. 81-104.

Newman, J. P., & Baskin-Sommers, A. R. (2012). *Early selective attention abnormalities in psychopathy: Implications for self-regulation*. In M. I. Posner (Ed.), Cognitive Neuroscience of Attention (2 ed., pp 421-439). United States of America: The Guilford Press.

Newman, J. P., Curtin, J. J., Bertsch, J. D., & Baskin-Sommers, A. R. (2010). Attention moderates the fearlessness of psychopathic offenders. *Biological Psychiatry, 67*(1), 66-70.

Newman, J. P., & Kosson, D. S. (1986). Passive avoidance learning in psychopathic and nonpsychopathic offenders. *Journal of Abnormal Psychology, 95*(3), 252-256.

Newman, J. P., & Lorenz, A. R. (2003). *Response modulation and emotion processing: Implications for psychopathy and other dysregulatory psychopathology.* Handbook of Affective Sciences. Series in Affective Science. New York, NY, US: Oxford University Press, pgs 904-929.

Nieuwenhuis, S., Yeung, N., Van Den Wildenberg, W., & Ridderinkhof, K. R. (2003). Electrophysiological correlates of anterior cingulate function in a go/nogo task: Effects of response conflict and trial type frequency. *Cognitive, Affective, & Behavioral Neuroscience, 3*, 17-26.

Obradovic, J., Pardini, D. A., Long, J. D., Loeber, R., & 2007. (2007). Measuring interpersonal callousness in boys from childhood to adolescence: An examination of longitudinal invariance and temporal stability. *Journal of Clinical Child and Adolescent Psychology, 36*(3), 276-292.

Ogilvie, J. M., Stewart, A. L., Chan, R. C. K., & Shum, D. H. K. (2011). Neuropsychological measures of executive function and antisocial behavior: A meta-analysis. *Criminology, 49*(4), 1063-1107.

Oishi, K., Faria, A. V., Hsu, J., Tippett, D., Mori, S., & Hillis, A. E. (2015). Critical role of the right uncinate fasciculus in emotional empathy. *Annals of Neurology, 77*, 68-74.

Olson, E. A., Collins, P. F., Hooper, C. J., Muetzel, R., Lim, K. O., & Luciana, M. (2009). White matter integrity predicts delay discounting behavior in 9- to 23-year-olds: A diffusion tensor imaging study. *Journal of Cognitive Neuroscience, 21*, 1406-1421.

Olvet, D. M., & Hajcak, G. (2008). The error-related negativity (ERN) and psychopathology: Toward an endophenotype. *Clinical Psychology Review, 28*(8), 1343-1354.

Olvet, D. M., & Hajcak, G. (2009). The stability of error-related brain activity with increasing trials. *Psychophysiology, 46*(5), 957-961.

Onifade, E., Petersen, J., Bynum, T. S., & Davidson, W. S. (2011). Multilevel recidivism prediction: Incorporating neighborhood socioeconomic ecology in juvenile justice risk assessment. *Criminal Justice and Behavior, 38*(8), 840-853.

Overbeek, T. J. M., Nieuwenhuis, S., & Ridderinkhof, K. R. (2005). Dissociable components of error processing. *Journal of Psychophysiology, 19*(4), 319-329. doi: 10.1027/0269-8803.19.4.319

Pape, L. E., Cohn, M. D., Caan, M. W. A., van Wingen, G., van den Brink, W., Veltman, D. J., & Popma, A. (2015). Psychopathic traits in adolescents are associated with higher structural connectivity. *Psychiatry Research: Neuroimaging, 233*, 474-480.

Pardini, D., Obradovic, J., & Loeber, R. (2006). Interpersonal callousness, hyperactivity/impulsivity, inattention, and conduct problems as precursors to delinquency persistence in boys: A comparison of three grade-based cohorts. *Journal of Clinical Child and Adolescent Psychology, 35*, 46-59.

Parsons Winokur, K., Smith, A., Bontrager, S. R., & Blankenship, J. L. (2008). Juvenile recidivism and length of stay. *Journal of Criminal Justice, 36*(2), 126-137.

Passamonti, L., Fairchild, G., Goodyer, I. M., Hurford, G., Hagan, C. C., Rowe, J. B., & Calder, A. J. (2010). Neural abnormalities in early-onset and adolescence-onset conduct disorder. *JAMA Psychiatry, 67*(7), 729-738.

Patrick, C. J. (1994). Emotion and psychopathy: Startling new insights. *Psychophysiology, 31*(4), 319-330.

Petrides, M., & Pandya, D. N. (2007). Efferent association pathways from the rostral prefrontal cortex in the macaque monkey. *Journal of Neuroscience, 27*(43), 11573-11586.

Philippi, C. L., Mehta, S., Grabowski, T., Adolphs, R., & Rudrauf, D. (2009). Damage to association fiber tracts impairs recognition of the facial expression of emotion. *Journal of Neuroscience, 29*, 15089-15099.

Philippi, C. L., Pujara, M. S., Motzkin, J. C., Newman, J., Kiehl, K. A., & Koenigs, M. (2015). Altered resting-state functional connectivity in cortical networks in psychopathy. *Journal of Neuroscience, 35*(15), 6068-6078.

Picton, T. W., Stuss, D. T., Alexander, M. P., Shallice, T., Binns, M. A., & Gillingham, S. (2006). Effects of focal frontal lesions on response inhibition. *Cerebral Cortex, 17*(4), 826-838.

Pierpaoli, C., & Basser, P. J. (1996). Toward a quantitative assessment of diffusion anisotropy. *Magnetic Resonance in Medicine, 36*(6), 893-906.

Pinel, P. (1800). *Analectes sur le Traité Médico-Philosophique sur l'Aliénation Mentale ou la Manie Richard*. Paris: Caille et Ravier Libraries, No. 11, an ix.

Piquero, A. (2001). Testing Moffitt's neuropsychological variation hypothesis for the prediction of life-course persistent offending. *Psychology, Crime & Law, 7*(3), 193-215.

Pontifex, M. B., Scudder, M. R., Brown, M. L., O'Leary, K. C., Wu, C. T., Themanson, J. R., & Hillman, C. H. (2010). On the number of trials necessary for stabilization of error-related brain activity across the life span. *Psychophysiology, 47*(4), 767-773.

Posner, M. I., Rothbart, M. K., Sheese, B., & Tang, Y. (2007). The anterior cingulate gyrus and the mechanism of self-regulation. *Cognitive, Affective, & Behavioral Neuroscience, 7*, 391-395.

Puzzanchera, C. (2014). *Juvenile arrests, 2012*. US Department of Justice, Office of Juvenile Justice and Delinquency Prevention. Available: http://www.ojjdp.gov/pubs/248513.pdf. Accessed June 2019.

Quinque, E. M., Arélin, K., Dukart, J., Roggenhofer, E., Streitbuerger, D. P., & Villringer, A. (2012). Identifying the neural correlates of executive functions in early cerebral microangiopathy: A combined VBM and DTI study. *Journal of Cerebral Blood Flow and Metabolism, 32*, 1869-1878.

Rabbit, P. M. A. (1981). *Sequential reactions*. In D. Holding (Ed.), Human Skills (pp. 153-175). New York: Wiley.

Raschle, N. M., Menks, W. M., Fehlbaum, L. V., Steppan, M., Smaragdi, A., Gonzalez-Madruga, K., . . . Stadler, C. (2018). Callous-unemotional traits and brain structure: Sex-specific effects in anterior insula of typically-developing youths. *NeuroImage: Clinical, 17*, 856-864.

Rice, M. E., & Harris, G. T. (1997). Cross-validation and extension of the Violence Risk Appraisal Guide for child molesters and rapists. *Law and Human Behavior, 21*(2), 231-241.

Rizzolatti, G., & Luppino, G. (2001). The cortical motor system. *Neuron, 31*, 889-901.

Robbins, R. N., & Bryan, A. (2004). Relationships between future orientation, impulse sensation seeking, and risk behavior among adjudicated adolescents. *Journal of Adolescent Research, 19*(4), 428-445.

Romer, D., Betancourt, L., Giannetta, J. M., Brodsky, N. L., Farah, M., & Hurt, H. (2009). Executive cognitive functions and impulsivity as correlates of risk taking and problem behavior in preadolescents. *Neuropsychologia, 47*(13), 2916-2926.

Rosenfeld, R., Helene, R. W., & Finn-Aage, E. (2012). *Special categories of serious and violent offenders: Drug dealers, gang members, homicide offenders, and sex offenders*. in From Juvenile Delinquency to Adult Crime: Criminal Careers, Justice Policy, and Prevention, New York: Oxford University Press, pp. 118-149.

Roth, R. M., Saykin, A. J., Flashman, L. A., Pixley, H. S., West, J. D., & Mamourian, A. C. (2007). Event-related functional magnetic resonance imaging of response

inhibition in Obsessive-Compulsive Disorder. *Biological Psychiatry, 62*(8), 901-909.

Santesso, D. L., Segalowitz, S. J., & Schmidt, L. A. (2006). Error-related electrocortical responses in 10-year old children and young adults. *Developmental Science, 22*(2), 433-445.

Sarkar, S., Craig, M. C., Dell'Acqua, F., Fahy, T., Deeley, Q., & Murphy, D. G. M. (2013). Frontotemporal white-matter microstructural abnormalities in adolescents with conduct disorder: A diffusion tensor imaging study. *Psychological Medicine, 43*, 401-411.

Schmithorst, V. J., Holland, S. K., & Dardzinski, B. J. (2008). Developmental differences in white matter architecture between boys and girls. *Human Brain Mapping, 29*(6), 696-710.

Schmithorst, V. J., Wilke, M., Dardzinski, B. J., & Holland, S. K. (2002). Correlation of white matter diffusivity and anisotropy with age during childhood and adolescence: A cross-sectional diffusion-tensor MR imaging study. *Radiology, 222*(1), 212-218.

Schroder, H. S., Moran, T. P., Moser, J. S., & Altmann, E. M. (2012). When the rules are reversed: Action-monitoring consequences of reversing stimulus-response mappings. *Cognitive, Affective, & Behavioral Neuroscience, 12*(4), 629-643.

Schubert, C. A., Mulvey, E. P., & Glasheen, C. (2011). Influence of mental health and substance use problems and criminogenic risk on outcomes in serious juvenile offenders. *Journal of the American Academy of Child and Adolescent Psychiatry, 50*(9), 925-937.

Schwalbe, C. S., Fraser, M. W., Day, S. H., & Cooley, V. (2006). Classifying juvenile offenders according to risk of recidivism: Predictive utility, race/ethnicity, and gender. *Criminal Justice and Behavior, 33*, 305-324.

Seagrave, D., & Grisso, T. (2002). Adolescent development and the measurement of juvenile psychopathy. *Law and Human Behavior, 26*(2), 219-239.

Sebastian, C. L., De Brito, S. A., McCrory, E. J., Hyde, Z. H., Lockwood, P. L., Cecil, C. A. M., & Viding, E. (2016). Grey matter volumes in children with conduct problems and varying levels of callous-unemotional traits. *Journal of Abnormal Child Psychology, 44*, 639-649.

Sebastian, C. L., McCrory, E. J., Cecil, C. A. M., Lockwood, P. L., De Brito, S. A., Fontaine, N. M. G., & Viding, E. (2012). Neural responses to affective and cognitive theory of mind in children with conduct problems and varying levels of callous-unemotional traits. *Archives in General Psychiatry, 69*(8), 814-822.

Seghete, K. L., Herting, M. M., & Nagel, B. J. (2013). White matter microstructure correlates of inhibition and task-switching in adolescents. *Brain Research, 1527*, 15-28.

Sethi, A., Gregory, S., Dell'Acqua, F., Thomas, E. P., Simmons, A., Murphy, D. G. M., . . . Craig, M. C. (2015). Emotional detachment in psychopathy: Involvement of dorsal default-mode connections. *Cortex, 62*, 11-19.

Sharp, C., & Kine, S. (2008). The assessment of juvenile psychopathy: Strengths and weaknesses of currently used questionnaire methods. *Child and Adolescent Mental Health, 13*(2), 85-95.

Sheng, T., Gheytanchi, A., & Aziz-Zadeh, L. (2010). Default network deactivations are correlated with psychopathic personality traits. *PLoS ONE, 5*, e12611.

Sickmund, M., & Puzzanchera, C. (2014). *Juvenile offenders and victims: 2014 National Report*. National Center for Juvenile Justice, Pittsburgh, PA.

Sickmund, M., Sladky, T. J., Kang, W., & Puzzanchera, C. (2011). *Easy access to the census of juveniles in residential placement*. Available at: http://www.ojjdp.gov/ojstatbb/ezacjrp/ [June 2019].

Simmonds, D. J., Pekar, J. J., & Mostofsky, S. H. (2008). Meta-analysis of go/no-go tasks demonstrating that fMRI activation associated with response inhibition is task-dependent. *Neuropsychologia, 46*(1), 224-232.

Simonoff, E., Elander, J., Holmshaw, J., Pickles, A., Murray, R., & Rutter, M. (2004). Predictors of antisocial personality: Continuities from childhood to adult life. *The British Journal of Psychiatry, 184*, 118-127.

Skeem, J. L., & Cauffman, E. (2003). Views of the downward extension: Comparing the Youth version of the Psychopathy Checklist with the Youth Psychopathic Traits Inventory. *Behavioral Sciences & The Law, 21*(6), 737-770.

Skeem, J. L., & Cooke, D. J. (2010). Is criminal behavior a central component of psychopathy? Conceptual directions for resolving the debate. *Psychological Assessment, 22*(2), 433-445.

Skeem, J. L., & Mulvey, E. P. (2001). Psychopathy and community violence among civil psychiatric patients: Results from the MacArthur Violence Risk Assessment Study. *Journal of Consulting and Clinical Psychology, 69*(3), 358-374.

Smith, S. M., Jenkinson, M., Johansen-Berg, H., Rueckert, D., Nichols, T. E., Mackay, C. E., . . . Behrens, T. E. (2006). Tract-based spatial statistics: Voxelwise analysis of multi-subject diffusion data. *NeuroImage, 31*(4), 1487-1505.

Sobhani, M., Baker, L., Martins, B., Tuvblad, C., & Aziz-Zadeh, L. (2015). Psychopathic traits modulate microstructural integrity of right uncinate fasciculus in a community population. *NeuroImage: Clinical, 8*, 32-38.

Sohn, W. S., Yoo, K., Lee, Y.-B., Seo, S. W., Na, D. L., & Jeong, Y. (2015). Influence of ROI selection on resting state functional connectivity: An individualized approach for resting state fMRI analysis. *Frontiers in Neuroscience, 9*, 1-10.

Sowell, E. R., Thompson, P. M., Leonard, C. M., Welcome, S. E., Kan, E., & Toga, A. W. (2004). Longitudinal mapping of cortical thickness and brain growth in normal children. *Journal of Neuroscience, 24*, 8223-8231.

Spain, S. E., Douglas, K. S., Poythress, N. G., & Epstein, M. (2004). The relationship between psychopathic features, violence and treatment outcome: The comparison of three youth measures of psychopathic features. *Behavioral Sciences & The Law, 22*(1), 85-102.

Steele, V. R., Aharoni, E., Munro, G. E., Calhoun, V. D., Nyalakanti, P., Stevens, M. C., . . . Kiehl, K. A. (2013). A large scale (*n* = 102) functional neuroimaging study of response inhibition in a Go/NoGo task. *Behavioural Brain Science, 256*, 529-536.

Steele, V. R., Anderson, N. E., Claus, E. D., Bernat, E. M., Rao, V., Assaf, M., . . . Kiehl, K. A. (2016). Neuroimaging measures of error-processing: Extracting reliable signals from event-related potentials and functional magnetic resonance imaging. *NeuroImage, 132*, 247-260.

Steele, V. R., Claus, E. D., Aharoni, E., Harenski, C., Calhoun, V. D., Pearlson, G. D., & Kiehl, K. A. (2014). A large scale (*n* = 102) functional neuroimaging study of error processing in a Go/NoGo task. *Behavioural Brain Science, 268*, 127-138.

Steele, V. R., Claus, E. D., Aharoni, E., Vincent, G. M., Calhoun, V. D., & Kiehl, K. A. (2015). Multimodal imaging measures predict rearrest. *Frontiers in Human Neuroscience, 9*(425), 1-13.

Steele, V. R., Fink, B. C., Maurer, J. M., Arbabshirani, M. R., Wilber, C. H., Jaffe, A. J., . . . Kiehl, K. A. (2014). Brain potentials measured during a go/nogo task predict completion of substance abuse treatment. *Biological Psychiatry, 76*(1), 75-83.

Steele, V. R., Maurer, J. M., Bernat, E. M., Calhoun, V. D., & Kiehl, K. A. (2016). Error-related processing in adult males with elevated psychopathic traits. *Personality Disorders: Theory, Research, and Treatment, 7*(1), 80-90.

Steele, V. R., Rao, V., Calhoun, V. D., & Kiehl, K. A. (2017). Machine learning of structural magnetic resonance imaging predicts psychopathic traits in adolescent offenders. *NeuroImage, 145*, 265-273.

Stevens, M., Kiehl, K. A., Pearlson, G. D., & Calhoun, V. D. (2007). Functional neural networks underlying response inhibition in adolescents and adults. *Behavioural Brain Science, 181*, 12-22.

Sumich, A., Sarkar, S., Hermens, D. F., Ibrahimovic, A., Kelesidi, K., Wilson, D., & Rubia, K. (2012). Sex differences in brain maturation as measured using event-related potentials. *Developmental Neuropsychology, 37*(5), 415-433.

Sumich, A., Sarkar, S., Hermens, D. F., Kelesidi, K., Taylor, E., & Rubia, K. (2012). Electrophysiological correlates of CU traits show abnormal regressive maturation in adolescents with conduct problems. *Personality and Individual Differences, 53*(7), 862-867.

Sundram, F., Deeley, Q., Sarkar, S., Daly, E., Latham, R., Craig, M., . . . Murphy, D. G. M. (2012). White matter abnormalities in the frontal lobe of adults with antisocial personality disorder. *Cortex, 48*, 216-229.

Swick, D., Ashley, V., & Turken, U. (2008). Left inferior frontal gyrus is critical for response inhibition. *BMC Neuroscience, 9*(102), 1-11.

Swick, D., & Turken, A. U. (2002). Dissociation between conflict detection and error monitoring in the human anterior cingulate cortex. *Proceedings of the National Academy of Sciences, 9925*, 16354-16359.

Tekin, S., & Cummings, J. L. (2002). Frontal-subcortical neuronal circuits and clinical neuropsychiatry: An update. *Journal of Psychosomatic Research, 532*, 647-654.

Thomas, C., Avidan, G., Humphreys, K., Jung, K., Gao, F., & Behrmann, M. (2009). Reduced structural connectivity in ventral visual cortex in congenital prosopagnosia. *Nature Neuroscience, 12*, 29-31.

Tian, Y., & Yao, D. (2008). A study on the neural mechanism of inhibition of return by the event-related potential in the Go/NoGo task. *Biological Psychology, 79*, 171-178.

Tiihonen, J., Rossi, R., Laakso, M. P., Hodgins, S., Testa, C., Perez, J., . . . Frisoni, G. B. (2008). Brain anatomy of persistent violent offenders: More rather than less. *Psychiatry Research: Neuroimaging, 163*(3), 201-212.

Trulson, C. R., Marquart, J. W., Mullings, J. L., & Caeti, T. J. (2005). In between adolescence and adulthood: Recidivism outcomes of a cohort of state delinquents. *Youth Violence and Juvenile Justice, 3*, 355-387.

Turken, A., Whitfield-Gabrieli, S., Bammer, R., Baldo, J. V., Dronkers, N. F., & Gabrieli, J. D. (2008). Cognitive processing speed and the structure of white matter pathways: Convergent evidence from normal variation and lesion studies. *NeuroImage, 42*, 1032-1044.

Uhlmann, A., Fouche, J.-P., Lederer, K., Meintjes, E. M., Wilson, D., & Stein, D. J. (2016). White matter microstructure and impulsivity in methamphetamine dependence with and without a history of psychosis. *Human Brain Mapping, 37*(6), 2055-2067.

Ullsperger, M., Harsay, H. A., Wessel, J. R., & Ridderinkhof, K. R. (2010). Conscious perception of errors and its relation to the anterior insula. *Brain Structure & Function, 214*(5-6), 629-643.

van Dam, C., Janssens, J. M. A. M., & De Bruyn, E. E. J. (2004). PEN, Big Five, juvenile delinquency and criminal recidivism. *Personality and Individual Differences, 39*, 7-19.

van Veen, V., & Carter, C. S. (2002). The timing of action-monitoring processes in the anterior cingulate cortex. *Journal of Cognitive Neuroscience, 14*(4), 593-602.

Vermeij, A., Kempes, M. M., Cima, M. J., Mars, R. B., & Brazil, I. A. (2018). Affective traits of psychopathy are linked to white-matter abnormalities in impulsive male offenders. *Neuropsychology, 32*(6), 735-745.

Vermeiren, R., Schwab-Stone, M. E., Ruchkin, V., De Clippele, A., & Deboutte, D. (2002). Predicting recidivism in delinquent adolescents from psychological and psychiatric assessment. *Comparative Psychiatry, 43*(2), 142-149.

Viding, E., Sebastian, C. L., Dadds, M. R., Lockwood, P. L., Cecil, C. A. M., De Brito, S. A., & McCrory, E. J. (2012). Amygdala response to preattentive masked fear in children with conduct problems: The role of callous-unemotional traits. *American Journal of Psychiatry, 169*, 1109-1116.

Vincent, G. M., Cope, L. M., King, J., Nyalakanti, P., & Kiehl, K. A. (2017). Callous-unemotional traits modulate brain drug craving response in high-risk young offenders. *Journal of Abnormal Child Psychology*, 1-17.

Vincent, G. M., Odgers, C. L., McCormick, A. V., & Corrado, R. R. (2008). The PCL:YV and recidivism in male and female juveniles: A follow-up into young adulthood. *International Journal of Law and Psychiatry, 31*(3), 287-296.

Vitacco, M. J., Neumann, C. S., Robertson, A. A., & Durrant, S. L. (2002). Contributions of impulsivity and callousness in the assessment of adjudicated male adolescents: A prospective study. *Journal of Personality Assessment, 78*(1), 87-103.

Vitacco, M. J., & Rogers, R. (2001). Predictors of adolescent psychopathy: The role of impulsivity, hyperactivity, and sensation seeking. *Journal of the American Academy of Psychiatry and the Law, 29*(4), 374-382.

Vitale, J. E., Newman, J. P., Bates, J. E., Goodnight, J., Dodge, K. A., & Pettit, G. S. (2005). Deficient behavioral inhibition and anomalous selective attention in a community sample of adolescents with psychopathic traits and low-anxiety traits. *Journal of Abnormal Child Psychology, 33*(4), 461-470.

Wager, T. D., Sylvester, C. Y., Lacey, S. C., Nee, D. E., Franklin, M., & Jonides, J. (2005). Common and unique components of response inhibition revealed by fMRI. *NeuroImage, 27*, 323-340.

Wallace, G. L., White, S. F., Robustelli, B., Sinclair, S., Hwang, S., Martin, A., & Blair, R. J. R. (2014). Cortical and subcortical abnormalities in youths with conduct disorder and elevated callous-unemotional traits. *Journal of the American Academy of Child and Adolescent Psychiatry, 53*(4), 456-465.

Walters, G. D. (2003). Predicting institutional adjustment and recidivism with the Psychopathy Checklist factor scores: A meta-analysis. *Law and Human Behavior, 27*, 541-558.

Walters, G. D., Ermer, E., Knight, R. A., & Kiehl, K. A. (2015). Paralimbic biomarkers in taxometric analyses of psychopathy: Does changing the indicators change the conclusion? *Personality Disorders: Theory, Research, and Treatment, 6*(1), 41-52.

Walters, G. D., & Kiehl, K. A. (2015). Limbic correlates of fearlessness and disinhibition in incarcerated youth: Exploring the brain-behavior relationship with the Hare Psychopathy Checklist: Youth Version. *Psychiatry Research, 230*, 205-210.

Wasserman, G. A., McReynolds, L. S., Lucas, C. P., Fisher, P., & Santos, L. (2002). The Voice DISC-IV with incarcerated male youths: Prevalence of disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 41*(3), 314-321.

Wechsler, D. (1997). *WAIS-III: Wechsler Adult Intelligence Scale (3rd Edition)*. San Antonio, TX: The Psychological Corporation.

Wechsler, D. (2003). *Wechsler Intelligence Scale for Children - 4th Edition*. San Antonio, TX: Harcourt Assessment.

Wheeler, M. S., Arnkoff, D. B., & Glass, C. R. (2017). The neuroscience of mindfulness: How mindfulness alters the brain and facilitates emotion regulation. *Mindfulness, 8*(6), 1471-1487.

White, S. F., Brislin, S. J., Meffert, H., Sinclair, S., & Blair, R. J. R. (2013). Callous-unemotional traits modulate the neural response associated with punishing another individual during social exchange: A preliminary investigation. *Journal of Personality Disorders, 27*(1), 99-112.

White, S. F., Marsh, A. A., Fowler, K. A., Schechter, J. C., Adalio, C., Pope, K., . . . Blair, R. J. (2012). Reduced amygdala response in youths with disruptive behavior disorders and psychopathic traits: Decreased emotional response versus increased top-down attention to nonemotional features. *American Journal of Psychiatry, 169*(7), 750-758.

Williamson, S., Harpur, T. J., & Hare, R. D. (1991). Abnormal processing of affective words by psychopaths. *Psychophysiology, 283*, 260-273.

Winklewski, P. J., Sabisz, A., Naumczyk, P., Jodzio, K., Szurowska, E., & Szarmach, A. (2018). Understanding the physiopathology behind axial and radial diffusivity changes - What do we know? *Frontiers in Neurology, 9*(92), 1-8.

Winston, G. P. (2012). The physical and biological basis of quantitative parameters derived from diffusion MRI. *Quantitative Imaging in Medicine and Surgery, 2*, 254-265.

Wittfoth, M., Küstermann, E., Fahle, M., & Herrmann, M. (2008). The influence of response conflict on error processing: Evidence from event-related fMRI. *Brain Research, 1194*, 118-129.

Wolf, R. C., Plichta, M. M., Sambataro, F., Fallgatter, A. J., Jacob, C., Lesch, K.-L., . . . Vasic, N. (2009). Regional brain activation changes and abnormal functional connectivity of the ventrolateral prefrontal cortex during working memory processing in adults with attention-deficit/hyperactivity disorder. *Human Brain Mapping, 30*(7), 2252-2266.

Wolf, R. C., Pujara, M. S., Motzkin, J. C., Newman, J. P., Kiehl, K. A., Decety, J., . . . Koenigs, M. (2015). Interpersonal traits of psychopathy linked to reduced integrity of the uncinate fasciculus. *Human Brain Mapping, 36*, 4202-4209.

Woltering, S., Granic, I., Lamm, C., & Lewis, M. D. (2011). Neural changes associated with treatment outcome in children with externalizing problems. *Biological Psychiatry, 70*(9), 873-879.

Wozniak, J. R., Krach, L., Ward, E., Mueller, B. A., Muetzel, R., Schnoebelen, S., . . . Lim, K. O. (2007). Neurocognitive and neuroimaging correlates of pediatric traumatic brain injury: A diffusion tensor imaging (DTI) study. *Archives of Clinical Neuropsychology, 22*, 555-568.

Yang, Y., Raine, A., Colletti, P., Toga, A. W., & Narr, K. L. (2010). Morphological alterations in the prefrontal cortex and the amygdala in unsuccessful psychopaths. *Journal of Abnormal Psychology, 119*(3), 546-554.

Ystad, M., Eichele, T., Lundervold, A. J., & Lundervold, A. (2010). Subcortical functional connectivity and verbal episodic memory in healthy elderly -- A resting state fMRI study. *NeuroImage, 52*(1), 379-388.

Yu, C., Li, J., Liu, Y., Qin, W., Li, Y., Shu, N., . . . Li, K. (2008). White matter tract integrity and intelligence in patients with mental retardation and healthy adults. *NeuroImage, 40*(4), 1533-1541.

Yuan, W., Holland, S. K., Schmithorst, V. J., Walz, N. C., Cecil, K. M., Jones, B. V., . . . Wade, S. L. (2007). Diffusion Tensor MR Imaging reveals persistent white matter alteration after traumatic brain injury experienced during early childhood. *American Journal of Neuroradiology, 28*(10), 1919-1925.

Zhang, J., Gao, J., Shi, H., Huang, B., Wang, X., Situ, W., . . . Yao, S. (2014). Sex differences of uncinate fasciculus structural connectivity in individuals with Conduct Disorder. *BioMed Research International, 673165*, 1-9.

Ziegel, E. R., & Allison, P. D. (1997). Survival analysis using the SAS system by Paul D. Allison. *Technometrics, 39*, 344-345.

Table 1

List of MNI coordinates and regions corresponding to the functional maps for Components 24, 66, and 69

Component 24

Brain Region	Broadmann Area	MNI (x, y, z)
Precuneus	7	(-3, -64, 61) / (3, -61, 64)
Superior Parietal Lobule	7	(-6, -70, 58) / (6, -70, 58)
Postcentral Gyrus	5, 7	(-6, -58, 70) / (6, -55, 67)
Paracentral Lobule	4, 5	(-3, -49, 64) / (3, -49, 64)
Sub-Gyral	n/a	(-12, -55, 61) / (12, -52, 64)
Cingulate Gyrus	31	(-3, -52, 43) / (3, -52, 43)

Component 66

Brain Region	Broadmann Area	MNI (x, y, z)
Anterior Cingulate	10, 24, 25, 32	(-3, 38, -8) / (3, 38, -8)
Medial Frontal Gyrus	10, 11	(-3, 41, -11) / (3, 41, -11)
Superior Frontal Gyrus	10	(-3, 59, 1) / (3, 59, 1)
Subcallosal Gyrus	n/a	(-12, 23, -14) / (0, 0, 0)

| Middle Frontal Gyrus | n/a | (-18, 44, -11) / (18, 44, -11) |

Component 69

Brain Region	Broadmann Area	MNI (x, y, z)
Insula	13, 22, 40, 41	(0, 0, 0) / (48, -19, 16)
Transverse Temporal Gyrus	41, 42	(0, 0, 0) / (51, -19, 13)
Precentral Gyrus	6, 13, 43, 44	(0, 0, 0) / (48, -16, 13)
Postcentral Gyrus	2, 40, 43	(0, 0, 0) / (51, -16, 16)
Superior Temporal Gyrus	13, 22, 41, 42	(0, 0, 0) / (48, -16, 7)
Inferior Parietal Lobule	40	(0, 0, 0) / (51, -28, 22)
Lentiform Nucleus	n/a	(0, 0, 0) / (30, -15, 13)

Table 2

Multiple Regression Analyses with PCL:YV factor scores entered with covariates predicting FA in WM Tracts

Regression #14: Predicting FA values in the R Inferior Longitudinal Fasciculus

Predictors	B	SE B	t	β	Sig.
PCL:YV Factor 1	-0.001	.000	-2.226	-.089	.027
PCL:YV Factor 2	0.000	.000	0.580	.024	.562
Num. Sub. Dep.	0.001	.000	1.537	.057	.126
Age	0.001	.001	1.554	.055	.122
IQ	0.000	.000	2.669	.095	.008
Avg. FA	1.023	.042	24.417	.828	< .001

Regression #18: Predicting FA values in the R Uncinate Fasciculus

Predictors	B	SE B	t	β	Sig.
PCL:YV Factor 1	-0.001	.000	-2.472	-.130	.014
PCL:YV Factor 2	0.001	.000	1.665	.089	.097
Num. Sub. Dep.	0.000	.001	0.259	.012	.796
Age	0.001	.001	2.183	.106	.030
IQ	0.000	.000	1.306	.061	.193
Avg. FA	0.890	.056	15.992	.711	< .001

Note. Regression 14 (predicting FA values in the R inferior longitudinal fasciculus): $R^2 = .727$, $R = .853$, $F(253) = 109.564$, $p < .001$; Regression 18 (predicting FA values in the R uncinate fasciculus): $R^2 = .532$, $R = .729$, $F(253) = 46.708$, $p < .001$.

Table 3

Multiple Regression Analyses with PCL:YV facet scores entered with covariates predicting FA in WM Tracts

Regression #11: Predicting FA values in the L Inferior Fronto-Occipital Fasciculus

Predictors	B	SE B	t	β	Sig.
PCL:YV Facet 1	0.001	.000	1.539	.054	.125
PCL:YV Facet 2	-0.001	.000	-1.998	-.075	.047
PCL:YV Facet 3	0.000	.000	0.591	.021	.555
PCL:YV Facet 4	-0.001	.000	-1.111	-.038	.268
Num. Sub. Dep.	0.001	.000	1.428	.048	.154
Age	0.000	.001	0.609	.020	.543
IQ	0.000	.000	1.080	.035	.281
Avg. FA	1.180	.042	27.967	.863	< .001

Regression #12: Predicting FA values in the R Inferior Fronto-Occipital Fasciculus

Predictors	B	SE B	t	β	Sig.
PCL:YV Facet 1	0.000	.000	0.875	.024	.382
PCL:YV Facet 2	-0.001	.000	-2.024	-.060	.044
PCL:YV Facet 3	0.000	.000	1.276	.036	.203
PCL:YV Facet 4	0.000	.000	-1.334	-.036	.183
Num. Sub. Dep.	0.000	.000	0.795	.021	.427
Age	0.001	.000	2.243	.057	.026

IQ	0.000	.000	1.386	.035	.167
Avg. FA	1.192	.032	37.496	.910	< .001

Regression #14: Predicting FA values in the R Inferior Longitudinal Fasciculus

Predictors	B	SE B	t	β	Sig.
PCL:YV Facet 1	0.000	.000	-0.176	-.007	.860
PCL:YV Facet 2	-0.001	.000	-2.581	-.107	.010
PCL:YV Facet 3	0.000	.000	-0.297	-.012	.766
PCL:YV Facet 4	0.001	.000	1.465	.056	.144
Num. Sub. Dep.	0.001	.000	1.348	.050	.179
Age	0.001	.001	1.544	.055	.124
IQ	0.000	.000	2.450	.087	.015
Avg. FA	1.022	.042	24.399	.827	< .001

Regression #17: Predicting FA values in the L Uncinate Fasciculus

Predictors	B	SE B	t	β	Sig.
PCL:YV Facet 1	-0.001	.000	-1.500	-.063	.135
PCL:YV Facet 2	0.000	.001	-0.559	-.025	.577
PCL:YV Facet 3	0.001	.000	2.065	.088	.040
PCL:YV Facet 4	-0.001	.001	-0.979	-.040	.329
Num. Sub. Dep.	0.001	.001	1.187	.048	.236
Age	0.000	.001	-0.335	-.013	.738

IQ	0.000	.000	1.105	.043	.270
Avg. FA	1.090	.049	22.380	.822	< .001

Regression #18: Predicting FA values in the R Uncinate Fasciculus

Predictors	B	SE B	t	β	Sig.
PCL:YV Facet 1	-0.001	.000	-2.004	-.102	.046
PCL:YV Facet 2	-0.001	.001	-0.903	-.049	.367
PCL:YV Facet 3	0.001	.001	1.918	.100	.056
PCL:YV Facet 4	0.000	.001	-0.021	-.001	.983
Num. Sub. Dep.	0.000	.001	0.325	.015	.745
Age	0.001	.001	2.197	.108	.029
IQ	0.000	.000	1.409	.066	.160
Avg. FA	0.888	.056	15.893	.709	< .001

Note. Regression 11 (predicting FA values in the L inferior fronto-occipital fasciculus): $R^2 = .777$, $R = .882$, $F(253) = 106.859$, $p < .001$; Regression 12 (predicting FA values in the R inferior fronto-occipital fasciculus):$R^2 = .862$, $R = .929$, $F(253) = 191.663$, $p < .001$; Regression 14 (predicting FA values in the R inferior longitudinal fasciculus):$R^2 = .731$, $R = .855$, $F(253) = 83.186$, $p < .001$; Regression 17 (predicting FA values in the L uncinate fasciculus):$R^2 = .684$, $R = .827$, $F(253) = 66.271$, $p < .001$; Regression 18 (predicting FA values in the R uncinate fasciculus):$R^2 = .534$, $R = .731$, $F(253) = 35.114$, $p < .001$.

Table 4

Cox Proportional Hazard Regressions predicting time to rearrest with PC3 mean amplitude and Covariates

Model 1

Predictor	Boot-Strapped B	Boot-Strapped SE (B)	p-value	Exp(B)	Boot-Strapped CI (95%) for exp(B)
PC3 mean	0.694	0.264	.009	2.001	1.19 – 3.36

Model 2

Predictor	Boot-Strapped B	Boot-Strapped SE (B)	p-value	Exp(B)	Boot-Strapped CI (95%) for exp(B)
PC3 mean	0.775	0.259	.003	2.171	1.31 – 3.604
PCL:YV factor interaction	-0.001	0.002	.775	.999	.995 – 1.003
Num. Sub. Dep.	0.029	0.069	.673	1.029	.900 – 1.178
Release Age	0.196	0.087	.025	1.217	1.03 – 1.44

Model 1: PC3 mean amplitude entered alone to predict time to rearrest: -2 Log Likelihood: 594.269, Overall model: $X^2 = 6.921$, $p = .009$.
Model 2: PC3 mean amplitude entered with covariate measures to predict time to rearrest: -2 Log Likelihood: 589.092, Overall model: $X^2 = 12.459$, $p = .014$.

Table 5

Cox Proportional Hazard Regressions predicting time to rearrest with CST FA and Covariates

Model 1

Predictor	Boot-Strapped B	Boot-Strapped SE (B)	p-value	Exp(B)	Boot-Strapped CI (95%) for exp(B)
CST FA	-10.291	4.053	.011	0.000034	0.0 – 0.096 1.0

Model 2

Predictor	Boot-Strapped B	Boot-Strapped SE (B)	p-value	Exp(B)	Boot-Strapped CI (95%) for exp(B)
CST FA	-9.576	3.855	.013	0.000069	0.00 – 0.132
PCL:YV factor interaction	0.001	0.002	.541	1.001	0.998 – 1.004
Num. Sub. Dep.	0.214	0.077	.005	1.238	1.065 – 1.440
Release Age	0.072	0.072	.320	1.074	0.933 – 1.238

Model 1: FA values from the right corticosponal tract entered alone to predict time to rearrest: -2 Log Likelihood: 804.240, Overall model: X^2 = 5.986, *p* = .014.
Model 2: FA values from the right corticospinal tract entered with covariate measures to predict time to rearrest: -2 Log Likelihood: 804.240, Overall model: X^2 = 16.619, *p* = .002.

Figure 1 – *Component #24 obtained through Group Independent Component Analysis*
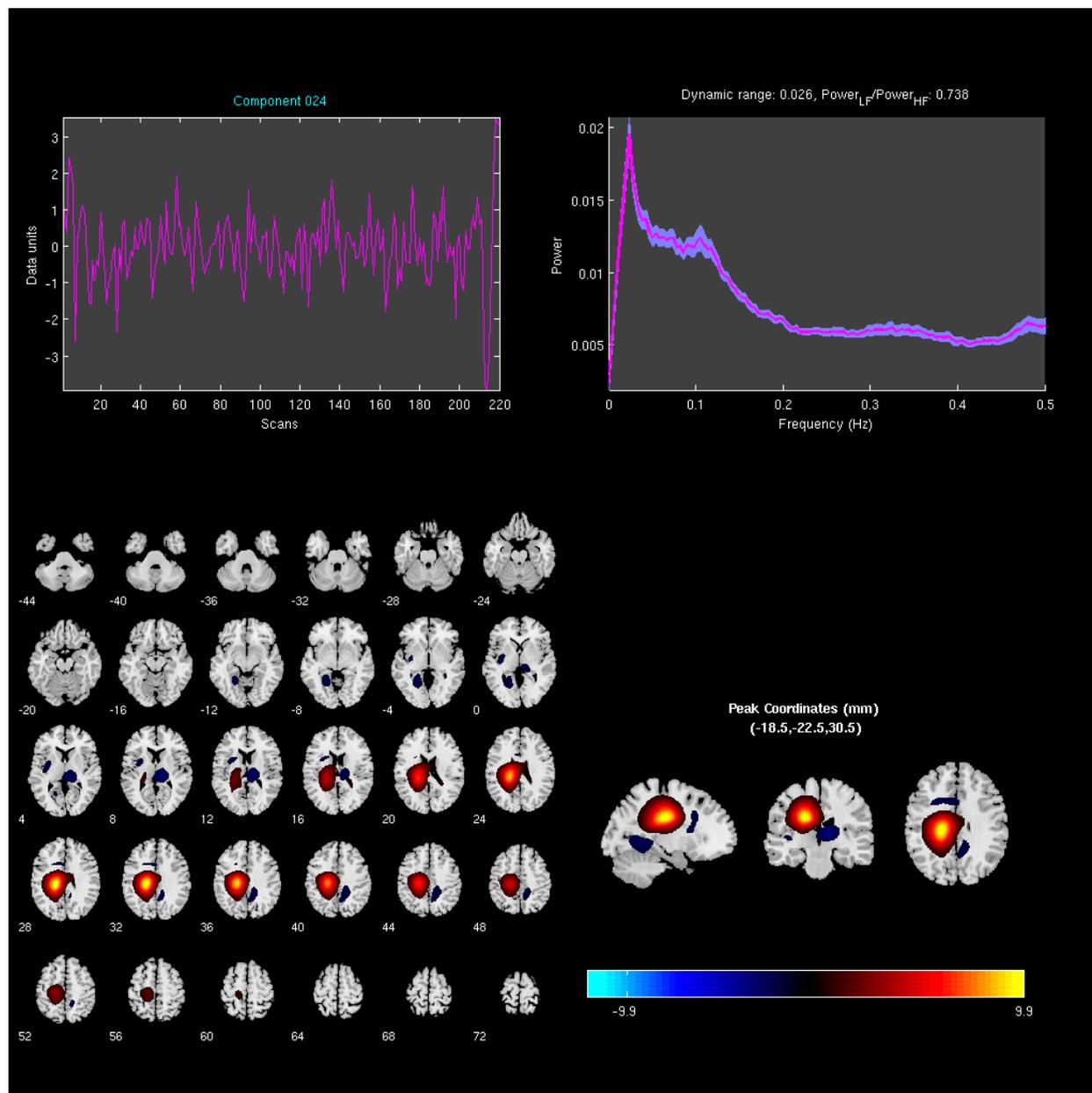

Figure 1. Regions included in Component 24 include the precuneus, superior parietal lobule, postcentral gyrus, paracentral lobule, and cingulate gyrus. Via independent-samples *t*-tests, youth scoring higher on the PCL:YV ($n = 67$) exhibited increased functional connectivity within brain regions incorporated in Component 24 compared to youth scoring low on the PCL:YV ($n = 67$).

Figure 2 – *Component #66 obtained through Group Independent Component Analysis*
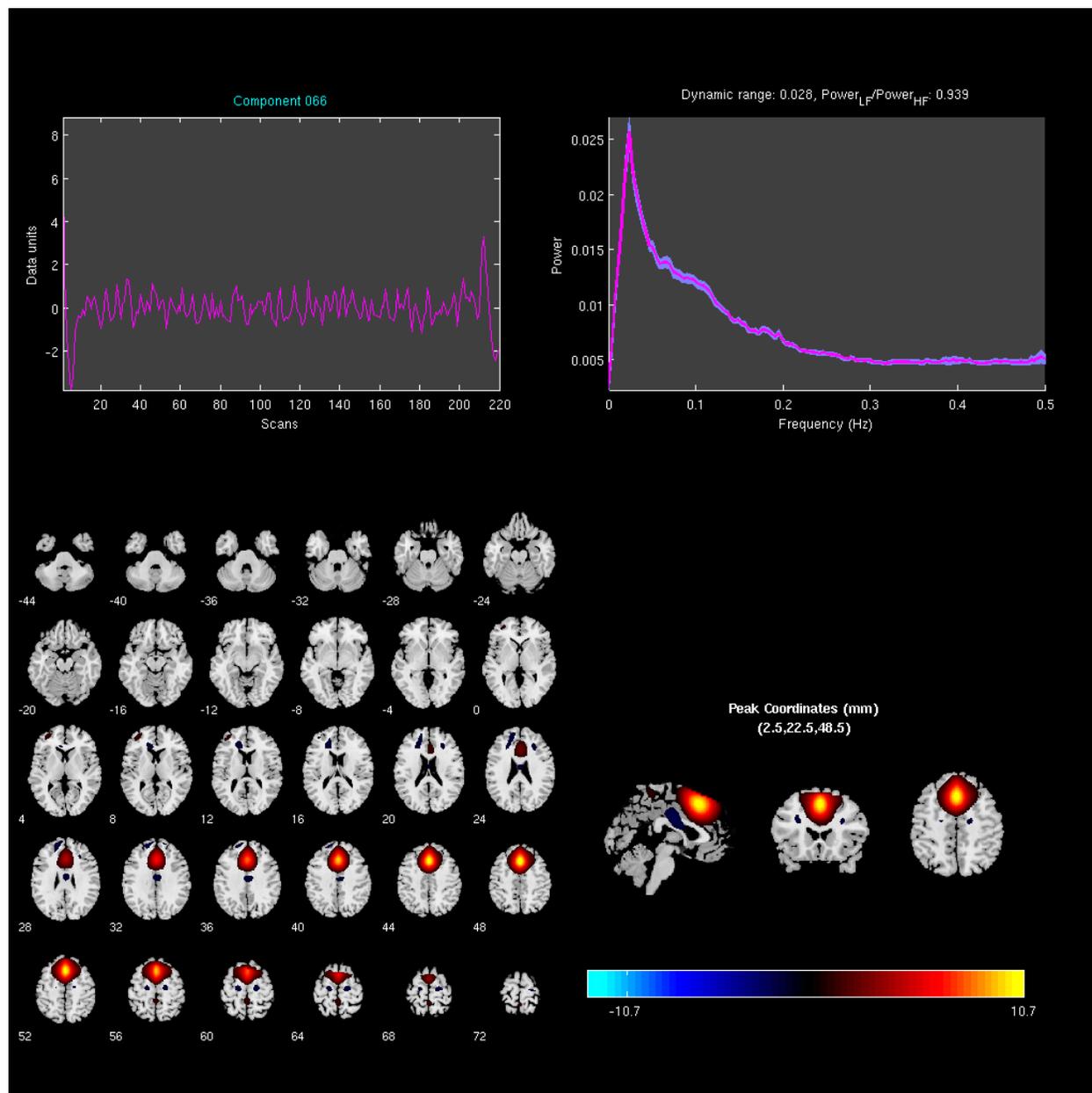
Figure 2. Regions included in Component 66 include the ACC, superior, medial, and middle portions of the frontal gyrus, and subcallosal gyrus. Via independent-samples *t*-tests, youth scoring higher on the PCL:YV ($n = 67$) exhibited increased functional connectivity within brain regions incorporated in Component 66 compared to youth scoring low on the PCL:YV ($n = 67$).

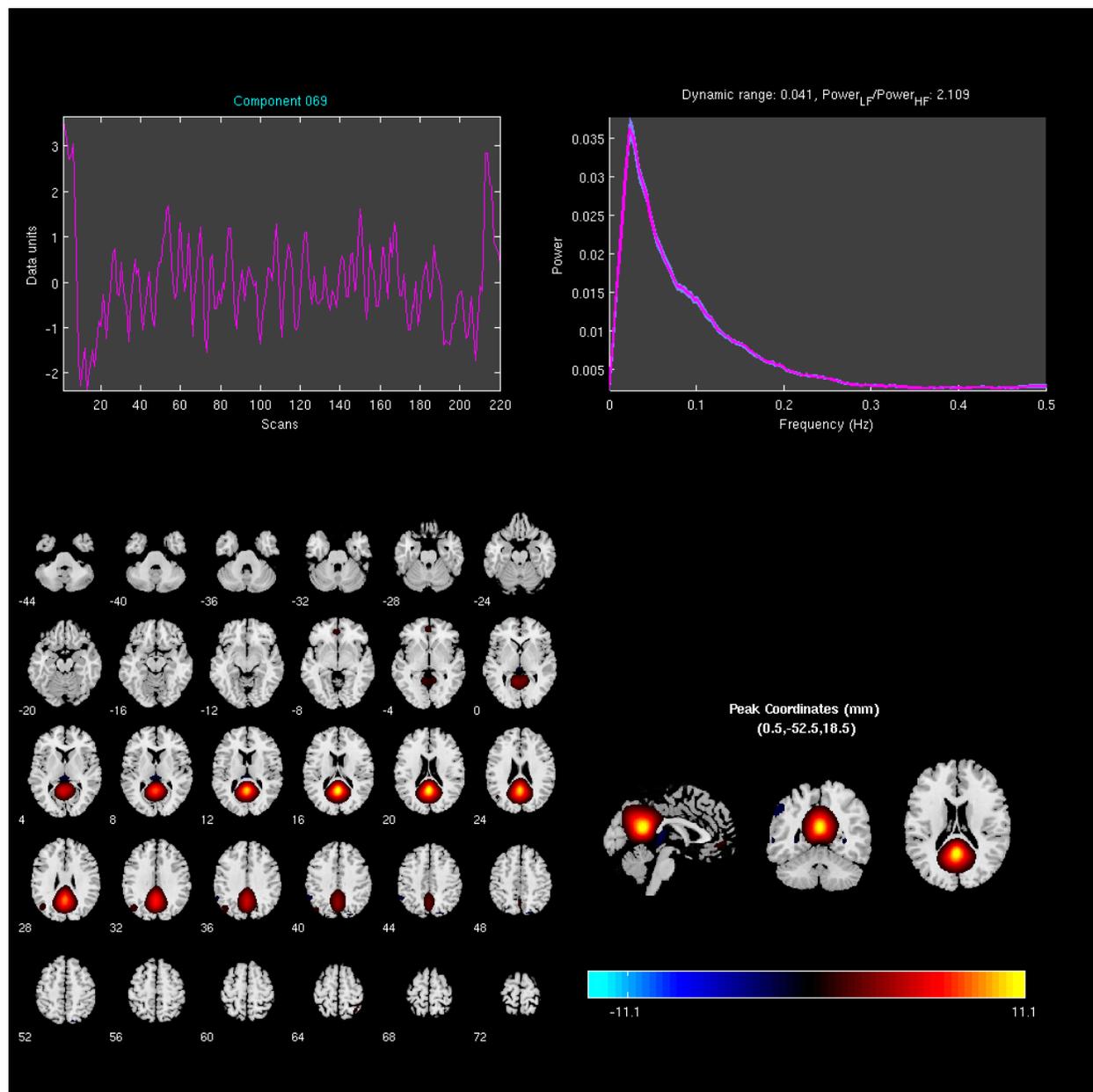**Figure 3 –** *Component #69 obtained through Group Independent Component Analysis*

Figure 3. Regions included in Component 69 include the insula, precentral gyrus, superior temporal gyrus, inferior parietal lobule, and lentiform nucleus. Via independent-samples *t*-tests, youth scoring higher on the PCL:YV ($n = 67$) exhibited reduced functional connectivity within brain regions incorporated in Component 69 compared to youth scoring low on the PCL:YV ($n = 67$).

Figure 4
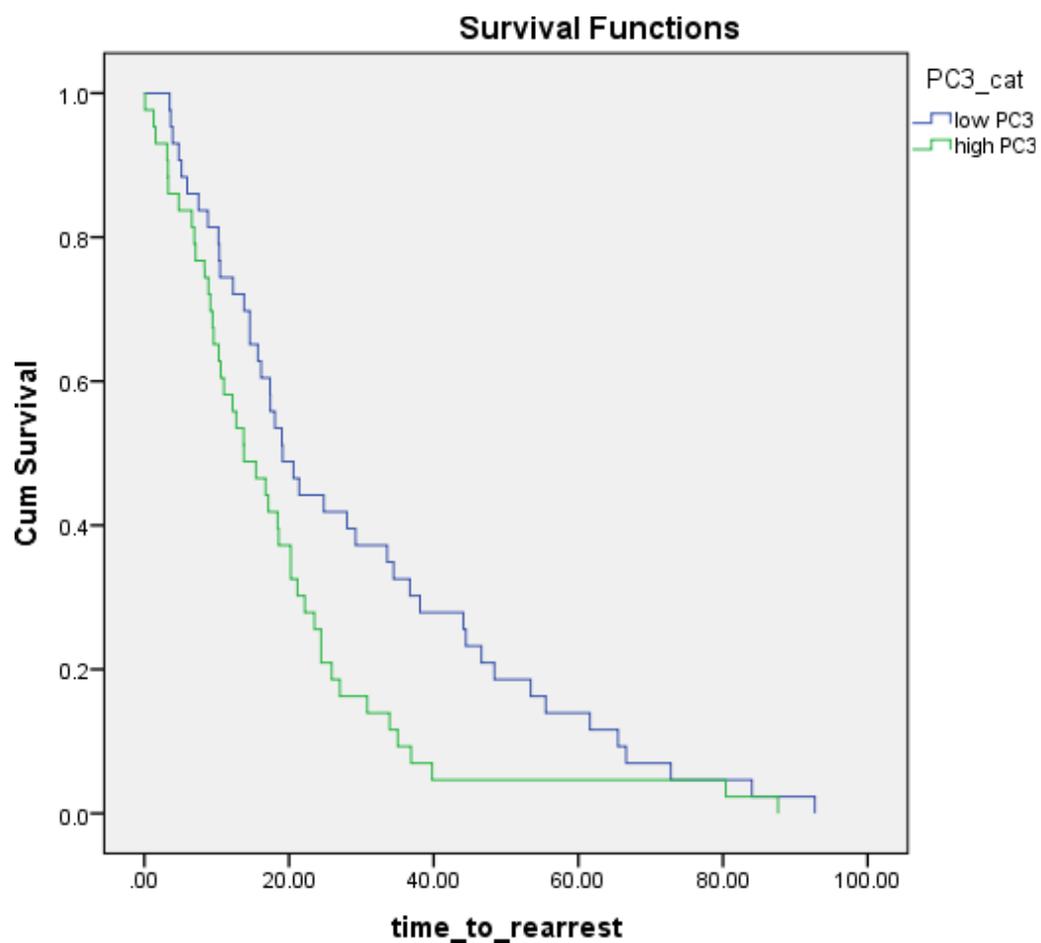
Note. Kaplan-Meier survival plot showing proportional rearrest survival rates of high (*green*) versus low (*blue*) PC3 response groups for any crime over a 100-month period.

Figure 5
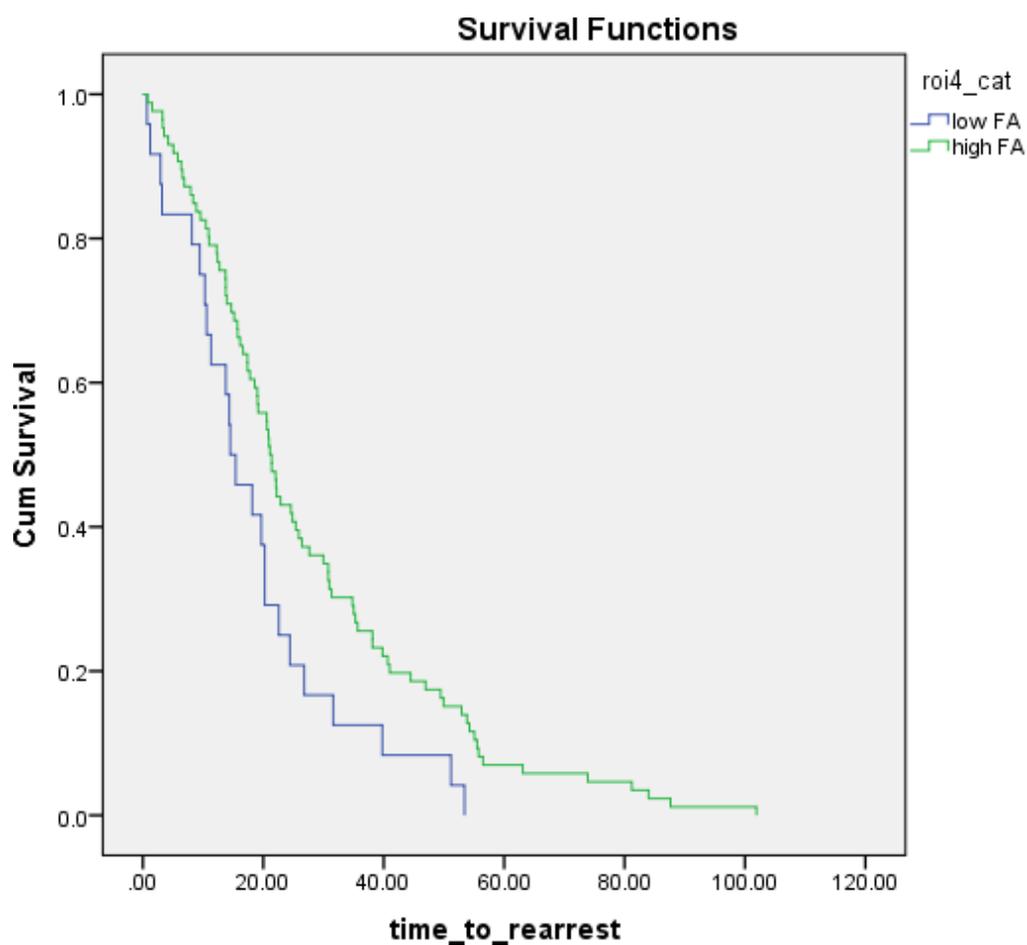
Note. Kaplan-Meier survival plot showing proportional rearrest survival rates of high (*green*) versus low (*blue*) corticospinal FA response groups for any crime over a 100-month period.