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

**by**

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

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**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.

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## 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, Gheytauchi, & 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) (Breden, 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 (Breedon et al., 2015; Pape et al., 2015; Sarkar et al., 2013). Disparate results between previous studies (e.g., reduced FA in the right UF (Breedon 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 – 3<sup>rd</sup> Edition (WAIS-III) (Wechsler, 1997) for participants sixteen years of age or older and from the Wechsler Intelligence Scale for Children – 4<sup>th</sup> 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](http://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 ( $\mu$ s)) 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 (Belleman 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 (Rabbitt, 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 (Rabbitt, 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; Irajii 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 (Breedon 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 (Breedon 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  $\text{s/mm}^2$ ). Five interleaved nondiffusion weighted ( $b$  value = 0  $\text{s/mm}^2$ ) 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  $\text{mm}^3$ , 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  $b_0$  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/](http://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  $\text{mm}^3$  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 ( $\beta = -.10, p = .046$ ), whereas PCL:YV Facet 2 scores emerged as a significant predictor of reduced FA in both the left inferior fronto-occipital fasciculus ( $\beta = -.08, p = .047$ ) and right inferior fronto-occipital fasciculus ( $\beta = -.06, p = .044$ ), and the right inferior longitudinal fasciculus ( $\beta = -.11, p = .010$ ). Alternatively, PCL:YV Facet 3 scores emerged as a significant predictor of increased FA in the left uncinate fasciculus ( $\beta = .09, p = .040$ ).

In addition, PCL:YV Facet 2 scores emerged as a moderate predictor of reduced FA in the left anterior thalamic radiation ( $\beta = -.08, p = .061$ ), PCL:YV Facet 3 scores emerged as a moderate predictor of increased FA in the forceps minor ( $\beta = .07, p = .079$ ) right superior longitudinal fasciculus ( $\beta = -.06, p = .061$ ), and uncinate fasciculus ( $\beta = .10, p = .056$ ). 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 (Breedon 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 (Breedon 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 (Breedon 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 (Breedem 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, Kuo, & 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](http://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 k $\Omega$ .

### **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  $\pm 100 \mu\text{V}$  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 (Rabbitt, 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 (Rabbitt, 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 ( $r = .82, p < .001, r = .72, p < .001, \text{ and } r = .94, p < .001$ , 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 (Himmelstein, 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 were 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 be 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 (Flechsigs, 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 – 3<sup>rd</sup> Edition (WAIS-III) (Wechsler, 1997) for participants sixteen years of age or older and from the Wechsler Intelligence Scale for Children – 4<sup>th</sup> 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  $b_0$  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/](http://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<sup>3</sup> 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 (Himmelstein 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 be 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 (Breedon 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).

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**Table 1**

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

<b>Component 24</b>		
<b>Brain Region</b>	<b>Broadmann Area</b>	<b>MNI (x, y, z)</b>
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)
<b>Component 66</b>		
<b>Brain Region</b>	<b>Broadmann Area</b>	<b>MNI (x, y, z)</b>
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**

<b>Brain Region</b>	<b>Broadmann Area</b>	<b>MNI (x, y, z)</b>
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)

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**Table 2**

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

<b>Regression #14: Predicting FA values in the R Inferior Longitudinal Fasciculus</b>					
<b>Predictors</b>	<b>B</b>	<b>SE B</b>	<b>t</b>	<b><math>\beta</math></b>	<b>Sig.</b>
<b>PCL:YV Factor 1</b>	-0.001	.000	-2.226	-.089	.027
<b>PCL:YV Factor 2</b>	0.000	.000	0.580	.024	.562
<b>Num. Sub. Dep.</b>	0.001	.000	1.537	.057	.126
<b>Age</b>	0.001	.001	1.554	.055	.122
<b>IQ</b>	0.000	.000	2.669	.095	.008
<b>Avg. FA</b>	1.023	.042	24.417	.828	< .001

<b>Regression #18: Predicting FA values in the R Uncinate Fasciculus</b>					
<b>Predictors</b>	<b>B</b>	<b>SE B</b>	<b>t</b>	<b><math>\beta</math></b>	<b>Sig.</b>
<b>PCL:YV Factor 1</b>	-0.001	.000	-2.472	-.130	.014
<b>PCL:YV Factor 2</b>	0.001	.000	1.665	.089	.097
<b>Num. Sub. Dep.</b>	0.000	.001	0.259	.012	.796
<b>Age</b>	0.001	.001	2.183	.106	.030
<b>IQ</b>	0.000	.000	1.306	.061	.193
<b>Avg. FA</b>	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*

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

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

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

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

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

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

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

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

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

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

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

<b>Predictors</b>	<b>B</b>	<b>SE B</b>	<b>t</b>	<b><math>\beta</math></b>	<b>Sig.</b>
<b>PCL:YV Facet 1</b>	-0.001	.000	-2.004	-.102	.046
<b>PCL:YV Facet 2</b>	-0.001	.001	-0.903	-.049	.367
<b>PCL:YV Facet 3</b>	0.001	.001	1.918	.100	.056
<b>PCL:YV Facet 4</b>	0.000	.001	-0.021	-.001	.983
<b>Num. Sub. Dep.</b>	0.000	.001	0.325	.015	.745
<b>Age</b>	0.001	.001	2.197	.108	.029
<b>IQ</b>	0.000	.000	1.409	.066	.160
<b>Avg. FA</b>	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 uncinata fasciculus):  $R^2 = .684$ ,  $R = .827$ ,  $F(253) = 66.271$ ,  $p < .001$ ; Regression 18 (predicting FA values in the R uncinata 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*

<i>Model 1</i>					
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
<i>Model 2</i>					
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

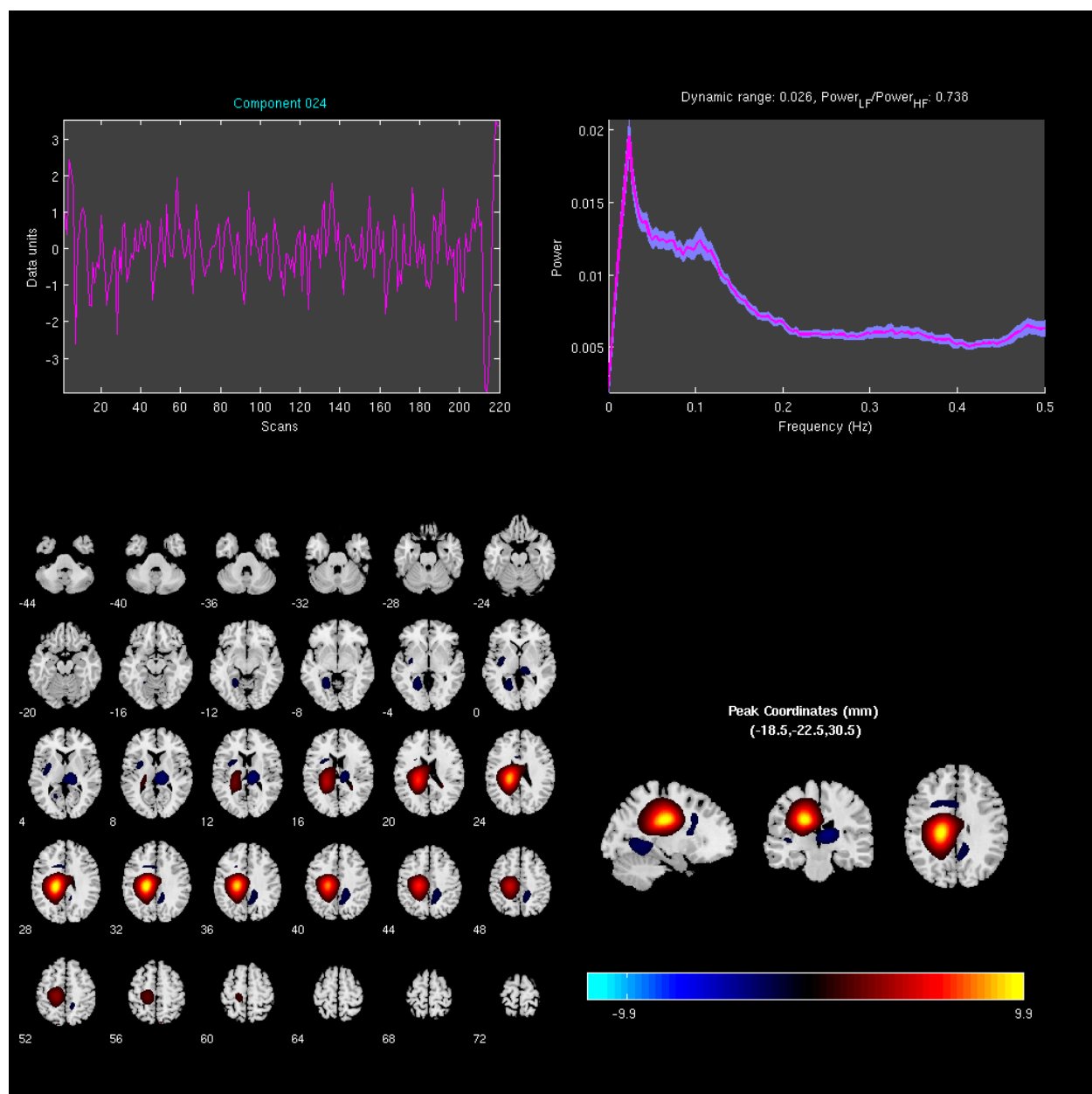
*Cox Proportional Hazard Regressions predicting time to rearrest with CST FA and Covariates*

<i>Model 1</i>					
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
<i>Model 2</i>					
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
<b>PCL:YV factor interaction</b>	0.001	0.002	.541	1.001	0.998 – 1.004
<b>Num. Sub. Dep.</b>	0.214	0.077	.005	1.238	1.065 – 1.440
<b>Release Age</b>	0.072	0.072	.320	1.074	0.933 – 1.238

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

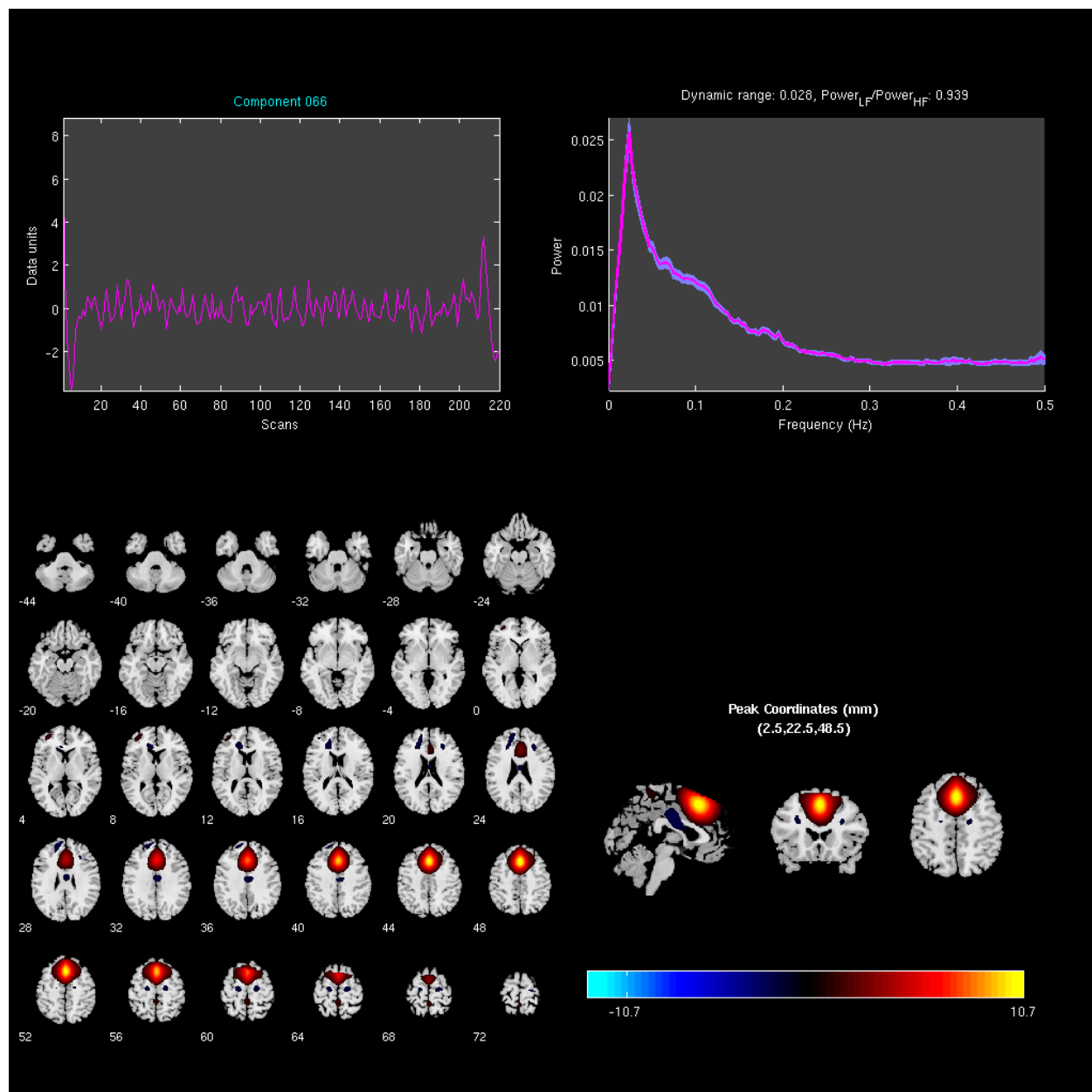
*Model 2:* FA values from the right corticospinal tract entered with covariate measures to predict time to rearrest: -2 Log Likelihood: 804.240, Overall model:  $X^2 = 16.619$ ,  $p = .002$ .

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



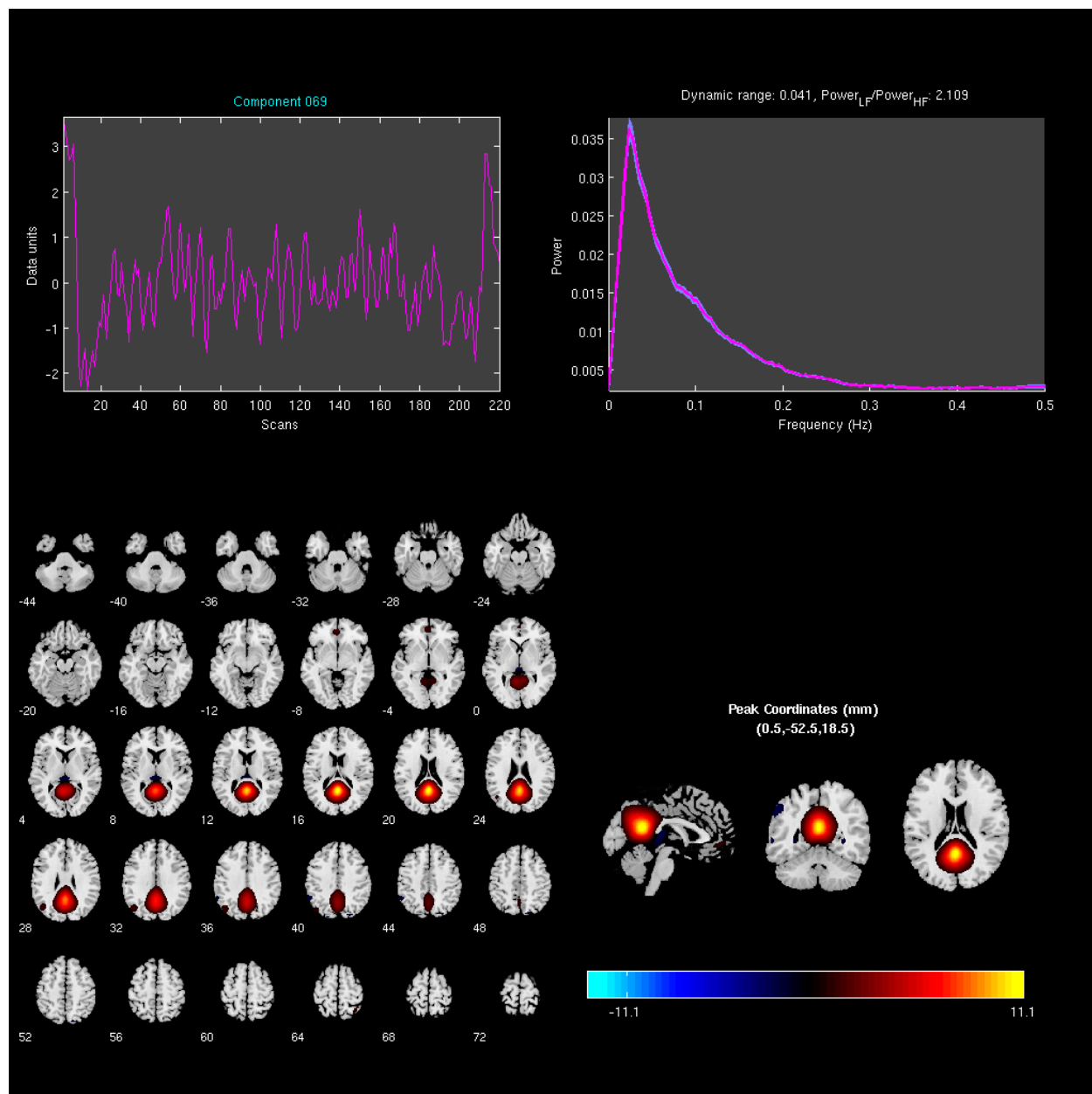
*Figure 1.* Regions included in Component 24 include the precuneus, superior parietal lobule, postcentral gyrus, paracentral lobule, and cingulate gyrus. Via independent-samples *t*-tests, youth scoring higher on the PCL:YV ( $n = 67$ ) exhibited increased functional connectivity within brain regions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

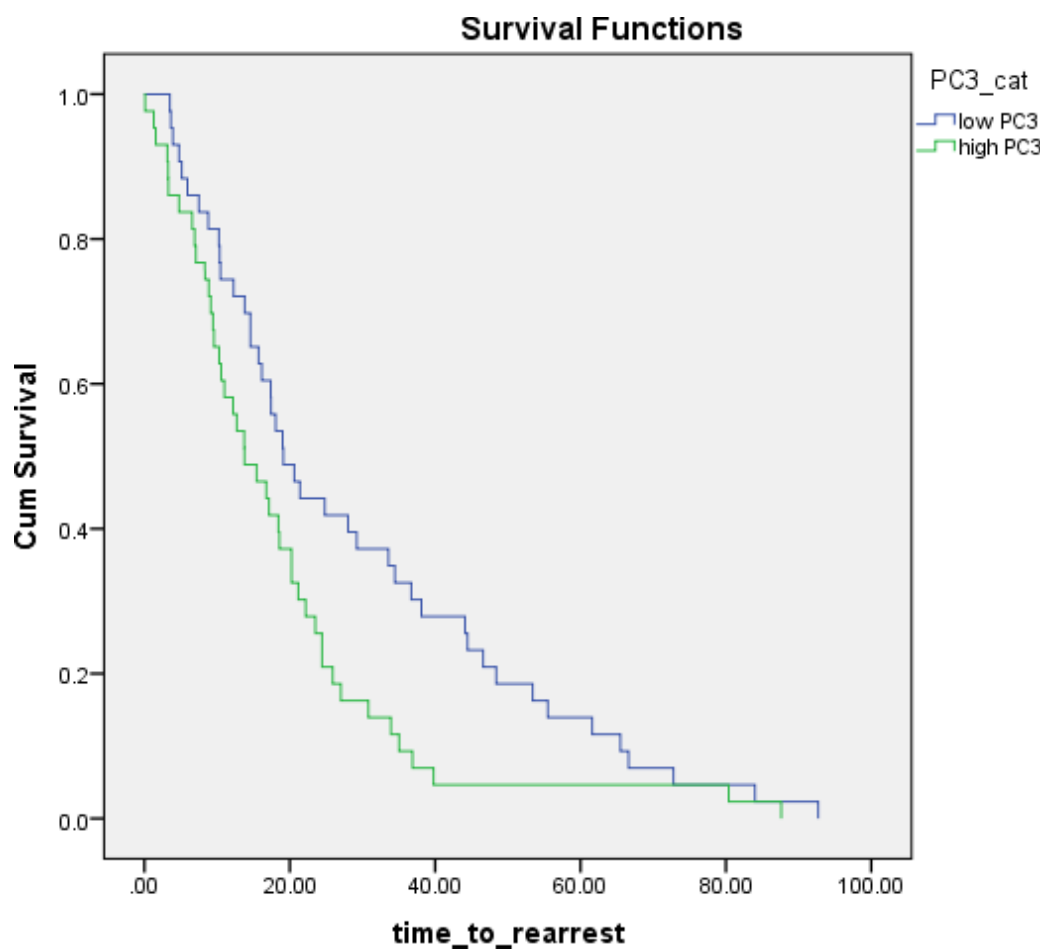
**Figure 3** – Component #69 obtained through Group Independent Component Analysis



*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 ( $n = 67$ ).

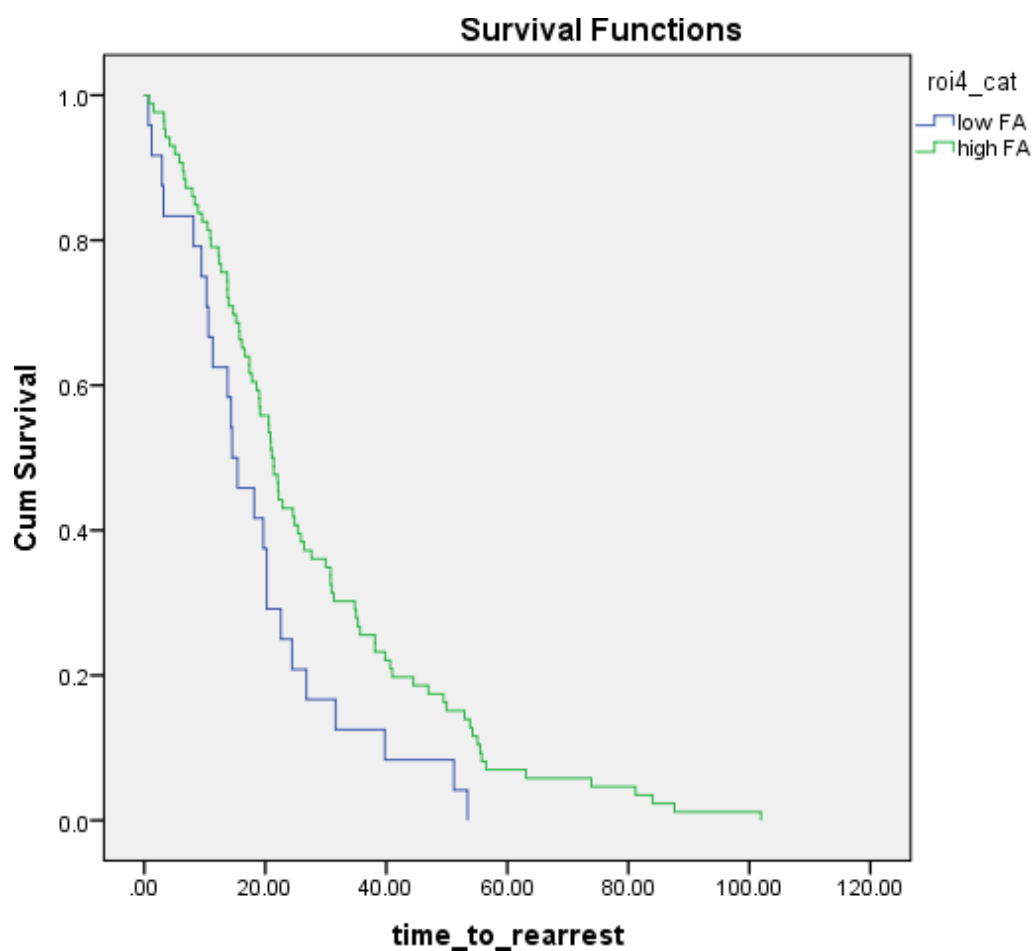


Figure 4



*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.