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INVESTIGATING ERROR-RELATED PROCESSING IN INCARCERATED ADOLESCENTS WITH ELEVATED PSYCHOPATHIC TRAITS

by

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THESIS

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ABSTRACT

Adult psychopathic offenders show an increased propensity towards violence, impulsivity, and recidivism. For a subsample of youth with elevated psychopathic traits, the disorder appears to remain stable throughout development. Such youth represent a particularly severe subgroup characterized by their extreme behavioral problems and comparable neurocognitive deficits as their adult counterparts, including perseveration deficits. Here, we investigated error-related processing using two distinct neuroimaging methodologies in response-locked event-related potentials (ERPs) and functional magnetic resonance imaging (fMRI) in two samples of incarcerated juvenile male offenders who performed a response inhibition Go/NoGo task. Adolescent psychopathic traits were assessed using the Hare Psychopathy Checklist: Youth Version (PCL:YV). In Study 1, PCL:YV scores were unrelated to the error-related negativity (ERN/Ne) amplitude linked to early error-monitoring processes, but were negatively related to error-related positivity (Pe) amplitude, reflecting later stages of error-related processing. In Study 2, PCL:YV scores were negatively associated with hemodynamic

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activity in subregions encompassing the basal ganglia, including the caudate, nucleus accumbens, globus pallidus, subthalamic nucleus, and substantia nigra during error-related processing. These two studies support the attentional bottleneck theory, whereby adolescents with elevated psychopathic traits devote attentional resources to early error-related processing, but exhibit a specific deficit in allocating attentional resources to further processing of error-related information, including the motivational significance of such information to ongoing behavior. These two studies provide the first evidence to suggest that youth with elevated psychopathic traits do not process error-related information effectively, which could potentially help explain this population's increased propensity towards negative outcomes including recidivism.

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BACKGROUND AND SIGNIFICANCE

Psychopathy is a multifaceted personality disorder characterized by interpersonal, affective, lifestyle, and behavioral dysfunction (Hare, 2003). Psychopaths are typically described by their dearth of moral emotions, and their impulsive, irresponsible lifestyle which significantly increases the likelihood of incarceration (Hemphill, Hare, & Wong, 1998). Only 0.5 – 1% of the general population meets the established diagnostic criteria for psychopathic personality (Hare, 2003), though the base rate increases significantly in incarcerated settings, with 15 – 25% of incarcerated offenders meeting the criteria for psychopathy (Hare, 2003). While only comprising a minority of the general population, psychopaths are responsible for a disproportionate amount of criminal offenses, yielding an estimated financial burden of $450 billion annually (Kiehl & Hoffman, 2011). Additionally, psychopaths are at least four times more likely to recidivate, particularly violently, in the twelve months following institutional release compared to non-psychopathic offenders (Rice & Harris, 1997).

The criteria for psychopathy was initially influenced by early physician's conceptions of related conditions. Most notably, Phillipe Pinel coined the phrase "*manie sans delire*", or "*insanity without delirium*" to describe an individual who engaged in antisocial behavior, without any traditional symptomatology common to psychosis (Pinel, 1800). The Italian criminologist Cesare Lombroso later developed the idea of a *born criminal*, suggesting that there that there may be potential neurobiological differences related to these specific individuals (Lombroso, 1911). The term psychopathy itself was first used by Julius Koch, although not in the

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modern sense of the definition. Koch, a German psychiatrist, developed the concept of *personality disorders*, calling them "*psychopathic inferiorities*" (Koch, 1889). Emil Kraeplin was the first to use the term psychopathy in the modern sense of the definition, in the 1904 edition of his classic psychiatry textbook. Here, he included a full section devoted towards psychopathic personality, referring exclusively to individuals characterized by antisocial, manipulative, impulsive, and aggressive traits (Kraepelin, 1909).

The modern study of psychopathic personality was first pioneered by the American psychiatrist Hervey Cleckley. Cleckley published *The Mask of Sanity* in 1941, in which he described his experience working in psychiatric hospitals. Here, he encountered and interacted with many individuals with elevated psychopathic traits (Cleckley, 1941). The current conception of a psychopath is largely based on the profiles Cleckley described in *The Mask of Sanity* (Cleckley, 1941). His work remains largely influential, directly motivating 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). The PCL-R contains twenty items measuring psychopathic traits, including: (1) glibness and superficial charm, (2) grandiose sense of self-worth, (3) need for stimulation and proneness to boredom, (4) pathological lying, (5) conning and manipulative behavior, (6) lack or remorse or guilt, (7) shallow affect, (8) callousness and/or lack of empathy, (9) parasitic lifestyle, (10) poor behavioral controls, (11) promiscuous sexual behavior, (12) early behavioral problems, (13) lack of realistic, long-term goals, (14) impulsivity, (15) irresponsibility, (16) failure to accept responsibility, (17) many short-term marital relationships, (18) juvenile delinquency, (19) revocation of conditional release, and (20) criminal versatility (Hare, 2003).

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The PCL-R has been established as the gold standard for the assessment of psychopathic traits across a variety of populations, including incarcerated samples, forensic and psychiatric patients, and substance users (Hare, 1996, 2003; McDermott et al., 2000). Each of the twenty items on the PCL-R can be scored based on the following criteria: a score of 0 reflecting *the item does not apply to the individual*, a score of 1 implying *the item applies somewhat to the individual*, or a score of 2 meaning *the item definitely applies to the individual*. Scores can then range from 0 to 40, with a score of 30 or above reflecting an individual who meets the diagnostic criteria for psychopathy (Hare, 2003). The mean PCL-R score in incarcerated settings is a 22 (Hare, 2003), whereas mean PCL-R scores are typically quite low in community samples, with individuals typically scoring under 3 (Neumann & Hare, 2008).

Factor analyses of the PCL-R reveal two factors of psychopathic traits (Harpur, Hare, & Hakstian, 1989). Factor 1 is composed of interpersonal and affective traits (items 1, 2, 4, 5, 6, 7, 8, and 16), whereas Factor 2 is composed of lifestyle and antisocial traits (items 3, 9, 10, 13, 14, 15, 18, 19, and 20) (Harpur et al., 1989). Note, PCL-R items 11 and 17 do not load onto either factor (Harpur et al., 1989). A four-facet model of psychopathic traits has also been developed, with Facet 1 reflecting interpersonal traits (items 1, 2, 4, and 5), Facet 2 reflecting affective traits (items 6, 7, 8, and 16), Facet 3 reflecting lifestyle traits (items 3, 9, 13, 14, and 15), and Facet 4 reflecting antisocial traits (items 10, 12, 18, 19, and 20). Again, items 11 and 17 do not load onto any of the four facets (Hare & Neumann, 2006). Others refer to a three-facet model of psychopathic traits, which rids of the items reflecting antisocial tendencies (Cooke & Michie, 2001). Using confirmatory factor analysis, the three-facet model of psychopathic traits identified the following factors: (1) arrogant and deceitful interpersonal lifestyle, (2) deficient affective experience, and (3) impulsive and irresponsible behavior (Cooke & Michie, 2001). Some remain

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apprehensive over the inclusion of an antisocial facet 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-facet model of psychopathic traits has been criticized due to severe statistical issues, such as resulting in impossible parameters, including negative variances (Hare & Neumann, 2010). As such, studies have typically incorporated either PCL-R Total Score, or the two-factor of four-facet models into subsequent analyses.

PCL-R Factor 2 traits are related to the Diagnostic and Statistical Manual of Mental Disorders (DSM-V) Axis II disorder Antisocial Personality Disorder (APD) (American Psychiatric Association, 2013). However, APD and psychopathy, while sharing many similarities, do not measure the same construct. APD criteria is often met by 80 – 90% of incarcerated offenders in maximum-security prisons, compared to the 15 – 25% meeting criteria for psychopathy (Hart & Hare, 1989). This difference is due to the strict reliance of antisocial behaviors when diagnosing APD, while ignoring the affective and interpersonal dysfunction central to the diagnostic criteria of psychopathy.

A number of theories have been proposed to potentially explain the deficits exhibited by psychopathic individuals. David Lykken (1995) proposed the *low-fear hypothesis*, whereby psychopaths show an innate deficiency in fear-responding (Lykken, 1995). Supporting this theory, psychopathic offenders typically 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). Joseph Newman proposed an alternative theory labeled the *response modulation hypothesis*, suggesting that the psychopath's fear conditioning and emotional dysfunction reflect a failure to process affective, inhibitory, or otherwise

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potentially important information when it is peripheral to their ongoing goal-directed behavior (Newman & Lorenz, 2003). Supporting this hypothesis, a number of studies have shown that those with elevated psychopathic traits can engage in normal fear conditioning or emotional processing when specifically directed to the stimuli of interest (Anderson & Stanford, 2012; Anderson et al., submitted; 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).

Additionally, a number of neurobiological models have been proposed to explain the deficits central to psychopathic personality. James Blair described affective deficits central to psychopathy due to dysfunctional amygdala and orbitofrontal cortex (OFC) functioning (Blair, 2003, 2006, 2007). Kent Kiehl later proposed the *paralimbic dysfunction hypothesis of psychopathy*, based on a number of studies from neuroimaging, patient, and brain lesion reports (Kiehl, 2006). In addition to the amygdala and OFC dysfunction Blair proposed, Kiehl postulated psychopaths exhibit dysfunctional processing in a number of paralimbic and surrounding regions, including the anterior superior temporal gyrus, anterior cingulate cortex (ACC), posterior cingulate cortex (PCC) insula, and parahippocampal gyrus (Kiehl, 2006). Subsequently, a number of neuroimaging studies have confirmed Kiehl's initial hypothesis, showing dysfunction in a number of paralimbic and surrounding regions in those with elevated psychopathic traits (Boccardi et al., 2011; de Oliveira-Souza et al., 2008; Ermer, Cope, Nyalakanti, Calhoun, & Kiehl, 2012; Juárez, Kiehl, & Calhoun, 2013; Tiihonen et al., 2008; Yang & Raine, 2009).

Despite intense interest in understanding the neurobiological underpinnings of psychopathic personality, treatment intervention approaches have often proven ineffective for

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this disconcerting population (Rice & Harris, 1997). Younger samples with extreme behavioral problems may prove more amenable to treatment compared to adults, as personality traits are still in nascent stages of development. For example, youth with externalizing disorders have shown improvement in self-regulation through cognitive-behavioral therapy (Woltering, Granic, Lamm, & Lewis, 2011). Furthermore, despite the ineffectiveness of treatment in adult psychopathic offenders, optimism exists that juveniles with elevated psychopathic traits can be treated successfully based on studies using an intensive decompression approach (Caldwell, 2011; Caldwell, McCormick, Umstead, & Van Rybroek, 2007). This approach has been shown to reduce recidivism rates and interpersonal and affective psychopathic traits in severely at-risk juvenile samples (Caldwell, 2011). With the advancement of effective treatment for juveniles with elevated psychopathic traits, researchers have attempted to identify abnormalities early in development consistent with adult psychopathic offenders.

UNDERSTANDING PSYCHOPATHY IN YOUTH SAMPLES

Due to the pejorative label of psychopathy, and the poor outcome measures associated with adult offenders, including poor treatment outcomes and increased recidivism rates (Hemphill et al., 1998; Rice & Harris, 1997), researchers often do not refer to youth as 'psychopaths'. Rather, terms such as "youth with elevated psychopathic traits" are preferred to reduce the stigma associated with the term psychopath and also to provide hope that these especially at-risk children can be treated successfully. In order to measure psychopathic traits in youth and adolescent samples, the Hare Psychopathy Checklist: Youth Version (PCL:YV) (Forth, Hart, & Hare, 1990; Forth, Kosson, & Hare, 2003) was developed. The PCL:YV is a downward extension of the PCL-R modified for age appropriateness. Scoring the PCL:YV is

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very similar to the PCL-R, resulting in the same factor and facet structure found in adult samples (Neumann, Kosson, Forth, & Hare, 2006).

Psychopathic traits, at least in low to moderate levels, often reduce naturally in a large proportion of youth samples (Frick, 2009; Lee, Klaver, Hart, Moretti, & Douglas, 2009; Lynam, 1996). However, for a subsample of youth with elevated psychopathic traits, the disorder appears to remain stable throughout development (Blonigen, Hicks, Krueger, Patrick, & Iacono, 2006; Frick et al., 2003; Lynam, 1996; Lynam, Caspi, Moffitt, Loeber, & Stouthamer-Loeber, 2007; Neumann, Wampler, Taylor, Blonigen, & Iacono, 2011; Obradovic, Pardini, Long, & Loeber, 2007). The best evidence of continuity comes from longitudinal research incorporating both self-report and interview-based measures of adolescent psychopathic traits, showing moderate stability from age 13 to 23 (Lynam et al., 2007). Much of the research investigating psychopathic traits in adolescent samples has focused on callous-unemotional (CU) traits, which are distinguished by persistent patterns of antisocial behavior reflecting a blatant disregard for others, lack of empathy, and deficient affect (Frick, 2009). Higher levels of CU traits typically distinguish youth on a life-course persistent trajectory towards severe antisocial behavior (Frick, 2009).

Although CU traits in youth reflect the interpersonal and affective dysfunction characteristic of adult psychopathic offenders, others have viewed lifestyle psychopathic traits as the most important predictor for identifying youth on a life-course persistent trajectory towards severe antisocial behavior (Lynam, 1996). These traits are typically associated with delinquency and antisocial indices (Frick, Bodin, & Barry, 2000; Lynam, 1996). Similar to adult psychopathic offenders, it appears to be the conglomeration of interpersonal, affective, lifestyle, and antisocial traits that help distinguish youth with elevated psychopathic traits from other externalizing

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populations, including youth who meet criteria for Conduct Disorder (CD) or Oppositional Defiant Disorder (ODD) (Neumann et al., 2011). Youth who meet criteria for these latter 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 who strictly meet criteria for externalizing disorders, youth scoring high on measures of psychopathic traits exhibit similar neurocognitive deficits as adult psychopathic offenders, including reduced sensitivity to punishment cues (Vitale et al., 2005), behavioral inhibition (Roussy & Toupin, 2000), passive avoidance learning (Finger et al., 2008), and perspective taking (Cheng, Hung, & Decety, 2012). Furthermore, both structural and functional neuroimaging studies reveal brain abnormalities in youth with elevated psychopathic traits consistent with adult psychopathic offenders as young as fourteen years of age (Budhani & Blair, 2005; Cope, Ermer, Nyalakanti, Calhoun, & Kiehl, 2014; Ermer, Cope, Nyalakanti, Calhoun, & Kiehl, 2013; Harenski, Harenski, & Kiehl, 2014; Lockwood et al., 2013; Marsh et al., 2008). Deficits appear in many of the regions outlined by Kiehl's paralimbic dysfunction hypothesis (Kiehl, 2006), including the OFC (Budhani & Blair, 2005; Ermer et al., 2013), insula (Lockwood et al., 2013), amygdala (Harenski et al., 2014; Marsh et al., 2008), PCC (Ermer et al., 2013), parahippocampal gyrus (Ermer et al., 2013), and ACC (Cope, Ermer, et al., 2014; Ermer et al., 2013; Marsh et al., 2008).

ERROR-RELATED PROCESSING

Targeting specific cognitive deficits characteristic of adult psychopathic offenders may allow researchers to identify a subset of youth with elevated psychopathic traits most receptive to treatment intervention approaches. One such cognitive deficit may involve the processing of error-related information, as youth with elevated psychopathic traits often perseverate during

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experimental learning paradigms, failing to adjust their behavior to meet the demands established by external sources (Finger et al., 2008; Roussy & Toupin, 2000).

The basal ganglia (BG) has been shown to play an important, though indirect, role in error-related processing. The BG is a subcortical relay station located at the base of the forebrain, comprised of the following regions: the dorsal striatum (composed of the caudate and putamen), ventral striatum (composed of the nucleus accumbens [NAcc] and olfactory tubercle), globus pallidus, ventral pallidum, substantia nigra, and subthalamic nucleus.

Dorsal Striatum (DS): The DS is comprised of the caudate and putamen. The caudate appears to play a large role in the generation of voluntary movement, as it has been continually implicated in Parkinson's Disease (Sardar, Czudek, & Reynolds, 1996). Recently, the caudate has been shown to play a role in executive functioning. Specifically, the caudate plays a role in goal-directed action, selecting behavior based on the continually changing values of one's goals, and associating specific actions with specific outcomes (Hollerman, Tremblay, & Schultz, 2000). The caudate is also involved in the shifting of attentional sets (Middleton & Strick, 2000; Monchi, Petrides, Strafella, Worsley, & Doyon, 2006; Ravizza & Ciranni, 2002). Damage to the caudate has been shown to reduce drive in humans, increasing inattentiveness and stimulus-bound perseverative behavior (Nys, van Zandvoort, van der Worp, Kappelle, & de Haan, 2006). The caudate has also been shown to be active during a variety of response inhibition tasks (Braet et al., 2011; Menon, Adleman, White, Glover, & Reiss, 2001; Verney, Brown, Frank, & Paulus, 2003). Poorer response inhibition, resulting in reduced behavioral performance such as increased error rates, may then be associated with reduced hemodynamic activity in the caudate. To date, seven studies have been performed investigating the role between the caudate and psychopathic personality, including structural magnetic resonance imaging (sMRI) studies and functional

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magnetic resonance imaging (fMRI) studies investigating reward processing (Boccardi et al., 2013; Cope et al., 2012; Cope, Vincent, et al., 2014; Finger et al., 2011; Finger et al., 2008; Schiffer et al., 2011; Yoder, Harenski, Kiehl, & Decety, 2015).

Also comprising the DS is the putamen, which plays a significant role in regulating movements and learning, particularly reinforcement learning (Haruno & Kawato, 2006). The putamen appears to specifically be involved in selecting actions in order to maximize outcomes. Lesions to the putamen typically result in deficits in category-based learning (Ell, Marchant, & Ivry, 2006). Nine studies have investigated the relationship between the putamen and psychopathic personality, typically with sMRI, but also fMRI studies investigating reward processing and drug-craving (Boccardi et al., 2013; Cope et al., 2012; Cope, Vincent, et al., 2014; de Oliveira-Souza et al., 2008; Decety, Chen, Harenski, & Kiehl, 2013; Glenn, Raine, Yaralian, & Yang, 2010; Marsh et al., 2013; Viera et al., in press; Yang et al., 2015).

Ventral Striatum (VS): The VS is comprised of the NAcc and olfactory tubercle. The NAcc plays a significant role in drug addiction, as activity within the NAcc is correlated with the euphoria associated with substance use, and plays a significant role in rewarding experiences in general (Knutson, Adams, Fong, & Hommer, 2001). Specifically, the NAcc plays a noteworthy role in processing information that is both rewarding and reinforcing (Dalley et al., 2007). Four studies have investigated the relationship between the NAcc and psychopathic personality, with fMRI studies investigating drug craving (Boccardi et al., 2013; Buckholtz et al., 2010; Cope, Vincent, et al., 2014; Schiffer et al., 2011).

Olfactory Tubercle (OF): The OF plays a similar role as the NAcc in processing rewarding and reinforcing information, but plays a role mainly in the gathering of sensory information from olfactory receptors (Wesson & Wilson, 2010). Deficits within the olfactory

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tubercle have been observed in a number of psychiatric disorders, including schizophrenia (Hurwitz, Kopala, Clark, & Jones, 1988). To date, the olfactory tubercle has never been investigated in relation to psychopathic personality.

Globus Pallidus (GP): The GP plays a similar role as regions that comprise the dorsal striatum, as it is involved in the regulation of voluntary movement. However, the GP appears to specifically play a role in movements that occur at a subconscious level (Liu et al., 2008). Damage to the GP results in a number of different types of movement disorders. The GP receives input from the dorsal striatum, and information is then projected towards the thalamus. Three studies have previously investigated the relationship between GP activity and psychopathic personality, with fMRI studies investigating reward processing and drug-craving (Cope, Vincent, et al., 2014; Decety et al., 2013; Glenn et al., 2010).

Ventral Pallidum (VP): The VP receives dopaminergic projections from the ventral tegmental area (VTA), and plays a prominent role in substance use addiction (Frankel, Alburges, Bush, Hanson, & Kish, 2008). Specifically, the use of addictive drugs facilitates the release of dopamine in the VP (Pierce & Kumaresan, 2006). To date, the ventral pallidum has never been investigated in relation to psychopathic personality.

Substantia Nigra (SN): The SN plays an important role in reward processing, addiction, and movement. Specifically, the pars reticulata of the SN plays an important role in processing information. Here, it sends processed information to the thalamus and superior colliculus. The pars compacta of the SN is involved in learning responses to stimuli. In primates, dopamine activity increases in the substantia nigra when a new stimulus is presented, and decreases through repeated presentations, through habituation (Ljungberg & Apicella, 1992). Behaviorally significant stimulus presentations continually activate dopaminergic neurons in the substantia

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nigra. To date, the substantia nigra has never been investigated in relation to psychopathic personality.

Subthalamic Nucleus (STN): The STN is typically involved in action selection processes. Specifically, the STN has been shown to contribute to the phenomenon of post-error slowing (PES) (Cavanagh, Sanguinetti, Allen, Sherman, & Frank, 2014; Siegert et al., 2014), described as the behavioral slowing that occurs after one commits an incorrect response, in order to prevent future errors from occurring (Rabbit, 1981). Additionally, damage to the STN has been shown to increase impulsivity in individuals when shown two equally competing rewarding stimuli (Uslaner & Robinson, 2006). To date, the STN has never been investigated in relation to psychopathic personality.

The BG as a whole is involved in processing information, cognition, and movement (Gehring, Goss, Coles, Meyer, & Donchin, 1993; Mathalon, Whitfield, & Ford, 2003). Additionally, the BG is involved in error-monitoring and error-processing indirectly, playing a significant role in error-detection and correction. Error commission is tightly monitored in the BG and plays a significant role in cognitive processing (Holroyd & Coles, 2002). Specifically, an increase in error commission is associated with a subsequent reduction in cognitive functioning (Welcome, Razvodovsky, Pereverzeva, & Pereverzev, 2010). The BG communicates with other brain regions and monitors error-detection and correction processes (Carter et al., 1998; Garavan, Ross, Kaufman, & Stein, 2003; Holroyd & Yeung, 2003). The functions of this error-monitoring system appear to be dependent upon dopaminergic transmission (De Bruijn, Sabbe, Hulstijn, Ruigt, & Verkes, 2006), though see evidence to the contrary (Larson, Clayson, Primosch, Leyton, & Steffensen, in press).

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In experimental paradigms, errors are frequently made as a result of incorrect behavioral responses (Stevens, Kiehl, Pearlson, & Calhoun, 2009). An error, simply defined, is a deviation from set goals (Welcome et al., 2010). The BG plays an important role in determining whether the end result of events will be favorable or not (Holroyd & Coles, 2002). The BG monitors and steadily predicts the result of ongoing events (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Mathalon et al., 2003). The BG is one of the many brain regions that sends command information to the ACC for further processing. One of these commands sent by the BG is for the generation of the error-related negativity event-related potential (ERP) component. This ERP component was discovered at roughly the same time by two independent laboratories, with Falkenstein and colleagues (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991) referring to this component as the "Ne", whereas Gehring and colleagues (Gehring, Coles, Meyer, & Donchin, 1990) referring to this component as the "ERN". To give credit to both discoveries, we will refer to this early error-related ERP component as the "ERN/Ne" throughout the remainder of the manuscript. The ERN/Ne reflects a reinforcement learning signal sent by phasic dips in mesencephalic dopamine activity to the ACC, which then updates the correct response selection. The ACC is believed to integrate this reinforcement history over time, in order to optimize the correct response selection (Kennerley, Walton, Behrens, Buckley, & Rushworth, 2006). Support for this theory comes from various neurological conditions associated with dopaminergic dysfunction, primarily Parkinson's and Huntington's Disease, which are associated with reduced ERN/Ne amplitude (Beste et al., 2008; Stemmer, Segalowitz, Dywan, Panisset, & Melmed, 2007). Additionally, ERN/Ne amplitude has been shown to increase in amplitude with the administration of dopaminergic agonist medications, including methylphenidate (Ritalin) (de Bruijn, Hulstijn, Verkes, Ruigt, & Sabbe, 2004; Groom et al., 2013).

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Researchers have typically investigated two ERPs in relation to error-related processing: the ERN/Ne defined above and the error-related positivity (Pe). Though closely related temporally, the ERN/Ne and Pe are believed to reflect distinct stages of error-related processing. Specifically, the ERN/Ne, which has a fronto-central distribution on the scalp, occurs 50 – 150 milliseconds (ms) after an erroneous response, and reflects initial, automatic error-detection and action monitoring processes (Falkenstein, 2004; Gehring et al., 1993). On the other hand, the Pe, which has a centro-parietal distribution, occurs around 200 – 500 ms after an incorrect response, and is involved in later, more elaborate error-processing stages, including the motivational (Ullsperger, Harsay, Wessel, & Ridderinkhof, 2010) or affective appraisal (Overbeek, Nieuwenhuis, & Ridderinkhof, 2005) of error-related information. Whereas the ERN/Ne is present whether or not the participant was consciously aware of the error's occurrence, the Pe is only present when participant were consciously aware of the error's occurrence (Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001). Additionally, source localization (Dehaene, Posner, & Tucker, 1994; Herrmann, Rommler, Ehlis, Heidrich, & Fallgatter, 2004) and fMRI (Edwards, Calhoun, & Kiehl, 2012; van Veen & Carter, 2002) studies converge on the ACC as the generator for both the ERN/Ne and Pe, albeit separate subregions. The ERN/Ne is said to arise within the cognitive, caudal division of the ACC (cACC), whereas both the caudal and the affective, rostral division of the ACC (rACC) contribute to the Pe amplitude (Edwards et al., 2012; van Veen & Carter, 2002).

While the ERN/Ne has been clearly linked to processes generated by the BG, the Pe's association with the BG has not been well established. Studies suggest that the ERN/Ne and Pe are dependent upon entirely independent neurotransmitter systems, as neurological conditions and drug administration affecting dopaminergic transmission have had differential effects on the

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ERN/Ne and Pe amplitude (de Bruijn et al., 2004). However, two studies performed with children who met criteria for attention deficit hyperactivity disorder (ADHD) have shown that the administration of methylphenidate (Ritalin), a dopamine agonist, increased both the ERN/Ne and Pe amplitude (Groom et al., 2013; Jonkman, van Melis, Kemner, & Markus, 2007).

Several studies have found comparable ERN/Ne amplitude between adult psychopathic offenders and control participants when using affectively neutral stimuli (Brazil et al., 2009; Brazil et al., 2011; Maurer et al., 2016; Munro et al., 2007; Steele, Maurer, Bernat, Calhoun, & Kiehl, 2016; von Borries et al., 2010). However, reduced ERN/Ne amplitude has been observed in adult psychopathic offenders when incorporating evocative angry or fearful facial stimuli (Munro et al., 2007). Compared to the ERN/Ne, disparate findings have been observed in the Pe amplitude in relation to psychopathic personality. Two previous reports, one with adult males (Brazil et al., 2009) and another with adult females (Maurer et al., 2016) associated reduced Pe amplitude with increased psychopathy scores. However, a recent report found increased Pe amplitude in incarcerated males with elevated psychopathic traits compared to incarcerated offenders with low levels of psychopathic traits (Steele, Maurer, et al., 2016).

Despite the interest in the electrophysiological correlates of error-related processing in adult psychopathic offenders, such processes have never been investigated in youth with elevated psychopathic traits. Here, we address this issue by reporting on error-related electrophysiological indices using ERPs and a response inhibition experimental paradigm in a sample of incarcerated male adolescent offenders. Furthermore, to our knowledge, fMRI studies have never been performed investigating error-related processing in either youth or adults with elevated psychopathic traits. By investigating error-related processing using two different neuroimaging methodologies in ERPs and fMRI, this will extend our understanding of unique cognitive deficits

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associated with youth with elevated psychopathic traits. By understanding these processes to a further degree, we may be able to develop specialized treatment intervention approaches aimed at reducing recidivism and improving treatment retention with this at-risk sample.

STUDY 1: Investigating error-related processing in incarcerated adolescents with elevated psychopathic traits using ERPs

INTRODUCTION

Based on previous error-related ERP studies performed with adult psychopathic offenders (Brazil et al., 2009; Brazil et al., 2011; Maurer et al., 2016; Munro et al., 2007; Steele, Maurer, et al., 2016; von Borries et al., 2010), we hypothesized adolescent psychopathy scores would be unrelated to early, action-monitoring processes, as indexed by intact ERN/Ne amplitude. In addition, we hypothesized adolescent psychopathy scores would be negatively related to Pe amplitude, consistent with two previous studies with adult psychopathic offenders (Brazil et al., 2009; Maurer et al., 2016), but inconsistent with a recent report with adult psychopathic offenders (Steele, Maurer, et al., 2016) who exhibited *increased* Pe amplitude. An increased Pe amplitude in adult male psychopathic offenders may result from a compensatory mechanism, attempting to overcome initial post-error processing deficits experienced early in development. In addition to the use of traditional time-domain ERP analyses, we incorporated an approach based on principal component analysis (PCA), which provides a robust decomposition of the overlapping variance both between and within ERP components (Bernat, Williams, & Gehring, 2005; Dien, Khoe, & Mangun, 2007). This approach has been incorporated in a number of reports from our laboratory (Anderson, Steele, Maurer, Bernat, & Kiehl, 2015; Fink et al., in press; Maurer et al., 2016; Steele et al., 2015; Steele, Fink, et al., 2014; Steele, Maurer, et al., 2016), providing a more sensitive and predictive measure compared to traditional time-domain

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ERP analyses. In regards to PCA analyses, we hypothesized that adolescent psychopathy scores would be negatively related to a middle subcomponent underlying the Pe, as this has been shown to be dysfunctional in previous reports (Maurer et al., 2016; Steele, Maurer, et al., 2016).

METHOD

PARTICIPANTS

Participants included 100 incarcerated adolescents at a maximum-security juvenile detention center who participated in a larger overall study (Southwest Advanced Neuroimaging Cohort – Youth (SWANC-Y)), ranging from 16 to 20 years of age (M = 17.38, SD = 0.86) at the time of electroencephalography (EEG) collection. The sample was predominantly right-handed (7% reported being left-hand dominant). Participants were predominantly Hispanic/Latino (76%), with the remaining self-identifying as Black/African American (12%), White (10%), or more than one ethnic category (2%).

Incarcerated adolescents are considered to be a vulnerable population for research, so extra precautions were taken in order to minimize the potential for coercive influences that could reduce one's ability to provide voluntary consent to participate (Edens, Epstein, Stiles, & Poythress, 2011; Gostin, Vanchieri, & Pope, 2007). For example, potential study participants may feel inclined to participate in research in order to interact with people from outside the correctional facilities (Edens et al., 2011). With the issue of coercion in mind, we did our best to ensure that study participants did not feel coerced in any way to participate. Accordingly, our recruitment procedure was as follows: Initial contact was made with potential study participants through announcements made at the correctional facility by trained research staff (not correctional staff). Meetings were then scheduled with interested participants, providing them the opportunity to make an informed choice about participating. Participants 18 years of age or older

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provided written informed consent and participants younger than 18 years of age provided written informed assent in conjunction with parent/guardian written informed consent. Participants were informed of their right to terminate participation at any point, the lack of direct institutional benefits, and that their participation would not affect their facility status or release. Participants also received remuneration at the hourly labor wage of the facility. The University of New Mexico Health Science Center Human Research Review Committee and the Office of the Human Research Protections approved all procedures.

ASSESSMENTS

Psychopathic traits were assessed by trained research assistants, graduate students, and postdoctoral researchers using the PCL:YV (Forth et al., 2003). In addition to PCL:YV Total Score, we investigated the two-factor and four-facet models for identification of specific adolescent psychopathic traits associated with electrophysiological error-related indices. The mean PCL:YV Total Score for the sample was 23.83 (SD = 9.46). The mean Factor 1 score was 6.75 (SD = 3.19) and the mean Factor 2 score was 12.78 (SD = 3.20). PCL:YV Factor 1 and 2 scores were significantly correlated ($r = .58$, $p < .001$), consistent with previous reports (Harpur et al., 1989). See Table 1 for the remainder of correlations and Table 2 for the remainder of descriptive statistics.

In addition to psychopathic traits, additional assessments were administered to assess intelligence quotient (IQ), substance dependence, mental illness, and traumatic brain injury (TBI) by trained research staff. Exclusionary criteria included: a full-scale IQ less than 70, a TBI accompanied with a significant loss of consciousness, poor behavioral performance or significant movement during data collection, or personal and/or familial history of bipolar or psychotic disorders. Participants were also excluded for meeting criteria for mood disorders, including

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major depression and anxiety disorders, including post-traumatic stress disorder (PTSD), due to the important role these disorders play for both the generation of the ERN/Ne (Chiu & Deldin, 2007; Olvet & Hajcak, 2008) and Pe (Bridwell, Steele, Maurer, Kiehl, & Calhoun, 2015) amplitude. Additionally, participants were excluded for making fewer than four errors. 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).

We included the following covariates in subsequent analyses: age, number of substance dependencies, and IQ. Mental illness and substance dependence were assessed using the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS) (Kaufman, Birmaher, & Brent, 1997). Number of substance dependencies were calculated by summing the total number of substances (both alcohol and drug) for which participants met lifetime dependence diagnoses (range 0 – 9, $M = 2.33$, $SD = 1.68$), as psychopathic traits are often comorbid with substance use (Edwards, Harenski, Maurer, & Kiehl, submitted; Smith & Newman, 1990; Walsh, Allen, & Kosson, 2007). Full-scale IQ was estimated using the Vocabulary and Matrix Reasoning sub-tests of the Wechsler Adult Intelligence Scale – 3rd Edition (WAIS-III) (Wechsler, 1997) for participants older than eighteen years of age, and from the Wechsler Intelligence Scale for Children – 4th Edition (WISC-IV) (Wechsler, 2003) for participants younger than eighteen years of age ($M = 93.90$, $SD = 10.93$); IQ scores were unavailable for $n = 9$ participants included in subsequent analyses.

STIMULI AND TASK

EEG data were collected in a quiet, dimly lit room, reserved for EEG data collection at the correctional facility, separate from the general population housing. After placement of electrodes, participants were seated in a comfortable chair 60 cm away from a computer monitor

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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, & Hopfinger, 2000), consisting of two experimental runs, each comprising 245 visual stimuli. After the first run, participants were afforded the opportunity to take a break in order to reduce fatigue. Stimuli were presented to participants through the computer-controlled Neurobehavioral Systems Inc. visual presentation software package, Presentation. Each stimulus appeared for 250 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 on a computer keyboard every time the target Go stimulus (a white "X") appeared and to withhold a response whenever the distracter NoGo stimulus (a white "K") appeared. The stimuli subtended approximately 3 x 5 visual degrees against a black background. Targets appeared with higher frequency (84%, 412 trials, with 206 on each run) than distracters (16%, 78 trials, with 39 on each run) to establish a strong stimulus-response mapping on Go trials. Two "K's" 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 to the constraint that three Go stimuli were presented within each 6 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 8 to 14 seconds. Hits were defined as successful responses to Go stimuli, whereas False Alarms (FA's) were defined as incorrect responses to NoGo stimuli. Prior to recording, each participant performed a block of ten practice trials in order to ensure that task instructions were clearly understood.

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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 collected with the BioSemi software were low-pass filtered using a fifth-order sinc filter with a half-power cut-off 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 point. Six electrodes were additionally placed on the participant's face to measure electrooculogram, above, below, and lateral to the canthus of each eye. All impedances were kept below 10 kΩ.

Analytic Strategy

Pre-processing 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 all other non-ocular channels. These channels were replaced using the mean of surrounding electrodes. ERP epochs were defined relative to the response, from 1000 pre- to 2000 ms post-response. An independent component analysis (ICA) eye-blink removal protocol was also performed. The ICA utility in EEGlab software (Delorme & Makeig, 2004) was used to derive components; then, using an in-house template matching algorithm (Jung, Makeig, Westerfield, Courschesne, & Sejnowski, 2000), blink components were identified and removed from the data.

Classic time-domain response-locked ERP components, relative to FA's were extracted: the ERN/Ne, the mean amplitude of the negative deflection occurring 0 – 100 ms after an

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incorrect behavioral response, and the Pe, the mean amplitude of the positive deflection occurring 94 – 500 ms after an incorrect behavioral response. 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 exceeding ± 100 μV were omitted from analyses. Applying these criteria, 20.72% of response-locked trials were excluded from analyses. An additional data reduction method, PCA, was also performed. Temporal PCA with Varimax rotation was carried out on the covariance matrix from all electrodes to define a four component response-locked solution for FA stimuli that accounted for 94.97% of the variance. One of these principal components reflected the ERN/Ne (PC3), whereas the remaining three principal components reflected early, middle, and late subcomponents underlying the Pe (PC1, PC2, and PC4 respectively). These three separate subcomponents underlying the Pe appear to reflect unique patterns of cognitive processing and hemodynamic activity in subregions of the ACC (Edwards et al., 2012). The ERN/Ne amplitude was maximal at FCz, whereas Pe amplitude was maximal at CPz. Therefore, these electrodes were selected as most representative of the ERN/Ne and Pe related activations and used in subsequent time-domain and PCA analyses. Stepwise linear regression analyses were carried out on all n = 100 participants to predict mean time-domain and PCA decompositions reflecting the ERN/Ne and Pe amplitude using adolescent psychopathy scores and three covariate measures: age, IQ, and number of substance dependencies in order to take full advantage of our large sample size.

RESULTS

BEHAVIORAL RESULTS

Response times (RTs) and frequency for Hits and FA's were analyzed. As expected, participants responded faster to NoGo stimuli (M = 381 ms, SD = 43 ms) compared to Go stimuli

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(*M* = 419 ms, *SD* = 51 ms), *t*(99) = 7.74, *p* < .001. Participants made significantly more errors (FA's) to NoGo stimuli (*M* = 23.76, *SD* = 11.85) compared to Go stimuli (*M* = 12.61, *SD* = 14.65), *t*(99) = 17.89, *p* < .001. There was a main effect for PES (*M* = 28 ms, *SD* = 73 ms), 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 slowing to Go stimuli compared to correct responses to NoGo stimuli (Rabbit, 1981). Participants indeed did respond more slowly after error trials (*M* = 384 ms, *SD* = 84 ms) than after correct trials (*M* = 356 ms, *SD* = 33 ms), *t*(99) = 3.86, *p* < .001. However, PES did not significantly correlate with adolescent psychopathy scores, covariate measures, or time-domain or PCA measures reflecting the ERN/Ne or Pe mean amplitude. Typically, PES is best observed in tasks that have a short response-stimulus interval (RSI) (Danielmeier & Ullsperger, 2011). As our current study and past studies (Brazil et al., 2009; Brazil et al., 2011; Maurer et al., 2016; Munro et al., 2007; Steele, Maurer, et al., 2016; von Borries et al., 2010) had longer RSIs than the optimal RSI for the detection of PES, we may not be able to accurately speak of PES effects.

GROUP DIFFERENCES

The original categorization of adult psychopathy was defined as one standard deviation above the mean PCL-R Total Score (*M* = 22, *SD* = 8), resulting in a standard cutoff score of 30 or above for adult psychopathy (Hare, 2003). Here, we report a similar cutoff score of 30 for our sample, with twenty-one participants meeting the categorical classification for psychopathy (*M* PCL:YV Total Score = 32.03, *SD* = 1.47). Using a quartile split of PCL:YV Total Scores, youth with low levels of psychopathic traits consisted of *n* = 22 participants (*M* PCL:YV Total Score = 14.64, *SD* = 3.53). Groups differed with respect to PCL:YV Total Score *t*(41) = -20.93, *p* < .001,

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and number of substance dependencies, $t(41) = -2.33$, $p = .025$. Groups did not significantly differ with respect to age, $t(41) = -1.01$, $p = .317$ or IQ, $t(35) = 0.13$, $p = .910$. Groups did not significantly differ with respect to ERN/Ne mean amplitude, $t(41) = -0.46$, $p = .649$, PC1 mean amplitude, $t(41) = 0.41$, $p = .586$, PC3 mean amplitude, $t(41) = -1.11$, $p = .273$, or PC4 mean amplitude, $t(41) = 1.67$, $p = .102$. Groups significantly differed with respect to PC2 mean amplitude, with those scoring higher on PCL:YV Total Score exhibiting reduced PC2 mean amplitude, $t(41) = 2.40$, $p = .021$ (see Figure 1). Finally, groups were marginally different with respect to the time-domain Pe mean amplitude, $t(41) = 1.72$, $p = .093$.

TIME-DOMAIN ERP STEPWISE LINEAR REGRESSION ANALYSES

Separate stepwise linear regression analyses were performed to assess unique contributions to mean ERN/Ne and Pe amplitude measured with traditional time-domain ERPs with all $n = 100$ participants. Each of the six regressions performed had an ERP component as the dependent measure (i.e., in three regressions, ERN/Ne mean amplitude was the dependent measure, and the remaining three regressions, Pe mean amplitude was the dependent measure). PCL:YV scores (Regression 1: PCL:YV Total, Regression 2: PCL:YV Facets 1 – 4 (interpersonal, affective, lifestyle, and antisocial traits, respectively), and Regression 3: PCL:YV Factor 1 (interpersonal and affective traits) and Factor 2 (lifestyle and antisocial traits)) and three covariate measures (age, IQ, and number of substance dependencies) were entered as simultaneous predictor variables in the stepwise linear regression analyses performed. PCL:YV Facet scores were measured before Factor scores, as they provide a more precise measure of specific adolescent psychopathic traits (Neumann et al., 2006).

In addition, we implemented a Simes-Hochberg multiple comparison correction to all stepwise linear regression analyses performed (Hochberg, 1988; Simes, 1986) to maintain the

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family wise error rate at an acceptable rate. This correction is in the class of sequential Bonferroni correction methods, which consists of arranging the obtained *p*-values within a family of tests from largest to smallest, and excluding tests on a sequential basis on whether they are associated with a *p*-value that is less than a previously adjusted alpha level. Therefore, all significant results reflect this correction.

After implementation of the Simes-Hochberg correction, neither PCL:YV scores (Total, Facets, or Factor scores) or covariate measures included in analyses (age, IQ, or number of substance dependencies) were significant predictors of the mean ERN/Ne or Pe time-domain ERP components.

PCA ERP STEPWISE LINEAR REGRESSION ANALYSES

Twelve separate stepwise linear regression analyses were also performed to assess the amount of variance in four PCA-derived subcomponents (one measuring ERN/Ne mean amplitude [PC3] and three components measuring early, middle, and late subcomponents of the Pe [PC4, PC2, and PC1, respectively]) explained by adolescent psychopathy scores. Like before, PCL:YV Total, Facet, and Factor scores were the predictor variables of interest in three separate regression models for each of the four PCA subcomponents, along with age, IQ, and number of substance dependencies entered as covariate measures.

After implementation of the Simes-Hochberg multiple comparison procedure, neither adolescent psychopathy scores or covariate measures were significant predictors of PC3 mean amplitude, reflecting the ERN/Ne. Similarly, adolescent psychopathy scores and covariate measures were not significant predictors of PC1 or PC4 mean amplitude, reflecting late and early subcomponents underlying the Pe, respectively. However, elevated adolescent psychopathy scores were negatively related to PC2 mean amplitude, reflecting a middle subcomponent

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underlying the Pe. In separate stepwise linear regression analyses, PCL:YV Total Score (β = -.22, p = .037, d = .45) and PCL:YV Facet 4 scores measuring antisocial traits (β = -.27, p = .011, d = .55) emerged as significant predictors of reduced PC2 mean amplitude (see Table 3). PCL:YV Factor 2 scores measuring lifestyle and antisocial traits emerged as a marginal predictor of reduced PC2 mean amplitude after the implementation of the Simes-Hochberg multiple comparison procedure (p = .039).

SUPPLEMENTARY ANALYSES FOR STUDY 1

The Pe may actually reflect an error-related subcomponent of the P300 ERP component, as both components show a similar topography and latency in response to errors (Arbel & Donchin, 2009; Leuthold & Sommer, 1999; Nieuwenhuis et al., 2001). The Pe may specifically reflect a P300 elicited by rare error trials, but also, involved in post-error response adjustment strategies, including the updating of environmental contexts (Arbel & Donchin, 2009; Leuthold & Sommer, 1999). As such, we performed additional stimulus-locked analyses, investigating the P300 ERP component. Here, the P300 was defined as the positive deflection occurring 340 – 650 ms post-stimulus onset, consistent with previous reports (Bekker, Kenemans, & Verbaten, 2005; Bokura, Yamaguchi, & Kobayashi, 2001; Donkers, Nieuwenhuis, & van Boxtel, 2005; Huster, Westerhausen, Pantev, & Konrad, 2010). Within each trial, individual electrodes in which activity exceeded ± 100 μV were omitted from analyses. Applying these criteria, 19.01% of stimulus-locked trials were excluded from analyses. An additional approach based on PCA was also performed. A five-component stimulus-locked solution was extracted from stimulus-locked NoGo trials which accounted for 92.96% of the variance. The P300 was maximal at Fz; therefore, this electrode was selected as most representative of the P300 and used in subsequent time-domain and PCA analyses. Performing stepwise linear regression analyses, number of

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substance dependencies emerged as a significant predictor of reduced P300 amplitude in both time-domain (β = -.44, p = .014, d = .45) and PCA analyses (β = -.46, p = .010, d = .45). Results are consistent with previous research associating reduced P300 amplitude in individuals who meet criteria for substance dependence (Bauer, 2001; Carlson, Katsanis, Iacono, & Mertz, 1999). Therefore, adolescent psychopathy scores were strictly related to deficits within response-locked ERP analyses, but not within stimulus-locked ERP analyses in the current manuscript.

DISCUSSION FOR STUDY 1

In Study 1, adolescent psychopathy scores were not significantly related to ERN/Ne amplitude, consistent with previous reports performed with adult psychopathic offenders (Brazil et al., 2009; Brazil et al., 2011; Maurer et al., 2016; Munro et al., 2007; Steele, Maurer, et al., 2016; von Borries et al., 2010). However, adolescent psychopathy scores were negatively related to Pe mean amplitude, consistent with two previous reports with adult psychopathic offenders (Brazil et al., 2009; Maurer et al., 2016), but inconsistent with a recent report in which incarcerated adult males exhibited *increased* Pe amplitude compared to controls (Steele, Maurer, et al., 2016). Our results suggest that adolescent psychopathy scores, specifically Facet 4 scores reflecting antisocial adolescent psychopathic traits, are not associated with dysfunctional error-detection or action-monitoring processes (Falkenstein et al., 1991; Gehring et al., 1990; Yeung & Summerfield, 2012), but are associated with specific deficits in post-error processing.

The results obtained in Study 1 were most strongly supported through the use of PCA to separate overlapping variance between and within traditional time-domain ERP components. PCA identified three subcomponents underlying the time-domain Pe component: an early, middle, and late subcomponent. Elevated adolescent psychopathy scores were negatively related to the middle subcomponent underlying the Pe in Principal Component 2. This subcomponent

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has a similar temporal distribution as a subcomponent identified in a previous report associated with cACC hemodynamic activity (Edwards et al., 2012). Reduced Pe amplitude has also been suggested to represent a specific deficit in using information received from errors in order to improve future behavior (Brazil et al., 2009). Thus, reduced Pe amplitude observed in youth with elevated psychopathic traits could help explain a variety of deficits this population experiences, including increased behavioral impulsivity (Roussy & Toupin, 2000) and perseveration deficits observed in tasks including passive avoidance learning paradigms (Finger et al., 2008). Specifically, reduced post-error processing may result in an inability for youth with elevated psychopathic traits to learn from their mistakes, resulting in an increased propensity towards severe antisocial behavior, incarceration, recidivism, and substance use proclivity (Edens, Campbell, & Weir, 2007; Gregory et al., 2015).

The examination of the Pe with an at-risk juvenile sample is particularly intriguing given the developmental context of this ERP component. Compared to the ERN/Ne, which increases in amplitude throughout adolescence, the Pe's development is rather invariant, showing comparable amplitude between youth and adult samples (Davies, Segalowitz, & Gawin, 2004; Ladouceur, Dahl, & Carter, 2007; Santesso, Segalowitz, & Schmidt, 2006). A reduced Pe amplitude then could suggest a potential biological vulnerability marker for the development of life-course persistent psychopathic traits. Furthermore, as a recent report associated increased Pe amplitude with adult offenders with elevated psychopathic traits (Steele, Maurer, et al., 2016), this observation could therefore reflect a potential compensatory mechanism in adults, attempting to overcome initial post-error processing deficits experienced in adolescence. The Pe has additionally been shown to increase in amplitude through mindfulness meditation training (Larson, Steffen, & Primosch, 2013), suggesting that the developmental anomaly in reduced Pe

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amplitude observed in youth with elevated psychopathic traits may be able to increase and stabilize in amplitude through specialized treatment intervention approaches.

In sum, in Study 1, adolescent psychopathy scores were unrelated to the ERN/Ne mean amplitude, and were negatively related to Pe mean amplitude. Results were most strongly supported through the use of PCA, whereby adolescent psychopathy scores were negatively related to a middle subcomponent underlying the Pe. Stepwise linear regression analyses associated reduced amplitude of this subcomponent underlying the Pe with increased PCL:YV Total and Facet 4 scores reflecting antisocial traits, including early behavioral problems. This is the first evidence to suggest a specific electrophysiological deficit in post-error processing in youth with elevated psychopathic traits.

STUDY 2: Investigating error-related processing in incarcerated adolescents with elevated psychopathic traits using fMRI

INTRODUCTION

Youth with elevated psychopathic traits exhibit a number of comparable neurocognitive deficits as adult psychopathic offenders, including perseveration deficits. These perseveration deficits suggest that youth with elevated psychopathic traits exhibit difficulty allocating attention, becoming hyper-focused on a single aspect of a task at the price of subsequent cognitive functioning, making it difficult to move beyond the current cognitive process (Newman & Baskin-Sommers, 2012). This "attentional bottleneck" can be used to help interpret the results obtained in Study 1, whereby youth with elevated psychopathic traits were characterized by normal ERN/Ne amplitude, and reduced Pe amplitude. Specifically, this suggests that this population devotes a large proportion of attentional resources towards the initial identification of an error, but lack attentional resources to devote to further error-

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processing stages, including processing the motivational significance of error-related information and relating it to ongoing behavior (Ullsperger et al., 2010). Again, the ERN/Ne and Pe are thought to arise from the ACC (Edwards et al., 2012; van Veen & Carter, 2002). However, error-related dysfunction observed in the ACC in youth with elevated psychopathic traits may reflect underlying dysfunction occurring in the basal ganglia. This region plays a significant, though indirect, role in error-related processing by sending continual projections to the ACC. The basal ganglia continually monitors and steadily predicts the result of ongoing events (Botvinick et al., 2001; Mathalon et al., 2003), determining whether the end result of events will be favorable or not (Holroyd & Coles, 2002). Additionally, dysfunction occurring in subregions of the basal ganglia, including the caudate, has been shown to increase stimulus-bound perseverative behavior (Nys et al., 2006). As such, dysfunction occurring within subregions of the basal ganglia could give rise to the error-related processing dysfunction characteristic of youth with elevated psychopathic traits observed in Study 1.

To date, no existing studies have investigated error-related processing in subregions of the basal ganglia in relation to youth with elevated psychopathic traits. Instead, previous studies have investigated various tasks using fMRI, including passive avoidance learning (Finger et al., 2011), probabilistic reversal learning (Finger et al., 2008), and pain perception (Marsh et al., 2013), and also sMRI (Yang et al., 2015). These studies have found mixed results, with two studies showing positive associations between youth psychopathy scores and subregions of the basal ganglia, including hemodynamic activity within the caudate (Finger et al., 2008) and gray matter volume in the putamen (Yang et al., 2015). However, two other studies reported a negative association with youth psychopathy scores and subregions of the basal ganglia, including hemodynamic activity within the caudate (Finger et al., 2011) and the putamen and

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globus pallidus (Marsh et al., 2013). Input nuclei of the basal ganglia include the DS, comprised of the caudate and putamen, and the VS, comprised of the NAcc and olfactory tubercle. Input nuclei receive direct projections from the thalamus and cerebral cortex, and send command information to output nuclei, including the internal segment of the globus pallidus and substantia nigra pars reticulata. The output nuclei send efferents to thalamic nuclei, which then project back to the cortex. Intermediate nuclei of the basal ganglia include the external segment of the globus pallidus, the subthalamic nucleus, and the substantia nigra pars compacta (Lanciego, Luquin, & Obeso, 2012). Thus, with existing studies, it appears youth with elevated psychopathic traits exhibit dysfunction within both input nuclei (including the caudate and putamen) (Finger et al., 2011; Finger et al., 2008; Marsh et al., 2013; Yang et al., 2015), and both intermediate/output nuclei, including the globus pallidus (Marsh et al., 2013) of the basal ganglia.

Existing studies have not investigated whether dysfunction occurring in subregions of the basal ganglia could give rise to error-related dysfunction associated with youth with elevated psychopathic traits. In addition, two subregions of the basal ganglia, including the substantia nigra and subthalamic nucleus, have never been investigated in relation to youth with elevated psychopathic traits. Here, we address this issue by reporting on error-related processing using fMRI and a response inhibition Go/NoGo paradigm in a sample of incarcerated male adolescent offenders. We specifically hypothesized that youth with elevated psychopathic traits would exhibit reduced hemodynamic activity within input nuclei of the basal ganglia, including the caudate, putamen, and NAcc, intermediate nuclei, including the subthalamic nucleus, and both intermediate/output nuclei including the globus pallidus and substantia nigra. Dysfunction within these regions would result in less elaborative processing of error-related information (Cavanagh et al., 2014; Dalley et al., 2007; Nys et al., 2006). Additionally, dysfunction within these

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subregions would support the attentional bottleneck theory (Newman & Baskin-Sommers, 2012), whereby youth with elevated psychopathic traits would devote attentional resources to the initial identification of an error, rather than further processing of error-related information, including the motivational significance of such information towards ongoing behavior.

METHOD

PARTICIPANTS

Participants included 202 incarcerated adolescent offenders at a maximum-security juvenile detention center who participated in a larger study (SWANC-Y) (n = 72 of which performed the EEG task described in Study 1). The sample age range was from 14 to 20 years of age (M = 17.61, SD = 1.09) at the time of fMRI data collection. The sample was predominantly right-handed (13% reported being left-hand dominant). Participants were predominantly Hispanic/Latino (78%), with the remaining self-identifying as White (10%), American Indian or Alaskan Native (6%), Black or African American (3%), or Native Hawaiian or Pacific Islander (1%). Two percent of the sample chose not to disclose their ethnicity. Initial contact with study participants and informed consent/assent procedures were identical to those reported in Study 1.

ASSESSMENTS

Psychopathic traits were assessed using the Hare PCL:YV (Forth et al., 2003). The mean PCL:YV Total Score for this sample was 23.54 (SD = 6.13). The mean Factor 1 score was 6.65 (SD = 3.09) and the mean Factor 2 score was 14.64 (SD = 3.31). PCL:YV Factor 1 and 2 scores were significantly correlated (r = .49, p < .001), consistent with previous reports (Harpur et al., 1989).

In addition to psychopathic traits, assessments were administered by trained research staff to assess IQ, substance dependence, mental illness, and TBI. Exclusionary criteria for Study 2

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was the same as Study 1 (namely, low IQ, TBI accompanied with a significant loss of consciousness, or history of personal and/or familial bipolar or psychotic disorders, or personal history of mood and/or anxiety disorders). Full-scale IQ was estimated using the Vocabulary and Matrix Reasoning sub-tests of the WAIS-III (Wechsler, 1997) for participants older than eighteen years of age, and with the WISC-IV for participants younger than eighteen years of age (Wechsler, 2003) (M = 93.36, SD = 11.94); IQ scores were unavailable for n = 15 participants. Substance dependence and mental illness 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) for which participants met lifetime dependence diagnoses (range: 0 – 11, M = 2.33, SD = 1.70); number of substance dependencies were unavailable for n = 8 participants.

STIMULI AND TASK

Participants performed the same response inhibition Go/NoGo task described in Study 1 (Kiehl et al., 2000). Images were collected with the Mind Research Network's mobile Siemens 1.5T Avanto stationed at the correctional facility, with advanced SQ gradients (max slew rate 200 T/m/s 346 T/m/s vector summation, rise time 200 μs) equipped with a 12 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. No participants were excluded due to excessive motion (defined as motion greater than 2 SD's away from the mean (> 2 mm translation, or 1.5° rotation)). Head motion was evaluated using INRIalign, a mutual information algorithm unbiased by local signal change (Freire & Mangin, 2001; Freire, Roche, & Mangin, 2002).

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DATA REDUCTION

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 half maximum) in the Statistical Parametric Mapping 12 (SPM12) software package (http://www.fil.ion.ucl.ac.uk/spm/software/spm12/). Response types (Hits and FA's) 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 6 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 hemodynamic response for each condition that controlled for differences between slices in timing and variation across regions in the latency of the hemodynamic response (Calhoun, Adali, & Pekar, 2004). Second-level analyses examined associations between these contrast estimates and adolescent psychopathy (via PCL:YV Total, Factor, or Facet scores) and covariate measures (age, IQ, and number of substance dependencies).

REGION OF INTEREST (ROI) ANALYSES

Several *a priori* ROIs were selected from subregions of the basal ganglia previously investigated in relation to youth with elevated psychopathic traits, including the caudate, putamen, NAcc, and globus pallidus (Finger et al., 2011; Finger et al., 2008; Marsh et al., 2013; Yang et al., 2015). In addition, two additional ROIs were selected, which have never been

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investigated in relation to youth with elevated psychopathic traits, including the substantia nigra and subthalamic nucleus.

ROIs were defined primarily by actual anatomical boundaries of brain regions contributing to the regions described above. Boundaries were defined by automated anatomical labels (AAL) featured in the Wake Forest University PickAtlas toolbox available in SPM12 to generate ROIs for the caudate, putamen, globus pallidus, subthalamic nucleus, and substantia nigra (Maldjian, Laurienti, & Burdette, 2004; Maldjian, Laurienti, Kraft, & Burdette, 2003). The NAcc ROI was made with a hand-drawn mask in a similar manner to a previous publication from our laboratory (Cope, Vincent, et al., 2014).

We did not investigate two additional subregions which comprise the basal ganglia, in the olfactory tubercle or the ventral pallidum in the current report. The ventral pallidum is typically investigated in relation to reward processing, rather than error-related processing (Pierce & Kumaresan, 2006), and the olfactory tubercle is typically investigated in relation to sensory processing of information received from olfactory receptors (Wesson & Wilson, 2010).

DATA ANALYSIS

All main effects were whole-brain corrected for Family-Wise Error Rate (FWE). The contrast of interest (FA's vs. Hits) was evaluated for linear relationships with PCL:YV Total Scores in the *a priori* ROIs. Additionally, PCL:YV Factor and Facet scores were also investigated in relationship with basal ganglia function. All models presented accounted for participant's age, IQ, and number of substance dependencies, holding these variables constant. All effects were small-volume corrected for the ROI. Due to some participants missing covariate assessments, final analyses were restricted to n = 183 participants.

RESULTS

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BEHAVIORAL RESULTS

RTs and frequency for Hits and FA's were analyzed. As expected, participants responded faster to NoGo stimuli (*M* = 399 ms, *SD* = 37 ms) compared to Go stimuli (*M* = 445 ms, *SD* = 50 ms), $t(201) = 20.40$, $p < .001$. Participants also made significantly more errors to NoGo stimuli (*M* = 24.88, *SD* = 11.12) compared to Go stimuli (*M* = 11.96, *SD* = 20.24), $t(201) = -8.67$, $p < .001$. There was a main effect for PES (*M* = 44 ms, *SD* = 68 ms). Participants did indeed respond more slowly after error trials (*M* = 454 ms, *SD* = 92 ms) than after correct trials (*M* = 409 ms, *SD* = 60 ms), $t(201) = -9.33$, $p < .001$.

GROUP DIFFERENCES

Similar to Study 1, we report a similar cutoff score of 30 for our sample for high-scoring participants, with thirty-seven participants meeting the categorical classification for psychopathy (*M* PCL:YV Total Score = 31.94, *SD* = 1.67). Using a quartile split of PCL:YV Total Scores, youth with low levels of psychopathic traits consisted of *n* = 39 participants (*M* PCL:YV Total Score = 14.56, *SD* = 3.28). Groups significantly differed with respect to PCL:YV Total Score, $t(74) = -28.90$, $p = < .001$, and number of substance dependencies, $t(69) = -3.02$, $p = .004$, with high-scoring participants scoring higher in both categories. However, groups did not significantly differ with respect to IQ, $t(66) = 0.31$, $p = .757$, or age, $t(74) = 0.93$, $p = .354$. See Table 5 for the remainder of group differences.

CORRELATIONAL ANALYSES

PCL:YV Total, Factor, and Facet scores were positively correlated with number of substance dependencies (see Table 4), consistent with previous studies (Edwards et al., submitted; Smith & Newman, 1990; Walsh et al., 2007). PCL:YV Facet 1 scores were positively correlated with IQ scores ($r = .16$, $p = .034$). IQ scores were not significantly correlated with any

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other PCL:YV scores or covariate measures. IQ scores were significantly negatively correlated with number of Hits (r = -.17, p = .024); no other significant correlations emerged with PCL:YV scores, covariate measures, or number of Hits or FA's. Age or PES did not significantly correlate with adolescent psychopathy scores or covariate measures. See Table 4 or the remainder of correlations between adolescent psychopathy scores and covariate measures.

FA's vs. HITS

Main Effects: This contrast produced significant activation in bilateral regions of the caudate, putamen, globus pallidus, NAcc, subthalamic nucleus, and substantia nigra (see Figure 2). See Table 6 for peaks.

Model 1: In this model, PCL:YV Total Score was entered along with covariate measures (age, IQ, and number of substance dependencies). PCL:YV Total Score showed a negative association with hemodynamic activity in both the left and right subthalamic nucleus and the right substantia nigra during error-related processing (see Table 7). In addition, IQ scores showed a negative association with activity in the right substantia nigra, right NAcc, right caudate, and left globus pallidus during error-related processing (see Table 7). No significant associations emerged with age or number of substance dependencies with any of the *a priori* ROIs.

Model 2: In this model, PCL:YV Factor 1 (reflecting interpersonal and affective traits) and Factor 2 (measuring lifestyle and antisocial traits) scores were entered along with the same covariate measures used in Model 1. Neither PCL:YV Factor scores, age, or number of substance dependencies were significantly associated with any of the *a priori* ROIs. IQ scores showed a negative association with activity in the right NAcc and the left globus pallidus during error-related processing in this model.

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Model 3: In this model, PCL:YV Facet 1 (measuring interpersonal traits), Facet 2 (measuring affective traits), Facet 3 (measuring lifestyle traits), and Facet 4 (measuring antisocial traits) scores were entered along with the same covariate measures used in Models 1 & 2. PCL:YV Facet 1 showed a negative association with activity in the left caudate and the right subthalamic nucleus, PCL:YV Facet 3 showed a negative association with activity in the left subthalamic nucleus, and PCL:YV Facet 4 showed a negative association with activity in the right NAcc, right caudate, and right globus pallidus during error-related processing (see Table 8). In addition, IQ scores showed a negative association with activity in the right NAcc and left globus pallidus during error-related processing (see Table 8). No other significant associations emerged with PCL:YV Facet 2 scores, age, or number of substance dependencies with any of the *a priori* ROIs.

SUPPLEMENTARY ANALYSES

In addition to performing ROI-specific analyses focusing strictly on the basal ganglia, we performed additional analyses investigating thirty-two other ROIs previously reported as being activated during the False Alarms vs. Hits condition during the same Go/NoGo task in a sample of n = 102 healthy control participants (Steele, Claus, et al., 2014). The regions investigated included the L/R superior frontal gyrus, L/R inferior frontal gyrus, L/R precentral gyrus, L/R ACC, L superior temporal gyrus, L/R middle temporal gyrus, L/R inferior parietal lobule, L middle occipital gyrus, L/R inferior occipital gyrus, R fusiform gyrus, R precuneus, L cuneus, R lingual gyrus, pons, brain stem, and two regions of the cerebellum: the culmen and declive (Steele, Claus, et al., 2014).

ROIs were defined primarily by actual anatomical boundaries of brain regions contributing to the regions described above. Boundaries were defined by automated anatomical

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labels (AAL) featured in the Wake Forest University PickAtlas toolbox available in SPM12 to generate ROIs (Maldjian et al., 2004; Maldjian et al., 2003).

As before, all main effects were whole-brain corrected for Family-Wise Error Rate (FWE). The contrast of interest (FA's vs. Hits) was evaluated for linear relationships with PCL:YV Total, Factor, and Facet scores and covariate measures in the *a priori* ROIs. All effects were small-volume corrected for the ROI.

Supplementary analyses revealed that PCL:YV Total and Facet 1 scores were positively related to hemodynamic activity in the left superior frontal gyrus during error-processing, whereas PCL:YV Facet 2 scores (measuring affective traits) were negatively related to hemodynamic activity in the left ACC during error-related processing. No other significant associations emerged with the remaining PCL:YV Factor or Facet scores.

The superior frontal gyrus is typically negatively correlated with age during error-related processing, whereby younger participants typically display increased hemodynamic activity in this region compared to older participants (Tamm et al., 2002). In the current report, youth with elevated psychopathic traits exhibited increased hemodynamic activity in the left superior frontal gyrus compared to youth with lower levels of psychopathic traits, which may reflect a compensatory mechanism attempting to overcome deficits observed in the ACC during error-related processing in order to properly perform the experimental paradigm.

DISCUSSION FOR STUDY 2

In Study 2, adolescent psychopathy scores were negatively related to hemodynamic activity within regions encompassing the basal ganglia during error-related processing. Specifically, adolescent psychopathy scores were negatively related to input nuclei (the caudate

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and NAcc), intermediate nuclei (the subthalamic nucleus), and both intermediate/output nuclei (the globus pallidus and substantia nigra) of the basal ganglia.

Specifically, interpersonal (Facet 1) and antisocial (Facet 4) adolescent psychopathy scores were negatively associated with hemodynamic activity within input nuclei of the basal ganglia, including the caudate, during error-related processing. Facet 1 includes traits such as impression management, pathological lying, conning and manipulative behavior, and a grandiose sense of self-worth (Neumann et al., 2006). The caudate is typically involved in the shifting of attentional sets (Middleton & Strick, 2000; Ravizza & Ciranni, 2002), with damage leading to stimulus-bound perseverative behavior (Nys et al., 2006). Dysfunction within the caudate may then lead to perseveration deficits that are typically associated with youth with elevated psychopathic traits (Budhani & Blair, 2005; Dadds et al., 2006; Finger et al., 2008). Additionally, antisocial (Facet 4) adolescent psychopathic traits were negatively associated with hemodynamic activity in input nuclei of the basal ganglia, including the right caudate and right NAcc. Facet 4 traits include poor behavioral controls, early behavioral problems, juvenile delinquency, and criminal versatility (Neumann et al., 2006). The NAcc also plays a significant role in substance use disorders (Weissman et al., 2015). Psychopathic traits, particularly Facet 4 traits, have been shown to be highly comorbid with substance use proclivity (Kennealy, Hicks, & Patrick, 2007). Furthermore, the NAcc plays a role in processing rewarding and reinforcing information, including errors (Dalley et al., 2007). With reduced NAcc activity in response to errors, this may partly explain why youth who score higher on PCL:YV Facet 4 traits have an increased propensity towards substance use, incarceration, and recidivism (Edens et al., 2007), by failing to consider the consequences of their actions.

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In addition to dysfunction within input nuclei, youth scoring higher on PCL:YV Total, Facet 1, and Facet 3 scores exhibited reduced hemodynamic activity within intermediate nuclei of the basal ganglia, including the subthalamic nucleus, during error-related processing. This region is typically involved in the phenomenon of PES (Cavanagh et al., 2014; Siegert et al., 2014), described as the behavioral slowing down after an incorrect response in order to prevent error occurrence in the future (Rabbit, 1981). The subthalamic nucleus has never been investigated in relation to psychopathic personality. PCL:YV Facet 3 traits include impulsivity, irresponsibility, stimulation seeking, and a lack of realistic, long-term goals (Neumann et al., 2006). Interestingly, damage to the subthalamic nucleus has been shown to increase impulsivity (Uslaner & Robinson, 2006). Therefore, reduced activity within the subthalamic nucleus during error-related processing may play a role in the impulsive nature that often characterizes youth with elevated psychopathic traits.

Finally, adolescent psychopathy scores were associated with reduced hemodynamic activity in both intermediate/output nuclei of the basal ganglia. Specifically, youth scoring higher on PCL:YV Total Score exhibiting reduced hemodynamic activity in the right substantia nigra and youth scoring higher on Facet 4 scores exhibiting reduced hemodynamic activity in the right globus pallidus during error-related processing. The substantia nigra is typically involved in information processing, particularly for behaviorally salient and novel information (Bunzeck & Duzel, 2006), and has never been investigated in relation to psychopathic personality. Errors are frequently described as behaviorally significant events, as they are quite novel events, deviating from normal processing (Harsay, Spaan, Wijnen, & Ridderinkhof, 2012). Similarly, the globus pallidus is typically involved in voluntary movement generation, and lesions specific to this region have been previously associated with poor motivation on behavioral tasks

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(Vijayaraghavan, Vaidya, Humphreys, Beglinger, & Paradoso, 2008). Reduced activity within the globus pallidus and substantia nigra therefore suggests that youth with elevated psychopathic traits may exhibit a specific deficit in processing the ongoing motivational significance of error-related information (Ullsperger et al., 2010), and applying this information to ongoing behavior. These results support the findings found in Study 1, whereby youth with elevated psychopathic traits exhibited reduced Pe mean amplitude. Combining the results from these two studies, it appears that youth with elevated psychopathic traits suffer from an attentional abnormality, in which they can detect an error has occurred, but lack attentional resources to devote to the motivational significance of error-related information, and applying such information to ongoing behavior.

In addition to psychopathy scores, in the current report, increased IQ scores were associated with reduced hemodynamic activity in several regions encompassing the basal ganglia, including the caudate, NAcc, substantia nigra, and globus pallidus during error-related processing. Several neuroimaging studies support the *neural efficiency hypothesis*, whereby those characterized by higher intelligence show lower (more efficient) brain activity compared to less intelligent individuals on cognitive tasks of low to moderate difficulty (Neubauer & Fink, 2009). Individuals with higher intelligence appear to be better at blocking out interfering information compared to those with lower intelligence. In addition, Burgaleta et al. (2014) suggests that regions of the striatum, including the NAcc, plays a significant role in processing novel, abstract problems that are highly dependent upon working memory, but not when dealing with problems requiring manipulation of previously acquired knowledge (Burgaleta et al., 2014). In the current study, we incorporated a response inhibition Go/NoGo task to investigate error-related processing. Compared to other experimental paradigms, including Stop-Signal tasks, the

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Go/NoGo tasks engages brain regions involved in successful response inhibition, without the additional confound of requiring working memory demands (Rubia et al., 2001). Therefore, as our Go/NoGo task is not dependent upon processing abstract problems, this may not engage regions of the basal ganglia, such as the NAcc, to the same degree as other experimental paradigms in individuals characterized by higher intelligence.

To summarize the results found in Study 2, we found youth with elevated psychopathic traits, particularly interpersonal (Facet 1), lifestyle (Facet 3), and antisocial (Facet 4) traits, exhibited reduced error-related hemodynamic activity in subregions encompassing the basal ganglia. Specifically, youth scoring high on the PCL:YV exhibited reduced hemodynamic activity within input nuclei (including the caudate and NAcc), intermediate nuclei (including the subthalamic nucleus), and both intermediate/output nuclei (including the globus pallidus and substantia nigra) during error-related processing. The results from this study support the attentional bottleneck theory (Newman & Baskin-Sommers, 2012), whereby youth with elevated psychopathic traits appear to exhibit intact processing of the initial detection of an error. However, such youth appear to exhibit a specific deficit in allocating attentional resources to further processing of error-related information, including processing the motivational significance of error-related information and applying it to ongoing behavior.

FINAL DISCUSSION

Youth with elevated psychopathic traits exhibit a number of neurocognitive deficits consistent with adult psychopathic offenders including reduced sensitivity to punishment cues (Vitale et al., 2005), behavioral inhibition (Roussy & Toupin, 2000), passive avoidance learning (Finger et al., 2008), and perspective taking (Cheng et al., 2012). Because of these similar deficits, researchers have attempted to delineate the adolescent manifestation of this condition, as

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personality traits are still in nascent stages of development. Intervention efforts targeted specifically towards at-risk youth may have a better chance of altering life-course persistent antisocial behavior if started early (Caldwell, 2011; Caldwell et al., 2007). One such cognitive deficit may involve the processing of error-related information, as youth with elevated psychopathic traits often perseverate during experimental learning paradigms, failing to adjust their behavior to meet the demands established by external sources (Budhani & Blair, 2005; Dadds et al., 2006; Finger et al., 2008; Roussy & Toupin, 2000). As such, in the current report, we investigated error-related processing in incarcerated adolescents with elevated psychopathic traits recruited from a maximum-security correctional facility using two different neuroimaging methodologies using ERPs and fMRI.

ERPs are commonly used to examine different components of cognitive control, including error-related processing, with the two most commonly investigated error-related ERPs including the ERN/Ne and Pe. Previous studies with adult psychopathic offenders suggest that adult psychopathic offenders can detect when an error has occurred, as indexed by intact ERN/Ne amplitude (Brazil et al., 2009; 2011; Maurer et al., 2016; Munro et al., 2011; Steele et al., 2016; von Borries et al., 2010), but exhibit a specific deficit in post-error processing, as exhibited by dysfunctional Pe amplitude. Specifically, in two previous reports, one with adult males (Brazil et al., 2009) and another with adult females (Maurer et al., 2016) associated reduced Pe amplitude with increased psychopathy scores. However, a recent report found increased Pe amplitude in incarcerated males with elevated psychopathic traits compared to incarcerated offenders with low levels of psychopathic traits (Steele, Maurer, et al., 2016).

In Study 1, we found with both group-based and stepwise linear regression analyses, youth with elevated psychopathic traits, particularly PCL:YV Total and Facet 4 traits, exhibited

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intact ERN/Ne mean amplitude, but reduced Pe mean amplitude. Intact ERN/Ne amplitude suggests that youth with elevated psychopathic traits can detect that an error has occurred, but reduced Pe amplitude suggests that this population exhibits a specific deficit in using information received from errors in order to improve future behavior (Brazil et al., 2009). This reduced Pe amplitude observed in youth with elevated psychopathic traits could help explain a variety of deficits this population experiences, including increased behavioral impulsivity (Roussy & Toupin, 2000) and perseveration deficits observed in a number of experimental conditions (Budhani & Blair, 2005; Dadds et al., 2006; Finger et al., 2008). Specifically, reduced post-error processing may result in an inability for youth with elevated psychopathic traits to learn from their mistakes, resulting in an increased propensity towards severe antisocial behavior, incarceration, recidivism, and substance use proclivity (Edens et al., 2007; Gregory et al., 2015).

Intact ERN/Ne amplitude and reduced Pe amplitude observed in youth with elevated psychopathic traits best supports the attentional bottleneck theory (Newman & Baskin-Sommers, 2012), whereby youth with elevated psychopathic traits appear to exhibit intact processing of the initial detection of an error, but appear to exhibit a specific deficit in allocating attentional resources to further processing of error-related information, including applying the motivational significance of error-related information to ongoing behavior (Ullsperger et al., 2010).

The examination of the Pe with an at-risk juvenile sample is particularly intriguing given the developmental context of this ERP component. Compared to the ERN/Ne, which increases in amplitude throughout adolescence, the Pe's development is rather invariant, showing comparable amplitude between youth and adult samples (Davies et al., 2004; Ladouceur et al., 2007; Santesso et al., 2006). A reduced Pe amplitude hen could suggest a potential biological vulnerability marker for the development of life-course persistent psychopathic traits.

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Furthermore, as a recent report associated increased Pe amplitude with adult offenders with elevated psychopathic traits (Steele, Maurer, et al., 2016). Increased Pe amplitude could therefore reflect a potential compensatory mechanism, attempting to overcome initial post-error processing deficits experienced in adolescence in adult offenders with elevated psychopathic traits. Pe amplitude has additionally been shown to increase in amplitude through mindfulness meditation training (M. J. Larson et al., 2013), suggesting that the developmental anomaly in reduced Pe amplitude observed in youth with elevated psychopathic traits may be able to increase and stabilize in amplitude through specialized treatment intervention approaches.

The ERN/Ne and Pe are thought to arise from the ACC (Edwards et al., 2012; van Veen & Carter, 2002). However, error-related dysfunction observed in the ACC in youth with elevated psychopathic traits may reflect underlying dysfunction occurring in the basal ganglia. This region plays a significant role in error-related processing by sending continual projections to the ACC. The basal ganglia continually monitors and steadily predicts the result of ongoing events (Botvinick et al., 2001; Mathalon et al., 2003), determining whether the end result of events will be favorable or not (Holroyd & Coles, 2002). Additionally, dysfunction occurring in subregions of the basal ganglia, including the caudate, has been shown to increase stimulus-bound perseverative behavior (Nys et al., 2006). As such, dysfunction occurring within subregions of the basal ganglia could give rise to the error-related processing dysfunction characteristic of youth with elevated psychopathic traits observed in Study 1.

In Study 2, we found adolescent psychopathy scores, particularly interpersonal (Facet 1), lifestyle (Facet 3), and antisocial (Facet 4) traits, were associated with reduced error-related hemodynamic activity in subregions encompassing the basal ganglia. Specifically, youth scoring high on the PCL:YV exhibited reduced hemodynamic activity within input nuclei (including the

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caudate and NAcc), intermediate nuclei (including the subthalamic nucleus), and intermediate/output nuclei (including the globus pallidus and substantia nigra) during error-related processing. Reduced hemodynamic activity within these regions suggests that deficits within the basal ganglia could help contribute to the electrophysiological abnormalities observed in Study 1. The basal ganglia plays a significant, though indirect, role in error-related processing by sending continual projections to the ACC, where the Pe ERP component is believed to arise (Edwards et al., 2012; van Veen & Carter, 2002).

Additionally, dysfunction within subregions of the basal ganglia could help contribute to some of the characteristic deficits associated with youth with elevated psychopathic traits. Reduced hemodynamic activity within the caudate could help explain the perseveration deficits typically associated with this population (Budhani & Blair, 2005, whereas reduced hemodynamic activity in the subthalamic nucleus could help could contribute to the impulsive nature distinguishing of youth with elevated psychopathic traits (Roussy & Toupin, 2000). Furthermore, dysfunction within the substantia nigra, NAcc, and globus pallidus could help contribute to the profile of an individual who is not significantly motivated to process novel, salient events that have the potential to be quite reinforcing, such as errors. Errors are quite aversive events, but they do not appear to impact juveniles with elevated psychopathic traits to the same degree as other populations. Rather, reduced Pe amplitude and hemodynamic activity in subregions of the basal ganglia suggest that adolescents with elevated psychopathic traits can detect an error has occurred, but lack resources to fully process error-related information, including how to apply such information to ongoing behavior (Ullsperger et al., 2010). This could have tremendous implications in terms of the heightened propensity for youth with elevated psychopathic traits to engage in reckless, impulsive behavior, without fully processing the consequences of their

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actions. By not taking into consideration such consequences, this could potentially lead to an increased propensity towards substance use, incarceration, and the eventual recidivism that typically characterizes youth with elevated psychopathic traits (Gregory et al., 2015).

LIMITATIONS

Several limitations from the two studies performed should be noted. First, psychopathic traits, at least at low to moderate levels detected early in life, often reduce naturally in a large proportion of youth samples (Frick et al., 2003; Lee et al., 2009; Lynam et al., 2007). The best evidence of continuity from adolescence to adulthood comes from longitudinal research incorporating both self-report and interview-based measures of psychopathic traits, showing moderate stability from age 13 to 23 (Lynam et al., 2007). As such, there exists the possibility that youth in our current study may not grow up to meet the established criteria for psychopathic personality. Longitudinal research is desperately needed to see whether reduced Pe amplitude or error-related hemodynamic activity in youth samples can serve as a potential biological vulnerability marker for the future development of psychopathic personality.

Second, our study recruited participants from a maximum-security correctional facility. While recruiting participants from incarcerated settings compared to community samples leads to a substantially higher base rate of psychopathic traits (Neumann & Hare, 2008), youth in incarcerated settings differ on a number of variables compared to youth from community samples. Such differences include substance use history, general intelligence, and trait anxiety (Foley, 2001; Wasserman, McReynolds, Lucas, Fisher, & Santos, 2002). In the two studies performed, we note that we had PCL:YV Total Scores ranging from the low to the extreme range of scores, with means in line with previously published incarcerated youth samples. Thus, our samples should be considered to have clinical levels of psychopathy, which may not extrapolate

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to samples with lower overall psychopathy scores. Moreover, there continues to be little agreement between various self-report and interview-based measures of psychopathic traits in adolescent samples (Fink, Tant, Tremba, & Kiehl, 2012; Maurer et al., submitted). We recommend that future studies compare samples on identical measures of adolescent psychopathic traits; comparison across assessment instruments is not likely to lead to replication.

Finally, in the two studies performed, as in common practice in psychopathy research, we tested our hypotheses by examining participants with high to low levels of psychopathic traits within the samples. This allows us to carefully control for potentially moderating variables (i.e., substance use history, etc.). However, we did not compare our results to a 'healthy' population. Thus, our findings need to be considered in this light. Future research should attempt to replicate and extend our findings incorporating a non-incarcerated control group, carefully attending to critical moderating variables, including IQ, age, and substance use history.

FUTURE DIRECTIONS

EEG and fMRI have complimentary strengths and weaknesses. The advantage of EEG is the tremendous temporal resolution on the order of milliseconds associated with the technique, and the ability to measure neuronal activity directly. However, due to the inverse problem, it is actually mathematically impossible to truly localize the given source of neuronal firing (Luck, 2014). In contrast, fMRI has excellent spatial resolution, but measures an indirect metabolic correlate of neuronal function in the blood oxygenation level dependent (BOLD) signal, over a considerably longer time period of seconds. Though both ERP and fMRI provide spatial and temporal information, the strengths of each technique differ in a complementary manner. Thus, an approach which combines both techniques can draw potentially on the strengths associated with each technique not afforded by either technique alone. We are currently analyzing a

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combination of ERP and fMRI data on a subsample of participants in the two samples reported here (n = 72) using joint independent component analysis (ICA) (Maurer et al., in prep), to improve the identification of the specific error-related cognitive process associated with youth with elevated psychopathic traits.

Additionally, it would be interesting to perform functional network connectivity (FNC) analyses with the full n = 202 sample involved in Study 2. Here, we could see if subregions of the basal ganglia actually do show reduced functional connectivity with the ACC in youth with elevated psychopathic traits, which would fully enhance our understanding of the error-related dysfunction associated with this population.

FINAL SUMMARY

Youth with elevated psychopathic traits exhibit a number of comparable neurocognitive deficits as adult psychopathic offenders. The results from the two studies performed in this review suggest that youth with elevated psychopathic traits exhibit comparable error-related processing deficits as adult psychopathic offenders. Namely, in Study 1 using error-related ERPs, adolescent psychopathy scores were not significantly related to ERN/Ne amplitude, but were negatively related to Pe amplitude. Despite the excellent temporal resolution associated with ERPs, the technique suffers from a lack of specific temporal resolution. Using fMRI in Study 2, we show that error-related dysfunction associated with youth with elevated psychopathic traits may actually be due to underlying dysfunction occurring in subregions of the basal ganglia. Specifically, adolescent psychopathy scores were negatively related to hemodynamic activity within input nuclei of the basal ganglia (the caudate and NAcc), intermediate nuclei (subthalamic nucleus), and intermediate/output nuclei (globus pallidus and substantia nigra) during error-related processing. The results obtained from these two studies

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best support the attentional bottleneck theory (Newman & Baskin-Sommers, 2012), whereby youth with elevated psychopathic traits devote attentional resources to the initial identification of an error, but exhibit a specific deficit in allocating attentional resources to further processing of error-related information, including applying the motivational significance of error-related information to ongoing behavior. Reduced processing of later error-related information may result in an inability for youth with elevated psychopathic traits to learn from their mistakes, resulting in an increased propensity towards severe antisocial behavior, incarceration, recidivism, and substance use proclivity (Edens et al., 2007; Gregory et al., 2015).

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Table 1

Correlations among PCL:YV Variables and Covariates for Study 1 (n = 100)

Variable	PCLYV Total	PCLYV Factor1	PCLYV Factor2	PCLYV Facet 1	PCLYV Facet 2	PCLYV Facet 3	PCLYV Facet 4	Age	IQ	Sub. Use
PCLYV Total	____									
PCLYV Factor 1	0.86**	_____								
PCLYV Factor 2	0.90**	0.58**	____							
PCLYV Facet 1	0.66**	0.85**	0.39**	____						
PCLYV Facet 2	0.78**	0.84**	0.60**	0.42**	____					
PCLYV Facet 3	0.80**	0.50**	0.90**	0.34**	0.50**	____				
PCLYV Facet 4	0.78**	0.50**	0.86**	0.30**	0.54**	0.56**	____			
Age	0.08	0.08	0.02	0.15	-0.02	0.05	-0.02	____		
IQ	-0.05	0.07	-0.12	0.19	-0.07	-0.14	-0.08	0.05	____	
Sub. Use	0.33**	0.24**	0.30**	0.15	0.26**	0.32**	0.22**	-0.07	0.02	_____

Note. Assessments: PCL:YV Total is the total score derived from the Psychopathy Checklist: Youth Version (PCL:YV); PCL:YV Factor 1 and 2 are Factor 1 and 2 scores derived from the PCL:YV; PCL:YV Facet 1, Facet 2, Facet 3, and Facet 4 scores are Facet 1, 2, 3, and 4 scores derived from the PCL:YV (Forth, Kosson, & Hare, 2003); Intelligence Quotient (IQ) was calculated from the Wechsler Adult Intelligence Scale – Third Version (WAIS-III) (Wechsler, 1997) and Wechsler Intelligence Scale for Children – Fourth Edition (Wechsler, 2003); Sub. Use is the number of substance dependencies calculated by summing the total number of substances (alcohol and drug) for which participants met lifetime dependence diagnoses from the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS) (Kaufman, Birmaher, & Brent, 1997).
**$p < .05$; *$p < .01$.

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Table 2

Descriptive Statistics and Independent Samples t-tests for Variables for Study 1 (n = 100)

	All Participants (n=100)			PCL:YV Lower Quartile (n=22)			PCL:YV Highest Quartile (n=21)					
Variable	N	Mean	SD	N	Mean	SD	N	Mean	SD	t	df	p
PCL:YV Total	100	23.82	6.46	22	14.64	3.53	21	32.03	1.47	-20.93	41	< .001
PCL:YV Factor 1	100	6.75	3.19	22	3.45	2.11	21	11.05	1.36	-13.96	41	< .001
PCL:YV Factor 2	100	12.78	3.20	22	8.36	2.74	21	15.86	1.49	-11.08	41	< .001
PCL:YV Facet 1	100	2.24	1.91	22	1.09	1.60	21	4.76	1.14	-8.63	41	< .001
PCL:YV Facet 2	100	4.51	1.87	22	2.36	1.33	21	6.29	1.10	-10.51	41	< .001
PCL:YV Facet 3	100	6.52	2.02	22	3.91	1.54	21	8.19	1.17	-10.24	41	< .001
PCL:YV Facet 4	100	8.18	1.81	22	6.09	2.05	21	9.67	0.66	-7.64	41	< .001
Age	100	17.38	0.86	22	17.36	0.85	21	17.62	0.81	-1.01	41	.317
IQ	91	93.90	10.97	18	95.06	12.98	19	94.63	9.60	0.13	35	.910
Substance Use	100	2.33	1.67	22	1.55	1.14	21	2.67	1.93	-2.33	41	.025
ERN/Ne	100	-3.17	4.39	22	-3.69	4.96	21	-3.08	3.57	-0.46	41	.649
Pe	100	6.25	6.78	22	8.61	7.05	21	4.84	7.37	1.72	41	.093
PC1	100	0.31	0.41	22	0.37	0.48	21	0.30	0.38	0.55	41	.586
PC2	100	0.64	0.50	22	0.87	0.50	21	0.50	0.51	2.40	41	.021
PC3	100	-0.13	0.21	22	-0.17	0.21	21	-0.10	0.17	-1.11	41	.273
PC4	100	0.37	0.42	22	0.52	0.48	21	0.29	0.38	1.67	41	.102

Note. Assessments: PCL:YV Total is the total score derived from the Psychopathy Checklist: Youth Version (PCL:YV); PCL:YV Factor 1 and 2 are Factor 1 and 2 scores derived from the PCL:YV; PCL:YV Facet 1, Facet 2, Facet 3, and Facet 4 scores are Facet 1, 2, 3, and 4 scores derived from the PCL:YV (Forth et al., 2003); Intelligence Quotient (IQ) was calculated from the Wechsler Adult Intelligence Scale – Third Version (WAIS-III) (Wechsler, 1997) and Wechsler Intelligence Scale for Children – Fourth Edition (Wechsler, 2003); Substance Use is the number of substance dependencies calculated by summing the total number of substances (alcohol and drug) for which participants met lifetime dependence diagnoses from the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS) (Kaufman et al., 1997); ERN/Ne, Pe, PC1, PC2, PC3, and PC4 refer to the mean amplitude of the time-domain and principal components pertaining to ERN/Ne and Pe amplitude.

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Table 3

Linear Stepwise Regression Analyses with Psychopathy Variables entered with Covariates predicting Principal Component 2 (PC2) Mean Amplitude

Predictors	B	SE B	t	β	Sig.
Regression 1:					
PCL:YV Total	-0.018	0.008	-2.112	-.218	.037
IQ			0.857		.394
Age			-1.643		.104
Sub. Use			-1.263		.210
Regression 2:					
PCL:YV Facet 1			0.687		.494
PCL:YV Facet 2			0.091		.927
PCL:YV Facet 3			-0.183		.855
PCL:YV Facet 4	-0.073	0.028	-2.609	-.267	.011
IQ			0.755		.452
Age			-1.825		.071
Sub. Use			-1.305		.195

Regression1: R^2 = .048, R = .218, $F(1,89)$ = 4.461.
Regression 2: R^2 = .071, R = .267, $F(1,89)$ = 6.806.
Note. Assessments: PCL:YV Total is the total score derived from the Psychopathy Checklist: Youth Version (PCL:YV); PCL:YV Factor 1 and 2 are Factor 1 and 2 scores derived from the PCL:YV; PCL:YV Facet 1, Facet 2, Facet 3, and Facet 4 scores are Facet 1, 2, 3, and 4 scores derived from the PCL:YV (Forth et al., 2003); Intelligence; Intelligence Quotient (IQ) was calculated from the Wechsler Adult Intelligence Scale – Third Version (WAIS-III) (Wechsler, 1997) and Wechsler Intelligence Scale for Children – Fourth Edition (Wechsler, 2003); Sub. Use is the number of substance dependencies calculated by summing the total number of substances (alcohol and drug) for which participants met lifetime dependence diagnoses from the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS) (Kaufman et al., 1997). All significant findings corrected for multiple comparisons using the Simes-Hochberg multiple comparison procedure (Hochberg, 1988; Simes, 1986).

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Table 4

Correlations among PCL:YV Variables and Covariates for Study 2 (n = 202)

	PCL: YV Total	PCLYV Factor 1	PCLYV Factor 2	PCLYV Facet 1	PCLYV Facet 2	PCLYV Facet 3	PCLYV Facet 4	IQ	Age	Sub. Use
PCLYV Total	____									
PCLYV Factor 1	.842**	_____								
PCLYV Factor 2	.867**	.493**	____							
PCLYV Facet 1	.653**	.858**	.301**	____						
PCLYV Facet 2	.783**	.838**	.546**	.442**	____					
PCLYV Facet 3	.774**	.433**	.894**	.284**	.460**	____				
PCLYV Facet 4	.732**	.417**	.284**	.228**	.490**	.530**	____			
IQ	-.039	.088	-.142	.155*	.002	-.119	-.139	____		
Age	-.008	.027	-.038	.050	.003	-.082	.020	-.107	____	
Sub. Use	.287**	.181*	.287**	.155*	.159*	.278**	.216**	.086	-.041	____

Note. PCL:YV Total is the total score derived from the Psychopathy Checklist: Youth Version (PCL:YV); PCL:YV Factor 1 and 2 are Factor 1 and 2 scores derived from the PCL:YV; PCL:YV Facet 1, Facet 2, Facet 3, and Facet 4 are Facet 1, 2, 3, & 4 scores derived from the PCL:YV (Forth et al., 1983); Intelligence Quotient (IQ) was calculated from the Wechsler Adult Intelligence Scale – Third Version (WAIS-III) (Wechsler, 1997) and Wechsler Intelligence Scale for Children – Fourth Edition (Wechsler, 2003); Sub. Use is the number of substance dependencies calculated by summing the total number of substances (alcohol and drug) for which participants met lifetime dependence diagnoses from the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS) (Kaufman et al., 1997). $^{**}p < .05$; $^{*}p < .01$.

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Table 5

Descriptive Statistics and Independent Samples t-tests for Variables for Study 2 (n = 202)

	All Participants (n = 202)			PCL:YV Highest Quartile (n = 37)			PCL:YV Lowest Quartile (n = 39)					
Variable	N	Mean	SD	N	Mean	SD	N	Mean	SD	t	df	p
PCL:YV Total	202	23.54	6.13	37	31.94	1.66	39	14.56	3.28	-28.90	74	< .001
PCL:YV Factor 1	202	6.65	3.09	37	11.29	1.62	39	3.44	1.85	-19.66	74	< .001
PCL:YV Factor 2	202	14.64	3.31	37	17.54	1.72	39	9.83	3.10	-13.29	74	< .001
PCL:YV Facet 1	202	2.20	1.90	37	4.95	1.41	39	1.03	1.22	-12.95	74	< .001
PCL:YV Facet 2	202	4.45	1.76	37	6.38	0.95	39	2.41	1.04	-17.27	74	< .001
PCL:YV Facet 3	202	6.40	2.03	37	8.02	1.54	39	3.67	1.42	-12.86	74	< .001
PCL:YV Facet 4	202	8.25	1.71	37	9.46	0.84	39	6.24	2.07	-8.79	74	< .001
Age	202	17.61	1.09	37	17.45	1.28	39	17.69	1.06	0.93	74	.354
IQ	187	93.36	11.94	33	94.24	11.58	35	95.23	14.36	0.31	66	.757
Sub. Use	194	2.29	1.70	34	2.85	1.81	37	1.57	1.77	-3.02	69	.004
Hit RT	202	445.04	50.50	37	445.60	52.52	39	441.15	39.82	-0.42	74	.688
Mean Misses	202	11.96	20.24	37	8.97	10.11	39	10.08	1.61	-0.23	74	.817
False Alarm RT	202	399.44	37.08	37	394.47	32.06	39	402.36	34.52	1.02	74	.312
Mean FA's	202	24.88	11.12	37	23.14	9.66	39	23.97	10.99	0.35	74	.725
Post-Error Slowing	202	44.76	68.18	37	64.35	81.40	39	47.08	68.31	-1.00	74	.319

Note. PCL:YV Total is the total score derived from the Psychopathy Checklist: Youth Version (PCL:YV); PCL:YV Factor 1 and 2 are Factor 1 and 2 scores derived from the PCL:YV; PCL:YV Facet 1, Facet 2, Facet 3, and Facet 4 are Facet 1, 2, 3, & 4 scores derived from the PCL:YV (Forth et al., 1983); Intelligence Quotient (IQ) was calculated from the Wechsler Adult Intelligence Scale – Third Version (WAIS-III) (Wechsler, 1997) and Wechsler Intelligence Scale for Children – Fourth Edition (Wechsler, 2003); Sub Use is the number of substance dependencies calculated

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by summing the total number of substances (alcohol and drug) for which participants met lifetime dependence diagnoses from the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS) (Kaufman et al., 1997).
**$p < .05$; *$p < .01$.

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Table 6

Main Effects: False Alarms vs. Hits

Region	x	y	z	FWE *p*-value	*t*-value
L Caudate	-6	0	9	< .001	7.52
R Caudate	9	0	12	< .001	7.15
L Putamen	-33	6	-6	< .001	16.89
R Putamen	30	12	-9	< .001	15.64
L Globus Pallidus	-12	-3	0	< .001	7.20
R Globus Pallidus	15	-3	-6	< .001	8.14
L NAcc	-12	6	-6	< .001	3.97
R NAcc	12	6	-6	< .001	4.89
L Subthalamic Nucleus	-9	-15	-9	< .001	11.73
R Subthalamic Nucleus	9	-12	-9	< .001	12.54
L Substantia Nigra	-6	-15	-12	< .001	13.14
R Substantia Nigra	9	-21	-12	< .001	12.91

Note. Investigating error-related processing in the contrast False Alarms (FA's) vs. Hits. ROIs were defined primarily by actual anatomical boundaries contributing to the regions listed above. Boundaries were defined by automated anatomical labels (AAL) featured in the WFU PickAtlas toolbox available in SPM to generate ROIs for the caudate, putamen, globus pallidus, subthalamic nucleus, and substantia nigra (Maldjian et al., 2003; 2004). The NAcc ROI was made with a hand-drawn mask in a similar manner to a previous publication from our laboratory (Cope, Vincent, et al., 2014). Coordinates are given in MNI space. All *p*'s < .001, and results were whole-brain corrected for Family-Wise Error Rate (FWE).

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Table 7

Psychopathy-Related Effects: FA's vs. Hits

Model 1: PCL:YV Total Score entered with IQ, age, and number of substance dependencies.

	Region	x	y	z	FWE *p*-value	Peak Voxel *t*-value
PCL:YV Total	R Substantia Nigra	15	-21	-6	0.014	2.61
	L Subthalamic Nucleus	-12	-12	-3	0.036	2.19
	R Subthalamic Nucleus	12	-15	-6	0.022	2.42
IQ	R Caudate	18	24	-9	0.027	3.18
	L Globus Pallidus	-15	-3	-6	0.023	2.89
	R NAcc	9	6	-9	0.009	2.87
	R Substantia Nigra	15	-21	-9	0.035	2.25

Note. Investigating error-related processing in the contrast False Alarms (FA's) vs. Hits. ROIs were defined primarily by actual anatomical boundaries contributing to the regions listed above. Boundaries were defined by automated anatomical labels (AAL) featured in the WFU PickAtlas toolbox available in SPM to generate ROIs for the caudate, putamen, globus pallidus, subthalamic nucleus, and substantia nigra (Maldjian et al., 2003; 2004). The NAcc ROI was made with a hand-drawn mask in a similar manner to a previous publication from our laboratory (Cope, Vincent, et al., 2014). Coordinates are given in MNI space. Results were whole-brain corrected for Family-Wise Error Rate (FWE). PCL:YV Total is the Total Score derived from the Hare Psychopathy Checklist: Youth Version (PCL:YV) (Forth et al., 2003). Intelligence Quotient (IQ) was calculated from the Wechsler Adult Intelligence Scale – Third Version (WAIS-III) (Wechsler, 1997). No significant associations were observed between age or IQ scores.

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Table 8

Psychopathy-Related Effects: FA's vs. Hits:

Model 3: PCL:YV Facet scores entered with IQ, age, and number of substance dependencies.

	Region	x	y	z	FWE *p*-value	*t*-value
PCL:YV Facet 1	L Caudate	-21	-18	21	0.009	3.52
	R Subthalamic Nucleus	12	-12	-3	0.045	2.11
PCL:YV Facet 3	L Subthalamic Nucleus	-12	-12	-3	0.013	2.58
PCL:YV Facet 4	R Caudate	6	9	-3	0.025	3.17
	R Globus Pallidus	12	3	-3	0.019	2.91
	R NAcc	9	6	-6	0.005	3.08
IQ	L Globus Pallidus	-15	-3	-6	0.019	2.92
	R NAcc	9	6	-9	0.011	2.78

Note. Investigating error-related processing in the contrast False Alarms (FA's) vs. Hits. ROIs were defined primarily by actual anatomical boundaries contributing to the regions listed above. Boundaries were defined by automated anatomical labels (AAL) featured in the WFU PickAtlas toolbox available in SPM to generate ROIs for the caudate, putamen, globus pallidus, subthalamic nucleus, and substantia nigra (Maldjian et al., 2003; 2004). The NAcc ROI was made with a hand-drawn mask in a similar manner to a previous publication from our laboratory (Cope, Vincent, et al., 2014). Coordinates are given in MNI space. Results were whole-brain corrected for Family-Wise Error Rate (FWE). PCL:YV Facet 1, 3, and 4 scores are the Facet scores derived from the Hare Psychopathy Checklist: Youth Version (PCL:YV) (Forth et al., 2003). Intelligence Quotient (IQ) was calculated from the Wechsler Adult Intelligence Scale – Third Version (WAIS-III) (Wechsler, 1997). No significant associations were observed between PCL:YV Facet 2, age, or IQ scores.

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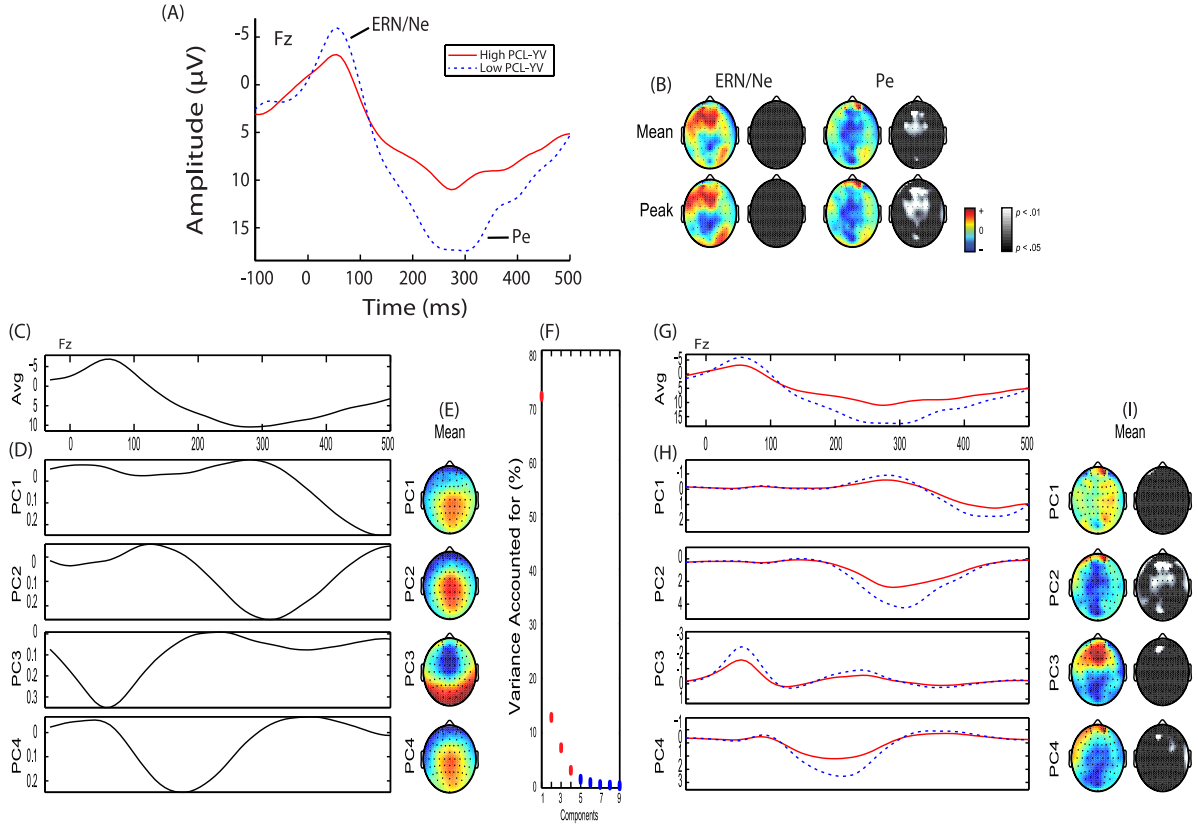

Figure 1. Response-locked event-related potential (ERP) and principal component analysis. **(A)** Representative ERP waveform plotted at Fz for each group with negative plotted up. Youth with elevated psychopathic traits (PCL:YV Total score > 30, *n* = 21) (red) and youth with low levels of psychopathic traits (PCL:YV Total score < 20, *n* = 22) (dotted blue) are plotted. ERP components of interest (the error-related negativity [ERN/Ne] and error-related positivity [Pe]) are identified. **(B)** Topographic difference (color) and statistical (black and white) maps are plotted for each component window highlighting youth with elevated psychopathic traits show reduced Pe amplitude. **(C)** Grand average waveform plotted at Fz. **(D)** Principal components extracted accounting for 94.97% of the variance. **(E)** Topographic depiction of the mean spatial distribution for each principal component. **(F)** Scree plot of singular values which was used to determine a four-component solution. **(G)** Group average waveform for youth with elevated psychopathic traits (red line) and low levels of psychopathic traits (blue line) are plotted at Fz. PC2 reflects Pe mean amplitude. **(H)** Principal components plotted by group. **(I)** Topographic difference (color) and statistical (black and white) maps are plotted for each principal component highlighting youth with elevated psychopathic traits show reduced Pe amplitude. Adapted from "Dysfunctional error-related processing in incarcerated adolescents

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with elevated psychopathic traits" by J. M. Maurer, V. R. Steele, L. M. Cope, G. M. Vincent, J. M. Stephen, V. D. Calhoun, & K. A. Kiehl, 2016, *Developmental Cognitive Neuroscience*, *19*, 70 – 77. Copyright 2016 by Elsevier.

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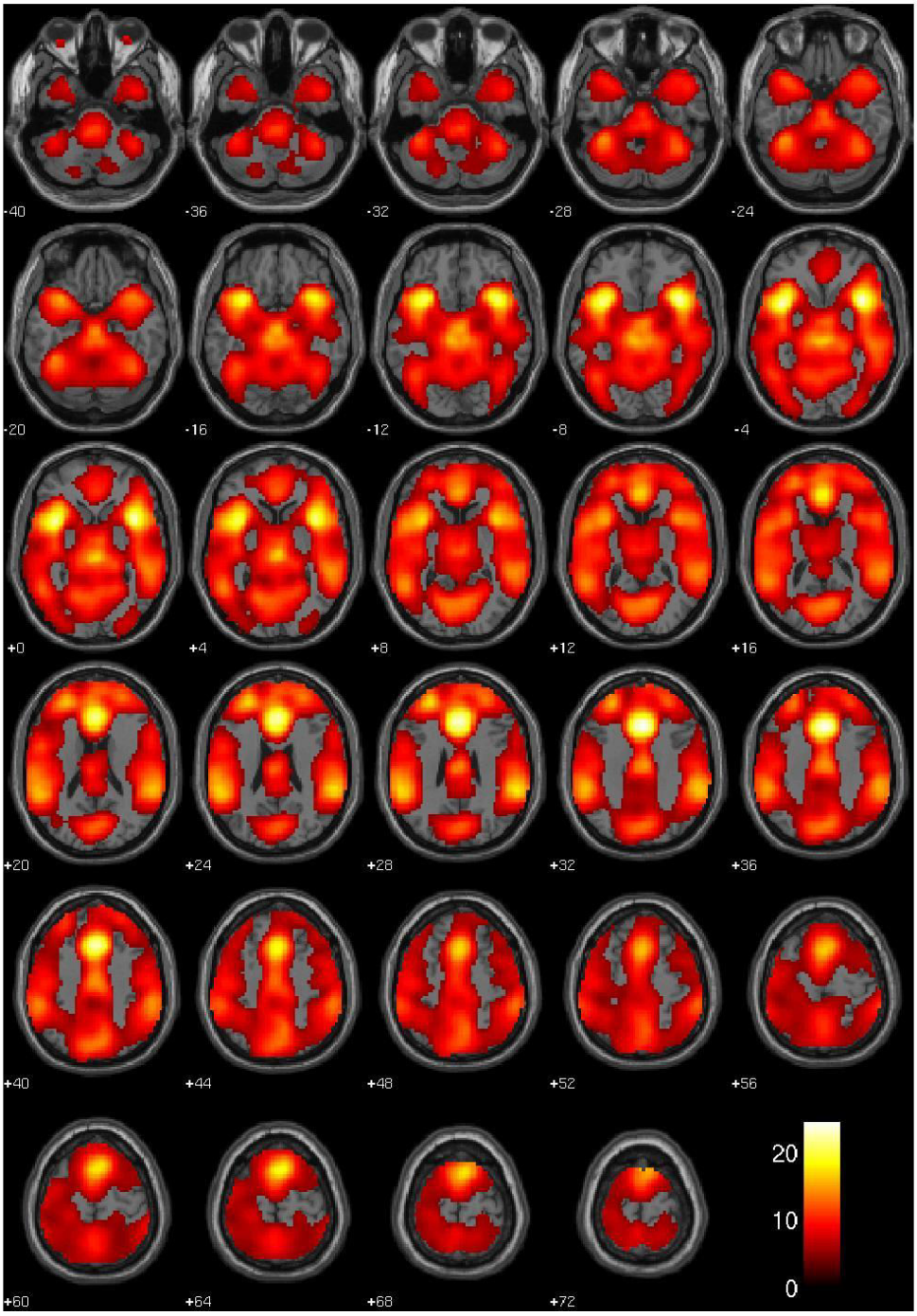

Figure 2. *False Alarms vs. Hits*, Main Effects (all subjects, n = 202, regardless of PCL:YV score). Red-yellow scale represents t-values for signal where FA exceeds that of Hits. Threshold at $p < .001$, FWE-corrected.

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