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Dysfunctional error-related processing in incarcerated youth with elevated psychopathic traits

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A B S T R A C T

Adult psychopathic offenders show an increased propensity towards violence, impulsivity, and recidivism. A subsample of youth with elevated psychopathic traits represent a particularly severe subgroup characterized by extreme behavioral problems and comparable neurocognitive deficits as their adult counterparts, including perseveration deficits. Here, we investigate response-locked event-related potential (ERP) components (the error-related negativity [ERN/Ne] related to early error-monitoring processing and the error-related positivity [Pe] involved in later error-related processing) in a sample of incarcerated juvenile male offenders (n = 100) who performed a response inhibition Go/NoGo task. Psychopathic traits were assessed using the Hare Psychopathy Checklist: Youth Version (PCL:YV). The ERN/Ne and Pe were analyzed with classic windowed ERP components and principal component analysis (PCA). Using linear regression analyses, PCL:YV scores were unrelated to the ERN/Ne, but were negatively related to Pe mean amplitude. Specifically, the PCL:YV Facet 4 subscale reflecting antisocial traits emerged as a significant predictor of reduced amplitude of a subcomponent underlying the Pe identified with PCA. This is the first evidence to suggest a negative relationship between adolescent psychopathy scores and Pe mean amplitude.

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1. Introduction

Psychopathy is a multifaceted personality disorder characterized by affective, interpersonal, and behavioral dysfunction. Psychopaths have been classically defined by their overall absence of moral emotions and their impulsive, irresponsible lifestyle (Hare, 1991, 2003). About 15–25% of incarcerated offenders meet the diagnostic criteria for psychopathy, with increased prevalence in higher security levels (Hare, 2003). This disconcerting population has often proven impervious to treatment intervention approaches, as highlighted by their increased propensity towards violent recidivism (Hemphill et al., 1998; Rice & Harris, 1997). Researchers have recently attempted to delineate the adolescent manifestation of this condition, as personality traits are still in nascent stages of development. Intervention efforts targeted at youth may have a better chance of altering life-course persistent antisocial behavior if started early (Caldwell, 2011; Caldwell et al., 2007).

Youth scoring high on measures of psychopathic traits exhibit similar neurocognitive deficits as adult psychopathic offenders. For example, youth with elevated psychopathic traits exhibit increased behavioral impulsivity (Roussy & Toupin, 2000), reduced sensitivity to punishment cues (Vitale et al., 2005), and passive avoidance learning (Finger et al., 2008) deficits. Furthermore, using both functional and structural neuroimaging, abnormalities have been observed in youth with elevated psychopathic traits consistent with adult psychopathic offenders as young as fourteen years of age (Cope et al., 2014; Ermer et al., 2013; Harenski et al., 2014; Lockwood et al., 2013; Marsh et al., 2008). Reduced hemodynamic
activity and reduced gray matter have been found in a number of paralimbic regions, including the orbitofrontal cortex (Cope et al., 2014; Ermer et al., 2013), insula (Lockwood et al., 2013), amygdala (Harenski et al., 2014; Marsh et al., 2008), posterior cingulate cortex (PCC) (Ermer et al., 2013), parahippocampal gyrus (Ermer et al., 2013), and anterior cingulate cortex (ACC) (Cope et al., 2014; Ermer et al., 2013; Marsh et al., 2008).

One additional cognitive deficit juveniles with elevated psychopathic traits may experience is the processing of error-related information. Youth with elevated psychopathic traits often perseverate during behavioral inhibition and experimental learning paradigms, failing to adjust their behavior to meet the demands established by external sources (Budhani & Blair, 2005; Finger et al., 2008; Roussy & Toupin, 2000; Vitale et al., 2005). Event-related potentials (ERPs) are commonly used to examine different components of cognitive control including error-related processing. The two most frequently investigated error-related ERPs are the error-related negativity (the ERN or Ne) and the error-related positivity (Pe). Though closely related temporally, the ERN/Ne and Pe reflect distinct stages of error-related processing. The ERN/Ne reflects initial, automatic error-correction and action-monitoring processes (Falkenstein et al., 1991; Gehring et al., 1993; Yeung & Summerfield, 2012). However, the Pe is involved in later, more elaborate error-processing stages, indexing the accumulation of error-related information (Yeung & Summerfield, 2012), including the motivational (Ullsperger et al., 2010) or affective (Overbeek et al., 2005) appraisal of such stimuli. Additionally, the ERN/Ne is said to arise within the cognitive, caudal division of the ACC (cACC), whereas both the caudal and rostral portions (rACC) of the ACC contribute to Pe amplitude (Edwards et al., 2012; van Veen & Carter, 2002). However, recent evidence suggests that the ERN/Ne may be generated by the PCC (Agam et al., 2011), whereas the insula may additionally contribute to the Pe (Schröder et al., 2012; Ullsperger et al., 2010).

In adult psychopathic offenders, several studies have found comparable ERN/Ne amplitudes between adult psychopaths and control groups when using affectively neutral stimuli (Brazil et al., 2009; Brazil et al., 2011; Maurer et al., in press; Munro et al., 2007; Steele et al., 2016; von Borries et al., 2010). However, reduced ERN/Ne amplitude has been observed in adult psychopathic offenders when incorporating evocative angry and fearful facial stimuli (Munro et al., 2007).

Compared to the ERN/Ne, disparate findings have been observed regarding Pe amplitude in adult psychopathic offenders. Two previous reports with adult males and females have associated reduced Pe amplitude with increased psychopathy scores (Brazil et al., 2009; Maurer et al., in press). However, a recent report associated increased Pe amplitude with higher psychopathy scores in an incarcerated male sample (Steele et al., 2016). Intact ERN/Ne and deficits in Pe amplitude in adult offenders with elevated psychopathic traits suggests that this population can detect that an error has occurred, but exhibit specific dysfunction in regards to post-error processing. Reduced Pe amplitude in adult psychopathic offenders suggests a specific deficit in using information received from errors to improve future behavior (Brazil et al., 2009; Maurer et al., in press), which may partly explain this population’s increased propensity towards perseverance in experimental learning paradigms (Newman & Kosson, 1986). Importantly, the Pe component has been shown to be malleable, increasing in amplitude through mindfulness meditation intervention (Larson et al., 2013). Thus, the present study sought to examine whether adolescent psychopathy scores were associated with reduced Pe amplitude. If hypotheses are confirmed, the Pe may be a target for future interventions, such as mindfulness, to help ameliorate dysfunctional post-error processing.

Despite interest in the electrophysiological correlates of 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 Go/NoGo paradigm in a sample of incarcerated male adolescents. Psychopathic traits were assessed using the Hare Psychopathy Checklist: Youth Version (PCL:YV) (Forth et al., 2003), a downward extension of the Hare Psychopathy Checklist–Revised (PCL–R) (Hare, 2003) modified for age appropriateness.

Based on previous error-related ERP studies performed with adult psychopathic offenders (Brazil et al., 2009; Brazil et al., 2011; Maurer et al., in press; Munro et al., 2007; Steele 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 previous studies with adult psychopathic offenders (Brazil et al., 2009; Maurer et al., in press), but contrary to a recent report with adult psychopathic male offenders (Steele et al., 2016). An increased Pe amplitude observed with adult psychopathic male offenders may result from a compensatory mechanism, attempting to overcome initial post-error processing deficits experienced as adolescents. 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 overlapping variance both between and within ERP components (Bernat et al., 2011; Dien et al., 2007). This approach has been incorporated in a number of reports (Anderson et al., 2013; Maurer et al., in press; Steele et al., 2015; Steele et al., 2014; Steele et al., 2016), providing a more sensitive and predictive measure compared to traditional time-domain ERP analyses. In the current report, four principal components were extracted, one reflecting mean ERN/Ne amplitude, and the remaining three reflecting early, middle, and late subcomponents underlying the Pe (see Fig. 1). The 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). In particular, the early Pe subcomponent has been previously associated with both cACC and rACC activity, the middle Pe subcomponent has been associated with cACC activity, and the late Pe subcomponent has been associated with rACC activity (Edwards et al., 2012). In regards to PCA analyses, we hypothesized adolescent psychopathy scores would be negatively related to a middle subcomponent underlying the Pe, which has been shown to be dysfunctional in previous reports (Maurer et al., in press; Steele et al., 2016).

2. Method

2.1. Participants

Participants were 142 incarcerated adolescents at a maximum-security juvenile detention center who participated in a larger study (SWAINTS Intervention: Youths Incarcerated and Non-Incarcerated). 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 category (2%).

Incarcerated adolescents are considered a vulnerable population for research, so extra precautions were taken in order to minimize the potential for coercive influences that could reduce their ability to provide voluntary consent to participate (Edens et al., 2011; Costin et al., 2007). For example, potential study participants may feel inclined to participate in research in order to relieve boredom and interact with people from outside the prison (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 detention center by trained research staff (not correctional staff). Meetings were scheduled with interested participants, providing them the opportunity to make an informed choice about participating. Participants 18 years of age or older 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.

2.1.1. Assessments

Psychopathic traits were assessed by trained research assistants, graduate students, and postdoctoral researchers using the PCL:YV (Forth et al., 2003). The PCL:YV assesses interpersonal, affective, behavioral, and lifestyle features related to psychopathic traits in adolescents. Total scores can range from 0 to 40. There is currently no accepted diagnostic cutoff for youth psychopathy. For identification of specific psychopathic traits associated with electrophysiological error-related indices, we used a two-factor model of psychopathic traits, with Factor 1 comprising interpersonal and affective traits and Factor 2 consisting of lifestyle and antisocial traits (Harpur et al., 1989). To allow for increased specificity, we also examined the four-facet model with four latent dimensions representing the underlying dimensions of psychopathy: interpersonal, affective, behavioral/lifestyle, and antisocial traits, respectively (Neumann et al., 2006). The mean PCL:YV Total score for this sample was 23.83 (SD = 6.46). 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 remaining correlations.

In addition to psychopathic traits, assessments were administered to assess intelligence quotient (IQ), substance dependence, mental illness, and traumatic brain injury (TBI) by trained research assistants, graduate students, and postdoctoral researchers. Exclusion criteria included: a full-scale IQ less than 70 ($n = 0$), a TBI accompanied with a significant loss of consciousness ($n = 4$), poor behavioral performance or significant movement during data collection ($n = 16$), or personal history of bipolar or psychotic disorders ($n = 0$). Participants were also excluded for mood disorders, including major depression ($n = 10$), and anxiety disorders, including post-traumatic stress disorder (PTSD) ($n = 3$), due to the important role these disorders play for both the ERN/Ne (Chiu & Deldin, 2007; Olvet & Hajcak, 2008) and Pe (Bridwell et al., 2015) amplitude.
Table 1
Correlations among PCL:YV Variables and Covariates.

<table>
<thead>
<tr>
<th>Variable</th>
<th>PCLYV Total</th>
<th>PCLYV Factor1</th>
<th>PCLYV Factor2</th>
<th>PCLYV Factor3</th>
<th>PCLYV Factor 4</th>
<th>Age</th>
<th>IQ</th>
<th>Sub. Use</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCLYV Total</td>
<td>–</td>
<td>0.86**</td>
<td>0.90**</td>
<td>0.66**</td>
<td>0.78**</td>
<td>0.80**</td>
<td>0.78**</td>
<td>0.83**</td>
</tr>
<tr>
<td>PCLYV Factor 1</td>
<td>0.86**</td>
<td>–</td>
<td>0.58**</td>
<td>–</td>
<td>0.84**</td>
<td>0.60**</td>
<td>0.86**</td>
<td>0.86**</td>
</tr>
<tr>
<td>PCLYV Factor 2</td>
<td>0.90**</td>
<td>0.58**</td>
<td>–</td>
<td>0.39**</td>
<td>0.42**</td>
<td>0.34**</td>
<td>0.30**</td>
<td>0.26**</td>
</tr>
<tr>
<td>PCLYV Factor 3</td>
<td>0.66**</td>
<td>0.85**</td>
<td>0.39**</td>
<td>–</td>
<td>–</td>
<td>0.54</td>
<td>0.56**</td>
<td>0.26**</td>
</tr>
<tr>
<td>PCLYV Factor 4</td>
<td>0.78**</td>
<td>0.84**</td>
<td>0.60**</td>
<td>0.42**</td>
<td>–</td>
<td>0.34**</td>
<td>0.56**</td>
<td>0.32**</td>
</tr>
<tr>
<td>Age</td>
<td>0.08</td>
<td>0.08</td>
<td>0.15</td>
<td>0.15</td>
<td>0.05</td>
<td>0.02</td>
<td>0.05</td>
<td>0.05</td>
</tr>
<tr>
<td>IQ</td>
<td>–0.05</td>
<td>0.07</td>
<td>–0.12</td>
<td>0.19</td>
<td>–0.07</td>
<td>–0.14</td>
<td>0.05</td>
<td>0.05</td>
</tr>
<tr>
<td>Sub. Use</td>
<td>0.33**</td>
<td>0.24**</td>
<td>0.30**</td>
<td>0.15</td>
<td>0.26**</td>
<td>0.32**</td>
<td>0.22**</td>
<td>0.07</td>
</tr>
</tbody>
</table>

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 Factor 1, Facet 1, Facet 2, and Facet 3, and Facet 4 scores are Factor 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 (WASI-III) (Wechsler, 1997); 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.

Table 2
Descriptive statistics and independent samples t-tests for variables.

<table>
<thead>
<tr>
<th>Variable</th>
<th>All Participants (N = 100)</th>
<th>PCL:YV Lower Quartile (N = 22)</th>
<th>PCL:YV Higher Quartile (N = 21)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>PCL:YV Total</td>
<td>100</td>
<td>23.82</td>
<td>6.46</td>
</tr>
<tr>
<td>PCL:YV Factor 1</td>
<td>100</td>
<td>6.75</td>
<td>3.19</td>
</tr>
<tr>
<td>PCL:YV Factor 2</td>
<td>100</td>
<td>12.78</td>
<td>3.20</td>
</tr>
<tr>
<td>PCL:YV Factor 1</td>
<td>100</td>
<td>2.24</td>
<td>1.91</td>
</tr>
<tr>
<td>PCL:YV Factor 2</td>
<td>100</td>
<td>4.51</td>
<td>1.87</td>
</tr>
<tr>
<td>PCL:YV Factor 3</td>
<td>100</td>
<td>6.52</td>
<td>2.02</td>
</tr>
<tr>
<td>PCL:YV Factor 4</td>
<td>100</td>
<td>8.18</td>
<td>1.81</td>
</tr>
<tr>
<td>Age</td>
<td>100</td>
<td>17.38</td>
<td>0.86</td>
</tr>
<tr>
<td>IQ</td>
<td>91</td>
<td>93.90</td>
<td>10.97</td>
</tr>
<tr>
<td>Substance Use</td>
<td>100</td>
<td>2.33</td>
<td>1.67</td>
</tr>
<tr>
<td>ER/N/Ne</td>
<td>100</td>
<td>–3.17</td>
<td>4.39</td>
</tr>
<tr>
<td>PC1</td>
<td>100</td>
<td>6.25</td>
<td>6.78</td>
</tr>
<tr>
<td>PC2</td>
<td>100</td>
<td>0.31</td>
<td>0.41</td>
</tr>
<tr>
<td>PC3</td>
<td>100</td>
<td>0.64</td>
<td>0.50</td>
</tr>
<tr>
<td>PC4</td>
<td>100</td>
<td>0.03</td>
<td>0.21</td>
</tr>
</tbody>
</table>

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 Factor 1, Facet 1, Facet 2, Facet 3, and Facet 4 scores are Factor 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 (WASI-III) (Wechsler, 1997); 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); ER/N/Ne, Pe, PC1, PC2, PC3, and PC4 refer to the mean amplitude of the time-domain and principal components pertaining to ER/N/Ne and Pe amplitude.

Female participants (n=9) were excluded from final analyses, as there were not enough female participants to power gender effects. Additionally, participants (n=0) were excluded for making fewer than four errors. Reliability analyses suggest that the ER/N/Ne and Pe cannot be quantified as few as four to six trials (Olvet & Hajcak, 2009; Pontefix et al., 2010; Steele et al., in press). This resulted in a final sample of n=100 male participants, ranging from 16 to 20 years of age (M=17.38, SD=0.86) at the time of electroencephalography (EEG) collection (Table 2).

We included the following covariates in addition to psychopathy variables in linear regression analyses: age, IQ, and number of substance dependences. Full-scale IQ was estimated using the Vocabulary and Matrix Reasoning sub-tests of the Wechsler Adult Intelligence Scale (WASI-III) (Wechsler, 1997) (M=93.90, SD=10.93); IQ scores were unavailable for n=9 participants. Mental illness and substance dependence were assessed using the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS) (Kaufman et al., 1997). Number of substance dependences were calculated by summing the total number of substances (alcohol and drug) for which participants met lifetime dependence diagnoses (possible range 0–9, M=2.33, SD=1.68), as psychopathic traits are often comorbid with substance use (Smith & Newman, 1990; Walsh et al., 2007). Complete collection of the full assessment protocol is difficult due to the nature of the prison institutional environment (e.g. unannounced facility transfers, early release, disciplinary actions, etc.).

2.2. Stimuli and task

EEG data were collected in a quiet, dimly lit room, reserved for EEG data collection at the corrective facility, separate from the general population housing. Participants were collected between 8:00 AM to 5:00 PM. After placement of electrodes, participants were seated in a comfortable chair 60 cm away from a computer monitor on which the task stimuli were presented and were instructed to refrain from excessive blinking and movement during data collection. Participants then performed a response inhibition Go/NoGo task (Kiehl et al., 2000) consisting of two experimental runs, each comprising 245 visual stimuli, each about seven minutes in duration. After the first run, participants were afforded the opportunity to take a break to minimize fatigue. Stimulation was 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 distractor NoGo stimulus (a white ‘K’) appeared. The stimuli subtended approximately 3 × 5 visual degrees against a black background. Targets appeared with higher frequency (84%, 412 trials, with 206 on each run) than distractors (16%, 78 trials, with 39 on each run) to establish a strong stimulus-response mapping on Go trials. Two ‘Ks’ were never presented sequentially. The inter-stimulus interval was pseudo-randomly jittered (1–3 s stimulus onset asynchrony [SOA] averaging 1.5 s). The SOA between Go stimuli varied to the constraint that three Go stimuli were presented within each 6 s 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 8 to 14 s. Hits were defined as successful responses to Go stimuli; False Alarms (FAs) were defined as incorrect responses to NoGo stimuli. Prior to recording, each participant performed a block of ten practice trials to ensure that directions were clearly understood.

2.3. EEG recordings

EEG data were collected using two computers and a 64-channel Biopac 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 acquired electroencephalographic data using Biopac software and amplifiers. All signals collected with the Biopac software were low-pass filtered using a fifth-order sinc filter with a half-power cutoff of 204.8 Hz, then digitized to 1024 Hz during data collection. EEG activity was recorded using sintered Ag-AgCl active electrodes placed in accordance with the 10–20 International System. The participant’s nose was used as a reference. Six electrodes were 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.

2.4. 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 et al., 2000) blink components were identified and removed from the data.

Classic time-domain response-locked ERP components, relative to a False Alarm were extracted: the ERN/Ne, the mean amplitude of the negative deflection occurring 0–100 ms and the Pe, the mean amplitude of the positive deflection, occurring 94–500 ms. Response-locked components were baseline corrected with a −200 to −50 ms window relative to FAs. 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 component solution for FA stimuli accounting for 94.97% of the variance. A subset of nine electrodes representing maximal time-domain component activation were selected for the ERN/Ne and Pe (AF3, AFz, AF4, F3, Fz, F4, FC3, FCz, and FC4) and used in both time-domain and PCA analyses below. Linear regression analyses were carried out on all n = 100 participants to predict mean time-domain and PCA decompositions reflecting mean ERN/Ne and Pe amplitude using psychopathy variables and three covariate measures: age, IQ, and number of substance dependencies to take full advantage of our large sample size. Effects that did not reach statistical trend (p > .10) are not reported.

3. Results

3.1. Behavioral results

Response times (RTs) and frequency for Hits and FAs were analyzed. As expected, participants responded faster to NoGo stimuli (M = 381 ms, SD = 43 ms) than Go stimuli (M = 419 ms, SD = 51 ms), t(99) = 7.74, p < .001. Participants made significantly more errors (FAs) to NoGo stimuli (M = 23.76, SD = 11.85, range 4–63) compared to Go (Hits) stimuli (M = 12.61, SD = 14.65), t(99) = 17.89, p < .001. There was a main effect for post-error slowing (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 responded more slowly after error trials (M = 384 ms, SD = 84) than after correct trials (M = 356 ms, SD = 33 ms), t(99) = 3.86, p < .001. PES did not correlate with PCL:YV variables (Total, Factor, or Facet scores); covariate measures, time-domain, or PCA measures reflecting ERN/Ne and Pe mean amplitude (r’s < .15). Psychopathy variables and other covariates were not significantly correlated with response times or error rates (r’s < .15).

3.2. Time-domain ERP linear regression analyses

Separate linear regressions were performed to assess unique contributions to mean ERN/Ne and Pe amplitude measured with traditional classic windowed ERP components and PCA 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 variable and in the other three regressions Pe mean amplitude was the dependent variable). PCL:YV measures (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. PCL:YV Facet scores were measured before Factor scores, as they provide a more precise measure of specific psychopathic traits (Neumann et al., 2006).

In addition, we implemented a Simes-Hochberg correction to all linear regression analyses (Hochberg, 1988; Simes, 1986) to maintain the 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.
Using the Simes-Hochberg correction, neither PCL:YV scores (Total, Factor, and Facet scores) or covariate measures included in analyses (age, IQ, and number of substance dependencies) were significant predictors of either ERN/Ne or Pe mean amplitude.

3.3. PCA ERP linear regression analyses

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 [PC1, PC2, and PC4]) explained by PCL:YV variables. Like before, PCL:YV Total, Factor, or Facet scores were the predictor variables of interest in three separate regression models for each ERP subcomponent, along with age, IQ, and number of substance dependencies as covariates.

Using the Simes-Hochberg correction, neither psychopathy variables or the three covariates were significant predictors of PC3 mean amplitude, reflecting the ERN/Ne. Similarly, psychopathy variables and covariate measures were not significant predictors of PC1 or PC4 mean amplitude, reflecting early and late subcomponents underlying the Pe. Elevated psychopathy scores were negatively related to PC2 mean amplitude, reflecting the middle subcomponent underlying the Pe. In separate linear regression analyses, PCL:YV Total (p = .037) and Facet 4 (antisocial traits) (p = .011) emerged as significant predictors of reduced PC2 mean amplitude (Table 3). PCL:YV Factor 2 scores emerged as a marginally significant predictor of reduced PC2 mean amplitude (p = .039) with the implementation of the Simes-Hochberg correction.

4. Discussion

Psychopathy is a serious personality disorder with enormous societal cost. Given the importance of this construct, research has sought to understand the adolescent manifestation of this condition. The prevailing view is that intervention efforts targeted towards youth will have a better chance of altering life-course persistent antisocial behavior if started early (Caldwell, 2011; Caldwell et al., 2007). Here, we examined whether adolescent psychopathy scores were associated with reduced Pe amplitude. Participants provided informed consent, and were not coerced in any way to participate in the current study, which is especially important when collecting data from incarcerated settings (Edens et al., 2011; Gostin et al., 2007).

In the current report, psychopathy scores were not related to ERN/Ne amplitude, consistent with previous reports with adult offenders with elevated psychopathic traits when using affectively neutral stimuli (Brazil et al., 2009; Brazil et al., 2011; Maurer et al., in press; Munro et al., 2007; Steele et al., 2016; von Borries et al., 2010). However, adolescent psychopathy scores were negatively related to Pe amplitude, consistent with two previous reports with adult males and females with elevated psychopathic traits (Brazil et al., 2009; Maurer et al., in press), but inconsistent with a recent report with incarcerated adult males with elevated psychopathic traits (Steele et al., 2016). Taken together, our current results suggest that adolescent psychopathy scores, specifically Facet 4 (antisocial) traits, are not associated with dysfunctional error-correction and action-monitoring processes (Falkenstein et al., 1991; Gehring et al., 1993; Yeung & Summerfield, 2012), but are associated with specific deficits in post-error processing.

Our results were most strongly supported through the use of PCA to separate overlapping variance between and within ERP components. PCA identified three subcomponents underlying the time-domain Pe component: an early, middle, and late subcomponent. Elevated psychopathic scores were negatively related to the middle subcomponent underlying the Pe in Principal Component 2. This subcomponent has a similar temporal distribution as a subcomponent defined in a previous report associated with cACC activity (Edwards et al., 2012). Reduced Pe amplitude has also been suggested as a specific deficit in using information received from errors to improve future behavior (Brazil et al., 2009). This Pe reduction could help explain a variety of deficits youth with elevated psychopathic traits experience, including increased behavioral impulsivity (Roussy & Toupin, 2000), and reduced performance in passive avoidance learning paradigms (Finger et al., 2008).

PCL:YV Facet 4, reflecting antisocial and criminogenic behavioral traits, emerged as a significant predictor of reduced Pe mean amplitude. This antisocial facet includes traits directly tapping into the early onset of psychopathic traits, including early behavioral problems before the age of 12 (Forth et al., 1990; Neumann et al., 2011). The emergence of early antisocial behavior in youth is an important predictor for the development of psychopathy in adulthood (Frick et al., 2003). Some remain apprehensive over the inclusion of this 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). Others argue that eliminating this antisocial facet may result in a considerable narrowing of the psychopathy construct, particularly in regards to the developmental course of psychopathic traits (Hare & Neumann, 2010; Lynam, 1997; Neumann et al., 2011).

Results of the present study indicate Facet 4 traits contribute considerable importance for future research investigating the neurodevelopmental course of psychopathy. A negative association between Facet 4 traits and Pe mean amplitude may enable researchers to identify a subset of youth on a life-course persistent trajectory towards severe antisocial behavior. Specifically, reduced post-error processing of errors may result in an inability for such youth 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).

The examination of the Pe with an at-risk juvenile sample is particularly intriguing within 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 amplitudes between youth and adult samples (Davies et al., 2004; Ladouceur et al., 2007; Santesso et al., 2006). Reduced Pe amplitude could suggest a potential biological vulnerability marker for the development of life-course persistent psychopathic traits. Furthermore, a recent report associated increased Pe amplitude with elevated psychopathic traits in an incarcerated adult male sample (Steele et al., 2016). Increased Pe amplitude in adulthood may reflect a compensatory mechanism, attempting to overcome initial post-error processing deficits experienced in adolescence in individuals with elevated psychopathic traits. Additionally, Pe amplitude has been shown to increase through mindfulness meditation training (Larson et al., 2013). These results suggest 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.

4.1. Limitations

Psychopathic traits, at least at 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 using 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 diagnostic criteria of psychopathy. Longitudinal research is desperately needed to measure whether reduced Pe amplitude in youth samples can serve as a potential biomarker for the development of psychopathic personality.

Additionally, our study recruited participants from a maximum-security detention center. Compared to youth in community samples, youth in incarcerated settings differ on a number of variables, including substance use history, general intelligence, and trait anxiety (Foley, 2001; Wasserman et al., 2002). We note that our sample 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 sample should be considered one with clinical levels of psychopathy, which may not extrapolate to samples with lower psychopathy scores. Moreover, there appears to be little agreement between various self-report and interview-based measures of psychopathic traits in adolescent samples (Fink et al., 2012). We recommend that future studies compare samples on identical measures of psychopathic traits; comparison across assessment instruments is not likely to lead to replication.

In addition, the present study reported a negative relationship between adolescent psychopathy scores and Pe mean amplitude within an incarcerated sample with clinical levels of psychopathy. As it is common practice in the psychopathy field, we tested our hypotheses by examining participants with low to high levels of psychopathy within this sample. This allows us to carefully control for potential moderating variables (i.e., substance use, etc.). However, we did not compare our results to a 'healthy' population; thus, our results need to be considered in this light. Future research should attempt to replicate and extend our current results incorporated a non-incarcerated control group, carefully attending to critical moderating variables (IQ, substance use, etc.).

5. Conclusions

In sum, adolescent psychopathy scores were unrelated to the ERN/Ne mean amplitude, and 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. Linear regression analyses associated reduced amplitude of this subcomponent underlying the Pe with PCL:YV Facet 4 (antisocial traits), including early behavioral problems. This is the first evidence to suggest a potential negative relationship between adolescent psychopathy scores and Pe mean amplitude.

References


Table 3

<table>
<thead>
<tr>
<th>Predictors</th>
<th>B</th>
<th>SE B</th>
<th>t</th>
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<th>Sig.</th>
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<tr>
<td>PCL:YV Total</td>
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<td>IQ</td>
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<td>Age</td>
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<td>Regression 2:</td>
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Regression 1: $R^2 = .048$, $R = .218$, $F(1,89) = 4.461$.
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 Factor 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); 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) (Kauffman et al., 1997).


