Electrophysiological indices of information processing in psychopathy

by

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ABSTRACT

Psychopathy is a severe personality disorder associated with a range of affective, interpersonal, and behavioural abnormalities. Evidence suggests that psychopaths show marked deficits in processing emotional information, although it is unclear whether they also show more general deficits in error monitoring, attention allocation and response control. It is also unclear whether any variation in neurophysiological performance is also reflected in subclinical populations. In this thesis, event-related potentials (ERPs) were used to examine these issues and involved two separate samples. The first included incarcerated offenders with a range of scores on the Hare Psychopathy Checklist –Revised (PCL-R) and non-offender (staff) controls. The second included a large group of healthy undergraduate males with a full range on scores on the Self-Report Psychopathy Scale (SRP-III).

Error monitoring was examined in both samples using a standard letter-flanker task and a modified version of the task in which faces with angry or fearful expressions were used instead of the usual letter stimuli. In general, psychopathy in both samples was associated with attenuated ERN amplitudes on the face flanker task only. Source modeling of the ERN indicated that, while the ERN is generally modeled as having a dipole in the ACC, the psychopath group showed no evidence of ACC activity in this region in conjunction with face-flanker errors. These data suggest that the affect-based neurophysiological deficits associated with psychopathy in the clinical range are observed in a graduated fashion among subclinical samples.

Inhibitory control processes were also examined in the incarcerated group using the inhibitory N2 and anteriorized P3 as indices of inhibitory processes evident in correctly withholding prepotent response tendencies on a Go-NoGo task. Despite the common assumption that poor inhibitory control is a central aspect of psychopathy, there was no sign that those at higher levels of psychopathy showed any inhibitory control problems and they produced a robust NoGo N2 and P3. In fact, there were signs that the incarcerated offenders who were low on psychopathy were more likely to produce diminished inhibitory-related components.

Finally, years of controversy regarding attention allocation deficits in psychopathy was addressed by collecting standard P3 components during a traditional visual oddball task in the university sample. Behavioural response and P3 amplitudes were unrelated to psychopathy. However, consistent with data from incarcerated samples, higher scores on psychopathy were associated with larger amplitude P2 and N5 responses to target relative to nontarget stimuli, again suggesting some continuity with respect to a distinct, although not necessarily deficient, attentional style at subclinical levels of psychopathy. In general, across these four data sets, the only clear evidence of impaired processing involved a reduced error-monitoring response during the face-flanker task when emotional stimuli formed the basis of the required discrimination and this reduced response was found to vary with the degree of psychopathy even within a subclinical range. These findings support a model of psychopathy involving limbic and paralimbic structures rather than a general

reduction in neural function affecting error monitoring, attention allocation and response control.

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CHAPTER ONE. INTRODUCTION

Psychopathy is a personality disorder characterized by a range of affective and behavioral abnormalities (Cleckley, 1941; Hare, 1991, 2003). Early descriptions of the condition can be traced back to the writings of the Greek philosopher Theophrastus (c. 319 BC/2004), although psychopathy did not receive a great deal of clinical attention until the work of Hervey Cleckley in the twentieth century. The American Psychological Association's Diagnostic and Statistical Manual of Mental Disorders (2000) describes psychopathy as an analogue to antisocial personality disorder although there are conceptual differences that separate these conditions (Hare, 1996). The prevalence of psychopathy is thought to be lower than that of antisocial personality disorder, at approximately one percent in the general population and between 15% and 25% in forensic populations (Hare, 1991, 2003). Psychopathy poses a significant challenge to the criminal justice system, as psychopaths commit a disproportionate number of serious offenses, and are at a higher risk of recidivism than are nonpsychopaths (Hemphill, Hare, & Wong, 1998) but treatment has been found to be generally ineffective, and at times can lead to worse outcomes than no treatment at all (Rice, Harris, & Cormier, 1992). However, despite this disorder presenting significant costs to society and the justice system, relatively little is known about the etiology or the mechanisms underlying this condition.

It is currently unclear whether psychopathy is qualitatively different from other types of antisocial behaviours, or whether it is a point on a spectrum

of antisocial or malevolent personality traits. There is evidence supporting both perspectives. Several studies have indicated that psychopathy likely reflects a latent taxometric class (Harris, Rice, Hilton, Lalumiere, & Quinsey, 2007; Harris, Rice, & Quinsey, 1994; Skilling, Quinsey, & Craig, 2001). For example, Harris, Rice and Quinsey (1994) used several methods to conduct taxometric analyses of 653 offenders scored on the Hare Psychopathy Checklist – Revised (PCL-R Hare, 1991, 2003), a clinical measure of psychopathy. When considered in combination, PCL-R score, and a childhood history of antisocial behaviour suggested that psychopathy was a discrete class of antisocial behaviour, and that this was particularly true of for Factor Two traits. In a similar study with antisocial juveniles, Skilling, Quinsey and Craig (2001) examined over one thousand boys with an average age of 12. Scores on a range of factors relating to measures such as conduct disorder and the Youth Version of the Psychopathy Checklist (Forth, Hart & Hare, 1990). This provided evidence that a discrete taxon underlay the measures of antisocial behaviour they had used. Unlike Harris et al (1994), Skilling et al. found that it was the personality traits associated with PCL Factor One items that were the strongest taxon indicators. However, these results have been inconsistently replicated, and even Harris et al (1994) found no evidence supporting a latent taxonomy for Factor One of the PCL-R. Factor One reflects the affective and interpersonal aspects of the disorders, including personality traits such as grandiosity and lack of emparthy.

Conversely, there is also persuasive evidence that psychopathic personality traits may vary within the normal population (Edens, Marcus, Lilienfeld, & Poythress, 2006; Marcus, John, & Edens, 2004; Walters, Diamond, Magaletta, Geyer, & Duncan, 2007; Walters, Duncan, & Mitchell-Perez, 2007; Walters, Gray et al., 2007). For example, Guay, Ruscio, Knight, and Hare (2007) conducted a taxometric analysis of over 4000 offenders and found no evidence that psychopathic personality traits reflect a unique or discrete class, but rather concluded that it should be considered as a cluster of extreme personality traits - all of which are normally distributed throughout the population. Likewise, Edens et al (2006) examined almost 900 offenders and substance abusers who had been assessed with the PCL-R, and found no evidence supporting a latent taxonomy. Thus, it is currently unclear whether psychopathy should be treated as a discrete category of personality disorder or as the extreme on a continuum of normally-distributed psychopathic personality traits. However, if there were evidence of a continuum, this would offer support for the study of this condition in nonclinical (and nonforensic) populations.

Laboratory Findings

Psychopathy has been associated with a broad spectrum of laboratory findings involving both affect and cognition. For example, psychopaths have been reported to be slower (Blair et al., 2004) and less accurate (Habel, Kuhn, Salloum, Devos, & Schneider, 2002; Kosson, Suchy, Mayer, & Libby, 2002; Marsh & Blair, 2008; Montagne et al., 2005) than nonpsychopaths when recognizing affect in voices and facial expressions, an effect that has been

shown to correlate with degree of emotional detachment (Habel et al., 2002). Psychopaths have also been reported to show less affective interference in the recall of emotional events (Christianson et al., 1996), and less emotional priming than nonpsychopaths when shown affectively negative images (Benning, Patrick, & Iacono, 2005; Patrick, Cuthbert, & Lang, 1993; Patrick, Cuthbert, & Lang, 1994). It also appears that psychopaths do not show the expected autonomic differentiation between unpleasant and pleasant visual images (Patterson & Newman, 1993), an effect which positively correlates with the psychopaths' degree of emotional detachment.

There are also reports that psychopaths process language abnormally under a variety of circumstances (Day & Wong, 1996; Intrator et al., 1997; Kiehl et al., 2004; Reidy, Zeichner, Hunnicutt-Ferguson, & Lilienfeld, 2008; Williamson, Harpur, & Hare, 1991). One example of this occurs on lexical decision tasks, which require participants to recognize and identify whether neutral and affective words are real words or nonword letter strings. In nonpsychopaths, affective words prompt faster reaction times, a process known as response facilitation because it is believed that the presence of semantic information concerning the emotionality of affective words aids in their identification. Williamson et al. (1991) used such a task, and found that affective words failed to elicit response facilitation in psychopathic offenders. Psychopaths in their sample also failed to show different electrophysiological responses for emotional and neutral classes of words. This suggests that

psychopaths may fail to utilize the affective component of semantic information when responding to emotional relative to neutral stimuli.

Atypical behavioural and neurophysiological responses to aversive stimuli have also been reported and include deficits in passive avoidance learning (Lykken, 1957; Newman & Schmitt, 1998), electrodermal hypoactivity in anticipation of aversive stimuli (Fung et al., 2005; Gatzke-Kopp, Raine, Loeber, Stouthamer-Loeber, & Steinhauer, 2002; Hare, 1978), and an attenuated startle response to unpleasant images (Patrick et al., 1993; Patrick et al., 1994). In addition, psychopaths are relatively insensitive to aversive conditioning contingencies (e.g. Flor, Birbaumer, Hermann, Ziegler, & Patrick, 2002) and generally do not show autonomic responses to conditioned stimuli under such conditions. Results from several imaging studies indicate that this phenomenon is accompanied by a pattern of attenuated neurological responses to aversive conditioning, primarily in limbic regions such as the orbitofrontal cortex, anterior cingulate, and amygdala. These areas are responsible for both the activation of conditioned responses and the processing of emotionally-relevant information (Birbaumer et al., 2005; Veit et al., 2002).

Although a major focus of research in psychopathy has been on the affective component of the disorder, there are also concerns about various aspects of attention allocation and control. For example, psychopaths have been reported to show deficits in orienting, disengaging, and reorienting selective attention during task performance (Kosson, 1996), especially during goal- or reward-driven tasks, and they appear to be less responsive to peripheral task

contingencies and motivationally neutral cues than nonpsychopaths (Bernstein, Newman, Wallace, & Luh, 2000; Hiatt, Schmitt, & Newman, 2004; Newman, Schmitt, & Voss, 1997). Not only are psychopaths reported to have difficulty spontaneously using such motivationally neutral cues to adjust their dominant response pattern (Newman & Kosson, 1986; Newman et al., in press), they seem also to be less sensitive to motivationally neutral peripheral information of all kinds. For example, Hiatt, Schmitt, and Newman (2004) reported that psychopaths showed reductions in interference on some variations of the Stroop task. This suggests that psychopaths may be less sensitive to information which is not integrated with, or central to, goal- or reward-driven behaviour.

There have also been a number of reports of reduced inhibitory control in psychopaths, especially when the inhibition of a prepotent response is required (Howland, Kosson, Patterson, & Newman, 1993; Kiehl, Smith, Hare, & Liddle, 2000; LaPierre, Braun, & Hodgins, 1995). One electrophysiological marker of response inhibition is the frontal N2. This component is thought to represent response inhibition processes likely initiated in orbital frontal cortex (Goldstein & Volkow, 2002) and the anterior cingulate cortex (ACC), a region implicated in behavioural control (Bekker, Kenemans, & Verbaten, 2005; Bokura, Yamaguchi, & Kobayashi, 2001; van Veen & Carter, 2002a). There is also some evidence that psychopathy is associated with attenuated N2 amplitudes during a visual Go/NoGo task (Kiehl, Smith et al., 2000) and such data have been used to support the view that psychopathy involves a fairly broad-based series of deficits that extend beyond abnormal affective response.

Theoretical Perspectives

The most influential model of psychopathy in the twentieth century was proposed by Cleckley (1941), who described the disorder as being essentially an emotion-specific form of "semantic dementia" in which psychopaths were unable to appreciate the emotional ramification of life events. Many of the models that followed also accepted the presence of a fundamental deficit in some aspect of emotional processing in psychopaths. A prominent example of this is the low fear hypothesis (Lykken, 1957), a position supported by electrodermal hyporeactivity in anticipation of aversive stimuli (Hare, 1978) and deficits in passive avoidance learning and punishment avoidance (Newman, Widom, & Nathan, 1985). A similar model proposed that psychopathy might be associated with a weak Behavioural Inhibition System (BIS). Reductions in BIS activity could result in lower levels of anxiety and increased approach responses under circumstances in which most individuals would show avoidance responses. This would result in lower trait fear and lower levels of anxiety. Some authors (e.g. Fowles & Dindo, 2006; Lykken, 1995) argue that this model accounts for many of the physiological deficits seen in psychopaths. According to this model, atypical septohippocampal function may also be associated with the condition (Gray & McNaughton, 2000).

According to the response modulation hypothesis (Newman, 1998) psychopaths display a failure to monitor, and hence learn from, the response-based feedback they encounter. This perspective suggests that atypical autonomic responses such as decreased skin conductance as well as the failure

to acquire classically conditioned responses may reflect a deficiency in selective attention rather than indicating affective deficits *per se*. Like the previous model, Newman and colleagues (in press) also argue that these cognitive deficits may be the result of septohippocampal (and by extension, BIS) hypoactivation, although there is limited evidence supporting this position. However, there is support for the presence of attentional abnormalities in psychopaths, such as difficulty orienting and reorienting selective attention during task performance (Kosson, 1996).

Proponents of this atypical attention perspective argue that suboptimal autonomic responses such as decreased skin conductance and failure to acquire classically conditioned responses reflect deficits in selective attention – psychopaths simply do not attend to the relevant feedback cues or peripheral affective information that would normally guide behaviour under such circumstances. Thus, based on this model, deficits in emotional processing seen in psychopathy are not due to limbic dysfunction, but rather to failures of attention.

A second attention-based theory was postulated by Raine and Venables (1988), who hypothesized that the presence of atypical attention in psychopathy does not necessitate a deficit model. Rather, they thought that psychopathy may be associated with abnormalities in attention, such that psychopaths may over-focus and have a heightened ability to direct selective attention to events that are of immediate interest (Raine & Venables, 1987, 1988) while ignoring (potentially useful) peripheral information. Raine and Venables (1987; 1988)

conducted a series of studies examining the P3, an ERP index thought to relate to attentional allocation. Using a continuous performance task to elicit the P3 component, the authors reported that both antisocial adolescents and psychopathic offenders generated P3s of significantly larger amplitudes than did normal adolescents or nonpsychopathic offenders. From this, they concluded that although psychopaths may demonstrate atypical attentional allocation, it may not necessarily be due to a generalized deficit in attentional processes *per se*.

There are also a number of theoretical perspectives that are based more directly on specific types of brain dysfunction. A number of these models support the centrality of amygdala dysfunction. For example, according to Blair's violence inhibition mechanism model (Blair, 1995; Blair, Jones, Clark, & Smith, 1997; Blair, Peschardt, Budhani, Mitchell, & Pine, 2006), amygdala dysfunction impairs the normal development of cognitive mechanisms that promote empathy and discourage antisocial behaviour. This results in impaired socialization and antisocial behaviour. Likewise, Patrick and colleagues (1993) argue that the decreased levels of startle potentiation in the presence of aversive stimuli seen in psychopaths maybe due to a disconnection between normal stimulus-response patterns occurring as a consequence of amygdala dysfunction.

The paralimbic dysfunction hypothesis (Kiehl, 2006) goes beyond the amygdala-dysfunction models, and implicates the entire limbic and paralimbic system in the development of psychopathy. This model draws upon data

indicating that dysfunction in numerous neural regions has been associated with psychopathy. This includes the amygdala (Gordon, Baird, & End, 2004; Kiehl, Smith et al., 2001; Müller et al., 2003), the cingulate cortex (Kiehl, Smith et al., 2001; Müller et al., 2003; Sterzer, Stadler, Krebs, Kleinschmidt, & Poustka, 2005), and several frontal and temporal regions (Intrator et al., 1997; Kiehl, Bates, Laurens, Hare, & Liddle, 2006; Kiehl et al., 2004). For example, Kiehl et al. (2001) examined limbic function in psychopaths using emotional and neutral words in a memory paradigm and found that emotional stimuli produced less activation in limbic and paralimbic regions, including the ACC, but elicited more activation in fronto-temporal regions outside the limbic system. Data from these studies have been taken to mean that psychopaths process emotional information in a fundamentally different way than do nonpsychopaths, i.e., they may rely on nonlimbic neural regions for the processing of affective information (see also, Gordon et al., 2004; Intrator et al., 1997).

One region which has been reported as dysfunctional in many of the studies supporting the paralimbic dysfunction hypothesis is the ACC. For example, a number of imaging studies have indicated that psychopaths show decreased activity in the ACC during the processing of emotionally-relevant stimuli (Birbaumer et al., 2005; Kiehl, Smith et al., 2001; Müller et al., 2003; Sterzer et al., 2005). The ACC has rich connections with both limbic and cortical regions (Devinsky, Morrell, & Vogt, 1995) and is involved in the integration of cognitive, affective, and visceral information (Allman, Hakeem, Erwin, Nimchinsky, & Hof, 2001; Thayer & Lane, 2000). The ACC has been

associated with the control and modulation of ongoing behavior and with assessing the motivational significance of external stimuli (Devinsky et al., 1995). Furthermore, the ACC has been modeled as the source generator of the N2 and ERN, two ERP components that have been associated with the on-line monitoring of one's own performance (Carter et al., 1998; Coles, Scheffers, & Holroyd, 2001).

The ACC has been differentiated into two functionally distinct and reciprocally inhibitory regions (Devinsky et al., 1995; Vogt, Finch, & Olson, 1992). The more dorsal portions of the ACC, which include portions of regions 24 and 32 as defined by Brodmann's cytoarchitechtonic maps of cortical regions (Brodmann, 1909), have reciprocal projections to cortical regions involved in the control of attention, whereas ventral regions including portions of Brodmann areas 24, 25, and 32 share projections with limbic, paralimbic, and brainstem regions, and are involved in the processing of affective information (Bush, Luu, & Posner, 2000; Devinsky et al., 1995). See Figure 1.1 for a depiction of the relevant neural structures. Functionally distinct neural systems in the ACC have also been observed during the resolution of response conflict. For example, Egner, Etkin, Gale, and Hirsch (2008) used fMRI to record brain activation of healthy individuals during a task that had emotional and nonemotional conditions. In the emotionally-neutral condition, participants had to identify the gender of a face, while ignoring simultaneously presented conflicting lexical stimuli (e.g., a fearful male face with the word "FEMALE" superimposed on it). In the emotional condition, participants were required to identify the facial

expression while ignoring the conflicting word (e.g., a fearful male face with the word "ANGRY' superimposed on it). They found that during the nonemotional task, activity in the lateral prefrontal cortex (LPFC) and dorsal regions of the ACC was associated with better task performance. LPFC activity was also associated with increased activity in the fusiform face area. Conversely, enhanced performance on the emotional task was associated with increased activity in rostral regions of the ACC, and decreases in amygdala activity. The authors interpret this as reflecting the presence of two dissociable and functionally independent neural systems.

To summarize, it is possible that neural dysfunction in one or more limbic regions underlies the major deficits associated with psychopathy. However, it is unclear whether the basis of this disorder lies primarily in the inability to process and respond appropriately solely to emotional information (e.g., Blair, 2003; Kiehl, Smith et al., 2001) or whether the disorder can also be characterized by broader neural abnormalities. These would include atypical aspects of attention and cognition and whether these are best characterized as a deficits or as an enhanced ability to (hyper) focus attention on personally relevant information remains a matter of debate (e.g., Hiatt et al., 2004; Kosson, 1996; Patterson & Newman, 1993; Raine & Venables, 1988).

Measuring Brain-Behavioural Relations

Although techniques such as functional magnetic resonance imaging (fMRI) allow for excellent spatial localization, they do not permit the on-line temporal analysis of attentional and cognitive function - processes that take just

hundredths of a second to unfold. On-line neural responses are better examined using event-related potentials (ERPs) because of their higher temporal resolution. Researchers have made attempts to examine attentional processes of psychopaths using ERPs but the results have been inconclusive. For example, Raine and Venables (1987; 1988) reported enhanced P3 amplitudes in this population. However, attenuated P3s have also been found. Kiehl and colleagues (2006; 1999) reported that psychopathic offenders generated P3s of significantly smaller amplitude than did nonpsychopathic offenders, showing marked deficits in the electrophysiological differentiation of nonemotional stimuli, and suggesting disrupted attentional allocation.

Another electrophysiological marker of cognition that has been used in the study of psychopathy is the error-related negativity (ERN e.g., Dehaene, Posner, & Tucker, 1994; Holroyd & Coles, 2002). It is a negative ERP component specific to errors (Coles et al., 2001; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1990) and is thought to originate in the ACC. To test this model Dikman and Allen (2000) examined ERPs relating to error monitoring in undergraduates who scored in the top and bottom ranges of a measure of socialization (a construct theoretically related to psychopathy), and reported that under some circumstances, the low-socialized group showed less electrocortical responsivity to the commission of errors than did the high-socialized group.

Santesso, Segalowitz, and Schmidt (2005) reported similar results from their examination of the ERN in a group of children. Poor socialization was associated with smaller amplitude ERNs. Similarly, Hall, Bernat, and Patrick

(2007) reported that higher scores on a measure of externalizing were associated with smaller amplitude ERNs. Many conditions associated with behavioral inhibition, such as conduct disorder, antisocial personality disorder, and alcoholism, are thought to reflect this externalizing factor (Krueger, 2002), although it is currently unclear how strongly this factor is associated with psychopathy

Current Investigation

Although much work has been done examining the cognitive and neuropsychological correlates of psychopathy, much of this work is inconsistent and difficult to interpret. In general, the electrophysiological, neuroimaging, and behavioural evidence suggests that psychopaths show marked deficits in processing emotional information (e.g., Blair et al., 2004; Kiehl, Smith et al., 2001), but is inconsistent in regards to the presence of general information-processing or response-inhibition deficits (e.g., Hiatt et al., 2004; Kiehl, Bates et al., 2006; Kiehl et al., 1999; Kosson, 1996). That said, several studies have reported attentional differences between psychopaths and nonpsychopaths (Dvorak-Bertsch, Sadeh, Glass, Thornton, & Newman, 2007; Hiatt et al., 2004; Smith, Arnett, & Newman, 1992).

Attempts to explain the basis of an attentional disorder in psychopathy include Newman's response monitoring hypothesis (Newman, 1998). This theory suggests that many of the behavioural traits seen in psychopathy (e.g.,relative insensitivity to punishment and response perseveration) may be the result of impairments in the ability to monitor and utilize peripheral or non-

motivational information during goal-directed behaviour. Raine and Venables (1988) postulated that the disorder is also associated with atypical, but not necessarily deficient, attention that may allow psychopaths to hyper-focus on information that seems relevant to them and to avoid processing peripheral or seemingly irrelevant detail. Alternatively, according to Kiehl's (2006) paralimbic dysfunction hypothesis, the disorder is primarily a result of deficits in the processing of affective information, specifically due to atypical function in limbic and paralimbic regions such as the orbitofrontal cortex, amygdala, and cingulate gyrus (including the ACC). These hypotheses also lead to dissociable predictions about expected electrophysiological performance in psychopaths, which will be addressed in the current work by using a range of tasks to evoke ERPs. These tasks were selected to allow for the separate examination of emotional and non-emotional information processing in individuals who vary with respect to psychopathic personality traits. Specific methods and hypotheses will be reviewed in the subsequent chapters.

The data presented in this thesis were collected from two samples. The first sample was from a population of incarcerated violent offenders with a range of scores on a clinical measure of psychopathy. The results from tasks conducted in this population are presented in chapters two and three. The second sample was a group of undergraduate males with a range of scores on a non-clinical measure of psychopathy. Results from this sample are presented in chapters four and five. The issues addressed involve first a follow up on the results of my previous work. In my MA work (some of which is reported in

chapter two), results indicated that whereas psychopaths generated normal ERNs to an error-monitoring task using non-emotional stimuli, they generated attenuated ERNs in response to an error-monitoring task with affective stimuli. Furthermore, the degree of attenuation of the ERN for emotional stimuli was correlated with the offenders' psychopathy scores such that higher scores were associated with diminished ERNs. When considering research implicating the ACC as the neural generator of the ERN, an important extension of this work would involve data exploration using source analysis techniques to identify the neural generator of the ERN in both the offender and control groups. These source analyses will be conducted in chapter two. Concretely, source analyses would allow for us to confirm that normally the ACC is the main generator of the ERN in non-psychopathic individuals, and that atypical ACC function during the processing of emotional information is specifically associated with psychopathy.

A second focus in this investigation was to examine response inhibition in incarcerated offenders. This is an issue of interest because psychopathy has been associated with problems of impulse control and response inhibition (Howland et al., 1993; LaPierre et al., 1995). This was done by examining electrocortical components, such as the N2 and inhibitory anterior P3, that have been associated specifically with inhibitory control during Go NoGo tasks (e.g. Bekker, Kenemans et al., 2005; Bokura et al., 2001; Bruin, Wijers, & van Staveren, 2001; Eimer, 1993; Falkenstein, Hoormann, & Hohnsbein, 1999; Fallgatter, Bartsch, & Herrmann, 2002; Fallgatter et al., 2004; Kok, 1986). The

N2 and anterior P3 components are reportedly generated in the ACC, so examining them offered another window on the function of this region as it related to psychopathy.

The third and fourth data sets provided an opportunity to examine attentional and affective performance in a subclinical population that varied with respect to psychopathic tendencies but who were all functioning well as undergraduate students. Besides replicating and extending the results of data sets one and two with incarcerated individuals, this work provided an opportunity to examine the "continuity hypothesis," i.e., that psychopathy is a normally-distributed personality trait rather than a separate taxonomic category (e.g. Edens et al., 2006). It is currently unclear whether those who express psychopathic personality traits in subclinical populations will perform similarly to clinically diagnosed psychopaths on psychophysiological measures. The last issue addressed involved the examination of general attention allocation processes, as reflected by the P3 component. Several models of psychopathy have predicted the presence of cognitive or attentional deficits, while others have predicted normal or even enhanced attentional processes associated with psychopathy. The P3 offers an index of selective attention, and provides a direct means of evaluating information-processing models of psychopathy within the normative ranges of psychopathic tendencies.

CHAPTER TWO. NEUROCOGNITIVE PROCESSES INVOLVED IN RESPONSE MONITORING

Introduction

The ACC is considered central to the integration of attentional, affective, and visceral information (Allman et al., 2001; Critchley, 2005; Thayer & Lane, 2000). It has been associated with the control and modulation of ongoing behavior and with assessing the motivational significance of external stimuli (Devinsky et al., 1995). The ACC has been differentiated into two functionally distinct regions (Vogt et al., 1992). The more dorsal and dorsal portions of the ACC have reciprocal projections with cortical regions involved in the control of attention, whereas ventral regions share projections with limbic, paralimbic, and brainstem regions, and are involved in the processing of affective information (Bush et al., 2000). The ACC provides entry for limbic influence on the voluntary motor system (Morecraft & Van Hoesen, 1998).

The ACC is specifically involved in the brain's error processing system (e.g. Miltner et al., 2003). The dorsal regions of the ACC have consistently been modeled as the generator site of the ERN (e.g. Dehaene et al., 1994; Holroyd & Coles, 2002; Mathewson, Dywan, & Segalowitz, 2005). The ERN is observed as a negative deflection in the response-locked event-related potential (ERP) that is maximal at frontocentral sites and specific to errors (Coles, Gehring, Gratton, & Donchin, 1991; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991). There is some debate as to the functional significance of the ERN, with researchers linking it alternately to response conflict (e.g. van Veen & Carter,

2002a) or reinforcement learning (Holroyd, Praamstra, Plat, & Coles, 2002). Others believe it to be an index of a generic response monitoring system, reflecting a process by which actual and desired outcomes are compared or evaluated and related to their consequences (see also Rushworth, Walton, Kennerley, & Bannerman, 2004; Scheffers & Coles, 2000).

A number of subjective motivational factors have also been linked to the ERN (Luu, Flaisch, & Tucker, 2000). Hajcak, Moser, Yeung, and Simons (2005) reported that participants generated larger-amplitude ERNs to errors committed on highly-rewarded versus minimally-rewarded trials. Similar findings have been reported under other reward conditions (e.g., Gehring, Goss, Coles, Meyer, & Donchin, 1993). The ERN also appears to be sensitive to personality factors associated with anxiety (e.g., Hajcak, McDonald, & Simons, 2003; Pailing & Segalowitz, 2004c). ERNs of increased amplitude have been reported in individuals diagnosed with obsessive compulsive disorder, a condition associated with over-activation of cingulate cortex and related brain regions (Gehring, Himle, & Nisenson, 2000; Hajcak & Simons, 2002; Santesso, Segalowitz, & Schmidt, 2006). Such data raise questions as to whether personality traits associated with under-activation of the ACC would be linked with impairments in response monitoring and a reduced ERN, reflecting that under-activation.

Neurocognitive correlates of psychopathy

As described in chapter one, psychopathy is a personality disorder accompanied by a spectrum of affective abnormalities such as lack of empathy,

callousness, and lack of remorse (Cleckley, 1941; Hare, 1991, 2003). It has also been associated with abnormal ACC function under some circumstances, specifically during the processing of affective information (Kiehl, Smith et al., 2001; Müller et al., 2003), including emotional facial expressions – especially fear (Blair et al., 2004; Montagne et al., 2005). There is also evidence that psychopaths may show impairments in other processes associated with the limbic system and ACC, including reduced neurophysiological responses to aversive conditioning (e.g. Birbaumer et al., 2005) and electrodermal hyporeactivity in anticipation of aversive stimuli (Fung et al., 2005; Gatzke-Kopp et al., 2002). These arousal-based deficits seem to occur in the presence of largely intact higher-order cognitive abilities (Hart, Forth, & Hare, 1990).

There is, however, some evidence that psychopaths may also demonstrate more general difficulties with response monitoring. They have been shown to differ from controls in passive avoidance learning (Newman & Schmitt, 1998) and in their failure to exhibit post-error slowing after negative feedback (Newman, 1987). Indeed, it has been suggested that psychopaths may display a failure to monitor, and hence learn from, the response-based feedback they encounter (a response monitoring deficit). However, an alternate explanation is that, in the presence of ACC or paralimbic dysfunction, their visceral response to negative feedback is simply not sufficient to engage their attention or to be experienced as sufficiently aversive to lead to behavioural change (an emotional reactivity deficit).

Dikman and Allen (2000) explored the relation between antisocial behaviour, ERNs and reward. They recorded ERNs associated with errors made on the Eriksen Flanker task (Eriksen & Eriksen, 1974) under avoidance-learning or reward conditions. They used a sample of undergraduates divided on the basis of whether they scored high or low on the socialization scale of the California Psychological Inventory (CPI Gough, 1994), an instrument designed to capture variance in socialization in healthy populations. These authors used socialization to create a subclinical analog sample of psychopathic and non-psychopathic individuals. They found that highly socialized participants produced consistently large ERNs, whereas low socialized individuals showed smaller amplitude ERNs under some conditions.

Using a standard flanker task, Santesso et al. (2005) recorded ERNs from 10 year old children who varied on socialization (within a normative range) as measured by the Junior Eysenck Personality Questionnaire-Revised (Corulla, 1990). The researchers did not introduce reward contingencies but nonetheless found that higher scores on the socialization questions of the psychoticism scale were associated with a reduction in the amplitude of the ERN. Although none of the Santesso participants would have been diagnosed as psychopaths, this variation in level of socialization could be seen as relevant for those studying psychopathy because, as Eysenck has shown, low socialization scores in normal young populations can be predictive of antisocial behaviors in adulthood (Eysenck, 1997). Nonetheless, no one had actually reported data relevant to those at the highest end of the range with respect to psychopathic

tendencies, i.e., those actually diagnosed as criminal psychopaths. To that end, we examined error monitoring in a group of incarcerated violent offenders who varied with respect to the degree of psychopathy shown on the PCL-R, i.e., the scale used in the diagnosis of forensic populations. This allowed us to examine the degree to which error monitoring would be predictive of psychopathy at the highest end of the range. The offenders performance was compared with that of a non-offender population of prison staff who shared the same environment to some degree but who had no known history of violent behaviour.

The other strategy used was to include an error monitoring task that was specifically designed to engage those regions of the brain associated with the processing of emotional information. For this, an error monitoring task that corresponded with the letter-flanker task but included an emotional component was designed. Thus, instead of having to make speeded decisions as to whether the centre letter in the display was an H or an S, participants had to decide whether the centre stimulus was a frightened or angry face. As in the letter flanker task, the centre stimulus was flanked by either congruent or incongruent items. Concretely, the a priori predictions for my MA thesis were that the degree of error monitoring deficits should vary with the degree of psychopathy within the offender group but that the deficit in error monitoring would be most pronounced when the task required the decoding of emotional facial expression in the face-flanker task. The data were generally supportive of these hypotheses, and are fully described in my MA thesis (Munro, 2004) and in a subsequent publication (Munro et al., 2007). Nonetheless, these procedures and data are

described in enough detail here to provide a context in which to present the dipole analyses and to describe issues associated with participant selection and the nature of the sample, which will be relevant here and set the stage for the following study of inhibitory control in the same population.

As indicated in the introduction (chapter one), the data presented here primarily involve the degree to which the error-related ERP components could be modeled using source analyses. Specifically, I sought to uncover the ERN generators when errors were committed in a task involving emotion processing (the emotional face flanker task), and in an emotionally neutral task (the standard letter flanker task). The specific goal was to determine whether the generators of the ERN in these tasks would differ depending on both task-type, offender status, and level of psychopathy within the offender group. Given basic theoretical (e.g. Kiehl, 2006) and experimental evidence (Bush et al., 2000; Kiehl, Smith et al., 2001; Sterzer et al., 2005) as to the neural regions that may be associated with psychopathic tendencies, it was expected that, in the control group, the ERN dipole associated with errors during the letter flanker task would be in the region of the dorsal ACC as has been reported now by numerous researchers (e.g. Miltner et al., 2003; van Veen & Carter, 2002a). It was also expected that, during the face flanker task, the ERN dipole would be located in more ventral regions as compared to the letter task. It was hypothesized, however, that this ability to shift the ERN dipole to the ventral region of the ACC would not occur to the same degree in the offender group, especially for those high in PCL-R ratings of psychopathy. Such results would

support the view that to the degree that ACC is involved in psychopathy (Kiehl, Laurens, Duty, Forster, & Liddle, 2001; Müller et al., 2003) it would be those regions of the ACC that link most closely with limbic structures such as the amygdala that are involved and that such deficits would primarily be seen when the task involves these regions.

Methods

Participants

Violent offenders (N = 15; all male) were inmates at a maximum security forensic hospital. They were incarcerated for a range of violent offenses, ranging from arson to homicide. Staff members recommended offenders who were considered suitable candidates for participation in this research. Control participants (N = 15; all male) were recruited from among staff members (largely registered nurses) of that facility. All participants were free from recent psychotic illness and history of severe head injury, although overall offenders had a higher degree of pathology than did controls. Age did not differ between offenders (M = 45.9, SE = 3.5 yrs) and controls (M = 46.6, SE = 1.78 yrs), t (27) = 0.16, p = .87, d = .06, showing a moderate effect size. Offenders had fewer years of education (M = 10.9, SE = .67 yrs) than controls (M = 14.8, SE = .42 yrs), t(28) = 4.89, p <.001, d = 1.9[1], but all participants scored within the average range on a standard index of general intelligence (Shipley Institute of Living Scale, Shipley, 1986), with groups not differing on

[1] Variance due to education was subsequently examined with respect to all effects of interest and did not change the outcome.

this measure ($M_{Controls}$ = 106.5, SE = 2.10; $M_{Offenders}$ = 102.4, SE = 2.13), t (25) = 1.35, p = .19, d = .54. Although a history of severe head injury served as an exclusion criterion for this study, mild to moderate head injuries were reported by individuals in both the control and offender groups. An index developed to rate the severity of head injury (0 = no history of head injury to 3 = history of one moderate head injury or multiple minor head injuries) did not differentiate the groups, ($M_{Controls}$ = .80, SE = .26; $M_{Offenders}$ = 1.3, SE = .30), t (28) = 1.27, p = .22, d = .48. Offenders were more likely than controls to be currently prescribed psychoactive medication (most commonly sleeping medication or tranquilizers) but use was not extensive and was not related to any effects of interest (all p's > .4). Participants were also tested on a range of psychometric measures of attention and face-processing ability, but there were no group differences on any psychometric measure, all p's > .1.

The PCL-R (Hare, 1991, 2003), a measure reported to be robust with respect to indices of reliability and validity (Hare et al., 1990), was used to assess psychopathy in the offender group. The scores were calculated on the basis of file review by a trained and experienced staff member. Scores ranged from five (very low) to 36 (very high); M = 24.4, SD = 10.61, thus allowing for a full range of scores on this measure with nine of the offenders surpassing the customary cut-off of 25 for psychopathy[2]. Besides being able to examine behavioural and electrophysiological differences between the offender and non-

[2] The PCL-R was based on file information alone, excluding interview data. Although reliable (Harris et al., 2003), such ratings tend to be more conservative (Wong, 1988), which justified the use of a PCL-R score of 25 or above as the customary criterion for psychopathy rather than the usual cut-off of 30.

offender groups, we were able to use correlational methods within the offender group to examine individual differences with respect to degree of psychopathy. As well, for dipole analyses, it was possible to isolate only those offenders who would be clearly designated as meeting the criteria for psychopathy. Due to practical constraints at the facility, control participants were not assessed for psychopathy.

Offenders who participated in this study received a stipend of CAN$40. Participating staff (the non-offender control group) were paid CAN$15 when tested during their regular work hours and CAN$40 when they came in on their own time.

Procedures

Electrophysiological and psychometric testing took place in a quiet room in the research wing of the institution. There were two separate testing sessions. Electrophysiological testing was usually done first, with psychometric testing done the following day. This sequence was reversed on occasion due to scheduling difficulties, and a number of control participants completed all tasks in a single session due to constraints on their time.

Letter Flanker Task. Electroencephalography (EEG) data was collected while participants were engaged in two flanker tasks. The letter flanker task (Eriksen & Eriksen, 1974) involved the presentation of a series of five-letter strings made up of the letter H and/or the letter S presented on a computer monitor as white letters against a black background. Two letters on each end of the string were either congruent (SSSSS, HHHHH) or incongruent (SSHSS,

HHSHH) with the center letter. Instructions appeared on the screen and were read aloud. Participants were required to respond by key press with one hand if the central letter was an "H" and with the other hand if it were an "S." Test stimuli appeared on the screen for 190 ms, with an inter-trial interval of 1100 ms. A total of 480 trials were presented as 4 blocks of 120 trials. One-third of all trials were congruent. The session began with four practice trials and brief breaks were taken between blocks.

Face Flanker Task. The face flanker task (Figure 2.1) was designed to be as similar as possible to the letter flanker task while introducing an affective component. Stimuli were presented on a computer monitor and consisted of a series of black-and-white photographs of faces that had been modified (Santamaria, 2003) from an initial set of emotional faces developed by Gur and colleagues (e.g. Gur et al., 2002). The images selected for this task were of five individuals, each presenting an angry and a fearful expression. As there is no evidence that gender differentially effects facial expression recognition in offender populations, both male and female faces were incorporated in this task. On a given trial an emotional face was presented in the centre flanked on each side by faces with either congruent or incongruent expressions. Participants were asked to indicate by key press whether the expression on the centre face was angry or fearful. Images of multiple individuals portraying one of two emotions were used to encourage participants to process the emotional expression when making a judgement rather than allowing them to rely on an easily learned alteration in the features of one individual to distinguish between

emotions. In addition, all the faces with their different expressions were presented to participants for identification prior to their commencing the task followed by four self-paced practice trials. No errors were made in the identification of either fearful or angry faces under these conditions by participants in either the offender or the non-offender control group.

At test, stimuli appeared on the screen for 390 ms, with an inter-trial interval of 1100 ms. Stimulus duration was slower for the face flanker than letter flanker task to compensate for the increased visual complexity of the face stimuli. There were 480 trials in total, presented as four blocks of 120 trials with one-third being congruent. Brief breaks (one to two minutes) were given between blocks of trials. During both tasks, the importance of both speed and accuracy were emphasized. Due to discrepancies in task difficulty, all participants were given the letter flanker task first to allow them to familiarize themselves with the general flanker paradigm prior to having to deal with the more complex face flanker stimuli.

Electrophysiological Recording and Processing. EEG was collected using a 128-channel Active Two Biosemi system (BioSemi, Amsterdam). Eye-movements were recorded with three electrodes placed on the outer canthus, supra-orbital ridge, and cheekbone of the right eye. Signals were sampled at the rate of 512 points per second and digitized with a 24 bit ADC. Because the BioSemi system does A-D conversion at the electrode site, the amplifier gain was one. A bandpass filter from one Hz (time constant 0.1592s) to 30 Hz was used. The roll-off had a slope of 12 dB/oct. All electrodes were re-referenced

offline to averaged mastoids. The stimulus presentation and data acquisition program was E-Prime (Psychological Software Tools, Inc., 2004).

Vision Analyzer (Brain Products GmbH, München) was used to correct for both vertical and horizontal ocular artefacts. This automated eye-movement correction was based on a method developed by Gratton, Coles, and Donchin (1983). For both flanker tasks, response-locked, artefact-free EEG recordings were averaged relative to a -600 ms to -400 ms pre-response baseline[3]. The ERN amplitude was defined as the most negative value at fronto-central sites in the 150 ms following the response. The error positivity (Pe) was defined as the most positive value at fronto-central sites in the 150 – 350 ms post-response period. Peak values of these error-related ERP components were analyzed across tasks using repeated measure ANOVAs, with group (offenders vs. controls) as the between-group factor. Within-group factors consisted of task (letter-flanker vs. face-flanker) and midline sites equivalent to Fz, FCz, Cz, and Pz of the standard extended 10/20 system (Jasper, 1958). Correlations and forced-entry regression analyses were carried out to determine the relations between the amplitude of the ERN and Pe, error rate, and degree of psychopathy among the violent offenders. When necessary, all statistical analyses were corrected for violations of Mauchley's Test of Sphericity, utilizing the Huynh-

[3] This early baseline was selected in order to quantify the ERN because the period immediately preceding the response includes the P3 component, which could reflect variance due to stimulus evaluation that would be independent of error-related response. However, this is controversial, so all analyses were also done using the P3 as a covariate, which is equivalent to using the immediate pre-stimulus baseline, and the effects of interest were not affected.

Feldt correction for estimating the F-Statistics with original degrees of freedom presented in the text. The Bonferroni correction for multiple comparisons was used where appropriate.

The locations of the major generators of the ERN to letter and face flanker tasks were modeled on grand average error waveforms for each group using Brain Electrical Source Analysis software (BESA 5.0 Scherg, 2003), a software program that estimates sources of electrical activity in a four-shell, spherical model of the head with ellipsoidal correction. Site co-ordinates were digitized using a Polhemus FASTRAK digital tracker (www.polhemus.com).

Statistical Analyses. Of the 30 participants, data from four offenders and three controls were dropped from the analyses. One control participant and one offender were excluded because of outlying error rates on the face flanker task. Three offenders (two psychopaths and one nonpsychopath) and two control participants were excluded from electrophysiological analyses because they had made too few errors to allow for reliable ERN averages on one or both tasks. Thus, there was a full data set from 12 controls and 11 offenders, seven of whom met the PCL-R cut-off for psychopathy. The average number of trials per ERP average was 46.87 (± 26.2) with no average based on fewer than 12 trials.

Results

Behavioural Responses

Error and response time (RT) data are presented in Table 2.1. Initial analyses of error responses included the congruency factor. There was, as would be expected, a main effect of congruency, F (1,26) = 106.9, $p < .001$, $\eta^2 = .80$,

such that there were more errors on incongruent than congruent trials. However, this occurred for both tasks and did not interact with group, so data are presented collapsed across congruent and incongruent flanker conditions for all analyses. These collapsed error and response time (RT) data are presented in Table 2.1. Overall error rate differences between groups were not significant (p = .18). The proportion of errors made during the face flanker task (M = .21 ± .02) indicated that it was more difficult than the letter flanker task (M = .10 ± .01), $F(1, 21) = 52.1$, $p < .001$, $\eta^2 = .71$. However, there was a group by task interaction $F(1, 21) = 8.67$, $p = .008$, $\eta^2 = .29$, caused by offenders committing more errors than controls on the face flanker but not the letter flanker task. Within the offender group PCL-R scores correlated with fear expression errors, $r = .73$, $p = .01$, but not with angry expression errors, $r = .35$, $p > .1$. Thus, the likelihood of making a face flanker error when confronted with a fearful target face was directly associated with the degree of psychopathic symptomatology within the offender group.

Response times were analysed in a similar fashion[4]. There was a main effect of task such that participants generally responded faster during the letter flanker task (M = 443 ± 11 ms) than the face flanker task (M = 662 ± 29 ms), $F(1, 21) = 80.1$, $p = < .001$, $\eta^2 = .79$. There was also a main effect of group, such that controls were generally faster than offenders, $F(1, 21) = 4.89$, p =

[4] Although RTs to error trials are typically not examined in standard cognitive paradigms, they are typically reported in error monitoring studies as this they do convey a great deal of information regarding the nature of the error. For example, faster RTs on error trials are commonly seen and interpreted as indicating impulsive responses or behavioural slips. Flanker tasks are designed to elicit such responding, and hence the examination of RTs provides an index of the success of the task manipulation. It also allows for the documentation of whether one group is responding in a more impulsive way than the other.

.038, η^2 = .19, but there was no interaction between group and task nor between group and accuracy with respect to RT.

Electrophysiological Response

Grand-mean response-locked ERP waveforms at midline sites for the offender and control groups for each task are presented in Figures 2.2 and 2.3. Analyses revealed that there were no consistent group or task differences in regards to the P3 component. As expected, the ERN was larger on error than correct trials, $F(1, 21) = 88.51$, $p < .001$, η^2 =.81, and there was a task by group interaction, $F(1, 21) = 11.34$, $p = .003$, η^2 =.35. While the groups generated ERNs of comparable size to the letter task, offenders generated a markedly reduced ERN to the face flanker task. The amplitude of the ERN in the emotional condition was significantly correlated with offenders' scores on the PCL-R, $r = .79$, $p = .004$, indicating that the attenuated ERN seen in the offender group was closely linked to psychopathy. There were no differences in the amplitude of this component on correct trials.

A possible confound in these data involves the fact that offenders had a higher error rate in the face flanker task than controls so that their reduced face-flanker ERN could be a consequence of that task being more difficult for them. To explore this possibility, 4 controls who had the lowest error rates on the face flanker task were dropped from the analysis, as were 4 offenders who had the highest error rates on this task. As a result, the error rate no longer differed between groups, $F(1,14) = .41$, $p = .54$, and there was no group by task interaction on errors, $F(1, 14) = .62$, $p = .44$, or with respect to behavioural RTs,

$F(1, 14) = .80$, $p = .39$, $\eta^2 = .05$. However, despite equalizing error rate between the two groups, an analysis of ERN amplitudes still revealed a significant interaction between task and group, $F(1,14) = 10.56$, $p = .006$, $\eta^2 = .43$, which was consistent with that obtained for the whole group, $F(1, 21) = 11.34$, $p = .003$, $\eta^2 = .35$. Taking another approach, ERN amplitudes were again subjected to an ANOVA with face-flanker error rate used as a covariate. Even after adjusting for error rate in this way, there was a significant interaction between task and group, $F(1, 21) = 9.31$, $p = .006$, $\eta^2 = .31$, confirming the specific reduction in the amplitude of the ERN for offenders in the face flanker task. Thus, it is unlikely that the ERN amplitude effects can be explained solely on the basis of group differences in response to task difficulty levels.

The ERN is followed by the error positivity (Pe), peaking approximately 150-350 ms after the erroneous response. There was also a marginal trend, $F(1, 21) = 3.36$, $p = .08$, $\eta^2 = .14$, for those in the offender group to produce a Pe of reduced amplitude ($M = 4.86 \pm 1.06$ μV) relative to controls ($M = 7.55 \pm 1.02$ μV), although this did not correlate with ERN amplitude.

Source Modelling of the ERN

A major issue in this work was to determine the best dipole solutions for the ERP components associated with errors during both tasks and to determine whether those high in psychopathy responded differently at the electrocortical level to task conditions. To best model the dipoles associated with the effects of interest, source analyses were conducted only for those offenders who met the PCL-R cut-off for psychopathy (PCL-R $\geq$ 25, n = 7) and controls (n = 12). The topographical maps of the ERN response of the controls and the selected offenders who met the criteria for psychopathy can be seen in Figure 2.4. The results of the source localization can be found in Figure 2.5. Modelling the responses for these groups to each task generally resulted in satisfactory fits with less than 10% of the variance left unaccounted for. The only exception was in the case of the psychopath group's responses to the face flanker task.

Modelling the ERN during the letter flanker task in the control group resulted in a three-dipole solution. A single dipole located in the area of the dorsal ACC was recorded between 53 – 109 ms post response (x = 4, y = 0.1, z = 44.7, Talairach), which accounted for a large proportion of the variance (60 percent). Two symmetrical dipoles located in the posterior cingulate gyrus were also identified (x = 5.5, y = -49.2, z = 27.2; x = -5.5, y = -49.2, z = 27.2), which accounted for a further 20 percent of the variance. Together these three dipoles accounted for 95 percent of the variance associated with errors in the letter flanker task.

Modelling the error response during the letter flanker task in the psychopath group, based on a 46 – 89 ms window, also resulted in a three-dipole solution. A single dipole was localized approximately within the dorsal ACC (x = 9.8, y = 1.24, y = 48) and accounted for 81% of the variance. The addition of two symmetrical dipoles located in the posterior cingulate gyrus (x = 7.4, y = -47, z = 8.2; x = -7.4, y = -47, z = 8.2) increased the variance accounted for to 93.6 percent.

When the ERN response to face flanker task errors was modeled for the control group (25 – 85 ms), it resulted in a solution comprised of a pair of symmetrical dipoles with a more ventral generator localized in the ACC (x = 7.6, y = -17, z = 40; x = -7.6, y = -17, z = 40), accounting for 94.5 percent of the variance. The solution for the psychopath group was considerably different from the solution for the control participants. Two pairs of symmetrical dipoles were localized in a window of 68 – 97 ms post response. One symmetrical pair was localized in the region of the insula (x = 34, y = -0.1, z = 17.3; x = -34, y = -0.1, z = 17.3) and accounted for the majority of the variance (60 percent), while two minor, symmetrical dipoles were localized in the region of the parahippocampal gyrus (x = 16, y = -40.0, z = -3.4; x = -16, y = -40, z = -3.4), which raised the variance accounted for to 82 percent. Thus, it would appear that although psychopaths were able to produce ERNs of similar scalp topography and similar dipoles when engaging in the letter flanker task, their error monitoring response was markedly different during the rapid discrimination of facial expressions.

Discussion

This study was designed to examine the degree to which error monitoring, and by extension, ACC function, are altered in psychopathic violent offenders. Results indicated that offenders and controls did equally well on the standard letter flanker task, suggesting that violent offenders, even those with high levels of psychopathy, may not necessarily be deficient in their error monitoring performance per se. These results are consistent with other data suggesting that psychopathy is not necessarily related to general deficits in higher order cognitive functions (e.g. Hart et al., 1990), a position bolstered by the equivalent performance between the offender and control groups on the various psychometric measures of attention and memory used here. However, there were marked differences between the two groups' electrophysiological and behavioural performance on the emotional face flanker task.

Response Monitoring Capability and ACC Function

Error monitoring has been linked to differential amplitude of the ERN (e.g. Falkenstein et al., 1991) and considerable evidence has now accrued linking the generator site of the ERN to the ACC (Dehaene et al., 1994). The ACC, in addition to being involved in the brain's error-processing system (Miltner, Braun, & Coles, 1997), is thought to provide a link between limbic function and voluntary activity (Morecraft & Van Hoesen, 1998) and to be involved in the processing of emotional facial expressions, particularly fear (George et al., 1993). There is growing evidence that psychopathy may be specifically associated with a deficit in the ability to recognize this emotion

(Marsh & Blair, 2008). Thus, an alteration of the ERN in the offender population, especially if linked specifically with emotional stimuli, would suggest that ACC activity is selectively influenced in psychopathy in the context of affective tasks.

Psychopathy and Cingulate Function

The link between psychopathy and the ACC was more specifically examined through source modelling of the ERN. For controls, ERNs elicited by errors during both the letter and face tasks appeared to be generated primarily in the ACC. However, those offenders designated as psychopaths on the PCL-R produced ERNs with ACC-based dipoles in response to letter-flanker errors, but not in response to errors on the face-flanker task. These dipole solutions are generally consistent with the results of imaging studies that have shown a differential response in psychopaths relative to controls when faced with emotional, but not neutral, stimuli. For example, Intrator et al. (1997), using a lexical decision task for emotional and neutral words, found that for the emotional words psychopaths showed an increase in regional cerebral blood flow not in limbic and paralimbic areas as seen in controls, but rather in fronto-temporal and medial frontal regions. Kiehl et al. (2001) replicated these effects using emotional and neutral words in a memory paradigm and found that emotional stimuli produced less activation in limbic and paralimbic regions, including the ACC, but elicited more activation in fronto-temporal regions outside the limbic system.

One could argue that the ERN was smaller for psychopaths when making face-flanker errors because they were less aware of their errors. Although we do not have specific indices of awareness, it has been shown that uncertainty, while decreasing the ERN, also increases negativity in the ERP waveform associated with correct trials, the CRN (Pailing & Segalowitz, 2004a; Scheffers & Coles, 2000). Supplementary post-hoc analyses revealed that this did not occur for psychopaths in the present study. There were no differences in amplitude of CRNs between offenders and controls during the face flanker task and there was no relation between offenders' scores on the PCL-R and CRN amplitude.

Both controls and offenders were able to make the appropriate discrimination between angry and fearful faces during the self-paced practice trials that preceded the actual testing situation, which is consistent with data reported by other authors (Book, Quinsey, & Langford, 2007). Thus, the offenders were able to identify fearful and angry faces during the familiarization phase of the experiment. The deficits in emotion processing observed in the offender group emerged only when speeded decisions were required. Thus, it may be that psychopaths process emotional expressions by depending on slower, top-down strategies rather than on emotion-based visceral responses, especially in the case of fearful facial expressions. The failure to process fearful faces using the more automatic and viscerally linked region of the ACC could prevent them from developing patterns of social behavior more typically associated with the expression of fear in the face of another person.

Thus, it is possible that when task goals require the recruitment of ventral ACC, psychopaths are less able to engage this region resulting in both reduced accuracy, diminished amplitude of the ERN, and the recruitment of other brain regions (Intrator et al., 1997; Kiehl, Smith et al., 2001). There is precedence for such effects. The examination of flanker task performance by patients with ACC lesions has shown that such individuals are capable of completing this task, and while they seemed to know that they were making errors their errors simply did not elicit the expected ERN (Stemmer, Segalowitz, Witzke, & Schoenle, 2003). Similarly, in the present study, offenders responded behaviorally to having made an error during the face-flanker task in the same way as controls, i.e., they often showed obvious signs of frustration when they hit the wrong key, but this acknowledgement of error did not produce a substantive ERN.

Study Limitations

A limitation of this study is the relatively small sample size (12 controls and 11 offenders, only seven of whom met the customary cut-off for psychopathy). Nonetheless, the sample size was sufficient to demonstrate the interaction between group and task on the face-flanker task. It may be that, in a larger sample, an association between psychopathy and error monitoring on the letter-flanker task would be evident as well, although relevant effect sizes in this sample suggest such an outcome would be unlikely. Another avenue for future work is to utilize a more complex non-emotional task to examine group difference in general error monitoring capacity to follow up on the current work,

to ensure that the observed results were not simply a bi-product of differences in task difficulty between the face flanker and letter flanker task as opposed to differences attributable to the face-flanker's task requiring the processing of emotional stimuli. Another concern is that the shallowness of the face-flanker ERN in the psychopath group resulted in relatively less stable dipole solutions than those obtained for the offenders in the face-flanker conditions and for the control group in both conditions. Thus, replication would be required before one can confirm that psychopaths are more dependent on the insula and parahippocampal gyrus when performance monitoring involves emotional information. Nonetheless, it does seem fairly safe to conclude that they do process emotional information differently than controls when involved in the same performance monitoring condition. Furthermore, despite the relatively small sample size and other limitations, the results are consistent with current theory about the neural basis of psychopathy and underline the importance of adjusting standard error monitoring paradigms to more specifically test the questions of interest, especially for special populations.

Summary

Despite some limitations, these data suggest that while violent offenders generally performed in a similar fashion to controls under standard error monitoring conditions, they made significantly more errors when faced with emotional stimuli. The ERN was similar across groups for letter-flanker errors but was markedly reduced in the offenders for face-flanker errors, an effect related to the degree of psychopathy within the offender group. Source

modelling placed the primary dipoles associated with the ERN in the region of the anterior cingulate for both groups when making errors concerning non-emotional stimuli. For offenders who met the criteria for psychopathy, the attenuated ERN elicited by the emotional face-flanker errors showed little evidence of ACC involvement. One interpretation of these results is that psychopathy is associated with an atypical response only when error monitoring requires the discrimination of affectively-based information.

CHAPTER THREE. RESPONSE INHIBITION IN PSYCHOPATHY: THE NOGO N2 AND P3

Introduction

Abnormal anterior cingulate cortex (ACC) function has been associated with a number of conditions for which issues of strategic control are of concern. Psychopathy is such a condition. Psychopathy is associated with a range of performance-monitoring deficits that may be consistent with abnormal ACC function. Moreover, recent data from imaging studies (Kiehl, Smith et al., 2001; Müller et al., 2003; Sterzer et al., 2005) suggest some degree of atypical function in the ACC and adjacent brain regions in this population.

The ACC is implicated in response-withholding paradigms. The stimuli in Go/NoGo tasks elicit two characteristic components of the event-related potential (ERP), the N2 and P3. These components are typically larger on NoGo compared to Go trials and are thought to reflect response inhibition (Bokura et al., 2001; Bruin et al., 2001; Eimer, 1993; Falkenstein et al., 1999; Kok, 1986). Both the NoGo N2 (Bekker, Kenemans et al., 2005; Bokura et al., 2001; van Veen & Carter, 2002b) and the NoGo P3 (Fallgatter et al., 2002; Fallgatter et al., 2005; Fallgatter et al., 2004) have been associated with activity in the ACC and associated regions.

In addition to the reports of reduced inhibitory control among psychopaths (Howland et al., 1993; LaPierre et al., 1995), Kiehl and colleagues have reported that psychopaths failed to demonstrate a NoGo N2 effect during a visual Go/NoGo task (Kiehl, Smith et al., 2000). It is the goal of the current

study to replicate these results in a group of violent offenders who represented a range on the PCL-R ratings of psychopathy so as to take advantage of the power of correlational techniques to uncover the relations between psychopathy and ERP components elicited during tasks requiring response inhibition. Finding a diminished NoGo N2 effect and a reduced or less anterior inhibitory P3 in the offender group that also correlated with degree of psychopathy within the group would be consistent with the hypothesized ACC involvement in psychopathy and would confirm the sensitivity of the inhibitory N2 and P3 as indices of ACC function.

Methods

Procedures

Participants and general procedure in this study were the same as reported in chapter two. In the current study, EEG was collected while participants responded to a Go/NoGo task (Garavan, Ross, Murphy, Roche, & Stein, 2002; Lewis, Lamm, Segalowitz, Stieben, & Zelazo, 2006). This task involved the serial presentation of a series of letters in alternating fashion (x-y-x-y-x-x-y). Participants were required to press a key when the stimulus letter was different from the preceding one (Go condition), and to withhold a response to lures (e.g., the second of the consecutive x's in the example above), which occurred when the stimulus letter was the same as on the preceding trial (NoGo condition). A total of 550 stimuli were presented in two blocks of 200 trials and one block of 150 trials. No Go trials occurred only on 1/3 of the trials, so that in each block the large majority of trials involved key-press responses. Using this

proportion of go to no-go trials ensured that the withholding of a response involved overcoming an established response tendency. The stimuli differed across blocks of trials such that "x" and "y" were used in the first block, "o" and "p" in the second, and "d" and "u" in the third. The inter-stimulus interval (ISI) increased following errors and decreased following correct responses, thus maintaining a moderate overall error rate and avoiding the potential of random responding for those unable to keep pace with a standard stimulus presentation speed. There were 10 practice trials at the beginning of each stimulus set that were not used for averaging.

Stimulus-locked, artifact-free EEG recordings associated with successful Go and NoGo trials were averaged relative to a 200 ms pre-stimulus baseline. N2 amplitude was defined as the most negative peak at frontocentral midline sites in the 150 - 350 ms period following stimulus onset on correctly inhibited no-go trials; the P3 was defined as the most positive value in the 300 - 600 ms window on correct no-go trials. Peak values were analyzed for each component using separate repeated measure ANOVAs with trial type and site (Fz, FCz, and two Cz sites, Cz_1 and Cz_2, slightly anterior and posterior to the standard Cz, respectively) as within-group factors and group (offenders and controls) as the between-group factor. Subsequent correlational analyses were carried out to determine specific associations between the behavioral and ERP measures and psychopathy within the offender group. All statistical analyses were corrected for violations of Mauchley's Test of Sphericity, where necessary, utilizing the Huynh-Feldt correction for estimating the F-Statistics; however, the original

degrees of freedom (not the corrected values) are reported below. The Bonferroni correction for multiple comparisons was used where appropriate. Scores from two control participants were dropped from the behavioral analyses due to missing data, and from two offenders due to response times or error rates that were more than two standard deviations from their group average.

Results

Behavioural Responses

Proportion of error responses were entered into a 2 (Group) x 2 (Trial-Type) repeated measures ANOVA. There was a main effect of Trial Type $F(1, 22) = 83.62$, $p < .001$, $\eta^2 = .79$, such that participants made more errors of commission on NoGo trials ($M = .37 \pm .03$) than errors of omission on Go trials ($M = .10 \pm .01$). There was also a significant effect of Group, $F(1, 22) = 6.45$, $p = .019$, $\eta^2 = .23$, such that offenders ($M = .27 \pm .02$) made more errors across both trial types than controls ($M = .21 \pm .02$). However, these effects were superseded by a significant Trial-Type by Group interaction, $F(1, 22) = 4.63$, $p = .04$, $\eta^2 = .17$, which indicated that offenders ($M = .43 \pm .04$) made more errors of commission on NoGo trials than Controls ($M = .31 \pm .04$), but errors of omission on Go trials were the same ($M = .10 \pm .01$) for both groups (see, Table 3.1). The higher level of commission errors did not relate to PCL-R scores within the offender group, $r = -.46$, $p = .13$, suggesting that this effect was not specific to psychopathy[5].

[5] Analyses done on the average amplitude across the latency window associated with the entire P3 (300 – 600 ms) or with the early P3 (300 – 400 ms) yielded similar results.

With respect to response times (RTs), there was a main effect for group, $F(1, 22) = 6.23$, $p = .02$, $\eta^2 = .22$, such that, in general, offenders ($M = 410 \pm 14.1$ ms) responded more slowly than controls ($M = 360 \pm 14.1$ ms). There was no effect of trial-type (Go versus NoGo) nor was there an interaction between trial-type and group, suggesting no obvious difference in impulsive responding on NoGo trials. As well, RTs were unrelated to psychopathy within the offender group, all *p's* > .80. Because ISI varied with task performance, the mean ISI of the two groups were entered into a one-way ANOVA revealing that offenders ($M = 1086$ ms ± .58ms) had longer ISIs on average than did controls ($M = 846$ ms ± 52 ms), $F(1, 24) = 9.52$, $p = .005$. This is consistent with the higher rate of errors of commission on NoGo trials seen in the offender group but was also uncorrelated with their scores on the PCL-R, $r = -.19$, $p = .52$.

Electrophysiological Responses

N2 amplitudes for correct trials were entered into a 2 (Group) x 2 (Trial-Type) x 4 (Site) repeated measures ANOVA. As is evident in Figure 3.1, the N2 was larger when generated on NoGo ($M = -3.04 \pm .54$ μV) relative to Go trials ($M = -2.27 \pm .43$μV), $F(1, 26) = 7.96$, $p = .009$, $\eta^2 = .23$. There was also a quadratic effect of site, $F(1, 26) = 10.88$, $p = .003$, $\eta^2 = .30$, such that the N2 was larger at frontocentral sites (FCz and Cz_1) than at the more frontal or posterior sites. The latency of the N2 did not differ across groups, but latencies were slightly longer at central relative to posterior and anterior sites, $F(1, 26) = 5.24$, $p = .03$, $\eta^2 = .17$.

P3s were analyzed in a similar fashion. They were larger on NoGo (M = 5.20 ± .45 μV*)* relative to Go trials (M = 4.19 ± .39 μV) and were larger at FCz than other sites, $F(1,26) = 44.07$, $p < .001$, $\eta^2 = .63$. There was no main effect of group, but an interaction between group and site, $F(1, 26) = 4.73$, $p = .023$, $\eta^2 = .15$, was based on offenders generating P3s of smaller amplitude at the most frontal site (Fz) compared with controls, an effect that was unrelated to trial-type. P3 latencies were shorter for Go (M = 416 ± 14.5 ms*)* than NoGo trials (M = 471 ± 16.3 ms), $F(1, 26) = 9.10$, $p = .006$, $\eta^2 = 26$, but were slightly longer at the two most frontal sites for the offender group, $F(3, 78) = 3.36$, $p = .035$, $\eta^2 = .11$. There was no relation between PCL-R scores in the offender group and any of the electrophysiological measures, all p's > .65. For illustrative purposes (Figure 3.2) we divided the offender group into those who met the criterion for psychopathy ($M_{\text{PCL-R}} = 31.8 \pm 4.3$) and those who did not ($M_{\text{PCL-R}} = 15.0 \pm 5.5$). As is evident, there is absolutely no suggestion that high levels of psychopathy are associated with a diminished NoGo N2 or inhibitory P3. The topography based on difference waves associated with NoGo relative to Go responses (Figure 3.3) indicates that the N2 and P3 effects were quite similar across groups.

Discussion

Individuals in this sample who exceeded the cut-off for psychopathy on the PCL-R produced robust NoGo N2s and inhibitory P3s. Although offenders made more errors of commission on NoGo trials, these did not correlate with level of psychopathy within the offender group suggesting that such errors were

not specific to psychopathy. Response times were longer for offenders than controls, but again, RTs did not relate to psychopathy. Although the offender group produced smaller inhibitory P3 amplitudes at frontal sites, this effect was not associated with condition and did not correlate with psychopathy within the group.

A larger sample size could increase power to find differences between the offenders and controls. However, there was no sign of a group difference between controls and offenders with respect to the inhibitory N2 and P3 and no sign that higher levels of psychopathy within the group diminished these effects at all. The waveforms of those high on psychopathy (Figure 3.2) show what appeared to be an even more robust neural response on NoGo trials than was apparent in the control group (Figure 3.1). In fact, if there were any sign of a deviation from a normal ERP response, it would be in offenders low in psychopathy (Figure 3.2). While not significant in this small sample, the finding that offenders low in psychopathy have more difficulty with response control would be interesting to follow up using a larger sample of impulsive violent offenders who do not meet the criterion for psychopathy. Indeed, there is some evidence that such a group would likely produce diminished NoGo N2 effects (Chen, Tien, Juan, Tzeng, & Hung, 2005). We are also aware that any comparison between Go and NoGo trials are inherently confounded by such things as motor preparation (Bekker, Kenemans, & Verbaten, 2004; Bekker, Kenemans et al., 2005). However, in the present study, the effect of interest was not whether the absolute amplitude of either the N2 or P3 components in the

NoGo condition differed across groups. What was of interest was whether the inhibitory N2/P3 effect. i.e., the difference in response between Go and NoGo trials, was larger in one group relative to the other. Since the potential confounds of concern would be operative in both groups, they should not diminish the validity of group comparisons regarding the condition effects.

These results differ from those of Kiehl et al. (2000), who report that the N2 effect is absent in their psychopath group relative to two other incarcerated groups, one made up of individuals with schizophrenia and another of nonpsychopathic offenders. It should be noted, however, that even for the nonpsychopathic offenders the N2 effect was small, possibly due to having used data from a 50% Go/NoGo ratio paradigm, which has been shown to diminish the N2 effect (Bekker et al., 2004). Since Kiehl et al. did not include a non-offender control group, these effects are hard to interpret.

Finally, these data raise questions about how we conceptualize impulsivity in the context of psychopathy. The impulsivity item on the PCL-R refers to unpremeditated conduct lacking in forethought or reflection and not to frankly disinhibited behavior. Even though psychopaths may fail to resist impulses (e.g., for material gain or sexual gratification), they typically do so in a planful, or even predatory, manner (Hare, 1999; Harris & Rice, 1997). The present results suggest that the impulsivity associated with an exploitative and predatory lifestyle may not necessarily be reflected in aberrant inhibitory control at the level of motor response tendencies.

CHAPTER FOUR: ERROR MONITORING AND EMOTION IN SUBCLINICAL PSYCHOPATHY.

Introduction

As described before, psychopathy is a personality disorder associated with affective, interpersonal and behavioral anomalies (Hare, 1991, 2003). There is evidence of atypical function of limbic and paralimbic brain regions such as the ACC (Kiehl, Smith et al., 2001; Müller et al., 2003). Although psychopathy has been related to deficits in both emotional (Christianson et al., 1996; Habel et al., 2002; Kosson, 1996; Marsh & Blair, 2008; Montagne et al., 2005), and cognitive functions (Bernstein et al., 2000; LaPierre et al., 1995; Newman, 1998), the data reported in this thesis so far suggests the potential for a more focused deficit in ACC function, i.e., one that primarily involves those aspects of error-monitoring that have to do with affectively-relevant stimuli. To explore this issue further, the purpose of this study was to replicate the specificity of this affectively based error-monitoring deficit but within a potentially more normative range of psychopathic traits as would be available within a healthy undergraduate population.

Affective Processing in Psychopathy

As described in previous chapters, affective and interpersonal deficiencies have long represented a major characteristic of psychopathic behaviour (Cleckley, 1941; Habel et al., 2002; Hare, 1991, 2003), and have spawned a majority of the research in this field. There have been reports that psychopaths show deficits in the identification of some emotional facial

expressions and the identification of vocal affect (Blair, Budhani, Colledge, & Scott, 2005; Blair, Mitchell, Richell, Kelly, & Leonard, 2002; Hiatt, Lorenz, & Newman, 2002). For example, Blair et al (2004) displayed slides of faces. Each consecutive face showed a greater amount of affect than its predecessor. They found that when fearful facial expressions were shown in these sequences psychopaths responded more slowly than did nonpsychopaths, requiring on average a greater number of slides (and hence more intense affect) to recognize the facial expression being portrayed. Psychopaths in this study also made more errors identifying fearful faces than did the nonpsychopaths.

The debate continues regarding the specificity of any deficits in emotional processing associated with psychopathy. Some researchers suggest that psychopaths may be impaired in the identification of both negatively and positively valenced expressed emotion (Dolan & Fullam, 2006; Habel et al., 2002; Hiatt et al., 2002), whereas others suggest that the deficit may be specific to negative emotions (Blair et al., 2005; Blair et al., 2004; Blair et al., 2002; Kosson, Suchy, Libby, & Mayer, 2002; Marsh & Blair, 2008). There have also been conflicting results regarding whether the processing of specific emotions are impaired. Some authors (e.g., Kosson, Suchy, Libby et al., 2002) have indicated psychopaths are particularly unable to identify disgust, while others suggest the processing of fearful affect is most problematic. For example, in a recent meta-analysis, Marsh and Blair (2008) examined 20 studies, and found that psychopathy was associated with a specific deficit in the recognition of fear, a result that was unrelated to general task difficulty. It was also the

processing of fear that presented the greatest challenge to those psychopaths engaging in the emotional face flanker task reported earlier in this thesis (chapter two).

However, some investigations do not report this association (Glass & Newman, 2006), and others have suggested that, instead of being associated with deficits in emotional processing, psychopaths may be able to understand and judge the meaning of emotional expressions while being unable to react in emotionally appropriate ways to the social cues offered by other individuals (Lorenz & Newman, 2002). For example, Book et al. (2007) reported that offenders with a range of scores on psychopathy performed no differently than controls on a measure requiring the categorization of emotional facial expressions. However, psychopathy was associated with the ability to correctly judge non-verbal cues of assertiveness and vulnerability. Book et al. christened the ability to comprehend other's emotional states but not respond empathetically to them "callous empathy." These data suggest that psychopaths do know what other people are feeling (and can use this information to exploit and victimize them), but that they show a marked lack of concern regarding those emotions (Book et al., 2007).

Neurocognitive Function in Psychopathy

As noted earlier, the ACC, particularly the ventral regions, has been associated with the processing of affective information through its connection to limbic regions like the orbitofrontal cortex, amygdala, and insula (Bush et al., 2000; Devinsky et al., 1995). These links would be consistent with the growing

evidence that psychopathy may be associated with deficient amygdala function (Blair, 2003). Amygdala dysfunction could compromise the ability to react appropriately to emotional information (particularly fear) appropriately, preventing the psychopath from integrating the perception of fear with cognition and motor actions. It is also possible that even if some emotional processing does occur it does not get integrated at the level of the ventral ACC.

The debate also continues regarding the adequacy of cognitive and attentional processes in psychopaths. Such processes are often associated with dorsal ACC function (Bush et al., 2000). As described earlier, these deficits involve orienting and disengaging selective attention during task performance (Kosson, 1996), responsivity to peripheral task contingencies (Bernstein et al., 2000; Hiatt et al., 2004; Newman & Kosson, 1986; Newman et al., 1997), reduced inhibitory control (Howland et al., 1993; Kiehl, Smith et al., 2000; LaPierre et al., 1995), and so on. Thus, it may be that deficits occur in all major aspects of ACC function, cognitive, affective, and visceral. As such, it would not simply be a matter of failed integration across these domains but a general impairment of neurovisceral function involving both cognition and affect. The fact that attentional and inhibitory control deficits were not observed in the two previous data sets reported in this thesis may reflect a lack of power due to a relatively small sample size and/or the possibility that the letter flanker task reported in chapter two was not as difficult a discrimination as the face-flanker task where deficits were shown.

Another issue that has been central in the context of psychopathy research involves the classification of psychopathy as representing either a separate taxonomic category (e.g. Harris et al., 2007; Harris et al., 1994; Skilling et al., 2001) or the end of a continuum of normally distributed personality traits thought to make up the disorder (e.g. Edens et al., 2006; Marcus et al., 2004; Marcus, Lilienfeld, Edens, & Poythress, 2006). While many studies to date have examined this question from a psychometric perspective, there has been little investigation of whether psychophysiological traits associated with psychopathy vary in nonclinical populations in the same fashion as they do in clinical populations. It is currently unclear whether psychopaths represent a discrete class of individuals that are qualitatively different from nonpsychopaths, or simply represent an extreme point on a continuum of psychopathic personality traits that are normally distributed throughout the population.

The current study is intended to replicate and extend the work reported in chapter two, in which it was found that psychopathy was associated with error monitoring deficits during affectively-salient tasks, in an incarcerated sample of violent offenders that varied on psychopathic traits. The current study was designed using the same parameters as described for the incarcerated sample in chapter two. An index of ACC function was based on the ERN and Pe, which typically appear to be generated in the ACC (Falkenstein et al., 1991; Gehring et al., 1993). The ERN is thought to reflect performance monitoring processes (Bernstein, Scheffers, & Coles, 1995; Coles et al., 2001; Falkenstein

et al., 1990; van Veen & Carter, 2002a) while the Pe, associated with dorsal anterior or posterior regions of cingulate cortex (Herrmann, Rommler, Ehlis, Heidrich, & Fallgatter, 2004; O'Connell et al., 2007; van Boxtel, van der Molen, & Jennings, 2005; van Veen & Carter, 2002a), is usually thought to reflect the conscious components of error processing (Endrass, Franke, & Kathmann, 2005; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001; Overbeek, Nieuwenhuis, & Ridderinkhof, 2005).

If the traits associated with psychopathy are continuous, one would expect to see a distribution of sublicinical levels of psychopathic personality traits even among a high-functioning sample of University students. Furthermore, one would predict similar patterns of deficits among those higher in psychopathic personality traits to those noted in the psychopaths in chapter two. In this study, in addition to the (potentially) too easy letter-flanker task, a more complicated version of the letter flanker task developed by van Veen and Carter (2002a) was included to better equate task demands (such as the presence of multiple stimuli mapped onto a single response) between the letter and face-flanker tasks

Methods

Participants

Sixty-seven male University of Waterloo undergraduates, 17 to 25 years of age were selected from a screening of 1,500 students who completed the Self-Report Psychopathy scale (Paulhus, Hemphill, & Hare, in press), a measure designed to capture psychopathic personality traits in nonclinical populations. This scale measures psychopathy along four factors: Erratic Lifestyle, Callous Affect, Interpersonal Manipulation, and Antisocial Behaviour. Selection was done in a pseudo-random manner to ensure adequate representation across the full range of test scores. All participants were screened for psychiatric disorders and history of head injury. Those who participated for course credit received two credits towards their final course grade; those who participated for pay received CAN$16. After indicating their informed consent, participants completed a 2-hour testing session comprised of several ERP tasks.

Procedures

The Easy Letter Flanker Task. EEG was collected while participants were engaged in three flanker tasks. The letter flanker task (Eriksen & Eriksen, 1974) involved the presentation of a series of five-letter strings made up of either the letter H or the letter S presented on a computer monitor as white letters against a black background and are described fully in chapter two (pp. 21-22).

The Difficult Letter Flanker Task. The more difficult letter flanker task (van Veen & Carter, 2002b) involved the presentation of a series of five-letter

strings made up of the letters H, P, S and X. As in the previous task, these were presented on a computer monitor as white letters against a black background. Two letters on each end of the string were either congruent (SSSSS, PPPPP) or incongruent (SSXSS, HHSHH) with the center letter. Instructions appeared on the screen and were read to participants who were required to respond by key press with one hand if the central letter was an "H" or a "P," and with the other hand if it were an "S" or an "X". Prior to each trial a fixation cross appeared in the centre of the screen for 300 ms. The flanking letters then appeared for 100 ms before the target stimuli in order to increase the interference effect of the flanking stimuli. There were 180 trials, with test stimuli appearing on the screen for 190 ms. Inter-trial intervals varied from 500 – 1500 ms.

The Face Flanker Task. The face flanker task consisted of the same series of black-and-white photographs of faces that had been modified (Santamaria, 2003) from an initial set of emotional faces developed by Gur and colleagues (Gur et al., 2002). Again, task parameters and procedures are described in full in chapter two (pp. 22-23). As was the case with the incarcerated sample, participants made no errors in the identification of emotions when presented with no time constraints prior to task initiation.

Stimulus Presentation

Tasks were presented as previously described. The letter flanker tasks were presented first, followed by the face flanker task. Brief breaks (1 to 2 minutes) were given between blocks of trials. During all tasks, the importance of both speed and accuracy were emphasized. Due to discrepancies in task

difficulty, all participants received the easy letter flanker tasks first to allow them to familiarize themselves with the general flanker paradigm prior to having to deal with the more difficult tasks to follow. The stimulus presentation and data acquisition program used was E-Prime (Psychological Software Tools, Inc., 2004).

Electrophysiological recording and processing. Rather than using the BioSemi EEG data collection system as described in previous chapters, for this study a 64-channel QuickAmps system (www.brainproducts.com) was used due to its availability for installation at the Waterloo testing site. Eye-movements were recorded with three electrodes placed on the outer canthus, supra-orbital ridge, and cheekbone of the right eye. Signals were sampled at the rate of 500 points per second and digitized with a 22 bit ADC. A bandpass filter from 1 Hz (time constant 0.16s) to 30 Hz is used to filter the EEG data. All electrodes were re-referenced offline to averaged mastoids. Vision Analyzer (Brain Products GmbH, München) was used to correct for both vertical and horizontal eye artefacts using automated eye-correction procedures (Gratton et al., 1983).

Stimulus-locked, artifact-free EEG recordings were averaged relative to a -400 ms to -600 ms pre-response baseline, with response-locked P3 amplitude defined as the most positive component around the time of response, ERN amplitude defined as the most negative value at frontocentral sites in the 50 – 200 ms following the response. The Pe was defined as the most positive component in the 200 ms – 400 ms period following the response. When examining ERPs, amplitudes on correct trials were entered into the equation

prior to those on error-related trials to control for any effect of interpersonal differences in general electrophysiological power. The data from 21 participants were excluded due to excess artifact, too few trials to permit reliable analyses, or outlying behavioural or electrophysiological performance on one of the three tasks. The mean number of trials per ERP average was 101 (± 3.58). No average contained fewer than nine trials. Because of technical issues that occurred during recording, source analyses cannot be performed on this data set.

Statistical analysis. Analyses across tasks were conducted using repeated-measures ANOVAs, with task (easy letter flanker, difficult letter flanker and face flanker) and midline sites (Fz, FCz, Cz, and Pz) as within-group factors. Correlations and forced-entry regression analyses were carried out to determine the relations between behavioural indices of performance and the amplitude of each component with psychopathy (as indexed by SRP-III score). All statistical analyses were corrected for violations of Mauchley's Test of Sphericity where necessary, utilizing the Huynh-Feldt correction for estimating the F-Statistics with original degrees of freedom presented in the text. The Bonferroni correction for multiple comparisons was used where appropriate.

Results

Behavioural Responses

Error responses included the congruency factor, although there were no a priori hypotheses regarding this factor. As the complexity of the difficult flanker task did not lend itself to simple congruency analyses, only data from the easy letter

flanker and face flanker tasks were examined. As is common, a main effect of congruency was noted, F (1,44) = 14.41, p <.001, η^2 = .25, such that there were more errors on incongruent than congruent trials. Because the face flanker task had an overall higher error rate, there was also a main effect of task, but this was superceded by a task by congruency interaction, F (1,44) = 9.75, p = .003, η^2 = .18, such that the most errors were committed on incongruent face flanker trials; however, congruency was unrelated to psychopathy (all *p's* > .6), so trials were collapsed across congruency for further analyses.

Accuracy and response time (RT) data are presented in Table 4.1. Behavioural data were analysed with a one-way ANOVA using error rate as the dependent variable. This showed that the task difficulty manipulation was successful, such that participants made more errors on the face flanker task (M = .22 ± .02) and difficult letter flanker task (M = .24 ± .02) than on the easy letter flanker task (M = .15 ± .01), F(2, 88) = 12.81, p = < .001, η^2 = .23. Regression analyses were conducted to follow up on these results. SRP-III scores (the Total Psychopathy summary score as well as each subscale) were used as the dependent variables, and performance (proportion of errors) was regressed against them for each of the tasks separately. These analyses showed that error rate, irrespective of task, was unrelated to Total Psychopathy score (all *p*s >.05). For the face flanker task the degree to which type of emotion played a part in error rate was also examined. In contrast to the results reported in chapter two, those higher in psychopathy were not more likely to make errors on fearful than angry faces, r = .23, p = .13.

Response times were analysed using a 2 (response time on correct and error trials) by 3 (easy and difficult letter flanker, face flanker task) repeated-measures ANOVAs. This analysis indicated that there was a main effect of accuracy, such that erroneous responses (M = 450 ± 10 ms) had shorter RTs than did correct responses (M = 489 ± 10 ms), $F(1, 44) = 112.85$, $p < .001$ $\eta^2 = .72$. As is typical in ERN studies, the shorter RTs on error trials suggests that error responses represent impulsive slips rather than considered decisions. There was also a main effect of task, such that participants had longer RTs on the face flanker task (M = 567 ± 17 ms) than on either the easy (M = 416 ± 8 ms) or difficult (M = 427 ± 7 ms) letter flanker tasks, $F(2, 88) = 101.64$, $p < .001$ $\eta^2 = .70$. There was also a task by accuracy interaction, such that correct responses on the face flanker task were longer than responses in the other conditions, $F(2, 88) = 9.15$, $p < .001$ $\eta^2 = .17$.

The next question was whether response times were associated with psychopathy. To answer this, total psychopathy was entered into a regression equation as the dependent variable, with RT on correct trials as the predictor. RT was not associated with Total Psychopathy on any of the three tasks (all *p's* > .4).

Electrophysiological Responses

Although psychopathy was treated as a continuous variable during data analyses, for the purpose of illustration, grand-mean results from the top and bottom one-third of SRP-III scores across all tasks are shown in Figures 4.1, 4.2 and 4.3. Topographical maps of the same are shown in Figure 4.4. ERN

amplitude differed as a function of accuracy in the expected fashion: ERNs were larger on error trials (M = -4.41 ± .27) than on correct trials (M = -1.07 ± .19), $F(1, 44) = 159.27$, $p < .001$, $\eta^2 = .78$. There was also a task by accuracy interaction, such that face-flanker errors elicited generally smaller-amplitude ERNs (M = -3.36 ± .29 μV) than did errors on either the easy (M = -4.93 ± .42 μV) or hard (M = -4.94 ± .36 μV) letter flanker tasks, $F(2, 88) = 7.04$, $p = .002$ $\eta^2 = .14$. ERNs associated with error trials on the various tasks were then used in a series of follow-up regression analyses to examine their relationship to psychopathy scores. Results indicated that only face-flanker ERN amplitude predicted SRP-III Total Psychopathy score, $F(1, 42) = 4.77$, $p = .035$ (see, Figure 4.5). As can be seen in Figures 4.6 - 4.8, this was primarily driven by a relation between the Erratic Lifestyle factor and ERN amplitude on this task, $F(1, 42) = 4.84$, $p = .03$.

The Pe occurred as two consecutive peaks approximately 150-350 ms after the erroneous response and was analysed in the same fashion as the ERN. There was a main effect of accuracy such that the earlier Pe was markedly larger on error trials (M = 4.10 ± .32μV) than on correct trials (M = 1.83 ± .19μV), $F(1, 44) = 33.91$, $p = < .001$, $\eta^2 = .44$. Like the ERN, the Pe was larger on the easy (M = 3.01 ± .23μV) and hard (M = 3.48 ± .28μV) letter flanker tasks than on the face flanker task (M = 2.37 ± .23μV), $F(2, 88) = 7.58$, $p = .001$, $\eta^2 = .15$. However, Pe amplitude on all three tasks was associated with psychopathy (see, Figures 4.9 – 4.12). After adjusting for Pe amplitude on correct trials, the Pe amplitude associated with error trials on the easy letter flanker task explained

a significant amount of variance of Total Psychopathy scores, $F(1, 42) = 4.17$, $p = .047$. This was primarily driven by the Erratic Lifestyle subscale, $F(1, 42) = 5.44$, $p = .025$. Participants with higher SRP-III scores generated smaller-amplitude Pe's on this task. Performance on the difficult flanker task showed the same general pattern. Higher Total Psychopathy scores were associated with decreased Pe amplitude, $F(1, 42) = 5.0$, $p = .031$. In this case, variance in Pe amplitude was primarily related to the Callous Affect, $F(1, 42) = 5.75$, $p = .021$ and Erratic Lifestyle, $F(1, 42) = 5.27$, $p = .027$, subscales.

Performance on the face flanker task was consistent with that on the other tasks. Participants with elevated Total Psychopathy scores produced smaller Pe's, $F(1, 42) = 9.32$, $p = .004$, an effect driven by the Interpersonal Manipulation, $F(1, 42) = 6.02$, $p = .018$ and Erratic Lifestyle subscales, $F(1, 42) = 13.82$, $p = .001$. Pe latencies were unrelated to psychopathy on all tasks. Examination of the second Pe peak revealed that while it was larger for error than correct trials, it was unrelated to psychopathy in both flanker tasks (all p's $< .1$).

These results indicate that participants with higher Total Psychopathy scores had decreased Pe amplitudes to errors on all tasks. Whereas this is not consistent with the results reported in chapter two, using criminal psychopaths, the sample used in the previous study ($M = 2.81$ $SE = .77$ vs $M = 3.55$, $SE = .34$, in the current study) had a large amount of variability in Pe amplitude, which may have reduced effect sizes. However, it does suggest that some aspects of error monitoring may be sensitive to subclinical psychopathy. While multiple

comparisons such do increase the possibility of Type I error in the current analyses all relations are in the predicted direction, while true false positive relations may be more likely to be random, arguing against this as a factor in the results of these analyses.

Discussion

The focus of this study was the investigation of error monitoring, and by extension, ACC function, in a sample of university students with a full range of scores on a self-report measure of psychopathic traits. As the ACC is implicated in both error monitoring and the processing of emotional stimuli, tasks were included that required participants to make judgements about both types of information. Results indicated that psychopathy was unrelated to the actual error rate irrespective of task or type of error. As well, psychopathy scores were unrelated to ERN amplitude on either of the emotionally-neutral letter flanker tasks. These data support the view that psychopathy is not associated with deficient ACC-based error monitoring functions in general. However, as was found in the incarcerated sample reported in chapter two, higher psychopathy scores were related to smaller ERNs elicited by error during the emotional face flanker task. These results are consistent with the hypothesis that psychopathy involves problems processing emotional stimuli. These results are also consistent with those of a number other studies that have reported ERP abnormalities in psychopaths in the absence of any other behavioural differences (Kiehl et al., 1999; Kiehl et al., 2004).

In the current study, decreased Pe amplitudes were associated with psychopathy across all the tasks, suggesting that this aspect of error monitoring may be generally disrupted in those with psychopathic personality traits. These results are consistent with those of Hall et al., (2007). While these authors do not report Pe amplitude results, visual inspection of the figures provided suggests that the Pe on error trials is much smaller in the high- than low-externalizing group, with externalization serving as a proxy measure for psychopathy.

Whereas the ERN is thought to reflect automatic performance monitoring processes, the processes reflected in the Pe are less clear. Nieuwenhuis et al. (2001) used an antisaccade task to examine error monitoring. This paradigm generally elicits reflexive errors of which the participant is supposedly unaware. They found that, while both perceived and unperceived errors were followed by ERNs of similar amplitude, Pe amplitude was significantly larger to errors that the participant was aware of committing. The authors interpreted these results as suggesting that the Pe may reflect conscious error monitoring processes. Other researchers have reached similar conclusions using a number of different paradigms (Davies, Segalowitz, Dywan, & Pailing, 2001; Falkenstein et al., 1991; Leuthold & Sommer, 1999; Nieuwenhuis et al., 2001; Pailing & Segalowitz, 2004b; Vidal, Hasbroucq, Grapperon, & Bonnet, 2000), and have suggested as well that it may represent the degree to which one cares about having made the error.

If the Pe does reflect an index of either error salience or the emotional reaction to having made an error, then the reduced Pe in those with higher levels of psychopathic traits suggests a tendency on the part of these participants to either pay less attention to or have smaller emotional reactions to the errors they commit. This interpretation is consistent with behavioural studies indicating that psychopaths may be relatively insensitive to exogenous performance feedback such as that given in passive avoidance tasks (Lykken, 1957; Newman, 1998), so it is possible that they are also insensitive to endogenously generated indicators of performance, such as the processes associated with the generation of the ERN.

The inclusion of the more difficult flanker task allowed us to control for the potentially confounding effect of task difficulty, since the face flanker task is more visually complex and requires more response decisions than the standard Eriksen letter flanker task. There was no evidence of a relationship between either error rates or the size of ERNs on this task and levels of psychopathy, suggesting that the ERN effects seen on the face flanker task in this and the previous investigation with incarcerated offenders were not likely due to discrepancies in task difficulty, but rather related to the affective relevance of the stimuli. Indeed, there were few differences in the electrophysiological response to the easy and difficult letter flanker tasks, despite differences in error rates.

Whereas this is not entirely consistent with what was seen in the sample of PCL-R diagnosed psychopaths, it is interesting that this effect can be

observed in a group of university students, a population significantly removed from the incarcerated violent offenders included in the previous study. The fact that the neurophysiological patterns that have been observed in clinically–assessed populations can be replicated in subclinical populations who score high on psychopathy, suggest that psychopathy traits are normally distributed (with clinically diagnosed psychopaths representing one very extreme end of this continuum).

One of the findings observed in the incarcerated offender sample that was not replicated here was the relation between psychopathy and deficits in identifying fearful facial expressions. In the earlier investigation, psychopaths had higher error rates and smaller ERP amplitudes when the target face was expressing fear. There is a body of evidence supporting an association between psychopathy and deficits in the processing affective stimuli, especially fearful emotions. For example, Blair et al (2004)reported that both children and adults with psychopathic personality traits took longer to recognize fearful, but not other emotional, facial expressions. Similar results have been reported by other studies examining emotional facial expression recognition (Montagne et al., 2005), and also fearful vocal affect (Blair et al., 2002; Hiatt et al., 2002). However, many of the studies reporting fear-specific deficits have been conducted with clinically diagnosed psychopaths, who would be at the highest end of the distribution, i.e., they would be more psychopathic than even those who scored at high levels of psychopathy on the SRP-III. Thus, even if those at the upper end of our sample of university students were having some difficulty

processing or responding to fearful faces (as seen in their reduced ERN to face-flanker errors), this may not have markedly impaired their ability to make the discrimination between fear and anger in the context of the face flanker task.

Psychophysiologically, the abnormal ERN amplitude during the processing of emotional stimuli is consistent with the presence of limbic and paralimbic dysfunction in psychopathy. ACC and amygdala dysfunction in particular have been associated with this disorder (Kiehl, Smith et al., 2001; Müller et al., 2003). Functional limbic abnormalities have also been reported in normal participants scoring highly on a measure of psychopathy (Gordon et al., 2004) and in youths diagnosed with conduct disorder (Sterzer et al., 2005). In several cases, this decreased limbic activation is associated with increased cortical activity, especially in prefrontal and frontal-temporal cortical regions (Intrator et al., 1997; Kiehl, Smith et al., 2001). This pattern of limbic hypo- and cortical hyper-activity has been interpreted as reflecting a unique response strategy – namely, that because psychopaths are impaired in their ability to utilize limbic regions to process affective information, other cognitive regions are instead recruited (Intrator et al., 1997). Given the multiple aspects of ACC function (Bush et al., 2000; Devinsky et al., 1995), it is possible that the ACC dysfunction seen in psychopaths is either the result of amygdala or hippocampal dysfunction, abnormal ventral ACC activity, or both. However, because dorsal cingulate regions remain relatively unaffected, the processing of neutral stimuli remains normal, as do many other measures of ACC function (Dvorak-Bertsch et al., 2007).

There are several limitations that should be noted when considering the results of this study. Primarily of concern was the use of a self-report measure of psychopathy, which may be more susceptible to deceit or the effects of social desirability. Many clinical measures of psychopathy avoid this by considering data from both interview and the review of personal records (Hare, 1991, 2003). However, if social desirability influenced responses to the SRP in the current sample, in general this would increase the noise in the data, as more psychopathic participants would have received scores lower than they deserved, thus decreasing the likelihood of finding a statistically significant association between psychopathy and electrophysiological activity.

The results of this study have shown that the diminished ERN to emotional stimuli observed in the incarcerated offender sample was also observed in a young, healthy sample of university undergraduates for those at the highest levels of the SRP-III. As was the case in that other sample, there were no signs of reduced ERNs associated with errors in either letter flanker task, irrespective of level of task difficulty. This, and the fact that the diminished ERN was associated with psychopathic personality characteristics even in the absence of differences in task performance, leaves little doubt that the sensitivity of ERN amplitude to psychopathic tendencies is not due to task difficulty levels or generally poorer performance. These data offer support for the paralimbic dysfunction hypothesis, suggesting that psychopaths may show functional neural abnormalities when forced to process affectively-relevant information. These data also suggest that psychopathic personality traits are

normally distributed within the population, and are associated with the same psychophysiological markers as those shown by those with clinically significant levels of this disorder.

CHAPTER FIVE: SUBCLINICAL PSYCHOPATHY, ERPS, AND ATTENTION.

Introduction

The examination of attentional anomalies in psychopaths has a fairly long history. Many early studies suggest that psychopaths show a range of attentional abnormalities (e.g. Lykken, 1957; Newman & Kosson, 1986). As previously described, Newman and colleagues (Newman, 1998) suggest that the central problem may involve a deficit in the automatic, bottom-up processing of secondary stimuli. This manifests itself in several ways, such as the reduction in electrocortical responses during the performance of dual tasks (Hare & Jutai, 1988) and reduced interference during Stroop tasks from the to-be-ignored dimension on incongruent trials (Hiatt et al., 2004). Another approach has been to record event-related potentials (ERPs) while participants engage in various types of attentional tasks. However, variation in the definition of psychopathy and in the nature of the tasks used to elicit ERPs may have contributed to a continuing lack of consensus regarding some aspects of these data.

One well-studied ERP component has been the P3 or P300 (e.g., Sutton, Braren, Zubin, & John, 1965), a positive-going component that occurs approximately 300 ms after stimulus onset (Polich, 2007). It is commonly elicited by infrequent targets presented within the context of frequent non-target stimuli, i.e., the standard oddball task (e.g. Donchin & Coles, 1988) and has been thought to represent the updating of a mental representation (Donchin, 1981) or to serve as an index of attentional control and arousal (e.g. Kok, 1990;

Polich & Kok, 1995). In recent reviews, the P3 has been described as an attention-driven stimulus signal generated in temporal/parietal structures (Polich, 2007; Polich & Criado, 2006). Recent evidence also links the P3 to the locus coeruleus-norepinephrine system (see Nieuwenhuis, Aston-Jones, & Cohen, 2005), and suggests that it can be considered the scalp representation of internal decision processes involving motivationally significant events, although it has also been linked to memory processes by Polich and colleagues (Polich, 2007; Polich & Criado, 2006).

Researchers have used the P3 as a means to understand aspects of attention in the context of both internalizing and externalizing disorders. Results with respect to internalizing disorders have been somewhat inconsistent (e.g., Bange & Bathien, 1998; Bruder et al., 2002; Bruder et al., 1995; Kayser, Bruder, Tenke, Stewart, & Quitkin, 2000; Pfefferbaum, Wenegrat, Ford, Roth, & Kopell, 1984), although there has been some suggestion that the contradictory results may be the result of inconsistencies in task difficulty and design (Tenke et al., 2008).

In contrast, there is an extremely well-established association between P3 amplitude and externalizing psychopathologies, particularly antisocial personality disorder (e.g. Bauer & Hesselbrock, 1999; Bauer, O'Connor, & Hesselbrock, 1994; Iacono, Carlson, Malone, & McGue, 2002; Iacono & McGue, 2006; Patrick et al., 2006). Not only are individuals diagnosed with an externalizing disorder more likely to display attenuated P3 amplitudes, but disruptions in the attentional processes indexed by this component may reflect a

vulnerability to the development of an externalizing disorder in healthy individuals with family histories of these conditions (e.g., Iacono et al., 2002; O'Connor, Bauer, Tasman, & Hesselbrock, 1994). This has been supported by at least one large-scale study indicating that this relation is strongly mediated by genetic factors (Hicks et al., 2007). Taken together, these data suggest that the P3 may be an endophenotypic marker of a genetic vulnerability to disinhibitory conditions, especially conduct disorder and antisocial personality disorder (e.g. Patrick et al., 2006). It is currently unclear whether this relation extends to psychopathy, a condition which shares many common features with antisocial personality disorder.

Attempts have been made to explore the P3 index of attentional allocation in psychopathy. For example, Raine and Venables (1988), using a continuous performance task, reported larger amplitude P3s in a psychopath group compared to controls. They concluded that psychopathy is associated with an increased ability to attend to task-relevant events, and potentially enhanced information processing abilities. However, based on more recent work, Kiehl et al. (1999) have challenged these results. They tested 21 male prison inmates using a visual oddball task and found that those diagnosed with psychopathy generated markedly smaller P3s to target stimuli than did nonpsychopath controls. The authors concluded that these results were consistent with a reduced ability on the part of psychopaths to adequately sustain or orient attentional resources during task performance. However, these results have not been consistently replicated in subsequent studies using

auditory oddball and go/nogo tasks (Kiehl, Bates et al., 2006; Kiehl, Smith et al., 2000). Likewise, many early studies reported no consistent group differences in P3 amplitude between psychopaths and nonpsychopaths (e.g. Forth & Hare, 1989; Syndulko, Parker, Jens, Maltzman, & Ziskind, 1975; Williamson et al., 1991). Thus, the degree to which psychopathy is associated with an abnormal P3 is still unclear, as is the larger issue of whether psychopathy is associated with general attentional abnormalities (Kiehl, 2006; Kosson, Miller, Byrnes, & Leveroni, 2007) or is specific to abnormal function when dealing with emotional information.

More recently, there have been reports of an association between psychopathy and a negative ERP component occurring approximately 500 ms after stimulus onset referred to as the N5 (Kiehl, Bates et al., 2006; Kiehl et al., 1999). The N5 directly follows the P3 and is maximal at frontal sites. It also appears to be enhanced in psychopaths relative to nonpsychopaths during a variety of decision-based visual tasks (Kiehl, Bates et al., 2006; Kiehl et al., 1999). This component has also been elicited in psychopaths during the processing of emotional and nonemotional words (Williamson et al., 1991). However, the generators of the N5 and the processes reflected in this component are not well defined.

The goal of the current study is to further explore the degree to which psychopathy is associated with attentional allocation deficits that would be manifest in abnormal P3 amplitudes. Given that the current understanding that the P3 is dependent on the locus-coeruleus-norepinephrine (LC-NE) system

(e.g., Nieuwenhuis et al., 2005; Polich, 2007; Polich & Criado, 2006), a marked reduction in P3 amplitudes in conjunction with high levels of psychopathy would suggest that psychopathy is associated with alterations in the LC-NE system, although there is no evidence supporting this supposition. This would support the view that psychopathy is associated with an impairment in attentional systems (Newman, 1998). If however, the P3 is of equivalent or greater amplitude relative to controls, it would support the view that attentional abilities *per se* are not markedly diminished in psychopathy or if psychopathic individuals show greater than normal P3s, that attentional focus might actually be enhanced (Raine & Venables, 1987, 1988).

The current discrepancies in the literature may be due to various aspects of the research paradigms used to date. Because much but not all (see Kiehl, Bates et al., 2006) of the electrophysiological evidence of abnormal attention in psychopaths comes from studies using small groups and a variety of tasks, including go/nogo, visual and auditory oddball tasks, and tasks designed to elicit the contingent negative variation component, the plan here was to examine this issue in a relatively large sample of university undergraduates with a full range of scores on the SRP-III. The P3 was elicited using a standard version of a visual oddball task. If psychopathy is indeed associated with a general deficit in attention orientation, one would expect to see attenuated P3 amplitudes in those who scored higher on the psychopathy rating scale. Also examined was the relation between psychopathic tendencies and the N5. Reproducing the relations between the N5 and psychopathy reported by Kiehl and colleagues (Kiehl,

Bates et al., 2006; Kiehl et al., 1999) in this subclinical sample would speak to the strength and reproducibility of this effect and prompt researchers to more fully examine the actual nature of this association, i.e., what it means with respect to the ability to allocate attentional resources, an issue that has still to be explored.

Methods

Procedures

Participants and general procedure were the same as reported in chapter four. EEG was collected while participants were engaged in a visual oddball task involving the presentation of two cartoon-like images: one of a red convertible car, and the other of a predominantly green deciduous tree. Instructions appeared on the screen and were read aloud. Participants were required to respond by key press if the tree was presented and to withhold their response on trials when presented with the car. Test stimuli appeared on the screen for 100 ms, with an inter-trial interval of 1000 – 1600 ms. A total of 200 trials were presented in one block, with targets making up 25% of all trials. The session began with 16 practice trials to ensure participants understood task requirements. The stimulus presentation and data acquisition program used was E-Prime (Psychological Software Tools, Inc., 2004).

EEG recording and processing. Stimulus-locked, artifact-free EEG recordings were averaged relative to a -200 ms to 0 ms pre-response baseline, with P2 amplitude defined as the most positive value at frontal sites in the 100 – 300 ms following stimulus onset. P3 amplitude was defined as the most positive

value at central-parietal sites in the 200 – 400 ms period. The N5 was defined as the most negative component in the 200 ms following the return of the P3 to baseline.

Statistical analyses. Correlations and forced-entry regression analyses were carried out to determine the relationship between behavioural indices of performance and the amplitude of each component with psychopathy (as indexed by SRP-III score). The data from one participant were excluded because of excess artifact. The mean number of trials per ERP average was 70 (± 25). Because of technical issues that occurred during recording, source analyses cannot be performed on this data set.

Results

Behavioural Responses

Participants had no difficultly identifying or responding to target stimuli correctly (*M* accuracy = 98.9%). They committed relatively few false alarms to nontarget stimuli (*M* = 0.003%). The mean response time for target trials was 400 ms. Neither error rate nor response times were related to psychopathy. Although psychopathy was treated as a continuous variable, for the purposes of illustration, the depiction of ERP waveforms in Figure 5.1 are based on SRP-III scores from the top and bottom quartiles of the distribution for purposes of comparison.

Electrophysiological Responses

Grand-average EEGs for the top and bottom quartiles can be seen in Figure 5.1. The relation between SRP-III scores and ERPs was examined using

Total Psychopathy and scores from the four SRP-III subscales as dependant variables, and regressing ERP averages against them. Unless otherwise specified, results are reported for the Total Psychopathy score (Figure 5.2), which represents the sum of scores on the SRP-III subscales.

P2 amplitude on nontarget trials was unrelated to SRP-III scores, (all *p's* > .1). Next, with P2 amplitude on nontarget trials entered on the first step, P2 amplitude on target trials was entered on the second step. This accounted for a significant amount of variance in Total Psychopathy score, $F(1,46) = 5.05$, $p = .029$, as well as on the Antisocial Behaviour subscale, $F(1,46) = 7.13$, $p = .01$. Scatterplots depicting the relation between P2 amplitude and the SRP-III subscales can be seen in Figure 5.3. N2 amplitude was analysed in a similar fashion; however, it was unrelated to psychopathy irrespective of trial type (all *p*'s > .1).

P3 amplitude was analysed in the same way. Entering the P3 amplitude to nontarget trials on the first step did not account for a significant proportion of variance, $F(1,47) = .36$, *ns*. Entering P3 amplitude on target trials also failed to account for a significant proportion of variance in Total Psychopathy, $F(1,46) = .11$, $p = ns$, or on any of the subscales (all *p's* > .49), suggesting quite clearly that in this sample P3 amplitude was generally unrelated to variance in the SPR-III (see, Figure 5.4).

As was the case with previous analyses, entering nontarget N5 amplitude on the first step did not account for a significant proportion of variance in SRP-III Total Psychopathy scores, $F(1, 47) = .42$, $p = ns$, all other *p's* > .19 .

However, when the N5 to target trials was entered on the second step, it explained a significant amount of variance in both the SRP-III Total score, $F(1, 46) = 5.28$, $p = .026$, and Erratic Lifestyle subscale, $F(1, 46) = 7.12$, $p = .01$. There was also a trend towards a relation between the N5 and the Interpersonal Manipulation subscale, $F(1, 46) = 3.39$, $p = .072$. Participants with higher SPR-III scores, especially those with elevated scores on Erratic Lifestyle subscale, tended to generate N5s of larger (more negative) amplitude on target trials (see, Figure 5.5).

To ensure that this effect was related to the N5 itself and was not an artefact of minor variation in P3 amplitude, further regression analyses were conducted. Using the same dependent variables as in the previous analyses, nontarget P3 amplitude was entered on the first step, target P3 amplitude on the second, and then nontarget and target N5s on the third and fourth steps respectively. While none of the variables entered on the first three steps produced significant F changes, target N5 amplitude explained variance beyond that accounted for by the previous variables, $R^2 = .12$, $F(1, 44) = 6.03$, $p = .018$, in the prediction of the total SRP-III score. To examine whether P2 and N5 predicted different variance on the SRP-III, another regression equation was conducted. With total SRP-III score serving as the dependent variable, nontarget and then target N2 followed by nontarget and then target N5 were entered on sequential steps of a regression equation. Target P2 amplitude added significantly to the prediction of psychopathy ratings, $F(1, 46) = 5.05$, $p = .029$. Likewise, target N5 amplitude generally accounted for unique variance in Total

Psychopathy score beyond the earlier component, $F(1, 44) = 3.81$, $p = .057$. When examined as unique variance in the full model, each component contributes unique variance to the prediction of psychopathy (P2 = 6%; N5 = 7%).

ERP latencies were examined using the same pattern of analyses. Regressing ERP average latencies against SRP-III scores indicated that P2 nontarget latencies had no predictive value (all p's > .15); however, latencies associated with target trials entered on the second step it did account for variance on Total Psychopathy, $F(1,46) = 3.99$, $p = .052$, although this did not quite reach significance. P2 latencies did account for a significant amount of variance on the Callous Affect subscale, $F(1,46) = 7.32$, $p = .01$. In both cases, longer latencies were associated with higher psychopathy scores.

While N2 and P3 latencies were unrelated to psychopathy (all p's > .1), N5 latencies were associated with psychopathy. Entering N5 latency to nontarget trials on the first step did not account for a significant proportion of variance in Total Psychopathy score or for any of the subscales (all p's > .1). However, when N5 latency on target trials was entered it was evident that shorter N5 latencies were associated with higher scores on Callous Affect, $F(1,46) = 7.41$, $p = .009$, and showed a trend to be associated with higher scores on Total Psychopathy, $F(1,46) = 3.66$, $p = .062$.

Discussion

All individuals in the current sample produced robust P3s irrespective of their SPR-III scores, suggesting that, at least in nonforensic samples, the P3 is not

diminished as a function of psychopathic tendencies *per se*. These results are consistent with other reports using a range of tasks (Forth & Hare, 1989; Jutai, Hare, & Connolly, 1987), although Kiehl and colleagues have reported P3 amplitude reductions in large forensic samples (Kiehl, Bates et al., 2006; Kiehl et al., 1999). It is, of course, always possible that this may be due to the reduced range in psychopathic severity in the present sample to find a diminished P3. However, even in forensic populations, P3 amplitude reductions are inconsistently found and one might have to wonder about differences in the rate of other factors such as head injury within a particular group that might be reflected in the P3 data.

Nonetheless, neurocognitively, there is limited evidence to lead one to hypothesize abnormal P3 amplitude in this population. While there is substantial evidence linking paralimbic dysfunction with psychopathy (Blair, 2003; Kiehl, 2006; Kiehl, Bates et al., 2006; Kiehl, Smith et al., 2001; Müller et al., 2003), the P3 is associated with activation in the temporal-parietal junction, reflecting phasic activation of the LC-Ne system (Nieuwenhuis et al., 2005). The LC is the main source of Ne innervation to the forebrain, and its activity is thought to be associated with the determination of motivationally-significant responses. There is no evidence for dysfunction in the LC or the temporal-parietal junction in psychopaths. While the LC also provides the primary NE innervation of the hippocampus and amygdala, regions that some studies have shown to be dysfunctional in psychopaths (e.g. Müller et al., 2003; Raine et al., 2004), there is no evidence that these regions are involved in the generation of the P3

(Birbaumer, Elbert, Canavan, & Rockstroh, 1990; Johnson, 1988; Polich & Squire, 1993).

Whereas we found no evidence of an association between psychopathy and the P3, relations between psychopathy and other ERP components were evident. N5 amplitude at frontal sites on target trials negatively related to level of psychopathy, such that those with higher SRP-III scores tended to produce larger (more negative) and faster N5s. Latency was related to the Callous Affect subscale, while the relation with N5 amplitude was evident for the subscales Erratic Lifestyle and Interpersonal Manipulation but not for Callous Affect or Antisocial Behaviour. Erratic Lifestyle is analogous to the PCL-R's Lifestyle factor (Hare, 2003), which indexes traits such as irresponsibility, parasitic lifestyle, and impulsivity. Interestingly, Neumann, Hare, and Newman (2007) have recently reported that, in Hare's four-factor model, the Lifestyle dimension may be the most strongly related to psychopathy.

These N5 results are consistent with other reports (e.g., Kiehl, Bates et al., 2006), and directly replicate those of Kiehl et al. (1999), who also found N5 amplitude differences between groups for target trials. These authors speculated that enhanced N5s might be partially due to the decreased P3 amplitudes observed in that study. However, the current findings indicate that enhanced N5 amplitudes can be found among those high in psychopathy, even when P3 amplitudes are unrelated to psychopathy. In other words N5 amplitudes are a unique predictor of psychopathy. Kiehl et al. (2006) have suggested that this component may represent some aspect of motor or response modulation

although this model has yet to be tested directly. However, the presence of the N5 cannot necessarily be taken as evidence of an attentional deficit since there is very little corroborating evidence for a general response monitoring deficit in psychopathy (Dikman & Allen, 2000; Hall et al., 2007).

It is of interest, however, that a negative component similar to the N5 has been noted under certain conditions in nonpsychopathic populations. Koelsch, Gunter, Friederici, and Schroeger (2000) recorded an N5 in their participants during a music listening task. They found that the N5 was larger following musical chords that were unexpected or inconsistent with the existing musical context, suggesting that the N5 may be related to participants' sensitivity to violations of musical syntax. Thus, in these studies, the N5 may serve a similar purpose as the N4 does in linguistic tasks, reflecting some form of semantic integration (Kutas & Federmeier, 2000; Kutas & Hillyard, 1980). If the negativity seen in psychopaths is reflecting the same cognitive processes as the N5 seen during studies of musical syntax or the N4 in semantic priming studies, it would suggest that those aspects of psychopathy that lead to erratic lifestyle are also involved in the tendency to view the infrequent target stimuli as being unexpected. However, it must be noted that the only study to date examining the N4 in this population found no evidence of any association with psychopathy (Kiehl, Laurens, Bates, & Liddle, 2006). Likewise, the frontal scalp topography of the N5 is not entirely consistent with that of the N4, which tends to be maximal at centro-parietal sites (Williamson et al., 1991). Thus, it is clear that there is more to learn about the meaning and implications of the N5,

but from what is known, there is little evidence to suggest that it is a sign of impairment rather than enhancement of focussed attentional capacity.

There was also an association between enhanced P2 amplitudes and psychopathy. P2 amplitude at frontal sites during target trials was positively related to psychopathy, such that those with higher SRP-III scores, especially on the Antisocial Behaviour subscale, tended to produce P2s of larger amplitude and longer latencies. The P2 has been associated with basic information processing functions such as stimulus classification (e.g., Crowley & Colrain, 2004). Like the results reported in chapter three, which suggested that incarcerated violent offenders who scored highly on the PCL-R showed no sign of impairment on electrophysiological indices of inhibitory control, the increase in P2 amplitude and latency also argues against increased impulsivity or electrocortical hyper-reactivity in this population. Moreover, the P2 and N5 were shown to account for separate unique variance in the prediction of psychopathy. However, in both cases, these components are associated with increased precision in the parsing of an ongoing stream of information.

Flor et al. (2002) have also reported evidence of electrocortical over-responding without the presence of abnormality in other earlier components. They found that psychopaths tended to generate contingent negative variations (CNVs) of larger amplitude than their nonpsychopathic counterparts. The CNV is generally considered to be an index of attentional expectancy, i.e., the attention allocated to the expected onset of a targeted event (Walter, Cooper, Aldridge, McCallum, & Winter, 1964). On the basis of these findings, Flor et al.

(2002) suggest that psychopaths demonstrate superior attentional mobilization when anticipating events. This, along with the current data, is consistent with Raine and Venables' (1988) over-focusing hypothesis, i.e., that psychopathy is associated with the enhanced processing of the most central or task-relevant information.

A potential consequence of over-focusing on the most central items during an attentional task is the tendency to under-respond to peripheral information (Newman & Kosson, 1986; Newman et al., 1997). There are, for example, a number of reports showing that psychopaths show normal Stroop interference during color-letter Stroop tasks (Dvorak-Bertsch et al., 2007; Hiatt et al., 2004; Smith et al., 1992), but demonstrate reduced interference when the unattended dimension (i.e., the word) is spatially separated from the attended dimension (i.e., the to-be-named colour). Newman (Newman & Schmitt, 1998) hypothesized that, while psychopaths show normal goal-directed (top-down) processing, they differ from nonpsychopaths in their ability to ignore peripheral stimuli that should elicit a reflexive (bottom-up) attentional response (like the automatic reading of the word in a Stroop task). We note, however, that the ability to inhibit the processing of task-irrelevant stimuli is usually seen as a good thing, i.e., it speaks to the efficiency of attention allocation in the face of interference (e.g., Posner & Rothbart, 2000).

In psychopathic individuals, these effects appear to primarily be associated with the behavioural or externalizing factor of psychopathy, as comprised by the Antisocial Behaviour and Erratic Lifestyle scales of the SRP-

III. Atypical attention to peripheral information, whether the result of over-focusing on central items (Raine & Venables, 1988) or diminished attention to non-central information (Newman, 1998), may lead to impulsive or reckless behaviour and lifestyle choices if this over-focusing should prevent the consideration of a full range of options, and the disregard of important sources of information that lay outside of immediate goals. To date, this is still a matter of conjecture, and has received limited empirical support. As well, the Erratic Lifestyle construct is associated with factors that underlie the antisocial behaviours and lifestyles associated with many externalizing disorders. Therefore, these results may not necessarily be related to psychopathy *per se*, but rather reflect attentional abnormalities associated with externalizing disorders such as antisocial personality disorder, which lack many of the other interpersonal and affective traits which characterize psychopathy.

In summary, this study involved the examination of ERP responses as elicited by target and nontarget stimuli during a visual oddball task in a large population of undergraduates with a range of psychopathic personality traits. There was little evidence that psychopathy was related to P3 amplitude, suggesting that atypical attention processes in this population are not reflected in the P3 under standard testing conditions. However, psychopathy was related to enhanced amplitudes of the P2, a mid-latency positivity, and the N5, a late negativity. Enhancement of the P2 and N5 cannot be assumed to constitute a deficit in attentional processing but the exact implications of these enhanced responses will require further study in the context of actual behavioural

anomalies. Of particular interest is the N5, as this component may be relatively more sensitive to psychopathy than other ERP components, and warrants further investigation.

CHAPTER SIX. GENERAL DISCUSSION

The primary focus in this thesis was to investigate attentional and affective processing in psychopaths using a variety of evoked potentials reflecting aspects of cognitive function such as error monitoring, response control, and attentional allocation – processes purported to be abnormal in psychopaths. However, despite the range of psychophysiological, behavioural, and neuroimaging data available, there is little consensus in the field regarding the cognitive or neurological underpinnings of the disorder. Nonetheless, researchers have attempted to develop models of the disorder on the basis of both clinical and experimental evidence. Cleckley (1941) initially proposed that psychopathy was a type of semantic dementia, whereas more recent models propose various levels of attentional and/or emotional dysfunction. However, according to Newman's response monitoring hypothesis (Newman, 1998), psychopathy is associated with a deficit in automatic, bottom-up information processing and attention allocation. Raine and Venebles (1988) argued that the disorder is associated with atypical, but not necessarily deficient, attentional processes, whereas neural models implicate a disorder in amygdala function (Blair et al., 2006) as well as dysfunction in broader paralimbic areas, particularly the ACC (Kiehl, 2006).

The main hypothesis underlying the work presented here was that psychopathy is not necessarily associated with any general cognitive deficits but rather with deficits in processing or evaluating emotionally relevant information. This hypothesis was based on distinctions in the ACC as described

by Bush et al. (2000). They have shown that the ACC is divisible into two regions. The dorsal portion is associated with primarily cognitive and motor functions and has connections with the caudal limbic system, including regions such as the hippocampus, posterior cingulate, and basal ganglia. Ventral regions of the ACC are part of the rostral regions of the limbic system and have reciprocal connections to the orbitofrontal cortex, amygdala, insula, and other limbic (Bush et al., 2000; Devinsky et al., 1995). It was hypothesized that it is dysfunction in the ventral (emotional) regions of the ACC that is specifically associated with psychopathy.

Error Monitoring and Emotional Processing

The primary purpose of the studies examining error monitoring was to investigate ACC function in those with psychopathic personality traits under circumstances requiring primarily cognitive or primarily emotional processing, which was intended to selectively activate either the dorsal or ventral ACC. The first round of data collection involved examining the performance of a group of incarcerated violent offenders with a range of scores on a clinical measure of psychopathy and a group of noncriminal, nonpsychopathic control participants. The second round of data collection extended the range to include normal, healthy undergraduates who varied on a measure of psychopathic personality traits.

Error monitoring was initially examined in a group of incarcerated offenders and controls. These participants completed the easy letter flanker and the face flanker task. It was expected that source modeling of the ERN generator during

the letter flanker task would show a dipole in the more dorsal (cognitive) regions of the ACC, but that the emotional face flanker task might draw more on the ventral ACC regions. Results suggested that the location of the ERN dipole did indeed vary by task: During the non-emotional letter flanker task both groups generated ERNs with sources in the vicinity of the dorsal ACC. This is consistent with other work suggesting that the ERN often has a source generator in the dorsal regions of the ACC (e.g. Kiehl, Liddle, & Hopfinger, 2000; van Veen & Carter, 2002a). The control group also showed the expected functional dissociation between dorsal and ventral ACC during the emotional task, generating a ventrally located source. This is consistent with other work suggesting that the ventral ACC with its connections to limbic regions (Devinsky et al., 1995) is associated with the processing of affective information (Bush et al., 2000). However, the psychopath group failed to show ERN generators anywhere near the ACC. Rather, there was evidence of activity in the insula and parahippocampal gyrus, although these unusual dipoles resulted in a model that did not achieve a satisfactory solution for the ERN data. The important finding here is not the location of the atypical dipoles, but rather the absence of source generators within the ACC – a finding that supports the presence of ACC abnormalities in psychopathy.

Also examined was a sample of university students who varied on a range of psychopathic traits. Participants were required to make judgements about both neutral and affective information. They were presented with the face- and letter-flanker tasks from the first study as well as a difficult flanker

task that was intended to better equate face and letter flanker tasks in terms of their difficulty. If psychopathy was associated with general paralimbic dysfunction, then one would expect to see performance deficits across all tasks because the ACC would be generally impaired and unable to monitor performance. If psychopathy were associated with general information-processing deficits, but not ACC deficits, then one would expect to see deficits on only the two more challenging tasks. If psychopathy were associated only with deficits in affective processing associated with rostral limbic dysfunction, then psychopathy should be associated with deficits on the emotional task, but not on the other two tasks. In general, the results of this study supported this latter alternative: psychopathy was unrelated to behavioural performance or electrocortical responses on either letter flanker task (irrespective of level of difficulty), but was associated with attenuated ERN amplitudes on the face-flanker task. The main findings from these studies do not offer unconditional support to any one of the current models of psychopathy but are compatible with the views of Raine and Venables (e.g. Raine & Venables, 1988), who claim that attentional processes may be atypical but not deficient in psychopathy. The data presented here are also compatible with the work of Blair and colleagues (e.g. Blair, 2003) that suggests a deficit primarily in the processing or responding to emotional information.

Error monitoring involves regions other than the ACC – the insula, regions of prefrontal cortex, and some areas of parietal cortex have all been implicated in this process (e.g. Mathalon, Whitfield, & Ford, 2003). These are

also part of a larger circuit known as the rostral limbic system (Devinsky et al., 1995), which is comprised of a number of regions including the amygdala, ACC, insula, and orbitofrontal cortex. These regions have also been associated with electrodermal responses to motivational and emotional stimuli (Critchley, Corfield, Chandler, Mathias, & Dolan, 2000; Frederikson et al., 1998) and sympathetic arousal (Critchley et al., 2003). The insula and ACC are also active when individuals experience empathy, and activity in these regions correlate with scores on a measure of empathy (Singer et al., 2004). Interestingly, these are all functions which have been identified as abnormal in psychopaths. Thus, it is possible that psychopathy is associated with dysfunction in this system. Dysfunction in this region could also potentially explain the smaller Pe amplitudes associated with psychopathy, as the Pe is also associated with ventral ACC activation (van Boxtel et al., 2005). One potential explanation for the activation of the parahippocampal gyrus during the face flanker task is that because the rostral limbic system was impaired, the more caudal region of the limbic system, which includes hippocampus, posterior cingulate, and certain parietal regions (Devinsky et al., 1995), was instead recruited. This is consistent with the results of behavioural studies, which have found that when psychopaths are given unlimited time, they show no deficits in the ability to recognize faces (Book et al., 2007). It is possible that when given sufficient time psychopaths are able to use top-down cognitive strategies or to recruit alternative cortical regions (e.g. Intrator et al., 1997) to recognize facial expressions and also raises

questions about whether emotion, even if identified, is appropriately integrated with cognition and action.

Response Inhibition

There have been a number of reports of reduced inhibitory control in psychopaths, particularly during the withholding of a prepotent response (e.g. Kiehl, Smith et al., 2000; LaPierre et al., 1995). Psychopathy is also associated with behavioural impulsivity (Dikman & Allen, 2000; LaPierre et al., 1995). To investigate response inhibition in psychopathy, the frontal N2, an ERP component thought to have a source in the region of the ACC (Bekker, Bocker, Van Hunsel, van den Berg, & Kenemans, 2005), and the NoGo P3, which is thought to represent response inhibition processes likely initiated in both orbital frontal cortex and ACC (Bokura et al., 2001; Goldstein & Volkow, 2002), were examined. Results indicated that that there was no relation between psychopathy and any aspect of behavioural or electrophysiological performance on this task, results which do not support the presence of the general response-inhibition deficit suggested by some authors (e.g. Howland et al., 1993). However, attenuated NoGo P3 amplitudes were seen at frontal sites. Kiehl et al. (2000) reported a similarly reduced NoGo P3 in a sample of violent offenders, but in the current sample the amplitude of the frontal P3 was not correlated with psychopathy and so this may reflect issues associated more generally with a prison population.

The lack of a relation between psychopathy and poor inhibitory control was further supported when the offender group was divided on the basis of

PCL-R score. This resulted in two groups of offenders. Those who were higher in psychopathy had N2s that appeared more robust than those produced by controls. It was the low-scoring offenders who generated particularly shallow and poorly-defined N2s. While this suggests that psychopathy may not be associated with response inhibition deficits at a neurological level, it is consistent with data that indicates that non-psychopathic offenders, especially those with a greater susceptibility to externalizing disorders such as antisocial personality disorder, may show abnormal neurological activity during response inhibition (Dolan & Park, 2002; Horn, Dolan, Elliot, Deakin, & Woodruff, 2003). These data also raise questions about how well the impulsivity associated with psychopathy is captured in laboratory tests of response inhibition (Dolan & Fullam, 2004). The impulsivity commonly associated with psychopathy refers to the inability to resist impulses or unpremeditated conduct lacking in forethought or reflection, however, it may not reflect aberrant inhibitory control at the level of motor response tendencies.

Attention Allocation and Information Processing

Information-processing models, such as the response monitoring hypothesis (Newman, 1998) suggest that psychopathy is essentially a disorder of attention, and that psychopaths display a failure to monitor or react to response-based feedback. According to this perspective, many core features of psychopathy (i.e., failure to acquire classically conditioned responses, deficits in processing affective information, relative insensitivity to peripheral task contingencies) may reflect a reduced efficiency in selective attention rather than

affective deficits. According to this model, psychopaths would be expected to have error monitoring deficits (behavioural and/or electrocortical) on all flanker tasks, regardless of whether the task dealt with affective information or not. However, the letter-flanker tasks in the current data sets provided little evidence that psychopathy was associated with disrupted attention. The only effect associated with psychopathy from the letter-flanker response monitoring tasks was an attenuated Pe. If the Pe can be taken to reflect the conscious aspects of error monitoring, as proposed by (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000), then these data would suggest that psychopaths may attend less to endogenous feedback cues signaling error commission than do their nonpsychopathic counterparts.

Another commonly-examined electrophysiological index of attention is the P3, a positive-going component associated with the allocation of attention (e.g. Donchin, 1981; Kok, 1990; Polich & Kok, 1995; Sutton et al., 1965). Despite strong relations between attenuated P3 amplitudes and externalizing disorders such as antisocial personality disorder (e.g. Bauer et al., 1994; Iacono et al., 2002; Iacono & McGue, 2006; Patrick et al., 2006), it is unclear whether this relation generalizes to psychopathy. Much of the work in this area has produced conflicting results, with reports of both enhanced (e.g. Raine & Venables, 1988) and attenuated (Kiehl et al., 1999) P3 amplitudes. This issue was examined here in a population of undergraduate males who varied on a measure of psychopathic personality traits using a visual oddball task. Despite a large sample size and a wide range of scores on the SRP-III, there was

absolutely no evidence of an association between psychopathy and task performance or attentional allocation. That is to say, on this task there was no association between psychopathy and attentional deficits as indexed by P3 amplitude.

However, higher scores on psychopathy were associated with larger P2 and N5 responses to target relative to nontarget stimuli. The larger N5s are consistent with other reports (Kiehl, Bates et al., 2006; Kiehl et al., 1999), and although the neural correlates of this component are not well defined, it may represent a form of semantic integration, not unlike the N4 (Kutas & Federmeier, 2000; Kutas & Hillyard, 1980). What is important to note, however, is that there is little evidence to suggest that enhanced N5 amplitudes are a sign of impairment rather than enhancement of focused attentional capacity. Likewise, larger P2 amplitudes do not necessarily reflect attentional deficits: the P2 has been associated with basic information processing functions such as stimulus classification (e.g. Crowley & Colrain, 2004) and, like the N5, may be associated with the parsing of an ongoing stream of information.

These data do not support the hypothesis that psychopathy is associated with a general information-processing deficit but are more consistent with Raine's over-focusing model of psychopathy. Raine and colleagues (1988) do not interpret traits such as decreased sensitivity to peripheral information as reflective of attentional deficits. Instead, they suggest that these may actually represent enhanced attention to task-relevant information. Based on the results of previous oddball tasks (e.g. Raine & Venables, 1987, 1988), this model

would predict that larger P3 amplitudes should be associated with psychopathy. Although, this effect was not noted in the current study, larger P2s and N5s were correlated with the disorder. A better understanding of the meaning of these components will hopefully provide greater insight into the nature of attentional differences associated with psychopathy.

Psychopathy as a Continuum

All of the data presented here are consistent with the view that psychopathic personality traits may be normally distributed throughout the population and do not represent a latent taxometric class. Support for this position comes from the significant correlations between psychopathy and ERP indices. For example, the results of the study reported in chapter four, in which ERN amplitudes were examined during emotional and nonemotional tasks in the undergraduate sample, showed that psychopathy was correlated with ERP indices of error monitoring – those with higher psychopathy scores also had attenuated ERN and Pe amplitudes. Likewise, the results from the oddball task reported in chapter five, which examined attention orientation in the undergraduate sample, indicated that psychopathy was positively correlated with P2 and N5 amplitudes. The data from these data sets are consistent with a number of other reports that psychopathic personality traits may vary in the normal population (e.g. Edens et al., 2006; Marcus et al., 2004; Walters, Duncan et al., 2007). This suggests that psychopaths are not qualitatively different from non-psychopaths, but rather display these personality types to a greater degree than the majority of the population. This has significant implications for the

study of psychopathy and for the way this condition is dealt with in the legal system: most treatment choices and legal decisions are based on the assumption that psychopaths are a discreet class of individuals. This also suggests that approaches towards the study of psychopathy may need to be altered, supporting the validity of examining this disorder in nonclinical samples as well as institutionalized individuals (Lilienfeld, 1998).

Limitations

There are a number of limitations that should be considered when interpreting the results of this investigation. Of concern is the small size of the offender sample. There were 12 control participants and 11 offenders, only seven of whom met the customary cut-off for psychopathy. The small sample does have some potential implications for generalizability, but the effects seen in these data sets were strong. For example, our sample size was sufficient to demonstrate the interaction between group and task on the face-flanker task. It may be that, in a larger sample, an association between psychopathy and error monitoring on the letter-flanker task would be evident as well, although relevant effect sizes in this sample suggest such an outcome would be unlikely. The second sample, consisting of university students, rectified the sample size issue, as 67 male undergraduates were recruited. However, this increased sample size came at the cost of the full range of psychopathy. Because this was a community sample, it did not represent the same degree of psychopathy at the higher ends of the scale found in the incarcerated population.

A second area which requires further examination is the dipole model described in chapter two. The shallowness of the face-flanker ERN in the psychopathic offender group resulted in unstable dipole solutions, which will require replication before any strong conclusions can be drawn from these data. Nonetheless, despite the relatively small sample size and other limitations, the results are at least consistent with the hypothesis that psychopathy is associated with functional ACC abnormalities during emotional processing, and that this dysfunction may be specifically associated with ventral ACC and perhaps the rostral limbic system. This also underlines the importance of adjusting standard error monitoring paradigms to more specifically test the questions of interest, especially for special populations. Lastly, because of technical problems during data collection, source analyses could not be carried out on any of the data from the undergraduate sample. Thus, the conclusions from the investigations in the undergraduate sample cannot be confirmed with dipole analysis.

Conclusion

The purpose of this investigation was to examine aspects of attentional processing in psychopaths through evoked potentials reflecting various aspects of cognitive function. The tasks were designed to allow for the evaluation of both information-processing and limbic dysfunction models of psychopathy. A secondary issue was the examination of the "continuity" theories of psychopathy, which suggest it is a normally-distributed personality trait. Taken together, results from the four data sets do not support theories suggesting a general impairment in the ability of psychopaths to allocate or control attention.

Indeed, both violent offenders and high-scoring undergraduate males showed little evidence of deficits in error monitoring, attention allocation, or response inhibition under non-affective testing conditions, although results were suggestive of some level of atypical function across all tasks. The greatest abnormality occurred in the processing of emotional information - a finding consistent with theories that propose focal functional abnormalities in the limbic system, and which the current data suggests may occur specifically the ventral ACC.

Future directions for this line of research include the development of a cognitively-based model of neurological function in psychopathic personality disorder. This work has suggested that the ventral ACC may be particularly associated with psychopathy, especially under testing conditions which require activation of the rest of the limbic system, although this region appears to function normally when testing conditions require activation of connecting cortical, but not subcortical, regions. However, to fully test this model requires expanding the scope of this research beyond electrophysiological and into functional imaging techniques. There is a relative dearth of well-conducted functional imaging research in this population. However, that which has been conducted generally confirms the hypothesis that this condition is associated with abnormal limbic activation in the presence of affective stimuli (e.g. Müller et al., 2003). Some studies have also reported increased activation in cortical regions (e.g. Intrator et al., 1997; Kiehl, Smith et al., 2001). One hypothesis to explain this atypical cortical activation is that, because some regions of the

limbic system are dysfunctional, psychopaths recruit alternate brain regions instead – perhaps using a top-down information processing strategy. This interpretation is supported by behavioural studies: when psychopaths are given plenty of time to identify emotional facial expressions, they do not show any of the deficits seen during speeded response tasks (Book et al., 2007). One approach to examine this issue is through the use of backward masking paradigms that elicit bottom-up limbic activation. This issue can also be addressed electrophysiologically by examining early components such as the N170, which occur prior to conscious processing.

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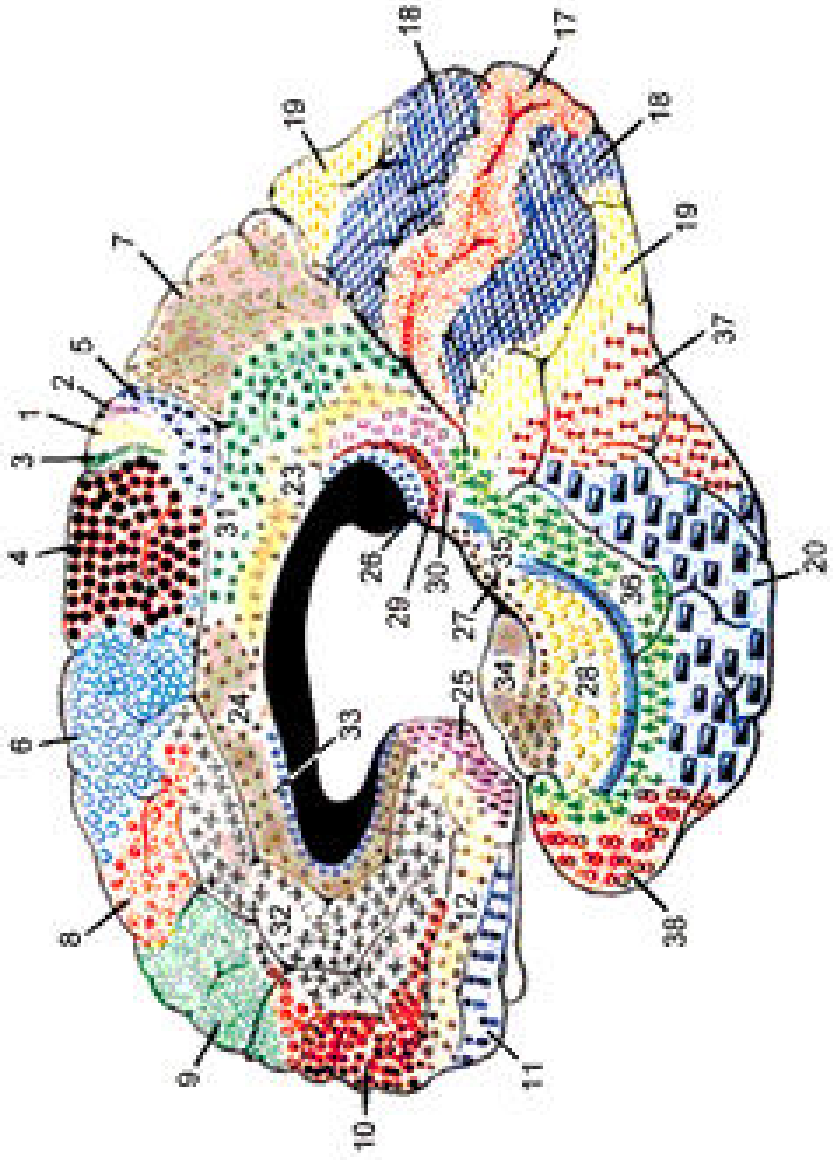

Figure 1.1. Map depicting Brodmann areas (Brodmann, 1909).

Figure 2.1. Sample stimuli for the face flanker task.

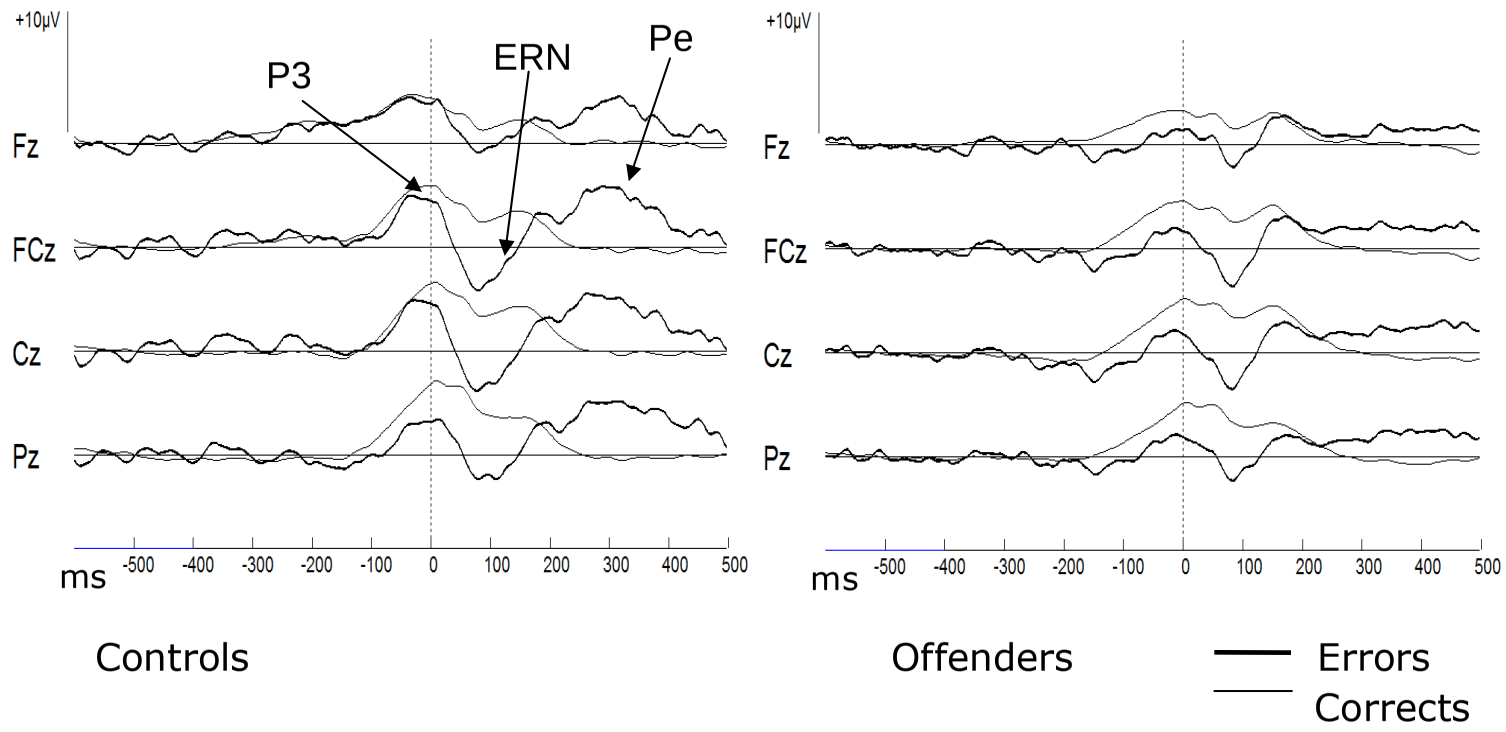

Figure 2.2. Overlaid ERP waveforms for correct and incorrect trials for control and offender groups during the letter flanker task (Study 1).

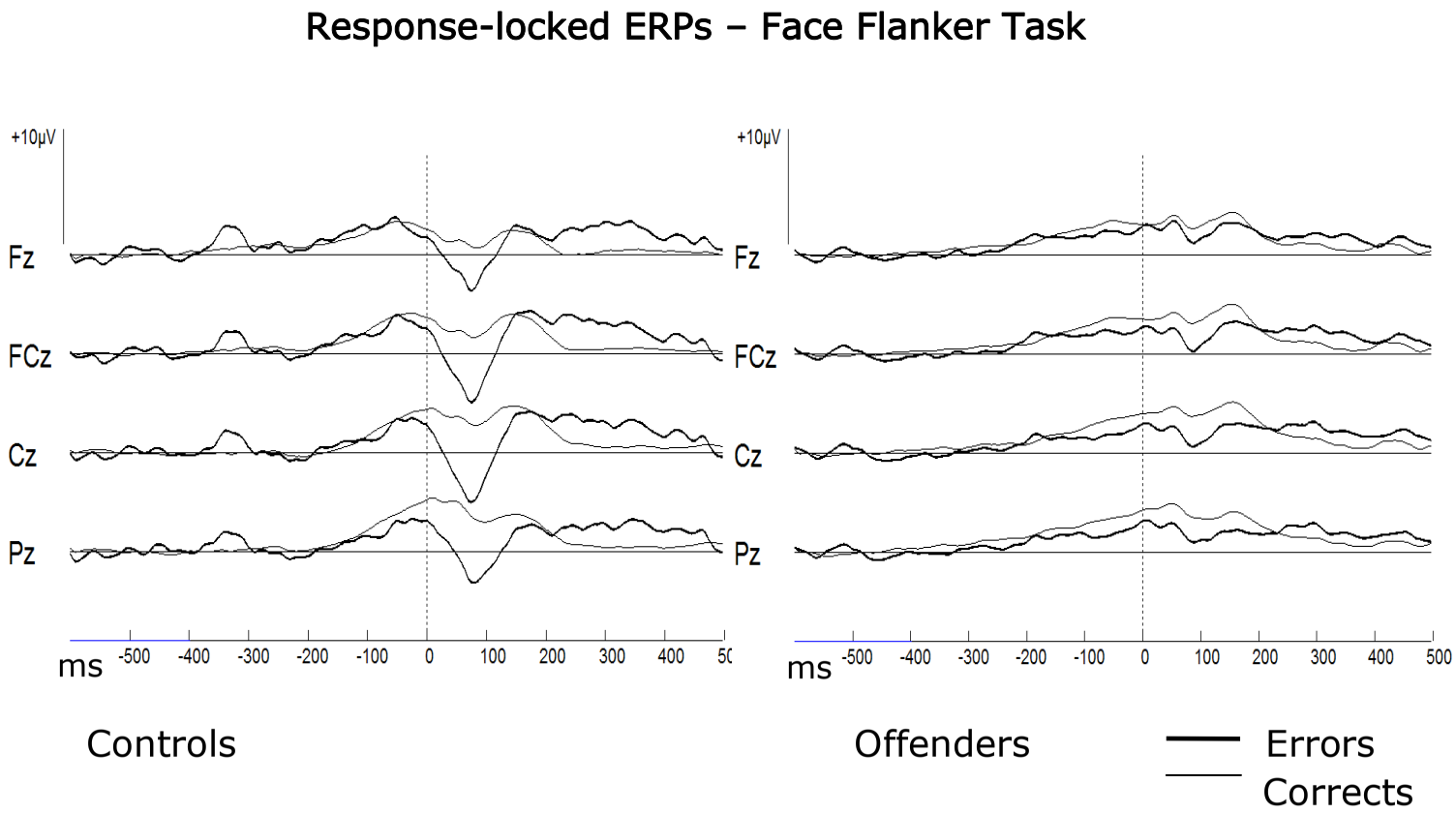

Figure 2.3. Overlaid ERP waveforms for correct and incorrect trials for control and offender groups during the face flanker task (Study 1).

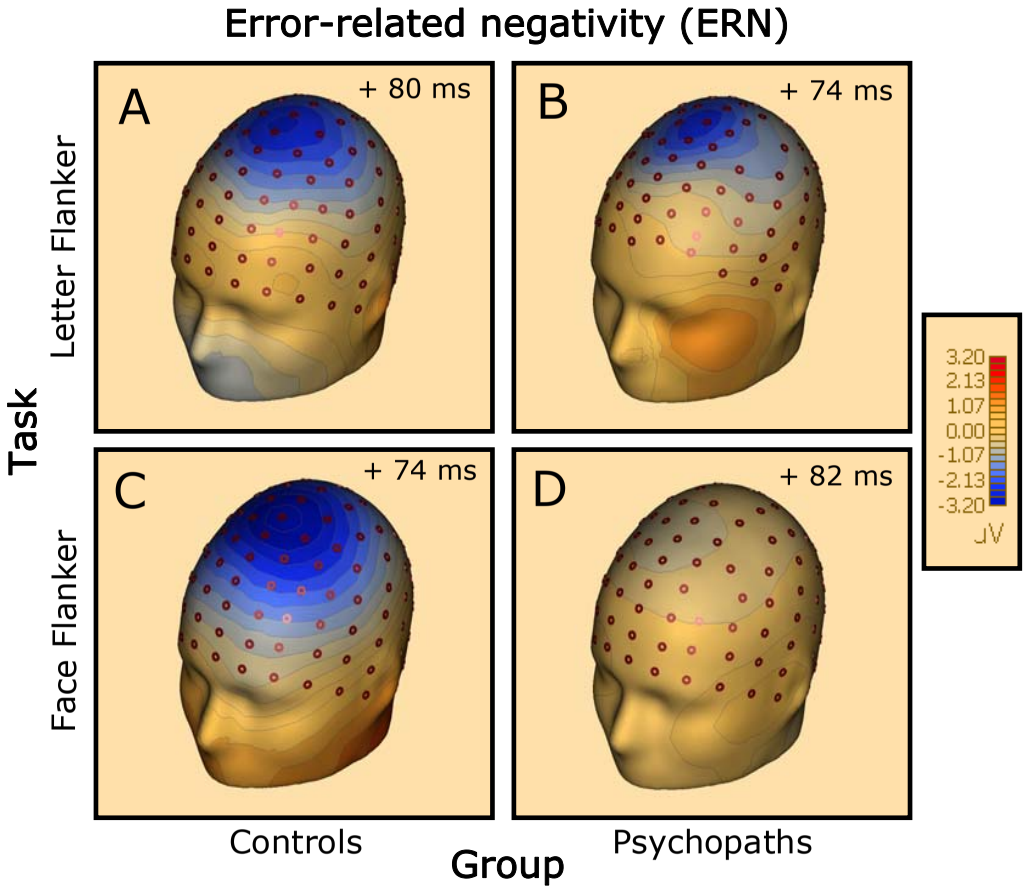

Figure 2.4. Voltage scalp distribution maps indicating scalp topography of the ERN as elicited by letter flanker and face flanker errors (Study 1).

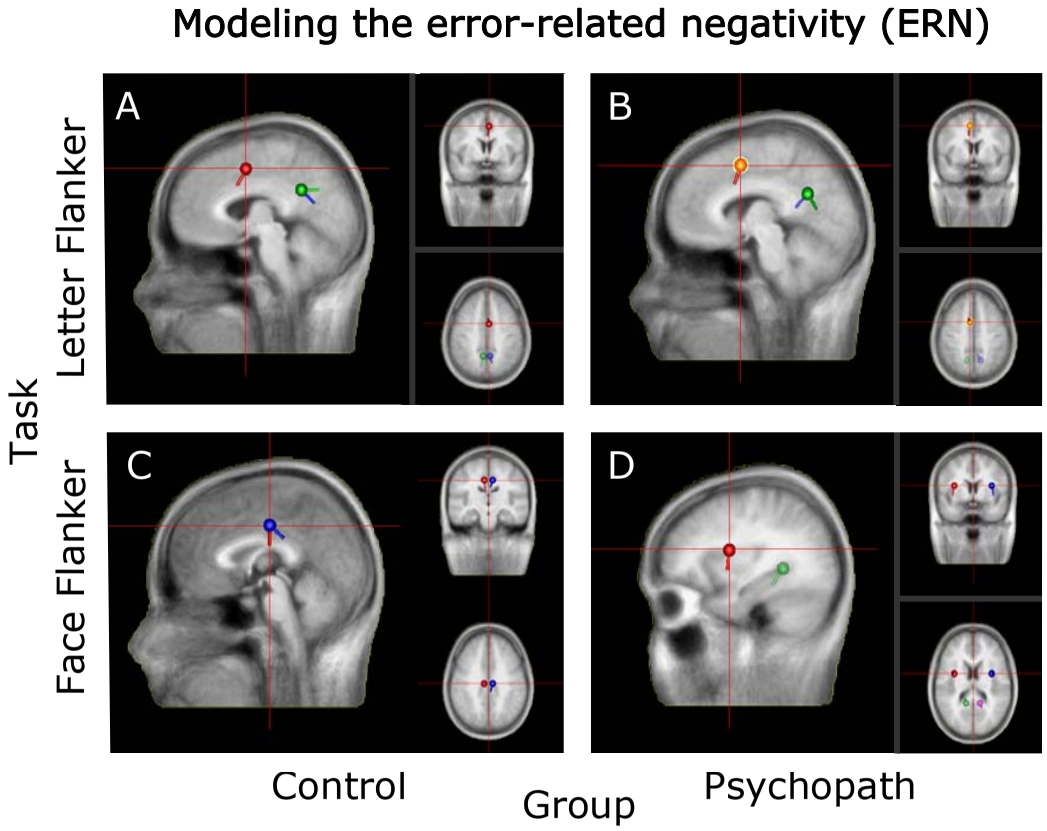

Figure 2.5. Sagittal, transversal, and coronal views of major dipoles superimposed on MRI-based head models indicating dipole source solutions for the ERN as elicited by letter flanker errors and face flanker errors for the control and psychopath groups (Study 1).

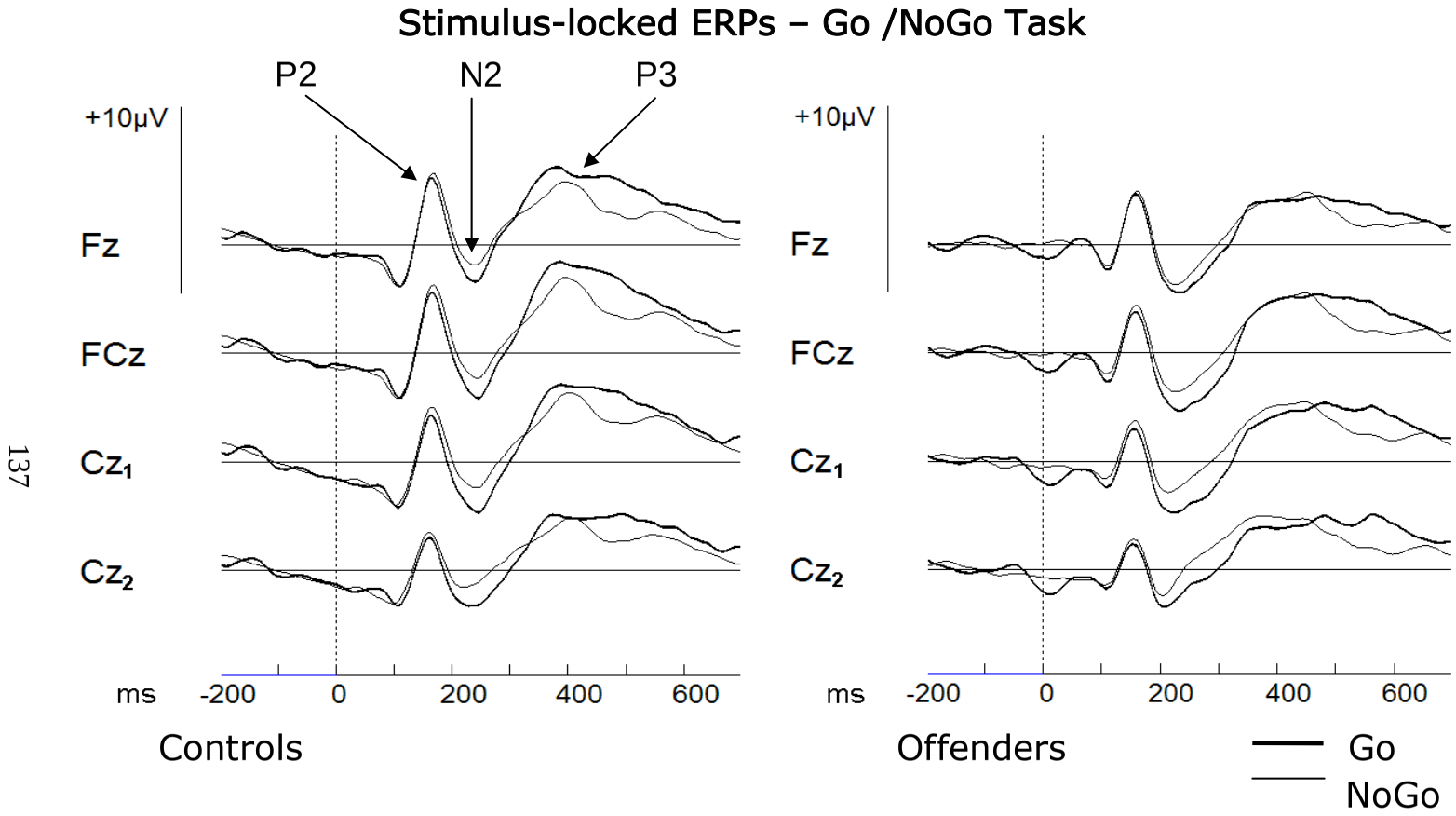

Figure 3.1. Overlaid stimulus-locked ERP waveforms for correct go and no-go trials for control and offender groups during the XY task (Study 2).

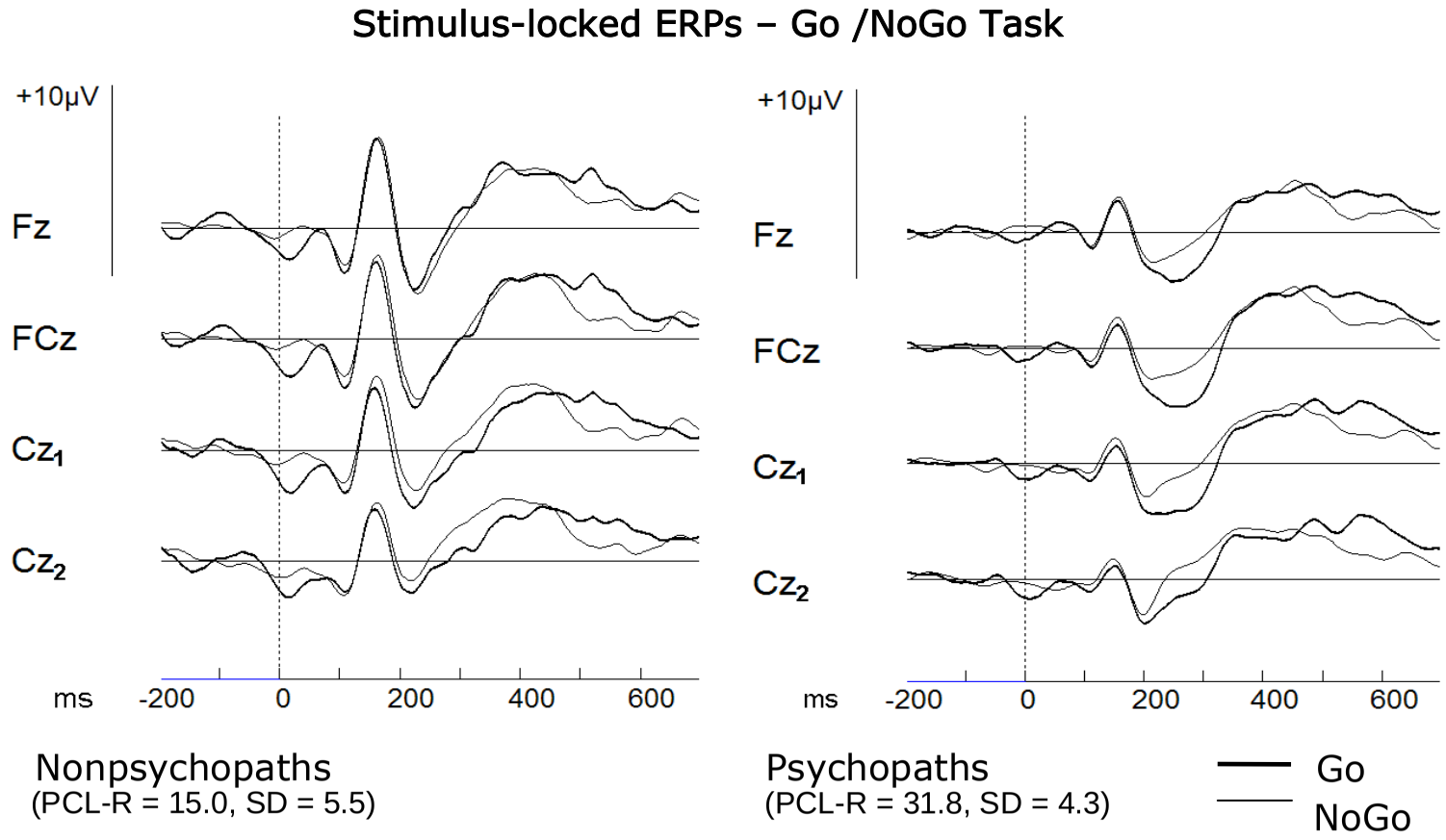

Figure 3.2. Overlaid stimulus-locked ERP waveforms for correct go and no-go trials for psychopath and nonpsychopath offender subgroups, during the XY task (Study 2).

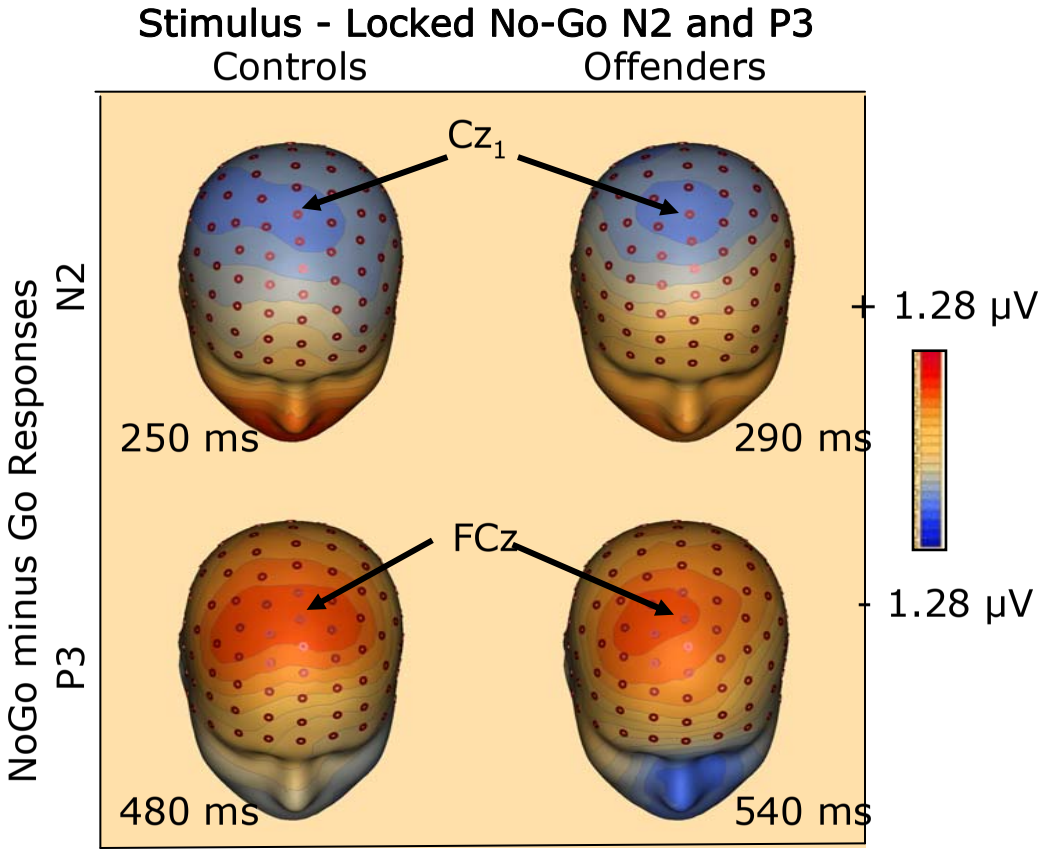

Figure 3.3. Topographical maps indicating greater N2 negativity for NoGo relative to Go trials and greater P3 positivity for NoGo relative to Go trials for control participants and offenders (Study 2).

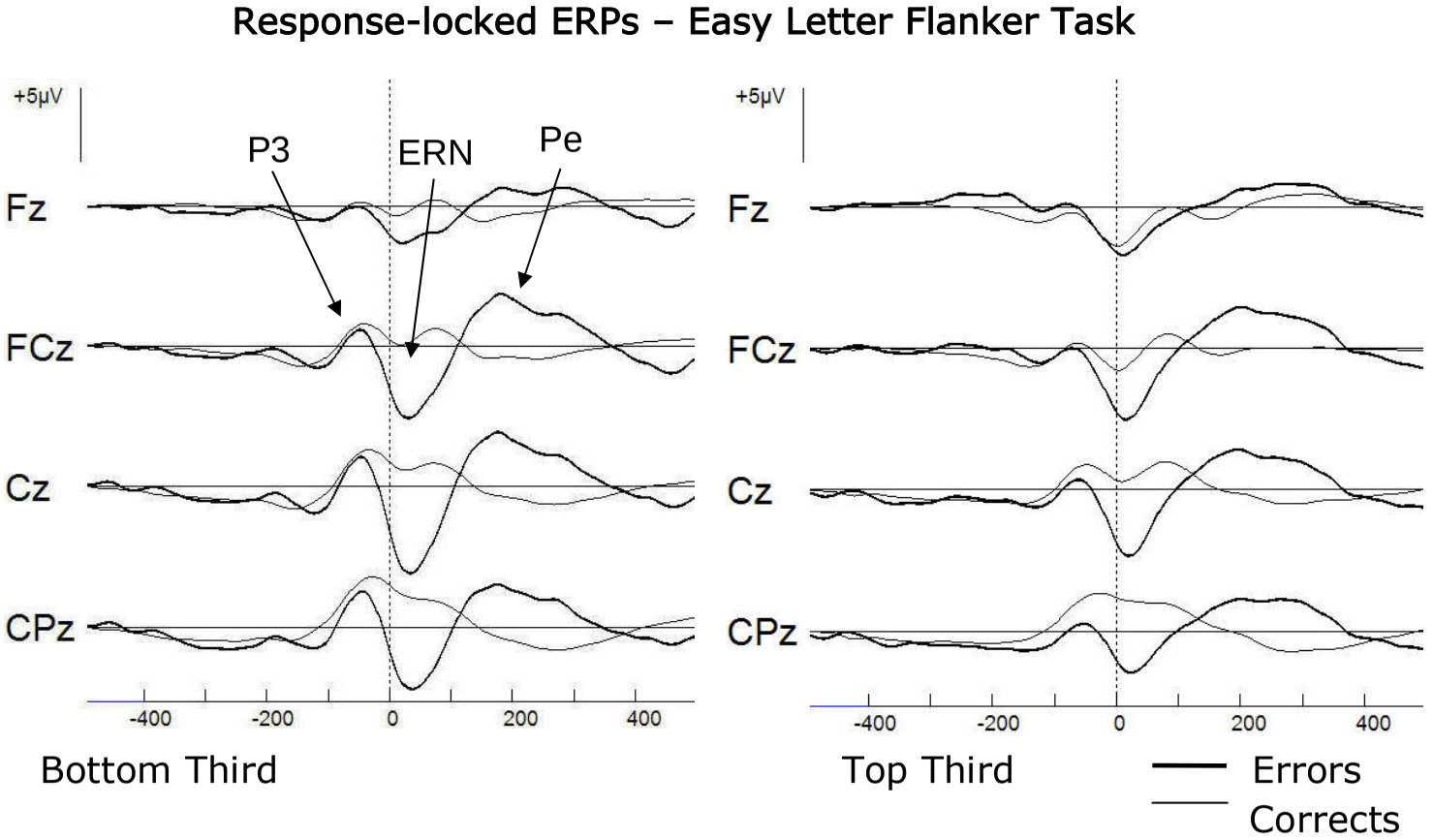

Figure 4.1. Overlaid ERP waveforms for correct and error trials on the easy letter flanker task for the top and bottom thirds for SRP-III Total Psychopathy scores (Study 3).

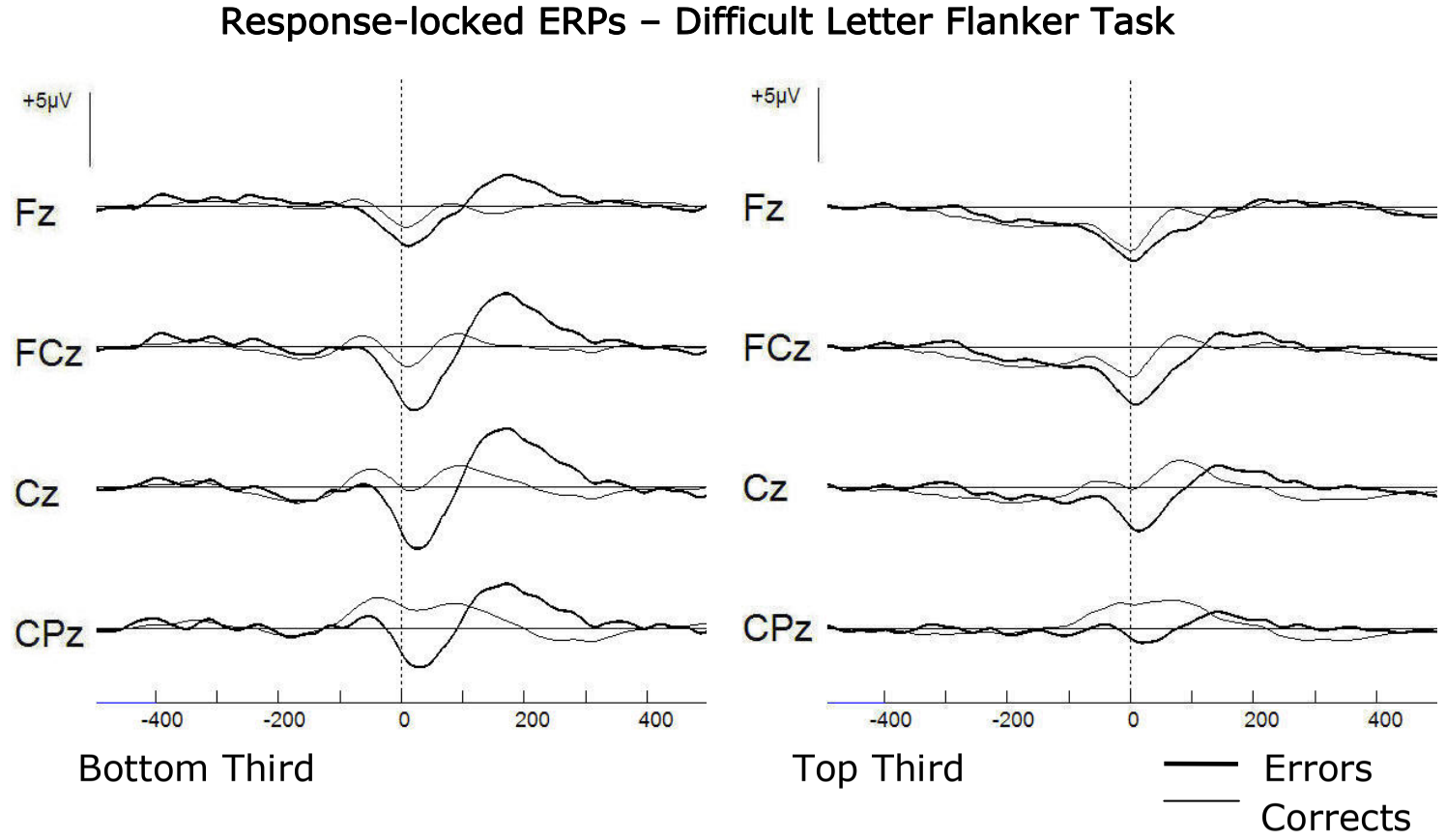

Figure 4.2. Overlaid ERP waveforms for correct and error trials on the difficult letter flanker task for the top and bottom thirds for SRP-III Total Psychopathy scores (Study 3).

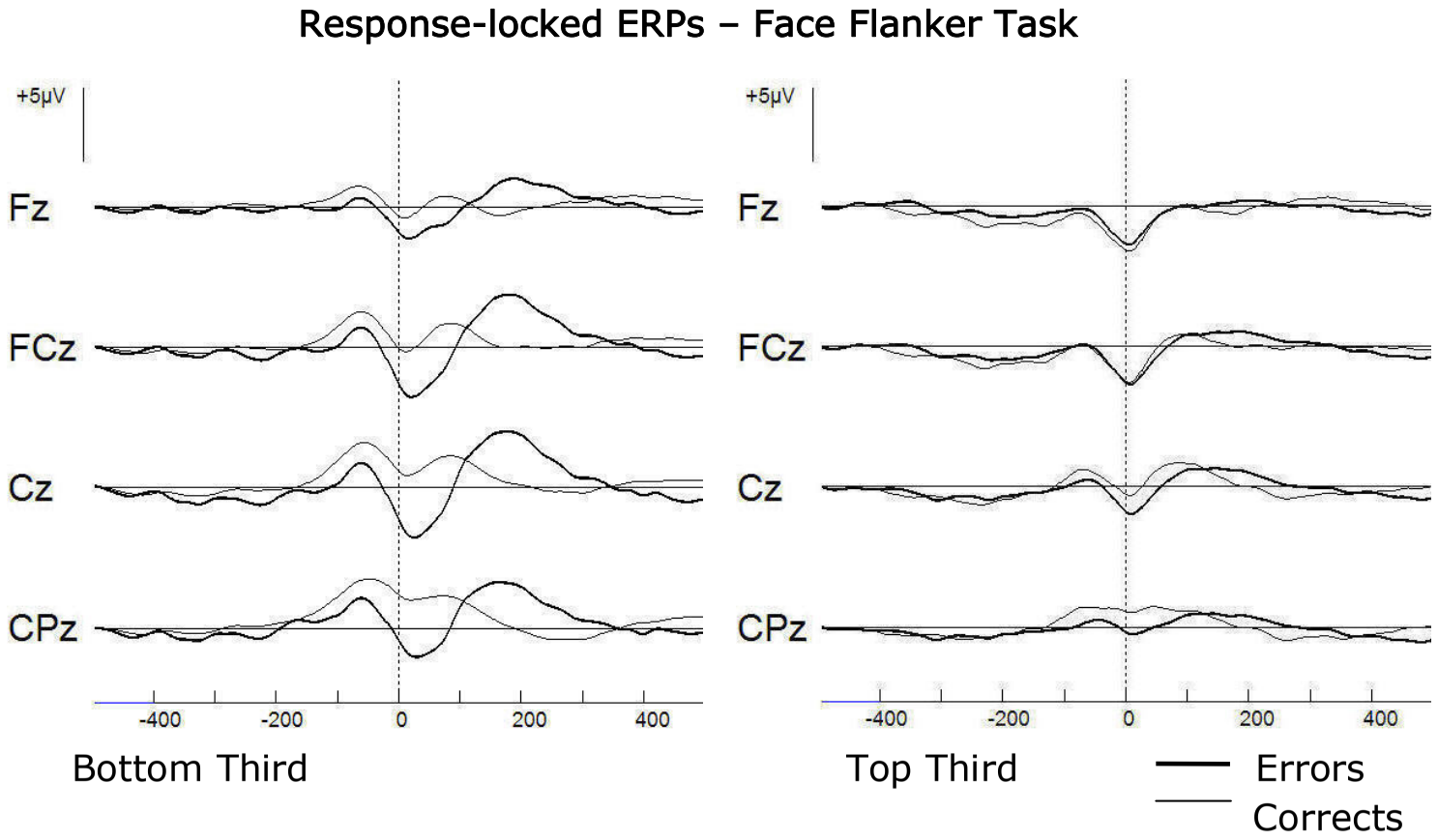

Figure 4.3. Overlaid ERP waveforms for correct and error trials on the face flanker task for the top and bottom thirds for SRP-III Total Psychopathy scores.

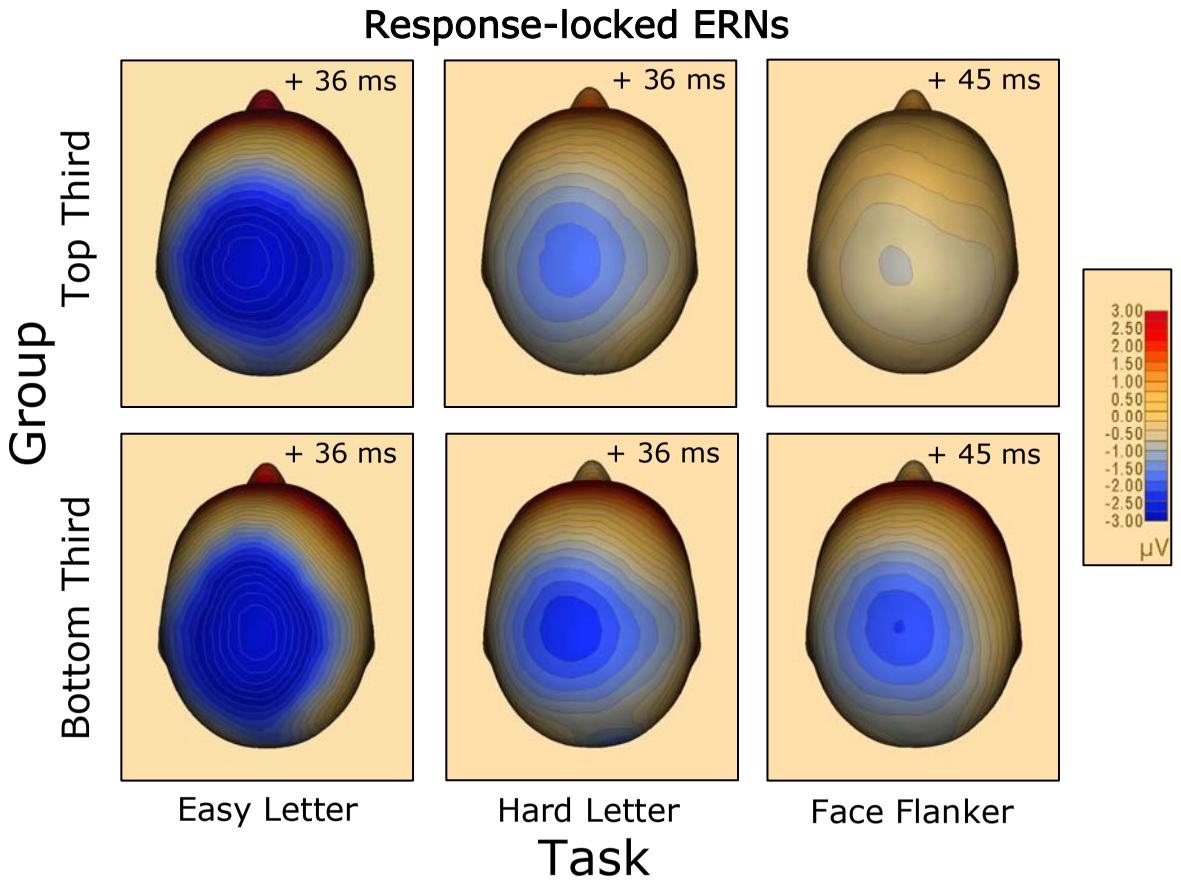

Figure 4.4. Topographical maps indicating ERN amplitude on the easy letter, hard letter, and face flanker tasks for the top and bottom thirds for SRP-III psychopathy scores

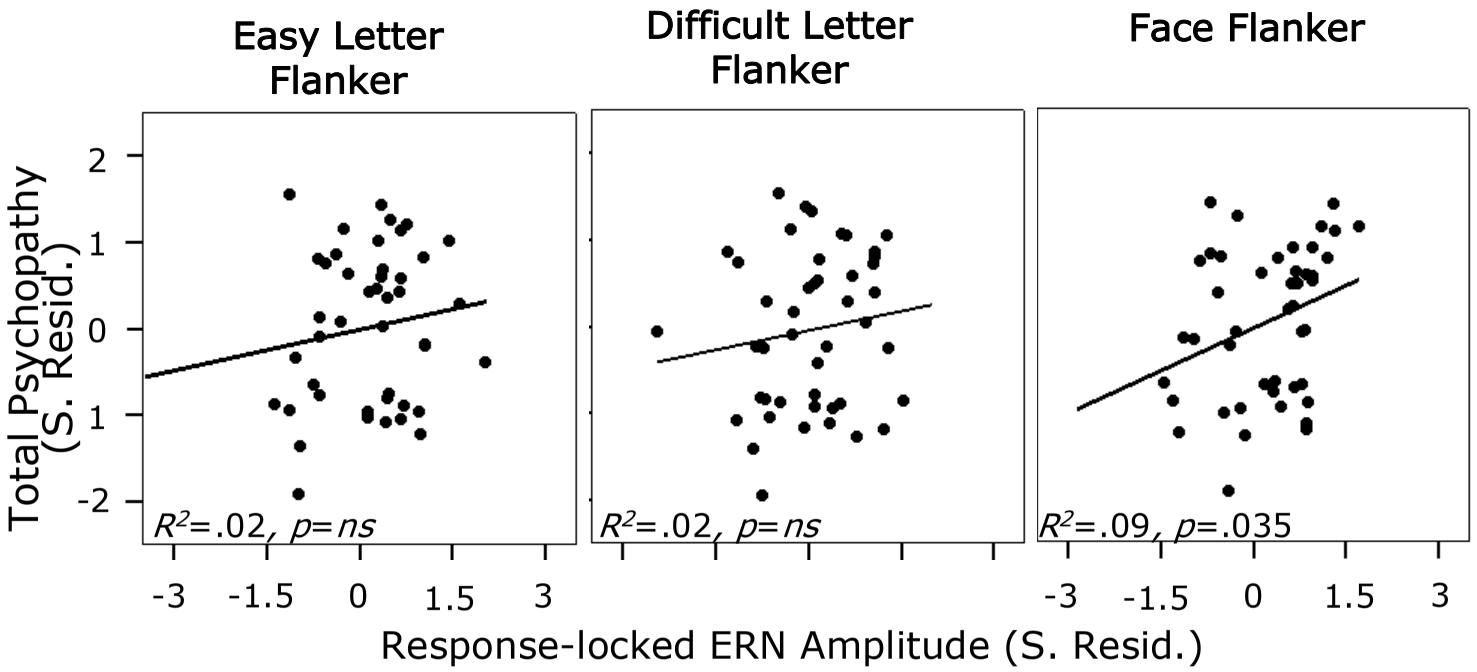

Figure 4.5. Scatter plots indicating the relationship between standardized SRP-III Total Psychopathy scores for the standardized residual (S. Resid) of responses-locked ERN amplitudes at FCz, adjusted for correct trials, across all three tasks (Study 3).

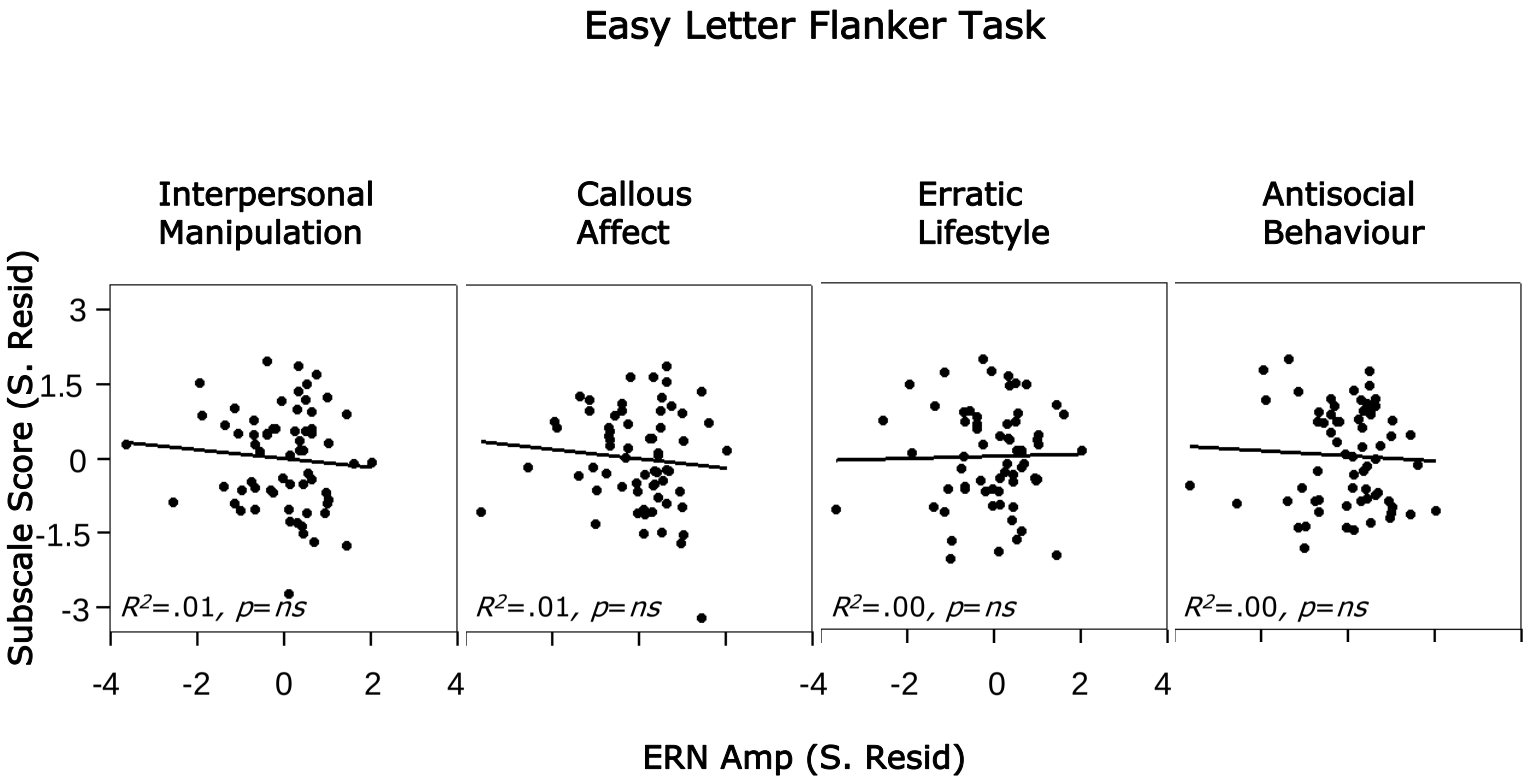

Figure 4.6. Scatter plots indicating the relationship between SRP-III subscale scores for the standardized residual (S. Resid) of response-locked ERN amplitude at FCz on error trials, adjusting for correct trials, on the easy letter flanker task (Study 3).

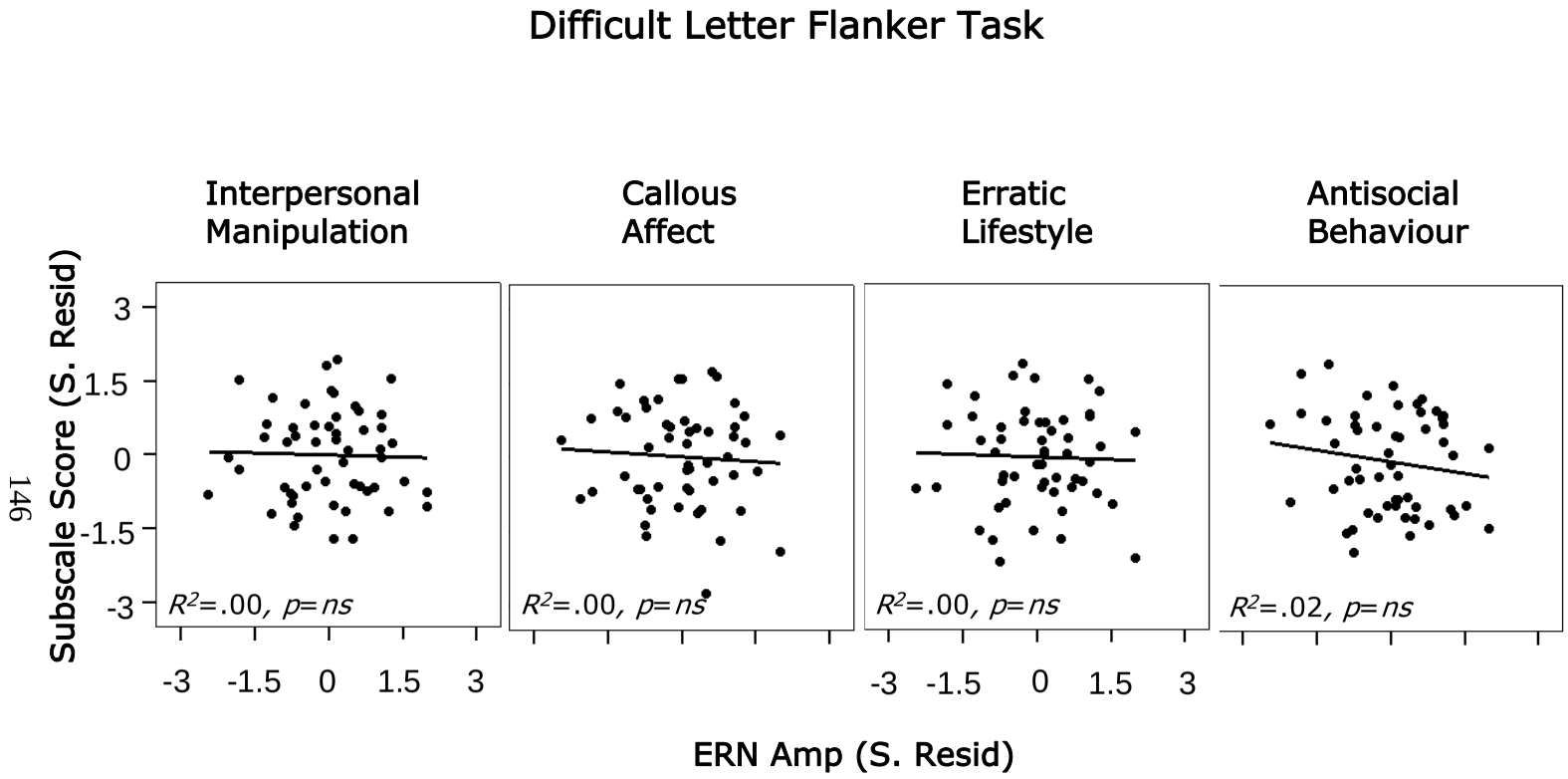

Figure 4.7. Scatter plots indicating the relationship between SRP-III subscale scores for the standardized residual (S. Resid) of response-locked ERN amplitude at FCz on error trials, adjusting for correct trials, on the difficult letter flanker task (Study 3).

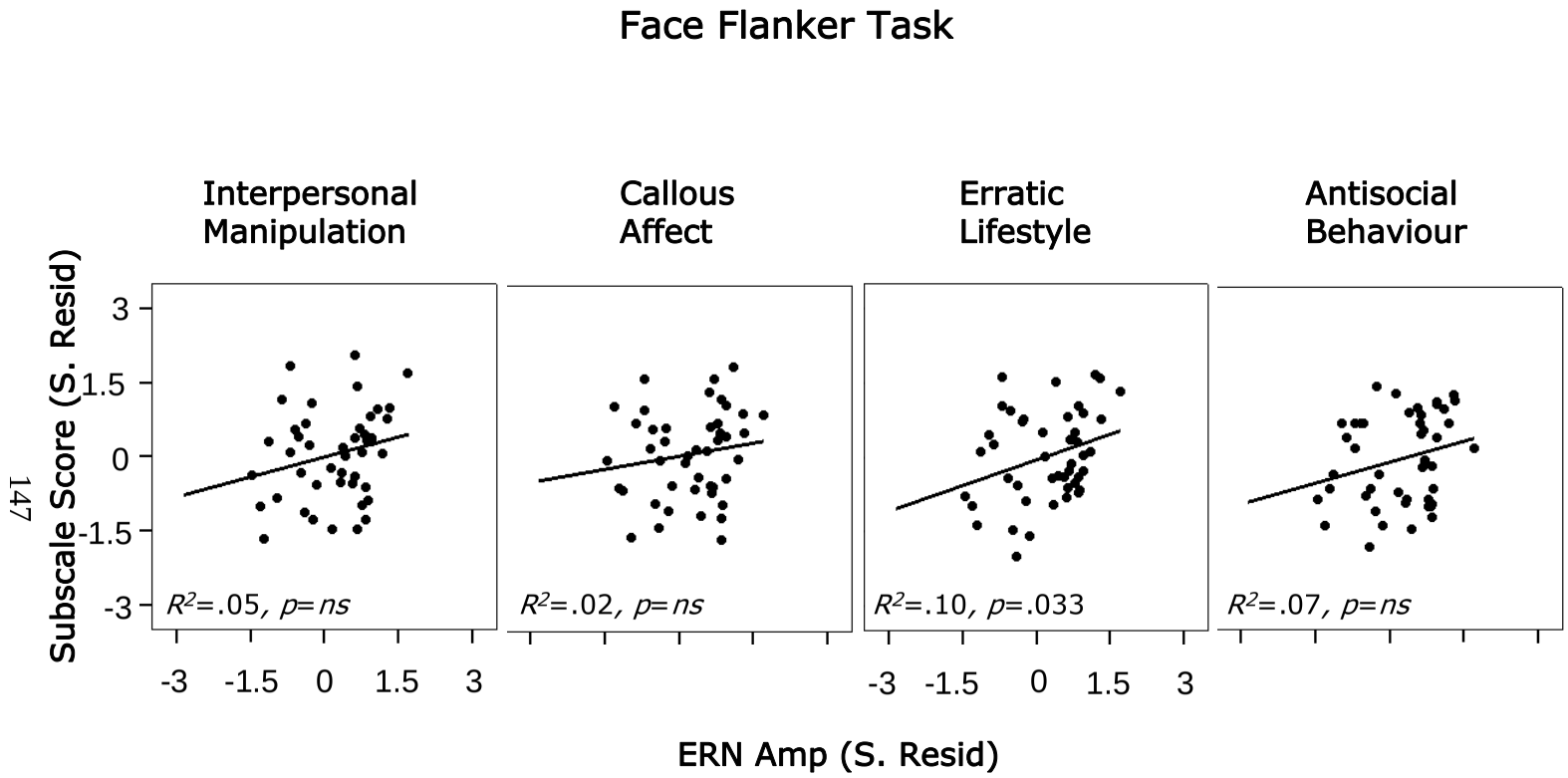

Figure 4.8. Scatter plots indicating the relationship between SRP-III subscale scores for the standardized residual (S. Resid) of response-locked ERN amplitude at FCz on error trials, adjusting for correct trials, on the face flanker task (Study 3).

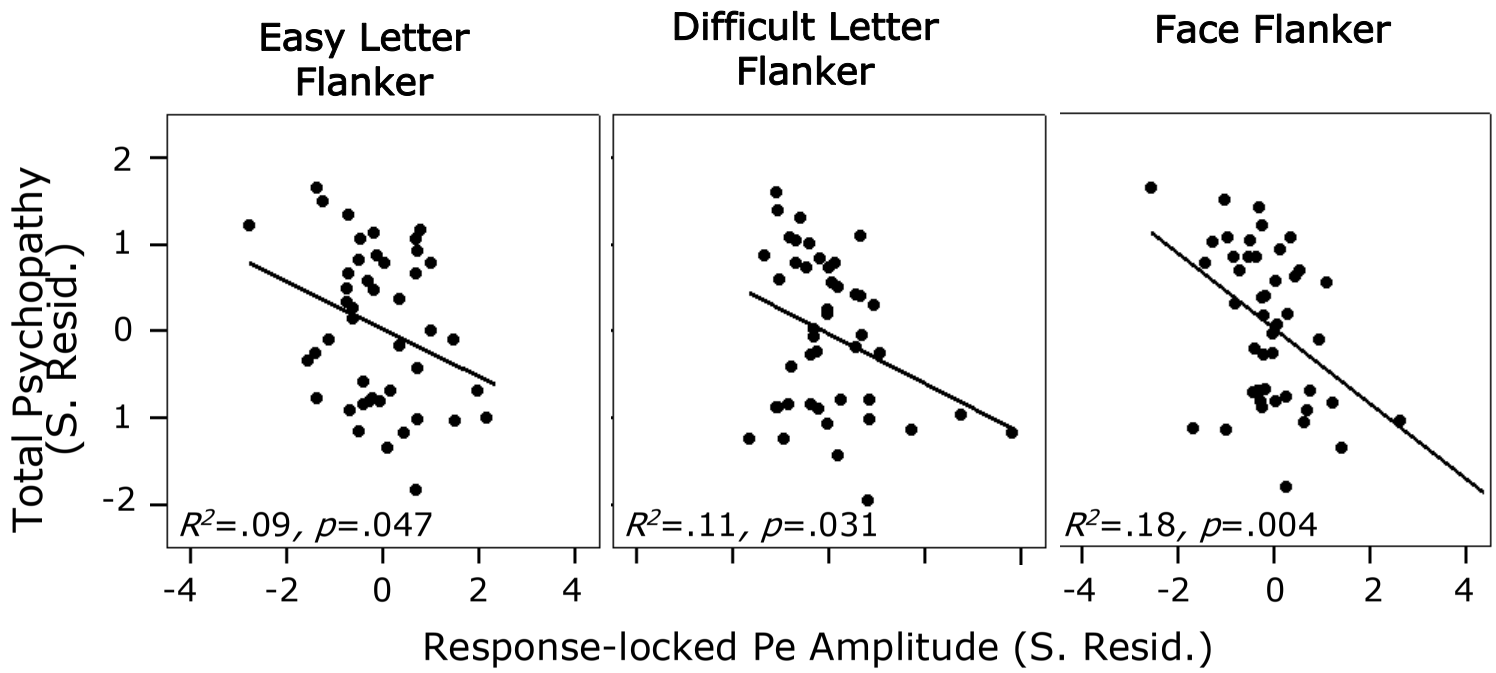

Figure 4.9. Scatter plots indicating the relationship between standardized SRP-III Total Psychopathy scores for the standardized residual (S. Resid) of Pe amplitudes at Cz, both adjusted for correct trials, across all three tasks (Study 3).

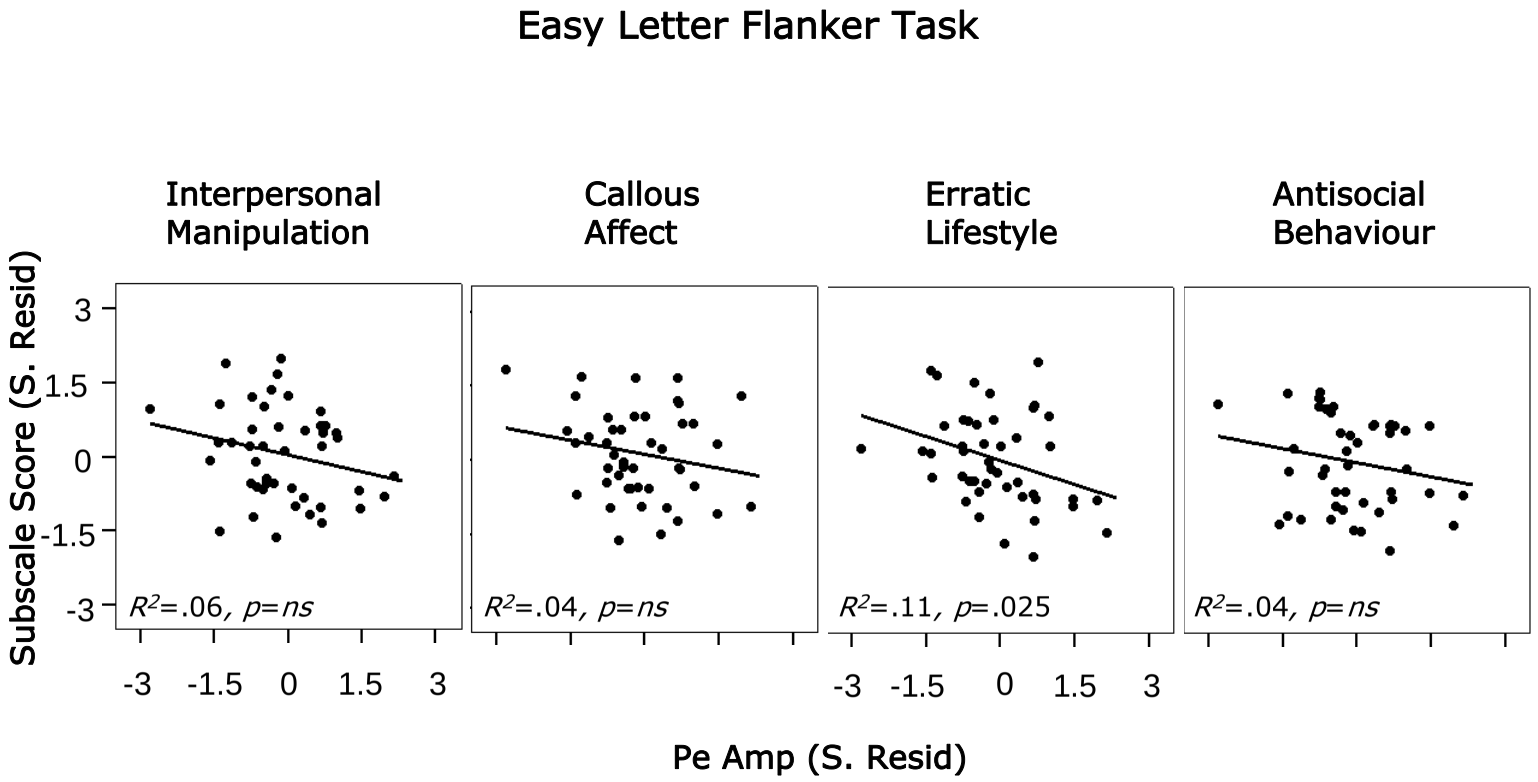

Figure 4.10. Scatter plots indicating the relationship between SRP-III subscale scores for the standardized residual (S. Resid) of response-locked Pe amplitude at Cz on error trials, adjusting for correct trials, on the easy letter flanker task (Study 3).

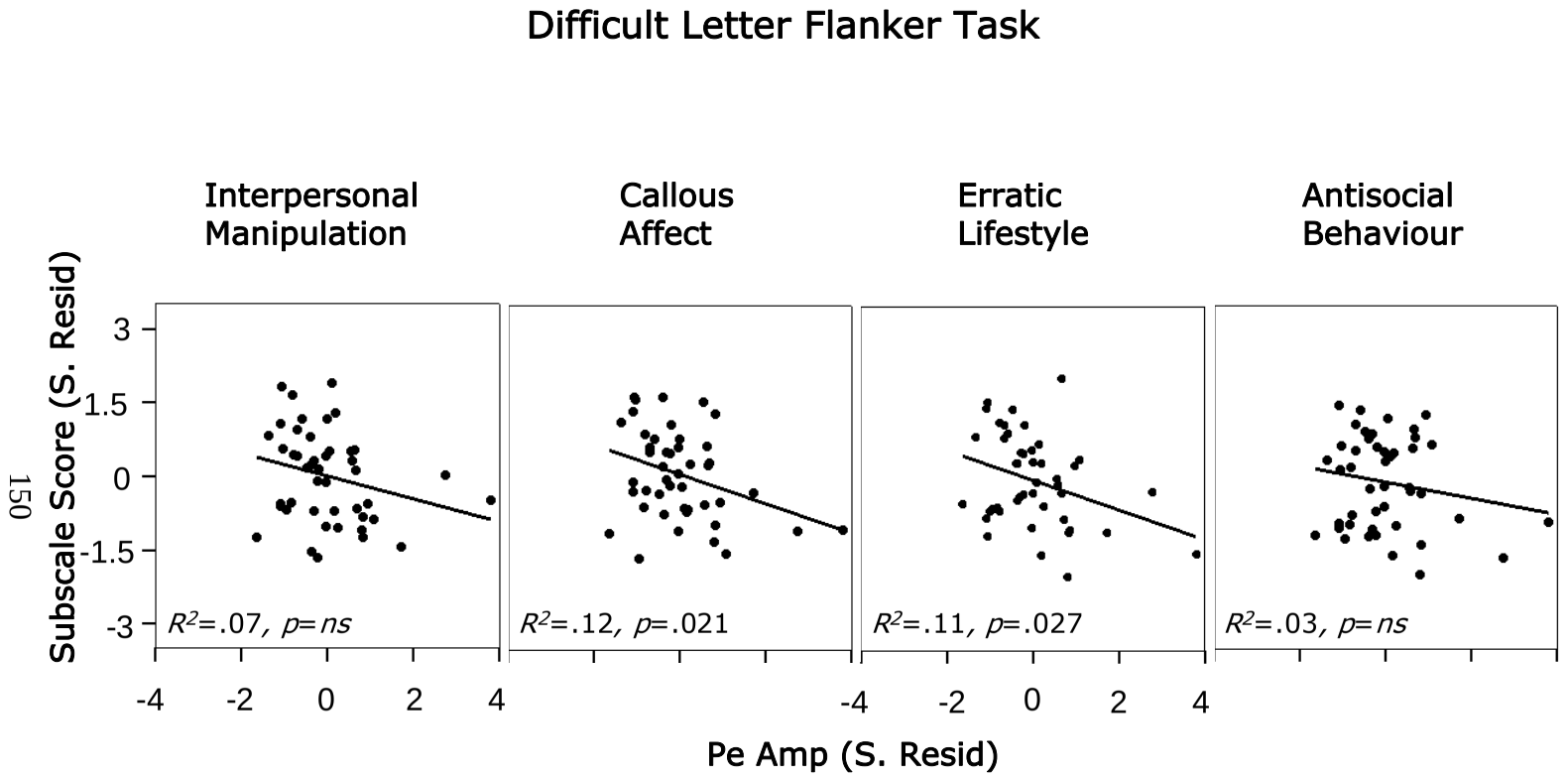

Figure 4.11. Scatter plots indicating the relationship between SRP-III subscale scores for the standardized residual (S. Resid) of response-locked Pe amplitude at Cz on error trials, adjusting for correct trials, on the difficult letter flanker task (Study 3).

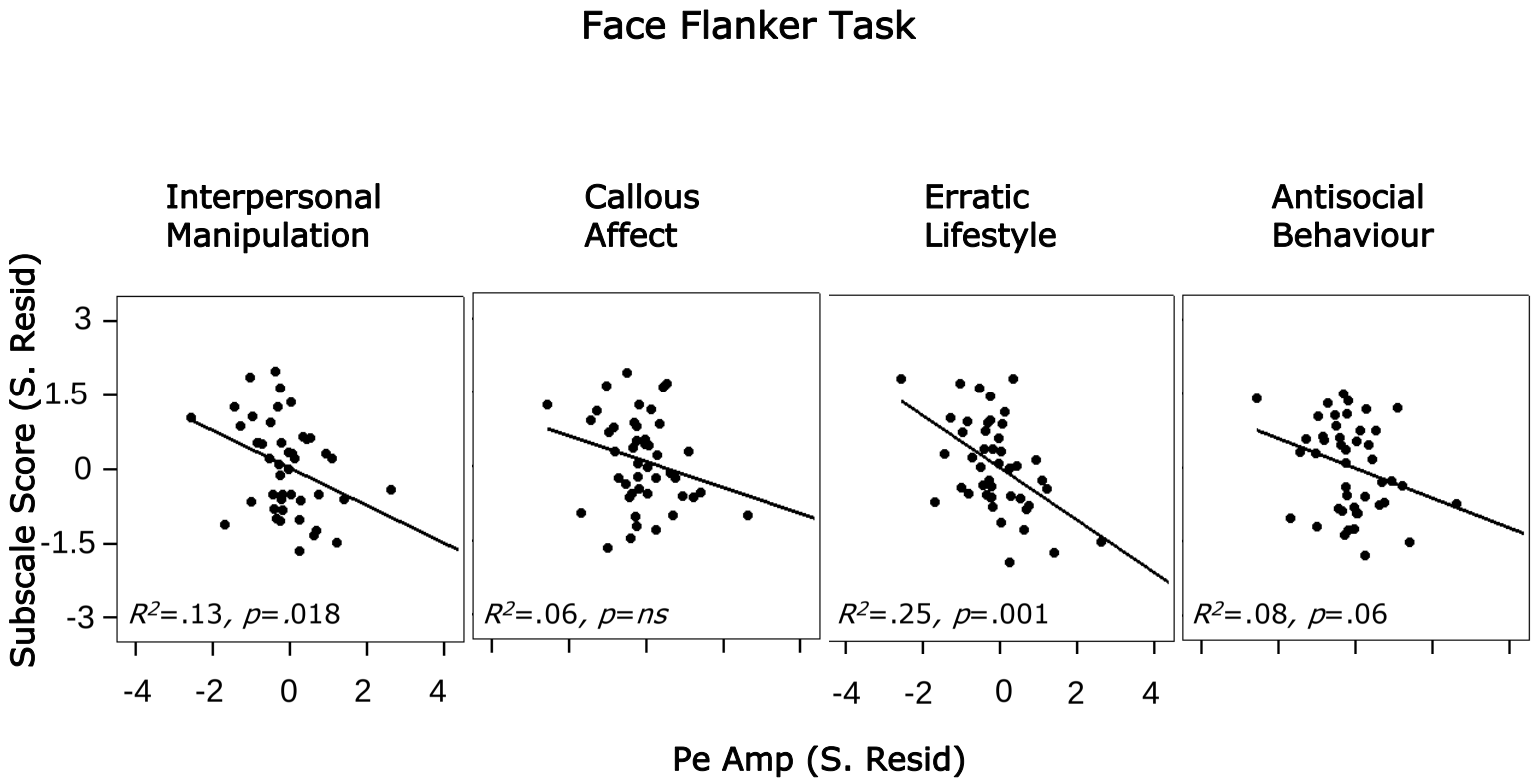

Figure 4.12. Scatter plots indicating the relationship between SRP-III subscale scores for the standardized residual (S. Resid) of response-locked Pe amplitude at Cz on error trials, adjusting for correct trials, on the face flanker task (Study 3).

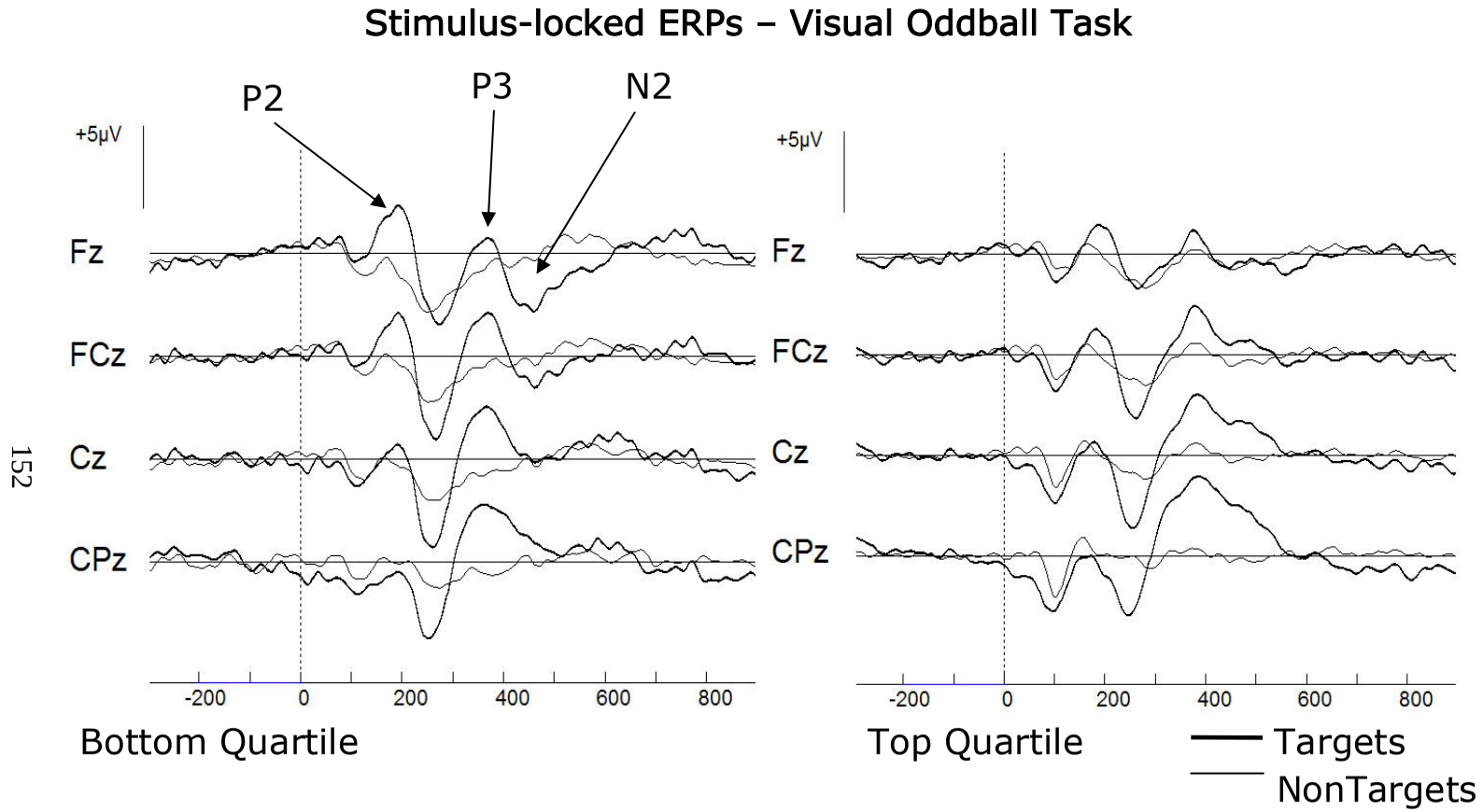

Figure 5.1. Overlaid ERP waveforms for target and nontarget trials for the top and bottom thirds for SRP-III Total Psychopathy scores (Study 4).

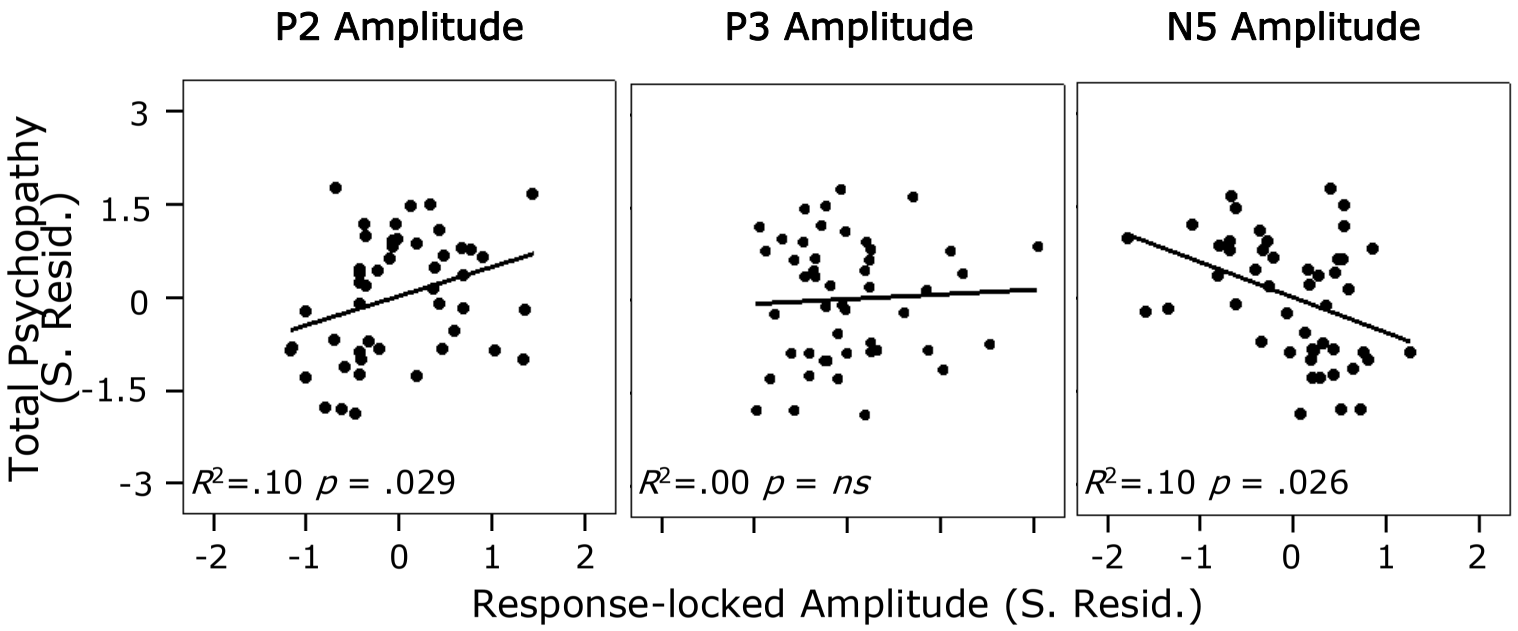

Figure 5.2. Scatter plots indicating the relationship between standardized SRP-III Total Psychopathy scores and the standardized residual (S. Resid) of the stimulus-locked components, P2, P3, and N5 for target trials, adjusted for nontarget trials, during the visual oddball task (Study 4).

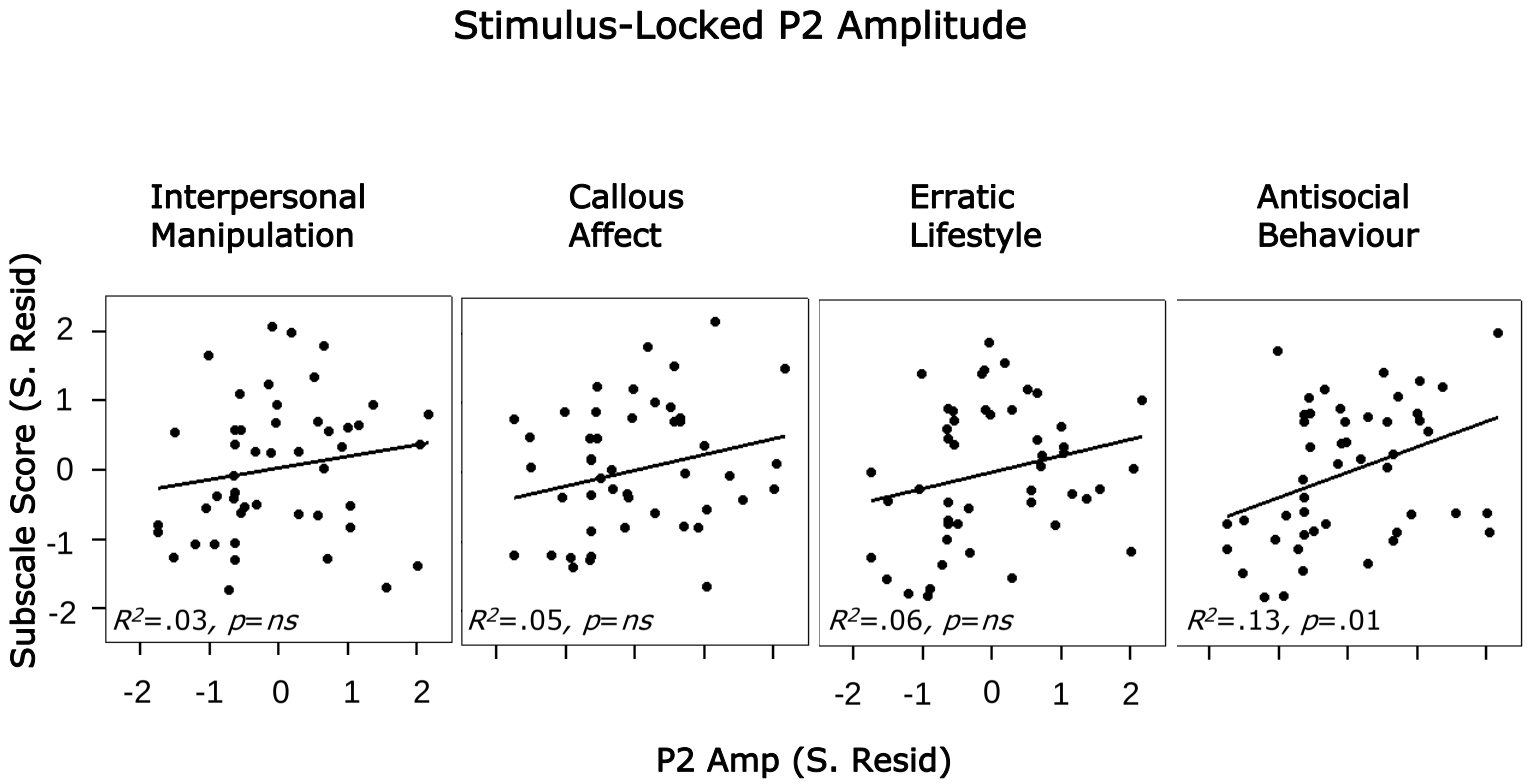

Figure 5.3. Scatter plots indicating the relationship between standardized SRP-III Subscale scores and the standardized residual (S. Resid). of the stimulus-locked P2 at Fz for target trials, adjusted for nontarget trials, during the visual oddball task (Study 4).

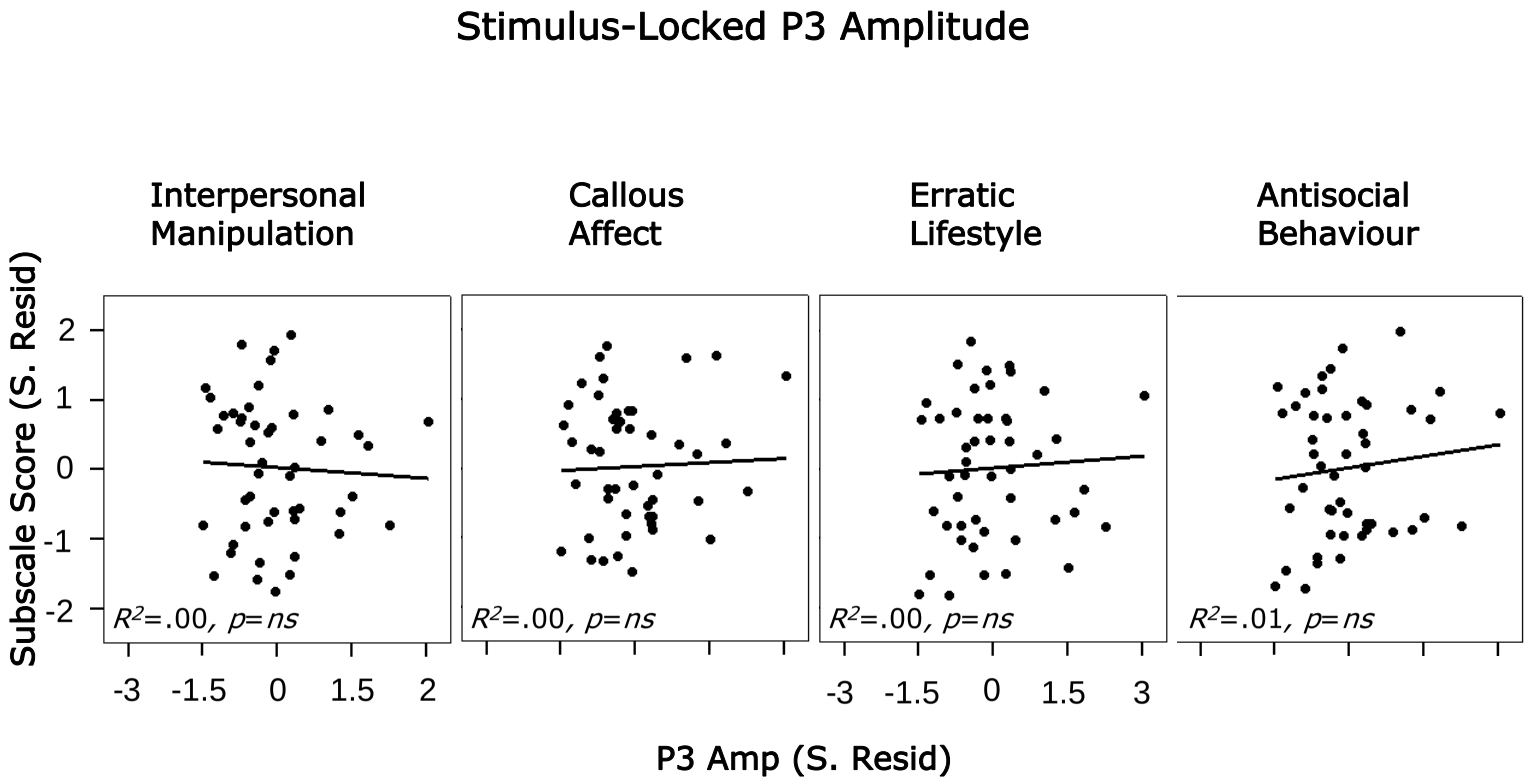

Figure 5.4. Scatter plots indicating the relationship between standardized SRP-III Subscale scores and the standardized residual (S. Resid) of the stimulus-locked P3 at CPz for target trials, adjusted for nontarget trials, during the visual oddball task (Study 4).

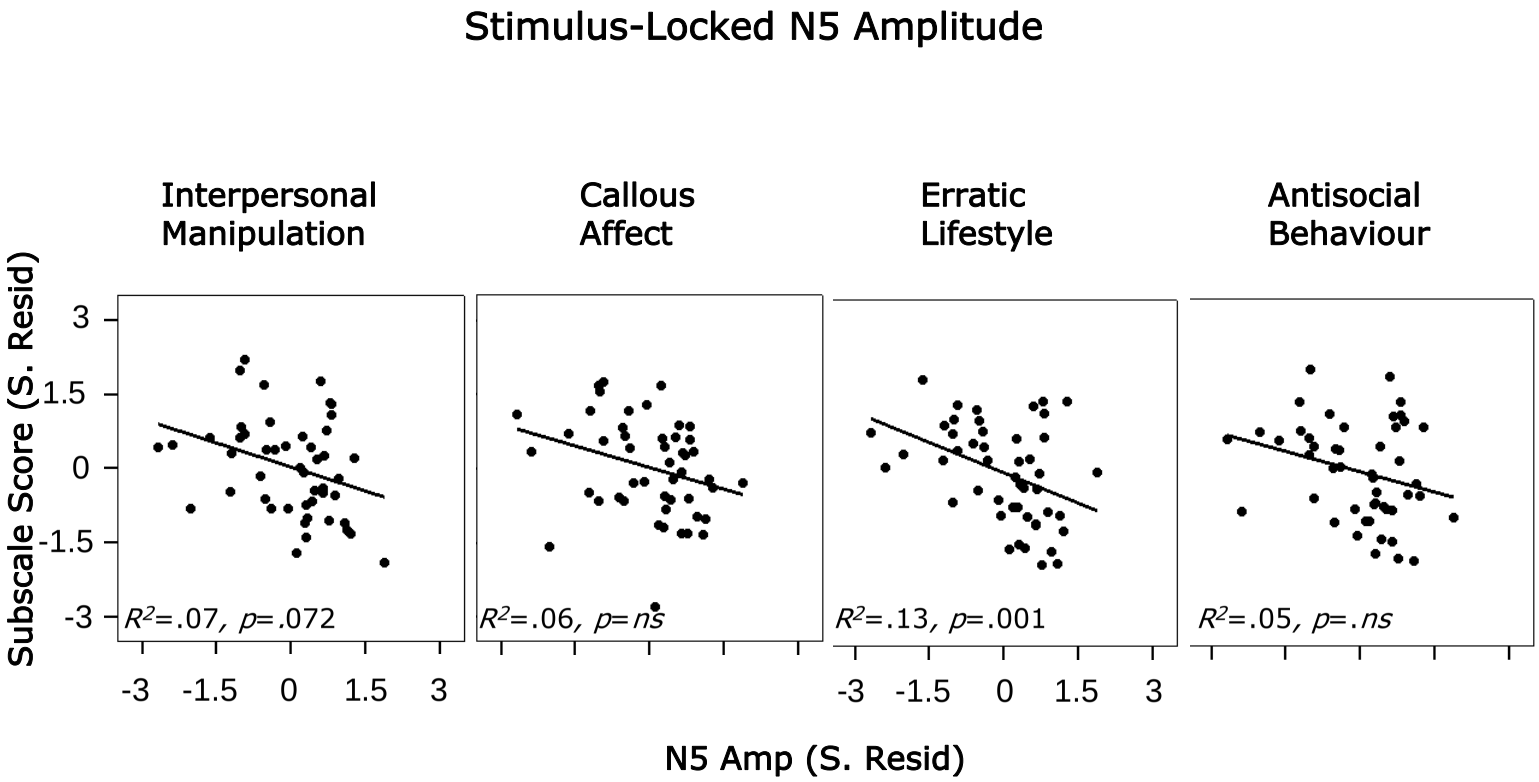

Figure 5.5. Scatter plots indicating the relationship between standardized SRP-III Subscale scores and the standardized residual (S. Resid) of the stimulus-locked N5 at Fz for target trials, adjusted for nontarget trials, during the visual oddball task (Study 4).

Appendix A
Tables

Table 2.1. *Mean Proportion of Errors and Response Times (ms) for Letter Flanker and Face Flanker Tasks for Control and Offender Groups.*

	Letter Flanker		Face Flanker	
Group	Error (SE)	RT (SE)	Error (SE)	RT (SE)
Controls	.10 (.02)	438 (15)	.16 (.03)	625 (38)
Offenders	.09 (.02)	510 (16)	.25 (.03)	723 (40)

Note. Response times are for correct trials.

Table 3.1 Mean Proportion of Errors and Response Times (ms) for the Go/NoGo task for Control and Offender Groups.

	Errors of Commission on NoGo Trials		Errors of Omission on Go Trails		Response Times	
Groups	Mean	SE	Mean	SE	Mean	SE
Controls	.10	.01	.31	.04	360	14.1
Offenders	.10	.01	.43	.04	410	14.1

Table 4.1. *Mean Proportion of Errors and Response Times (ms) for the Easy Letter Flanker, Difficult Letter Flanker, and Face Flanker Tasks.*

	Accuracy	Response Time	
	Error Rate (S.E.)	Correct RT (S.E.)	Error RT (S.E.)
Easy Letter Flanker	.15 (.01)	436 (8)	396 (8)
Hard Letter Flanker	.24 (.02)	454 (7)	400 (8)
Face Flanker	.22 (.02)	578 (17)	556 (18)

Appendix B
Source Tables

Appendix B-2.1.

2 (Task) x 2 (Group) Repeated Measures ANOVA for Mean Percent Error during Study 1.

	ANOVA summary				
Source	*SS*	*df*	*F*	*p*	η^2
Within-Subject Effects					
Task (T)	704.49	1	44.37	.000	.62
T x G	120.79	1	7.61	.010	.22
Error	428.74	27			
Between-Subjects Effects					
Group (G)	112.91	1	2.04	.165	.07
Error	1497.80	27			

Appendix B-2.2.
2 (Emotion) x 2 (Group) Repeated Measures ANOVA for Mean Error Percent in the Emotional Flanker during Study 1.

	ANOVA summary				
Source	SS	df	F	p	η^2
Within-Subject Effects					
Emotion (E)	488.40	1	1.39	.248	.05
E x G	186.95	1	.53	.471	.02
Error	9463.98	27			
Between-Subjects Effects					
Group (G)	3120.46	1	2.10	.159	.07
Error	4.0060.60	27			

Appendix B-2.3.
2 (Task) x 2 (Accuracy) x 2 (Group) Repeated Measures ANOVA for Response Time during Study 1.

ANOVA summary

Source	SS	df	F	p	η^2
Within-Subject effects					
Task (T)	127856.27	1	103.47	.000	.79
T x G	852.39	1	.07	.795	.00
Error	333644.15	27			
Accuracy (A)	55513.97	1	28.03	.000	.51
A x G	2106.14	1	1.06	.312	.04
Error	53468.77	27			
T x A	10494.96	1	7.78	.010	.22
T x A x G	655.65	1	.49	.492	.02
Error	36412.90	27			
Between-Subjects Effects					
Group (G)	135651.38	1	4.82	.037	.15
Error	759290.92	27			

Appendix B-2.4.
2 (Task) x 2 (Accuracy) x 4 (Site) x 2 (Group) Repeated Measures ANOVA for response-locked P3 during Study 1.

ANOVA summary

Source		SS	df	F	p	η^2
Within-Subject Effects						
Task (T)		35.44	1	2.10	.162	.09
T x G		21.10	1	1.25	.276	.06
	Error	354.84	21	.		
Accuracy (A)		.84	1	.06	.808	.00
A x G		5.35	1	.39	.542	.01
	Error	291.62	21			
Site (S)		12.88	3	1.62	.215	.07
S x G		3.21	3	.40	.627	.02
	Error	166.84	63			
T x A		2.33	1	.19	.668	.01
T x A x G		2.98	1	.24	.627	.01
	Error	258.01	21			
T x S		1.67	3	.80	.451	.04
T x S x G		3.43	3	1.65	.205	.07
	Error	43.58	63			
A x S		18.88	3	6.14	.003	.23
A x S x G		9.12	3	2.97	.054	.12
	Error	63.59	63			
T x A x S		1.83	3	.88	.455	.04
T x A x S x G		1.15	3	.55	.648	.03
	Error	43.51	63			
Between-Subjects Effects						
Group (G)		48.88	1	.95	.342	.04
	Error	1084.45	21			

Appendix B-2.5.
2 (Task) x 2 (Accuracy) x 4 (Site) x 2 (Group) Repeated Measures ANOVA for ERN during Study 1.

ANOVA summary

Source		SS	df	F	p	η^2
Within-Subject Effects						
Task (T)		50.60	1	5/24	.033	.20
T x G		122.99	1	12.73	.002	.38
	Error	202.88	21			
Accuracy (A)		439.58	1	19.29	.000	.48
A x G		57.02	1	2.50	.129	.11
	Error	478.45	21			
Site (S)		28.54	3	3.24	.045	.13
S x G		13.34	3	1.51	.23	.07
	Error	185.12	63			
T x A		15.82	1	1.55	.228	.07
T x A x G		38.90	1	380	/065	.15
	Error	215.13	21			
T x S		5.05	3	1.35	.271	.06
T x S x G		3.57	3	.95	.396	.04
	Error	78.70	63			
A x S		38.07	3	6.36	.001	.23
A x S x G		11.27	3	1.88	.15	.08
	Error	125.69	63			
T x A x S		1.50	3	.45	.69	.02
T x A x S x G		.84	3	.25	.83	.01
	Error	69.30	63			
Between-Subjects Effects						
Group (G)		157.07	1	2.49	.13	.11
	Error	1326.56	21			

Appendix B-2.6.
2 (Task) x 2 (Accuracy) x 4 (Site) x 2 (Group) Repeated Measures ANOVA for the Pe during Study 1.

ANOVA summary

Source		SS	df	F	p	η^2
Within-subject effects						
Task (T)		3.17	1	.11	.746	.01
T x G		.04	1	.001	.970	.00
	Error	620.56	21			
Accuracy (A)		3.52	1	.13	.718	.01
A x G		10.27	1	.39	.538	.02
	Error	551.45	21			
Site (S)		9.66	3	1.28	.29	.06
S x G		2.71	3	.36	.72	.02
	Error	158.81	63			
T x A		1.96	1	.09	.774	.00
T x A x G		2.76	1	.12	.733	.01
	Error	485.62	21			
T x S		5.05	3	1.44	.246	.06
T x S x G		3.68	3	1.05	.363	.05
	Error	73.37	63			
A x S		10.56	3	2.24	.13	.10
A x S x G		3.28	3	.67	.488	.03
	Error	99.26	63			
T x A x S		6.41	3	1.96	.15	.09
T x A x S x G		4.94	3	1.51	.232	.07
	Error	68.55	63			
Between-Subjects Effects						
Group (G)		6.13	1	.14	.715	.01
	Error	942.83	21			

Appendix B-3.1.
2 (Trial-Type) x 2 (Group) Repeated Measures ANOVA Error Rate during Study 2.

ANOVA summary

Source	SS	df	F	p	η^2
Within-subject effects					
Trial-Type (T)	739076.27	1	394.31	.000	1.0
T x G	366.93	1	.20	.662	.07
Error	46859.07	25			
Between-subjects effects					
Group (G)	99855.61	1	.97	.335	.16
Error	2578636.76	25			

Appendix B-3.2.
2 (Accuracy) x 2 (Group) Repeated Measures ANOVA Response Time during Study 2.

ANOVA summary

Source	SS	df	F	p	η^2
Within-subject effects					
Accuracy (A)	2091.24	1	3.06	.092	.11
A x G	701.44	1	1.03	.321	.04
Error	17073.77	25			
Between-subjects effects					
Group (G)	923023	1	3.05	.123	.12
Error	6575854.96	25			

Appendix B-3.3.
2 (Condition) x 4 (Site) x 2 (Group) Repeated Measures ANOVA for the P2 during Study 2.

ANOVA summary

Source		SS	df	F	p	η^2
Within-Subject Effects						
Condition (C)		1.04	1	.43	.519	.02
C x G		.78	1	.32	.578	.01
	Error	58.59	24			
Site (S)		31.40	3	10.37	001	.30
S x G		10.18	3	3.36	.064	.12
	Error	72.66	72			
C x S		.65	3	1.14	.33	.05
C x S x G		.16	3	.29	.765	.01
	Error	13.60	72			
Between-Subjects Effects						
Group (G)		27.79	1	1.06	.314	.04
	Error	631.25	24			

Appendix B-3.4.
2 (Condition) x 4 (Site) x 2 (Group) Repeated Measures ANOVA for the N2 during Study 2.

ANOVA summary

Source		SS	df	F	p	η^2
Within-subject effects						
Condition (C)		27.67	1	6.65	.016	.21
C x G		.08	1	.02	.888	.00
	Error	103.99	25			
Site (S)		8.21	3	1.34		
S x G		6.95	3	1.13	.316	.04
	Error	153.85	25			
C x S		1.58	3	4.25	.014	.15
C x S x G		.17	3	.29	.790	.01
	Error	15.21	75			
Between-Subjects Effects						
Group (G)		65.37	1	1.31	.263	.05
	Error	1246.16	25			

Appendix B-3.5.
2 (Condition) x 4 (Site) x 2 (Group) Repeated Measures ANOVA for the P3 during Study 2.

ANOVA summary

Source		SS	df	F	p	η^2
Within-subject effects						
Condition (C)		51.46	1	5.40	.029	.18
C x G		7.58	1	.80	.381	.03
	Error	238.15	25			
Site (S)		18.98	3	5.29	.017	.18
S x G		17.26	3	4.81	.023	.16
	Error	89.77	75			
C x S		.32	3	.37	.733	.01
C x S x G		.14	3	.16	.888	.01
	Error	21.97	75			
Between-Subjects Effects						
Group (G)		15.16	1	.50	.484	.02
	Error	751.89	25			

Appendix B-4.1.
2 (Task) x 2 (Congruency) Repeated Measures ANOVA for Error Trials during Study 3.

ANOVA summary

Source		SS	df	F	p	η^2
Task (T)		10610.69	1	9.91	.003	.18
	Error	47134.81	44			
Congruency (C)		583.20	1	14.41	.000	.25
	Error	1780.30	44			
T x C		381.36	1	9.75	.003	.18
	Error	1721.14	44			

Appendix B-4.2.
One-Way ANOVA for Task and Error Trials during Study 3.

ANOVA summary

Source	SS	df	F	p	η^2
Task (T)	.21	2	12.81	.000	.23
Error	.73	88			

Appendix B-4.3.
2 (Task) x 2 (Accuracy) Repeated Measures ANOVA for Response Time during Study 3.

ANOVA summary

Source		SS	df	F	p	η^2
Task (T)		1275192.91	2	101.64	.000	.70
	Error	552055	88			
Accuracy (A)		101893.73	1	112.85	.000	.72
	Error	39728.80	44			
T x A		11293.71	2	9.15	.000	.17
	Error	54286.99	88			

Appendix B-4.4.
3 (Task) x 2 (Accuracy) x 4 (Site) Mixed-Model ANOVA for the P3 during Study 3.

ANOVA summary

Source		SS	df	F	p	η^2
Task (T)		5.38	2	.29	.748	.01
	Error	793.04	86			
Accuracy (A)		22.70	1	2.90	.096	.06
	Error	336.13	43			
Site (S)		625.13	3	86.52	.000	.67
	Error	310.68	129			
T x A		123.70	2	13.62	.00	.24
	Error	390.60	86			
T x S		11.24	6	2.55	.055	.06
	Error	189.47	258			
A x S		68.51	3	33.58	.000	.44
	Error	87.75	129			
T x A x S		11.24	6	3.71	.014	.08
	Error	130.44	258			

Appendix B-4.5.
3 (Task) x 2 (Accuracy) x 4 (Site) Mixed-Model ANOVA for the ERN during Study 3.

ANOVA summary

Source		SS	df	F	p	η^2
Task (T)		119.49	2	6.28	.004	.13
	Error	837.58	88			
Accuracy (A)		2975.83	1	188.97	.000	.81
	Error	692.90	44			
Site (S)		408.68	3	34.57	.000	.44
	Error	520.18	132			
T x A		172.94	2	88.98	.000	.18
	Error	796.90	88			
T x S		12.53	6	2.14	.092	.05
	Error	257.10	264			
A x S		347.04	3	61.16	.000	.58
	Error	249.67	132			
T x A x S		61.51	6	13.33	.000	.23
	Error	203.06	264			

Appendix B-4.6.
3 (Task) x 2 (Accuracy) x 4 (Site) Mixed-Model ANOVA for the Pe during Study 3.

ANOVA summary

Source		SS	df	F	p	η^2
Task (T)		159.65	2	7.50	.001	.15
	Error	936.32	88			
Accuracy (A)		1102.11	1	39.94	.000	.48
	Error	1214.13	44			
Site (S)		253.28	3	48.08	.000	.52
	Error	231.80	132			
T x A		73.44	2	3.86	.027	.08
	Error	837.04	88			
T x S		12.35	6	2.76	.05	.06
	Error	197.13	264			
A x S		30.07	3	8.84	.001	.17
	Error	149.72	132			
T x A x S		5.77	6	1.77	.147	.04
	Error	143.66	264			

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