# Response Monitoring and Adjustment: Differential Relations with Psychopathic Traits

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## Abstract

Studies on the relation between psychopathy and cognitive functioning often show mixed results, partially because different factors of psychopathy have not been considered fully. Based on previous research, we predicted divergent results based on a two-factor model of psychopathy (interpersonal-affective traits and impulsive-antisocial traits). Specifically, we predicted that the unique variance of interpersonal-affective traits would be related to increased monitoring (i.e., error-related negativity) and adjusting to errors (i.e., post-error slowing), whereas impulsive-antisocial traits would be related to reductions in these processes. Three studies using a diverse selection of assessment tools, samples, and methods are presented to identify response monitoring correlates of the two main factors of psychopathy. In Studies 1 (undergraduates), 2 (adolescents), and 3 (offenders), interpersonal-affective traits were related to increased adjustment following errors and, in Study 3, to enhanced monitoring of errors. Impulsive-antisocial traits were not consistently related to error adjustment across the studies, although these traits were related to a deficient monitoring of errors in Study 3. The results may help explain previous mixed findings and advance implications for etiological models of psychopathy.

## Keywords

In the last few decades, psychometric research has indicated that psychopathy is likely a multidimensional entity composed of at least two factors, which themselves are a collection of traits and behaviors (Benning, Patrick, Hicks, Blonigen, & Krueger, 2003; Cooke & Michie, 2001; Hare, 2003; Patrick, Fowles, & Krueger, 2009). Furthermore, multivariate analyses indicate that different factors of psychopathy show unique and sometimes opposing correlates in cognitive, affective, and behavioral domains (Hare, 2003; Hicks & Patrick, 2006; Patrick & Zempolich, 1998; Sadeh & Verona, 2008; Vaidyanathan, Hall, Patrick, & Bernat, 2011; Vitacco, Neumann, & Jackson, 2005). It is important to understand the differential correlates of these factors as a way of getting closer to identifying distinct etiological pathways that manifest in psychopathy and further building the nomological

network of psychopathy factors and their unique influence on outcomes of interest (cf. Verona & Miller, in press).

In comparison to the affective and behavioral domains, there has been less research and theory devoted to understanding differential correlates of psychopathic traits and cognitive functioning. Moreover, much of the research that does exist has focused on general aspects of cognition (e.g., intelligence: Salekin, Neumann, Leistico, & Zalow, 2004; Vitacco et al., 2005; executive functioning: Morgan & Lilienfeld, 2000; Ogilvie, Stewart, Chan, & Shum, 2011) or focused on total psychopathy scores instead of specific factors (Brazil et al., 2009; Munro et al., 2007; though see Baskin-Sommers, Zeier, & Newman, 2009 for a recent counter example). Thus, more work is needed to explicate basic processes that govern relations between cognitive functions and each factor of psychopathy. With these gaps in mind, the goal of this paper was to examine the relation between the two main factors of psychopathy (i.e., interpersonal-affective and impulsive-antisocial) and two basic processes that are important to many aspects of cognitive functioning—specifically, monitoring and adjusting to errors (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Rabbitt, 1966).

## Factors of Psychopathy

Though classic clinical descriptions of psychopathy describe a unitary construct (e.g., Cleckley, 1976), factor analytic work on psychopathy assessments support a multi-factor solution (e.g., Benning et al., 2003; Hare & Neumann, 2008; Lynam & Widiger, 2007; Patrick et al., 2009). Indeed, across many common psychopathy assessments, a two-factor model has been proposed (Benning et al., 2003; Harpur, Hare, & Hackstain, 1989). For instance, the Psychopathic Personality Inventory (PPI; Lilienfeld & Andrews, 1996), a self-report measure designed for the general population, is usually represented by two general factors: Fearless Dominance and Impulsive Antisociality, with one of the subscales, Coldheartedness, not loading on either factor (Benning et al., 2003; Patrick, Poythress, Edens, Lilienfeld, & Benning, 2006; although see Neumann, Malterer & Newman, 2008 for an alternative factor structure). Likewise, Hare's Psychopathy Checklist instruments (PCL-R, PCL: SV; Hare, 2003; Hart, Cox, & Hare, 1999), designed for forensic populations, have commonly been decomposed into two factors (termed Factor 1 and Factor 2; Harpur et al., 1989; Verona, Patrick, & Joiner, 2001). Although recent recommendations suggest a four-facet structure, this is a higher order model and can be reduced to the two-factor model (e.g., Hare & Neumann, 2008). Across the PPI and PCL-based measures, the two factors can loosely be described as interpersonal-affective (i.e., Fearless Dominance and Factor 1) and impulsive-antisocial traits (Impulsive Antisociality and Factor 2). However, the exact nature of the traits captured in these factors varies by assessment type.

The differential coverage of the PPI and PCL-based measures is most apparent when comparing the Fearless Dominance scale of the PPI and Factor 1 of the PCL-R (i.e., interpersonal-affective traits). Whereas Fearless Dominance focuses on interpersonal traits related to social dominance (e.g., an ability to influence) and affective traits focused on the absence of anxiety (e.g., low fear, stress immunity), Factor 1 focuses on interpersonal traits of a more antagonistic variety (e.g., deceitfulness, conning, superficial charm) and affective traits focused on the absence of empathy and remorse. Given that each scale assesses

different types of interpersonal-affective traits, it is not surprising that the correlation between Fearless Dominance and Factor 1 is small (Marcus et al., 2013; Miller & Lynam, 2012). In spite of this small correlation, research suggests that there is overlap between the nomological networks of the two scales (Poythress et al., 2010). For example, both Fearless Dominance and the unique variance of Factor 1 (i.e., covarying Factor 2) are negatively related to neuroticism (Harpur et al., 1989; Hicks & Patrick, 2006; Marcus et al., 2013; Miller & Lynam, 2012; Patrick, Hicks, Nichol, & Krueger, 2007; Verona et al., 2001) and reduced fear-potentiated startle (Benning, Patrick, & Iacono, 2005; Vaidyanathan et al., 2011). Moreover, both scales are positively related to aspects of positive adjustment (e.g., social potency; Marcus et al., 2013; Miller & Lynam, 2012; Patrick et al., 2007; Verona et al., 2001).

In comparison to the differential coverage of interpersonal-affective traits in the PPI and PCL-based measures, the impulsive-antisocial traits across both measures have stronger convergence. For instance, both Impulsive Antisociality and Factor 2 assess similar types of impulsivity (e.g., failure to plan ahead, proneness to boredom). Not surprisingly, the correlation between them is moderate to large (Marcus et al., 2013; Miller & Lynam, 2012). Moreover, the two scales have very similar correlates, being positively related to substance use, aggression, and neuroticism (Harpur et al., 1989; Marcus et al., 2013; Miller & Lynam, 2012; Verona et al., 2001), suggesting similar nomological networks.

Despite the presence of multiple factors, psychopathy is often defined as the combination of high levels of both interpersonal-affective traits and impulsive-antisocial traits (Lilienfeld, 2013; Patrick et al., 2009). Specifically, it has been proposed that it is the interpersonal-affective traits that distinguish psychopathy from other disorders that are composed of impulsive-antisocial traits (e.g., antisocial personality disorder; Patrick et al., 2013). Still, there is utility in studying each factor separately, as the unique correlates may reflect different etiological pathways to psychopathy. This idea is referenced in theoretical work on subtypes of psychopathy (Karpman, 1941; Lykken, 1995), with one subtype (i.e., primary psychopathy) being driven mostly by the etiological processes thought to give rise to interpersonal-affective traits and another subtype (i.e., secondary psychopathy) being influenced mostly by impulsive-antisocial traits (e.g., development of low empathy or shallow affect as a result of social experiences linked to impulsivity). There is growing research to suggest that such subtypes exist among individuals high in total psychopathy scores (e.g., Falkenbach, Stern, & Creevy, 2014; Hicks, Markon, Patrick, Krueger, & Newman, 2004; Skeem, Johansson, Andershed, Kerr, & Louden, 2007). Thus, understanding the unique correlates of interpersonal-affective traits and impulsive-antisocial traits may help identify distinct pathways to psychopathy manifested by different subtypes.

## Cognition and the Two Psychopathy Factors

One interesting area of divergence between interpersonal-affective traits and impulsive-antisocial traits is cognitive abilities. Though there is a negative relationship between criminality and intelligence (Rushton & Templer, 2009), classical theories of psychopathy posit that individuals with psychopathy have "good intelligence" (Cleckley, 1976). This discrepancy fits with the unique relations between interpersonal-affective/impulsive-

antisocial traits and cognitive ability. Studies using both the PPI and PCL have found that interpersonal-affective traits are unrelated or positively related to cognitive functioning, whereas impulsive-antisocial traits are negatively related to intelligence, particularly when adjusting for the overlap between the two factors (Benning et al., 2003; Heinzen, Köhler, Godt, Geiger, & Huchzermeir, 2011; Neumann & Hare, 2008; Salekin et al., 2004; Vitacco et al., 2005). In terms of executive functioning, research with the PCL has found that impulsive-antisocial traits are related to deficits in executive functioning (Bernat, Nelson, Steele, Gehring, & Patrick, 2011; Morgan & Lilienfeld, 2000), although null results have been found when using the PPI (Carlson & Thái, 2010). In contrast, interpersonal-affective traits as indexed by the PCL or PPI are either unrelated to executive functioning (e.g., Hart, Forth, & Hare, 1990), or for some aspects, related to superior executive functioning (Baskin-Sommers et al., 2009; Sadeh & Verona, 2008; Sellbom & Verona, 2007). In sum, there seems to be evidence of divergent relations between the two psychopathy factors and cognitive functioning.

Though these results are important, intelligence and executive functioning are broad constructs. Relatively less research has focused on specific processes that may have differential relations to psychopathic traits (though see Baskin-Sommers et al., 2009; Carlson & Thái, 2010; Sadeh & Verona, 2008 for exceptions). Therefore, further research is necessary to clarify relations between basic cognitive processes and factors of psychopathy that generalize across samples and instruments. Two processes that may be relevant to understanding the divergent relations of interpersonal-affective and impulsive-antisocial traits and general cognitive functioning are monitoring and adjusting to errors.

## Error Monitoring and Adjustment

In order to regulate behavior and pursue goals, it is necessary to monitor discrepancies between one's current state and the desired goal (Carver & Scheier, 1990; Robinson, Schmeichel, & Inzlicht, 2010). When large discrepancies are detected, the individual must adjust his or her behavior to be more in line with relevant goals. These two processes map quite well onto the cognitive processes of monitoring and adjusting to errors. The error-related negativity (ERN), an event-related potential (ERP) that consists of a negative deflection following an error of commission (Gehring, Gross, Coles, Meyer, & Donchin, 1993), is thought to reflect the monitoring process. The ERN is larger (i.e., more negative) for error trials compared to correct trials (Gehring et al., 1993) and is thought to reflect the conflict or discrepancy between response options (Botvinick et al., 2001; Yeung, Botvinick, & Cohen, 2004). Similarly, there is a general tendency for individuals to slow their reaction time (RT) on trials following an error, consistent with behavioral adjustment (Rabbitt, 1966). It has been proposed that this post-error slowing (PES) reflects an increase in response caution following an error (Botvinick et al., 2001; Dutilh et al., 2012).

Based on the differential correlates of the two factors of psychopathy, it might be predicted that the two factors would have divergent relations with monitoring and adjusting to errors. First, because individuals high (versus low) in interpersonal-affective traits are manipulative and able to influence others, they should be highly attentive to errors, and be better able to adjust their behavior as a way of avoiding detection (i.e., an outward appearance of mental

health disguising interpersonal, emotional, and behavioral problems; Cleckley, 1976). This also fits with the findings of enhanced intelligence (e.g., Neumann & Hare, 2008) and enhanced executive functioning in some domains (e.g., Sadeh & Verona, 2008) in individuals high in interpersonal-affective traits of psychopathy. Second, because individuals high (versus low) in impulsive-antisocial traits may lack planning and foresight, they may be less able to notice errors and less able to adjust following mistakes. This proposal is consistent with previous findings of negative relations between impulsive-antisocial traits and intelligence and executive functioning (Neumann & Hare, 2008).

Multiple studies have examined PES in individuals high (versus low) in psychopathy, including early work by Newman and colleagues (1987; see Patterson & Newman, 1993 for a review of some of this work), with recent work showing mixed results (e.g., Brazil et al., 2009). However, this work did not differentiate between subcomponents of psychopathy. To our knowledge, only two studies, presented in the same paper, have examined the relation between the psychopathy factors and PES. In this paper, Wilkowski and Robinson (2008) found that undergraduate students scoring high on the secondary psychopathy scale (impulsive-antisocial traits) of the Levenson Self-Report Psychopathy Scales (LSRPS; Levenson, Kiehl, & Fitzpatrick, 1995) displayed deficient RT slowing following errors. There was no relation between the primary psychopathy scale (interpersonal-affective traits) and PES. One limitation of this study is that the primary and secondary psychopathy subscales of the LSRPS both seem to index antagonism, callousness, and impulsivity (Lynam, Whiteside, & Hones, 1999). Indeed, some have argued that this scale does not have accurate coverage of the interpersonal aspects of psychopathy as defined by Cleckley (see Lilienfeld & Fowler, 2006). Moreover, the authors focused on zero-order relationships and did not account for the overlap between the primary and secondary psychopathy scales when examining relationships with PES. Many measures of psychopathy, including the LSRPS, show moderate to large correlations between the factors (e.g., Hare, 2003; Levenson et al., 1995), and the divergent correlates are only apparent when adjusting for this overlap, particularly for interpersonal-affective traits. Although caution is warranted in interpreting the residual variance (Lynam, Hoyle, & Newman, 2006), the unique variance of each factor in previous research has been shown to be meaningful and revealing of putative etiological processes (Hicks & Patrick, 2006; Neumann & Hare, 2008; see Verona & Miller, in press). Thus, it is important to look at results for zero-order relations and relations with the other factors partialled out.

Previous research has also examined the relation between psychopathy and error monitoring (i.e., ERN) with mixed results. Two studies have found evidence for decreased ERN for individuals high (versus low) in psychopathy (Brazil et al., 2011; Munro et al., 2007), and one found no group differences (Brazil et al., 2009). These studies, however, did not examine the two factors of psychopathy separately. A recent study by Heritage and Benning (2013) examined the unique relations between ERN and interpersonal-affective and impulsive-antisocial traits, as measured by the PPI (i.e., Fearless Dominance and Impulsive Antisociality). The results showed that Fearless Dominance was not related to ERN amplitude, whereas Impulsive Antisociality was related to reduced ERN. The results for Impulsive Antisociality traits are consistent with other ERN studies that have found that constructs composed of high impulsivity (e.g., substance dependence, externalizing

psychopathology) are related to reduced ERN amplitude (Franken, van Strien, Franzek, & van de Wetering, 2007; Hall, Bernat, & Patrick, 2007). Thus, the extant research supports predicted negative relations between impulsive-antisocial traits and monitoring and adjusting to errors. However, the evidence in support of a positive relationship with regard to interpersonal-affective traits is absent from the literature.

## Current Studies

In summary, there are theoretical reasons to assume that the two general factors of psychopathy (interpersonal-affective and impulsive-antisocial traits) have differential relations to monitoring and adjusting to errors. However, most studies on psychopathy and monitoring and adjusting to errors have conceptualized psychopathy as a unitary construct. Moreover, few studies have examined relations across different samples and psychopathy assessment tools, which may help in uncovering the constituent traits shared across instruments that are playing a role in psychopathic cognitive functioning. To expand upon the current literature, we examined data from three studies. Due to the novelty of our predictions, the first two studies involved secondary data analyses of two existing data sets – one a sample of college students and the other a sample of community adolescents – to examine relations between psychopathic traits and PES, measured as RT slowing following errors. The third study examined both ERN and PES in a sample of individuals with a history of involvement in the criminal justice system. Based on theory and limited data, our general predictions were that interpersonal-affective traits as measured by PPI Fearless Dominance and PCL Factor 1would be related to increased monitoring and adjusting to errors, whereas impulsive-antisocial traits would be related to reduced monitoring and adjusting to errors.

## Study 1

The primary goal of Study 1 was to examine the relation between PES and the two factors of psychopathy. To do this, we examined data from an unpublished task in a sample previously collected in our lab (Sprague & Verona, 2010). The main goal of the original project was to determine whether individuals high (relative to low) on dysregulation (represented by high levels of both borderline and antisocial traits) would show emotion-modulated behavioral dyscontrol. Thus, participants completed an emotional-linguistic go/no-go (GNG) task, which measured the effects of negative emotional context on response inhibition. Participants from this study also completed a letter-flanker task, which was not analyzed in the previous publication (Sprague & Verona, 2010). They also completed a self-report measure of psychopathy, specifically the short form of the PPI, which allowed us to examine the relation between PES and factors of psychopathy in the current paper.

Study 1 served as a replication and extension of Wilkowski and Robinson (2008) in that the latter study assessed psychopathy using the LSRPS, based partly on the PCL-R, in which the two factors are highly correlated with each other. In contrast, the PPI was developed based on trait models, and the two main factors (i.e., Fearless Dominance and Impulsive Antisociality) have substantially smaller correlations with each other than the LSRPS or PCL-based measures (Benning et al., 2003; Marcus et al., 2013). Due to the uncorrelated

nature of Fearless Dominance and Impulsive Antisociality, the present study provided a strong test of the unique relations between the two factors and PES.

## Method

### Participants

Participants were recruited from a larger pool of 318 undergraduates who completed self-report measures of personality pathology to screen for traits related to emotional and behavioral dysregulation. Potential participants who scored in the upper 65$^{th}$ or lower 35$^{th}$ percentile of a composite measure of dysregulated personality disorder traits (i.e., borderline and antisocial) were invited to participate (see Sprague & Verona, 2010 for more details on screening measures). As detailed below, this process produced adequate spread in scores for the two factors of psychopathy. A total of 83 participants (43 women) were recruited to participate in the study for course credit. Two participants were missing RT data and thus excluded from the analyses. The majority of the sample was between the ages of 18 and 21 (94%). The ethnic breakdown was 60% Caucasian, 22% Asian, 8% Hispanic, 8% other, and 1% African-American. Approximately 62% of the sample reported a family income above $60,000 a year. Data from these participants on a different task were reported by Sprague and Verona (2010). However, results from the flanker task have not been published.

### Psychopathy Assessment

Psychopathy was measured using the Psychopathic Personality Inventory - Short Form (PPI-S; Lilienfeld & Andrews, 1996). Participants indicated the extent to which 56 items applied to them on a 4-point Likert scale (1= *false*, 4 = *true*). Based on previous factor analytic work (Benning et al., 2003), we computed two scores for each participant: Fearless Dominance (α = .85) and Impulsive Antisociality (α = .85; also known as Self-Centered Impulsivity; Lilienfeld & Widows, 2005). The subscale Coldheartedness is typically not represented in either factor and thus was not used in main analyses.[1] Consistent with other research (see Marcus et al., 2013 for a review), the correlation between Fearless Dominance and Impulsive Antisociality was close to zero ($r$ = .02, $p$ = .843).

Given that participants were recruited based on extreme scores on personality traits related to dysregulation (i.e., symptoms of borderline and antisocial personality disorders), we carefully examined the distributions of the PPI scores to ensure normality. We did this because we were interested in using continuous scores, consistent with research indicating that psychopathy is a dimensional, and not taxonic, construct (e.g., Marcus, John, & Edens, 2004). To establish the normality of the distributions, we calculated four indices: skew, kurtosis, bimodality index, and the Kolmogorov-Smirnov test. All skew (Fearless Dominance = −.48, Impulsive Antisociality = −.09) and kurtosis (Fearless Dominance = −.08, Impulsive Antisociality = −.85) values were less than 1. Moreover, the $Z$-tests indicated

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[1]For completeness, we also examined results including the Coldheartedness subscale (α = .64). Coldheartedness was not significantly correlated with accuracy ($r$ = −.10, $p$ = .780). When we included this scale in our main analyses, there were no significant effects involving Coldheartedness. Moreover, including Coldheartedness did not affect the significance of the main results. Finally, the zero-order correlation between Coldheartedness and PES was positive, but not significant, $r$ = .15 $p$ = .19. We did not conduct analyses involving Coldheartedness in Study 2 because Coldheartedness is usually not estimated in studies using estimated PPI scores (e.g., Benning et al., 2005; Blonigen et al., 2006), so the nomological network of estimated Coldheartedness scores is unclear.

that neither of the variables had significant skew (*p*'s .078 & .374) or kurtosis (*p*'s .393 & .108). Neither of the variables had a bimodality index greater than .55 (Fearless Dominance = .422, Impulsive Antisociality = .468), which is suggestive of a bimodal distribution (Freeman & Dale, 2013; Pfister, Shwarz, Janczyk, Dale, & Freeman, 2013). Finally, neither of the Kolmogorov-Smirnov tests was significant, suggesting that the data did not depart from normality. Hence, we used untransformed continuous scores.

## Flanker Task

The flanker task was a modified version of the Eriksen and Eriksen (1974) task. Participants were required to respond to a central target (e.g., H) among flanking distracters (e.g., *SSHSS*). However, we used a go/no-go version of the task in which participants were instructed to respond to one target (i.e., *H*) that occurred on 80% of trials and to withhold responding when the central target was a different letter (i.e., *S*) that occurred on 20% of trials, thus encouraging a dominant response set. Participants were told to respond as quickly and accurately as possible to emphasize both speed and accuracy. Flanking distracters were either congruent or incongruent with the target. Following a practice block, participants completed four blocks of 40 trials each, with two of the blocks involving a noise stressor. In this report, we focus only on the two no-stress blocks[2], which consisted of a total of 80 trials per participant. Within a trial, the stimulus was presented on the screen for 500 milliseconds (ms), and following onset, participants had 2000 ms to respond. The inter-trial interval varied for each trial (1500, 1750, or 2500 ms). Feedback was not given for incorrect responses. All participants completed an emotional go/no-go task (involving word stimuli; Sprague & Verona, 2010) before the completion of the flanker task.

To reduce the influence of outliers, we discarded trials that were aberrantly fast (< 150 ms) or slow (> 1000 ms). Trials were coded as to the accuracy of the current trial (i.e., trial$_n$) and the accuracy of the previous trial (i.e., trial$_{n-1}$). For both current and previous trials, trials involving no response (either correct rejections or errors of omission) were not considered due to our interest in PES following errors of commission. We also discarded trials in which an error of commission was made (i.e., trial$_n$ is an error) in analysis of PES (although those trials were important in determining whether slowing occurred in trials following error trials). This resulted in 4.93% of trials being discarded. Accuracy in the task was quite high (*M* = 95.07%). Interestingly, in this study, accuracy (or percentage of trials correct) had a small to medium correlation with Fearless Dominance (*r* = .23, *p* = .036), but not Impulsive Antisociality, (*r* = −.03, *p* = .780). Despite the relationship between Fearless Dominance and accuracy, accuracy was not significantly correlated with PES (*r* = −.11, *p* = .340). Participants made an average of 3.25 (*SD* = 2.25, *Max* = 13) errors leaving an average of 2.70 (*SD* = 2.09, *Max* = 12) errors on the previous trial. Though there are no published guidelines on the number of trials necessary to calculate PES, this number of errors is lower

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[2]In a second set of blocks (80 trials total), an aversive boat horn noise was administered on 30% of trials, and not all individuals consistently received the noise due to technical difficulties. Given that stress was not the focus of the current study and that all participants received both types of blocks in a within-subject design, we did not expect that removing these trials would influence the results. Interestingly, there was a significant Fearless Dominance by lag-error by stress block type interaction, $\gamma = 2.63$, $t = 3.21$, $p = .001$. Follow-up tests indicated that the two-way Fearless Dominance by lag-error interaction was significant for the no-stress blocks, $\gamma = 1.99$, $t = 3.25$, $p = .001$, but not the stress blocks, $\gamma = −.47$, $t = −.88$, $p = .378$.

than other published studies, which usually average around 10 errors per participant (e.g., Rabbitt, 1968; Wilkowski & Robinson, 2007).

## Results

Due to the nested nature of the data (trials within subjects), we used multi-level modeling (MLM; Singer & Willett, 2003) to examine relations between psychopathic personality traits and PES. For our purposes, MLM had three distinct advantages over repeated measures ANOVA. First, because participants had different numbers of errors, they had different numbers of trials available for analysis, and MLM is robust to unbalanced data (Judd, Westfall, & Kenny, 2012). Second, MLM does not require that the assumption of sphericity be met. Instead, MLM allows for the modeling of an unstructured variance-covariance matrix, which reduces Type I errors (Judd et al., 2012). Finally, the MLM framework provides a more intuitive set-up for follow-up tests with a continuous between-subject moderator and a categorical within-subject variable (Preacher, Curran, & Bauer, 2006). The Level 1 (within-person) model was:

$$y_{ij}=b_{0j}+b_{1j}\,(lag\text{-}error_{ij})+r_{ij} \quad (1)$$

where $y_{ij}$ is the RT for the ith trial for participant j, $b_{0j}$ is the within-person average RT for trials that follow a correct trial, $b_{1j}$ is the within-person difference between post-correct and post-error trials (i.e., PES), and $r_{ij}$ is the within-person residual. Lag-error was dummy coded such that 0 = the previous trial was correct, 1 = the previous trial was an error.

The Level 2 (between-person) models were:

$$\begin{aligned}
b_0 = \gamma_{00} &+ \gamma_{01}\,(Fearless\ Dominance_j) \\
&+ \gamma_{02}\,(Impulsive\ Antisociality_j) \\
&+ \gamma_{03}\,(Fearless\ Dominance_j * Impulsive\ Antisociality_j) + u_{10}
\end{aligned} \quad (2)$$

$$\begin{aligned}
b_1 = \gamma_{10} &+ \gamma_{11}\,(Fearless\ Dominance_j) \\
&+ \gamma_{12}\,(Impulsive\ Antisociality_j) \\
&+ \gamma_{13}\,(Fearless\ Dominance_j * Impulsive\ Antisociality_j)
\end{aligned} \quad (3)$$

and the combined model was:

$$\begin{aligned}
y_{ij} = \gamma_{00} &+ \gamma_{01}\,(Fearless\ Dominance_j) \\
&+ \gamma_{02}\,(Impulsive\ Antisociality_j) \\
&+ \gamma_{03}\,(Fearless\ Dominance_j * Impulsive\ Antisociality_j) \\
&+ \gamma_{10}\,(lag\text{-}error_{ij}) \\
&+ \gamma_{11}\,(Fearless\ Dominance_j * lag\text{-}error_{ij}) \\
&+ \gamma_{12}\,(Impulsive\ Antisociality_j * lag\text{-}error_{ij}) \\
&+ \gamma_{13}\,(Fearless\ Dominance_j * Impulsive\ Antisociality_j * lag\text{-}error_{ij}) + r_{ij} + u_{10}
\end{aligned} \quad (4)$$

where $\gamma_{00}$ is the grand average RT for lag-correct trials, $\gamma_{10}$ is the grand average effect of PES, $\gamma_{01}-\gamma_{03}$ are the slopes associated with PPI factors and their interaction, $\gamma_{11}-\gamma_{13}$ are the slopes associated with the interaction between PPI factors and lag-error, and $u_{10}$ is the between-person residual. To reduce collinearity and facilitate parameter estimate interpretation, Fearless Dominance and Impulsive Antisociality were grand mean-centered (Enders & Tofighi, 2007). We experimented with alternative error structures (e.g., autocorrelation), but the unstructured model provided the best fit by having the lowest AIC and BIC values.

The results from this analysis are presented in the left column of Table 1. All tests are based on Type III sums of squares, as these sums of squares account for other effects in the model. Consistent with previous research (Rabbitt, 1966), there was a significant effect of lag-error, which indicated that the average participant slowed down 23.97 ms following an error. There was also a positive relation between Fearless Dominance and RT (regardless of lag-error). Of more importance and consistent with one of our predictions, there was a significant interaction between Fearless Dominance and lag-error. Inconsistent with our other prediction, analyses failed to reveal an interaction between Impulsive Antisociality and lag-error.

To follow-up the significant Fearless Dominance by lag-error interaction, we first plotted estimated PES scores for prototypical individuals low ($-1$ $SD$) and high ($+1$ $SD$) in Fearless Dominance using the MLM output (see Figure 1 top panel). Second, we conducted simple slopes analyses to examine the effect of lag-error for individuals high and low in Fearless Dominance (cf. Preacher et al., 2006). The results indicated that at high levels of Fearless Dominance, there was a significant medium sized effect of lag-error ($\gamma = 41.27$, $t = 4.32$, $p < .001$, $d = .52$), in that there was a slowing in RT following an error. However, at low levels of Fearless Dominance, the effect of lag-error was close to zero ($\gamma = 8.78$, $t = 1.14$, $p = .253$, $d = .10$). Taken together, these results indicate that individuals high in Fearless Dominance displayed PES, while individuals low in Fearless Dominance did not modulate their behavior following an error.[3]

Finally, there was a significant Fearless Dominance by Impulsive Antisociality by lag-error interaction, $\gamma = .08$, $t = 2.06$, $p = .039$. The follow-up tests showed that while no significant PES was observed among individuals high in Impulsive Antisociality and low in Fearless Dominance, $\gamma = -6.23$, $t = -.60$, $p = .309$, $d = -.07$, PES was significantly different from zero for individuals among the three other combinations (low on both factors, $\gamma = 22.69$, $t = 2.23$, $p = .025$, $d = .28$; high Fearless Dominance/low Impulsive Antisociality, $\gamma = 31.81$, $t = 2.35$, $p = .018$, $d = .39$; and high on both factors, $\gamma = 47.43$, $t = 3.41$, $p < .001$, $d = .58$). Together, the results suggest that high Fearless Dominance serves to increase behavioral adjustment following errors, including among those also high on Impulsive Antisociality.

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[3]Some previous research has found that when using the PPI-S, the Fearlessness subscale cross-loads on the Fearless Dominance and Impulsive Antisociality factors (Benning et al., 2005; Edens & McDermott, 2010). Hence, we also conducted the analyses without including the Fearlessness subscale in the former factor. The interaction between Fearless Dominance and lag-error was still significant, $\gamma = 1.88$, $t = 2.27$, $p = .023$. Moreover, the follow-up test still showed a significant PES at high levels of Fearless Dominance, $\gamma = 35.75$, $t = 4.55$, $p < .001$, $d = .44$. The only slight difference was a significant PES at low levels of Fearless Dominance, $\gamma = 13.30$, $t = 1.98$, $p = .047$, $d = .16$).

To show that the results were not exclusive to the unique variance of the factors, we also computed zero-order correlations between PES (average across trials) and the psychopathy factors. Zero-order correlations were similar to the multivariate analyses (Fearless Dominance: $r = .32$, $p = .015$; Impulsive Antisociality: $r = .05$, $p = .710$). One potential concern is that participants are more likely to make errors early in the task and have faster reaction times later in the task. To rule out this alternative, we added trial number as a covariate, which did not affect the results. We also ran separate models using overall accuracy and gender as covariates, which also did not affect the results.

## Discussion and Study 2

The results of Study 1 were consistent with one of our predictions. Individual differences in interpersonal-affective traits, as defined by the PPI, moderated the relation between accuracy on the previous trial and RT. More specifically, participants high in interpersonal-affective traits slowed down after making an error on the flanker task, while individuals low in interpersonal-affective traits displayed no modulation in response to errors. Inconsistent with our other hypothesis and Wilkowski and Robinson (2008), the only instance in which impulsive-antisocial traits were related to reduced PES was for individuals who were also low in interpersonal-affective traits. One possible reason for these divergent results is that our use of the PPI-S allowed us to better examine the unique variance of interpersonal-affective and impulsive-antisocial traits (as they are uncorrelated in the PPI-S), whereas Wilkowski and Robinson (2008) did not adjust for overlap between the two correlated factors in the LSRPS. However, it is unclear if the divergence of results is meaningful based on the findings from a single study. Although the three-way interaction suggests that being high on interpersonal-affective traits modulates RT slowing in impulsive-antisocial traits, replication is needed before this result is considered further, particularly because this interaction was not predicted.

Further, there are limitations to this study. Besides the need for replication, there was a high accuracy rate and limited number of overall trials; thus, PES was based on very few errors. Also, the correlation between Fearless Dominance and accuracy may suggest a speed-accuracy trade-off. Finally, the current study was conducted with a fairly homogeneous sample of college students, and it is not clear whether results would generalize to younger samples or to a wider array of the population residing in the community. Therefore, in Study 2 we attempted to determine whether the results would replicate in an archival data set of mid- to late adolescents (see Finy, Bresin, Korol, & Verona, in press)

The goal of the original project was to examine the interactions between psychosocial stress and the personality traits of negative emotionality and constraint on impulsivity, risk-taking, and cortisol reactivity in an adolescent sample (Finy et al., in press). Participants were first assigned to a stress or no-stress condition, and then they completed a go/no-go task to measure impulsivity. Saliva samples were obtained at four different time points to assess stress reactivity. Participants in this project also completed the Multidimensional Personality Questionnaire – Simplified-Wording Form (MPQ-SF; Javdani, Finy, & Verona, in press; Patrick, Kramer, Tellegen, Verona, & Kaemmer, 2013), which allowed us to estimate PPI

scores (cf. Benning et al., 2003), and we used the data from the go/no-go task to measure PES.

## Method

### Participants

Participants for Study 2 consisted of a subsample of a larger study examining cortisol reactivity and disinhibition in adolescents (Finy et al., in press). A total of 88 adolescent participants were recruited for the full study. However, in this report we only focus on the 43 participants (26 males) in the control (i.e., no-stress) condition.[4] Participants were recruited from a previous study of adolescents in our lab (see Verona, Javdani, & Sprague, 2011) and anew from flyers posted in the community for a study on decision-making. Youth with psychotic symptoms or a pervasive developmental disorder were excluded based on a phone screening completed with the parent or guardian. The mean age of these participants was 15.97 years (*SD* = 1.64, *range* 15–19), and the ethnic breakdown was 67% Caucasian, 13% Biracial, 9% Hispanic, 6% African-American, and 2% Asian. Though the majority of the sample (55%) had a household income over $60,000, 15% earned less than $30,000 a year, indicating a range of incomes.

### Procedure

After obtaining assent from the adolescent (or consent if the youth was over 18) and consent from the parent or guardian, participants completed the MPQ-SF and other measures not relevant to the current study. Following this, participants in the no-stress condition were asked to read neutral passages from the text of three popular adolescent books (e.g., *The Rescue*, by Kathryn Lasky). Each book was provided for a 5-minute interval to match the timing of tasks in the stress condition (see Finy et al., in press for more details). Participants were told that they could read at their leisure and would not be tested on the material in any way. Following the 15 minutes of reading, participants completed the go/no-go task. Families were paid $25 for their participation.

### Psychopathy Assessment

Psychopathy factor scores were calculated as estimated PPI scores based on the MPQ-SF, as done in previous work in adults (Benning et al., 2003; Benning, Patrick, Blonigen, Hicks, & Iacono, 2005) and adolescents (Blonigen, Hicks, Krueger, Patrick, & Iacono, 2006).[5] The version of the MPQ-SF used in this study consists of 155 items, which were modified from original versions of the MPQ to be at a 6th or 7th grade reading level. This version has been

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[4]A second set of 42 participants completed a youth version of the Trier Social Stress Test (Kirschbaum, Pirke, & Hellhammer, 1993) prior to completing the go/no-go task (See Finy et al., in press for more details). Because we were interested in replicating our results from Study 1, these participants were not used in the main analyses. When all participants were used, there was a significant Fearless Dominance by lag-error by stress group interaction, $\gamma = -18.84$, $t = -2.35$, $p = .019$. Follow-up tests indicated that the two-way Fearless Dominance by lag-error interaction was significant for the no-stress condition, $\gamma = 15.55$, $t = 2.86$, $p = .004$, but not for the stress condition, $\gamma = -3.20$, $t = -.54$, $p = .589$.

[5]We also calculated Fearless Dominance and Impulsive Antisociality based upon individual items (see Blonigen et al., 2005). The results were consistent with those using the beta weight method. The Fearless Dominance by lag-error interaction was marginally significant, $\gamma = 1.75$, $t = 1.94$, $p = .052$. The follow-up tests showed that at both high, $\gamma = 39.64$, $t = 7.54$, $p < .001$, and low levels, $\gamma = 25.87$, $t = 5.50$, $p < .001$, of Fearless Dominance, there was an effect of PES. Hence, the results are almost identical with both methods.

validated in young adults (Patrick et al., 2013) and adolescents (Javdani et al., in press). Participants were instructed to rate items as either true or false based on which choice best described them. Following the recommendations of Benning et al. (2003), Fearless Dominance scores were estimated based on a composite of the social potency ($\alpha = .78$), stress reaction ($\alpha = .81$), and harm avoidance ($\alpha = .75$) subscales. Impulsive Antisociality scores were based on a composite of the aggression ($\alpha = .80$), traditionalism ($\alpha = .66$), control ($\alpha = .78$), alienation ($\alpha = .82$), and social closeness ($\alpha = .80$) subscales. Relevant subscales were $Z$-scored and multiplied by the standardized beta weights reported by Benning et al. (2003) before being summed to create the factor scores. The two factors displayed a small non-significant correlation in this sample ($r = -.12$, $p = .439$). Though there are likely disadvantages to estimating PPI scores rather than measuring them directly, particularly in adolescents where there has been less research on estimated PPI scores, we felt it was justified given that this study was a replication and could reveal whether the results from Study 1 were generalizable to community adolescents.

### Go/No-go Task

Similar to the flanker task in Study 1, the go/no-go task required participants to respond to one stimulus (*X*) but withhold responding to a rare (occurring on 20% of trials) non-target stimulus (*K*). However, in Study 2 there were no flanking distracters presented. Nonetheless, the basic parameters involving a main focus on target stimuli and a dominant response set were both present. After a practice block, participants completed four blocks of 76 trials. Within each block, stimuli were presented in a random order for 250 ms, and then participants had 1000 ms to respond. The inter-trial interval varied from trial to trial (1500, 2000, 2500 ms). No feedback was provided for incorrect responses. Participants were instructed to respond as quickly and as accurately as possible.

We again discarded trials for three reasons: they were abnormally fast ($< 150$ ms) or slow ($> 1000$ ms), no response was made, or an error was made, which led us to discarding 5.59% of trials. The overall accuracy rate was 94.57%, and participants made an average of 14.36 (*SD* = 7.74, *Max* = 36) errors and 14.16 (*SD* = 6.60, *Max* = 13) lag-errors during the task, allowing for a larger number of trials available for analysis relative to Study 1. In this study, Fearless Dominance ($r = -.05$, $p = .771$) and Impulsive Antisociality ($r = -.17$, $p = .259$) were not related to accuracy. There was a small to medium, but non-significant, correlation between accuracy and PES ($r = .20$, $p = .190$).

## Results

MLM models were similar to those in Study 1. The results are displayed in the middle column of Table 1. The effect of lag-error was significant and indicated that on average participants slowed down 31 ms following an error. Consistent with Study 1 and one of our predictions, the interaction between Fearless Dominance and lag-error was significant. Inconsistent with our other prediction, none of the effects involving Impulsive Antisociality were significant, and the three-way interaction was not replicated (see Table 1 for parameter estimates). To follow-up the interaction involving Fearless Dominance, we again plotted estimated means (see Figure 1, bottom panel) and calculated simple slopes tests. As in Study

1, at high levels of Fearless Dominance, there was a significant medium sized effect of lag-error ($\gamma = 40.65$, $t = 8.91$, $p < .001$, $d = .51$), indicating PES. At low levels of Fearless Dominance, the effect of lag-error was also significant ($\gamma = 22.60$, $t = 4.74$, $p < .001$, $d = .28$), but small according to Cohen's (1992) standards. The significant interaction indicates that PES was significantly smaller for low versus high scorers (22 ms versus 40 ms, respectively). Adjusting for trial number, overall accuracy, gender, and age did not affect these results. As in Study 1, the zero-order correlations were similar to the results of the multivariate tests (Fearless Dominance: $r = .23$, $p = .124$; Impulsive Antisociality: $r = .09$, $p = .530$), though in this study it appears that adjusting for the overlap between the factors increased the effect for Fearless Dominance (and decreased its $p$-value to below significance level).

## Discussion and Study 3

The results for Study 2 were in line with those of Study 1. Adolescents with high (versus low) levels of interpersonal-affective traits, as defined by the PPI, displayed greater RT slowing after errors. In Study 2, adolescents with lower levels of interpersonal-affective traits also displayed PES, but to a lesser degree than individuals high in interpersonal-affective traits. Also, in this study, there was no evidence of a speed-accuracy trade-off, in that accuracy was not correlated with interpersonal-affective traits. It is noteworthy that these results were replicated in an adolescent sample performing a slightly different task than in Study 1. Further, PPI scores were estimated and not measured directly, indicating that these results are likely robust across samples, tasks, and psychopathy measurements. None of the effects involving impulsive-antisocial traits were significant, and we did not replicate the significant three-way interaction from Study 1 in this study.

In spite of the consistency of the results for the interpersonal-affective traits across the first two studies, there were unanswered questions, which led us to conduct data analyses for a third study. First, the participants recruited for Studies 1 and 2 involved college students and adolescents in the community who typically score on the lower range of psychopathic traits, especially the more malignant forms. Thus, we deemed it important to examine the generalizability of these results to participants scoring at the higher range of these psychopathic traits. Second, the Fearless Dominance scale has been criticized for primarily indexing traits related to positive adjustment (e.g., low fear, social potency; Miller & Lynam, 2012). In contrast, PCL-based measures assess interpersonal-affective traits of a more maladaptive variety (e.g., deceitfulness, conning, lack of empathy). Given this distinction, we considered it important to generalize these results to forensic-based assessments of psychopathy as a way of clarifying constituent traits that account for relations to PES. In particular, if Factor 1 relates to PES in a similar way as PPI Fearless Dominance, this would suggest that what these two psychopathy-related constructs share (and not what is different between them) may explain the higher cognitive control observed. Third, Studies 1 and 2 were focused on behavioral adjustments following errors and thus do not identify differences in cognitive processing involved in error monitoring, specifically the ERN. Therefore, Study 3 involved a clinical-forensic sample of individuals who completed the standard letter-flanker task, while we measured both ERN and PES. Consequently, Study

3 allowed us to fully test our predictions concerning both monitoring (ERN) and adjusting to errors (PES).

The goal of the original study was to examine cognition-emotion interactions among community dwelling offenders differing on scores of psychopathy and antisocial personality disorder (Verona, Sprague, & Sadeh, 2012). We recorded event-related brain potentials during an emotional-linguistic go/no-go task to challenge both emotional processing and inhibitory control syndromes. Participants high on psychopathy showed decreased negative emotional processing across inhibitory control conditions (go and no-go), whereas participants with only antisocial personality disorder showed enhanced negative emotional processing even under inhibitory control conditions. Participants in this study also completed a letter-flanker task, the results of which were not reported in the original publication and form the basis for Study 3.

## Method

### Participants

Seventy-one participants (49 males) were recruited from a larger assessment study (see Schoenleber, Sadeh, & Verona, 2011) targeting individuals with criminal histories. Participants were selected for inclusion in the current study based on scores of psychopathy and antisocial personality disorder. Although in the original study, we analyzed results for groups of individuals high on psychopathy, antisocial personality disorder, or neither, we had recruited individuals scoring across the whole range of psychopathy total scores to participate in the laboratory experiment (see more details below). Participants were recruited from parole and probation (39.43% of this sample), substance use treatment centers (9.86 % of this sample), local jails (8.45% of this sample), and newspaper ads (42.26% of this sample). The mean age of the laboratory sample was 33 years old (*SD* = 9.08, *range* 19–53). The ethnic breakdown was 49% Caucasian, 42% African-American, 2% Native American, 2% mixed ethnicity, 1% Hispanic, and 1% other. The majority of the sample (59%) earned less than $15,000 annual income, and only 5.63% earned more than $75,000. Data from a subsample of these participants from another task were reported in Verona et al. (2012). However, data from the flanker task have not yet been published.

### Psychopathy Assessment

Psychopathy was assessed using the Psychopathy Checklist: Screening Version (PCL: SV; Hart et al., 1999). Ratings on the PCL: SV were made based on a semi-structured interview and a review of public criminal records. Each of the 12 items was rated on a 3-point scale (0 = *not at all characteristic*, 2 = *extremely characteristic*). Interviews were conducted by trained doctoral students supervised by a Ph.D.-level psychologist. Secondary ratings were available for 112 of the participants from the full assessment sample (*N* = 493). The interclass correlations (ICC) were high for Factor 1 (ICC = .95) and Factor 2 (ICC = .94), indicating adequate inter-rater reliability.

As mentioned previously, participants who were recruited for the laboratory portion of the study were low scorers (below 12), middle scorers (between 12 and 18), and high scorers on

the PCL: SV to adequately represent the whole range of psychopathy total scores (range: 1–23; possible range of scores 0–24). Fifty-one percent had a diagnosis of antisocial personality disorder. We focused our analyses on the two PCL: SV factors: Factor 1 (*skew =.41; kurtosis = −.98*), which represents an antagonistic affective-interpersonal style involving conning, deceitfulness, and lack of empathy; and Factor 2 (*skew =−.32; kurtosis = −.39*), which represents impulsive-antisocial traits, as per previous work (Harpur et al., 1989; Hicks & Patrick, 2006; Patrick & Zempolich, 1998; Verona et al., 2001). We also examined correlations with the four-facet model in supplementary analyses, for the sake of thoroughness. The four-facet model breaks Factor 1 into interpersonal and affective facets and Factor 2 into impulsive-lifestyle and antisocial facets. As is typical with PCL-based measures (Hare, 2003), the two main factors were significantly correlated ($r = .53$, $p < .001$) with a large effect size.

**Flanker Task**

Similar to the flanker task in Study 1, participants were required to respond to a central target among flanking distracters (Eriksen & Eriksen, 1974; Hall et al., 2007). However, in this task, the two letters (*S* and *H*) each served as targets on different trials. Targets were displayed with congruent flankers for half the trials and incongruent flankers on the other half. Each target type required a unique response on the keyboard (i.e., right or left shift keys), which was counterbalanced across blocks. To increase task complexity, a non-target (*X*) was displayed (with congruent or incongruent flankers) on 16% of trials, and participants were instructed to withhold responses to these non-targets (see Hall et al., 2007). After a practice block, participants completed six blocks of 100 trials. At the end of each block, participants received feedback about their performance in that block. However, consistent with Studies 1 and 2, no feedback was given at the trial level. For each trial, the stimulus array was displayed for 150 ms, and participants then had up to 1400 ms to respond. The inter-trial interval varied between 1500, 2000, and 2500 ms. Participants were told to respond as quickly and as accurately as possible. Participants also completed an emotional go/no-go task (see Verona et al., 2012) in a counterbalanced order. Task order did not affect any of the results reported below and thus is not discussed further.

As in Studies 1 and 2, we discarded trials that were too fast (< 150 ms) or too slow (> 1000 ms), trials where no response was made, and trials in which an error of commission was made on the current trial (i.e., trial$_n$ is an error). We also discarded blocks where the accuracy rate was less than 50% based on the assumption that the correct response mappings were not being used. In total, 7.66% of trials were discarded. The accuracy rate ($M = 88.22\%$) was comparable to other studies using similar paradigms (Hall et al., 2007; Munro et al., 2007). Participants made an average of 23.70 errors ($SD = 22.54$, $Max = 126$) and 18.44 ($SD = 19.16$, $Max = 115$) lag-errors. As in Study 2, accuracy rate was not correlated to Factor 1 ($r = .00$, $p = .988$) or Factor 2 ($r = .03$, $p = .741$), with both having an effect size close to zero. However, in this study the correlation between accuracy and PES was significant and small to medium in size ($r = .25$, $p = .027$), suggesting that individuals who were more accurate tended to slow down more after errors, regardless of level of psychopathy.

### Error-Related Negativity

Event-related potentials were measured with a stretch lycra electrode cap (Electrocap, Eaton, OH) using the 10–20 international system. Analog signals were digitized at 2000 Hz with a .15–200 HZ bandpass filter using Neuroscan 2 amplifiers (Compumedics, Charlotte, NC). The left mastoid served as the online reference electrode, but off-line the data were re-referenced to the average of the mastoids (Miller, Lutzenberger, & Elbert, 1991). Vertical and horizontal eye movements were recorded for eye blink correction and detection of artifacts. Data reduction was completed using the PhysBox add-on to EEGLAB in Matlab (Curtin, 2011; Delorme & Makeig, 2004). First, the data were filtered using a low pass (30 Hz) Butterworth filter. Second, a blink correction was applied. Third, trials that had deflections greater than 75 µV in absolute value were discarded. Waveforms were then averaged by trial accuracy. The response locked ERN was defined as the most negative peak in the window from the response until 250 ms after the response, relative to the baseline, which consisted of the activity between 250 and 50 ms before the response. Sixteen participants had data that was not used due to either having too few blinks to apply the correction procedure ($n = 11$) or excessive artifacts ($n = 5$), leaving 55 participants for ERN analyses.

## Results

### Post-error Slowing: Replication

First, we examined whether the results from Studies 1 and 2 would replicate in this clinical-forensic sample. In this analysis, we used the same models as in the previous studies, with PCL: SV Factor 1 and Factor 2 representing the interpersonal-affective and impulsive-antisocial traits of psychopathy, respectively. The results are displayed in the right panel of Table 1. Consistent with Studies 1 and 2, there was a significant main effect of lag-error indicating that, overall, participants slowed down following errors. More importantly, the interaction between Factor 1 and lag-error was significant. The follow-up tests indicated that at high levels of Factor 1, there was significant PES ($\gamma = 46.77$, $t = 6.97$, $p < .001$, $d = .33$). There was also an effect of lag-error at low levels of Factor 1 ($\gamma = 18.78$, $t = 2.77$, $p = .005$, $d = .13$), but this effect was smaller in magnitude (see Figure 3). It is worth noting that Factor 1 was not correlated with accuracy, suggesting no evidence of a speed-accuracy trade-off. As in Studies 1 and 2, there was no significant Factor 2 by lag-error interaction, and like Study 2, no significant three-way interaction. Adjusting for age, gender, recruitment site, ethnicity, overall-accuracy, and trial number did not affect any of these results. The zero-order correlations between PES and Factor 1 ($r = .13$, $p = .227$) and Factor 2 ($r = -.07$, $p = .522$) were similar to the multivariate analysis, though somewhat weaker in magnitude and not significant, indicating that adjusting for the overlap strengthens the correlation for Factor 1 (cooperative suppressor effects; Hicks & Patrick, 2006).

To better understand which facets may drive the effect for Factor 1, we explored correlations between PES and the four-facet model, using partial correlations for each facet with the others partialled out. These results showed that the relation between PES and Factor 1 was driven by the interpersonal facet ($r = .22$, $p = .074$), and not the affective facet ($r = -.05$, $p$

= .651). Both Factor 2 facets were negatively but not significantly related to PES (lifestyle $r$ = −.07, antisocial $r$ = −.07).

## Error-related Negativity: Expansion

We tested whether the two factors of the PCL: SV were related to ERN using a general linear model. In this model, Factor 1 and Factor 2 scores (mean-centered) and their interaction were between-subjects continuous variables, and accuracy of the current trial (correct, incorrect) was a categorical within-subjects variable. We focused on the Cz site, as this was where the differentiation between accurate and inaccurate trials was numerically maximal. This site has also been the focus of previous ERN research in psychopathy (Brazil et al., 2011; Munro et al., 2007). There was a significant effect of accuracy, $F(1, 53) =$ 37.86, $p < .001$, partial $\eta^2 = .41$. As would be expected, incorrect trials ($M = −3.00$, $SD$ =4.15) produced a more negative amplitude than correct trials ($M = .05$, $SD$ =2.23). There were no significant main effects of the psychopathy factors, and the two-way interaction between the factors was not significant ($p$'s > .31). Consistent with our prediction, there was a significant interaction between Factor 1 and accuracy, $F(1, 53) = 7.67$, $p = .007$, partial $\eta^2$ = .12. The interaction between Factor 2 and accuracy approached significance, $F(1, 53) =$ 3.05, $p = .086$, partial $\eta^2 = .05$. The three-way interaction was not significant ($p = .830$).

To follow-up the significant Factor 1 by accuracy interaction, we created a difference score that represented the within-person effect (Judd, McClelland, & Smith, 1996) by subtracting correct trials from incorrect trials, so that more negative values indicated a larger ERN relative to correct trials. We then used this variable as the dependent measure for two multiple regressions. In each regression, Factor 1 was modified to reflect high (+ 1 $SD$) or low (−1 $SD$) levels. Factor 2 and the interaction between the modified Factor 1 and Factor 2 were also added as covariates. In these models, the significance of the intercept indicates an effect of accuracy for that level of Factor 1 (Judd et al., 1996). Estimated means are displayed in the top panel of Figure 3. At high levels of Factor 1, the intercept was significant, $b = −5.05$, $t = −5.69$, $p < .001$, $d = −1.31$, suggesting that there was differentiation between errors and correct trials at high levels of Factor 1. Similarly, at low levels of Factor 1, the intercept was significant but smaller in magnitude than for high levels of Factor 1, $b = −1.79$, $t = −2.41$, $p = .019$, $d = −.46$. Taken together, these results suggest that at all levels of Factor 1, individuals are attending to errors, but individuals higher in Factor 1 traits do this to a greater extent than individuals low in Factor 1.

We also followed up the marginal Factor 2 by accuracy interaction due to our a priori hypotheses. Similar to other research (Hall et al., 2007), the differentiation between correct and error trials was larger at low levels, $b = −7.04$, $t = −2.25$, $p = .002$, $d = −1.83$, compared to high levels of Factor 2, $b = −4.97$, $t = −4.36$, $p < .001$, $d = −1.29$, and both were significantly different from zero (see the bottom panel of Figure 3). Given that the interaction was marginally significant, these results could be interpreted as indicating some evidence that individuals high in Factor 2 are less aware of or reactive to their errors. For completeness, the zero-order correlations between the factors and ERN were $r = −.27$, $p = .$ 035 for Factor 1, and $r = .04$, $p = .714$, indicating a suppressor effect for Factor 2.

We also explored the correlations between ERN and the four-facet indices of the PCL: SV (with the other facets partialled out). In terms of Factor 1, the relation with ERN was similar for the interpersonal facet ($r = -.21$, $p = .118$) and the affective facet ($r = -.12$, $p = .379$), although slightly stronger in the former. In terms of Factor 2, the facet correlations showed that the effect was specific to the lifestyle facet ($r = .32$, $p = .018$), as the antisocial facet was not significantly related to ERN ($r = -.09$, $p = .487$).

### Post-error Slowing: Meta-analysis

Given that each study had relatively modest sample sizes, we used meta-analytic techniques to combine the effect size for PES (but not ERN) across the 3 samples to provide a more powerful test of our hypothesis regarding PES. We examined Cohen's *d*, calculated as the RT following a correct trial subtracted from RT following an error, divided by pooled standard deviation, as estimated by the square root of the model-based within-subject variance. This was done at high and low levels of interpersonal-affective traits (acknowledging that these traits were operationalized differently across the 3 studies), which were then compared using confidence intervals. Standard errors for the effect size were calculated based on the recommendations of Dunlap, Cortina, Vaslow, and Burke (1996), taking into account the correlation between the repeated measures. The weighted effect size and 95% confidence intervals were calculated using the metafor package in R (R development Core Team, 2010; Viechtbauer, 2010) based on a random effects model.

At high interpersonal-affective traits, the effect size for PES was medium based upon Cohen's (1992) standards and significantly different from zero ($d = .57$, $Z = 8.69$, $p < .001$), and there was no significant heterogeneity in the effect across studies, $Q(2) = .99$, $p = .608$. At low interpersonal-affective traits, the effect was small in size and marginally different from zero ($d = .13$, $Z = 1.69$, $p = .089$), and the test for heterogeneity was not significant, $Q(2) = 3.45$, $p = .178$. Critically, the confidence intervals for the effect size at high and low levels of interpersonal-affective traits did not overlap (low interpersonal-affective traits: [−.02, .28], high interpersonal-affective traits: [.44, .70]), suggesting a robust difference in the effect of PES for different levels of interpersonal-affective traits across studies.

## Discussion

Consistent with our predictions, the results of Study 3 showed that individuals high in interpersonal-affective traits, as defined by the PCL: SV, displayed enhanced adjusting to (consistent with Studies 1 and 2) and monitoring of errors. This study extended the results of Studies 1 and 2 to a clinical-forensic sample using a widely validated interview-based assessment of psychopathy, as well as by using two different indices of response monitoring and/or adjusting (i.e., PES and ERN). It was important to show these results in such a sample using a PCL-based assessment given that the correlation between Factor 1 and Fearless Dominance is small (Miller & Lynam, 2012), likely because they index very different types of interpersonal-affective traits. Our results show that the unique variance of Factor 1, which is characterized by antisocial forms of interpersonal-affective traits (e.g., conning, deceitfulness), has the same relation with PES as Fearless Dominance. Thus, our study highlights an interesting point of convergence between these different

operationalizations of psychopathic traits across instruments. Counter to the null results found for the impulsive aspects of psychopathy and PES in Studies 1 and 2, Study 3 found an effect, albeit marginal, relating impulsive-antisocial traits and ERN. Consistent with research on similar traits (e.g., externalizing; Hall et al., 2007), individuals high in impulsive-antisocial traits displayed reduced ERN relative to individuals low in impulsive-antisocial traits.

## General Discussion

The goal of the current set of studies was to examine the relations of the two-factor model of psychopathy with monitoring and adjusting to errors, as an attempt to expand upon previous research seeking to understand the trait dimensions associated with regulation problems in psychopathy. Across studies, we supported our prediction that individuals high in interpersonal-affective traits would be better able to adjust their behavior following errors (Studies 1, 2, and 3) and to detect errors (Study 3). However, we found limited support for our prediction that individuals high in impulsive-antisocial traits would have deficits in monitoring and adjusting to errors. Together, these highly novel findings are likely to add to our understanding of not only the unique aspects of psychopathy, but psychopathy and cognitive control more generally.

The results for interpersonal-affective traits and PES were very robust. In spite of the differences among samples, assessment tools, and tasks, individuals high in interpersonal-affective traits showed a very similar RT slowing following errors across the studies. This is particularly interesting when contrasting Studies 1 and 2 with Study 3. Though the Fearless Dominance subscale of the PPI and Factor 1 of the PCL both assess interpersonal-affective traits, they are operationalized quite differently with presumably different nomological networks. Fearless Dominance assesses a more adaptive variant of interpersonal-affective traits (e.g., assertiveness, persuasiveness, lack of fear), whereas Factor 1 taps into more maladaptive forms of interpersonal-affective traits (e.g., deceitfulness, manipulation, lack of guilt). The replication across measures clearly indicates that PES is a cognitive function that overlaps across different operationalizations of psychopathic traits.

It may be useful to examine the results of these studies through the lens of the triarchic theory of psychopathy (Patrick et al., 2009), which suggests that psychopathy is a composite of boldness (low stress reactivity, high social efficacy), meanness (lack of empathy, exploitativeness), and disinhibition (i.e., lack of planfulness, inability to control urges). It has been suggested that Fearless Dominance is a relatively pure measure of boldness, whereas Factor 1 is predominantly a measure of meanness, with boldness being weakly represented mostly by the interpersonal facet (Patrick et al., 2009; Patrick, Venables, & Drislane 2013). The consistency of our PES results across studies may suggest that they are a function of boldness. This is further supported by the partial correlations in Study 3 involving the four-facet model, which showed that the interpersonal facet was driving the PES results. However, the ERN results showed similar correlations between the interpersonal and affective facets, possibly suggesting that meanness might be driving these results as well. The facet level analyses suggest that the ERN Factor 2 results are a function of disinhibition (impulsive lifestyle and not antisocial aspects of psychopathy).

The PES and ERN results are in line with previous research, suggesting that interpersonal-affective traits (or at least the unique variance when partialling impulsive-antisocial traits) are not associated with gross deficits in cognitive functioning and are in fact related to better cognitive functioning in some areas (Neumann & Hare, 2008; Sadeh & Verona, 2008). The fact that these advantages only occur in certain contexts (e.g., following an error) is also consistent with other cognitive theories of psychopathy (e.g., Kosson & Harpur, 1997; Newman & Baskin-Sommers, 2012). Our results add to this literature by indicating specific processes that may also play a role in the relation between certain aspects of psychopathy and general cognitive functioning (e.g., intelligence).

There are many possible explanations for why the PES results for impulsive-antisocial traits of psychopathy were not consistent with our predictions and previous work (Wilkowski & Robinson, 2008). Previous research has generally found deficits in executive functioning related to impulsive-antisocial traits in clinical-forensic samples (Kiehl, Bates, Laurens, Hare, & Liddle, 2006; Ogilvie et al., 2011), but not college or community samples as used in Studies 1 and 2 (e.g., Carlson & Thái, 2010; Sellbom & Verona, 2007). However, it is not fully clear why there was no association between PES and impulsive-antisocial traits in Study 3, which used a clinical-forensic sample, suggesting that further research is necessary.

Our findings with PES and impulsive-antisocial traits are inconsistent with two other sources of data. First, Wilkowski and Robinson (2008) found that the impulsive-antisocial traits, as defined by the LSRPS, were related to reduced PES. There are some key methodological differences of theoretical importance between these two sets of studies that might explain these differences. For instance, we did not provide feedback following errors at the trial level, whereas Wilkowski and Robinson (2008) did. Work by Newman and colleagues (1987) shows that psychopathy is related to perseverative errors in response to feedback, which might explain this difference. Another key difference involves the stimuli used. In contrast to our simple stimuli, Wilkowski and Robinson (2008) used words that belonged to salient categories, some of which involved affective words. It is possible that the inclusion of affective words in their studies may have exacerbated regulation deficits among those high in the impulsive aspects of psychopathy (e.g., Verona et al., 2012).

Second, there is work by Newman and colleagues showing that individuals high versus low on psychopathy tend to display deficits in PES following punishing feedback (Newman et al., 1987; Newman, Patterson, Howland, & Nichols, 1990). However, this work does not parse psychopathy into different factors, making it difficult to know whether interpersonal-affective traits, impulsive-antisocial traits, or the combination is driving the results. It is also worth noting that the reduced PES shown by Newman and colleagues is only seen when there is a need to switch between response sets (e.g., reward and punishment are part of the task; Newman et al., 1990). It could be argued that our task only required one response set, which may explain the facilitated performance for individuals high in interpersonal-affective traits and the absence of effects for those high in impulsive-antisocial traits (Newman & Baskin-Sommers, 2012; Patterson & Newman, 1993).

Nonetheless, the marginal results for Factor 2 and ERN do fit with previous research showing that individuals with differing forms of externalizing behaviors, similar to

impulsive-antisocial traits, have a reduced ability to detect errors (Franken et al., 2007; Hall et al., 2007). In that regard, it is not uncommon for psychophysiology studies to find relations among ERP components and not behavioral measures (e.g., Hall et al., 2007; Kiehl et al., 2006; Weinberg et al., 2011). One common interpretation of this discrepancy is that psychophysiological measures may reveal deficits that individuals can compensate for behaviorally when performing simple tasks (Miller, 1996). Indeed, intact behavioral performance in the face of alterations in cognitive processing are commonly reported in ERP studies of psychopathology (e.g., Hall et al., 2007; Kiehl et al., 2006; Verona et al., 2012)

## Error Monitoring and Adjustment in Individuals High in Psychopathy

Even though our approach was to decompose psychopathy into two factors, it is important to understand our findings in the context of explaining the behavior of individuals high on both factors. Previous theory suggests that the ability to monitor and adjust to errors is necessary for the pursuit of goals (Carver & Scheier, 1990; Robinson et al., 2010). In the context of high impulsive-antisocial traits (antisocial lifestyle), individuals high in interpersonal-affective traits may use their ability to monitor and adjust to errors to pursue antisocial goals. For example, monitoring and adjusting to errors may be useful in conning/manipulating others, in that it requires the presentation of a coherent story to ensure deception (i.e., monitor for errors) and adjustments to remove the doubt in the victim (i.e., adjust to errors). Similarly, monitoring and adjusting to errors may be advantageous in premeditated crimes (e.g., instrumental aggression), which are sometimes correlated with the interpersonal-affective traits (Cornell et al., 1996; Patrick & Zempolich, 1998; though see Camp, Skeem, Barchard, Lilenfeld, & Poythress, 2013). Future research may wish to examine whether monitoring and adjusting to errors are related to certain types of antisocial behavior among individuals high in interpersonal-affective traits.

Monitoring and adjusting to errors may also manifest in total psychopathy (i.e., individuals high on both factors) as cognitive processes that hide the impulsive-antisocial traits, similar to Cleckley's (1976) "mask of sanity." Previous research in other areas has found that individual differences in monitoring and adjusting to errors (i.e., higher versus lower) are related to some forms of adjustment including lower levels of depression, higher levels of well-being, greater expressions of happiness, and better academic performance (Hirsh & Inzlicht, 2009; Robinson, 2007). Thus, monitoring and adjusting to errors might be processes that lead to the outward appearance of adjustment (e.g., an adept social presence, "good" intelligence, the limited experience of anxiety) that, at least at first glance, obscure the maladaptive or destructive aspects of psychopathy (lack of empathy, recklessness, deceit). Hence, among people high in psychopathic traits, monitoring and adjusting to errors are cognitive processes that partly represent the psychopathic ability to display a mentally healthy outward appearance. Conversely, in the absence of impulsive-antisocial traits, monitoring and adjusting to errors may lead individuals high in interpersonal-affective traits to be well adjusted (cf. Lilienfeld et al., 2012; Patrick et al., 2009).

Given that we examined the two main factors of psychopathy individually and did not consistently find significant interactions between the factors, these interpretations are purely speculative. However, this approach fits with theory and research suggesting unique

underlying diatheses associated with distinct paths to psychopathy (e.g., Karpman, 1941; Lykken 1995; Patrick et al., 2009). It is possible that monitoring and adjusting to errors may reflect differential mechanisms associated with different pathways by which individuals may look phenotypically psychopathic. For instance, within the larger group of persons high on a measure of psychopathy, there may be those who followed the interpersonal-affective trajectory (and thus would show enhanced PES and ERN), while others would show a more externalizing trajectory (with no enhancements in PES and reduced ERN). This interpretation is consistent with cluster analyses revealing at least two distinct temperamental and behavioral profiles among individuals showing high levels of PCL-R psychopathy (e.g., Hicks et al., 2004). However, longitudinal studies are required to fully understand how our results help understand divergent etiological pathways.

## Limitations and Strengths

The results of these studies should be interpreted within the bounds of their limitations. First, the sample size for each study was relatively modest. Though the size is consistent with recommendations for MLM (van der Leeden & Busing, 1994), the ability to recruit individuals high in psychopathy is enhanced with larger samples, particularly for non-incarcerated populations. Nonetheless, our use of a meta-analytic approach to estimate the effect size across the three studies enhances confidence in the robustness of our results for PES. Second, these studies only considered affectively neutral stimuli, and it is unclear whether these results would generalize to affective contexts. Finally, we used the two-factor model of psychopathy based primarily on research on the PCL and PPI. There are multiple models of psychopathy that may further clarify distinct processing deficiencies in psychopathy (Cooke & Michie, 2001; Patrick et al., 2009). For instance, new assessment tools like the Triarchic Psychopathy Measure (Drislane, Patrick, & Arsal, in press) or the Comprehensive Assessment of Psychopathic Personality (Hoff, Rypdal, Mykletun, & Cooke, 2012) may deliver a different set of results or more specification of traits linked to enhanced or deficient cognitive control. Therefore, until more studies are conducted, it is unclear how these results might generalize to other models of psychopathy.

Our set of studies also has a number of strengths worth noting. Given the current focus on replication in psychological science (e.g., Pashler & Harris, 2012), it is notable that we were able to replicate the relation between interpersonal-affective traits and PES in three independent samples. Moreover, the meta-analytic results provide additional support to the robustness of our findings. The use of very different samples (e.g., adolescents versus adults) and assessment methods of psychopathy (e.g., PPI versus PCL-R) allows for some generalizability of our findings. This is particularly noteworthy given the small correlation between Fearless Dominance and Factor 1 (e.g., Marcus et al., 2013). We also used a statistical technique (MLM) in our examination of PES that has advantages over traditional methods (Judd et al., 2012). In summary, these studies help establish differential relations between interpersonal-affective and impulsive-antisocial traits and monitoring and adjusting to errors. Future research should seek to understand the boundary conditions of these effects and further clarify how these relations may help us understand their role in psychopathy.

## Acknowledgments

## References

Baskin-Sommers A, Zeier JD, Newman JP. Self-reported attentional control differentiates the major factors of psychopathy. Personality and Individual Differences. 2009; 47:626–630. [PubMed: 19997525]

Benning SD, Patrick CJ, Blonigen DM, Hicks BM, Iacono WG. Estimating facets of psychopathy from normal personality traits: A step toward community epidemiological investigations. Assessment. 2005; 12:3–18. [PubMed: 15695739]

Benning SD, Patrick CJ, Hicks BM, Blonigen DM, Krueger RF. Factor structure of the Psychopathic Personality Inventory: Validity and implications. Psychological Assessment. 2003; 15:340–350. [PubMed: 14593834]

Benning SD, Patrick CJ, Iacono WG. Psychopathy, startle blink modulation, and electrodermal reactivity in twin men. Psychophysiology. 2005; 42:753–62. [PubMed: 16364071]

Bernat EM, Nelson LD, Steele VR, Gehring WJ, Patrick CJ. Externalizing psychopathology, and gain-loss feedback in a simulated gambling task: Dissociable components of brain responses revealed by time-frequency analysis. Journal of Abnormal Psychology. 2011; 120:352–364. [PubMed: 21319875]

Blonigen DM, Hicks BM, Krueger RF, Patrick CJ, Iacono WG. Continuity and change in psychopathic traits as measured via normal-range personality: A longitudinal-biometric study. Journal of Abnormal Psychology. 2006; 115:85–95. [PubMed: 16492099]

Botvinick MM, Braver TS, Barch DM, Carter CS, Cohen JD. Conflict monitoring and cognitive control. Psychological Review. 2001; 108:624–652. [PubMed: 11488380]

Brazil IA, de Bruijn ERA, Bulten BH, von Borries AKL, van Lankveld JJDM, Buitelaar JK, Verkes RJ. Early and late components of error monitoring in violent offenders with psychopathy. Biological Psychiatry. 2009; 65:137–143. [PubMed: 18814860]

Brazil IA, Mars RB, Bulten BH, Buitelaar JK, Verkes RJ, de Bruijn. A neurophysiological dissociation between monitoring one's own and other's actions in psychopathy. Biological Psychiatry. 2011; 69:693–699. [PubMed: 21215385]

Camp JP, Skeem JL, Barchard K, Lilienfeld SO, Poythress NG. Psychopathic predators? Getting specific about the relation between psychopathy and violence. Journal of Consulting and Clinical Psychology. 2013; 81:467–480. [PubMed: 23316742]

Carlson SR, Thái S. ERPs on a continuous performance task and self-reported psychopathic traits: P3 and CNV augmentation are associated with fearless dominance. Biological Psychology. 2010; 85:318–330. [PubMed: 20723576]

Carver CS, Scheier MF. Origins and functions of positive and negative affect: A control-process view. Psychological Review. 1990; 97:19–35.

Cleckley, H. The mask of sanity. 5. St Louis, MO: Mosby; 1976.

Cohen J. A power primer. Psychological Bulletin. 1992; 112:155–159. [PubMed: 19565683]

Cooke DJ, Michie C. Refining the construct of psychopathy: Towards a hierarchical model. Psychological Assessment. 2001; 13:171–188. [PubMed: 11433793]

Cornell DG, Warren J, Hawk G, Stafford E, Oram G, Pine D. Psychopathy in instrumental and reactive violent offenders. Journal of Consulting and Clinical Psychology. 1996; 64:783–790. [PubMed: 8803369]

Curtin, JJ. PhysBox: The Psychophysiology toolbox. An open source toolbox for psychophysiological data reduction within EEGLab. 2011. http://dionysus.psych.wisc.edu/PhysBox.htm

Delorme A, Makeig S. EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics. Journal of Neuroscience Methods. 2004; 134:9–21. [PubMed: 15102499]

Drislane LE, Patrick CJ, Arsal G. Clarifying the content coverage of differing psychopathy inventories through reference to the triarchic psychopathy measure. Psychological Assessment. (in press).

Dunlap WP, Cortina JM, Vaslow JB, Burke MJ. Meta-analysis of experiments with matched groups or repeated measures designs. Psychological Methods. 1996; 1:170–177.

Dutilh G, Vandekerckhove J, Forstmann BU, Keuleers E, Brysbaert M, Wagenmakers E. Testing theories of post-error slowing. Attention, Perceptions, and Psychophysics. 2012; 74:454–465.

Edens JF, McDermott BE. Examining the construct validity of the Psychopathic Personality Inventory–Revised: Preferential correlates of fearless dominance and self-centered impulsivity. Psychological Assessment. 2010; 22:32–42. [PubMed: 20230149]

Enders CK, Tofighi D. Centering predictor variables in cross-sectional multilevel models: A new look at an old issue. Psychological Methods. 2008; 12:121–138. [PubMed: 17563168]

Eriksen BA, Eriksen CW. Effects of noise letters upon identification of a target letter in a nonsearch task. Perception and Psychophysics. 1974; 16:143–149.

Falkenbach DM, Stern SB, Creevy C. Psychopathy variants: Empirical evidence supporting a subtyping model in a community sample. Personality Disorders: Theory, Research, and Treatment. 2014; 5:10–19.

Finy MS, Bresin K, Korol DL, Verona E. Impulsivity, risk taking, and cortisol reactivity as a function of psychosocial stress and personality in adolescents. Development and Psychopathology. (in press).

Fowles, DC.; Dindo, L. A dual-deficit model of psychopathy. In: Patrick, CJ., editor. Handbook of psychopathy. New York: Guilford Press; 2006. p. 14-34.

Franken IHA, van Strien JW, Franzek EJ, van de Wetering BJ. Error-processing deficits in patients with cocaine dependence. Biological Psychology. 2007; 75:45–51. [PubMed: 17196732]

Freeman JB, Dale R. Assessing bimodality to detect the presence of a dual cognitive process. Behavior Research Methods. 2013; 45:83–97. [PubMed: 22806703]

Gehring WJ, Gross B, Coles MG, Meyer DE, Donchin E. A neural system for error detections and compensation. Psychological Science. 1993; 4:385–390.

Hall JR, Benning SD, Patrick CJ. Criterion-related validity of the three-factor model of psychopathy: Personality, behavior, and adaptive functioning. Assessment. 2004; 11:4–16. [PubMed: 14994949]

Hall JR, Bernat EM, Patrick CJ. Externalizing psychopathology and the error-related negativity. Psychological Science. 2007; 18:326–333. [PubMed: 17470258]

Hare, RD. The Hare Psychopathy Checklist-Revised. Toronto, ON: Multi-Health System; 2001.

Hare, RD. The Hare Psychopathy Checklist-Revised. 2. Toronto, ON: Multi-Health System; 2003.

Hare RD, Neumann CS. Psychopathy as a clinical and empirical construct. Annual Review of Clinical Psychology. 2008; 4:217–246.

Harpur TJ, Hare RD, Hakstain AR. Two-factor conceptualization of psychopathy: Construct validation and assessment implications. Psychological Assessment. 1989; 1:6–17.

Hart, SD.; Cox, DN.; Hare, RD. Manual for the Psychopathy Checklist: Screening Version PCL: SV. Toronto, ON: Multi-Health Systems; 1999.

Hart SD, Forth AE, Hare RD. Performance of criminal psychopaths on selected neuropsychological tests. Journal of Abnormal Psychology. 1990; 99:374–379. [PubMed: 2266211]

Heinzen H, Köhler D, Godt N, Geiger F, Huchzermeir C. Psychopathy, intelligence and conviction history. International Journal of Law and Psychiatry. 2011; 34:336–340. [PubMed: 21899890]

Heritage AJ, Benning SD. Impulsivity and response modulation deficits in psychopathy: Evidence from the ERN and N1. Journal of Abnormal Psychology. 2013; 122:215–222. [PubMed: 22985014]

Hicks BM, Markon KE, Patrick CJ, Kruger RF, Newman JP. Identifying psychopathy subtypes on the basis of personality structure. Psychological Assessment. 2004; 16:276–288. [PubMed: 15456383]

Hicks BM, Patrick CJ. Psychopathy and negative emotional: Analyses of suppressor effects reveal distinct relations with emotional distress, fearfulness, and anger-hostility. Journal of Abnormal Psychology. 2006; 115:276–287. [PubMed: 16737392]

Hirsh JB, Inzlicht M. Error-related negativity predicts academic performance. Psychophysiology. 2009; 47:192–196. [PubMed: 19674391]

Hoff HA, Rypdal K, Mykletun A, Cooke DJ. A prototypicality validation of the Comprehensive Assessment of Psychopathic Personality model (CAPP). Journal of Personality Disorders. 2012; 26:414–427. [PubMed: 22686229]

Javdani S, Finy MS, Verona E. Evaluation of the validity of the Multidimensional Personality Questionnaire – Simplified-Wording Form (MPQ-SF) in adolescents with treatment histories. Assessment. (in press).

Judd CM, McClelland GH, Smith ER. Testing treatment by covariate interaction when treatment varies within subjects. Psychological Methods. 1996; 1:366–378.

Judd CM, Westfall J, Kenny DA. Treating stimuli as a random factor in social psychology: A new and comprehensive solution to a pervasive but largely ignored problem. Journal of Social and Personality Psychology. 2012; 103:54–69.

Karpman B. On the need for separating psychopathy into two distinct clinical types: Symptomatic and idiopathic. Journal of Criminal and Psychopathology. 1941; 3:112–137.

Kiehl KA, Bates AT, Laurens KR, Hare RD, Liddle PF. Brain potentials implicate temporal lobe abnormalities in criminal psychopaths. Journal of Abnormal Psychology. 2006; 115:443–453. [PubMed: 16866585]

Kirschbaum C, Pirke KM, Hellhammer DH. The "Trier Social Stress Test" – a tool for investigating psychobiological stress responses in a laboratory setting. Neuropsychobiology. 1993; 28:76–81. [PubMed: 8255414]

Kosson, KD.; Harpur, TK. Attentional functioning of psychopathic individuals: Current evidence and developmental implications. In: Burack, JA.; Enns, JT., editors. Attention, development, & psychopathology. New York: Guilford Press; 1997. p. 339-402.

Lasky, K. The rescue. New York, NY: Warner Books, Inc.; 2004.

Levenson MR, Kiehl KA, Fitzpatrick CM. Assessing psychopathic attributes in a noninstitutional population. Journal of Personality and Social Psychology. 1995; 68:151–158. [PubMed: 7861311]

Lilienfeld SO. Is psychopathy a syndrome? Commentary on Marcus, Fulton, and Edens. Personality Disorders: Theory, Research, and Treatment. 2013; 4:85–86.

Lilienfeld SO, Andrews BP. Development and preliminary validation of a self-report measure of psychopathic personality traits in noncriminal populations. Journal of Personality Assessment. 1996; 66:488–524. [PubMed: 8667144]

Lilienfeld, SO.; Fowler, KA. The self-report assessment of psychopathy. In: Patrick, CJ., editor. Handbook of psychopathy. New York: Guilford Press; 2006. p. 107-132.

Lilienfeld SO, Patrick CJ, Benning SD, Berg J, Sellbom M, Edens JF. The role of fearless dominance in psychopathy: Confusions, controversies, and clarifications. Personality Disorders: Theory, Research, and Treatment. 2012; 3:327–340.

Lilienfeld, SO.; Widows, MR. Professional manual for the Psychopathic Personality Inventory-Revised: (PPI-R). Lutz, FL: Psychological Assessment Resources; 2005.

Lykken, DT. The antisocial personalities. Hillsdale, NJ: Erlbaum;

Lynam DR, Hoyle RH, Newman JP. The perils of partialling cautionary tales from aggression and psychopathy. Assessment. 2006; 13:328–341. [PubMed: 16880283]

Lynam DR, Whiteside S, Jones S. Self-reported psychopathy: A validation study. Journal of Personality Assessment. 1999; 73:110–132. [PubMed: 10497804]

Lynam DR, Widiger TA. Using a general model of personality to identify the basic elements of psychopathy. Journal of Personality Disorders. 2007; 21:160–178. [PubMed: 17492919]

Marcus DK, Fulton JJ, Edens JF. The two-factor model of psychopathic personality: Evidence from the Psychopathic Personality Inventory. Personality Disorders: Theory, Research, and Treatment. 2013; 4:67–76.

Marcus DK, John SL, Edens JR. A taxometric analysis of psychopathic personality. Journal of Abnormal Psychology. 2004; 113:626–635. [PubMed: 15535794]

Miller GA. How we think about cognition, emotion, and biology in psychopathology. Psychophysiology. 1996; 33:615–628. [PubMed: 8961782]

Miller GA, Lutzenberger W, Elbert T. The linked-reference issue in EEG and ERP recording. Journal of Psychophysiology. 1991; 5:273–276.

Miller JD, Lynam DR. An examination of the Psychopathic Personality Inventory's nomological network: A meta-analytic review. Personality Disorders: Theory, Research, and Treatment. 2012; 3:305–326.

Morgan AB, Lilienfeld SO. A meta-analytic review of the relation between antisocial behavior and neuropsychological measures of executive function. Clinical Psychology Review. 2000; 20:113–156. [PubMed: 10660831]

Munro GES, Dywan J, Harris GT, McKee S, Unsal A, Segalowitz SJ. ERN varies with degree of psychopathy in an emotion discrimination task. Biological Psychology. 2007; 76:31–42. [PubMed: 17604898]

Neumann CS, Hare RD. Psychopathic traits in a large community sample: Links to violence, alcohol use, and intelligence. Journal of Consulting and Clinical Psychology. 2008; 76:893–899. [PubMed: 18837606]

Neumann CS, Malterer MB, Newman JP. Factor structure of the Psychopathic Personality Inventory (PPI): Findings from a large incarcerated sample. Psychological Assessment. 2008; 20:167–174.

Newman, JP.; Baskin-Sommers, AR. Early selective attention abnormalities in psychopathy: Implications for self-regulation. In: Posner, MI., editor. Cognitive neuroscience of attention. 2. New York: Guilford Press; 2012.

Newman JP, Patterson CM, Howland EW, Nichols SL. Passive avoidance in psychopaths: The effects of reward. Personality and Individual Differences. 1990; 11:1101–114.

Newman JP, Patterson CM, Kosson DS. Response preservation in psychopaths. Journal of Abnormal Psychology. 1987; 96:145–148. [PubMed: 3584663]

Ogilvie JM, Stewart AL, Chan RCK, Shum DHK. Neuropsychological measures of executive function and antisocial behavior: A meta-analysis. Criminology: An Interdisciplinary Journal. 2011; 49:1063–1107.

Pashler H, Harris CR. Is the replicability crisis overblown? Three arguments examined. Perspectives on Psychological Science. 2012; 7:531–536.

Patrick, CJ.; Bernat, EM. Neurobiology of psychopathy: A two process theory. In: Berntson, GG.; Cacioppo, JT., editors. Handbook of neuroscience for the behavioral sciences. Vol. 2. Hoboken, NJ: John Wiley & Sons; 2009. p. 1110-1131.

Patrick CJ, Edens JF, Poythress NG, Lilienfeld SO, Benning SD. Construct validity of the psychopathic personality inventory two-factor model with offenders. Psychological Assessment. 2006; 18:204–208. [PubMed: 16768596]

Patrick CJ, Fowles DC, Krueger RF. Triarchic conceptualization of psychopathy: Developmental origins of disinhibition, boldness, and meanness. Development and Psychopathology. 2009; 21:913–938. [PubMed: 19583890]

Patrick CJ, Hicks BM, Nichol PE, Krueger RF. A bifactor approach to modeling the structure of the Psychopathy Checklist-Revised. Journal of Personality Disorders. 2007; 21:118–141. [PubMed: 17492917]

Patrick CJ, Kramer MD, Tellegen A, Verona E, Kaemmer BA. Development and preliminary validation of a simplified-wording form of the multidimensional personality questionnaire. Assessment. 2013; 20:405–418. [PubMed: 23180862]

Patrick CJ, Venables NC, Drislane LE. The role of fearless dominance in differentiating psychopathy from antisocial personality disorder: Comment on Marcus, Fulton, and Edens. Personality Disorders: Theory, Research, and Treatment. 2013; 4:80–82.

Patrick CJ, Zempolich KA. Emotion and aggression in the psychopathic personality. Aggression and Violent Behavior. 1998; 3:303–338.

Patterson CM, Newman JP. Reflectivity and learning from aversive events: Toward a psychological mechanism for syndromes of disinhibition. Psychological Review. 1993; 100:716–736. [PubMed: 8255955]

Pfister R, Schwarz KA, Janczyk M, Dale R, Freeman JB. Good things peak in pairs: A note on the bimodality coefficient. Frontiers in Psychology. 2013; 4:1–4. [PubMed: 23382719]

Poythress NG, Lilienfeld SO, Skeen JL, Douglas KS, Edens JF, Epstein M, Patrick CJ. Using the PCL-R to help estimate the validity of two self-report measures of psychopathy with offenders. Assessment. 2010; 17:206–219. [PubMed: 19915197]

Preacher KJ, Curran PJ, Bauer DJ. Computational tools for probing interactions in multiple linear, multilevel modeling, and latent curve analysis. Journal of Educational and Behavioral Statistics. 2006; 31:437–448.

R Development Core Team. R: A language and environment for statistical computing. Vienna, Austria: R Foundation for Statistical Computing; 2010.

Rabbitt PMA. Errors and error-correction in choice-response tasks. Journal of Experimental Psychology. 1966; 71:264–272. [PubMed: 5948188]

Rabbitt PMA. Three kinds of error-signaling responses in a serial choice task. Quarterly Journal of Experimental Psychology. 1968; 20:179–188. [PubMed: 5653421]

Robinson MD. Gassing, breaking, and self-regulating: Error self-regulation, well-being, and goal-related processes. Journal of Experimental Social Psychology. 2007; 43:1–16.

Robinson MD, Schmeichel BJ, Inzlicht M. A cognitive control perspective of self-control strength and its depletion. Social and Personality Psychology Compass. 2010; 4:189–200.

Rushton PJ, Templer DI. National differences in intelligence, crime, income, and skin color. Intelligence. 2009; 37:341–346.

Sadeh N, Verona E. Psychopathic personality traits associated with abnormal selective attention and impaired cognitive control. Neuropsychology. 2008; 22:669–680. [PubMed: 18763886]

Salekin RT, Neumann CN, Leistico AR, Zalot AA. Psychopathy in youth and intelligence: An investigation of Cleckley's hypothesis. Journal of Clinical Child and Adolescent Psychology. 2004; 33:731–742. [PubMed: 15498740]

Schoenleber M, Sadeh N, Verona E. Parallel syndromes: Two dimensions of narcissism and the facets of psychopathic personality in criminally involved individuals. Personality Disorders: Theory, Research, and Treatment. 2011; 2:113–127.

Sellbom M, Verona E. Neuropsychological correlated of psychopathic traits in a non-incarcerated sample. Journal of Research in Personality. 2007; 2:276–294.

Skeem J, Johansson P, Andershed H, Kerr M, Louden JE. Two subtypes of psychopathic violent offenders that parallel primary and secondary variants. Journal of Abnormal Psychology. 2007; 116:395–409. [PubMed: 17516770]

Sprague J, Verona E. Emotional conditions disrupt behavioral control among individuals with dysregulated personality traits. Journal of Abnormal Psychology. 2010; 119:409–419. [PubMed: 20455613]

Vaidyanathan U, Hall JR, Patrick CJ, Bernat EM. Clarifying the role of defensive reactivity deficits in psychopathy and antisocial personality using startle reflex methodology. Journal of Abnormal Psychology. 2011; 120:235–258.

van der Leeden, R.; Busing, F. First iteration versus IGLS/RIGLS estimates in two-level models: A Monte Carlo study with ML3. Leiden, Netherlands: Leiden University; 1994. (Preprint PRM 94-03)

Verona E, Javdani S, Sprague J. Comparing factor structures of adolescent psychopathology. Psychological Assessment. 2011; 23:545–551. [PubMed: 21280958]

Verona, E.; Miller, GA. Analysis of covariance and related approaches. In: Cautin, R.; Lilienfeld, S., editors. Encyclopedia of Clinical Psychology. (in press)

Verona E, Patrick CJ, Joiner TE. Psychopathy, antisocial personality, and suicide risk. Journal of Abnormal Psychology. 2001; 110:462–470. [PubMed: 11502089]

Verona E, Sprague J, Sadeh N. Inhibitory control and negative emotional processing in psychopathy and antisocial personality disorder. Journal of Abnormal Psychology. 2012; 121:489–510.

Viechtbauer W. Conducting meta-analyses in R with the metafor package. Journal of Statistical Software. 2010; 36:1–48.

Vitacco MJ, Neumann CS, Jackson RL. Testing a four-factor model of psychopathy and its association with ethnicity, gender, intelligence, and violence. Journal of Consulting and Clinical Psychology. 2005; 73:466–476. [PubMed: 15982144]

Weinberg A, Riesel A, Hajcak G. Integrating multiple perspectives on error-related brain activity: The ERN as a neural indicator of trait defensive reactivity. Motivation and Emotion. 2012; 36:84–100.

Wilkowski BM, Robinson MD. Putting the brakes on antisocial behavior: Secondary psychopathy and post-error adjustments in reaction time. Personality and Individual Differences. 2008; 44:1807–1818.

Yeung N, Botvinick MM, Cohen JD. The neural basis of error detection: Conflict monitoring and the error-related negativity. Psychological Review. 2004; 111:931–959. [PubMed: 15482068]
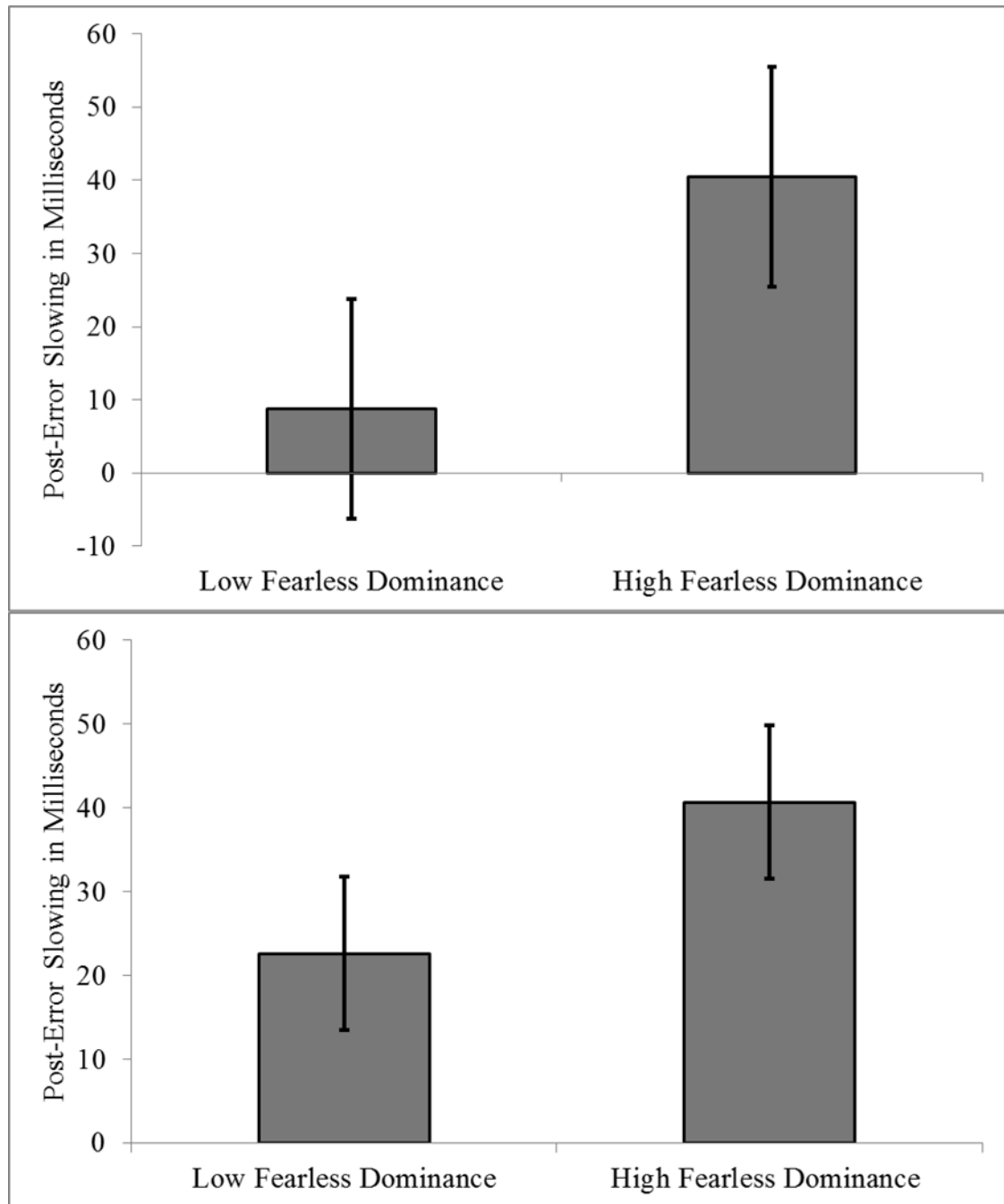
**Figure 1.**
Post-error Slowing (i.e., Lag-error – Lag-Correct) as a Function of Fearless Dominance for Study 1 (top panel) and Study 2 (bottom panel)
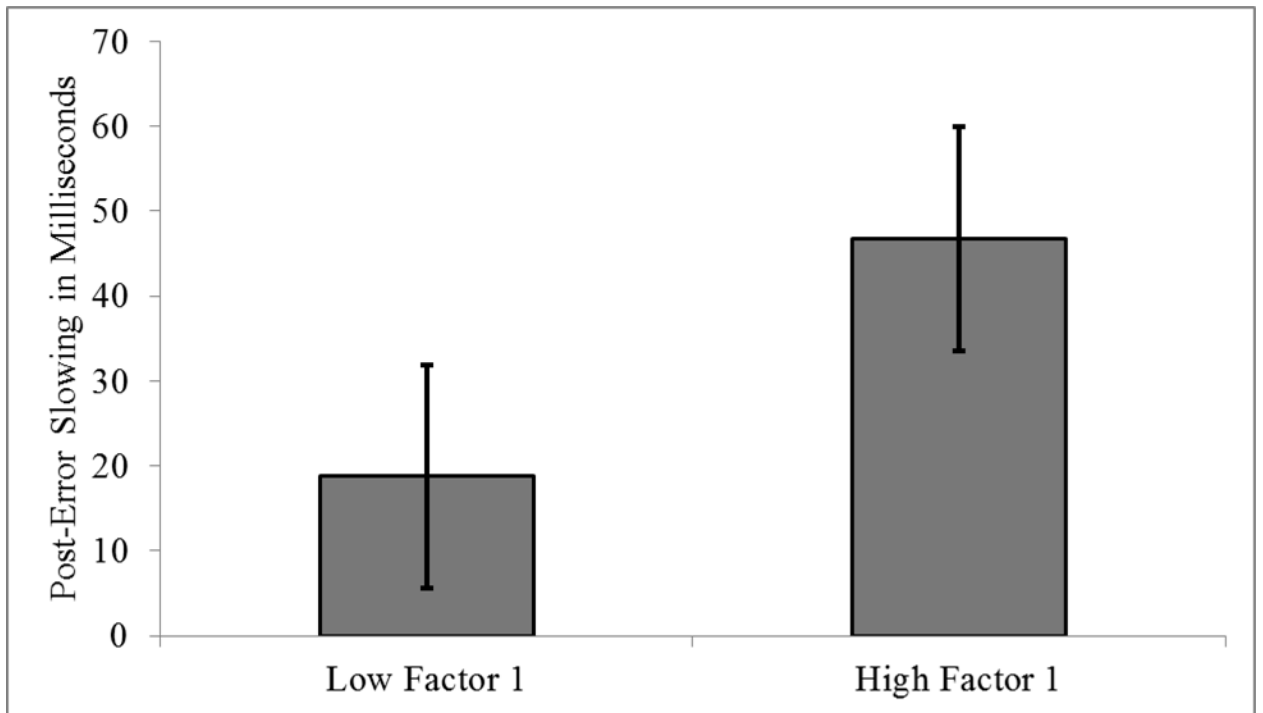
**Figure 2.**
Post-error Slowing (i.e., Lag-error – Lag-Correct) as a Function of Factor 1 for Study 3
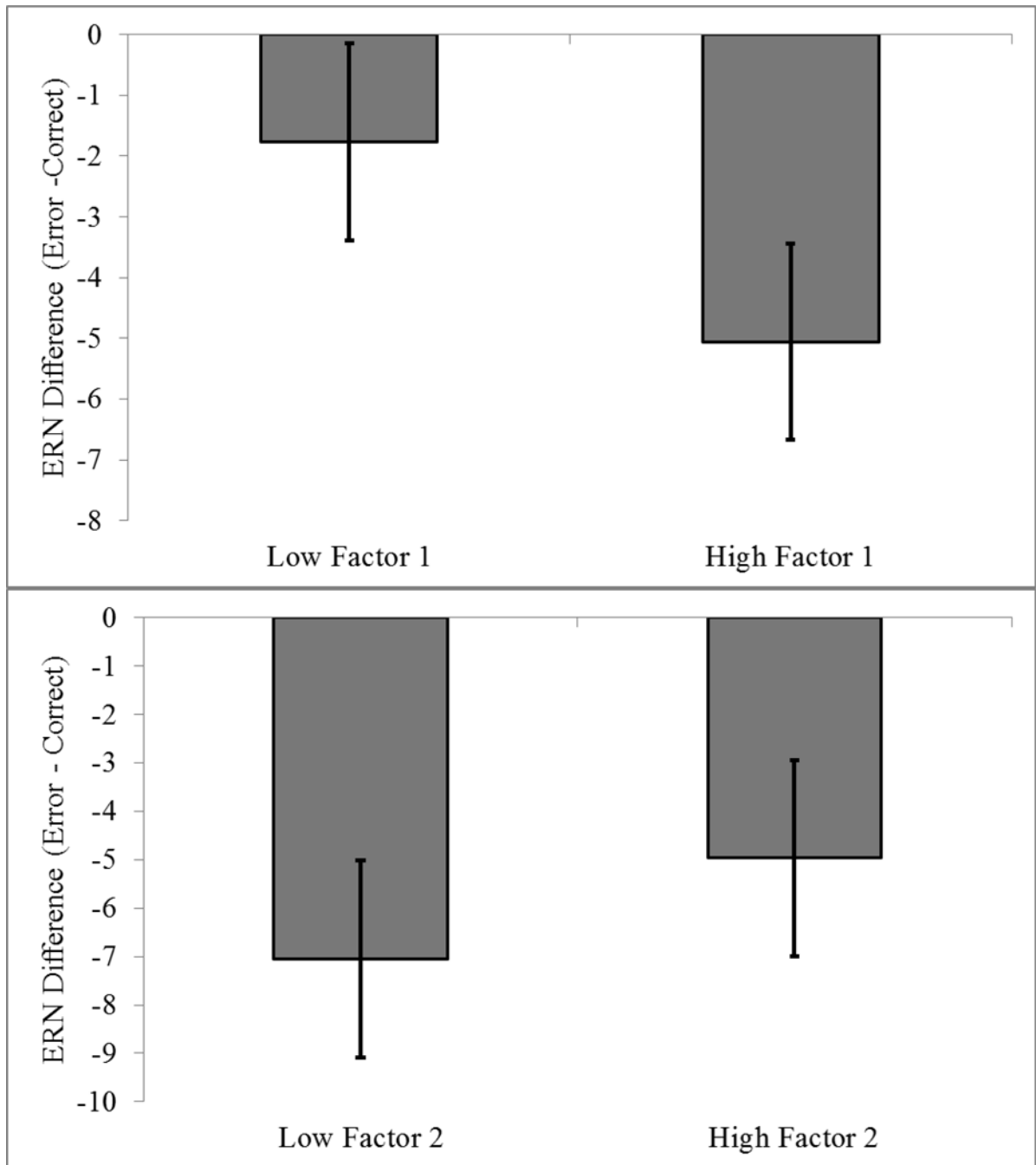
**Figure 3.**
Error-related Negativity (i.e., Error – Correct) as a Function of Factor 1 (top panel) and
Factor 2 (bottom panel)

## Table 1

Unstandardized Regression Coefficients, Variance Components, and Standard Errors for Reaction Time in Studies 1, 2, and 3 Multi-level Models

| | Fixed Effects | | |
|---|---|---|---|
| | Study 1 ($N = 81$) | Study 2 ($N = 42$) | Study 3 ($N = 71$) |
| Intercept ($\gamma_{00}$) | 449.02** (5.21) | 362.19** (6.56) | 609.27** (9.68) |
| Int-Aff ($\gamma_{01}$) | −1.12* (.49) | 3.44 (10.74) | 6.56 (3.30) |
| Imp-Anti ($\gamma_{02}$) | −0.06 (.44) | −1.04 (11.54) | 2.77 (3.90) |
| Int-Aff*Imp-Anti ($\gamma_{03}$) | −0.03 (.03) | −8.04 (26.28) | −0.44 (1.01) |
| Lag Error ($\gamma_{10}$) | 23.92** (5.96) | 31.12** (3.40) | 34.25** (4.19) |
| Int-Aff*Lag-Error ($\gamma_{11}$) | 1.46* (.57) | 15.55** (5.43) | 3.90** (1.36) |
| Imp-Anti*Lag-Error ($\gamma_{12}$) | −0.28 (.48) | 10.57 (6.28) | −2.51 (1.78) |
| Int-Aff*Imp-Anti*Lag-Error ($\gamma_{13}$) | 0.09* (.04) | −8.14 (13.37) | −0.35 (.45) |
| | Variance Components | | |
| Within-person ($\sigma^2$) | 6479.58** (129.85) | 6151.81** (81.30) | 5214.77** (911.37) |
| Between-person ($\tau_{00}$) | 2088.55** (344.80) | 1682.17** (385.31) | 19311** (156.36) |

Note.

** $p < .001$,

* $p < .05$.

Int-Aff = Interpersonal-affective traits (Fearless Dominance in Studies 1 and 2, Factor 1 in Study 3); Imp-Anti = Impulsive-antisocial traits (Impulsive Antisociality in Studies 1 and 2, Factor 2 in Study 3).