Early and Late Components of Error Monitoring in Violent Offenders with Psychopathy

Inti A. Brazil, Ellen R. A. de Bruijn, Berend H. Bulten, A. Katinka L. von Borries, Jacques J. D. M. van Lankveld, Jan K. Buitelaar, and Robbert J. Verkes

Background: One of the most recognizable features of psychopathy is the reduced ability to successfully learn and adapt overt behavior. This might be due to deficient processing of error information indicating the need to adapt controlled behavior.

Methods: Event-related potentials (ERPs) and behavioral components of error-monitoring processes were investigated in 16 individuals with psychopathy and in 18 healthy subjects. A letter version of the Eriksen flanker task was used in two conditions. The first condition (normal condition) required participants to press one of two buttons depending on the identity of the target stimulus. The second condition (signaling condition) required them to signal each time they had committed an error by making a second press on a signaling button. Early stages of error monitoring were investigated by using the error-related negativity (ERN/Ne) and post-error slowing as indexes. Later stages were explored by examining the error positivity (Pe) and signaling rates.

Results: Both groups showed similar ERN amplitudes and amounts of post-error slowing. The psychopathic group exhibited both reduced Pe amplitudes and diminished error-signaling rates compared with the control group.

Conclusions: Individuals with psychopathy show intact early error processing and automatic behavioral adaptation but have deficits in later stages of error processing and controlled behavioral adaptation. This is an indication that individuals with psychopathy are unable to effectively use error information to change their behavior adequately.

Key Words: Automatic processing, behavioral adaptation, error positivity, error-related negativity, error signaling, psychopathy

One of the most recognizable characteristics of psychopathy is the reduced ability to successfully learn and adapt overt behavior to comply with social rules and norms. The deficient behavioral adaptation exhibited by psychopathic individuals has been investigated using different kinds of learning paradigms. These studies consistently point out that psychopathic individuals fail to adapt their behavior to meet the rules provided by external sources (1–3). Newman *et al.* (1) indicated that individuals with psychopathy are deficient in avoiding monetary loss in situations in which they have to avoid punishment and earn monetary rewards. More recent research conducted by Budhani *et al.* (3) demonstrated that individuals scoring high on psychopathy showed impaired behavioral adaptation on a probabilistic reversal learning task. In this task, participants were expected to implicitly learn stimulus-reinforcement associations based on trial-by-trial feedback on performance. At some point, the contingencies were reversed without the participants knowing this, and they had to adapt their behavior to continue to receive positive feedback. Psychopathic individuals failed to make this reversal, providing further evidence for their inability to effectively adjust their behavior to meet the demands of the environment. Furthermore, these studies suggest that individuals with psychopathy are less sensitive to negative feedback following erroneous responses, consequently showing impairments in reinforcement-guided decision making.

From the Department of Psychiatry (IAB, AKLvB, JKB, RJV), Donders Centre for Neurosciences, University Medical Center St Radboud, Nijmegen; Donders Institute for Brain, Cognition and Behaviour (ERAdB), Radboud University Nijmegen, Nijmegen; Department of Clinical Psychological Science (JJDMvL), University of Maastricht, Maastricht; and Pompestichting (IAB, BHB, AKLvB, RJV), Nijmegen, The Netherlands.

Address reprint requests to Inti A. Brazil, MSc, Pompekliniek, PO Box 31435, 6503 CK Nijmegen, The Netherlands; E-mail: i.brazil@pompestichting.nl.

Received February 13, 2008; revised July 8, 2008; accepted August 8, 2008.

Rushworth *et al.* (4) have proposed a functional neuroanatomical model of reinforcement-based decision making. In their model, decision making is guided by the involvement of the anterior cingulate cortex (ACC) and orbitofrontal cortex (OFC). These areas are anatomically interconnected to other areas involved in encoding reward and reinforcement information, such as the ventral striatum and the amygdala (5,6). The amygdala has been found to be responsive to both aversive and reinforcing stimuli (7). Functionally, the OFC and the ACC are responsible for different aspects of reinforcement-guided decision making. The OFC shows greater involvement in the processing of information regarding stimuli, such as the formation of stimulus-reinforcement associations and representations of reward expectations. The ACC, on the other hand, is thought to use reinforcement information to adapt behavior (4).

The possibility that psychopaths are unable to adequately use error feedback to adapt their future behavior and the anatomical relationship between the ACC and the OFC suggest that there may also be deficiencies in one or more facets of error monitoring, which include the involvement of the ACC according to the reinforcement learning theory proposed by Holroyd and Coles (8). This theory states that an error signal is conveyed by the dopamine system from the basal ganglia to the ACC, resulting in the generation of an electrocortical waveform with a negative deflection. This waveform has been termed the error negativity (Ne) or error-related negativity (ERN) and is succeeded by a second component known as the error positivity (Pe) (9,10). The ERN is generated after error commission and negative feedback (8) and peaks between 0 to 100 msec after an erroneous response has been given (11). Source localization studies have localized the source of the ERN in the ACC (12,13), which is in accordance with functional magnetic resonance imaging (fMRI) studies demonstrating ACC involvement in error monitoring (for an overview, see Ridderinkhof *et al.* [14]). The Pe is a slow wave with maximum amplitude peaking between 200 to 400 msec after response onset (10) and can be regarded as a reflection of a later stage in error processing.

0006-3223/09/$36.00
doi:10.1016/j.biopsych.2008.08.011

Previous studies have shown that the ERN is a reflection of early stages in error processing that is not dependent on error awareness, while the Pe has been linked to later stages involving conscious error recognition (11,15–17).

Further evidence for the dichotomy between early and late components of error monitoring on the behavioral level has been provided by Debener *et al.* (18). They found that the amplitude of the ERN predicts the magnitude with which participants adapted their behavior by slowing down on the trial following an error. Slowing down after an error is a type of behavioral adjustment known as post-error slowing, first reported by Rabbitt (19), and has been interpreted as an involuntary and cautionary response strategy. To explore the impact of remedial actions, Ullsperger and von Cramon (20) investigated the differences between immediately correcting an error and signaling an error. The results showed that error correction is a fast, often involuntary, process that does not necessarily have to be preceded by conscious detection (see also Rabbitt [21]). In contrast, signaling errors is an intentional, much slower, and complex process based on conscious error recognition. Recognizing and signaling an error implies that at least some degree of error awareness is involved in the process. A study conducted by O'Connell *et al.* (17) demonstrated that the Pe was only present when participants were aware that they had committed an error, which was measured by pressing an "awareness button" to signal error commission.

Thus, the ERN has been associated with early unconscious processing of errors and the automatic adaptive processes of post-error slowing, while the Pe is believed to be related to conscious behavioral adaptations such as error signaling.

Research on error monitoring in psychopathic individuals has recently begun to emerge. In one study, Munro *et al.* (22) compared ERN amplitudes of psychopathic individuals on both neutral and emotional stimuli. In this study, psychopathic participants did not show abnormal ERN amplitudes on neutral stimuli when compared with healthy control subjects. However, the size of the ERN was significantly smaller in psychopathic offenders when the stimuli carried negative emotional valence.

Considering the findings on error monitoring together with the behavioral maladaptation that psychopathic individuals exhibit, we hypothesized that psychopathic individuals may show normal early processing in emotionally neutral conditions but are unable to effectively use error signals to guide their behavior. If this is the case, we expect that this inability should be reflected in a diminished Pe and lower error-signaling rates.

Methods and Materials

Subjects

The psychopathic group was recruited from the inpatient population of the Pompestichting Forensic Psychiatric Institute Nijmegen, The Netherlands.[1] Patients were selected based on available information about clinical status and prior history. Educational level was coded according to the Dutch educational system into three levels (level 1 = primary education; level 2 = secondary education; level 3 = higher education). The patient group consisted of 16 male patients (mean age = 39 years, SD = 9.5, mean education = 2.3), who were violent offenders diagnosed with psychopathy, as assessed with the Hare Psychopathy Checklist-Revised (PCL-R) (23). In this study, participants with a PCL-R score $\geq$ 26 were considered psychopaths and thus suitable for the first group. The psychopathic group had a mean PCL-R score of 32 (SD = 3.6).

The control group consisted of 18 healthy male volunteers (mean age = 37, SD = 6.4, mean education = 2.9). They were recruited by use of advertisements among the staff of the Forensic Institute who were not directly involved in patient care and known to have no criminal records and an absent history of psychiatric disorders. They were matched with the patients on age and educational level. Compliance to the exclusion criteria was determined for both groups using the Dutch version of Mini International Neuropsychiatric Interview (MINI) (24) and the Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II) (25). Exclusion criteria included all major Axis I and Axis II disorders (except antisocial personality disorder in the patient group), somatic disorders, pretest use of medication, and chronic use of intoxicating substances. All assessments were conducted by trained psychologists based on interviews with the participants and on available information from each patient's clinical files.

The protocol was approved by the local medical ethical committee. All participants received written information about the experiment and gave written informed consent. All participants received financial reward for their participation.

Task and Procedure

All subjects participated in two sessions, a screening session and a test session, during which experimental recordings were made. During the screening session, a number of self-report questionnaires[2] were completed and compliance to the exclusion criteria was determined.

Behavioral and electroencephalography (EEG) data were collected during the execution of a simple computer task. A modified version of the Eriksen flanker task (29) was used for the purposes of this study. In this task, participants responded with a button press of either their left or right index finger to the central letter (H or S) of a letter string. Four different letter strings were presented randomly with equal probabilities. The letter strings were either congruent (HHHHH or SSSSS) or incongruent (SSHSS or HHSHH) and appeared in black on a white background on a 100-Hz monitor at a distance of approximately 75 cm from the participant. Participants responded with a response button device with four buttons placed in a row. The left and right outer buttons were used to respond to the central letter of the target string.

The experiment consisted of two conditions. In both conditions, participants were instructed to focus on a fixation spot and to press the button corresponding to the letter presented in the center of the array as fast as possible. When an error was made in the first (normal) condition, no additional responses were required. However, when an error was made in the second (signaling) condition, participants were additionally asked to

[1]The Pompestichting is a "TBS-clinic" located in Nijmegen. TBS is a disposal to be treated, on behalf of the state, for people who have committed serious criminal offenses in connection with having a mental disorder. TBS is not a punishment but an entrustment act for mentally disordered offenders (diminished responsibility). These court orders are an alternative to either long-term imprisonment or confinement in a psychiatric hospital, with the goal to strike a balance between security, treatment, and protection.

[2]To identify possible covariates, anger, anxiety, and impulsivity were also measured using Dutch versions of the State-Trait Anxiety Inventory (STAI) (26), the State-Trait Anger Expression Inventory (STAXI) (27), and the Behavioral Inhibition System (BIS) and Behavioral Activation System (BAS) scales (28). However, inclusion of these as covariates did not show any significant group differences (all p's $>$ .093) or any significant within-subject effects (all p's $>$ .220).

signal the error by pressing the button located on the inside of the target button (i.e., the button on the right of the left button or the button on the left of the right button).

Each experimental condition was preceded by a practice block of 40 trials. The experimental phase was divided into four blocks of 100 trials. A fixation point was displayed in the center on the screen for 750 msec. After this, the "flanking" letters, that is, the surrounding letters without the central target letter was presented for 80 msec followed by the entire letter string for another 30 msec. After presentation of the stimulus, a blank screen was presented for 1000 msec during which the participants had to respond. After an intertrial interval of 300 msec, the next trial was presented. The entire experimental session lasted about 1.5 hours, including preparation and breaks.

Apparatus and Recordings

Scalp potentials were collected using active electrodes (Acticap, BrainProducts, Munich, Germany) arranged according to an extended version of the 10–20 system at F7, F3, Fz, F4, F8, FC5, FC1, FCz, FC2, FC6, T7, C3, Cz, C4, T8, CP5, CP1, CP2, CP6, P7, P3, Pz, P4, P8, O1, Oz, and O2. All electrodes were referenced to the left ear during recording and were rereferenced to the average of the earlobes during analysis. Electrooculography (EOG) recordings were also obtained: vertical eye movements were recorded by placing electrodes above and below the left eye, and another set located at the outer canthi recorded horizontal eye movements. The recorded signals were digitized with a sampling rate of 500 Hz using the Brain Products QuickAmp amplifier (BrainProducts) and filtered offline using a .02 Hz to 20 Hz bandpass filter.

Reaction times faster than 150 msec (1.9%) and slower than 1000 msec (.2%) were removed from the behavioral and EEG data for both groups. Brain activity was recorded continuously during the whole experiment. Electrooculography artifacts were removed using independent component analysis (ICA) (30). Electroencephalography signals for incongruent trials were time-locked to response onset and were averaged separately for each participant to event-related potentials (ERPs) for correct and incorrect responses relative to a 200 msec preresponse baseline.

Difference waves were computed on individual averages by subtracting the correct ERP waveforms from the incorrect ERPs (31). The ERN was defined on this difference wave as the most negative peak between the 0 msec to 150 msec period following response onset. These analyses were conducted at FCz and Cz, where ERN amplitudes were at a maximum. The Pe is a waveform known to evolve relatively slow and to be susceptible to jittering. For these reasons, it was defined as the average of the rectified amplitude between 250 msec to 400 msec following response onset in the difference wave. Analyses of the Pe activity were conducted at Cz, where Pe activity was maximal.

Values for the ERN were analyzed using a 2 × 2 × 2 repeated measures general linear model (GLM) with electrode site (FCz, Cz) and condition (normal, signaling) as within-subject variables and group (psychopaths, control subjects) as between-subject factor. The Pe was examined using a univariate GLM with mean activity at Cz as dependent variable and group as between-subjects variable. The analyses of Pe values were only conducted for the normal condition to avoid ERP distortion of motor activity related to the second button press in the signaling condition (32).

Behavioral data were analyzed by entering individual averages of reaction times (RTs) and error rates into different repeated measures GLMs with condition (normal, signaling), correctness (correct, incorrect), congruency (congruent, incongruent), and post-correctness (post-correct, post-error) as possible within-subject factors and group as between-subject factor. Post-error slowing analyses were limited to the normal condition because of the different instructions and additional signaling responses in the signaling condition. Error signaling rate was examined using a one-sided independent samples *t* test with group as independent variable.

Results

Behavioral Analyses

The RT analyses revealed a main effect for group, with patients responding slower (347 msec) than control subjects (325 msec) [$F(1,32) = 6.21$, $p = .018$]. There was a marginal trend for condition indicating slightly slower RTs in the signaling condition [$F(1,32) = 3.91$, $p = .057$]. Incorrect responses (293 msec) were faster than correct responses (379 msec) [$F(1,32) = 831$, $p < .001$]. A significant interaction with group indicated that this effect was larger for psychopathic individuals (93 msec) compared with control subjects (79 msec) [$F(1,32) = 5.89$, $p = .021$].

As expected, a main effect for congruency was present [$F(1,32) = 616$, $p < .001$]. Participants responded faster to congruent stimuli (338 msec) than to incongruent ones (430 msec). The interaction between group and congruency was not significant [$F(1,32) = 1.45$, $p = .238$]. Also, the interaction between congruency and condition was significant [$F(1,32) = 18.7$, $p < .001$], indicating that the congruency effect was larger in the signaling condition (99 msec) than in the normal condition (85 msec). The three-way interaction did not reach significance [$F(1,32) = .843$, $p = .366$].

With regard to error rates (Table 1), only a main effect for congruency was present [$F(1,32) = 276$, $p < .001$], indicating that participants made more errors on incongruent trials (9.7%) than on congruent ones (1.2%). The interaction between congruency and group was not significant [$F(1,32) = 3.11$, $p = .087$]. There was a marginal trend for the main effect of group, indicating that control subjects made slightly more errors (6.0%) than psycho-

Table 1. Mean Percentages of Error Rates for Congruent and Incongruent Trials for Each Condition and Mean Percentages of Signaling Rate Measured in the Signaling Condition

	Errors				
	Condition 1		Condition 2		
Group	Congruent	Incongruent	Congruent	Incongruent	Signaling
Control Group	1.4 (1.0)	10 (3.4)	1.3 (.9)	11 (3.3)	87 (19)
Psychopathic Group	1.3 (1.2)	9.1 (3.9)	1.0 (.9)	8.2 (2.6)	97 (3.4)

Standard deviations are displayed in parentheses.

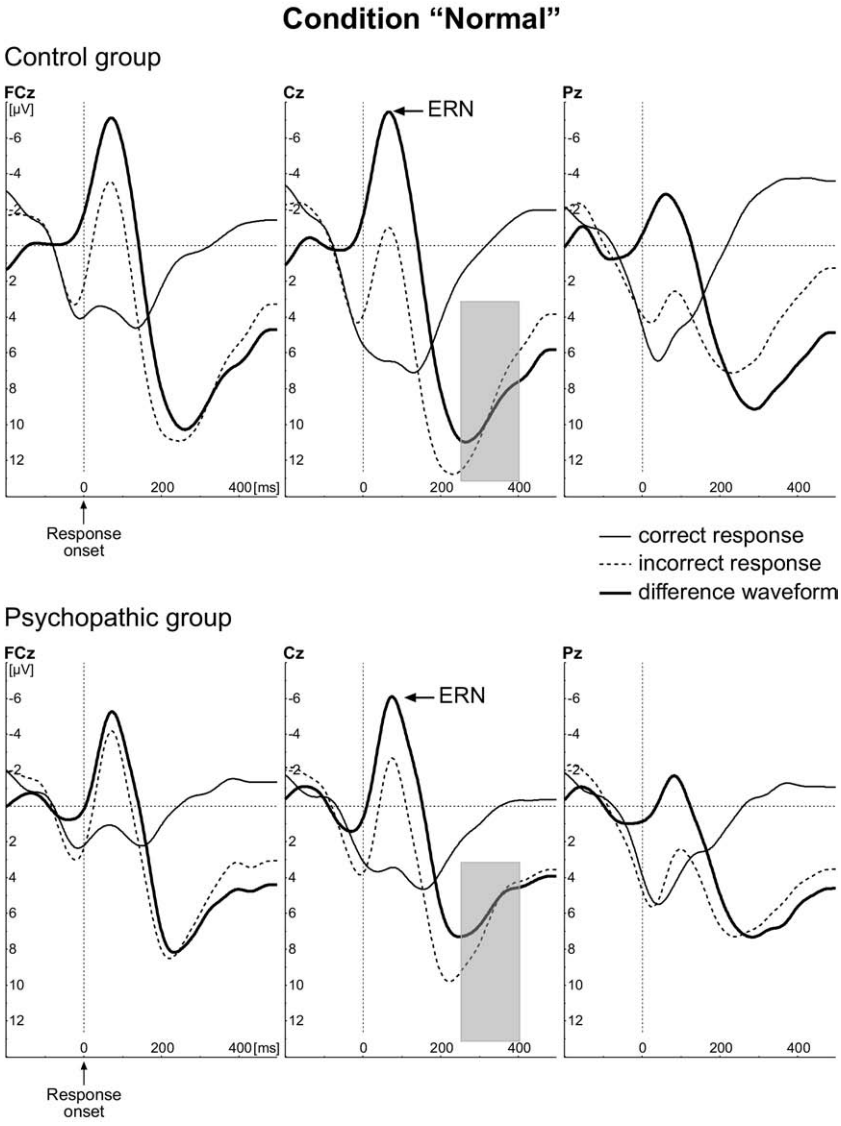

Figure 1. Grand average response-locked waveforms in the normal condition for correct and incorrect responses and the average difference waveform for the control and the psychopathic groups. Electrodes FCz, Cz, and Pz are depicted. ERN, error-related negativity.

pathic subjects (4.9%) [$F(1,32) = 4.04$, $p = .053$] and no significant differences were found between the two conditions [$F(1,32) = .106$, $p = .75$].

Reaction times for post-error trials (351 msec) were significantly slower than for post-correct trials (336 msec) in the normal condition [$F(1,32) = 9.99$, $p = .003$]. However, the performance of the groups did not differ on post-correct and post-incorrect trials, as the interaction between group and post-correctness failed to reach significance [$F(1,32) = .11$, $p = .75$].

ERP Analyses

The difference waves and the average waveforms for correct and incorrect trials for both groups are depicted in Figures 1 and 2 for each condition. With regard to ERN amplitudes, the main effect for group was not significant, demonstrating that ERN amplitudes did not differ between control subjects (−8.41 μV) and psychopathic subjects (−6.32 μV) [$F(1,32) = 2.12$, $p = .16$]. There was no effect of electrode site with a slightly larger ERN on electrode Cz (−7.47 μV) [$F(1,32) = .33$, $p = .57$] (Figure 3). Both groups showed comparable latencies with the ERN peaking around 73 msec on average [$F(1,32) = .10$, $p = .31$].[3]

Analyses of Pe activity revealed a significant main effect for group at Cz [$F(1,32) = 4.22$, $p = .048$], indicating larger Pe activity for the control group (10.5 μV) compared with the psychopathic group (7.4 μV) (Figure 3).

Signaling Rate Analyses

Analyses of signaling rate showed that patients signaled less errors (87%) compared with the control group (97%) [$t(16) = -1.98$, $p = .033$].

[3]The same analyses were also conducted using peak-to-peak differences of the ERN on incorrect response waveforms obtained by subtracting the most positive peak within -120 to 80 msec time window from the most negative peak within 0 msec to 150 msec relative to the response. These analyses yielded similar results. There was no main effect for condition [$F(1,32) = 3.28$, $p = .079$], but the analysis did show a main effect for electrode, indicating larger peak differences at FCz (−8.82 uV) compared with Cz (−7.38 uV) [$F(1,32) = 21.9$, $p < .001$]. More importantly, there was no main effect for group [$F(1,32) = .060$, $p = .808$].

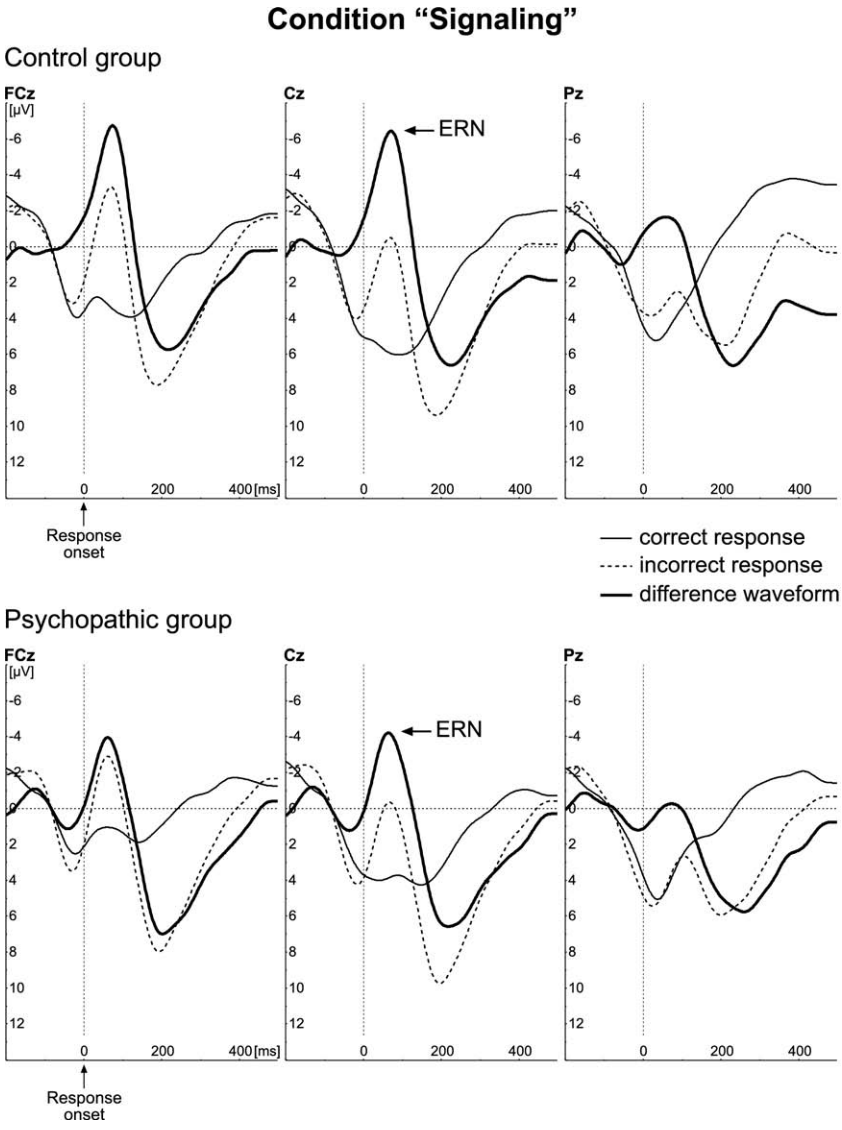

Figure 2. Grand average response-locked waveforms in the signaling condition for correct and incorrect responses and the average difference waveform for the control and the psychopathic groups. Electrodes FCz, Cz, and Pz are depicted. ERN, error-related negativity.

Discussion

Our results indicate that psychopathic individuals show unimpaired early processing of error information, while showing deficits in the later stages implicated in controlled behavioral adaptation.

The behavioral results indicated that the psychopathic group had error rates comparable with the control group, though displaying longer overall reaction times. This finding has previously also been reported by Munro *et al.* (22). Also, the increased RT differences between correct and incorrect responses for the psychopathic group might be interpreted as reflecting a more impulsive response style, with erroneous responses given relatively too fast (33). This interpretation is also in line with the general clinical image of psychopathy. However, recent findings from Munro *et al.* (34) did not provide evidence for a more impulsive response style in individuals with psychopathy. So, although the currently found RT patterns suggest a more impulsive response style, it is still rather unclear whether increased impulsivity of individuals with psychopathy is always reflected in these speeded choice reaction tasks.

Psychopathic subjects did not show differences in ERN amplitudes compared with healthy control subjects. This provides further evidence demonstrating that individuals with high levels of psychopathy show normal early error detection processes when presented with affectively neutral stimuli. The same pattern was found for post-error slowing, with both groups showing a comparable amount of slowing after error commission. As such, current outcomes are in line with recent findings by Munro *et al.* (22), demonstrating similar ERNs in psychopathic individuals and healthy individuals on a letter version of the flanker task highly comparable with the task currently used. They also found that the behavioral performance of the psychopathic group on trials following correct and error trials resembled that of healthy subjects. Hence, the current study and the study by Munro *et al.* (22) both show that individuals with psychopathy and healthy control subjects share commonalities in early unconscious error detection processes.

Interestingly, in a previous study by Dikman and Allen (35), reduced ERN amplitudes were reported in response to punishment in low socialized individuals compared with high socialized subjects. However, while Dikman and Allen (35)

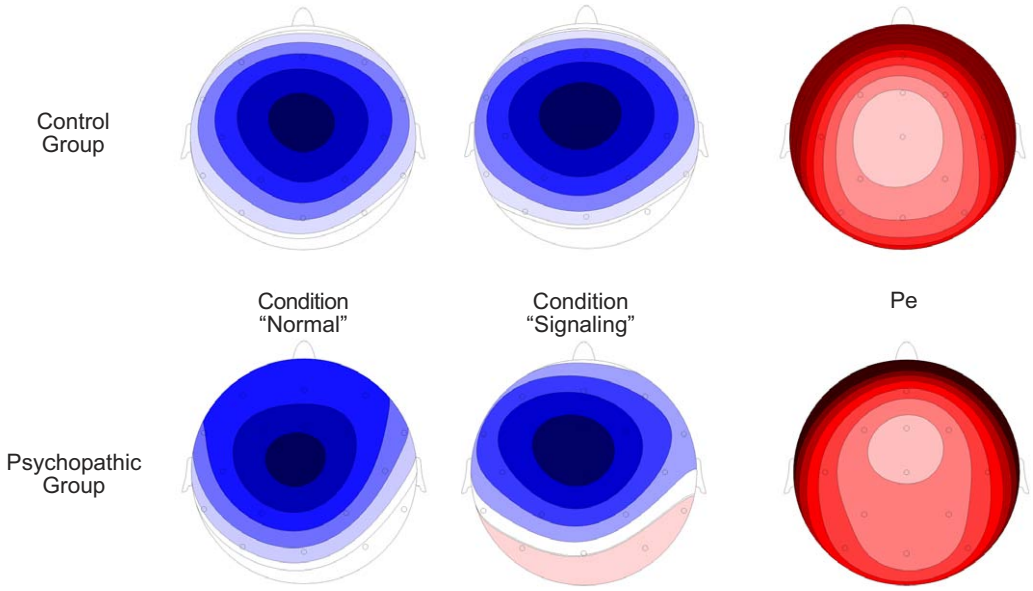

Figure 3. Scalp topographies of the ERNs at 70 msec for each group at Cz in the normal condition, in the signaling condition, and the mean Pe activity of each group in the normal condition (250–400 msec). Dark colored shades indicate negative polarities and lighter shades depict positive polarities. ERN, error-related negativity.

used low socialization in healthy subjects as an analogue for psychopathy, our experimental sample consisted of incarcerated patients actually diagnosed with psychopathy. Also, our task did not include reward/punishment manipulations. These large differences in sample characterization and task make a direct comparison between the two studies rather difficult and may explain the divergent outcomes regarding ERN amplitudes.

Contrary to the ERN outcomes but in line with our expectations, the current results show decreased Pe amplitudes for individuals with psychopathy compared with healthy control subjects. Munro *et al.* (22) did not demonstrate differences in Pe amplitudes between psychopathic subjects and healthy control subjects, but they did report a marginal trend that suggests decreased Pe sizes in the psychopathic group. However, it is possible that their Pe analysis did not reach significance due to the relatively small sample size of nine subjects meeting the criteria for psychopathy. So, psychopathic subjects showed a smaller Pe compared with healthy control subjects, with a reduction of approximately 30%. These findings demonstrate that individuals with psychopathy show deficits in a later stage involved in conscious error processing.

Finally, the behavioral findings of intact post-error slowing on the one hand and diminished error signaling on the other corroborate the ERP findings. Apparently, automatic behavioral adaptations resulting from early error detection processes are unaffected, while more controlled adaptive behavior related to later stages of error processing seems to be diminished. The signaling rate of the healthy control subjects (97%) is comparable with a previous study (95% in Ullsperger and von Cramon [20]), but the individuals with psychopathy were only capable of signaling 87% of their errors.

An alternative explanation for the functional significance of the Pe has been discussed by Overbeek *et al.* (36). The affective-processing hypothesis states that the Pe is involved in affective processes in such a way that the Pe could be a manifestation of emotional appraisal following an error. Emotional bluntness is considered to be a core feature of psychopathy (23). Research has shown that psychopathic individuals show reduced eye blink reflexes in response to stimuli with negative emotional valence (37). An fMRI study conducted by Müller *et al.* (38) demonstrated that psychopathic individuals exhibit reduced activation in the anterior cingulate, among other areas, in response to negative slides. Reports of abnormal affective processing in highly psychopathic subjects concord with the reduced Pe we found in our study, suggesting that psychopathic offenders have deviant emotional appraisal following errors.

However, Munro *et al.* (22) mention that their analyses indicate that the processing of affective information might not have a specific influence on the Pe. Note that our results also converge with outcomes of studies of error awareness in healthy individuals (11,15–17), providing further support for the dissociation between early unconscious components of error processing and later components leading to controlled adaptation of behavior.

Additionally, our results provide evidence for our suggestion that ACC functioning is compromised in psychopathy. Source localization studies of the Pe indicate that this component is generated within the ACC (17,39). The reduced Pe activity shown by our psychopathic subjects supports the idea that the ACC is involved in the anatomical networks that are considered to be deficient in psychopathy and might play a role in the abnormal learning behavior associated with this disorder.

We would like to note that we do not believe that possible ERN differences are precluded by relatively small sample sizes. On the contrary, our group sizes are comparable or even larger than previous between-group studies on error monitoring that did show ERN differences (33,40). Moreover, in the previous study by Munro *et al.* (22), similar results were obtained using the same neutral letter version of the flankers task.

Conclusion

In summary, these results indicate that early error processing and automatic adaptive behavior are intact in highly psycho-

pathic individuals, as reflected in normal ERN amplitudes and normal post-error slowing. More importantly, individuals with psychopathy display impairments in later stages of error processing and controlled adaptive behavior, as reflected in decreased Pe amplitudes and lower signaling rates. These findings may help us develop a better understanding of the relationship between the abnormal behavioral characteristics of psychopathy and broader concepts encountered in everyday life, such as learning and adapting their behavior to the ever-changing demands of their environment.

The authors reported no biomedical financial interests or potential conflicts of interest.

1. Newman JP, Widom CS, Nathan S (1985): Passive avoidance in syndromes of disinhibition: Psychopathy and extraversion. *J Pers Soc Psychol* 48:1316–1327.
2. Newman JP, Kosson DS (1986): Passive avoidance learning in psychopathic and nonpsychopathic offenders. *J Abnorm Psychol* 95:252–256.
3. Budhani S, Richell RA, Blair RJ (2006): Impaired reversal but intact acquisition: Probabilistic response reversal deficits in adult individuals with psychopathy. *J Abnorm Psychol* 115:552–558.
4. Rushworth MF, Behrens TE, Rudebeck PH, Walton ME (2007): Contrasting roles for cingulate and orbitofrontal cortex in decisions and social behaviour. *Trends Cogn Sci* 11:168–176.
5. Schoenbaum G, Roesch MR, Stalnaker TA (2006): Orbitofrontal cortex, decision-making and drug addiction. *Trends Neurosci* 29:116–124.
6. Haber SN, Kim KS, Mailly P, Calzavara R (2006): Reward-related cortical inputs define a large striatal region in primates that interface with associative cortical connections, providing a substrate for incentive-based learning. *J Neurosci* 26:8368.
7. Everitt BJ, Cardinal RN, Hall J, Parkinson JA, Robbins TW (2000): Differential involvement of amygdala subsystems in appetitive conditioning and drug addiction. In: Aggleton JP, editor. *The Amygdala: A Functional Analysis*. Oxford: Oxford University Press, 353–390.
8. Holroyd CB, Coles MGH (2002): The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychol Rev* 109:679–709.
9. Gehring WJ, Goss B, Coles MGH, Meyer DE, Donchin E (1993): A neural system for error detection and compensation. *Psychol Sci* 4:385–390.
10. Falkenstein M, Hohnsbein J, Hoormann J, Blanke L (1990): Effects of errors in choice reaction tasks on the ERP under focused and divided attention. *Psychophysiol Brain Res* 1:192–195.
11. Falkenstein M, Hoormann J, Christ S, Hohnsbein J (2000): ERP components on reaction errors and their functional significance: A tutorial. *Biol Psychol* 51:87–107.
12. Dehaene S, Posner MI, Tucker DM (1994): Localization of a neural system for error detection and compensation. *Psychol Sci* 5:303–305.
13. Carter CS, Braver TS, Barch DM, Botvinick MM, Noll D, Cohen JD (1998): Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science* 280:747–749.
14. Ridderinkhof KR, Ullsperger M, Crone EA, Nieuwenhuis S (2004): The role of the medial frontal cortex in cognitive control. *Science* 306:443–447.
15. Nieuwenhuis S, Ridderinkhof KR, Blom J, Band GP, Kok A (2001): Error-related brain potentials are differentially related to awareness of response errors: Evidence from an antisaccade task. *Psychophysiology* 38:752–760.
16. Endrass T, Franke C, Kathmann N (2005): Error awareness in a saccade countermanding task. *Int J Psychophysiol* 19:275–280.
17. O'Connell RG, Dockree PM, Bellgrove MA, Kelly SP, Hester R, Garavan H, *et al.* (2007): The role of cingulate cortex in the detection of errors with and without awareness: A high-density electrical mapping study. *Eur J Neurosci* 25:2571–2579.
18. Debener S, Ullsperger M, Siegel M, Fiehler K, von Cramon DY, Engel AK (2005): Trial-by-trial coupling of concurrent electroencephalogram and functional magnetic resonance imaging identifies the dynamics of performance monitoring. *J Neurosci* 25:11730–11737.
19. Rabbitt PM (1966): Errors and error correction in choice-response tasks. *J Exp Psychol* 71:264–272.
20. Ullsperger M, von Cramon DY (2006): How does error correction differ from error signaling? An event-related potential study. *Brain Res* 1105: 102–109.
21. Rabbitt P (2002): Consciousness is slower than you think. *Q J Exp Psychol* 55:1081–1092.
22. Munro GES, Dywan J, Harris GT, McKee S, Unsal A, Segalowitz SJ (2007): ERN varies with degree of psychopathy in an emotion discrimination task. *Biol Psychol* 76:31–42.
23. Hare RD, Hart SD, Harpur TJ (1991): Psychopathy and the DSM-IV criteria for antisocial personality disorder. *J Abnorm Psychol* 100:391–398.
24. Van Vliet IM, Leroy H, Van Megen HJM (2000): *De MINI-Internationaal Neuropsychiatrisch Interview: Een Kort Gestructureerd Diagnostisch Interview voor DSM-IV en ICD-10 Psychiatrische Stoornissen*. Leiden, The Netherlands: Leiden University Medical Center.
25. Groenestijn MAC, Akkerhuis GW, Kupka RW, Schneider N, Nolen WA (1999): *Gestructureerd klinisch interview voor de vaststelling van DSM-IV As-I stoornissen (SCID-I)*. Lisse, The Netherlands: Swets Test Publishers.
26. Van Der Ploeg HM, Defares PB (1980): *ZBV: A Dutch-Language Adaptation of the Spielberger State-Trait Anxiety Inventory*. Lisse, The Netherlands: Swets & Zeitlinger.
27. Van Der Ploeg HM, Defares PB (1982): *ZAV: A Dutch-Language Adaptation of the Spielberger State-Trait Anger Scale*. Lisse, The Netherlands: Swets & Zeitlinger.
28. Carver CS, White TL (1994): Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS scales. *J Pers Soc Psychol* 67:319–333.
29. Eriksen BA, Eriksen CW (1974): Effects of noise letters upon the identification of a target letter in a nonsearch task. *Percept Psychophys* 16:143–149.
30. Jung TP, Makeig S, Westerfield M, Townsend J, Courchesne E, Sejnowski TJ (2000): Removal of eye activity artifacts from visual event-related potentials in normal and clinical subjects. *Clin Neurophysiol* 111:1745–1758.
31. Holroyd CB, Dien J, Coles MGH (1998): Error-related scalp potentials elicited by hand and foot movements: Evidence for an output-independent error-processing system in humans. *Neurosci Lett* 242: 65–68.
32. Fiehler K, Ullsperger M, Von Cramon DY (2005): Electrophysiological correlates of error correction. *Psychophysiology* 42:72–82.
33. De Bruijn ERA, Grootens KP, Verkes R, Buchholz V, Hummelen JW, Hulstijn W (2006): Neural correlates of impulsive responding in borderline personality disorder: ERP evidence for reduced action monitoring. *J Psychiatr Res* 40:428–437.
34. Munro GES, Dywan J, Harris GT, McKee S, Unsal A, Segalowitz SJ (2007): Response inhibition in psychopathy: The frontal N2 and P3. *Neurosci Lett* 418:149–153.
35. Dikman Z, Allen J (2000): Error monitoring during reward and avoidance learning in high-and low-socialized individuals. *Psychophysiology* 37:43–54.
36. Overbeek TJM, Nieuwenhuis S, Ridderinkhof KR (2005): Dissociable components of error processing. *Int J Psychophysiol* 19:319–329.
37. Patrick CJ, Bradley MM, Lang PJ (1993): Emotion in the criminal psychopath: Startle reflex modulation. *J Abnorm Psychol* 102:82–92.
38. Müller JL, Sommer M, Wagner V, Lange K, Taschler H, Röder CH, *et al.* (2003): Abnormalities in emotion processing within cortical and subcortical regions in criminal psychopaths evidence from a functional magnetic resonance imaging study using pictures with emotional content. *Biol Psychiatry* 54:152–162.
39. Hermann M, Rommler J, Ehlis A, Heidrich A, Fallgatter A (2004): Source localisation (LORETA) of the error-related negativity (ERN) and positivity (Pe). *Brain Res Cogn Brain Res* 20:294–299.
40. Ruchsow M, Walter H, Buchheim A, Martius P, Spitzer M, Kächele H, *et al.* (2006): Electrophysiological correlates of error processing in borderline personality disorder. *Biol Psychol* 72:133–140.