A Neurophysiological Dissociation Between Monitoring One's Own and Others' Actions in Psychopathy

Inti A. Brazil, Rogier B. Mars, Berend H. Bulten, Jan K. Buitelaar, Robbert J. Verkes, and Ellen R.A. De Bruijn

Background: Psychopathy is a severe personality disorder often leading to violent and disruptive antisocial behavior. Efficient and proper social behavior crucially relies on monitoring of one's own as well as others' actions, but the link between antisocial behavior in psychopathy and action monitoring in a social context has never been investigated.

Methods: Event-related potentials were used to disentangle monitoring of one's own and others' correct and incorrect actions in psychopathic subjects ($n = 18$) and matched healthy control subjects ($n = 18$). The error-related negativity (ERN) was investigated following own and other's responses in a social flanker task.

Results: Although both groups showed similar event-related potentials in response to own actions, amplitudes after the observation of others' action-outcome were greatly reduced in psychopathy. More specifically, the latter was not unique to observed errors, because the psychopathic group also showed reduced brain potentials after the observation of correct responses. In contrast, earlier processing of observed actions in the motor system, as indicated by the lateralized readiness potential, was unimpaired.

Conclusions: Monitoring of own behavior is not affected in psychopathy, whereas processing of the outcome of others' actions is disturbed. Specifically, although psychopathic individuals do not have a problem with initial processing of the actions of others, they have problems with deeper analyses of the consequences of the observed action, possibility related to the reward value of the action. These results suggest that aspects of action monitoring in psychopathy are disturbed in social contexts and possibly play a central role in the acquisition of abnormal social behavior.

Psychopathy is a personality disorder characterized by distortions in emotional processing and antisocial behavior (1). Psychopathic individuals are known to show an almost total lack of empathy, guilt, or remorse combined with antisocial behavior fueled by impulsivity, poor planning skills, and frequently criminal intents. In clinical practice, psychopathy is often labeled as highly resistant to treatment. The antisocial lifestyle of psychopathic offenders indicates that they have experienced severe problems in acquiring social norms and rules (2). One way of acquiring social norms and rules and appropriate behavior is by observing others. More specifically, we learn by monitoring other individuals' performance and imitating behavior leading to desired outcomes, while avoiding others' behavior ending in undesired outcomes (3). This implies that we need to be susceptible to errors committed by others to learn appropriately.

Research on performance monitoring has predominantly focused on processing of one's own errors. The detection of error commission by oneself is associated with the generation of the error-related negativity (rERN) (4–6), an event-related brain potential (ERP) in posterior medial frontal cortex (7). This component has been linked to the processing of the reward value of the action and subsequent behavioral adjustments (8,9). Previous results on monitoring of own actions in psychopathy are mixed, although there seems to be a dissociation between studies using students with psychopathic traits (10,11) and actual psychopathic offenders (12,13). Although the former studies reported reduced error-related negativity (ERNs) in tasks consisting of affectively neutral stimuli, these deficiencies were not demonstrated in diagnosed psychopathy.

More recently, investigations of ERPs during action monitoring in social contexts have been initiated, focusing on two aspects of processing others' actions. First, components related to initial processing of the action. Studies on motor resonance have shown that the observation of movements activates brain systems in the motor cortices similar to those activated by the self-generation of the same actions (14–16). Motor activation can be measured with the lateralized readiness potential (LRP), a marker for automatic motor preparation, visible before the execution of a movement over the contralateral hemisphere. During observation, the development of the LRP seems to be susceptible to the correctness of the observed response. The LRPs for both correct and incorrect responses start to develop in the same direction before the onset of the observed response ("anticipation") and continue to increase in amplitude after the observation of a correct response but will decrease if the observed response was incorrect (17). Thus, motor resonance during action observation extends further than only making copies of observed movements by showing differential activation susceptible to response correctness, a function that might play an important role in observational learning (18).

The second component identified during observation of others' actions is a later ERN-like component, which is generated when participants observe other individuals commit errors, the so-called "observed ERN" (oERN) (17,19,20). The source of the oERN has been localized in the same medial frontal areas as the traditional rERN, suggesting that both waveforms are a reflection of the same underlying mechanism (17). This was confirmed by functional magnetic resonance imaging data showing that both the detection of own and others' errors activate the same networks (21,22).

The aim of the present study is to investigate error-monitoring during the observation of actions in psychopathy. We hypothesized that deeper processing of others' erroneous outcomes is compromised in psychopathy, made evident by reduced oERN amplitudes in the psychopathic group. We expected, in contrast and in line with earlier research, normal ERNs to own errors, reflecting unaffected monitoring of own actions (13). Additionally, we investigated the onset and course of the LRP as a marker for differential involuntary motor activation during the commission and observation of correct and erroneous responses.

Methods and Materials

Subjects

The psychopathic group was recruited from the in- and out-patient population of the Pompestichting Forensic Psychiatric Institute in The Netherlands, a treatment facility for mentally disordered offenders. Stay in the clinic is designed to resemble everyday life outside of detention, requiring patients to follow treatment, schooling, work, practice sports, and the like.

Patients were selected on the basis of available information about clinical status and prior history. An estimation of the IQ level of each participant was obtained with the Dutch version of the National Adult Reading Test (23) (Table 1). The patient group consisted of 18 male violent offenders diagnosed with psychopathy, as assessed with the Hare Psychopathy Checklist–Revised (PCL-R) (1). Patients scoring above the cutoff score (PCL > 25), according to European standards, were considered suitable for inclusion in the psychopathic group (24,25).

The control group consisted of 18 healthy male volunteers without criminal records and no history of psychiatric disorders recruited by use of advertisements. They were matched with the patients on age and IQ. The Dutch version of MINI Psychiatric Interview (26) and the Structured Clinical Interview for DSM-IV Axis II Personality Disorders (27) were used in both groups to determine compliance with the inclusion criteria. Exclusion criteria included all major Axis-I and Axis-II disorders, somatic disorders, any form of (self-)reported or documented head trauma, chronic use of intoxicating substances, use of psychotropic medication up to 5 days before the test session, pretest use of alcohol or tobacco, and for the patient group, a positive result on any of the unannounced randomly administered urinal drug tests. All assessments were conducted by trained psychologists on the basis of interviews with the

participants and on available information from the clinical files of each patient.

The protocol was approved by the local medical ethics committee. All participants received written information about the experiment, gave written informed consent, and received a financial reward.

Task and Procedure

All clinical assessments were conducted during a screening session. In a second session, behavioral and electroencephalography data were collected during the execution of a modified version of the arrowhead Eriksen flanker task (17,28). Participants were seated across the table facing the experimenter. A light-emitting diode (LED) device was situated at the center of the table with a custom-made joystick device in front of it at a distance of approximately 25 cm on both the right and the left side of the LED device. The LED device had two display sides, one facing the participant, and the other toward the actor at a viewing distance of approximately 75 cm. Stimuli consisted of arrowheads pointing to the left or to the right in four arrays ($<<<<<,>>>>>,<< ><<,>> < >>$) occurring randomly with equal probabilities.

The experiment was divided into two conditions. In the first condition (Perform condition), participants were instructed to respond as quickly and accurately as possible by using their thumb to push the lever on the joystick in the same direction indicated by the arrowhead in the center of the array displayed.

In the second condition (Observe condition), participants received instructions to observe the actor (experimenter) while he performed the same flanker task and to count and report the amount of errors committed by the actor after each block. The counting provided an accuracy measure for the engagement of the observer in the task. Only the center arrowhead was displayed to the observers, to ensure that error detection was not compromised by the presence of flankers. Observers were able to see both the LED device and the actor's responses without moving their eyes and were instructed to stay focused on the fixation point and to identify responses without making eye movements (cf. van Schie *et al.*) (17). All subjects participated in the Perform condition first, establishing their understanding of the task, before participating in the Observe condition (17,21,22).

The experimental conditions started with a practice block of 40 trials. Each condition consisted of six blocks of 100 trials. A trial started with the presentation of a fixation point presented at the center of the LED device for 200 msec, followed by a stimulus-free interval of 200 msec. In succession, one of the four stimulus arrays was displayed for 300 msec followed by a response window of 900 msec. An error-check was added to the task to ensure participants committed enough errors. After 15 consecutive correct trials an array of hash marks (#####) was presented, indicating that the performer had to increase his response speed. In the observe condition, subjects were instructed to write down the amount of errors they had observed at the end of each block.

Data Acquisition

Scalp potentials were collected with 27 active electrodes (Acti-Cap, Brain Products, Munich, Germany) arranged according to an extended version of the 10–20 system. All electrodes were referenced to the left ear during recording and were re-referenced to the linked earlobes during analysis. Electro-oculography recordings were also collected for vertical and horizontal eye movements by placing electrodes above and below the left eye and at the outer canthi. The recorded signals were digitized with a sampling rate of 500 Hz with a QuickAmp amplifier (Brain Products) and filtered

Table 1. Demographic Data of Control and Psychopathic Groups

	Control Group (n = 18)	Psychopathic Group (n = 18)
Age	36 (8)	39 (8)
IQ	101 (6)	98 (9)
PCL-R	—	31 (3)

No significant group differences. Means are reported with SDs between parentheses.

PCL-R, Hare Psychopathy Checklist–Revised.

offline with a .02–20 Hz band-pass filter for analyses of all ERPs. No filtering was applied during acquisition.

Reaction times (RTs) below 150 msec (1.6%) and slower than 3 SDs from the mean (558 msec; .8%) were removed from the data for both groups. Ocular artifacts were removed using Independent Component Analysis (29).

ERN

A matching procedure was used to diminish the impact of stimulus-related activity on the ERN and the LRP (30,17). Through this procedure, each incorrect trial was randomly matched to a corresponding correct trial on the basis of RT ($\pm$ 4 msec), for each participant in both conditions.

Electroencephalography signals for correct and incorrect trials in both conditions were time-locked to response onset (700-msec epoch) and were averaged separately for each participant for correct and incorrect responses relative to a 200-msec pre-response baseline. The rERN was defined as the most negative peak within the 50–150-msec period after response onset. For the oERN, the most negative peak between 150 and 350 msec time-locked to the response of the actor was determined. These analyses were conducted at FCz and Cz, where ERN amplitudes were at a maximum.

LRP

The LRPs were calculated with signals recorded from C3 and C4 electrodes. The average asymmetry, defined as the difference between C3 and C4, was derived by averaging the asymmetries associated with trials where the left movements were correct and those where right movements were correct according to the following equation (31):

$$LRP = (\text{left hand } [C4 - C3] + \text{right hand } [C3 - C4])/2$$

Negative values of the LRP indicate relative activation of the correct response, and positive values indicate relative activation of the incorrect response. As for the ERN, this analysis was performed on trials matched for RTs.

Peak LRP amplitudes were determined in a window around the response (-150–50 msec for the Perform condition; 50–400 msec for the Observed condition). To determine when the LRPs first significantly differed between correct and incorrect responses, the difference between correct and incorrect trial waveforms was assessed by a stepwise series of one-tailed serial t tests (step size of 2 msec; cf. Schmitt *et al.*) (32). For each test, data were averaged from a time window of 40 msec. The latency of the significant difference between the two waveforms was defined as the first point at which 10 consecutive t tests show a significant difference at $p < .05$. This procedure was applied in a time window around the response (-350–300 msec for the Perform condition; 50–470 msec for the Observe condition).

Additional Analyses

Stimulus-locked P3 amplitudes were computed on unmatched correct incongruent trials in the Observe condition to check for abnormal stimulus processing and attention. The P3 was defined as the most positive peak between 400 and 800 msec at Cz, where this component was maximal.

Also, correlations among the oERN, the PCL-R scores, and its subscales were investigated for the psychopathic group.

Generalized Linear Models (GLMs)

Behavioral data were analyzed by entering individual averages of RTs and error rates from the Perform condition into different repeated-measures GLMs with Correctness (correct, incorrect),

Congruency (congruent, incongruent), and Post-correctness (post-correct, post-error) as possible within-subject (WS) variables and Group (patients, control subjects) as between-subject (BS) factor. For the Observe condition, accuracy rates were determined by calculating the ratio between the amount of errors reported by the participants and the actual amount of errors committed by the actor. Also, the amount of errors committed by the actor in the Observe condition and the percentage of observed errors reported by the subjects were entered in Univariate GLMs with Group as a BS-factor.

The rERN was analyzed for the Perform condition, and the oERN was examined for the Observe condition with separate $2 \times 2 \times 2$ repeated measures GLMs with Electrode site (FCz, Cz) and Correctness as WS-factors and Group as BS-factor.

The LRP amplitudes and latencies were analyzed separately by entering Condition (perform, observe), Correctness, and Group as possible factors. For the additional P3 analyses, Group was entered as BS-factor in a Univariate GLM.

Results

Behavioral Analyses

The RT analyses yielded a significant result for Correctness [$F(1,34) = 274$, $p < .001$], with incorrect responses being faster than correct ones (Table 2). There was no main effect for Group [$F(1,34) < 1$, $p = .402$] and no significant interaction of Group $\times$ Correctness [$F(1,34) < 1$, $p = .973$].

A main effect was found for Congruency [$F(1,34) = 112$, $p < .001$], indicating that subjects were faster on congruent trials compared with incongruent ones. There was no main effect for Group [$F(1,34) < 1$, $p = .420$], but a significant Group $\times$ Congruency interaction showed that the congruency effect was larger for the psychopathic group (24 msec) compared with the control group [16 msec; $F(1,34) = 4.78$, $p = .036$].

There was also a main effect for Post-correctness [$F(1,34) = 4.98$, $p = .032$]. Participants responded more slowly on post-error trials compared with post-correct trials. The groups did not differ on this measure [$F(1,34) < 1$, $p = .522$], and the interaction also failed to reach significance [$F(1,34) = 1.91$, $p = .176$].

For error rates, a main effect for Congruency was found [$F(1,34) = 66.7$, $p < .001$], indicating that more errors were committed on incongruent trials compared with congruent trials (Table 3). Neither the Congruency $\times$ Group interaction [$F(1,34) = 1.74$, $p = .197$] nor the main effect for Group [$F(1,34) < 1$, $p = .852$] reached significance. Analyses of the amount of errors committed by the actor in the Observe condition revealed that both groups had the opportunity to observe a comparable amount of errors [119 vs. 118; $F(1,34) < 1$, $p = .907$] and that the groups did not differ significantly

Table 2. Mean RTs for Control and Psychopathic Groups

Measure	Trial Type	Control Group (*n* = 18)	Psychopathic Group (*n* = 18)	Overall Mean
Correctness	Correct	345 (41)	355 (35)	351 (38)
	Incorrect	295 (26)	304 (35)	300 (31)
Congruency	Congruent	327 (37)	333 (33)	330 (35)
	Incongruent	343 (42)	357 (40)	350 (41)
Post-Correctness	Post-error	350 (44)	356 (36)	353 (40)
	Post-correct	343 (42)	354 (36)	349 (39)

No significant group differences. Mean reaction times (RTs) (msec) for the control and the psychopathic group (SD between parentheses).

Table 3. Mean Percentage Error Rates in the Perform Condition for the Control and the Psychopathic Groups

Measure	Control Group (n = 18)	Psychopathic Group (n = 18)	Overall Mean
Congruent	7.8 (5.5)	6.1 (6.0)	6.9 (5.7)
Incongruent	15.0 (5.1)	16.1 (5.0)	15.6 (5.0)

No significant group differences. Mean percentage error rates in the Perform condition for the control and the psychopathic group (SD between parentheses).

on the percentage of observed errors reported [89.4% vs. 93.5%; $F(1,34) < 1, p = .373$].

ERP Analyses

A main effect for Correctness was found for the rERN [$F(1,34) = 65.5, p < .001$] (Figure 1). Neither the main effect for Group [$F(1,34) < 1, p = .593$] nor the interaction [$F(1,34) < 1, p = .788$] was significant. The main effect for Electrode was significant [$F(1,34) = 39.8, p < .001$], with larger negativity at FCz (.2 μV) compared with Cz (1.7 μV). The groups showed comparable latencies for the rERN at FCz [64 msec; $F(1,34) = 4.25, p = .519$].
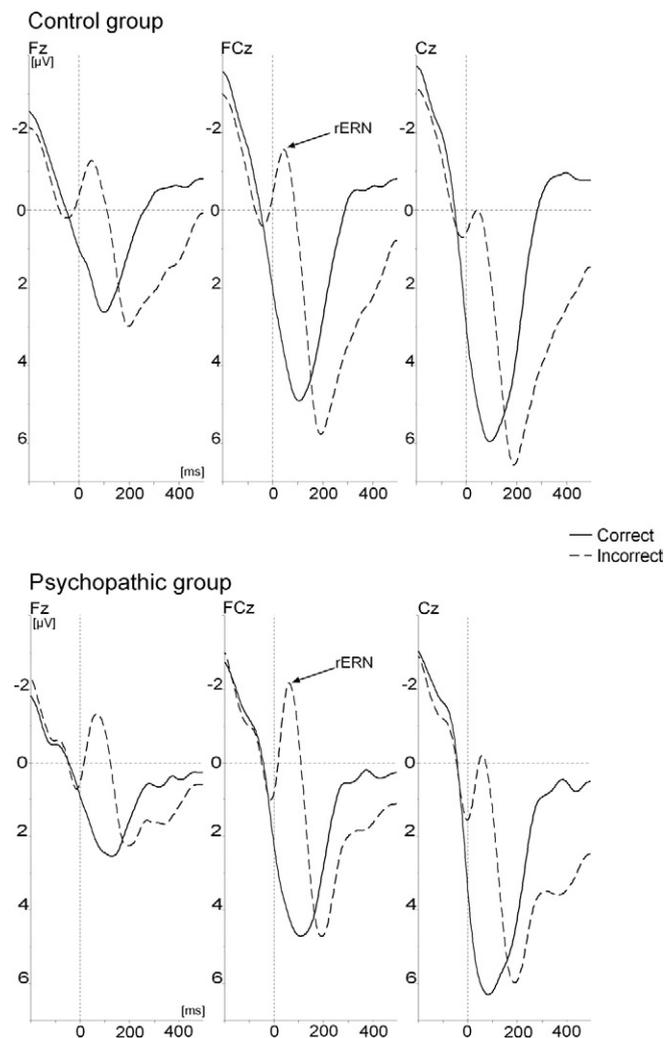

Figure 1. Grand average response-locked waveforms for correct and incorrect responses in the Perform condition for the control (n = 18) and the psychopathic (n = 18) groups. rERN, response error-related negativity.
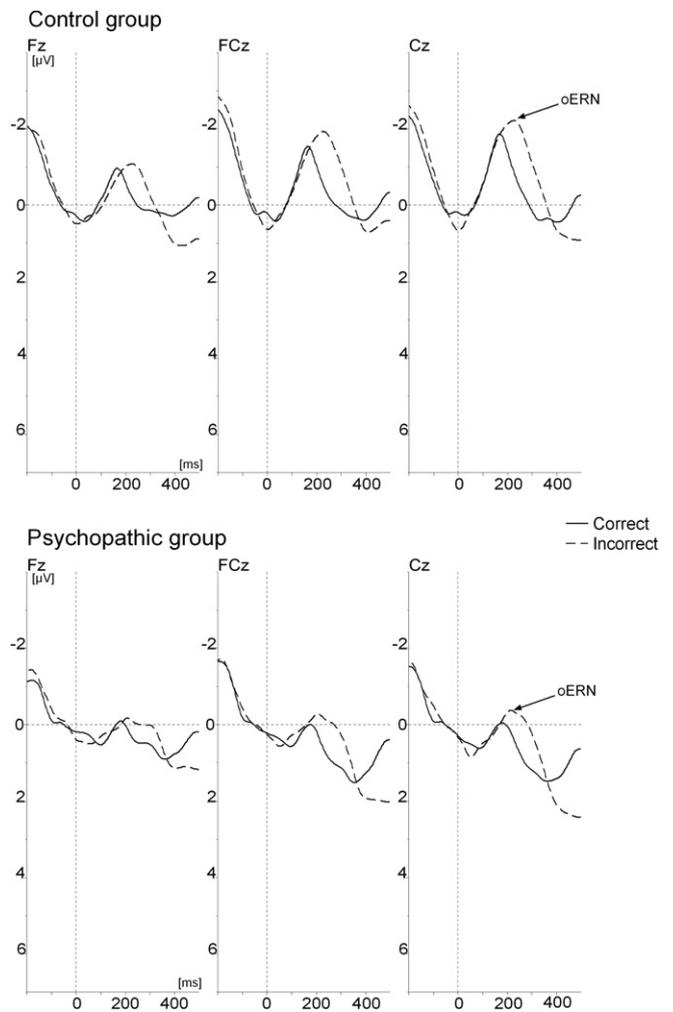

Figure 2. Grand average response-locked waveforms for correct and incorrect responses in the Observe condition for the control (n = 18) and the psychopathic (n = 18) groups. oERN, observed error-related negativity.

Results for the oERN (Figures 2 and 3) revealed a main effect for Correctness [$F(1,34) = 4.84, p = .035$] but not for Electrode [$F(1,34) = 1.21, p = .277$]. The maximum oERN amplitudes was at Cz (-1.38 μV). Although there was a main effect for Group [$F(1,34) = 6.60, p = .015$], the Group × Correctness interaction failed to reach significance [$F(1,34) < 1, p = .936$].[1,2] The latency of the oERN did not differ between the control and the psychopathic group [227 msec; $F(1,34) < 1, p = .548$].

---

[1]An alternative analysis suggested by one of the reviewers also included Condition as a WS-factor. As expected, a main effect for Condition [$F(1,34) = 23.1, p < .001$] and an interaction for Group × Condition [$F(1,34) = 4.68, p = .038$] were found. Further examination revealed reduced overall amplitudes in the Observe condition for the psychopathic group [$F(1,34) = 6.60, p = .015$]. Importantly, the 3-way interaction of Group × Condition × Correctness was not significant [$F(1,34) < 1, p = .782$], thus confirming the findings obtained in our initial analyses.

[2]The obtained results are not due to a potential confounding difference in N2 amplitude between the patient and control groups, because recent results have shown normal N2 amplitudes in the psychopathic population in an oddball paradigm (Brazil et al., in prep).
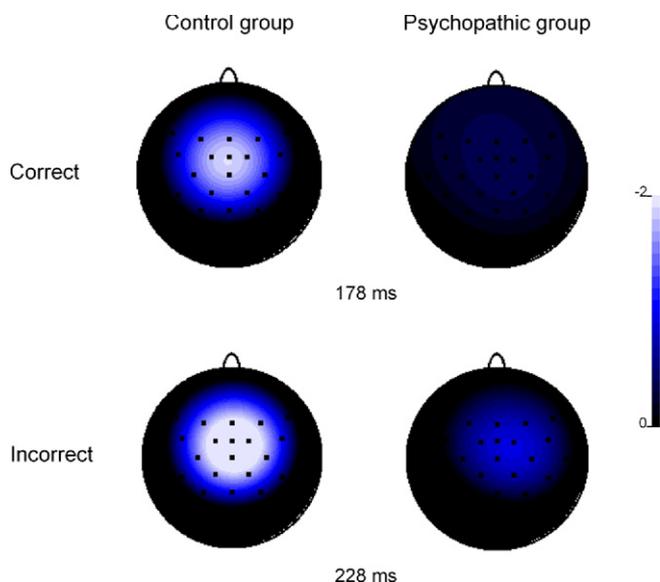
Figure 3. Scalp topographies of the peak amplitudes of the correct and incorrect waveforms for each group (*n* = 18) in the Observe condition. Light-colored shades indicate negative polarities, and darker shades depict more positive polarities.

LRP

Figure 4 depicts the LRPs from both groups in the Perform condition. As expected, LRPs just peaked before the response showing opposite sign amplitudes for correct and incorrect responses (32,15). An analysis of variance on peak LRP amplitudes showed that the difference between correct (-8.5 μV) and incorrect (7.1 μV) waveforms was significant [$F(1,34) = 181, p < .001$] but did not differ between groups [main effect of Group: $F(1,34) = 2.06$, $p = .16$; Group × Correctness interaction: $F(1,34) < 1, p = .403$].

The LRPs from both groups in the Observe condition peaked at or just after the response of the actor was recorded by the response device (Figure S1 in Supplement 1). As in the Perform condition, there was a significant difference between correct (-3.2 μV) and incorrect (1.9 μV) LRP peak amplitudes, as shown by a main effect of Correctness [$F(1,34) = 71.9, p < .001$]. Again, these effects were similar for both groups [main effect of Group: $F(1,34) = 1.04, p = .314$; Group × Correctness interaction: $F(1,34) < 1, p = .381$].

Correct and incorrect LRPs differed significantly from one another at -152 msec (relative to the response) in the control subjects and at -120 msec in the patients in the Perform condition. During observation, correct and incorrect LRPs first differed significantly from one another 182 msec and 174 msec after the response was registered in control subjects and patients, respectively. A WS analysis of variance on the LRP peak latencies with factors Condition, Correctness, and Group showed main effects of Condition [$F(1,34) = 641, p < .001$], Correctness [$F(1,34) = 10.9, p = .002$], and a Condition × Correctness interaction [$F(1,34) = 29.8, p < .001$], reflecting: 1) that LRPs peaked before the response in the Perform condition but only after the response was observed in the Observe condition, and 2) that the LRP peak latency was modulated by correctness in the Observe condition only. These effects indicate that LRPs in the Observe condition are due to the observation of the action rather than covert task performance.

Additional Analyses

The stimulus-locked P3 peak amplitudes did not differ between the two groups [$F(1,34) < 1, p = .423$]. No correlations were found between the PCL-R scores and oERN amplitudes at Cz or between the factor scores and the oERN (all *p* values > .35).

Discussion

The main goal of the present study was to dissociate monitoring of own and others' actions in psychopathic individuals. Our results show that, although there were no deficits in rERN in psychopathy, monitoring the outcome of another individual's responses is compromised in this disorder, as indicated by reduced ERPs after the observation of both correct and incorrect outcomes.

Although the rERN can be regarded as the result of a cognitive mechanism relying completely on internal processes, the oERN is a reflection of a mechanism additionally reliant on external processes. Monitoring the outcome of others' actions during social interaction requires the integration of information from different modalities and external sources into an own internal representation of the action. Although the latter aspect of action monitoring during observation is deficient in psychopathy, both automatic motor preparation—as indexed by the observed LRP—and stimulus processing—as indexed by the P3—are unaffected. Together these results provide robust neurophysiological corroboration of previous suggestions that psychopathy is associated with disorders in the processing of social information (2,33).
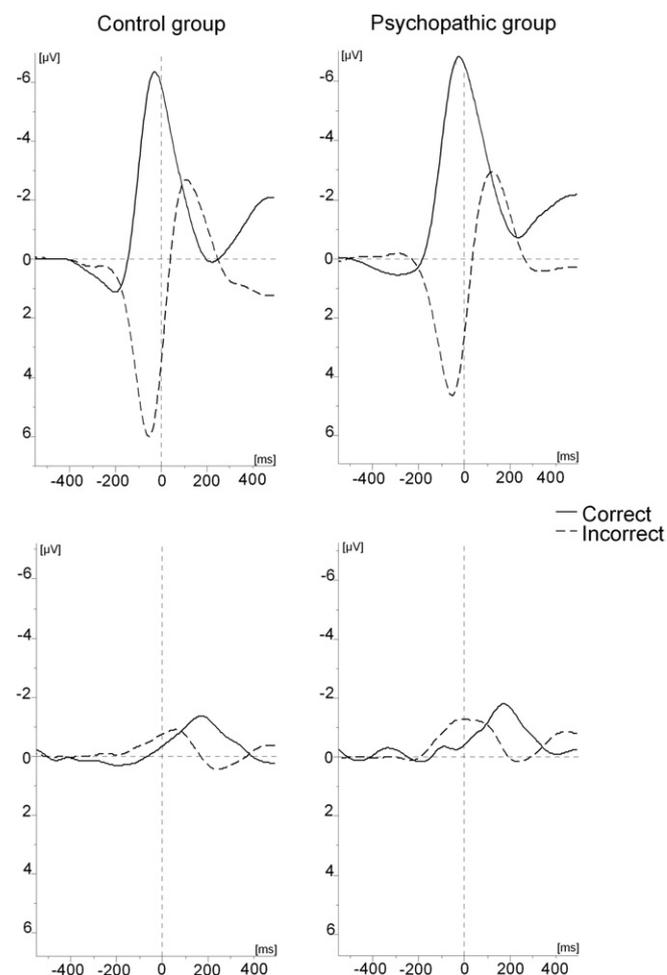

Figure 4. Grand average response-locked lateralized readiness potentials for correct and incorrect responses in the Perform condition (top row) and the Observe condition (bottom row) for the control (*n* = 18) and the psychopathic (*n* = 18) groups.

Behaviorally, action execution was unimpaired in psychopathic patients. Both groups showed the expected congruency effects, comparable accuracy levels, and post-error slowing. Post-error slowing is a cautionary response strategy, in which participants slow down their responses on trials after incorrect trials (34). Psychopathic subjects did not show slower RTs, in contrast to previous studies (12,13). The presence of the observer during task execution might have led to faster RTs in the psychopathic group, as predicted by the drive theory of social facilitation (35).

The rERNs were of similar amplitudes in both groups, in line with prior reports, showing unimpaired monitoring of one's own actions in psychopathy (13). Results obtained during the observation of the actions of others are more complicated. Initial automatic processing of others' actions in terms of the identity of the response (left or right joystick movement) was unimpaired in patients, as indicated by a normal pattern of oLRPs reflecting correct processing of the kinematic properties of the observed actions. The oLRPs on incorrect trials initially developed in the same direction as for correct responses but showed deactivation after observation of the incorrect responses. The deactivation points out that participants did not commit the errors themselves covertly. Thus, automatic processing of the observed action is similar to healthy control subjects.

In contrast, deeper processing of the consequences of the action, as indexed by the oERN, shows a different pattern in patients compared with control subjects. Psychopathic patients showed diminished oERNs after observing errors. Surprisingly, brain potentials were also reduced after observation of correct responses. In other words, the psychopathic group showed an overall reduced neural response for the outcome of observed actions but still differentiated between observed correct and incorrect responses. These findings suggest a broader deficiency in processing the consequence of others' actions rather than abnormal processing of observed errors only.

The ERN has been linked to reward-based learning mechanisms (8), but monitoring of correct behavior is also crucial to optimize gains. Recent insights point out that an organism's learning and adaptation rate is driven by the value of the information that becomes available with the outcome of each action (36,37). Thus, both incorrect and correct responses can be regarded as useful cues for behavioral adaptation through their informative value. The reduced neural activity after the observation of both correct and incorrect outcomes in psychopathy is a clear indicator that performance monitoring is disturbed in social contexts, and we believe this might play an important role in the abnormal acquisition of social behavior. More specifically, our results suggest that psychopathic individuals are less able to process observed cues in social settings, leading to reduced availability of usable information about outcomes. Consequently, the association of the outcome of a specific observed action to the action itself could be compromised. Thus, deficient processing of this type of social cue (human action) might be the first stage where "things go wrong" during action observation in psychopathy, probably also altering subsequent stages of behavioral adaptation and social learning.

An alternative interpretation is that psychopathic individuals would simply care less about others' actions, especially in a neutral context in which the observed actions had no direct consequences for themselves. However, we do not believe that a lack of motivation was the driving force behind our results. If the psychopathic subjects were less motivated, they would be expected to miss more errors in the observe condition and subsequently report significantly fewer errors compared with control subjects. This was not the case, as evident by comparable accuracy between the groups in the amount of observed errors reported. Additionally, the analyses

of the stimulus-locked P3 amplitudes in the observe condition did not show any significant group differences, indicating that the psychopathic group paid attention and processed the stimuli equally well and that our results cannot be attributed to a more general stimulus-processing deficiency. Nonetheless, it would be interesting to address this issue more in future research by, for example, introducing an evaluation of the observed action on a trial-to-trial basis or by introducing dependency between the observer and the actor. The first will not only enable an objective measure of accuracy in the observation condition. It will also allow for investigating whether any observed error-related processes are reflected in the ERPs after the observation of correct actions and also the functional significance of the reduced amplitude of the negativity during observation of correct responses in patients. Dependency can be achieved by making the outcome of the response have relevant consequences for the observer, such as monetary loss or reward in a cooperative or competitive context. These manipulations would allow for more objective measures of attention and motivation in future studies.

A limitation of the present study is that it did not include any measures of learning; therefore the actual relationship between the reduced signals and using them to learn and adapt behavior through observation is not made evident by our results. There has been only one study on the electrophysiological correlates of external feedback cues and learning in psychopathy (38). The results showed that, although psychopathic participants elicited normal electrophysiological responses to external feedback, they were less able to learn by using negative feedback optimally in a computerized reinforcement learning task. Thus, in such settings, using negative signals to adapt behavior seems compromised. Although the latter study was focused on processing of nonsocial external error cues, our findings suggest that in social settings, which are also more complex by nature, both negative and positive external cues are processed deficiently in psychopathy.

Conclusions

In conclusion, the current study demonstrates that psychopathic individuals show unaffected monitoring of own performance but specifically show altered processing of others' action-outcomes. The impact of the latter is more likely to be reflected in behavior during daily social situations, which are obviously richer in nature and more complex than the task currently used. This alteration might play an important role in the acquisition of disturbed social behavior in psychopathic offenders. As previous studies have demonstrated that healthy individuals learn from both positive and negative feedback (37) and that posterior medial frontal cortex plays a crucial role in these learning processes (39), the current study might provide support for disturbed observational learning in social contexts in psychopathic individuals. Obviously, future studies should address this question more directly to investigate how specific these disturbances are in psychopathy. Finally, the results show a potential new direction for future investigations of performance monitoring in clinical populations, particularly in psychiatric disorders characterized by severe social deficits, like autism and schizophrenia.

1. Hare RD, Hart SD, Harpur TJ (1991): Psychopathy and the DSM-IV criteria for antisocial personality disorder. *J Abnorm Psychol* 100:391–398.
2. Blair RJR (2007): The amygdala and ventromedial prefrontal cortex in morality and psychopathy. *Trends Cogn Sci* 11:387–392.
3. Yu R, Zhou X (2006): Brain responses to outcomes of one's own and other's performance in a gambling task. *Neuroreport* 17:1747–1751.
4. Falkenstein M, Hohnsbein J, Hoormann J, Blanke L (1990): Effects of errors in choice reaction tasks on the ERP under focused and divided attention. In: Brunia CHM, Gaillard AWK, Kok A, editors. *Psychophysiological Brain Research*. Tilburg, The Netherlands: Tilburg University Press, 192–195.
5. Gehring WJ, Goss B, Coles MGH, Meyer DE, Donchin E (1993): A neural system for error detection and compensation. *Psychol Sci* 4:385–390.
6. Holroyd CB, Nieuwenhuis S, Yeung N, Nystrom L, Mars RB, Coles MGH, Cohen JD (2004): Dorsal anterior cingulate cortex shows functional magnetic resonance imaging response to internal and external error signals. *Nat Neurosci* 7:497–498.
7. Ridderinkhof KR, Ullsperger M, Crone EA, Nieuwenhuis S (2004): The role of the medial frontal cortex in cognitive control. *Science* 306:443–447.
8. Holroyd CB, Coles MGH (2002): The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychol Review* 109:679–709.
9. 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.
10. Dikman ZV, Allen JJB (2000): Error-monitoring during reward and avoidance learning in high- and low-socialized individuals. *Psychophysiology* 37:43–54.
11. Hall JR, Bernat EM, Patrick CJ (2007): Externalizing psychopathology and the error-related negativity. *Psychol Sci* 18:326–333.
12. Munro GE, 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.
13. Brazil IA, de Bruijn ERA, Bulten BH, von Borries AKL, van Lankveld J, Buitelaar JK, Verkes RJ (2009): Early and late components of error monitoring in violent offenders with psychopathy. *Biol Psychiatry* 65:137–143.
14. Van Overwalle F (2008): Social cognition and the brain: A meta-analysis. *Hum Brain Mapp* 30:829–858.
15. Flanagan JR, Johansson RS (2003): Action plans used in action observation. *Nature* 424:769–771.
16. Iacoboni M, Woods RP, Brass M, Bekkering H, Mazziotta JC, Rizzolatti G (1999): Cortical mechanisms of human imitation. *Science* 286:2526.
17. van Schie HT, Mars RB, Coles MGH, Bekkering H (2004): Modulation of activity in medial frontal and motor cortices during error observation. *Nat Neurosci* 7:549–554.
18. Koelewijn T, van Schie HT, Bekkering H, Oostenveld R, Jensen O (2008): Motor-cortical beta oscillations are modulated by correctness of observed action. *Neuroimage* 40:767–775.
19. Bates AT, Patel TP, Liddle PF (2005): External behavior monitoring mirrors internal behavior monitoring: Error-related negativity for observed errors. *J Psychophysiol* 19:281–288.
20. Miltner WHR, Brauer J, Hecht H, Trippe R, Coles MGH (2004): Parallel brain activity for self-generated and observed errors. In: Ullsperger M, Falkenstein M, editors. *Errors, Conflicts, and the Brain: Current Opinions on Performance Monitoring*. Leipzig: Max Planck Institute for Cognitive Neuroscience, 124–129.
21. De Bruijn ERA, de Lange FP, von Cramon DY, Ullsperger M (2009): When errors are rewarding. *J Neurosci* 29:12183–12186.
22. Shane MS, Stevens M, Harenski CL, Kiehl KA (2008): Neural correlates of the processing of another's mistakes: A possible underpinning for social and observational learning. *Neuroimage* 42:450–459.
23. Schmand B, Bakker D, Saan R, Louman J (1991): The Dutch reading Test for Adults: A measure of premorbid intelligence level. *Tijdschr Gerontol Geriatr* 22:15.
24. Hildebrand M, De Ruiter C, Nijman H (2004): PCL-R psychopathy predicts disruptive behavior among male offenders in a Dutch forensic psychiatric hospital. *J Interpers Violence* 19:13–29.
25. Rasmussen K, Storsæter O, Levander S (1999): Personality disorders, psychopathy, and crime in a Norwegian prison population. *Int J Law Psychiatry* 22:91–97.
26. 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: LUMC.
27. 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 Publishing Group.
28. 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.
29. 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.
30. Coles MGH, Scheffers MK, Holroyd CB (2001): Why is there an ERN/Ne on correct trials? Response representations, stimulus-related components, and the theory of error-processing. *Biol Psychol* 56:173–189.
31. Coles MGH (1989): Modern mind-brain reading: Psychophysiology, physiology, and cognition. *Psychophysiology* 26:251–269.
32. Schmitt BM, Münte TF, Kutas M (2000): Electrophysiological estimates of the time course of semantic and phonological encoding during implicit picture naming. *Psychophysiology* 37:473–484.
33. Raine A, Yang Y (2006): Neural foundations to moral reasoning and antisocial behavior. *Soc Cognit Affect Neuroscience* 1:203.
34. Rabbitt PM (1966): Errors and error correction in choice-response tasks. *J Exp Psychol* 71:264–272.
35. Zajonc RB (1965): Social facilitation. *Science* 149:269–274.
36. Walton ME, Devlin JT, Rushworth MFS (2004): Interactions between decision making and performance monitoring within prefrontal cortex. *Nat Neurosci* 7:1259–1265.
37. Rushworth MFS, Behrens TEJ (2008): Choice, uncertainty and value in prefrontal and cingulate cortex. *Nat Neurosci* 11:389–397.
38. von Borries AKL, Brazil IA, Bulten BH, Buitelaar JK, Verkes RJ, de Bruijn ERA (in press): Neural correlates of error-related learning deficits in individuals with psychopathy. *Psychol Med*:1–10.
39. Hester R, Barre N, Murphy K, Silk TJ, Mattingley JB (2008): Human medial frontal cortex activity predicts learning from errors. *Cereb Cortex* 18:1933–1940.