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Neurophysiological correlates of error monitoring and inhibitory processing in juvenile violent offenders

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ABSTRACT

Performance monitoring is crucial for well-adapted behavior. Offenders typically have a pervasive repetition of harmful-impulsive behaviors, despite an awareness of the negative consequences of their actions. However, the link between performance monitoring and aggressive behavior in juvenile offenders has not been closely investigated. Event-related brain potentials (ERPs) were used to investigate performance monitoring in juvenile non-psychopathic violent offenders compared with a well-matched control group. Two ERP components associated with error monitoring, error-related negativity (ERN) and error-positivity (Pe), and two components related to inhibitory processing, the stop-N2 and stop-P3 components, were evaluated using a combined flanker-stop-signal task. The results showed that the amplitudes of the ERN, the stop-N2, the stop-P3, and the standard P3 components were clearly reduced in the offenders group. Remarkably, no differences were observed for the Pe. At the behavioral level, slower stop-signal reaction times were identified for offenders, which indicated diminished inhibitory processing. The present results suggest that the monitoring of one's own behavior is affected in juvenile violent offenders. Specifically, we determined that different aspects of executive function were affected in the studied offenders, including error processing (reduced ERN) and response inhibition (reduced N2 and P3). However, error awareness and compensatory post-error adjustment processes (error correction) were unaffected. The current pattern of results highlights the role of performance monitoring in the acquisition and maintenance of externalizing harmful behavior that is frequently observed in juvenile offenders.

1. Introduction

Crime and violent behavior continues to be a significant problem in Western societies despite investments in implementing delinquency-prevention programs and other education interventions (Greenwood, 2008). Despite the impact of criminal behavior, few neuroscientific studies have examined the role of cognitive control mechanisms in the regulation of violent behavior using fine-grained electroencephalographic measures (Event-Related Brain potentials, ERPs). These measures allow a very accurate evaluation of certain cognitive control processes such as error monitoring and inhibitory processing, by tracking specific ERP components that tap into their neural dynamics. At the temporal level these measures are very reliable and might allow a better characterization of the role of interindividual variability in certain cognitive control processes that could explain the association previously observed between cognitive control and aggressive-violent behavior (Blair et al., 2006, 2007; Giancola, 2004; Hoaken, Shaughnessy, & Pihl, 2003; Krakowski, 2003; Krämer et al., 2007; Krämer, Kopyciok, Richter, Rodriguez-Fornells, & Münte, 2011; LeMarquand et al., 1998). A common explanation for this relationship is that low values of cognitive control might be associated to a lack of capacity to control aggressive behavior, highlighting the important role of inhibitory processing in the regulation of violent behavior (Gorenstein & Newman, 1980). Another core aspect

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of cognitive control that has recently attracted a lot of attention is error monitoring, especially its relationship to individual differences in aggressive behavior (Brazil et al., 2009; Dikman & Allen, 2000; Krämer et al., 2011; Munro et al., 2007a). In the present study, we investigated both core aspects of cognitive control, error monitoring and inhibitory processing, in a selected sample of juvenile violent offenders using ERPs. In legal terms, the word offender refers to an individual who violates or transgresses the law and is often linked to violent behavior. Specifically, the term juvenile offender refers to an individual who has not yet reached adulthood (age range of 15–20 years old). This period between 15 and 20 years is critical for the development of cognitive control processes primarily because relevant prefrontal cerebral structures attain their neural maturation during this time (Diamond, 2002; Segalowitz & Dywan, 2009).

A central function of cognitive control is to monitor and regulate our behavior. Thus, an important aspect of cognitive control is the self-regulation of our own performance, which comprises several processes such as the constant monitoring of our actions, detection of conflict, implementation of cognitive control mechanisms after conflict-detection or error-commission and subsequent behavioral adjustments (Logan, 1985; Rabbitt & Rodgers, 1977; Ridderinkhof, van den Wildenberg, Segalowitz, & Carter, 2004). These crucial functions are supported, to a great extent, by the prefrontal cortex and, more specifically, by the medial prefrontal cortex (MPFC), which includes the anterior cingulate cortex (ACC), the inferior frontal gyrus (IFG), the dorsolateral prefrontal cortex (DLPFC), and the insular cortex (Carter, Braver, Barch, Botvinick, Noll, & Cohen, 1998; Gehring & Knight, 2000; Krämer et al., 2007; Marco-Pallarés, Camara, Münte, & Rodríguez-Fornells, 2008; Ullsperger & von Cramon, 2001).

As a component of the performance monitoring system, *error detection* plays a critical role in action regulation and cognitive control, which are critical processes of correct socialization and adaptive behavior (Logan, 1985; Rabbitt & Rodgers, 1977; Ridderinkhof et al., 2004). A negative event-related potential (ERP), labeled error-related negativity (ERN, or Ne), has been shown to appear immediately after committing errors (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1990; Gehring, Goss, Coles, Meyer, & Donchin, 1993; Gehring, Coles, Meyer, & Donchin, 1995). The dopaminergic system from the basal ganglia to the MPFC (including the ACC) plays a key role in the generation of the ERN (Yeung, 2004). The ERN component exhibits a clear fronto-central topographical distribution; it peaks at approximately 60–80 ms after error commission and has been associated with the commission of errors and the processing of negative feedback (Holroyd & Coles, 2002; Yeung, 2004). According to the error detection theory (Falkenstein, Hohnsbein, Hoorman, & Blanke, 1991; Gehring et al., 1993) and the conflict monitoring theory (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Carter et al., 1998; Yeung, Botvinick, & Cohen, 2004), the ERN can be considered a reliable index of performance monitoring, thereby reflecting the output of a general evaluative system concerned with the motivational significance of the outcomes of our actions (reinforcement-learning theory of the ERN; Holroyd & Coles, 2002). It has been suggested that theta oscillatory activity recorded at frontal midline electrodes may be the electrophysiological mechanism that underlies the ERN (Cavanagh, Cohen, & Allen, 2009; Luu, Tucker, & Makeig, 2004; Trujillo & Allen, 2007). After the appearance of the ERN, a positive ERP component is observed (error positivity, Pe) which exhibits a centro-parietal distribution that peaks at approximately 200–600 ms after the error (Ullsperger, Harsay, Wessel, & Ridderinkhof, 2010). Despite the lack of a consensus regarding the specific functional significance of the Pe, it has been argued that Pe might reflect the following: (i) error awareness (Leuthold & Sommer, 1999; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001, Falkenstein, 2004), (ii) a motivational significance or emotional assessment of an error (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Overbeek, Nieuwenhuis, & Ridderinkhof, 2005; Ridderinkhof, Ramautar, & Wijnen, 2009), and (iii) an orienting response to an error commission (Arbel & Donchin, 2009, 2011; Davies, Segalowitz, Dywan, & Pailing, 2001; Hajcak, McDonald, & Simons, 2003; Overbeek et al., 2005; Ridderinkhof et al., 2009). Therefore, it appears that the Pe is primarily modulated by conscious error detection and the corresponding adjustments observed in future responses (Hajcak et al., 2003; Nieuwenhuis et al., 2001).

Few studies have been devoted to investigating error monitoring in adult offenders, with most studies focused on adult psychopathic violent offenders. However, to our knowledge, no previous studies have focused on juvenile non-psychopathic offenders. In adult offenders, Munro et al. (2007a) reported no amplitude differences for the ERN and Pe between offenders and healthy controls in a letter-flanker task. However, these authors encountered a reduced ERN amplitude in the face-flanker task for offenders (when psychopathic and non-psychopathic offenders were combined) compared with healthy controls. In contrast, Brazil et al. (2009) found no amplitude differences for the ERN, but they found decreased Pe amplitude for adult psychopathic violent offenders compared with healthy controls. Moreover, using measures of dispositional dimensions related to delinquency, some authors have reported only reduced ERN amplitude, but not reduced Pe amplitude, in adults with poor sociability scores (Dikman & Allen, 2000) and high scores in the personality trait of externalization (Hall, Bernat, & Patrick, 2007).

The spectrum of externalizing behaviors has been related to different personality traits associated with violent or offensive behavior. The core trait of the externalizing spectrum (Patrick & Bernat, 2006; Iacono, Carlson, Malone, & McGue, 2002) is the difficulty in inhibiting inappropriate responses or impulses (Gorenstein & Newman, 1980). Interestingly, compared with healthy control children, a reduced ERN amplitude was encountered in children with poor sociability (Santesso, Segalowitz, & Schmidt, 2005) and with high externalizing symptomatology (Stieben et al., 2007). In a more recent study, Bernat, Nelson, Steele, Gehring, and Patrick (2011) failed to identify differences in the feedback-related negativity (FRN, or theta oscillatory activity) in a comparison between high and low externalizing undergraduate students. This ERP component is an index of external performance monitoring (i.e., feedback related information regarding the outcome of an action), which is thought to be highly associated with the ERN component (Holroyd & Coles, 2002). Considering this pattern of results, Bernat et al. (2011) proposed that high externalizing individuals might have deficits in the endogenous (internally cued) performance monitoring signals (ERN) but not in exogenous (externally cued) performance monitoring (FRN).

Another cognitive control aspect that is very important in aggressive behavior is the ability to inhibit or avoid certain behaviors or thoughts (Gorenstein & Newman, 1980; Logan, 1994). Inhibitory processes have been studied using different electrophysiological measures and typically use the go/nogo or stop-signal tasks (Krämer et al., 2007; Logan, 1994; Rodríguez-Fornells, Kurzbuch, & Münte, 2002). For example, the stop-N2 component (with a fronto-central topographical distribution that peaks approximately 250–350 ms after the target to inhibit) is related to conflict detection, inhibition or revision of inappropriate response tendencies (Kok, 1986). Similar to the ERN component, the MPFC plays a critical role in the generation of the stop-N2 (Amodio, Master, Yee, & Taylor, 2008; Bekker, Kenemans, & Verbaten, 2005; Bokura, Yamaguchi, & Kobayashi, 2001; Gründler, Cavanagh, Figueroa, Frank, & Allen, 2009; Jonkman, Sniedt, & Kemner, 2007; Mathalon, Whitfield, & Ford, 2003; Ullsperger & von Cramon, 2001;

Van Veen & Carter, 2002a, 2002b). As postulated for the ERN, some authors have suggested that the observed theta oscillatory activity in stop trails reflects inhibitory processing in N2 (Harmony, Alba, Marroquín, & González-Frankenberger, 2009; Kirmizi-Alsan et al., 2006). Additionally, Cavanagh, Zambrano-Vázquez, and Allen (2012) claimed that mid-frontal theta activity could be a generic processing mechanism that coordinates endogenous and exogenous performance-relevant information for monitoring actions. The theta activity would reflect a principal and common neural activity for the different ERP components of action monitoring, such as the N2, the ERN, the FRN (feedback-related negativity), and the CRN (correct-response negativity). Following the stop-N2 component, the stop-central-P3 can be identified, with a fronto-central topographical distribution that peaks approximately 300–500 ms after the target to be inhibited. The stop-P3 has been associated with inhibitory processing (Bruin, Wijers, & Staveren, 2001). Only few studies have focused on studying the inhibitory function in a population of offenders using EPRs, and their findings are contradictory. Chen, Tien, Juan, Tzeng, and Jung (2005) using a go/nogo task, identified a reduced N2 amplitude in high-impulsive-violent offenders, but they did not find differences in the amplitude of the P3. On the contrary, Munro et al. (2007b) found opposite results using a similar go/nogo task. They failed to identify a reduced N2 amplitude, although they found a reduced amplitude for the P3. The P3 has also been largely studied in relation to externalizing and aggressive behavior. For example, Bernat et al. (2011) found a reduced P3 amplitude in feedback processing for high externalizing students. This reduction in the P3 amplitude has also been previously reported in individuals with aggressive behavior performing an oddball task (Patrick, 2008) and is considered a link between aggressive behavior and ERPs.

In the present study, we aimed to evaluate, for the first time, whether performance monitoring and inhibitory processing are altered in juvenile violent offenders. From the previous review, it is clear that there are inconsistent findings between psychopathic and non-psychopathic offenders in adult samples. To carefully understand the neurobiological substrates of aggressive behaviors, it is important to consider separately the individuals who exhibit the core-affective features of psychopathy from other types of violent non-psychopathic offenders (Patrick, 2008). Thus, we restricted our sample selection to *juvenile non-psychopathic violent offenders* and focused on different electrophysiological components associated to performance monitoring and inhibitory processing (Folstein & Van Petten, 2008). With this aim, we used a combined flanker-stop-signal paradigm that has been extensively used to investigate error detection/correction (Gehring et al., 1993; Krämer et al., 2007; Rodríguez-Fornells et al., 2002) and inhibitory processes (Krämer et al., 2007; Liotti, Pliszka, Perez, Kothmann, & Woldor, 2005; Pliszka, Liotti, & Woldorff, 2000).

Previous neuroimaging studies conducted in adults have identified lower ERN amplitudes, but no difference in Pe in individuals with poor sociability (Dikman & Allen, 2000) or individuals with high scores in the externalization dispositional dimension (Hall et al., 2007) compared with controls. Based on these results, we expected to observe reduced ERN amplitude in juvenile violent offenders, but no differences in the Pe component. We specifically used theta-filtered ERPs (Bernat et al., 2011) to better isolate ERN oscillatory activity (Cavanagh et al., 2009; Luu et al., 2004; Trujillo & Allen, 2007), expecting similar results in this analysis as in the standard ERN analysis. Notice that the Pe is partially associated with theta and delta oscillatory activity (Cavanagh et al., 2012), but as for the standard ERPs, we did not expect differences in this component between groups in theta- and delta-filtered data.

Furthermore, there is no clear evidence of an association with diminished response inhibition in adult offenders. Chen et al. (2005) identified reduced N2 amplitude in high-impulsive violent offenders, whereas Munro and colleagues (2007b) found no differences between offenders and controls. Therefore, the current evidence regarding response inhibition and violent offenders are not sufficient to enable clear predictions for the evaluated sample in the current study. In addition and consistent with a large set of experimental data (Bernat et al., 2011; Iacono et al., 2002; Munro et al., 2007b; Patrick, 2008; but see Chen et al., 2005), diminished amplitude of the P3 component (stop-P3 and stimulus-related P3) was expected in violent offenders.

2. Methods

All procedures were approved by the local ethics committee of the university, and written informed consent was obtained from all participants.

2.1. Participants

Juvenile male offenders ($N = 17$; mean age of 18.3; $SD = .3$) were recruited for the study from the Juvenile Justice and Educative Center of Girona, a youth detention center. The juvenile violent offenders were inmates at the center and were serving time for violent crimes; they were incarcerated because of extremely offensive violent behavior according to the jurisdiction of a Spanish juvenile court. The average sentence length of the participants in the study was 19.79 months ($SD = 9.76$). The total number of convictions for all participants in the offender group included the following number of convictions: 38 robberies (88% of offenders had committed a robbery), 32 thefts (64%), 27 injuries (58%), 22 larcenies (47%), 14 threats (52%), 11 motor vehicle thefts (44%), 6 resistance to disobedience of an individual in authority (29%), 7 property damage (23%), 5 direct assaults against individuals in authority or their agents (29%), 4 unauthorized possession of firearms (23%), 4 libels (23%), 3 disorderly conduct (17%), 3 coercion (17%), 2 vandalism (11%), 2 domestic abuse (11%), 2 violation of court order (11%), 1 unjust vexation (5.8%), 1 sexual assault (5.8%), and 1 homicide (5.8%). Importantly, all participants were diagnosed with non-psychopathic conduct disorder by the staff at the center. The sex-, age- and IQ-matched control participants ($N = 17$; mean age of 18.6; $SD = .3$) were recruited from the University of Girona. All participants included in both groups had normal IQs (less than ± 1 S.D., i.e., scores between 85 and 115; see Table 1), had no previous history of severe head injury and were free from drug abuse or intake of psychiatric medication.

2.2. Personality battery and IQ testing

Each participant completed the Spanish version of the following questionnaires: (i) the I7 Questionnaire (Eysenck, Pearson, Easting, & Allsopp, 1985; Luengo, Carrillo-De-La-Pena, & Otero, 1991), which is a measure of impulsiveness and risk-taking; (ii) the Aggression Questionnaire (Andreu, Peña, & Graña, 2002; Buss & Perry, 1992), which is a measure of aggressiveness (total AQ score) that includes the subscales Physical Aggression (AQ-PA), Verbal Aggression (AQ-VA), Anger (AQ-A) and Hostility (AQ-H); (iii) the Cognitive Failures Questionnaire (CFQ; Broadbent, Cooper, FitzGerald, & Parkes, 1982; Garcia & Sanchez-Canovas, 1994), which is a measure of the proneness to committing cognitive slips and errors (e.g., failures of memory, action, and perception) in everyday life; (iv) the Revised NEO Personality Inventory (NEO-PI-R; Cordero, Pamos, & Seisdedos, 1999; McCrae & Costa, 2004), which was used to evaluate Conscientiousness facets (e.g., self-efficacy, orderliness, dutifulness, achievement striving, self-discipline and cautiousness); and (v) the dimensions of Conscientiousness and Emotional Stability (Rodríguez-Fornells, Lorenzo-Seva, & Andrés-Pueyo, 2001) of the Five Factor Personality Inventory (FFPI; Hendriks, Hofstee, & De Raad, 1999). Finally, (vi) the Raven's Progressive Matrices was used to assess IQ (Raven, 1989).

2.3. Procedure

Electrophysiological and psychometric testing was performed in an ERP research lab at the institution. The participants individually completed all tasks in a single session; they began with the psychometric testing and continued with the electrophysiological recording while performing an experimental task. We presented the participants with a modified variant of the Eriksen flanker task (Eriksen & Eriksen, 1974) that required them to respond, using the index finger of each hand, to the pointing direction (right or left) of a central arrow that appeared in the middle of a horizontal five arrows array (see Fig. 1). All four surrounding arrows were either compatible or incompatible with the direction of the central arrow, thus favoring performance errors in the incompatible condition (Krämer et al., 2007; Rodríguez-Fornells et al., 2002). The stimuli were presented in the middle of the screen at a viewing distance of 100 cm for 300 ms and with a stimulus asynchrony fixed to 900 ms. In 33.3% of the trials, the direction of the central arrow was compatible with the direction of the surrounding arrows, whereas in 50% of the trials, the direction was incompatible. The remaining 16.6% of the trials consisted of no-go trials, which followed a variant of a stop-signal paradigm (Band, van der Molen, & Logan, 2003). In stop trials, the central green arrow changed to red after a variable delay, which

Table 1
Personality mean and IQ scores and *t*-test comparisons between groups.

	Students		Juvenile offenders		*t*	*p*	*d*
	M	SD	*M*	SD			
Impulsiveness	5.88	3.84	13.82	4.46	**−5.46**	**0.000**	**−1.907**
Risk taking	9.00	3.87	9.59	3.59	−0.55	0.589	−0.158
Aggression total – AQ	66.18	14.67	98.94	13.34	**−6.38**	**0.000**	**−2.336**
Physical aggression	18.00	2.50	34.00	6.29	**−9.40**	**0.000**	**−3.342**
Verbal aggression	13.18	3.24	16.06	3.44	**−2.15**	**0.047**	**−0.861**
Anger	17.41	4.96	25.18	4.19	**−4.49**	**0.000**	**−1.692**
Hostility	17.59	5.99	23.71	4.31	**−3.23**	**0.005**	**−1.172**
CFQ Total	36.59	12.35	40.82	8.46	−1.26	0.230	−0.399
Conscientiousness – NEO-PI-R	159.88	19.94	124.53	14.34	**4.97**	**0.000**	**2.035**
Self-efficacy	28.18	3.97	19.88	3.46	**6.12**	**0.000**	**2.230**
Orderliness	23.76	4.99	24.41	3.04	−0.40	0.693	−0.157
Dutifulness	28.76	5.91	22.47	3.61	**3.39**	**0.004**	**1.284**
Achievement striving	26.88	4.81	21.12	3.39	**3.35**	**0.004**	**1.384**
Self-discipline	26.12	4.09	20.82	2.88	**3.85**	**0.001**	**1.498**
Cautiousness	26.18	4.98	15.82	4.65	**5.39**	**0.000**	**2.150**
Conscientiousness – FFPI	67.18	7.88	56.59	8.68	**3.17**	**0.006**	**1.277**
Emotional stability – FFPI	73.47	7.32	54.94	10.35	**6.03**	**0.000**	**2.067**
IQ	113.47	9.31	111.47	7.28	0.70	0.490	0.239

The significant values ($p < 0.05$) are in bold type.

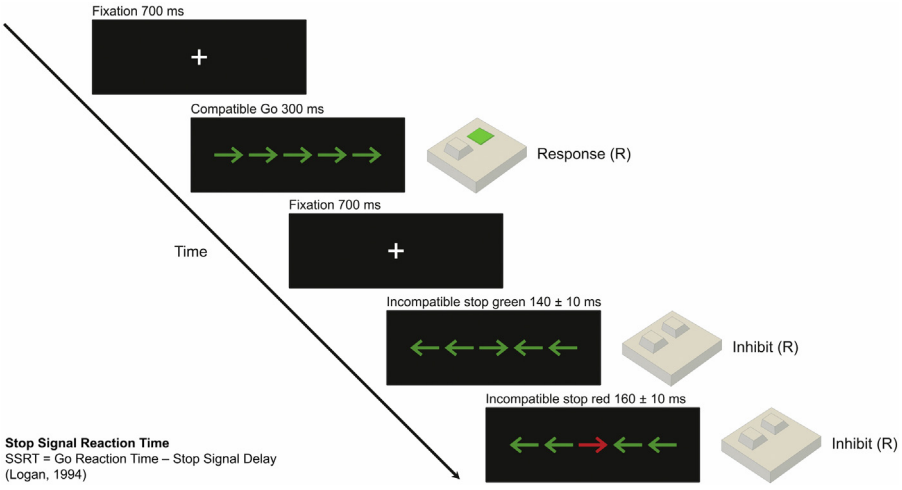

Fig. 1. Modified variant of the Eriksen flanker task (Eriksen & Eriksen, 1974) that required the participants to respond, using the index finger of each hand, to the pointing direction (right or left) of a central arrow from an array of five arrows. All four surrounding arrows were either compatible or incompatible with the central arrow. We presented 33.3% compatible trials and 50% incompatible trials. In the remaining 16.6%, we included no-go trials, which followed a variant of the stop-signal paradigm (Band et al., 2003). In these stop trials, the central green arrow changed to red after a variable delay, which indicated to the participants to inhibit the response. The delay was adapted to the participants' behavior by means of a staircase tracking algorithm (Band & van Boxtel, 1999). For go trials, only one green target stimulus (300 ms) was presented; for stop trials, two trials were presented, including the green trial (that started with 140 ms and was modulated by ±10 ms depending on the participants' responses) and the red trial (that started with 160 ms and was modulated by ±10 ms depending on the participants' responses). The stop-signal reaction time (SSRT) (Band et al., 2003) was subsequently calculated first individually and then averaged for each condition by subtracting the participant's mean stop-signal delay from the median reaction time of the correct go responses. (For interpretation of the references to color in text, the reader is referred to the web version of this article.)

indicated to the participant to withhold the response. The stop-signal delay was adapted to the participants' behavior by means of a staircase tracking algorithm (Band & van Boxtel, 1999). After a successful inhibition, the stop-signal delay was increased by 10 ms, which increased the difficulty of the inhibition. After an inhibitory failure, the stop-signal delay was decreased 10 ms, which made the inhibition easier. The stop-signal delay was initially set to 140 ms. This procedure was applied to yield an inhibition rate of 50%. The stop-signal reaction time (SSRT) (Band et al., 2003) was subsequently calculated individually for each participant and condition by subtracting the participant's mean stop-signal delay from the median reaction time (RT) of the correct go responses.

The participants received 20 training trials to enable them to become acquainted with the task. They were requested to immediately correct errors committed on the go trials, if possible. The experiment was divided into eight blocks, and each block comprised 240 trials, which resulted in 1920 trials.

2.4. Electrophysiological recording

Electroencephalogram (EEG) was recorded continuously (digitized with a sampling rate of 250 Hz, bandpass from 0.01 to 70 Hz) using SynAmp Neuroscan amplifiers from 29 tin electrodes that were mounted on an elastic cap and located at standard positions (FP1/2, F3/4, C3/4, P3/4, O1/2, F7/8, T3/4, T5/6, Fz, Cz, Pz, FC3/4, FT7/8, CP3/4, TP7/8, FCz, CPz). The EEG was referenced on-line to the right ocular canthus. Biosignals were re-referenced offline to the mean activity at the two mastoid processes. Electrode impedances were maintained below 5 kΩ. Vertical eye movements were monitored by an electrode placed below the right eye.

2.5. Data processing

The EEG rejection rate was similar between groups (controls 7.12 ± 5.02%; offenders 6.84 ± 5.59%; $t(32) = .2$, $p > .9$, $d = .05$). ERP averages were also obtained for the different conditions (time-range from −100 to 924 ms for stimulus-locked averages and from −400 to 600 ms for response-locked ERPs). In the stimulus-locked ERPs, the baseline referred to the 100 ms prior to the stimulus; for the response-locked ERPs, the baseline referred to the 50 ms prior to the button press. Epochs that exceeded ±100 μV in electrooculogram (EOG) or EEG were removed offline for further analysis using the extreme value function of the EEGlab toolbox. In the behavioral and ERP analyses, only the RT responses that were produced between 120 and 750 ms after the stimulus presentation were considered for the analyses (Krämer et al., 2007). All artifact-free error trials were included regardless of a subsequent correct response. A specific analysis on filtered data was conducted with the aim of better isolating the ERN/Pe components from the overlapping slow positive components in the correct and error trials. For this analysis, we filtered the ERP response-locked

averages within the theta band range that characterize the underlying oscillatory activity of these components (band-pass filtering, 3–9 Hz; see Luu & Posner, 2003; Luu et al., 2004; Marco-Pallarés et al., 2008; Bernat et al., 2011). Furthermore, we also low-pass filtered the waveforms to isolate delta oscillatory activity (delta range, low-pass filtering < 3 Hz) (see for a similar procedure Bernat et al., 2011). As for the response-locked analysis, we conducted a new filtered analysis to isolate the N2 component. For this analysis, we filtered the stop-locked ERPs averages within the theta band range that characterize the underlying oscillatory activity for this component (band-pass filtered, 3–9 Hz; see Kirmizi-Alsan et al., 2006; Harmony et al., 2009).

A time-frequency analysis of the electrophysiological activity elicited by choice-errors and correct responses was conducted (epochs that comprised ± 2000 ms around the response). Epochs that exceeded ±100 μV in the electrooculogram (EOG) or EEG were removed offline from further analysis using the EEGlab toolbox. A 100 ms time-range prior to the button press was defined as baseline. Single trial data were convoluted using a complex Morlet wavelet:

$$w(t, f_0) = (2\pi\sigma_t^2)^{-1/2} e^{-t^2/2\sigma_t^2} e^{2i\pi f_0 t}$$

with the relation (where) set to 6.7 (Tallon-Baudry, Bertrand, Delpuech, & Permier, 1997). Changes in time varying energy (i.e., the square of the convolution between the wavelet and signal) in the studied frequencies (from 1 to 40 Hz; linear increase) with respect to baseline were computed for each trial and averaged for each subject prior to the calculation of a grand average.

2.6. Data analysis

For all ERP data, ANOVAs with condition and electrode location (Fz, Cz, Pz) as the within-subject factors and group (controls, offenders) as the between-subject factor were conducted utilizing the Greenhouse–Geisser epsilon correction when necessary (Jennings & Wood, 1976); the corrected *P*-value is reported. The Cohen's *d* and *f* were used as a measure of the effect sizes for the *t*-tests and the ANOVAs, respectively (Cohen, 1992). Mean amplitude measures were measured at different time-windows (TW) for each ERP component of interest based on previous studies (Krämer et al., 2007; Rodríguez-Fornells et al., 2002) and visual inspection. Thus, to compute each TW, we used the peak-based method. We first localized the peak of a specific component on the grand average waveform and then defined a symmetric TW centered on the peak. The time-range of these TWs was set to 50 or 100 ms depending of the size of the component. Thus, the TWs for the response-locked ERP were: choice-ERN (35–85 ms), stop-ERN (65–115 ms), choice-Pe (120–220 ms), and stop-Pe (160–260 ms); for the stop-signal locked ERPs: stop-N2 (200–300 ms) and stop-P3 (300–350 ms); and N1 (100–150 ms) in the stimulus-locked ERPs. Because the offender group committed more errors compared with the control group, we re-analyzed the choice-error analysis and introduced the error rate as a covariate to discard that the decrement in the ERN amplitude observed in the offender group could be accounted for by the higher error rate.

To better isolate the differences between groups, we performed a set of different complementary rmANOVA. First, for the Pe component, we conducted a new set of analyses with an extended time-window of 150 ms mean amplitude (choice-Pe: 95–245 ms; stop-Pe: 135–285 ms) centered to the peak, a similar time-window as the one used in Brazil et al. (2009), despite they used a different method (rectified mean amplitude). Second, in order to discard the potential Pe amplitude and latency differences, we performed a peak-to-peak analysis between the groups, which contrasted the choice vs. stop-error trials. Similarly, we conducted an additional peak-to-peak analysis for the N2 component at the Fz electrode in the stop-triggered data, thus calculating the differences in amplitude between the P2 and the N2 peaks. Third, for the stimulus-locked data, we conducted a new peak-to-peak analysis between the N2 and the P3 components (TW control group: 280–380 ms; TW offender group: 300–400 ms). Finally, we conducted a final analysis to isolate the interference effect detected for the P3 component. The TW for the P3 was adapted accordingly to the latency differences between the groups (315–415 ms for the control group and 335–435 ms for the offender group).

3. Results

3.1. Personality assessment

Table 1 shows the comparison between the controls and offenders in the administered questionnaires. As expected, the mean scores on the Aggression Questionnaire were higher for the offenders compared with the controls in all evaluated dimensions. Furthermore, the offenders showed larger Impulsiveness scores compared with the controls, except for the Risk Taking dimension. In contrast, the offenders scored lower compared with the controls on all facets of the Conscientiousness scale of the NEO-PI-R (except for Orderliness) and on the Conscientiousness and Emotional Stability scales of the FFPI.

3.2. Behavioral results

Similar to previous findings in the flanker task, the participants responded faster in the compatible trials (347.2 ± 55.8 ms) compared with the incompatible trials (367.1 ± 58.6 ms) (main effect of condition: $F(1,32) = 99.7$, $p < .001$, $f = 1.8$). Erroneous responses (303.0 ± 51.1 ms) were faster compared with the correct responses (358.9 ± 57.0 ms) (main effect of condition: $F(1,32) = 220.1$, $p < .001$, $f = 2.6$). Importantly, a significant main effect of group ($F(1,32) = 7.7$, $p = .009$, $f = .5$) indicated that the offenders were slower in both the compatible and incompatible conditions.

Regarding performance quality, the participants committed less errors in the compatible (11.0 ± 7.0% for controls; 12.8 ± 9.1% for offenders) compared with the incompatible trials (22.7 ± 7.4% for controls; 20.3 ± 7.6% for offenders) (main effect of condition: $F(1,32) = 41.8$, $p < .001$, $f = 1.1$). The mean percentage of correct trials was larger for the control group compared with the offenders group ($F(1,32) = 12.4$, $p = .001$, $f = .6$). No group differences were identified for the corrected errors (70.5 ± 28.4% for controls; 60.5 ± 24.0% for offenders; $t(32) = 1.1$, $p > .2$, $d = .4$).

In agreement with previous findings on the flanker task, we observed slower RT in the correct trials after erroneous responses compared with the trials after correct responses (post-error-slowing effect, 8.1 ± 18.6 ms) (Rabbitt, 1966); the same result was identified in the trials following failed inhibitions (post-non-inhibition-slowing, 10.6 ± 29.0 ms). No group differences were detected for the post-error-slowing (6.9 ± 12.8 ms for controls; 9.4 ± 23.4 ms for offenders; $t(32) = -.4$, $p > .6$, $d = -.1$) or the post-non-inhibition-slowing effects (9.0 ± 15.3 ms for controls; 12.1 ± 38.8 ms for offenders; $t(32) = -.3$, $p > .7$, $d = -.1$).

Finally, the percentage of inhibited trials was 55.8 ± 8.5% for the controls and 51.8 ± 12.0% for the offenders with no differences between the groups ($t(32) = 1.1$, $p > .2$, $d = .4$). Importantly, the offenders (307.9 ± 49.2 ms) had larger SSRT compared with the control (276.6 ± 19.5 ms) participants ($t(32) = -2.5$, $p = .024$, $d = .8$), which reflects slower inhibitory processing for the offenders (Chen et al., 2005).

3.3. Event-related brain potentials (ERPs)

3.3.1. ERPs: response-locked data

As shown in Fig. 2a, both the errors in the go trials (choice-errors) and the errors in the no-go trials (stop-errors) led to an increased negativity that peaked approximately 50 ms after the error; this negativity was associated with a clear fronto-central distribution in both groups, which was identified as the ERN component (Falkenstein et al., 1990; Gehring et al., 1993).

3.3.1.1. Choice-errors, ERN. Visual inspection of Fig. 2a suggested a larger ERN amplitude for the control group compared with the offender group. The increased negativity after choice-errors, which characterizes the ERN component, was confirmed by the significant main effect of condition ($F(1,32) = 70.4$, $p < .001$, $f = 1.5$). Although the main effect of group was not significant ($F(1,32) = 1.2$, $p > .2$, $f = .2$), a significant interaction between the condition and group was observed ($F(1,32) = 9.5$, $p = .004$, $f = .6$), which reflected a larger ERN amplitude in the error condition for the control group compared with the offender group. Further pairwise *t*-test comparisons that contrasted the error vs. correct trials showed a reduced ERN for the offender group compared with the control group, with the largest difference encountered on the Cz electrode ($t(32) = -3.1$, $p = .004$, $d = -1.0$). For the choice-ERN, an additional analysis was performed that introduced the error rate as a covariate. The same result was obtained (main effect of group: $F(1,31) = 3.2$, $p > .08$, $f = .3$; condition x group: $F(1,31) = 8.7$, $p = .006$, $f = .5$) as for the standard

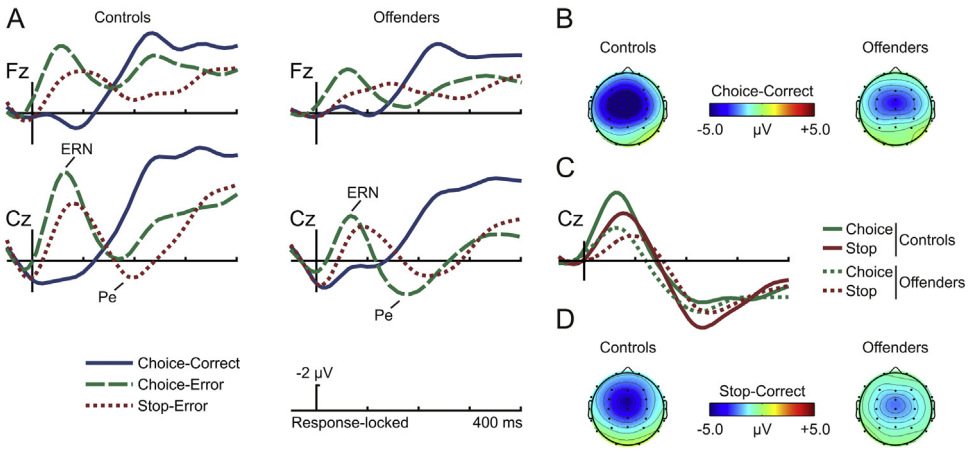

Fig. 2. (A) Grand average of the response-locked ERPs at the midline electrodes in the control and juvenile non-psychopathic violent offender groups [correct trials (solid lines), choice-error trials (dashed lines) and stop-error trials (pointed lines)]. For illustration purposes, these averages were low-pass filtered (lowpass filter, 12 Hz). (B). Scalp distribution for choice-error vs. choice-correct subtraction for the time window 35–85 ms and for the control and offender groups (minimum and maximum values, −5.0/+5.0 μV). (C) ERPs for choice-error and correct difference, and for stop-error and correct difference, at the Cz electrode for the control (solid lines) and offender (pointed lines) participants. (D) Topography for the subtraction between the stop-error vs. choice-correct conditions for the time window of 65–115 ms for the control and offender groups (min. and max. amplitude values, −5.0/+5.0 μV).

analysis, which confirmed the reduction of the ERN amplitude for the offenders in erroneous trials.

3.3.1.2. Stop-errors, ERN. Similar to choice-errors, the ERN component for the stop trials was larger for the control group. A main effect of condition was identified ($F(1,32) = 46.9$, $p < .001$, $f = 1.2$), as well as a significant interaction between condition and group ($F(1,32) = 4.4$, $p = .044$, $f = .4$), despite the non-significant main effect of group ($F(1,32) = .2$, $p > .6$, $f = .08$). This interaction reflected the lower amplitude of the stop-ERN for the offenders. The post hoc *t*-test (that contrasted the stop-errors vs. correct trials) showed a reduced ERN for the offender group compared with the control group at the fronto-central sites, with the largest difference localized on the Fz ($t(32) = -2.5$, $p = .016$, $d = -.4$; see Fig. 2a).

3.3.1.3. Error positivity (Pe). Following the ERN, a clear positivity (Pe) was identified in the waveform for both groups and conditions. The Pe was clearly observed when the correct and choice-errors trials were compared (main effect of condition: $F(1,32) = 22.8$, $p < .001$, $f = .8$) and when the correct and stop-error trials were compared (main effect of condition: $F(1,32) = 51.6$, $p < .001$, $f = 1.3$). No group differences were identified for the Pe for the correct vs. choice-error or the correct vs. stop-error analyses (all *p*-values > .09); the same results were obtained using a larger TW (all *p*-values > .08). Moreover, no differences in the amplitude of the Pe were identified when the choice-error and stop-error trials were compared (main effect of condition: $F(1,32) = .4$, $p > .5$, $f = .1$), and no differences were identified between the groups (all *p*-values > .10). Moreover, no group differences were identified when a broader TW was used (all *p*-values > .1). The results of an additional peak-to-peak analysis for the Pe that compared the choice vs. stop-error discarded differences between groups (all *p*-values > .1). Although the visual inspection of the peak latency of the Pe appeared similar between the groups for the choice-Pe, but slower for the stop-Pe for the offender group compared with the control group, there were no significant differences between the groups (all *p*-values > .7).

3.3.2. Isolation of theta and delta components

Fig. 3 shows the results of the isolated theta and delta activity for the correct and error trials in the two groups. The analysis of the filtered data corroborated that for the choice-error (at Cz, TW 40–90 ms, $t(32) = -2.8$, $p = .009$, $d = -.9$), and for the stop-error condition (at Cz, TW 70–120 ms, $t(32) = -2.3$, $p = .027$, $d = -.8$), the theta (ERN) peak was larger in the control group compared with the offender group. Similar to the previous analysis conducted on unfiltered data (see above), the analysis conducted on the filtered data corroborated that the Pe peak at the Cz location was similar in both groups for choice-errors ($t(32) = -1.5$, $p > .1$, $d = .5$) and stop-errors ($t(32) = -.5$, $p > .6$, $d = -.2$). In sum, the analysis of theta oscillatory activity confirmed a decreased ERN amplitude in the offenders and an equivalent amplitude for the Pe component in both groups. No differences in the delta activity were observed at Cz for choice-errors (ERN: $t(32) = -1.2$, $p > .2$, $d = -.4$; Pe: $t(32) = -1.4$, $p > .1$, $d = .5$) or stop-errors (ERN: $t(32) = -.3$, $p > .7$, $d = .1$; Pe: $t(32) = -.4$, $p > .6$, $d = .1$).

In addition, the results of the isolated theta activity for the inhibited and non-inhibited trials are shown in Fig. 4. As for the ERPs, a peak-to-peak analysis was conducted to clarify the effect of the N2 component, which corroborated the significant main effect of group ($F(1,32) = 99.7$, $p < .001$, $f = .5$). Furthermore, pairwise comparisons confirmed that the magnitude of the N2 was reduced in the offender group compared with the control group in both the inhibited ($t(32) = -3.0$, $p = .007$, $d = -1.3$) and non-inhibited ($t(32) = -2.2$, $p = .039$, $d = .6$) conditions.

3.3.3. ERPs: stop triggered inhibitory effects

3.3.3.1. N2 Inhibitory effects. ERP waveforms time-locked to the appearance of the stop-signal showed a sharp central negativity at ~200 ms followed by a broad posterior positivity (see Fig. 5). This negativity was clearly observed in both the inhibited (stop-correct) and non-inhibited trials (stop-errors) and for both groups, although a larger N2 amplitude was observed for the control group compared with the offender group. In the trials where participants could not withhold the response, i.e., failed inhibitions, a second negative peak was observed that peaked at ~300 ms. The statistical analyses confirmed that the N2 component was larger for the inhibited compared with the non-inhibited trials (main effect of condition: $F(1,32) = 33.0$, $p < .001$, $f = .9$), but non-significant differences were identified between the groups (all *p*-values > .1).

As shown in Fig. 5 and taking into consideration the P2 component, the amplitude of the N2 component appears clearly reduced in the offender group. A peak-to-peak analysis was conducted to clarify this effect on the N2 component. The results of this analysis confirmed that the larger P2–N2 observed for the inhibited trials compared with the non-inhibited trials was significant (main

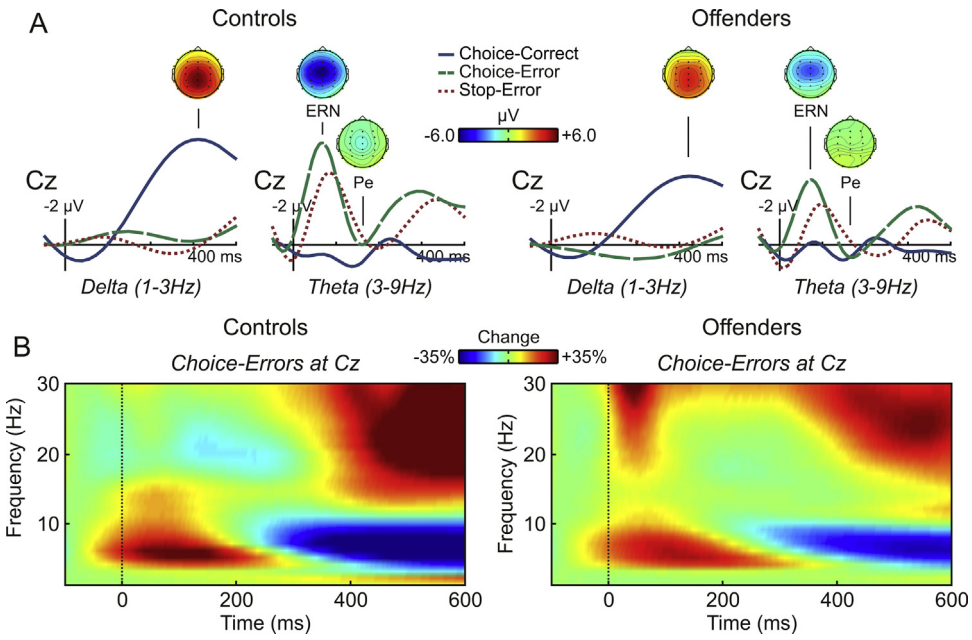

Fig. 3. (A) Grand average of the response-locked ERPs at the Cz electrode, filtered for delta activity (3 Hz low-pass) and theta activity (3–9 Hz bandpass), for the control and juvenile non-psychopathic violent offender groups [correct trials (solid lines), choice-error trials (dashed lines) and stop-error trials (pointed lines)]. Scalp distribution of delta (3 Hz low-pass filter) choice-error activity for the time window of 275–355 ms for each group (min. and max. amplitude values, −6.0/+6.0 μV). Scalp distributions of theta (3–9 Hz bandpass filter) choice-error activity were calculated for the two time windows of 30–90 ms and 120–220 ms (min. and max. amplitude values, −6.0/+6.0 μV). (B) Grand average of the spectral power modulation for the choice-error trials at the Cz electrode (baseline 100 ms prior to the erroneous response) for the control and juvenile non-psychopathic violent offender groups.

effect of condition: $F(1,32) = 19.6$, $p < .001$, $f = .8$). More importantly, this analysis revealed a significant main effect of group, which confirmed a reduced N2 component for the offenders ($F(1,32) = 8.0$, $p = .008$, $f = .5$). A non-significant interaction between condition and group was identified ($F(1,32) = 1.3$, $p > .2$, $f = .2$). Thus, the reduction of the amplitude of the N2 component for the offenders was identified in both the inhibited ($t(32) = 2.1$, $p = .048$, $d = .7$) and non-inhibited ($t(32) = 3.3$, $p = .002$, $d = 1.1$) conditions.

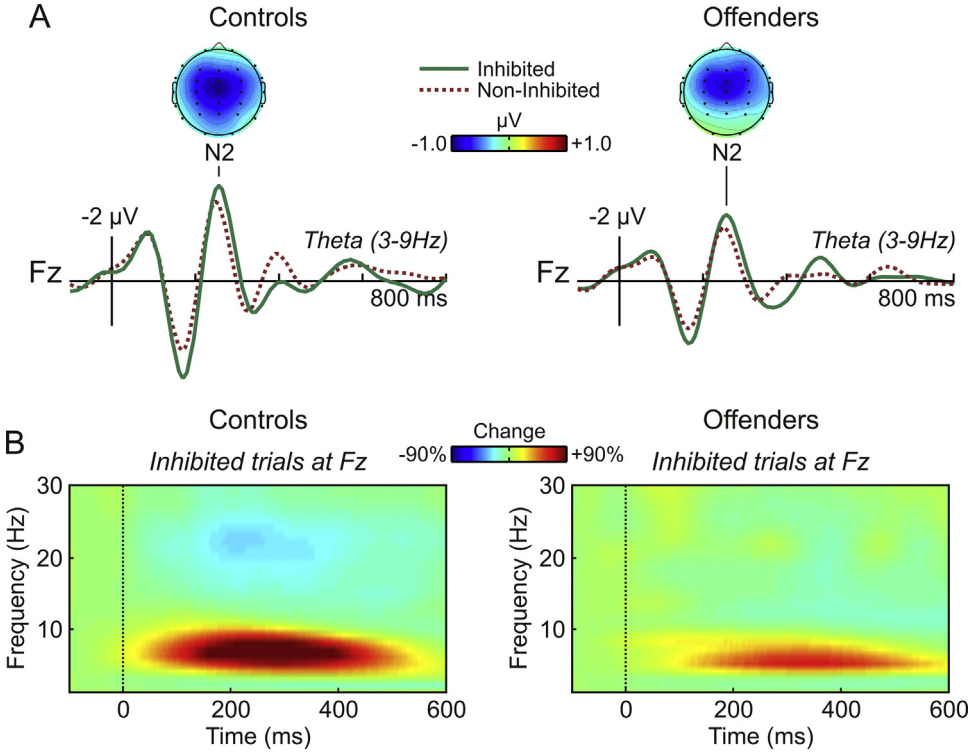

Fig. 4. (A) Grand average of the stop-triggered ERPs at the Fz electrode, filtered for theta activity (3–9 Hz bandpass), for the control and juvenile non-psychopathic violent offender groups [inhibited (solid lines) and non-inhibited trials (pointed lines)]. Scalp distribution of theta (3–9 Hz bandpass filter) inhibited vs. non-inhibited differences was calculated for the time window of 200–300 ms (min. and max. amplitude values, −1.0/+1.0 μV). (B) Grand average of the spectral power modulation for inhibited trials at the Cz electrode (baseline 100 ms prior to the stop-signal) for the control and juvenile non-psychopathic violent offender groups.

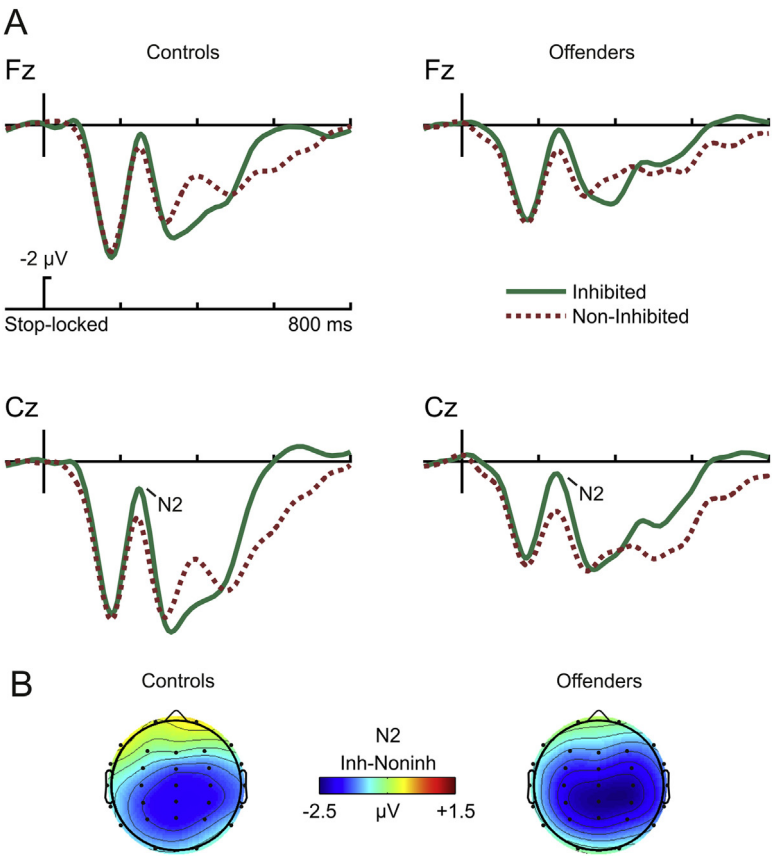

Fig. 5. (A) Grand average of the stop-locked ERPs at the midline electrodes for the control and juvenile non-psychopathic offender groups [stop-inhibited trials (solid lines), stop-non-inhibited trials (pointed lines); low-pass filtered data at 12 Hz]. (B) Topography for the stop-inhibited vs. stop-non-inhibited subtraction for the controls and the juvenile non-psychopathic offenders; time window of 240–260 ms (min. and max. amplitude values, −2.5/+1.5 μV).

In regard to the P3 component, non-significant differences were found between the inhibited and non-inhibited trials (main effect of condition: $F(1,32) = .06$, $p > .8$, $f = .05$). As we expected and consistent with previous findings in the literature, the P3 amplitude was reduced in the offender group compared with the control group (main effect of group: $F(1,32) = 7.1$, $p = .012$, $f = .5$). Finally, a non-significant interaction between condition and group was observed ($F(1,32) = .8$, $p > .3$, $f = .2$).

3.3.4. ERPs: stimulus-locked data (N1/N2–P3 interference effect)

To rule out overall differences between groups in the amplitude of other ERP components, we analyzed the N1 component and the N2–P3 interference effects in the stimulus-locked data (see Fig. 6). This analysis was important to discard the possible explanation of a general reduction of the amplitude in the different ERP components evaluated for the offenders.

3.3.4.1. N1 component. Previous to the N2, the first negative component, the N1, was clearly observed in both groups. No differences on the N1 were identified between the compatible and incompatible conditions or between the groups (all p-values > .1).

3.3.4.2. N2–P3 effects. To test the interference effect between the compatible and incompatible trials, we conducted a new peak-to-peak analysis between the N2-peak and P3-peak. The incompatibility effect on the N2–P3 amplitude was confirmed (main effect of condition: $F(1,32) = 35.4$, $p < .001$, $f = 1.0$), although no differences were encountered between the groups (all p-values > .08).

Visual inspection suggested a reduced P3 amplitude in the offender group compared with the control group (see Fig. 6). We conducted a new analysis that isolated the P3 in the interference effect and adapted the TW according to the latency differences between the groups ($F(1,32) = 9.7$, $p = .004$, $f = .6$). As expected, a reduced P3 amplitude for the incompatible trials compared with the compatible trials was identified (main effect of condition: $F(1,32) = 8.2$, $p = .008$, $f = .5$). Furthermore, a reduced P3 amplitude was identified for the offender group compared with the control group ($F(1,32) = 5.1$, $p = .032$, $f = .4$), and a non-significant interaction between condition and group was found ($F(1,32) = .03$, $p > .8$, $f = .03$).

4. Discussion

In the present electrophysiological study, we investigated error processing and response inhibition in juvenile non-psychopathic violent offenders. A flanker-stop-signal paradigm was used to assess both processes in the control and offender groups. The personality assessment revealed greater impulsivity and aggression scores in the offender group, which confirmed a clear externalizing behavioral pattern. However, we did not observe differences between the groups for the risk-taking dimension. In agreement with this finding, Daderman, Meurling, and Hallman (2001) reported that young offenders were not interested in socially desirable forms of sensation seeking and that this group engaged in other types of sensations, such as drug use, drinking and/or gambling, rather than socially desirable activities. The majority of the risk-taking items included in the I7 risk-taking scale (Eysenck et al., 1985) are social and desirable forms of engaging in risk-taking

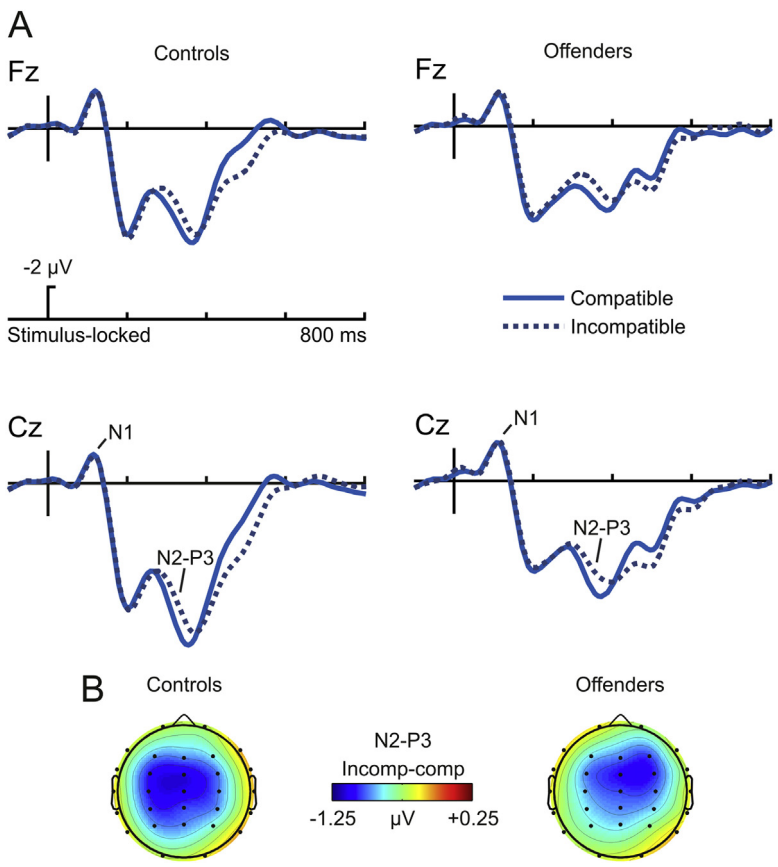

Fig. 6. (A) Grand average of the stimulus-locked ERPs at the midline electrodes for the control and juvenile non-psychopathic offender groups [compatible correct trials (solid lines), incompatible correct trials (pointed lines); averages have been low-pass filtered at 12 Hz]. (B) Topography for the incompatible vs. compatible condition differences for the controls (time window of 280–380 ms) and the juvenile non-psychopathic offenders (time window of 300–400 ms) (min. and max. amplitudes, −1.25/+0.25 μV).

activities (e.g., 'Would you enjoy parachute jumping?'). For this reason, we believe that this questionnaire might not be sufficiently sensitive to detect the engagement of the offender population in non-accepted forms of risk-taking behaviors.

At the behavioral level and despite the lack of differences in post-error adjustments, the RT was slower for the offenders (see similar results in Munro et al., 2007b and Brazil et al., 2009). In the paragraphs below, we develop these ideas in additional detail considering the previous literature and theoretical explanations on this topic.

4.1. Error monitoring

Our results show that juvenile violent offenders had compromised error monitoring that was indicated by a diminution of the ERN after both choice-errors and stop-errors (see Fig. 2 and Fig. 7). This pattern of results is convergent with the findings of diminished error-monitoring performance in adults with low socialization scores (Dikman & Allen, 2000), as well as in children with high externalizing symptomatology (Stieben et al., 2007). Interestingly, the juvenile violent offenders in our sample had been diagnosed with conduct disorder, which has been associated with the externalizing spectrum and high impulsivity scores. Hall et al. (2007) suggested that externalizing individuals could be characterized by a deficit in their ability to self-monitor their own behavior. This pattern could explain the pervasive repetition of harmful behaviors despite 'being aware' of the negative consequences of these actions on themselves or others, which were observed in the offenders.

Based on the association between the ERN component and reinforcement learning (Holroyd & Coles, 2002; Holroyd, Yeung, Coles, & Cohen, 2005), it could also be speculated that juvenile violent offenders have problems in correctly learning from the negative outcomes that originate from their actions. According to this model, the amplitude of the ERN indexes the amount of discrepancy between the expected outcome of our actions and the actual outcome, which serves as an error-teaching signal that allows the organism to adjust and optimize its performance in future similar actions. Therefore, the diminished ERN observed in the offenders group could explain part of its behavior because this population repeatedly exhibits non-adaptive behaviors, such as not taking advantage of learning from errors. However, the fact that we did not identify differences in the amplitude of the Pe component between the groups suggests that we must be

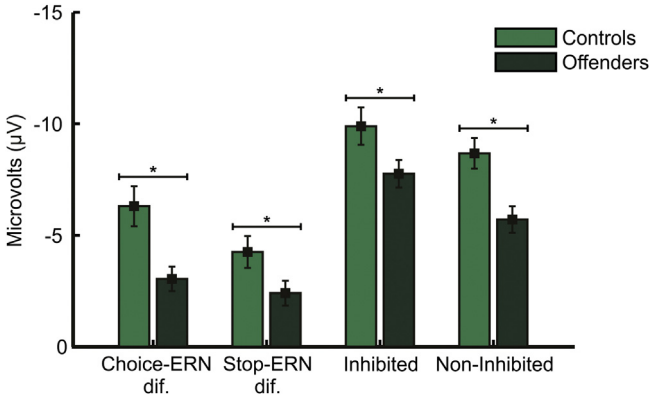

Fig. 7. (A) Mean amplitude difference at the Cz electrode for the Choice and Stop ERNs for both groups; peak-to-peak (N2-P2) differences at Fz for the inhibited and non-inhibited conditions and for both groups.

cautious when considering how error awareness influences behavior in the population of offenders. Recently, the implication of the Pe component in error awareness (Falkenstein, 2004), as well as the motivational significance of the errors, was clarified (Overbeek et al., 2005; Ridderinkhof et al., 2009). The finding of non-affected Pe amplitudes for offenders may suggest that the deficits detected in the offenders were selectively related to the ability to self-monitor ongoing behavior or conflict detection rather than a lack of error awareness (Leuthold & Sommer, 1999). Thus, our findings are in accordance with the externalizing literature that suggests a non-affected Pe (Hall et al., 2007) but an impaired P3 (Costa et al., 2000; Patrick & Bernat, 2006). It is important to note that these findings are somehow contradictory to the recent suggestions regarding the similarity of the neural sources that underlie the Pe and P3 components (Arbel & Donchin, 2009, 2011; Davies et al., 2001; Hajcak et al., 2003; Overbeek et al., 2005; Ridderinkhof et al., 2009). Thus, our findings of a reduced P3 amplitude in the offender group with an unimpaired Pe component might suggest that both components are not completely equivalent. Recent studies that utilized principal component analysis have shown that although the Pe and P3 components share a very similar centro-parietal component, it is also true that an additional fronto-central positive component appears between 250 and 300 ms that is responsible for the development of the Pe (Arbel & Donchin, 2009; Potts, Martin, Kamp, & Donchin, 2011; Endrass, Klawohn, Preuss, & Kathmann, 2012). Thus, considering the robust and consistent findings in the previous literature on the reduction of the P3 amplitude associated with externalizing symptoms, as well as our findings in relation to the P3 (which showed the same pattern), we suggest the only plausible explanation is that because of the overlap of these two-positive components in the morphology of the Pe, the lack of differences observed in our offender group with respect to the Pe amplitude should be associated with compensatory differences in this early fronto-central positive component. Further studies are necessary to disentangle the contribution of these positive components associated with Pe and the lack of differences observed in the present study with respect to this component (see also Hall et al., 2007). Based on these findings, we believe that an interpretation founded on defectual error-monitoring and conflict detection processes fits better with the current observations compared with the interpretation of a deficit in the realization of the importance of the errors committed. Indeed, it is interesting to note that no differences between groups were observed regarding the percentage of errors that were immediately corrected. As error correction reflects post-error adjustment processes (Marco-Pallarés et al., 2008), this finding supports our interpretation that the ERN reduction for offenders could be more easily associated with a lack in monitoring errors than in error-awareness.

4.2. Response inhibition (stop-N2)

The results for the inhibition-related stop-N2 component indicate altered inhibitory processing in the offender group, which was marked by a reduced N2 amplitude in both successful and failed inhibited trials (see Fig. 5 and Fig. 7). It has been suggested that the stop-N2 component might reflect prefrontal monitoring processes associated with a "red flag" signal that is triggered during inhibitory processing (Kok, 1986). The present pattern of results is consistent with the increase in the SSRT encountered in Band et al. (2003), which also suggests slower inhibitory processing in offenders. This specific deficit in the inhibitory function of juvenile violent offenders may explain the lack of self-control or the presence of impulsive and inappropriate behaviors typically observed in these population. Our ERP results are also consistent with previous results obtained in an adult population for the N2 component. For example, Chen et al. (2005) identified a similar reduction in the N2 amplitude in high-impulsive-violent offenders. However, this study was not conclusive because the group of adult-offenders was compared with a group of juvenile controls. In contrast, Munro et al. (2007b) did not find significant differences between adult offenders and adult controls for the inhibitory N2 component. We believe that the present results suggest that inhibitory processing reflected by the reduced N2 in offenders could be generalized to different externalizing traits, assuming that violent behavior is one facet of disinhibitory control. This idea receives some support from a study in children with externalizing symptoms (Stieben et al., 2007), although the encountered reduction of the N2 was only marginally significant.

4.3. General findings regarding standard ERP components

To discard possible confounds (e.g., attentional or motivational factors) that could explain the present findings, we analyzed the stimulus-locked ERP data for the compatible and incompatible go-correct trials. Indeed, we did not observe group differences for the N1 or N2 components, but we did identify differences for the P3 component.

Importantly, the present results enabled us to indirectly evaluate the hypothesis that the P3 amplitude is associated with aggressive and violent behavior and externalizing patterns (see Bernat et al., 2011; Munro et al., 2007b; Patrick, 2008; but see Chen et al., 2005). Our results are in agreement with this consistent finding in previous studies of non-psychopathic populations. We observed a reduction in the amplitude of the P3 component in stop trials (stop-locked P3) and in the stimulus-locked P3 amplitude after standard go trials for the offender group compared with the control participants.

5. Conclusions

To our knowledge, the current study is the first to provide evidence of impaired performance monitoring in juvenile non-psychopathic violent offenders. Deficits in monitoring and inhibitory function were evidenced by a reduced amplitude of the ERN, N2-inhibitory, and P3 components. This deficit may explain the typical inappropriate behavior observed in juvenile violent offenders, and most important, may provide clues to understand the risk of committing offending-impulsive behaviors for this population. Our results regarding performance monitoring in non-psychopathic violent offenders supports the theory stated by Patrick and colleagues, which suggests that a deficit in performance monitoring is an endophenotype of externalizing disorders (Patrick & Bernat, 2006; Hall et al., 2007; Bernat et al., 2011). Thus, the present study contributes to delineating the relationship between performance monitoring deficits, high externalization traits and offending behavior in juvenile non-psychopathic offenders. Considering our results along with Chen et al.'s (2005) findings in adult offenders, it is possible to argue that violent criminals might present deficits in monitoring their behavior over time, a dysfunctional feature that could appear during childhood and then be consolidated later during the adolescence period. Future investigations should be conducted to assess the temporal stability of altered performance monitoring and the importance of these deficits as a risk factor for criminal behavior. Additionally, future studies are necessary to evaluate potential new preventive or rehabilitation programs based on the idea that deficits in performance monitoring and inhibitory function are present in violent offenders.

Conflict of interest

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