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Diminished error processing in smokers during smoking cue exposure

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ABSTRACT

Deficits in error processing may contribute to the continuation of impulsive behaviors such as smoking. Previous studies show deficits in error processing among substance abuse patients. However, these studies were all conducted during affectively neutral conditions. Deficits in error processing in smokers may become more pronounced under affectively challenging conditions, such as during smoking cue exposure. The aim of the present study was to investigate whether smokers showed initial error processing deficits, as measured with the error-related negativity (ERN), and decreased motivational significance attributed to an error, as measured with the error positivity (Pe) when exposed to smoking cues. Additionally, we examined the nature of the ERN and Pe amplitudes in more detail by investigating their associations with trait impulsivity, nicotine dependence levels and cigarette craving. Event-related potentials were measured during a modified Erikson flanker task in both smokers and non-smoking controls. Smokers showed reduced ERN and Pe amplitudes after making an error, accompanied by diminished post-error slowing of reaction times. These results suggest that initial error processing and motivational significance attributed to an error are affected in smokers during smoking cue exposure. Furthermore, individual variation in impulsivity and nicotine dependence was associated with reduced ERN amplitudes.

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1. Introduction

Substance abuse is characterized by a variety of impulsive behaviors including diminished inhibitory control and the preference of immediate rewards over delayed larger rewards (Dawe et al., 2004; Li and Sinha, 2008; Reynolds, 2006; Verdejo-Garcia et al., 2008). A common behavioral pattern that accompanies these processes is an apparent failure to learn from harmful behavior for self or others (Franken et al., 2007). The ability to monitor ongoing performance is a crucial function of the human brain in order to adapt behavior appropriately to situational demands and to continue goal directed behavior (Ridderinkhof et al., 2004b). Deficits in error processing may, therefore, contribute to the continuation of impulsive behaviors (such as drug use) despite negative consequences. Hypothetically, the impulsivity observed in substance abuse patients may result from the fact that errors are processed in a limited way and are therefore not detected optimally.

The processing of errors can be measured both at behavioral and physiological levels. On the electrophysiological level, at least two different error-related brain waves can be distinguished in the Event-Related Potential (ERPs; Falkenstein et al., 2000; Herrmann et al., 2004). The Error-Related Negativity (ERN) arises after 50–80 ms after making an error in speeded response tasks and is followed by the ongoing error positivity (Pe) potential. The ERN and the Pe are regarded as two independent components of error processing (Herrmann et al., 2004; Overbeek et al., 2005). The ERN is a fast and automatic response reflecting initial error detection (Bernstein et al., 1995). A growing body of evidence supports the notion that ERN is modulated by dopaminergic brain systems (Holroyd and Coles, 2002). Haloperidol, a dopamine (DA) antagonist, significantly attenuated ERN amplitudes to self-detected errors during a flanker task (Zirnheld et al., 2004). In contrast, the indirect DA agonist D-amphetamine leads to an enlargement of ERN amplitudes (de Bruijn et al., 2004). The reinforcement learning theory predicts that a disruption of the mesencephalic dopamine system should affect the ERN (Holroyd and Coles, 2002; Holroyd et al., 2009). This theory further suggests that the ERN arises from a dopaminergic midbrain learning signal that is conveyed to the anterior cingulate cortex. Converging evidence indeed indicates that the anterior cingulate cortex is the neural generator of the ERN (Gehring and Knight, 2000; Herrmann et al., 2004; Mathalon et al., 2003; Miltner et al., 2003; Ridderinkhof et al., 2004a; Stemmer et al., 2004; van Veen and Carter, 2002).

The Pe has been linked with the motivational significance attributed to an error (Falkenstein et al., 2000) and the more conscious reflection on an error (Overbeek et al., 2005). Recent research confirmed that the Pe covaried with the stimulus locked P3 that is known to be involved in conscious processing of motivationally significant events (Ridderinkhof et al., 2009).

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Very few ERP studies investigated error processing in substance dependence (Franken et al., 2007, 2010; Sokhadze et al., 2008). Results of studies among cocaine dependent patients suggest a disruption in the brain's error processing system as indicated by reduced ERN (Franken et al., 2007; Sokhadze et al., 2008) and Pe amplitudes (Franken et al., 2007). In a study among smokers, Franken et al. (2010) did not show reduced ERN amplitudes, but did show reduced Pe amplitude as compared to controls, suggesting that initial error processing seems to be intact, while the motivational significance attributed to errors might be compromised. Interestingly, these findings of affected error processing are not specific to substance abusers. Reduced error processing has also been observed in ADHD patients (Liotti et al., 2005; van Meel et al., 2007; Zhang et al., 2009), in psychopaths (Brazil et al., 2009; Munro et al., 2007) and in borderline personality disorder patients (Ruchsow et al., 2006). Results of these studies are in line with a recent theory proposing that patients with externalizing psychopathology share the inability to monitor performance errors (Hall et al., 2007; Olvet and Hajcak, 2008). These similarities in error processing among clinical populations may be the result of shared personality traits in externalizing psychopathology including sensitivity to reward and enhanced impulsivity levels. Reduced ERN components in high impulsive people in the normal population provide further support for the idea that reduced error processing may be related to personality traits (Potts et al., 2006; Ruchsow et al., 2005).

Although Franken et al. (2010) suggest that initial error processing is intact in smokers, possible error processing deficits may remain undetected unless the smoker is tested in more challenging environments, such as during smoking cue exposure. A possible mechanism for enhanced cognitive deficits during cue exposure is that through the course of developing nicotine addiction, increased incentive salience has been assigned to smoking-related cues, which results in increased attentional priority given to these cues (Field and Cox, 2008; Franken, 2003; Littel and Franken, 2007; Robinson and Berridge, 2008). This attentional bias for smoking-related cues might reduce the overall cognitive resources available to monitor ongoing behavior resulting in reduced ERN and Pe amplitudes. The possibility that cue exposure may interfere with error processing is further supported by the idea that both cue exposure and error processing are depending on dopamine release in the ventral striatum (Brody et al., 2004; Holroyd and Coles, 2002) suggesting that cue exposure may change the ERN by changing the underlying dopaminergic system. A previous study among psychopaths indeed showed a reduced ERN during an emotion recognition task, but not during an affective neutral task (Munro et al., 2007), suggesting that error processing is dependent on the presence of environmental, motivational relevant, stimuli. Therefore, the current study investigated error processing in smokers and non-smoking controls while being exposed to smoking cues. For this purpose, the Erikson Flanker task was adapted by adding smoking-related pictures. It is expected that error processing will be reduced in smokers as compared to controls while being exposed to these smoking-related cues. More specifically, we expect to find reduced post-error slowing of reaction times on the behavioral level and reduced ERN and Pe components at the physiological level. Additionally, we examined the nature of the ERN and Pe amplitudes in more detail by investigating their associations with trait impulsivity, severity of nicotine dependence and cigarette craving.

2. Methods

2.1. Participants

Nineteen smokers and 20 non-smoking controls participated in this study. Exclusion criteria for both groups were (a) drug abuse other than nicotine and alcohol, and (b) indications of current physical or psychological illness. Six smokers and 6 non-smokers were excluded from analyses because they had less than ten artifact-free error-related EEG epochs (due to too few errors, n = 5, or too much artifacts, n = 7). The final group consisted of 13 smokers (mean age = 20.7 years, S.D. = 1.3, 9 male) and 14 non-smokers (mean age = 21.4 years, S.D. = 2.6, 10 male). The mean age (t = .96; ns) and gender ratio (chi-square = .02; ns) of the smoker and non-smoker groups did not differ. Smokers smoked at least 10 cigarettes a day (mean = 16.8 cigarettes per day, range = 10–25) for a duration of at least two years (mean = 4.6 years, range = 2–7). The Fagerström test for nicotine dependence (FTND) served as a measure of nicotine dependence in smokers (mean score = 5.0, range = 0–8) and suggested medium levels of nicotine dependence (Heatherton et al., 1991; Vink et al., 2005). Non-smokers had smoked ten or less cigarettes lifetime (mean = 1.6 cigarettes lifetime, range = 0–10). Participants consisted of undergraduate psychology students, who received course credit or a small financial compensation for participation. The study was conducted in accordance with the Declaration of Helsinki and all procedures were carried out with the adequate understanding and written informed consent of the subjects. The ethics committee of the Institute of Psychology of the Erasmus University Rotterdam approved the study.

2.2. Instruments

Breath carbon monoxide concentration was measured using a Micro + Smokerlyzer (Bedfont Scientific Ltd., Rochester, UK) to objectively define smokers and non-smokers. The *impulsiveness* subscale of the Dutch version of the I_7 questionnaire (Lijffijt et al., 2005) was used to measure trait impulsivity. Several questionnaires were used in order to investigate possible confounders. Alcohol consumption, both quantity and frequency, was measured using a QF-index (Lemmens et al., 1992). The positive affect negative affect scale (PANAS; Watson et al., 1988) and the Snaith–Hamilton Pleasure Scale (SHAPS; Snaith et al., 1995) were used to measure mood state and anhedonia. In addition, smokers completed the FTND to measure nicotine dependence and the Questionnaire of Smoking Urges (QSU; Cox et al., 2001) to indicate their subjective craving for a cigarette.

2.3. Task paradigm

A modified version of the Erikson Flanker Task was developed for the purpose of the current study (see Fig. 1). Participants had to indicate the direction of the middle arrow with a button press using the left and right index fingers. To increase the amount of errors, participants were instructed to respond as fast as possible. In the beginning of each trial participants saw a warning sign (^) for a random duration between 300 and 500 ms, after which two rows of five horizontal flanker arrows appeared. The middle arrow in both rows was either congruent or incongruent with the direction of the flanker arrows. The proportion of congruent and incongruent trials was equal. Pictures with smoking-related content or non-smoking-related content were semi-randomly presented in between the five flanker arrows. Arrows and pictures remained on the screen until the button press. A blank screen appeared with a randomly varying duration between 600 and 800 ms before a feedback symbol (a green plus sign for correct trials or a red minus sign for incorrect trials) was presented for 400 ms. Seventy-five smoking-related pictures and 75 non-smoking pictures were each presented six times during the total of 900 trials. Sixteen practice trials were presented before the start of the task and four rest periods were included during task presentation. Smoking-related pictures showed people engaged in smoking behavior or smoking-related objects, whereas non-smoking pictures showed people engaged in non-smoking behavior or neutral objects. The proportion of smoking and non-smoking pictures displaying persons versus objects was equal for both picture categories. In addition, smoking and non-smoking pictures

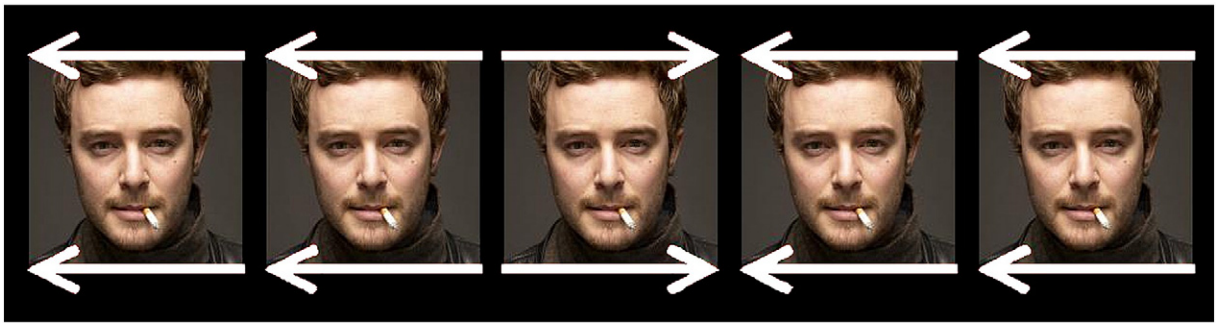

Fig. 1. Example of an incongruent trial combined with a smoking cue picture during the modified affective Flanker task.

were matched on gender of the displayed persons and visual complexity (e.g. number of objects on the picture).

2.4. Procedure

Smokers were instructed to abstain from smoking for at least 1 h before the experiment. This short period of smoking deprivation was introduced in order to reduce the acute effects of nicotine on ERP amplitudes (Houlihan et al., 1996; Houlihan et al., 2001) without introducing strong withdrawal effects. After arrival, participants approved participation by signing informed consent. The CO breath sample was taken and the questionnaires were completed. Subsequently, participants were seated in a comfortable EEG chair in a light and sound-attenuated room. Electrodes were attached and task instructions were explained, after which the smoking Flanker task was started. Smokers completed the QSU again after completing the task.

2.5. EEG recording and data reduction

The EEG was recorded using the Biosemi Active-Two amplifier system from 34 scalp sites (10-10 system, and two additional electrodes at FCz and CPz) mounted in an elastic cap. Six additional electrodes were attached to the left and right mastoids, two outer canthi of both eyes (HEOG), and infraorbital and supraorbital regions of the right eye (VEOG). All signals were digitalized with a sample rate of 512 Hz and 24-bit A/D conversion with a bandpass of 0–134 Hz. Data were off-line re-referenced to computed mastoids. Off-line, EEG and EOG activities were filtered with a bandpass of .10–30 Hz (phase shift-free Butterworth filters; 24 dB/octave slope). Data were segmented in epochs of 1 s (200 ms before and 800 ms after response or stimulus presentations). After ocular correction (Gratton et al., 1983) epochs including an EEG signal exceeding ± 75 μV were excluded from the average. The mean 200 ms pre-response or pre-stimulus period served as baseline. After baseline correction, average ERP waves were calculated for artifact-free trials at each scalp site for correct and incorrect responses separately. The ERN was defined as the mean value in the 25–75 ms time segment after onset of the response. The Pe was defined as the mean value in the 250–350 ms time segment after onset of the response. Both ERN and Pe were studied at the midline electrodes, FCz, Cz and CPz. The chosen time windows include ERN and Pe peaks at these midline electrodes as observed in many studies (Overbeek et al., 2005). In addition, stimulus locked ERPs were calculated by the mean ERP activity between 200 and 300 ms for the N2, 300 and 500 ms for the P3 and 500 and 800 ms for the slow wave at the same midline electrodes.

2.6. Statistical analysis

Group differences on demographics and questionnaire data were analyzed using independent sample t-tests. The difference in self-reported craving before and after task performance was analyzed by means of a paired sample t-test. Repeated measures (RM) ANOVA's were used to analyze task performance and ERP data with Greenhouse–Geisser adjusted p-values. Group (smokers versus non-smokers) was used as a two-level between subjects factor in all RM-ANOVAs. Post-hoc tests for interactions were performed only for interactions including the between subject factor Group. For all analyses, the .05 level of significance was employed and a Bonferroni correction was applied in post-hoc analyses.

The current task design resulted in the following two-level within subject factors of interest (a) Congruency (congruent versus incongruent arrow direction); (b) Picture (smoking versus neutral pictures); (c) Correctness (correct versus incorrect trials) and (d) Post-correctness (reaction times on post-correct versus post-incorrect trials; a commonly used measure for between group comparisons on post-error slowing; Brazil et al., 2009; Franken et al., 2007, 2010; Jonkman et al., 2007; Munro et al., 2007; Potts et al., 2006; Rabbitt, 1966a,b; van Meel et al., 2007). For the behavioral accuracy (percentage of errors) we employed a Group × Congruency × Picture RM-ANOVA. Three RM-ANOVAs were employed for mean reaction time (RT) data: (1) Group × Congruency × Picture, (2) Group × Correctness and (3) Group × Post-correctness. Electrode (FCz, Cz, and CPz) was included as a three-level within subject factor in all ERP analyses. The number of analyzable ERN and Pe epochs did not differ between smokers (mean = 23.8, S.D. = 10.3) and controls (mean = 20.1, S.D. = 11.3), $t(25) = .46$, ns. However, the number of epochs was too small (i.e., resulted in too few segments for each category) to include the Picture within subject factor in the ERN and Pe analyses. Therefore a Group × Electrode × Correctness RM-ANOVA was conducted for the ERN and Pe. To further investigate the nature of the ERN and Pe peaks we calculated Spearman's *rho* correlation coefficients with trait impulsivity across groups and with nicotine dependence and the increase in self-reported craving in smokers only. These correlations were performed separately for correct and incorrect trials and with FCz, Fz and CPz averaged together in order to avoid multiple comparisons. For each stimulus locked ERP (N2, P3, slow wave) a Group × Electrode × Congruency × Picture RM-ANOVA was conducted.

3. Results

3.1. Breath CO levels and questionnaires

As expected, smokers showed higher carbon monoxide (CO) parts per million concentration (mean CO = 11.6, S.D. = 6.4) than non-smoking controls (mean CO = 1.1, S.D. = 1.2), $t(25) = 6.1$, $p < .001$. Smokers and controls did not differ on positive and negative affect as measured by the PANAS, on anhedonia as measured by the SHAPS, and on habitual alcohol drinking patterns, that is alcohol drinking quantity and frequency. However, smokers scored higher (mean = 10.1, S.D. = 4.4) than controls (mean = 6.4, S.D. = 4.3) on the *impulsiveness* subscale of the Dutch version of the I_7 questionnaire, $t(25) = 2.3$, $p < .05$, which indicates that smokers reported higher trait impulsivity levels than non-

Table 1
Percentage errors and reaction times in milliseconds on the affective flanker task. Standard deviations are displayed in brackets.

	Non-smokers	Smokers
Percentage errors smoke incongruent	6.2 (2.6)	5.7 (4.0)
Percentage errors smoke congruent	3.8 (3.2)	4.0 (2.8)
Percentage errors neutral incongruent	5.7 (3.9)	7.2 (4.2)
Percentage errors neutral congruent	3.0 (1.9)	3.4 (2.9)
Reaction time smoke incongruent	390 (54)	376 (40)
Reaction time smoke congruent	381 (52)	362 (35)
Reaction time neutral incongruent	394 (53)	376 (42)
Reaction time neutral congruent	378 (52)	364 (35)
Reaction time correct trials	389 (57)	374 (37)
Reaction time incorrect trials	328 (55)	302 (51)
Reaction time post-correct trials	384 (52)	370 (37)
Reaction time post-incorrect trials	418 (62)	381 (44)

smokers. Subjective craving in smokers was significantly increased after task performance, $t(12) = 4.7$, $p = .001$.

3.2. Behavioral data

Table 1 shows the percentages of errors and reaction times for both groups on the affective flanker task. A robust main effect for congruency, $F(1,25) = 34.0$, $p < .001$, $\eta^2 = .58$ showed that more errors were made on incongruent than on congruent trials (mean difference = 2.7%). No main effect of Picture was observed. There was no overall effect of Group on percentage of errors, neither an interaction effect including Group, suggesting similar percentage of errors between smokers and non-smokers regardless of Congruency and Picture type.

A main effect for Congruency, $F(1,25) = 67.3$, $p < .001$, $\eta^2 = .73$ on reaction times, showed the expected effect that reaction times to incongruent trials were longer than to congruent trials (mean difference 13.23 ms). No main effect of Picture was observed. Also as expected, a main effect of Correctness showed that reaction times to incorrect trials were faster than reaction times to incorrect trials, $F(1,25) = 42.0$, $p < .001$, $\eta^2 = .63$ (mean difference 66.65 ms). Furthermore, a main effect for Post-correctness, $F(1,25) = 17.6$, $p < .001$, $\eta^2 = .41$ showed that reaction times to trials following an incorrect trial were longer than reaction times to trails that followed a correct trial (mean difference 22.93 ms). No main effect for Group was observed nor interactions between group and one or more of the within-factors Congruency, Picture and Correctness. However, a significant interaction effect of Group × Post-correctness, $F(1,25) = 4.4$, $p < .05$, $\eta^2 = .15$ was found. Post-hoc tests showed that the difference between post-incorrect and post-correct was significant for non-smokers, $t(13) = 4.7$, $p < .001$, but not for smokers. These results indicate that non-smokers adjusted their behavior after making an error by slowing down reaction times, whereas smokers did not. In addition, a negative correlation, $r = -.40$, $p < .05$, between post-error slowing (defined as the difference between averaged reaction times for post-error trials versus post-correct trials) and the overall percentage of errors showed that post-error slowing is related to more accurate task performance.

3.3. Event-related potentials

3.3.1. ERN

ERN and Pe amplitudes at the midline electrodes on correct and incorrect trials are displayed in Fig. 2. As expected, a significant main effect was found for Correctness $F(1,25) = 120.14$, $p < .001$, $\eta^2 = .83$ on the ERN at the midline electrodes showing that ERN amplitudes were larger for incorrect trials than for correct trials. We also found a

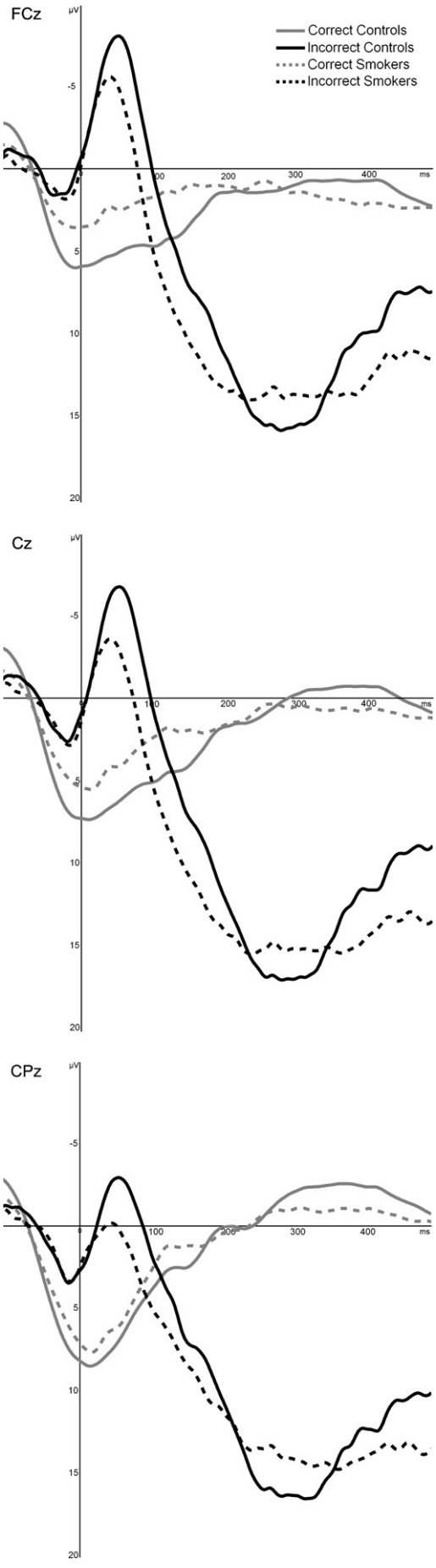

Fig. 2. Grand-average response-locked waveforms at FCz, Cz, and CPz of correct and incorrect responses for smokers and non-smoking controls.

significant main effect for Electrode, $F(2,50) = 56.10$, $p < .001$, $\eta^2 = .69$. No main effect was found for Group. The Group × Correctness × Electrode interaction was not significant $F(2,50) = 1.74$, ns. The interaction effect for Electrode × Correctness was significant $F(2,50) = 11.81$, $p = .001$, $\eta^2 = .32$ and, most importantly, an interaction effect for Group × Correctness was found $F(1,25) = 7.83$, $p = .01$, $\eta^2 = .24$. Post-hoc analysis indicated that the ERN to incorrect trials was significantly reduced in smokers as compared to non-smokers $t(25) = 2.09$, $p < .05$. Smokers and non-smokers did not differ on correct trials.

3.3.2. Pe

As expected, a main effect for Correctness was found $F(1,25) = 15.02$, $p = .001$, $\eta^2 = .38$ on the Pe amplitude, being larger for incorrect trials than for correct trials. No significant main effect was found for Electrode. A significant main effect of Group was found, $F(1,25) = 8.82$, $p < .01$, $\eta^2 = .26$ which showed that smokers have overall lower Pe amplitudes. The Group × Correctness × Electrode interaction was not significant. Furthermore, the interaction effect for Electrode x Correctness was non-significant. Importantly, the interaction effect for Group x Correctness was significant $F(1,25) = 5.07$, $p < .05$, $\eta^2 = .17$. Post-hoc analysis indicated that Pe to incorrect trials was significantly reduced in smokers as compared to non-smokers $t(25) = 3.00$, $p < .01$. Smokers and non-smokers did not differ on correct trials.

3.3.3. Correlations

Correlations between ERN and Pe components with trait impulsivity, FTND scores and self-reported craving are displayed in Table 2 and Fig. 3. Results show that reduced ERN amplitudes on incorrect trials across groups are associated with higher levels of impulsiveness, $r = .44$, $p < .05$, just like stronger nicotine dependence in smokers, $r = .69$, $p < .01$. Higher self-reported craving is not related to magnitude of ERN response. No significant correlations were found on Pe amplitudes.

3.3.4. Stimulus locked ERPs

Data analysis did not reveal any significant main effect of Group, or interaction effects including Group on stimulus locked ERP components including the N2, P3 and slow wave.

4. Discussion

The current study showed behavioral and physiological evidence of reduced error processing in smokers during a task in which participants were exposed to smoking cues. More specifically, this study showed reduced ERN and Pe amplitudes following incorrect responses and accompanied diminished post-error slowing in smokers as compared to non-smoking controls. In addition, self-reported levels of impulsivity, which were higher in smokers, were associated with a reduced ERN across smokers and non-smokers. Moreover, higher nicotine dependence levels among smokers were also associated with smaller ERN responses. On the behavioral level, smokers showed less post-error slowing than non-smoking controls suggesting that also behavioral adaptation (e.g., slowing down after an incorrect response in order prevent another error) is reduced in smokers. However, it must be noted that smokers and non-smokers made comparable numbers of errors. Both groups made more errors on incongruent trials and were faster to respond on error trials. Analyses of stimulus locked ERP waves further suggest that the findings of reduced error processing in smokers are not influenced by an overall reduced cognitive ability that may arise as a result of possible withdrawal effects, as no differences between smokers and non-smokers were found on the stimulus locked ERPs.

Table 2
Correlations between impulsiveness, nicotine dependence, craving and mean ERN and Pe responses over FCz, Cz and CPz (collapsed).

	ERN incorrect trials	ERN correct trials	Pe incorrect trials	Pe correct trials
Impulsiveness[a]	r = .44[b]	r = .04	r = −.09	r = .20
Nicotine dependence[c]	r = .69[d]	r = −.20	r = .19	r = .09
Craving[c]	r = .14	r = .23	r = −.03	r = .00

[a] Correlations includes smokers and non-smokers.
[b] Significant at the .05 level.
[c] Correlations include smokers only.
[d] Significant at the .01 level.

Reduced error processing in smokers is in line with previous ERP studies in cocaine users (Franken et al., 2007, 2010; Sokhadze et al., 2008) and smokers (Franken et al., 2010). Furthermore, several functional imaging studies show reduced activation in the ACC related to error processing in various substance use disorder patients including opiate (Forman et al., 2004), cocaine (Kaufman et al., 2003), cannabis (Hester et al., 2009), and methamphetamine (London et al., 2005) abusers. As expected, we did find a reduced ERN in smokers, in contrast to a previous study of our lab (Franken et al., 2010). A plausible explanation for this discrepancy is the exposure to smoking-related cues during task performance in the current study. Unfortunately, this hypothesis could not directly been tested because participants made not enough errors to analyze error trials for smoking and neutral pictures separately. However, the idea that smoking cues influence the cognitive state of smokers is supported by the significant increase in craving for cigarettes following task performance. The current results, in combination with those of Franken et al. (2010), therefore suggest that fast and automatic error processing may be specifically compromised in smokers when limited cognitive resources are available for error monitoring such as during exposure to smoking cues. Munro et al. (2007) found similar results related to psychopathy. Violent offenders showed reduced ERN amplitudes only during emotion recognition and not during a neutral task paradigm. However, findings of a study of Wiswede et al. (2009) offer an alternative explanation for the reduced ERN in smokers. Wiswede et al. found that ERN amplitudes in healthy controls are enlarged after viewing unpleasant pictures. It may be that the current sample of non-smoking controls considers the smoking pictures as unpleasant and consequently had larger ERN amplitudes than the smokers.

The reduced Pe in smokers confirms the Franken et al. (2010) finding that the motivational significance attributed to an error may be diminished in smokers. It appears that smokers not only process their errors less intensely, they seem to be less worried by their mistakes. However, self-report studies are needed to confirm this finding. Furthermore, the Pe in the current study is, in contrast to the ERN, not correlated with trait impulsivity or nicotine dependence levels, which is in line with the idea that the Pe and ERN reflect independent processes (Overbeek et al., 2005; Ridderinkhof et al., 2009).

The finding in the current study that higher levels of self-reported trait impulsivity across groups are related to lower ERN amplitudes provides further evidence for the idea that personality traits may be associated with reduced error processing. Ruchsow et al. (2006) demonstrated similar results in borderline personality disorder patients. They showed reduced ERN amplitudes in borderline personality disorder patients and correlations with enhanced impulsivity and reduced ERN components. Studies performed in the normal population also confirm that high levels of impulsivity are related to lower ERN amplitudes (Potts et al., 2006; Ruchsow et al., 2005). These studies, together with the findings of the current study, provide evidence for the idea that impulsivity may explain reduced error processing in smokers. Note, however, that enhanced impulsivity in smokers was found on self-reported trait impulsivity, while smokers did not show diminished impulse control on behavioral performance indices of the adapted Flanker task. This clearly suggests that although

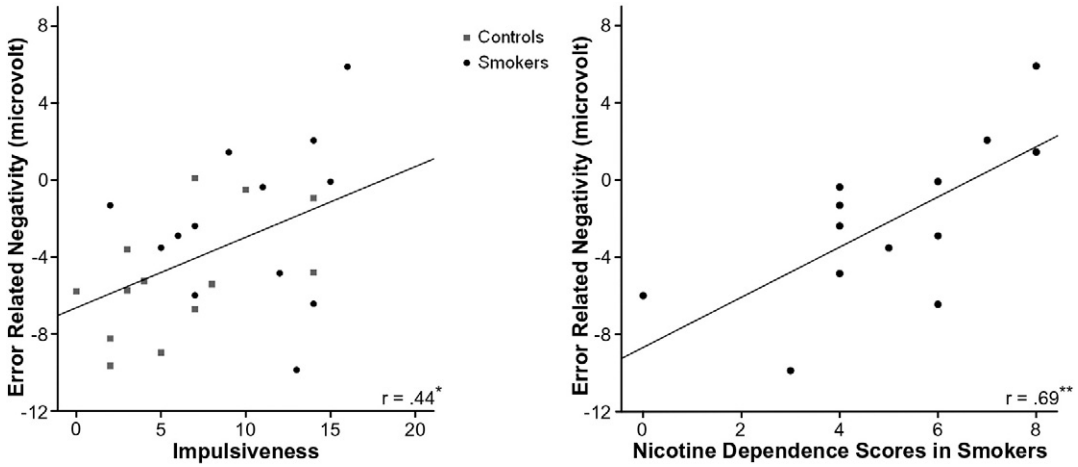

Fig. 3. Correlation between the mean amplitude of the error related negativity for incorrect responses and self-reported trait impulsivity (left panel) and nicotine dependence levels (right panel). * significant at the .05 level, ** significant at the 0.01 level.

self-reported trait impulsivity and behavioral errors both reflect impulsivity, they tap different aspects of impulsivity (Alderson et al., 2007; van Mourik et al., 2005). More research is needed to elucidate the discrepancy between self-reported and behavioral impulsivity. Furthermore, the current study design does not allow drawing conclusions on causality. It may be that impulsivity and reduced error processing are a predisposition to start smoking, or that impulsive behavior, including smoking, contributes to diminished error processing. However, since the ERN in smokers in the current study also varied with the degree of nicotine dependence, an impulsive predisposition cannot fully explain diminished error processing. Other characteristics specific for nicotine dependence may have a complementary effect on the deficit in error processing. A possible explanation for the association between the level of nicotine dependence and reduced ERN amplitudes is the compromised function of the dopaminergic system in the ventral striatum in addiction (Volkow et al., 2009). In either case, reduced error processing undermines the ability to monitor ongoing behavior and may be related to the continuation of addiction related behaviors.

A limitation of the current study is the relatively small size of the samples, such that replication of the current results in larger groups of participants is essential. In addition, it must be kept in mind that the present smokers are relative young smokers in an early stage of smoking dependence. Although generalization to other categories of smokers is limited, the current sample of smokers can be considered heavy smokers within the student population of smokers (Berg et al., 2010), which is further supported by moderate levels of FTND scores (Heatherton et al., 1991).

To conclude, results of the current study showed reduced error processing in smokers both at the behavioral and physiological level. Decreased ERN and Pe amplitudes in smokers were accompanied by reduced post-error slowing. Furthermore, self-reported impulsivity levels were associated with reduced ERN amplitudes in smokers and non-smokers and nicotine dependence was associated with lower ERN amplitudes in smokers specifically. Together, these results suggest that both personality traits and specific nicotine dependent characteristics, such as a disturbed dopamine system, are associated with diminished error processing. Since adequate error processing is required to adapt behavior properly, reduced error processing may contribute to the development and maintenance of addictive behaviors.

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