Neuroscience Letters 563 (2014) 61–65

Contents lists available at ScienceDirect

Neuroscience Letters

journal homepage: www.elsevier.com/locate/neulet

The N2 ERP component as an index of impaired cognitive control in smokers

George A. Buzzell*, John R. Fedota, Daniel M. Roberts, Craig G. McDonald

George Mason University, Fairfax, VA, USA

HIGHLIGHTS

- Cognitive control was evaluated in individuals with low levels of nicotine dependence.
- The N2 and P3 ERP components of smokers and non-smokers were compared.
- Smokers exhibited a reduced N2 component, but no behavioral deficits.
- The N2 may provide a sensitive index of cognitive control deficits in smokers.

ARTICLE INFO

Article history:
Received 2 August 2013
Received in revised form 9 January 2014
Accepted 18 January 2014

Keywords:
Smoking
Nicotine
Cognitive control
Executive function
ERP

ABSTRACT

Impaired cognitive control has been proposed as a hallmark of nicotine dependence and is thought to arise, in part, from synaptic alterations in anterior cingulate cortex (ACC), a primary component of the dopamine reward pathway. The N2 component of the event-related potential (ERP) appears to index a cognitive control process in paradigms such as the visual go/no-go task. Moreover, as dipole-modeling has suggested that the neural generator of the N2 component can be localized to the ACC, this component may prove useful for investigating impairments of cognitive control in smokers. Given conflicting reports of whether the N2 is reduced in smokers (as compared to non-smoker controls), the current study further examined the suitability of this component as an index for impaired cognitive control in smokers. Smokers and non-smokers performed a visual go/no-go task while electroencephalogram (EEG) was recorded. As predicted, the no-go N2 of smokers was significantly smaller than that of non-smoker controls, while the no-go P3 did not differ between groups. Importantly, behavioral performance (reaction time and accuracy) did not differ between smokers and nonsmokers, which might reflect the low levels of nicotine dependence (assessed by the Fagerstrom test) in our sample. The observed N2 modulation in the absence of behavioral impairments provides evidence for the utility of the N2 component as a sensitive measure of impaired cognitive control in smokers, even in those with low levels of nicotine dependence.

1. Introduction

The physiological and cognitive deficits that accompany nicotine addiction have prompted substantial research interest in the underlying etiology of this disorder. Based on neuroimaging and animal model research, Kalivas and Volkow [1] propose that addiction can be conceptualized as a failure in cognitive control. Further, Dawe and colleagues [2] suggest that one aspect of cognitive control, inhibitory control, is a hallmark of drug dependence. Nonetheless, a well-established index of the cognitive control deficits that accompany nicotine addiction remains unsubstantiated. Given that event-related potentials (ERPs) provide a relatively inexpensive and sensitive measure of cognitive deficits, a well-established electrophysiological index of impaired cognitive control in smokers would be a valuable addition to further investigations of nicotine addiction. Therefore, the current study was designed to further investigate the validity of ERPs in assessing cognitive control deficits in young adults that smoke.

Impairments in the executive functioning of smokers, and specifically cognitive control, are thought to partly arise from synaptic alterations in the nucleus accumbens (NAcc) and anterior cingulate cortex (ACC) [1], two primary components of the dopamine reward pathway and executive attention network. The ACC in particular, is responsive to nicotine administration in humans [3] and is consistently activated in tasks that induce prepotent response tendencies that must be inhibited [4]. For example, in a "go/no-go" task, participants must respond to a frequently

* Corresponding author at: George Mason University, 4400 University Drive MS 3F5, Fairfax, VA 22030, Tel.: +1703 851 5843.
E-mail addresses: gbuzzell@gmu.edu (G.A. Buzzell), john.fedota@nih.gov (J.R. Fedota), drobertc@gmu.edu (D.M. Roberts), cmcdona3@gmu.edu (C.G. McDonald).

0304-3940/$ – see front matter © 2014 Elsevier Ireland Ltd. All rights reserved.
http://dx.doi.org/10.1016/j.neulet.2014.01.030

presented stimulus ("go" trials), while withholding responses to a second, infrequently presented stimulus ("no-go" trials) [5,6]. The high frequency of go trials results in a prepotent response tendency, which conflicts with the required withholding of responses on infrequent no-go trials. Therefore, no-go trials require both the detection [6] and inhibition [5] of prepotent response tendencies in order to respond correctly on these trials. While neuroimaging data has consistently provided evidence that ACC activation is increased on no-go trials [4], the high temporal resolution provided by ERPs allows for unique incite into the rapid cognitive processing elicited by no-go trials.

Analysis of the ERP waveform during a go/no-go task consistently reveals two characteristic differences on no-go trials, compared to go trials. Approximately 250–350 ms after stimulus onset, an increased frontocentral negativity, termed the N2, is observed [7]. While it is debated whether this N2 component reflects inhibition per se [5], as opposed to a conflict monitoring process [6], both of these interpretations are consistent with the N2 serving as an index of a cognitive control process. Further, dipole-modeling work suggests that the neural generator of the N2 can be localized to the ACC, a structure known to play a key role in cognitive control [6,8]. Following the N2 component, and peaking approximately 300–600 ms after stimulus onset, a large, positive deflection, termed the P3 component, is observed [9,10]. In contrast to the N2, the P3 component is thought to arise from a more distributed network of cortical generators [9] and reflect inhibitory, attentional and working memory related processes [9–11]. While it is less clear if the P3 component directly reflects cognitive control, reductions in this component have been previously related to risk for alcoholism [12]. In addition, the no-go P3 has been associated with attentional impairments [10] a deficit often related to nicotine abuse [13,14].

Given the sensitivity of ERPs to dynamic cognitive processes, the ERP technique may provide a well-suited index of impairments in cognitive control associated with nicotine dependence. However, few prior studies have examined how electrophysiological indices of response inhibition, an aspect of cognitive control, differs in smokers vs. non-smokers [15,16]. One investigation by Evans and colleagues [15] did not report on any differences in the no-go N2 of nicotine-dependent participants, although a reduction in the no-go P3 was observed for moderately addicted smokers (whether abstaining or not) as compared to non-smoking controls. In contrast, a more recent investigation by Luijten and colleagues [16] found that the no-go N2 was indeed diminished in moderately addicted smokers (as compared to non-smoking controls), while the no-go P3 was not. Given these conflicting findings, the current study further investigated the suitability of using either the N2 or P3 components as an index of impaired cognitive control in smokers.

While the work of both Evans and colleagues [15] and Luijten et al. [16] used a go/no-go task in their investigations of cognitive control, it should be noted that there were subtle differences in the nature of the go/no-go tasks employed. Specifically, while Luijten and colleagues [16] employed a traditional go/no-go task, Evans and colleagues [15] used what could be described as a hybrid go/no-go task, involving a working memory component. It is possible that this working memory component might occlude the direct analysis of cognitive control deficits. Therefore, because of the strong theoretical basis specifically implicating impaired cognitive control in drug abuse [1,2] we opted to employ a more traditional go/no-go task in the present investigation (similar to that used by Luijten and colleagues [16]). Given the strong link between the no-go N2 and cognitive control processes in a traditional go/no-go task, we hypothesized that the N2 elicited in this task may provide a particularly sensitive marker for cognitive control deficits and would be reduced for smokers in this task. In addition, in line with the work of Evans et al. [15] and other substance abuse studies [12], we also investigated the potential for the no-go P3 to be reduced in smokers, as compared to non-smoking controls.

2. Method

2.1. Participants

Participants were recruited from the George Mason University undergraduate population and provided either course credit or monetary compensation ($10/h) for their participation. Fifteen smokers (mean age = 21.93 years, SD = 4.08, 8 female) and 15 non-smoking controls (mean age = 21.4, SD = 4.22, 8 female) participated in the study. Smokers and non-smokers were matched for age, sex and education level. In addition, both groups had no known neurological deficits and were not currently taking any psychoactive medications. The smokers showed, on average, a very low level of nicotine dependence (mean Fagerstrom score = 1.87, SD = 1.51, range = 0–5) and only five of the smokers indicated smoking more than 10 cigarettes per day [17]. All smokers had been smoking for at least one year prior to the study and 11 had been smoking for 2 years or longer. Prior to participating in the study, all smokers abstained from smoking for at least one hour (13 of the smokers abstained for 2 h or longer). All non-smokers had no current or previous history of cigarette smoking. The Office of Research Subject Protections at George Mason University approved all procedures for the study.

2.2. Procedure

Participants arrived at the research laboratory and took part in the experimental task between the hours of 9 am and 5 pm. After providing written informed consent, participants' vision was tested using a combination of the Snellen and Rosenbaum eye charts to ensure that all participants had normal, or corrected-to normal vision. Participants then completed a demographic questionnaire and smokers additionally completed a questionnaire assessing smoking behavior (time since last cigarette, years smoking) and the Fagerstrom Test for Nicotine Dependence (FTND) assessment [17]. Participants than completed a visual go/no-go task (described below). All experimental procedures lasted no longer than 2 h.

2.3. Go/no-go task

Participants performed a go/no-go task similar to that described by Nieuwenhuis et al. [6], in which the "go" and "no-go" stimuli were presented at probabilities of 0.80 and 0.20, respectively. The stimuli—either an upper case "M" or "W" presented in Ariel font—were presented for 100 ms, 0.5° below a central fixation dot (subtending 0.3°). Stimuli subtended 0.5° vertically and 0.7° horizontally and were presented on an LCD monitor located approximately 1 m from the participant. The initial assignment of go and no-go stimuli (M and W) was counterbalanced across participants and changed halfway through the testing of each participant in order to control for low-level stimulus features. The task was comprised of four 200 trial blocks (800 trials total). Participants responded to the go stimuli with the index finger of their dominant hand, using the spacebar of a standard desktop computer keyboard. Participants were instructed to place an equal emphasis on accuracy and response time. Total time to complete the experimental task was 30 min.

2.4. Electrophysiological recording

The EEG was recorded using a Neuroscan NuAmps amplifier. Recordings were made at 32 scalp sites (extended 10–20 system) with Ag/AgCl electrodes mounted in an elastic cap. In addition,

Ag/AgCl electrodes were placed at the left supraorbital and suborbital sites, as well as the left and right outer canthal sites, to monitor vertical and horizontal electro-oculographic (EOG) activity, respectively. Customized MATLAB scripts (The MathWorks, Natick, MA) and the Psychophysics Toolbox [18–20] were used to present the stimuli, while SCAN 4.01 software (Compumedics, North Carolina, USA) was used to digitize the EEG at a sampling frequency of 500 Hz. All scalp electrodes were referenced to the left mastoid on-line and re-referenced to the average of the left and right mastoids following data collection. All electrode impedances were maintained below 5 kΩ and recorded with a 70 Hz low-pass filter. Following data acquisition, EEG was band-pass filtered from .1–30 Hz, using a Butterworth filter from the ERPLAB plug-in [21].

2.5. Behavioral analysis

Accuracy data was analyzed using a two-factor (stimulus type × smoking behavior) mixed design ANOVA, with stimulus type (go, no-go) serving as a within-subjects factor and smoking behavior (smoker, non-smoker) serving as a between-subjects factor. For response time data, only correct go trials and errors of commission elicit a measurable behavioral response. Correct go and error of commission trials were separately analyzed using independent-samples *t*-tests to investigate possible differences between smokers and non-smokers.

2.6. Electrophysiological quantification and analysis

Error trials, as well as those contaminated by muscle activity, body movements and eye blinks were discarded (rejection threshold: 50 μV), and separate artifact-free averages were computed for each category of trial (go, no-go). After artifacts were removed, the number of correct, artifact free trials remained comparable across groups (smoker mean go trials = 459.1, no-go trials = 89.9; non-smoker mean go trials = 419.9, no-go trials = 86.5). All EEG processing was accomplished using EEGLAB [22], a toolbox for the MATLAB programming environment. For each component of interest (N2, P3) the time window and electrode locations used for ERP quantification was based on previous literature [5,6,16] and inspection of the grand-average ERP waveforms, collapsed across groups. In order to improve the robustness of any effects and as means to protect against false positive findings, ERPs were analyzed as a cluster of electrodes where the component was shown to be maximal in the grand-average waveform. For the N2 component, mean amplitudes were extracted from the 268–308 ms time period, using a cluster of three medial, frontocentral electrode locations (Fz, FCz, Cz). For the P3 component, mean amplitudes were extracted from the 362–462 ms time period, using a cluster of three medial, centroparietal electrode locations (Cz, CPz, Pz).

A series of two-way (stimulus type × smoking behavior), mixed design ANOVAs were used to analyzed the N2 and P3 component mean amplitudes. Stimulus type (go, no-go) was included as a within-subjects factor and smoking behavior (smoker, non-smoker) as a between-subjects factor. Follow-up comparisons were made using independent samples *t*-tests, using a Bonferonni correction for multiple comparisons.

3. Results

3.1. Behavior

Analysis of the accuracy data revealed that the expected main effect of stimulus type (go, no-go) was significant, $F(2,28) = 78.27$, $p = .001$, $\eta_p^2 = .74$; participants were more accurate when responding to go trials ($M = 98.32\%$, SD = 1.87%) compared to no-go trials ($M = 76.5\%$, SD = 14.52%). However, no significant main effect of smoking behavior, $F(2,28) = 3.27$, $p = .081$, nor an interaction between stimulus type and smoking behavior was identified, $F(2,28) = 2.11$, $p = .157$. This suggests that the go trials of smokers ($M = 97.66\%$, SD = 2.29%) did not significantly differ from the go trials of non-smokers ($M = 98.99\%$, SD = 1.02%). Similarly, smoker no-go trials ($M = 72.25\%$, SD = 16.71%) did not significantly differ from the no-go trials of non-smokers ($M = 80.75\%$, SD = 10.91%).

Analysis of the response time data revealed no significant differences between the correct go trials of smokers ($M = 384.75$ ms, SD = 36.45 ms), as compared to non-smokers ($M = 399.63$ ms, SD = 43.51 ms), $t(1,28) = 1.02$, $p = .32$. Similarly, no significant response time differences were identified for the error of commision trials for smokers ($M = 330.68$ ms, SD = 29.01 ms), as compared to non-smokers ($M = 340.18$ ms, SD = 29.01 ms), $t(1,28) = .897$, $p = .38$.

3.2. Electrophysiology

Inspection of the topographic plots and grand-average ERPs revealed that the N2 component exhibited the predicted frontocentral distrubution [5,6], with maximal amplitudes over the three midline, frontocentral electrodes (Fz, FCz, Cz) used in the cluster-based analysis (see Fig. 1b). Analysis of the N2 component revealed both a significant main effect of stimulus type (go, no-go), $F(1,28) = 29.66$, $p = .001$, $\eta_p^2 = .514$, and a trend for smoking behavior (smoker, non-smoker), $F(1,28) = 3.85$, $p = .06$, $\eta_p^2 = .121$. However, there was also a significant stimulus type by smoking behavior interaction, $F(1,28) = 4.23$, $p = .049$, $\eta_p^2 = .131$. Follow-up analyses showed that smokers had a smaller no-go N2 amplitude ($M = 0.29$, SD = 3.58) compared to non-smokers ($M = -3.20$, SD = 4.13), $t(28) = 2.47$, $p = .02$ (see Fig. 1b). However, the N2 did not differ on go trials between smokers ($M = 2.11$, SD = 3.53) and non-smokers ($M = 0.82$, SD = 3.27), $t(28) = 1.04$, $p = .309$.

Inspection of the topographic plots and grand-average ERPs of the P3 component revealed the predicted centroparietal distribution [9], with maximal amplitudes over the three midline, central-parietal electrodes (Cz, CPz, Pz) used in the cluster-based analysis (see Fig. 1d). Analysis of the P3 component revealed a significant main effect of stimulus type (go, no-go), $F(1,28) = 73.8$, $p = .001$, $\eta_p^2 = .725$, (see Fig. 1b). However, no main effect of smoking behavior, $F(1,28) = 0.01$, $p = .924$, or a stimulus type by smoking behavior interaction was observed, $F(1,28) = 0.03$, $p = .858$. That is, the smoker go P3 ($M = 6.55$, SD = 3.63) did not significantly differ from the non-smoker go P3 ($M = 6.60$, SD = 3.38); similarly, the smoker no-go P3 ($M = 11.34$, SD = 5.25) did not significantly differ from the non-smoker no-go P3 ($M = 11.59$, SD = 5.16).

4. Discussion

The present study provides confirmatory evidence that the no-go N2 component, a well-established index of cognitive control, is significantly reduced in individuals who smoke, relative to non-smoking controls. In contrast, the no-go P3, a component that is less directly related to aspects of cognitive control [23], did not significantly differ between smokers and non-smoking controls. The present results are in line with previous results showing a reduced N2 component for moderately addicted smokers in a go/no-go task [16]. However, the present results augment these prior findings by identifying a reduction in the N2 component with only a mildly nicotine-dependent sample (mean FTND = 1.87, SD = 1.51, compared to $M = 5.05$, SD = 2.27 as reported in Luijten et al. [16]). Further, the present study identified a decrease in the N2 component without the presence of significant behavioral impairments. This suggests that the N2 component is a particularly sensitive index of executive functioning deficits in smokers. In addition, the

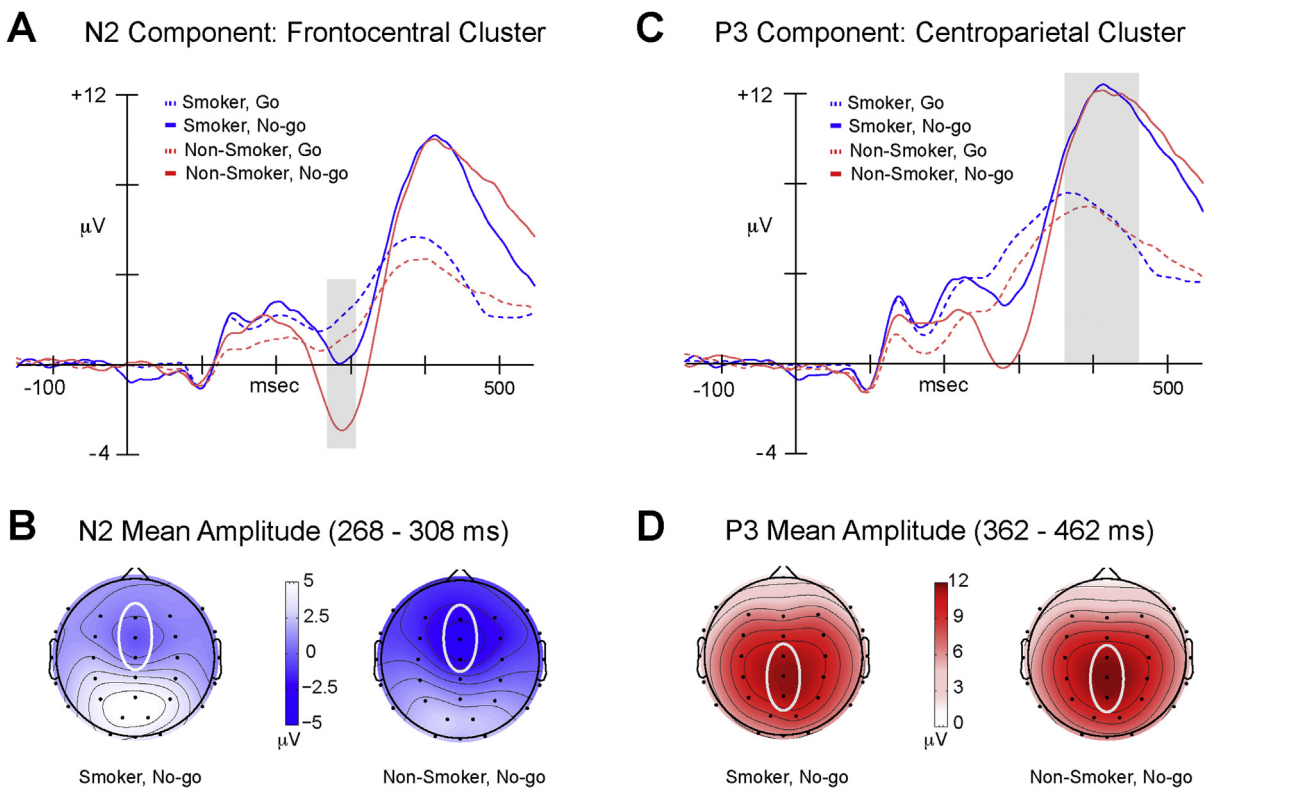

Fig. 1. Stimulus-locked, grand average ERP waveforms and topographic plots reflecting the N2 and P3 components elicited by the go/no-go task. Go and no-go ERP waveforms (A, C) were derived from averaging across the three electrode locations depicted with white ellipses in each topographic plot (B, D). Shaded areas (A, C) reflect the analysis window and time period plotted in the topographic plots (B, D).

present results clarify conflicting reports [15,16] and reinforce the notion that the N2 is a useful index of cognitive control deficits in smokers.

Prior evidence suggests that the neural generator for the N2 component is the ACC [6,8], a structure implicated in cognitive control. Moreover, this brain region has been shown to be activated by nicotine administration in humans [3] and morphological changes within this region are associated with addiction more generally [1]. In addition, ACC activation is reduced in substance abusers [24,25]. In conjunction with previous electrophysiological [16] and functional neuroimaging [24,25] work, the present findings illustrate the presence of diminished ACC functioning in smokers, which is in line with prevailing theoretical models of addiction [26].

The finding that the N2 was attenuated in the absence of significant behavioral deficits suggests that light smokers may be able to rely on other compensatory mechanisms to maintain a level of behavioral performance comparable to non-smokers [14]. Such an interpretation is in line with research showing a dissociation between ACC activation and behavioral performance measures [10,27,28]. It is also possible these findings are indicative of the fact that the participants did not exhibit strong signs of nicotine dependence. However, it should be noted that although accuracy did not differ significantly between groups, there was a relatively weak statistical trend for reduced accuracy in light smokers. Therefore, while the present study provides evidence that the N2 component provides a particularly sensitive index of the cognitive control deficits observed in smokers, a larger sample might have revealed that light smokers differ from non-smokers in terms of behavioral performance.

While the N2 likely indexes disruption to ACC functioning in light smokers, it remains unclear whether such dysfunction reflects changes due to chronic nicotine exposure, or vulnerability to nicotine usage. Longitudinal studies have previously shown behavioral deficits on executive functioning tasks to predict the initiation of smoking or the use of other substances of abuse [29,30]. Therefore, it is also possible that a reduction in the N2 component would have been observed in the present sample of light-smokers even prior to their initiation of smoking. While the present results do not permit this conclusion, they do support the validity of the N2 in assessing cognitive control deficits (even in a sample of light-smokers) and suggest the need for additional longitudinal work employing this method.

While developmental research has established a link between behavioral measures of impaired executive functioning and the likelihood of initiating smoking [29], an electrophysiological marker of addiction liability may better capture susceptibility. For example, one might hypothesize that while behavioral methods can be used to predict smoking initiation, the reliability of such predictions might be improved with the complementary use of ERPs. Further, longitudinal data may be able to provide more direct evidence as to whether the N2 component predicts smoking initiation, or indexes neurobiological changes that result from chronic nicotine exposure.

In contrast to Evans et al. [15], but consistent with the work by Luijten and colleagues [16], we found no evidence of a reduced no-go P3 in our sample of light smokers. However, this difference might stem from the fact that Evans and colleagues [15] used a hybrid go/no-go task, in comparison to the more traditional go/no-go task employed in the present experiment and by Luijten et al. [16]. The task employed by Evans and colleagues [15] can essentially be characterized as a 1-back working memory task, which would place additional load on working memory processes [15,31]. Therefore, the P3 difference identified by Evans and colleagues [15] might point toward working memory deficits in smokers, which have similarly been observed in relation to alcohol abuse [12]. In addition, it should be noted that the participants used in the study

by Evans and colleagues [15] were recruited from the local Tampa Bay community, in contrast to the undergraduate population used by the present investigation and that of Luijten et al. [16]. Therefore, it is also possible that the present study (and that of Luijten et al. [16]) reflects a sample from a qualitatively different population as that sampled by Evans and colleagues [15].

Within traditional, go/no-go tasks, the no-go P3 has been interpreted as an index of a later inhibitory control process more closely linked to the act of motor inhibition [11]. This interpretation is largely based on the finding that the P3 is larger on no-go trials than on go trials. However, as the no-go stimulus is typically infrequently presented in most go/no-go paradigms, the enhanced P3 may reflect the low probability of the no-go stimulus. Therefore, an alternative interpretation of the P3 is that it indexes attentional engagement [9,23], suggesting that both groups were equally attentive when performing the go/no-go task. Such an interpretation is further reinforced by the lack of any significant speed or accuracy differences between the two groups. Regardless of the functional interpretation of the no-go P3, these findings suggest that smokers were not simply slower or less attentive when performing the go/no-go task in the present experiment. Rather, the present findings suggest a degree of selectivity in the neurocognitive differences between smokers and non-smokers.

5. Limitations and future directions

The present investigation was careful to control for many demographic characteristics across groups, however, future investigations could expand on the present findings by controlling for other potential confounds, such as the use of other substances of abuse. In addition, while smoking status and addiction severity was characterized in the present study using self-reported measures and the FTND, a more objective measure, such as exhaled carbon dioxide (ppm CO), could be employed in future investigations. In addition, it should be noted that while the present results provide support for N2 differences between smokers and non-smoking controls, this was found in a college-age sample of smokers with low-levels of nicotine dependence.

Acknowledgment

This study was funded by the Virginia Foundation for Healthy Youth.

References

[1] P.W. Kalivas, N.D. Volkow, The neural basis of addiction: a pathology of motivation and choice, Am. J. Psychiatry 162 (2005) 1403–1413.
[2] S. Dawe, M.J. Gullo, N.J. Loxton, Reward drive and rash impulsiveness as dimensions of impulsivity: implications for substance misuse, Addict. Behav. 29 (2004) 1389–1405.
[3] V. Kumari, J.A. Gray, D.H. Ffytche, M.T. Mitterschiffthaler, M. Das, E. Zachariah, G.N. Vythelingum, S.C.R. Williams, A. Simmons, T. Sharma, Cognitive effects of nicotine in humans: an fMRI study, NeuroImage 19 (2003) 1002–1013.
[4] M.M. Botvinick, J.D. Cohen, C.S. Carter, Conflict monitoring and anterior cingulate cortex: an update, Trends Cogn. Sci. 8 (2004) 539–546.
[5] M. Falkenstein, J. Hoormann, J. Hohnsbein, ERP components in go/nogo tasks and their relation to inhibition, Acta Psychol. (Amst.) 101 (1999) 267–291.
[6] S. Nieuwenhuis, N. Yeung, W. Van Den Wildenberg, K.R. Ridderinkhof, Electrophysiological correlates of anterior cingulate function in a go/no-go task: effects of response conflict and trial type frequency, Cogn. Affect. Behav. Neurosci. 3 (2003) 17–26.
[7] J.R. Folstein, C. Van Petten, Influence of cognitive control and mismatch on the N2 component of the ERP: a review, Psychophysiology 45 (2008) 152–170.
[8] N. Yeung, M.M. Botvinick, J.D. Cohen, The neural basis of error detection: conflict monitoring and the error-related negativity, Psychol. Rev. 111 (2004) 931–959.
[9] J. Polich, Updating, P300: an integrative theory of P3a and P3b, Clin. Neurophysiol. Off. J. Int. Fed. Clin. Neurophysiol. 118 (2007) 2128.
[10] R.A.P. Roche, H. Garavan, J.J. Foxe, S.M. O'Mara, Individual differences discriminate event-related potentials but not performance during response inhibition, Exp. Brain Res. 160 (2004) 60–70.
[11] A. Kok, J.R. Ramautar, M.B. De Ruiter, G.P.H. Band, K.R. Ridderinkhof, ERP components associated with successful and unsuccessful stopping in a stop-signal task, Psychophysiology 41 (2004) 9–20.
[12] C. Kamarajan, B. Porjesz, K.A. Jones, D.B. Chorlian, A. Padmanabhapillai, M. Rangaswamy, A.T. Stimus, H. Begleiter, Spatial-anatomical mapping of NoGo-P3 in the offspring of alcoholics: evidence of cognitive and neural disinhibition as a risk for alcoholism, Clin. Neurophysiol. 116 (2005) 1049–1061.
[13] P.A. Newhouse, A. Potter, A. Singh, Effects of nicotinic stimulation on cognitive performance, Curr. Opin. Pharmacol. 4 (2004) 36–46.
[14] D.E. Evans, D.J. Drobes, Nicotine self-medication of cognitive-attentional processing: nicotine and attention, Addict. Biol. 14 (2009) 32–42.
[15] D.E. Evans, J.Y. Park, N. Maxfield, D.J. Drobes, Neurocognitive variation in smoking behavior and withdrawal: genetic and affective moderators, Genes Brain Behav. 8 (2009) 86–96.
[16] M. Luijten, M. Littel, I.H.A. Franken, Deficits in inhibitory control in smokers during a go/noGo task: an investigation using event-related brain potentials, PLoS ONE 6 (2011) e18898.
[17] T.F. Heatherton, L.T. Kozlowski, R.C. Frecker, K.-O. Fagerstrom, The Fagerström test for nicotine dependence: a revision of the Fagerstrom tolerance questionnaire, Br. J. Addict. 86 (1991) 1119–1127.
[18] D.H. Brainard, The psychophysics toolbox, Spat. Vis. 10 (1997) 433–436.
[19] M. Kleiner, D. Brainard, D. Pelli, What's new in psychtoolbox-3? Perception 36 (2007), ECVP Abstract supplement.
[20] D.G. Pelli, The VideoToolbox software for visual psychophysics: transforming numbers into movies, Spat. Vis. 10 (1997) 437–442.
[21] J. Lopez-Calderon, S. Luck, ERPLAB, UC-Davis Center for Mind & Brain, 2010.
[22] A. Delorme, S. Makeig, EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis, J. Neurosci. Methods 134 (2004) 9–21.
[23] A. Kok, On the utility of P3 amplitude as a measure of processing capacity, Psychophysiology 38 (2001) 557–577.
[24] D.H. Fishbein, D.L. Eldreth, C. Hyde, J.A. Matochik, E.D. London, C. Contoreggi, V. Kurian, A.S. Kimes, A. Breeden, S. Grant, Risky decision making and the anterior cingulate cortex in abstinent drug abusers and nonusers, Cogn. Brain Res. 23 (2005) 119–136.
[25] F. Musso, F. Bettermann, G. Vucurevic, P. Stoeter, A. Konrad, G. Winterer, Smoking impacts on prefrontal attentional network function in young adult brains, Psychopharmacology (Berl.) 191 (2006) 159–169.
[26] R.Z. Goldstein, N.D. Volkow, Dysfunction of the prefrontal cortex in addiction: neuroimaging findings and clinical implications, Nat. Rev. Neurosci. 12 (2011) 652–669.
[27] K.I. Erickson, M.P. Milham, S.J. Colcombe, A.F. Kramer, M.T. Banich, A. Webb, N.J. Cohen, Behavioral conflict, anterior cingulate cortex, and experiment duration: implications of diverging data, Hum. Brain Mapp. 21 (2004) 98–107.
[28] G.A. Buzzell, D.M. Roberts, C.L. Baldwin, C.G. McDonald, An electrophysiological correlate of conflict processing in an auditory spatial Stroop task: the effect of individual differences in navigational style, Int. J. Psychophysiol. 90 (2013) 265–271.
[29] Z. Harakeh, L. de Sonneville, R.J.J.M. van den Eijnden, A.C. Huizink, S.A. Reijneveld, J. Ormel, F.C. Verhulst, K. Monshouwer, W.A.M. Vollebergh, The association between neurocognitive functioning and smoking in adolescence: the TRAILS study, Neuropsychology 26 (2012) 541–550.
[30] S.F. Tapert, M.V. Baratta, A.M. Abrantes, S.A. Brown, Attention dysfunction predicts substance involvement in community youths, J. Am. Acad. Child Adolesc. Psychiatry 41 (2002) 680–686.
[31] A. Gevins, B. Cutillo, Spatiotemporal dynamics of component processes in human working memory, Electroencephalogr. Clin. Neurophysiol. 87 (1993) 128–143.