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# A SMOKING-RELATED BACKGROUND HELPS MODERATE SMOKERS TO FOCUS: AN EVENT-RELATED POTENTIAL STUDY USING A GO-NOGO TASK

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## Abstract

Objective: Cognitive impairment is a major component in addiction. However, research has been inconclusive as to whether this is also the case for smokers. The present study aims at providing electrophysiological clue for altered inhibitory control in smokers and at investigating whether reduced inhibition was more pronounced during exposure to a smoking cue. Methods: ERPs were recorded during a visual Go-NoGo task performed by 18 smokers and 23 controls, in which either a frequent Go signal (letter ‘‘M”) or a rare No-Go signal (‘‘letter W”) were superimposed on three different long-lasting background contexts: black-neutral, smoking-related and non smoking-related. Results: (1) Smokers performed worse and had an earlier NoGo-N2 latency as compared to controls and independently of context, suggesting a general inhibition impairment; (2) with smoking-related backgrounds specifically, smokers made fewer mistakes than they did in other contexts and displayed a larger NoGo P3 amplitude. Conclusion: These data might suggest that background cues related to addiction may help smokers to be more accurate in an inhibition task. Significance: Our results show the classical inhibitory impairment in smokers as compared to non-smokers. However, our data also suggest that a smoking-related background may bolster the inhibitory ability of smokers specifically.

## 1. Introduction

Smoking tobacco is known as being one of the most common health-damaging behaviors, which seems to persist, despite awareness of its negative consequences on health and intensive prevention and treatment efforts (Le Faou and Scemama, 2005). Indeed, tobacco seems to be the most addictive substance among addictive drugs (32% of users become dependent; Inserm, 2015) and a smoker dies, on average, 15 years earlier than a non-smoker (U.S. Department of Health and Human Services, 2010).

The role of cognitive functions has been emphasized as a major component in the development and persistence of addiction (Luijten et al., 2014; Ehrman et al., 2002; Waters and Feyerabend, 2000). Specifically, the incentive salience properties of the addiction–related stimuli on the one hand and a deficit in inhibition on the other hand are core mechanisms of addictive behavior (the dual-process model; Field and Cox 2008; Volkow et al., 2004; Wiers et al., 2007). Accordingly, as was shown before in heroin-addicts (Yang et al., 2009), cocaine users (Waters et al., 2012), alcohol users (Noël et al., 2007), smokers (1) show a smoking-cue reactivity, manifested by a processing enhancement in the brain striatal regions related to motivation and reward (David et al., 2005; Lydon et al., 2014; Luijten et al., 2016) and (2) typically fail to inhibit drug-oriented behavior even when the consequences are deleterious. Interestingly, all those behaviors are independent of physiological needs: when obese people are satiated they still want to eat, when smokers have smoked they are still oriented towards smoking-related cues (Tibboel et al., 2011; Watson et al., 2013). Note that addictions ‘‘without substance” like gambling or internet addiction show similar patterns as drug addictions (Luijten et al.,

2014). This highlights the fact that we cannot easily understand the complexity of addiction pharmacologically: it is not merely the physiological deficit of the drug, which predicts drugs seeking behaviors.

Cortical measures are known to be affected by attentional bias towards salient stimuli as sanctioned by event-related potentials (ERPs) (Carretie et al., 2006; Franken, 2003), startle electromyography (EMG) (Franken, 2003) and on behavioral measures of attention (RTs) (Mogg and Bradley, 1998). However, although the attentional bias has been widely examined for substances such as alcohol (Field et al., 2008; Noël et al., 2016), cocaine, (Franken et al., 2007), heroin (Forman et al., 2004) and food (Loeber et al., 2012), only a few studies have investigated this attentional bias in smokers (Evans et al., 2009; Luijten et al., 2011; Luijten et al., 2014) and results have been inconsistent (Dinn et al., 2004; Luijten et al., 2014; Reynolds et al., 2007). This heterogeneity in the data has been attributed to differences in the level of smoking addiction (Machulska et al., 2015; Piper et al., 2006) or to the type of paradigm used (Tibboel et al., 2015). However, one main reason for these inconclusive results could be related to the fact that controlling an addictive behavior often requires the management of affective situations, whereas behavioral and neural reactions incensed by brief experimental stimuli (which are normally presented for 200–500 ms) are clearly not as intense nor as complex as those generated by real-life, longer-lasting emotional contexts (Carretie et al., 2006; Albert et al., 2010; Campanella et al., 2016). The original contribution of the present paper, then, will be to use a variant of the Go-NoGo task, in which Go and No-go trials are displayed on a longer-lasting background context (i.e., for two minutes). Indeed, such a ‘‘contextual Go-NoGo task” has already been used in our laboratory with social drinkers during an ERP recording, revealing more commission errors in an alcoholrelated context (Petit et al., 2012).

It is generally reported that, two major brain waves, the NoGoN2 and the NoGo P3, are enhanced for NoGo trials (as compared to Go trials). This change is supposed to reflect the modifications in brain activity related to inhibition (Falkenstein et al., 1999). In particular, ERP measures have shown to be more sensitive than behavioral outcomes (Ridderinkhof et al., 2002; Yang et al., 2009) and might reveal subclinical differences which are not yet behaviorally visible.

First, concerning the NoGo-N2, this is a negative wave emerging between 200–300 ms after stimulus presentation and maximum peaks emerge from frontal scalp sites. The mechanism underlying the inhibition of an automatic tendency is supposed to be reflected by the NoGo-N2 (Luijten et al., 2014). In a general population, N2 is shown to be larger when there is time pressure and smaller and delayed when there is a high error rate (Gajewski and Falkenstein, 2013). Even though some research argues that NoGo-N2 reflects the monitoring of conflicts emerging from competition between achievement and inhibition of the action (Nieuwenhuis et al. 2003), this interpretation is also consistent with the N2 serving as an index of a cognitive control process (Buzzell et al., 2014; Nakata et al., 2004). Assuming that the enhancement of the N2 is a proactive strategy to control task performance (in our case not responding during the NoGo trials), we expect this component to vary as a function of the smoking status of the participant, and as a function of the experimental context elements.

Second, concerning the NoGo P3, this component appears approximately 300–500 ms after the stimulus and has a more widespread distribution over the frontocentral areas of the brain. As it is a late component, it would not reflect the initial inhibition process (as is the case for N2) but rather a later stage more closely related to the inhibition of premotor and motor systems (Huster et al, 2010; Luijten et al., 2011).

Together, growing evidence suggests that the N2 and P3 reflect functionally and structurally distinct mechanisms linked to inhibitory control (Gamma et al., 2005; Luijten et al., 2011). Accordingly,

less pronounced N2 or P3 amplitudes in addiction compared to controls are considered to index neural default in inhibitory control. The distinction of the two systems is reinforced by the fact that, on a neuropharmacological level, although the neurotransmitter systems underlying the generation of the N2 and P3 waves are still unclear, growing evidence shows they are subserved by different basal ganglia subsystems. The nigrostriatal system would modulate the NoGo-N2 while the mesocortico-limbic dopamine system would modulate the NoGo-P3 (Beste et al., 2010; Hansenne, 2000; Polich, 2007).

Despite this, to our knowledge, there only are three studies that combine a NoGo paradigm with EEG in a smoker population, and they all show a general inhibition impairment on the ERP components and no difference on a behavioral level in smokers as compared to non-smokers (except for a general deficit on task performance in smokers shown by Luijten et al., 2011). The first two studies (Buzzell et al., 2014; Evans et al., 2009) investigated inhibitory control through a classical NoGo task. Buzzell and colleagues (2014) found that the NoGo-N2 of smokers is significantly smaller than that of non-smoker controls (no differences were found on the P3) while behavioral performance (reaction time and accuracy) does not differ between smokers and nonsmokers. Evans and colleagues (2009) found that non-smokers relative to smokers have a greater NoGo P3 amplitude but again, no behavioral difference was found. Importantly, the inhibition bias found in these studies was general and not specifically tied to addiction-related stimuli, as no smoke-related stimuli were used in the Go-NoGo paradigms.

However, both the 2011 and 2016 studies conducted by Luijten and colleagues (2011, 2016) manipulated the type of stimulus presented (smoking-related or not). In the 2011 study, they investigated whether inhibitory control in smokers that were nicotine-deprived (for one hour) is modulated by the presence of smoking-related cues. They used a paradigm in which a cue, which was either smoking-related or not, was surrounded by a frame on a computer screen, the color of which indicated if a trial is either a Go or a NoGo trial. Compared with controls, those with a nicotine dependency were less accurate on the NoGo tasks (regardless of whether the picture was smoking-related or not) and exhibited lower NoGo-N2 amplitudes. The P3 amplitudes did not differ between the groups. The 2016 study compared smokers who relapsed to smokers who did not with the same paradigm. The authors found that the NoGo P3 was reduced in relapsed smokers; moreover, this NoGo P3 predicted the belonging to one of the two groups. In contrast, there were no differences in N2 and P3 enhancement in the Go trials (reflecting stimulus salience) between the two groups.

In the present study, the experimental logic is adapted to what we think is more ecologically valid, that is to say more proximal to the everyday life experience of smokers as the smoking-related background will appear for a long time. More precisely, in our tasks, the cue itself is not-smoking related (it is, in fact, a capitalized letter) and, second, importantly, while Luijten and colleagues' smoking cues were only presented for a maximum of 200 ms, our smoking-related background (either a pack of cigarettes or a lit cigarette) stayed on screen for the duration of the task (up to 10 minutes depending of participant' speed). Indeed, under real-life conditions, smoking-related stimuli always appear for longer than 200 ms, and required the individual to control their reaction to the stimuli within a longer time-frame (Albert et al., 2010). In addition to this, short-delay cues require more cognitive resources for processing and inhibiting the stimuli, and therefore induce a higher risk of variability linked to factors which cannot be investigated, such as, for example, initial attention allocation, attention shift, information monitoring, etc. (Luijten et al., 2016).

Specifically, the smoking-related Go-NoGo task was as follows: either a frequent Go signal (letter ''M''), or a rare NoGo signal (letter ''W'') were superimposed on two different types of contexts, a smoking-related (a cigarette lit or a pack of cigarettes) and two non-smoking-related contexts, taken as non-emotional baseline cues. For this second type, we presented both a non-smokingrelated

(respectively a pen or a basket) and a black-background context, for which we expect basically no differences in results. We hypothesize that smokers will show generally reduced response inhibition as compared to non-smoking controls whatever the background. This will result behaviorally in more commission errors and longer reaction times in NoGo trials and electrophysiologically, in reduced amplitudes and shortened latencies for both NoGo-N2 and NoGoP3 – this electrophysiological signature highlighting the decrease of the cognitive resources assigned to the treatment of inhibition. We also hypothesize that this response inhibition will be even more reduced when smokers will face the smoking-related backgrounds. However, in the present study the cues are in and by themselves neutral (the letters ‘‘M” and ‘‘W”), and it is the (permanent) background, which is or not smoking-related. Therefore, an alternative prediction could be that a smoking-related background helps smokers to concentrate. Indeed, it has also been shown that nicotine can actually alleviate clinical symptoms such as cognitive deficits (De Beaurepaire, 2012; Dervaux and Laqueille, 2008; Evans and Drobes, 2008) even if the results of clinical and laboratory research remain inconclusive (see Evans and Drobes, 2008 review). In this alternative scenario, we expect fewer commission errors as well as NoGo-N2 and -P3 amplitudes and latencies revealing an effective inhibition process, i.e. which, for smokers specifically, are relatively less diminished and delayed in smoking related as compared to non-smoking related NoGo trials.

# 2. Materials and method

## 2.1. Participants

Forty-one participants (mean age = 30.8 ± 9.7 see Table 1 for demographic data) were recruited via the hospital where the experiment was located (CHU Brugmann Hospital), via email, through personal contacts and through announcements on social networks. Through a brief phone screening, major medical problems, as well as past or current drug consumption (other than moderate levels of alcohol and tobacco) were excluded. Smokers had to abstain from smoking 2 hours before the experiment to avoid floor or ceiling effects of the urge to smoke during the task hewissen et al., 2007). The local ethics committee of the Brugmann Hospital approved the study (OM_026).

**Table 1**
Characteristics of the study participants: mean scores (±standard deviation) of the clinical characteristics of control and smokers participants.

| | Smokers (n = 18) | Controls (n = 23) | *p*-value |
|---|---|---|---|
| Women (n) | 10 | 19 | 0.08 |
| Mean age in years (SD) | 29.8 (7) | 30.2 (10) | 0.92 |
| Education level* | 14 (2) | 15 (2) | 0.87 |
| Anxiety state (STAI-A) | 35.9 (10) | 32.7 (9) | 0.28 |
| Impulsivity (UPPS scale) | 96.7 (11) | 77.7 (11) | <0.001 |
| Urgency | 23.9 (5) | 18.9 (4) | <0.001 |
| Premeditation | 19.5 (4) | 15.5 (3) | <0.001 |
| Perseverance | 22.9 (5) | 17.6 (4) | <0.001 |
| Sensation Seeking | 29.4 (5) | 25.7 (6) | <0.05 |
| TQSU | 59.83 (15) | / | |
| FTND | 3.99 (1) | / | |
| Cigarettes smoked/day | 13.67 (5) | / | |
| Minutes without smoking | 294.1 (279) | | |

* Number of years of education since completing primary school.

## 2.2. Measures

### *2.2.1. Personality and behavioral questionnaires*

#### 2.2.1.1. Smoking and nicotine dependence and craving questionnaires.

Participants had to complete both the Fagerström Test for Nicotine Dependence (FTND) and the Tiffany Questionnaire for Smoking Urges (TQSU). The FTND is a 7-item questionnaire (with different response types, most of them either binary or four-way) which is a widely used, reliable and valid self-

report measure aimed at capturing the degree of nicotine dependence (Etter and Perneger, 1999; Etter, 2005; IARC, 2008; Kozlowski et al., 1994; Piper et al., 2006). The TQSU is a 12-item questionnaire (with responses on a 7-point Likert scale) as a classic self-report measure of craving (e.g. ‘‘I have a desire for a cigarette right now”; ‘‘Smoking would make me less depressed”).

#### 2.2.1.2. The state trait anxiety inventory (STAI).

The STAI (Gauthier and Bouchard, 1993) is a self-report questionnaire used to assess self-reported anxiety. In this study, only the state version of the STAI was used (how people feel regarding their level of anxiety at the moment they are assessed).

#### 2.2.1.3. The urgency premeditation perseverance and sensation seeking impulsive behavior scale (UPPS).

The UPPS (Whiteside and Lynam, 2003) is a well-validated and frequently used self-report questionnaire, composed of 45 items (with responses on a four– point Likert scale), which illustrates ‘‘the trouble of restraining general behavioral reactions in situations that elicit strong emotion (Urgency), the difficulty to anticipate habitual situations (Lack of Premeditation), the difficulty to sustain in drawn-out activity (lack of Perseverance), and the tendency to look for new emotionallyexciting situations (Sensation seeking)” (Cirilli et al., 2011: 1) Both the FTND and the TQSU are quick assessment tools with acceptable psychometric properties for smoking behavior. The UPPS, on the other hand, is an elaborate measure of general impulsivity, which thoroughly investigates its different facets and might therefore be more sensitive to how people represent themselves. Links between tobacco dependency and UPPS have been consistently demonstrated (Mitchell, 1999, 2004; Reynolds et al., 2007) with smokers being more impulsive than non-smokers.

#### 2.2.1.4. SCID MINI (structured clinical interview for DSM MINI international neuropsychiatric interview.

The SCID MINI (Lecrubier et al., 1997) is a widely used well-validated structured interview, which assesses 17 disorders (including substance use disorders - SUD) related to DSM IV. This allowed us to eliminate participants who had other SUD’s than cigarette (Finally, three of them had to be excluded).

### *2.2.2. Go-NoGo modified for somking*

We used the same paradigm as Petit et al. (2012) except that we used smoking-related instead of alcohol-related stimuli. During the Go-NoGo tasks, the participants were instructed ‘‘to press a button on a joystick with their right index finger, as quickly and accurately as possible, whenever the letter M (Go) was displayed (...) and to refrain from pressing the button when the letter W (No-Go) was presented ” (Petit et al, 2012: 2). Either ‘‘M” or a ‘‘W” layed over three different long-lasting background contexts: a smoking context (SC), and non-smoking-related context (NSC), and a black-screen background with no context (NC) (Fig. 1). Two different pictures were used for each of the smoking and non-smoking-related contexts. The order in which the contexts were displayed was counterbalanced across participants. Overall, following the Petit et al. paradigm (2012: 2) ‘‘the task comprised of six separate stimulation blocks. Each block contained 133 letters, divided into 93 Go (70%) and 40 No-Go (30%) stimuli. Trials were semi-randomized in order to avoid the consecutive presentation of two No-Go letters within each block. One to four Go trials could precede No-Go trials. Each task consisted of the presentation of a background screen (SC, NSC or NC; 500 ms), then the letter M or W appeared on this background screen for 200 ms after which the initial background screen came back (1300 ms). Thus, a maximum of 1500 ms was possible for subjects to press the button before the next letter appeared. Participants were asked to look at the center of the screen continuously and to refrain from moving and blinking during blocks to reduce interference caused by movements”.

#### 2.2.2.1. Stimuli.

The stimuli consisted of two yellow capitalized letters (M and W; size of 500x400 mm; Arial font) with a black outline in order to make them as visible as possible, superimposed on four different background pictures (displayed on a 17-inch monitor). For the neutral backgrounds, we selected the same backgrounds as the control ones from Petit et al. (2012), that is to say, a pencil and a basket. For the smoking-related ones, we first selected 16 pictures from the Internet and the International Affective Picture System. Then, 31 people, independent from the ERP study, rated these pictures for cigarette-relatedness and their emotional level: ‘‘(1) for cigarette-relatedness, participants were asked to rate whether the picture was strongly related to cigarettes on a scale from zero (not at all) to five (extremely); (2) for emotional level, participants were asked to rate how pleasant the picture was on a scale from zero (very unpleasant) to nine (very pleasant)” (Petit et al., 2012: 2). On this basis, five images were retained and reproduced in a way that enhanced their brightness and color quality. Ninety-one participants rated these images once again in order to verify whether there was any difference with the first results obtained. This subsequent evaluation confirmed the suitability of the same five images and from this pool we selected the 2 which where the most appropriate for the NoGo experimental design.

### 2.3. Design and procedure

Participants were tested one by one in a quiet room from the Brugmann hospital. They signed a consent form and completed a pretest of subjective cigarette craving (only smokers): participants self-reported their craving of a cigarette by answering in% to the question ‘‘how much do you want to have a cigarette right now?”. This was followed by the modified Go-NoGo task, the rest of the questionnaires assessing an identical posttest craving, FTND, TQSU (only smokers) as well as UPPS and STAI (all participants) and a debriefing.

### 2.4. EEG recording

‘‘Electric brain potentials were recorded from 32 electrodes mounted on a Quik-Cap and placed in standard (based on the 10–20 system) and intermediate positions. A common physical linked mastoids reference was used. The ground electrode (AFz) was positioned between Fpz and Fz along the midline, and the impedance of all electrodes was maintained below 10 kX. The EEG was recorded continuously at a sampling rate of 1024 Hz with the ANT Eeprobe software The EEG was amplified by batteryoperated ANT amplifiers (Advanced Neuro Technology - ANT Ltd, Enschende, the Netherlands) with a gain of 30,000 through a band-pass of 0.01-100 Hz” (Petit et al., 2012: 4), which is a classical set-up used in our lab (Campanella et al., 2010). Data were then filtered offline with a band-pass of 0.3–30 Hz. Epochs of 1000 ms were created, from -200 to 800 ms after stimulus onset (with - 200 to 0 considered as the baseline). Go and NoGo trials were processed separately. Based on these epochs filtered, we investigated the FP1, FPz and FP2 electrodes to check the artefacts. Approximately 29% of trials were contaminated (cut-off of 30 mV was used to define trials that were contamined either by eye movements or muscular artifacts) and were eliminated offline in order to analyze only the artefact-free trials. A 2-way ANOVA, imputing context (SC, NSC and NC) as a within-subject variable, and group (smokers and non-smokers) as a between-subjects variable was done separately for each condition (Go and NoGo trials). This showed that the number of rejected trials was similar in each group and context, and this for the two conditions (for the Go trials: Context $F(2,78) = 0.23$, $p = 0.80$; Context／Group $F(2,78) = 2.70$, $p = 0.80$ and for the NoGo trials: Context $F(2,78) = 1.18$, $p = 0.31$; Context／Group $F(2,78) = 0.10$, $p = 0.91$).

### 2.5. Data collection

Given the hypothesis, at a behavioral level, we recorded and investigated the effects of the (1) stimulus type (Go or NoGo); (2) context (SC, NSC or NC); and (3) response (keypress to the Go stimuli or no keypress to the NoGo stimuli) both in terms of accuracy as in terms of speed. Following the usual

procedure of our lab (see Petit et al., 2012), at the EEG level, for each context, the maximum peak amplitude and latency to peak amplitude of the Go and No-Go N2 and the Go and No-Go P3 components were recorded. The component values were measured with frontocentral electrodes (Fz, FC1, FC2 and Cz) and the most negative peak value was around 200–300 ms after stimulus onset for the N2, and the most positive peak value was round 300–500 ms for the P3.

## 2.6. Data analysis

ERP data were analyzed with a repeated measures 3-way ANOVA with the condition (Go-NoGo) and stimulation (background C, NSC, and NC) as within-subjects variable and with the group (control participants and smokers) as a between-subject variable. Simple effects and interactions were systematically examined. Student's t-tests, ANOVA, Bonferroni's post-hoc test and Spearman's correlation were used when appropriate. Omission error rates (i.e., no response in Go trials), commission error rates (i.e., keypress in No-Go trials), and reaction times (RTs) to Go stimuli were also analyzed separately by a 2-way ANOVA with the stimulation as within-subjects variable and with the group as a between-subject variable. Finally, a stepwise regression, known to evaluate the order of importance among a set of variables (Montgomery et al., 2012), was done in order to investigate if any of the independent variables (electrophysiological and behavioral data) predicted the group belonging (non-smokers vs smokers).

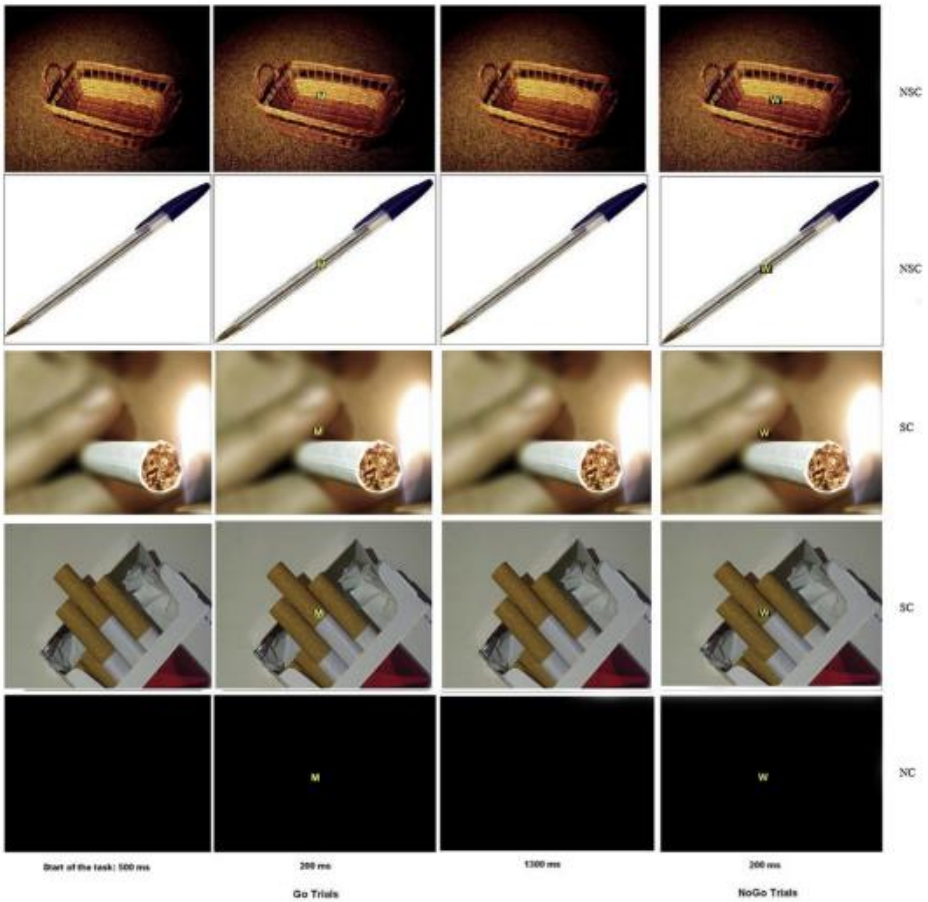


**Fig. 1.** Go-NoGo task. Six blocks of 133 stimuli each (93 Go, M letter; 40 NoGo, W letter) were presented. The Go or NoGo letters were superimposed on two smoking-related backgrounds (SC), two non-smoking related backgrounds (NSC) or a neutral black background (NC).

# 3. Results

## 3.1. Questionnaires

Table 1 summarizes the characteristics of the population and differences between the smokers and the non-smokers on the questionnaires. Both groups were matched on gender, age and educational level. The groups did not differ on STAI. As expected, smokers had higher scores on the UPPS (and all its subscales). A stepwise regression reinforces these results as it shows that, when introducing all electrophysiological and behavioral variables, gender, age and the UPPS scores (and its subscales), UPPS scores significantly predict which group the participant belonged to ($b = 0.33$, $F(1,39) = 4.71$ $p < 0.05$, $r2 = 0.11$; $t = 2.17$ $p < 0.05$).

## 3.2. Behavioral data

The accuracy rates and reaction times for both the smoking and non-smoking group on the smoking-related Go-NoGo task are displayed in Table 2.

**Error rates**: Participants' performance were investigated under three different contexts: the smoking context (SC); the nonsmoking context (NSC) and no context (NC) and two conditions (NoGo and Go, differences between those two conditions revealing an inhibition process). A robust main effect of inhibition was found ($F(1,39) = 113.75$, $p < 0.001$, $g2 = 0.75$, observed power 1.00) showing that participants were less accurate on NoGo trials (99.98% accuracy for Go trials versus 87.49% for NoGo trials). There was also a main effect for group ($F(1,39) = 6.36$, $p < 0.05$, $g2 = 0.14$, observed power 0.69) which indicated that overall task performance was, as expected, less accurate in smokers than in nonsmoking controls (92.27% and 95.21%, respectively). An interaction was found for Group ⁄ Condition ($F(1,37) = 6.29$, $p < 0.05$, $g2 = 0.14$, observed power 0.69). Post-hoc t-tests revealed that, specifically on NoGo trials, smokers performed less accurately than non-smoking controls ($p < 0.05$; 84.45% vs. 90.43%), whereas there was no difference in accuracy between the groups for Go trials. A main effect was also found for the Context type ($F(2,78) = 14.33$, $p < 0.001$, $g2 = 0.27$, observed power 1.00) showing that participants made more errors with the NC background (NC: 7.33%; NSC: 6.22% and SC: 5.25%). An interaction for Group ⁄ Context was found ($F(2,78) = 3.28$, $p < 0.001$, $g2 = 0.08$, observed power 0.61) and post-hoc t-tests showed that smokers were always less accurate than controls. Differences were significant for the NSC (smokers had 8.20% of errors and non-smokers 4.24%) and NC (smokers had 8.78% and non-smokers 5.87%) and marginally significant ($p = 0.07$) for the SC (smokers had 6.23% and nonsmokers 4.26%). An interaction between Condition and Context was also found ($F(2,78) = 14.96$, $p < 0.001$, $g2 = 0.28$, observed power 1.00). Post-hoc t-tests revealed that, specifically on NoGo trials, more commission error rates were committed in NC. Finally a triple interaction of Condition ⁄ Group ⁄ Context ($F(2,78) = 3.31$, $p < 0.001$, $g2 = 0.08$, observed power 0.60) was found.

For this purpose and because there were differences in the distribution for Go and NoGo accuracy, which may lead to subsequent differences in the magnitude of effects for Go and NoGo accuracy, we additionally performed two separate ANOVAs for Go and NoGo accuracy scores. Results showed the same pattern as the combined analysis. A main effect for Group was found for NoGo accuracy ($F(1,39) = 6.33$, $p < 0.01$, $g2 = 0.15$, observed power 0.69) confirming that smokers were less accurate than controls on NoGo trials. No difference on accuracy between the groups was found for Go trials. A main effect of Context was found ($F(2,38) = 15.79$, $p < 0.01$, $g2 = 0.45$, observed power 0.99) confirming the NC condition elicited more false alarms (commission errors). And finally a Group ⁄ Context interaction ($F(2,38) = 4.63$, $p < 0.05$, $g2 = 0.08$, observed power 0.61) with smokers making more errors than controls irrespective of the context. However, Student's t-tests on each group showed that, for the SC condition only, smokers made significantly fewer errors as compared to the

NSC and NC condition, while controls made as many errors in the SC condition as in the NSC condition and significantly more errors in the NC condition.

A significant correlation, only for smokers, was found between the rate of commission errors and scores on urgency subscale of the UPPS: $t(18) = 0.47$ $p < 0.05$. No other correlation was found with the cigarette-related measures (number of cigarettes smoked per day, TQSU and FTND).

**Reaction time**: With regard to the reaction time data, we found no significant effects. No significant correlations were found between the number of cigarettes smoked per day, the TQSU and the level of dependency as measured by the FTND with reaction time. However, a significant correlation was found between accuracy rates and RT for NoGo trials only in smokers $t(18) = -0.48$ $p < 0.05$ revealing a speed accuracy trade-off.

**Table 2**
Reaction times on Go trials, omission and commission percentage rates are displayed for the three contexts and the mean of the three contexts for the two groups.

| | Smokers (n = 18) | Controls (n = 23) | p-value |
|---|---|---|---|
| *Smoking context* | | | |
| Go RTs (ms) | 318.52 (54) | 335.76 (51) | 0.15 |
| Omission error rates (%) | 0.06 | 0.04 | 0.86 |
| Commission error rates (%) | 12.4 (8)[a] | 8.48 (5) | 0.07 |
| *No Smoking context* | | | |
| Go RTs (ms) | 323.56 (55) | 346.79 (75)[b] | 0.30 |
| Omission error rates (%) | 0 | 0 | |
| Commission error rates (%) | 16.39 (11) | 8.48 (5) | p < 0.01 |
| *Without context* | | | |
| Go RTs (ms) | 319.51 (60) | 345.30 (51) | 0.15 |
| Omission error rates (%) | 0 | 0 | |
| Commission error rates (%) | 17.56 (10) | 11.74 (7)[*] | p < 0.05 |
| *Mean of the 3 contexts* | | | |
| Go RTs (ms) | 320.55 (56) | 342.62 (48) | 0.19 |
| Omission error rates (%) | 0.02 | 0.01 | 0.09 |
| Commission error rates (%) | 15.44 (9) | 9.57 (6) | p < 0.05 |

[*] Without context commission error rates (False Alarms) significantly different at $p < 0.05$ from the two other contexts (smoking and no smoking) for the controls.
[a] Smoking context commission error rates (False Alarms) significantly different at $p < 0.05$ from the two other contexts (no smoking and without context) for smokers.
[b] GO RT significantly different from the SC condition.

## 3.3. Electrophysiological data

In order to investigate the potentials elicited by the task, ANOVAs were computed for the N2 and P3 components, imputing condition type (Go-NoGo), context (NC, AC, NAC) and parameters (amplitude, latency) as within-subject variables, and group belonging as the between-subject variable. N2 and P3 amplitudes and latencies for smoking-related, non-smoking-related and no context backgrounds in both groups are displayed in Table 3.

### *3.3.1. N2*

**N2. amplitude**: In line with the hypotheses, a robust main effect was found for Inhibition ($F(1,39) = 15.13$, $p < 0.001$, $g2 = 0.28$, observed power 0.97) on the N2 component at the frontocentral electrode cluster, the Go-No-Go effect is represented in Fig. 2. This result demonstrates that N2 amplitudes were generally larger for NoGo trials (M = -1.33 mV) than for Go trials (M = 0.17 mV). A second main effect for Context was found ($F(2,38) = 21.59$, $p < 0.001$, $g2 = 0.36$, observed power 1.00) showing that N2 was larger in the NC condition (NC M :-2.15 mV, NS M:0.12 mV, SC M: 0.29 mV). No

group main or interaction effects were found for the N2 component. No significant correlations were found between the number of cigarettes smoked per day, the TQSU and the level of dependency and the cluster combined N2 peak amplitudes for NoGo trials.

**N2 latency**: Here again, a main effect was found for Inhibition ($F(1,39)= 40.87$, $p < 0.001$, $g2 = 0.51$, observed power 0.97) on the N2 latency showing N2 was later in NoGo condition (NoGo M: 256 ms, Go M : 226 ms), the Go-No-Go effect is represented in Fig. 2. A second main effect for Group was found ($F(1,39) = 9.52$, $p = 0.016$, $g2 = 0.20$, observed power 0.85) with smokers displaying a shorter latency (M: 231 ms) than non-smokers (M: 251 ms). A Group⁄Condition interaction ($F(1,39) = 18.44$, $p < 0.001$, $g2 = 0.32$, observed power 0.99) revealed that, in post-hoc t-tests, the difference between the two groups was on NoGo trials with smokers showing a shorter latency (M: 236 ms) as compared to controls (M: 275 ms) and this effect is represented in Fig. 3. No difference was found between groups for the Go trials. No significant correlations were found among the number of cigarettes smoked per day, the TQSU and the level of dependency and N2 latency.

### *3.3.2. P3*

**P3. amplitude**: The Inhibition main effect was also found for the P3 amplitude ($F(1,39) = 62.62$, $p < 0.001$, $g2 = 0.62$, observed power 1.00) with P3 waves being generally larger for NoGo trials (M = 14.43 mV) than for Go trials (M = 9.18 mV), the Go-No-Go effect is represented in Fig. 2. An interaction for Group⁄Context was found ($F(2,78) = 3.84$, $p = 0.015$, $g2 = 0.10$, observed power 0.75) and Post-hoc t-tests showed different patterns regarding the group. Indeed, smokers had a P3 amplitude significantly larger for SC backgrounds ($p < 0.05$, SC M: 13.19 mV) and no difference was found between the two other contexts (NSC M: 11.79 mV and NC M: 11.29 mV) while, for controls, the P3 amplitude was the largest for the NC background ($p < 0.05$, NC M:12.44 mV) and no difference was found regarding the two other contexts, (SC M: 11.17 mV and NSC M: 10.94 mV). These results are shown in Figs. 4 and 5. No group or context main effects were found for the P3 amplitude component. No significant correlations were found between the number of cigarettes smoked per day, the TQSU and the level of dependency and the cluster combined P3 peak amplitudes for NoGo trials.

**P3 latency**: The Inhibition main effect was also found for the P3 latency ($F(1,39) = 6.82$, $p = 0.013$ $g2 = 0.15$, observed power 0.72) with P3 waves being generally later for NoGo trials (M = 404) than for Go trials (Go M = 391), the Go-No-Go effect is represented in Fig. 2. No group or context main or interaction effects were found for the P3 latency component. No significant correlations were found among the number of cigarettes smoked per day, the TQSU and the level of dependency and the cluster combined P3 peak amplitudes for NoGo trials.

**Additional analysis**: In order to investigate if the differences between groups were due to spatial differences, as there is a possibility that the smoking context modifies the brain state or networks that are activated, we investigated the spatiotemporal characteristics of ERPs (lateralization and localization). This was done with a repeated measures 3-way ANOVA with the localization (frontal, central, parietal, occipital electrodes) and lateralization (left, center, right) as within-subjects variable and with the group (control participants and smokers) as a between-subject variable. The component values were measured with the following electrodes: F3, Fz, F4, C3, Cz, C4, P3, Pz, P4, O1, Oz, 02. Data are displayed in Table 4. Please note that no interaction with group and lateralization and/or localization was found. All reported topographical effects were then observable in both groups.

**N2 amplitude Smoking context**: Two main effects were found: localization ($F(3,102) = 9.52$, $p < 0.001$, $g2 = 0.22$, observed power 1.00) and lateralization ($F(2,68) = 4.95$, $p < 0.05$, $g2 = 0.13$, observed power 0.79). The N2 amplitude, in the smoking context was the most ample frontally (Mean = ——— 1.22) and the less ample parietally (Mean = 2.08) ($p < 0.05$ when compared to any other localization). Regarding lateralization, it was the most ample centrally (Mean =

0.66) and the less ample on both sides (Mean of the left side: 0.61; Mean of the right side = 0.77) ($p < 0.05$ when compared to any other lateralization). No main effect of status or interaction between smoking status and localization or lateralization were found.

**P3 amplitude Smoking context**: Two main effects were found: localization ($F(3,102) = 55.66$, $p < 0.001$, $g2 = 0.62$, observed power 1.00) and lateralization ($F(2,68) = 8.45$, $p < 0.01$, $g2 = 0.20$, observed power 0.96). The P3 amplitude, in the smoking context was the less ample occipitally (Mean = 4.63; $p < 0.05$ when compared to any other localization) and the most ample centrally (Mean = 14.50; $p < 0.05$ when compared both to parietal localization Mean: 11.13 and occipital). Regarding lateralization, it was the most ample centrally (Mean = 11.61; $p < 0.05$ when compared to the right side: Mean = 10.11). No main effect of status or interaction between smoking status and localization or lateralization were found.

**N2 amplitude Non-Smoking context**: A main effect was found: localization ($F(3,102) = 5.25$, $p < 0.01$, $g2 = 0.12$, observed power 0.92) with the N2 amplitude, in the non-smoking context which was the less ample parietally (Mean = 1.85; $p < 0.05$ when compared to any other localization). No main effect of status or interaction between smoking status and localization or lateralization were found.

**P3 amplitude Non-Smoking context**: Two main effects were found: localization ($F(3,102) = 42.29$, $p < 0.001$, $g2 = 0.52$, observed power 1.00) and lateralization ($F(2,68) = 11.04$, $p < 0.001$, $g2 = 0.2é$, observed power 0.99). The P3 amplitude, in the nonsmoking context was the less ample occipitally (Mean = 6.13; $p < 0.05$ when compared to any other localization). Regarding lateralization, it was the most ample centrally (Mean = 11.77; $p < 0.05$ when compared to any other lateralization). No main effect of status or interaction between smoking status and localization or lateralization were found.

**Table 3**
N2 and P3 amplitude and latency are displayed by condition (Go-NoGo) for the three contexts for the two groups.

| | Controls (n = 23) | Smokers (n = 18) | p-value |
|---|---|---|---|
| *Smoking context* | | | |
| Go-N2 amplitude | 1.75 (3.1) | 1.01 (3.8) | 0.50 |
| Go-N2 latency | 229 (39) | 224 (25) | 0.65 |
| NoGo-N2 amplitude | −0.55 (3.6) | −1.07 (4.2) | 0.67 |
| NoGo-N2 latency | 266 (38) | 240 (36) | 0.03 |
| Go-P3 amplitude | 8.77 (3.4) | 10.71 (3.8) | 0.09 |
| Go-P3 latency | 382 (36.4) | 388 (47) | 0.66 |
| NoGo-P3 amplitude | 13.57 (5.8) | 15.68 (5.6) | 0.25 |
| NoGo-P3 latency | 406 (40) | 405 (45) | 0.94 |
| *No Smoking context* | | | |
| Go-N2 amplitude | 0.96 (2.8) | 0.19 (4.2) | 0.49 |
| Go-N2 latency | 220 (34) | 225 (33) | 0.61 |
| NoGo-N2 amplitude | −0.38 (3.6) | −0.30 (4.3) | 0.94 |
| NoGo-N2 latency | 285 (48) | 235 (42) | 0.001 |
| Go-P3 amplitude | 7.93 (3.0) | 9.15 (3.6) | 0.25 |
| Go-P3 latency | 414 (37 | 387 (48) | 0.05 |
| NoGo-P3 amplitude | 13.94 (5.7) | 14.44 (5.9) | 0.79 |
| NoGo-P3 latency | 409 (36.5) | 393 (40) | 0.20 |
| *Without context* | | | |
| Go-N2 amplitude | −0.96 (4.2) | −1.93 (4.4) | 0.48 |
| Go-N2 latency | 231 (32) | 230 (19) | 0.96 |
| NoGo-N2 amplitude | −2.99 (2.8) | −2.71 (5.5) | 0.84 |
| NoGo-N2 latency | 275 (43) | 234 (29) | 0.001 |
| Go-P3 amplitude | 9.12 (3.9) | 9.40 (3.5) | 0.81 |
| Go-P3 latency | 391 (40) | 384 (45) | 0.61 |
| NoGo-P3 amplitude | 15.75 (7.7) | 13.18 (8.5) | 0.32 |
| NoGo-P3 latency | 420 (40) | 394 (57) | 0.09 |

# 4. Discussion

## 4.1. Discussion of main results

Although the literature has consistently proposed that smokers, as other addict populations, have high smoking-related cue reactivity and impaired inhibitory processing, results have been inconsistent until now (Dinn et al., 2004; Evans et al., 2009; Littel and Franken, 2007; Luijten et al., 2011). Indeed, there is also some evidence suggesting that nicotine may work as a cognitive enhancer (Evans and Drobes, 2008; Gehricke et al., 2006; Gehricke et al., 2007). The main purpose of the present study was so to investigate differences between smokers and controls in general response inhibition as well as specific inhibition on a behavioral and on an electrophysiological level, using a modified ‘‘contextual” Go-NoGo paradigm in combination with the recording of ERPs.

Agreeing with the notion that the N2 and P3 reflect an inhibitory process, both their amplitudes were significantly increased on NoGo trials as compared to Go trials in the whole population. More importantly, and as stated in our hypothesis, execution on the Go-NoGo task was generally less accurate in the NoGo trials in smokers than in non-smokers and the N2 latency in NoGo was shorter in smokers as compared to controls. No difference on N2 amplitude was found regardless of the context. However, and contrary to our initial hypothesis, specifically on the smoking-related background (SC), smokers made significantly fewer errors on NoGo trials and displayed an enhanced P3 amplitude for this context only, when compared to controls. Finally, surprisingly, the blackbackground context (No Context: NC) primed larger N2 waves in both groups as well as larger P3 waves and more behavioral errors in controls only.

Our first result thus shows that, behaviorally, smokers exhibited significantly worse motor response inhibition compared to the control group independent of the background. This population is known to exhibit more impulsivity than non-smokers (Mitchell, 2004; Verdejo-García et al., 2008; Zhou et al., 2010). Moreover, the urgency subscale was correlated with the general false alarms scores in the smokers only. This correlation is of particular interest as Billieux et al. (2007) showed that tobacco cravings are significantly predicted by urgency, while depression and anxiety are not. These authors actually advise that the influence of urgency on inhibition capacities evaluated by NoGo tasks should be explored.

Detandt, S., Bazan, A., Schroder, E., Olyff, G., Kajosch, H., Verbanck, P., & Campanella, S. (2017). A smoking-related background helps moderate smokers to focus. An Event-Related Potentials study during a Go-NoGo Task in smokers. *Clinical Neurophysiology, 128, 1872-1885.* (IF : 3.866)

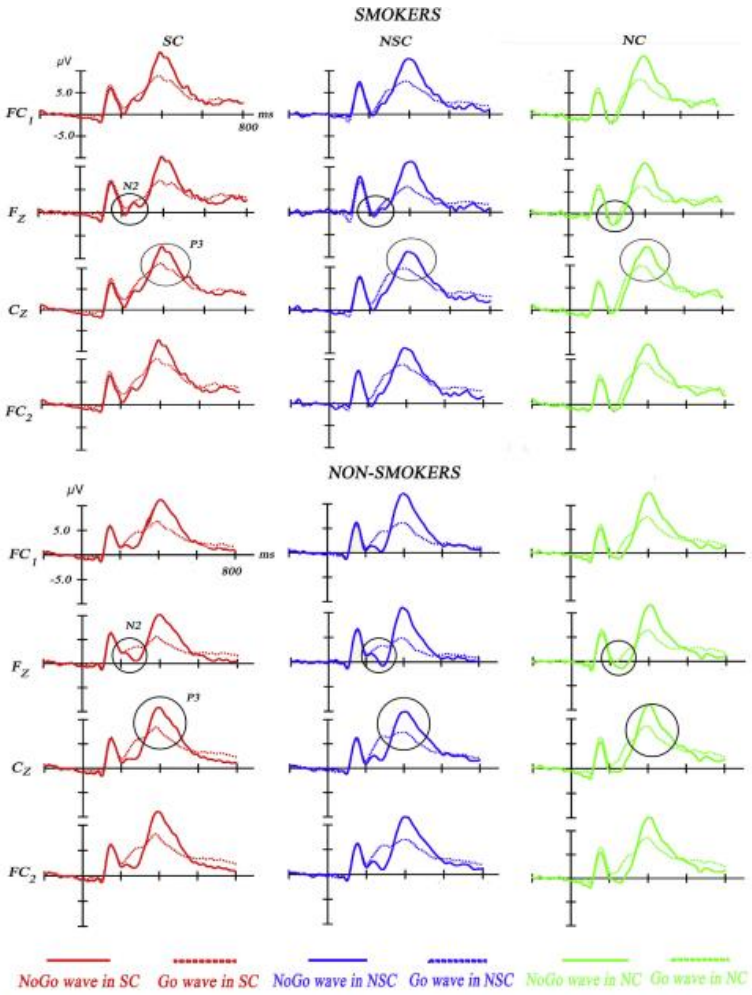


**Fig. 2.** Original Go-NoGo N2 and P3 waves (grand averages) in the smokers and non-smokers groups at the FC1, Fz, Cz and FC2 sites in the three contexts. Higher N2 and P3 amplitude are visible in No-Go as compared to Go trials in both groups and in the three contexts.

Interestingly, Billieux et al. (2010) have even suggested that impairments in the inhibition of prepotent responses might be one of the individual factors related to cigarette smoking dependency. So, this trait suggests that smokers may impulsively respond before having completely processed the stimulus, which would also explain the precocity of their NoGo-N2. Thus, it may be that non-smokers, who make fewer errors and have a later N2, are more cautious and process the stimulus more slowly.

Detandt, S., Bazan, A., Schroder, E., Olyff, G., Kajosch, H., Verbanck, P., & Campanella, S. (2017). A smoking-related background helps moderate smokers to focus. An Event-Related Potentials study during a Go-NoGo Task in smokers. *Clinical Neurophysiology, 128, 1872-1885.* (IF : 3.866)

Interestingly, the study of Wu et al. (2010) with a PTSD population (known for having inhibition deficits) sheds a complementary light on our results as their population also exhibited a shorter NoGo-N2 latency. The authors suggest that this behavior reflects a faster monitoring or detection of the response conflict, which may therefore be ‘‘an enhanced motor readiness or an increased prepotency to respond”, and consequently represent an ‘‘increased demand on inhibitory control systems” and ‘‘more urgent inhibition” (Wu et al., 2010: 120). This puts forward the idea that the impairment in behavioral inhibition is related to prior impulsive functioning. In the same line, Gajewski and Falkenstein (2013) have shown that the NoGo-N2 is sensitive to task complexity. A NoGo condition is logically more complex to handle than a Go condition. This, indeed, is seen in the control group, but the smokers, however, have no N2 latency increase in the NoGo condition, and make more errors in general. We propose then that this is again due to a general increase in impulsivity, which does not allow smokers to exert particular precautions when the task requires it.

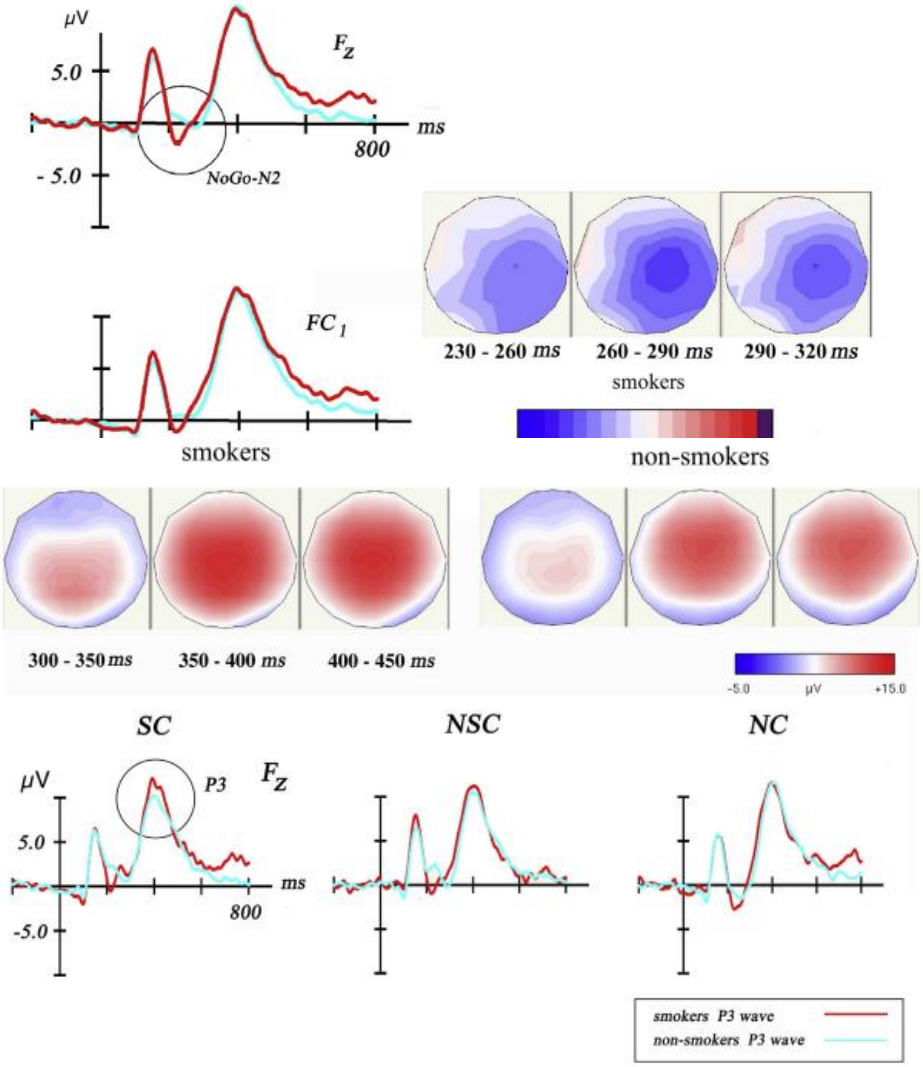


**Fig. 4.** Original NoGo P3 waves (grand averages) in the smokers and non-smokers groups in the three contexts illustrated on the Fz site. The topographical distribution of corresponding waves is also shown. Smokers show a significantly enhanced P3 compared to non-smokers, on the SC specifically.

**Fig. 3.** Original NoGo N2 waves (grand averages) in the smokers and non-smokers groups (mean of the three contexts) at the FC1, Fz, Cz and FC2 sites. The topographical distribution of corresponding waves is also shown. Smokers show a significantly reduced NoGo-N2 compared to non-smokers, whatever the context.

Second, we found no difference on N2 amplitude between smokers and non-smokers, contrary to our expectations and to what has been found by Luijten et al. (2011), who found a general difference between groups (independent of context and stimulus). This absence of difference might reflect a similar level of conflict monitoring in both our groups (O’Connell et al., 2009). Studies on alcoholics

(Ridderinkhof et al., 2002) have similarly found no difference on the N2 amplitude but have shown a reduced ErrorRelated Negativity (ERN). Recent work on the specificity of N2 (Yeung and Cohen, 2006), indeed, shows that error monitoring and conflict monitoring are indexed by two separates waves: the ERN and the N2. The ERN, then, would be the index of an early warning of conditions in which errors are expected and potentially warning that increased attention is required. In our smokers, the conflict monitoring does not seem impaired, but speculatively, this does not preclude an earlier deficiency, at the ERN stage.

Third, remarkably, smokers make significantly fewer errors in a smoking-related context as compared to other contexts, and the amplitude of the P3 is specifically enhanced in this context. The significant increase of the NoGo-P3 generally reflects successful inhibition (Falkenstein et al., 1999; O'Connell et al., 2009) but, this P3 enhancement is shown on both the Go P3 and the NoGo P3. This, then, may reflect a general attentional enhancement, related to both incentive salience and inhibition. According to the incentive salience theory (IST; Robinson and Berridge, 2003), the motor investment towards addiction-related cues should push the for ward grasping movement for the addictive object (Bazan and Detandt, 2013). In our paradigm a classical prediction would have anticipated smokers to be less accurate on NoGo trials when confronted with a smoking-related background, as the addictionrelated forward move would forcefully interfere with the inhibition task. However, we found that this was not the case. However, it is important to note that the stimulus to grasp – a capitalized letter – has, in fact, no particular salience and thus the logic of the salience theory might not apply as such here. It is in fact the background, which is addiction-related. Instead, we propose that the general P3-enhancement reflects some sort of compensatory mechanism exerted by the cigarette-related background, allowing smokers to achieve better performance – indeed at almost the same level as controls.

The reason why we could uncover these discrete differences may be related to the use of longer-lasting backgrounds, which provide a more ecological design than those used in previous research. Moreover, while in many studies, behavioral differences could only be uncovered at a high level of dependency (Luijten et al., 2014; Monterosso et al., 2005), our smoker group showed light-to-medium levels of dependency and, compared to smoker groups in similar studies, didn't consume more cigarettes or didn't have a higher dependency rate on the FTND (it was

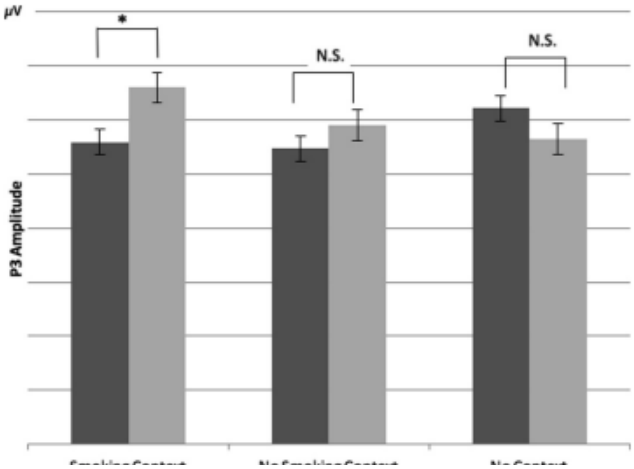


**Table 4**
N2 and P3 amplitudes displayed for the NoGo condition in the Non-Smoking and Smoking context for both localization (Frontal-Central-Parietal-Occipital) and lateralization (Left-Central-Right).

| | Frontal | | | Central | | | Parietal | | | Occipital | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Left | Central | Right | Left | Central | Right | Left | Central | Right | Left | Central | Right |
| N2 Non-Smoking context | −0.82 (0.71) | −0.81 (0.73) | −0.29 (0.67) | 0.73 (0.62) | 0.31 (0.68) | 0.6 (0.52 | 1.90 (0.64) | 1.99 (0.56) | 1.673 (0.50) | 0.52 (0.49) | 0.43 (0.52) | 0.54 (0.51) |
| P3 Non-smoking context | 11.06 (0.72) | 12.91 (0.94) | 11.77 (0.77) | 13.40 (0.78) | 14.99 (1.00) | 13.03 (0.75) | 11.09 (0.79) | 12.69 (0.76) | 10.51 (0.67) | 6.42 (0.58) | 6.50 (0.54) | 5.46 (0.55) |
| N2 Smoking context | −1.36 (0.63) | −1.84 (0.69) | −0.45 (0.63) | 1.23 (0.65) | −0.38 (0.65) | 1.77 (0.71) | 2.30 (0.63) | 2.13 (0.71) | 1.83 (0.64) | 0.29 (0.58) | −0.18 (0.50) | −0.06 (0.59) |
| P3 Smoking context | 12.41 (0.77) | 13.53 (0.89) | 12.83 (0.78) | 14.11 (0.92) | 15.65 (1.11) | 13.74 (0.91) | 10.60 (0.83) | 12.65 (1.02) | 10.13 (0.92) | 5.53 (0.90) | 4.60 (0.71) | 3.75 (0.64 |

even the opposite, see Supplementary Table S1). Therefore, the observed differences in the present research cannot be explained by higher levels of nicotine in the participants of the present study. Our results corroborate other findings, which suggest that addiction cannot be reduced to the quantity of nicotine inhaled as this quantity cannot explain the variety of smoking behaviors observed. Shiffman and colleagues (2015) already proposed that smoking patterns can be better categorized by a model of smoking that also allows for stimulus control to influence smoking. In a recent study (Smoking addiction: the shift from head to hands), we show that a shift seems to have operated from a mental preoccupation with smoking in low dependent smokers (based on the TND cut-off of 4) to smoking as a motor habit in dependent smokers while, remarkably, there was no difference in the number of cigarettes consumed. (Detandt, Bazan, Quertemont, & Verbanck, in preparation). In this study, participants had to complete a battery of declarative questionnaires on their attitudes towards smoking and to perform another modified Go-NoGo task using tobaccorelated words and neutral words as stimuli. Results showed that smokers generally made more mistakes both on neutral and smoking-related words and tended to be faster for smokingrelated cues. But, interestingly, smokers with low dependency were more eager to acknowledge their addiction in declarative questionnaires while making more errors and being slower on smoking cues in their motor approach behavior, while dependent smokers were less prone to indicate their addiction declaratively while having more accurate and fast responses when it came to selecting the smoking cues in the motor approach task. This second result is in line what we found out in the present study that is to say that the quantity of cigarettes smoked per day cannot define the level of addiction.

Finally, the NC primed larger N2 waves in both groups as well as larger P3 waves and more behavioral errors in controls only. We can speculate that this result can be explained by the fact that, in controls - who were generally good at inhibition - the absence of a background cue aroused less interest in the task and tired them more (it has to be noted that some participants specifically indicated the NC background required more effort than the SC background as the NC background was visually exhausting). Therefore, the N2 and P3 enhancements might reflect an increase in cognitive resources, which is high enough to maintain arousal not to fall asleep but not enough to be attentive. Further investigations should be done and a way to avoid this concern could be to use an extremely difficult situation of inhibitory control in a challenging stop task by using an algorithm that adjusts the task individually on the basis of individual RTs (e.g., Rubia et al., 2003).

### 4.2. Smoking as a self-remedy?

Based on the present findings, we would like to discuss more broadly some of the questions elicited by our observations. First, in the present study, we haven't been able to indicate any direction of causality whatsoever regarding (reduced) inhibition, impulsivity and smoking. It is possible that long-term smoking leads to neuronal modifications (specifically in the dopamine system), which could result in reduced inhibition. But, it is also possible that, inversely, reduced inhibition and enhanced impulsivity actually predispose to start smoking. Our results would actually lean towards the second interpretation as there is no correlation between inhibitory control and nicotine exposure (i.e. the number of cigarettes smoked per day). Furthermore, we have found both a general inhibition bias in smokers together with an improvement of performance and increased P3 with smoke-related backgrounds specifically.

The fact that smokers actually seem helped by the smokingrelated background challenges the current idea that smoking-related stimuli would grab their attention and limit their other abilities, such as their performance on NoGo trials. While many studies have shown a bias towards addiction-related cues, most of them either try to answer why this kind of bias exists or to which extent the intensity of

it will predict relapse (Volkow et al., 2003). But, to our knowledge, it has rarely been investigated what function this bias might serve for the person, while with the present data we propose to see smoking also as a form of (effective) auto-treatment.

Taking both observations together, then, a speculative perspective on cigarette addiction, suggested by these data, would be that a predisposition to general impulsivity promotes cigarette addiction as smoking effectively improves focusing and performance. Whereas this proposition might seem controversial, it is in line with e.g. Evans and colleagues (2009), who propose that smokers might use cigarettes precisely to alleviate difficulties in focusing on stimuli. Indeed, growing research (Mihailescu and DruckerColín, 2000; Kumari and Postma, 2005) has shown that cigarette smoking has the ability to remediate cognitive impairments. Research shows that nicotine and other tobacco constituents modulate specifically the dopaminergic activity in the prefrontal cortex, amygdala, nucleus accumbens and cingular gyrus (Berrendero et al., 2010; Rezvani and Levin, 2001) and this has a significant impact on attention regulation. Moreover, it has also been shown that other populations with attention deficits and impulsivity (ADHD; Gehricke et al., 2007; schizophrenia, Dervaux and Laqueille, 2008; Harris et al., 2004) may smoke to reduce symptoms associated with their inhibitory impairment, such as attention and working memory deficits. In other words, smoking might reveal here as a partial self-remedy to the biases induced by impulsivity, and, unfortunately, this cognitive enhancing ability may potentially be a factor in the maintenance of smoking.

This proposition is only preliminary but, the notion that some people may use smoking as a cognitive enhancing drug may have important implications for smoking cessation strategies. These strategies might, for example, also take into account the possibility of potential cognitive benefits lost when one stops smoking.

### 4.3. Considerations for future research

Before concluding, we propose some suggestions for future research resulting from the present study. Considering the target population, distinctions between subclinical categories (low to moderate and heavy smokers) should specify how the level of addiction may interact with: (1) general cognitive biases; and (2) the processing of specific smoking-related cues. In addition, establishing long-term follow-up studies in which individuals are tested t least one year would allow new hypotheses concerning the predictive factors of relapse (including impulsivity). Specifically concerning impulsivity, we suggest that studies with non-dependent populations with high urgency scores are required to explore the mechanisms underlying the urgency component of impulsivity. Also, proposing the same set-up to a population of ex-smokers who take nicotine replacement therapy should be of great interest in order to disentangle the direct impact of nicotine exposure on the one hand and smoking in its broader sense on the other hand on inhibitory functions: if nicotine is the decisive element, then results should be similar in ex-smokers on nicotine replacement, however if addiction as a psychological concept is at stake, then results should be quite different. In the same line, biochemical measures (in order to control for the smoking status in both smokers and non-smokers independently of self-reports) should be added in any other design in order to disentangle the craving variations due to the initial level of cotinine in plasma levels. In terms of electrophysiology, adding the measure of the ERN component should help to disentangle our results concerning the N2 amplitude and provide insight concerning performance monitoring. The use of Transcranial Magnetic Stimulation (TMS) in combination with EEG should also be of interest to measure cortical inhibition (Daskalakis et al., 2008) and possibly give further support to the hypothesis of differential inhibitory behaviours among smokers and non-smokers.

Finally, in order to determine if our results contradict the IST which predicts that a smoking-related background should compete with other cues in smokers, inducing them to fail to disengage

their attention from the background, leading to less accuracy in this group, a paradigm in which the Go-NoGo cue is directly related to smoking (such as a cigarette) should be implemented. In that case, when there is a cigarette on which participants can click, it is predicted that smokers will indeed be less accurate when they have to inhibit their response.

### 4.4. Conclusion

Based a narrow definition of the dual-process theory, a smoking-related background is predicted to compete with other cues in smokers, inducing them to fail to disengage their attention from the background, leading to less accuracy in a Go-Nogo task. However, this prediction is disavowed by our data and another reading emerges, which proposes that smokers are, in a certain way, given mental support by the smoke-related background, which is automatically processed and may help sustain attention for the principal task. Interestingly, what should be stressed here is that smokers are not helped by merely smoking cigarettes, i.e. by a direct influence of nicotine, but by the representation of a smoking-related background.

In conclusion, much research has still to be done to show how we can maximize the benefits of smoking prevention and cessation programs keeping in mind the plurality of the functions smoking addiction can hold and therefore what is the most acceptable, most efficient, most tolerable and profitable for individuals. The present study, however, might bring empirical support to the very idea of self-remedy through smoking and, together with other observations, leads to considering smoking addiction as a mental concept which transcends the concept of mere physiological dependency.

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