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Changes in the Electroencephalographic Spectrum in Response to Smoking Cues in Smokers and Ex-Smokers

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Key Words
Smoking · Cue reactivity · EEG · Beta power · Craving · Processing bias

Abstract
Aims: To investigate the changes in the electroencephalographic (EEG) spectrum in smokers during exposure to a neutral and a smoking-related cue to determine whether these EEG changes are still present in ex-smokers after prolonged abstinence and to examine the relationship between the power in each spectral bandwidth and subjective craving. ***Methods:*** EEG frequencies in response to a smoking-related and a neutral cue were examined in 23 smokers and 21 ex-smokers, who quit smoking for 1.4 years on average. Additionally, self-report measures of cigarette craving and nicotine dependence were obtained. The spectral power of each bandwidth was computed, log-transformed, and analyzed using a within-subject design. Differences between EEG activity under neutral and smoking conditions were correlated with differences between pre- and postexperimental subjective craving. ***Results:*** Increases in reward craving (desire and intention to smoke) were associated with reduced theta activity, whereas increases in withdrawal craving (reduction of negative affect and withdrawal symptoms) were correlated with increases in both delta and higher alpha power. Furthermore, in smokers, but not in ex-smokers, a significant beta power increase was observed between the neutral condition and the smoking condition. ***Conclusion:*** Since the beta band is associated with arousal, attention, and alertness, it is suggested that the beta increase in response to the smoking cue might reflect an enhanced allocation of resources to smoking-related stimuli, i.e. a processing bias, which is an important feature of substance abuse. Since ex-smokers do not respond to the smoking cue with beta activity enhancement, we preliminarily conclude that smoking cues do not arouse ex-smokers or capture their attention as much as they do in smokers.

Introduction

Cigarette smoking has been associated with increases in alertness and mood changes [e.g. 1–7]. These findings are supported by measures using electroencephalography (EEG) techniques, which reveal that nicotine administration leads to strong increases in electrophysiological activity, i.e. scalp-recorded activity shifts from low (delta, theta, alpha-1) to high (alpha-2, beta) frequencies, indicating a state of arousal. The reverse is true for nicotine abstinence: deprivation causes increases in theta power, and leads to reductions in both alpha and beta frequency [for an overview, see 8, 9]. Decreases in alpha frequency

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have been associated with slow reaction time [10, 11], diminished arousal and decreased vigilance [12, 13]. Increases in theta power are correlated with drowsiness [13, 14] and the transition from wakefulness to sleep [15]. These changes in EEG spectrum in response to smoking abstinence persist until at least 1 month after quitting [16]. As far as we know, there are no studies investigating EEG activity in prolonged (>1 month) smoking abstinence, and therefore it is unknown how EEG power and frequency will develop after a 1-year period of abstinence.

Besides shifts from high to low frequencies, smoking deprivation has been associated with shifts regarding the balance of alpha activity between the left versus the right frontal hemisphere [17]. When people are presented with appetitive stimuli, and are motivated to approach these, they tend to display relatively greater left than right activation; conversely, people presented with aversive stimuli or people under withdrawal-associated conditions display relatively greater right than left frontal activation [18–22]. If smokers who are deprived for 24 h are exposed to cigarette cues or anticipate smoking, they show left frontal asymmetry, i.e. greater left than right frontal hemispheric activation, suggesting an enhanced approach motivation as a result of the deprivation [17].

Most EEG spectrum research in smokers is focussed on the spontaneous electrophysiological changes during nicotine intake and/or abstinence. Only few studies have addressed the question whether EEG power and frequency are affected by (imaginary) exposure to cigarette cues. Knott et al. [23] found smoking urge scripts, depicting scenes with persons experiencing a desire to smoke, to increase both beta and theta activity, whereas they found no-urge scripts, depicting the same scenes without smoking desires, to have no effects on the EEG frequency domains. Comparable results were observed in cocaine users: during cocaine-craving-related guided imagery, both theta and beta power increased. During active cocaine paraphernalia handling and video viewing only beta activity was enhanced. Changes in delta power were more dependent on task: during imagery, there was an increase in delta, whereas a drop was observed in the paraphernalia and video task [24]. In contrast, Knott et al. [23] found delta activity to be decreased in response to urge-related imagery, but only in males. Alpha activity remained unaffected by urge and no-urge scripts and drug-related stimuli in both smokers and cocaine users [23, 24].

It has been suggested [23, 24] that the EEG changes in the frequency domain as a result of urge-related imagery reflect drug craving. However, either no correlation analyses between the power in each frequency band and self-reported craving were performed [23] or they led to non-significant results [24]. It might also be possible that the EEG changes reflect other processes such as a general enhancement of arousal, or an enhanced allocation of cognitive resources to smoking-related cues, i.e. a processing bias, which is a concept associated with craving [25–28]. At present, it is not clear whether self-reported cigarette craving is related to changes in EEG frequency and power.

Changes in beta, theta and delta might be neurophysiological indices of self-reported craving, since urge scripts are able to provoke changes only in these frequency domains. Encouraged by the structure of the Questionnaire on Smoking Urges (QSU-brief [29]), which subdivides craving into 'desire and intention to smoke' (reward craving) and 'reduction of negative affect and withdrawal symptoms' (withdrawal craving), based on respectively appetitive-incentive models of craving [e.g. 30–33] and associative-withdrawal models of craving [e.g. 34–36], Knott et al. [23] make several assumptions. First, they assume delta reductions and beta increments, which are smoking-like EEG changes, to be correlated with reward craving. The second subscale, or withdrawal craving, is expected to correlate with theta increases, a withdrawal-like EEG change. However, these assumptions need to be confirmed.

Within the present study, we examined the EEG spectrum changes in smokers during exposure to a neutral and a smoking-related cue. In line with the results of Knott et al. [23], we expect smokers' beta and theta activity to increase in response to the smoking-related cue, but not in response to the neutral cue. Because of conflicting findings concerning the direction of the changes in the delta frequency [23, 24], we only hypothesize a change in delta in response to the smoking cue.

Furthermore, the present study addressed the question whether EEG frequency domain changes in response to smoking cues are still present in ex-smokers after prolonged abstinence (>1 year). Recently, research has shown that ex-smokers, at least to some extent, exhibit an extinction of the cortical reactivity towards smoking cues [37]. In the present study, we expected to find differences between smokers and ex-smokers in line with these results, i.e. that ex-smokers will not respond with changes in delta, beta and theta activity to the smoking-related cues, or at least to a lesser degree than smokers.

Moreover, we expected increases in self-reported craving, as measured with the QSU-brief, to be correlated with increases in beta and theta EEG activity and de-

creases in delta EEG activity. In accordance with the assumptions of Knott et al. [23], we expected reward craving ('desire and intention to smoke') to be associated with delta reductions and beta increases, and withdrawal craving ('reduction of negative affect and withdrawal symptoms') with theta increases.

Finally, because of their greater approach motivation in smoking-related contexts, we expect more left frontal alpha asymmetry (left > right) in smokers than in ex-smokers during the presentation of the smoking cue. Since ex-smokers tend to evaluate smoking cues as less pleasurable than smokers do and also tend to evaluate smoking cues as less pleasurable than neutral cues [37], they will probably show more alpha activity in the right frontal hemisphere than smokers.

Method

Subjects

Twenty-two smokers and 21 ex-smokers [partly the same as reported in 37] participated in this study, which was approved by the institutional ethical board. Smokers (23.3% males, mean age 21.5 years, SD = 2.4) smoked at minimum 10 cigarettes a day. Ex-smokers (9.3% males, mean age 23.3 years, SD = 3.9) quit smoking at least 6 months ago and did not smoke a single cigarette within that period. Smokers and ex-smokers did not differ significantly in age ($t(41) = 1.9$, $p = 0.07$), smoking duration (smokers = 4.8 years, SD = 2.7 years; ex-smokers = 5.3 years, SD = 3.1 years; $t(41) = 1.6$, $p = 0.56$) or nicotine dependence [smokers' Fagerström Test of Nicotine Dependence (FTND) score = 3.5, SD = 2.3; ex-smokers' FTND score = 2.7, SD = 2.5; $t(41) = 1.0$, $p = 0.31$]. Furthermore, sex ratio was equal in both groups (χ^2 (1, n = 43) = 3.4, $p = 0.07$). The mean quit duration of ex-smokers was 1.4 years (SD = 1.7). Because of the marginally significant sex ratio, sex was added as covariate in all analyses. No significant main or interaction effect of sex was found. Therefore, we report the analyses without sex as covariate.

The groups consisted predominantly of undergraduate psychology students, who received course credit or a small financial compensation for participation.

Procedure

Smokers were asked to abstain from smoking for at least 1 h before the experiment. This short period of smoking deprivation served to reduce the acute effects of nicotine on electrocortical arousal and accordingly to decrease the differences between smokers and ex-smokers.

First of all, participants completed a questionnaire about demographics and smoking history: the FTND [38]. After completion, participants were seated in an EEG chair in a sound- and light-attenuated room; electrodes were attached, and a pictorial task was presented [reported elsewhere, see 37]. Instructions were to sit relaxed and still and to carefully attend to the cues without employing distracting thoughts. After the task was completed, the subjects filled out the QSU-brief [29].

In the present experiment, two cues were presented. The first cue consisted of a pen on a small dish (control/neutral condition), the second consisted of a lit cigarette on the same dish (smoking-related condition). Each cue was located at approximately eye level about 1 m in front of the participants and was presented for 30 s.

After the cue presentation, electrodes were removed and subjects filled out the QSU-brief for the second time. After having completed the experiment, subjects received their course credit or financial compensation.

Self-Report Measures

Demographic and smoking history data were self-reported [age, period(s) of abstinence, and smoking duration]. Craving was assessed twice by means of the QSU-brief [29], before and after cue presentation. This 10-item questionnaire is adapted from the QSU [39] and consists of two subscales: 'desire and intention to smoke', and 'reduction of negative affect and withdrawal symptoms'. These subscales have adequate psychometric properties [29].

Strength of smoking habit was measured with the Dutch version of the FTND [38, 40]. This questionnaire consists of 6 items, which are scored according to the scoring system described by Heatherton et al. [38]. The FTND has good reliability and correlates significantly with number of cigarettes smoked per day. Ex-smokers answered the questions retrospectively. Retrospectively assessed FTND scores also have adequate psychometric properties [41].

Physiological Measures

EEG was recorded with a digital BioSemi Active-Two system, using active Ag/AgCl electrodes at 34 scalp sites according to the International 10/10 system [42] (32 standard channels mounted in an elastic cap and two mastoid locations, M1 and M2, which were used for off-line re-referencing). The vertical electro-oculogram (VEOG) was recorded with two active Ag/AgCl electrodes located above and underneath the left eye. The horizontal electro-oculogram (HEOG) was recorded with two Ag/AgCl electrodes located at the outer canthus of each eye. An additional active electrode (CMS, common mode sense) and a passive electrode (DRL, driven right leg) were used to comprise a feedback loop for amplifier reference. All signals were digitized with a sample rate of 1,024 Hz and 24-bit A/D conversion with a low-pass filter of 134 Hz. Offline, the EEG signals were referenced to the mathematically linked mastoids, and EEG and EOG were phase-shift-free filtered using a 0.1- to 40-Hz (24 dB/octave roll off) band-pass filter.

Data Reduction and Analysis

EEG and EOG recordings were segmented in two 30-second epochs and corrected for vertical and horizontal eye movements and eye blinks using the Gratton and Coles algorithm [43]. After the EOG correction, we excluded segments containing artifacts. The absolute difference between two values in a segment was not allowed to exceed 200 μV. To analyze band power changes during exposure to the smoking, each EEG epoch was divided in 30 1-second segments. For smokers under the neutral condition, the mean number of artifact-free segments was 28.41. For ex-smokers, this was 29.29. For smokers under the smoking condition, the mean number of artifact-free segments was 29.18. For ex-smokers

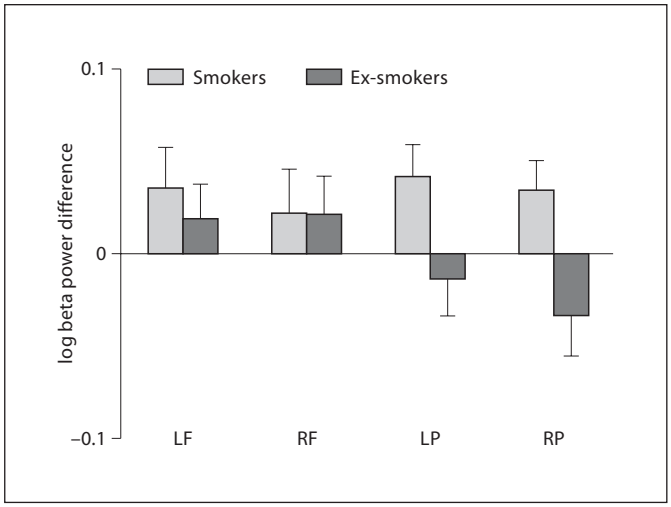

Fig. 1. Differences between log beta power in response to the smoking cue and the neutral cue for smokers and ex-smokers at left frontal (LF), right frontal (RF), left posterior (LP), and right posterior (RP) clusters (including error bars).

under the same condition, this was 29.10. Each segment was Fast Fourier transformed using a Hanning window of 10%. For each condition, the Fast Fourier transforms (FFTs) were averaged and delta (0.75–3.75 Hz), theta (3.75–7.75 Hz), alpha-1 (7.75–10.75 Hz), alpha-2 (10.75–13.75 Hz), alpha-total (7.75–13.75 Hz) and beta (13.75–29.75 Hz) band power was measured. For each of these 6 frequency bands and each of the 32 electrodes, log-transformed [log (x)] power (μV^2) was calculated.

Statistical Analyses

The electrodes were divided into 4 clusters: left frontal (LF; Fp1, AF3, F7, F3, FC5), right frontal (RF; Fp2, AF4, F8, F4, FC6), left posterior (LP; CP5, CP1, P3, PO3, O1), and right posterior (RP; CP6, CP2, P4, PO4, O2) [44]. For each of the 6 frequency bands, a 4 (cluster) × 2 (condition: pen, cigarette) × 2 (group: smokers, ex-smokers) repeated-measures analysis of variance (RM ANOVA) was performed. Greenhouse-Geisser correction was applied when applicable.

To assess relationships between EEG activity changes and increase in craving as a consequence of exposure to the cue, Spearman's ρ coefficients were calculated between the difference between the mean cluster activity in the smoking and neutral condition (mean increase or decrease in cluster activity between the two conditions) in each of the bands and the difference between the post- and preexposure QSU-brief scores. We selected Spearman correlation because of nonnormal distributed data of the QSU-brief.

In order to measure left frontal alpha asymmetry, two RM ANOVAs were performed. The first was a 2 (frontal clusters) × 2 (condition) × 2 (group) RM ANOVA; the second was almost identical except for the frontal clusters. In this analysis, frontal clusters were replaced by the specific frontal electrodes F3 and F4, which are similar to the locations used by Zinser et al. [17]. An alpha level of 0.05 was used for all statistical tests.

Results

Because our interest mainly concerned group differences, only group and group-interaction effects are reported. Furthermore, and in order to reduce the number of results, we report only significant (or borderline significant) effects.

EEG Frequency Bands

Beta exhibited a significant cluster × condition × group interaction effect ($F_{3, 123} = 4.37$, $p < 0.05$). For smokers, post-hoc tests with Bonferroni adjustments revealed a significant beta power increase between the neutral and smoking condition at the left posterior cluster ($p < 0.05$). Additionally, this beta increase was nearly significant at the left frontal and right posterior clusters (both p values = 0.08). In contrast, for ex-smokers, no significant beta increase was observed. However, at the right posterior cluster, a decrease in beta power almost reached significance ($p = 0.09$). These results are confirmed by a follow-up t test, which showed a significant difference between smokers and ex-smokers for the difference between the neutral and the smoking condition at both the left posterior cluster ($t(41) = -2.14$, $p < 0.05$) and the right posterior cluster ($t(41) = -2.50$, $p < 0.05$). To summarize, when exposed to smoking-related cues, smokers' beta increased, yet ex-smokers' beta did not change (or even displayed a nonsignificant tendency to decrease; fig. 1). These group differences are found particularly at posterior sites.

For the theta band, a cluster × condition × group effect was also found ($F_{3, 123} = 6.48$, $p < 0.01$). However, post-hoc analyses revealed no significant differences between groups or conditions. In addition, we computed group differences on cue-induced change scores at each cluster using t tests. Again, this did not result in significant effects. The significant interaction was probably caused by differences between clusters dependent on condition and group. These effects are beyond our scope of interest.

No interactions were found between clusters, conditions, and groups in the other EEG bands.

Alpha Asymmetry

When comparing activity at all left hemisphere electrodes to activity at all right hemisphere electrodes, no significant differences are found between groups or conditions for alpha-1, alpha-2, and alpha-total (all p values > 0.42). In addition, when comparing activity at F3 (left) and F4 (right) electrodes, no significant alpha differences are found either (all p values > 0.143).

Self-Reported Craving

A time (pre-QSU vs. post-QSU) × group ANOVA revealed significant differences between groups on scores at pre-QSU ($F_{1, 41} = 57.27$, $p < 0.001$) and post-QSU ($F_{1, 41} = 59.68$, $p < 0.001$). The difference between the mean postexposure QSU-brief total score and the mean preexposure QSU-brief total score was significantly larger for smokers (M = 6.0, SD = 6.7) than for ex-smokers (M = 1.8, SD = 4.2; $t(41) = 2.36$, $p < 0.05$). This difference between smokers and ex-smokers was mainly the result of smokers' larger difference between pre- and postexperiment scores on the first QSU subscale 'desire and intention to smoke' ($t(41) = 2.36$, $p < 0.05$). Smokers did not differ from ex-smokers on differences between the pre- and posttest on the second subscale 'reduction of negative affect and withdrawal symptoms' ($t(41) = 0.85$, $p = 0.40$). When analyzing groups separately, ex-smokers report no increase in craving at all ($t(20) = 1.98$, $p = 0.06$), whereas smokers report a strong increase ($t(21) = 4.23$, $p < 0.001$).

EEG and Craving among Smokers

Because ex-smokers did not show any increase in craving in response to the smoking-related cue, we only examined the data of the smokers for the EEG craving correlation analysis.

Only correlations between QSU subscales, i.e. 'desire and intention to smoke', and 'reduction of negative affect and withdrawal symptoms', and mean increase or decrease in activity per frequency band will be reported below.

Increases in scores on the first subscale of the QSU (desire and intention) are significantly correlated with a left posterior decrease in theta activity ($\rho = -0.51$, $p < 0.05$). Furthermore, increases in scores on the second subscale of the QSU (negative affect and withdrawal) are significantly correlated with left and right frontal increases in delta activity ($\rho = 0.45$, $p < 0.05$ and $\rho = 0.53$, $p < 0.05$, respectively) and a left posterior increase in higher alpha (alpha-2) activity ($\rho = 0.57$, $p < 0.01$).

Conclusions

The present study investigated EEG spectrum changes in smokers and ex-smokers in response to a neutral (pen) and a smoking-related cue (lit cigarette). In line with our hypotheses, and in line with the results of Knott et al. [23], a significant increase in beta power between the two conditions was observed in the smokers. Ex-smokers did not show such an increase in beta power. If anything, their beta tended to decrease (borderline significance) as a consequence of the smoking cue.

In general, beta power increments are thought to reflect increases in cortical arousal [45] and have been associated with perception, cognition, the orienting response, attentive-like behavior and increased activation in an attentional alertness/vigilance network [46–50]. In studies addressing addiction, beta power increases have been associated with exposure to a cocaine cue, cocaine craving-related guided imagery [24], exposure to smoking urge scripts [23], and both actual nicotine [8, 9] and cocaine administration [51–53].

Although we cannot exclude the possibility that our beta power enhancement merely reflects an arousal increment, the beta power enhancement may reflect a processing bias for drug-related stimuli. Processing bias refers to the enhanced processing of drug-related stimuli, which is a consequence of their strong acquisition of incentive motivational properties [33]. During the course of drug use, drug-associated stimuli become extremely salient and a greater proportion of attentional resources is allocated to them than to other (rewarding) stimuli [25, 33]. Studies investigating smoking cue reactivity by means of Stroop tasks, visual probe tasks and the later components of event-related potentials (ERP: P300; late positive potential) have shown a processing bias in smokers [e.g. 54–57], but not in ex-smokers [37, 58]. Because of the association of beta with arousal and attention, the above-mentioned processing bias might be in accordance with a beta increase in the processing of drug-related cues. Egner and Gruzelier [46, 47] have demonstrated this relationship more directly: beta training appears to be reliably correlated with the enhancement of P300 ERP component amplitudes.

In the present study, the smokers' increase in beta activity probably reflects their enhanced processing of smoking-related cues, i.e. their processing bias. This idea is strengthened by the topography of the beta increase, which is predominantly posterior in nature and thus reflects arousal in temporal and occipital parts of the brain. Activation of the visual cortex is in agreement with previous findings that smokers, compared to non-smokers, maintain their gaze longer on smoking-related cues than on neutral cues [26], which is also considered an indication of enhanced attention and processing bias. Since ex-smokers do not show increases in beta activity, we can preliminarily conclude that this might reflect an absence of processing bias in this population, which is in line with results from previous studies [37, 58]. The smoking-re-

lated cue does not lead to the same amount of arousal and attention in ex-smokers as it does in smokers. Oddly enough, arousal and attention tend to decrease in ex-smokers. Although this result is not significant and should therefore be interpreted cautiously, it might reflect some kind of avoidance mechanism, e.g. caused by disgust, or automated coping strategy, i.e. a cognitive distraction or relaxation technique. Further research on this topic is necessary.

In contrast to the results of Knott et al. [23], we found no significant changes in delta and theta activity between conditions, nor did we find any differences between the two groups. These differences between our results and those of Knott et al. may have been caused by differences in the experimental manipulation. Knott et al. obtained their recordings during imagery-elicited cravings, and not in response to in vivo cues, where craving processes may be experienced differently.

However, we did find delta and theta frequency bands to be correlated with subjective craving. Knott et al. [23] suggested that reductions in delta and increments in beta, both smoking-like EEG changes, and increases in theta, a withdrawal-like EEG change, could be paralleled by neural substrates reflecting reward craving and neural substrates reflecting withdrawal craving, respectively.

As for theta and delta, just about the opposite was observed. Increases in reward craving were significantly correlated with reductions in left posterior theta activity. Furthermore, increases in withdrawal craving were associated with increases in frontal delta activity. This latter result is in line with results of Reid et al. [52], who found that self-reported cocaine craving was correlated with delta power during the first 5 min following cocaine self-administration. Nevertheless, correlations between self-reported craving and EEG activity were absent in most other studies [24, 59]. However, in contrast to the present study, these studies concern cocaine addiction, cocaine paraphernalia and cocaine-related craving questionnaires. Differences in substances of abuse might cause the inconsistencies in correlations between EEG activity and subjective craving. There are no studies on nicotine addiction that have adequately addressed the correlation between EEG oscillations and craving.

In addition to reductions in theta and increases in delta, we found increases in alpha-2 activity to be correlated with craving. Specifically, increases in left posterior higher alpha were significantly associated with increases in withdrawal craving. This is in contrast with results from all other EEG frequency domain studies on exposure to drug cue and craving, which reported either no relation [23, 24, 52] or a borderline significant negative correlation [59] between alpha power and craving. Finally, neither alpha-1 nor beta activity was associated with self-reported craving.

Besides differences between smokers and ex-smokers in EEG spectrum changes as a result of the presented cues, and a relation between these EEG changes and craving, we hypothesized that smokers, compared to ex-smokers, would display greater approach motivation to the smoking cue than to the neutral cue. We expected this to be reflected by a greater left frontal alpha asymmetry in the smokers than in the ex-smokers. However, we did not find any alpha power differences between groups or hemispheres. This is not in line with the results of Zinser et al. [17], who found EEG asymmetry to be increased as a consequence of seeing a cigarette. Nevertheless, their study differed from ours in many ways. First of all, smokers had been abstinent for 24 h, whereas smokers in our study had been deprived for only 1 h. Second, smokers in the above-mentioned study were told that they were allowed to smoke immediately after the experiment, whereas our smokers did not receive any instructions, except for concentrating on the stimulus at hand. Both deprivation and anticipation of drug use are thought to augment drug motivation [e.g. 55, 60–64], and although we successfully manipulated craving levels, which also reflect augmented drug motivation [33], the differences in deprivation and anticipation might have had an influence on the absence of left frontal alpha asymmetry in the present study.

A limitation of the current study is that the subjects performed a pictorial task first [reported elsewhere, see 37]. This task, in which 16 different smoking-related pictures and 16 neutral pictures were presented 4 times, may have impacted the present measurements. Although the pen and the lit cigarette were new, unexpected, multimodal (sight and smell) and more realistic than the stimuli in the pictorial task, and although the presentation of these cues led to a significant increase in subjective craving in smokers, the continuous presentation of so many smoking cues may have caused some habituation, which may have changed or reduced the effects. Research investigating the effects of repeated drug exposure has shown that both physiological reactivity and self-reported cue reactivity decrease over time [65], which makes it plausible that the EEG activity of smokers in the current experiment could have been subject to habituation. However, both groups were presented with the same cues and the same amount of cues, and in spite of possible habituation, the significant group differences still remain.

Another limitation is that we did not counterbalance the order of the two cues across participants. Counterbalancing was not possible because of the smoke and smell a lit cigarette produces. During the presentation of the pen, the participants would still have smelled the cigarette and the condition would not have been neutral anymore.

A third limitation is that we used self-report to validate ex-smoker status. In the future, smoking status should be validated with a biochemical marker.

The main conclusion of the present study is that smokers, but not ex-smokers, show an increase in beta activity in response to a smoking-related cue compared to a neutral cue. Since activity in the beta frequency band has been associated with heightened arousal, attention, alertness and enhancement of the P300 component of the ERP, the increase in beta activity in the current study might reflect an enhanced allocation of resources to smoking-related stimuli, i.e. a processing bias, which was found in smokers in previous studies and is a very important feature of substance abuse. Since ex-smokers do not respond to the smoking cue with beta activity enhancement, we preliminarily conclude that smoking cues do not arouse ex-smokers as much as they arouse smokers.

Furthermore, we are the first to establish the relationship between EEG spectrum changes in response to smoking-related cues and self-reported cigarette craving by means of correlation analyses. In smokers, reward craving ('desire and intention to smoke') is associated with reduced theta activity. Withdrawal craving ('reduction of negative affect and withdrawal symptoms'), on the other hand, holds positive correlations with increases in both delta and alpha-2 activity.

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