



Electroencephalography reveals a selective disruption of cognitive control processes in craving cigarette smokers

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Abstract

Addiction to nicotine is extremely challenging to overcome, and the intense craving for the next cigarette often leads to relapse in smokers who wish to quit. To dampen the urges of craving and inhibit unwanted behaviour, smokers must harness cognitive control, which is itself impaired in addiction. It is likely that craving may interact with cognitive control, and the present study sought to test the specificity of such interactions. To this end, data from 24 smokers were gathered using EEG and behavioural measures in a craving session (following a three-hour nicotine abstinence period) and a non-craving session (having just smoked). In both sessions, participants performed a task probing various facets of cognitive control (response inhibition, task switching and conflict processing). Results showed that craving smokers were less flexible with the implementation of cognitive control, with demands of task switching and incongruency yielding greater deficits under conditions of craving. Importantly, inhibitory control was not affected by craving, suggesting that the interactions of craving and cognitive control are selective. Together, these results provide evidence that smokers already exhibit specific control-related deficits after brief nicotine deprivation. This disruption of cognitive control while craving may help to explain why abstinence is so difficult to maintain.

KEYWORDS

addiction, craving, EEG, event-related potentials, nicotine

Abbreviations: ACC, anterior cingulate cortex; CNV, contingent negative variation; DLPFC, dorsal lateral prefrontal cortex; EEG, electroencephalography; ERP, event-related potential; fMRI, functional magnetic resonance imaging; FTND, Fagerström test for nicotine dependence; ICA, independent component analysis; ITI, inter-trial interval; Pe, error positivity; pre-SMA, pre-supplementary motor area; QSU, questionnaire of smoking urges; ROI, region of interest; RT, response time; SEM, standard error of the mean; VEOG, vertical electro-oculogram.

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1 | INTRODUCTION

Despite the many health warnings and known negative consequences, smoking remains a relatively popular habit throughout the world, with the World Health Organization projecting that over 1 billion people will regularly smoke tobacco by 2025 (Bilano et al., 2015). Although quitting is a goal for many smokers, the state of craving that accompanies nicotine deprivation is a formidable obstacle in maintaining abstinence, with the measured strength of this urge positively correlating with relapse (Killen & Fortmann, 1997). Smokers who successfully quit recruit cognitive control mechanisms to actively suppress their feelings of craving, thereby avoiding relapse (Potenza, Sofuoglu, Carroll, & Rounsaville, 2011). Specifically, to maintain abstinence, a smoker must inhibit the automatic urge to smoke, ignore the distracting smoking-related cues around them and focus on other non-smoking-related tasks, so as to avoid cue-induced craving effects (Carter & Tiffany, 1999). In this way, craving and cognitive control mechanisms during abstinence are pitted against one other, with successful cessation or relapse representing alternative outcomes of this competition. The strength of craving, however, is twofold, as it can be both an acute reaction, often triggered by smoking-related cues, but also a tonic state, independent of such cues (Ferguson & Shiffman, 2009). An important question is if, in the absence of smoking-related cues, the more tonic state of craving is disruptive to cognitive control mechanisms in a way that might account for the failures of nicotine abstinence during nicotine deprivation.

Cognitive control, a form of executive function, is a term that encompasses the subcomponents of cognition necessary for goal-directed behaviour, including (but not limited to) the inhibition of unwanted responses, flexible preparation for upcoming events, and selecting that which is relevant in the presence of conflicting input (see Banich, 2009 and Gratton, Cooper, Fabiani, Carter, & Karayanidis, 2017 for reviews). These cognitive control processes are thought to entail dynamic interactions involving frontal-lobe networks (Helfrich & Knight, 2016), with specific frontal subregions being implicated in different control processes (Badre & Nee, 2018). The various facets of cognitive control have been characterized using EEG and fMRI measures, with each control process showing a unique signature in terms of the timing and distribution of underlying activations. For example, in Go/NoGo tasks requiring response inhibition, inhibition-related activity is often found in the right inferior frontal gyrus, the insula and pre-SMA (Garavan, Ross, & Stein, 1999; Sebastian et al., 2012; Zhang, Geng, & Lee, 2017), and manifests electrophysiologically as an N2/P3 ERP difference (e.g. Albert, López-Martín, Hinojosa, & Carretié, 2013; Harper, Malone, & Bernat, 2014; Huster, Enriquez-Geppert, Lavalée, Falkenstein, & Herrmann, 2013; Pfefferbaum, Ford, Weller, & Kopell, 1985). Preparatory activity for a

given task has been associated with a fronto-centrally distributed, slow negative-polarity ERP wave, referred to as the contingent negative variation (CNV; Astle, Jackson, & Swainson, 2006; Poljac & Yeung, 2014), which increases when more effort is required (e.g. when the participant must switch tasks and maintain the correct task in working memory (Astle, Jackson, & Swainson, 2008; Poljac & Yeung, 2014; Vandamme, Szmalec, Liefoghe, & Vandierendonck, 2010)). This component has been reported to have contributing sources in regions that include the insula and supplementary motor areas (Nagai et al., 2004), and fMRI has revealed that, in addition to activation of pre-SMA, task switching recruits the basal ganglia and DLPFC (Crone, Wendelken, Donohue, & Bunge, 2006). Finally, conflict processing has been most often associated with the ACC, manifesting in EEG data as a greater negativity for incongruent relative to congruent trial types, known as the N_{inc} or N450 (Carter, 1998; Carter & van Veen, 2007; Donohue, Appelbaum, Appelbaum, McKay, & Woldorff, 2016; Siltan et al., 2010; West & Alain, 1999). Together, these processes, in conjunction with selective attention, allow for the control of actions, and when these processes are not effectively invoked, unwanted behaviours such as compulsive substance usage in addiction can emerge.

One hallmark of addiction is indeed poor cognitive control, and inhibitory control is the specific control process that is most often noted as being deficient in individuals who regularly use nicotine, alcohol or illegal drugs (Belin, Belin-Rauscent, Murray, & Everitt, 2013; Flaudias et al., 2016; Goldstein & Volkow, 2011). Although cognitive control is more often characterized in users of other addictive substances, there is evidence that such cognitive control deficits also exist in smokers (e.g. Billieux et al., 2010; Dinn, Aycicegi, & Harris, 2004; Wagner et al., 2012). Indeed, a meta-analysis of behavioural studies concluded that inhibitory control deficits are present in smokers compared with controls, particularly in the Go/NoGo task (Smith, Mattick, Jamadar, & Iredale, 2014). Further, such reduced inhibitory control in smokers can be observed with neural measures, even if no obvious behavioural deficit is present (Luijten et al., 2014). The evidence for neural inhibitory deficits, however, is not entirely consistent, with two studies comparing smokers to controls in a Go/NoGo task finding differences on the N2 but not on the P3 (Buzzell, Fedota, Roberts, & McDonald, 2014; Luijten, Littel, & Franken, 2011), and another study showing that non-smokers had a greater difference in P3 amplitude for NoGo versus Go trials than smokers (Evans, Park, Maxfield, & Drobles, 2009). Moreover, in an fMRI study of a Go/NoGo task, the only differences observed between smokers and controls were found in non-frontal regions, such as the cerebellum (Weywadt, Kiehl, & Claus, 2017), whereas another study found decreased neural activity in prefrontal regions for smokers relative to controls (Nestor, McCabe, Jones, Clancy, & Garavan, 2011). Interestingly,

only one of the aforementioned neural studies (Nestor et al., 2011) found a behavioural difference between smokers and controls in inhibitory control, suggesting the importance of neural measures to pick up on more subtle effects and compensatory mechanisms. As such, although there is evidence to suggest that inhibitory control is impaired in smokers, the exact nature of the impairment is not very clear.

Others aspects of cognitive control have been less thoroughly examined in smokers, and the few studies that have sought to test these facets have also yielded mixed results. A behavioural study implementing a wide variety of executive function tasks in smokers found that the level of smoking (the product of average daily use \times years of smoking) was a significant predictor of some executive function measures, including task switching and response speed in the inhibition-related Go/NoGo and Stop-Signal tasks, but this did not influence conflict processing as measured with a Stroop task (Glass et al., 2009). In an fMRI study investigating conflict processing in smokers versus controls, smokers showed decreased accuracy and reduced activity in the right anterior insula for one conflict condition relative to controls, but not for all conflict conditions (Fedota et al., 2016). Yet another study found that smoking history only influenced performance on task switching and not on conflict or other attention-related executive function measures (Razani, Boone, Lesser, & Weiss, 2004). Importantly, the participants there were almost all former smokers, and only those who had once been heavy smokers showed the task-switching impairment. Together, these data suggest that in addition to the aforementioned inhibitory impairments, smokers may have impairments in cognitive control related to task switching, but other aspects, such as conflict, may only be moderately affected.

Given the impairments of cognitive control in smokers, it is reasonable to expect that the state of craving would only exacerbate these problems. Surprisingly, however, this is not necessarily the case. Dawkins and colleagues (Dawkins, Powell, West, Powell, & Pickering, 2007) found that deprived smokers had impaired inhibitory control in responding to stimuli, which was rescued by the administration of nicotine, but also that other aspects of cognitive control such as working memory were not impaired. These results would suggest that craving selectively influences certain control processes. Nevertheless, their study used only behavioural measures, and it is entirely possible that there may have been differences in the underlying neural activity, which the behavioural measures were unable to capture. That is, craving could still operate in a non-specific manner, altering all attention and cognitive control processes, with behavioural ramifications only showing up for certain cognitive control tasks. In a previous study (Donohue, Woldorff, et al., 2016), we observed that when smokers were in a state of craving, they had an enhanced sensory-evoked P1, suggesting a higher general level

of arousal. Surprisingly, this had no consequences for an attentional-shift-related task, with smokers showing comparable behavioural and neural effects in both sated and craving conditions. The task we used previously, however, primarily engaged ventral visual regions (Hopf et al., 2000) and was not designed to test more control-related areas.

To specifically test the behavioural and neural ramifications of craving on cognitive control, we implemented a cuing paradigm with manipulations of task switching, conflict processing and response inhibition while recording EEG measures of brain activity. This paradigm was run on each smoker twice: once when they had smoked immediately prior to the experiment (i.e. a non-craving condition) and once when they had abstained from smoking for the three hours leading up to the session (i.e. a craving condition). If craving were exclusively a low-level process that mainly influences arousal, then we would not expect to see any task-specific influences as a function of craving, but perhaps just a general impairment of performance. In contrast, if craving does have specific detrimental effects on cognitive control, as previous behavioural results have suggested (Dawkins et al., 2007), then these effects would manifest in some but not all of the cognitive control tasks implemented here.

While the task used here focused on various subcomponents of cognitive control, a final aspect to consider is the motivation of the participants, and specifically, how they respond to feedback. When a participant receives feedback, this can translate into performance monitoring, where behaviour on subsequent trials may be adjusted according to this feedback (Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004). Because the task we used was relatively challenging, we gave our participants feedback on their performance at the end of every trial. They were specifically awarded points (more for faster accurate responses), and once they had reached 3,000 points, the session ended and they were free to go smoke. We included this to keep subjects engaged in the task, but we also sought to examine how craving influenced the response to feedback. Smokers who have been deprived of nicotine for 12 hr have been found to show decreased sensitivity to monetary rewards (Lydon, Roberts, & Geier, 2015) and have decreased signatures of performance monitoring (Schlitz, Hawk, & Rosch, 2013). We therefore hypothesized that it could be the case that the smokers in our study would be overall less sensitive to the feedback when they were craving, but also that they may differentiate more between low and high feedback, as high feedback would bring them closer to their reward of smoking. To fully assess the response to feedback, we examined this reward-related feedback not only on the current trial, but also on the subsequent trial, to see how it ramified. If smokers are less sensitive to feedback when they are craving, then this should not only be reflected in a decreased feedback-related P3 response (Ullsperger, Fischer, Nigbur, & Endrass, 2014) on the current trial, but it

should also manifest in a lack of feedback-related changes in preparation and performance on the subsequent trial.

2 | METHODS

2.1 | Subjects

Smokers were recruited from the Otto-von-Guericke University of Magdeburg and the surrounding community. In total, data from 24 participants (mean age 27.2 years, range 18–45, 12 female) were included in the final analysis. A sample size of 23 participants was calculated with G*Power (Faul, Erdfelder, Lang, & Buchner, 2007) to be sufficient for a moderate within-subject effect size (Cohen's $f > 0.25$) at an alpha of 0.05. Additionally, studies examining similar components have used a similar (or smaller) sample size (e.g. 23 subjects for the CNV (Morie, Sanctis, & Foxe, 2014); 20 subjects for the N_{inc} (McKay, Berg, & Woldorff, 2017)). All included participants had neither a history of (or current) drug or alcohol abuse nor any diagnosis of neurological disease/psychiatric illness. Data from several additional participants were excluded due to an excessive amount of physiological noise in their EEG data in one or both sessions ($N = 5$), or due to poor performance on the task ($N = 3$). All smokers reported smoking on average at least 10 cigarettes per day (mean = 15) and had all smoked regularly for at least two years (mean years smoked = 10). All participants gave written, informed consent, and all procedures were approved by the Ethics Committee of the medical school at the Otto-von-Guericke University, Magdeburg, Germany.

2.2 | General experiment design

Each participant participated in two separate sessions, occurring on different days. In one session, the *non-craving session*, the participant smoked immediately prior to the start of the experiment. In the other session, the *craving session*, the participant arrived at the laboratory, smoked a cigarette and then waited for three hours under direct experimenter supervision to ensure he/she did not smoke within this time period. After these three hours, the experiment began. The order of the sessions was randomized and counterbalanced across participants. In both sessions, the experimental task was the same, with the only difference being whether or not the participant had recently smoked.

2.3 | Stimuli and task

The paradigm used here was a task-switching paradigm, designed to invoke switching, incongruity and inhibitory

effects. For each trial, a circle of one of four colours (e.g. green, blue, red and brown) appeared, indicating which task the participants should prepare for (i.e. a cue). Two of the four colours corresponded to an even/odd judgement on an upcoming number stimulus. The other two colours indicated that participants should make a greater than/less than five judgements on the upcoming number stimulus. Two colours were used instead of one for each type of judgement to prevent the observed effects being attributable to cue-colour-switching costs rather than task-switching costs (Mayr & Kliegl, 2003). This cue was presented for 300 ms. After a delay period (during which only a fixation cross was present for 1,100–1,400 ms, randomly jittered), the target stimulus (single-digit number, 1° wide \times 1.5° high) was presented for 300 ms. On 80% of the trials, the target stimulus was presented in the same colour as its preceding cue, and participants were instructed to make the relevant judgement as quickly as possible. On 20% of the trials, the target stimulus would be presented in a fifth colour (e.g. magenta), and in these cases, participants were asked to withhold their response (i.e. a NoGo condition). After the target stimulus was presented, another fixation cross was present for 1,000–1,300 ms (jittered), to allow participants enough time to respond. Following the response, participants were given feedback (lasting 700 ms) as to their accuracy and how many points they had won for that trial. The cue stimulus was 2.5° of visual angle in diameter, and all stimuli (cue, target, feedback) were presented at 1.5° below fixation using Presentation (Neurobehavioral Systems). The colour corresponding to which judgement/NoGo signal was randomized across participants, but remained the same for a given participant across both sessions.

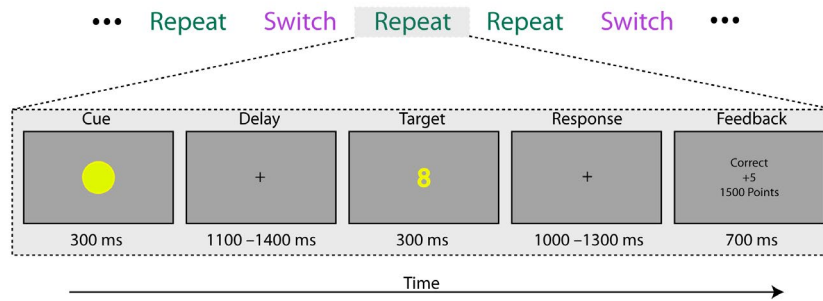
2.3.1 | Task-switching component

From trial to trial, the task assigned by the cue would either switch (i.e. go from an even/odd judgement on the previous trial to a greater than/less than judgement on the current trial, or vice versa) or repeat (i.e. both the current trial and the previous trial would be even/odd judgements, or both be greater than/less than judgements). The ratio of switch to repeat trials was 40% to 60%. An example excerpt from the trial sequence is shown in Figure 1a.

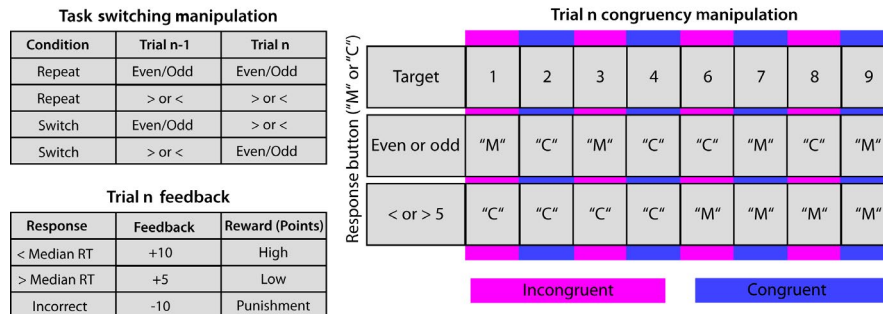
2.3.2 | Congruency component

For each task, a response button was assigned to a specific judgement. For example, if a number were odd, participants were asked to press “M,” whereas if it were even, they were asked to press “C.” The same response buttons were used for the greater than/less than five judgement task, such that “C”

(a) Example trial sequence



(b) Experimental conditions



(c) Time-lock points for analyses

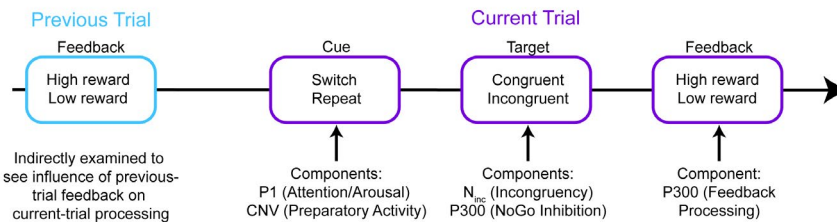


FIGURE 1 (a) Example Trial Sequence. From trial to trial, the task could either repeat (e.g. an even/odd judgement followed by another even/odd judgement), or a greater than/less than judgement followed by another greater than/less than judgement) or switch (e.g. an even/odd judgement followed by a greater than or less than five judgement, or vice versa). Displayed here is one trial (a repeat), showing the cue, delay, target, response and feedback periods. Following the feedback, a jittered ITI was presented before the next trial began. (b) Experimental Conditions. The three task manipulations (task switching, congruency and feedback) are depicted here. Of note, the incongruency effects were induced by a response conflict, where a given target had either one response regardless of task or two possible responses, contingent upon task. The feedback was given on every trial, and once the participant had earned a total of 3,000 points, the experiment was ended (and he/she was free to smoke). (c) Time-lock Points for Analyses. Each of the arrows points to an event to which the time-locked ERP averaging was calculated, and listed below these arrows are the components of interest that were extracted. [Colour figure can be viewed at wileyonlinelibrary.com]

could be used for numbers less than five and "M" was used for numbers greater than five. This created two different levels of congruency as a function of response. That is, for half the numbers, regardless of which task the participants were cued to do, the response would be the same (i.e. a congruent condition). For the other half of the numbers, the response was task-dependent (i.e. would have been "M" for one task and "C" for another task), thereby representing an incongruent condition. Participants were not told which numbers were congruent or incongruent, and, based on debriefing, if this were learned at all, it was learned implicitly. A depiction of the congruency by stimulus mapping is shown in the right panel of Figure 1b.

2.3.3 | Response inhibition

As mentioned above, for 20% per cent of the trials the target number would appear in a fifth colour, and participants needed to withhold from making a response (NoGo trials). All the other trials were thus considered "Go" trials.

2.3.4 | Feedback

In order to motivate participants to perform quickly and accurately, performance feedback was provided following every trial and indicated points earned for each correct

response given. For the first trial of each task type, participants earned 10 points for every correct response given. After this, the median RT was obtained and if a participant responded more quickly than this value, he/she earned 10 points (high feedback), and if a participant responded slower than this value, he/she earned 5 points (low feedback). During piloting, we observed that, for many participants, their overall RT was a bit faster for the greater than five/less than five task and thus to avoid biasing one task, two separate median RT counters were kept, one for each task and the speed for a given trial was only calculated relative to that task's previous responses. Responses that fell outside the response window (150–1,200 ms after target onset) were counted as misses. For the NoGo trials, correctly abstaining from responding earned participants 10 points, and all incorrect responses, including false alarms on NoGo trials, cost participants 10 points. At the start of the experiment, participants were told that the session ended once they reached 3,000 total points. The total value of points earned was displayed on every trial, along with the feedback for that particular trial. Participants were told that if they had not achieved 3,000 points after an hour of recording time, the experiment would be ended automatically. Of note, the data reported here were from participants who earned 3,000 points in both sessions before the hour was up.

2.4 | General procedure

Each session took place in a dimly lit, electrically shielded recording chamber. At the start of each session, participants received one or more practice blocks to ensure that they had learned the response mapping and were comfortable with the task. Within the task, after every ~7 min, participants were given a break and could press a button when they wished to resume the experiment. The first trial of the experiment and the first trial after each break were excluded from analysis.

2.5 | Questionnaires

At the start of each EEG session, participants were given the Questionnaire of Smoking Urges (QSU; Tiffany & Drobes, 1991) to determine their level of self-reported craving. This questionnaire results in data that are sorted into two factors. Factor one captures the anticipation of the pleasure of smoking (positive aspect), and the second factor measures the anticipated relief of the unpleasant symptoms of nicotine deprivation (negative aspect). Both factors were analysed to determine whether our craving manipulation was effective insofar as self-report measures can provide.

Additionally, the Fagerström Test for Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerström, 1991), as well as a questionnaire on smoking history, were administered.

2.6 | EEG measurement

Continuous EEG data were recorded using Brain Products Amplifiers and caps (the 32-Channel ActiChamp System with an Acticap and Vision Recorder software (Brain Products Inc., Gilching, Germany)) during both the craving and non-craving sessions while participants performed the task. The EEG data consisted of 32 channels, arranged in a standard 10–20 montage and referenced online to the right mastoid. The data were sampled at a frequency of 500 Hz, and impedances were maintained at or below 5 kOhms. One VEOG channel was placed under the right ocular orbit to monitor blinks.

2.7 | EEG analysis

Offline, the EEG data were processed using EEGLab (Delorme & Makeig, 2004) and ERPLab (Lopez-Calderon & Luck, 2014) software toolboxes in MATLAB. The EEG data were epoched (–400 to 1,600 ms), separately for time-lock points at the cue, target, and feedback onsets. An initial blink-based artefact rejection was performed for ± 200 ms after the onset of each event to ensure that only trials for which the subject had perceived the stimulus were retained (i.e. that he/she had not blinked during that display). After this, ICA decomposition was run on the epoched data, separately for the cues, targets and feedback. The output of this decomposition (i.e. 32 components for each trial period and subject) was then inspected by examination of the spatial distribution of each component and its respective time course. Components that could be clearly identified as noise (e.g. blink-related) were removed. After these components were removed, a second round of artefact rejection was implemented to remove any trials still containing major physiological artefacts not removed during the ICA. The total amount of trials rejected ended up being: cue (non-craving) mean = 1.69%; range = 0%–9.31%; cue (craving) mean = 1.46%; range = 0%–4.24%; target (non-craving) mean = 1.25%; range = 0%–8.17%; target (craving) mean = 0.77%; range = 0%–4.45%; feedback (non-craving) mean = 1.55%; range = 0%–9.15%; feedback (craving) mean = 2.07%; range = 0%–11.18%. Importantly, for the cue, target and feedback, there were no significant differences between rejection rates for craving and non-craving sessions (all p 's > 0.3). The data were then selectively averaged, low-pass filtered at 30 Hz, re-referenced

to the algebraic average of the left and right mastoids, and relevant difference waves were obtained for the respective conditions.

2.7.1 | Statistical analysis of ERP data

Each event (cue, target and feedback) had different components of interest. Figure 1c delineates which components were expected to be elicited during each time period. The precise time windows and analysis for each are detailed, below, and a summary of the ERP analyses conducted is presented in Table 1.

Cue-locked ERPs

For the cue period, the initial sensory-evoked responses (the P1 at occipital sites O1 and O2 from 110 to 150 ms) were examined as a function of task, the feedback from the previous trial, and craving. Based on our previous work, we hypothesized that craving would influence this component (Donohue, Woldorff, et al., 2016). The time window and sites were selected by collapsing across both sessions/conditions and visually inspecting the data to see the location/time of the first occipitally located positive-going deflection in the data, and taking ± 20 ms around the peak. The mean amplitudes at these sites and for this time period were then extracted for each session and condition, and submitted to a $2 \times 2 \times 2$ repeated measures ANOVA, with the factors of craving (craving, non-craving), task switching (switch, repeat) and feedback (previous-trial high, previous-trial low). The second cue-locked component of interest was the CNV. Again, here, the data were collapsed across all sessions and conditions and the location of the maximal negative-going deflection (at site Cz) was obtained. The time period for the CNV was divided into an early (700–1,000 ms) and late (1,000–1,300 ms) time period. For each time period, a $2 \times 2 \times 2$ repeated measures ANOVA was conducted on the mean amplitude

values with the factors of craving (craving, non-craving), task switching (switch, repeat) and feedback (previous-trial high, previous-trial low).

Target-locked ERPs

For the ERP time-locked to the onset of the target, we examined an incongruity-related effect (the N_{inc}) and a Go/NoGo response inhibition-related effect. Based on previous literature (Donohue, Appelbaum, et al., 2016; Liotti, Woldorff, Perez, & Mayberg, 2000), we expected a centrally distributed negativity for the N_{inc} (incongruent minus congruent), occurring roughly between 300 and 600 ms. To refine the time window and distribution for this experiment, we isolated the effect via a collapsed localizer in which the difference wave of all incongruent minus all congruent trials was obtained. This revealed a time range (350–550 ms) and distribution (maximal at Cp1, Cp2, Pz, P3, P4) of this difference, which was subsequently examined for the effects of craving, switching and feedback. That is, the mean amplitudes for this time window averaged across the aforementioned sites were analysed in a $2 \times 2 \times 2 \times 2$ repeated measures ANOVA, with the factors of craving (craving, non-craving), task switching (switch, repeat), feedback (previous-trial high, previous-trial low) and incongruity (incongruent, congruent). Of note, as this effect was defined based on incongruity, a significant main effect of incongruity is circular; nevertheless, this was included as a factor in the ANOVA to track modulations of the incongruity effects by the other factors (i.e. to examine potential interactions). Based on previous literature, we expected to see two primary effects for the Go/NoGo component of the task: an N2 and a P300. Visual inspection of all NoGo minus all Go trials revealed no clear N2, and this component was therefore not analysed further. A clear P300 was present, however, maximal at site Cz, and lasting from 300 to 700 ms. The mean amplitudes were then extracted at this site for this time range and submitted to a $2 \times 2 \times 2 \times 2$ repeated measures ANOVA, with the factors of craving

TABLE 1 Task period, components, time windows and factors used in ERP analyses

Task Period	Component	Time Window	ANOVA Factors
Cue	P1	110–150 ms	Craving (craving, non-craving); Task Switching (switch, repeat); Previous Feedback (prev-high, prev-low)
Cue	CNV (early)	700–1,000 ms	Craving (craving, non-craving); Task Switching (switch, repeat); Previous Feedback (prev-high, prev-low)
Cue	CNV (late)	1,000–1,300 ms	Craving (craving, non-craving); Task Switching (switch, repeat); Previous Feedback (prev-high, prev-low)
Target	N_{inc}	350–550 ms	Craving (craving, non-craving); Task Switching (switch, repeat); Previous Feedback (prev-high, prev-low); Congruency (congruent, incongruent)
Target	P300 (Go/NoGo)	300–700 ms	Craving (craving, non-craving); Task Switching (switch, repeat); Previous Feedback (prev-high, prev-low); trial type (Go, NoGo)
Feedback	P300	300 – 400 ms	Craving (craving, non-craving); Feedback (high, low)

(craving, non-craving), task switching (switch, repeat), feedback (previous-trial high, previous-trial low) and trial type (Go, NoGo).

Feedback-locked ERPs

For feedback-locked activity, we examined the P300 component. Here, the distribution of this component (collapsed across types of feedback) revealed a brief peak around 350 ms at site Cz, and the time period around this peak (300–400 ms) was examined. Specifically, a 2×2 repeated measures ANOVA with the factors of craving (craving, non-craving) and feedback (current trial high, current trial low) was run to determine whether the P300 was modulated by these factors. As the number of incorrect trials was small, those were not included in this analysis. Of note, the results of all ANOVAs reported for all analyses are Greenhouse–Geisser corrected.

2.8 | Behavioural analysis

The behavioural analysis examined only those trials associated with artefact-free EEG data. Accuracy (error rates) and response times (RTs) were submitted to a $2 \times 2 \times 2 \times 2$ repeated measures ANOVA with the factors of craving (craving, non-craving), task switching (switch, repeat), congruency (incongruent, congruent) and feedback (previous-high vs. previous-low). Because incorrect feedback occurred on a very small number of trials, those trials following an incorrect response and corresponding feedback were excluded from this analysis in lieu of its inclusion as a 3rd level of the feedback factor. All values reported are Greenhouse–Geisser corrected.

2.9 | Supplementary analysis with gender as a factor

Previous work has found that males and females exhibit differential effects of smoking-related attentional capture (Perlato, Santandrea, Libera, & Chelazzi, 2014). To determine whether gender also influenced any of the effects in our study, we conducted an additional analysis on the behavioural and ERP data using gender as an additional factor in our ANOVAs (with all other factors kept the same). The results of these analyses are reported in the Supporting Information.

3 | RESULTS

3.1 | Questionnaires

Participants smoked on average 15 cigarettes per day (range: 10.0–22.5) and had an average FTND score of 4.1 (range = 1.0–6.0). To determine whether the craving

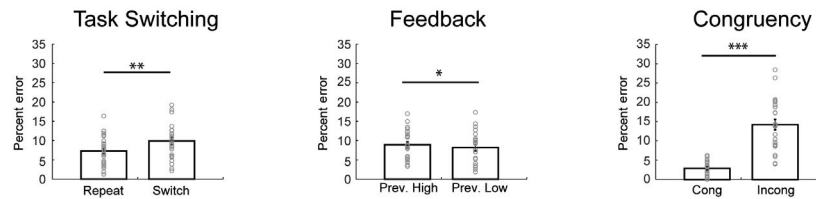
manipulation was effective, we compared the two factors of the QSU (the anticipation of pleasure from smoking, and the anticipation of relief from negative symptoms by smoking; see Methods) for when subjects were craving and when they were non-craving. For both QSU factors, there was a highly significant effect of craving (Factor 1: mean non-craving = 4.18, mean craving = 5.65, $t(23) = 9.03$, $p < .001$; Factor 2: mean non-craving = 1.98, mean craving = 3.08, $t(23) = 7.54$, $p < .001$).

3.2 | Behaviour

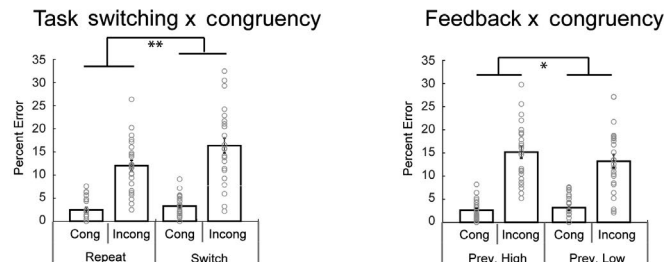
The error rate data were submitted to a $2 \times 2 \times 2 \times 2$ repeated measures ANOVA with the factors of craving (craving, non-craving), task switching (switch, repeat), congruency (incongruent, congruent) and feedback (previous-high, previous-low). This analysis revealed a main effect of task switching ($F(1,23) = 15.01$, $p = .001$, $\eta_p^2 = 0.40$), with switch trials showing higher error rates than repeat trials, a main effect of feedback ($F(1,23) = 4.31$, $p = .049$, $\eta_p^2 = 0.16$), with participants being more accurate following a trial with low feedback, and a main effect of congruency ($F(1,23) = 86.89$, $p < .001$, $\eta_p^2 = 0.79$), with participants committing fewer errors on congruent trials than incongruent ones. Additionally, there was a trending interaction of craving by congruency ($F(1,23) = 3.00$, $p = .097$, $\eta_p^2 = 0.12$), a significant interaction of switching by congruency ($F(1,23) = 15.45$, $p = .001$, $\eta_p^2 = 0.40$) and a significant interaction of feedback by congruency ($F(1,23) = 7.91$, $p = .01$, $\eta_p^2 = 0.26$). No other main effects or interactions were significant (all p 's > 0.1). The interaction of switching by congruency was driven by a difference between the incongruent conditions as a function of switching (repeat incongruent vs. switch incongruent, $t(23) = 4.45$, $p < .001$), with the switch incongruent condition inducing poorer performance, and no significant difference between the congruent conditions as a function of switching (repeat congruent vs. switch congruent, $p > .1$). The feedback by congruency interaction was driven by a significant difference between the incongruent conditions as a function of feedback (incongruent when previous-high feedback vs. incongruent when previous-low feedback, $t(23) = 3.88$, $p = .001$), with the performance on incongruent trials following low feedback being better than those following high feedback. There was no significant difference between the congruent trials as a function of feedback ($p > .1$). Figure 2a-b depicts the main effects and significant interactions for the error rate data. Of note, it is also the case that the number of false alarms to the NoGo stimuli did not differ as a function of craving (mean craving = 5.58, mean non-craving = 5.13, $p > .1$).

The RT data were also submitted to a $2 \times 2 \times 2 \times 2$ repeated measures ANOVA with the factors of craving

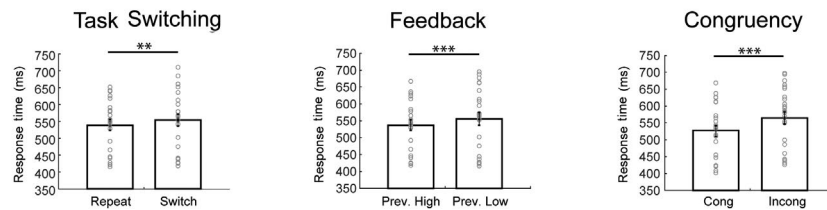
(a) Significant main effects of condition on error rates



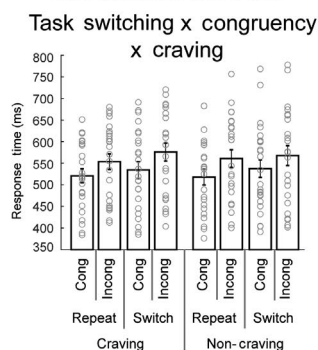
(b) Significant interactions between condition on error rates



(c) Significant main effects of condition on response times



(d) Significant interaction of conditions on RTs



(e) Significant correlations between addiction level and response times

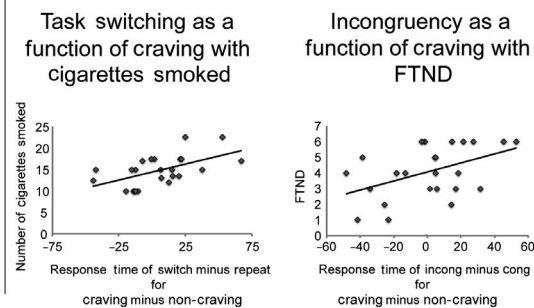


FIGURE 2 (a) Main Effects of Condition on Error Rates. All significant main effects are shown, with the expected modulations of switch costs and incongruency-related decreases in accuracy. (b) Significant interactions of condition on Error Rates. The effects of incongruency were larger under conditions of task switching, and the incongruency effect was larger for high previous feedback than for low previous feedback. (c) Main Effects of Condition on Response Times (RTs). Task switching and incongruency gave rise to slower RTs, as did trials following low feedback. (d) Significant 3-way interaction between Conditions on RTs. Here, craving interacted with switching and incongruency, indicating that craving modulated these control processes. Although significant differences are denoted in the above graphs with asterisks, as these interactions are more complicated, significant effects are not marked here in the figure (see text for details of significant differences). (e) Correlations Across Subjects between Addiction Level and RTs. The left graph shows the significant correlation between the average amount of cigarettes smoked per day and the switch costs as a function of craving. The right graph shows the significant correlation between the FTND (Fagerström Test for Nicotine Dependence) and the incongruency-related effects as a function of craving. In both cases, the more an individual smoked and was addicted to nicotine, the more these switch costs and incongruency-related costs were present when craving relative to when non-craving. * $p < .05$ to $.01$, ** $p < .01$ to $.001$, *** $p < .001$. All error bars represent the standard error of the mean (SEM)

(craving, non-craving), task switching (switch, repeat), congruency (incongruent, congruent) and previous-trial feedback (high vs. low). This revealed a main effect of task switching ($F(1,23) = 13.17$, $p = .001$, $\eta_p^2 = 0.36$), a main effect

of feedback ($F(1,23) = 21.97$, $p < .001$, $\eta_p^2 = 0.49$), a main effect of congruency ($F(1,23) = 54.35$, $p < .001$, $\eta_p^2 = 0.70$), a three-way interaction of craving by switching by congruency ($F(1,23) = 4.46$, $p = .046$, $\eta_p^2 = 0.16$) and a significant

four-way interaction of craving by feedback by switching by congruency ($F(1,23) = 11.52, p = .002, \eta_p^2 = 0.33$). The three-way interaction primarily appeared to be driven by a significant difference between the repeat incongruent and switch incongruent RTs in the craving condition ($t(23) = 2.99, p = .007$), with no such difference present in the non-craving condition between the repeat incongruent and switch incongruent RTs ($p < .1$). Figure 2c and d show the plots for the various significant effects for the response time data, and Table S1 shows the mean RT values for all conditions. Table S2 shows the results of the post hoc comparisons for the four-way interaction on the RT data.

In order to determine whether the level of nicotine dependence influenced the RTs as a function of condition and craving, we performed an exploratory correlational analysis on the RTs with two measures we obtained from the questionnaires (total FTND, total cigarettes smoked) as individual differences in task performance have previously been observed to be related to smoking behaviour (Libera, Zandonai, Zamboni, Santandrea, Sandri, Lugoboni, Chiamulera, & Chelazzi, 2019). Of note, although these two measures are closely related and tend to show a relationship with each other, they were not significantly correlated with each other ($r = .376, p = .07$) and were therefore examined independently. We hypothesized that the stronger the level of addiction, the more influence craving would have on task performance. To this end, we obtained the switch costs separately for craving and non-craving conditions (switch minus repeat); in this case, a greater number would suggest participants had more difficulty switching tasks. We then took the difference of these differences (i.e. craving minus non-craving). A positive number would suggest that the switch cost was greater under craving, and a negative number would suggest the switch cost was greater under non-craving conditions, with a difference of zero indicating these costs did not differ as a function of craving. Although there was no correlation between the FTND and the switch costs as a function of craving ($p = .5$), there was a significant correlation between the number of cigarettes smoked and the switch costs ($r = 0.562, p = .004$). Further, to determine how the incongruency effects that emerged as a function of craving were influenced by the level of nicotine dependence, we conducted an analogous analysis for the dependence measure. Specifically, we took the incongruent minus congruent RTs separately for craving and non-craving, and then subtracted the differences for non-craving from the differences for craving. As above, a positive number would suggest that greater incongruency effects were present when subjects were craving, and a more negative number would suggest that greater incongruency effects were present when subjects were non-craving. Although no significant correlation with the number of cigarettes per day was observed ($p = .28$), there was a

significant relationship between the FTND and congruency effects ($r = .471, p = .02$). In both of these cases (as seen in Figure 2e), as the level of addiction to nicotine increased, so did the costs of switching and incongruency when subjects were craving.

3.3 | ERP results

3.3.1 | Cue processing

P1 effects

The sensory-evoked P1 component in response to the cue was examined as a function of craving, task switching and previous-trial feedback. Specifically, a $2 \times 2 \times 2$ repeated measures ANOVA with the factors of craving (craving, non-craving), task switching (switch, repeat) and feedback (prev-high, prev-low) was conducted on the mean amplitude of the P1 (110–150 ms). For the P1, a marginal effect of craving emerged ($F(1,23) = 4.12, p = .054, \eta_p^2 = 0.15$), with the P1 tending to be larger when subjects were in a state of craving. Additionally, there was a significant interaction between craving and feedback ($F(1,23) = 5.84, p = .024, \eta_p^2 = 0.20$), and a significant 3-way interaction for craving \times switching \times feedback ($F(1,23) = 6.85, p = .02, \eta_p^2 = 0.23$). The two-way interaction was driven by a significant difference between craving and non-craving status in response to cues following high feedback ($t(23) = 2.65, p = .009$), with subjects showing a larger P1 following high feedback when craving. The 3-way interaction was mainly driven by the P1 amplitude for the craving repeat following high feedback being larger than that for several other conditions (versus craving repeat after low ($t(23) = 3.08, p = .005$); versus non-craving repeat after high ($t(23) = 3.48, p = .002$), versus craving switch after high ($t(23) = 3.08, p = .005$)).

CNV

A $2 \times 2 \times 2$ repeated measures ANOVA was conducted on the mean amplitudes of the early (700–1,000 ms) and late (1,000–1,300 ms) phases of the CNV component, with the factors of craving (craving, non-craving), task switching (switch, repeat) and previous-trial feedback (prev-high, prev-low). In the early phase, there was a main effect of craving ($F(1,23) = 4.36, p = .048, \eta_p^2 = 0.16$), with subjects having a larger CNV when they were *not* craving. There was also a significant switching by feedback interaction ($F(1,23) = 6.92, p = .015, \eta_p^2 = 0.23$). In the later phase of the CNV, the main effect of craving was no longer present ($p = .8$), but a main effect of switching emerged ($F(1,23) = 5.78, p = .025, \eta_p^2 = 0.20$), with the CNV being larger for switch trials than repeat, and the switching by feedback interaction remained significant ($F(1,23) = 7.85, p = .01, \eta_p^2 = 0.25$). For both the early and the late phases of the CNV, the switching by feedback interaction was driven by a significant difference between repeat versus switch after

high feedback (early: $t(23) = 4.02, p = .001$; late: $t(23) = 4.58, p > .001$). Figure 3 shows the waveforms and mean amplitude graphs for the processing of the cue.

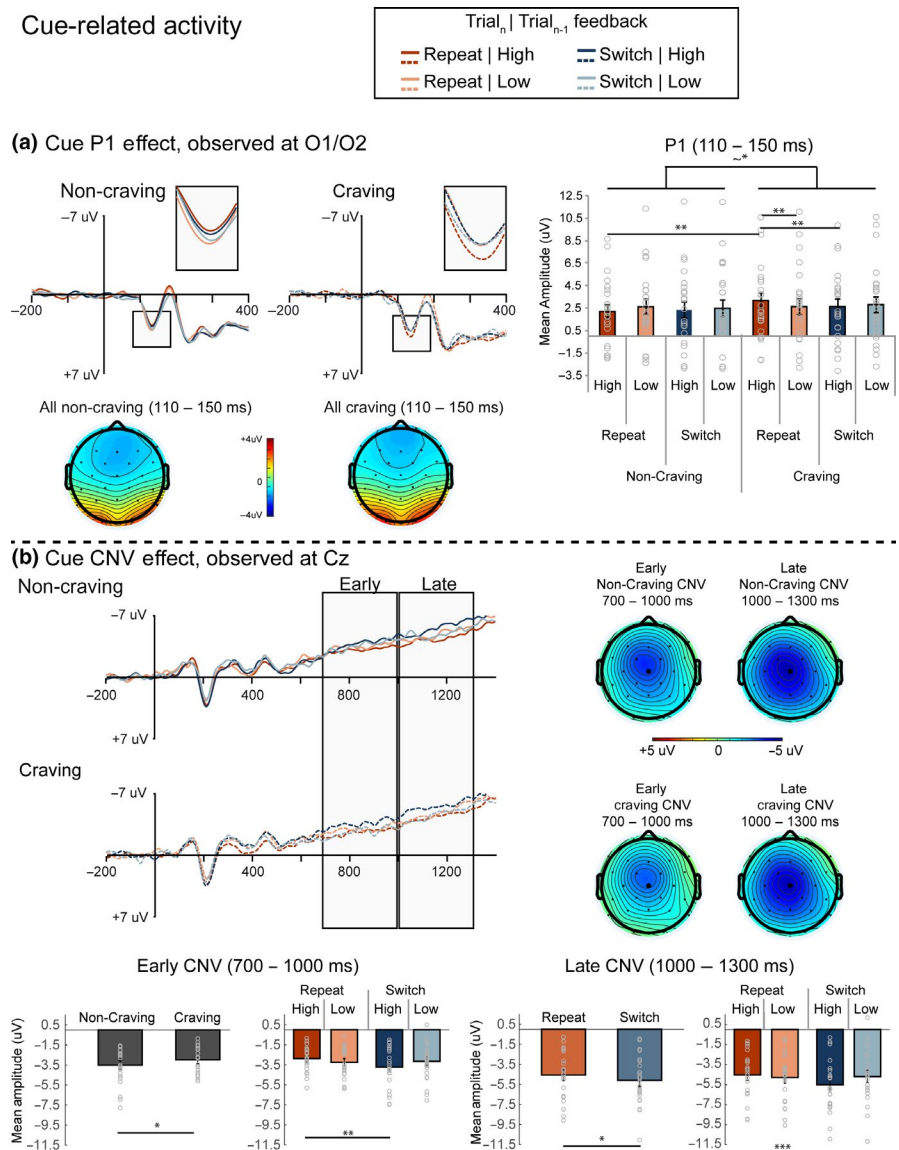
3.3.2 | Target processing

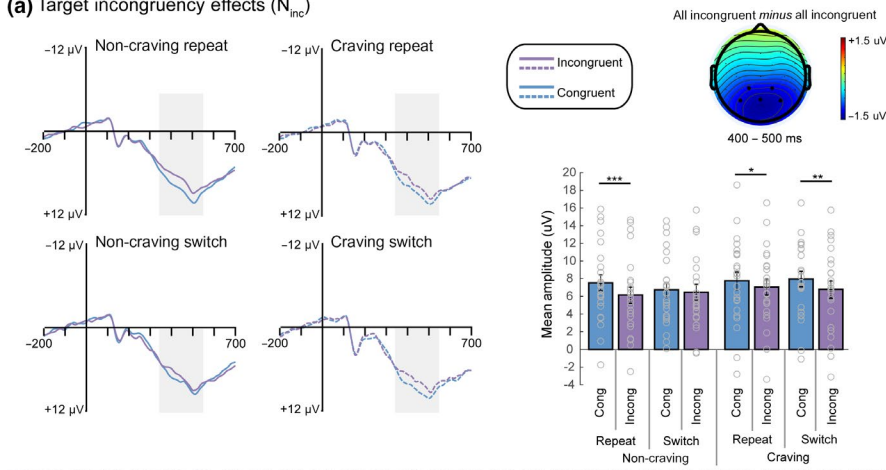
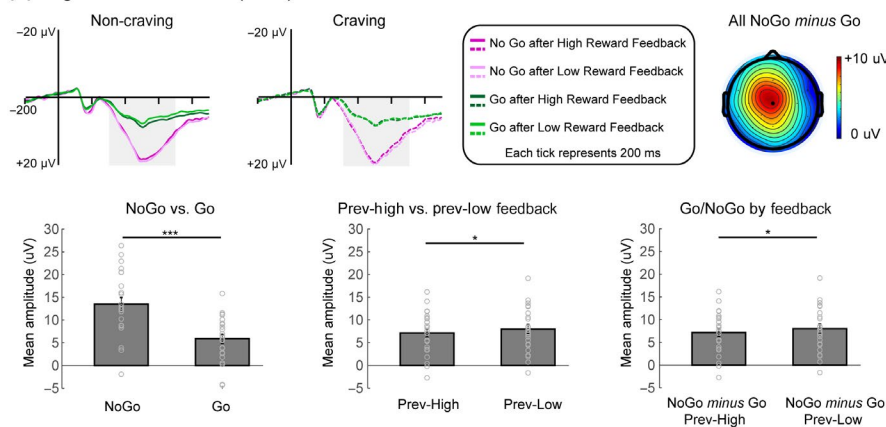
Based on previous work, we expected to see an incongruency-related effect occurring roughly 350–550 ms following target onset over posterior central scalp sites (see Methods for details). The mean response amplitude during this period was submitted to a $2 \times 2 \times 2 \times 2$ repeated measures ANOVA with the factors of craving (craving, non-craving), task switching (switch, repeat), feedback on the previous trial (prev-high, prev-low), and congruency (congruent, incongruent). This revealed a main effect of congruency ($F(1,23) = 16.99, p < .001, \eta_p^2 = 0.43$), with incongruent trials showing the expected enhanced relative negativity compared

to congruent trials. There was also a trending main effect of feedback ($F(1,23) = 4.04, p = .06, \eta_p^2 = 0.15$), with trials following low feedback tending to show more of a negativity relative to trials following high feedback. Additionally, a significant 3-way interaction between craving, task switching and congruency ($F(1,23) = 6.76, p = .02, \eta_p^2 = 0.23$) was present. Figure 4 depicts the significant effects. Post hoc t tests revealed that this interaction was primarily driven by the presence of significant differences between incongruent and congruent trial types for non-craving repeat congruent versus non-craving repeat incongruent ($t(23) = 4.76, p < .001$), craving repeat congruent versus craving repeat incongruent ($t(23) = 2.12, p = .04$), and craving switch congruent versus craving switch incongruent ($t(23) = 3.90, p = .001$), but not between non-craving switch congruent versus non-craving switch incongruent ($p = .54$). Further, a significant difference between non-craving repeat congruent and non-craving switch congruent was the final significant

FIGURE 3 Cue-Related Activity.

Note, legend presented above A applies to both parts of the figure, and all instances where “high” or “low” are written refers to the feedback received on the previous trial. (a) P1. The sensory-evoked P1 (110–150 ms) is depicted here, zoomed in for the traces and with mean amplitudes for each condition shown in the bar graphs. The topographic maps (lower right) show the distribution of this effect (posterior positivity) collapsed across all conditions for craving and for non-craving. (b) CNV. The CNV was analysed in an early (700–1,000 ms) and a late (1,000–1,300 ms) time period, depicted separately for non-craving and craving for task switching and previous-trial feedback conditions. Topographic distributions (right) show the CNV for these respective time periods, collapsed across all conditions for craving and all conditions when non-craving. The thick black dot in the centre of the topomaps highlights site Cz where the statistical analyses were conducted. $*p < .05$ to $.01$, $**p < .01$ to $.001$, $***p < .001$. All error bars represent the standard error of the mean (SEM). [Colour figure can be viewed at wileyonlinelibrary.com]



(a) Target incongruity effects (N_{inc})**(b) Target Go-NoGo effects (P300)****FIGURE 4** Target-Related Effects.

(a) The incongruity-related effect, the N_{inc} (measured from 350–550 ms, grey shaded area), is depicted here for each craving condition and task type separately. As can be seen, the N_{inc} was significantly present as the difference of incongruent versus congruent trial types, except in the instance of the non-craving switch condition.

Topographic distribution is shown for all conditions of incongruent minus congruent, with black dots representing the ROI used for statistical analyses. (b) The Go/NoGo-related effects were observed as a larger P300 (measured from 300 to 700 ms depicted in the grey shaded area) for NoGo trials relative to Go. The black dot on the topographic distribution indicates the site, Cz, where the P300 was measured, and the topographic distribution is averaged from 400 to 600 ms. * $p < .05$, ** $p < .01$ to .001, *** $p < .001$. All error bars represent the standard error of the mean (SEM). [Colour figure can be viewed at wileyonlinelibrary.com]

difference observed, which appeared to be driving the interaction ($t(23) = 3.25$, $p = .004$).

The Go/NoGo component of the task was also examined to determine whether there were any effects or interactions with craving. Specifically, for the P300 (300–700 ms) at site Cz, a $2 \times 2 \times 2 \times 2$ repeated measures ANOVA was conducted with the factors of craving (craving, non-craving), task switching (switch, repeat), feedback on the previous trial (prev-high, prev-low) and trial type (Go, NoGo). As expected, there was a highly significant effect of trial type, with NoGo trials having a larger P300 response than Go trials ($F(1,23) = 60.91$, $p < .001$, $\eta_p^2 = 0.73$). There was a significant interaction between trial type and feedback ($F(1,23) = 6.89$, $p = .015$, $\eta_p^2 = 0.23$), and a trending interaction between task switching and previous feedback ($F(1,23) = 3.64$, $p = .07$, $\eta_p^2 = 0.14$). The trial type by feedback interaction was the result of there being a larger difference between go and NoGo trials following low compared with high previous feedback.

3.3.3 | Feedback processing

The neural response to the feedback was reflected in the presence of a P300 component, and the mean amplitudes of

which were submitted to a 2×2 repeated measures ANOVA with the factors of craving (craving, non-craving) and current feedback level (high reward, low reward). This revealed only a significant effect of feedback level, with the P300 being larger when participants received a low reward ($F(1,23) = 27.12$, $p < .001$, $\eta_p^2 = 0.54$). Craving did not impact the processing of the feedback stimulus, either through a main effect or by interacting with the type of feedback the participants received. Figure 5 depicts the feedback-related waveforms.

4 | DISCUSSION

In the present study, we investigated the impact of craving on several cognitive control processes (task switching, response inhibition, incongruity) in smokers when they had recently smoked (non-craving) and when they had been deprived of smoking for three hours (craving). The interactions of craving with these control processes were complex, as discussed in detail below, but its influence seemed to primarily manifest as decreased preparation for an upcoming target and subsequent increased incongruity effects. Importantly, craving did not interact with inhibitory

Feedback-related P300

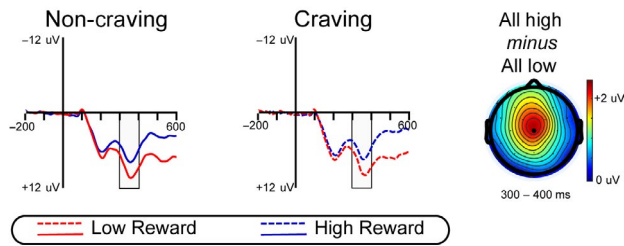


FIGURE 5 Feedback-Related Effects. The waveforms shown are for the P300 (300 to 400, indicated in the box) at site Cz. The P300 was larger in response to low reward than high reward, but this did not interact with craving. The topographic distribution shows the P300 for high reward minus low reward, collapsed across craving. [Colour figure can be viewed at wileyonlinelibrary.com]

control processes or current trial feedback-related processes, suggesting that a brief period of nicotine deprivation selectively influences cognitive control processes.

4.1 | Behavioural effects of switching and incongruency regardless of craving state

Regardless of whether or not smokers were craving, the task used here elicited the expected behavioural effects. Specifically, for both the accuracy and RT data, participants showed the expected performance decrements when they had to switch tasks and when the target stimulus was incongruent. Such behavioural effects are in line with what has previously been reported for tasks involving task switching (e.g. Braver, Reynolds, & Donaldson, 2003) and incongruency (MacLeod, 1991), and confirm that our paradigm was able to elicit similar robust effects. Together, these main effects on behaviour indicate under conditions of craving and non-craving this task was tapping into the intended cognitive control processes.

4.2 | Main effects of craving

As we found previously (Donohue, Woldorff, et al., 2016), craving has a general effect on overall arousal. Here, when we examined the effects of craving on the P1 component in response to the cue stimuli, we found that this component was (marginally) enhanced when participants were in a state of craving. This effect thus replicates our prior results and suggests that across multiple tasks smokers are in a heightened attentional state when they have not smoked for several hours. Interestingly, in the current study, this did not ramify as overall faster RTs or enhancement across all later components, suggesting that this heightened arousal does not necessarily provide any general cognitive benefit,

particularly in tasks where control-related processes must be recruited.

The other main effect of craving observed was a decreased amplitude in the CNV under conditions of craving. This difference was short-lived and was observed only in the early time window of the CNV. Nevertheless, it is likely that this lack of initial preparation had consequences for subsequent target processing. As the CNV appears to increase with more cognitive effort (Falkenstein, Hoormann, Hohnsbein, & Kleinsorge, 2003), it could be the case that early on, smokers were not able to mobilize as much preparatory effort (e.g. keeping in working memory what the upcoming task would be, what the corresponding response buttons were, attentional-focus preparation), and this happened regardless of whether or not the task was repeated or switched. It is conceivable that craving puts smokers in an attentional state that is less proactive (Braver, 2012), which, in this case, manifested as reduced preparatory activity reflected in the CNV.

4.3 | Interactions between craving, task switching and incongruency

Although smokers were able to essentially “catch up” on the amount of preparatory activity they invoked when craving, as indicated by a lack of a difference in the late CNV amplitude, it would appear that the lack of early preparation did indeed have consequences when the target appeared, particularly in the case when participants had to switch tasks and/or an incongruent stimulus were present. In both the behavioural data and the target-related N_{inc} response, there was a three-way interaction present between craving, task switching and incongruency, which, albeit through different effects, manifested as a lack of preparatory up-regulation when smokers were craving and needed to switch tasks, resulting in larger incongruency-related effects for the target stimulus.

When smokers were in a non-craving state, the incongruency-related differences (in the N_{inc} component) were not present under conditions of switching, whereas they were under conditions of repeating. What this suggests is that when subjects were switching tasks and not craving, they had sufficient cognitive control resources and were so focused on the task that, once the target appeared, regardless of whether or not it was congruent or incongruent, they were able to attend to the relevant feature and block out the irrelevant information. When the task repeated, and they were not in a state of craving, the smokers appeared to have invoked less control and therefore had a greater reaction to the incongruent stimuli. This general pattern of activity has been seen in other domains of conflict-related cognitive control. For example, tasks looking at conflict-adaptation effects have

demonstrated that when incongruent trials follow an incongruent trial, responses are faster, ACC conflict-related activity is decreased, and DLPFC activity is increased, relative to when incongruent trials follow a congruent trial (Egner & Hirsch, 2005; Gratton, Coles, & Donchin, 1992; Kerns, Cohen, & MacDonald, Cho, Stenger, & Carter, 2004). In those cases, the increased preparation comes from a previous-incongruent trial, which is a challenging event and subjects often respond with increased preparation for the next trial. Here, the switch itself is what is more challenging and necessitates the increased preparation. This would suggest that when smokers were not craving, they were more flexibly using cognitive control and thus modulating the amount of activity necessary as a function of the task.

In contrast, when the smokers were craving, the incongruity effects for switches and repeats were similar. That is, these conflict-adaptation-like effects were not present, suggesting that they were not modulating the level of control as a function of switching/repeating tasks. This ramified behaviourally in that when participants were craving, the incongruent trials on the switch condition elicited longer RTs than incongruent trials on the repeat condition. This suggests that participants were experiencing more conflict under conditions of craving and switching. This was confirmed in the neural data, where, for both switching and repeating, a significant incongruity-related N_{inc} effect was present, whereas when smokers were not in a state of craving, this incongruity-related effect was only present for repeat trials. Increased incongruity effects in a Stroop conflict task have also been observed in a study looking at smokers who failed to quit (i.e. those who could not overcome their level of craving) relative to those who successfully quit (Krönke, Wolff, Benz, & Goschke, 2015). Although that study did not measure the levels of craving in its participants, their data do suggest that smokers who are able to successfully quit (and inhibit/overcome craving overall) have enhanced levels of cognitive control, which our participants were not showing in their state of craving.

Together, the current results suggest that craving shifts participants to a more reactive state, wherein cognitive control processes are not as strongly (or reliably) invoked. A behavioural study specifically looking at task switching found that deprived smokers showed less flexibility compared with non-deprived smokers and controls (Lyvers, Maltzman, & Clinical, 1994), which would generally support our neural findings here. Although we had hypothesized, based on previous literature, that task switching would be more strongly impacted by craving than incongruity, the current data suggest that they are both impacted. Importantly, however, there was no significant craving by incongruity interaction either behaviourally or neurally (with the N_{inc} component), and there was also no craving by switching interaction (behaviourally or

neurally) present. Only when both incongruity and task switching were considered, did we observe a significant (3-way) interaction between these factors and craving. That is, in the early stages of nicotine deprivation, smokers exhibit impairment in cognitive control when these processes are pitted together. Moreover, the level of smoking here, as measured in cigarettes per day, strongly correlated with the behavioural task switching costs as a function of craving, with heavier smokers showing increased performance decrements when craving, while the incongruity-related costs correlated with the level of nicotine dependence in the FTND. It may, therefore, be the case that had we measured smokers with a larger consumption (> a pack/day) and/or given our smokers a longer period of abstinence, individual craving by switching and craving by incongruity interactions would have emerged. Regardless, speaks to the disruptiveness that craving can cause when it comes to these control processes, and underscores the necessity to curb its influence for successful smoking cessation.

4.4 | Craving and response inhibition

Importantly, craving did not modulate all control processes. It did not have an influence on the inhibitory processes, with the caveat that only the P300 and behaviour were examined here, as there was no clear difference in the N2 between the Go and NoGo conditions. At first glance, this lack of a difference on the P300 is a bit surprising, both given that craving did influence the other control-related tasks and that smokers appear to have deficits in inhibitory control, as has been observed in other Go/NoGo tasks (e.g. Evans et al., 2009). In fact, given the interactions between craving and task switching and incongruity, one would predict that, at the very least, craving would have also interacted with inhibition as a function of task switching. Nevertheless, no influence of craving on inhibitory control in and of itself or in combination with other factors was observed. The task used here, however, was a hybrid of many control-related tasks, and not just a Go/NoGo task, and it is possible that because an increased level of control may have been required (Chmielewski & Beste, 2017), the smokers were already at ceiling for their inhibition, even when craving. Moreover, given that subjects knew that they needed to remain in the room and not go out and smoke, they may have already activated a certain level of longer-lasting inhibitory control to accomplish this, which could have transferred over to the task in the craving session, during which remaining in the experiment and not smoking would have been particularly challenging. It could also be the case that after only a short deprivation, inhibitory deficits are not found, as they have been shown to emerge only after longer (e.g. 10–72 hr) periods of abstinence in other studies examining inhibition in

smokers (Charles-Walsh, Furlong, Munro, & Hester, 2014; Harrison, Coppola, & McKee, 2009; Lydon et al., 2015; Tsaur, Strasser, Souprountchouk, Evans, & Ashare, 2015).

4.5 | The influence of feedback

Feedback, like inhibitory control, did not modulate the P300 response as a function of craving. Because this rami-fied into how soon the smokers could leave the experiment (the more high feedback they got, the sooner the experi-ment ended), we had expected to see a stronger differen-tiation between high and low feedback under conditions of craving versus non-craving, but this was not the case. Regardless of the session, participants displayed a stronger neural response to the low feedback versus the high, sug-gesting that they were either surprised by it (Squires, Wickens, Squires, & Donchin, 1976) or that their atten-tion was more captured by it, perhaps because of the moti-vational salience (Nieuwenhuis, Aston-Jones, & Cohen, 2005) of this feedback (i.e. low feedback requires more time spent in the experiment).

Although the feedback-related response was not modu-lated by craving on the current trial, the feedback partici-pants received on the previous trial did influence both the cue and target processing on the current one, where it did interact with craving. The first manifestation of this oc-curred at the level of the P1, where the amplitude of this component was highest when subjects were in a craving state and when they received a cue for a repeat trial after receiving high feedback. In addition to overall arousal, the amplitude of the P1 is known to vary with the amount of attention allocated to a stimulus, with higher amplitudes indicating greater attention (e.g. Heinze et al., 1994). Here, it is likely that the smokers were in fact motivated by the high feedback (both with the inherent value of more points, and what that meant for them leaving earlier and getting to smoke sooner), and by the fact that a repeat trial indicated that they needed less cognitive control to solve the task, making this trial a “cognitive win” of sorts. The high feed-back also yielded a stronger difference in the CNV activity on the switch versus repeat trials, with smokers showing an increased CNV following high feedback on a switch trial compared with a repeat trial. Perhaps the reward from hav-ing just received a high feedback was motivation enough for the smokers to engage these preparatory regions a bit more when the upcoming task was a switch and therefore involved more control.

Behaviourally, feedback on the previous trial modulated the response on the current trial, with participants being more accu-rate but slower after low versus high feedback. The modulation of behaviour by feedback from the previous trial suggests that the smokers were sensitive to this form of reward manipulation.

Although they were not receiving money based on their per-formance, the positive but low feedback and the notion that wining enough points would let them go early was enough to consistently slow their performance after a lower amount of points to ensure accuracy. To date, there is some evidence to suggest that feedback processing (on correct vs. incorrect trials) may be altered in smokers compared with controls, particularly for the later-stage components of feedback processing (the Pe; Franken, Strien, & Kuijpers, 2010). Our data suggest that crav-ing may also influence this processing, not on the present trial, but moving forward to the next trial, indicating the complex interaction craving may have with motivation.

4.6 | Nicotine enhancement versus deprivation

The results presented here have been considered in terms of the influence of craving on these control processes. It is also im-portant, however, to consider that nicotine itself has a modulat-ing effect on cognition (see Newhouse, Potter, Dumas, & Thiel, 2011 for review). When non-smokers have been administered nicotine, nicotine has been found to increase response speed in attention-related tasks (Foulds et al., 1996; Griesar, Zajdel, & Oken, 2002; Meinke, Thiel, & Fink, 2006), despite not show-ing any effects on early EEG attentional components (P1 and N1; Meinke et al., 2006). Interestingly, the P3a and P3b com-ponents (indices of cognitive control processes) were not al-tered in an oddball task when non-smokers were administered nicotine versus placebo (Evans, Jentink, Sutton, Rensburg, & Drobos, 2014). Taken together, these results suggest that while nicotine does improve response speed, its direct effect on at-tention and control-related processes may be limited, too small to be characterized with EEG, or only present in a subgroup of subjects (see Logemann, Böcker, Deschamps, Kemner, & Kenemans, 2014 who observed effects on the N1 in subjects who also showed large changes in blood pressure in response to nicotine administration).

In smokers, of course, it cannot be fully determined if the effects observed as a function of smoking/nicotine depri-vation versus administration are due to the enhancement of performance from nicotine, or the relative decrement of per-formance from craving. Given that the neural effects of nicot-ine administration in non-smokers are rather limited, it is likely that the results we see here are the result of craving altering these measures, rather than nicotine altering them. Moreover, we did not see overall changes in RTs as a func-tion of smoking, suggesting that our pattern of results is more complex than just the nicotine-enhanced response speed. Nevertheless, more research needs to be conducted to fully assess the influence of nicotine on a wider variety of cog-nitive tasks and circumstances. Of note, there is some evidence (albeit in a small sample), that in a smoker three hours after

smoking a cigarette, the majority of receptors in the brain are still occupied by nicotine (Brody et al., 2006), suggesting that our deprivation period may not have been long enough to see strong nicotine-related effects, while still long enough to induce the feeling of craving, as assessed by the questionnaire (QSU) data.

5 | CONCLUSION

The present study demonstrated that cognitive control processes are impaired in smokers when they are craving as compared to when they are sated. Specifically, craving smokers were less able to flexibly implement cognitive control, which manifested as increased deficits on task switching and incongruency processing. Importantly, the disruption in cognitive control by craving was not present for every form of control, indicating that craving is not general in its actions, but specific control-related processes are affected. Even in the absence of nicotine-related cues, and after only three hours of deprivation, craving had an impact on both behavioural and neural measures of cognitive control. This further bolsters the notion that the difficulty many people have in quitting an addiction is not just due to a general impairment in control systems, but that they may be further impaired when trying to abstain (and thus are craving), increasing the likelihood of a relapse.

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CONFLICT OF INTEREST

The authors have no conflicts of interest to declare.

AUTHOR CONTRIBUTIONS

S.E.D., J.A.H. and M.A.S. designed the experiment. S.E.D. collected the data and S.E.D. analysed the data. K.L. provided analytical tools. J.M.H. and H.J.H. provided conceptual input throughout the project. S.E.D. wrote the manuscript. M.G.W., J.A.H. and M.A.S. edited the manuscript.

DATA AVAILABILITY STATEMENT

The data from this study are available from the corresponding author upon reasonable request.

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REFERENCES

- Albert, J., López-Martín, S., Hinojosa, J. A., & Carretié, L. (2013). Spatiotemporal characterization of response inhibition. *NeuroImage*, *76*, 272–281. <https://doi.org/10.1016/j.neuroimage.2013.03.011>
- Astle, D. E., Jackson, G. M., & Swainson, R. (2006). Dissociating neural indices of dynamic cognitive control in advance task-set preparation: An ERP study of task switching. *Brain Research*, *1125*, 94–103. <https://doi.org/10.1016/j.brainres.2006.09.092>
- Astle, D. E., Jackson, G. M., & Swainson, R. (2008). Fractionating the cognitive control required to bring about a change in task: A dense-sensor event-related potential study. *Journal of Cognitive Neuroscience*, *20*, 255–267. <https://doi.org/10.1162/jocn.2008.20015>
- Badre, D., & Nee, D. E. (2018). Frontal cortex and the hierarchical control of behavior. *Trends in Cognitive Sciences*, *22*, 170–188. <https://doi.org/10.1016/j.tics.2017.11.005>
- Banich, M. T. (2009). Executive function: The search for an integrated account. *Current Directions in Psychological Science*, *18*, 89–94. <https://doi.org/10.1111/j.1467-8721.2009.01615.x>
- Belin, D., Belin-Rauscent, A., Murray, J. E., & Everitt, B. J. (2013). Addiction: Failure of control over maladaptive incentive habits. *Current Opinion in Neurobiology*, *23*(4), 564–572. <https://doi.org/10.1016/j.conb.2013.01.025>
- Bilano, V., Gilmour, S., Moffiet, T., Turasan d'Espaignet, E., Stevens, G. A., Commar, A., ... Shibuya, K. (2015). Global trends and projections for tobacco use, 1990–2025: An analysis of smoking indicators from the WHO Comprehensive Information Systems for Tobacco Control. *The Lancet*, *385*, 966–976. [https://doi.org/10.1016/S0140-6736\(15\)60264-1](https://doi.org/10.1016/S0140-6736(15)60264-1)
- Billieux, J. L., Gay, P., Rochat, L., Khazaal, Y., Zullino, D., & Van der Linden, M. (2010). Lack of inhibitory control predicts cigarette smoking dependence: Evidence from a non-deprived sample of light to moderate smokers. *Drug and Alcohol Dependence*, *112*, 164–167. <https://doi.org/10.1016/j.drugalcdep.2010.06.006>
- Braver, T. S. (2012). The variable nature of cognitive control: A dual mechanisms framework. *Trends in Cognitive Sciences*, *16*, 105–112. <https://doi.org/10.1016/j.tics.2011.12.010>
- Braver, T. S., Reynolds, J. R., & Donaldson, D. I. (2003). Neural mechanisms of transient and sustained cognitive control during task switching. *Neuron*, *39*, 713–726. [https://doi.org/10.1016/S0896-6273\(03\)00466-5](https://doi.org/10.1016/S0896-6273(03)00466-5)
- Brody, A. L., Mandelkern, M. A., London, E. D., Olmstead, R. E., Farahi, J., Scheibal, D., ... Mukhin, A. G. (2006). Cigarette smoking saturates brain alpha 4 beta 2 nicotinic acetylcholine receptors. *Archives of General Psychiatry*, *63*, 907–915.
- Buzzell, G. A., Fedota, J. R., Roberts, D. M., & McDonald, C. G. (2014). The N2 ERP component as an index of impaired cognitive control in smokers. *Neuroscience Letters*, *563*, 61–65. <https://doi.org/10.1016/j.neulet.2014.01.030>
- Carter, B. L., & Tiffany, S. T. (1999). Meta-analysis of cue-reactivity in addiction research. *Addiction*, *94*, 327–340. <https://doi.org/10.1046/j.1360-0443.1999.9433273.x>
- Carter, C. S. (1998). Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science*, *280*, 747–749. <https://doi.org/10.1126/science.280.5364.747>
- Carter, C. S., & van Veen, V. (2007). Anterior cingulate cortex and conflict detection: An update of theory and data. *Cognitive, Affective, & Behavioral Neuroscience*, *7*, 367–379. <https://doi.org/10.3758/CABN.7.4.367>

- Charles-Walsh, K., Furlong, L., Munro, D. G., & Hester, R. (2014). Inhibitory control dysfunction in nicotine dependence and the influence of short-term abstinence. *Drug and Alcohol Dependence, 143*, 81–86. <https://doi.org/10.1016/j.drugalcdep.2014.07.008>
- Chmielewski, W. X., & Beste, C. (2017). Testing interactive effects of automatic and conflict control processes during response inhibition – A system neurophysiological study. *NeuroImage, 146*, 1149–1156. <https://doi.org/10.1016/j.neuroimage.2016.10.015>
- Crone, E. A., Wendelken, C., Donohue, S. E., & Bunge, S. A. (2006). Neural evidence for dissociable components of task-switching. *Cerebral Cortex, 16*, 475–486. <https://doi.org/10.1093/cercor/bhi127>
- Dawkins, L., Powell, J. H., West, R., Powell, J., & Pickering, A. (2007). A double-blind placebo-controlled experimental study of nicotine: II—Effects on response inhibition and executive functioning. *Psychopharmacology (Berl), 190*, 457–467. <https://doi.org/10.1007/s00213-006-0634-6>
- Delorme, A., & Makeig, S. (2004). EEGLAB: An open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *Journal of Neuroscience Methods, 134*, 9–21. <https://doi.org/10.1016/j.jneumeth.2003.10.009>
- Dinn, W. M., Aycicegi, A., & Harris, C. L. (2004). Cigarette smoking in a student sample: Neurocognitive and clinical correlates. *Addictive Behaviors, 29*, 107–126. <https://doi.org/10.1016/j.addbeh.2003.07.001>
- Donohue, S. E., Appelbaum, L. G., McKay, C. C., & Woldorff, M. G. (2016). The neural dynamics of stimulus and response conflict processing as a function of response complexity and task demands. *Neuropsychologia, 84*, 1–15. <https://doi.org/10.1016/j.neuropsychologia.2016.01.035>
- Donohue, S. E., Woldorff, M. G., Hopf, J.-M., Harris, J. A., Heinze, H.-J., & Schoenfeld, M. A. (2016). An electrophysiological dissociation of craving and stimulus-dependent attentional capture in smokers. *Cognitive, Affective, & Behavioral Neuroscience, 16*, 1–13. <https://doi.org/10.3758/s13415-016-0457-9>
- Egner, T., & Hirsch, J. (2005). Cognitive control mechanisms resolve conflict through cortical amplification of task-relevant information. *Nature Neuroscience, 8*, 1784–1790. <https://doi.org/10.1038/nn1594>
- Evans, D. E., Jentink, K. G., Sutton, S. K., Van Rensburg, K. J., & Drobles, D. J. (2014). 7 mg nicotine patch fails to enhance P300 neural indices of cognitive control among nonsmokers. *Pharmacology, Biochemistry and Behavior, 126*, 77–82. <https://doi.org/10.1016/j.pbb.2014.09.001>
- Evans, D. E., Park, J. Y., Maxfield, N., & Drobles, D. J. (2009). Neurocognitive variation in smoking behavior and withdrawal: Genetic and affective moderators. *Genes, Brain and Behavior, 8*, 86–96. <https://doi.org/10.1111/j.1601-183X.2008.00445.x>
- Falkenstein, M., Hoormann, J., Hohnsbein, J., & Kleinsorge, T. (2003). Short-term mobilization of processing resources is revealed in the event-related potential. *Psychophysiology, 40*, 914–923. <https://doi.org/10.1111/1469-8986.00109>
- Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods, 39*, 175–191. <https://doi.org/10.3758/BF03193146>
- Fedota, J. R., Matous, A. L., Salmeron, B. J., Gu, H., Ross, T. J., & Stein, E. A. (2016). Insula demonstrates a non-linear response to varying demand for cognitive control and weaker resting connectivity with the executive control network in smokers. *Neuropsychopharmacology, 41*, 2557–2565. <https://doi.org/10.1038/npp.2016.62>
- Ferguson, S. G., & Shiffman, S. (2009). The relevance and treatment of cue-induced cravings in tobacco dependence. *Journal of Substance Abuse Treatment, 36*, 235–243. <https://doi.org/10.1016/j.jsat.2008.06.005>
- Flaudias, V., Picot, M. C., Lopez-Castroman, J., Llorca, P.-M., Schmitt, A., Perriot, J., ... Guillaume, S. (2016). Executive functions in tobacco dependence: Importance of inhibitory capacities. *PLoS ONE, 11*, e0150940–e151012. <https://doi.org/10.1371/journal.pone.0150940>
- Foulds, J., Stapleton, J., Swettenham, J., Bell, N., McSorley, K., & Russell, M. A. (1996). Cognitive performance effects of subcutaneous nicotine in smokers and never-smokers. *Psychopharmacology (Berl), 127*, 31–38. <https://doi.org/10.1007/BF02805972>
- Frankel, I. H. A., Van Strien, J. W., & Kuijpers, I. (2010). Evidence for a deficit in the salience attribution to errors in smokers. *Drug and Alcohol Dependence, 106*, 181–185. <https://doi.org/10.1016/j.drugalcdep.2009.08.014>
- Garavan, H., Ross, T. J., & Stein, E. A. (1999). Right hemispheric dominance of inhibitory control: An event-related functional MRI study. *Proceedings of the National Academy of Sciences of the United States of America, 96*, 8301–8306.
- Glass, J. M., Buu, A., Adams, K. M., Nigg, J. T., Puttler, L. I., Jester, J. M., & Zucker, R. A. (2009). Effects of alcoholism severity and smoking on executive neurocognitive function. *Addiction, 104*, 38–48. <https://doi.org/10.1111/j.1360-0443.2008.02415.x>
- Goldstein, R. Z., & Volkow, N. D. (2011). Dysfunction of the prefrontal cortex in addiction: Neuroimaging findings and clinical implications. *Nature Reviews Neuroscience, 12*, 652–669. <https://doi.org/10.1038/nrn3119>
- Gratton, G., Coles, M. G., & Donchin, E. (1992). Optimizing the use of information: Strategic control of activation of responses. *Journal of Experimental Psychology: General, 121*, 480–506. <https://doi.org/10.1037/0096-3445.121.4.480>
- Gratton, G., Cooper, P., Fabiani, M., Carter, C. S., & Karayanidis, F. (2017). Dynamics of cognitive control: Theoretical bases, paradigms, and a view for the future. *Psychophysiology, 310*, 1–29.
- Griesar, W. S., Zajdel, D. P., & Oken, B. S. (2002). Nicotine effects on alertness and spatial attention in non-smokers. *Nicotine & Tobacco Research, 4*, 185–194. <https://doi.org/10.1080/14622200210123617>
- Harper, J., Malone, S. M., & Bernat, E. M. (2014). Theta and delta band activity explain N2 and P3 ERP component activity in a go/no-go task. *Clinical Neurophysiology, 125*, 124–132. <https://doi.org/10.1016/j.clinph.2013.06.025>
- Harrison, E. L. R., Coppola, S., & McKee, S. A. (2009). Nicotine deprivation and trait impulsivity affect smokers' performance on cognitive tasks of inhibition and attention. *Experimental and Clinical Psychopharmacology, 17*, 91–98. <https://doi.org/10.1037/a0015657>
- Heatherington, T. F., Kozlowski, L. T., Frecker, R. C., & Fagerström, K. O. (1991). The Fagerström test for nicotine dependence: A revision of the Fagerström tolerance questionnaire. *British Journal of Addiction, 86*, 1119–1127.
- Heinze, H. J., Mangun, G. R., Burchert, W., Hinrichs, H., Scholz, M., Münte, T. F., ... Hillyard, S. A. (1994). Combined spatial and temporal imaging of brain activity during visual selective attention in humans. *Nature, 372*, 543–546. <https://doi.org/10.1038/372543a0>
- Helfrich, R. F., & Knight, R. T. (2016). Oscillatory dynamics of prefrontal cognitive control. *Trends in Cognitive Sciences, 20*, 916–930. <https://doi.org/10.1016/j.tics.2016.09.007>

- Hopf, J.-M., Luck, S. J., Girelli, M., Hagner, T., Mangun, G. R., Scheich, H., & Heinze, H. J. (2000). Neural sources of focused attention in visual search. *Cerebral Cortex*, *10*, 1233–1241. <https://doi.org/10.1093/cercor/10.12.1233>
- Huster, R. J., Enriquez-Geppert, S., Lavallee, C. F., Falkenstein, M., & Herrmann, C. S. (2013). Electroencephalography of response inhibition tasks: Functional networks and cognitive contributions. *International Journal of Psychophysiology*, *87*, 217–233. <https://doi.org/10.1016/j.ijpsycho.2012.08.001>
- Kerns, J. G., Cohen, J. D., MacDonald, A. W., Cho, R. Y., Stenger, A., & Carter, C. S. (2004). Anterior cingulate conflict monitoring and adjustments in control. *Science*, *303*, 1023–1026. <https://doi.org/10.1126/science.1089910>
- Killen, J. D., & Fortmann, S. P. (1997). Craving is associated with smoking relapse: Findings from three prospective studies. *Experimental and Clinical Psychopharmacology*, *5*, 137–142. <https://doi.org/10.1037/1064-1297.5.2.137>
- Krönke, K.-M., Wolff, M., Benz, A., & Goschke, T. (2015). Successful smoking cessation is associated with prefrontal cortical function during a Stroop task: A preliminary study. *Psychiatry Research: Neuroimaging*, *234*, 52–56.
- Libera Della, C., Zandonai, T., Zamboni, L., Santandrea, E., Sandri, M., Lugoboni, F., Chiamulera, C., & Chelazzi, L. (2019). Revealing dissociable attention biases in chronic smokers through an individual-differences approach. *Scientific Reports*, *9*, 1–12.
- Liotti, M., Woldorff, M. G., Perez, R., & Mayberg, H. S. (2000). An ERP study of the temporal course of the Stroop color-word interference effect. *Neuropsychologia*, *38*, 701–711. [https://doi.org/10.1016/S0028-3932\(99\)00106-2](https://doi.org/10.1016/S0028-3932(99)00106-2)
- Logemann, H. N. A., Böcker, K. B. E., Deschamps, P. K. H., Kemner, C., & Kenemans, J. L. (2014). The effect of the augmentation of cholinergic neurotransmission by nicotine on EEG indices of visuo-spatial attention. *Behavioural Brain Research*, *260*, 67–73. <https://doi.org/10.1016/j.bbr.2013.11.044>
- Lopez-Calderon, J., & Luck, S. J. (2014). ERPLAB: An open-source toolbox for the analysis of event-related potentials. *Frontiers in Human Neuroscience*, *8*, 213. <https://doi.org/10.3389/fnhum.2014.00213>
- Luijten, M., Littel, M., & Franken, I. H. A. (2011). Deficits in inhibitory control in smokers during a Go/NoGo task: An investigation using event-related brain potentials. *PLoS ONE*, *6*, e18898–e18907. <https://doi.org/10.1371/journal.pone.0018898>
- Luijten, M., Machielsen, M., Veltman, D., Hester, R., de Haan, L., & Franken, I. (2014). Systematic review of ERP and fMRI studies investigating inhibitory control and error processing in people. *Journal of Psychiatry and Neuroscience*, *39*, 149–169. <https://doi.org/10.1503/jpn.130052>
- Lydon, D. M., Roberts, N. J., & Geier, C. F. (2015). Reduced influence of monetary incentives on Go/NoGo performance during smoking abstinence. *Nicotine & Tobacco Research*, *17*, 1178–1181.
- Lyvers, M., Maltzman, I., & Clinical, Y. M. E. A. (1994). (n.d.) Effects of cigarette smoking and smoking deprivation on Wisconsin Card Sorting Test performance. *Journal of Experimental Psychology: General*, *2*, 283–289. <https://doi.org/10.1037/1064-1297.2.3.283>
- MacLeod, C. M. (1991). Half a century of research on the Stroop effect: An integrative review. *Psychological Bulletin*, *109*, 163–203. <https://doi.org/10.1037/0033-2909.109.2.163>
- Mayr, U., & Kliegl, R. (2003). Differential effects of cue changes and task changes on task-set selection costs. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *29*, 362–372.
- McKay, C. C., van den Berg, B., & Woldorff, M. G. (2017). Neural cascade of conflict processing: Not just time-on-task. *Neuropsychologia*, *96*, 184–191.
- Meinke, A., Thiel, C. M., & Fink, G. R. (2006). Effects of nicotine on visuo-spatial selective attention as indexed by event-related potentials. *Neuroscience*, *141*, 201–212. <https://doi.org/10.1016/j.neuroscience.2006.03.072>
- Morie, K. P., De Sanctis, P., & Foxe, J. J. (2014). Reward contingencies and the recalibration of task monitoring and reward systems: A high-density electrical mapping study. *Neuroscience*, *273*, 100–117. <https://doi.org/10.1016/j.neuroscience.2014.05.002>
- Nagai, Y., Critchley, H. D., Featherstone, E., Fenwick, P. B. C., Trimble, M. R., & Dolan, R. J. (2004). Brain activity relating to the contingent negative variation: An fMRI investigation. *NeuroImage*, *21*, 1232–1241. <https://doi.org/10.1016/j.neuroimage.2003.10.036>
- Nestor, L., McCabe, E., Jones, J., Clancy, L., & Garavan, H. (2011). Differences in “bottom-up” and “top-down” neural activity in current and former cigarette smokers: Evidence for neural substrates which may promote nicotine abstinence through increased cognitive control. *NeuroImage*, *56*, 2258–2275. <https://doi.org/10.1016/j.neuroimage.2011.03.054>
- Newhouse, P. A., Potter, A. S., Dumas, J. A., & Thiel, C. M. (2011). Functional brain imaging of nicotinic effects on higher cognitive processes. *Biochemical Pharmacology*, *82*, 943–951. <https://doi.org/10.1016/j.bcp.2011.06.008>
- Nieuwenhuis, S., Aston-Jones, G., & Cohen, J. D. (2005). Decision making, the P3, and the locus coeruleus–norepinephrine system. *Psychological Bulletin*, *131*, 510–532. <https://doi.org/10.1037/0033-2909.131.4.510>
- Perlato, A., Santandrea, E., Della Libera, C., & Chelazzi, L. (2014). Biases of attention in chronic smokers: Men and women are not alike. *Cognitive, Affective, & Behavioral Neuroscience*, *14*, 742–755. <https://doi.org/10.3758/s13415-014-0287-6>
- Pfefferbaum, A., Ford, J. M., Weller, B. J., & Kopell, B. S. (1985). ERPs to response production and inhibition. *Electroencephalography and Clinical Neurophysiology*, *60*, 423–434. [https://doi.org/10.1016/0013-4694\(85\)91017-X](https://doi.org/10.1016/0013-4694(85)91017-X)
- Poljac, E., & Yeung, N. (2014). Dissociable neural correlates of intention and action preparation in voluntary task switching. *Cerebral Cortex*, *24*, 465–478. <https://doi.org/10.1093/cercor/bhs326>
- Potenza, M. N., Sofuoglu, M., Carroll, K. M., & Rounsaville, B. J. (2011). Neuroscience of behavioral and pharmacological treatments for addictions. *Neuron*, *69*, 695–712. <https://doi.org/10.1016/j.neuron.2011.02.009>
- Razani, J., Boone, K., Lesser, I., & Weiss, D. (2004). Effects of cigarette smoking history on cognitive functioning in healthy older adults. *American Journal of Geriatric Psychiatry*, *12*, 404–411. <https://doi.org/10.1097/00019442-200407000-00008>
- Ridderinkhof, K. R., Ullsperger, M., Crone, E. A., & Nieuwenhuis, S. (2004). The role of the medial frontal cortex in cognitive control. *Science*, *306*, 443–447. <https://doi.org/10.1126/science.1100301>
- Schliez, N. J., Hawk, L. W., & Rosch, K. S. (2013). The effects of acute abstinence from smoking and performance-based rewards on performance monitoring. *Psychopharmacology (Berl)*, *229*, 701–711. <https://doi.org/10.1007/s00213-013-3131-8>
- Sebastian, A., Pohl, M. F., Klöppel, S., Feige, B., Lange, T., Stahl, C., ... Tüscher, O. (2012). Disentangling common and specific neural subprocesses of response inhibition. *NeuroImage*, *64*, 1–15.
- Silton, R. L., Heller, W., Towers, D. N., Engels, A. S., Spielberg, J. M., Edgar, J. C., ... Miller, G. A. (2010). The time course of activity in

- dorsolateral prefrontal cortex and anterior cingulate cortex during top-down attentional control. *NeuroImage*, 50, 1292–1302. <https://doi.org/10.1016/j.neuroimage.2009.12.061>
- Smith, J. L., Mattick, R. P., Jamadar, S. D., & Iredale, J. M. (2014). Deficits in behavioural inhibition in substance abuse and addiction: A meta-analysis. *Drug and Alcohol Dependence*, 145, 1–33. <https://doi.org/10.1016/j.drugalcdep.2014.08.009>
- Squires, K. C., Wickens, C., Squires, N. K., & Donchin, E. (1976). The effect of stimulus sequence on the waveform of the cortical event-related potential. *Science*, 193, 1142–1146. <https://doi.org/10.1126/science.959831>
- Tiffany, S. T., & Drobes, D. J. (1991). The development and initial validation of a questionnaire on smoking urges. *British Journal of Addiction*, 86, 1467–1476. <https://doi.org/10.1111/j.1360-0443.1991.tb01732.x>
- Tsaur, S., Strasser, A. A., Souprountchouk, V., Evans, G. C., & Ashare, R. L. (2015). Time dependency of craving and response inhibition during nicotine abstinence. *Addict Res Theory*, 23, 205–212. <https://doi.org/10.3109/16066359.2014.953940>
- Ullsperger, M., Fischer, A. G., Nigbur, R., & Endrass, T. (2014). Neural mechanisms and temporal dynamics of performance monitoring. *Trends in Cognitive Sciences*, 18, 259–267. <https://doi.org/10.1016/j.tics.2014.02.009>
- Vandamme, K., Szmalec, A., Liefvooghe, B., & Vandierendonck, A. (2010). Are voluntary switches corrected repetitions? *Psychophysiology*, 36, 1176–1181. <https://doi.org/10.1111/j.1469-8986.2010.01032.x>
- Wagner, M., Schulze-Rauschenbach, S., Petrovsky, N., Brinkmeyer, J., von der Goltz, C., Gründer, G., ... Winterer, G. (2012). Neurocognitive impairments in non-deprived smokers—results from a population-based multi-center study on smoking-related behavior. *Addiction Biology*, 18, 752–761. <https://doi.org/10.1111/j.1369-1600.2011.00429.x>
- West, R., & Alain, C. (1999). Event-related neural activity associated with the Stroop task. *Cognitive Brain Research*, 8, 157–164. [https://doi.org/10.1016/S0926-6410\(99\)00017-8](https://doi.org/10.1016/S0926-6410(99)00017-8)
- Weywadt, C. R., Kiehl, K. A., & Claus, E. D. (2017). Neural correlates of response inhibition in current and former smokers. *Behavioural Brain Research*, 319, 207–218. <https://doi.org/10.1016/j.bbr.2016.11.030>
- Zhang, R., Geng, X., & Lee, T. M. C. (2017). Large-scale functional neural network correlates of response inhibition: An fMRI meta-analysis. *Brain Structure and Function*, 222, 3973–3990. <https://doi.org/10.1007/s00429-017-1443-x>

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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