Psychopharmacology
https://doi.org/10.1007/s00213-020-05475-2

ORIGINAL INVESTIGATION

Can we boost attention and inhibition in binge drinking? Electrophysiological impact of neurocognitive stimulation

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Received: 9 August 2019 / Accepted: 28 January 2020

Abstract

Rationale Binge drinking (i.e. excessive episodic alcohol consumption) among young adults has been associated with deleterious consequences, notably at the cognitive and brain levels. These behavioural impairments and brain alterations have a direct impact on psychological and interpersonal functioning, but they might also be involved in the transition towards severe alcohol use disorders. Development of effective rehabilitation programs to reduce these negative effects as they emerge thus constitutes a priority in subclinical populations.

Objectives The present study tested the behavioural and electrophysiological impact of neurocognitive stimulation (i.e. transcranial direct current stimulation (tDCS) applied during a cognitive task) to improve attention and inhibition abilities in young binge drinkers.

Methods Two groups (20 binge drinkers and 20 non-binge drinkers) performed two sessions in a counterbalanced order. Each session consisted of an inhibition task (i.e. Neutral Go/No-Go) while participants received left frontal tDCS or sham stimulation, immediately followed by an Alcohol-related Go/No-Go task, while both behavioural and electrophysiological measures were recorded.

Results No significant differences were observed between groups or sessions (tDCS versus sham stimulation) at the behavioural level. However, electrophysiological measurements during the alcohol-related inhibition task revealed a specific effect of tDCS on attentional resource mobilization (indexed by the N2 component) in binge drinkers, whereas later inhibition processes (indexed by the P3 component) remained unchanged in this population.

Conclusions The present findings indicate that tDCS can modify the electrophysiological correlates of cognitive processes in binge drinking. While the impact of such brain modifications on actual neuropsychological functioning and alcohol consumption behaviours remains to be determined, these results underline the potential interest of developing neurocognitive stimulation approaches in this population.

Keywords Binge drinking · Inhibition · Event-related potentials · Neuromodulation · tDCS

Introduction

Alcohol remains the most widespread psychoactive substance consumed worldwide. A hallmark of alcohol-related disorders is the presence of executive impairments, underlain by reduced activity in frontal regions. Indeed, the dominant neurobiological (e.g. Koob 2014; Volkow and Baler 2015) and neuropsychological (e.g. Rochat et al. 2019; Wiers et al. 2007) models of severe alcohol use disorders (as defined by the DSM-5) underline that this psychiatric disorder is simultaneously characterized by (1) increased activation of the reward system (notably indexed by craving and alcohol-related biases) and (2) reduced activation of the frontal networks responsible for inhibition and executive control. This imbalance between an over-activated automatic system and an under-activated control system leads to the persistence of excessive alcohol consumption. Many studies have supported these models by repeatedly observing lower cognitive abilities (e.g. Stavro et al. 2013) and anatomical/functional

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impairments in a large range of frontal (e.g. Bühler and Mann 2011; Moselhy et al. 2001) and prefrontal (e.g. Goldstein and Volkow 2011) areas in severe alcohol use disorders.

Capitalizing on these data among patients, a new research field has emerged during the two last decades, exploring whether such cognitive and brain deficits might already be observed in subclinical populations who present excessive alcohol consumption (but who do not fulfil the criteria for severe alcohol use disorders). In this context, many studies (e.g. Carbia et al. 2018; Hermens et al. 2013 for recent reviews) have been conducted on binge drinking, a frequent alcohol consumption pattern in youth, characterized by large alcohol intakes over short periods (leading to repeated alternations between drunkenness and withdrawal episodes; Kraus et al. 2016). It has indeed been shown that adolescents who present binge drinking habits have a higher risk of developing severe alcohol use disorders in adulthood (e.g. Bonomo et al. 2004; Viner and Taylor 2007). Binge drinking, while not considered an addictive disorder per se, might thus constitute a first step in the transition between pleasurable/controlled (drug "liking", as proposed in the incentive-sensitization theory; Robinson and Berridge 2001) and compulsive/uncontrolled (drug "wanting") alcohol consumption. Although few studies have reported reduced behavioural executive performance among binge drinkers (BD) when compared with that of non-/low drinkers (e.g. Czapla et al. 2015; Townshend and Duka 2005), neuroimaging and electrophysiological techniques have identified latent brain indexes of attentional and executive difficulties related to binge drinking (e.g. Campanella et al. 2013; Crego et al. 2012; Holcomb et al. 2019; Lannoy et al. 2017; López-Caneda et al. 2013). For example, modified brain activity has been observed in BD compared with controls, despite identical behavioural performance (López-Caneda et al. 2013). Moreover, these brain modulations were stronger when BD had to explicitly inhibit alcohol-related stimuli (Lannoy et al. 2019; Petit et al. 2012). In the same vein, neuroimaging evidence also suggests that BD show reduced activation of the brain areas usually involved in attention, executive and working memory tasks (i.e. frontal and temporal regions), together with increased activation in other regions classically not associated with such tasks (e.g. Herman et al. 2018; López-Caneda et al. 2012). Together, these results have led to the emergence of the compensation hypothesis (Maurage et al. 2013), suggesting that the altered functioning of the brain network related to attentional or executive functions in binge drinking might initially be undetectable at the behavioural level due to the compensatory activation of alternative and preserved brain structures.

Given the key role of executive functions for the regulation of alcohol consumption, these latent brain impairments might be involved in the maintenance of binge drinking habits or even promote the onset of severe alcohol use disorders (Field et al. 2008; Maurage et al. 2013). The transition between binge drinking and severe alcohol use disorders could be characterized by a worsening of alcohol-related brain consequences, leading to the disappearance of the brain compensation mechanisms and to the emergence of patent cognitive impairments, as repeatedly reported in severe alcohol use disorders (Stavro et al. 2013). It thus appears crucial to identify the early brain correlates of binge drinking, even when behavioural measures cannot yet index them, and, most importantly, to rehabilitate these difficulties before their expansion. To this end, a promising perspective is offered by neuromodulation techniques, particularly by transcranial direct current stimulation (tDCS), a non-invasive and easily implemented tool, which could enhance cognitive abilities through brain stimulation (Elmasry et al. 2015).

tDCS improves executive performance in healthy participants (Ditye et al. 2012; Friehs and Frings 2018), but this technique has also recently been used in populations with alcohol use disorders (Spagnolo and Goldman 2016). Overall, these studies have shown that frontal tDCS can reduce craving in patients with severe alcohol use disorders (Boggio et al. 2008; da Silva et al. 2013) and in young heavy drinkers (den Uyl et al. 2015), as well as lower relapse rates in recently detoxified patients (Klauss et al. 2014). The modulation of frontal regions, which are directly involved in executive processing and the cognitive control of drinking behaviour, might thus lead to positive behavioural and clinical outcomes. Notably, although tDCS has mostly been applied offline (i.e. stimulation at rest), a larger impact of this procedure has recently been shown when the participant is involved in a cognitive task (i.e. online tDCS; Elmasry et al. 2015). Following this proposal, trials were conducted in heavy drinkers (den Uyl et al. 2016) and in patients with severe alcohol use disorders (den Uyl et al. 2017; den Uyl et al. 2018) by coupling frontal tDCS with cognitive training sessions to modify approach tendencies and attentional bias towards alcohol. Although the joint tDCS/cognitive training approach has shown a slight positive impact on craving in young heavy drinkers (den Uyl et al. 2016) or on the relapse rate in recently detoxified patients (den Uyl et al. 2017, 2018), no strong evidence for a specific enhancement effect of tDCS on cognitive performance has been reported.

These mixed results might be partly related to issues with clinical outcome variables (e.g. using self-reported craving measures), sample characteristics (e.g. low initial craving levels, low motivation to change) or discrepancies in stimulation parameters (e.g. localization, intensity, number of sessions), as well as centrally to the nature of the cognitive processes targeted. Indeed, the processes underlying cognitive bias modification measured in these studies were mainly related to implicit and automatic behaviours, rather than being based on limbic areas and the reward system (Noël et al. 2010). Moreover, it has been shown that tDCS effects can

be influenced by the cognitive load, as they are increased when the cognitive process is sufficiently demanding to require the recruitment of higher cognitive and neuronal resources (Elmasry et al. 2015; Gill et al. 2015). It is thus not surprising that tDCS stimulation focused on frontal areas has only limited impact on processes that require low involvement of frontal regions and their related cognitive resources, in comparison with more controlled executive processes (Shiffrin and Schneider 1977).

Therefore, to address these issues, we tested whether applying online (i.e. performed simultaneously with a cognitive task) frontal neuromodulation through tDCS might increase efficiency in a cognitive task (Go/No-Go task) by directly mobilizing the neural resources (i.e. frontal areas) involved in controlled processes (i.e. attention and inhibitory control). We chose the Go/No-Go paradigm because it presents a high level of difficulty and resource mobilization (Vocat et al. 2008). Self-reported impulsivity was also measured at baseline (through the UPPS-P; Billieux et al. 2012), as this factor is related to binge drinking, predicts excessive alcohol consumption (Henges and Marczinski 2012; Townshend and Duka 2005) and may also have an impact on the processing speed in a Go/No-Go task (Lannoy et al. 2019). Moreover, to further evaluate whether neurocognitive online tDCS can enhance attentional and inhibitory performance in BD and to observe the potential post-stimulation effect of the tDCS, we performed behavioural measures after tDCS by using soft drink and alcohol-related stimuli while participants performed a Go/No-Go task (Lannoy et al. 2018). Although slightly different from the Neutral Go/No-Go task, this adapted version was chosen because (1) it requires similar cognitive and brain executive mechanisms; (2) it has already demonstrated specific differences between BD and non-BD (Lannoy et al. 2018); and (3) it avoids repeated administration of the same task across sessions, which might generate training effects. This behavioural measure was combined with a simultaneous electrophysiological recording to determine the brain correlates of neurocognitive stimulation. Interestingly, the high temporal resolution and large sensitivity of event-related potentials (ERPs) can index the extent (and modification through neurocognitive stimulation) of subtle alcohol-related effects, still undetectable at the behavioural level (Campanella and Noël 2016). To specifically explore the brain correlates of attentional and inhibitory processes, we recorded two electrophysiological components indexing high-level cognitive abilities, namely N2 and P3. N2 is classically considered to reflect the amount of attentional resources involved in the task (i.e. attentional focus; Knight 1991; Smith et al. 2013), but it is also related to response inhibition and conflict (Donkers and van Boxtel 2004; Nieuwenhuis et al. 2003) and modulated by response-related activation (Bruin et al. 2001). P3 reflects high-level decisional processes preceding response initiation (Polich 2004; Sutton et al. 1965) or response inhibition (Wessel and Aron 2015). N2 and P3 were also chosen because they have been repeatedly explored in binge drinking. Although several studies have shown delayed latency or reduced amplitude for N2 (Maurage et al. 2009, 2012) and P3 (Ehlers et al. 2007; Petit et al. 2014) among BD, other studies have conversely shown increased amplitudes (Crego et al. 2009, 2012; López-Caneda et al. 2013) or an absence of group differences (Park and Kim 2018; Petit et al. 2012; Watson et al. 2016). The current literature is thus characterized by mixed results regarding the electrophysiological modifications related to this drinking pattern.

The main aim of our study was to directly explore whether a neurocognitive stimulation approach, using online tDCS, can improve attentional and executive functioning at the behavioural and electrophysiological levels in binge drinking. We used a single-blinded within-subject design with two groups (BD, non-BD) and two sessions (sham stimulation, active tDCS). We centrally hypothesised that (1) BD would not present patent behavioural deficits for attention or inhibitory control, but might have modified brain activations related to cognitive processes (despite mixed results reported by earlier electrophysiological studies), and (2) neurocognitive stimulation would improve attentional and inhibition abilities in both groups and counter the potential electrophysiological modifications related to binge drinking.

Methods

Participants

Participants were selected through an online screening questionnaire sent by email or through social networks to university students. The screening assessed socio-demographic variables (age, gender) and alcohol consumption variables (i.e. consumption speed in units per hour, mean number of alcohol units per week, mean number of alcohol units (10 g of pure ethanol) per drinking occasion, mean number of drinking occasions per week, drunkenness frequency). Inclusion criteria were as follows: no personal/family history of moderate/severe alcohol use disorders and epilepsy, no history of head injury, no brain surgeries, no psychological/neurological disorders, no psychotropic medications, normal or corrected-to-normal vision and absence of past or current drug consumption (except for alcohol/tobacco). These inclusion criteria were measured through self-reported items.

Two groups (BD, non-BD) were formed based on their binge drinking score (Townshend and Duka 2005) and the number of alcohol units per occasion (López-Caneda et al. 2014). The binge drinking score was computed according to the following formula: (4 × consumption speed) + number of drunkenness episodes + (0.2 × percentage of drunkenness episodes). Cut-off scores for group assignation (Townshend and

Duka 2005) were adapted for Belgium's alcohol unit measurement (Lannoy et al. 2017) and reinforced by other alcohol-related measures (Table 1). Twenty BD (binge drinking score > 16; alcohol units/occasion ≥ 6) and 20 non-BD (binge drinking score < 12; alcohol units/occasion ≤ 3) were selected. All BD reported beer as their most regular alcoholic drink.

All participants (10 women per group) were between 18 and 26 years old (BD: 21.3 ± 2.0; non-BD: 21.6 ± 2.6). To control the potential effects of psychopathological comorbidities, we asked participants to fill in questionnaires that assessed depressive symptoms (Beck Depression Inventory, BDI-II; Beck et al. 1996), anxiety (State-Trait Anxiety Inventory, STAI; Spielberger et al. 1983), impulsivity (UPPS-P; Billieux et al. 2012) and alcohol-related disorders (Alcohol Use Disorder Identification Test, AUDIT; Babor et al. 2001). All participants were asked to refrain from consuming alcohol during the 3 days preceding each experimental session.

General procedure

The experiment consisted of two 2-h sessions (one session with active tDCS, one with sham stimulation), separated by 7 days. The order of the sessions was counterbalanced across participants to avoid potential learning and/or training effects. After we checked the exclusion criteria for tDCS use and informed participants about the whole procedure, they provided written informed consent. They were seated in front of a Dell computer (1280 × 1024 pixels) at a 60-cm viewing distance and tested individually.

The procedure was identical across sessions: participants first had to perform a cognitive inhibition task (Neutral Go/No-Go task) coupled with neuromodulation (see tDCS section), followed by an evaluation task (Alcohol-related Go/No-Go task) during which electrophysiological data were recorded (see EEG section) to assess the effect of online tDCS (Fig. 1b). The two Go/No-Go tasks were presented by using E-Prime 2 Professional software (Psychology Software Tools, Pittsburgh, PA, USA). After the online tDCS was completed, an electrophysiological recording cap was immediately placed on the participant. Between sessions, participants had to complete the different questionnaires online by using Qualtrics software (Qualtrics, LLC). At the end of the second session, participants were debriefed and received compensation (40 euros). None of the participants reported having detected a difference between the sham stimulation and tDCS sessions. The study protocol was approved by the local ethics committee and carried out according to the principles of the Declaration of Helsinki.

Neurocognitive stimulation

Cognitive inhibition task

In this Neutral Go/No-Go task (adapted from Vocat et al. 2008), arrows presented centrally on a white background were used as visual stimuli (subtending a visual angle of 11.4°×0.05°). Each trial started with a black arrow, oriented upward or downward, presented for a variable duration (1000–2000 ms). The arrow could then turn either green or turquoise (these two colours were matched for luminance), either in a similar or in the opposite direction (Fig. 1a). The

Table 1 Demographic, psychopathological and alcohol consumption measures (mean (SD)) for binge drinkers (BD) and non-binge drinkers (non-BD) participants

	BD ($n = 20$)	non-BD ($n = 20$)
Demographic measures		
Gender ratio (male/female)[ns]	10/10	10/10
Age[ns]	21.25 (2.0)	21.60 (2.6)
Laterality (right/left)[ns]	19/1	19/1
Psychopathological measures		
Beck Depression Inventory[ns]	6.80 (4.5)	5.65 (5.0)
State Anxiety Inventory[ns]	37.25 (10.7)	35.40 (8.8)
Trait Anxiety Inventory[ns]	38.90 (8.4)	39.40 (7.6)
UPPS-P*	48.70 (8.1)	41.75 (7.9)
Alcohol consumption measures		
Alcohol Use Disorder Identification Test**	17.65 (5.6)	2.80 (2.9)
Binge Drinking Score**	40.04 (19.5)	3.00 (4.0)
Total units per week**	26.95 (12.3)	1.20 (1.9)
Number of occasions per week**	3.10 (1.0)	0.65 (1.1)
Number of units per occasion**	9.11 (3.3)	0.71 (1.1)

[ns] Non-significant, *$p < .01$, **$p < .001$

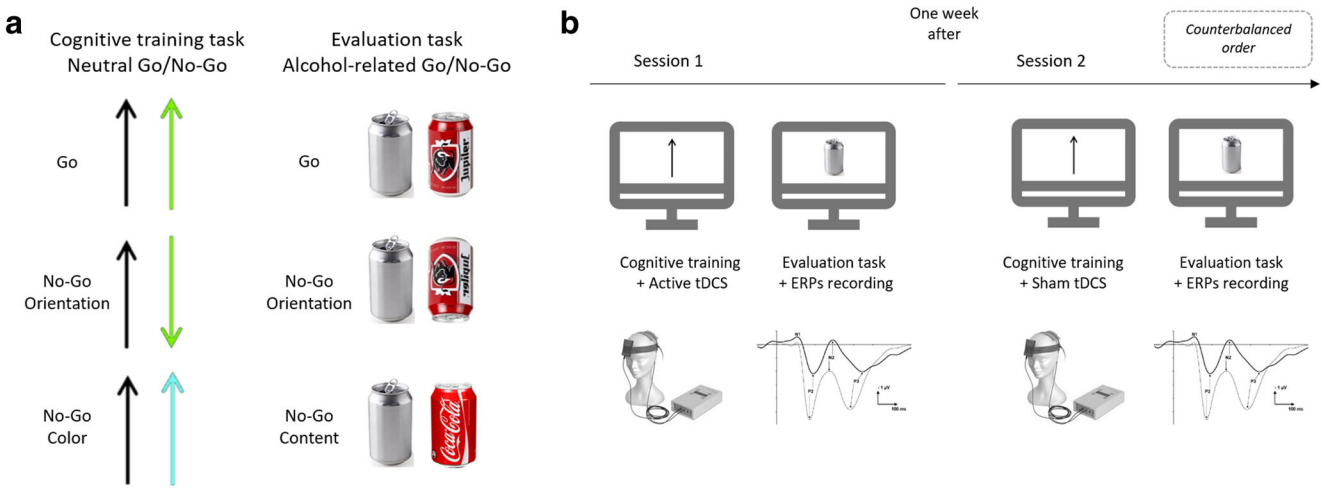

Fig. 1 **a** Trial examples for the Neutral (left) and Alcohol-related (right) Go/No-Go tasks (illustrated here for the neutral upward orientation). The neutral arrow/can was followed either by a green arrow/alcohol drink can in the same orientation (Go trial), by a green arrow/alcohol drink can in the opposite orientation (No-Go orientation trial) or by a turquoise arrow/soft drink can in the same orientation (No-Go colour/content trial). **b** General experimental procedure. The experiment consisted of two sessions, separated by 7 days. During each session, participants had to perform (1) a Neutral Go/No-Go task during which they received 20 min of active or sham tDCS (the order of the session was counterbalanced), followed by (2) an Alcohol-related Go/No-Go task while electrophysiological data were recorded

coloured arrow remained on the screen for a maximum of 1500 ms. Trials were separated by a blank screen (500 ms), followed by a central fixation cross (500 ms).

Participants were instructed to respond as fast as possible by pressing the space bar with their dominant hand each time the black arrow turned green and maintained the same direction (Go trials). Participants had to refrain from responding each time the black arrow turned turquoise or changed its direction (No-Go colour and No-Go orientation trials, respectively). The task comprised two parts, each consisting of a calibration block (14 trials; 10 Go, 2 No-Go colour, 2 No-Go orientation), directly followed by two test blocks (60 trials each; 40 Go, 10 No-Go of each type). The whole task lasted around 20 min and included 268 stimuli.

During each calibration block, the mean reaction time (RT) for Go trials was computed online and used to define a reference threshold for the following test blocks. Participants were not informed about this procedure. For the first session, the upper answer time limit was set to 80% of the mean RT of the corresponding calibration block, and the upper limit was set to 90% for the second session (for more details, see Vocat et al. 2008). When participants were too slow, a feedback screen related to their speed was displayed ("Too late" on-screen message). Participants were informed that these slow hits were considered errors and that they reduced their percentage of correct responses (indicated on the top right of the screen). This procedure forced participants to respond as quickly as possible and thus promoted frequent error occurrences (Vocat et al. 2008). This speedy version of the Go/No-Go paradigm increased the complexity of the task and thus improved the discrimination of participants' performance by preventing ceiling effects usually observed with simple tasks (Campanella et al. 2017). As underlined earlier, the main aim of this Neutral Go/No-Go task used during tDCS stimulation (i.e. online tDCS) was to expose participants to a cognitive task by using high-level inhibition processes and recruiting brain areas specifically involved in this type of processing. This recruitment of inhibitory-related processes and brain networks was used to maximize the tDCS impact, as it is increased when performed simultaneously with a complex cognitive task (Elmasry et al. 2015; Li et al. 2019).

tDCS

While participants performed the Neutral Go/No-Go task, a 1.5-mA current was administered by using two 35-cm^2 (7 × 5 cm) electrodes inserted in saline-soaked sponges and connected to a DC stimulator (Neuroconn, Ilmenau, Germany). The electrode position was established by using the international 10–20 EEG system, with the anodal electrode placed at the F3 position (corresponding to the left dorsolateral prefrontal cortex area) and the cathodal electrode placed above the right eye, at the Fp2 position (corresponding to the right supraorbital region). In the active tDCS condition, the current was held constant during the whole Neutral Go/No-Go task (20 min). During the sham condition, the device was turned off after a 30-s stimulation. In both conditions, gradual 20-s fade-in and fade-out phases were used.

Evaluation task and EEG recording

Alcohol-related Go/No-Go task

The same procedure as that used in the Neutral Go/No-Go task was followed, except for the type and number of stimuli

displayed. This adapted version used soft drink- and alcohol-related stimuli (Lannoy et al. 2018). Each trial started with a neutral grey can, oriented upward or downward. The can could then be replaced by an alcohol drink can (beer can) or a soft drink can, either in a similar or in the opposite direction (Fig. 1a). Several beer and soft drink cans were used during this task, matched on perceptual parameters (colour, size, luminosity). Participants have seen an illustration of all cans beforehand to ensure their correct identification.

Participants had to respond as quickly as possible each time the grey can was replaced by an alcohol drink and maintained the same direction (Go trials) and to refrain from responding each time the can became a soft drink or changed its direction (No-Go content trials and No-Go orientation trials, respectively). This second task was divided into three parts, each consisting of a calibration block (14 trials; 10 Go, 2 No-Go content, 2 No-Go orientation), directly followed by two test blocks (60 trials each; 40 Go, 10 No-Go of each type). The whole task lasted around 30 min and included 402 stimuli. The same speedy procedure and mean RT computation during the calibration block were applied, including the feedback screen displayed after a slow hit and the percentage of correct responses indicated on the top right of the screen.

EEG acquisition and pre-processing

Electrophysiological data were recorded with a 128-channel (pin-type) Biosemi ActiveTwo system referenced to the CMS-DRL ground at 1024 Hz (0–208 Hz bandwidth). EEG processing was performed by using the BrainVision Analyzer. First, EEG data were band-pass filtered (0.1–30 Hz, Butterworth zero phase filters, 12 dB/oct), followed by a notch filter at 50 Hz. Ocular artefact removal was carried out through an independent component analysis ICA-based strategy. Signals were re-referenced in order to average and build EEG segments beginning 200 ms before and ending 800 ms after stimulus onset. Baseline correction was applied for the mean activity during the 200 ms prior to stimulus. Algorithmic artefact rejection of voltage exceeding ± 100 μV was conducted, and segments with artefacts were manually rejected. Finally, individual participants' averages for correct trials were built separately for each condition (i.e. correct No-Go and Go). The two main components classically considered to be ERP markers of response inhibition, namely N2 and P3 (Donkers and Van Boxtel 2004; Wessel and Aron 2015), were identified. In line with previous studies (Kreusch et al. 2014; López-Caneda et al. 2014; Maurage et al. 2012) and following visual data inspection, the N2 component was quantified by measuring peak amplitude/latency at three frontal electrodes (Fz-F3-F4) in a 150- to 300-ms time interval, and the P3 component was computed at three parietal electrodes (Pz-P3-P4) in a 300- to 500-ms time interval. Finally, the mean peak amplitude and latency obtained for the three electrodes were averaged for each region (López-Caneda et al. 2014).

Statistical analyses

First, between-group comparisons (i.e. independent *t* tests and chi-square independent test) were performed on demographic and psychopathological characteristics, as well as on alcohol consumption variables. Second, behavioural performance related to the Neutral and Alcohol-related Go/No-Go tasks was explored by using analyses of variance (ANOVA) separately for correct Go responses (RT hits) and correct No-Go responses (%). To observe the potential effect of neurocognitive stimulation on inhibition performance of both groups, a 2 × 2 ANOVA with GROUP (BD, non-BD) as the between-subject factor and SESSION (Active, Sham) as the within-subject factor was computed. At the electrophysiological level, two 2 × 2 ANOVAs (amplitude, latency) were performed for each component (N2, P3) in both trial types (correct Go, correct No-Go trials), with GROUP (BD, non-BD) as the between-subject factor and SESSION (Active, Sham) as the within-subject factor. Finally, correlational analyses were performed between behavioural and electrophysiological measures that significantly differed across groups. Moreover, to further explore the links between binge drinking habits and inhibition difficulties, we performed correlations between inhibition performance during Go/No-Go tasks and alcohol consumption.

Results

Demographic and psychopathological measures (Table 1)

No significant group difference was observed for age [$t(38) = 0.477$, $p = .636$], gender [$\chi^2(1, N = 40) = 0.000$, $p = 1$], depressive symptoms [$t(38) = 0.761$, $p = .451$] and state [$t(38) = 0.596$, $p = .555$] or trait anxiety [$t(38) = 0.198$, $p = .844$]. Regarding impulsivity, BD had a larger UPPS-P score than non-BD did [$t(38) = 2.754$, $p = .009$]. Significant differences regarding alcohol consumption were observed: BD had a larger binge drinking score [$t(38) = 8.327$, $p < .001$], AUDIT score [$t(38) = 10.461$, $p < .001$], number of units per week [$t(38) = 9.245$, $p < .001$], number of occasions per week [$t(38) = 7.339$, $p < .001$] and number of units per occasion [$t(38) = 10.868$, $p < .001$].[1]

[1] Complementary statistical analyses, including gender as a between-subject factor for all experimental variables (alcohol consumption, behavioural and electrophysiological measures), did not show any significant difference between females and males, either for the whole sample or within BD/non-BD groups.

Behavioural data (Table 2)

In both tasks (Neutral and Alcohol-related Go/No-Go), there neither were significant differences between groups (BD, non-BD) or sessions (Active, Sham) nor were there significant interactions for any of the behavioural variables analysed (RT Go trials, percentage of correct No-Go responses).

Correlational analyses between inhibition performance and alcohol consumption measures showed a significant negative relationship between percentage of correct No-Go responses and binge drinking score in both tasks (Neutral and Alcohol-related Go/No-Go) and both sessions (Active and Sham) for BD only (all p values $< .001$). Moreover, significant correlations were observed between the UPPS-P score and percentage of correct No-Go responses in both tasks (Neutral and Alcohol-related Go/No-Go) in the Sham condition (all p values $< .05$).

Electrophysiological analyses (Table 3)

Correct Go trials

- N2 amplitude: A GROUP × SESSION interaction was found [$F(1,38) = 4.728$, $p = .036$, $\eta^2 = 0.111$], showing a larger amplitude after stimulation than after a sham session in BD [$t(19) = 2.438$, $p = .025$], whereas no significant difference was observed in non-BD [$t(19) = 0.486$, $p = .632$; Fig. 2].
- N2 latency: There was a main effect of SESSION [$F(1,38) = 4.107$, $p = .050$, $\eta^2 = 0.098$], showing a longer latency in the sham session than in the active session. No main effect of GROUP [$F(1,38) = 1.161$, $p = .288$, $\eta^2 = 0.030$] and no GROUP × SESSION interaction [$F(1,38) = 1.916$, $p = .174$, $\eta^2 = 0.048$] was observed.
- P3 amplitude: No significant main effect of GROUP and SESSION and no significant interaction were found (all p-values $> .05$).
- P3 latency: No significant main effect of GROUP and SESSION and no significant interaction were found (all p-values $> .05$).

Correct No-Go trials

- N2 amplitude: No significant main effect of GROUP and SESSION and no significant interaction were found (all p-values $> .05$).
- N2 latency: No significant main effect of GROUP and SESSION and no significant interaction were found (all p-values $> .05$).
- P3 amplitude: A GROUP × SESSION interaction was found [$F(1,38) = 7.616$, $p = .009$, $\eta^2 = 0.167$], showing a larger amplitude after stimulation than after a sham session in non-BD [$t(19) = 2.666$, $p = .015$], whereas no significant difference was observed in BD [$t(19) = 1.427$, $p = .170$; Fig. 3].
- P3 latency: No significant main effect of GROUP and SESSION and no significant interaction were found (all p-values $> .05$).

Correlational analyses

No significant correlations were found between behavioural and electrophysiological measures. A significant negative correlation was revealed between the UPPS-P score and the effect of stimulation on the N2 latencies for Go trials ($r = -.335$, $p = .034$).

Table 2 Behavioural data for cognitive training (i.e. Neutral Go/No-Go) and evaluation (i.e. Alcohol-related Go/No-Go) tasks (mean (SD)) as a function of stimulation session (Active, Sham) for binge drinkers (BD) and non-binge drinkers (non-BD) participants

Behavioural performance	BD		non-BD	
	Active	Sham	Active	Sham
Neutral Go/No-Go				
RT Hits (ms)	330 (53)	324 (37)	323 (41)	320 (40)
Correct No-Go (%)	65.75 (25.1)	70.75 (16.6)	73.50 (18.2)	76.57 (14.8)
Alcohol-related Go/No-Go				
RT Hits (ms)	368 (62)	369 (36)	380 (50)	391 (64)
Correct No-Go (%)	50.11 (26.3)	48.32 (21.6)	52.36 (26.2)	54.32 (25.3)

RT reaction time

Table 3 Amplitude (in μV) and latency (in ms) (mean (SD)) of the N2 component for the frontal region (mean of Fz, F3 and F4) and P3 component for the parietal region (mean of Pz, P3 and P4) in each experimental condition (correct Go and correct No-Go) as a function of session (Active, Sham) for binge drinkers (BD) and non-binge drinkers (non-BD) participants

	Variable	Condition	BD		non-BD	
			Active	Sham	Active	Sham
N2	Amplitude	Go	0.43 (2.76)	1.84 (2.93)	1.53 (2.55)	1.29 (2.54)
		No-Go	− 2.98 (2.56)	− 3.21 (3.05)	− 2.55 (2.76)	− 3.23 (3.02)
	Latency	Go	215 (26)	233 (25)	230 (33)	234 (33)
		No-Go	249 (24)	246 (31)	248 (28)	259 (20)
P3	Amplitude	Go	− 0.55 (1.58)	0.23 (2.05)	0.42 (1.30)	0.08 (1.58)
		No-Go	4.49 (2.80)	5.05 (2.45)	5.05 (1.87)	4.24 (2.21)
	Latency	Go	397 (55)	389 (56)	394 (50)	383 (59)
		No-Go	408 (43)	408 (41)	409 (44)	399 (37)

Discussion

The cognitive and brain consequences of binge drinking have been established during the last decade (Hermens et al. 2013). Neurocognitive stimulation has emerged as a potential tool to counter alcohol-related negative effects (Spagnolo and Goldman 2016). The present study measured the behavioural and electrophysiological correlates of neurocognitive stimulation in university students, testing whether this technique can modify attentional and inhibitory processes in BD and non-BD.

No significant behavioural group difference was observed regarding executive functioning. Contrary to what is found in severe alcohol use disorders, binge drinking is not associated with massive executive impairments at the behavioural level (Lannoy et al. 2017; López-Caneda et al. 2012), despite higher self-reported impulsivity and a (non-significant) trend to commit more errors in BD than in non-BD in both Go/No-Go tasks. In addition, anodal tDCS over the left frontal area did not improve inhibition performance, as no difference in RT and the correct response rate was detected between sessions. As suggested earlier (Campanella et al. 2018), the behavioural outcomes classically measured in the Go/No-Go task might not be sensitive enough to detect subtle modifications resulting from neurocognitive stimulation. This absence of behavioural differences does not imply that tDCS has no impact on the brain, as demonstrated by recent studies using neuroscience tools among healthy participants (Campanella et al. 2017; Cunillera et al. 2016; Sallard et al. 2018). The joint use of electrophysiological measures in our study has thus revealed the modulation effects of tDCS on brain activity, beyond the absence of a detectable behavioural counterpart.

A reduced N2 latency for correct Go trials was observed after stimulation in both groups, suggesting a positive offline influence of tDCS. In Go trials, this component is usually interpreted as being related to the speed and intensity of attentional resource mobilization (Knight 1991; Smith et al. 2013) and also indexes response-related activation (Bruin et al. 2001). The first main electrophysiological result is thus that neurocognitive stimulation leads, in both groups, to a global

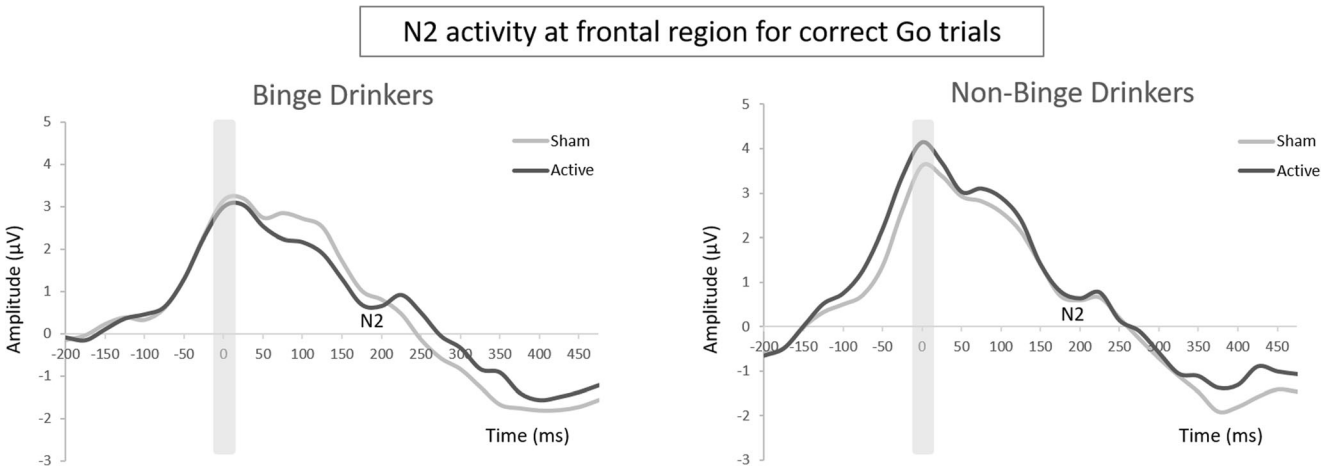

Fig. 2 Grand average event-related potential waveforms of correct Go trials at the frontal region for sham (light grey line) and active (dark grey line) sessions for binge drinkers (left) and non-binge drinkers (right)

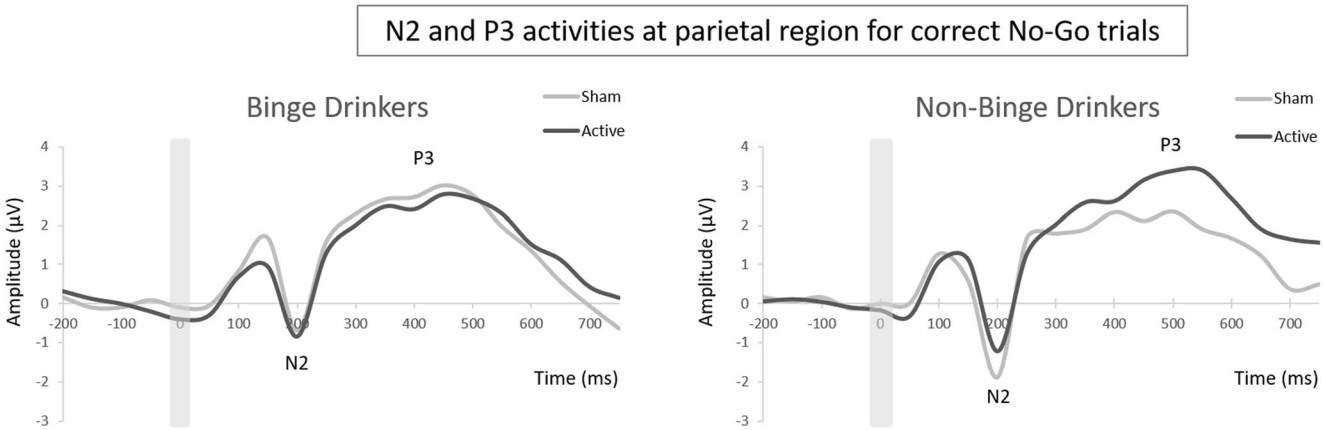

Fig. 3 Grand average event-related potential waveforms of correct No-Go trials at the parietal region for sham (light grey line) and active (dark grey line) sessions for binge drinkers (left) and non-binge drinkers (right)

boost of attentional abilities for Go trials, which is in line with earlier studies showing that tDCS impacts attentional performance (e.g. Filmer et al. 2017; Heeren et al. 2017). This boost is even stronger among BD, also showing increased N2 amplitudes following neurocognitive stimulation (Fig. 2). This result should, however, be interpreted with caution among BD, as our paradigm exclusively used alcohol-related stimuli as Go trials. It can thus not be excluded that tDCS in fact enhanced the processing of salient alcohol cues in this group (i.e. favours the attentional processing of alcohol), which might be counterproductive at the therapeutic level. Future studies comparing the electrophysiological effect of tDCS for Go trials related to alcoholic versus non-alcoholic stimuli are thus needed to determine whether the attentional boost in binge drinking is general (as suggested by the fact that this effect was also observed here among non-BD) or specific to alcohol-related stimuli. It should also be underlined that this attentional boost is not found in No-Go trials, where N2 indexes inhibitory or response conflict processes (Botvinick et al. 2001; Nieuwenhuis et al. 2003). The tDCS-related modifications are thus specific to attentional resources, as no effect is observed when inhibitory abilities are recruited.

Regarding the P3 component, which constitutes the main electrophysiological index of cognitive inhibition (Smith et al. 2007; Wessel and Aron 2015), results showed no impact of neurocognitive stimulation among BD. Conversely, the P3 amplitude for correct No-Go trials was increased following stimulation in the non-BD group, indexing a tDCS impact on successful response inhibition (Jacobson et al. 2012; Lapenta et al. 2014). Although inhibition abilities may be compromised in binge drinking (Campanella et al. 2013), neurocognitive stimulation did not significantly impact the specific abilities related to motor response inhibition in this group. As a whole, neurocognitive stimulation can boost attentional resources among BD but, conversely, has no impact on inhibitory processes in this population.

These findings clarify the usefulness of neuromodulation techniques in binge drinking, as well as their limits. On the one hand, our study confirms that online tDCS constitutes a powerful way to modulate brain activity in subclinical populations (Elmasry et al. 2015; Sathappan et al. 2018). The validity of our results is reinforced by the within-subject design used (each participant receiving both active and sham tDCS), which took into account the individual baseline level and minimized the impact of inter-individual differences. Moreover, our choice to use a task involving cognitive processes (which are involved in the persistence of binge drinking; Gill et al. 2015) related to frontal regions (Chikazoe 2010) proved to have stronger sensitivity than was the case in previous studies that focused on craving/automatic bias modification (den Uyl et al. 2016). Finally, our findings confirm the importance of electrophysiological measures, beyond behavioural indexes, to objectify subtle modifications of brain activity and improve the understanding of the processes involved in excessive alcohol consumption (Houston and Schlienz 2018). All these methodological choices pave the way for the development of gold standards for neurocognitive stimulation in subclinical populations. On the other hand, our results suggest that neurocognitive stimulation does not lead to global improvement of brain activity, as it influences attentional components (i.e. N2), but does not modify the electrophysiological correlates of inhibition (i.e. P3), which is at the core of addictive disorders. This lack of impact might be related to the absence of a strong pre-existing inhibition deficit among BD. Indeed, although BD tended to commit more errors than non-BD did, and although a positive correlation was found between binge drinking scores and error rates, our hypothesis that BD would present pre-stimulation cognitive or electrophysiological deficits was not globally confirmed. As mentioned earlier, previous electrophysiological studies among BD led to mixed results, some showing N2-P3 impairments (Ehlers et al. 2007; Maurage et al. 2009, 2012; Petit et al. 2014) while others did not (Park and Kim 2018; Petit et al. 2012; Watson et al. 2016).

The absence of deficit observed among BD in our study is thus not at odds with several earlier results, but it could have lowered the improvement range allowed by tDCS, particularly regarding inhibition. This confirms the importance of considering the baseline cognitive state when defining tDCS paradigms in experimental studies or rehabilitation programs (e.g. Dubreuil-Vall et al. 2019; Li et al. 2019). Nevertheless, even though our results cannot prove that neurocognitive stimulation is useful to compensate for brain deficits among BD (as they did not present such deficits at baseline), the findings do show that tDCS can enhance brain activity related to attentional processes in binge drinking. Boosting such processes, which are involved in the emergence and maintenance of alcohol-related disorders, might have a positive impact on excessive alcohol consumption (future work being needed to quantify this impact). Notably, the absence of baseline group differences does not lessen the interest of our results: tDCS-related changes here constitute an improvement of preserved abilities rather than the rehabilitation of impaired abilities. Many studies have indeed measured the boosting effect of tDCS on a wide range of cognitive functions in healthy populations (e.g. Ditye et al. 2012; Filmer et al. 2017; Friehs and Frings 2018; Heeren et al. 2017) and have shown that a beneficial effect (i.e. further boosting already efficient abilities) can be observed even in the absence of a pre-existing deficit.

This study being the first to explore both the behavioural and electrophysiological impact of neurocognitive stimulation in BD, the current findings should be extended, notably by exploring the impact of such an approach on other cognitive processes (e.g. memory, emotional processing). Their modulation by demographic factors (e.g. gender, as the absence of gender effect reported here was based on a limited sample size), psychological factors (e.g. impulsivity) or alcohol-related factors (e.g. craving intensity and alcohol consumption during the days preceding the experiment, which were not measured here) should also be documented. As disease stage (e.g. subclinical versus clinical status) strongly modulates the efficiency of neurocognitive rehabilitation (e.g. Wiers et al. 2018), the variation of the impact of tDCS according to the intensity of binge drinking habits should also be further explored (notably to extend our correlational analyses that show that inhibitory deficits are influenced by the binge drinking score).

Moreover, future studies should explore how methodological choices modulate the impact of neuromodulation on behavioural or electrophysiological measures. First, although the technical characteristics of tDCS used here are totally in line (e.g. regarding stimulation intensity/duration and electrode size) with the most recent studies that apply neuromodulation in addictive disorders (e.g. Den Uyl et al. 2017, 2018; Klauss et al. 2018), modifying such characteristics might modulate the impact of tDCS. Large variations in executive enhancement (after frontal stimulation) have been reported, depending on current density (Dedoncker et al. 2016) or electrode size/montage (Imburgio and Orr 2018). Second, the effect of repeated tDCS sessions should be measured: this study proposed only one active session and the impact was measured just after stimulation, which did not allow us to measure the increased behavioural and brain impact related to multiple tDCS sessions or to determine the long-term evolution of such an impact. A recent review showed that a single stimulation session was frequently insufficient in revealing reliable improvement effects (Horvath et al. 2015). In the same vein, we applied anodal stimulation to the left frontal cortex, which is consistent with previous studies on craving modulation in heavy drinkers (den Uyl et al. 2015, 2016). However, other studies have reported effects when stimulating the right frontal cortex in similar Go/No-Go tasks (Cunillera et al. 2016; López-Caneda et al. 2014). A more systematic exploration of the influence of stimulation site should determine the optimal location and define standardized guidelines. Finally, it should be underlined that the impact of neurocognitive stimulation might be modulated by behavioural or brain abnormalities preceding alcohol consumption. Indeed, although alcohol neurotoxicity has a direct effect on brain functioning in binge drinking (e.g. Maurage et al. 2009), some BD might also present pre-existing genetic or neurobiological vulnerabilities (Goldstein and Volkow 2011; Volkow et al. 2012), leading to cognitive and brain alterations before the initiation of alcohol consumption. Such predisposing factors, notably related to reduced frontal activation (e.g. Norman et al. 2011), might be more stable and thus less sensitive to neurocognitive stimulation.

Conclusions

The main outcome of the present research is that, whereas earlier studies had observed a limited impact of neuromodulation on craving in heavy drinkers, the use of online tDCS can modify the brain correlates of cognitive processing. Neurocognitive stimulation had a significant and specific impact on attentional resource mobilization in BD (indexed by N2), whereas the later processing stages (i.e. inhibition or motor response preparation, indexed by P3) remained unchanged by the intervention in this group. The modulation of electrophysiological activity that results by following our approach proves that neurocognitive stimulation can efficiently boost specific brain processes in BD, which may initiate the application of such interventions in subclinical populations. However, future studies are needed to clarify the concrete influence of these brain modifications on cognitive functioning and alcohol consumption habits before applying neurocognitive remediation approaches in this population.

Acknowledgements We thank Laura Ninane, Celia Rotha and Stéphane Acke for their help in data collection.

Funding information Pierre Maurage (Senior Research Associate) is funded by the Belgian Fund for Scientific Research (F.R.S.-FNRS, Belgium), and this research has been supported by a grant from the Fondation pour la Recherche en Alcoologie (FRA), but these funds did not exert any editorial direction or censorship on any part of this article.

Compliance with ethical standards

The study protocol was approved by the local ethics committee and carried out according to the principles of the Declaration of Helsinki.

Conflict of interest The authors declare that they have no conflict of interest.

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