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# Delta, theta, and alpha event-related oscillations in alcoholics during Go/NoGo task: Neurocognitive deficits in execution, inhibition, and attention processing

Ashwini K. Pandey *, Chella Kamarajan, Niklas Manz, David B. Chorlian, Arthur Stimus, Bernice Porjesz

*Henri Begleiter Neurodynamics Laboratory, Department of Psychiatry and Behavioral Sciences, SUNY Downstate Medical Center, 450 Clarkson Avenue, MSC #1203, Brooklyn, NY 11203, USA*

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ABSTRACT

Higher impulsivity observed in alcoholics is thought to be due to neurocognitive functional deficits involving impaired inhibition in several brain regions and/or neuronal circuits. Event-related oscillations (EROs) offer time-frequency measure of brain rhythms during perceptual and cognitive processing, which provide a detailed view of neuroelectric oscillatory responses to external/internal events. The present study examines evoked power (temporally locked to events) of oscillatory brain signals in alcoholics during an equal probability Go/NoGo task, assessing their functional relevance in execution and inhibition of a motor response. The current study hypothesized that increases in the power of slow frequency bands and their topographical distribution is associated with tasks that have increased cognitive demands, such as the execution and inhibition of a motor response. Therefore, it is hypothesized that alcoholics would show lower spectral power in their topographical densities compared to controls. The sample consisted of 20 right-handed abstinent alcoholic males and 20 age and gender-matched healthy controls. Evoked delta (1.0–3.5 Hz; 200–600 ms), theta (4.0–7.5 Hz; 200–400 ms), slow alpha (8.0–9.5 Hz; 200–300 ms), and fast alpha (10.0–12.5 Hz; 100–200 ms) ERO power were compared across group and task conditions. Compared to controls, alcoholics had higher impulsiveness scores on the Barrett Impulsiveness Scale (BIS-11) and made more errors on Go trials. Alcoholics showed significantly lower evoked delta, theta, and slow alpha power compared to controls for both Go and NoGo task conditions, and lower evoked fast alpha power compared to controls for only the NoGo condition. The results confirm previous findings and are suggestive of neurocognitive deficits while executing and suppressing a motor response. Based on findings in the alpha frequency ranges, it is further suggested that the inhibitory processing impairments in alcoholics may arise from inadequate early attentional processing with respect to the stimulus related aspects/semantic memory processes, which may be reflected in lower posterio-temporal evoked fast alpha power. It can thus be concluded that alcoholics show neurocognitive deficits in both execution and suppression of a motor response and inadequate early attentional processing with respect to the semantic memory/stimulus related aspects while suppressing a motor response.



## 1. Introduction

Inhibition of undesired actions, automatic/pre-potent or deliberatx`e, is an essential component of self-regulation of behavior. Inadequate response inhibition has been implicated as a core dysfunction in a spectrum of externalizing psychiatric disorders characterized by impulsive behaviors, such as substance abuse disorders (SUDs), attention deficit hyperactivity disorder, conduct disorder, and antisocial personality disorder (Bauer, 2001; Bauer and Hesselbrock, 1999a,b, Brandeis et al., 2002; Chamberlain and Sahakian, 2007; Kaufman et al., 2003; Kiehl et al., 1999, 2000; Rubia et al., 2005; Young et al., 2000). In humans, neural substrates of response inhibition have been studied using various types of Go/NoGo tasks that require a speeded motor response to a Go stimulus and withholding a response when a NoGo stimulus is presented. Studies using event related brain potentials (ERPs) have identified various neuroelectric components that discriminate between Go and NoGo conditions and purportedly reflect activation of distinct functional networks involved in response execution and inhibition (Albert et al., 2013; Bekker et al., 2005; Falkenstein, 2006; Jonkman, 2006; Jonkman et al., 2003, 2007; Kirmizi-Alsan et al., 2006; Lavric et al., 2004; Nieuwenhuis et al., 2003, 2004; Pandey et al., 2012; Smith et al., 2007, 2008; van Veen and Carter, 2002). The vast majority of studies have implicated N2 and P3 components as reflecting different aspects and temporal features of inhibitory processing in healthy normal subjects as well as reflecting dysfunction in these processes in subjects with several neurological and psychiatric conditions, including alcoholism (Bekker et al., 2005; Falkenstein, 2006; Kamarajan et al., 2004,

* Corresponding author.
*E-mail address:* akp@hbnl.downstate.edu (A.K. Pandey).

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2005a, 2005b; Kirmizi-Alsan et al., 2006; Munro et al., 2007; Nieuwenhuis et al., 2003, 2004; Smith et al., 2006, 2007, 2008).

The ERP components, such as P3 and N2, that are obtained by averaging procedures in the time domain, are not unitary neuroelectric phenomena elicited during cognitive processing, but consist of the simultaneous oscillatory activity of different frequencies that are generated by multiple sources within the brain with contributions from frontal cortex (including anterior cingulate), parietal cortex, and hippocampus (Halgren et al., 1980, 1995a, 1995b; Menon et al., 2001). Averaged sensory evoked potential waveforms have been shown to develop from the phase resetting of the ongoing EEG oscillations with different frequencies as well as modulation of their amplitude (cited from Muller and Anokhin, 2012). Similarly, recent research using advanced methods for time-frequency analysis of event-related oscillations (EROs) has provided evidence that ERP waveforms can be at least partially accounted for by phase resetting of EEG oscillations (Freunberger et al., 2007, 2008; Gruber et al., 2005; Klimesch et al., 2004, 2007; Sauseng and Klimesch, 2008; Yeung et al., 2004, 2007). It has also been demonstrated that averaged ERPs provide limited representation of the underlying event-related neural dynamics, whereas ERO analysis permits the separation of phase and amplitude effects of different frequencies that contribute to the averaged ERP waveform and therefore provide important insights into the neural dynamics underlying the ERP response (Fell et al., 2004; Makeig et al., 2002, 2004; Onton and Makeig, 2006; Roach and Mathalon, 2008). Another advantage of ERO analyses is the potential for transfer of knowledge obtained by the analysis of spontaneous EEG to findings of ERP research and vice versa (e.g., Button et al., 2007), thereby facilitating the understanding of how different cortical networks are integrated in response to an external stimulus and how information can be transferred between such circuits (e.g., Carr et al., 2004), as well as dissociating cognitive processes which were not dissociable by ERPs (e.g., Branchey et al., 1988, see for review Sauseng and Klimesch, 2008).

Different frequency bands in EROs have been attributed to underlie various task-specific cognitive processes. Event-related delta activity is generated by cortico-cortical interactions (Devrim et al., 1999), and is a product of the distributed network system of the brain (Basar-Eroglu et al., 1992; Basar, 1999a,b) involved in mediating attention and task demand, signal detection and decision making (Basar-Eroglu et al., 1992; Basar, 1999a,b, Basar et al., 2001; Schurmann et al., 2001). On the other hand, event-related theta oscillations are related to cortico-hippocampal (Basar, 1999b; Miller, 1991) or fronto-limbic interactions (Karakas et al., 2000), and are associated with a complex set of cognitive processes including alertness, arousal or readiness (Basar, 1999b), episodic encoding and retrieval processes (Klimesch, 1999; Klimesch et al., 1994, 1996a, 1997a, 1997c, 2001), selective attention and short-term memory (Basar-Eroglu et al., 1992; Demiralp and Basar, 1992; Karakas, 1997; Klimesch, 1999), error processing (Luu et al., 2003, 2004; Trujillo and Allen, 2007), reward processing (Cohen et al., 2007; Gehring and Willoughby, 2004; Kamarajan et al., 2008; Marco-Pallares et al., 2008), realizing the need for top-down control (cited from Cavanagh and Frank, 2014; Cavanagh et al., 2012; Itthipuripat et al., 2013; Jacobs et al., 2006), mid-frontal substrate for action monitoring processes (Cavanagh et al., 2012), and response conflict (Cohen and Cavanagh, 2011). Alpha has been reported to be linked with a cessation of activity or inhibition (Knyazev et al., 2006, 2010; Knyazev and Slobodskaya, 2003), and to reflect anticipatory preparedness (Knyazev et al., 2002, 2004a, 2004b, 2005, 2006; Knyazev and Slobodskaya, 2003). The slow alpha (8–10 Hz) has been reported to modulate as a function of attentional demands (Basar and Schurmann, 1997; Basar et al., 1997; Klimesch et al., 1996b, 1997b, 1998), and fast alpha activity (10–12 Hz) has been shown to mediate semantic memory processes as well as stimulus-related aspects (Klimesch, 1996; Klimesch et al., 1994, 1997a, 1997b). Therefore, based on findings in the literature that are related to action/conflict monitoring and response inhibition, assessed by the Go/NoGo task and the functional significance of various frequency bands as mentioned above, analyses in the present study was restricted to frequency bands below 12.5 Hz. Changes in the N1, N2, and P3 ERP components, which probably reflect modulations in low-frequency oscillations (e.g., in the delta-alpha range), have been observed with respect to the Go/NoGo task.

Relatively few studies have examined EROs during a Go/NoGo task. Muller and Anokhin (2012) examined temporal (phase-locking index across trials) (a.k.a. PLI) and spatial (phase coherence across electrode sites) synchrony of neuroelectric activity during a continuous performance Go/NoGo task in university students; they found that both synchrony measures increased in Go and NoGo conditions, followed by warning, and lowest in neutral stimuli. Both measures showed their strongest effects in delta and theta frequency bands, where the Go condition accounted for the increase in the first post-stimulus time interval (0–300 ms) whereas NoGo accounted for the increase during the second post-stimulus time interval (300–600 ms) in the delta band. Although phase synchrony, especially as measured by PLI, was in general highest in centro-parietal sites, it was stronger for the Go compared to the NoGo condition at centro-parietal sites during the first post-stimulus time interval (0–300 ms). On the other hand, PLI for the NoGo condition was stronger compared to the Go condition at frontocentral sites during the second post-stimulus time interval (300–600). Another study using a cued Go/NoGo task found stronger inter-trial coherence (ITC) in the NoGo compared to the Go condition at theta frequency during the time interval between 200 and 600 ms after stimulus onset (Schmiedt-Fehr and Basar-Eroglu, 2011). The results of these studies are suggestive of anteriorization of transient increases in neural synchrony while suppressing a motor response, as well as its relatively later occurrence in mental chronometry compared to executing a motor response. It is to be noted here that measures such as PLI and ITC used in both studies are similar and are the means by which event-locked evoked power for the various frequencies, (the dependent measure used in the present study), are calculated.

With respect to alcoholism, in a study on alcoholics using an equal probability visual Go/NoGo task, Kamarajan et al. (2004) reported lower delta and theta oscillatory activity at frontal regions, particularly during NoGo trials, in abstinent alcoholics compared to controls. The authors also found significantly lower ERO activity in delta, theta, and slow alpha (8–9 Hz) bands during the NoGo condition, as well as lower delta and theta activity during the Go condition in offspring of alcoholic parents (Kamarajan et al., 2006). Other than this study by Kamarajan et al. (2006) on high-risk subjects, ERO studies have mainly examined slow waves such as delta and theta while evaluating performance during a Go/NoGo task. Therefore, considering the activation-inhibition dimension being examined in a Go/NoGo task, along with the previously reported literature regarding the functional significance of various frequency bands, it would be important to examine spectral power of delta, theta, and alpha frequency bands that are time-locked to the execution (Go) and suppression (NoGo) of a motor response as well as their patterns and topographical densities of relative appearances in the mental chronometry.

The goal of the present study was to investigate evoked power density and its topographical distributions of neural oscillatory dynamics underlying response execution and its inhibition using S-transform analysis as well as to assess its differences between controls and alcoholics. Based on the previously reported literature, it was hypothesized that (i) evoked power would increase with increasing cognitive control demand, such that response execution (Go) and suppression (NoGo) involving competing response tendencies (response conflict), and necessitating decision making will be characterized by a higher evoked power, (ii) evoked power density in NoGo trials would be distributed more anteriorly as compared with the Go trials in slow frequency bands, especially delta and theta, based on the evidence regarding the role of the prefrontal cortex in conflict monitoring, decision making, and response inhibition, and (iii) alcoholics would show lower evoked power compared to controls in different frequency bands. This effect

would be substantial in the brain regions that respond to task situations with increased cognitive control demand, such as frontal regions. For this purpose, we derived evoked power of different frequency bands for both task conditions at all electrode locations.

## 2. Methods

### 2.1. Subjects

Twenty right-handed abstinent alcoholic males (mean age = 28.08 ± 3.65 years) and 20 healthy age-matched right-handed male controls (mean age = 27.33 ± 4.49 years) who met the criteria for inclusion were recruited. Initial screening was performed over the telephone for all participants. Control subjects were recruited through newspaper advertisements and did not have any personal and/or family history of major medical, psychiatric, or substance-related disorders. They were instructed to abstain from alcohol and other substances with CNS effects for at least five days prior to the recordings and assessments. The alcoholic subjects (Alcohol Dependence as per DSM-IV criteria) were recruited from alcoholism treatment centers in and around New York City. Before testing, they had been detoxified in a 30-day treatment program and were not in withdrawal. The Bard/Porjesz Adult Alcoholism Battery (BAAB; cited from (Kamarajan et al., 2004)), a semi-structured clinical assessment schedule, was used to obtain the clinical data related to alcohol dependence and alcohol-related medical problems; information about other substances and family history were obtained. Subjects who had a family history of psychiatric disorders in their first degree relatives, as well as those with moderate and severe cognitive deficits based on their score (<21) on the mini mental state examination (MMSE; (Folstein et al., 1975)) were excluded from the study. Subjects who were found to be positive (for recent drug use) on the urine screen and Breathalyzer test as well as those with a history of hallucinogen abuse (e.g., LSD) were excluded from the study to avoid the possible interaction of drugs with the EEG profile. However, given the nature of the disorder, subjects with a history of other substance use and/or ASPD as co-existing conditions and with a past history of CD, ADHD, and oppositional defiant disorder (ODD) were included in the alcoholic group. For both groups, subjects with hearing or visual impairment, liver disease, or head injury were also excluded. Experimental procedures and ethical guidelines were in accordance with approval from the institutional review board (IRB). Alcohol and other drug use information obtained using the BAAB for the period of six months prior to detoxification for the alcoholic group are shown in Table 1 along with similar information for the controls.

### 2.2. Task procedure and measures

The Go/NoGo task used was identical to the task described in a previous study from our lab ((Pandey et al., 2012); see Fig. 1 for schematic illustration). Briefly, each subject was presented with four types of visual stimuli consisting of white isosceles triangles pointing in either the up, down, right, or left direction. The stimuli were presented for 100 ms at the center of a computer screen (17″ diagonal, 75 Hz refresh rate, and 1024 × 768 resolution) against a dark background that subtended a visual angle of approximately 1°.

In the practice session, subjects were instructed to press a key whenever a white triangle pointed either up or down (Go stimulus) and refrain from pressing the key whenever the triangle pointed towards the right or left (NoGo stimulus). A dollar sign ($) appeared on the screen for 200 ms at 1200 ms after stimulus onset when subjects responded correctly whereas a cross sign (X) appeared on the screen for 200 ms at 1200 ms after stimulus onset when subjects responded incorrectly. Subjects were instructed that speed and accuracy were equally important for making a correct response. The EEG was recorded during the experimental phase. In this phase, subjects were informed that each correct response would earn a reward. However, each subject received a predetermined fixed amount at the end of the experiment without deductions for errors, although they were not informed of this while performing the task. The probabilities of occurrence of Go and NoGo stimuli were equal (50/50), and the order of stimulus presentation was randomized. The inter-trial interval was 2400 ms. Reaction time, Go, NoGo errors, and total correct response percentage were obtained and used for the statistical analysis.

The Barratt Impulsiveness Scale (BIS-11; (Patton et al., 1995)) was used to assess the personality/behavioral construct of impulsiveness. It is the most widely cited instrument for the assessment of impulsiveness and has been used to advance our understanding of this construct and its relationship to other clinical phenomena for 50 years (see for review Stanford et al., 2009). Nonplanning, Motor, Attentional, and Total impulsiveness subscales were derived by scoring and used for the statistical analysis.

**Table 1**
Alcohol and drug use profile of alcoholic before detoxification (BAAB) and control subjects for last six months.

| | Alcoholic | | | Control | | |
|---|---|---|---|---|---|---|
| | *N* | Mean | SD | *N* | Mean | SD |
| Alcohol: days/month | 20 | 21.10 | 7.52 | 13 | 2.38 | 1.98 |
| Alcohol: drinks/day | 20 | 9.18 | 6.29 | 13 | 2.00 | 1.00 |
| Tobacco: days/month | 18 | 28.67 | 4.70 | 2 | 8.50 | 9.19 |
| Tobacco: times/day | 18 | 15.17 | 7.57 | 2 | 3.50 | 2.12 |
| Marijuana: times in the last 6 months | 15 | 96.33 | 84.67 | 2 | 8.50 | 9.19 |
| Cocaine times in the last 6 months | 13 | 69.69 | 71.00 | | | |
| Opiate times in the last 6 months | 3 | 60.67 | 103.35 | | | |

SD = standard deviation.

### 2.3. Data acquisition and analysis

#### 2.3.1. Data recording

The subjects were comfortably seated in front of a computer monitor screen placed one meter away in a dimly lit sound-attenuated RF-shielded room (IAC, Industrial Acoustics, The Bronx, NY). The EEG was recorded on a Neuroscan System (Versions 4.1, 4.2, and 4.3; (Neurosoft, Inc., El Paso, TX)) using a 61 channel electrode cap (Electro-cap International, Inc., Eaton, OH) that had electrode placements based on the 10–10 International System (Electrode Position Nomenclature, (Society, 1991); Fig. 2) with the notch filter off. The electrodes were referenced to the tip of the nose and subjects were grounded using an electrode placed on the forehead (frontal midline, 2 cm above nasion). Eye movements were recorded using a supraorbital vertical lead and a horizontal lead on the external canthus of the left eye. Electrode impedance was maintained below 5 kΩ throughout the recording. The continuous EEG signals were recorded marked with all stimulus, response, and feedback event codes at the sampling rates of 512 (16 bit A/D), and 500 Hz (32 bit A/D) depending on the amplifier version, with a band pass filter set at 0.02–100 Hz and were amplified 10,000 times using a set of amplifiers (SynAmps$^2$, Neuroscan, TX).

#### 2.3.2. Data reduction and analysis

All recordings were digitally re-sampled offline at 256 samples per second with 0.3–45 Hz band pass filter in order to control for the DC drifts and AC power artifacts in the waveform. The EEG segments were divided into epochs of 1625 ms (187.5 ms pre- and 1437.5 ms post stimulus). All epochs exceeding ± 100 μV amplitude were automatically excluded from further processing. Each participant's successful trials were averaged based on task conditions and visually inspected using a display program to further eliminate those trials that show evidence of any kind of artifact. Only artifact-free correct trials were considered for the analysis. In order to reduce within subject variability due to the effect of averaging on the computed power values for each participant, a minimum of 15 and a maximum of 25 artifact-free correct trials out of a maximum of 50 trials in each task condition

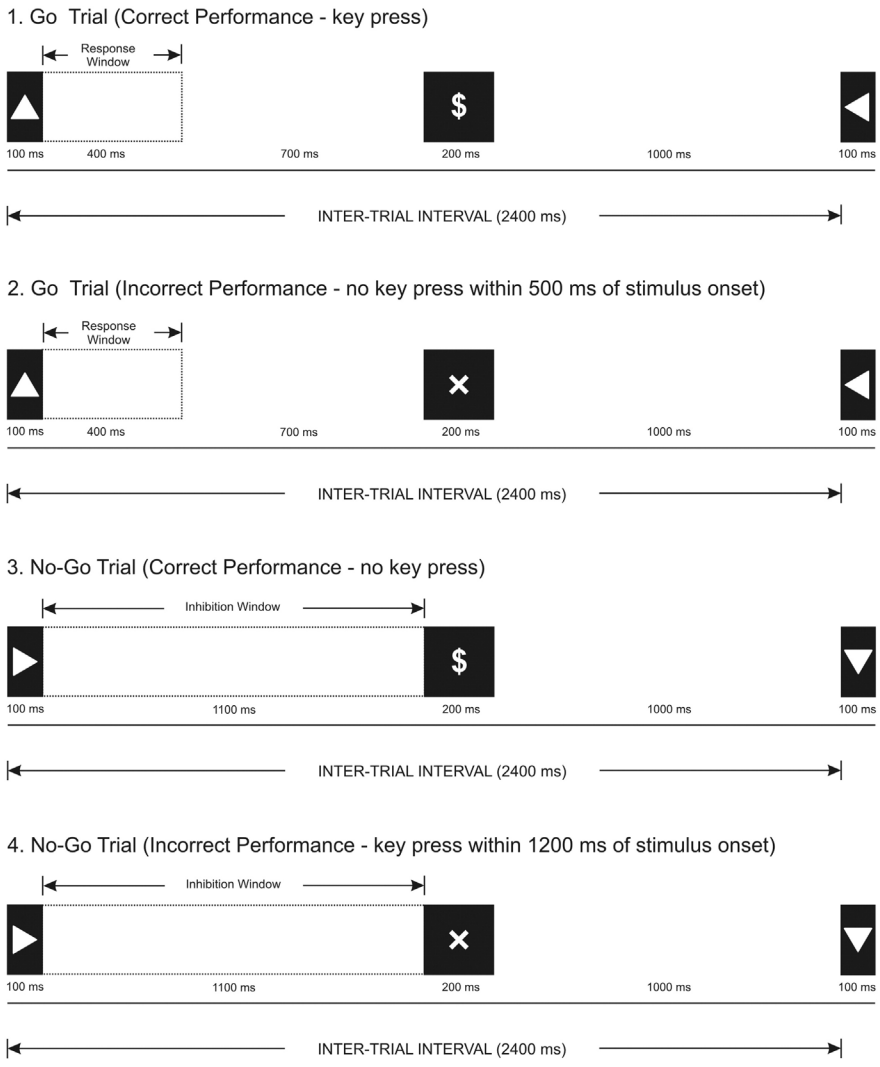


**Fig. 1.** Illustration of Go/NoGo Task showing four different response possibilities.

(Go and NoGo) was taken for further analysis. If there were more than 25 artifact-free correct trials in a condition, 25 trials were selected randomly from the pool. The S-transform method was used to obtain the power values in different frequency bands for each successful trial for both task conditions (Stockwell et al., 1996). The S-transform is a variable window of short-time Fourier transform (STFT) or an extension of wavelet transform (WT). It is based on a scalable localizing Gaussian window and supplies the frequency dependent resolution of a time-frequency domain and entirely refers to local phase information. This method has been shown to produce reliable estimates of localized power of nonstationary evoked potential time series (Chu, 1996; Theophanis and Queen, 2000) and has been applied in several studies from our lab to analyze time-frequency signals of EROs (for details see Chen et al., 2009; Jones et al., 2004, 2006a, 2006b; Kamarajan et al., 2004, 2006, 2008, 2012; Kang et al., 2012; Padmanabhapillai et al., 2006a, 2006b; Rangaswamy et al., 2007).

To compute power values that are time-locked to an event condition, resultant power values in different frequency bands for each trial were then averaged to derive evoked power values. The resultant evoked power values (in $\mu V^2$) were extracted for delta (1.0–3.5 Hz; 200–600 ms), theta (4.0–7.5; 200–400 ms), slow alpha (8.0–9.5 Hz; 200–300 ms), and fast alpha (10.0–12.5 Hz; 100–200 ms) bands for each participant across electrode locations. Power values from 36 region-representative channels (Fig. 2; highlighted channels) were subsequently subjected to statistical analyses. The grand averages of S-transform analyses were computed for the control and alcoholic groups separately for the purpose of illustrating TFR profiles of different frequency bands and their group and condition differences. The TFRs were computed, z-scored, and plotted (1.0–12.5 Hz frequency bands) for the Fz, FCz, Pz, and Oz electrodes for both groups and task conditions (2nd and 3rd columns of Figs. 3b, 4b, 5b, 6b). For the purpose of illustrating group and condition differences in relative localization of the power density of these frequency bands, scalp surface Laplacian measures were computed and plotted for the evoked power of delta (200–600 ms), theta, (200–400 ms), slow alpha (200–300 ms), and fast alpha (100–200 ms) bands for both groups and task conditions (1st and 4th columns of Figs. 3b, 4b, 5b, 6b). The TFR areas used for the computation of scalp surface Laplacian measures for each of these frequency bands are shown within the white dashed rectangular boxes with an arrow indicating the specific frequency that is represented in the plots of the respective figure. The selection of the TFR plot from a specific electrode shown with the surface Laplacian plot of specific frequency bands was made based on the relative regional densities of the frequency bands as well as on previous literature.

### 2.4. Statistical analysis

For statistical comparisons, the electrode sites were grouped into six scalp regions, and six representative electrodes from each region were included in the analysis (see Fig. 2; highlighted channels). The evoked

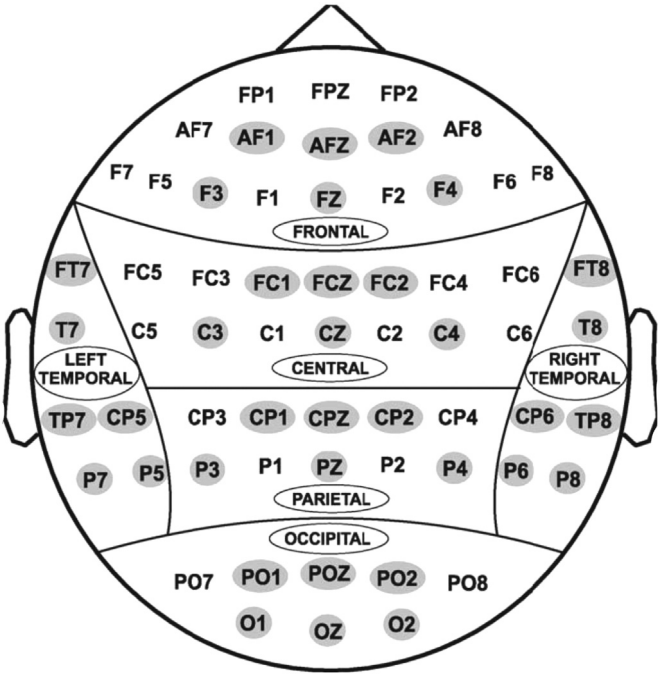


**Fig. 2.** 61 Electrode locations are illustrated and shown according to regional groupings. Electrodes selected for statistical analysis from each group included are highlighted.

power values obtained for the delta, theta, slow alpha, and fast alpha frequency bands were analyzed using a linear mixed-effects model of SAS Proc Mixed Procedure (SAS 9.3, SAS Institute Inc., NC, USA). The mixed-effects model included group (2: controls, alcoholics), condition (2: Go/NoGo), region (6: frontal, central, parietal, occipital, left temporal, and right temporal), electrode (6: representative electrode sites nested within each region) and their interactions as fixed effects, where condition (Go/NoGo) and electrode coordinates ($x$, $y$, $z$) were treated as repeated measures. To determine direct (Kronecker) product structures based on distance between electrodes (i.e., $x$, $y$, $z$), a spatial anisotropic exponential (EXPA) matrix was used to model within subject covariance structure of the data. A backward stepwise method was used to remove insignificant effects. Further exploration of main and interaction effects was performed using Wald's tests (Kenward and Roger, 1997) for pairwise comparisons and the significance levels were adjusted with Bonferroni correction for multiple comparisons. The demographic, cognitive, and behavioral data (i.e., age, MMSE score, BIS-11 scores, reaction time, and error responses) were analyzed using $t$-tests.

## 3. Results

### 3.1. Demographic, cognitive, and behavioral performance

Comparisons between alcoholics and control groups on demographic measures, cognitive, and behavioral performance during the task and scales are shown in Table 2. Significant differences between groups were found for errors made on the Go trials $t$ (22.24) = 2.50, $p < 0.05$, Nonplanning $t$ (38) = 3.45, $p < 0.01$, and Motor $t$ (38) = 3.28, $p < 0.01$, subscales of the BIS-11 scale, as well as Total impulsiveness $t$ (38) = 3.87, $p < 0.001$; alcoholics made more errors on Go trials, and scored higher on Nonplanning and Motor subscales, as well as Total impulsiveness of the BIS-11 scale compared to control subjects. No statistically significant differences were found for age, MMSE, NoGo error, correct response percentage, reaction time, and the attentional impulsiveness subscale of BIS-11, indicating that the groups were comparable on these measures.

### 3.2. Event-related oscillations (EROs)

Statistical analysis yielded significant main and interaction effects (Table 3). For evoked delta (Table 3, 1st column) and theta (Table 3, 2nd column) power, all main and interaction effects, except the three-way interaction i.e., Group × Condition × Region, were found to be highly significant. For evoked slow alpha power, all main and interaction effects except Group × Region and Group × Condition × Region were found to be highly significant (Table 3, 3rd column). For evoked fast alpha power, all main and interaction effects except Group × Region were found to be highly significant (Table 3, 4th column). In order to illustrate and compare results obtained for different frequency bands in light of the significant three-way interaction effect results for the evoked fast alpha power, post hoc analyses of interaction effects of the pairwise differences for all frequency bands were examined, corrected for multiple comparison using Bonferroni correction, and are plotted and described below (refer to plots in Figs. 3a, 4a, 5a, 6a).

#### 3.2.1. Evoked delta power

As evidenced in Fig. 3a, Go evoked delta power was higher overall than NoGo evoked delta power at all except frontal regions in both groups; evoked power density was found to be highest at the parietal region for the Go condition and highest at the central region for the NoGo condition in both groups. Significantly higher evoked delta power was found for the Go compared to the NoGo condition at all but frontal regions in the control group (Fig. 3a, top left panel), and all except frontal and central regions in alcoholics (Fig. 3a, top right panel). The alcoholic group displayed lower evoked delta power compared to the control group at all regions regardless of the condition. This lower evoked delta power in alcoholics compared to controls was statistically significant for the Go condition at all regions (Fig. 3a, bottom left panel) and for the NoGo condition at all except left temporal regions (Fig. 3a, bottom right panel).

The alcoholic group displayed lower power compared to controls in all frequency bands at all time-points for the Go as well as NoGo conditions at the midline parietal location (refer to respective TFR plots in Fig. 3b). Further, 2D surface Laplacian head-maps showed that for the Go condition, the control group had evoked delta power density localized bilaterally in centeroposterior regions whereas in the alcoholics, this density was localized bilaterally at posterior regions. For the NoGo condition, the control group showed localized power density in frontocentral regions whereas the alcoholic group showed this density localized in central regions. Regardless of the topographical similarities and differences shown, the alcoholics displayed much lower and region-restricted density of evoked delta compared to the controls for both Go and NoGo conditions.

#### 3.2.2. Evoked theta power

As evidenced in Fig. 4a, Go evoked theta power was higher overall than NoGo evoked theta at all except frontal regions in the control group, where power density was found to be highest at the central region for the Go and at the frontal region for the NoGo condition. Furthermore, NoGo evoked theta power was higher overall than Go at all regions in the alcoholic group, where power density was found to be highest at the occipital region for both task conditions. However, no significant condition differences were found in either group at any of the six scalp regions (Fig. 4a, top panels). The alcoholic group displayed lower evoked theta power compared to the control group at all regions, regardless of condition; this lower evoked theta power in alcoholics compared to controls was statistically significant for the Go condition at frontal, central, and parietal regions (Fig. 4a, bottom left panel) and for the NoGo condition at frontal and central regions (Fig. 4a, bottom right panel).

As illustrated in Fig. 4b, the alcoholic group displayed lower power compared to controls in all frequency bands at all time-points for the Go as well as NoGo conditions (refer to respective TFR plots) at midline

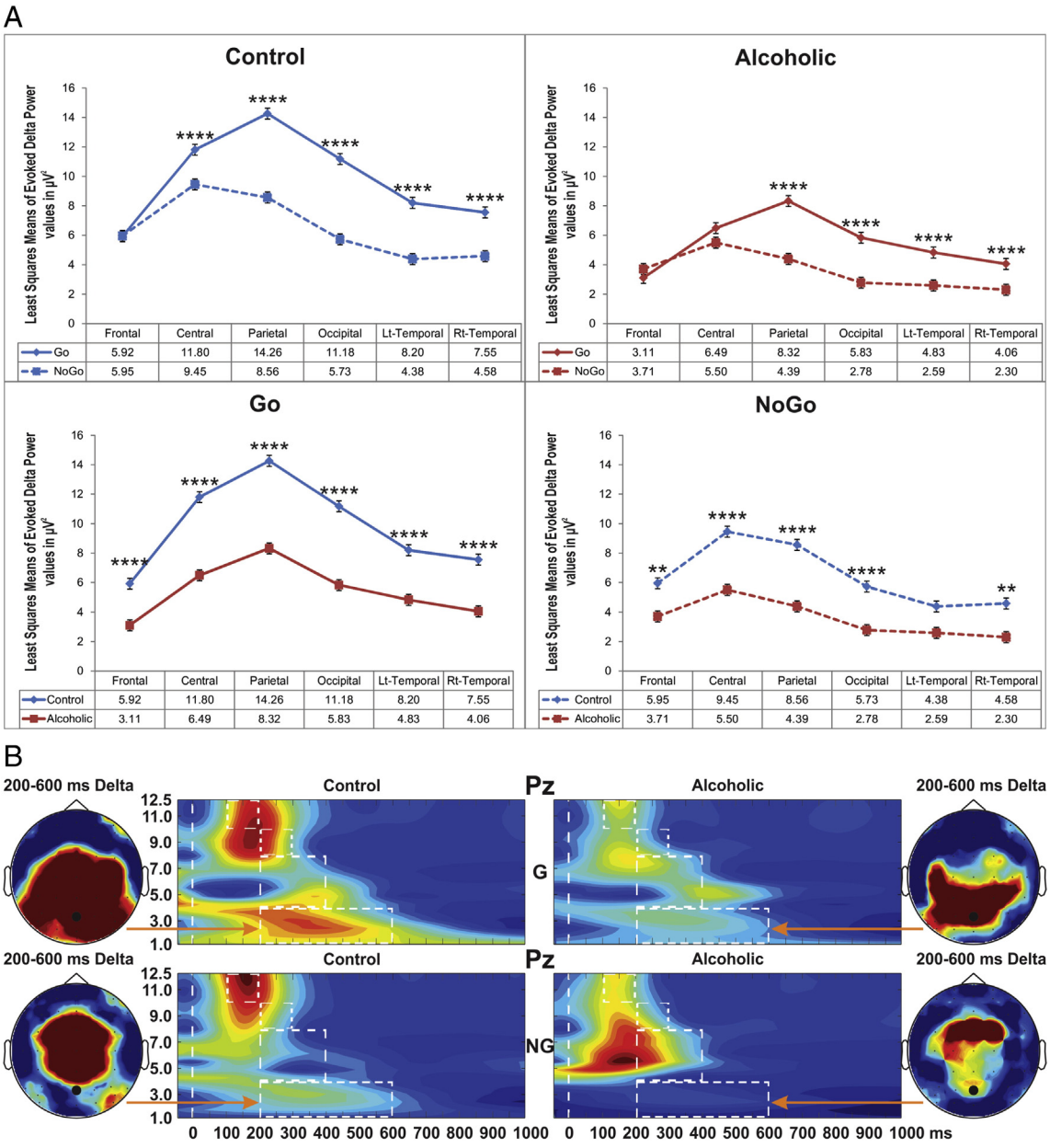


**Fig. 3.** a: Least Squares mean differences between task conditions (top panels) and groups (bottom panels) for evoked delta power and their significance levels for the six scalp regions. Values are in $\mu V^2$. $^{****}p < 0.0001$, $^{**}p < 0.01$. b: Time-frequency representation plots of z-scored mean evoked power values (up to 12.5 Hz) for the control (left) and alcoholic (right) groups and for the Go (top) and NoGo (bottom) task conditions at the Pz scalp location (middle panels; highlighted in surface head plots). The surface Laplacian 2D head plots illustrate localized evoked delta power density within 200–600 ms post-stimulus duration (white dashed box indicated with the arrow) for the control (left) and alcoholic (right) groups and for the Go (G: top) and NoGo (NG: bottom) task conditions. Blue color indicates low and red color indicates high values. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

frontal location. Further, 2D surface Laplacian head-maps showed that although both groups had evoked theta power localized bilaterally at frontocentral as well as bilateral parietotemporal regions of the brain (similar topography), the alcoholics displayed much lower and region-restricted density compared to the controls for both Go and NoGo conditions.

### 3.2.3. Evoked slow alpha power

As evidenced in Fig. 5a, NoGo evoked slow alpha power was higher than Go at all except occipital and right temporal regions in the control group and at all regions in the alcoholic group, where power density was found to be highest at the occipital region in both groups and conditions. However, only the alcoholic group showed significantly higher NoGo evoked slow alpha power compared to the Go condition at frontal and central regions (Fig. 5a, top right panel) whereas the control group did not display condition differences at any of the six regions (Fig. 5a, top right panel). The alcoholic group displayed lower evoked slow alpha power compared to control group at all regions regardless of the task condition. This lower evoked slow alpha power density in alcoholics compared to controls was significant for the Go condition at central and parietal regions (Fig. 5a, bottom right panel) and at central regions for the NoGo condition (Fig. 5a, bottom right panel).

As illustrated in Fig. 5b, the alcoholic group displayed lower power compared to controls in all frequency bands at all time-points for the Go as well as NoGo conditions at midline frontocentral location (refer to respective TFR plots). Further, 2D surface Laplacian head-maps

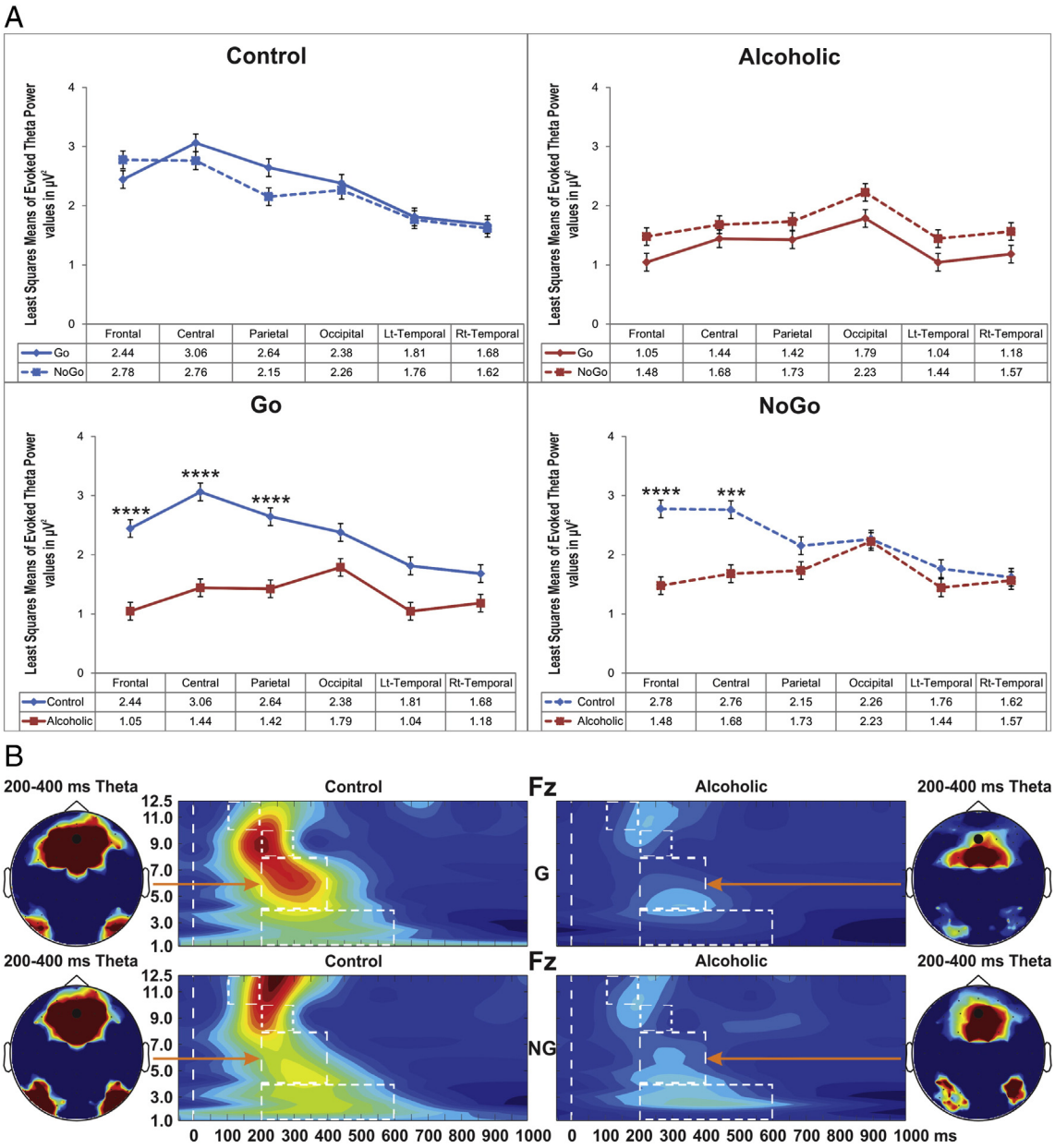


**Fig. 4.** a: Least Squares mean differences between task conditions (top panels) and groups (bottom panels) for evoked theta power and their significance levels for the six scalp regions. Values are in $\mu V^2$. $^{****}p < 0.0001$, $^{***}p < 0.001$. b: Time-frequency representation plots of z-scored mean evoked power values (up to 12.5 Hz) for the control (left) and alcoholic (right) groups and for the Go (top) and NoGo (bottom) task conditions at the Fz scalp location (middle panels; highlighted in surface head plots). The surface Laplacian 2D head plots illustrate localized evoked theta power density within 200–400 ms post-stimulus duration (white dashed box indicated with the arrow) for the control (left) and alcoholic (right) groups and for the Go (G: top) and NoGo (NG: bottom) task conditions. Blue color indicates low and red color indicates high values. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

showed that although both groups had evoked slow alpha power density localized bilaterally at parieto–occipito–temporal regions of the brain, the alcoholics displayed much lower and region-restricted density compared to the controls for both task conditions. In addition, only control group displayed higher density of evoked slow alpha for the NoGo (left bottom panel) compared to the Go (left top panel) task condition at frontocentral midline region. Furthermore, although alcoholic did not show evoked slow alpha density at frontocentral regions for either task condition as illustrated (Fig. 5b; rightmost column), they displayed significantly more evoked slow alpha power compared to the Go condition at frontocentral regions (Fig. 5a top right panel). It is interesting to note that despite higher density of the evoked slow alpha shown posteriorly, group differences on evoked slow alpha power were statistically significant at centroparietal regions for the Go and central regions for the NoGo task conditions.

### 3.2.4. Evoked fast alpha power

As evidenced in Fig. 6a, NoGo evoked fast alpha displayed an overall higher power than Go at all regions, where power density was found to be highest at the occipital region for both groups and conditions. However, only the control group showed significantly higher NoGo evoked fast alpha power compared to the Go condition at parietal, occipital, and right temporal regions (Fig. 6a, top left panel), whereas the alcoholic group did not display condition differences at any of the six regions (Fig. 6a, top right panel). The alcoholic group displayed lower evoked fast alpha power compared to the control group at all regions regardless

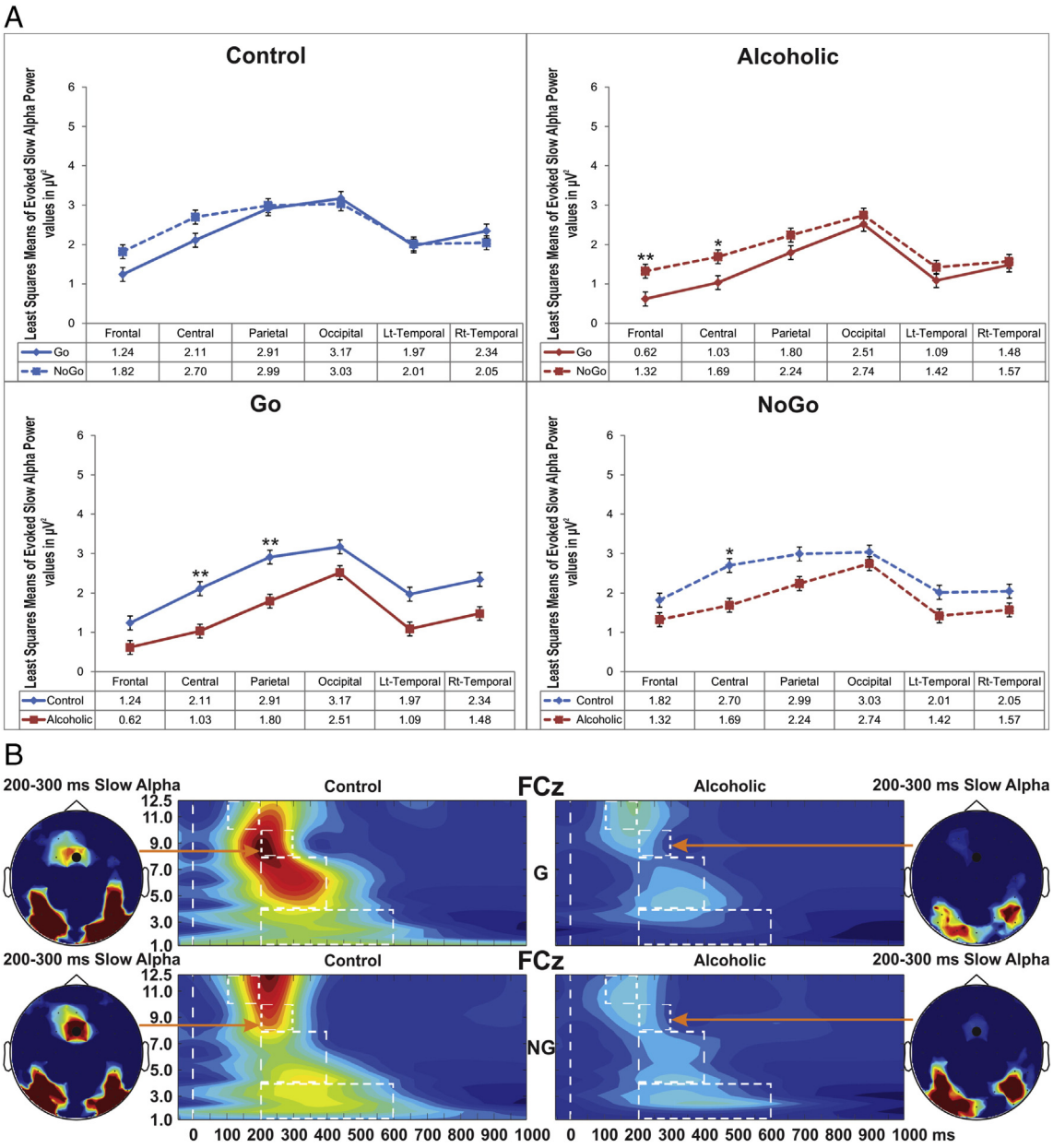


**Fig. 5.** a: Least Squares mean differences between task conditions (top panels) and groups (bottom panels) for evoked slow alpha power and their significance levels for the six scalp regions. Values are in $\mu V^2$. $^{**}p < 0.01$, $^{*}p < 0.05$. b: Time-frequency representation plots of z-scored mean evoked power values (up to 12.5 Hz) for the control (left) and alcoholic (right) groups and for the Go (top) and NoGo (bottom) task conditions at the FCz scalp location (middle panels; highlighted in surface head plots). The surface Laplacian 2D head plots illustrate localized evoked slow alpha power density within 200–300 ms post-stimulus duration (white dashed box indicated with the arrow) for the Control (left) and Alcoholic (right) groups and for the Go (G: top) and NoGo (NG: bottom) task conditions. Blue color indicates low and red color indicates high values. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

of the task condition. However, this lower evoked fast alpha power in alcoholics was significant for only the NoGo condition at parietal, occipital, left-temporal, and right-temporal regions (Fig. 6a, bottom right panel), and not for the Go condition at any of the six regions (Fig. 5a, bottom left panel).

Fig. 6b illustrates that the alcoholic group displayed lower power in all frequency bands at all time-points for the Go as well as NoGo conditions at midline occipital location (refer to respective TFR plots). Further, 2D surface Laplacian head-maps showed that although both groups had evoked fast alpha density localized bilaterally at parieto–occipito–temporal regions of the brain, the alcoholics displayed much lower and region-restricted density compared to the controls for both Go and NoGo conditions.

## 4. Discussion

### 4.1. Main findings

The results of this study may be considered in two different ways: group differences within each condition (hypothesis 3) and condition differences within each group (hypotheses 1 and 2).

Group differences: (1) alcoholics scored higher compared to controls on the Nonplanning and Motor impulsiveness subscales of the BIS-11 scale as well as on the Total impulsiveness, indicating higher impulsiveness in alcoholics, (2) alcoholics committed more omission (Go) errors compared to controls, (3) alcoholics displayed lower evoked delta, theta, slow alpha, and fast alpha frequency band power compared

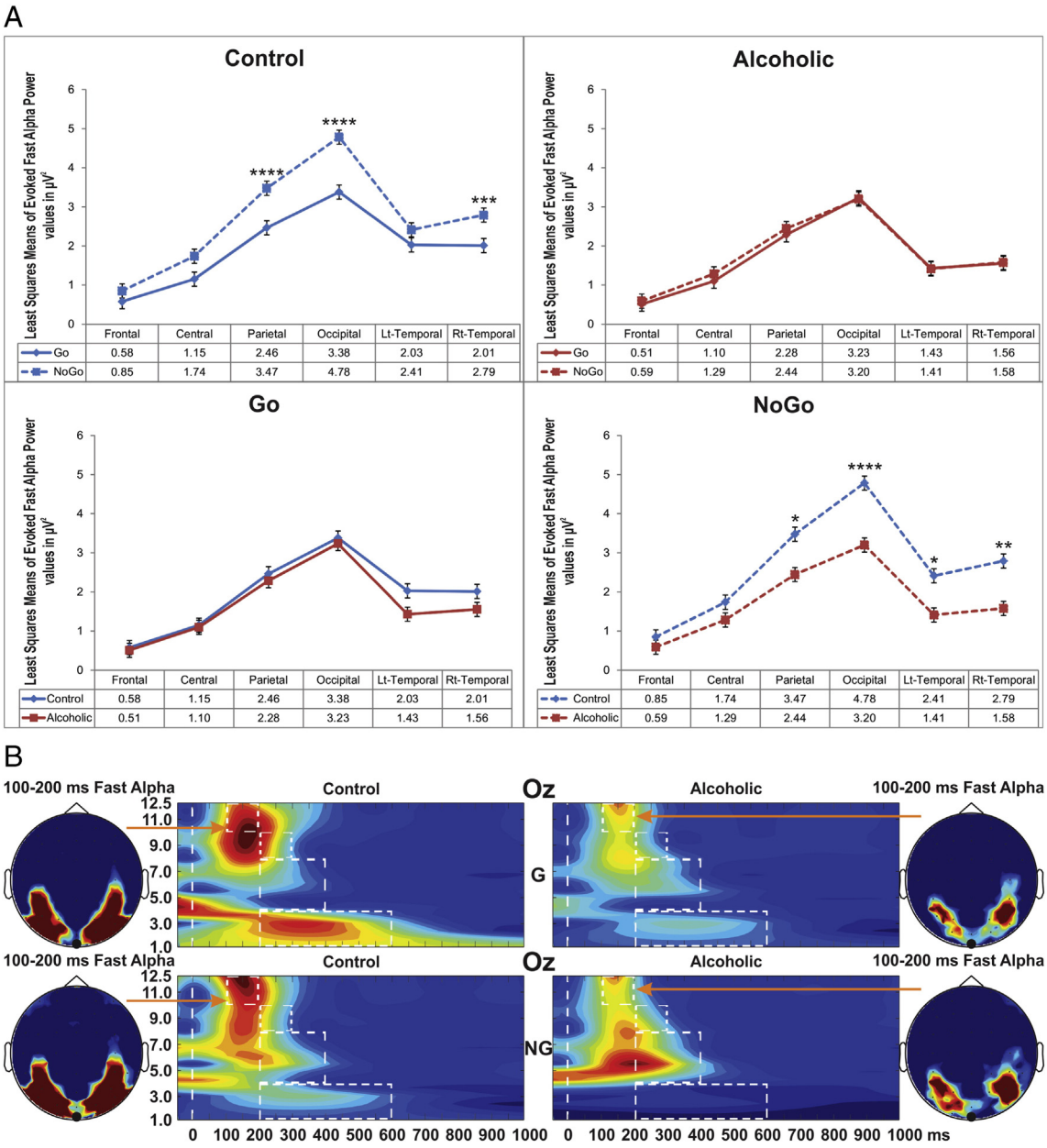


**Fig. 6.** a: Least Squares mean differences between task conditions (top panels) and groups (bottom panels) for evoked fast alpha power and their significance levels for the six scalp regions. Values are in $\mu V^2$. $^{****}p < 0.0001$, $^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$. b: Time-frequency representation plots of z-scored mean evoked power values (up to 12.5 Hz) for the control (left) and alcoholic (right) groups and for the Go (top) and NoGo (bottom) task conditions at the Oz scalp location (middle panels; highlighted in surface head plots). The surface Laplacian 2D head plots illustrate localized evoked fast alpha power density within 100–200 ms post-stimulus duration (white dashed box indicated with the arrow) for the Control (left) and Alcoholic (right) groups and for the Go (G: top) and NoGo (NG: bottom) task conditions. Blue color indicates low and red color indicates high values. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

to controls regardless of condition, indicating lower resource allocation for the activation/inhibition dimension, (4) these findings were highly significant in both Go and NoGo conditions for the evoked delta, theta, and slow alpha power, whereas they were significant in only the NoGo condition for evoked fast alpha power, indicating its specific relative contribution to the suppression of a motor response.

Condition differences: (5) evoked delta power in the Go condition was higher compared to the NoGo condition at all except frontal regions in controls and all except frontocentral regions in alcoholics, indicating its relative contribution to the activation of a motor response, (6) evoked slow alpha power was higher in the NoGo compared to the Go condition in both groups, indicating its relative contribution to the suppression of a motor response; however, this difference was significant only in alcoholics at the frontocentral regions, indicating higher resource allocation in these brain regions during inhibition in alcoholics, (7) evoked fast alpha power was higher in the NoGo compared to the Go condition in both groups, but this difference was significant only in controls at posteriotemporal regions, indicating higher resource allocation in these brain regions during inhibition, (8) both groups had their highest evoked delta power at parietal regions for the Go condition and at central regions for the NoGo condition, indicating relative activation of these respective brain regions while executing and inhibiting a motor response, and (9) the control group had their highest evoked theta power at central regions for the Go condition and at frontal regions for the NoGo condition, indicating relative activation of these brain regions while executing and inhibiting a motor response, whereas the alcoholic group had its highest evoked theta power in both Go and NoGo conditions at the occipital region.

**Table 2**
Mean, SD, df, and t values of demographic, cognitive, and behavioral measures.

| Variable | Alcoholics ($N = 20$) | | Controls ($N = 20$) | | | $t$ value |
|---|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | df | |
| Age (in years) | 28.08 | 3.65 | 27.33 | 4.49 | 38 | 0.58 |
| MMSE | 28.15 | 2.03 | 28.05 | 1.50 | 38 | 0.18 |
| Go error (in %) | 11.51 | 11.18 | 5.00 | 3.28 | 22.24 | 2.50* |
| NoGo error (in %) | 3.60 | 3.15 | 6.80 | 6.57 | 27.32 | −1.97 |
| Correct response (in %) | 92.48 | 5.96 | 94.09 | 3.86 | 32.54 | −1.02 |
| Reaction time | 355.00 | 30.39 | 337.95 | 26.66 | 38 | 1.89 |
| BIS-11: Nonplanning | 25.70 | 4.93 | 20.20 | 5.15 | 38 | 3.45** |
| BIS-11: Motor | 26.00 | 6.27 | 20.60 | 3.87 | 38 | 3.28** |
| BIS-11: Attentional | 16.25 | 2.90 | 14.05 | 3.98 | 38 | 2.00 |
| BIS-11: Total impulsiveness | 67.95 | 11.58 | 54.85 | 9.77 | 38 | 3.87*** |

SD = standard deviation, df = degrees of freedom.
*** $p < 0.001$.
** $p < 0.01$.
* $p < 0.05$.

*4.1.1. Trait impulsivity and inhibitory control differences between groups*

Higher BIS-11 scores in alcoholics confirm previously reported findings of alcoholics having high trait impulsiveness (Chen et al., 2007, 2005). Alcoholics also showed higher omission (Go) errors but did not differ from controls in commission (NoGo) errors as well as on reaction time on the Go trials. There are several studies that have reported higher omission errors (Noel et al., 2007; Pandey et al., 2012; Rubio et al., 2007) and comparable commission errors in alcoholics with equal probability Go/NoGo tasks (Kamarajan et al., 2005a; Karch et al., 2008; Pandey et al., 2012; Rubio et al., 2007), as well as with frequent Go/rare NoGo tasks (Easton et al., 2008; Fallgatter et al., 1998). On the other hand, comparable omission errors (Bjork et al., 2004; Goudriaan et al., 2005; Kamarajan et al., 2005a; Karch et al., 2008) and higher commission errors have been reported in alcoholics using equal probability Go/NoGo tasks (Bjork et al., 2004; Goudriaan et al., 2005; Noel et al., 2007; Thoma et al., 2007) as well as with frequent Go/rare NoGo tasks (Salgado et al., 2009). Similarly, results for reaction time to the Go trials are also mixed (refer to Smith et al., 2014 for a meta-analysis). Therefore, with respect to behavioral performance on Go/NoGo tasks, findings are at best equivocal in the literature. However, based on a meta-analysis, Smith et al. (2014) have concluded that their findings are generally consistent with the view that substance use disorders and addiction-like behavioral disorders are associated with impairments in inhibitory control. On the other hand, the direction of the behavioral performance results on the Go/NoGo task, i.e., high (significant) omission and low (nonsignificant) commission errors, coupled with longer (nonsignificant) reaction time on the Go trials in the present study, suggest general slowness in the suppression as well as execution of a motor response in alcoholics. It is probable that by virtue of the psychomotor slowness, alcoholics had more omission errors, fewer commission errors, and slower reaction time on the Go trials. This general slowness in the activation of goal-directed responses is confirmed in previous studies as well. Ortner et al. (2003) found alcohol intoxication reduced impulsivity during a delayed discounting task and suggested that alcohol intoxication may lead to more cautious decision-making under certain conditions. Similarly, based on their results on alcoholic and problem gamblers, Lawrence et al. (2009) have concluded that inhibitory control is impaired in alcohol dependence but it occurs in the context of psychomotor slowing. In addition, alcoholics failed to show behavioral adjustments following failed stops on a stop signal task. The findings are interpreted as deficits due to the direct effects of chronic alcohol administration on fronto-striatal circuitry.

In a study that addressed the relationship between trait impulsivity and inhibitory control (two features known to be impaired in a number of psychiatric condition, including alcoholism), Aichert et al. (2012) found significant associations of BIS-11 impulsivity with commission errors on a Go/NoGo task and directional error on an anti-saccade task, but not on stop signal and Stroop tasks. Latent variable analysis revealed that 12% of variance of the "prepotent response inhibition" construct could be explained by the BIS-11 impulsivity. However, the magnitude of the associations was small, indicating that while a portion of variance in prepotent response inhibition can be explained by psychometric trait impulsivity, the majority of variance remains unexplained. Thus, these findings suggest that prepotent response inhibition paradigms can account for psychometric trait impulsivity only to a limited extent. In another study, Lijffijt et al. (2004) investigated the association between trait impulsivity in the normal population and inhibitory motor control, as assessed by both the stop task and by a short meta-analysis of three studies. They did not find any difference between high-impulsives and low-impulsives on the speed to stop the response (SSRT). However, the meta-analysis revealed that the high-impulsives are marginally slower in stopping than low-impulsives (effect size = −0.26, $p$ = 0.06). The authors concluded that there is only minor evidence that impulsivity in the common population is associated with poor inhibitory control.

The evidence of low associations between inhibitory performance measures and personality traits, such as impulsivity, may not be surprising (cf. de Wit et al., 2007) given that self-report measures reflect participants' assessment of their cognitive and behavioral styles across different (social) contexts, whereas various response inhibition paradigms measure a specific behavior in the laboratory. Another reason for the failure to observe strong correlations between psychometric trait impulsivity and response inhibition may be the role of decision making: The tendency to make rapid disadvantageous decisions may be argued to be at the core of dysfunctional impulsivity, whereas these cognitive mediation processes may play a lesser role in laboratory-based motor response inhibition tasks.

**Table 3**
Main and interaction effects for the evoked delta, theta, slow alpha, and fast alpha power.

| Effect | df | Delta | Theta | Slow alpha | Fast alpha |
|---|---|---|---|---|---|
| | | F (Sig.) | F (Sig.) | F (Sig.) | F (Sig.) |
| Group | 1.38 | 374.17**** | 101.59**** | 67.09**** | 39.08**** |
| Condition (Go/NoGo) | 1.38 | 670.22**** | 10.44** | 33.77**** | 77.13**** |
| Region | 5.190 | 67.27**** | 11.01**** | 28.43**** | 84.17**** |
| Group × Condition | 1.38 | 52.69**** | 37.99**** | 7.81** | 53.89**** |
| Condition × Region | 5.190 | 55.49**** | 3.13** | 6.69**** | 3.36** |
| Group × Region | 5.190 | 5.81**** | 6.61**** | 0.98 | 1.79 |
| Group × Condition × Region | 5.190 | 1.48 | 1.39 | 0.34 | 3.97** |

df = degrees of freedom.
**** $p < 0.0001$.
** $p < 0.01$.

*4.1.2. ERO differences between groups*

Several studies have examined ERO measures in alcoholism, including evoked, induced, and total power using various tasks and paradigms and found lower power in different frequency bands (Andrew and Fein, 2010; Jones et al., 2006a, 2006b; Kamarajan et al., 2004, 2006, 2008, 2012; Padmanabhapillai et al., 2006a, 2006b; Rangaswamy et al., 2007). Findings of the present study of group differences are largely consistent with the previously reported literature (please refer to points #3–4 under the subsection 4.1 above). In addition, compared to the studies mentioned above, the present study also found lower evoked slow alpha power in both Go and NoGo conditions and lower evoked fast alpha power in only the NoGo condition in alcoholics.

*4.1.3. ERO differences between Go/NoGo task conditions*

The studies mentioned above primarily focused on group differences and did not specifically comment on the task condition differences, wherever applicable. The present study further extends its scope by reporting condition differences (please refer to points #5–7 under subsection 4.1 above) that suggest relative contributions of these frequency bands and their topographical distributions during executing and inhibiting a motor response.

*4.1.4. Topographical differences between groups and conditions in EROs*

With respect to the topographical distribution of evoked power in different frequency bands for both task conditions (please refer to points #8–9 under the subsection 4.1 above), the obtained results are largely in sync with the previous findings, where evoked delta and alpha power have been shown to be posterior phenomena and evoked theta power has been shown to be an anterior phenomenon (Basar-Eroglu and Demiralp, 2001; Brier et al., 2010; Demiralp et al., 1994; Kamarajan et al., 2008, 2012; Klimesch et al., 2005; Pizzagalli et al., 2003; Sauseng et al., 2002, 2005). With respect to the topography based on Go/NoGo task conditions, it is evident that NoGo evoked delta and theta power showed relatively higher density more anteriorly compared to the Go condition. However, evoked delta and theta power in the Go condition was higher overall compared to the NoGo condition.

*4.2. Delta, theta, and alpha EROs and their functional relevance*

There is a wealth of literature that links spontaneous slow frequency oscillations with activation or approach motivational behavior that may be implicated as a driving force behind impulsivity, while alpha oscillations are involved with cessation of activity or inhibition processes (see for a review Knyazev, 2007, 2011; Knyazev et al., 2008). Developmentally, a shift from a preponderance of spontaneous theta oscillations to alpha has been linked with the development of prefrontal inhibitory control in humans (Segalowitz et al., 2010). Consistent with this interpretation, spontaneous slow wave power (e.g., theta) has been found to be higher in children compared to adults as well as in different psychiatric conditions characterized by impulsivity, such as conduct disorder, antisocial behavior, and alcoholism compared with normal controls (Rangaswamy et al., 2003, see for review Segalowitz et al., 2010). By the same token, tonic and phasic slow frequencies, such as theta power, have been interpreted to behave differently with respect to cognitive performance. Phasic theta power increases with increased cognitive performance and decreases with decreasing performance (e.g., Gevins et al., 1998). On the other hand, tonic theta is reported to increase with decreased cognitive activity and decrease with increased cognitive activity (cf. Rangaswamy et al., 2003).

However, unlike children and adolescents, individuals with psychiatric conditions related to impulsiveness, including alcoholism have consistently been found to have lower power of slow waves (time-locked to the events) while performing a behavioral or cognitive task compared to controls (Andrew and Fein, 2010; Jones et al., 2006a, 2006b; Kamarajan et al., 2004, 2006, 2008, 2012; Knyazev et al., 2008; Padmanabhapillai et al., 2006a, 2006b; Rangaswamy et al., 2007). Higher spontaneous slow oscillation power and lower slow ERO power that contributes substantially to different chronological peak components of the ERP may appear at first counterintuitive, in light of the evidence that in addition to additive evoked power superimposed on oscillations (Fell et al., 2004), ERP waveforms can be accounted for by the partial phase resetting of EEG oscillations (Fuentemilla et al., 2006; Min et al., 2007). It thus appears reasonable to conclude that if the power in a spontaneous oscillatory frequency is high, there is a relatively higher probability that partial phase resetting of the frequency would render higher power to the phase-locked oscillatory frequency compared to a spontaneous oscillatory frequency that had lower power. However, there is evidence that points to lower phase locking rates among impulsive university students (Knyazev et al., 2008), schizophrenia patients (Basar-Eroglu et al., 2009), and alcohol preferring rats (Criado and Ehlers, 2010). Further, the diffused brain dysfunction hypothesis that postulates that alcohol causes generalized deficits that involve many structures of the brain, is associated with an abnormal EEG profile, and dysfunction resembles that of premature aging (Glenn et al., 1994; Kamarajan et al., 2004; Newman, 1978; Parsons, 1994; Tivis et al., 1995) may also lend additional support to the present observation showing lower evoked power during task demands. In addition, structural magnetic resonance imaging (MRI) evidence of less white matter integrity and cellular volume loss in various parts of the brain in alcoholics may also help explain the relatively lower additive evoked power contributions, the lower rates of partial phase resetting during ERP generation, and therefore lower evoked power in various ERO frequencies (Kroenke et al., 2014; Le Berre et al., 2014; Mann et al., 2001). Studies that have directly correlated EEG/ERP measures to the structural MRI also lend support to this interpretation. (Valdes-Hernandez et al., 2010; Westlye et al., 2009). Colrain et al. (2011) have reported lower NoGo P3 and delta power in alcoholics and its inverse relation with diffusivity (an MRI measure) in the left and right cingulate bundles and have concluded that this relation provides correlational evidence for a functional role of frontoparietal white matter tracts in inhibitory processing.

*4.3. Integrative interpretation of results with respect to functional significance*

The results of the present study are discussed in view of the hypothesis stated earlier that the evoked power in the respective frequency bands would increase with increased cognitive control demand. Therefore, based on the interpretation that slow oscillations are related to activation and alpha is related to inhibition, or that its desynchronization is related to the preparatory phase of a motor response, lower slow oscillations (i.e., delta and theta) and lower alpha oscillations in alcoholics (especially lower fast alpha power only for the NoGo condition in alcoholics as observed in the present study), suggest deficits in both activation and inhibition of neural circuits underlying the desired/required behavior. This deficient processing was observed at all regions (except left-temporal regions for the NoGo condition) as evident in lower evoked delta power in alcoholics compared to controls for the Go and NoGo conditions; lower evoked theta power in alcoholics compared to controls for the Go condition at fronto-centro-parietal regions, and for the NoGo condition at fronto-central regions, respectively; lower evoked slow alpha power in alcoholics compared to controls for the Go condition at centroparietal regions, and for the NoGo condition at central regions; lower evoked fast alpha power in alcoholics compared to controls only in the NoGo condition at posteriotemporal regions.

Similarly, based on the observed region-specific condition differences in all frequency bands, it may also be concluded that higher evoked delta power, especially in posteriotemporal regions, appears to contribute to the execution (Go) of a motor response (activation) while evoked slow alpha power at frontocentral regions and evoked fast alpha power at posteriotemporal regions appear to contribute to the suppression (NoGo) of a motor response (inhibition). Further, only evoked fast alpha power can reliably be interpreted as differentially reflecting suppression (NoGo) of a motor response based on the specificity of group and condition differences that are specific to the NoGo condition. Therefore, based on regions of significant group (posteriotemporal regions) and condition (posteriotemporal regions) discriminability of evoked fast alpha power within the 100–200 ms time duration, as well as previous studies indicating its functional significance (e.g., Klimesch, 1996; Klimesch et al., 1994, 1997a, 1997b), it is plausible to conclude that NoGo evoked fast alpha activity may reflect the semantic memory processes and/or stimulus related aspects of cognitive inhibitory processing during the suppression of a motor response and present findings indicate towards its deficiency in alcoholics. Further, NoGo evoked slow alpha activity may reflect modulation to

attentional demands during the suppression of a motor response and its deficiency in alcoholics compared to controls at central regions appears to reflect the involvement of brain regions that are thought to be associated with modulation of attentional demands during the suppression of a motor response. It is to be noted that although evidence of task condition based modulation of attention was observed in alcoholics, the involvement of these brain regions was still significantly lower compared to controls.

### 4.4. Conclusion

Therefore, based on the observed topographical contributions of different frequencies, their functional relevance, and their group and condition differences in the current study as well as the previous literature, it may be concluded that alcoholics manifest neurocognitive processing deficits compared to controls that involve both activation and inhibition processes. These deficits are more pronounced in the frontocentral regions for the slower frequency bands (e.g., delta and theta) that have traditionally been found to be associated with the activation aspects of higher order complex cognitive processing involved in decision making and adaptive control of behavior. Furthermore, it appears that only evoked fast alpha power reliably reflects the early attentional component of inhibitory processing (suppression of a motor response for the NoGo condition), as well as its deficits in alcoholics. On the other hand, evoked slow alpha power reflects the relative contributions of the anterior regions while suppressing (inhibition) a motor response.

### 4.5. Limitations and future directions

Although the present study has several strengths, such as carefully age- and gender-matched sample as well as a strong statistical design, future studies based on larger sample sizes are required to strengthen the findings. Due to the small to moderate effect sizes reported in the electrophysiological literature with respect to alcoholism, larger sample sizes would also make it possible to assess the role of smoking and polydrug use contributing to the differences between alcoholics and controls in light of growing strong evidence in the literature about structural brain effects seen in alcoholics, which was beyond the scope of the present study. Furthermore, tasks that are designed to assess specific functional processing could better elucidate the specificity of the cognitive processing deficits as well as the functional relevance of various frequency bands. Given the opportunity presented by oscillation analysis regarding: 1) testing hypotheses with respect to the functional relevance of different frequency bands, and 2) to facilitate the transfer of knowledge between spontaneous oscillations and EROs, more studies that examine spontaneous oscillations and ERO together are needed. Additionally, future research must also include both temporal/local (phase-locked across trials) and spatial (phase-locked across electrode sites) synchrony measures to examine the strength of transient increases in localized (temporal) as well as topographically distributed (spatial) synchronization of various frequency bands and their chronometry, in order to fully understand functional connections within the brain while performing a Go/NoGo task and potential dysfunction in conditions such as alcoholism.

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