# Hazardous alcohol consumption and risk of alcohol dependence present different neurophysiological correlates

Wendy V. Herrera-Morales, Leticia Ramírez-Lugo, Efraín Santiago-Rodríguez, Julián V. Reyes-López, Luis Núñez-Jaramillo

**Introduction.** Hazardous alcohol consumption (HAC) is a pattern of alcohol use that may result in harm for the user and/or for those around them. Prior research has suggested that HAC and alcohol dependence share some neurophysiological features but differ in others.

**Aim.** To determine whether HAC and alcohol dependence presented different neurophysiological correlates.

**Subjects and methods.** Two hundred subjects were screened for HAC or alcohol dependence. A quantitative electroencephalographic analysis of delta, theta, alpha and beta absolute power, relative power and mean frequency in subjects with HAC but not alcohol dependence, subjects with risk of alcohol dependence and controls was performed.

**Results.** One hundred and fourteen subjects met inclusion criteria. The HAC group presented with higher beta absolute power and relative power, as well as a lower beta mean frequency than the control group, while the group with risk of alcohol dependence presented lower delta absolute power than controls.

**Conclusions.** HAC and risk of alcohol dependence present different neurophysiological correlates. There is an important effect of the severity of alcohol dependence on neurophysiological correlates of this condition. Our results support the existence of two different types of behavioral disinhibition.

**Key words.** Alcohol dependence. Behavioral disinhibition. Beta activity. Delta activity. Hazardous alcohol consumption. Quantitative EEG.

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## Introduction

Alcohol is a psychoactive substance with abuse rates among the five top risk factors for disease, disability and death worldwide. A global study revealed that alcohol use disorders (AUDs) represent 9.6% of mental and substance disorders, which in turn are responsible for 7.4% of disability-adjusted life years [1].

The most frequently studied AUD is alcohol dependence, however, hazardous alcohol consumption (HAC), 'a pattern of alcohol consumption that increases the risk of harmful consequences for the user or others', which is not necessarily accompanied by alcohol dependence [2], is another AUD that strongly impacts society. Research has shown that HAC is strongly correlated with alcohol-related injuries [3,4]. HAC is frequent among college students and leads to risky behavior such as drunk-driving and the use of other substances, as well as risky sexual practices [5-7].

Quantitative electroencephalography (qEEG) has been used to study AUDs. qEEG is an affordable and non-invasive technique that allows the study of brain electrical activity both at resting state and associated with cognitive processes [8]. An increase in theta [9], and beta absolute power (AP) [10,11] are the main resting qEEG correlates consistently found in alcohol dependent subjects. Regarding delta frequency band in alcohol dependent patients, there are reports for both increased and decreased delta activity, thus remaining inconclusive at the moment [8]. Different reports have addressed alpha activity in relation with familiar history of alcohol consumption and ethnicity [12,13]. Some reports have described a decrease in alpha activity in alcoholics, but other studies have failed to replicate this result [8].

An increase in beta and theta AP has been implicated as a homeostatic imbalance in cortical excitability [8] and as such, it has been proposed that subjects with a genetic predisposition towards developing alcohol dependence present a homeostatic imbalance leading to disinhibition/hyperexcitability in the central nervous system [8]. Behavioral disinhibition is found not only in alcohol dependence, but also in other disorders such as HAC [14], implying a deficit in impulse control [15].

Prior research on binge drinkers has found deficits in white matter integrity, cortical thickness and neurocognitive markers that are similar to that observed in alcoholic patients, suggesting that binge drinking may be the predecessor to alcoholism [16]. A study addressing basal qEEG activity found that binge drinking is associated with high delta and fast beta (20-35 Hz) activity in the eyes open condition [17].

Alcohol dependence produces important plastic changes within the central nervous system [18] and as such, it is important to rule out alcohol dependence when investigating the neurophysiological correlates of patients with HAC. We have previously reported that subjects with HAC, in the absence of alcohol dependence, present with a decrease in beta mean frequency (MF) in frontal and centro-parietal areas, as well as an increase in beta AP in centro-parietal areas [19] compared to controls. Previous reports of qEEG analyses in alcohol dependent subjects revealed increased beta and theta AP [9,11]. Thus, subjects with HAC present with some similar neurophysiological correlates observed in alcohol dependent subjects (increased beta AP) but differ in others. As mentioned earlier, both HAC and alcohol dependence are associated with behavioral disinhibition. However, alcohol dependence includes neurophysiological mechanisms underlying substance dependence, while HAC does not share this set of mechanisms. Since it has been suggested that beta activity is involved in the regulation of cortical excitability [15,20], it is important to determine whether the decrease in beta MF observed in subjects with HAC [19] represents a neurophysiological feature unique to HAC and not shared with subjects with alcohol dependence. Moreover, since no conclusive results have been obtained on delta and alpha activity in AUDs we will study these frequency bands in HAC and risk of alcohol dependence in the same population.

## Subjects and methods

### Participants

Participants were first year health sciences students and were verbally invited during class to participate in the study. All procedures and data management were carried out in accordance with the Declaration of Helsinki, and the protocol was approved by the Committee of Ethics in Research of the Health Sciences Division of the university. All participants signed an informed consent.

### Clinical evaluation

The Spanish version of the Alcohol Use Disorders Identification Test (AUDIT) was used to assess HAC and risk of alcohol dependence. The AUDIT consists of 10 items that explore the frequency, amount of consumption, negative consequences, and associated psychosocial problems related to alcohol consumption [2]. Different cut-points in the AUDIT have been used in college students, but a study comparing four different alcohol screening tests in adolescents, found that for this age group a cut-point of two was optimal for detecting alcohol problem use and a cut-point of 3 for identifying risk of abuse or dependence [21]. In order to avoid false positives we confirmed the results analyzing specific sections of the AUDIT test. A score of 1 or above on items 2 and 3 met criteria for HAC, while a score over 0 on items 4, 5 and 6 met criteria for risk of alcohol dependence [2]. Subjects rating 3 or more in total AUDIT score, but who did not fulfill the criteria for the section assessing risk of alcohol dependence, were included in the HAC group. Thus, subjects rating positive for HAC presented a total AUDIT score of 2 or more, a score of 1 or above on items 2 and 3, and a score of 0 on items 4, 5 and 6; while subjects rating positive for risk of alcohol dependence presented a total AUDIT score of 3 or more, and a score over 0 on items 4, 5 and 6 of this test. While the AUDIT has been validated in different countries such as México, Brazil and Venezuela [2,22,23], no further clinical evaluation was performed for alcohol dependence in this work, thus subjects rating positive for alcohol dependence are considered as 'at risk'.

All participants were non-smokers. Consumption of other drugs was not determined for the subjects in this study.

Participants completed a self-reported questionnaire (ASRS v. 1.1) to test for attention deficit hyperactivity disorder (ADHD) [24,25], and the Plutchick Suicide Risk test. These instruments were selected based on our previous study [19] and are conditions related to AUDs and present in the population [26-28]. Participants had no current or previous history of neurological illness and were not taking any medication that could affect EEG activity.

### Quantitative electroencephalography

#### *EEG recording*

Subjects were comfortably seated in a dimly lit room, were awake, and had their eyes closed. EEGs were recorded with a 19-channel Medicid Fenix

electroencephalograph. Amplifier was set with a low frequency filter of 0.5 Hz, a high frequency filter of 30 Hz, and a 50/60 Hz notch filter. A sampling frequency of 240 Hz, with a 16-bit resolution was used. The signal was collected through 19 electrodes from the international 10/20 system (FP1, FP2, F7, F8, F3, F4, T3, T4, C3, C4, T5, T6, P3, P4, O1, O2, FZ, CZ and PZ), fitted in an electrode cap, and linked mastoids were used as references. All EEG recordings were rigorously analyzed by a clinical neurophysiologist (E.S.R).

***Quantitative analysis***

Quantitative analysis was performed on EEG segments of at least 2.56 s. The segments were manually selected until summing to at least one minute. Only segments during which the subject was awake and presented alpha activity were included. Segments with muscular activity and blinking were also eliminated.

Quantitative EEG analysis software was used to calculate the AP, relative power (RP) and MF for delta (1.56-3.52 Hz), theta (3.91-7.42 Hz), alpha (7.81-12.5 Hz) and beta (12.89-19.14 Hz) frequency bands [29] using a fast Fourier transform algorithm. In order to decrease non-physiological variability, subtraction of the global scale factor (GSF) was applied to AP measures in all frequency bands. The mathematical model used to obtain GSF can be summarized as a logarithmic transformation applied to the brain electrical signal. This equation is $V_i\,(e,t) = \gamma_1\beta_1\,(e,t)$, where the brain electrical activity obtained in the EEG may be represented by a matrix where $V\,(e,t)$, $V$ being the potential recorded at electrode $e$ at the moment $t$. A global factor scale is represented by $\gamma$. The mathematical model used to obtain it is described in detail in the work published by Hernández et al [30]. The subtraction of GSF has been incorporated into some EEG software analysis, such as the one used in this study, and many papers have been published since then. EEG recordings were reformatted to the average reference for quantitative analysis [31].

## Statistical analysis

For the analysis of AUDIT, suicide risk and ADHD raw scores, as well as age, we performed ANOVA's tests followed by Bonferroni's post hoc test.

For qEEG analysis a mixed design analysis of variance (mixed ANOVA) with electrode as a within-subject variable and group as between-subject variable was performed. Greenhouse-Geisser corrected degrees of freedom were used to assess the significance of the corresponding *F*-value when necessary. Partial eta squared ($\eta^2_p$) was used to determine effect size. The AP, RP and MF for delta, theta, alpha and beta frequency bands were analyzed through separated mixed ANOVAs. Where an effect of group was found, pairwise comparison revealed specific inter-group differences, and an ANOVA test was carried out to determine in which electrodes this effect was found. *P*-values were adjusted for multiple comparisons using a Bonferroni correction (Table I).

**Table I.** Outcomes of mixed ANOVAs performed for absolute power, relative power and mean frequency values of delta, theta, alpha and beta frequency bands. Specification of the groups differing is indicated in those measures where an effect of group was found.

| | Absolute power | Relative power | Mean frequency |
|---|---|---|---|
| Delta | DEP < control ($p < 0.05$) | Non-significant | Non-significant |
| Theta | Non-significant | Non-significant | Non-significant |
| Alpha | Non-significant | Non-significant | Non-significant |
| Beta | HAC > control ($p < 0.01$) | HAC > control ($p < 0.01$) | HAC < control ($p < 0.05$) |

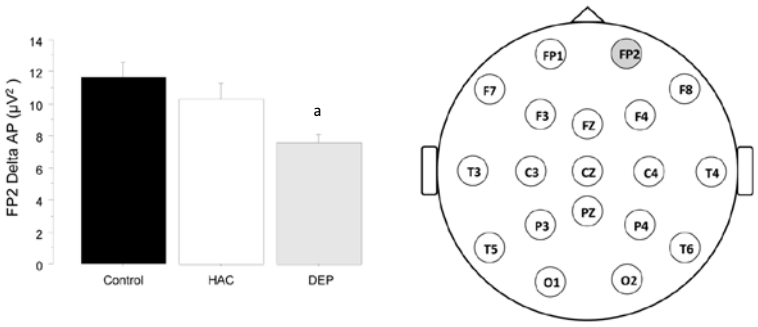
DEP: risk of alcohol dependence; HAC: hazardous alcohol consumption.

## Results

A total of 200 students participated in the study (106 females, age 19.6 ± 0.25, and 94 males, age 19.8 ± 0.35). Subjects older than 22 years, with a present or previous history of neurological disorders, and those in which EEG visual analysis revealed abnormalities were excluded from the analysis.

Only 114 subjects were included in the statistical analysis. Of the 114 subjects, 48 subjects scored negatively for all clinical conditions evaluated (HAC, risk of alcohol dependence, suicide risk and ADHD) and were included in the control group; 45 subjects scored positive for HAC but negatively for risk of alcohol dependence, as well as negative for all other tests (suicide risk, ADHD) and were included in the HAC group; and 21 subjects scored positively for risk of alcohol dependence and were included in the risk of alcohol dependence group (DEP). The majority of subjects within the DEP group presented comorbidities; 18 rated positive for HAC, 6 rated positive for suicide risk and 8 rated positive for ADHD. Age and gender composition of each group is depicted in table II.

**Figure 1.** Site where ANOVA revealed an effect of group on delta AP (mean ± standard error). Differences revealed by Bonferroni post hoc test are shown. [a] $p < 0.05$ (DEP vs control). Electrode in grey indicate lower value in DEP when compared with controls. HAC: hazardous alcohol consumption; DEP: risk of alcohol dependence.




There was no difference in age between groups. ANOVA performed on raw scores for the tests applied revealed a difference in total scores for ADHD ($F_{2,111}$ = 11.59; $p < 0.0001$) and suicide risk ($F_{2,111}$ = 16.49; $p < 0.0001$) tests. For suicide risk, DEP group presented higher scores than control (DEP 4.434 ± 0.6; control 2.0 ± 0.17; $p < 0.0001$) and HAC (2.33 ± 0.21; $p < 0.0001$) groups, while in the ADHD test DEP group scored higher than control (DEP 2.67 ± 0.37; control 1.39 ± 0.16; $p < 0.01$) and HAC (0.18 ± 0.15; $p < 0.0001$). The three groups also differed in AUDIT total score ($F_{2,111}$ = 57.11; $p < 0.0001$), as well as in the score analyzed for HAC ($F_{2,111}$ = 240.66; $p < 0.0001$) and DEP ($F_{2,111}$ = 70.86; $p < 0.0001$). In AUDIT total score DEP group presented higher scores than HAC (DEP 5.24 ± 0.62; HAC 2.89 ± 0.33; $p < 0.0001$) and control (control 0.208 ± 0.09; $p < 0.0001$) groups, while HAC presented a higher score than control group ($p < 0.0001$). In the AUDIT section analyzed for HAC, Bonferroni's post hoc test revealed that control group presented lower scores than HAC (control 0.0 ± 0.0; HAC 1.11 ± 0.47; $p < 0.0001$) and DEP (DEP 0.86 ± 0.078; $p < 0.0001$) groups, while DEP and HAC also differed from each other ($p < 0.01$). In the section evaluated to determine risk of alcohol dependence, Bonferroni's post hoc test showed that DEP group presented higher scores than HAC (DEP 1.48 ± 0.26; HAC 0.0 ± 0.0; $p < 0.0001$) and control (control 0.0 ± 0.0; $p < 0.0001$) groups, while no difference was found between control and HAC groups.

## Delta activity

There was an effect of the electrode studied on delta AP ($F_{4.71,\ 522.35}$ = 57.98; $p < 0.001$; $\eta^2_p$ = 0.343), as well as an effect of group ($F_{2,\ 111\ =}$ 3.25; $p < 0.05$; $\eta^2_p$ = 0.055). Pairwise comparison revealed a difference between control and DEP groups ($p < 0.05$). ANOVA revealed an effect of group at Fp2 ($F_{2,11}$ = 3.12; $p < 0.05$) (Fig. 1).

There was an effect of electrode on delta RP ($F_{7.45,\ 827.84}$ = 153.91; $p < 0.001$; $\eta^2_p$ = 0.581) and MF ($F_{10.67,\ 1185.15}$ = 28.83; $p < 0.001$; $\eta^2_p$ = 0.206). No effect of group or electrode × group interaction was found for these measures.

**Table II.** Age and gender composition of control, hazardous alcohol consumption (HAC) and risk of alcohol dependence (DEP) groups.

| | Female (mean age) | Male (mean age) |
|---|---|---|
| Control | 26 (19.15 ± 0.21 years) | 22 (19.08 ± 0.13 years) |
| HAC | 17 (19.11 ± 0.21 years) | 28 (19.30 ± 0.19 years) |
| DEP | 16 (19.05 ± 0.23 years) | 5 (18.90 ± 0.18 years) |

## Theta activity

There was an effect of the electrode on theta AP ($F_{5.08,\ 563.4}$ = 50.99; $p < 0.001$; $\eta^2_p$ = 0.315), RP ($F_{8.6,\ 954.38}$ = 92.58; $p < 0.001$; $\eta^2_p$ = 0.455) and MF ($F_{8.97,\ 995.58}$ = 32.95; $p < 0.001$; $\eta^2_p$ = 0.229). There was no effect of group or electrode × group interaction on any measure of theta activity.

## Alpha activity

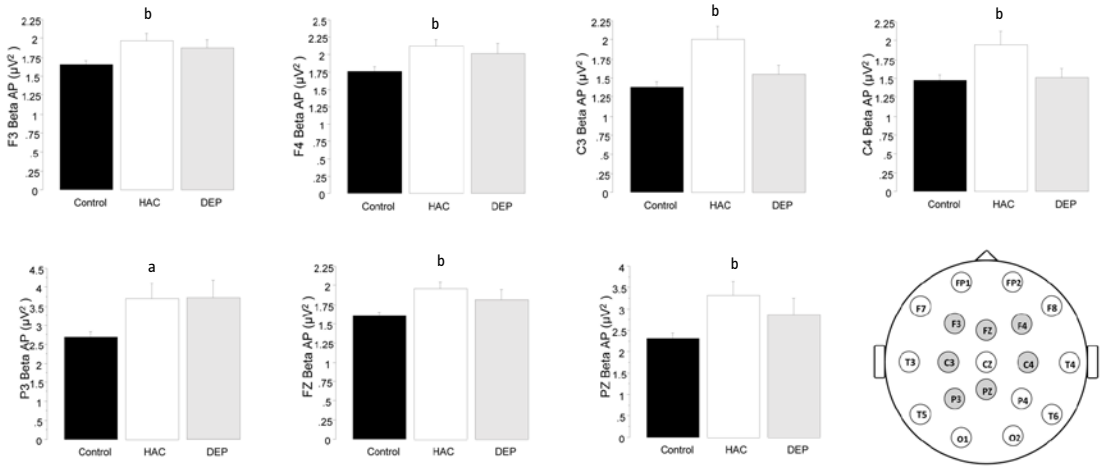
Mixed ANOVA revealed an effect of electrode on alpha AP ($F_{3.73,\ 374.42}$ = 90.35; $p < 0.001$; $\eta^2_p$ = 0.449), RP ($F_{70.9,\ 787.19}$ = 149.73; $p < 0.001$; $\eta^2_p$ = 0.574) and MF ($F_{7.72,\ 856.61}$ = 28.39; $p < 0.001$; $\eta^2_p$ = 0.204). However, no effect of group or electrode × group interaction was found for any of them.

## Beta activity

Mixed ANOVA on beta AP values revealed an effect of electrode ($F_{6.89,\ 764.42}$ = 60.59; $p < 0.001$; $\eta^2_p$ = 0.353) and an effect of group ($F_{2,111}$ = 5.81; $p$ = 0.004; $\eta^2_p$ = 0.095). There was no electrode × group interaction. Pairwise comparison revealed a difference between control and HAC groups ($p$ = 0.003). Figure 2 shows the sites in which a difference between these groups was found.

There was an effect of electrode ($F_{60.3,\ 669.2}$ = 29.78; $p < 0.001$; $\eta^2_p$ = 0.212) and an effect of group ($F_{2,111}$

**Figure 2.** Sites where ANOVA revealed an effect of group on beta AP (mean ± standard error). Differences revealed by Bonferroni post hoc test are shown. [a] $p < 0.05$; [b] $p < 0.01$. Electrodes in grey indicate higher values in HAC when compared with controls. HAC: hazardous alcohol consumption; DEP: risk of alcohol dependence.




= 6.08; $p = 0.003$; $\eta^2_p = 0.099$) on beta RP. There was no electrode × group interaction. Pairwise comparison showed a difference between control and HAC groups ($p = 0.002$). Figure 3 shows the sites in which a difference between these groups was found.

There was an effect of electrode ($F_{8.18,\ 908.19} = 27.34$; $p < 0.001$, $\eta^2_p = 0.198$) and an effect of group ($F_{2,111} = 4.26$; $p = 0.017$; $\eta^2_p = 0.071$) on beta MF. However, there was no electrode × group interaction. Pairwise comparison showed a difference between control and HAC groups ($p = 0.004$). Figure 4 shows the sites in which a difference between these groups was found.

## Discussion

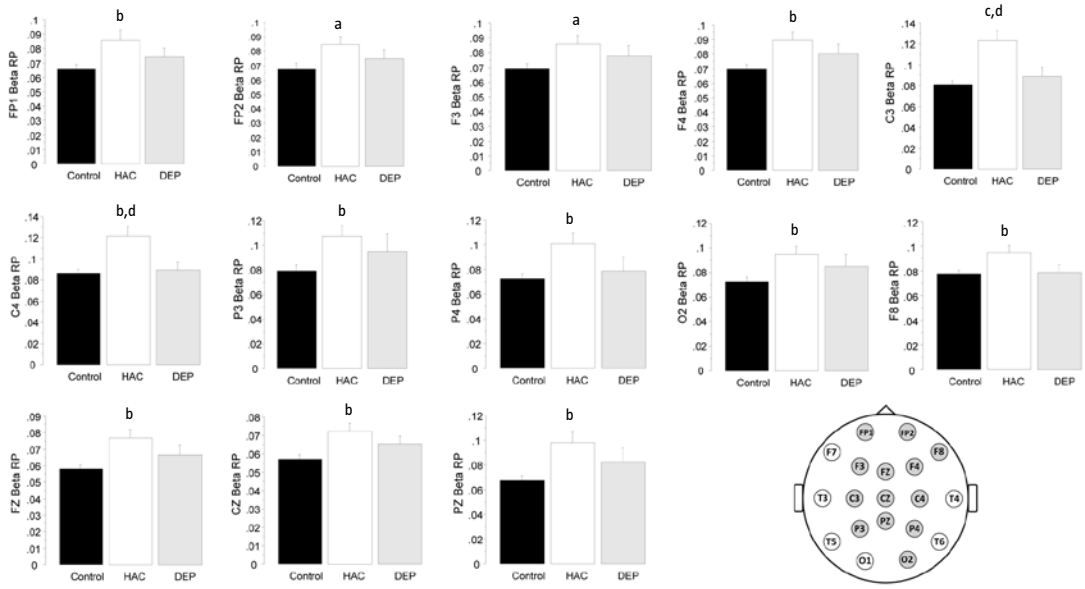
The current study explored qEEG activity between subjects with HAC and risk of alcohol dependence along with a control population without an alcohol-related disorder. Subjects with HAC presented higher beta AP, higher beta RP, and lower beta MF when compared with controls. Subjects with risk of alcohol dependence presented lower delta AP when compared with controls.

Beta activity has been linked with activity of cortical GABAergic interneurons, and as such has been related to inhibition/excitability homeostasis in the cortex. Changes in beta activity have therefore been associated with a homeostatic imbalance leading to central nervous system disinhibition/hyperexcitability [8], and this association has been supported by electrophysiological and genetic analyses [15]. Both HAC and alcohol dependence are manifestations of behavioral disinhibition [8,14,15], and prior research has proposed that behavioral and central nervous system disinhibition share common neurophysiological markers [8,15]. Thus, the higher beta AP and RP, as well as the lower beta MF present in the HAC group suggest an imbalance in central nervous system disinhibition/hyperexcitability.

As mentioned earlier, available data on delta activity in alcoholics is non-conclusive, while earlier works point to an increase in delta activity, more recent works reveal an decrease in delta activity in these patients [8]. A very interesting study performed on alcohol dependent subjects identified two different profiles within the same population based on qEEG analysis, one with increased delta and theta activity, and another with decreased delta and theta activity. Both groups presented a delay in P300 and impairments in attention and memory, although these effects were stronger in the first group (increased delta and theta activity). Authors suggest that this could be reflecting different stages of alcohol dependence [32], although other factors (different types of alcohol dependence, ethnic influence, etc.) cannot be discarded.

Thus, decreased delta AP is present only in some patients with alcohol dependence. We believe that

**Figure 3.** Sites where ANOVA revealed an effect of group on beta RP (mean ± standard error). Differences revealed by Bonferroni post hoc test are shown. [a] $p < 0.05$; [b] $p < 0.01$; [c] $p < 0.0001$ (HAC vs control); [d] $p < 0.01$ (HAC vs DEP). Electrodes in grey indicate higher values in HAC when compared with controls. HAC: hazardous alcohol consumption; DEP: risk of alcohol dependence.

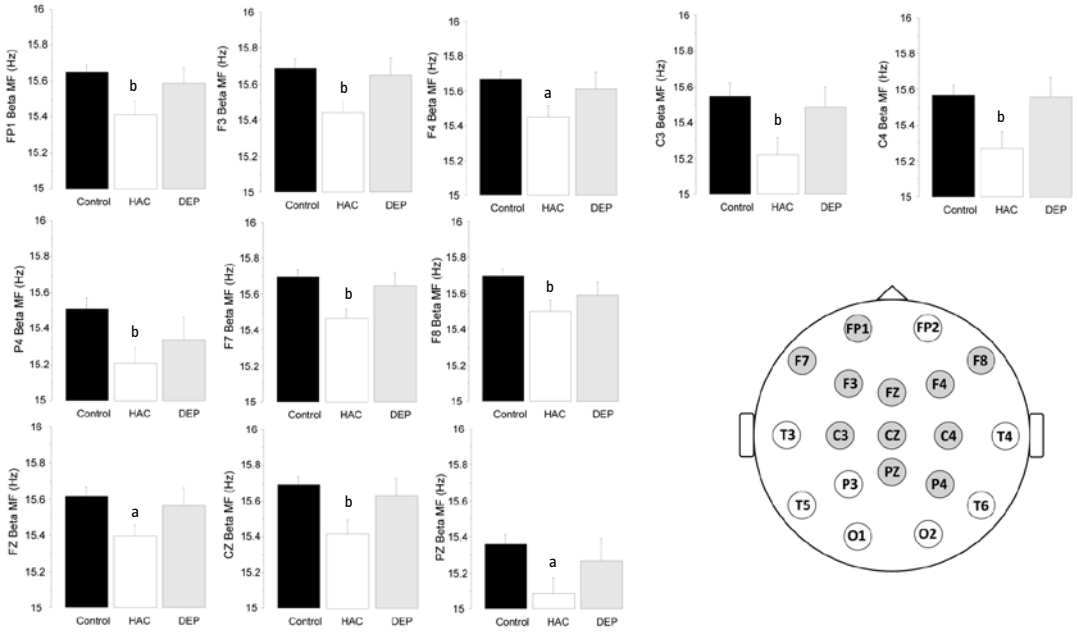



this might represent variations in the type of alcohol dependence. One of the studies showing a decrease in delta activity in alcohol dependent patients shed some light about the possible relevance of this finding, since decreased slow-wave activity (decreased delta and theta activity) correlated with cortical atrophy and onset age of alcohol consumption below 20 years [33]. While cortical atrophy and decreased delta activity might be the result of prolonged alcohol abuse, it might also be possible that decreased delta activity be found primarily in subjects beginning alcohol consumption at an early age, which would also be the case of our study. The implications of this requires further studies characterizing the two subtypes of alcohol dependent subjects, since variation in delta activity probably is not the only neurophysiological difference.

In this work we found statistically significant differences revealed by the ANOVA, but small effect sizes. It is noteworthy that effect size and statistical significance serve different purposes, while statistical significance test reveals the probability of obtaining the between groups difference by chance, effect size provides information about the magnitude of that difference and its possible practical implications [34]. Different works have assessed the importance of small changes on qEEG activity. For example, carbamazepine treatment produces impairment in neuropsychological performance in those patients whose alpha mean frequency in the occipital region slows 0.5-0.6 Hz after treatment, and has been used to determine possible outcome of different antiepileptic drugs [35]. Even though effect size is not always reported along with statistical significance in qEEG studies, previous reports have revealed important findings in studies of alcohol use disorders with small (but significant) differences between groups in different qEEG parameters such as P300 [32,36,37], AP [38] and event related oscillations [39,40].

Gender composition was not equal in all groups, particularly in the DEP group we had a small number of males. Previous reports addressing possible gender differences in neurophysiological correlates of alcohol dependence have found that males present with higher theta AP at central and parietal regions, while females present with higher theta AP at parietal regions only [9]. Another study found that both males and females with alcohol dependence had an increase in beta AP, although for fe-

**Figure 4.** Sites where ANOVA revealed an effect of group on beta MF (mean ± standard error). Differences revealed by Bonferroni post hoc test are shown. [a] $p < 0.05$; [b] $p < 0.01$ (HAC vs control). Electrodes in grey indicate lower values in HAC when compared with controls. HAC: hazardous alcohol consumption; DEP: risk of alcohol dependence.




males this increase did not reach statistical significance [11]. However, a more recent study performed on only women with alcohol use disorders found that alcohol dependent women presented a significant increase in beta AP and RP [10]. Thus, previous studies addressing gender influence on the neurophysiological correlates of alcohol use disorders suggest a mild influence, mostly in regard to the particular distribution of qEEG changes in both genders. However, further studies are warranted to fully confirm the precise influence of gender.

The majority of studies on the neurophysiological correlates of alcohol dependence to date have been carried out in adult populations of medically treated alcohol dependent subjects, which by definition have a more severe alcoholism than the population in the current study. However, studies performed with adult treatment-naïve alcohol dependent subjects have revealed a mild non-significant ($p = 0.05$) decrease in P300 [37], and treatment-naïve adolescents with alcohol dependence do not present any reduction in P300 [41], while studies on adult treated population revealed a significant decrease in P300 amplitude [8]. P300 signifies brain activity during cognitive functioning, and as such, it is a very different type of neurophysiological marker than the resting EEG activity reported herein. However, taken together the results allow us to have a better understanding of the differences in brain electrical activity in subjects with early and more severe alcohol dependence.

Although the subjects in the current study presented with alcohol use disorders, they had not yet sought medical treatment, and therefore, it is not surprising that our neurophysiological results differ from those that studied participants with a more severe alcoholism who had sought treatment or were already in treatment [37,41]. The results from resting qEEGs of subjects with risk of alcohol dependence in the current study were inconsistent with features previously described in alcoholic patients, namely, increased theta and beta AP [9-11]. The participants in the current study were young (age 17 to 22 years old) and treatment-naïve, meaning that their alcohol use disorder was not yet severe enough to seek for medical help. All subjects were first-year university students, and attended classes and other academic activities on a regular basis. Therefore, the risk of alcohol dependence had not yet caused a serious impairment in daily activi-

ties, suggesting an early stage of alcohol addiction [42]. Thus, we believe that a low addiction severity may account for the lack of differences in theta and beta activity between subjects with risk of alcohol dependence and control subjects in the current study. However, we found a lower delta AP, significant at Fp2, in the group with risk of alcohol dependence when compared versus controls, indicating a differential qEEG activity associated with risk of alcohol dependence. Moreover, in this work we did not analyze other EEG parameters reported to be altered in association with alcohol dependence such as event related potentials, thus differences in these measures cannot be ruled out.

Despite the fact that most of the subjects with risk of alcohol dependence in the current study also presented with HAC (82.6%); high beta AP and RP, as well as low beta MF, were found only in subjects with HAC, but not alcohol dependence. One possible interpretation suggests the existence of a different etiology for HAC in the two groups (HAC only and DEP).

There is some controversy on whether HAC is a prelude of alcohol dependence or if HAC and alcohol dependence are two separate conditions [16]. If these two conditions are not linked, then alcohol dependence is not a 'higher level' of HAC, allowing the existence of neurophysiological correlates exclusive to HAC, as may be the case for the lower beta MF. However, the question remains as to why differences in neurophysiological correlates differ between the HAC and DEP groups when most of the DEP group also presents with HAC.

HAC can be present in an individual with or without alcohol dependence [2] since alcohol dependence includes factors associated with addiction. The possibility exists that HAC that is associated with alcohol dependence has a different etiology than HAC not associated with addiction. Interestingly, a previous study among cocaine abusers with and without comorbid personality disorders, found that subjects with cocaine dependence and comorbid personality disorders reported higher behavioral disinhibition than those without comorbid personality disorders and this disinhibition was present prior to the development of cocaine dependence. Comparatively, while cocaine dependent subjects without comorbidities also presented increased disinhibition, this behavior developed along with cocaine dependence, suggesting a difference in disinhibition trajectory between these two groups [43]. These results suggest the existence of at least two subtypes of disinhibition: one resulting from the development of addiction and another that precedes addictive behavior. The current results suggest that these two types of disinhibition may have different neurophysiological correlates. Applied to our results, this theory implies that HAC observed in individuals in the DEP group would be secondary to an addiction process, while in the HAC group this condition would not be related with the development of alcohol dependence.

While HAC and DEP are manifestation of behavioral disinhibition [8,14,15], it is reasonable that they differ in their neurophysiological correlates, since alcohol dependence implies neurophysiological mechanisms associated with addiction. However, if higher beta activity is related with cortical and behavioral disinhibition [8], why is it present only in the HAC group, and not in the DEP group? In the work of Albein-Urios et al [43] they report two different types of disinhibition, one preceding addiction and the other developing along with addiction. We believe that our subjects in the HAC group have the first type and our subjects in the DEP group the second. According to this, and given that we are observing the first stages of alcohol dependence, the increase in beta activity associated with disinhibition would be under development in these subjects. In agreement with this proposal we can observe that, although DEP group does not differ from controls in beta AP, RP and MF, it does not differ from the HAC group either in most of the cases observed in this study. However, further studies on subjects with more advanced alcohol dependence are necessary in order to prove this idea.

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## El consumo de riesgo de alcohol y el riesgo de dependencia al alcohol presentan correlatos neurofisiológicos diferentes

**Introducción.** El consumo de riesgo de alcohol (CRA) es un patrón de consumo que puede resultar dañino para el usuario o para los demás. Investigaciones previas sugieren que el CRA y la dependencia al alcohol comparten algunas características neurofisiológicas, pero difieren en otras.

**Objetivo.** Determinar si el CRA y la dependencia al alcohol presentan correlatos neurofisiológicos diferentes.

**Sujetos y métodos.** Doscientos sujetos realizaron la prueba de detección de CRA y riesgo de dependencia al alcohol (DEP). Se realizó un estudio de electroencefalografía cuantitativa para determinar la potencia absoluta, la potencia relativa y la frecuencia media de las bandas delta, theta, alfa y beta en sujetos con CRA, con DEP y controles.

**Resultados.** Un total de 114 sujetos cumplió los criterios de inclusión. El grupo con CRA presentó mayor potencia absoluta, mayor potencia relativa y menor frecuencia media de la banda beta en comparación con los controles, mientras que el grupo con DEP presentó menor potencia absoluta de la banda delta que los controles.

**Conclusiones.** El DEP y el CRA presentan diferentes correlatos neurofisiológicos. Hay un efecto importante de la gravedad de la dependencia al alcohol sobre sus correlatos neurofisiológicos. Nuestros resultados apoyan la existencia de dos tipos distintos de desinhibición conductual.

**Palabras clave.** Actividad beta. Actividad delta. Consumo de riesgo de alcohol. Dependencia al alcohol. Desinhibición conductual. EEG cuantitativo.