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ORIGINAL ARTICLE

Effects of a Persistent Binge Drinking Pattern of Alcohol Consumption in Young People: A Follow-Up Study Using Event-Related Potentials

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Abstract — Aims: The objective of this study was to examine brain activity related to visual attention processes in youths who had maintained a binge drinking (BD) pattern of alcohol consumption for >2 years. **Methods:** The participants were 57 university students (26 binge drinkers: BDs) with no personal or family history of alcoholism or psychopathological disorders in first-degree relatives. Event-related potentials (ERPs) were recorded while participants performed a visual oddball task (twice within a 2-year interval). The latency and amplitude of the P3b component of the ERPs were analysed. **Results:** The P3b amplitude was larger in young BDs than in aged-matched controls at both evaluation times, and the difference was more pronounced after 2 years of maintenance of a BD pattern of consumption. The larger P3b amplitude was associated with an earlier onset of regular drinking and with a greater quantity and intensity of consumption. **Conclusions:** These findings suggest that young BDs exhibit anomalies in neural activity involved in attentional/working memory processes, which increase after 2 years of maintenance of BD. This anomalous neural activity may reflect underlying dysfunctions in neurophysiological mechanisms as well as the recruitment of additional attentional/working memory resources to enable the binge drinkers to perform the task adequately.

INTRODUCTION

Human adolescence is characterized by an increase in peer-directed social interaction, as well as by an increase in sensation or novelty seeking and risk taking behaviour (Spear, 2002), such as those linked to the consumption of recreational drugs (Van Amsterdam and Van den Brink, 2010). Alcohol is by far the most commonly used drug, and >90% of adolescents report having consumed it at some point in their lives (Anderson and Baumberg, 2006).

The intake of large amounts of alcohol in a short time followed by a period of abstinence is a very common pattern of consumption among young people. This pattern of abusive drinking is known as binge drinking (BD) and has generally been defined as the consumption of five or more drinks (four or more for females) on one occasion within a 2-h interval at least once in the last 2 weeks or in the last month (Courtney and Polich, 2009; Parada *et al*., 2011a). Given the increased incidence of BD, with around 10–40% of teenagers (depending on the country) reporting this pattern of alcohol use (Wechsler *et al*., 2002; Miller *et al*., 2007; Viner and Taylor, 2007; Caamaño-Isorna *et al*., 2008; Assanangkornchai *et al*., 2009), the effects of BD on neurocognitive development are of particular concern.

Throughout adolescence, important changes occur in brain morphology, including synaptic pruning, myelination and profound modifications in neurotransmitter receptor levels and sensitivity (Spear, 2000; Lenroot and Giedd, 2006). As a consequence of this brain maturation, cognitive functions such as attention, working memory and inhibitory control also develop during adolescence (Luna and Sweeney, 2004). Consequently, significant exposure to alcohol during this period may adversely affect a wide variety of neuromaturational processes, with behavioural, cognitive and psychosocial consequences, including an increased liability to alcohol use disorders (AUDs) (Hingson *et al*., 2006).

Given that BD is the most common form of problematic drug consumption among young people, the short, mid and long-term effects of BD on the brain are of great interest. However, as opposed to chronic alcoholism, whose structural, cognitive and functional consequences have been well documented (Oscar-Berman and Marinkovic, 2007), the effects of BD are poorly known.

Evidence from animal studies shows that BD induces more brain damage in adolescent than in adult rats (Crews *et al*., 2000), and that the former exhibit a greater degree of cognitive impairment than the latter (Markwiese *et al*., 1998; White and Swartzwelder, 2005). Likewise, it has been demonstrated that BD episodes may be more harmful for the brain than an equivalent amount of alcohol without withdrawal episodes (Becker, 1994; Duka *et al*., 2004).

Studies in human adolescents with AUD have shown several alterations at cognitive, structural and functional levels. Thus, AUD during adolescence has been associated with poorer performance on neuropsychological tasks requiring attention and visuospatial skills, verbal and non-verbal retrieval, problem solving and working memory (Moss *et al*., 1994; Tapert *et al*., 2002; Brown and Tapert, 2004). Similarly, AUD has been associated with reduced hippocampal and prefrontal volumes (De Bellis *et al*., 2000, 2005; Nagel *et al*., 2005) as well as altered brain response during tasks involving working memory and attention (Tapert *et al*., 2004; Caldwell *et al*., 2005).

With regard to BD, neuropsychological studies have reported different types of cognitive impairment in adolescents and young people with this consumption pattern (Hermens *et al*., 2013). Along with executive functioning (Sanhueza *et al*., 2011; Parada *et al*., 2012) and memory (Scaife and Duka, 2009; Parada *et al*., 2011b), attention is one of the cognitive processes that is most often affected in this population (Hartley *et al*., 2004; Townshend and Duka, 2005; Squeglia *et al*., 2009). Furthermore, diffusion tensor imaging (DTI) and

magnetic resonance imaging (MRI) studies have demonstrated that adolescent binge drinkers (BDs) have poorer white matter integrity and different brain morphometry than control adolescents (McQueeny *et al.*, 2009; Squeglia *et al.*, 2012). Finally, functional MRI (fMRI) studies have also shown abnormal brain activity in BDs during performance of learning and memory tasks (Schweinsburg *et al.*, 2010, 2011; Squeglia *et al.*, 2011).

Event-related potentials (ERPs) are commonly used in the study of brain functioning. Numerous studies have used the technique to assess the neurofunctional effects of alcoholism, and anomalies in several ERP components have been reported in relation to attentional processes (Porjesz and Begleiter, 1996; Rodríguez Holguín *et al.*, 1999; Malone *et al.*, 2001; Cohen *et al.*, 2002). Of these, the P3b component has been the most frequently used to assess the effects of alcoholism. This positive wave, which peaks at around 300–600 ms after the stimulus onset and reaches its maximum amplitude at centro-parietal sites, is elicited by visual or auditory 'task-relevant' events and has been functionally associated with attention and memory access (Polich, 2007).

With regard to chronic alcoholics, smaller P3b amplitude has been observed in these subjects compared with non-alcoholic controls (Porjesz and Begleiter, 2003). Given that low P3b amplitude has also been observed in children of alcoholics prior to any alcohol exposure (Polich *et al.*, 1994), and that, unlike other electrophysiological signals, the P3b component does not return to normal values after withdrawal (Parsons, 1994; Fein and Chang, 2006), it has been hypothesized that the low P3b amplitude constitutes a biological marker of genetic vulnerability to alcoholism, and that it precedes the development of alcoholism rather than being a consequence of the alcohol intake (Porjesz *et al.*, 2005; Perlman *et al.*, 2009; Euser *et al.*, 2012).

Several ERP studies in recent years have revealed that the BD pattern also leads to electrophysiological anomalies (Ehlers *et al.*, 2007; Crego *et al.*, 2009, 2010, 2012; Maurage *et al.*, 2009, 2012; López-Caneda *et al.*, 2012; Petit *et al.*, 2012). Thus, a recent study carried out by our research group reported larger P3b amplitudes in young BDs than in controls during the performance of a visual oddball task (Crego *et al.*, 2012). The larger amplitude was interpreted in terms of additional neural recruitment, associated with attentional processing, which would compensate for a possible BD-related neurocognitive decline.

The present study is a continuation of this work and is therefore framed as a follow-up study. Follow-up studies on BD are scarce, and the psychophysiological consequences of the maintenance of this intensive pattern of alcohol consumption remain unclear. To our knowledge, only two studies have carried out electrophysiological follow-up tests; both of these studies have shown that the maintenance of BD leads to an increase in electrophysiological anomalies during emotional processing (Maurage *et al.*, 2009), and response inhibition (López-Caneda *et al.*, 2012) in young university students.

This work attempts to provide new evidence about how maintenance of the BD pattern in the mid-long term may affect brain functioning and, in particular, the attentional processes that mediate the detection of a relevant stimulus or event. Therefore, the specific aim of this study was to determine whether, after 2 years maintenance of a BD pattern, the electrophysiological anomalies initially observed in young people persisted, increased or (eventually) decreased.

METHODS

Participants

Fifty-seven students of the University of Santiago de Compostela (Galicia, Spain) participated in the study. Thirty-one were classified as controls (16 females) and 26 as BDs (11 females). The procedure for subject selection is fully described in López-Caneda *et al.* (2012). The participants were evaluated twice within a 2-year interval (at 18–19 and 20–21 years of age).

The subjects were initially selected on the basis of their responses to the Alcohol Use Disorder Identification Test (AUDIT) (Varela *et al.*, 2005), and other questions regarding use of alcohol and other drugs. They were included in the BD group if they (a) drank six or more standard alcoholic drinks (SADs) (10 g of alcohol, according to the Spanish Health Authority's reference) on the same occasion one or more times per week or (b) drank six or more SADs on the same occasion at least once a month and during these episodes drank at least three drinks per hour. Only participants who met these criteria at the time of both evaluations were selected. Participants who drank less than this amount at the time of both assessments were classified as controls. The consumption and demographic characteristics are shown in Table 1.

The exclusionary criteria were non-corrected sensory deficits, any episode of loss of consciousness for >20 min, history of traumatic brain injury or neurological disorder, personal history of psychopathological disorders (according to DSM-IV criteria), use of illegal drugs except cannabis and family history of major psychopathological disorders in first-degree relatives,

Table 1. Demographic and drinking characteristics of the control and binge drinking groups (mean ± SD)

	First evaluation		Second evaluation	
	Controls	Binge drinkers	Controls	Binge drinkers
N (females)	31 (16)	26 (11)	31 (16)	26 (11)
Age	18.5 ± 0.5	18.8 ± 0.5	20.4 ± 0.6	20.8 ± 0.6
Handedness (right/left)	28/3	24/2	28/3	24/2
Caucasian ethnicity (%)	100	100	100	100
Regular tobacco smokers	1	3	1	5
Regular use of cannabis (once a week)	0	3	0	0
Age of onset of regular drinking	15.7 ± 1	14.6 ± 1.4*	15.7 ± 1	14.6 ± 1.4*
Total grams of alcohol in a standard week	–	–	39.67 ± 35.08	216.44 ± 98.26**
Number of drinks in a standard drinking episode	1.7 ± 1.3	5.6 ± 2.6**	2.1 ± 1.4	4.7 ± 1.9**
Total AUDIT score	2.8 ± 2.6	12.4 ± 4.1**	3.2 ± 3	11.2 ± 3.1**

*$t < 0.05$ significant group differences.
**$t < 0.001$ significant group differences.

including alcoholism and substance abuse. Participants with AUDIT scores ≥20 (i.e. with AUD) were excluded.

Some of the subjects included in this study were part of the sample used in the Crego *et al.* study (2012). From that sample, those subjects who abandoned (13 BD, 16 control), those who did not meet the inclusion criteria at the second evaluation (6 BD, 7 control) and those with a poor quality of the electroencephalographic (EEG) recording at this time (1 BD, 3 control) were excluded. On the contrary, those subjects excluded in the previous study due to their being left-handed, regular tobacco smokers or consumers of cannabis (18 in total) were included here, in order to increase the sample size. All the participants were paid volunteers, and all signed an informed consent at each assessment. The experiment was undertaken in compliance with Spanish legislation and the code of ethical principles for medical research involving human subjects present in the Declaration of Helsinki (Williams, 2008). The protocol was approved by the Bioethics Committee of the University of Santiago de Compostela.

Procedure

Participants were asked to abstain from consuming drugs and alcohol for 12 h before the experiment and not to partake in BD episodes in the 24 h before the experiment, to prevent effects of acute alcohol intake and to rule out withdrawal effects. The subjects were also instructed not to smoke or drink tea or coffee for at least 3 h before the assessments.

The task and procedure are fully described in Crego *et al.* (2012). In brief, the subjects performed a visual oddball task consisting of 150 stimuli, in which they had to respond to an infrequent ($P = 0.20$) white star while ignoring a frequent white square. The stimuli were briefly (45 ms) presented with an interstimulus interval of 1000–1400 ms. The EEG was recorded from 32 scalp locations (Extended 10–20 International System) referred to the nose tip, with a 500 Hz A/D rate.

Data analysis

Behavioural analysis

Only responses occurring between 100 and 1000 ms after the onset of a target stimulus were considered as correct. Responses to standard stimuli were rated as false alarms and failures to respond to target stimuli were scored as omissions. Reaction times (RTs) and the percentages of correct responses and false alarms were analysed by Student's *t*-tests for independent samples.

EEG analysis

The procedure for extracting the ERP components is fully described in Crego *et al.* (2012). After being corrected for ocular artefacts and digitally filtered (0.1–30 Hz), the EEG was epoched (−100 ms prestimulus to 900 ms poststimulus), and noisy segments and those associated to incorrect responses were excluded before averaging. The number of rejected segments was similar for the two groups (16% in the control group, 13% in the BD group). P3b was identified in the target-locked waveforms as the largest positive peak between 300 and 500 ms poststimulus at frontal (F3-Fz-F4-FC3-FCz-FC4), central (C3-Cz-C4-CP3-CPz-CP4) and parietal (P3-Pz-P4-PO3-POz-PO4) electrodes. Mixed-model ANOVAs with two between-subject factors (group: BD and control; gender: male and female) and three within-subject factors (evaluation: first and second evaluation; region: frontal, central and parietal; electrode: six channels) were used to examine the data (alpha level ≤ 0.05). Degrees of freedom were corrected, when appropriate, by the Greenhouse–Geisser estimate, and *post hoc* paired comparisons were performed with the Bonferroni adjustment for multiple comparisons, also with an alpha level of 0.05.

Earlier ERP components (P1, N2, N2) were also analysed, but results are not reported here because no significant group-related effects were found.

Finally, correlation analyses were performed to determine the relations among the alcohol use variables and the amplitude of P3b in the parietal region. The measures of alcohol-related behaviour considered were as follows: age of onset of regular drinking; quantity (total grams in a standard week) and intensity (number of drinks in a standard drinking episode).

RESULTS

Behavioural performance

The behavioural data are summarized in Table 2. There were no significant differences in RTs between the control and BD group, and the percentage of correct responses was ~99% in both groups.

Electrophysiological data

The grand averages of target-locked ERP waveforms for each group and the voltage maps corresponding to the peak of P3b are shown in Figs 1 and 2, respectively.

Analysis of the P3b amplitude revealed significant effects for the factors, evaluation [$F(1,53) = 5$, $P = 0.03$], with a larger amplitude in the second evaluation, region [$F(2,53) = 138.79$, $P < 0.001$], with a larger amplitude over the parietal region, and gender [$F(1,53) = 6.41$, $P = 0.014$], with a larger P3b amplitude in females than in males. The analysis also revealed significant differences between the groups [$F(1,53) = 4.8$, $P = 0.033$], with a larger P3b in the BD than in the control group. The group–region interaction was significant [$F(2,53) = 8.54$, $P < 0.001$], but the group–gender interaction was not significant. The group–evaluation interaction did not reveal significant effects. However, independent analysis for each evaluation moment showed a significant Group effect in the second [$F(1,53) = 6.47$, $P < 0.014$], but not in the first evaluation. Finally, although there were no significant interactions involving group–region–evaluation, separate analyses were performed for each evaluation moment, because of the interest

Table 2. Behavioural data for the control and binge drinking groups in the two evaluations (mean ± SD)

Behavioural performance	Controls	Binge drinkers
First evaluation		
Response time (ms)	402.47 ± 48.89	397.31 ± 39.97
% correct responses	99.40 ± 1.23	98.60 ± 2.07
% false alarms	0.07 ± 0.23	0.12 ± 0.45
Second evaluation		
Response time (ms)	391.57 ± 44.55	396.02 ± 40.35
% correct responses	99.68 ± 0.96	98.83 ± 1.51
% false alarms	0.02 ± 0.14	0.03 ± 0.16

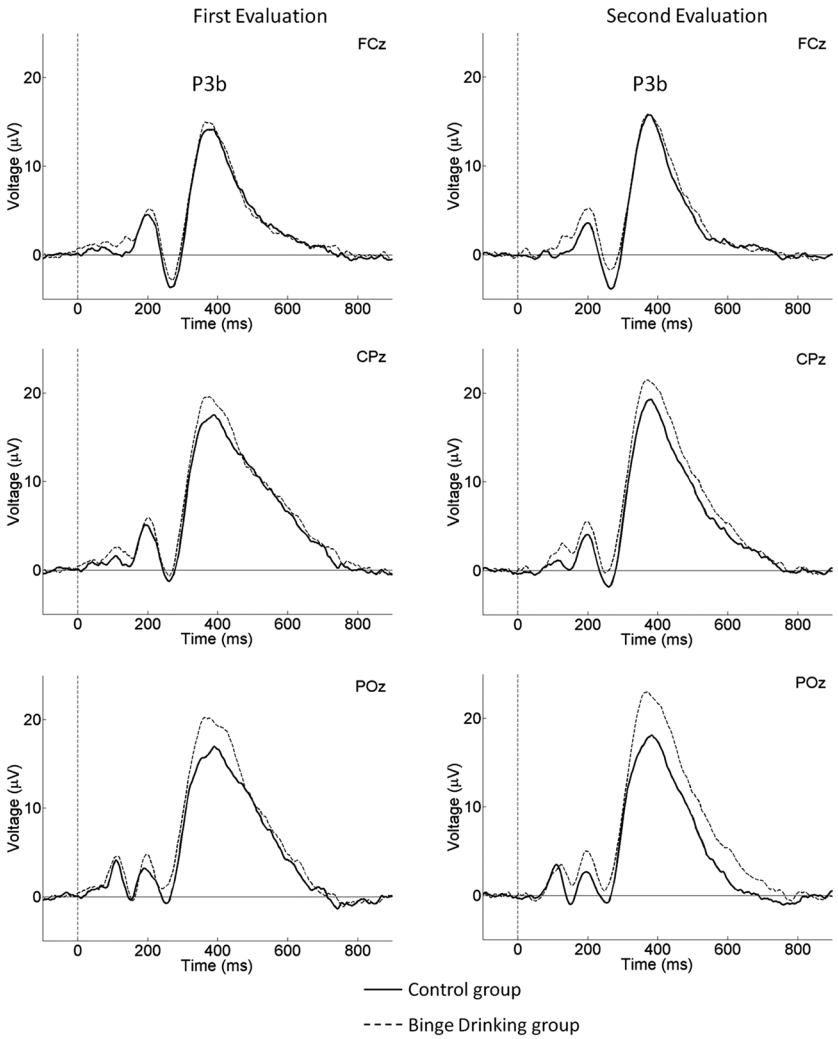

Fig. 1. Grand average ERPs for the control (solid line) and binge drinking (dashed line) groups in response to target stimuli during the first and second evaluations. Averages are shown for FCz, CPz, POz electrodes.

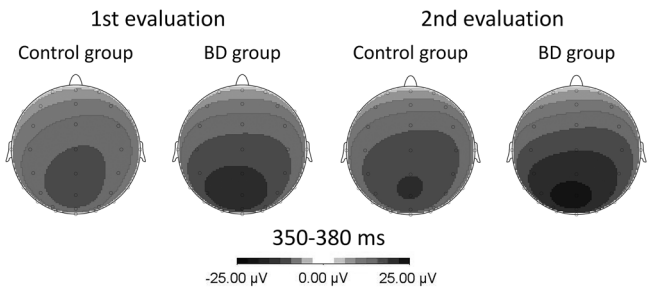

Fig. 2. Voltage maps corresponding to the peak P3b component during the first and second evaluations in the control and binge drinking (BD) groups in response to the target stimuli.

in knowing whether the differences between group were more pronounced over time. These analyses revealed significantly larger P3b amplitude in the BD than in the control group only over the parietal region [$F(1,53) = 4.07$, $P = 0.048$] in the first evaluation, and over the central [$F(1,53) = 4.63$, $P = 0.033$] and parietal regions [$F(1,53) = 15.01$, $P < 0.001$] in the second evaluation.

The correlation analysis revealed a negative correlation between age of onset of regular drinking and the P3b amplitude over the parietal region in the first [$r = -0.332$, $P = 0.016$] and the second evaluation[$r = -0.403$, $P = 0.003$]. Moreover, during the second evaluation, the parietal P3b amplitude was positively correlated with quantity [$r = 0.345$, $P = 0.009$], and intensity [$r = 0.286$, $P = 0.03$] of consumption (see Fig 3).

With regard to the latency of P3b, there was no significant main effect or interaction involving the group factor.

DISCUSSION

This follow-up study obtained ERP data to enable examination of the effects of the maintenance of a BD pattern of alcohol consumption, during at least 2 years, on brain electrical activity in young students. The results demonstrated that, despite similar behavioural performance, the P3b amplitude was larger in young BDs than in aged-matched controls during performance of a visual oddball task; the difference was more pronounced after persistent BD consumption, and the P3b amplitude was negatively correlated with age of onset of

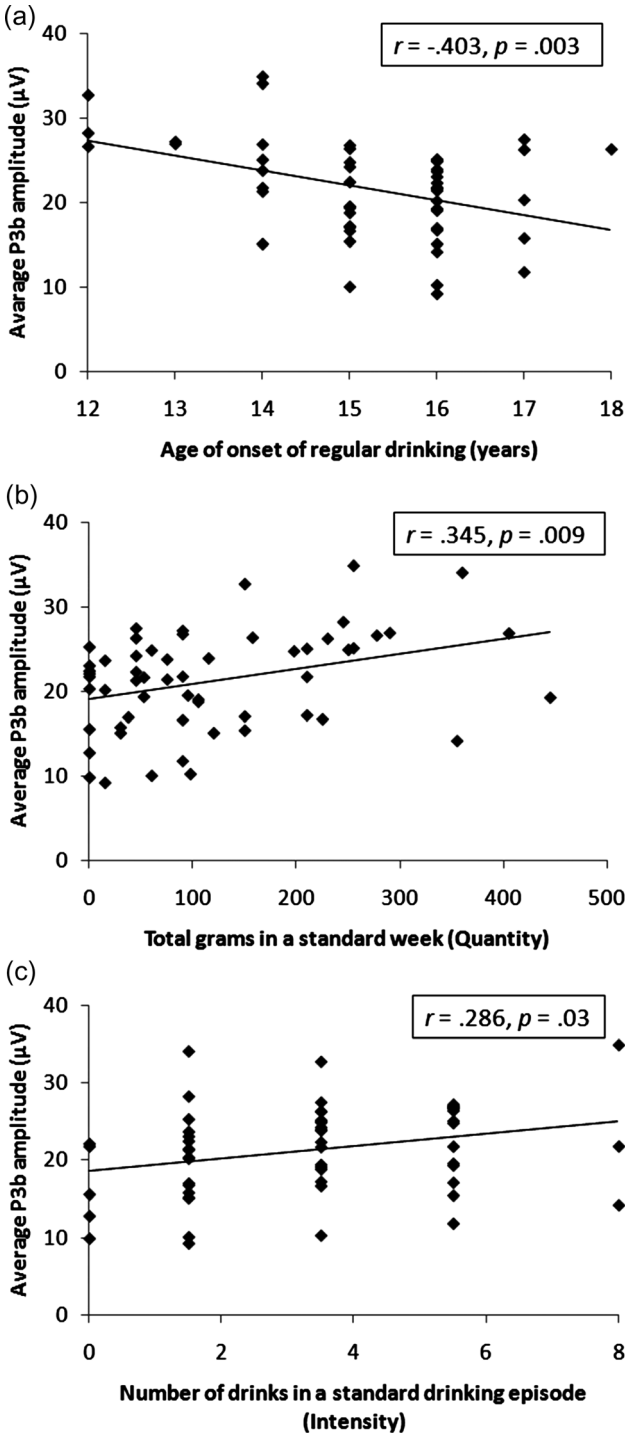

Fig. 3. Correlation analysis. Larger P3b amplitude over the parietal region during the second evaluation was associated with (**a**) an earlier onset of regular drinking, (**b**) greater quantity of alcohol consumed in a standard week and (**c**) more drinks in a standard drinking episode.

regular drinking and positively with quantity and intensity of consumption.

In the first part of this follow-up study, larger P3b amplitudes were recorded in the BD than in the control group (Crego *et al*., 2012). The results of the present study corroborate and extend the initial results. The results continued to reflect significantly larger P3b amplitude observed in the BD group in the first evaluation, and showed that differences became greater in the second evaluation, after the students had continued BD for a further 2 years. Therefore, the results appear to indicate that the electrophysiological differences between control and BD subjects continue to increase with the length of BD pattern of consumption.

The present findings add to evidence from previous longitudinal studies carried out in adolescents and young BDs showing that maintenance of a BD pattern for several years leads to poorer performance of various neuropsychological tasks requiring decision-making, visuospatial memory and sustained attention (Goudriaan *et al*., 2007; Squeglia *et al*., 2009). In ERP studies, 9 months of BD produced marked latency abnormalities in P1, N2 and P3b components in adolescents in an emotional valence judgment task (Maurage *et al*., 2009) and >2 years of BD in young people resulted in larger Go-P3 and NoGo-P3 amplitudes and hyperactivation of right inferior frontal cortex during response inhibition in a Go/NoGo task (López-Caneda *et al*., 2012).

ERP studies have revealed a smaller P3b amplitude in abstinent chronic alcoholics than in the respective controls (Campanella *et al*., 2009). In the present study, in which the sample of BDs had no first-degree family history of alcoholism (FHA) or psychopathological disorders, the P3b amplitude was larger in the BD group than in the control group. Given that the reduced P3b amplitude observed in alcoholics has been interpreted as a biological marker of genetic vulnerability to alcoholism (Porjesz *et al*., 2005; Perlman *et al*., 2009; Euser *et al*., 2012), a smaller P3b in BDs with no FHA was not expected. On the contrary, several studies have shown that adolescents and young BDs without FHA have larger amplitudes than their control peers (either abstinent or with moderate alcohol intake) in various ERPs. Similarly, a recent study in treatment-naïve adolescents with alcohol dependence (TNAD), who meet criteria for alcohol dependence but who drink around 50% less and have a lower FHA and psychiatric comorbidity than treated alcoholics, showed that the P3b amplitude was 22% (although not statistically significant) larger in TNAD adolescents boys than in age-matched controls (Cuzen *et al*., 2013). This study highlights the major differences that may exist in the P3b amplitude in individuals with alcohol abuse when variables such as alcohol exposure pattern, genetic vulnerability to alcoholism or psychiatric comorbidity are taking into account.

With regard to BD studies, Petit *et al*. observed larger P100 amplitudes in BD undergraduate students than in controls on performance of a visual oddball test with alcohol-related pictures (Petit *et al*., 2012). This finding was interpreted as an increased allocation of early attention toward alcohol-related stimuli in BDs. Our research group has also reported an augmented N2 amplitude in BDs, relative to controls, in a visual continuous performance task (Crego *et al*., 2009). This larger N2 amplitude was considered as indicative of the greater 'attentional effort' required by BDs to perform the task adequately. Finally, larger P3 amplitudes to Go and NoGo stimuli have been also recorded in our laboratory in a follow-up study (López-Caneda *et al*., 2012). This larger amplitude was interpreted as indicating that BDs may use additional neural mechanisms to allow them to perform the task at the same level as controls.

Given that P3b amplitude reflects the attentional capacity or the attentional resources invested in categorization of task

relevant events (Kok, 2001)—where categorization is understood to be the process that leads to the decision that the external stimulus matches, or does not match, an internal representation of the relevant stimulus, the larger P3b amplitude in BDs observed in this study may reflect the use of additional endogenous attentional/working memory resources to enable efficient memory access and, thus, to perform the task adequately.

In recent years, several fMRI studies on BD have shown that adolescents and young BDs do indeed recruit additional neural resources to enable proper task performance. Thus, the few studies performed to date have reported that, although there were no significant differences at the behavioural level, adolescent BDs showed enhanced brain response in frontal and parietal regions during a verbal learning task (Schweinsburg *et al*., 2010, 2011) as well as in frontal and anterior cingulate regions during spatial working memory tasks (Squeglia *et al*., 2011).

We also observed that the P3b amplitude was negatively associated with age of onset of regular drinking and positively associated with quantity and intensity of alcohol intake. An earlier age of onset of AUD has been associated with greater cognitive deficits and greater neurological damage (Pishkin *et al*., 1985; Chanraud *et al*., 2007), and a high intensity of drinking has predicted low performance on attention and executive function in adolescents with AUD (Thoma *et al*., 2011). In young BDs, poorer decision-making has been linked to an earlier onset of BD (Goudriaan *et al*., 2007), and to higher quantity and frequency of alcohol use (Goudriaan *et al*., 2011). Likewise, more drinking days in the previous year predicted a decline in visuospatial functioning in BD girls, and more hangover symptoms were linked to worsening in sustained attention in BD boys (Squeglia *et al*., 2009). Taking into account these correlations and the development of anomalies in the amplitude of P3b throughout the follow-up period, we propose that the anomalous brain activity of BDs, reflected in the larger P3b, may be a result of the adverse effects of a BD pattern on the young brain, which precludes the possibility of this being a premorbid characteristic.

Other types of studies are necessary to elucidate the underlying mechanisms of these electrophysiological alterations, but some hypotheses may be proposed on the basis of other findings. In this sense, it is known that moderate to heavy alcohol intake leads to compensatory changes in neurotransmitter systems, mainly glutamate (reducing its excitatory activity) and GABA (enhancing its inhibitory activity) (Valenzuela, 1997). When the alcohol is withdrawn (for instance, during a period of abstinence), a rebound effect characterized by glutamatergic hyperactivity takes place and may result in cellular death (Tsai and Coyle, 1998). This may explain why abstinent chronic alcoholics with multiple withdrawal episodes show more severe effects than alcoholics who do not undergo detoxifications or withdrawal episodes (Becker, 1998). It is possible that, as young BDs have been drinking alcohol for less time and consume lower amounts than alcoholics, cellular death is still minimal, but synaptic functioning may be already compromised. The changes in synaptic functioning would not necessarily lead to immediate impairment of behaviour because compensatory mechanisms may appear and thus conceal the underlying or latent deficit.

Ultimately, such neurocompensatory mechanisms (such as the larger P3b amplitude) may herald future problems in performance if the BD pattern is continued, since the brain may no longer be able to counteract the harmful effects derived from this type of drinking. Nevertheless, longitudinal studies covering longer periods are necessary to test this hypothesis.

It should be stated that the interpretation of our results must be cautious, especially because P3b amplitude is sensitive to different psychological and physiological factors (i.e. motivation, anxiety, craving) that could differ between the groups. Although the procedure in this study tried to prevent the influence of these factors (e.g. subjects did not know the group's classification criteria; differences between groups on motivation were minimized by providing identical monetary incentive to participants; there were no differences in the anxiety level measured by the Symptom Check List-90-Revised (Derogatis, 2002) and craving or withdrawal symptoms were unlikely because AUD were excluded), further studies are necessary to replicate the results.

Finally, gender effects should be considered. In this study, females presented longer P3b than males, but this factor did not interact with group. Although several studies have reported greater vulnerability to alcohol in females, data from ERP studies with BD subjects without AUD have not found consistent gender effects; furthermore, this result is consistent with previous data from our laboratory (Crego *et al*., 2009; 2010, 2012; López-Caneda *et al*., 2012).

In summary, the results of the present study reveal anomalous brain activity in young university binge drinkers, relative to aged-matched controls, during the performance of a visual oddball task; this was reflected by larger P3b amplitudes, which increased after 2 years of this pattern of alcohol consumption. The larger P3b amplitude is associated with an earlier onset of regular drinking and with a greater quantity and intensity of consumption. These findings suggest that young people with a BD pattern experience anomalies in neural activity involved in attentional/working memory processes, which may reflect underlying dysfunctions in neurophysiological mechanisms as well as the recruitment of additional attentional resources to enable the BDs to perform the task adequately.

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Conflict of interest statement. None declared.

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