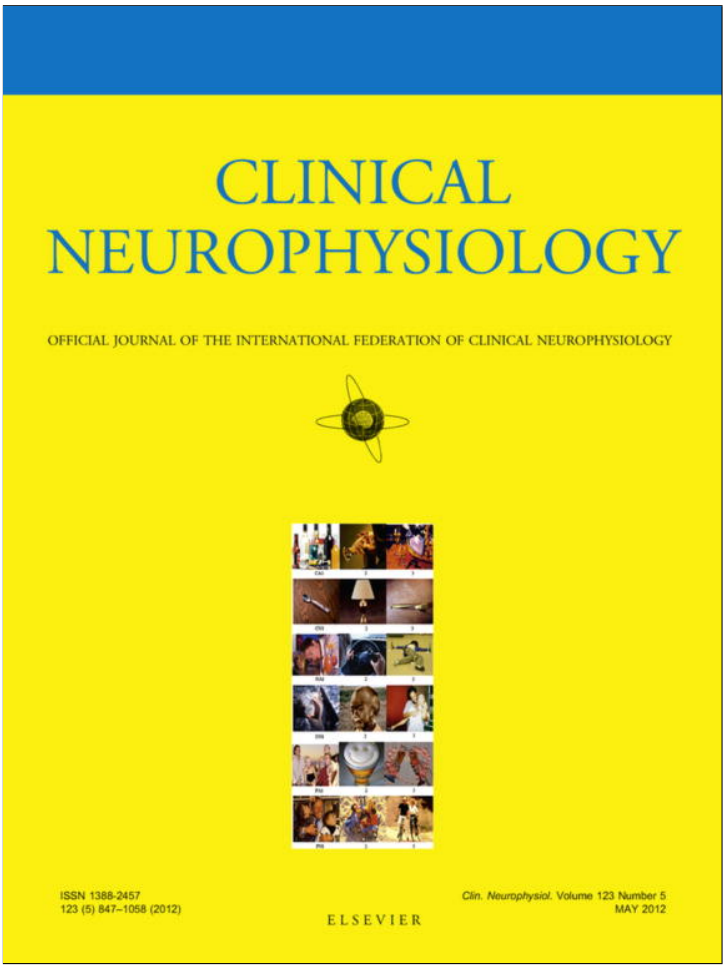

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Early attentional modulation by alcohol-related cues in young binge drinkers: An event-related potentials study

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HIGHLIGHTS

- ERP data showed that significantly larger P100 amplitudes are elicited by alcohol-related pictures in binge drinkers than in control students.
- All students had significantly faster responses to alcohol-related stimuli compared to non alcohol-related ones.
- The valence of the cues did not influence the observed heightened electrophysiological or behavioural reactivity elicited by alcohol cues.

ABSTRACT

Objective: Episodic excessive alcohol consumption (i.e., binge drinking) is now considered to be a major concern in our society. Previous studies have shown that alcohol cues can capture attentional resources in chronic alcoholic populations and that the phenomenon is associated with the development and maintenance of alcoholism. Using event-related potentials (ERPs), we investigated the responses of binge drinkers to alcohol-related pictures.
Methods: Two groups of college students (n = 18 in each group) were recruited for the study. One group was composed of binge drinkers and the other of controls. Each student completed a simple visual oddball paradigm in which alcohol-related and non-alcohol-related pictures (positive, neutral or negative) were presented. ERPs were recorded to explore the electrophysiological activity associated with the processing of each cue during the different cognitive steps.
Results: Although there were no behavioural differences between the two groups after detection of alcohol- and non-alcohol-related cues, the ERP data indicated that processing of alcohol-related stimuli was modulated by binge drinking: in the binge drinkers, the P100 amplitudes elicited by the alcohol-related pictures were significantly larger than those elicited by the non-alcohol pictures.
Conclusions: The present study provides evidence for an early processing enhancement, indexed by increased P100 amplitude, in binge drinkers when confronted with alcohol cues.
Significance: These findings suggest that higher reactivity to alcohol cues is not a phenomenon limited to adult alcoholics, but that young binge drinkers exhibit signs of prioritizing processing related to alcohol. Prevention intervention for alcohol misuse in young people should consider approaches that address this automatic cue reactivity.

1. Introduction

Binge drinking refers to the consumption of a large number of drinks over a short interval of time followed by periods of abstinence. Although its definition continues to stimulate debate, binge drinking is commonly defined as the consumption of 5 or more alcoholic drinks (4 or more for women) on one occasion, within a 2-h interval (according to the National Institute on Alcohol Abuse and Alcoholism), at least once in a 2-week (Keller et al., 2007; Presley and Pimentel, 2006) or 1 month (Jennison, 2004; Xing et al., 2006) period, with periods of abstinence between each episode. Binge drinking is a common occurrence among younger people, especially university students. These financially squeezed students often live

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in community dorms or apartments and the cheap prices and special promotions offered in university bars combined with environmental and social aspects of university life may encourage binge drinking. In addition, since these students seldom have early morning commitments, it is not surprising that they frequently drink to excess and fulfill the criteria for binge drinking (Johnston et al., 1997; Lange et al., 2002; Wechsler et al., 2002).

The effects of alcohol on the central nervous system (CNS) have been extensively studied, and the neurocognitive, neuroanatomical and neurofunctional consequences of chronic alcohol abuse in alcohol-dependent adults have been documented (for a review see Oscar-Berman and Marinković (2007)). Epidemiological data have demonstrated that adolescence and young adulthood is a key period for the development of chronic alcoholism (Harford et al., 2005; Kessler et al., 2005; Grant and Dawson, 1998; Li et al., 2004; Hingson et al., 2006). In particular, in their 6-year cohort study, Bonomo et al. (2004) showed that binge drinking in teenagers was one of the main risk factors for alcohol dependence in adulthood. Given the high prevalence (approximately 30–40%), of binge drinking in young people aged between 18 and 24 years old (Miller et al., 2007), these data raise the question as to whether there is a link between binge drinking in adolescence and the development of alcohol dependence in both adolescence and adulthood and as such warrants further investigation.

Compared with adults, relatively little is known about adolescent binge drinking and its neurocognitive consequences. This is particularly surprising for at least three reasons. Firstly, several animal studies (e.g., Tokunaga et al., 2006; White et al., 2000; Crews et al., 2000, 2006) have suggested that adolescence is a critical stage in the development of the brain, which is particularly sensitive to the neurotoxic effects of alcohol, in particular brain structures that mature during this period, such as the hippocampus and the prefrontal cortex (Hunt, 1993; Monti et al., 2005; White and Swartzwelder, 2004). Studies of human clinical samples have consistently revealed that adolescents with alcohol-use disorders (AUDs) have decreased hippocampus (De Bellis et al., 2000; Nagel et al., 2005) and prefrontal cortex (De Bellis et al., 2005; Medina et al., 2008; Schweinsburg et al., 2005) volumes compared with adolescents without AUDs, as well as deficits in functions related to these areas, such as visuospatial capacities (Tapert and Brown, 1999), learning processes and working memory (Brown and Tapert, 2004; Tapert et al., 2002). Secondly, binge drinking habits are characterized by multiple withdrawals (caused by repeated phases of alcohol intoxication and abstinence over a short period of time), which are known to be particularly damaging to brain function (Obernier et al., 2002; Pascual et al., 2007). Thirdly, heavy drinking is generally acceptable within the community, such that binge drinkers do not recognize their alcohol use as problematic and are unlikely to be motivated to reduce alcohol intake. In most cases, this tendency to "bingeing" only lasts during the academic years and stops with the assumption of adult roles, such as employment, marriage and/or parenthood (e.g., Chen and Kandel, 1995; Bachman et al., 2002; Dawson et al., 2006). However, as stopping their excessive alcohol intake is not part of the binge drinking student's plans, it is reasonable to assume that the deficits and/or permanent neurobiological changes that occur during this period of extensive brain development could play a role in the maintenance of alcohol use and abuse. This may be associated with difficulties in stopping alcohol consumption at a later stage, which could then develop into problem-use (e.g., Hiller-Sturmhofel and Swartzwelder, 2004; King et al., 2006; Haller et al., 2010).

A few recent studies have explored the cerebral consequences of binge drinking in nonclinical samples of adolescents and university students. Electrophysiological studies identified abnormalities in some event-related potentials (ERPs) (Ehlers et al., 2006; Crego et al., 2009, 2010) after only 9 months of binge drinking experience and in subjects without behavioural deficiencies (Maurage et al., 2009), as well as EEG rhythm modifications similar to those observed in alcoholics (Courtney and Polich, 2010). In functional magnetic resonance imaging (fMRI) studies, adolescent binge drinkers, with relatively short drinking histories, demonstrated different patterns of brain functioning and somewhat poorer performance during verbal encoding compared with non-drinkers (Schweinsburg et al., 2011a,b). Overall, these few studies have shown that binge drinking provokes considerable cerebral dysfunction, similar to that observed in alcohol dependence, although it may not be expressed at a behavioural level and may be less serious than that provoked by chronic alcoholism. These observations strengthen the hypothesis that this drinking pattern may be a precursor of chronic alcoholism and that binge drinking and chronic alcoholism may correspond to two stages of the same phenomenon (e.g., Kilty, 1990; Chassin et al., 2002; Wagner and Anthony, 2002; Enoch, 2006; Li et al., 2007).

With respect to the neurocognitive effects of binge drinking in youths, it is important to evaluate similarities with deficits induced by long-term alcohol consumption in adults so as to be able to develop adapted information and prevention programs for young people. In the present study, we focused on the fact that when alcoholic patients are exposed to alcohol-related cues, changes are observed in several physiological parameters, such as skin-conductance level (Kaplan et al., 1985; Turkkan et al., 1989), salivation level (Pomerleau et al., 1983) and heart rate (Kaplan et al., 1985; Turkkan et al., 1989; Payne et al., 1992), as well as changes in the cognitive processing of these alcohol-related stimuli (Field and Cox, 2008). A dominant theoretical model of addiction proposes that cues associated with alcohol gain incentive value with repeated drug use through sensitization of the brain's reward system, such that the incentive value of these stimuli intensifies making them increasingly "wanted" (Robinson and Berridge, 1993, 2001). Various tasks have been developed to assess this "bias" in cognitive processing of alcohol cues in chronic alcoholism, which together demonstrate that chronic alcoholics exhibit excessive attentional focusing on alcohol-related cues (e.g., Bauer and Cox, 1998; Cox et al., 1999, 2002; Stormark et al., 2000; Sharma et al., 2001; Ryan, 2002; Townshend and Duka, 2001; Field et al., 2004; Noël et al., 2006). Interestingly, models of alcohol cue reactivity indicate that a number of pairings with alcohol consumption are needed for the associated stimuli to become conditioned (see Field and Cox, 2008). Nevertheless, findings support that adolescents with AUDs experience alcohol cue reactivity, which is evidenced by increased salivation in the presence of alcohol-related stimuli (Thomas et al., 2005) and heightened brain response to alcohol cues in brain regions that have been conditioned with the positive, rewarding aspects of alcohol use (Tapert et al., 2003, 2004). Given that adolescent alcoholics rarely reach the upper range of dependent criteria, as physical withdrawal symptoms are unusual in this population (Thomas et al., 2005), such observations therefore question the relative importance of dependence severity and drinking pattern habits in alcohol cue reactivity.

The aim of the present study was to investigate whether binge drinking students, with their brief history of alcohol exposure compared to adults, and without a diagnosis of AUD, exhibit any modulation in alcohol-cue processing. More specifically, we wanted to examine whether there was any difference in cerebral reactivity between students with binge drinking habits and paired controls when they were confronted by alcohol-related compared to non-alcohol-related (control) cues. Our main hypothesis was that binge drinkers (as compared to controls) will display an enhanced attentional processing of alcohol-related stimuli as compared to unrelated alcohol ones. This will be tested by focusing our analyses on ERP components well-known to be modulated by attention. In this view, a variant of the classical oddball paradigm was used in which participants had to detect deviant stimuli from a series of frequent standard stimuli, an experimental design that has already

been shown to be highly informative in psychopathological populations (e.g., Campanella et al., 2005; Campanella and Philippot, 2006; Maurage et al., 2007; Mejias et al., 2005; Rossignol et al., 2007, 2008; Vermeulen et al., 2008). Indeed, many 'ERP oddball' studies have been conducted to investigate neurophysiological markers in psychiatric disorders. For example, a reduction in P3b amplitude and prolongation of P3b latency, which suggest attentional and/or memory alterations, have been noted in depression (e.g., Sara et al., 1994; Bruder et al., 2001), alcoholism (e.g., Enoch and Goldman, 2002; Suresh et al., 2003) and schizophrenia (e.g., Duncan, 1990). This type of paradigm has also been used to define the level of the information processing stream (perceptive, attentional or decisional) at which the differences between control participants and clinical patients originate (in anxiety: e.g., Li et al., 2007; in depression: e.g., Yang et al., 2011).

In the current study, deviant stimuli were or were not related to alcohol, which allowed processing of both categories of stimuli to be investigated. Moreover, recording of the brain ERPs during the task enabled information processing to be monitored during the entire information-processing stream, ranging from sensory to higher cognitive processes. This permitted the distinction to be made between early automatic, unconscious sensory processing (P100) and more conscious attentional orienting mechanisms (N2b) or even decisional cognitive components (P300). Indeed, the P100, recorded at around 100 ms at posterior sites, is sensitive to physical stimulus factors but also to visual attention (e.g., Hillyard et al., 1996, 1998). For example, enhanced P100 amplitudes have been reported during perception of fearful faces (e.g., Batty and Taylor, 2003; Pourtois et al., 2005a), supporting the hypothesis of a faster detection of motivationally relevant stimuli (Vuilleumier and Pourtois, 2007), that links with ongoing feedback from the amygdala (Eimer and Holmes, 2007). These top-down attentional influences have also been demonstrated, as P100 amplitudes are, for example, typically larger for attended than for unattended stimuli (Hillyard et al., 1998). In contrast, the N2b component, maximally recorded at around 250 ms in occipital sites is a negativity that is the result of a deviation in form or context of a prevailing stimulus (Hoffman, 1990). This is typically found in oddball tasks, in which participants have to detect sporadic deviant stimuli among a series of standard frequent stimuli (Näätänen and Picton, 1986). In several studies using this paradigm, the N2b has been typically evoked before the motor response, indicating its connection with the cognitive processes of stimulus identification and distinction (Hoffman, 1990). Furthermore, the N2b only arises during conscious stimulus attention which suggests it relates to the voluntary attentional switch needed to take new information into account (Halgren and Marinkovic, 1995; Campanella et al., 2002). Changes in the N2b have been shown to indicate a deficit in the voluntary allocation of attentional resources. For example, Vermeulen et al. (2008) showed that alexithymic individuals had reduced N2b components to emotional deviant stimuli. Finally, the P3b component, P300 (sometimes called late positive potential or late positive complex), peaking at parietal sites at around 450 ms, arises when an attended, infrequent task-relevant stimulus is detected (Halgren, 1990; Knight et al., 1995) and reflects different higher level mechanisms, such as inhibition, cognitive closure, decision making and premotor response-related stages (see Polich, 2007 for a review). P300 amplitude is linked to increased resource deployment (e.g., Yee and Miller, 1994) and is related to task difficulty, stimulus probability, motivation, and vigilance. P300 amplitude has been shown to be larger for emotional than for neutral stimuli (e.g., Fischler and Bradley, 2006; Herbert et al., 2008; Schupp et al., 2004), which reflects preferential emotion processing. P300 latency is influenced by task complexity and stimulus evaluation and can more precisely indicate stimulus duration (Donchin and Coles, 1988; Duncan-Johnson and Donchin, 1982).

In this study, we expected the binge drinkers' reactivity in response to alcohol-related stimuli to be different from the reactivity in response to control stimuli, in the sense that an enhanced response to the alcohol-associated stimuli would be observable. This difference would not be observed in controls, and may affect one or more ERP components. More precisely, if attentional bias specific to the binge drinking group occurs early in an automatic/unconscious way, the P100 component should present larger amplitude for alcohol-related cues than for non alcohol-related cues. In contrast, differences in the N2b would reflect modulation starting at the level of voluntary attentional resources allocation. Finally, if extended resources are deployed to process alcohol pictures, this modulation should be observable on the P300. Attentional bias for specific cues cannot only be indexed by enhanced amplitudes of some ERP components (depending on when it appears in the cognitive processing stream), but also by longer latencies. Indeed, ERP latencies represent the timing of a cognitive process related to a stimulus, thus they index speed of information processing. Latencies of the P3b component can also be matched with the reaction times as these index higher processes. Moreover, ERP has an advantage over reaction time measures, in that the dependence on motor responses and possibly nerve conduction velocity is eliminated, making ERP measures more sensitive.

Finally, personal alcohol use by college students has been found to be correlated with the experience of pleasure while viewing a visual alcohol cue (Pulido et al., 2009). This suggests that more positive affective experiences of alcohol stimuli may cause increased alcohol use, which in turn may reinforce the positive affective responses to cues, thereby developing a cycle that will perpetuate heavy drinking. As an affective response (i.e., pleasant vs. unpleasant) to alcohol advertisements predicts alcohol consumption and escalation (Atkin et al., 1983; Snyder et al., 2006), youths who consume large amounts of alcohol may be vulnerable to the affective tint of alcohol cues, which would influence alcohol consumption. We, therefore, investigated the potential interaction between the valence of the stimuli and alcohol cue reactivity. For this purpose, we included in our oddball experimental procedure deviant images that combined alcohol beverage scenes and non-alcohol (control) scenes with neutral, negative and positive valence. We tested the hypothesis that the context of the scene would modulate the response to alcohol images, potentiating the effect of alcohol-related images, but only in the binge drinkers.

2. Methods

2.1. Participants

We first conducted a general screening phase among students at the Faculty of Psychology of Brussels (Belgium) University. Three hundred students completed a questionnaire that assessed psychological measures as well as alcohol and drug consumption characteristics. To be included in our study, students had to meet the following selection criteria: no major medical problems, no history of CNS disease (including epilepsy and history of brain trauma), no visual impairment, no past or current drug consumption (other than alcohol), no family history of alcoholism, very low alcohol consumption and absence of binge drinking habits before starting university studies but maintenance of the same drinking pattern since then. All participants were assessed for several psychological measures: State and Trait Anxiety (STAI A and B, Spielberger et al., 1983), depression (BDI, Beck and Steer, 1987) and alexithymia (TAS 20, Bagby et al., 1994). On this basis, 36 students were selected. According to their alcohol consumption characteristics while at university, students were divided into two groups, each of 18 participants: controls and binge drinkers. According to the quantitative definition

of binge drinking used in European countries, where a standard alcoholic drink equals about 10 g of alcohol, binge drinking pattern was defined in this study as the consumption of six or more standard alcoholic drinks on the same occasion at least one time per month (Crego et al., 2009). In addition to this quantity/frequency criterion, speed of alcohol consumption was also considered. Three criterions were thus used to classify subjects: quantity, frequency and speed of alcohol consumption. Participants who drank six or more standard alcoholic drinks on the same occasion, three or four times maximum per week and, during these episodes, drank at a speed of consumption of at least three drinks per hour, were classified as binge drinkers. Those who drank 1–30 days a month but never more than five standard alcoholic drinks on the same occasion and at a maximum speed of consumption of two drinks per hour, were classified as controls. It was therefore possible that a subject classified as control drank the same amount of alcohol doses per week than a binge drinker, but not at the same frequency, which made the first being a low daily consumer and the latter a binge drinker. Participants were provided with full details regarding the aims of the study and the procedure to be followed and gave their informed consent after receiving this information. The local ethical committee of Brugmann Hospital approved the study. All the participants were between the ages of 19 and 26, with normal/corrected vision, normal hearing, no medication and no history of neurological disease. We matched the groups for age, sex and psychological measures: State anxiety, trait anxiety, depression and alexithymia (as these variables have been shown to influence ERPs for emotional processing, see for sex, Campanella et al., 2004; for anxious and depressive state, Rossignol et al., 2008; for age, Vermeulen et al., 2008). The groups' characteristics are shown in Table 1.

2.2. Preparation of visual stimuli

Alcohol-related pictures and non-alcohol-related (control) pictures were used as target deviant stimuli placed among frequent neutral stimuli (see Fig. 1 for illustration). Moreover, all these deviant stimuli (alcohol and control) presented a neutral, negative or positive scene. To construct this set of picture stimuli, we started with 44 pictures, chosen from the International Affective Picture System (IAPS) (Lang et al., 1997), or from our own selection on the internet. All visual stimuli were standardized for brightness level and net colour by Photoshop 6.0 and resized to 12 × 12 cm. To select the stimuli, 40 students who did not take part in the electrophysiological investigation rated the 44 pictures for alcohol specificity and emotionality. For specificity rating, these students were asked how strongly the item was related to alcohol. The range of the specificity scale was 0 (not at all) to 5 (extremely). For emotionality rating, subjects were asked "How pleasant or unpleasant is the picture for you?". The range of the emotionality scale was 0 (very unpleasant) to 9 (very pleasant). Based on the results of this rating, six categories of pictures were created. The seven pictures with the lowest specificity and the most neutral emotionality were selected as neutral stimuli (control neutral, CN). The three pictures with the lowest specificity and the highest emotional evaluation were selected as "positive neutral, PN" stimuli. The three pictures with the lowest specificity and the more negative emotional evaluation were selected as "negative neutral, NN" stimuli. The three pictures with the highest specificity and the lowest emotionality were selected as "control alcohol, CA" stimuli. The three pictures with the highest specificity and the more positive emotional evaluation were selected as "positive alcohol, PA" stimuli. The three pictures with the highest specificity and the more negative emotional evaluation were selected as "negative alcohol, NA" stimuli.

2.3. Procedure

Having signed an informed consent document, all participants were assessed with the Beck Depression Inventory, the State-Trait Anxiety Inventory, and the Alexithymia Scale and completed a questionnaire about their alcohol and drug consumption before commencing the EEG session. The task and EEG recording were then started. Participants sat in a dark room on a chair placed 1 m from the screen. The task used was a visual oddball paradigm, consisting of four blocks. In each block, participants were confronted with one regularly repeated standard stimulus and various deviant ones. The frequent neutral stimulus was chosen from the "CN" group and was different in each block. In each block, 320 stimuli were presented: the same neutral target occurred 230 times (72%) and the three deviant pictures for each condition (CA–CN–PA–PN–NA–NN) were each presented five times (28%) (see Fig. 1 for illustration).

A practice block of pictures was shown first to adapt the subjects to the task ahead and to the experimental environment. This practice block included presentation of a simple neutral figure as the frequent stimulus, and a deviant neutral stimulus. The pictures used were not included in the four experimental blocks and data from this block were not included in the data analysis. Each picture was presented for 800 ms. A black screen was displayed between pictures, for a random duration of between 600 and 1000 ms. From the stimulus onset, participants had 1200 ms to answer. Subjects were instructed to indicate as quickly as possible the occurrence of a deviant stimulus with a right fingertap. The order of the four blocks varied across participants. Response times and percentages

Table 1
The means and standard deviations (in parentheses) of the control and binge drinkers groups' scores on the BDI, STAI-T and TAS-20 psychological tests and on alcohol consumption characteristics.

	Binge drinkers	Controls
Gender (♂:♀) ($\chi^2(1) = 1.8$; $p = .18$)	12:6	8:10
Age (year) ($t(34) = .789$; $p = .436$)	21.28(1.67)	21.94(3.17)
Number of alcohol doses per week ($t(34) = -7.101$; $p < .001$)	30.38(18.63)	1.58(1.11)
Number of drinking occasions per week ($t(34) = -6.546$; $p < .001$)	3.42(1.2)	0.89(0.91)
Duration of alcohol drinking habits (in months) ($t(34) = -1.151$; $p = .258$)	39.5(18.64)	31.5(22.7)
BDI ($t(34) = -.124$; $p = .902$)	3.00(2.4)	2.89(2.95)
STAI trait ($t(34) = -.557$; $p = .581$)	45.44(7.07)	43.78(10.55)
STAI State ($t(34) = .312$; $p = .757$)	44.89(6.28)	45.61(7.56)
TAS-20 ($t(34) = -.207$; $p = .838$)	50.16(10.01)	49.44(10.94)

BDI = Beck Depression Inventory.
STAI = State and Trait Anxiety Inventory.
STAI = State and Trait Anxiety Inventory.
TAS-20 = 20-item Toronto Alexithymia Scale.
SD = standard deviation.
One dose represents 10 g of alcohol.

Fig. 1. Illustration of the pictures used as frequent and deviant stimuli.

of correct answers were recorded. Participants were told that speed was important but not at the cost of accuracy. Only correct answers (i.e., deviant stimuli for which the subject gave a finger tap) were considered for analysis of reaction times and ERP. After the task, subjects were asked to rate the pictures they saw during the task for alcohol specificity and emotionality. The scales used were the same as the ones used for stimuli preparation (see Section 2.2). The mean scores of the valence and alcohol specificity evaluations for each stimulus in both groups are reported in Table 2.

Table 2
Means scores and SDs in parentheses of stimuli's valence (1: negative; 9: positive) and alcohol specificity evaluation (1: non-alcohol; 5: alcohol). Control alcohol, CA; control neutral, CN; negative alcohol, NA; negative neutral, NN; positive alcohol, PA; positive neutral, PN. IAPS pictures are indicated in parentheses in the control and binge drinkers groups.

	Valence		Alcohol-related	
	Controls	Bingers	Controls	Bingers
CA	4.7(0.87)	5.5(0.84)	4.3(0.55)	4.2(0.54)
CN	5.0(0.22)	4.7(0.93)	1.0(0.00)	1.0(0.31)
NA	2.0(0.58)	2.1(0.93)	4.7(0.34)	4.8(0.28)
NN	3.0(0.6)	2.7(0.55)	1.1(0.32)	1.0(0.07)
PA	7.2(0.87)	7.2(0.75)	3.9(0.60)	4.1(0.58)
PN	8.7(0.56)	8.2(1.02)	1.0(0.07)	1.0(0.00)

2.4. EEG recording and analysis

During the testing phase, the EEG was recorded by 32 electrodes mounted in an electrode Quick-Cap. Electrode positions included the standard 10–20 system locations and intermediate positions. Recordings were taken with a linked mastoid physical reference but re-referenced using a common average. The EEG was amplified by battery-operated A.N.T.® amplifiers with a gain of 30,000 and a band-pass of 0.01–100 Hz. The impedance of all electrodes was always kept below 5 kΩ. The EEG was recorded continuously (sampling rate 1024 Hz, A.N.T. Eeprobe software) and trials contaminated by EOG artifacts (mean of 8%) were manually eliminated off-line. Epochs were created starting 200 ms prior to stimulus onset and lasting for 800 ms. Data were filtered using a 30 Hz low-pass filter. To compute averages of different ERP target stimuli for each subject individually, three parameters were coded for each stimulus: (1) the valence of the stimulus (positive, negative, neutral); (2) the stimulus type (A, alcohol; C, control); and (3) the response type (keypress for deviant stimulus, no keypress for standard stimulus). A general time window was first determined globally for the identification of each ERP component on the basis of the ERP literature (90–160 ms for P100 (Pourtois et al., 2005b; Campanella and Philippot, 2006), 200–300 ms for N2b (Halgren and Marinkovic, 1995; Rossignol et al., 2007), 350–650 ms for P3b (Polich, 2004; Campanella et al., 2010). Peak selection was then conducted as follows: for each participant and each component of interest, individual peak amplitudes and maximum peak latencies were obtained for the ERPs resulting either from the waveforms evoked by the rare stimuli (P100 and P3b) or from the subtraction of waveforms evoked by frequent and deviant stimuli (N2b), and separately from the classical electrodes used to define the P100, N2b and P300 components and on which the maximum amplitudes have been recorded for these components: Oz, O1, O2, P7, P8 and POz for P100, Oz, O1, O2 for N2b (Bentin et al., 2011; Joassin et al., 2004; Rossignol et al., 2008) and Pz, P3, P4 for P3b (Polich, 2004). These values were tested using repeated measures of analysis of variance (ANOVA – Greenhouse–Geisser correction was applied when appropriate), paired sample *t*-tests and two-tailed Pearson correlations.

3. Results

All data are summarized in Tables 3–6.

3.1. Behavioural data

The participant's responses were 98% correct. Only the correct response latencies were analyzed statistically. The behavioural results are presented in Table 3. A $2 \times 3 \times 2$ ANOVA on reaction times (RTs) for correct responses was computed, with group (control,

Table 3
The means and standard deviations (in parentheses) of the reaction times (ms) for deviant stimuli detection as a function of group (control, binge drinkers), type (alcohol, non-alcohol) and valence (neutral, positive, negative).

	Neutral		Positive		Negative	
	Alcohol	Non-alcohol	Alcohol	Non-alcohol	Alcohol	Non-alcohol
Control RTs	406(44)	425(40)	409(35)	399(45)	401(42)	403(43)
Binge RTs	387(42)	409(38)	398(45)	382(43)	381(45)	384(45)

Table 4
The means and standard deviations (in parentheses) of P100 amplitudes (μV) at Oz, O1, O2, POz, P7 and P8 for deviant stimuli detection as a function of group (control, binge drinkers), type (alcohol, non-alcohol) and valence (neutral, positive, negative).

		Neutral		Positive		Negative	
		Alcohol	Non-alcohol	Alcohol	Non-alcohol	Alcohol	Non-alcohol
Control P100 ampl.	Oz	4.4(4.53)	5.8(4.7)	5.0(4.1)	4.7(4.2)	5.8(4.6)	5.3(4.6)
	O1	6.5(4.69)	6.3(4.5)	6.6(4.0)	7.5(4.3)	6.9(4.8)	6.8(4.6)
	O2	4.8(4.02)	6.0(4.8)	5.4(3.2)	5.5(4.3)	5.8(4.4)	6.2(4.9)
	POz	3.2(2.92)	2.9(3.0)	3.1(2.2)	3.2(3.1)	3.3(3.0)	2.9(2.5)
	P7	3.4(3.3)	1.8(2.7)	2.9(2.7)	2.8(3.2)	3.5(3.4)	2.9(3.1)
	P8	7.3(6.0)	6.8(5.5)	7.3(5.0)	8.6(6.2)	7.8(6.4)	8.0(5.3)
Binge P100 ampl.	Oz	5.4(4.7)	4.9(5.3)	5.1(5.6)	4.4(5.0)	4.9(5.3)	4.49(5.07)
	O1	7.4(5.8)	7.0(4.6)	8.4(5.4)	6.9(5.7)	7.0(5.5)	6.5(5.3)
	O2	7.6(4.7)	6.4(3.9)	7.8(5.2)	6.1(5.4)	6.6(5.4)	6.1(4.4)
	POz	3.0(5.4)	2.3(4.0)	3.5(4.0)	2.1(5.3)	2.2(4.6)	1.2(4.8)
	P7	3.2(3.6)	2.8(2.6)	3.7(2.9)	3.6(3.9)	3.2(3.3)	3.2(3.5)
	P8	8.1(4.4)	5.4(2.9)	7.2(4.4)	8.0(4.6)	7.4(4.3)	6.9(3.9)

Table 5
The means and standard deviations (in parentheses) of N2b amplitudes (μV) at Oz, O1 and O2 for deviant stimuli detection as a function of group (control, binge drinkers), type (alcohol, non-alcohol) and valence (neutral, positive, negative).

		Neutral		Positive		Negative	
		Alcohol	Non-alcohol	Alcohol	Non-alcohol	Alcohol	Non-alcohol
Control N2b ampl.	Oz	−6.8(3.1)	−5.3(2.1)	−4.9(3.4)	−6.4(4.0)	−4.9(2.8)	−5.5(2.0)
	O1	−3.5(3.4)	−4.7(2.3)	−3.4(2.8)	−2.7(3.1)	−2.5(2.8)	−3.1(2.8)
	O2	−7.2(3.6)	−5.2(2.4)	−5.4(2.6)	−6.3(4.2)	−5.0(3.0)	−4.8(3.4)
Binge N2b ampl.	Oz	−5.5(4.8)	−4.5(4.0)	−4.9(3.6)	−6.7(4.5)	−5.0(3.9)	−6.2(3.8)
	O1	−3.1(4.7)	−5.4(3.0)	−2.6(2.9)	−3.7(5.0)	−3.0(3.9)	−3.5(3.5)
	O2	−4.7(4.6)	−5.6(2.6)	−3.8(3.5)	−3.8(3.5)	−5.5(4.5)	−5.6(3.3)

Table 6
The means and standard deviations (in parentheses) of P3b amplitudes (μV) at Pz, P3 and P4 for deviant stimuli detection as a function of group (control, binge drinkers), type (alcohol, non-alcohol) and valence (neutral, positive, negative).

		Neutral		Positive		Negative	
		Alcohol	Non-alcohol	Alcohol	Non-alcohol	Alcohol	Non-alcohol
Control P3b ampl.	Pz	10.7(5.1)	10.2(5.6)	10.5(4.7)	10.9(4.4)	12.2(3.9)	11.6(4.7)
	P3	8.9(5.3)	7.4(4.8)	8.6(4.1)	9.1(3.6)	9.6(3.8)	9.4(4.4)
	P4	10.7(4.7)	9.7(4.6)	9.8(4.4)	11.2(4.5)	11.8(3.6)	10.8(4.1)
Binge P3b ampl.	Pz	11.8(4.9)	11.3(4.8)	12.9(5.3)	11.3(5.7)	13.7(5.0)	13.8(5.8)
	P3	9.8(5.1)	8.5(4.2)	11.5(4.7)	10.0(5.5)	11.3(5.3)	11.9(5.4)
	P4	10.8(4.6)	9.5(4.1)	11.5(4.4)	11.0(4.9)	12.6(4.7)	12.5(5.0)

binge) as the between-subject factor, and stimulus valence (C, Control; P, Positive; N, Negative) and stimulus type (A, Alcohol; N, Non-alcohol) as within-subject variables. There was no significant effect for group [$F(1,34) = 1.936$, $p = .173$, Eta-Squared = .054, observed Power = .272], but there was a significant main effect of stimulus valence [$F(2,68) = 4.164$, $p = .04$, Eta-Squared = .109]. Post hoc Bonferroni tests showed that the Positive stimuli were detected more quickly than the neutral stimuli (397 ms(6) vs. 407 ms(7 ms), $p = .048$). There was also a significant valence × type interaction [$F(2,68) = 14.852$, $p < .001$, Eta-Squared = .304,]. Paired t tests suggested that, independently of the group, the response latencies were shorter for CA stimuli than for CN stimuli [396 ms(7 ms) vs. 417 ms(6 ms), $t(35) = -6.288$, $p < .001$].

Overall, behavioural data showed that independently of their group, participants detected the deviant Positive stimuli faster than the neutral ones, and that in the neutral emotional context, reaction times were faster for alcohol- than for non-alcohol-related stimuli.

3.2. Stimuli evaluation

$2 \times 3 \times 2$ ANOVAs were computed for stimuli evaluation, with group (Control, Binge) as between-factor and stimulus valence

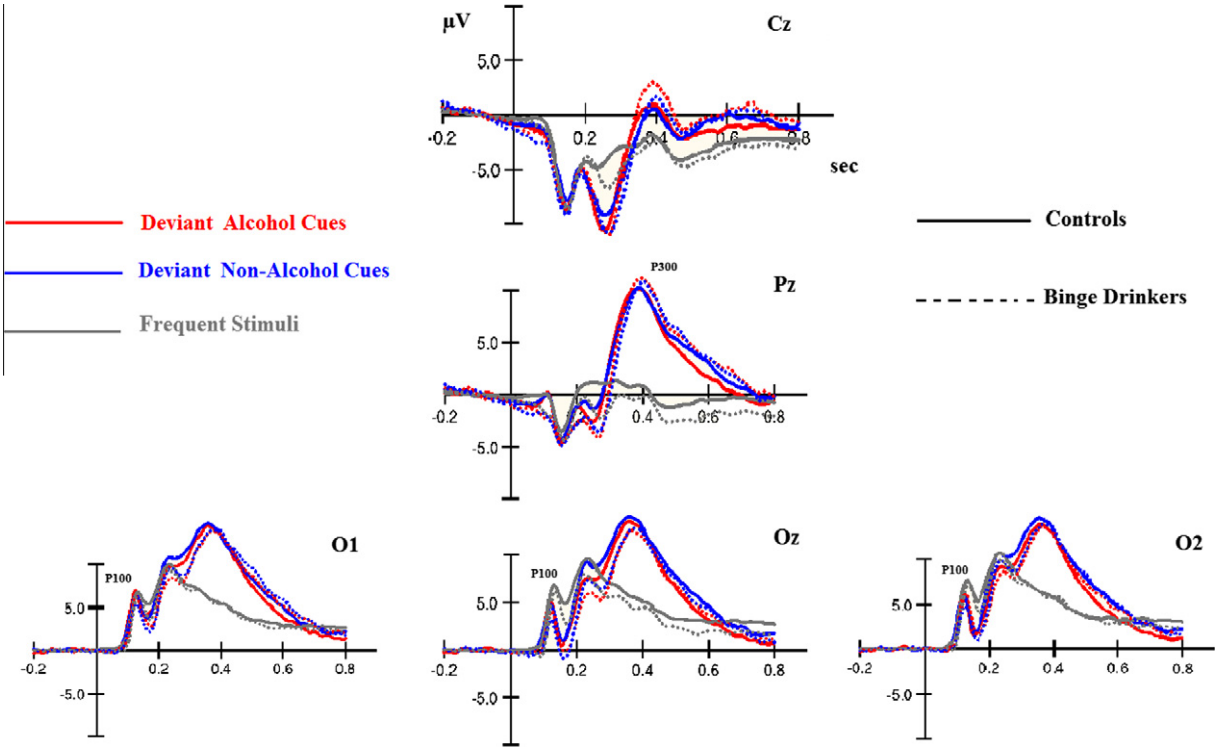

Fig. 2. Illustration of the base waveforms on three midline scalp electrodes (Cz, Pz, Oz) and two occipital electrodes (O1, O2) for frequent stimuli and the two types of deviant stimuli in the control and the binge drinker groups.

(C, Control; N, Negative; P, Positive) and stimulus type (A, Alcohol; N, Non-alcohol) as within-factors separately for valence and alcohol specificity ratings. No significant effect of group was found, but a type × group interaction was observed [$F(1,34) = 6.373$, $p = .016$, Eta-Squared = .158], which suggests that independently of the stimuli valence, controls subjects evaluated non-alcohol related pictures as more positive than alcohol-related ones [5.46(0.21) vs. 4.63(0.58); $t(17) = -6.589$, $p < .001$], whereas there was no significant difference between the two stimulus types in the binge drinkers group [5.22(0.55) vs. 5.00(.64); $t(17) = -1.077$, $p = .297$].

3.3. ERP data

Our main hypothesis was that the binge drinkers' reactivity in response to alcohol-related stimuli would be different from the reactivity in response to control stimuli, and that this may be observed in one or more ERP components. Therefore, in order to check mainly for group interactions, 2 × 3 × 3 × 2 ANOVAs were first computed separately for latencies and amplitudes, with group (Control, Binge) as between-factor and electrode (Oz, O1, O2, P7, P8 and POz for P100, Oz, O1, O2 for N2b and Pz, P3, P4 for P3b), stimulus valence (C, Control; N, Negative; P, Positive) and stimulus type (A, Alcohol; N, Non-alcohol) as within-factors, for each component of interest (P100, N2b and P3b) (see Fig. 2 for illustration).

3.3.1. Latencies

There was no significant effect of group or of group × stimulus type interaction for any ERP component's latencies, which is congruent with the absence of group differences for behavioural data ($p > .05$). As group differences were the main interest of our study, we focused on ERP amplitudes. Indeed, it is still interesting to perform statistical analyses on the amplitude of ERP components, as ERPs are able to detect even minor neurocognitive restrictions that are undetectable at the behavioural level (Rugg and Coles, 1995).

3.3.2. Amplitudes

3.3.2.1. P100. There was no significant group effect [$F(1,34) = .017$, $p = .898$; Eta-Squared = .000, observed Power = .052] but there was a significant type × group interaction [$F(1,34) = 4.337$, $p = .045$, Eta-Squared = .113], which suggests that, independent of the valence of the stimulus, P100 amplitudes were larger for alcohol stimuli compared to non-alcohol stimuli [5.78μ(0.84μ) vs. 4.93μ(0.77μ); $t(17) = -2.534$, $p = .021$] in the binge drinkers group, whereas there was no significant difference between the two stimulus types in the control group [5.19μ(0.84μ) vs. 5.23μ(0.77μ); $t(17) = .161$, $p = .874$]. Another significant type × group × electrode interaction [$F(5,170) = 8.214$, $p = .005$, Eta-Squared = .113] was found. Paired t tests suggested that the increased P100 amplitudes observed in binge drinkers for alcohol-related pictures compared to non-alcohol related ones were present on the central [Oz–POz: 4.26μ(4.65μ) vs. 3.24μ(4.44μ); $t(17) = 2.455$; $p = .025$] and on the right sided [O2–P8: 7.49μ(4.1μ) vs. 6.5μ(3.4μ); $t(17) = 2.218$; $p = .04$] scalp electrodes, whereas there was only a tendency on the left sided scalp electrodes [O1–P7: 5.59μ(3.86μ) vs. 5.04μ(3.74μ); $t(17) = 1.857$; $p = .081$]. Overall, these data showed that, compared to control participants, binge drinkers had increased P100 amplitudes in response to alcohol-related pictures compared to non-alcohol related ones (see Fig. 3) and this effect seemed to be lateralized, the differences being less pronounced on the left hemisphere.

3.3.2.2. N2b. There was no significant group effect [$F(1,34) = .098$, $p = .756$.] or any group × stimulus type interaction for the N2b component.

3.3.2.3. P3b. There was no significant group effect [$F(1,34) = .695$, $p = .41$] but analyses revealed a main effect of valence

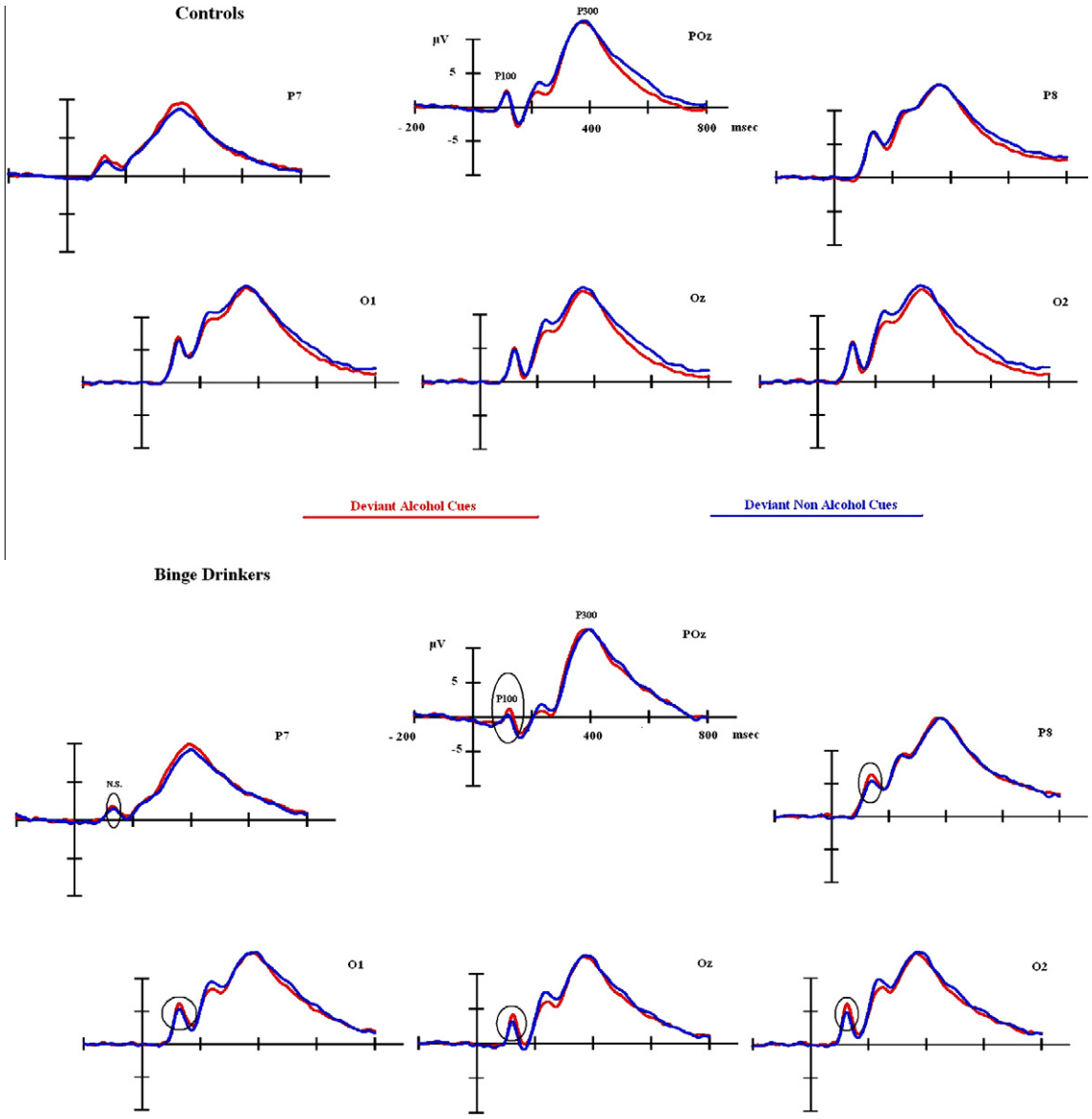

Fig. 3. Illustration of the P100 component on six posterior electrodes (P7, O1, POz, Oz, P8, O2) for alcohol-related cues and non-alcohol-related cues in the control and the binge drinker groups.

[$F(2,68) = 9.705$, $p < .001$, Eta-Squared = .222]. Post hoc Bonferroni tests showed that, in both groups, negative stimuli elicited significant larger P3b amplitudes compared to neutral ($p = .002$) or Positive stimuli ($p = .002$); no differences were found between amplitudes elicited by neutral compared to Positive stimuli ($p = .975$).

Thus, these data showed that in both groups, independent of the stimulus type, negative stimuli elicited larger P3b amplitudes than Positive and Neutral stimuli.

3.3.3. Correlations

Pearson correlations were performed to test the hypothesis that the increased P100 amplitude to alcohol-related cues observed in the binge drinker group is linked to the period of time they have been drinking (in months) and the amount of alcohol doses consumed per week. The results showed that the longer the duration of binge drinking habits, the larger the P100 amplitude to alcohol-related cues ($r = .666$; $p = .003$), and the greater the number of doses consumed per week, the larger the P100 amplitude to alcohol-related cues ($r = .491$; $p = .039$).

4. Discussion

The primary aim of the present study was to examine whether binge drinking university students have abnormal cognitive processing of alcohol-related cues compared to non-binge drinkers. ERP data provided evidence that binge drinkers and control participants differ with respect to their electrophysiological response to alcohol-related pictures.

The differences between binge drinkers and control participants specifically involved the P100 component. P100 amplitudes elicited by alcohol-related pictures were significantly larger than those elicited by neutral pictures in individuals with binge drinking habits, whereas there were no significant differences in the P100 amplitudes of controls with the different stimuli. An increase in P100 amplitude reflects an increased recruitment of extrastriate neurons of the visual cortex during processing of the stimulus because of an increased allocation of attention to the visual stimulus (e.g., Luck et al., 2000). It is assumed that the P100 component indexes an early, still preconscious stage of

attention allocation (Hillyard et al., 1995). In the present study, later cognitive processing stages expressed by the N2b and the P3b components were unaffected by the stimulus type; the response to the different cues was not significantly different for these waves in either group. Therefore, the elevated electrophysiological reactivity in response to alcohol-relevant pictures observed in this experiment may be interpreted as an indication of a shift in attention toward alcohol-related stimuli that occurs at an early phase without awareness by the binge drinkers.

This early discrimination of cues was notably stronger in the right hemisphere. This observation is consistent with previous studies that used current source density analysis and found that early emotion discrimination was bilateral over the occipital cortex, with an adjacent source over the right parietal cortex (Junghöfer et al., 2001; Schupp et al., 2003). fMRI research has also demonstrated greater activity over occipital and parietal sites for emotionally relevant pictures, with a right hemisphere emphasis (Lang et al., 1998). Our results, however, do not provide precise spatial information, and it may be appropriate to use multichannel ERP recording or fMRI to improve spatial resolution.

It is important to note that, even though such an increase in the P100 component was not observed in the control group, the results showed that, at a behavioural level, despite the fact that controls subjects evaluated alcohol-related stimuli as less positive than non-alcohol related ones, all participants showed significantly faster responses to alcohol-related stimuli compared to non-alcohol-related ones when the context was neutral. This suggests that all subjects are more vigilant to alcohol-related cues leading to a more salient conscious processing. This observation is in line with other studies in which behavioural measures showed that alcohol-related cues tend to capture attention in heavy but also in light drinkers and that the phenomenon often correlates with the level of alcohol involvement (Cox et al., 1999, 2003; Townshend and Duka, 2001; Bruce and Jones, 2004; Field et al., 2004). Such a link was apparent in our sample, as the P100 modulations in binge drinkers increased with the number of drinks consumed per week. This also highlights an important distinction that can be made between the two groups of the study: whereas a greater motivational-driven reaction to alcohol related-stimuli compared to non-alcohol stimuli appeared at a conscious behavioural level in all participants no matter what pattern of alcohol consumption they had, a specific excessive attention allocation toward alcohol-related stimuli occurred without conscious awareness only in the binge drinkers group.

To our knowledge, only two studies have previously attempted to explore the precise chronology of cognitive processing of alcohol-associated cues in non-dependent alcohol users. Ceballos et al. (2009), who used eye-tracking methodology, revealed a positive relationship between the quantity and frequency of alcohol consumption among college students and the amount of time that students spent viewing alcohol-related images compared to control ones, as well as the frequency with which they were attracted to the alcohol-related scene within the first milliseconds. Ceballos et al., therefore, suggested that the presence of bias toward alcohol-related stimuli occurred at both an automatic stage and a controlled stage of information processing. Herrmann et al. (2001) investigated cue reactivity together with ERP recordings in non-dependent drinkers and identified heightened P300 amplitude but a diminished N100 in response to alcohol-related words in heavy drinkers (compared to light drinkers). This finding indicated a greater salience of alcohol-related stimuli in late processing stages, but a shift away from alcohol-related stimuli in early stages of attention. However, a direct comparison of these studies with our present study is not appropriate, as they investigated different populations. In the study by Hermann et al., heavy drinkers were invariably adults who had been drinking on a regular basis for a longer time than binge drinkers. In the study by Ceballo and colleagues, the experimental population was a group of young college students with drinking styles varying from 1 to 3 drinks on an average day, which did not permit knowledge of the drinking patterns of users. Therefore, when measuring the cognitive consequences of alcohol consumption, it is necessary to focus more directly on defined drinking patterns rather than on the global amount of alcohol consumed in a certain period of time. For example, it would be appropriate to compare adolescent binge drinkers with adolescents drinking the same global amount of alcohol in a week but in a less concentrated consumption manner. According to Robinson and Berridge's theory, which suggested that attentional bias develops progressively with each new occasion of substance use, one could expect that heavy but more regular drinkers would have more opportunities to pair alcohol consumption with alcohol-associated stimuli for these to become conditioned and, therefore, to show more intense reactivity than less regular drinkers. The link between the period of time during which binge drinkers had started to drink in this fashion and the degree of enhanced P100 for alcohol cues found in our study is also consistent with this idea.

These present findings suggest that attention bias toward alcohol stimuli involves separate processes that can be altered selectively by different aspects of both alcohol use and history. Further research assessing the chronometry of cognitive bias toward alcohol-related indices in different types of alcohol users and abusers is needed to create and to prescribe appropriate tools that will directly target the cognitive processes responsible for cue reactivity and extinguish the cue response. Procedures inspired, for example, from research into social phobias could be envisaged to tackle binge drinking. Indeed, recent studies suggest that phobic individuals repeatedly trained to disengage attention from threat cues, acquire abilities to turn their attention away from similar negative cues and are, therefore, able to process less threatening aspects of the situation (e.g., see the Attention Modification Program, Amir et al., 2008). In alcoholism, varieties of attentional retraining (e.g., ABM, Fadardi and Cox, 2009; Schoenmakers et al., 2010; AAT, Wiers et al., 2009) have also shown promise in reducing the risk of relapse in alcoholics. With this in mind, alcohol cue tasks could be used to identify individuals at risk of heavy alcohol abuse and in need of rapid alcohol prevention interventions. Moreover, knowing which stage(s) of the processing stream is (are) affected by the cues would permit to apply the best attentional retraining procedure, focusing on the altered stage(s) of each individual.

The second goal of the present study was to investigate whether the valence of the stimuli would have an influence on alcohol-cue reactivity. Our results did not show any evidence of an interaction between stimuli valence and stimuli type, indicating that the valence of the cues had no influence on the observed enhanced electrophysiological or behavioural reactivity elicited by alcohol cues. However, analyses showed that in all participants, Positive stimuli led to shorter reaction times, which suggests a higher attractive power of these stimuli, thereby influencing the processing speed. This observation has been previously reported in numerous studies that have shown, for example, that facial expressions of happiness elicit faster behavioural responses (Kirita and Endo, 1995; Leppänen et al., 2003; Leppänen and Hietanen, 2004). It is also well-known that positive emotions facilitate approach-related behaviours (Cacioppo et al., 1999; Lang et al., 1997). Thus, it is not surprising that subjects responded faster to positive cues than to other cues. In addition, ERP data showed that, independently of the stimulus type, negative stimuli lead to increased P3b amplitudes compared to positive and neutral ones, which reflects a greater salience of these stimuli, indicating the presence of so

called "negativity bias". This tendency to pay more attention and give more weight to negative than to positive information is explained by the necessity of reacting preferentially to negative stimuli compared to positive ones because of the potentially harmful consequences of a negative stimulus. Existence of this bias is supported by a number of neurophysiological studies (e.g., Pourtois and Vuilleumier, 2006; Schupp et al., 2004).

5. Limitations and future directions

Firstly, self-report measures about alcohol consumption are mainly accepted as reliable (Townshend and Duka, 2002; Del Boca and Noll, 2000). However, it has been argued that self-report binge drinking estimation may not be representative of actual alcohol intake (Walker and Cosden, 2007). To make sure alcohol intake and any other drug use was as accurately reported as possible in this study, strong assurances of confidentiality were offered and questions were formulated as clearly and directly as possible. Nevertheless, we cannot certify the absence of errors due to difficulties in recall of drinking amounts. Also, we cannot be sure that social desirability did not keep people from honestly report their drug use. Prospective diaries completed on a daily basis and physiological assessments as hair sample or urine testing could be used in further research to get around these limitations. Secondly, the present study was designed to examine binge drinking effects in university students. To address this issue, all subjects were obtained from universities and alcohol abstainers, polysubstance users as well as individuals with any family history for alcoholism were excluded. A generalization of these findings to the general population may therefore have its limitations. Finally, as several neuroimaging and neurofunctional studies on alcohol consumption have noted that women with AUDs are more sensitive to the neurotoxic effects of alcohol than men (e.g., Caldwell et al., 2005; Hommer et al., 2003; Mann et al., 2005; Medina et al., 2008; Townshend and Duka, 2005), we initially assessed the possible influence of gender in the results. The fact that we did not discern any significant gender-related effects in our binge drinkers sample could be due to the small number of subjects in the group and should be investigated properly in further studies.

6. Conclusion

In conclusion, the present study provides evidence for an amplification of early visual processing of alcohol cues in binge drinkers, indicating that alcohol cue reactivity is not a phenomenon limited to alcohol-dependent individuals. Although the observed alcohol-related reactivity among episodic heavy drinking adolescents may be more subtle compared to alcohol-dependent or treatment-seeking populations, given the absence of significant behavioural manifestation of the bias, pursuing these risky drinking habits (started in adolescence) could generate permanent neurobiological modulations and/or the development of a chronic addictive state. It seems therefore crucial that further research addressing alcohol-cue reactivity in non-clinical populations focuses more on (1) defining and comparing different types of alcohol users and abusers, and (2) assessing the chronometry of cognitive bias toward alcohol-related indices to create adequate prevention programs to target the cognitive processes underlying cue reactivity.

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References

Amir N, Weber G, Beard C, Bomyea J, Taylor CT. The effect of a single-session attention modification program on response to a public-speaking challenge in socially anxious individuals. J Abnormal Psychol 2008;117:860–8.
Atkin CK, Neuendorf K, Mcdermott S. The role of alcohol advertising in excessive and hazardous drinking. J Drug Educ 1983;13:313–25.
Bachman JG, O'Malley PM, Schulenberg JE, Johnston LD, Bryant AL, Merline AG. The decline of substance use in young adulthood, changes in social activities, roles, and beliefs. Mawah, NJ: Lawrence Erlbaum Associates, Inc; 2002.
Bagby MR, Taylor GJ, Parker JD. The 20-item Toronto Alexithymia Scale-II. Convergent, discriminant, and concurrent validity.. J Psychosom Res 1994;38:33–40.
Batty M, Taylor MJ. Early processing of the six basic facial emotional expressions. Cogn Brain Res 2003;17:613–20.
Bauer D, Cox WM. Alcohol-related words are distracting to both alcohol abusers and non-abusers in the Stroop colour-naming task. Addiction 1998;93:1539–42.
Beck AT, Steer RA. Beck depression inventory manual. San Antonio: Psychological Corporation; 1987.
Bentin S, Allison T, Puce A, Perez E, McCarthy G. Electrophysiological studies of face perception in humans. J Cogn Neurosci 2011;8:551–65.
Bonomo YA, Bowes G, Coffey C, Carlin JB, Patton GC. Teenage drinking and the onset of alcohol dependence: a cohort study over 7 years. Addiction 2004;99:1520–8.
Brown SA, Tapert SF. Adolescence and the trajectory of alcohol use: basic to clinical studies. Ann NY Acad Sci 2004:234–44.
Bruce G, Jones BT. A pictorial Stroop paradigm reveals an alcohol attentional bias in heavier compared to lighter social drinkers. J Psychopharmacol 2004;18:527–33.
Bruder GE, Stewart JW, Tenke CE, McGrath PJ, Leite P, Bhattacharya N, et al. Electroencephalographic and perceptual asymmetry differences between responders and nonresponders to an SSRI antidepressant. Biol Psychiatry 2001;49:416–25.
Cacioppo JT, Gardner WL, Berntson GG. The affect system has parallel and integrative processing components: form follows function. J Pers Soc Psychol 1999;76:839–55.
Caldwell LC, Schweinsburg AD, Nagel BJ, Barlett VC, Brown SA, Tapert SF. Gender and adolescent alcohol use disorders on BOLD (blood oxygen level dependent) response to spatial working memory. Alcohol Alcsm 2005;40(3):194–200.
Campanella S, Philippot P. Insights from ERPs into emotional disorders: an affective neuroscience perspective. Psychol Belg 2006;46:37.
Campanella S, Gaspard C, Debatisse D, Bruyer R, Crommelinck M, Guerit J. Discrimination of emotional facial expressions in a visual oddball task: an ERP study. Biol Psychol 2002;59:171–86.
Campanella S, Rossignol M, Mejias S, Joassin F, Maurage P, Debatisse D, et al. Human gender differences in an emotional visual oddball task: an ERP study. Neurosci Lett 2004;367:14–8.
Campanella S, Vanhoolandt ME, Philippot P. Emotional deficit in subjects with psychopathic tendencies as assessed by the Minnesota Multiphasic Personality Inventory-2: an event-related potentials study. Neurosci Lett 2005;373:26–31.
Campanella S, Bruyer R, Froidbise S, Rossignol M, Joassin F, Kornreich C, et al. Is two better than one? A cross-modal oddball paradigm reveals greater sensitivity of the P300 to emotional face-voice associations. Clin Neurophysiol 2010;121:1855–62.
Ceballos NA, Komogortsev OV, Turner GM. Ocular imaging of attentional bias among college students: automatic and controlled processing of alcohol-related scenes. J Stud Alcohol Drugs 2009;70:652–9.
Chassin L, Pitts SC, Prost J. Binge drinking trajectories from adolescence to emerging adulthood in a high-risk sample: predictors and substance abuse outcomes. J Consult Clin Psychol 2002;70:67–78.
Chen K, Kandel DB. The natural history of drug use from adolescence to the mid-thirties in a general population sample. Am J Public Health 1995;85:41–7.
Courtney KE, Polich J. Binge drinking effects on EEG in young adult humans. Int J Environ Res Public Health 2010;7:2325–36.
Cox WM, Yeates GN, Regan CM. Effects of alcohol cues on cognitive processing in heavy and light drinkers. Drug Alcohol Depend 1999;55:85–9.
Cox WM, Hogan LM, Kristian MR, Race JH. Alcohol attentional bias as a predictor of alcohol abusers' treatment outcome. Drug Alcohol Depend 2002;68:237–43.
Cox WM, Brown MA, Rowlands LJ. The effects of alcohol cue exposure on non-dependent drinkers' attentional bias for alcohol-related stimuli. Alcohol Alcohol 2003;38:45–9.
Crego A, Holguín SR, Parada M, Mota N, Corral M, Cadaveira F. Binge drinking affects attentional and visual working memory processing in young university students. Alcohol Clin Exp Res 2009;33:1870–9.
Crego A, Rodriguez-Holguín S, Parada M, Mota N, Corral M, Cadaveira F. Reduced anterior prefrontal cortex activation in young binge drinkers during a visual working memory task. Drug Alcohol Depend 2010;109:45–56.
Crews FT, Braun CJ, Hoplight B, Switzer RC, Knapp DJ. Binge ethanol consumption causes differential brain damage in young adolescent rats compared with adult rats. Alcohol Clin Exp Res 2000;24:1712–23.
Crews FT, Mdzinarishvili A, Kim D, He J, Nixon K. Neurogenesis in adolescent brain is potently inhibited by ethanol. Neuroscience 2006;137:437–45.
Dawson DA, Grant BF, Stinson FS, Chou PS. Maturing out of alcohol dependence: the impact of transitional life events. J Stud Alcohol 2006;67:195–203.

De Bellis MD, Clark DB, Beers SR, Soloff PH, Boring AM, Hall J, et al. Hippocampal, volume in adolescent-onset alcohol use disorders. Am J Psychiatry 2000;157:737.
De Bellis MD, Narasimhan A, Thatcher DL, Keshavan MS, Soloff P, Clark DB. Prefrontal cortex, thalamus, and cerebellar volumes in adolescents and young adults with adolescent-onset alcohol use disorders and comorbid mental disorders. Alcohol Clin Exp Res 2005;29:1590–600.
Del Boca F, Noll J. Truth or Consequences: The validity of self-report data in health services research on addictions. Addiction 2000;95:347–60.
Donchin E, Coles MGH. Is the P300 component a manifestation of context updating? Behav Brain Sci 1988;11:357–427.
Duncan CC. Current issues in the application of P300 to research on schizophrenia. In: Straube E, Hahlurg K, editors. Schizophrenia: model. Vulnerability and intervention. New York: Springer-Verlag; 1990. p. 117–34.
Duncan-Johnson CC, Donchin E. The P300 component of the event-related brain potential as an index of information processing. Biol Psychol 1982;14:1–52.
Ehlers CL, Phillips E, Finnerman G, Gilder D, Lau P, Criado J. P3 components and adolescent binge drinking in Southwest California Indians. Neurotoxicol Teratol 2006;29:153–63.
Eimer M, Holmes A. Event-related brain potential correlates of emotional face processing. Neuropsychologia 2007;45:15–31.
Enoch M. Genetic and environmental influences on the development of alcoholism. Ann NY Acad Sci 2006:193–201.
Enoch MA, Goldman D. Problem drinking and alcoholism: diagnosis and treatment. Am Fam Physician 2002;65:441–54.
Fadardi JS, Cox M. Reversing the sequence: reducing alcohol consumption by overcoming alcohol attentional bias. Drug Alcohol Depend 2009;10:137–45.
Field M, Cox WM. Attentional bias in addictive behaviors: a review of its development, causes, and consequences. Drug Alcohol Depend 2008;97:1–20.
Field M, Mogg K, Zetteler J, Bradley BP. Attentional biases for alcohol cues in heavy and light social drinkers: the roles of initial orienting and maintained attention. Psychopharmacology 2004;176:88–93.
Fischler IS, Bradley MM. Event-related potential studies of language and emotion: words, phrases and task effects. Prog Brain Res 2006;156:185–204.
Grant BF, Dawson DA. Age of onset of drug use and its association with DSM-IV drug abuse and dependence: results from the National Longitudinal Alcohol Epidemiologic Survey. J Subst Abuse 1998;10:163–73.
Halgren E. Human evoked potentials. In: Boulton AA, Baker GB, Vanderwolf C, editors. Neurophysiological techniques – applications to neural systems. Clifton, NJ: Humana; 1990. p. 147–275.
Halgren E, Marinkovic K. Neurophysiological networks integrating human emotions. In: Gazzaniga MS, editor. The cognitive neurosciences. 1st ed. Cambridge: MIT Press; 1995. p. 1137–51.
Haller M, Handley E, Chassin L, Bountress K. Developmental cascades: linking adolescent substance use, affiliation with substance use promoting peers, and academic achievement to adult substance use disorders. Dev Psychopathol 2010;22:899–916.
Harford TC, Grant B, Yi H, Chen CM. Patterns of DSM-IV alcohol abuse and dependence criteria among adolescents and adults: results from the 2001 National Household Survey on Drug Abuse. Alcohol Clin Exp Res 2005;29:810–28.
Herbert C, Junghöfer M, Kissler J. Event related potentials to emotional adjectives during reading. Psychophysiology 2008;45:487–98.
Herrmann MJ, Weijers HG, Wiesbeck GA, Boning J, Fallgatter AJ. Alcohol cue-reactivity in heavy and light social drinkers as revealed by event-related potentials. Alcohol Alcohol 2001;36:588.
Hiller-Sturmhofel S, Swartzwelder HS. Alcohol's effects on the adolescent brain. Alcohol Res Health 2004;28:213–21.
Hillyard SA, Mangun GR, Woldorff MG, Luck SJ. Neural systems mediating selective attention. In: Gazzaniga MS, editor. The cognitive neurosciences. Cambridge, MA: MIT Press; 1995. p. 665–81.
Hillyard SA, Anllo-Vento L, Clark VP, Heinze HJ, Luck SJ, Mangun GR. Neuroimaging approaches to the study of visual attention: a tutorial. In: Kramer AF, Coles MGH, Logan GD, editors. Converging operations in the study of visual selective attention. Washington, DC: American Psychological Association; 1996. p. 107–38.
Hillyard SA, Vogel EK, Luck SJ. Sensory gain control (amplification) as a mechanism of selective attention: electrophysiological and neuroimaging evidence. Philos Trans R Soc Lond B 1998;353:1257–70.
Hingson RW, Heeren T, Winter MR. Age of alcohol-dependence onset: associations with severity of dependence and seeking treatment. Pediatrics 2006;118:755–63.
Hoffman JE. Event-related potentials and automatic and controlled processes. In: Rohrbaugh JW, Parasuraman R, Johnson Jr R, editors. Event related brain potentials. New York: Oxford University Press; 1990. p. 145–57.
Hommer DW, Momenan R, Kaiser E, Rawlings RR. Evidence for a gender-related effect of alcoholism on brain volumes. Focus 2003;1:171.
Hunt WA. Are binge drinkers more at risk of developing brain damage? Alcohol (Fayetteville, NY) 1993;10:559–61.
Jennison KM. The short-term effects and unintended long-term consequences of binge drinking in college: a 10-year follow-up study. Am J Drug Alcohol Abuse 2004;30:659–84.
Joassin F, Maurage P, Bruyer R, Crommelinck M, Campanella S. When audition alters vision: an event-related potential study of the cross-modal interactions between faces and voices. Neurosci Lett 2004;369:132–7.
Johnston LD, O'Malley PM, Bachman JG. National survey results on drug use from the monitoring the future study, 1975–1995. College students and young adults, vol. II. U.S. Department of Health and Human Services: NIH, 1997. p. 98–4140.
Junghöfer M, Bradley MM, Elbert TR, Lang PJ. Fleeting images: a new look at early emotion discrimination. Psychophysiology 2001;38:175–8.
Kaplan RF, Cooney NL, Baker LH, Gillespie RA. Reactivity to alcohol-related cues: physiological, subjective responses in alcoholics, nonproblem drinkers. J Stud Alcohol Drugs 1985;46:267.
Keller S, Maddock JE, Laforge RG, Velicer WF, Basler H. Binge drinking and health behavior in medical students. Addict Behav 2007;32:505–15.
Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, Walters EE. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. Arch Gen Psychiatry 2005;62:593–602.
Kilty KM. Drinking styles of adolescents and young adults. J Stud Alcohol Drugs 1990;51:556.
King A, Munisamy G, de Wit H, Lin S. Attenuated cortisol response to alcohol in heavy social drinkers. Int J Psychophysiol 2006;59:203–9.
Kirita T, Endo M. Happy face advantage in recognizing facial expressions. Acta Psychol 1995;89:149–63.
Knight RT, Grabowecky MF, Scabini D. Role of human prefrontal cortex in attention control. In: Jasper HH, Riggio S, Goldman-Rakic PS, editors. Epilepsy and the functional anatomy of the frontal lobe, Sage, New York; 1995. p. 21–36.
Lang PJ, Bradley MM, Cuthbert BN. International affective picture system (IAPS): technical manual and affective ratings. NIMH Center for the Study of Emotion and Attention; 1997.
Lang PJ, Bradley MM, Fitzsimmons JR, Cuthbert BN, Scott JD, Moulder B, et al. Emotional arousal and activation of the visual cortex: an fMRI analysis. Psychophysiology 1998;35:199–210.
Lange JE, Clapp JD, Turrisi R, Reavy R, Jaccard J, Johnson MB, et al. College binge drinking: what is it? Who does it? Alcohol Clin Exp Res 2002;26:723–30.
Leppänen JM, Hietanen JK. Positive facial expressions are recognized faster than negative facial expressions, but why? Psychol Res 2004;69:22–9.
Leppänen JM, Tenhunen M, Hietanen JK. Faster choice-reaction times to positive than to negative facial expressions: the role of cognitive and motor processes. Int J Psychophysiol 2003;17:113–23.
Li T, Hewitt BG, Grant BF. Alcohol use disorders and mood disorders: a National Institute on Alcohol Abuse and Alcoholism perspective. Biol Psychiatry 2004;56:718–20.
Li T, Hewitt BG, Grant BF. Is there a future for quantifying drinking in the diagnosis, treatment, and prevention of alcohol use disorders? Alcohol Alcohol 2007;42:57–63.
Luck SJ, Woodman GF, Vogel EK. Event-related potential studies of attention. Trends Cognit Sci 2000;4:432–40.
Mann K, Ackermann K, Croissant B, Mundle G, Nakovics H, Diehl A. Neuroimaging of gender differences in alcohol dependence: are women more vulnerable? Alcohol Clin Exp Res 2005;29:896–901.
Maurage P, Philippot P, Verbanck P, Noel X, Kornreich C, Hanak C, et al. Is the P300 deficit in alcoholism associated with early visual impairments (P100, N170)? An oddball paradigm. Clin Neurophysiol 2007;118:633–44.
Maurage P, Pesenti M, Philippot P, Joassin F, Campanella S. Latent deleterious effects of binge drinking over a short period of time revealed only by electrophysiological measures. J Psychiatry Neurosci 2009;34:111–8.
Medina KL, McQueeny T, Nagel BJ, Hanson KL, Schweinsburg AD, Tapert SF. Prefrontal cortex volumes in adolescents with alcohol use disorders: unique gender effects. Alcohol Clin Exp Res 2008;32:386–94.
Mejias S, Rossignol M, Debatisse D, Streel E, Servais L, Guérit JM, et al. Event-related potentials (ERPs) in ecstasy (MDMA) users during a visual oddball task. Biol psychol 2005;69:333–52.
Miller JW, Naimi TS, Brewer RD, Jones SE. Binge drinking and associated health risk behaviors among high school students. Pediatrics 2007;119:76–85.
Monti PM, Miranda R, Nixon K, Sher KJ, Swartzwelder HS, Tapert SF, et al. Adolescence: booze, brains, and behavior. Alcohol Clin Exp Res 2005;29:207–20.
Näätänen R, Picton TW. N2 and automatic versus controlled processes. Electroencephalogr Clin Neurophysiol 1986;38:169–86.
Nagel BJ, Schweinsburg A, Phan V, Tapert SF. Reduced hippocampal volume among adolescents with alcohol use disorders without psychiatric comorbidity. Psychol Res 2005;139:181.
Noël X, Colmant M, Van Der Linden M, Bechara A, Bullens Q, Hanak C, et al. Time course of attention for alcohol cues in abstinent alcoholic patients: the role of initial orienting. Alcohol Clin Exp Res 2006;30:1871–7.
Obernier JA, Bouldin TW, Crews FT. Binge ethanol exposure in adult rats causes necrotic cell death. Alcohol Clin Exp Res 2002;26:547–57.
Oscar-Berman M, Marinković K. Alcohol: effects on neurobehavioral functions and the brain. Neuropsychol Rev 2007;17:239–57.
Pascual M, Blanco AM, Cauli O, Minarro J, Guerri C. Intermittent ethanol exposure induces inflammatory brain damage and causes long-term behavioural alterations in adolescent rats. Eur J Neurosci 2007;25:541–50.
Payne TJ, Rychtarik RG, Rappaport NB, Smith PO, Etscheidt M, Brown TA, et al. Reactivity to alcohol-relevant beverage and imaginal cues in alcoholics. Addict Behav 1992;17:209–17.
Polich J. Clinical application of the P300 event-related brain potential. Phys Med Rehabil Clin N Am 2004;15:133–61.
Polich J. Updating P300: an integrative theory of P3a and P3b. Clin Neurophysiol 2007;118:2128–48.

Pomerleau OF, Fertig J, Baker L, Cooney N. Reactivity to alcohol cues in alcoholics and non-alcoholics: implications for a stimulus control analysis of drinking. Addict Behav 1983;8:1–10.
Pourtois G, Vuilleumier P. Dynamics of emotional effects on spatial attention in the human visual cortex. Prog Brain Res 2006;156:67–91.
Pourtois G, Dan E, Grandjean D, Sander D, Vuilleumier P. Enhanced extrastriate visual response to band-pass spatial frequency filtered fearful faces: time course and topographic evoked. Hum Brain Mapp 2005a;26:65–79.
Pourtois G, Thut G, Grave de Peralta R, Michel C, Vuilleumier P. Two electrophysiological stages of spatial orienting towards fearful faces: early temporo-parietal activation preceding gain control in extrastriate visual cortex. NeuroImage 2005b;26:149–63.
Presley CA, Pimentel ER. The introduction of the heavy and frequent drinker: a proposed classification to increase accuracy of alcohol assessments in postsecondary educational settings. J Stud Alcohol 2006;67:324–31.
Pulido C, Mok A, Brown SA, Tapert SF. Heavy drinking relates to positive valence ratings of alcohol cues. Add Biol 2009;14:65–72.
Robinson TE, Berridge KC. The neural basis of drug craving: an incentive-sensitization theory of addiction. Brain Res Brain Res Rev 1993;18:247–91.
Robinson TE, Berridge KC. Incentive-sensitization and addiction. Addiction 2001;96:103–14.
Rossignol M, Anselme C, Vermeulen N, Philippot P, Campanella S. Categorical perception of anger and disgust facial expression is affected by non-clinical social anxiety: an ERP study. Brain Res 2007;1132:166–76.
Rossignol M, Philippot P, Crommelinck M, Campanella S. Visual processing of emotional expressions in mixed anxious-depressed subclinical state: an event-related potential study on a female sample. Neurophysiol Clin 2008;38:267–75.
Rugg MD, Coles MGH. Electrophysiology of mind: event-related brain potentials and cognition. Oxford: Oxford University Press, Oxford Psychology Series; 1995.
Ryan F. Attentional bias and alcohol dependence: a controlled study using the modified Stroop paradigm. Addict Behav 2002;27:471–82.
Sara G, Gordon E, Kraiuhin C, Coyle S, Howson A, Meares R. The P300 ERP component: an index of cognitive dysfunction xin depression? J Affect Disorders 1994;31:29–38.
Schoenmakers TM, Tim M, de Bruin M, Lux IFM, Goertz AG, Van Kerkhof DHAT, Wiers RW. Clinical effectiveness of attentional bias modification training in abstinent alcoholic patients. Drug Alcohol Depend 2010;109:30–6.
Schupp HT, Junghöfer M, Weike AI, Hamm AO. Emotional facilitation of sensory processing in the visual cortex. Psychol Sci 2003;14:7–13.
Schupp HT, Ohman A, Junghöfer M, Weike AI, Stockburger J, Hamm AO. The facilitated processing of threatening faces: an ERP analysis. Emotion 2004;4:189–200.
Schweinsburg AD, Schweinsburg BC, Cheung EH, Brown GG, Brown SA, Tapert SF. FMRI response to spatial working memory in adolescents with comorbid marijuana and alcohol use disorders. Drug Alcohol Depend 2005;79:201–10.
Schweinsburg AD, McQueeny T, Nagel BJ, Eyler LT, Tapert SF. A preliminary study of functional magnetic resonance imaging response during verbal encoding among adolescent binge drinkers. Alcohol 2011a;44:111–7.
Schweinsburg AD, Schweinsburg BC, Nagel BJ, Eyler LT, Tapert SF. Neural correlates of verbal learning in adolescent alcohol and marijuana users. Addiction 2011b;106:564–73.
Sharma D, Albery IP, Cook C. Selective attentional bias to alcohol related stimuli in problem drinkers and non-problem drinkers. Addiction 2001;96:285–95.
Snyder LB, Milici FF, Slater M, Sun H, Strizhakova Y. Effects of alcohol advertising exposure on drinking among youth. Arch Pediatr Adolesc Med 2006;160:18–24.
Spielberger CD, Gorsuch RL, Lusthene RE. Manual for the State and Trait Anxiety Inventory. Palo Alto: Consulting Psychologist Press; 1983.
Stormark KM, Laberg JC, Nordby H, Hugdahl K. Alcoholics' selective attention to alcohol stimuli: automated processing? J Stud Alcohol Drugs 2000;61:18.
Suresh S, Porjesz B, Chorlian DB, Choi K, Jones KA, Wang K, et al. Auditory P3 in female alcoholics. Alcohol Clin Exp Res 2003;27:1064–74.
Tapert SF, Brown SA. Neuropsychological correlates of adolescent substance abuse: four-year outcomes. J Int Neuropsychol Soc 1999;5:481–93.
Tapert SF, Granholm E, Leedy NG, Brown SA. Substance use and withdrawal: neuropsychological functioning over 8 years in youth. J Int Neuropsychol Soc 2002;8:873–83.
Tapert SF, Cheung EH, Brown GG, Frank LR, Paulus MP, Schweinsburg AD, et al. Neural response to alcohol stimuli in adolescents with alcohol use disorder. Arch Gen Psychiatry 2003;60:727–35.
Tapert SF, Brown GG, Baratta MV, Brown SA. FMRI BOLD response to alcohol stimuli in alcohol dependent young women. Addict Behav 2004;29:33–50.
Thomas SE, Drobes DJ, Deas D. Alcohol cue reactivity in alcohol-dependent adolescents. J Stud Alcohol 2005;66:354–61.
Tokunaga S, Silvers JM, Matthews DB. Chronic intermittent ethanol exposure during adolescence blocks ethanol-induced inhibition of spontaneously active hippocampal pyramidal neurons. Alcohol Clin Exp Res 2006;30:1–6.
Townshend JM, Duka T. Attentional bias associated with alcohol cues: differences between heavy and occasional social drinkers. Psychopharmacology 2001;157:67–74.
Townshend JM, Duka T. Patterns of alcohol drinking in a population of young social drinkers: a comparison of questionnaire and diary measures. Alcohol Alcsm 2002;37:187–92.
Townshend JM, Duka T. Binge drinking, cognitive performance and mood in a population of young social drinkers. Alcohol Clin Exp Res 2005;29:317–25.
Turkkan JS, McCaul ME, Stitzer ML. Psychophysiological effects of alcohol-related stimuli: II. Enhancement with alcohol availability. Alcohol Clin Exp Res 1989;13:392–8.
Vermeulen N, Luminet O, Cordovil de Sousa M, Campanella S. Categorical perception of anger is disrupted in alexithymia: evidence from a visual ERP study. Cognit Emotion 2008;22:1052–67.
Vuilleumier P, Pourtois G. Distributed and interactive brain mechanisms during emotion face perception: evidence from functional neuroimaging. Neuropsychologia 2007;45:174–94.
Wagner FA, Anthony JC. From first drug use to drug dependence; developmental periods of risk for dependence upon marijuana, cocaine, and alcohol. Neuropsychopharmacology 2002;26:479–88.
Walker S, Cosden M. Reliability of college student self-reported drinking behavior. J Subst Abuse Treat 2007;33:405–9.
Wechsler H, Lee JE, Kuo M, Seibring M, Nelson TF, Lee H. Trends in college binge drinking during a period of increased prevention efforts. Findings from 4 Harvard School of Public Health College Alcohol Study surveys: 1993–2001. J Am Coll Health 2002;50:203–17.
White AM, Swartzwelder HS. Hippocampal function during adolescence: a unique target of ethanol effects. Ann NY Acad Sci 2004;1021:206–20.
White AM, Ghia AJ, Levin ED, Swartzwelder HS. Binge pattern ethanol exposure in adolescent and adult rats: differential impact on subsequent responsiveness to ethanol. Alcohol Clin Exp Res 2000;24:1251–6.
Wiers RW, Rinck M, Kordts R, Houben K, Strack F. Retraining automatic action-tendencies to approach alcohol in hazardous drivers. Addiction 2009;105:279–87.
Xing Y, Ji C, Zhang L. Relationship of binge drinking and other health-compromising behaviors among urban adolescents in China. J Adolesc Health 2006;39:495–500.
Yang W, Zhu X, Wang X, Wu D, Yao S. Time course of affective processing bias in major depression: an ERP study. Neurosci Lett 2011;487:372–7.
Yee CM, Miller GA. A dual-task analysis of resource allocation in dysthymia and anhedonia. J Abnormal Psychol 1994;103:625–36.