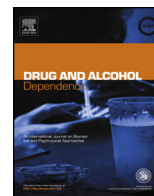




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## Drug and Alcohol Dependence

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# Binge drinking trajectory and neuropsychological functioning among university students: A longitudinal study

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### ARTICLE INFO

#### Article history:

Received 27 September 2012  
Received in revised form 13 May 2013  
Accepted 15 May 2013  
Available online xxx

#### Keywords:

Binge drinking  
Alcohol  
Adolescence  
Executive  
Memory

### ABSTRACT

**Background:** Adolescence is a time of considerable neurodevelopment. Binge drinking (BD) during this period increases the vulnerability to its neurotoxic effects. This longitudinal study aimed to investigate the relationship between BD trajectory over university years and neuropsychological functioning.

**Methods:** Cohort-study. Two-year follow-up. A total of 89 university students were assessed: 40 Non-BD (at Initial and Follow-up), 16 Ex-BD (BD at Initial but not at Follow-up) and 33 BD (at both times). Neuropsychological assessment of working memory, episodic memory and executive abilities was carried out during their first (Initial) and third (Follow-up) academic year at the University of Santiago de Compostela.

**Results:** BD subjects performed less well on the Wechsler Memory Scale-III (WMS-III) Logical Memory Subtest (immediate theme recall,  $P = .034$ ; delayed theme recall,  $P = .037$ ; and percent retention,  $P = .035$ ) and committed more perseverative errors on the Self-Ordered Pointing Task (SOPT) ( $P = .021$ ) than Non-BD. There were no differences between Ex-BD and Non-BD.

**Conclusions:** Binge drinking trajectory during adolescence is associated with neuropsychological performance. Persistent BD, but not Ex-BD, is associated with verbal memory and monitoring difficulties. This is compatible with the hypothesis that heavy alcohol use during adolescence may affect cognitive functions that rely on the temporomesial and dorsolateral prefrontal cortex.

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## 1. Introduction

Alcohol is the main risk factor for incident disability-adjusted life years (i.e., lost years of healthy life) in young people aged 10–24 years worldwide (Gore et al., 2011). Special attention has been given to binge drinking (BD), which is a prevalent pattern of alcohol consumption in many European countries (Hibell et al., 2009). BD is characterized by the intake of large amounts of alcohol in a short and irregular period of time (e.g., only on some days of the week).

The socio-sanitary relevance of the adverse consequences of BD (Mota et al., 2010; Okoro et al., 2004; World Health Organization, 2000) is highlighted by the evidence of an important degree of neural development during adolescence. Animal and human studies

indicate non-linear decreases in grey matter, which are more marked in frontal regions (Giedd et al., 2009; Sowell et al., 1999; Whitford et al., 2007). Besides, synaptic and cellular changes take place in the hippocampus and prefrontal cortex during late adolescence and continue until adulthood (Kornack and Rakic, 1999; Petanjek et al., 2011). These transformations involve development and strengthening of neural circuits, which are accompanied by improvements in neurocognitive functions related to these areas, such as episodic declarative memory (Anderson and Lajoie, 1996; Sowell et al., 2001), working memory (Conklin et al., 2007; Luciana et al., 2005; Silveri et al., 2004) and executive abilities (Luciana and Nelson, 2002; Welsh et al., 1991).

Binge drinking during adolescence appears to interact with neural development in a way that increases the vulnerability to its neurotoxic effects. Structural abnormalities in prefrontal and mesolimbic areas have been observed in binge drinking animal models (Crews et al., 2000; Taffe et al., 2010). Also, human clinical studies have reported hippocampal and prefrontal abnormalities in

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adolescents with alcohol use disorder (AUD; De Bellis et al., 2000, 2005; Medina et al., 2007, 2008).

Binge drinking is prevalent among students (Wicki et al., 2010) and structural and functional differences (in cerebral activation patterns and in psychophysiological responses to cognitive tasks) between binge drinkers and abstainers or light drinkers have been reported (Ehlers et al., 2007; Crego et al., 2009, 2010; Courtney and Polich, 2010; Maurage et al., 2009; Schweinsburg et al., 2010; Squeglia et al., 2012). These differences are in line with those reported on neuropsychological functioning. Several studies have showed poorer performance among BD students on neuropsychological tasks assessing inhibitory control, cognitive interference, sustained attention, verbal working memory and episodic declarative memory (García-Moreno et al., 2008; Goudriaan et al., 2007; Hartley et al., 2004; Heffernan et al., 2010; Johnson et al., 2008), functions known to be supported by prefrontal and/or hippocampal regions. Consistently, we have recently reported that BD results in poorer performance in neuropsychological tests assessing both verbal declarative memory (i.e., immediate and delayed recall) (Parada et al., 2011) and executive aspects of working memory (Parada et al., 2012), processes known to depend on the integrity of the hippocampus and dorsolateral prefrontal cortex, respectively.

However, despite the fact that adolescent binge drinkers show neurocognitive difficulties in population-based cross-sectional studies, little is known about the evolution of these difficulties in relation to their binge drinking trajectories. A ten-year follow-up study with AUD adolescents has shown that persistent heavy use of alcohol from adolescence to young adulthood is associated with poorer verbal memory over time (Hanson et al., 2011). These authors also suggest that, whereas early abstinence improves the performance at similar levels as healthy youths, heavy alcohol use during certain neurodevelopmental stages may lead to persistent difficulties. Similarly, recovery of neuropsychological functioning in chronic alcoholics seems related to both age and length of abstinence (Rourke and Grant, 2009). Unlike data from clinical studies, so far there is no evidence, to the best of our knowledge, on the effects of the binge drinking trajectory on neuropsychological functioning in general adolescent population. Longitudinal studies providing repeated observations at individual level of both alcohol consumption and neuropsychological performance are more suitable to elucidate causal-relationships, that is, whether neuropsychological deficits represent a consequence of heavy episodic drinking during adolescence.

Considering that prefrontal and hippocampal brain regions continue to develop until adulthood, and both are especially vulnerable to alcohol effects, as shown in clinical and animal studies, we carried out a longitudinal study to investigate the relationship between BD trajectory over a 2-year time period and neuropsychological performance in tests sensitive to temporomesial and prefrontal functioning among university students who began their alcohol consumption pattern during adolescence. This study extends on previous work and allows us to test the following hypotheses: (i) participants with a persistent binge drinking trajectory would show poorer performance in neuropsychological tests related to prefrontal and temporomesial functioning, such as working memory and declarative memory tasks, than participants who left their pattern of consumption and than non-binge drinkers; and (ii) participants who left the binge drinking pattern would improve their performance on these tasks from the initial to the follow-up assessment, to the level of the non-binge drinkers.

## 2. Methods

### 2.1. Participants

Participants were part of a longitudinal research project about the epidemiology and neurocognitive effects of binge drinking among university students conducted

at the University of Santiago de Compostela (Caamaño-Isorna et al., 2008). The neuropsychological assessment of students was performed during their first (Initial) and third (Follow-up) academic year of attendance at the University of Santiago de Compostela. At the Initial assessment time, first-year university students were screened by means of an anonymous and confidential questionnaire that they completed in class (see Section 2.2 for details). The classification criteria were based on their responses to the third question of the Alcohol Use Disorders Identification Test (AUDIT) (How often do you have six or more drinks on a single occasion? Never/Less than monthly/Monthly/Weekly/Daily or almost daily) and to one question related to the speed of consumption (drinks per hour). Subjects were classified as binge drinkers if they reported drinking six or more alcoholic drinks on the same occasion weekly, or monthly and during these episodes drank at least three drinks per hour. They were classified as controls (Non-BD) if they reported drinking six alcoholic drinks on the same occasion never, or less than monthly and at a maximum speed of consumption of two drinks per hour. Taking into account that, in Spain, a standard alcoholic drink is equivalent to 10 g of alcohol, six drinks consumed at a speed of more than two drinks per hour brings blood alcohol concentration (BAC) to 0.08 g percent or above. Once classified as a function of BD, participants were interviewed to obtain information about their clinical and sociodemographic status. To reduce the potential confusion of other factors, the following exclusion criteria were considered: personal history of neurological disorders (including loss of consciousness for at least 20 min); history of diagnosis or current psychopathological diseases (DSM-IV-TR Axis I and II; American Psychiatric Association, 2000); current psychopathological symptoms as assessed by the Symptom Checklist-90-R (SCL-90-R; Degoratis, 1983); regular consumption of other drugs (opiates, hallucinogens, cocaine, amphetamine compounds or medically prescribed psychoactive substances), except nicotine and cannabis; alcohol use disorders; severe non-corrected motor or sensory deficits; family history of major mental disorder and history of alcoholism in first- and second-degree relatives. Moreover, an AUDIT score of 20 or over was adopted as a cut-off for identifying possible alcohol abuse or dependence (Babor et al., 2001).

Participants who did not fulfil exclusion criteria were invited to undertake a neurocognitive assessment. Participants were required to not take alcohol or any other drug the day of the assessment, and to attend rested and on good health condition. Two years later, participants were contacted again for a follow-up interview and neuropsychological assessment. The same exclusion criteria were considered. The average time elapsed was  $22.01 \pm 2.1$  months. Ninety-four subjects of the one hundred forty-three assessed in the initial phase (66%) agreed to carry out the follow-up interview. Experimental mortality did not alter the sample representation. According to ANOVAs and chi-square tests, the characteristics of the total sample and follow-up sample at Initial assessment are not significantly different on sex, age, SCL-90-R scores and aspects related to alcohol consumption pattern (AUDIT total score, frequency of six drinks/occasion, drinks per hour, percentage of drunkenness, age of onset of alcohol consumption and cannabis occasional use).

At Follow-up, subjects were reclassified according to their trajectory of alcohol use. Those who reported a binge drinking pattern of consumption at both times (Initial and Follow-up) were considered BD. Those who were classified as binge drinkers at the Initial assessment but not at Follow-up were characterized as Ex-BD. Those who did not report a binge drinking pattern of alcohol consumption were classified as Non-BD. Two subjects were excluded at Follow-up interview because they met exclusion criteria and one subject did not complete the assessment. Besides, for sample consistency, three Non-BD participants at the Initial assessment who reported a binge drinking pattern at Follow-up were excluded from the analysis. Finally, 89 participants were included in the analysis: 40 Non-BD, 16 Ex-BD and 33 BD.

Neuropsychological assessments were performed by psychologists specialized in neuropsychology. All participants gave written informed consent and received monetary compensation for their participation (15€ at the Initial assessment and 30€ at Follow-up). The research was performed in accordance with the ethical principles for research involving human subjects outlined in the Helsinki Declaration, European Council Agreements and Spanish Bioethics Legislation.

### 2.2. Measures

**2.2.1. Sociodemographic and clinical data.** Sociodemographic, academic and substance use data were collected through a questionnaire and the Alcohol Use Disorders Identification Test (Babor et al., 2001). The AUDIT is a brief written screening method developed by the World Health Organization (WHO) to identify current harmful and hazardous drinking that has demonstrated reasonable psychometric properties in college students (Kokotailo et al., 2004). The Galician version of the AUDIT (Varela et al., 2005) was used to assess the frequency of binge drinking.

Personal and family history of alcohol use disorder and psychopathological or medical diseases information was collected through a semi-structured interview that included a translated and adapted version of the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA), Individual Assessment Module (IAM) and Family History Assessment Module (FHAM), designed by the Collaborative Study on the Genetics of Alcoholism (COGA; Bucholz et al., 1994), and the Symptom Checklist-90-R (Degoratis, 1983). The SCL-90-R evaluates a broad range of symptoms of psychopathology and provides a global index of psychological distress.

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