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Evolution of the binge drinking pattern in college students: Neurophysiological correlates

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ABSTRACT

It is well known that alcohol impairs response inhibition and that adolescence is a critical period of neuromaturation where cognitive processes such as inhibitory control are still developing. In recent years, growing evidence has shown the negative consequences of alcohol binge drinking on the adolescent and young human brain. However, the effects of cessation of binge drinking on brain function remain unexplored. The objective of the present study was to examine brain activity during response execution and inhibition in young binge drinkers in relation to the progression of their drinking habits over time. Event-related potentials (ERPs) elicited by a Go/NoGo task were recorded twice within a 2-year interval in 57 undergraduate students (25 controls, 22 binge drinkers, and 10 ex-binge drinkers) with no personal or family history of alcoholism or psychopathological disorders. The results showed that the amplitude of NoGo-P3 over the frontal region correlated with an earlier age of onset of regular drinking as well as with greater quantity and speed of alcohol consumption. Regression analysis showed that NoGo-P3 amplitude was significantly predicted by the speed of alcohol intake and the age of onset of regular drinking. The group comparisons showed that, after maintaining a binge drinking pattern for at least 2 years, binge drinkers displayed significantly larger NoGo-P3 amplitudes than controls, whereas ex-binge drinkers were in an intermediate position between the two other groups (with no significant differences with respect to controls or binge drinkers). These findings suggest that binge drinking in young people may impair the neural functioning related to inhibitory processes, and that the cessation of binge drinking may act as a *brake* on the neurophysiological impairments related to response inhibition.

Introduction

Inhibitory control is a core component of human behavior, generally defined as the ability to withhold or suppress actions or thoughts that are inappropriate. The broad range of psychiatric impairments that have been associated with inhibitory deficits, such as attention deficit hyperactivity disorder (Barkley, 1997; Nigg, 2001), obsessive-compulsive disorder (Chamberlain et al., 2007; Penadés et al., 2007), or substance-use disorder (Verdejo-García, Lawrence, & Clark, 2008), underlines the importance of this executive function. Alcohol abuse/dependence is one of the most common addictive behaviors (Rehm, Room, van den Brink, & Jacobi, 2005), and several studies point to a possible inhibitory deficit in alcoholics (Begleiter & Porjesz, 1999; Kamarajan et al., 2005).

Inhibitory control impairments related to alcohol use are especially worrying because failures in inhibition may weaken the ability to stop the alcohol intake and consequently promote the continuation of drinking (Field, Schoenmakers, & Wiers, 2008; Leeman, Patock-Peckham, & Potenza, 2012; López-Caneda, Rodríguez Holguín, Cadaveira, Corral, & Doallo, 2014). Adolescence is a period of life that is typically characterized by reduced inhibitory control (Casey, Jones, & Somerville, 2011; Luna, Padmanabhan, & O'Hearn, 2010), which might partially explain the increase in sensation/novelty seeking and risk-taking behaviors observed during this stage (Dahl, 2004; Strang, Chein, & Steinberg, 2013). Partly due to this elevated risk-taking behavior, adolescents and young people exhibit high rates of experimental drug use and substance-use disorders (Rohde, Lewinsohn, Kahler, Seeley, & Brown, 2001; Young et al., 2002). Alcohol is by far the most commonly used substance in occidental countries, with binge drinking (BD) being the dominant type of alcohol misuse during adolescence and youth (Substance Abuse and Mental Health Services Administration, 2009). This type of abusive drinking has

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generally been defined as the consumption of 5 or more drinks (4 or more for females) on one occasion within a 2-h interval (which corresponds to blood alcohol concentrations around .08% or above), at least once in the last 2 weeks or in the last month (Courtney & Polich, 2009; NIAAA, 2004).

In addition to the high prevalence of BD in adolescents and young people (around 10–40% of college students) (Archie, Zangeneh Kazemi, & Akhtar-Danesh, 2012; Caamaño-Isorna, Corral, Parada, & Cadaveira, 2008; Johnston, O'Malley, Bachman, & Schulenberg, 2012; Miller, Naimi, Brewer, & Jones, 2007), and the major social and health consequences associated with it (traffic crashes, aggression, unsafe sexual relations, low academic achievement, etc.) (Mota et al., 2010; Svensson & Landberg, 2013; Valencia-Martín, Galán, & Rodríguez-Artalejo, 2008; Viner & Taylor, 2007), the growing importance of the study of this phenomenon in the last decade comes from studies in animal models. These studies have shown that several BD episodes may cause more brain damage than an equivalent amount of alcohol without withdrawal episodes (Becker, 1994; Duka et al., 2004), and that this pattern of consumption may induce more harmful effects on the brain, as well as more cognitive impairments in adolescents than in adults (Crews, Braun, Hoplight, Switzer, & Knapp, 2000; Risher et al., 2013; White & Swartzwelder, 2005).

Likewise, BD appears to be of particular concern in adolescents and young adults due to the major neuromaturational changes (such as synaptic pruning and myelination) that occur throughout this period (Guerri & Pascual, 2010; Squeglia, Jacobus, & Tapert, 2009). These developmental changes essentially involve the high-order association cortices, with the prefrontal cortex (PFC) as the last region to reach maturity (Blakemore & Choudhury, 2006a; Gogtay et al., 2004). As a consequence of this brain maturation, cognitive functions supported in part by the PFC such as attention, working memory, or inhibitory control are also developing and refining during this period of transition to adulthood (Casey, Giedd, & Thomas, 2000; Luna & Sweeney, 2004; Yurgelun-Todd, 2007). Given that alcohol preferentially affects PFC (Moselhy, Georgiou, & Kahn, 2001; Oscar-Berman & Marinković, 2007) and directly reduces processes controlling inhibitory control (Field, Wiers, Christiansen, Fillmore, & Verster, 2010; Perry & Carroll, 2008), the assessment of the neural dynamics of this still immature cognitive function in adolescents and young people with a BD pattern is of great interest.

A very common way to explore the neural correlates of response inhibition (the behavioral or motor dimension of inhibitory control [Diamond, 2013]) is the recording of ERPs during a Go/NoGo task. This task requires subjects to respond to some trials (Go stimuli) and to refrain from responding to others (NoGo stimuli). Two major ERP components related to response inhibition have been recorded: the NoGo-N2 component, a negative deflection peaking approximately 200–300 ms post-stimulus and reaching maximum amplitude at fronto-central electrodes; and the NoGo-P3 component, a positive wave peaking around 300–500 ms post-stimulus and with a more anterior distribution than the Go-P3 (Falkenstein, Hoormann, & Hohnsbein, 1999; Kok, Ramautar, De Ruiter, Band, & Ridderinkhof, 2004). Although the debate about their functional meanings is still open, strong evidence relates the NoGo-N2 to conflict-monitoring processes and the NoGo-P3 to response inhibition (Bruin, Wijers, & van Staveren, 2001; Nieuwenhuis, Yeung, van den Wildenburg, & Ridderinkhof, 2003; Smith, Johnstone, & Barry, 2008).

In a previous study (López-Caneda et al., 2012), our research group examined the consequences of maintaining a BD pattern for more than 2 years on the electrophysiological correlates of response inhibition. The main finding was the hyperactivation of the right inferior frontal cortex (rIFC) – a region typically involved in inhibitory control (Aron, Robbins, & Poldrack, 2004; Chambers, Garavan, & Bellgrove, 2009) – during response inhibition, as well as a larger NoGo-P3 amplitude in youths engaged in BD for at least 2 years relative to aged-matched controls. This greater neural activation was interpreted as a compensatory neurofunctional mechanism that would allow binge drinkers (BDs) to achieve similar task performance as controls. Additional findings showing altered inhibitory functioning in young people with excessive alcohol intake come from other recent studies with heavy social drinkers, which reported delayed NoGo-P3 latencies in these subjects in an alcohol-related context (Petit, Kornreich, Noël, Verbanck, & Campanella, 2012).

In adult chronic alcoholics, several studies have shown that neuropsychological performance as well as brain structure and function recover after a period of abstinence (Bartsch et al., 2007; Sullivan & Pfefferbaum, 2005). Similarly, behavioral and electrophysiological recovery of inhibitory control processes has been observed in drug abusers as a function of abstinence (Bell, Foxe, Ross, & Garavan, in press; Morie et al., 2013). Regarding BD, despite the growing evidence indicating that this drinking pattern can be associated with poor neuropsychological performance (Heffernan & O'Neill, 2012; Parada et al., 2011) as well as with anomalies in brain structure (Lisdahl, Thayer, Squeglia, McQueeny, & Tapert, 2013; Squeglia, Sorg, et al., 2012), connectivity (Jacobus et al., 2009; McQueeny et al., 2009), and functioning (Crego et al., 2010; Schweinsburg, Schweinsburg, Nagel, Eyler, & Tapert, 2011), the nature and the extent of the neuropsychological and neurofunctional impairments after giving up the BD pattern remain unknown. Recently, our research group reported that, while maintenance of BD was associated with lower verbal episodic memory and response monitoring than Non-BD, no significant differences were found between BD and Ex-BD (BD at initial but not at follow-up) nor between Non-BD and Ex-BD, i.e., the young Ex-BDs were in an intermediate position between controls and BDs (Mota et al., 2013).

In the present study, ERPs were recorded during a Go/NoGo task in a sample of young people who maintained or gave up this pattern of consumption over a 2-year interval and in a control group of abstemious and light drinkers. The aim was to assess the neurophysiological correlates of response inhibition associated with the evolution of drinking habits. We tested the hypotheses that measures related to the BD pattern of alcohol consumption correlate and predict the amplitude of the NoGo-P3 component of the ERPs, and that BD cessation may act as a *brake* on the neural processing alterations related to response inhibition observed in BD subjects in our previous study (López-Caneda et al., 2012); this would be reflected in the ERPs as an *intermediate* NoGo-P3, i.e., lower amplitudes of the NoGo-P3 component in Ex-BDs than in BDs but larger than in controls.

Materials and methods

Participants

Fifty-seven undergraduate students participated in the study. They were part of a longitudinal research following subjects from 18 to 23 years of age. They were evaluated at two different times, when they were 18–19 and 20–21 years old. On the basis of their responses to a questionnaire that included the Galician validated version of the Alcohol Use Disorder Identification Test (AUDIT) (Varela, Braña, Real, & Rial, 2005), items 10, 11, and 12 of the Alcohol Use Questionnaire (AUQ) (Townshend & Duka, 2002), as well as other items regarding use of alcohol and other drugs, the subjects were included in three different groups: Control, BD, and Ex-BD. The BD group consisted of those participants who 1) drank 6 or