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Executive performance and dysexecutive symptoms in binge drinking adolescents

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ABSTRACT

Alcohol is probably the most common legal drug of abuse in Western countries. The prevalence of binge drinking (BD) pattern of alcohol consumption among adolescents is a worrisome phenomenon. Adolescents and university students who practice a BD pattern have difficulty performing tasks involving prefrontal cortex functions, such as working memory, planning, attention, and decision making. The aim of the present study was to investigate the association between BD and executive functioning in adolescents. Two hundred twenty-three high-school students between 12 and 18 years old (15.19 ± 2.13) participated in our study. They were assigned to one of three groups according to their pattern of alcohol consumption: BD (subjects who consumed alcohol intensively, $n = 48$), MAC (subjects who consumed alcohol moderately, $n = 53$), and CTR (non-drinking subjects, $n = 122$). The students were evaluated with two groups of testing tools: a set of performance neuropsychological tests and two questionnaires of executive functioning. The results showed that the students who drank alcohol exhibited a more pronounced dysexecutive symptomatology (disinhibition, executive dysfunction, intentionality, executive memory), but they obtained better results than controls on some of the neuropsychological tests such as Spatial Location, Five Digit Tests, or Stroop Test. According to the results, we can deduce that heavy alcohol drinking in adolescents brings a certain dysfunction of prefrontal circuits. This prefrontal dysfunction is not so clearly demonstrated in the neuropsychological tests used, but it was observed in the performance of daily activities. In the Discussion section we raise issues about sociodemographic features of the sample and ecological validity of the traditional neuropsychological tests. The neurotoxic effects of BD on prefrontal cortex can be less evident throughout adolescence, but if alcohol consumption persists, the executive dysfunction would be exacerbated.

Introduction

Alcohol is probably the most common legal drug of abuse in Western countries. In recent decades, there has been increasing concern regarding the neurocognitive effects of alcohol in adolescents and young people because of the high prevalence of alcohol abuse among this population (European Commission, 2010). Alcohol use during adolescence is particularly concerning because it can interfere with the achievement of key developmental tasks such as forming an identity, building mature relationships, and preparing for a career during this critical developmental period (Arnett, 2005). Despite this, there was little research outside the psychopathological framework on the potential adverse consequences of underage drinking on social, emotional, behavioral, and neurobiological development or on future drinking patterns. The lack of research with normal adolescents was partly due to the fact that drinking at this age was viewed as a transient phenomenon, with the majority of high-school and college students aging out of heavy drinking as they transitioned to new roles in adulthood (Bachman et al., 2002). Currently, there is enough empirical evidence to support the idea that alcohol consumption represents a serious danger to the health of young people (Cadaveira, 2009). Students who drink alcohol are more likely than non-drinkers to have academic problems, engage in high-risk sex, sustain an injury, overdose on alcohol, or drive while intoxicated (Jennison, 2004; Vik, Carrello, Tate, & Field, 2000; Wechsler, Lee, Kuo, & Lee, 2000).

Epidemiological studies have noted a high prevalence of the Binge Drinking (BD) pattern of consumption among adolescents and young adults, especially university students (Anderson, 2007; Caamaño-Isorna, Corral, Parada, & Cadaveira, 2008; White, Kraus, & Swartwelder, 2006). According to the last report by the World Health Organization (2014), the prevalence of binge drinking

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episodes among regular drinkers reaches high levels, e.g., 39% in Ireland, 29.4% in France, 28% in the United Kingdom, 19.6% in the United States, 13.4% in Spain, and 2.5% in Germany. Of course, BD prevalence is even higher among adolescents and youth (World Health Organization, 2014). Most studies that have reported the prevalence of binge drinking show a lack of consensus regarding the operational definition of this pattern of alcohol consumption. The National Institute on Alcohol Abuse and Alcoholism (NIAAA) has defined 'Binge Drinking' as a pattern of drinking alcohol that brings blood alcohol concentration (BAC) to about 0.08% or above in about 2 h. This pattern corresponds to consuming 5 or more drinks (male) or 4 or more drinks (female) in a session at least once in the previous 15–30 days (Courtney & Polich, 2009). This is the reason why some definitions have been proposed in relation to the number of standard drink units (SDUs) consumed per occasion, frequency of episodes, and their duration. The criterion most widely accepted by the international scientific community is the consumption of 5 or more SDUs for men and 4 or more for women, on a single occasion at least once in the previous 2 weeks. However, aspects such as differences in the grams of alcohol of SDUs hinder the establishment of an international definition of the term, making it necessary to adapt this approach to the country in which the study is being carried out (Parada et al., 2011b).

BD can also be defined as the alternation between uncontrolled alcohol intake and abstinence periods. This pattern is characterized by repeated bouts of drinking leading to high levels of alcohol in the brain followed by periods in which brain alcohol levels return to zero. The BD pattern may lead to brain damage and resultant cognitive dysfunction, which may be similar to the neurotoxicity induced by repeated withdrawals from alcohol in dependent animals and humans (Duka et al., 2004; Duka, Townshend, Collier, & Stephens, 2003; Scaife & Duka, 2009). Several studies have indicated that intermittent alcohol consumption may constitute a greater risk to neurocognitive functioning than regular alcohol consumption (Stephens & Duka, 2008). One study about intermittent alcohol consumption in rats, approximating the BD pattern in humans, has demonstrated the existence of inflammatory damage in the brain. It also showed an increase in cell death in the neocortex, hippocampus, and cerebellum, which could produce long-term disorders of brain functionality and affect cognitive and motor processes (Pascual, Blanco, Cauli, Miñarro, & Guerri, 2007). Thus, BD is deleterious for the brain because of alcohol consumption *per se*, and also because of its specific consumption pattern (Maurage et al., 2012), which leads to detrimental effects such as poorer academic results, less adequate social integration, and also cognitive impairments (Brumback, Cao, & King, 2007; Goudriaan, Grekin, & Sher, 2007; Hartley, Elsabagh, & File, 2004; Read, Merrill, Kahler, & Strong, 2007; Zeigler et al., 2005).

The effects of alcohol on the central nervous system have been widely studied and their neurocognitive, neuroanatomical, and neurofunctional consequences are well known (see Oscar-Berman & Marinković, 2007). During adolescence, the brain is in a critical period of development and is particularly sensitive to alcohol (Barron et al., 2005; Ehlers & Criado, 2010). In addition, animal studies with rats have demonstrated that the adolescent brain is more vulnerable to the neurotoxic effects of alcohol than the adult brain (Guerri & Pascual, 2010; Spear, 2014). In humans, the prefrontal lobe continues to mature into the early 20s (Casey, Giedd, & Thomas, 2000; Gogtay et al., 2004); this late developing area may therefore be especially sensitive to heavy alcohol use. Important structural and functional changes take place, especially in the prefrontal cortex and the mesolimbic regions (Crews, He, & Hodge, 2007; Lenroot & Giedd, 2006; Spear, 2000; Yurgelun-Todd, 2007) and many of the behavioral impairments seen in binge drinkers can be ascribed to alterations in the function of these areas (Duka et al. 2003, 2004).

Structural abnormalities in the prefrontal cortex and in the temporomedial areas have been observed both in binge drinking animal models (Crews et al., 2007; Taffe et al., 2010) and in adolescents with alcohol use disorders (AUD). In humans, it has been found that young people with AUD have a smaller prefrontal cortex volume and perform less well in neuropsychological attention and working memory tasks than control subjects (De Bellis et al., 2005; Medina et al., 2008; Tapert, Baratta, Abrantes, & Brown, 2002; Tapert & Brown, 1999). However, most of these studies were performed with subjects who already had problems with alcohol; few studies have investigated the neurobiological and neurocognitive effects of the BD pattern of alcohol consumption in non-clinical samples of young people. Adolescents and university students who practice the BD pattern have difficulty carrying out tasks involving prefrontal cortex functions, such as working memory, planning, attention, and decision making. However, not all studies address executive functions in a broad sense, and not all results are in agreement. Some of them have found deficits especially in attention and verbal and visuospatial working memory (García-Moreno, Expósito, Sanhueza, & Angulo, 2008; Hartley et al., 2004; Parada et al., 2012; Sanhueza, García-Moreno, & Expósito, 2011; Scaife & Duka, 2009; Townshend & Duka, 2005; Weissenborn & Duka, 2003), while others have found deficits in decision making (Goudriaan et al., 2007; Johnson et al., 2008) or in tasks of speed processing, behavioral inhibition, planning ability, and cognitive flexibility (Hartley et al., 2004; Sanhueza et al., 2011; Scaife & Duka, 2009; Townshend & Duka, 2005; Weissenborn & Duka, 2003). One likely explanation for this variability seems to be the differences observed in selected samples, the tests used for assessment, or the criteria for calculating alcohol intake. We believe there is a need to study more thoroughly the deleterious effects of alcohol consumption in young people to detect early signs of effects on the brain, and to design more effective interventions.

The aim of the present study was to investigate the association between BD and executive functioning in adolescents with no neurologic, psychiatric, or alcohol- and drug-related problems. Taking into account the greater vulnerability of the prefrontal cortex to the neurotoxic effects of alcohol, it is possible that executive functions supported by this brain region may be particularly susceptible to alteration during adolescence. Most of the executive functions from our study have been evaluated previously by other authors but, as we have said, with different results, probably because of the use of different samples and different binge drinking criteria. The researchers who study the cognitive performance of people with alcohol abuse problems often use data from subjects18 years old and older. In our study, we have used a younger sample than usual. We have recruited a sample from the age when alcohol consumption begins up to age of 18 years old in order to better understand how alcohol-related executive impairment progressed. We hypothesize that BD subjects will show greater dysexecutive symptomatology (e.g., disinhibition, impulsivity, poor decision making, poor perseveration, aggressiveness, or lack of mental flexibility) and will perform less well than non-BD subjects in tasks that evaluate executive functioning, such as selective attention, working memory, mental set shifting, self-monitoring, cognitive flexibility, or self-control and ability to display inhibition.

Materials and methods

Participants

Two hundred and twenty-three students (age range 12–18 years) participated in the study; 43.5% were woman ($n = 97$, mean age 15.123 ± 2.05 years) and 56.5% were men ($n = 126$, mean age 15.246 ± 2.19 years). Participants were selected through an