Binge Drinking in Adolescents: A Review of Neurophysiological and Neuroimaging Research

Géraldine Petit1, Pierre Maurage2, Charles Kornreich1, Paul Verbanck1 and Salvatore Campanella1,*

1Laboratory of Psychological Medicine and Addictology, ULB Neuroscience Institute (UNI), Université Libre de Bruxelles (ULB), Brussels, Belgium and 2Laboratory for Experimental Psychopathology, Institute of Psychology, Catholic University of Louvain, Louvain-la-Neuve, Belgium

*Corresponding author: The Belgian Fund for Scientific Research (FNRS), CHU Brugmann, Department of Psychiatry, 4 Place Vanghelichten, 1020 Brussels, Belgium. Tel.: +32-24773465; E-mail: salvatore.campanella@chu-brugmann.be

Abstract — Aims: While the relationship between chronic exposure to alcohol and neurobiological damage is well established, deleterious brain effects of binge drinking in youths have only recently been studied. Methods: Narrative review of studies of brain disturbances associated with binge drinking as assessed by neuroimaging (EEG and fMRI techniques in particular) in adolescent drinkers. Results: Some major points still deserved to be investigated; directions for future research are suggested. Conclusions: Information and prevention programs should emphasize that binge drinking is not just inoffensive social fun, but if carried on, may contribute to the onset of cerebral disturbances possibly leading to alcohol dependence later in life.

INTRODUCTION

The practice of drinking to intoxication (i.e. binge drinking) has become the peer norm among some groups of young people, and alcohol stands as the central favorite in a repertoire of psychoactive substances employed to facilitate pleasure and the enjoyment of time out with friends (Johnston et al., 2009). It has been known for a long time that, because of alcohol neurotoxicity, alcohol abuse leads to deleterious effects on the central nervous system, such as brain atrophy and/or dysfunction (e.g. Nicolas et al., 1997). Until recently, most research on brain damage from alcohol drinking has concentrated on heavy long-term use in adults (usually around age 45). A few studies focused on the effects of alcohol use disorders (AUDs) in adolescents and young adults (of 13–24 years). AUDs in 13- to 21-year age group have been linked to abnormal thalamic and putamen volumes and decreased gray matter density (Fein et al., 2000) and in prefrontal lobe volumes (De Bellis et al., 2005), to increased functional anisotropy (FA), decreased mean diffusivity in the corpus callosum (De Bellis et al., 2008), and to increased parietal activity during a spatial memory task (Tapert et al., 2004). Researchers in recent years have focused on the effects of binge drinking patterns in younger individuals. It is known from animal studies that (a) during maturation (which is typically not completed until up to 25 years in humans), the brain is particularly sensitive to the effects of alcohol (e.g. Baron et al., 2005) and (b) the repeated withdrawals due to alternation between periods of alcohol intoxication and abstinence may be deleterious for brain function due to excitotoxic cell death (Obernier et al., 2002; Bijl et al., 2007). Thus the binge drinking population seems to be at risk of brain damage.

At the structural level, binge drinking has been linked to compromised white matter (WM) in frontal, cerebellar, temporal and parietal regions (McQueeny et al., 2009), reduced WM integrity in both association fiber pathways as well as in the corona radiate (Jacobs et al., 2009), smaller cerebellar volumes (Lisdahl et al., 2013) and changes in cortical thickness (Squeglia et al., 2012a). These structural changes may account for the discovery by Courtney and Polich (2010) of alterations of delta and fast-beta activity among binge drinkers during electroencephalographic passive recording (EEG).

On the other hand, at the functional level, event-related potentials (ERPs) and functional magnetic resonance imaging (fMRI) recorded during diverse cognitive tasks showed that binge drinking is associated with impaired neural processes. Using ERPs, Ehlers et al. (2007) showed a decrease in the P3a component latency during a facial discrimination task, a component linked to automatic attention processing of stimulus deviance (see Polich and Criado, 2006). Crego et al. (2009, 2010) reported anomalies of the N2, the P3 and the late positive (LP) components combined with hypoactivation of the right anterior prefrontal cortex in binge drinkers during a visual working memory task, possibly reflecting some impairment of working memory processes. Similar to López-Caneda et al. (2013), they also demonstrated increased P3b amplitudes in a simple visual oddball task (Crego et al., 2012), suggesting anomalies in neural processes mediating attention processing. Maurage et al. (2009) showed diminished activity during an emotional auditory task, indexed by delayed latencies for the P1, the N2 and the P3b components and delayed P3 latencies and impairments related to earlier processes (P100, N100, N170, P2, N2b) during a facial detection task (Maurage et al., 2012). Finally, López-Caneda et al. (2012) reported altered Go- and NoGo-P3 components during response execution and inhibition which may represent a neural antecedent of difficulties in cognitive control.

fMRI demonstrated in binge drinkers increased frontal and parietal activations and decreased occipito-hippocampal activation during verbal encoding (Schweinsburg et al., 2010, 2011), thereby indicating greater engagement of working memory systems during encoding and disadvantaged processing of novel verbal information. Binge drinkers also display abnormal patterns of activation during working memory tasks (Squeglia et al., 2011; Campanella et al., 2013). Finally, Xiao et al. (2013) showed higher activity in the left amygdala and insula during a decision-making task in binge drinkers, suggesting alterations of the neural circuitry implicated in the execution of emotional and incentive-related behaviors.

PRE-EXISTING OR ALCOHOL-INDUCED DEFICITS?

While all of these studies undeniably describe cerebral aberrations associated with binge drinking, the majority of them...
either carried out only one testing session or followed binge drinkers without controlling for past alcohol consumption, which makes it difficult to be certain of the cause of the deficits. To our knowledge, only three studies explored this question by performing a test–retest study on groups of young individuals with no past alcohol consumption and with comparable baseline results on the tasks proposed. Squeglia et al. (2009, 2012b) showed that after 3 years of heavy alcohol use, adolescents exhibited lower visuospatial memory and sustained attention performances as well as abnormal frontal, parietal and occipital activation during a visual working memory task, compared with controls. Using ERPs, Maurage et al. (2009) showed delayed latencies of P100, N200 and P300 components only after 9 months in students who had initiated binge drinking habits when compared with students who had persisted in very low alcohol consumption. These few studies indicated that heavy and binge drinking can lead to marked cerebral dysfunction, in the absence of any pre-existing cerebral impairment.

EFFECT OF DRINKING PATTERN

One could wonder whether the deficits identified are due to the particular binge drinking pattern (i.e. intense but episodic alcohol consumption episodes) or to the global heavy alcohol intake, independent of the rate and frequency of alcohol absorption. As almost all of the studies cited above only compared binge drinkers with control non-drinkers or very low drinkers, they did not differentiate impairments due to cumulative total alcohol intake versus a specific binge pattern. Maurage et al. (2012) explored this hypothesis by comparing the results in groups with different drinking pattern in quantity and frequency, in a simple ERP cognitive task. Where there was a similar cumulative consumption (15–29 doses per week), binge drinkers (i.e. concentrating their weekly 15–59 doses to 2–3 drinking occasions) presented stronger cerebral impairments than daily drinkers (i.e. spreading the weekly 15–59 doses on 5–7 occasions). Similarly, in their recent work that explored alcohol-related bias in binge drinkers, Petit et al. (submitted) showed that among the different drinking characteristics of the binge drinkers studied (i.e. the number of alcohol doses per week, the number of alcohol doses per occasion, the number of occasion of drinking per week), the best predictor of the alcohol-related processing bias was the number of alcohol doses consumed on each occasion. Finally, in their study, Campanella et al. (2013) identified a specific link between the effects they observed in binge drinkers (i.e. higher activity in the dorsomedial prefrontal cortex) and the number of alcohol doses consumed per occasion. These latter observations indicate that, apart from the traditional AUDs, and the different patterns of misuse of alcohol (as risky, harmful, hazardous alcohol use), the repeated alternation between intoxication and withdrawal appears to have the more deleterious consequences.

BINGE DRINKING COMPARED CHRONIC ALCOHOL MISUSE

There are two important observations from these studies on binge drinking. Firstly, despite the broad variety of neuronal mechanisms and structures that they explored, it is evident that binge drinking is not only associated with damages or deficits, but that these anomalies mirror those observed in alcoholism. Reduced cerebellar volumes, WM integrity and abnormal cortical thickness observed in binge drinkers (Jacobus et al., 2009; McQueeny et al., 2009; Squeglia et al., 2012a; Lisdahl et al., 2013) align volume deficits, FA diminishations and aberrant thickness observed in adults with AUDs (e.g. Sullivan et al., 2000; Momenan et al., 2012). Abnormal central nervous system neuroelectric activity (Courtney and Polich, 2010) seen in binge drinkers refers to abnormalities in EEG profile shown alcoholics (e.g. Bauer, 2001). Defects in the LPC and the P3 components of the ERP (Ehlers et al., 2007; Crego et al., 2009, 2010, 2012; Maurage et al., 2009, 2012, 2013; López-Caneda et al., 2012) and in components associated with earlier cognitive processes in bingers (Crego et al., 2009; Maurage et al., 2009, 2012; Petit et al., 2012a) are in line with changes in both the amplitude and latency of the LPC and the P300 component (e.g. Brecher et al., 1987; Porjesz and Begleiter, 2003; George et al., 2004; Easdon et al., 2005) and with low-level cognitive function impairments observed in chronic alcoholism (e.g. Nicolas et al., 1997; Verma et al., 2006; Maurage et al., 2007; Fein et al., 2009). Aberrant patterns of brain activations during verbal encoding (Schweinsburg et al., 2010, 2011), working memory (Crego et al., 2010; Squeglia et al., 2011; Campanella et al., 2013) and decision-making tasks (Xiao et al., 2013) observed in binge drinkers align poor verbal learning capacities evidenced in chronic heavy drinkers (for a review, see Grant, 1987), abnormal brain activation during working memory task observed in adolescents with AUDs (Tapert et al., 2004) and decision-making deficits linked to dysfunctional brain activity reported in alcoholics (Bechara et al., 2001), respectively. Secondly, these studies also show that the similarities invariably observed in binge drinkers are either less serious than those observed in chronic alcohol misuse or are detected by neuroimaging tools but remain unexpressed at a behavioral level. Indeed, if a few studies (Squeglia et al., 2011, 2012a; Xiao et al., 2013) linked binge drinking with decreased performance, most of the others (Ehlers et al., 2007; Crego et al., 2009, 2010, 2012; Maurage et al., 2009, 2012; Schweinsburg et al., 2010, 2011; López-Caneda et al., 2012, 2013; Petit et al., 2012a, submitted) found neurophysiological differences without any significant behavioral modification. These results emphasize the necessity of using neuroimaging techniques to correctly estimate the actual level of impairment that could go unnoticed at the behavioral level, in a population of binge drinkers, in which abnormalities are not as marked as in pathological populations (Maurage et al., 2009; Campanella et al., 2013) (Fig. 1).

IS BINGE DRINKING A PATHWAY TO CHRONIC ALCOHOLISM?

Although less obvious, binge drinkers appear to present with the same pattern of impairments as alcohol-dependent individuals, since the deficits concern the same cognitive functions. The similarities in brain alterations between adolescent binge drinkers and adult alcohol dependents have led some authors (e.g. Wagner and Anthony, 2002; McCarthy et al., 2004; Enoch, 2006) to suggest the ‘continuum hypothesis’, in which binge drinking and chronic alcohol dependence should be regarded as two stages of the same phenomenon, inducing
analogous deficits, and not as independent pathologies. This further encouraged the strong suggestion that binge drinking during adolescence could constitute a first step towards the development of alcohol dependence during adulthood (Schulenberg et al., 1996; Tucker et al., 2003). Others have presented contrary data which show that binge drinking is an adolescence’s related normative feature (e.g. Gotham et al., 1997) which declines with the increased responsibility due to life transitions such as employment, marriage or parenthood (e.g. Muthén and Muthén, 2000; Wood et al., 2000). Should one therefore be concerned, ‘Youth will have its fling’. But what if, for some individuals, this transitional period leaves damage influencing their future? Indeed, knowing that current epidemiological studies have suggested that binge drinking in youths is associated with an increased risk of alcohol abuse/dependence in adulthood (Chassin et al., 2002; Bonomo et al., 2004; McCarty et al., 2004; Viner and Taylor, 2007), it is reasonable to believe that some deficits and/or neurobiological changes that occur during the binge drinking period could play a role in the maintenance of alcohol use and abuse, and could cause difficulties in curtailing consumption leading to long-term alcohol problems (e.g. Hiller-Sturmhofel and Swartzwelder, 2004; King et al., 2006; Haller et al., 2010). However, while this idea sounds relevant, as it has not been explored in depth, the causal relationship between binge drinking deficits and the development of alcohol dependence remains unclear. To elucidate the question of the possible role of such emerging brain abnormalities in binge drinking as risk factors to chronic alcoholism, future research should focus on two objectives. The first must be to investigate the similarities that exist between the neurocognitive deficits and/or abnormalities detectable in binge drinkers and those observed in adult chronic alcoholics by focusing specifically on those that have shown to play a role in the emergence and/or the maintenance of persistent drinking habits in chronic alcoholics. In this regard, as impaired inhibitory control has been identified as a risk factor for substance abuse (Porjesz et al., 2005), López-Caneda et al. (2012) focused on analyzing the inhibitory capacity of young binge drinkers. Neurophysiological aberrations indexing impaired inhibition capacities were evident in young binge drinkers which suggested that these alterations could constitute a risk factor for developing alcohol dependence in these individuals. Similarly Petit et al. (2012a,b, 2013, submitted) identified a differential electrophysiological processing of alcohol-related stimuli in binge drinkers, a feature demonstrated in alcoholism (e.g. Herrmann, 2000; Namkoong, 2004) and overall, proposed to play a role in the maintenance of drug dependence (Field and Cox, 2008) and considered to be a risk marker for alcohol dependence (Bartholow et al., 2007, 2010). The second important point should be to study the evolution of these deficits and/or abnormalities over time in parallel with the evolution of the consumption of the study population. Although their studies have not been conducted over a lengthy period (bingers were followed for almost 2 years), this allowed López-Caneda et al. (2012) and Petit et al. (submitted) to demonstrate that the emergence of the electrophysiological
<table>
<thead>
<tr>
<th>Authors (year)</th>
<th>Types of study</th>
<th>Sample characteristics</th>
<th>Age (years)</th>
<th>Cognitive task</th>
<th>Neuroimaging tools</th>
<th>Cognitive outcome</th>
<th>Neurophysiological and neuroimaging outcome</th>
<th>Examples of studies and reviews depicting similar defects in alcoholic population</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crego et al. (2009)</td>
<td>Cross-sectional</td>
<td>BD ($n = 42$): ≥6 drinks on 1 occasion at a speed of ≥3 drinks/h C ($n = 53$): ≤6 drinks on 1 occasion at a speed of ≤3 drinks/h</td>
<td>NA</td>
<td>Visual working memory task</td>
<td>ERP</td>
<td>BD = C</td>
<td>N2 amplitudes: BD &gt; C P3 amplitudes: BD ≠ C</td>
<td>Porjesz and Begleiter (2003), Fein et al. (2009), and Verma et al. (2006)</td>
</tr>
<tr>
<td>Jacobus et al. (2009)</td>
<td>Cross-sectional</td>
<td>BD ($n = 28$): history of at least 1 episode of ≥5 (M) or 4 (F) drinks on one occasion C ($n = 14$): very limited if any substance use history</td>
<td>NA</td>
<td>None</td>
<td>DTI</td>
<td>NA</td>
<td>WM indexed by FA: BD &lt; C</td>
<td>Pfefferbaum et al. (2006)</td>
</tr>
<tr>
<td>McQueeny et al. (2009)</td>
<td>Cross-sectional</td>
<td>BD ($n = 14$): ≥5 (M) or 4 (F) drinks in 1 sitting during the last 3 months C ($n = 14$): no history of BD episodes</td>
<td>NA</td>
<td>None</td>
<td>DTI</td>
<td>NA</td>
<td>WM integrity (indexed by FA): BD &lt; C</td>
<td>Pfefferbaum et al. (2006)</td>
</tr>
<tr>
<td>Maurage et al. (2009)</td>
<td>Cohort (prospective) - over 9 months</td>
<td>BD ($n = 18$): 12.52 DPO; 2.33 NOW; 35 DPW C ($n = 18$): no BD</td>
<td>Yes</td>
<td>Emotional auditory discrimination task</td>
<td>ERP</td>
<td>T1 and T2: BD = C</td>
<td>T1: BD = C T2: P1, N2, P3b latencies: BD &gt; C</td>
<td>Maurage et al. (2008) and Kathmann et al. (1996)</td>
</tr>
<tr>
<td>Courtney and Polish (2010)</td>
<td>Cross-sectional</td>
<td>Low-BD ($n = 32$): 5/4–7/6 drinks in within 2 h on &gt;1 occasion within the past 6 months High-BD ($n = 32$): ≥10 drinks within 2 h on &gt;1 occasion within the past 6 months Controls ($n = 32$): &gt;1 to 5/4 drinks within 2 h on &gt;1 occasion within the past 6 months Controls ($n = 32$): &gt;1 to 5/4 drinks within 2 h on &gt;1 occasion within the past 6 months Controls ($n = 32$): &gt;1 to 5/4 drinks within 2 h on &gt;1 occasion within the past 6 months</td>
<td>NA</td>
<td>None</td>
<td>EEG</td>
<td>NA</td>
<td>Spectral power in the delta (0–4 Hz) and fast-beta (20–35 Hz) bands : C = low-BD &lt; high-BD</td>
<td>Bauer (2001)</td>
</tr>
<tr>
<td>Crego et al. (2010)</td>
<td>Cross-sectional</td>
<td>BD ($n = 42$): ≥6 drinks on 1 occasion at a speed of ≥3 drinks/h 1 or more times per month. C ($n = 53$): ≤6 drinks on 1 occasion at a speed of ≤3 drinks/h</td>
<td>NA</td>
<td>Visual working memory task</td>
<td>ERP and eLORETA</td>
<td>BD = C</td>
<td>LPC amplitude and aPFC: BD &lt; C</td>
<td>Tapert et al. (2004) and Brecher et al. (1987)</td>
</tr>
<tr>
<td>Study</td>
<td>Design</td>
<td>KD</td>
<td>C</td>
<td>Ne</td>
<td>Measures</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-------------------------------------------</td>
<td>----------------------------</td>
<td>----</td>
<td>---</td>
<td>----</td>
<td>---------------------------------------------------------------------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Schweinsburg et al. (2011)</td>
<td>Cross-sectional</td>
<td>NA</td>
<td>16–18</td>
<td>Verbal encoding task fMRI</td>
<td>BD = C Frontal BOLD response during novel verbal encoding: BD ≠ C</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Squeglia et al. (2011)</td>
<td>Cross-sectional</td>
<td>NA</td>
<td>16–19</td>
<td>Neuropsychological battery + spatial working memory task MRI</td>
<td>Working memory performance: BD ≠ C Neural activation: BD ≠ C</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Crego et al. (2012)</td>
<td>Cross-sectional</td>
<td>NA</td>
<td>18–20</td>
<td>Visual oddball task ERP</td>
<td>BD = C P3b amplitudes: BD &gt; C</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lopez-Caneda et al. (2012)</td>
<td>Cohort (prospective) – over 2 years</td>
<td>No</td>
<td>T1: 18–19 Go/NoGo task ERP and eLORETA</td>
<td>T1 and T2: BD = C NoGo-P3 amplitude: T1: BD = C; T2: BD &gt; C Go-P3 amplitude: T1 and T2: BD &gt; C Activation in rIFC: T1: BD = C; T2: BD &gt; C</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maurage et al. (2012)</td>
<td>Cross-sectional</td>
<td>NA</td>
<td>19–24</td>
<td>Visual oddball task (face detection) ERP</td>
<td>BD1 = BD2 = C P100, N100, N170/P2, N2b/P3a and P3b amplitudes: BD1 &lt; C; BD2 &lt; C P100, N100 and N2b/P3a latencies : BD2 &gt; C P3b latency: BD1 &gt; C; BD2 &gt; C</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Petit et al. (2012a)</td>
<td>Cross-sectional</td>
<td>NA</td>
<td>19–25</td>
<td>Alcohol-modified oddball task ERP</td>
<td>BD = C P100 amplitude in response to alcohol cues: BD &gt; C</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Squeglia et al. (2012a)</td>
<td>Cross-sectional</td>
<td>NA</td>
<td>16–19</td>
<td>Neuropsychological battery MRI</td>
<td>Visuospatial, inhibition and attention performances: BD ≠ C Cortical thickness: BD ≠ C</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Campanella et al. (2013)</td>
<td>Cross-sectional</td>
<td>NA</td>
<td>18–25</td>
<td>Working memory N-back task fMRI</td>
<td>BD = C Activity in the pre-supplementary motor area: BD &gt; C</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lisdahl et al. (2013)</td>
<td>Cross-sectional</td>
<td>NA</td>
<td>16–19</td>
<td>None MRI</td>
<td>NA Cerebellar volumes: BD &lt; C</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Authors (year)</td>
<td>Types of study</td>
<td>Sample characteristics</td>
<td>Age (years)</td>
<td>Cognitive task</td>
<td>Neuroimaging tools</td>
<td>Cognitive outcome</td>
<td>Neurophysiological and neuroimaging outcome</td>
<td>Examples of studies and reviews depicting similar defects in alcoholic population</td>
</tr>
<tr>
<td>------------------------------------</td>
<td>----------------</td>
<td>----------------------------------------------------------------------------------------</td>
<td>-------------</td>
<td>-------------------------------------</td>
<td>--------------------</td>
<td>------------------</td>
<td>---------------------------------------------</td>
<td>----------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Lopez-Caneda et al. (2013)</td>
<td>Cohort (prospective) – over 2 years</td>
<td>BD (n = 26): ≥6 drinks on 1 occasion at a speed of ≥3 drinks/h ≥1 times per month or ≥6 drinks on 1 occasion ≥1 times per week C (n = 31): ≤6 drinks on 1 occasion ≥1 times per week</td>
<td>No T1: 18–19</td>
<td>Visual oddball task</td>
<td>ERP</td>
<td>T1 and T2: BD = C</td>
<td>P3b amplitudes: T1: BD &gt; C T2: BD &gt; + C</td>
<td>Porjesz and Begleiter (2003)</td>
</tr>
<tr>
<td>Petit et al. (2013)</td>
<td>Cross-sectional</td>
<td>BD (n = 29): ≥6 drinks on 1 occasion at a speed of ≥3 drinks/h C (n = 27): ≤6 drinks on 1 occasion at a speed of ≤3 drinks/h</td>
<td>NA 18–27</td>
<td>Alcohol-modified oddball task</td>
<td>ERP</td>
<td>BD = C</td>
<td>P3 amplitudes to alcohol cues: BD &gt; C</td>
<td>Herrmann et al. (2000)</td>
</tr>
<tr>
<td>Petit et al. (submitted)</td>
<td>Cohort (prospective) – over 1 year</td>
<td>BD (n = 15): ≥6 drinks on 1 occasion at a speed of ≥3 drinks/h C (n = 15): ≤6 drinks on 1 occasion at a speed of ≤3 drinks/h</td>
<td>No 19–25</td>
<td>Alcohol-modified oddball task</td>
<td>ERP</td>
<td>T1 and T2: BD = C</td>
<td>T1: P3 amplitudes to alcohol vs. non-alcohol cues: BD = C T2: P3 amplitudes to alcohol vs. non-alcohol cues: BD ≠ C</td>
<td>Herrmann et al. (2000)</td>
</tr>
</tbody>
</table>

C, controls; BD, binge drinkers; C = B, no significant difference between control and binge group; BD ≠ C, binge drinkers significantly differed from controls; BD > + C, more pronounced difference in BD vs. controls (here, compared with T1); EEG, electroencephalogram; ERP, event-related potentials; DTI, diffusion tensor imaging; WM, white matter; FA, fractional anisotropy; eLORETA, exact low-resolution brain electromagnetic tomography; LPC, late positive component; A PFC, activation of the right anterior prefrontal cortex; MRI, magnetic resonance imaging; fMRI, functional magnetic resonance imaging; ADO, mean number of alcohol doses per drinking occasion; DOW, mean number of drinking occasions per week; ADH, mean number of alcohol doses per hour (consumption speed); ADW, mean number of alcohol doses per week; rIFC, right Inferior Frontal Cortex.
abnormalities observed was associated with the continuation of binge drinking habits. These preliminary results suggest that a vicious circle could start with the habit of binge drinking. In other words, alterations in cue reactivity or reduced inhibition, two characteristics that the contemporary dual process model theories associate with the development of alcohol abuse (e.g. Stacy and Wiers, 2010), could arise with the emergence of binge drinking habits and then be amplified due to alcohol neurotoxicity. The stronger neuropsychological impairments and their link to neurobiological changes would then reinforce the alcohol consumption, preventing the normal decrease of heavy drinking with maturation and leading to the perpetuation of risky drinking habits and eventually to alcoholism.

CONCLUSION: STILL A LONG WAY TO GO

The neuroscience approach of binge drinking has already drawn attention to the structural and functional brain damage associated with this practice (Hermens et al., 2012). Research on binge drinking in young people is however still in its infancy and many questions remain unresolved. A first important question is whether the brain alterations observed among binge drinkers are the result of alcohol misuse or whether these changes may be pre-existing. This issue has only been properly assessed in a few studies and still needs to be confirmed and extended in more longitudinal studies, notably in regards of the control of other variables that precede and could also predispose to the development of binge drinking habits, as for example brain modifications related to family history of alcoholism. In their neuroimaging longitudinal study, Squeglia et al. (2012b) suggested that some cerebral deficits observed in heavy drinkers could already be present before alcohol misuse and be involved in the onset of alcohol consumption. Also, the earlier Ehlers et al.’s study (2007) already mentioned that a positive family history for alcohol dependence acted as a significant covariate in some of their findings on binge drinking while Norman et al. (2011) and Wetherill et al. (2013) showed in their longitudinal studies that pre-existing abnormalities in neural activity during response inhibition observed in early adolescence were predictors of future alcohol heavy use. Controlled longitudinal studies will be crucial. Finally, longitudinal studies on binge drinkers may reveal some who start to abstain from binge drinking and reversibility of abnormalities should be looked for. Meanwhile, given the data available, there is a case to develop adapt information and prevention programs to emphasize the message that binge drinking is not just inoffensive social fun, but if carried on, may contribute to the onset of cerebral disturbances leading to alcohol dependence later in life, even at a stage at which behavioral manifestation is not yet evident (Campanella et al., 2013). In Table 1, we summarize neuropsychological and neuroimaging studies of binge drinking. We especially focused on (1) whether these studies reported cognitive impairments in addition to neural mechanisms impairments (2) the similarities of the neurobiological alterations underlined with defects known in chronic alcoholism and (3) whether the alterations are proven to have occurred after binge drinking started rather than possibly be present before.

REFERENCES


CONFLICT OF INTEREST STATEMENT

None declared.


By guest on December 4, 2013 http://alcalc.oxfordjournals.org/ Downloaded from
Neurocognitive findings in binge drinking


