Alcohol and Alcoholism Advance Access published December 2, 2013

Alcohol and Alcoholism Vol. 0, No. 0, pp. 1–9, 2013 doi: 10.1093/alcalc/agt172

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

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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 IRMf 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*., 2013), reductions in bilateral hippocampal (De Bellis *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 (Jacobus *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 diminutions 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, 2013, 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

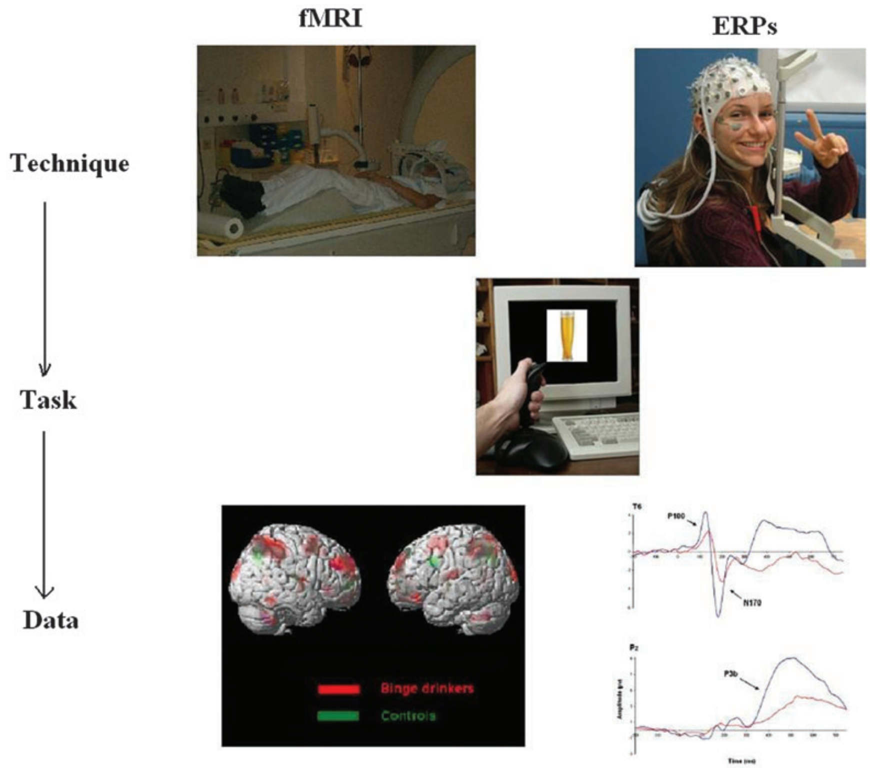

Fig. 1. The use of imaging techniques as event-related potentials (ERPs) and functional magnetic resonance imaging (fMRI) allowed measuring the brain's response during each processing steps involved in specific cognitive tasks. fMRI and ERPs data allowed to highlight brain areas that are activated by the presentation of stimuli in binge drinkers when compared with matched controls, and to stress the presence of abnormal electrophysiological processes when participants are for instance confronted with alcohol vs. non-alcohol cues. Such highly sensitive procedures allowed visualizing even minor cognitive restrictions, which may be observable despite normal behavioral performance.

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 1. Neurophysiological and neuroimaging studies of binge-drinking

Authors (year)	Types of study	Sample characteristics	For longitudinal studies: verification of the absence of difference *before starting* BD habits	Age (years)	Cognitive task	Neuroimaging tools	Cognitive outcome	Neurophysiological and neuroimaging outcome	Examples of studies and reviews depicting similar defects in alcoholic population
Ehlers *et al.* (2007)	Cross-sectional	BD (*n* = 30): >5 drinks in 1 occasion during adolescence (before age 18) C (*n* = 36): no BD history during adolescence	NA	18–25	Facial discrimination task	ERP	BD = C	P3 latency: BD < C	Porjesz and Begleiter (2003)
Crego *et al.* (2009)	Cross-sectional	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	NA	18–20	Visual working memory task	ERP	BD = C	N2 amplitudes: BD > C P3 amplitudes: BD ≠ C	Porjesz and Begleiter (2003), Fein *et al.* (2009) and Verma *et al.* (2006)
Jacobus *et al.* (2009)	Cross-sectional	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	NA	16–19	None	DTI	NA	WM indexed by FA: BD < C	Pfefferbaum *et al.* (2006)
McQueeny *et al.* (2009)	Cross-sectional	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	NA	16–19	None	DTI	NA	WM integrity (indexed by FA): BD < C	Pfefferbaum *et al.* (2006)
Maurage *et al.* (2009)	Cohort (prospective) – over 9 months	BD (*n* = 18) : 12.52 DPO; 2.33 NOW; 35 DPW C (*n* = 18) : no BD	Yes	T1: 18–19	Emotional auditory discrimination task	ERP	T1 and T2: BD = C	T1: BD = C T2: P1, N2, P3b latencies: BD > C	Maurage *et al.* (2008) and Kathmann *et al.* (1996)
Courtney and Polich (2010)	Cross-sectional	Low-BD (*n* = 32): 5/4–7/6 drinks in within 2 h on >1 occasion within the past 6 months High-BD (*n* = 32): ≥10 drinks within 2 h on >1 occasion within the past 6 months Controls (*n* = 32): >1 to 5/4 drinks within 2 h on >1 occasion within the past 6 months	NA	18–22	None	EEG	NA	Spectral power in the delta (0–4 Hz) and fast-beta (20–35 Hz) bands : C = low-BD < high-BD	Bauer (2001)
Crego *et al.* (2010)	Cross-sectional	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	NA	18–20	Visual working memory task	ERP and eLORETA	BD = C	LPC amplitude and aPFC: BD < C	Tapert *et al.* (2004) and Brecher *et al.* (1987)

Schweinsburg *et al.* (2010)	Cross-sectional	BD ($n = 12$): ≥5 (M) or 4 (F) drinks in 1 occasion C ($n = 15$): ≤5 (M) or 4 (F) drinks in 1 occasion	NA	16–18	Verbal encoding task	fMRI	BD = C	BOLD response during novel verbal encoding: BD > C	Tapert *et al.* (2004)
Schweinsburg *et al.* (2011)	Cross-sectional	BD ($n = 16$): ≥5 (M) or 4 (F) drinks in 1 occasion during the last 3 months C ($n = 22$): very limited alcohol use experience	NA	16–18	Verbal encoding task	fMRI	BD = C	Frontal BOLD response during novel verbal encoding: BD ≠ C	Tapert *et al.* (2004)
Squeglia *et al.* (2011)	Cross-sectional	BD ($n = 40$) C ($n = 55$)	NA	16–19	Neuropsychological battery + spatial working memory task	fMRI	Working memory performance: BD ≠ C	Neural activation: BD ≠ C	Tapert *et al.* (2004) and Ambrose *et al.* (2001)
Crego *et al.* (2012)	Cross-sectional	BD ($n = 32$): ≥6 drinks on 1 occasion at a speed of ≥3 drinks/h ≥1 times per month C ($n = 53$) : ≤6 drinks on 1 occasion at a speed of ≤3 drinks/h	NA	18–20	Visual oddball task	ERP	BD = C	P3b amplitudes: BD > C	Porjesz and Begleiter (2003)
Lopez-Caneda *et al.* (2012)	Cohort (prospective) – over 2 years	BD ($n = 23$): ≥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 ($n = 25$): ≤6 drinks on 1 occasion	No	T1: 18–19	Go/NoGo task	ERP and eLORETA	T1 and T2: BD = C	NoGo-P3 amplitude: T1: BD = C; T2: BD > C Go-P3 amplitude: T1 and T2: BD > C Activation in rIFC: T1: BD = C; T2: BD > C	Cohen *et al.* (1997) and Kamarajan *et al.* (2004)
Maurage *et al.* (2012)	Cross-sectional	BD1 ($n = 20$): 5–12 ADO; 2–3 DOW; ADH >3; 15–29 ADW BD2 ($n = 20$): ADO >10; 3–4 DOW; ADH >3; ADW >30 C ($n = 20$): ADO <2; DOW <0.5; ADH <1; ADW <2	NA	19–24	Visual oddball task (face detection)	ERP	BD1 = BD2 = C	P100, N100, N170/P2, N2b/P3a and P3b amplitudes: BD1 < C; BD2 < C P100, N100 and N2b/P3a latencies : BD2 > C P3b latency: BD1 > C; BD2 > C	Porjesz and Begleiter (2003), Maurage *et al.* (2007) and Nicolas *et al.* (1997)
Petit *et al.* (2012a)	Cross-sectional	BD ($n = 18$): ≥6 drinks on 1 occasion at a speed of ≥3 drinks/h C ($n = 18$): ≤6 drinks on 1 occasion at a speed of ≤3 drinks/h	NA	19–25	Alcohol-modified oddball task	ERP	BD = C	P100 amplitude in response to alcohol cues: BD > C	Herrman *et al.* (2000)
Squeglia *et al.* (2012a)	Cross-sectional	BD ($n = 29$) C ($n = 30$)	NA	16–19	Neuropsychological battery	MRI	Visuospatial, inhibition and attention performances: BD ≠ C	Cortical thickness: BD ≠ C	Momenan *et al.* (2012)
Campanella *et al.* (2013)	Cross-sectional	BD: 9.2 ADO, 3.4 ADH, 2.7 DOW C: 3.7 ADO, 1.5 ADH, 0.5 DOW	NA	18–25	Working memory N-back task	fMRI	BD = C	Activity in the pre-supplementary motor area: BD > C	Moselhy *et al.* (2001)
Lisdahl *et al.* (2013)	Cross-sectional	BD ($n = 46$) C ($n = 60$)	NA	16–19	None	MRI	NA	Cerebellar volumes: BD < C	Sullivan *et al.* (2000)

Continued

Table 1. *Continued*

Authors (year)	Types of study	Sample characteristics	For longitudinal studies: verification of the absence of difference *before starting* BD habits	Age (years)	Cognitive task	Neuroimaging tools	Cognitive outcome	Neurophysiological and neuroimaging outcome	Examples of studies and reviews depicting similar defects in alcoholic population
Lopez-Caneda *et al.* (2013)	Cohort (prospective) – over 2 years	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	No	T1: 18–19	Visual oddball task	ERP	T1 and T2: BD = C	P3b amplitudes: T1: BD > C T2: BD > + C	Porjesz and Begleiter (2003)
Petit *et al.* (2013)	Cross-sectional	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	NA	18–27	Alcohol-modified oddball task	ERP	BD = C	P3 amplitudes to alcohol cues: BD > C	Herrmann *et al.* (2000)
Petit *et al.* (submitted)	Cohort (prospective) – over 1 year	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	No	19–25	Alcohol-modified oddball task	ERP	T1 and T2: BD = C	T1: P3 amplitudes to alcohol vs. non-alcohol cues: BD = C T2: P3 amplitudes to alcohol vs. non-alcohol cues: BD ≠ C	Herrmann *et al.* (2000)
Xiao *et al.* (2013)	Cross-sectional	BD ($n = 14$) C ($n = 14$)	NA	16–18	Iowa Gambling Task (IGT)	fMRI	Performance on IGT: BD ≠ C	Neural activity: BD > C	Bechara *et al.* (2001)

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 neurophysiological 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.

Funding — G.P. (research fellow), P.M. (research associate) and S.C. (research associate) are funded by the Belgian Fund for Scientific Research (F.R.S.-F.N.R.S., Belgium).

Conflict of interest statement. None declared.

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