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Commentary

Pathways to alcohol-induced brain impairment in young people: A review by Hermens et al., 2013

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1. Introduction

Alcohol-use disorders, and particularly binge drinking habits, have dramatically increased among adolescents and young adults during the last decade (Johnston et al., 2007; Valencia-Martín et al., 2007) and now constitute a crucial public health problem in Western countries (Gill, 2002; Miller et al., 2007). Nevertheless, if the deleterious consequences of this alcohol-consumption pattern have been largely explored at medical, cognitive and social levels (e.g., Brumback et al., 2007; Laghi et al., 2012), its influence on brain functioning has only been investigated recently. While still being in its infancy, this neuroscience approach of binge drinking has already offered major insights concerning the development and extent of alcohol-related cerebral impairments. Hermens et al. (2013) have recently proposed a review of these studies exploring the brain correlates of excessive alcohol-consumption in young people. By proposing an extensive description of the current knowledge concerning the neuroscience of alcohol-related problems in youth, this article undoubtedly constitutes an essential contribution to this blooming research field as it offers, for the first time, a comprehensive description of the neuropsychological and neuroimaging data collected during the last decade. More recently however, and in complement to the behavioural and functional magnetic resonance imaging (fMRI) results listed by Hermens and colleagues, several studies have used electroencephalographic tools, and particularly event-related potentials (ERPs), to offer new insights on the cerebral correlates of binge drinking in young people.

Due to their high sensitivity, ERPs have the potential to monitor brain electrical activity with a high temporal resolution (on the order of milliseconds). This way, it is possible, during a cognitive task, to observe in healthy subjects the different electrophysiological components representing the different cognitive stages needed to reach a "normal" performance (Rugg and Coles, 1995). Conversely, a highly valuable interest of cognitive ERPs is that it is also possible to identify the electrophysiological component(s) representing the onset of a dysfunction in people presenting cognitive deficits, and then to infer the impaired cognitive stages (Rugg and Coles, 1995). The present commentary thus aims at complementing the review proposed by Hermens and colleagues, by underlining the usefulness of electrophysiology to bring complementary information to the reported neuropsychological and neuroimaging explorations, and by mentioning the most recent results in this research field. In this view, we believe that the results obtained through ERPs offer important complementary precisions on three of the key themes identified by Hermens et al. (2013) in the conclusion of their review.

2. Specific drinking pattern in young people

The definition of binge drinking is still under debate but, as underlined by the authors, it clearly appears that the drinking pattern mainly observed among young people differs from those observed among adults. Indeed, this pattern is most often based on more episodic but more intense alcohol-consumption episodes. A crucial question concerning alcohol-related impairments in youth is thus, as underlined by Hermens et al. (2013), to determine whether this distinctive alcohol-consumption pattern is particularly harmful for the brain in

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comparison to the more regular and moderate consumption observed in adulthood. We would like to mention several recent ERP studies directly exploring the hypothesis that binge drinking consumption might lead to specifically increased cerebral consequences. Animal studies had already suggested that the frequent alternation between intense alcohol-consumption episodes and withdrawal periods, which is typically observed in binge drinking, could be heavily deleterious for the brain (Obernier et al., 2002; Pascual et al., 2007). The first electroencephalographic studies conducted among young human populations clearly demonstrated that binge drinking is associated with cerebral impairments, indexed by disorganized brain electrical activity (Courtney and Polich, 2010) and alterations of ERPs, particularly for the P300 component (Crego et al., 2010, 2012; Ehlers et al., 2007). Nevertheless, as these studies only compared binge drinkers with control non-drinkers (or very low drinkers), they did not allow to distinguish whether these impairments are due to the global amount of alcohol intake or to the specific binge drinking consumption pattern.

In order to answer this question, a very recent study by Maurage et al. (2012) has compared the ERPs in four groups of 20 participants during a simple cognitive task: control non-drinkers, daily drinkers (i.e., drinking 3–5 alcohol doses 5–7 times per week, with a total consumption of 15–29 doses per week), moderate binge drinkers (i.e., drinking 5–12 alcohol doses 2–3 times per week, with a total consumption of 15–29 doses per week) and intense binge drinkers (i.e., drinking more than 10 alcohol doses 3–4 times per week, with a total consumption above 30 doses per week). Importantly, this design allowed to distinguish the *quantity effect* (by comparing moderate and intense binge drinkers, who share the same consumption pattern but with far more intense consumption in the second group) and the *quality effect* (by comparing daily drinkers and moderate binge drinkers, who have an identical total consumption per week, but in a far more concentrated way among binge drinkers). The electrophysiological results showed that, while daily drinkers do not present any brain functioning impairment in comparison to control non-drinkers, moderate and intense binge drinkers present marked deficits in latency and amplitude for the different ERPs components observed (namely P100-N100, N170-P200, N2b-P3a and P3b). This study thus confirmed the *quantity effect* (as impairments were stronger among intense than moderate binge drinkers), but more centrally showed, for the first time among humans, the *quality effect*: while these two groups have a similar global consumption, the moderate binge drinkers presented far stronger cerebral impairments than daily drinkers. This indicates that the alcohol-consumption pattern observed among adolescent and young adult binge drinkers, characterized by a repeated alternation between intoxications and withdrawals, is specifically harmful for brain functioning. In other words, binge drinking is not only deleterious for the brain because of alcohol-consumption *per se*, but also because of the specific consumption pattern observed in this population.

3. Pre-existing versus alcohol-induced neurobiological changes

In the third key theme presented in their conclusion, Hermens et al. (2013) emphasize two main questions respectively concerning: (1) The causal link between brain impairments and binge drinking: are these impairments a direct consequence of alcohol-consumption, or are there pre-existing brain alterations facilitating the development of alcohol-related problems?; (2) The continuum between binge drinking and alcohol-dependence: are the cerebral alterations described in binge drinking comparable to those classically observed among alcohol-dependent adults?

Several neuropsychological or neuroimaging studies mentioned in this review have already given some insights concerning these debates, and led Hermens and colleagues to suggest that binge drinking could rapidly lead to cerebral impairment, and that these impairments might be parallel to those observed in alcohol-dependence. This section will complement these studies by showing that electrophysiological results have recently given some supplementary answers to these questions.

First, concerning the causal link between alcohol-consumption and brain alterations, several electrophysiological studies (Crego et al., 2010, 2012; Ehlers et al., 2007) and very recent fMRI results (Schweinsburg et al., 2011; Squeglia et al., 2011; Xiao et al., in press) have reinforced the neuroimaging results mentioned by Hermens et al. (2013) by clearly describing cerebral alterations in binge drinking, with an increased sensitivity to cerebral effects of alcohol among girls in comparison to boys (Squeglia et al., 2011, 2012a). Nevertheless, all these studies presented the same limit: as they only performed one testing session, they did not give any information concerning the causality of the deficit. To our knowledge, and as mentioned by Hermens et al. (2013), only three longitudinal studies have been conducted to evaluate this causal link up to now (Norman et al., 2011; Squeglia et al., 2009, 2012b), strongly suggesting that binge drinking rapidly leads to brain impairments. In line with these results, an electrophysiological study (Maurage et al., 2009) proposed a specific exploration of this causal link by performing a test–retest study among young binge drinkers. Namely, two groups of 18 first-year university students without past alcohol-consumption were selected on the basis of their expected alcohol-consumption during the forthcoming year: the binge drinker group was expected to initiate a high alcohol-consumption while the control group was expected to have very low personal consumption. ERPs were recorded at the beginning and at the end of the academic year. As expected, no group differences were observed at the first testing session as both groups did not have regular past alcohol-consumption. However, 9 months later, at the second testing session, binge drinkers (who had been initiating binge drinking habits since the first testing session, i.e., having a mean consumption of 35 doses per week distributed in 2–3 occasions) presented significantly slowed-down cerebral activity (i.e., delayed latencies of P100, N200 and P300 components) in comparison to controls (who persisted in a very low alcohol-consumption). This study thus reports the first direct evidence that short-term binge drinking can lead to marked cerebral dysfunction, in the absence of any pre-existing cerebral impairment. This test–retest experiment leads to the proposal that alcohol-consumption might be responsible for the brain alterations observed among young binge drinkers, as already suggested by longitudinal fMRI studies (Norman et al.,

2011; Squeglia et al., 2009), and that alcohol neurotoxicity might have a direct and very fast deleterious influence on brain functioning. These preliminary results will have to be confirmed and extended in future longitudinal studies, notably because it cannot be excluded that several brain modifications could precede and influence the development of binge drinking habits in certain populations (e.g., children of alcohol-dependent individuals). In line with this, a recent neuroimaging longitudinal study (Squeglia et al., 2012b) has confirmed that binge drinking leads to brain alterations, but also suggested that some cerebral deficits could already be present before binge drinking and be involved in the onset of alcohol-consumption.

Secondly, concerning the parallelism in brain alterations between binge drinking during adolescence and alcohol-dependence during adulthood, Hermens et al. (2013) interestingly mention that the same areas (frontal and temporal regions) might be affected in both states and that there might exist a continuity between the brain impairments presented by binge drinkers and those observed in alcohol-dependence. In line with this, several studies (e.g., Enoch, 2006; McCarty et al., 2004; Wagner and Anthony, 2002) elaborated the "continuum hypothesis", suggesting that binge drinking and chronic alcohol-dependence have to be considered as two stages of the same phenomenon, leading to parallel deficits, rather than as independent pathologies. In other words, and while most adolescent binge drinkers will obviously not become alcohol-dependent in adulthood, binge drinkers seem to present the same pattern of impairments as alcohol-dependent individuals, the difference being quantitative (i.e., deficits are more marked in alcohol-dependence) and not qualitative (i.e., deficits affect the same cognitive functions). Hermens et al. (2013) have already mentioned several empirical findings supporting this continuum hypothesis, and this proposal has recently received further empirical support as electrophysiological studies have shown that binge drinking among adolescents or young adults impairs the same ERPs components (notably the P100 and N200) as those classically known to be altered in alcohol-dependence (e.g., Courtney and Polich, 2010; Maurage et al., 2009), these components being preserved in other psychopathological states like major depression or generalized anxiety. This leads to the strong proposal that binge drinking or alcohol-related disorders during adolescence could constitute a favourable ground for the development of alcohol-dependence during adulthood (Schulenberg et al., 1996; Tucker et al., 2003).

4. Vulnerability markers

Hermens et al. (2013) underlined in their conclusions that early cognitive and cerebral deficits related to binge drinking habits may lead young binge drinkers to be more vulnerable for later alcohol-dependence. While these vulnerability markers favouring the extension of binge drinking remain very little understood, several neuropsychological studies identified cognitive impairments that could be involved in the increase and maintenance of binge drinking habits. Indeed, Hermens et al. (2013) mentioned several important studies identifying early neuropsychological alterations among binge drinkers. In complement to these, other studies described impairments in crucial cognitive functions like visuo-spatial abilities (Brumback et al., 2007), attention (Giancola, 2002; Zeigler et al., 2005), memory (Blume et al., 2000) or executive (Johnson et al., 2008) abilities, and this major impairment of memory and executive functions has been further confirmed by very recent results (Heffernan and O'Neill, 2012; Parada et al., 2011, 2012; Sanhueza et al., 2011). These cognitive alterations could appear at early stages of binge drinking and influence the development and maintenance of alcohol-consumption. Up to now however, this proposal has not been directly tested by means of longitudinal studies proposing test–retest measures, and it is thus unclear whether these cognitive impairments are partly preceding binge drinking habits or are a consequence of alcohol neurotoxicity.

Nevertheless, these behavioural data exploring the vulnerability markers of alcohol-related problems in young people have been reinforced and extended by recent electrophysiological investigations. At least three studies used ERPs to explore the brain correlates of executive functions, attention and memory impairments in binge drinking. Crego et al. (2010) showed that binge drinking is associated with a significant impairment of working memory abilities, and that this alteration seems to be due to reduced activations in the right anterior prefrontal cortex, a region classically involved in memory abilities. More recently (Crego et al., 2012), anomalies in the neural processes mediating attentional abilities have been observed (namely an abnormal amplitude of the P300 component), which constitutes the first description of the brain impairments responsible for the attention deficits in binge drinking. Finally, a very recent study (López-Caneda et al., 2012) explored the electrophysiological correlates of the inhibition impairments observed in binge drinking, and showed that the impulse control deficit frequently described among binge drinkers is related to an abnormal brain activity in the right inferior frontal cortex. Interestingly, this study was based on a longitudinal design with two ERPs recordings within a 2-year interval, and its results show that these inhibition impairments are present as soon as the first recording (i.e., short after the beginning of binge drinking habits), suggesting that neural alterations might arise rapidly in the development of binge drinking habits.

Moreover, it has also been shown (Petit et al., 2012) that binge drinkers, in comparison to control light drinkers, present a strong attentional bias towards alcohol, indexed by an increased amplitude of the P100 component when confronted with alcohol-related stimuli (e.g., bottles of alcoholic drinks) in comparison to non alcohol-related ones. These results complement earlier behavioural (Thomas et al., 2005) and neuroimaging (Tapert et al., 2003, 2004) ones obtained in alcohol-use disorders by proving the automatic higher reactivity to alcohol cues in binge drinkers, and their prioritized processing of alcohol-related stimuli. Interestingly, this attentional bias towards alcohol-related stimuli has been repeatedly described among alcohol-dependent individuals (e.g., Ingjaldsson et al., 2003; Sharma et al., 2001), and this parallelism between binge drinking and alcohol-dependence reinforces the continuum hypothesis. Moreover, as it has been shown among recently detoxified alcohol-dependent

individuals that the intensity of this bias is highly correlated with the intensity of craving during withdrawal and with the probability of relapse after detoxification (Field et al., 2009; Schoenmakers et al., 2010), the rapid development of this attentional bias among young binge drinkers could favour the extension and maintenance of binge drinking habits. Nevertheless, these causal links between attentional bias and binge drinking have not been explored yet by longitudinal designs and will thus have to be clarified in future studies.

These electrophysiological results, together with neuropsychological ones mentioned by Hermens et al. (2013), lead to the proposal that a vicious circle could appear during the early stages of binge drinking habits in adolescence and reinforce alcohol-consumption, potentially leading to alcohol-dependence. Specifically, initial binge drinking habits could rapidly lead to the appearance of attentional biases 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). These alterations could facilitate the extension of binge drinking habits, which would in turn increase the cognitive deficits due to alcohol neurotoxicity. This vicious circle between neuropsychological impairments and binge drinking would finally reinforce the alcohol-consumption and favour the progressive apparition of alcohol-dependence. Longitudinal studies at neuropsychological, electrophysiological and neuroimaging levels are nevertheless needed to directly test this hypothesis of a vicious circle in binge drinking.

5. Conclusion

The main purpose of this commentary was to underline that recent electrophysiological studies on binge drinking have brought important information that, alongside neuropsychological and neuroimaging measures, may help to propose a more complete description of the alcohol-induced impairments in young people. Main findings are:

(1) Binge drinking consumption pattern is indeed particularly deleterious for brain functioning, not only because of the quantity of alcohol consumed, but also because of the specific harmful effect of this consumption pattern, alternating strong intoxications and withdrawal periods (Maurage et al., 2012).
(2) Some neurobiological changes might precede the appearance of binge drinking habits, but the neurotoxicity due to excessive alcohol-consumption has a direct deleterious effect on brain functioning, rapidly leading to slowed-down cerebral activations as shown by a test–retest study (Maurage et al., 2009).
(3) Several vulnerability markers of binge drinking have been observed in recent ERPs studies, particularly concerning memory-executive functions (Crego et al., 2010, 2012; López-Caneda et al., 2012) and attentional bias (Petit et al., 2012). A vicious circle with reciprocal influence between cognitive impairments and excessive alcohol-consumption could thus be responsible for the stabilization of binge drinking habits.

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