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Drug and Alcohol Dependence

journal homepage: www.elsevier.com/locate/drugalcdep



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Cognitive control in young heavy drinkers: An ERP study

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ARTICLE INFO

Keywords:
Performance monitoring
Drinking
Alcohol
Error processing
Inhibitory control
Event-related potentials

ABSTRACT

Substance use disorders have been frequently linked to an impaired cognitive control system. Whether this impaired control is also present in young adults who heavily drink alcohol is still subject to debate. The present study investigated possible impairments in cognitive control in heavy drinkers using behavioral and electrophysiological (EEG) measures. We studied behavioral performance on an inhibitory control and an error-processing task, using a GoNogo task and an Eriksen Flanker task respectively, while ERPs (Nogo-N2/P3 and ERN/Pe) were measured in a group of heavy alcohol drinkers (n = 48) and a healthy control group of light drinkers (n = 49). Results showed very few impairments in the heavy drinking group either at the behavioral or physiological level. One exception was the error-related Pe amplitude. This ERP component was reduced in heavy drinkers as compared to controls. Given that the Pe reflects a motivational component (i.e., the salience attributed to the making of errors) rather than a basic cognitive deficit, it can be concluded that heavy drinking in this population is not associated with major impaired cognitive control, but rather with impairments that are associated with aberrant attribution of salience to the making of errors. The present EEG findings are consistent with recent reviews and large scale epidemiological studies showing that heavy drinking, in contrast to substance use disorders, in young persons is not necessarily associated with major behavioral impairments in cognitive control.

1. Introduction

Many studies show that substance use disorders (SUD) are characterized by problems with cognitive control (e.g., Garavan and Weierstall, 2012; Luijten et al., 2014; Noel et al., 2013; Wiers et al., 2013). In particular two aspects of cognitive control have been studied in SUD patients: error-processing and response inhibition (see for a review, Luijten et al., 2014).

Inhibitory control and error-processing can be regarded as two core components of cognitive control that are both associated with specific neural networks and are both crucial to control substance use. More specifically, inhibitory control is the process of inhibiting inappropriate and automatic behavior, whereas error-processing refers to the monitoring of performance errors and ongoing behavior to prevent future mistakes (Ridderinkhof et al., 2004). Deficits in either inhibitory control or error-processing may respectively result in the inability to inhibit substance use intake, and an apparent failure to adaptively learn from previous harmful behavior thereby hampering the ability to prevent excessive substance or alcohol use (Franken et al., 2007).

The GoNogo task is one of the most commonly used tasks to measure inhibitory control (Chambers et al., 2009). In this task,

participants have to respond as quickly as possible to frequent 'Go' stimuli, and inhibit the responses to infrequent 'Nogo' stimuli thereby requiring inhibitory control to overcome automatic response tendencies. Two ERP components have been reported to reflect changes in brain activity related to inhibitory control (Kok et al., 2004). The Nogo-N2 is a negative-going wave visible 200–300 ms after stimulus presentation and is thought to index a top-down mechanism necessary to inhibit the automatic tendency to respond (Falkenstein, 2006). The Nogo-N2 has also been related to early-stage conflict detection during the inhibition process (Nieuwenhuis et al., 2003). The Nogo-P3 is a positive-going wave visible 300–500 ms after stimulus onset. The Nogo-P3 arises from motor and pre-motor cortices (Huster et al., 2010). Hence, Nogo-P3 amplitudes likely reflect a later stage of the inhibitory process when actual inhibition of the motor system in the premotor cortex takes place (Band and Van Boxtel, 1999).

Several studies among alcohol dependent patients showed reduced response inhibition using behavioral indices (e.g., Lawrence et al., 2009; Noël et al., 2007; Rubio et al., 2008), which was confirmed in a recent meta-analyses clearly showing that substance use disorders, including alcohol dependence, are associated with impairments in inhibitory control on the behavioral level (Smith et al., 2014). At the

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neural level, some studies suggest Nogo-P3 deficits in individuals with alcohol dependence during inhibition-related task performance (see Luijten et al., 2014 for a systematic review).

For error-processing, the most commonly used paradigm is the Eriksen Flanker task. In a typical version of the Eriksen Flanker task (Eriksen and Eriksen, 1974), participants are exposed to series of letters and participants are asked to identify the middle letter. In the incongruent condition the middle letter differs from the other letters (e.g., SSHSS/HHSHH) as opposed to the congruent condition (HHHHH/SSSSS). The high stimulus conflict situation in the incongruent condition usually results in performance errors, making it possible to measure the brain's response to mistakes.

On the electrophysiological level, two independent error-related ERPs consistently emerge after performance errors, i.e., the error-related negativity (ERN) and the error- positivity (Pe) (Overbeek et al., 2005). The ERN arises 50–80 milliseconds after an error and reflects initial and automatic error detection with the anterior cingulate cortex as the neural generator (Bernstein et al., 1995). In contrast, the Pe is a positive EEG peak, emerging approximately 300 ms after incorrect responses with a centro-parietal distribution (Falkenstein et al., 2000). Conceptually, the Pe appears to be associated with the more conscious evaluation of errors, error-awareness (Overbeek et al., 2005), and the motivational significance attributed to an error (Ridderinkhof et al., 2009).

Previous studies consistently showed reduced ERN and Pe amplitudes in various addicted populations, including cocaine dependent patients, smokers as well as behavioral addictions such as food addiction and gaming (Franken et al. in press; Luijten et al., 2014). In contrast to the other addicted populations alcohol dependent patients showed increased ERN amplitudes, which could be explained by enhanced anxiety levels observed in this sample of alcohol dependent patients (Schellekens et al., 2010).

From the abovementioned studies, it can be concluded that there are behavioral and electrophysiological indications that alcohol dependence is associated with deficits in both inhibitory control and errorprocessing. However, it is not clear whether these deficits are specific for patient populations or whether these deficits might also be observed in high-risk populations. One important high-risk population is the group of young heavy drinkers (i.e., adolescents and young adults). It is known that heavy drinking in adolescence and young adulthood is associated with substance use disorders (SUDs) in adulthood (DeWit et al., 2000). Besides this risk, heavy drinking among students is related to a series of negative alcohol-related consequences and psychosocial problems (Perkins, 2002; Turrisi et al., 2006; Wechsler et al., 1998). In addition, there is growing literature on the relation between bingedrinking, particularly among college-aged populations, showing that an intermittent-but-high alcohol use pattern is associated with less inhibitory control (see a review by Lopez-Caneda et al., 2014), and a range of ERP indices of aspects of cognitive control (see a review by Petit et al., 2014). More specifically, several studies provide indications that heavy drinking during adolescence and young adulthood might indeed be associated with electrophysiological and behavioral indices of reduced cognitive control discussed above (i.e., error-processing and response inhibition measured using the GoNogo task). For example, Smith and Mattick (2013) found evidence for deficits in response inhibition and error processing in young female heavy drinkers. However, that sample size was relatively small (13 heavy drinkers), which makes it difficult to draw firm conclusions. A recent review and meta-analysis (Smith et al., 2014) showed that heavy drinkers, in contrast to patient populations did not show any significant deficits while performing a GoNogo task, but did show a small deficit in inhibitory capacity in the stop-signal task. In general, the deficits seem smaller in heavy non-dependent drinkers as compared to dependent drinkers as there is some evidence for the idea that the deficit is dosedependent (Smith et al., 2014). Remarkably, the deficits that are observed in heavy drinkers seem only to be observed in female

Table 1
QFV –categories of drinkers.

Average number of drinking days in a month		Units of alcohol taken		_
	6 or more	4 or 5	2 or 3	0 or 1
28 or more	Very excessive	Excessive	Average	Light
21–27	Very excessive	Excessive	Average	Light
15–20	Excessive	Average	Average	Light
9–14	Excessive	Average	Light	Light
3–8	Average	Light	Light	Light
0–2	Light	Light	Light	light

populations (Nederkoorn et al., 2009; Smith and Mattick, 2013; Smith et al., 2015), suggesting a gender-specific effect. In contrast to these experimental studies, recent large-scale epidemiological studies question the presence of these cognitive deficits in heavy drinking (non-addicted) populations. In an important large longitudinal study (Boelema et al., 2015) among 2230 adolescents who were followed for about 8 years, the authors conclude that four years of weekly heavy drinking did not result in impairments in basic executive function, including inhibitory control. However, in that study no psychophysiological indices of cognitive control were measured, which are arguably more suitable to detect subtle deficits.

Given the contrasting findings concerning the presence of deficits in cognitive control associated with heavy alcohol drinking, we investigated in the present study whether error- processing and inhibitory control are reduced in young heavy drinkers as compared to light drinking controls. We measured both electrophysiological correlates and behavioral correlates of these functions.

2. Methods

2.1. Participants

Participants were recruited by written and verbal advertisement on the university campus. The educational level for all subjects was equal for both groups as we recruited only subjects following higher education. Both groups (i.e., light and heavy drinkers) were selected from a larger population that was screened on alcohol use using the Quantity-Frequency-Variability index (QFV; Bongers et al., 1997; Lemmens et al., 1992). The QFV measures alcohol consumption by four questions: "Which alcoholic drinks do you usually drink when you drink?"; "How many days a month do you drink on average?"; "If you drink alcohol, how many glasses do you drink on average?"; "Have you ever drunk six or more glasses at one day in the past six months?" Based on this QFV, participants were categorized either as light drinkers or as heavy drinkers (i.e., the joined categories "very excessive" and "excessive" drinkers; see Table 1).1 The QFV was assessed also in the week of testing. If the person did not fill the criteria anymore, he or she was not invited to participate in the study.

The resulting group of participants consisted of 49 light drinkers and 48 heavy alcohol drinkers. Mean age of the heavy drinkers was 23.4 years (SD = 10.0) and mean age of the light drinkers was 22.9 (SD = 8.5). The groups were matched on gender, age and education. No differences between these variables were observed (see Table 2, all ps > .75).

For the ERN/Pe EEG analyses ten participants (4 heavy drinkers and 6 light drinkers) were excluded from the analyses because they either

 $^{^{1}}$ If all persons (n=23) who fulfill the criteria of having a (albeit infrequent) binge drink were removed from the light drinking group (i.e., by excluding persons who drink > 3 glasses on average per drinking day), the analysis yield the same results, except for the Pe difference, which becomes non-significant (probably due to reduced power).

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