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Sex differences in the relationship between heavy alcohol use, inhibition and performance monitoring: Disconnect between behavioural and brain functional measures

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ABSTRACT

Previous research has reported mixed evidence of sex differences in the relationship between heavy alcohol use and deficits in behavioural control. Here, we examine sex differences in behavioural and event-related potential (ERP) markers of deficient inhibition. Participants were 71 young adults aged 18–21, who either drank heavily regularly (i.e., four standard drinks on one occasion, at least once a month, n=33, 20 male) or drank heavily less often than this (including never, n=38, 21 male). They completed a stop-signal task while ERPs were recorded. Increases in stop-signal reaction time, the time required to stop a response, were related to heavy drinking only in female participants. P3 amplitude, ERN amplitude and ERN latency did not display a significant interaction between group and sex. Heavy drinkers, regardless of sex, displayed a marginally larger successful > failed effect for P3 amplitude, and a marginally smaller error-related negativity. An apparent disconnect exists in behavioural and psychophysiological measures of sex differences in the relationship between heavy alcohol consumption and inhibitory processing; male heavy drinkers display only psychophysiological but not behavioural deficits, while female heavy drinkers display both. Future research may determine whether sex differences are apparent for other substances besides alcohol.

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1. Introduction

Deficits in the control of overt behaviour have attracted growing interest as a factor behind the development and maintenance of substance abuse, as well as relapse (Goldstein and Volkow, 2002; Crews and Boettiger, 2009; Hester et al., 2010; Jentsch and Pennington, 2014), such that substance abusers are less able to control use behaviours given the opportunity to use. We recently published a meta-analysis of inhibitory deficits in users of a range of substances (Smith et al., 2014), and showed that a deficit in behavioural inhibition, as measured by the stop-signal task (Logan and Cowan, 1984; Logan et al., 1984), is apparent not only for alcohol dependence (with participants primarily aged 30–45), but also for heavy drinkers (with participants primarily aged 18–30). However, it was noted in the discussion of the meta-analysis that the appraised studies included mostly male participants (overwhelmingly so for alcohol dependence). Despite evidence of sex differences in alcohol consumption, prevalence of alcohol-related disorders and alcohol-related health problems (reviewed by Erol and Karpyak, 2015), few studies consider sex as a factor in their analyses; of those that do, some report no differences (van der Plas et al., 2009; Henges and Marczinski, 2012; López-Caneda et al., 2012; Rossiter et al., 2012), and others have reported strong behavioural inhibition deficits for female, but not male heavy drinkers (Townshend and Duka, 2005; Nederkoorn et al., 2009; Kreusch et al., 2013; Weafer et al., 2015), leading to the possibility that inhibitory deficits may be underestimated when mostly male participants are included. Other researchers also report sex differences in the relationship between heavy alcohol consumption and brain structure and function, particularly in inhibition-related areas (e.g., Hommer, 2003; Squeglia et al., 2011, 2012; Ide et al., 2014), and some reports event suggest greater brain activity linked to inhibitory processing in healthy male controls compared to female controls, regardless of alcohol consumption (e.g., Li et al., 2006). These observations indicate that examination of sex differences in the relationship between alcohol consumption and inhibitory capacity, in particular in relation to brain function, is warranted.

One measure of brain function reliably linked to inhibition is the P3 component of the event-related potential (ERP), which occurs around 300–600 ms after stimulus presentation and is

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reliably different for trials on which inhibition is required, compared to when inhibition is not required (e.g., Randall and Smith, 2011; Smith et al., 2013; Groom and Cragg, 2015), and more pertinently to the current study, when inhibition is successful compared to unsuccessful (e.g., Kok et al., 2004; Wessel and Aron, 2015). Evidence of deficits in alcohol dependence as measured by the P3 component is mixed (for a review, see Luijten et al., 2014); for example, some researchers have observed differences in the inhibitory P3 in alcohol dependence (Cohen et al., 1997; Kamarajan et al., 2005; Colrain et al., 2011), while others have not (Pfefferbaum et al., 1987; Fallgatter et al., 1998; Karch et al., 2008). Furthermore, some of these researchers also observe differences in the P3 for response execution trials (Pfefferbaum et al., 1987; Cohen et al., 1997; Kamarajan et al., 2005), suggesting that deficits are not specific to the inhibitory process (indeed, Euser et al. (2012) report a meta-analysis showing reduced P3 amplitude in alcohol dependent participants performing the oddball task, which does not require response inhibition). To our knowledge, there are no published studies of P3 for failed versus successful inhibition trials in alcohol dependent participants. A similar story of mixed evidence exists for heavy drinkers: for response inhibition trials, researchers have reported for heavy drinkers relative to controls, increases in P3 amplitude (López-Caneda et al., 2012, and marginally for Watson et al., 2014), no difference (Petit et al., 2012) and decreases in P3 amplitude (Oddy and Barry, 2009), again sometimes in the context of differences on response execution trials (López-Caneda et al., 2012). López-Caneda et al. (2012) report no interactions of group and sex for Go and NoGo P3 amplitude, but Watson et al. (2014) report a marginally larger NoGo P3 in male binge drinkers. In a previous article, we reported the first study of the relationship between heavy drinking and event-related potential measures of failed and successful response inhibition in young females aged 18–21 (Smith and Mattick, 2013, 2014). Heavy drinkers showed a larger successful > failed effect for P3 amplitude, indicating that they triggered the inhibitory process more weakly than controls, and had to trigger it more strongly in order to successfully inhibit a response. In the current study, we re-examine (with permission) this previously published data from females in relation to newly acquired data from males using the same experimental protocol.

In addition to examination of inhibitory ERPs, we also examine ERPs linked to error processing/performance monitoring. The error-related negativity, or ERN, is a frontally maximal negative component occurring in the first 100 ms after an erroneous response is made; it is larger in amplitude for errors of a larger magnitude (Falkenstein et al., 2000) and occurs irrespective of participants' awareness that an error has been made (Nieuwenhuis et al., 2001), thus is considered to index an unconscious error detection mechanism. ERN has been less well-studied in relation to substance abuse; only two studies (Schellekens et al., 2010; Padilla et al., 2011) have examined ERN in alcohol dependence, reporting larger amplitude (increased performance monitoring) in contrast to other substances, where decreased ERN amplitudes are observed (Luijten et al., 2014), possibly due to comorbid anxiety (which is linked with increased monitoring) in the alcohol dependent groups. Furthermore, the few studies of ERN in heavy drinkers have reported inconsistent results also: recent work from our lab shows decreased ERN in female heavy drinkers and increased ERN in male heavy drinkers (Smith et al., 2015), in contrast to the female heavy drinkers performing the stop-signal task in our earlier study (Smith and Mattick, 2013), who displayed *decreased* ERN amplitude relative to female controls. In the current study, we examine whether this effect is upheld in male heavy drinkers in the stop-signal task.

2. Methods

2.1. Participants

The participant recruitment and exclusion criteria, experimental protocol, recording parameters and ERP extraction processes were identical to those used in Smith and Mattick (2013, 2014). Participants were 41 males and 30 females aged 18–21 who were recruited into two drinker groups: those who engaged in heavy drinking (4 or more Australian standard drinks, totalling more than 40 g alcohol, on one occasion) at least monthly in the preceding 12 month period ('heavy drinkers' group, males: n=21; females: n=13), and those engaged in heavy drinking less often than this, including never ('control' group, males: n=20; females: n=17). The most recent Australian health guidelines concerning risky drinking provide the same recommendation for males and females (National Health and Medical Research Council, 2009); therefore we used identical criteria for our male sample here as for our female sample (Smith and Mattick, 2013, 2014). Table 1 shows the demographic characteristics of the participants in the study. Participants were recruited via posters displayed on the university campus, as well as an online research participation website, and were excluded if they had ever had a serious head injury, period of unconsciousness or epileptic seizure, uncorrected hearing or vision problems, or regular (more than once a month) use of other drugs. Additionally, participants reported no use of medication or past history of psychiatric disorders. All aspects of the protocol were approved by the University of New South Wales Human Research Ethics Committee and participants gave written informed consent before data collection began.

The experimenter described the experimental protocol to the participant before consent was obtained. Participants completed a demographics questionnaire and a modified version of the Alcohol Use Disorders Identification Test (AUDIT, Saunders et al., 1993). Question 3 of the AUDIT was modified from "How often do you have six or more standard drinks on one occasion? " to "four or more standard drinks" to reflect Australian alcohol consumption guidelines advising that both males and females should consume no more than four standard drinks on one occasion (National Health and Medical Research Council, 2009). The answer to this question was used to create control and heavy drinker groups as specified above. Participants were requested to reference a standard drinks guide provided while they completed this section. Participants also completed the first section of the Drug Use Disorders Identification Test-Extended (DUDIT-E, Berman et al., 2007), assessing the frequency of use of a range of drug classes, which was used to confirm eligibility of the study.

All participants underwent structured interviews assessing lifetime alcohol consumption and lifetime cannabis consumption (collected for a separate study) using modified versions of the Lifetime Drinking History interview (Skinner, 1977). This assesses the frequency and quantity of alcohol consumption in relatively homogenous phases from the onset of regular drinking (at least one standard drink per month). The amount consumed per month during each phase can be calculated (as the number of drinks per drinking day multiplied by the number of drinking days per month), multiplied by the number of months duration of that phase, and summed across phases, to estimate the lifetime number of standard drinks consumed. Because distributions were non-normal, a log transform was applied to the lifetime standard drinks measure (one standard drink was added to each score to avoid taking the log of zero). Participants again referred to the standard drinks guide during the alcohol section of the interview.

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