Behavioural disinhibition precedes heavy drinking in young adults
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BACKGROUND
The stop-signal task (SST) is a reliable test of behavioural inhibition (BI) which produces the stop-signal reaction time (SSRT), see figure 1. It has previously been observed that heavy drinkers have increased SSRT. Furthermore, P3 and N2 amplitude are known to be decreased in heavy adult drinkers.

Binge drinking causes impairment to frontal brain regions and it is unknown how it directly effects BI in a young adult population who are still undergoing brain development.

Additionally, impulsivity has neural correlates with frontal brain regions and has been linked with increased alcohol consumption.

AIMS
To examine changes to BI pre to post binge drinking induction, using SSRT and ERP components, P3, N2, and ERN amplitude.
To determine if a relationship exists between impulsivity and BI. Barrett Impulsivity Scale (BIS-11) scores were correlated with BI measures (SSRT, P3, N2 and ERN amplitude).

METHOD
Participants: 35 student participants (19 female), aged 17–25 years. Never consumed four or more standard drinks (binged) before session one.
Groups (sorted after session two based on drinking reports):
- Non-bingers (did not binge in 3 months between sessions, n = 16)
- Bingers (binged at least once in 3 months between sessions, n = 19)

Procedure
Session 1: Administered drinking habit, Alcohol use disorder identification test (AUDIT), and BIS-11 questionnaires. Then performed a visual SST while EEG was recorded.

3 months interval between sessions.
Session 2: Administered AUDIT and drinking habits in past 3 month questionnaires. Performed visual SST while EEG was recorded.

SST: Green arrow (go stimulus) presented on screen prompting participants to indicate direction of arrow with key press. On 25 percent of trials, green arrow turned red (stop signal), at different delays (mean reaction time = 450, 350, 250, 150, or 50 ms), signaling participants to inhibit their responses. SSRT was then calculated.

EEG recording: 64 channel EEG cap, impedances kept below 5 kΩ. Signals recorded DC to 200 Hz, amplified 10 times and sampled at 1000 Hz using Neuroscan software.

RESULTS

Table 1. Group results for drinking and impulsivity questionnaires, and SSRT data at each session. All measures had group effects (p < .05). Examining the effect of session, Bingers had a significant increase in drinks consumed at session 2 (p = .005) and AUDIT score (p = .000). There was no session effect for SSRT and no interactions.

![Figure showing SSRT distribution](Image)

Figure 1. The horse race model of response inhibition. The red arrows represent the different stop-signal delays, while the orange lines under the curve represent their associated calculated internal SSRT. The different delays result in a probability distribution of being able to successfully inhibit a response (right of the dotted line), or fail to inhibit a response (left of dotted line).

![Figure showing ERP waveforms](Image)

Figure 2. ERP grand averages at the FCZ electrode for failed (blue) and successful (red) stop-signal trials, for each group at session 1 and 2. (p < .05). N2 and P3 components shaded orange and P3 shaded grey. For both N2 and P3 amplitude there was an effect of trial type (p < .01). Session and group effects were insignificant (p > .05). Grand mean error-related waveforms at the FCZ electrode for Non-bingers (blue) and Bingers (red) at session 1 and 2 (f). ERN component in Bingers group significant effect on ERN amplitude (p = 0.024).

CONCLUSION
The present study revealed no changes behaviourally (SSRT) nor in the ERP components P3, N2, or ERN, between session one and two. This indicates the binge drinking that occurred in the 3 months between session did not have an effect on BI.

Group differences were found in both behavioural (SSRT) and electrophysiological measures (ERN amplitude) before any binge drinking commenced. Those who binged showed deficits in SSRT and reduced ERN amplitude at session 1, indicating reduced BI. This suggests that BI deficits may lead to risky drinking behaviours.

Participants who commenced binge drinking were more impulsive, which is concurrent with models of addiction. Impulsivity was only related to the AUDIT after the occurrence of binge drinking, which has been previously shown. P3 amplitude difference was the only BI measure to correlate with impulsivity, therefore there may be other factors attributing to addiction development separate from BI deficits.

More work is needed to examine the effect of binge frequency and intensity, as this study did not compare the difference between a single session or multiple session of binge drinking.

REFERENCES