



Behavioural disinhibition precedes heavy drinking in young adults

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BACKGROUND

The stop-signal task (SST) is a reliable test of behavioural inhibition¹ (BI) which produces the stop-signal reaction time (SSRT), see figure 1. It has previously been observed that heavy drinkers have increased SSRT². Furthermore, P₃ and N₂ amplitude are known to be decreased in heavy adult drinkers².

Binge drinking causes impairment to frontal brain regions³ and it is unknown how it directly effects BI in a young adult population who are still undergoing brain development⁴.

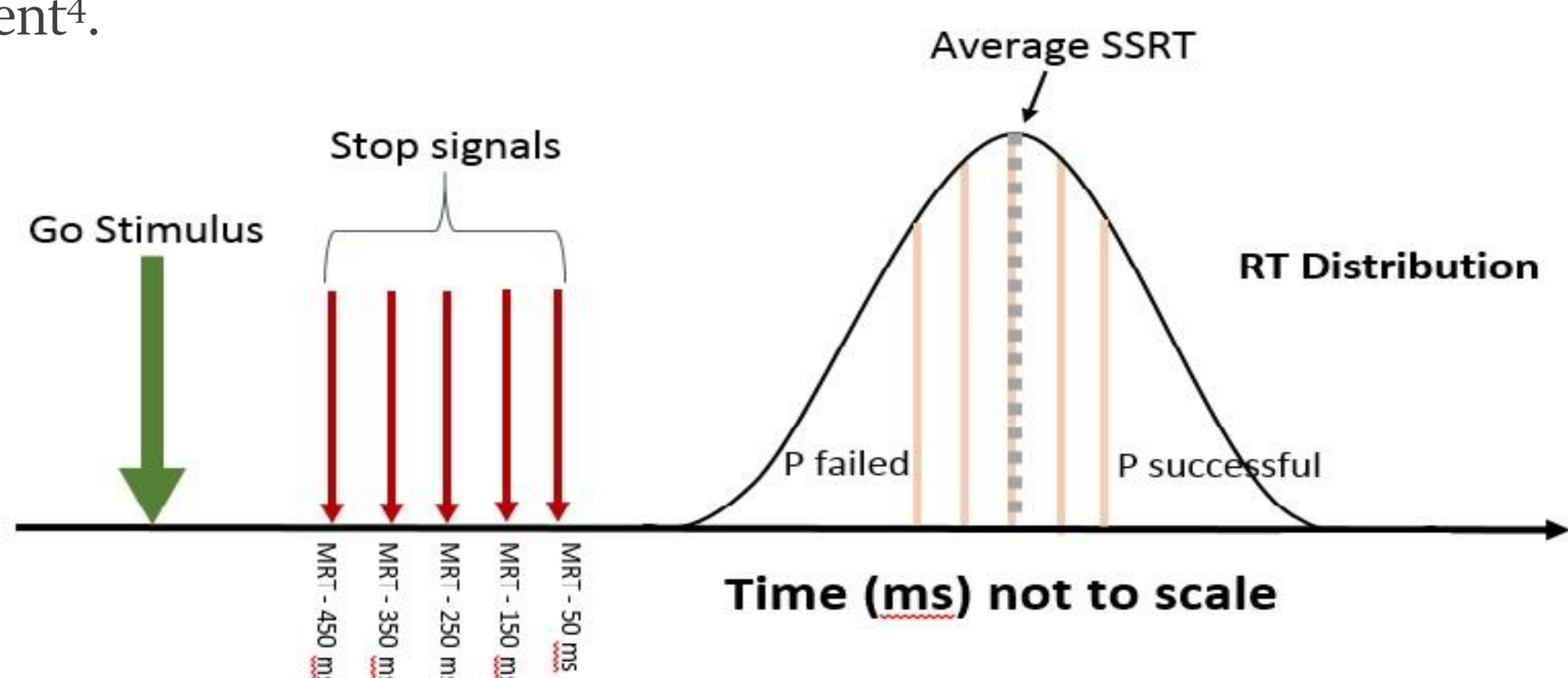


Figure 1. The horse race model of response inhibition⁵. The red arrows represent the different stop-signal delays, while the orange lines under the curve represent their associated calculated internal SSRT. The different delays result in a probability distribution of being able to successfully inhibit a response (right of the dotted line), or fail to inhibit a response (left of dotted line).

Additionally, impulsiveness has neural correlates with frontal brain regions and has been linked with increased alcohol consumption³.

AIMS

To examine changes to BI, pre to post binge drinking inducement, 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 calculated⁵.

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.

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.

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 < .05) and AUDIT score (p = .001). There was no session effect for SSRT and no interactions.

	Session	Mean (SD)		p value
		Non-bingers	Bingers	
Drinking habits (standard drinks/week)	1	0.74 (1)	2.30 (2.64)	.003
	2	0.94 (1.22)	4.04 (3.51)	.002
AUDIT	Overall	1.03 (1.09)	3.00 (1.15)	.000
	1	0.81 (0.75)	2 (1.45)	.006
	2	1.25 (1.53)	4 (1.94)	.000
BIS-11	1	56.75 (8.84)	64.63 (10)	.020
SSRT (ms)	Overall	218.09 (26.51)	248.12 (27.95)	.003
	1	213.78 (28.69)	245.19 (34.37)	.007
	2	222.41 (31.96)	251.04 (39.3)	.026

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 (a - d). N₂ 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 (all p values > .05). Grand mean error-related waveforms at the FCz electrode for Non-bingers (blue) and Bingers (red) at session 1 (e) and 2 (f). ERN component shaded in blue. There was a significant group effect on ERN amplitude (p = 0.024).

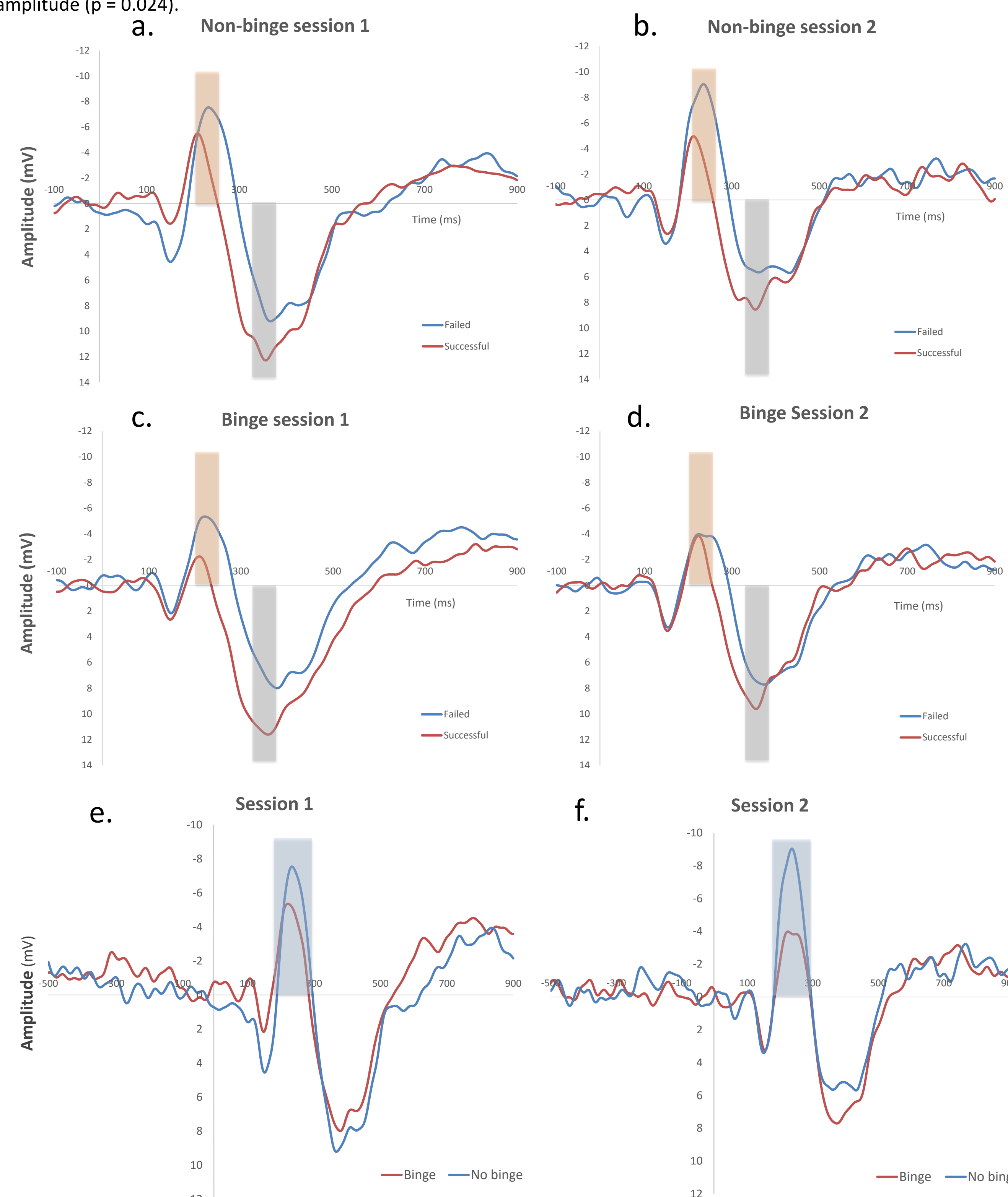
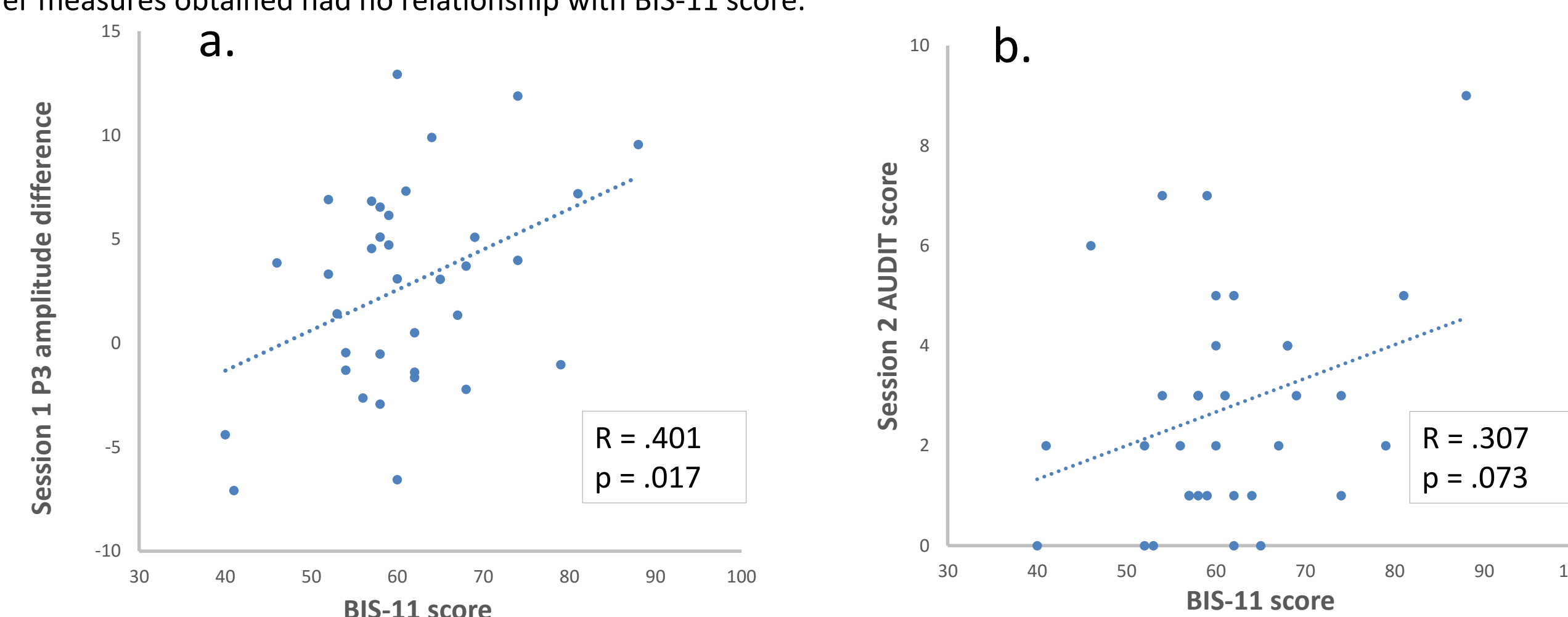


Figure 3. Strong correlations of impulsivity (BIS-11 score) and P₃ amplitude difference at session 1 (a), and AUDIT score session 2 (b). All other measures obtained had no relationship with BIS-11 score.



Participants who commenced binge drinking were more impulsive, which is concurrent with models of addiction⁷. Impulsivity was only related to the AUDIT after the occurrence of binge drinking, which has been previously shown³. 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.

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