No. of tables = 3

**The associations between proactive slowing, working memory, alcohol sensitivity and alcohol use.**

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**Abstract**

*Objective:* ‘Reactive’ inhibitory control is associated with heavy drinking and alcohol dependence. However, the majority of research ignores the downstream influence of proactive control – the preparation to withhold responses when examining alcohol-use behaviours. The potential mechanisms behind these relationships are also poorly understood. These studies aimed to investigate the role of proactive and reactive control in heavy drinkers, in the presence of alcohol-related cues and to examine the potential mediating effects of working memory capacity (WMC) and alcohol-sensitivity (AS). *Method:* In two studies, heavy drinkers completed online self-reported measures of alcohol use followed by a modified Stop-Signal task in the presence of alcohol related cues (images – study 1; words – study 2) and a Self-Ordered Pointing Task using neutral-related images (study 1) and alcohol-related images (study 2). *Results:* In Study 1 craving and WMC predicted alcohol use. In study 2 WMC was a negative predictor of alcohol use alongside Stop Signal reaction times, however the overall regression model was not significant. When conducting pooled analyses across both studies to increase power only a robust association between craving and alcohol use was demonstrated. *Conclusions:* This data provides no support for the associations between WMC, reactive control and proactive slowing and overall alcohol use.

**Key words:** alcohol, inhibitory control, proactive slowing, working memory, alcohol sensitivity

**Introduction**

Inhibitory control is the (in)ability to inhibit behaviours that are inappropriate under current circumstances, and is closely linked to impulsivity and self-regulation (Baumeister, 2014; Bickel et al., 2012). The inability to inhibit incongruous behaviour has been associated with hazardous drinking (Christiansen et al., 2012; Colder & O'Connor, 2002; Paz et al., 2016) and alcohol use disorders (Smith et al., 2014). Inhibitory control is thought to fluctuate *within individuals* in response to various psychological and environmental triggers, including alcohol intoxication and alcohol-cue exposure (de Wit, 2009; Jones, Christiansen, et al., 2013), with these fluctuations playing a causal role in alcohol consumption/(re)lapse. Meta-analyses suggest small but robust impairments in inhibitory control following alcohol cue-exposure (Jones, Robinson, et al., 2018), however there are also failures to demonstrate this effect (Baines et al., 2019; Jones, Rose, et al., 2013).

To date the majority of research in the field has focused on ‘reactive’ inhibitory control, which is the (unobservable) act of stopping or withholding a response, and is operationalized as inhibition errors/success or Stop Signal Reaction time on the Go/No-Go and Stop Signal tasks, respectively (Verbruggen, McLaren, et al., 2014). However, to effectively inhibit behavior requires a number of distinct downstream processes including action selection, the detection of an environmental signal to inhibit, and response execution, all of which may be influenced by proactive slowing (i.e. preparation) (Verbruggen, McLaren, et al., 2014). A failure to consider the role of preparation on these processes leads to over simplistic assumptions of the relationship between alcohol-related cues and inhibitory control. Indeed, Aron (Aron, 2011) suggests that proactive slowing may be a more appropriate model of inhibitory control in explaining real-world substance use behaviors. It seems more likely that individuals who are attempting to limit alcohol consumption will proactively adjust their behavior to suppress urges over a prolonged period of time, rather than relying on fast, reactive inhibition that acts as a late correction mechanism (Braver, 2012; Braver et al., 2009).

Research suggests that alcohol-related cues may induce cognitive biases that influence proactive slowing and the execution of a reactive stopping response (Stacy & Wiers, 2010). Recent research has developed methods to disentangle proactive from reactive control, in order to separately measure their effects. Verbruggen et al (Verbruggen, Stevens, et al., 2014) incorporated a block of trials in which there was no inhibition signal on a Stop Signal task (SST), and compared the reaction times on this block to a block of trials where inhibition was required. The slowing of reaction times when inhibitory control is required (compared to not being required) is indicative of strategic proactive adjustments in control (Verbruggen & Logan, 2009b). In two recent studies (Baines, et al., 2019), we used a similar version of this task to examine if i) heavy drinkers employed proactive control and ii) if this was impaired by alcohol intoxication or exposure to alcohol-related cues. We demonstrated that heavy drinkers did utilise proactive control (i.e. they proactively slowed responses in anticipation of inhibiting), but there was limited impairing effects of alcohol intoxication or cue-exposure. These findings contrast previous research by Sharma (2017) who demonstrated that light drinkers proactively adjusted behaviour in response to alcohol-related cues in a Stroop task, whereas heavy drinkers relied on their reactive control as a late correction mechanism (see also (Braver, 2012)).

It is important to attempt to clarify the contrasting findings above, and one potential reason for these conflicting results is that the mechanisms underlying the preparation to inhibit responses are not well understood (Criaud et al., 2012). Theoretical models suggest that individual differences in Working Memory Capacity (WMC) might account for variance in the ability to implement both proactive and reactive control (Braver, 2012; Richmond et al., 2015). Individuals with greater capacity and more efficient WMC are more able to actively maintain goal-directed behaviour, by actively remembering and updating task rules (e.g. ‘*inhibition is (not) required at this time, under these circumstances’*) (Braver, 2012; Richmond, et al., 2015). In support of this hypothesis, research has demonstrated that individuals with a high-WMC perform better than those with a low-WMC on the AX-Continuous Performance Test (e.g. (Redick & Engle, 2011; Wiemers & Redick, 2018)), a task which measures proactive and reactive control (Gonthier et al., 2016). Performance on this task has suggested that individuals with a lower-WMC tend to be less proactive than those with higher-WMC (Wiemers & Redick, 2018), and rely more on their reactive control (Richmond, et al., 2015). Therefore, these studies support the notion that individual differences in the use of proactive control may depend on WMC. This could have important implications for understanding substance misuse as evidence suggests that both substance dependent individuals and heavy drinkers show impairments in working memory (Bechara & Martin, 2004; Mahedy et al., 2018; Noël et al., 2001).

Event-related potential (ERP) research has also demonstrated that alcohol-related stimuli capture the attention of individuals who self –report low sensitivity (LS) to alcohol (e.g. (Bartholow et al., 2010; Fleming & Bartholow, 2014)). These individuals have a low level of response to the acute effects of alcohol, which may lead to increased consumption of alcohol per drinking session in order for the individual to experience the desired effects (Schuckit et al., 2011). A LS to alcohol is therefore considered a risk factor for alcohol misuse and dependence (Fleming & Bartholow, 2014; Schuckit & Smith, 2000). Alcohol sensitivity can be measured using self-report measure (discussed below) or by measuring blood alcohol concentration following a dose of alcohol (Schuckit, et al., 2011). Importantly, it has been demonstrated that when LS individuals are faced with task irrelevant alcohol-related stimuli, they experience conflict. When conflict is infrequent, individuals can overcome it by using reactive control effectively, however, when this conflict increases, these individuals have difficultly using proactive control efficiently (Bailey & Bartholow, 2016). Therefore, it is possible the individual differences in alcohol sensitivity may contribute to the effective use of proactive and/or reactive control in the presence of alcohol-cue exposure.

Therefore, the aim of these two online studies was to clarify the role of proactive slowing and reactive control in heavy drinkers, in the presence of alcohol-related cues (images – study 1, and words – study 2). We also sought to examine the potential mediating effects of WMC specifically in response to neutral images (study 1) and alcohol-related images (study 2), and alcohol-sensitivity (study 2). Study 1 was not pre-registered, however the design, statistical power calculations, hypotheses and analyses for study 2 were pre-registered on Open Science Framework [<https://osf.io/ctp2w/>]. Data is available for both studies on Open Science Framework.

**Study 1**

In this study heavy drinkers completed a modified SST (based on (Baines, et al., 2019)) designed to measure proactive slowing and reactive control in the presence of alcohol-related images. They also completed the Self-Ordered Pointing Task (SOPT) to measure their WMC and self-reported measures of alcohol consumption. We predicted that (i) individual differences in reactive control, proactive slowing, craving and WMC would be associated with individual differences in overall alcohol use. We included craving as previous research has demonstrated both inhibitory control and craving partially mediate alcohol consumption in the laboratory (Field and Jones, 2017). We also predicted that (ii) individual differences in WMC would be associated with individual differences in proactive slowing and (iii) WMC would mediate the relationship between proactive slowing and alcohol use.

**Methods**

**Participants**

Heavy drinkers (N=108; 82 female), with a mean age of 24.11 (±8.55) participated. The number of participants was decided upon using an a-prioi power calculation to detect a medium effect size (F² = .15) at α = .05, and 90% power with four predictors (craving, reactive control, proactive slowing, working memory), using G\*Power (R2 deviation from 0). Participants were recruited via opportunity sampling from the university and wider community using social media and advertisements. Inclusion criteria were; aged 18+, heavy drinking (> 14 units per week) and access to a PC/laptop/i-pad. Exclusion criteria involved a current or previous diagnosis of alcohol dependence, determined via self-report. All participants provided informed consent before completing the study, which was approved by the University of Liverpool's Research Ethics Committee.

**Materials**

**Questionnaires**

*The Timeline follow back (TLFB:(Sobell & Sobell, 1990))* was administered to measure retrospective alcohol consumption over the previous seven days in units (one UK unit = 8 g of alcohol). A visual guide providing the number of units in standard UK drinks was provided to assist participants in calculating their alcohol consumption. *The Alcohol Use Disorders Identification Test (AUDIT: (Saunders et al., 1993))* was also administered to measure hazardous drinking (study 1 α = .78; study 2 α = .78). Participants were asked when they last consumed alcohol (‘*When was the last time you drank alcohol?’* with the following options; more than one week ago, within the last week, in the last couple of days, yesterday, today, within the last couple of hours) (see (Jones & Field, 2015)). They were also asked about their motivation to reduce alcohol consumption (‘*On a scale of 0 (not at all) to 10 (extremely) how motivated are you to reduce your alcohol consumption?’*) and their current urge to drink alcohol (‘*What is your current craving for alcohol from 0 (no urge) to 10 (extreme urge)?’)* (or ‘*100 (extreme urge)?*’ in study 2).Lastly, participants were asked if they were distracted (‘*Were you distracted during the computer tasks?’* with the answers Yes or No). In both studies we included an attention check to ensure participants were paying attention as recommended for online research (Oppenheimer et al., 2009), by including a question (*‘If you are paying attention leave this question blank’*: with the answers No, Yes but not in the last year and Yes during the last year) in the middle of the AUDIT.

**Computer tasks**

***Modified Stop-signal task (SST: (Verbruggen et al., 2008)***

Participants completed a modified SST, which isolated proactive slowing and reactive control. On each trial, a letter (‘X’) or (‘O’) was displayed in the center of the screen (for up to 1200 ms). Participants were asked to respond as fast and as accurate as possible to these ‘go’ stimuli. There was an intertrial interval of 500 ms. They were asked to press the left (‘D’) key with the left index finger if an (‘X’) was displayed and the right (‘K’) key with the right index finger if an (‘O’) was displayed.An alcohol-related image (e.g. a scene in a bar) appeared in the background on each trial to induce cue-exposure, as inhibitory control should be a stronger predictor of alcohol consumption during exposure to alcohol cues (Jones and Field, 2017). There were 10 of these images that were approximately 230 mm x 130 mm in size. Participants first completed a practice block of 10 trials (not recorded).The main task then consisted of two blocks:

**No-signal block:** In this block participants were asked to respond to the letters (‘X’ or ‘O’) without interruption on 100% of trials (N=40). Participants were informed there would be no stop signals during this block.

**Signal block:** During this block, participants were asked to respond to the go-stimuli without interruption on 75% of trials (N=90). On the remaining 25% (N=30), two red lines “=” (Stop Signal) appeared superimposed over the go stimulus. Participants were informed to attempt to inhibit their response if they saw this. The stop-signal delay (SSD i.e. the delay between the presentation of the go stimulus and the stop signal) was adjusted on a trial-by-trial basis using a tracking procedure (Verbruggen & Logan, 2009a). The initial delay was 250 ms, if participants failed to inhibit the delay decreased by 50 ms making succeeding inhibition easier, if participants effectively inhibited then the delay increased by 50 ms making succeeding inhibition harder (minimum delay = 0, maximum delay = 1150 ms). Before starting the task, participants were also informed that they should respond as quickly as possible (i.e. not to wait for the stop-signal to appear) in line with standard SST instructions (Verbruggen et al., 2019). Reactive control was inferred from SSRTs in the stop-signal block.

***The self-ordered pointing task (SOPT:*** *(Petrides & Milner, 1982)*

Participants were shown a set of neutral images (e.g. couch, kettle) and asked to select one using the left hand mouse button. Following the selection of a picture, these were re-arranged into different positions. Participants were asked to try and avoid clicking the same picture more than once in a block and avoid clicking the same position in the array of images each time. Participants were first shown 6 images in a 2x3 array followed by 8-items in a 2x4 array, a 10-item block in a 2x5 array and finally a 12-item block in a 4x3 array. The number of trials in each block was in accordance with the number of images in the array. Participant’s scores were displayed at the end of the task informing them of the number of errors made in each block (i.e. clicking on the same image more than once) and the total number of errors. The total number of errors was used as a measure of WMC. Task schematics are presented on OSF [<https://osf.io/ucwj4/>].

**Procedure**

The study was completed using Inquisit Web 5.0 (Millisecond software). Participants were first presented with an information sheet and gave informed consent. Next, they completed the SST followed by the SOPT in a counterbalanced order. Participants then gave demographic information and completed the questionnaires. Lastly, participants were debriefed and thanked for participation. The session took approximately 10-15 minutes to complete, and participants could opt in to a prize draw for £50 in high street vouchers.

**Data reduction and analysis**

A composite measure of alcohol use was computed as our dependent variable. This was used as in previous research (see (Baines et al., 2016; Christiansen & Bloor, 2014; Fernie et al., 2013)) to capture a better picture of the general pattern of alcohol use rather than specific behaviours such as binge drinking. The overall measure of alcohol use consisted of the units consumed (measured by the TLFB), scores on the AUDIT and the frequency of heavy episodic drinking days (6+ units in a single session for females, 8+ for males (Office for National Statistics, 2018)), z-scored and combined. We ran a principal component analyses which confirmed that total AUDIT scores, units consumed, and heavy days drinking loaded onto a single component (eigenvalue = 2.25; accounting for 77.48% of variance with all factor loadings ≥ .74).

For the SST, we calculated SSRT using the integration method as outlined in Verbruggen et al (2019). All non-signal reaction times were retained in the distribution including those with a choice error (pressed the wrong key) and those with premature responses (eg. < 200ms). Trials with go omissions (where a response was required but not provided; 2.6% of total trials) were replaced with the max RT from eligible trials. To obtain SSRT the mean Stop Signal Delay was subtracted from the *Nth* RT in the distribution. The *Nth* RT is calculated as the number of reaction times in the ranked distribution \* p(respond|signal). For example, with 90 go trials and p(respond|signal) = .40, the 36th fastest RT would be used. We aimed to remove any SSRTs which were negative (N = 0), in line with previous research (Congdon et al., 2012). A minority of participants (N=7) also had <25% or >75% inhibition errors and/or a median RT on failed inhibition trials > go trials. We removed these participants based on Verbruggen et al (2009). For proactive control we subtracted the median go RT on the signal blocks from the median go RT on the non-signal blocks. We used the same go RT distributions, with the omission errors replaced with the maximum eligible RT. Data from Stop Signal tasks is reported in more detail in online supplementary materials. One participant did not complete the SOPT. Three participants failed the attention check; however, removal of these did not significantly alter the interpretation of our results.

**Results**

**Sample characteristics (see table 1)**

One hundred participants provided complete data on demographic variables. There were no significant differences between males and females in AUDIT scores (*t* (98) = -.360, p= .720, *d=* -0.07), heavy drinking days (*t* (98) = 0.09, p= .929, *d*= 0.02) or TLFB scores (*t* (27.84) = 1.52, p= .140, *d=* 0.57). There were also no significant differences in craving scores (*t* (98) = 1.86, p= .067, *d*= 0.38) or motivation to reduce alcohol consumption (*t* (98) = 0.02, p= .983, *d*= 0.00).

***The associations between individual differences in reactive control, proactive slowing, working memory and overall alcohol use (see table 2).***

One participant did not provide a WMC score. We conducted a multiple regression analysis on N=89 cases to investigate if individual differences in SSRTs, craving, proactive slowing and working memory predicted individual differences in overall alcohol use. Variance inflation factors (VIF) ranged between 1.04 and 1.17 suggesting there were no issues with multi-collinearity. The overall model predicted approximately 23% of variance (Adjusted R²= .23; F (4, 84) = 7.69, p < .001). Increased craving for alcohol was associated with increased overall alcohol use (β= .359, p= .002, 95% CI .139 to .579). WMC (β= .180, p=.009, 95% CI .046 to .313) also significantly predicted overall alcohol use with increased errors on the SOPT being associated with higher alcohol use. However, neither SSRTs (β= .002, p= .402, 95% CI -.007 to .010) nor proactive slowing (β= .001, p= .461, 95% CI -.002 to .005) were significant predictors of alcohol use. Given the uneven gender distribution in this study we repeated the analyses with Gender as a predictor variable. In this analysis gender was not a significant predictor (β = .186, p = .767, 95% CI -1.058 – 1.429), but WMC and Craving remained significant predictors. When running the models separately for AUDIT scores and units consumed craving was a consistent predictor, whereas WMC predicted units consumed but not AUDIT scores (see supplementary materials).

**Exploratory Analyses**

We also aimed to investigate whether individual differences in WMC mediated the relationship between proactive slowing and overall alcohol use. However, although WMC significantly predicted overall alcohol use, there was no association between individual differences in WMC and proactive slowing (see table 2). Therefore, we did not meet the assumptions required to examine mediation.

**Interim discussion**

Study 1 demonstrates that increased craving and poorer working memory were associated with increased overall alcohol use in a sample of heavy drinkers. However, individual differences in proactive slowing nor reactive control did not significantly predict individual differences in overall alcohol use or working memory.

**Study 2**

In study 2, participants completed a SST in which they responded directly to alcohol-related words (rather than ambiguous letters). This manipulation aimed to increase alcohol cue-reactivity, particularly since the failed to find a relationship between the inhibitory control measures and alcohol use in the presence of the alcohol-cues used in study 1. Participants also completed a SOPT in which they had to remember alcohol-related stimuli (rather than neutral-related stimuli). Participants also completed a questionnaire assessing their alcohol sensitivity. We predicted that (i) individual differences in proactive slowing, reactive control, WMC and alcohol sensitivity would be associated with individual differences in overall alcohol use. We also predicted that (ii) individual differences in working memory would be associated with individual differences in proactive slowing and (iii) individual differences in alcohol sensitivity would predict the ability to implement proactive slowing and reactive control. Lastly, we hypothesised that (iv) WMC and AS would mediate the relationship between proactive slowing and alcohol use (we did not pre-register this hypothesis).

**Methods**

**Participants**

Heavy drinkers (N=116; 63 female), with a mean age of 22.01 (6.09) were recruited from the university and wider community using social media and advertisements. The number of participants was decided upon using a power calculation to find a medium effect size (F² = .15) at α = .05, and 90% power with five predictors (craving, reactive control, proactive slowing, working memory, alcohol sensitivity). The inclusion and exclusion criteria were identical to those described in study 1.

**Materials**

**Computer Tasks**

*Modified Stop-signal task (Verbruggen, Stevens, et al., 2014)*

Participants also completed a modified SST, which isolated proactive slowing and reactive control. On each trial participants were shown a white horizontal line (approximately 70 mm) in the middle of the screen for 500ms. An alcohol-related word (e.g. ‘beer’) then appeared either above or below the line. If the word appeared above the line, participants pressed one key (‘T’), if the word appeared below the line, participants pressed another key (‘V’) using the keyboard (these ‘keys’ appeared at the bottom on the screen on touch screen devices). A neutral word (e.g. ‘sponge’) also appeared simultaneously but participants were asked not to respond to this. These were no-signal trials. We chose the words based on those used in previous research which developed matched alcohol and control words, specifically (Cox et al., 2003). There were 10 generic alcohol-related words (beer, vodka, whiskey, bar, alcopops, stout, cocktails, spirits, alcohol, shorts (shorts is a generic term for spirits e.g. whisky, gin, vodka served in 25 ml, 35 ml, or 50 ml measures, often used in the UK)) and 10 generic neutral-related words (brush, duster, polish, squeegee, shammy, shampoo, sponge, flannel, bucket, hoover). On stop-signal trials, the white line turned red and participants were told to try and withhold their response when this occurred. The blocks, stop-signal probability, tracking procedure and calculations of proactive/reactive control were the same described in study 1.

*Modified Self-ordered pointing* task *(Petrides & Milner, 1982)*

This task was identical to the task described in study 1. However, instead of neutral images participants completed the task using alcohol-related images (e.g. pint of beer, glass of wine). Task schematics are presented on OSF [<https://osf.io/fybgv/>].

**Questionnaires**

The questionnaires administered were identical to that of study 1, except the TLFB was administered for fourteen days instead of seven to capture a better picture of individuals’ drinking patterns. Additionally, participants also completed *The Alcohol Sensitivity Questionnaire (ASQ:* (Fleming et al., 2016)) (α = .94). This included 15 items asking participants how many alcoholic drinks they must typically drink to experience alcohol-related effects. Specifically, 9 of these items are associated with lower doses of alcohol and stimulation (e.g. increasing talkativeness) and 6 are associated with heavier doses of alcohol and sedation (e.g. passing out). Participants were first asked whether or not they have experienced each alcohol-related effect and if the answer was YES, they were asked to estimate the minimum number of drinks required to experience the lower dose effects or the maximum number of drinks they could consume without experiencing the higher dose effects. The total score is the number of drinks stated with higher scores on this questionnaire indicating low sensitivity to alcohol.

**Procedure**

The procedure is identical to that described in study 1.

**Data Analysis**

# The data was handled using identical procedures to those in study 1. Note we pre-registered using the mean method to calculate SSRTs. However we retained the integration method for Study 2 to ensure consistency with Study 1 and ‘this version of the integration method produces the most reliable and least biased non-parametric SSRT estimates’ (Verbruggen et al, 2019).We computed the same overall measure of alcohol use. We ran a principal component analyses which confirmed that total AUDIT scores, units consumed (measured by the TLFB) and heavy drinking days loaded onto a single component (eigenvalue = 2.07; accounting for 68.97% of variance with factor loadings of .64 to .93). Additional details regarding this and the analysis of each hypothesis can be found in the pre-registration on Open Science Framework. However, for the ASQ we calculated a composite score as missing data has previously been shown to result in biased ASQ scores. Therefore, we used the standardized person mean imputation approach (Bailey & Bartholow, 2016; Lee et al., 2015). We first standardised ASQ scores by transforming these into z-scores and then calculated the mean score across all non-missing items. On average participants answered 11.69 (± 2.86) questions, which is a similar average reported in previous research (e.g. Mean = 11.40 (Bailey & Bartholow, 2016)). This procedure was not pre-registered, however it provides more robust data estimates. Twenty-four participants also had <25% or >75% inhibition errors and/or a median RT on failed inhibition trials > go trials. Three participants failed the attention check; however removal of these did not significantly affect results.

**Results**

**Sample characteristics (see table 1)**

One hundred and nine participants provided full demographic information. There was no significant difference between males and females in AUDIT scores (*t* (107) = 0.81, p = .423, d= 0.16) or heavy drinking days (*t* (107) = -0.49, p= .623, d= -0.09). There were also no significant differences in craving (*t* (107) = 0.29, p= .772, *d*= 0.06) or motivation to reduce alcohol consumption (*t* (91) = 0.14, p= .890, *d*= 0.03). However, males did consume significantly more units than females (*t* (82) = 3.05, p= .003, d = 0.67).

***The associations between individual differences in proactive slowing, reactive control, WMC, alcohol sensitivity and overall alcohol use (see table 3).***

We conducted a multiple regression analysis to investigate if individual differences in SSRTs, proactive slowing, working memory and alcohol sensitivity predicted individual differences in overall alcohol use. Variance inflation factors (VIF) ranged between 1.03 and 1.13 suggesting there were no issues with multi-collinearity. The overall model predicted approximately 7% of variance (Adjusted R²= .07; F(5, 70) = 2.189, p = .065), but failed to reach statistical significance. SSRT (β= -.007, p= .031, 95% CI -.014 to -.001) and WMC (β = -.208, p = .045, 95% CI - .412 - -.004) were negative predictors of alcohol use. Craving (β= .019, p= .176, 95% CI -.009 to .048), alcohol sensitivity (β= .395, p= .301, 95% CI -.362 to .1.152) and proactive control (β= -.002, p= .500, 95% CI -.007 to .004) were not significant predictors. When performing the models separately SSRT and WMC were negative predictors of units consumed, and craving was the only significant predictor of AUDIT scores (see supplementary materials).

**Exploratory Analyses**

We also aimed to investigate whether individual differences in WMC/AS mediated the relationship between proactive slowing and overall alcohol use. However, although poorer WMC predicted poorer proactive slowing, there was no relationship between overall alcohol use and WMC or AS (see above). AS was also not related to proactive slowing (see table 3; p= .540). Therefore, we did not meet the assumptions required to examine mediation.

**Pooled Analyses**

In an exploratory pooled analysis, we combined the data from both studies. We did this for a number of reasons. First, our hypotheses were consistent across both studies (individual differences in proactive slowing, reactive control and WMC would predict alcohol use). Second, doing so would increase our statistical power to detect smaller effect sizes of interest, whilst maintaining our key variables of interest. Finally, our tasks were similar enough across both studies to allow for this (e.g. both Stop Signal tasks included the same number of critical Stop Signal trials, and exposure to alcohol-related cues). One notable difference is that the SOPT included neutral cues (study 1) and alcohol cues (study 2). However, this was the only difference. Within each data set we standardised complete cases for craving, alcohol use, WMC, SSRT and proactive slowing and pooled the two complete data sets (Study 1 N = 89; Study 2 = 76: total N = 165). We ran a multiple regression with alcohol use as the outcome and craving, WMC, SSRT and Proactive slowing as predictors. The model predicted 9% of variance (Adjusted R2 = .09; (F(4, 160) = 5.17, p =. 001). Craving was a significant predictor of alcohol use (β=.298, p < .001, 95% CI .150 -. 446). However, WMC (β = .085, p = .258, 95% CI -.063 - .234), SSRT (β = -.021, p =. 772, 95% CI -.167 - .124) and proactive slowing (β = .059, p = .426, 95%CI -.087 - .205) were not significant predictors.

**Discussion**

The current studies investigated if individual differences in reactive control and proactive slowing were associated with individual differences in overall alcohol use in heavy drinkers. We also aimed to investigate if WMC and AS mediated the relationship between the proactive slowing and overall alcohol use. However, contrary to our there were no robust associations between alcohol use and reactive control or proactive stopping. Although poorer working memory was associated with increased alcohol use in study 1, it was unrelated to the ability to implement proactive slowing and the opposite relationship was observed in study 2. Individual differences in alcohol sensitivity were also unrelated to alcohol use or proactive slowing in study 2. Therefore, there was no evidence that WMC or AS mediated the relationship between proactive slowing and alcohol use.

These findings support models (e.g. (Verbruggen, McLaren, et al., 2014)) which suggest that investigating reactive inhibition only is of limited theoretical benefits. We were able to isolate proactive slowing and reactive control in both studies. However, we failed to replicate studies that have demonstrated a relationship between reactive control and alcohol use (e.g. (Christiansen, et al., 2012; Colder & O'Connor, 2002; Paz, et al., 2016)), thus finding no empirical support for models of addiction which posit inhibitory control as a candidate mechanism of action (e.g. (de Wit, 2009; Fillmore, 2003; Goldstein & Volkow, 2002)). It is still plausible that a relationship exists between proactive slowing and alcohol use, however, it is also possible that the relationship between inhibitory control and alcohol use has been over-emphasised or is influenced by publication bias and small study effects. There are numerous studies which have reported null findings (e.g. (Czapla et al., 2015; Fernie et al., 2010; Nederkoorn et al., 2009)), or even the reverse association (improved inhibition is associated with alcohol use: Bø & Landrø (2017)). Furthermore, an updated meta-analyses by Smith et al (Smith & Mattick, 2018) suggested that inhibitory deficits are not associated with heavy drinking. Continuing well-powered and pre-registered studies should begin to correct any biases in the literature and elucidate the true nature of the relationship.

We demonstrated some support for research that has shown poorer WMC is associated with increased alcohol use in study 1 (e.g. (Mahedy, et al., 2018; Peeters et al., 2015; Thush, et al., 2008)). However, this relationship was reversed in study 2, which showed a negative association between WMC and alcohol use. This finding should be interpreted with caution though, as the overall regression model failed to reach statistical significance. We also found no support for the relationship between WMC and the ability to implement proactive slowing (e.g. (Richmond, et al., 2015; Wiemers & Redick, 2018)). Furthermore, we failed to replicate studies that have demonstrated that alcohol sensitivity is associated with increased risk for heavy drinking (Fleming & Bartholow, 2014) or associated with the ability to implement proactive slowing (Bailey & Bartholow, 2016).

These studies have limitations. We used a cross-sectional design and therefore we are unable to investigate these relationships over time. Furthermore, in study 1 there was an over-representation of females, thus future research should aim to recruit a more representative sample. We did not include a control measure of inhibitory control (in the absence of alcohol-related cues) and as such it is impossible to determine whether performance variability comes from alcohol-cue exposure, participants general inhibitory performance or a combination of both. Future research should attempt to disentangle this by providing a ‘neutral’ inhibitory control task (Field and Jones, 2017). Similarly, future studies should include a control group (e.g. light drinkers) to examine whether inhibitory control can differentiate these populations (Paz et al, 2016; Christiansen et al, 2012). Thirty-two participants study 1 and 34 in study 2 stated that they were distracted during the computer tasks. The proportion of reported distractions are similar to recent ecological momentary assessment studies examining SST and alcohol consumption in the real word (Jones, Tiplady, et al., 2018). However, completing these tasks online in the participant’s natural environment rather than in the laboratory does increase the ecological validity of the study, as in the real world inhibition occurs in ‘noisy’ surroundings (Verbruggen, Stevens, et al., 2014). Furthermore, only 3 participants in both studies responded incorrectly to the attention measure in the AUDIT and removal of these did not significantly affect results. Finally, removal of individuals who failed to complete the SSRT appropriately reduced our statistical power in each individual study, however our pooled analyses did not provide any evidence for the predictive associations between inhibitory control, working memory and alcohol use.

In conclusion, we have demonstrated no evidence that inhibitory control processes (reactive and proactive) are associated with alcohol use. Furthermore, we demonstrated no convincing evidence for our proposed mediators of WMC or alcohol sensitivity. Given the increasing number of null findings, it is possible the role of inhibitory control in alcohol use has been overemphasised.

Conflicts:

The authors report no conflicts of interest.

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**Tables**

Table 1: Descriptive statistics for AUDIT scores, TLFB scores, heavy drinking days, craving scores and motivation to reduce alcohol consumption, split by gender in Study 1 and Study 2 (values are mean (SD)).

Study 1 Study 2

Males (n=22) Females (n=78) Sample (N=100) Males (n=49) Females (n=60) Sample (N=109)

AUDIT 10.00 (5.59) 10.50 (5.81) 10.39 (5.73) 13.51 (6.20) 12.55 (6.20) 12.98 (6.19)

TLFB 24.77 (19.59) 17.95 (14.53) 19.45 (15.93) 43.73 (28.51) 29.13 (19.60) 35.70 (24.99)

Heavy drinking days 1.41 (1.30) 1.38 (1.10) 1.39 (1.14) 2.18 (1.81) 2.35 (1.71) 2.28 (1.75)

Craving 2.86 (2.98) 1.76 (2.31) 2.00 (2.50) 17.49 (22.54) 16.25 (21.89) 16.81 (22.09)

Motivation 2.59 (2.91) 2.58 (2.62) 2.58 (2.67) 2.59 (2.41) 2.53 (1.91) 2.56 (2.14)

AUDIT=Total scores on the AUDIT. TLFB = Total units reported in the TLFB. Heavy drinking days = occurrences of heavy episodic drinking days in the 7-day TLFB (Study 1) and 14-day TLFB (study 2) (6+ units in a single session for females, 8+ for males; (Office for National Statistics, 2018)). Craving = 0 (no urge) to 10 (extreme urge) or 100 (extreme urge; study 2). Motivation to reduce alcohol consumption = 0 (not at all) to 10 (extremely).

Table 2: Descriptive statistics and Pearson’s correlations for overall alcohol use, craving, reactive control, proactive slowing and working memory in Study 1.

Mean (SD) 2 3 4 5

Overall alcohol use 0.11 (2.60) .42\*\* .17 .15 .40\*\*

Craving 1.88 (2.35) - .20 .14 .35\*\*

SSRTs 255.50 (57.98) - - .18 .23\*

Proactive slowing 147.92 (148.61) - - - .08

Working memory 6.03 (3.88) - - - -

Overall alcohol use = units consumed (measured by the TLFB), scores on the AUDIT and the frequency of heavy episodic drinking days (6+ units in a single session for females, 8+ for males; (Office for National Statistics, 2018)), z-scored and combined. Craving = 0 (no urge for alcohol) to 10 (extreme urge for alcohol). SSRTS = reactive control. Higher scores = worse reactive control. Higher Proactive slowing = better proactive slowing. Working memory = errors on SOPT. Higher scores = worse working memory. \*\*p<.01, \*p<.05

Table 3: Descriptive statistics and Pearson’s correlations for overall alcohol use, craving, reactive control, proactive slowing, working memory errors and alcohol sensitivity, in Study 2.

Mean (SD) 2 3 4 5 6

Overall alcohol use -0.05 (2.46) .17 -.23\* .05 -.19 .08

Craving 13.41 (18.08) - -.16 .07 -.06 -.05

SSRTs 295.56 (89.00) - - -.24\* -.15 .04

Proactive slowing 55.96 (107.70) - - - -.18 -.11

Working memory errors 4.73 (3.01) - - - - .08

Alcohol sensitivity 0.02 (0.71) - - - - -

Overall alcohol use = units consumed (measured by the TLFB), scores on the AUDIT and the frequency of heavy episodic drinking days (6+ units in a single session for females, 8+ for males; (Office for National Statistics, 2018)) ,z-scored and combined. Craving = 0 (no urge for alcohol) to 100 (extreme urge for alcohol). SSRTS = reactive control. Higher scores = worse reactive control. Higher Proactive slowing = better proactive slowing. Working memory = errors on SOPT. Higher scores = worse working memory. Alcohol sensitivity = composite measure of Alcohol sensitivity. Higher scores = lower sensitivity to alcohol. \* p<.01, \*\*p<.05.

**Supplementary Information**

**The associations between proactive slowing, working memory, alcohol sensitivity and alcohol use.**

Laura Baines1,2🖃 Andrew Jones1,2

**Study 1:**

**Study 1**

**AUDIT scores**

We conducted a multiple regression analysis to investigate if individual differences in SSRTs, craving, proactive slowing and working memory predicted individual differences in AUDIT scores. Variance inflation factors (VIF) ranged between 1.05 and 1.17 suggesting there were no issues with multi-collinearity. The overall model predicted approximately 17% of variance (R²= .17; F (4, 84) = 5.48, p = .001). Increased craving for alcohol was associated with increased AUDIT scores (β= .965, p< .001, 95% CI .453 to 1.477). WMC (β= .214, p=.177, 95% CI -.098 to .525), SSRTs (β= -.007, p= .485, 95% CI -.027 to .013) and proactive slowing (β= .001, p= .833, 95% CI -.007 to .008) were not significant predictors of AUDIT scores.

**Units consumed.**

We conducted a multiple regression to investigate if individual differences in SSRTs, craving, proactive slowing and working memory predicted individual differences in units consumed. Variance inflation factors (VIF) ranged between 1.05 and 1.17 suggesting there were no issues with multi-collinearity. The overall model predicted approximately 25% of variance (R²= .25; F (4, 84) = 8.16, p = .001). Increased craving (β= 1.675, p= .013, 95% CI .357 to 2.994) for alcohol and WMC (β= 1.305, p=.002, 95% CI .502 to 2.107) were associated with increased units consumed. SSRTs (β= .024, p= .926, 95% CI -.028 to .076) and proactive slowing (β= .010, p= .313, 95% CI -.010 to .030) were not significant predictors of AUDIT scores.

**Study 2:**

**AUDIT scores**

We conducted a multiple regression analysis to investigate if individual differences in SSRTs, craving, proactive slowing and working memory predicted individual differences in AUDIT scores. Variance inflation factors (VIF) ranged between 1.03 and 1.13 suggesting there were no issues with multi-collinearity. The overall model predicted approximately 6% of variance (Adjusted R²= .06; F(5, 70) = 2.014, p = .087) and was not statistically significant. Increased craving for alcohol was associated with increased AUDIT scores (β= .091, p= .012, 95% CI .020 to .161). WMC (β= -.246, p = .334, 95% CI -.750 to .258), SSRTs (β= -.010, p= .251, 95% CI -.026 to .007), proactive slowing (β= -.008, p= .234, 95% CI -.022 to .005), and alcohol sensitivity (β= .289, p= .759, 95% CI -1.581 to 2.160) were not significant predictors of AUDIT scores.

**Units consumed**

We conducted a multiple regression analysis to investigate if individual differences in SSRTs, craving, proactive slowing and working memory predicted individual differences in AUDIT scores. Variance inflation factors (VIF) ranged between 1.03 and 1.13 suggesting there were no issues with multi-collinearity. The overall model predicted approximately 9% of variance (Adjusted R²= .09; F (5, 70) = 2.578, p = .034). SSRT (β= -.093, p= .008, 95% CI -.162 to -.025) and WMC (β= -2.430, p= .023, 95% CI -4.508 to .369) were negative predictors of units consumed. Proactive control (β= -.012, p= .672, 95% CI -.069 to .045), alcohol sensitivity (β= 4.824, p= .216, 95% CI -2.880 to .12.53) and craving (β= .087, p= .552, 95% CI -.202 to .376) were not significant predictors.

**Results**

*Distraction measure*

In both studies we asked participants whether they were distracted during the computer tasks. In study 1, 32 participants reported that they were distracted. In study 2, 34 participants stated that they were distracted during the computer tasks and 11 failed to answer the question. We did not remove participants based on this as it would have substantially reduced our statistical power.

**Supplementary Table 1: Outcome Variables from the Stop Signal tasks (standard deviations in brackets).**

**Study 1 Study 2**

Go RT No signal 425.1167 (69.36) 644.25 (117.82)

Go RT Signal 573.04 (155.08) 699.74 (135.06)

Stop RT Signal 486.18 (114.95) 610.33 (97.24)

Stop Signal Delay (mean) 297.19 (118.23) 370.00 (118.47)

Stop Errors (mean) 14.00 (2.02) 13.32 (2.50)

*Legend: Go RT No signal, Go RT Signal and Stop RT Signal are expressed as median reaction time (milliseconds - ms) on each. Median reaction times include any trials in which a go omission was replaced.* *Stop Signal Delay is the mean (ms). Stop Errors is the mean number of stop errors (min = 0, max = 30). For both Go RT No signal and Go RT signal the full distribution of reaction times were used (N = 40, N = 90, respectively). For Stop RT Signal, this was based on the number of failed stop trials for that individual (range: 8 - 22). For Stop Signal delay this was based on the delay from all 30 Stop trials in the task.*