SUBSTANCE USE & MISUSE
https://doi.org/10.1080/10826084.2025.2553304

Taylor & Francis
Taylor & Francis Group

ORIGINAL ARTICLE

An Ecological Momentary Assessment Study of Fluctuations in Inhibitory Control and Its Predictive Validity of Alcohol Use

Sam Burton[a,b] and Andrew Jones[a]

[a]School of Psychology, Liverpool John Moores University, Liverpool, United Kingdom; [b]Department of Women's & Children's Health, King's College London, London, United Kingdom

ABSTRACT

Introduction: Inhibitory control is associated with reports of alcohol use in cross-sectional and lab-based research. In the current study we investigated inhibitory control using an ecological momentary assessment paradigm to investigate 'in-the-moment' relationships with alcohol consumption and other factors (e.g., location, craving, emotions) in the real-world. We hypothesized that fluctuations in inhibitory control throughout the day would predict alcohol consumption.

Materials and methods: Heavy drinkers ($N=54$: mean age = 24.30, 47 females) were asked to complete a battery of questions and a stop signal task four times per day, at random intervals between 10am and 10 pm for one week. Participants were asked to record their location, craving, emotions and alcohol consumption at each assessment. Inhibitory control was assessed using stop signal task with personalized alcohol- and generic neutral-related cues.

Results: Multilevel modeling demonstrated that neutral Stop Signal Reaction Times (OR= 1.05: 95% CI 1.02, 1.08) and frequency of craving (OR = 1.65; 95% CI 1.48, 1.84) predicted subsequent alcohol use occasions. Intensity (B=-0.036; 95% CI −0.059, −0.013) and frequency ($B=0.026$; 95% CI 0.002, 0.050) of craving significantly predicted variance in alcohol consumption.

Discussion: Findings do not provide consistent evidence that fluctuations alone in inhibitory control predicts alcohol consumption. Future research should examine the interaction between inhibitory control and craving in the real-world, to better our understanding of the complex relationship.

KEYWORDS

Inhibitory control; alcohol use; ecological momentary assessment; disinhibition; substance use

Introduction

Inhibitory control, otherwise known as response inhibition, is the (in)ability to stop, change or delay inappropriate behavior under certain circumstances (Logan et al., 1984). This behavior is an underlying component of both impulsivity and executive functioning (Bickel et al., 2012), while also being encompassed under the broader construct of self-control (Fujita, 2011). The ability to inhibit behavioral responses has been operationalized in controlled environments using experimental tasks such as the 'stop signal; task (Frederick Verbruggen & Logan, 2008). In these tasks participants are required to execute a speeded motor response on the majority of trials without interruption (e.g., 75%), reinforcing a dominant response. On a minority of trials, they are required to withhold the speeded motor response following a 'stop signal'.

Deficits in inhibitory control are observed in individuals suffering from alcohol dependence, as well as those who are non-dependent but who drink 'heavily' (Christiansen et al., 2012; Houston et al., 2014; Smith et al., 2014), compared to 'light' drinking controls. Furthermore, poorer inhibitory control is associated with *ad-libitum* alcohol consumption in the laboratory ((commonly assessed using a 'bogus taste test') (Jones et al., 2013). This paradigm demonstrates good construct validity (Jones et al., 2016), and is thought to be representative of real world behavior (Leeman et al., 2010, 2013; Trautmann et al., 2024), in the laboratory (Bujarski & Ray, 2016; Trautmann et al., 2024). However, it is not clear whether such deficits are a cause or consequence of substance misuse (De Wit, 2009; López-Caneda et al., 2014; Verdejo-García et al., 2008). Longitudinally, inhibitory control predicts relapse following treatment (Rupp et al., 2016), the transition from heavy drinking to dependence (Rubio et al., 2008), along with the initiation and escalation of alcohol use in adolescents (Fernie et al., 2013; Nigg et al., 2006).

Much of the cross-sectional research into inhibitory control implies that it is a stable trait within individuals, however it is suggested that the ability to inhibit behavior can fluctuate *within* individuals over time (Jones et al., 2013), which makes it more difficult for individuals to engage their inhibitory control in response to temptation. Jones et al. (Jones et al., 2013) reviewed the evidence and observed that transient changes in inhibitory control were evident in response to; environmental influences (De Wit, 2009; Jones et al., 2013), stress (Roos et al., 2017), reward/extrinsic motivation (Burton et al., 2021) and exposure to alcohol-related cues and contexts which are thought to

CONTACT Sam Burton S.P.Burton@ljmu.ac.uk School of Psychology, Tom Reilly Building, Liverpool John Moores University; Byrom Street, Liverpool, L3 3AF.
Supplemental data for this article can be accessed online at https://doi.org/10.1080/10826084.2025.2553304.

increase craving (Czapla et al., 2016; Jones & Field, 2015). Furthermore, laboratory-based studies have demonstrated that fluctuations in inhibitory control, a result of experimental manipulations, may influence subsequent alcohol consumption suggesting a causal relationship (Field & Jones, 2017; Jones et al., 2011; Jones et al., 2011).

The evidence base to-date is mostly from laboratory-based studies. However, these studies are limited by retrospective recall of alcohol-use, demand characteristics and a suppression of craving/consumption behaviors in controlled settings (Jenkins et al., 2009; Monk et al., 2015). Substance use is contextually driven and time-sensitive, and in order to reliably examine the link between inhibitory control and alcohol use, assessments must be made repeatedly in congruent contexts and at strategically selected moments (Cathy Lau-Barraco & Linden, 2014). As such, Ecological Momentary Assessment (EMA) methods are well enabled to examine the precursors of substance use behaviors in real-world environments (Shiffman, 2009; Shiffman et al., 2008). EMA is the repeated sampling of participants' subjective states and behavior in naturalistic settings. EMA studies allow for the examination of temporal relationship between substance-related cues, fluctuations in craving, self-control and substance use (Fatseas et al., 2015; Remmerswaal et al., 2019; Serre et al., 2015). EMA allows for daily assessments of alcohol consumption providing more reliable estimates than retrospective diary measures (Monk et al., 2015). Such methods have been used to investigate cognitive precursors, such as attentional bias and inhibitory control, in substance use (Jones et al., 2018; Marhe et al., 2013; Waters et al., 2012).

To date, only one study has investigated the relationship between day-to-day fluctuations in inhibitory control and whether it can predict alcohol consumption in heavy drinkers (Jones et al., 2018). Jones et al. measured inhibitory control using a stop signal task twice per day, between 10am and 6pm. Their findings demonstrated that average daily inhibitory control did not predict daily alcohol use, however fluctuations over the course of the day did, suggesting fluctuations may be a risk factor for heavy drinking. However, these findings were exploratory, and focused on only two sessions administered per day, potentially not capturing the dynamic nature of inhibitory control.

The present study aimed to replicate and extend the findings of Jones et al. (2018) by administering four daily assessments allowing us to reliably allow us to investigate cue-specific inhibitory control (alcohol vs neutral, given alcohol cues are known to impair control), mood and craving in relation to alcohol consumption. By including more assessments our design will allow for a more detailed analysis of fluctuation in inhibitory control in relation to alcohol use, by including neutral and alcohol-specific estimates of inhibitory control (Shiffman, 2014; Shiffman et al., 2008). It also provides a clearer pattern of alcohol use throughout the day, directly building on limitations highlighted in the original paper of Jones et al. (2018). We also extended the daily testing period, from 6pm in Jones et al. to 10pm here in an attempt to capture more proximal associations between inhibitory control and drinking episodes which tend to occur later in the evenings (peaking between 6—8pm: (Liang & Chikritzhs, 2015). Further to this, baseline sessions of the Stop Signal Task (SST) were used to cross-validate findings from the mobile SST given to participants. Baseline taste-test results were compared to actual drinking behavior in EMA sessions to examine the predictive reliability of the taste test as an analogous measure of drinking behavior, to inform an unrelated project.

We hypothesized that i) daily fluctuations in inhibitory control would predict subsequent alcohol consumption, specifically decreased in inhibitory control (reduced SSRTs) will lead to increased alcohol consumption, ii) Alcohol consumed on an ad-libitum taste test will predict alcohol consumption in real world environment, iii) Inhibitory control performance will fluctuate as a result of location of the testing location and cue exposure, e.g., if participants are in an environment with alcohol-cues present inhibitory control will decrease. This study was pre-registered on the Open Science Framework (https://osf.io/q2xky).

Materials and method

Participants

We recruited 57 heavy drinking individuals into the study. Three participants withdrew from the study due as they were unable to commit to the testing schedule, leaving 54 (47 females, mean 24.30 ± 7.67 years old) in the final sample. Participants were recruited from the local community through the use of adverts both physically and *via* social media, this comprised both students and members of the general population. Heavy drinking was defined as regularly drinking in excess of UK government guidelines, which is 14+ UK units per week (Department of Health, 2016). To be eligible, participants had to be 18+ and own an iPhone (due to the experiment software only compatible with iOS operating systems). Participants were excluded if they self-reported a current or previous diagnosis of a substance use, psychiatric or neurological disorder. Our a-priori sample size estimation was 55 participants. This was based on simulation research by Maas and Hox (2005) suggesting that sample size >50 participants (as level 2 units) leads to unbiased standard errors in multilevel models. We aimed for 55 to account for 10% attrition. Upon completion of the full study participants were reimbursed with £20. The study protocol was approved by the local research ethics committee (approval number: 3854).

Baseline measures

Timeline follow-back (Sobell & Sobell, 1992)

Participants completed a two-week retrospective diary of all alcoholic beverages they consumed *via* the alcohol Timeline Follow Back (TLFB) (Sobell & Sobell, 1992). The TLFB is regularly used to assess the frequency and quantity of alcohol consumption. Participants were asked to record the number of units they consumed on a day-to-day basis for the previous 14 days, which was aggregated. A UK unit guide was provided for standard measurements of a variety of drinks, e.g., a small glass of wine or bottle of beer. Total units consumed during the previous 14 days and binge drinking frequency were the outcome measures.

Alcohol use Disorders Identification task (Saunders et al. 1993)

The Alcohol Use Disorders Identification Task (AUDIT) was used to assess hazardous drinking. The AUDIT is a ten-item scale, with each item given a score from 0 to 4, with a maximum score of 40. The internal consistency of the AUDIT in this sample was $\alpha = 0.75$.

Barratt impulsivity scale (BIS) (Patton et al., 1995)

The BIS measures self-reported trait impulsivity. The scale is comprised of 30 questions, scored from 1 to 4 'rarely', 'occasionally', 'often' and 'always'. The total score is made by summing the three subscales (max = 120); Attention, Non-Planning and Motor Impulsiveness. The internal consistency of the BIS total score was $\alpha = 0.57$, Attention subscale $\alpha = 0.44$, Non-Planning subscale $\alpha = 0.43$, Motor Impulsiveness subscale $\alpha = 0.54$.

Temptation and restraint inventory (TRI) (Collins & Lapp, 1992)

The TRI measures drinking restraint, using a 15-item scale, loading onto five subscales; Govern (difficulty controlling alcohol consumption), Restrict (attempts to limit drinking), Emotion (negative affect as a reason to drink), Cognitive Emotion Preoccupation (CEP; thoughts about drinking) and Cognitive Behavioral Control (CBC; plans to reduce drinking/worry about controlling drinking). Items are rated on a 9-point Likert scale from 1 'never' to 9 'always'. The internal consistency for the overall scale was $\alpha = 0.78$, CEP subscale $\alpha = 0.74$, CBC subscale $\alpha = 0.73$.

Brief self-control scale (SCS) (Tangney et al. 2018)

The self-control scale assesses an individual's general trait level self-control. The scale is scored using a 5-point Likert scale, from 1 'not at all' to 5 'very much', on 13 items, loading onto four factors; self-discipline, healthy habits, impulsivity and self-regulation. The internal consistency for the overall scale was $\alpha = 0.61$.

Brief desire for alcohol questionnaire (DAQ) (love, James, & Willner, 1998)

The DAQ allows the assessment of moment-to-moment craving for alcohol. The abbreviated DAQ is scored on a 7-point Likert scale, from 1'strongly disagree' to 7'strongly agree'. It is based upon three subscales that assess, intention to drink, negative reinforcement and positive reinforcement, and ability to control drinking (Kramer et al., 2010).

Ad-libitum alcohol taste test

In the ad-libitum taste test participants are given access to a set amount of alcohol and asked to rate it on multiple perceptual factors (Field & Eastwood, 2005; Jones et al., 2011), providing an unobtrusive measure of alcohol consumption (the rating scales are of secondary importance). In this study, participants were presented with two alcoholic beverages and one soft drink. Participants were presented with 3 units of alcohol, exact measure in ml varied across drink choice due to strength differences. Participants were also given a soft drink (cola) that was the same amount in ml as the alcoholic drinks. The dependent variable was the percentage of alcohol consumed. Additionally, a 100 mm Visual Analogue Scale (VAS) was administered to examine participants' thirst prior to the taste test ('*How thirsty are you right now on a scale of 0 (not thirsty at all) – 100 (extremely thirsty)*)'.

EMA measures

Stop signal task

The stop signal task (Frederick Verbruggen & Logan, 2008) was programmed in Inquisit 5, based on Jones et al. (2018), on the participants phone at all testing points. The screen background was white with a black fixation cross. On each trial, following the presentation of the fixation cross for 250 ms an alcohol- or neutral- stimulus was presented in the center of the screen, rotated 45 degrees to the right or left. On go trials, participants had to respond to the orientation of the image by pressing a left button on the touch screen if the image was rotated to the left and a right button if the image was rotated to the right. The categorization of this response was uninterrupted on 75% of trials, and these are referred to as 'go trials'. On the remaining 25% of trials a stop signal (a red '=' sign) was superimposed over the go stimuli, after a variable delay (stop signal delay) after the onset of go stimuli. Participants were instructed to withhold their categorization response on trials a stop signal was presented.

A dynamic tracking algorithm was used to set stop signal delays (Verbruggen & Logan, 2008). In a given session, the first delay was set at 250 ms following onset of the go stimulus. If participants were able to successfully inhibit, the delay increased by 50 ms on the subsequent stop trial and decreased on unsuccessful inhibition (min delay = 50 ms, max delay = 1000ms). Participants were presented with a total of 192 trials, with 48 stop trials and 144 go trials.

Stimuli

Eight personalized alcohol-related pictures for four different drink categories (beer, white wine, rose wine and cider), and eight neutral images (e.g., plug socket, shells, books) were used in the Stop Signal task, across all conditions. All images were presented in the same size and brightness, in an attempt to match the perceptual characteristics. Images were rotated 45 degrees to the right or left, as part of the classification component of the SST (see Jones & Field, 2015). Alcohol stimuli was personalized for the participant's preferred drink (e.g., if the individual preferred cider to beer, they were only shown cider-related images) (Christiansen & Bloor, 2014) to increase the strength of the manipulation and internal reliability (Christiansen et al., 2015).

EMA self-report measures

At the beginning of each assessment participants were asked "*How [energetic/sad/drowsy/happy] do you feel right now?*" with similar questions for craving "*How strong is your craving for alcohol right now?*" and were asked to respond on a 0—100 visual analogue scale (0 = not at all, 100 = extremely). Smoking behavior was assessed, "*Have you had a cigarette since your last assessment?*" since their last assessment, *via* a 'yes' or 'no' answer. Following completion of the stop signal task, participants were asked to report their location for the assessment into one of six categories ('work', 'home', 'traveling', 'bar', 'restaurant' or 'other'). To control for distractors, participants were asked to record whether they completed the session 'alone' or 'in the presence of others', and if they were interrupted and if so, how many times. Finally, participants were asked if they had consumed alcohol *"How much alcohol have you consumed since your last assessment?"*, in which the number of units consumed was reported, and to provide a breathalyzer reading using a portable breathalyzer supplied to them by the researcher.

Procedure

Participants were prescreened *via* an online questionnaire. Eligible participants were invited to take part in the baseline session in the Human Psychopharmacology Laboratories at the University of Liverpool. Upon arrival they provided informed consent. They then completed the battery of questionnaires for the baseline session (AUDIT, BIS, TRI, SCS, DAQ). Following completion, the EMA app (programmed on Inquisit 5) was loaded onto the participant's phone. Alcohol images were personalized to the individual. Participants then completed their first full session using the app and were asked if they had any questions. Finally, they completed the ad-libitum alcohol consumption measure. Before leaving the laboratory, participants were given a printed guide of units, instructions and contact details for the researcher should they incur any problems. They were also provided with a portable breathalyzer in an attempt to examine the feasibility of using these in future EMA studies (BAC data not reported here, however any positive readings were removed as alcohol intoxication impairs inhibitory control (Field et al., 2010; Weafer & Fillmore, 2016).

During the EMA phase participants were randomly prompted (Random Assessment: RA) four times per day, between the hours of 10am and 10pm, in 3-h time windows with a final breathalyzer session at 10pm. A testing session would take a maximum of 15min, if the participant did not respond at all once they had started, on average most participants took 8min. Notifications were sent *via* email to participants to complete a session at the next available opportunity, completing all self-report measures from the EMA session and the stop-signal task, a follow-up prompt was sent within 15min of the first prompt to remind the participant. Participants took part in the study for 7 full days, beginning the day after the baseline session. Upon completion, they were asked to return the breathalyzer and attend a debrief session. Any data that had been stored on the participants phone from EMA sessions *via* the app was uploaded to the database.

Data reduction and analysis

To compute Stop Signal Reaction Times (SSRT), SPSS 25 was used, and for subsequent analysis R was used with the 'dplyr' and 'Lme4' packages. We computed SSRTs separately for each image-type using the integration method (Verbruggen et al., 2013; Verbruggen & Logan, 2009), with replacement of incorrect/omitted go errors with the maximum reaction time from the distribution of correct reaction times on that trial type. We also removed any negative values ($N = 5$) for both alcohol and neutral SSRTs, which indicate strategic responding (Verbruggen et al., 2019).

Multilevel modeling is the most appropriate method for analysis of repeated measures using the link function of logit for Models A and B, and linear for Model C, due to the nature of nested data it takes into account the dependence between observations as a result of data clustering (e.g., stop signal performance may fluctuate over time, but should be highly correlated with other time points). Use of multilevel modeling allows for unequal number of data points across participants (resulting from missing data) (Hayes, 2006; Quené & Van den Bergh, 2004). A mean centered approach (Paccagnella, 2006) was adopted to assess an individual's fluctuations in SSRTs with respect to their own mean SSRT, rather than that of the groups, within the models. Improved model fit was assessed *via* reductions in AIC/BIC values (Burnham & Anderson, 2004) for binary outcomes (with reduction in AIC/BIC > 10 indicative of a better fitting model), and reductions in Log-likihood statistics (Leckie, 2019) for continuous outcomes. Mean centered time variant variables were scaled (divided by 10) to improve parameter interpretation (Statistic Consulting Center, n.d.). We used random intercepts for assessment day and participant, with SSRT values, Units, Location, Craving and Mood variables as fixed effects. Intraclass-correlations for each continous time varying variable were calculated using the 'performance' package, and marginal and conditional R2 values were reported for each model, where marginal R2 represents the variance explained by the fixed effects and conditional R2 represents total variance explained (fixed and random effects: Nakagawa & Schielzeth, 2013).

In a deviation from the pre-registered outcome, we did not use alcohol consumption in units as our primary outcome, but rather recoded the variable to a drinking occasion (vs no drinking occasion). This was due to the large proportion of sessions in which alcohol consumption was reported as zero (80.15%), skewing the distribution of quantity of alcohol units consumed. Location was initially coded as; home, work, travel, bar or restaurant, or other. Eight-hundred and sixteen of the data points were classified as being at home, with 484 being split across the remaining categories. For the purpose of the analysis, location was coded as either at home (vs not at home), due to some locations not having adequate data points for analysis. In supplementary models we included Gender as a fixed effect, however this did not influence any of the findings (changes in significance of any individual predictors) so we do not include it in reported models.

Results

Participant characteristics

Participants (Table 1) had a mean age of 24.30 (SD = 7.67). On average participants consumed 42.83 units of alcohol in a two-week period prior to the baseline session, exceeding the 'heavy drinking' threshold (~28 units over the two-week period), with an average of 3+ binge sessions over the same period. Thirty (52.63%) of the participants were classed as hazardous drinkers and 15 (26.32%) were indicative of alcohol dependence based upon their AUDIT score.

Compliance and practice effects

Participants completed 1298 Random Assessments (RA; of a possible 1512: 85.85%, none were removed from the analysis) and 326 Breathalyzer Assessments (BA; of a possible 378). Participants reported being interrupted during 401 RAs (30.89%) and completed 610 in the presence of others (47.00%). On 149 RAs participants completed the session while reporting a positive breathalyzer reading (11.03%). RAs were coded as confounded if there was a report of interruption or a positive breathalyzer reading, sensitivity analysis was carried out for the main analysis reported below, in which confounded RAs were removed. 11.04% (149) sessions were removed due to a positive alcohol breathalyzer reading. Further to sessions confounded by alcohol, those with interruptions and in which the participant had smoked were removed, as part of a sensitivity analysis due to previous lab work showing it may influence attention and inhibitory control (Wignall & de Wit, 2011), in total 37.75% of sessions (490) were removed. Results did not significantly differ when confounded sessions were removed and the main analyses re-run.

To examine the possibility of practice effects on inhibitory control we used assessment day (1—7) as a predictor of both Alcohol ($B = 0.91$ [95% CI: −1.20 to 3.01], $p = 0.398$) and Neutral SSRTS ($B = 0.96$ [95% CI: −1.09 to 3.01], $p = 0.358$), however there were no associations.

Hypothesis 1 and 2: Multilevel model predicting alcohol consumption, from laboratory alcohol use and EMA measures.

A two-level model (assessment > participant) was a significantly better fit than a single level model ($\chi^2(1)=6.03$, $p < 0.001$). A three-level model (assessment > day > participant) was a significantly better fit than a two level model ($\chi^2(1)=7.05$, $p < 0.01$), as such a three level model was used.

Model A (Table 2) included baseline alcohol consumption on a bogus taste test, alcohol and neutral SSRTs respectively for baseline and RA sessions, to examine if they can account for whether the participant consumed alcohol or not. Neutral SSRTs (OR = 1.05; 95% CI 1.02, 1.08, $p < 0.001$) explained a significant

Table 2. Multilevel model examining participant-level and daily-level predictors of alcohol consumption.

	Model A	Model B	Model C
Parameter	OR (95% CI)	OR (95% CI)	B (95% CI)
Outcome	Binary (drank vs not)	Binary (drank vs not)	Continuous (units cons.)
Participant Level			
Ad-lib alcohol	1.27 (0.90, 1.80)	1.26 (.83, 1.91)	1.11 (.26, 1.97)*
Daily Level			
Neutral SSRT	1.05 (1.02, 1.08)***	1.05 (1.02, 1.08)**	.07 (−0.01,.15)
Alcohol SSRT	1.12 (0.88, 1.43)	1.09 (0.85, 1.40)	.14 (−0.60, .87)
Craving intensity		1.01 (.91, 1.12)	−0.36 (−0.60, −0.13)*
Craving frequency		1.65 (1.48, 1.84)***	.26 (.02, .50)*
Location		1.22 (.86, 1.73)	−0.59 (−1.52, 0.34)
Sad		.89 (.78, 1.01)	.41 (.06, .76)*
Energetic		1.00 (.90, 1.10)	−0.04 (−0.31, .22)
Happy		1.08 (.95, 1.23)	.26 (−0.05, .57)
Drowsy		1.15 (1.05, 1.25)**	.03 (−0.21, .26)
Marginal R2	.026	.237	.107
Conditional R2	.146	.348	.177
N data points	1344	1344	268
ICC	.12	.15	.08

SSRT, Stop Signal Reaction Time. Location reference category is home.
*$p < 0.05$.
**$p < 0.01$.
***$p < 0.0001$.

Table 1. Participant characteristics and measurements from baseline, random and breathalyzer assessments.

		Participant-level baseline variables				
Age (years)	24.30 (7.58)					
TLFB consumption	42.83 (18.84)					
TLFB binge frequency	3.39 (2.08)					
AUDIT	11.94 (5.59)					
BIS total	74.52 (7.05)					
TRI	49.90 (18.38)					
SCS	32.87 (4.10)					
DAQ	29.22 (14.21)					
Daily level	**Baseline**	**1st RA**	**2nd RA**	**3rd RA**	**4th RA**	**ICC**
Alcohol SSRT	313.86 (62.36)	288.17 (69.35)	287.56 (56.73)	294.92 (71.90)	298.32 (71.32)	0.213
Neutral SSRT	307.07 (62.37)	289.50 (61.50)	287.58 (60.40)	293.15 (58.42)	297.97 (73.18)	0.265
Craving intensity	27.41 (21.51)	17.76 (18.66)	21.99 (21.58)	29.40 (26.32)	32.97 (28.48)	0.328
Craving frequency	24.83 (23.40)	21.46 (20.92)	20.75 (21.43)	60.04 (25.32)	31.89 (28.21)	0.357
Sad	24.38 (17.99)	28.70 (1.93)	26.39 (19.39)	27.33 (19.59)	24.58 (18.51)	0.254
Energetic	50.69 (21.89)	41.67 (23.70)	42.71 (23.57)	43.45 (22.70)	41.15 (24.71)	0.190
Happy	64.50 (15.23)	57.98 (19.78)	59.02 (18.63)	61.05 (18.91)	63.28 (18.77)	0.196
Drowsy	38.81 (26.05)	46.12 (26.24)	42.99 (26.48)	43.33 (26.27)	45.60 (26.48)	0.214
Units consumed	-	1.42 (3.18)	0.29 (1.48)	0.44 (1.55)	0.83 (2.34)	0.115

Values are means (standard deviation).
Abbreviations- TLFB, Timeline Follow back. AUDIT, Alcohol Use Disorders Identification Test. BIS, Behavioral Impulsivity Scale. TRI CEP, Temptation and Restraint Inventory Cognitive Emotional Preoccupation, TRI CBC, Temptation and Restraint Inventory Cognitive Behavioral Control. SCS, Self-Control Scale. DAQ, Desire for Alcohol Questionnaire. SSRT, Stop Signal Reaction Time. RA, Random Assessment.

amount of variance in the model, alcohol SSRTs (OR = 1.12, 95%CI 0.88, 1.43, $p=0.353$) and baseline alcohol consumption (OR = 1.27, 95% CI 0.90, 1.80, $p=0.170$), did not explain a significant proportion of variance. In Model B (Table 2), we added in the assessment-level variables of frequency of craving, location, sad, happy, energetic, and drowsy. Neutral SSRTs, frequency of craving and drowsiness significantly predicted variance in whether individuals consumed alcohol or not.

Sensitivity analyses

In sensitivity analysis we removed all sessions in which no alcohol consumption was reported and the outcome was amount (in units) consumed. A two-level model (assessment > participant) was a significantly better fit than a single level model ($\chi^2(1)=6.03$, $p<0.05$). A three-level model (assessment > day > participant) was not significantly better fit than a two level model ($\chi^2(1)=0.31$, $p=0.58$), as such a two level model was used. Model C (Table 2) used the same predictor variables as Model B, with reported alcohol consumption, as a continuous variable, as the dependent variable. Significant predictors were baseline alcohol consumption ($\beta=1.11$, 95% CI 0.26, 1.97, $p<0.05$), intensity of craving ($\beta=-0.04$, 95% CI −0.06, −0.01, $p<0.01$), frequency of craving ($\beta=0.03$, 95% CI 0, 0.05, $p<0.05$) and sad ($\beta=0.04$, 95% CI 0.01, 0.08, $p<0.05$). Note, that this model has considerably lower statistical power.

In further sensitivity analysis we removed all sessions in which there was an interruption or positive breathalyzer reading, the outcome was amount (in units) consumed. Significant predictors of alcohol consumed were Neutral SSRTs ($\beta=-0.00$, 95% CI 0.00, 0.00, $p<0.05$), intensity of craving (($\beta=-0.03$, 95% CI −0.04, −0.02, $p<0.001$) and frequency of craving ($\beta=-0.03$, 95% CI 0.02, 0.04, $p<0.001$), however the multilevel structure was not a good fit of the data, likely due to decreased power.

Hypothesis 3: Fluctuations in inhibitory control, both proactive and reactive, to alcohol stimuli & environment.

We examined if inhibitory control (SSRTs) fluctuated in response to alcohol stimuli. A multilevel model with predictors of cue type (alcohol vs. neutral) and environment (home, not home) with random intercepts for subject and day was conducted on SSRTs. There was no main effect of cue type ($B=-5.85$ [95% CI: −12.62, 0.92], $p=0.090$). However, there was an effect of location ($B=-9.10$ [95% CI: −15.71, −2.48], $p=0.007$). There, was also a weak significant interaction ($B=9.10$ [95% CI: 0.04, 17.80], $p=0.040$). Alcohol SSTs were increased outside of the home, and Neutral SSRTs decreased out of home. Alcohol SSRTs at home = 291.70 (SD = 56.48), Alcohol SSRTs out of home = 295.92 (SD = 81.84), Neutral SSRTs at home = 294.45 (SD = 61.31) and Neutral SSRTS out of home = 289.84 (SD = 67.12)

Discussion

The aim of this study was to examine if momentary fluctuations in inhibitory control could predict alcohol consumption. Hypothesis one was partially supported, as increased neutral SSRTs predicted a subsequent alcohol consumption occasion. Hypothesis two was supported, as baseline measurements of ad-libitum alcohol consumption accounted for a significant amount of variance in subsequent decisions to consume alcohol (and amount consumed). Hypothesis three was not supported, reactive inhibitory control performance did not fluctuate as a result of location nor cue exposure, however alcohol SSRTs were significantly slower outside of the home.

Whilst the findings from this study demonstrated some limited evidence for inhibitory control predicting alcohol use, this was only for SSRTS to generic neutral stimuli. These findings are broadly in line with Jones et al. (2018) who demonstrated daily fluctuations in inhibitory control were predictive of alcohol use, using arbitrary cues ('x' and 'o', on the stop signal task). However, it was surprising that there was no evidence for alcohol SSRTs given theoretical predictions and associations between alcohol-related cues, inhibitory control and alcohol consumption in the laboratory (Czapla et al., 2016; Jones et al., 2013; Weafer & Fillmore, 2015). Recent pre-registered work suggests that exposure to alcohol cues alone is not enough to create inhibitory deficits, but priming (consumption) may influence reactive components of inhibition (Baines et al., 2019). It is possible that participants demonstrated habituation to alcohol-related cues, and as such they exerted limited effects as the testing sessions persisted (Courtney et al., 2015). These findings then lend some support to wider theoretical models which posit the importance of inhibitory control on subsequent alcohol consumption (Goldstein & Volkow, 2011; Kalivas & Volkow, 2005; Paz et al., 2016), but less so the importance of cue-specific inhibition (Jones et al., 2013). These observations further support inhibitory control as a potential risk fact for alcohol use and (re)lapse (Gilbey & Wilcockson, 2024), and future studies might consider Ecological Momentary Interventions (Balaskas et al., 2021)which allow us to identify these fluctuations 'in the moment' and intervene by attempting to increase inhibitory control (Iannazzo et al., 2025)or prompting the individual to avoid temptation.

Location of the assessments did not explain a significant amount of variance in the alcohol use data. Due to the lack of variation in assessment locations outside of participant's home, data was recoded as either being at home or out of home, reducing the specificity of our measurement and potentially negating the effect of environment on alcohol use. Social and contextual changes can influence alcohol use (Correia et al., 2012), with bars and private residences, in particular, leading to high levels of alcohol consumption (Wray et al., 2014). Importantly, environments alone do not influence alcohol use. There is likely to be an interaction with social contexts, and the cognitive response evoked by situational cues (Vengeliene et al., 2020) and social context (Erskine-Shaw et al., 2017).

Interestingly, ad-libitum alcohol consumption in the laboratory predicted alcohol use in the real world. These findings extend the work of Jones et al. (2018) who had no baseline measure of alcohol consumption, showing that the bogus-taste test is analogous of real-world alcohol consumption. Lab-based consumption predicted real-world consumption, such findings are promising given previous speculation over the validity of the bogus taste-test (Leeman et al., 2009; Leeman et al., 2013;

Robinson et al., 2015; Robinson et al., 2014). Particularly for the field of experimental medicine the bogus taste-test appears to be an effective proxy for alcohol consumption, enabling its use to develop interventions to reduce alcohol consumption within a lab based environment (Field et al., 2021).

Our study has multiple strengths, we accounted for baseline measures of inhibitory control and alcohol use (*via* the ad-libitum taste test) allowing cross-validation between control conditions and real-world environments. In respect to alcohol use as a dependent variable, we conducted a variety of analyses such as daily consumption, only reported alcohol use session, and as a binary variable, allowing for robust analyses. Confounds (e.g., disturbances or alcohol intoxication) were accounted for as part of sensitivity analyses. The experimental paradigm used a greater number of testing sessions, allowing for dynamic changes in inhibitory control to be examined in comparison to previous studies (Jones et al., 2018), and more reliable estimates (Shiffman, 2014). Importantly, compliance was at acceptable levels for Ecological Momentary Assessment studies (Shiffman, 2014; Stone & Shiffman, 2002).

There were also a number of limitations. Firstly, we did not examine when alcohol was last consumed in relation to a testing session, and therefore cannot determine the effect of alcohol on inhibitory control or vice versa demonstrating the need for a more finite understanding of conditions during the initiation of drinking. Future research should ask participants to complete sessions following alcohol consumption (Collins et al., 2003). Secondly, participants were not given a cutoff as to when they had to complete the testing session by, meaning that poorer response inhibition may not have been caught (e.g., participants may delay their response if they felt their performance would be poor). Future EMA studies should control for the intended time of the RA and when the participant completed the session using cutoff points to examine the sensitivity of the paradigm. Further to this we did not differentiate between week and weekend RA sessions, future EMA research should examine drinking behaviors across these time frames given that motives may differ to consume alcohol (Lau-Barraco et al., 2016). Our sample was comprised of heavy drinkers limiting the generalizability of findings, future research should seek to examine different groups of drinkers such as, light drinkers and those with substance use disorders to examine fluctuations in inhibitory control in the real world. The majority of the sample were females, with inadequate numbers for gender analysis, given to the use of opportunity sampling, while prior research has found gender differences in inhibitory control (Weafer et al., 2015), we found no effect of gender on outcomes (see supplementary analysis for gender). Future research should seek to sample sufficient numbers of both genders to allow for the analysis of gender within an EMA paradigm, especially given the literature on gender differences in cue-reactivity (Kaag et al., 2019). Further to this future research should adopt purposive sampling to achieve a sample representative of the wider population to investigate differences between genders and ethnicity for example. It is possible that repeated testing of inhibitory control might lead to practice or fatigue effects (Spierer et al., 2013), however in exploratory analyses we note that assessment day did not predict SSRTs for either alcohol or neutral cues here. Finally, our ecological momentary assessments focused on alcohol consumption, but consumption of other drugs may also be associated with inhibitory control (e.g., Cannabis: Griffith-Lendering et al., 2012) and future studies may consider examining a more complete profile of psychoactive substance use.

To summarize, we found no consistent association between inhibitory control or fluctuations of inhibitory control and alcohol use. Despite previous research we found a limited effect of location or motivation to alcohol related stimuli on inhibitory control.

Disclosure statement

No potential conflict of interest was reported by the author(s).

Funding

This work was supported by a research studentship from Alcohol Change (grant number RS 17/01).

ORCID

Sam Burton http://orcid.org/0000-0003-3823-3275

References

Baines, L., Field, M., Christiansen, P., & Jones, A. (2019). The effect of alcohol cue exposure and acute intoxication on inhibitory control processes and ad libitum alcohol consumption. *Psychopharmacology*, *236*(7), 2187–2199. https://doi.org/10.1007/s00213-019-05212-4

Balaskas, A., Schueller, S. M., Cox, A. L., & Doherty, G. (2021). Ecological momentary interventions for mental health: A scoping review. *PloS One*, *16*(3), e0248152. https://doi.org/10.1371/journal.pone.0248152

Bickel, W. K., Jarmolowicz, D. P., Mueller, E. T., Gatchalian, K. M., & McClure, S. M. (2012). Are executive function and impulsivity antipodes? A conceptual reconstruction with special reference to addiction. *Psychopharmacology*, *221*(3), 361–387. https://doi.org/10.1007/s00213-012-2689-x

Bujarski, S., & Ray, L. A. (2016). Experimental psychopathology paradigms for alcohol use disorders: Applications for translational research. *Behaviour Research and Therapy*, *86*, 11–22. https://doi.org/10.1016/j.brat.2016.05.008

Burnham, K. P., & Anderson, D. R. (2004). Multimodel inference: Understanding AIC and BIC in model selection. *Sociological Methods & Research*, *33*(2), 261–304. https://doi.org/10.1177/0049124104268644

Burton, S. P., Knibb, G., & Jones, A. (2021). EXPRESS: A meta-analytic investigation of the role of reward on inhibitory control. *Quarterly Journal of Experimental Psychology*, *74*(10), 1818–1828. https://doi.org/10.1177/17470218211008895

Christiansen, P., & Bloor, J. F. (2014). Individualised but not general alcohol Stroop predicts alcohol use. *Drug and Alcohol Dependence*, *134*, 410–413. https://doi.org/10.1016/j.drugalcdep.2013.10.021

Christiansen, P., Cole, J. C., Goudie, A. J., & Field, M. (2012). Components of behavioural impulsivity and automatic cue approach predict unique variance in hazardous drinking. *Psychopharmacology*, *219*(2), 501–510. https://doi.org/10.1007/s00213-011-2396-z

Christiansen, P., Mansfield, R., Duckworth, JAY., Field, M., & Jones, A. (2015). Internal reliability of the alcohol-related visual probe task is increased by utilising personalised stimuli and eye-tracking. *Drug and Alcohol Dependence*, *155*, 170–174. https://doi.org/10.1016/j.drugalcdep.2015.07.672

Collins, R. L., Kashdan, T. B., & Gollnisch, G. (2003). The feasibility of using cellular phones to collect ecological momentary assessment data:

Application to alcohol consumption. *Experimental and Clinical Psychopharmacology*, *11*(1), 73–78. https://doi.org/10.1037/1064-1297.11.1.73

Correia, C. J., Murphy, J. G., & Barnett, N. P. (2012). *College student alcohol abuse: A guide to assessment, intervention, and prevention*. John Wiley & Sons.

Courtney, K. E., Ghahremani, D. G., & Ray, L. A. (2015). The effect of alcohol priming on neural markers of alcohol cue-reactivity. *The American Journal of Drug and Alcohol Abuse*, *41*(4), 300–308. https://doi.org/10.3109/00952990.2015.1044608

Czapla, M., Simon, J. J., Richter, B., Kluge, M., Friederich, H.-C., Herpertz, S., Mann, K., Herpertz, S. C., & Loeber, S. (2016). The impact of cognitive impairment and impulsivity on relapse of alcohol-dependent patients: Implications for psychotherapeutic treatment. *Addiction Biology*, *21*(4), 873–884. https://doi.org/10.1111/adb.12229

De Wit, H. (2009). Impulsivity as a determinant and consequence of drug use: A review of underlying processes. *Addiction Biology*, *14*(1), 22–31. https://doi.org/10.1111/j.1369-1600.2008.00129.x

Department of Health. (2016). *Alcohol guidelines review - Report from the guidelines development group to the UK Chief Medical Officers*. Department of Health.

Erskine-Shaw, M., Monk, R. L., Qureshi, A. W., & Heim, D. (2017). The influence of groups and alcohol consumption on individual risk-taking. *Drug and Alcohol Dependence*, *179*, 341–346. https://doi.org/10.1016/j.drugalcdep.2017.07.032

Fatseas, M., Serre, F., Alexandre, J.-M., Debrabant, R., Auriacombe, M., & Swendsen, J. (2015). Craving and substance use among patients with alcohol, tobacco, cannabis or heroin addiction: A comparison of substance- and person-specific cues. *Addiction (Abingdon, England)*, *110*(6), 1035–1042. https://doi.org/10.1111/add.12882

Fernie, G., Peeters, M., Gullo, M. J., Christiansen, P., Cole, J. C., Sumnall, H., & Field, M. (2013). Multiple behavioural impulsivity tasks predict prospective alcohol involvement in adolescents. *Addiction (Abingdon, England)*, *108*(11), 1916–1923. https://doi.org/10.1111/add.12283

Field, M., & Eastwood, B. (2005). Experimental manipulation of attentional bias increases the motivation to drink alcohol. *Psychopharmacology*, *183*(3), 350–357. https://doi.org/10.1007/s00213-005-0202-5

Field, M., & Jones, A. (2017). Elevated alcohol consumption following alcohol cue exposure is partially mediated by reduced inhibitory control and increased craving. *Psychopharmacology*, *234*(19), 2979–2988. https://doi.org/10.1007/s00213-017-4694-6

Field, M., Christiansen, P., Hardman, C. A., Haynes, A., Jones, A., Reid, A., & Robinson, E. (2021). Translation of findings from laboratory studies of food and alcohol intake into behavior change interventions: The experimental medicine approach. *Health Psychology: Official Journal of the Division of Health Psychology, American Psychological Association*, *40*(12), 951–959. https://doi.org/10.1037/hea0001022

Field, M., Wiers, R. W., Christiansen, P., Fillmore, M. T., & Verster, J. C. (2010). Acute alcohol effects on inhibitory control and implicit cognition: Implications for loss of control over drinking. *Alcoholism, Clinical and Experimental Research*, *34*(8), 1346–1352. https://doi.org/10.1111/j.1530-0277.2010.01218.x

Fujita, K. (2011). On Conceptualizing Self-Control as More Than the Effortful Inhibition of Impulses. *Personality and Social Psychology Review: An Official Journal of the Society for Personality and Social Psychology, Inc*, *15*(4), 352–366. https://doi.org/10.1177/1088868311411165

Gilbey, H., & Wilcockson, T. D. (2024). The effect of cognitive control on addiction relapse. *Addiction Research & Theory*, *33*, 1–8.

Goldstein, R. Z., & Volkow, N. D. (2011). Dysfunction of the prefrontal cortex in addiction: Neuroimaging findings and clinical implications. *Nature Reviews. Neuroscience*, *12*(11), 652–669. https://doi.org/10.1038/nrn3119

Griffith-Lendering, M. F., Huijbregts, S. C., Vollebergh, W. A., & Swaab, H. (2012). Motivational and cognitive inhibitory control in recreational cannabis users. *Journal of Clinical and Experimental Neuropsychology*, *34*(7), 688–697. https://doi.org/10.1080/13803395.2012.668874

Hayes, A. F. (2006). A primer on multilevel modeling. *Human Communication Research*, *32*(4), 385–410. https://doi.org/10.1111/j.1468-2958.2006.00281.x

Houston, R. J., Derrick, J. L., Leonard, K. E., Testa, M., Quigley, B. M., & Kubiak, A. (2014). Effects of heavy drinking on executive cognitive functioning in a community sample. *Addictive Behaviors*, *39*(1), 345–349. https://doi.org/10.1016/j.addbeh.2013.09.032

Iannazzo, L. H., Hayden, M. J., Lawrence, N. S., Kakoschke, N., Hughes, L. K., Van Egmond, K., Lum, J., & Staiger, P. K. (2025). Inhibitory control training to reduce appetitive behaviour: A meta-analytic investigation of effectiveness, potential moderators, and underlying mechanisms of change. *Health Psychology Review*, *19*(1), 66–96. https://doi.org/10.1080/17437199.2024.2410018

Jenkins, R. J., McAlaney, J., & McCambridge, J. (2009). Change over time in alcohol consumption in control groups in brief intervention studies: Systematic review and meta-regression study. *Drug and Alcohol Dependence*, *100*(1-2), 107–114. https://doi.org/10.1016/j.drugalcdep.2008.09.016

Jones, A., & Field, M. (2015). Alcohol-related and negatively valenced cues increase motor and oculomotor disinhibition in social drinkers. *Experimental and Clinical Psychopharmacology*, *23*(2), 122–129. https://doi.org/10.1037/pha0000011

Jones, A., Button, E., Rose, A. K., Robinson, E., Christiansen, P., Di Lemma, L., & Field, M. (2016). The ad-libitum alcohol 'taste test': secondary analyses of potential confounds and construct validity. *Psychopharmacology*, *233*(5), 917–924. https://doi.org/10.1007/s00213-015-4171-z

Jones, A., Christiansen, P., Nederkoorn, C., Houben, K., & Field, M. (2013). Fluctuating Disinhibition: Implications for the Understanding and Treatment of Alcohol and Other Substance Use Disorders. *Frontiers in Psychiatry*, *4*(, 140. https://doi.org/10.3389/fpsyt.2013.00140

Jones, A., Cole, J., Goudie, A., & Field, M. (2011). Priming a restrained mental set reduces alcohol-seeking independently of mood. *Psychopharmacology*, *218*(3), 557–565. https://doi.org/10.1007/s00213-011-2338-9

Jones, A., Field, M., Christiansen, P., & Stancak, A. (2013). P300 during response inhibition is associated with ad-lib alcohol consumption in social drinkers. *Journal of Psychopharmacology (Oxford, England)*, *27*(6), 507–514. https://doi.org/10.1177/0269881113485142

Jones, A., Guerrieri, R., Fernie, G., Cole, J., Goudie, A., & Field, M. (2011). The effects of priming restrained versus disinhibited behaviour on alcohol-seeking in social drinkers. *Drug and Alcohol Dependence*, *113*(1), 55–61. https://doi.org/10.1016/j.drugalcdep.2010.07.006

Jones, A., Rose, A. K., Cole, J., & Field, M. (2013). Effects of alcohol cues on craving and ad libitum alcohol consumption in social drinkers: The role of disinhibition. *Journal of Experimental Psychopathology*, *4*(3), 239–249. https://doi.org/10.5127/jep.031912

Jones, A., Tiplady, B., Houben, K., Nederkoorn, C., & Field, M. (2018). Do daily fluctuations in inhibitory control predict alcohol consumption? An ecological momentary assessment study. *Psychopharmacology*, *235*(5), 1487–1496. https://doi.org/10.1007/s00213-018-4860-5

Kaag, A. M., Wiers, R. W., de Vries, T. J., Pattij, T., & Goudriaan, A. E. (2019). Striatal alcohol cue-reactivity is stronger in male than female problem drinkers. *The European Journal of Neuroscience*, *50*(3), 2264–2273. https://doi.org/10.1111/ejn.13991

Kalivas, P. W., & Volkow, N. D. (2005). The neural basis of addiction: A pathology of motivation and choice. *The American Journal of Psychiatry*, *162*(8), 1403–1413. https://doi.org/10.1176/appi.ajp.162.8.1403

Kramer, J. R., Chan, G., Hesselbrock, V. M., Kuperman, S., Bucholz, K. K., Edenberg, H. J., Schuckit, M. A., Nurnberger, J. I., Foroud, T., Dick, D. M., Bierut, L. J., & Porjesz, B. (2010). A principal components analysis of the abbreviated Desires for Alcohol Questionnaire (DAQ). *Journal of Studies on Alcohol and Drugs*, *71*(1), 150–155. https://doi.org/10.15288/jsad.2010.71.150

Lau-Barraco, C., & Linden, A. N. (2014). Drinking buddies: Who are they and when do they matter? *Addiction Research & Theory*, *22*(1), 57–67. https://doi.org/10.3109/16066359.2013.772585

Lau-Barraco, C., Braitman, A. L., Linden-Carmichael, A. N., & Stamates, A. L. (2016). Differences in weekday versus weekend drinking among non-student emerging adults. *Experimental and Clinical Psychopharmacology*, *24*(2), 100–109. https://doi.org/10.1037/pha0000068

Leckie, G. (2019). *Multilevel models for continuous outcomes*.

Leeman, R. F., Corbin, W. R., & Fromme, K. (2009). Craving Predicts Within Session Drinking Behavior Following Placebo. *Personality and Individual Differences*, *46*(7), 693–698. https://doi.org/10.1016/j.paid.2009.01.024

Leeman, R. F., Corbin, W. R., Nogueira, C., Krishnan-Sarin, S., Potenza, M. N., & O'Malley, S. S. (2013). A human alcohol self-administration paradigm to model individual differences in impaired control over alcohol use. *Experimental and Clinical Psychopharmacology*, *21*(4), 303–314. https://doi.org/10.1037/a0033438

Leeman, R. F., Heilig, M., Cunningham, C. L., Stephens, D. N., Duka, T., & O'Malley, S. S. (2010). Ethanol consumption: How should we measure it? Achieving consilience between human and animal phenotypes. *Addiction Biology*, *15*(2), 109–124. https://doi.org/10.1111/j.1369-1600.2009.00192.x

Liang, W., & Chikritzhs, T. (2015). Weekly and daily cycle of alcohol use among the US general population. *Injury*, *46*(5), 898–901. https://doi.org/10.1016/j.injury.2015.01.029

Logan, G. D., Cowan, W. B., & Davis, K. A. (1984). On the ability to inhibit simple and choice reaction time responses: A model and a method. *Journal of Experimental Psychology. Human Perception and Performance*, *10*(2), 276–291. https://doi.org/10.1037/0096-1523.10.2.276

López-Caneda, E., Rodríguez Holguín, S., Cadaveira, F., Corral, M., & Doallo, S. (2014). Impact of alcohol use on inhibitory control (and vice versa) during adolescence and young adulthood: A review. *Alcohol and Alcoholism (Oxford, Oxfordshire)*, *49*(2), 173–181. https://doi.org/10.1093/alcalc/agt168

Maas, C. J., & Hox, J. J. (2005). Sufficient sample sizes for multilevel modeling. *Methodology*, *1*(3), 86–92. https://doi.org/10.1027/1614-2241.1.3.86

Marhe, R., Waters, A. J., van de Wetering, B. J. M., & Franken, I. H. A. (2013). Implicit and Explicit Drug-Related Cognitions during Detoxification Treatment are Associated with Drug Relapse: An Ecological Momentary Assessment Study. *Journal of Consulting and Clinical Psychology*, *81*(1), 1–12. https://doi.org/10.1037/a0030754

Monk, R. L., Heim, D., Qureshi, A., & Price, A. (2015). "I Have No Clue What I Drunk Last Night" Using Smartphone Technology to Compare In-Vivo and Retrospective Self-Reports of Alcohol Consumption. *PloS One*, *10*(5), e0126209. https://doi.org/10.1371/journal.pone.0126209

Nakagawa, S., & Schielzeth, H. (2013). A general and simple method for obtaining R2 from generalized linear mixed-effects models. *Methods in Ecology and Evolution*, *4*(2), 133–142. https://doi.org/10.1111/j.2041-210x.2012.00261.x

Nigg, J. T., Wong, M. M., Martel, M. M., Jester, J. M., Puttler, L. I., Glass, J. M., Adams, K. M., Fitzgerald, H. E., & Zucker, R. A. (2006). Poor response inhibition as a predictor of problem drinking and illicit drug use in adolescents at risk for alcoholism and other substance use disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, *45*(4), 468–475. https://doi.org/10.1097/01.chi.0000199028.76452.a9

Paccagnella, O. (2006). Centering or Not Centering in Multilevel Models? The Role of the Group Mean and the Assessment of Group Effects. *Evaluation Review*, *30*(1), 66–85. https://doi.org/10.1177/0193841x05275649

Patton, J. H., Stanford, M. S., & Barratt, E. S. (1995). Factor structure of the Barratt impulsiveness scale. *Journal of Clinical Psychology*, *51*(6), 768–774. https://doi.org/10.1002/1097-4679(199511)51:6<768::AID-JCLP2270510607>3.0.CO;2-1

Paz, A. L., Keim, C. A., & Rosselli, M. (2016). Inhibitory Performance Predicting Drinking Behaviours Among Young Adults. *Alcohol and Alcoholism (Oxford, Oxfordshire)*, *51*(6), 677–683. https://doi.org/10.1093/alcalc/agw017

Quené, H., & Van den Bergh, H. (2004). On multi-level modeling of data from repeated measures designs: A tutorial. *Speech Communication*, *43*(1-2), 103–121. https://doi.org/10.1016/j.specom.2004.02.004

Remmerswaal, D., Jongerling, J., Jansen, P. J., Eielts, C., & Franken, I. H. (2019). Impaired subjective self-control in alcohol use: An ecological momentary assessment study. *Drug and Alcohol Dependence*, *204*, 107479. https://doi.org/10.1016/j.drugalcdep.2019.04.043

Robinson, E., Hardman, C. A., Halford, J. C., & Jones, A. (2015). Eating under observation: A systematic review and meta-analysis of the effect that heightened awareness of observation has on laboratory measured energy intake. *The American Journal of Clinical Nutrition*, *102*(2), 324–337. https://doi.org/10.3945/ajcn.115.111195

Robinson, E., Kersbergen, I., Brunstrom, J. M., & Field, M. (2014). I'm watching you. Awareness that food consumption is being monitored is a demand characteristic in eating-behaviour experiments. *Appetite*, *83*, 19–25. https://doi.org/10.1016/j.appet.2014.07.029

Roos, L. E., Knight, E. L., Beauchamp, K. G., Berkman, E. T., Faraday, K., Hyslop, K., & Fisher, P. A. (2017). Acute stress impairs inhibitory control based on individual differences in parasympathetic nervous system activity. *Biological Psychology*, *125*, 58–63. https://doi.org/10.1016/j.biopsycho.2017.03.004

Rubio, G., Jiménez, M., Rodríguez-Jiménez, R., Martínez, I., Avila, C., Ferre, F., Jiménez-Arriero, M. A., Ponce, G., & Palomo, T. (2008). The role of behavioral impulsivity in the development of alcohol dependence: A 4-year follow-up study. *Alcoholism, Clinical and Experimental Research*, *32*(9), 1681–1687. https://doi.org/10.1111/j.1530-0277.2008.00746.x

Rupp, C. I., Beck, J. K., Heinz, A., Kemmler, G., Manz, S., Tempel, K., & Fleischhacker, W. W. (2016). Impulsivity and alcohol dependence treatment completion: Is there a neurocognitive risk factor at treatment entry? *Alcoholism, Clinical and Experimental Research*, *40*(1), 152–160. https://doi.org/10.1111/acer.12924

Serre, F., Fatseas, M., Swendsen, J., & Auriacombe, M. (2015). Ecological momentary assessment in the investigation of craving and substance use in daily life: A systematic review. *Drug and Alcohol Dependence*, *148*, 1–20. https://doi.org/10.1016/j.drugalcdep.2014.12.024

Shiffman, S. (2009). Ecological Momentary Assessment (EMA) in Studies of Substance Use. *Psychological Assessment*, *21*(4), 486–497. https://doi.org/10.1037/a0017074

Shiffman, S. (2014). Conceptualizing analyses of ecological momentary assessment data. *Nicotine & Tobacco Research: Official Journal of the Society for Research on Nicotine and Tobacco*, *16 Suppl 2*(Suppl 2), S76–S87. https://doi.org/10.1093/ntr/ntt195

Shiffman, S., Stone, A. A., & Hufford, M. R. (2008). Ecological momentary assessment. *Annual Review of Clinical Psychology*, *4*(1), 1–32. https://doi.org/10.1146/annurev.clinpsy.3.022806.091415

Smith, J. L., Mattick, R. P., Jamadar, S. D., & Iredale, J. M. (2014). Deficits in behavioural inhibition in substance abuse and addiction: A meta-analysis. *Drug and Alcohol Dependence*, *145*, 1–33. https://doi.org/10.1016/j.drugalcdep.2014.08.009

Sobell, L. C., & Sobell, M. B. (1992). Timeline follow-back. In *Measuring alcohol consumption* (pp. 41–72): Springer.

Spierer, L., Chavan, C. F., & Manuel, A. L. (2013). Training-induced behavioral and brain plasticity in inhibitory control. *Frontiers in Human Neuroscience*, *7*, 427. https://doi.org/10.3389/fnhum.2013.00427

Stone, A. A., & Shiffman, S. (2002). Capturing momentary, self-report data: A proposal for reporting guidelines. *Annals of Behavioral Medicine: A Publication of the Society of Behavioral Medicine*, *24*(3), 236–243. https://doi.org/10.1207/S15324796ABM2403_09

Trautmann, S., Kräplin, A., Muehlhan, M., Fuchs, F. O., Loesch, B., & Wittgens, C. (2024). The ad-libitum taste test as measure of momentary alcohol use in the laboratory: An investigation of construct validity and confounding factors. *Psychopharmacology*, *241*(5), 913–923. https://doi.org/10.1007/s00213-023-06518-0

Vengeliene, V., Foo, J. C., & Kim, J. (2020). Translational approach to understanding momentary factors associated with alcohol consumption. *British Journal of Pharmacology*, *177*(17), 3878–3897. https://doi.org/10.1111/bph.15180

Verbruggen, F., & Logan, G. D. (2008). Response inhibition in the stop-signal paradigm. *Trends in Cognitive Sciences*, *12*(11), 418–424. https://doi.org/10.1016/j.tics.2008.07.005

Verbruggen, F., & Logan, G. D. (2009). Models of response inhibition in the stop-signal and stop-change paradigms. *Neuroscience and Biobehavioral Reviews*, *33*(5), 647–661. https://doi.org/10.1016/j.neubiorev.2008.08.014

Verbruggen, F., Aron, A. R., Band, G. P. H., Beste, C., Bissett, P. G., Brockett, A. T., Brown, J. W., Chamberlain, S. R., Chambers, C. D., Colonius, H., Colzato, L. S., Corneil, B. D., Coxon, J. P., Dupuis, A., Eagle, D. M., Garavan, H., Greenhouse, I., Heathcote, A., Huster, R. J., … Boehler, C. N. (2019). A consensus guide to capturing the ability to inhibit actions and impulsive behaviors in the stop-signal task. *eLife*, *8*. https://doi.org/10.7554/eLife.46323

Verbruggen, F., Chambers, C. D., & Logan, G. D. (2013). Fictitious inhibitory differences: How skewness and slowing distort the estima-

tion of stopping latencies. *Psychological Science*, *24*(3), 352–362. https://doi.org/10.1177/0956797612457390

Verdejo-García, A., Lawrence, A. J., & Clark, L. (2008). Impulsivity as a vulnerability marker for substance-use disorders: Review of findings from high-risk research, problem gamblers and genetic association studies. *Neuroscience and Biobehavioral Reviews*, *32*(4), 777–810. https://doi.org/10.1016/j.neubiorev.2007.11.003

Waters, A. J., Marhe, R., & Franken, I. H. (2012). Attentional bias to drug cues is elevated before and during temptations to use heroin and cocaine. *Psychopharmacology*, *219*(3), 909–921. https://doi.org/10.1007/s00213-011-2424-z

Weafer, J., & Fillmore, M. T. (2015). Alcohol-related cues potentiate alcohol impairment of behavioral control in drinkers. *Psychology of Addictive Behaviors: Journal of the Society of Psychologists in Addictive Behaviors*, *29*(2), 290–299. https://doi.org/10.1037/adb0000013

Weafer, J., & Fillmore, M. T. (2016). Low-Dose Alcohol Effects on Measures of Inhibitory Control, Delay Discounting, and Risk-Taking. *Current Addiction Reports*, *3*(1), 75–84. https://doi.org/10.1007/s40429-016-0086-y

Weafer, J., De Arcangelis, J., & de Wit, H. (2015). Sex differences in behavioral impulsivity in at-risk and non-risk drinkers. *Frontiers in Psychiatry*, *6*, 72. https://doi.org/10.3389/fpsyt.2015.00072

Wignall, N. D., & de Wit, H. (2011). Effects of nicotine on attention and inhibitory control in healthy nonsmokers. *Experimental and Clinical Psychopharmacology*, *19*(3), 183–191. https://doi.org/10.1037/a0023292

Wray, T. B., Merrill, J. E., & Monti, P. M. (2014). Using Ecological Momentary Assessment (EMA) to Assess Situation-Level Predictors of Alcohol Use and Alcohol-Related Consequences. *Alcohol Res*, *36*(1), 19–27. Retrieved from https://pubmed.ncbi.nlm.nih.gov/26258997 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4432855/