

Acute effect of alcohol on working memory updating

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ABSTRACT

Aims To examine the acute effects of alcohol on working memory (WM) updating, including potential variation across the ascending limb (AL) and descending limb (DL) of the blood alcohol concentration (BAC) time-course. **Design** A two-session experiment in which participants were randomly assigned to one of three beverage conditions [alcohol (males: 0.80 g/kg; females: 0.72 g/kg), active placebo (0.04 g/kg) or non-alcohol control (tonic)] and one of two BAC limb testing conditions (AL and DL or DL-only) for the second session, yielding a 3 (beverage) × 2 (time-points tested) × 3 (time-point) mixed factorial design with repeated measures on the latter factor. One of the repeated assessments is 'missing by design' in the DL-only condition. **Setting** A psychology laboratory at the University of Missouri campus in Columbia, MO, USA. **Participants** Two hundred thirty-one community-dwelling young adults (51% female; aged 21–34 years) recruited from Columbia, MO, USA, tested between 2011 and 2013. **Measurements** Latent WM updating performance as indexed by shared variance in accuracy on three WM updating tasks (letter memory, keep track, spatial 2-back) at three time-points. **Findings** Multi-group modeling of latent WM updating indicated that performance among participants who consumed placebo or control beverages improved during the second session at time-points corresponding to AL (Δ from baseline in latent mean \pm standard error (SE) + 0.5 \pm 0.01, $P < 0.001$) and DL (+ 0.08 \pm 0.01, $P < 0.001$). Alcohol consumption did not impair WM updating (Δ from baseline in latent mean \pm SE, at AL: + 0.01 \pm 0.01, $P = 0.56$; at DL: + 0.05 \pm 0.01, $P < 0.001$), but attenuated performance improvements (equality of latent means across beverage groups at AL or DL: $\Delta\chi^2_{(1)} \geq 7.53$, $P < 0.01$). **Conclusions** Acute alcohol-induced impairment in working memory updating may be limited, but dampening of practice effects by alcohol could interfere with the completion of novel, unpracticed tasks.

Keywords Alcohol, executive functioning, pharmacology, practice, updating, working memory.

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Submitted 4 October 2020; initial review completed 2 December 2020; final version accepted 24 March 2021

INTRODUCTION

Numerous theoretical models [1–4] posit that alcohol-related hazards arise from alcohol-induced impairment in executive functioning (EF). EF refers to higher-order cognitive processes that support control over thoughts and actions during goal-directed behavior [5], which can be organized into general and specific components [6]. A subcomponent of EF, working memory (WM) updating, reflects the ability to maintain and manipulate existing information in WM while dynamically replacing or updating other information in WM. Broadly, acute impairment of EF is believed to underlie impaired control over drinking and negative consequences [7–10].

Intoxicated individuals may experience several difficulties due to WM updating impairment, including problems keeping track of numbers of drinks consumed and rendering accurate mental maps of locations, conversations and changing situations. In this way, intoxicated individuals may not maintain awareness of long-term goals and/or shield them against competing short-term goals and temptations [11].

Evidence for acute effects of alcohol on WM updating is mixed. A recent systematic review ($k = 13$) concluded that performance on auditory/speech-related WM tasks was reliably impaired by alcohol even at moderate doses, whereas performance on visuospatial WM tasks was commonly spared even at higher doses [12]. Others have questioned

that conclusion by showing that performance on auditory/speech-related WM tasks can be unaffected at low to moderate doses [13–15], and performance on visuospatial WM tasks can be impaired at moderate to high doses [16,17] and in daily life [18].

Limitations of previous studies

Previous studies of alcohol and WM have been limited in at least five ways. First, most studies have utilized a single WM updating task [19–22], making it difficult to generalize beyond a specific task to the broader construct due to ‘task impurity’ [6,23]. Laboratory tasks often suffer from low reliability [24], so scores on individual WM updating tasks can reflect the influence of other EF facets, non-EF-related cognitive processes and measurement error [6]. Thus, divergent effects reported in the literature may arise from task heterogeneity and unreliability. Latent variable models circumvent this issue by capturing shared variance among construct-relevant tasks and accounting for measurement error. Secondly, most studies use small samples ($n \approx 25$) [12], which are both underpowered and more likely to produce false-positive findings. Thirdly, acute alcohol effects are tested almost entirely against a single control condition, either placebo-alcohol [25,26] or a no-alcohol beverage [22,27]. Including placebo and no-alcohol control conditions allows testing whether acute effects are driven by expectancy, pharmacology or both [28].

Fourthly, most existing studies examined alcohol effects while blood alcohol concentration (BAC) is rising or at its peak [29–31], but alcohol’s effect on WM updating may differ within a drinking episode. For instance, WM updating may improve upon the descending relative to ascending limb of the BAC curve due to acute tolerance [32,33]. Subjective intoxication appears to be reliably affected by acute tolerance, but acute tolerance effects on impaired performance are neither reliable nor uniform across cognitive-behavioral domains [34,35]. Few studies have examined acute tolerance during WM updating [15,22,36], although this phenomenon could cause acute alcohol-related hazards. Fifthly, previous work has not considered practice effects. Repeated exposure to EF tasks improves task performance, which may indicate decreased difficulty or novelty and, in some cases, the development of better strategies [37,38]. Acute alcohol effects could manifest in impaired WM updating performance relative to baseline, or in the blunting or elimination of practice effects.

[†]We define a unit of alcohol as a serving of 14 g ethanol, which is the approximate amount of ethanol in 148 ml (5 fl. oz) of wine rated at 12% ethanol vol/vol or 355 ml of [12 fl. oz.] rated at 5% ethanol vol/vol or 44 ml (1.5 fl. oz) of liquor rated at 40% ethanol vol/vol [80].

[‡]We define a heavy use occasion as an occasion in which 5+ and 4+ units were consumed in a single sitting for males and females, respectively.

Current study

In this study, these limitations were addressed with several design features. We used a relatively large community sample ($n = 231$) of young adults, representing the developmental period in which heavy drinking, alcohol-related problems and alcohol use disorder (AUD) are most prevalent [39–42]. Three widely used WM updating tasks [43–50] were completed during two laboratory sessions separated by 1–3 weeks. To circumvent task impurity, we derived latent variables from performance across the three tasks. The two-session feature allowed for identifying between-subject differences in baseline ability and within-subject changes in performance between and within sessions. We also compared performance under alcohol to active placebo and no-alcohol control beverage conditions to disentangle alcohol’s expected and pharmacological effects. Finally, WM updating was assessed either once or twice after beverage consumption (either while BAC was descending or while it was both ascending and descending). This ‘missing by design’ [51] feature permits examination of whether any differences in alcohol’s effects during ascending and descending BAC reflect acute tolerance or practice effects.

The study had three main aims: (1) test the extent to which alcohol acutely impairs WM updating; (2) examine whether acute effects of alcohol on WM updating differ during ascending versus descending BAC; and (3) determine whether any such differences reflect acute tolerance or practice effects.

METHOD

The University of Missouri Institutional Review Board approved all procedures. Analyses were planned prior to data collection in the grant application that funded the study (P60 AA011998 5979). However, analyses were not formally pre-registered, so results should be considered exploratory.

Participants

Two hundred and thirty-one healthy young adults (51% female; 86% Caucasian) aged 21–34 years [median = 23.14, standard deviation (SD) = 2.74] who reported regular alcohol use (2–25 units[†] per week and at least one heavy use occasion[‡] during the past year) and no contraindications to alcohol administration (see Supporting information) were recruited from Columbia, MO, as in our two previous, related studies [52,53]. Table 1 presents demographics and recent alcohol use

Table 1 Characteristics of the sample (n = 231).

	Alcohol (n = 85)		Placebo (n = 71)		Control (n = 75)		χ^2	P
	A/D (n = 46)	D-only (n = 39)	A/D (n = 36)	D-only (n = 35)	A/D (n = 38)	D-only (n = 37)		
Age in years, median (IQR)	22.17 (3.81)	22.19 (1.98)	21.90 (1.65)	22.19 (1.61)	21.94 (1.70)	21.96 (1.96)	2.62	0.758
Sex, n, female (%)	23 (50)	20 (51)	18 (50)	17 (48)	19 (50)	19 (51)	0.08	0.999
Ethnicity, n Hispanic (%)	1 (2)	3 (8)	3 (8)	0 (0)	2 (5)	4 (11)	5.81	0.325
Race, n (%)							33.09	0.130
American Indian or Alaska Native	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (3)		
Asian	0 (0)	3 (8)	0 (0)	0 (0)	0 (0)	0 (0)		
Black or African American	5 (11)	0 (0)	1 (3)	5 (14)	3 (8)	0 (0)		
Native Hawaiian or Pacific Islander	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)		
White	39 (85)	32 (82)	33 (92)	28 (80)	33 (87)	34 (92)		
No information	2 (4)	4 (10)	2 (5)	2 (6)	2 (5)	2 (5)		
Alcohol use, median (IQR)								
Times used in past 3 months	19.29 (31.07)	19.29 (12.86)	19.29 (37.50)	19.29 (37.50)	19.29 (0.00)	19.29 (25.71)	3.06	0.691
Typical units in past 3 months	5.00 (3.00)	5.00 (2.00)	5.00 (2.50)	3.00 (2.50)	5.00 (3.00)	3.00 (2.00)	3.49	0.625
Times used in past 30 days	6.43 (0.00)	6.43 (4.29)	6.43 (3.93)	6.43 (12.5)	6.43 (8.57)	6.43 (0.00)	1.55	0.907
Typical units in past 30 days	4.00 (3.50)	4.00 (2.00)	4.00 (3.00)	4.00 (3.00)	5.00 (3.00)	3.00 (3.00)	3.88	0.567
Times 5+ units in past 30 days	2.50 (6.18)	2.50 (5.43)	1.00 (3.48)	1.00 (5.43)	2.50 (5.43)	1.00 (6.43)	7.37	0.194
Times 12+ units in past 30 days	0.00 (0.00)	0.00 (1.00)	0.00 (0.00)	0.00 (0.50)	0.00 (0.00)	0.00 (1.00)	1.15	0.950
Max. units used in past 30 days	7.50 (5.00)	8.00 (6.00)	7.00 (4.87)	7.00 (5.50)	8.00 (4.00)	5.00 (8.00)	5.63	0.344
Max. units used in life-time	15.00 (10.00)	16.00 (9.50)	14.50 (11.25)	13.00 (6.50)	12.25 (8.50)	15.00 (8.00)	5.14	0.399

A/D refers to the subset of participants assigned to complete working memory (WM) updating tasks on both the ascending and descending limbs of the BAC time-course or corresponding time-points. D-only refers to the subset of participants assigned to complete WM updating tasks only on the descending limb of the BAC time-course or corresponding time-points. IQR = interquartile range. Median and IQR are given for Supporting information alcohol use because these were not normally distributed. With respect to alcohol use, 1 unit here refers to US standard serving equivalents (14 g ethanol). See footnote v for alcohol use question language, response options and scaling. The similarity of the distributions of age and alcohol use across the six experimental cells to which participants were randomized was verified using Kruskal–Wallis χ^2 tests with 5 degrees of freedom (d.f.), whereas the similarity of the distributions of sex, ethnicity and race categories was verified using Pearson's χ^2 tests with 5, 5 and 25 d.f., respectively.

for individuals randomly assigned to each cell of the experiment.

Measures

Breath alcohol concentration (BrAC)

BrAC was measured using an Alco-Sensor IV (Intoximeters, St Louis, MO, USA) as g/210 l exhaled air, which is equivalent to g/dl whole blood, and is reported here as g%. BrACs are a reliable [54,55] proxy for expected post-absorption BACs [56,57]. In the alcohol and placebo conditions, BrAC was measured every 15–30 minutes after the end of the beverage administration period (24 minutes consumption, 5 minutes absorption), with care taken not to interrupt task set completion. BrACs were not shared with participants.

Placebo manipulation check items

At the end of the experimental session, participants assigned to the alcohol and placebo conditions were asked to rate their subjective intoxication (0–4, 'not at all' to 'a lot') after beverage consumption as well as during the AL and DL procedures. Additionally, they were asked to indicate 'the number of standard drinks you think would be equivalent to what you drank in the study today', using integers 0–20.

WM updating tasks

WM updating was measured with three widely used [43–50] tasks: keep track [6,44], letter memory [6,43], and spatial 2-back [45,46], each of which is described briefly below (see Supporting information for detailed descriptions). Task scores reflected the proportion of correct responses. Internal reliability varied across tasks, but was reasonable in all tasks (see Table 2). Test–re-rest reliability was also reasonable (see Table 3). Moreover, as in previous

reports [6,46,48, 58], tasks were moderately correlated with one another at each assessment (see Table 4), supporting the notion that they measure the same underlying construct.

Keep track task. After seeing a sequence of 15–25 words, participants were asked to recall and repeat aloud the most recently presented exemplar from three to five distinct categories (e.g. animals, countries).

Letter memory task. After seeing a sequence of nine to 13 letters, participants were asked to recall and repeat aloud the four most recent letters in the order of presentation.

Spatial 2-back task. Across a sequence of visual 'flashes' at different locations on a monitor, participants were asked to indicate via button press whether the location of the most recent 'flash' matched or did not match the location two 'flashes' back in the sequence.

Procedure

Figure 1 shows the overall design, including the baseline and experimental sessions, key events within each session and randomization of participants to one of six conditions for the experimental session.

Analytical strategy

Following previous work [45,46], task scores were winsorized at ± 3 SD from the mean (to reduce the influence of extreme values but retain their ordinal positions) and then subjected to angular transformation (arcsine of the square root) to normalize their distributions [59].

Latent variable models were estimated in Mplus version 7.3 [60] using the robust maximum likelihood estimator and full-information maximum likelihood [61], which can handle the 'planned missingness' feature of

Table 2 Internal consistency reliability of raw accuracy scores for each task at every assessment.

Tasks	ICC			$r_{\text{split-half}}$		
	Baseline	AL	DL	Baseline	AL	DL
KT	0.50	0.50	0.61	0.27	0.37	0.40
LM	0.57	0.68	0.72	0.47	0.55	0.60
SNB	0.88	0.87	0.89	0.82	0.82	0.83

KT = keep track task; LM = letter memory task; SNB = spatial 2-back task. Baseline represents assessment at the baseline session. AL = for ascending limb (or corresponding time-point) assessment in the experimental session; DL = descending limb for ascending limb (or corresponding time-point) assessment in the experimental session; ICC = intraclass correlation coefficient. Specifically, ICC (3, k) [79] was computed, using the k trials in each task as the k raters. Split-half correlation ($r_{\text{split-half}}$) was computed as Pearson's r for the average score across even- versus odd-numbered trials in each task, and it is shown without Spearman-Brown correction. The number of participants contributing pairwise complete data was: for KT, baseline $n = 231$, AL $n = 120$, DL $n = 228$; for LM, baseline $n = 231$, AL $n = 119$, DL $n = 227$; and for SNB, baseline $n = 230$, AL $n = 119$, DL $n = 230$. All ICC and $r_{\text{split-half}}$ were significantly different from 0 at $P < 0.001$.

Table 3 Test–re-test reliability of raw and transformed accuracy scores for each task across assessment.

Grp.	Tasks	ICC						$r_{\text{test-retest}}$							
		Baseline versus AL		Baseline versus DL		AL versus DL		Baseline versus AL		Baseline versus DL		AL versus DL			
		Raw	Transf	Raw	Transf	Raw	Transf	Raw	Transf	Raw	Transf	Raw	Transf		
All	KT	0.73	0.74	0.61	0.62	0.77	0.77	0.80	0.80	0.57	0.58	0.44	0.45	0.63	0.63
	LM	0.67	0.71	0.66	0.66	0.78	0.78	0.77	0.79	0.51	0.55	0.49	0.50	0.65	0.64
	SNB	0.81	0.82	0.78	0.76	0.87	0.87	0.87	0.87	0.69	0.71	0.64	0.64	0.78	0.77
Cntrl	KT	0.76	0.78	0.55	0.59	0.84	0.83	0.81	0.82	0.61	0.65	0.38	0.41	0.73	0.71
	LM	0.67	0.67	0.57	0.59	0.75	0.77	0.76	0.78	0.50	0.51	0.40	0.42	0.66	0.66
	SNB	0.83	0.82	0.72	0.69	0.91	0.88	0.89	0.97	0.72	0.74	0.58	0.56	0.82	0.78

Grp = groups included; Cntrl = control beverage only; Transf = transformed; KT = keep track task; LM = letter memory task; SNB = spatial 2-back task. Baseline represents assessment at the baseline session. AL = for ascending limb (or corresponding time-point) assessment in the experimental session; DL = descending limb for ascending limb (or corresponding time-point) assessment in the experimental session; ICC = intraclass correlation coefficient. Specifically, ICC (3, *k*) [79] was computed, using *k* = 2 or 3 tests, as the *k* raters. Test–re-test correlations ($r_{\text{test-retest}}$) were computed as Pearson’s *r*. For ease of comparison with the extant literature, correlations are presented for both raw and transformed (angularized and winsorized; see Analytical strategy) task score. For groups included = all, the number of participants contributing complete data was: for KT, baseline versus AL *n* = 120, baseline versus DL *n* = 228, AL versus DL *n* = 120, baseline versus AL versus DL *n* = 120; for LM, baseline versus AL *n* = 119, baseline versus DL *n* = 227, AL versus DL *n* = 119, baseline versus AL versus DL *n* = 119; and for SNB, baseline versus AL *n* = 119, baseline versus DL *n* = 228, AL versus DL *n* = 119, baseline versus AL versus DL *n* = 119. For groups included = control only, the number of participants contributing complete data was: for KT, baseline versus AL *n* = 38, baseline versus DL *n* = 73, AL versus DL *n* = 38, baseline versus AL versus DL *n* = 38; for LM, baseline versus AL *n* = 36, baseline versus DL *n* = 74, AL versus DL *n* = 38, baseline versus AL versus DL *n* = 38; and for SNB, baseline versus AL *n* = 36, baseline versus DL *n* = 72, AL versus DL *n* = 38, baseline versus AL versus DL *n* = 38. All ICC and $r_{\text{test-retest}}$ were significantly different from 0 at *P* < 0.001.

Table 4 Intercorrelation of raw and transformed task accuracy scores at every assessment.

Tasks	Baseline		AL		DL	
	Raw	Transformed	Raw	Transformed	Raw	Transformed
KT versus LM	0.18**	0.22***	0.37***	0.41***	0.28***	0.34***
KT versus SNB	0.11	0.14*	0.38***	0.37**	0.25***	0.26***
LM versus SNB	0.26***	0.28***	0.24**	0.28**	0.19**	0.24***

KT = keep track task; LM = letter memory task; SNB = spatial 2-back task. Baseline represents assessment at the baseline session. AL = ascending limb (or corresponding time-point) assessment in the experimental session; DL = descending limb (or corresponding time-point) assessment in the experimental session. Correlations were computed as Pearson’s *r*. For ease of comparison with the extant literature, correlations are presented for both raw and transformed (angularized and winsorized; see Analytical strategy) task scores. The number of participants contributing pairwise complete data was: for baseline, KT versus LM *n* = 231, KT versus SNB *n* = 229, LM versus SNB *n* = 229; for AL, KT versus LM *n* = 119, KT versus SNB *n* = 119, LM versus SNB *n* = 118; and for DL, KT versus LM *n* = 226, KT versus SNB *n* = 227, LM versus SNB *n* = 226. **P* < 0.05; ***P* < 0.01; ****P* < 0.001.

this study (i.e. D-only condition; see Fig. 1) as well as data missing at random.ⁱⁱⁱ Adequacy of model fit was based on the following guidelines suggested in the literature: comparative fit index (CFI) and Tucker–Lewis index (TLI) > 0.95 for reasonably good fit; and root mean square error of approximation (RMSEA) ≤ 0.08 for reasonable fit and ≤ 0.05 for close fit. We also report model fit χ^2 significance tests but did not rely upon them, because they are highly sensitive to *n* [62].

To examine groups’ latent WM updating performance across testing occasions, we specified a series of multi-group models (Fig. 2). We established strict measurement and temporal invariance on the structural model (equivalent task loadings, intercepts and residual variances on latent WM updating factors across groups and occasions; see Table S5 for invariance analyses), which is critical to rule out the possibility that observed differences in latent mean values across groups and occasions are due

ⁱⁱⁱDue to equipment malfunction, some data were lost at the time of collection. For the keep track task, baseline time-point data were available from all 231 participants, and ascending limb time-point data were available for all 120 participants assigned to the A/D condition, but descending time-point data were missing from three of 231 participants. For the letter memory task, baseline time-point data were available from all 231 participants, but ascending limb time-point data were missing for one of 120 participants assigned to the A/D condition, and descending time-point data were missing from four of 231 participants. For the spatial 2-back task, baseline time-point data were missing for two of 231 participants, ascending limb time-point data were missing for one of 120 participants assigned to the A/D condition and descending time-point data were missing for one of 231 participants.

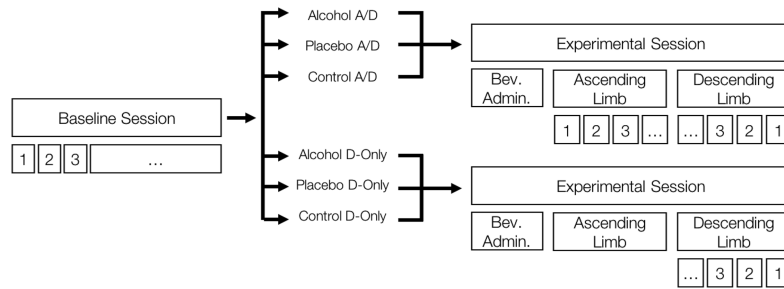


Figure 1 Schematic overview of experimental design. Bev. admin. = beverage administration. Baseline sessions (3–4 hours) were held between 9 a. m. and 1 p.m. Experimental sessions (4 hours) were held between 12 p.m. and 5 p.m. Sessions were held 1–3 weeks apart. Baseline session: after BrAC testing to confirm sobriety, participants provided informed consent and completed the three WM updating tasks (letter memory [LM], keep track [KT], and spatial N-back [SNB]; squares labeled ‘1’, ‘2’, ‘3’) and six other executive functioning (EF) tasks (rectangle labeled ‘...’; not reported here). Tasks were completed in a fixed order counterbalanced across participants. Experimental session: after BrAC testing to confirm sobriety, participants provided informed consent again and were asked to void the bladder, during which time female participants also self-administered a hormone-based (urine) pregnancy test in a private restroom (all tested negative). Participants were then administered a beverage and completed the three WM updating tasks (LM, KT, SNB; squares labeled ‘1’, ‘2’, ‘3’) and two other EF tasks (squares labeled ‘...’; not reported here) once or twice during the session according to randomly assigned experimental conditions. Participants were randomly assigned to one of six experimental cells resulting from fully crossing a three-level beverage condition with a two-level assessment condition. Beverage conditions were alcohol (0.80 g/kg for males, 0.72 g/kg for females), active placebo alcohol (0.04 g/kg) and no-alcohol control (tonic only). Participants assigned to the control condition were told that the beverage contained ‘no alcohol’ whereas participants in the alcohol and placebo conditions were told that the beverage contained ‘a moderate amount of alcohol’. Assessment conditions were A/D (tasks completed during both ascending and descending limbs of the BAC time-course or corresponding time-points) and D-only (tasks completed only during descending limb of the BAC time-course or corresponding time-points). Following beverage administration, participants assigned to the A/D condition completed the tasks when BrAC had risen to at least 0.065 g% (or corresponding time-points), whereas participants assigned to the D-only condition watched episodes of a popular sitcom (*The Office [U.S.]*). After peak BrAC (≈ 0.085 g%), participants in both the A/D and D-only conditions completed the tasks (in reverse order) when BrAC had fallen to at least 0.075 g% (or corresponding time-points).

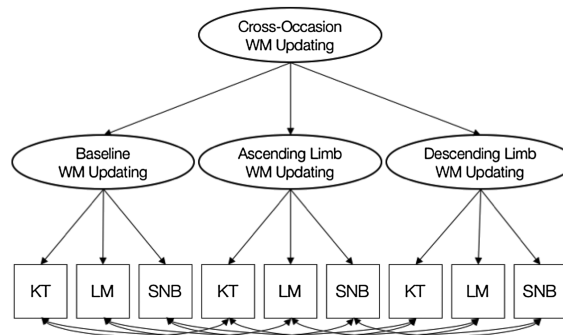


Figure 2 Conceptual diagram of the latent variable model of working memory (WM) updating performance. KT = keep track task; LM = letter memory task; SNB = spatial N (2)-back task. Baseline refers to the baseline session. Ascending limb and descending limb refer to different periods within the experimental session (completed 1–3 weeks after baseline session under one of six experimental conditions). See Fig. 1 for procedural details and Analytical strategy for multi-group modeling details. This figure depicts a conceptual diagram reflecting the multi-group models we tested. We examined pharmacological effects of alcohol by testing differences in latent WM updating means across ‘Alcohol’ groups (0 = no alcohol [placebo/control], 1 = Alcohol [alcohol]) and alcohol expectancy effects with ‘Expectancy’ groups (first, defined as: 0 = no expectancy [control], 1 = Expectancy [alcohol/placebo]). To disentangle ‘pure’ expectancy effects from pharmacological effects, we also tested ‘Pure expectancy’ groups (0 = no expectancy [control], 1 = expectancy [placebo]). Thus, multi-group models comprised two groups (no alcohol versus alcohol, no expectancy versus expectancy), each with three lower-order latent factors (i.e. one per testing occasion), one higher-order (cross-occasion) factor onto which each lower-order factor loaded and residual covariances among tasks across occasions and within groups. The WM updating latent factor from the baseline testing occasion of either the no alcohol or no expectancy group served as the reference factor. The most invariant multi-group models, which equated task factor loadings, intercepts and residual variances across groups and occasions, fit well (alcohol/no alcohol: $\chi^2 = 83$, degrees of freedom [d.f.] = 59, comparative fit index [CFI] = 0.961, Tucker–Lewis index [TLI] = 0.953, and root mean square error of approximation [RMSEA] = 0.059; expectancy/no expectancy: $\chi^2 = 82$, d.f. = 59, CFI = 0.963, TLI = 0.954, RMSEA = 0.058; see Supporting information, Table S5 for invariance analyses and Supporting information, Table S6 for final model parameters).

to measurement differences rather than substantive change [63]. We then tested whether latent means could be constrained to equality across groups and occasions

(see Supporting information, Table S5 for invariance analyses). Here, models were compared using the Δ CFI ($|0.002|$) and Δ RMSEA ($|0.015|$); we gave preference

Table 5 BrAC (g%) at start of AL and DL procedures for participants assigned to alcohol condition.

By WM updating task completion condition	AL		DL	
	Median	SD	Median	SD
A/D	0.075	0.014	0.067	0.008
D-only	0.071	0.013	0.067	0.007
Collapsing WM updating task completion condition	0.073	0.013	0.067	0.008

AL = ascending limb; DL = descending limb of the breath alcohol concentration (BrAC) curve; SD = standard deviation; WM = working memory. A/D refers to 46 participants who completed the WM updating tasks on both the AL and DL. D-only refers to 39 participants who completed the WM updating tasks only on the DL. Although D-only participants completed WM updating tasks only on the DL, their BrAC values were recorded on both the AL and DL.

to Δ CFI because it is more stringent and widely accepted [64,65]. We report Satorra–Bentler $\Delta\chi^2$ tests, but did not rely upon them because they are overly sensitive to peripheral factors (e.g. n) [65].

RESULTS

BrAC at start of AL and DL procedures

Table 5 presents BrAC medians and SDs. BrACs were analyzed using a 2 (task completion group: A/D, D-only) \times 2 (BAC curve limb: AL, DL) mixed factorial analysis of variance (ANOVA) with repeated measures on the latter factor. There was a significant main effect of BAC curve limb, $F_{(1, 83)} = 22.26$, $P < 0.001$, such that BrACs were lower at DL than AL, $t_{(84)} = 4.96$, $P < 0.001$. Neither the main effect of the task completion group, $F_{(1, 83)} = 1.52$, $P = 0.221$, nor the task completion group \times BAC curve limb interaction effect, $F_{(1, 83)} = 2.36$, $P = 0.128$, were significant.

Placebo manipulation check

Almost all placebo participants (69 of 71) estimated consuming a non-zero number of alcoholic drinks during the study, but those numbers, as well as retrospective subjective intoxication ratings, were lower among placebo compared to alcohol participants (see Table 6).

WM updating performance

Acute effects of alcohol

Alcohol versus no alcohol groups. Figure 3 presents the baseline,^{iv} AL and DL mean accuracy for each WM updating task as a function of beverage and task completion conditions.

A model with equal task factor loadings and intercepts across groups and testing occasions, and equal residual

task variances across groups fit the data well ($\chi^2 = 83$, degrees of freedom (d.f.) = 59, CFI = 0.961, TLI = 0.953, RMSEA = 0.059; see Supporting information, Table S5 for invariance analyses and Supporting information, Table S6 for final model parameters).^v

Baseline latent means did not differ across the alcohol and no alcohol groups, but there were group differences on AL and DL (Table 7, Fig. 4; see Supporting information, Table S7 for equality tests). On both AL and DL, latent means were higher for the no alcohol compared to alcohol groups.

Expectancy versus no expectancy groups. When the alcohol and placebo conditions were pooled into group expectancy, baseline and DL latent means did not differ between the expectancy and no expectancy groups, but there was a group difference on AL (Table 7, Fig. 4; see Supporting information, Table S7 for equality tests). On AL, latent means were higher for no the expectancy relative to expectancy groups. However, when the expectancy group contained only the placebo condition, there were no differences in WM updating between the expectancy and no expectancy groups at baseline, AL or DL (Table 7, Fig. 4; see Supporting information, Table S7 for equality tests). Together, these findings indicate that the expectancy effect in the traditional expectancy model that pooled alcohol and placebo conditions was a false positive reflecting the pharmacological effect of alcohol, and that there was no ‘pure’ expectancy effect.

Differences across limbs of the BAC curve

Alcohol versus no alcohol groups. For both groups latent means increased across occasions, but the pattern of means differed within groups (Table 7, Fig. 4; see Supporting information, Table S7 for equality tests). In the no alcohol group, latent means were significantly different among all testing occasions, such that:

^{iv}Baseline performance was equivalent among experimental cells. The relevant ANOVAs are presented in Supporting information. Baseline performance was in step with norms for this age group.[6,46]

^vThe constrained model is conceptually and statistically equivalent to a repeated-measures ANOVA design, but with latent factor variances estimated. Hierarchical regression analysis for each task and separate repeated-measures ANOVA within task completion conditions for each task are presented in the Supporting information. Results from these analyses mirrored those presented in the main text.

Table 6 Post-experiment expectancy manipulation checks.

	Alcohol						Placebo							
	Median	SD	Med	IQR	R	#0	%0	Median	SD	Median	IQR	R	#0	%0
Subjective intoxication immediately after beverage consumption														
A/D	2.49*	0.79	3	1	0-4	1*	2	1.47 ^a	0.86	2	1	0-3	5*	14
D-only	2.58*	0.81	3	1	1-4	0	0	1.48 ^a	0.79	1	1	0-4	2*	6
Collapsing WM Updating Task Completion condition	2.58*	0.80	3	1	0-4	1*	1	1.48 ^a	0.82	1	1	0-4	7 ^a	10
Subjective intoxication during AL procedures														
A/D	2.29*	0.79	2	1	0-4	2*	4	1.35 ^a	0.77	1	1	0-3	5*	14
D-only	2.79*	0.81	3	1	1-4	0	0	1.33 ^a	0.92	1	1	0-3	6 ^a	17
Collapsing WM Updating Task Completion condition	2.52*	0.83	2	1	0-4	2*	2	1.34 ^a	0.84	1	1	0-3	11 ^a	15
Subjective intoxication during DL procedures														
A/D	1.55	0.78	1	1	0-4	2*	4	0.68 ^a	0.81	0.5	1	0-3	17 ^a	47
D-only	1.92	0.85	2	1	0-4	1*	3	0.88 ^a	0.78	1	1	0-3	11 ^a	31
Collapsing WM Updating Task Completion condition	1.72*	0.83	2	1	0-4	3*	3	0.78 ^a	0.79	1	1	0-3	28 ^a	39
Perceived number of standard drink equivalents consumed														
A/D	4.09*	1.18	4	2	3-8	0	0	2.62 ^a	1.30	3	1	0-7	2*	5
D-only	4.10*	1.20	4	2	2-7	0	0	2.81 ^a	1.50	3	1	1-7	0	0
Collapsing WM Updating Task Completion condition	4.09*	1.19	4	2	2-8	0	0	2.71 ^a	1.39	3	1	0-7	2*	3

A/D = participants who completed the working memory (WM) updating tasks on both the ascending limb (AL) and descending limb (DL) of the breath alcohol concentration curve (alcohol $n = 46$; placebo $n = 36$). D-only = participants who completed the WM updating tasks only on the DL (alcohol $n = 39$; placebo $n = 35$). IQR = interquartile range. R = range, shown as min-max. #0 = number of participants for whom response was 0; %0 = percentage of participants for whom response was 0. Item and response language are provided in Supporting Information. Mean drink estimates and intoxication ratings were evaluated using t -tests ($*P < 0.05$ for null hypothesis test of group mean equivalence to 0; $^aP < 0.05$ for null hypothesis test of placebo group mean equivalence to alcohol group mean). Rank-based Wilcoxon s tests (on group medians) produced the same pattern of results. Pearson s^2 tests (degrees of freedom = 1) were used on the #0 ($*P < 0.05$ for null hypothesis test of uniform distribution of #0s and #non-0s within groups; $^bP < 0.05$ for null hypothesis test of similar distribution of #0s and #non-0s between placebo and alcohol groups). For drink estimate #0, the null hypothesis test of uniformly distributed 0s and non-0s could not be conducted for experimental cells in which no participants indicated 0 drinks.

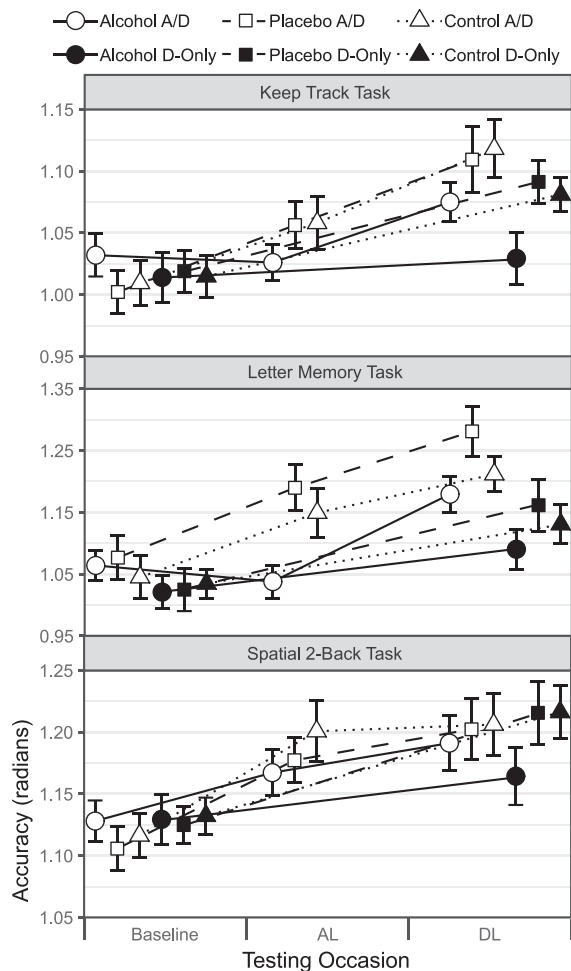


Figure 3 Mean accuracy across testing occasions for each task as a function of beverage and session 2 task completion condition. Transformed accuracy is the arcsine of the square root of the winsorized proportion correct. Testing occasion baseline refers to session 1. Testing occasion AL and DL are the ascending limb and descending limb of the blood alcohol concentration (BAC) curve, respectively, or corresponding time-points during session 2. Tasks were completed on DL only (D-only) or on both AL and DL (A/D). Beverage conditions were: alcohol, placebo or control. Sample sizes were 46, 36, 38, 39, 35 and 37 for alcohol A/D, placebo A/D, control A/D, alcohol D-only, placebo D-only and control D-Only groups, respectively. Error bars are ± 1 standard error of the mean.

baseline < AL < DL. In the alcohol group, the latent mean was not significantly different at baseline compared to AL, but was significantly lower at baseline compared to DL and at AL compared to DL.

Expectancy versus no expectancy groups. For both groups, latent means increased across occasions, and in both groups all latent means differed from each other (baseline < AL < DL; Table 7, Fig. 4; see Supporting information, Table S7 for equality tests).

Acute tolerance

Alcohol versus no alcohol groups. To determine whether improvement across post-consumption testing occasions

Table 7 Latent WM updating means across groups and testing occasions.

Model	SE	p	No alcohol (n = 146)		Alcohol (n = 85)	
			Median	SE	Median	SE
Alcohol effects model (n = 231)	0.00	-	0.00	0.01	0.00	0.634
Baseline	0.01	< 0.001	0.05	0.01	0.01	0.560
Ascending limb	0.01	< 0.001	0.08	0.01	0.05	< 0.001
Descending limb	0.01	< 0.001				
Expectancy effects model (n = 231)			No expectancy (n = 75)		Expectancy (n = 156)	
Baseline	0.00	-	0.00	0.01	0.00	0.705
Ascending limb	0.01	< 0.001	0.05	0.01	0.03	0.010
Descending limb	0.01	< 0.001	0.08	0.01	0.07	< 0.001
Pure expectancy effects model (n = 146)			No expectancy (n = 75)		Expectancy (n = 71)	
Baseline	0.00	-	0.00	0.01	0.00	1.00
Ascending limb	0.01	< 0.001	0.05	0.01	0.05	< 0.001
Descending limb	0.01	< 0.001	0.08	0.01	0.09	< 0.001

All means were estimated from models under strict measurement invariance constraints. P-values indicate the significance of change from the reference level, which was baseline WM updating no alcohol or no expectancy groups, depending on the model. SE = standard error; WM = working memory.

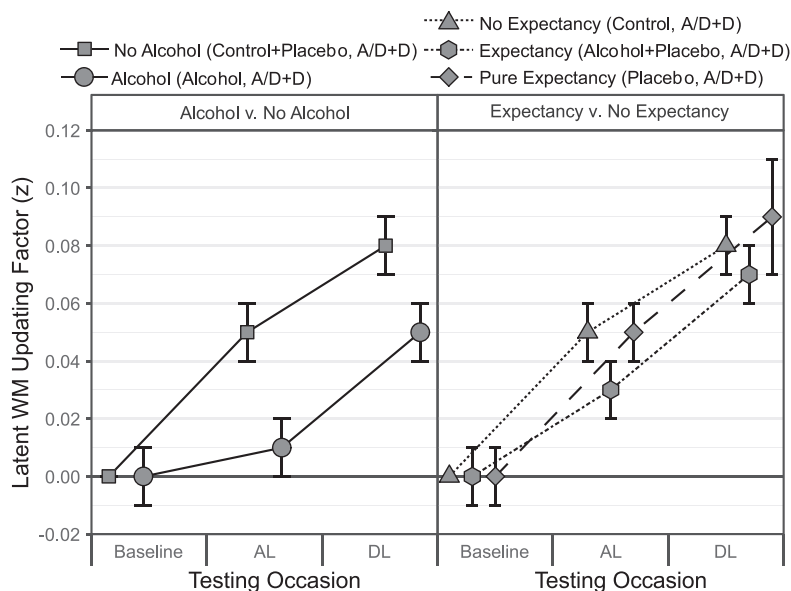


Figure 4 Latent working memory (WM) updating factor means across testing occasions for alcohol versus no alcohol and expectancy versus no expectancy models. Testing occasion baseline refers to session 1. Testing occasion AL and DL are the ascending limb and descending limb of the BAC curve, respectively, or corresponding time-points during session 2. Figure legend shows experiment cells pooled into each group in the multi-group latent variable model. A/D and D in the figure legend refer to session 2 assessment on both AL and DL or DL only, respectively. Baseline WM updating in the no alcohol or no expectancy groups served as the reference factor. Error bars are ± 1 standard error.

in the alcohol group was due to acute tolerance, we tested whether task completion condition (0 = A/D, 1 = D-only) predicted DL WM updating differently across the alcohol and no alcohol groups (Table S8). Task completion condition was negatively associated with DL WM updating to the same extent in both groups, indicating a lower WM updating performance when tasks were completed only once (on DL or corresponding time-points). This suggests that acute tolerance does not account for improved performance of the alcohol group at DL relative to AL.

Next, we re-estimated the latent means at each occasion after dropping D-only participants (Supporting information, Table S9) to examine whether the alcohol and no alcohol groups differed in the degree of performance improvement across testing occasions. WM updating latent means were significantly higher at AL than at baseline in the no alcohol group, but baseline and AL latent means were equivalent in the alcohol group. This suggests that alcohol prevented performance improvement from baseline to AL. In both groups, latent means were lower at AL than at DL. Latent means at DL were lower in the alcohol compared to no alcohol groups, but to a lesser degree than when D-only participants were included in both groups. Together, these findings indicate that alcohol also attenuated performance improvement from AL to DL.

DISCUSSION

By administering multiple laboratory tasks across separate sessions to a large sample, including both placebo and no-alcohol control conditions, and comparing performance across BAC curve limbs, the current study provided the most comprehensive test to date of alcohol's acute effects on WM updating. We found both between- and within-subject effects of alcohol pharmacology on WM updating. Across time, WM updating was lower among intoxicated compared with sober individuals, consistent with the previously reported between-subject effect on WM tasks [13,66,67]. Nonetheless, alcohol consumption did not diminish participants' WM updating performance relative to baseline ability. This finding is consistent with older reports [68,69], but is inconsistent with more recent reports [17,22,70]. Moreover, we found no between- or within-subject effects of alcohol expectancy on WM updating.^{vi} One possible explanation is that participants held no strong expectancies about negative effects of alcohol on task performance, and so did not attempt to compensate for anticipated impairment [28]. Another possibility is that, although the placebo manipulation convinced nearly all participants that they had consumed alcohol, they did not feel intoxicated enough to warrant compensatory efforts.

We also found that WM updating performance was better on the DL compared with baseline or the AL, but

^{vi}Dropping the two participants who were not convinced by the placebo (i.e. estimated having consumed 0 standard alcoholic drink equivalents during the study) did not change the latent mean patterns in any of the multi-group latent variable models (see Supporting information, Table S18).

there was essentially no evidence that DL relative to AL performance in the alcohol group was due to acute tolerance rather than repeated testing effects.^{vii} An acute tolerance account [32,33] would predict poorer performance on the AL among intoxicated relative to sober individuals, but sober-equivalent—or, at least, significantly improved—performance among intoxicated individuals on the DL. Instead, intoxicated individuals performed more poorly at both time-points relative to their sober counterparts. Furthermore, individuals tested only on the DL exhibited poorer performance on the DL than counterparts in the AL/DL condition, but that deficit did not differ between alcohol and no-alcohol groups. Thus, repeated testing effects were present in sober and intoxicated states, albeit attenuated somewhat in the latter. Taken together, our findings indicate that at a dose level sufficient to produce a peak BAC of 0.085 g% within 30 minutes, alcohol's acute effect on WM updating appears to manifest as attenuation of improvements in performance otherwise experienced upon repeated testing.^{viii}

The current findings should be considered in light of the study's limitations. First, although a relatively large dose was administered, within-person impairment and acute tolerance effects were not observed. One possible explanation is that participants' typical peak BAC impacted the opportunity to observe alcohol-induced impairment and, thus, acute tolerance. Arguing against this possibility, the latent mean pattern and the test for acute tolerance were unchanged by adjustment for between-person differences in typical alcohol use (a proxy for between-person differences in typical peak BAC; see Tables S19–20). Nonetheless, acute impairment and acute tolerance still might be observed at higher doses characterizing typical drinking experiences for high-intensity drinkers (e.g. ≥ 0.10 g/kg) [22,67]. Secondly, the tasks used here relied upon the same WM updating subprocesses (i.e. retrieval and substitution, but not transformation) [58], and the study was not designed for subprocess dissociation, so it remains to be seen whether different subprocesses are similarly (un)affected by alcohol. Thirdly, WM updating tasks were scored for accuracy but not response time (RT)—largely because they are not structured as RT tasks. Acute tolerance effects are observed more readily on response activation (indexed by RT) than mnemonic (indexed by accuracy) processes

[34,35,71]. However, alcohol-induced cognitive or psychomotor slowing is not unique to WM updating tasks, and its presence would not change the interpretation of the current findings. Fourthly, acute alcohol can affect cognitive abilities differently depending upon between-person differences in baseline (i.e. sober state) ability [27 52,53,72]. In some cases, higher baselines may 'buffer' against acute insult [73]. Alternatively, individuals with higher sober state ability may have 'more to lose' from acute insult and could regress to population-mean levels of EF when intoxicated [74,75]. Regression analyses (see the Supporting information) found no evidence that baseline ability moderated the acute effect of alcohol at the level of each task (Supporting information, Tables S3, S12). Nonetheless, latent variable models examining moderation were not viable, so it remains unclear whether sober state WM updating ability could moderate the acute effect of alcohol on the common variance across WM updating tasks. Finally, regression analyses, repeated-measures ANOVAs and within-subject AL (baseline) and DL (baseline) difference score analyses (see Supporting information) converged with the latent WM updating analyses, but also suggested that performance on the verbal WM tasks (keep track and letter memory) adhered more closely to the pattern of effects detected in the latent WM updating analyses than did performance on the visuospatial WM task (spatial 2-back). Moreover, those supplemental analyses suggested that alcohol attenuated performance improvements more strongly in the verbal WM tasks compared to the visuospatial WM task. Differential sensitivity of verbal versus visuospatial WM to acute effects of alcohol is consistent with findings from a recent literature review [12], but remains to be verified in a future study that can model WM subtypes (e.g. one using multiple verbal and visuospatial tasks).

Our findings have broader implications for understanding the acute effects of alcohol on EF and their implications for drinking-related negative consequences. Contemporary models emphasize 'unity and diversity' in EF [76], with unity captured by a common factor onto which tasks from all facets load, and diversity captured by residual shifting- and WM updating-specific factors [5,6,77]. It is possible that alcohol's impairment of different facets of EF reflects acute effects on the common factor.^{ix} This possibility is bolstered by congruence between the current findings and

^{vii}Improved WM updating performance at DL compared to AL could also be due to lower BrAC at the start of DL compared to AL testing in session 2. Additionally, the Mellanby method for acute tolerance measurement requires testing the acute effect of alcohol at the same BAC on AL and DL [32,33]. Consequently, we identified a subset of individuals in the alcohol + A/D cell of the experiment for whom BrAC at the start of AL and DL testing were statistically equivalent (≤ 10 mg% Δ), and repeated both the alcohol versus no alcohol multi-group latent variable modeling of WM updating as well as the hierarchical regressions and ANOVAs of performance in each WM updating task. These sensitivity analyses are presented in Supporting information. Results were unchanged.

^{viii}Our study is unable to determine the reason for repeated testing-related increases in WM updating performance. One possibility is that repeated testing increases familiarity with the task which, in turn, decreases its difficulty. A second possibility is that repeated testing produces short- and long-term enhancements of this cognitive ability, akin to a practice or training effect. A third possibility is that participants develop better task performance strategies [37,38].

^{ix}Given that there is no inhibition-specific factor when variance common across inhibition, shifting and WM updating tasks are accounted for, acute effects of alcohol on inhibition tasks would be subsumed as acute effects on the common EF factor. This would suggest that the oft-reported 'disinhibiting' acute effects of alcohol are more appropriately viewed as acute effects of alcohol on EF more broadly. This possibility remains to be tested directly.

findings of our previous work in independent samples testing acute alcohol effects on inhibition [52] and shifting [53]. Testing this idea in future studies requires modeling alcohol's acute effects on multiple EF facets, as well as on common EF, in the same individuals.

Although alcohol prevented the practice effects observed in the control and placebo conditions, it did not produce an absolute decrease in WM updating performance in our study. Such a decrease might be found in future studies employing experimental designs that, unlike ours, do not aim to evaluate potential practice effects but rather to overcome them (e.g. by having participants practice the tasks until asymptotic performance before beverage administration). Alcohol-induced deficits in WM updating performance could encourage excessive drinking, either through failure to keep track of drinks consumed as a drinking episode unfolds or through failure to maintain personal drinking reduction goals. Either process could contribute to the experience of negative consequences, including the potential to prolong or worsen AUD. These deficits may also limit the extent or duration of an individual's benefit from interventions such as motivational interviewing [78] that rely upon individuals maintaining awareness of set goals (e.g. drinking reduction) and implementing behavioral strategies to attain them (e.g. decreasing drinking episode frequency and the number of drinks consumed within episodes).

In conclusion, the acute effects of alcohol on WM updating attenuated performance improvement otherwise experienced upon repeated testing. Future studies should examine not only the acute effect of alcohol on other EF facets (e.g. access to, or strategic retrieval of, information in long-term memory), but also the extent to which similarity in the acute effect of alcohol on different EF facets can be accounted for by an acute effect of alcohol on the theorized common factor in EF [5,6,77]. Characterizing the acute effects of alcohol on EF is an important step towards understanding the cognitive-behavioral mechanisms that underlie problematic alcohol use.

Declaration of interests

The authors have no conflicts of interest to declare.

Acknowledgements

Funding and support for this work were provided by NIH grants P60 AA011998 (B.D.B., K.J.S., P.K.W., A.M., N.C.) and R01 AA025451 (B.D.B.), T32 AA013526 (K.J.S., R. U.C.), and the University of Missouri College of Arts and Science Mission Enhancement Fund (A.L.W., R.U.C.), as well as an advanced training doctoral fellowship (SFRH/BD/9261/2013) awarded to J.S.M. by the Fundação para a Ciência e a Tecnologia, IP, from the POPH/FSE funding program of the Portuguese government. We thank J. Scott

Saults for compiling and processing the executive functions task data and Sarah N. Mitchell for her work collecting the data. Jorge S. Martins is now at the Yale University Interdisciplinary Stress Center.

Author contributions

Roberto Cofresi: Formal analysis; validation; visualization. **Ashley Watts:** Formal analysis; validation; visualization. **Jorge Martins:** Formal analysis; validation; visualization. **Phillip K. Wood:** Conceptualization; funding acquisition; methodology; resources; supervision. **Ken Sher:** Conceptualization; funding acquisition; methodology; resources; supervision. **Nelson Cowan:** Conceptualization; funding acquisition; methodology; resources; supervision. **Akira Miyake:** Conceptualization; funding acquisition; methodology; resources. **Bruce Bartholow:** Conceptualization; funding acquisition; investigation; methodology; project administration; resources; supervision.

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Supporting Information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Data S1. Supporting information.

Table S1. Summary of Analyses of Variance for Updating Performance Across Tasks at Baseline (Session 1).

Table S2. Summary of Analyses of Variance for Updating

Performance Within Each Task at Baseline (Session 1).

Table S3. Summary of Hierarchical Multiple Regression Analyses Predicting Post-Drinking Updating Performance as a Function of Baseline Performance, Beverage Variables and Sex and (for Descending Limb data) Whether Tasks Were Completed Only on Descending Limb.

Table S4. Summary of Analyses of Variance for Updating Performance Across Testing Occasions (Baseline, Descending Limb, and for A/D condition: Ascending Limb) as a Function of Beverage Variables Within Session 2 Task Completion Conditions.

Table S6. Model Parameters for the Strict Invariance Alcohol Effects Model.

Table S7. Group and Testing Occasion Comparisons.

Table S8. Task Completion condition Predicting Descending Limb Task Performance.

Table S9. WM Updating Latent Means from Alcohol Effects Model Across Groups and Testing Occasions With and Without Descending Limb Only Participants Included.

Table S10. Summary of Analyses of Variance for Updating Performance Across Tasks at Baseline (Session 1) Retaining in the Alcohol + A/D Group Only Individuals With No More Than 0.01 g% Difference in BrAC at Start of Ascending Limb (AL) and Descending Limb (DL) Procedures.

Table S11. Summary of Analyses of Variance for Updating Performance Within Each Task at Baseline (Session 1) Retaining in the Alcohol + A/D Group Only Individuals With No More Than 0.01 g% Difference in BrAC at Start of Ascending Limb (AL) and Descending Limb (DL) Procedures.

Table S12. Summary of Hierarchical Multiple Regression Analyses Predicting Post-Drinking Updating Performance as a Function of Baseline Performance, Beverage Variables and Sex and (for Descending Limb data) Whether Tasks Were Completed Only on Descending Limb, Retaining in the Alcohol + A/D Group Only Individuals With No More Than 0.01 g% Difference in BrAC at Start of Ascending Limb and Descending Limb Procedures.

Table S13. Summary of Analyses of Variance for Updating Performance Across Testing Occasions (Baseline, Ascending Limb, and Descending Limb) as a Function of Beverage Variables Within Session 2 Task Completion Condition, Retaining in the Alcohol Group Only Individuals With No More Than 0.01 g% Difference in BrAC at Start of Ascending Limb (AL) and Descending Limb (DL) Procedure.

Table S14. Model Fit Indices for Invariance Testing When Alcohol + A/D Experimental Cell Represents Only

Individuals With No More Than 0.01 g% Difference in BrAC at Start of Ascending Limb (AL) and Descending Limb (DL) Procedures.

Table S15. Latent WM Updating Means Across Groups and Testing Occasions When Alcohol + A/D Experimental Cell Represents Only Individuals With No More Than 0.01 g% Difference in BrAC at Start of Ascending Limb (AL) and Descending Limb (DL) Procedures.

Table S16. Group and Testing Occasion Comparisons When Alcohol + A/D Experimental Cell Represents Only Individuals With No More Than 0.01 g% Difference in BrAC at Start of Ascending Limb (AL) and Descending Limb (DL) Procedures.

Table S17. Task Completion Condition Predicting Descending Limb Task Performance When Alcohol + A/D Experimental Cell Represents Only Individuals With No More Than 0.01 g% Difference in BrAC at Start of Ascending Limb (AL) and Descending Limb (DL) Procedures.

Table S18. Latent WM Updating Means Across Groups and Testing Occasions Dropping Placebo Participants Who Gave Post-Experiment Estimate of 0 Standard Drink Equivalents Consumed During the Study.

Table S19. Latent WM Updating Means Across Groups and Testing Occasions Covarying for Recent Alcohol Use.

Table S20. Task Completion Condition Predicting Descending Limb Task Performance Covarying for Recent Alcohol Use.

Figure S1. Baseline Performance for Each WM Updating Task Across Beverage and Session 2 Task Completion Conditions.

Figure S2. Baseline Performance for Each WM Updating Task Across Beverage and Session 2 Task Completion Conditions Retaining in the Alcohol + A/D Condition Only Individuals With No More Than 0.01 g% Difference in BrAC at Start of Ascending Limb (AL) and Descending Limb (DL) Procedures.

Figure S3. Mean Accuracy Across Testing Occasions for Each Task as a Function of Beverage Condition and Session 2 Task Completion Condition Retaining in the Alcohol + A/D Condition Only Individuals With No More Than 0.01 g% Difference in BrAC at Start of Ascending Limb (AL) and Descending Limb (DL) Procedures.

Figure S4. Latent WM Updating Factor Across Testing Occasions for Alcohol v. No Alcohol Group Model Retaining in the Alcohol + A/D Condition Only Individuals With No More Than 0.01 g% Difference in BrAC at Start of Ascending Limb (AL) and Descending Limb (DL) Procedures.