The effect of acute alcohol consumption on meal memory and subsequent food intake: Two laboratory experiments

Thomas Gough\*a, Paul Christiansena, Abigail K. Rosea,b, & Charlotte A. Hardmana

aDepartment of Psychology, University of Liverpool, Eleanor Rathbone Building, Bedford Street South, Liverpool, L69 7ZA, UK.

**b**Liverpool Centre for Alcohol Research, Liverpool Health Partners, IC3, Liverpool Science Park, Liverpool, UK

\*Corresponding author; tomgough@liverpool.ac.uk

**Abstract**

Altering the quality of episodic meal memories has been shown to affect subsequent food intake. Acute alcohol consumption disrupts memory formation and produces short-term overeating. In two studies, we investigated whether alcohol consumption can affect meal-related memories and later food intake. Study 1 (N = 60, 50% male) investigated how consumption of an alcoholic drink (0.5 g/kg) prior to consumption of a lunch meal affected meal memory of that lunch, and later food intake, compared with a placebo-alcohol. Findings revealed that alcohol consumption did not impair meal memory, and did not affect subsequent food intake. Study 2 (N = 72, 50% male) investigated whether, due to alcohol’s retrograde facilitation effect (the enhancement of recall due to reduced interference at the point of exposure) consuming alcohol *after* consumption of a lunch meal could enhance meal memory, compared with when consumed before a lunch meal (both a dosage of 0.6 g/kg), and compared with consumption of a soft drink. Contrary to prediction, alcohol consumed after a lunch meal did not significantly increase meal memory. But, certain types of meal memory were impaired when alcohol was consumed before the meal, compared with consumption of a soft drink. Subsequent food intake did not differ between conditions. Taken together, findings suggest that alcohol intoxication can impair some forms of meal memory recall, likely due to disruption of memory formation during the encoding phase. However, there was no evidence that this impairment contributes towards alcohol-induced overeating.

Keywords: alcohol; episodic memory; appetite; food intake

List of abbreviations: AUDIT (Alcohol Use Disorders Identification Test), BAES (Biphasic Alcohol Effects Scales), BMI (body mass index), BrAC (breath alcohol concentration), DEBQ (Dutch Eating Behaviour Questionnaire), IAPS (International Affective Picture System), TLFB (Timeline Follow-back).

**1. Introduction**

A multitude of cognitive processes have been identified as factors which influence eating behaviour (Higgs & Spetter, 2018). Such factors include attention and memory for recent eating in determining food intake. A growing body of research has demonstrated that impairments to episodic memories relating to recently consumed food can alter subsequent food intake. For example, animal research has demonstrated that lesions to the hippocampal region results in hyperphagia and weight gain (Clifton, Vickers, & Somerville, 1998; Davidson, Kanoski, Walls, & Jarrard, 2005). More recently, an animal study by Hannapel et al. (2019) revealed that inhibition of glutamatergic neurons to dorsal and ventral hippocampal areas after consumption of a meal, leads to an increase in the amount of food consumed during a subsequent meal. Further, evidence from amnesic patients has demonstrated that individuals who have an impaired ability of reporting memories for recent eating also display evidence of overeating (Higgs, Williamson, Rotshtein, & Humphreys, 2008).

Notably, manipulating the quality of episodic meal-related memories also affects subsequent food intake. This has been investigated by either enhancing or impairing the quality of a meal memory. Research has shown that cueing memory for a recently consumed meal (i.e., lunch consumed that day) reduces subsequent food intake, relative to no cue and when cued of a lunch meal consumed the previous day (Higgs, 2002; Higgs, Williamson, & Attwood, 2008), suggesting that cueing of only very recent eating affects subsequent food intake. Similarly, many studies have investigated the effect of enhancing the level of attention placed towards a meal (specifically relating to the sensory properties of the food) on subsequent food intake, which has been suggested to increase meal memory. Findings are mixed, with some experiments showing that this increase in focused attention leads to a reduction in later food intake in both samples exclusively of women (Higgs & Donohoe, 2011; Robinson, Kersbergen, & Higgs, 2014) and a mixed gender sample (Seguias & Tapper, 2018). Other studies, however, have failed to show this same reduction in a mixed sex (Whitelock et al., 2018), a male-only sample (Whitelock et al., 2019) and most recently a female-only sample (Tapper & Seguias, 2020).

Other research has focused on the effect of impairing memories of a recently consumed meal on subsequent food intake. This has been investigated by taking attention away from a meal whilst eating by using distractors such as television viewing (Higgs & Woodward, 2009; Mittal, Stevenson, Oaten, & Miller, 2011) and playing computer games (Oldham-Cooper, Hardman, Nicoll, Rogers, & Brunstrom, 2010). These studies have demonstrated that distracted participants display poorer levels of recall for meal memory, and greater subsequent food intake, compared with participants who eat in the absence of a distractor. This impairment of episodic meal memory is argued to be due to disruption during the encoding phase of memory formation.

Acute consumption of alcohol has also been shown to impair processes of episodic memory, resulting in impaired delayed recall of stimuli when exposure or learning occurs shortly after alcohol consumption (Hashtroudi et al., 1984; Nilsson, Bäckman, & Karlsson, 1989; Söderlund, Parker, Schwartz, & Tulving, 2005). This is believed to occur due to alcohol-induced disruptions to activity in the CA1 region of the hippocampus (White, Matthews & Best, 2000; Zola-Morgan, Squire, & Amaral, 1986). To date, no studies have investigated how acute alcohol consumption can impair recall of recently consumed food.

Acute alcohol consumption has also been shown to increase short-term levels of food intake, relative to consumption of alcohol-free drinks (Caton, Ball, Ahern, & Hetherington, 2004; Caton, Marks, & Hetherington, 2005; Kwok et al., 2019; Yeomans, 2010). Several mechanisms are likely to contribute to alcohol’s effect on increased intake, such as impairment of inhibitory control (Christiansen, Rose, Randall-Smith, & Hardman, 2016) and enhancing the reward value of certain foods (Schrieks et al., 2015). Furthermore, biological factors are also likely to contribute towards elevated levels of food intake, as acute alcohol consumption produces hyperactivity of agouti-related protein neurons (Cains et al., 2017) and produces inhibition of leptin and GLP-1 hormones (Raben et al., 2003; Röjdmark et al., 2001). A currently unexplored, but potentially important mechanism of this increased food intake may come from disruptions to meal memory if an alcoholic drink is consumed before consumption of food.

2. Study 1

Overview

In Study 1, participants either 1) consumed a pre-load meal after consuming an alcoholic drink, or 2) consumed a pre-load meal, after consuming a placebo-alcohol drink. After a delay of 30 minutes, all participants were given *ad libitum* access to chocolate chip cookies and recalled details of the pre-load meal. We hypothesised that participants who consumed an alcoholic drink would show greater impairment of meal memory and greater *ad libitum* food intake, compared with participants who consumed an alcohol-free placebo.

**2.1 Method**

2.1.1 Participants

Sample size was determined from previous investigations examining the effect of distraction on meal memory and subsequent food intake. Oldham-Cooper and colleagues (2010) found an effect size of *d* = 0.68 for the comparison between undistracted and distracted individuals on food intake and an effect size of *d* = 0.67 for meal memory between these two conditions. In order to detect an effect size of *d* = 0.67 with 80% power at an alpha level of 5%, 58 participants were required. 60 participants were recruited to allow for any cases which may need to be excluded. 60 participants (male = 30) aged between 18 and 62 y (M = 24.5, SD = 10.1) took part, and were recruited through online and email advertisement, and word-of-mouth. Participants were eligible to take part if they had no history of food allergies or intolerances, were not vegetarian or vegan, and were regular consumers of alcohol (consuming at least 10 UK alcohol units per week - one UK unit = 8 g of alcohol). Participants were excluded if they had a current or past alcohol use or eating disorder, had a current or recent illness that may increase sensitivity to alcohol (e.g., cold and flu), were taking medication that may be affected by alcohol, and were currently breastfeeding or pregnant. All participants provided written informed consent to participate in the experiment, which was approved by the University of Liverpool Health and Life Sciences Research Ethics Committee. Participants were reimbursed through either course credits or received a £10 shopping voucher.

2.1.2. Design

The study used a between-subjects, single-blind randomised design with drink type (alcoholic drink, placebo-alcohol) as an independent variable. The dependent variables were free recall and serial recall of the lunch meal, general memory recall, *ad libitum* intake (kcal) and total intake (test drink and *ad libitum* kcal combined).

2.1.3. Measures

*Beverage Preparation and Administration.* The present study used an alcohol dosage of 0.5 grams of alcohol per kilogram of participant bodyweight (g/kg) (35.76 grams of alcohol for a participant weighing 70 kg). The alcoholic drink contained vodka (Smirnoff Red, 37.5% ABV) up to a maximum of 200 ml of vodka (1 g of vodka = 2.08 kcal) and was mixed with chilled diet lemonade in the ratio one-part vodka to three parts diet lemonade. The placebo drink consisted of diet lemonade only; a vodka mist was sprayed on the surface of the drink to create the impression that it contained alcohol.

*Lunch meal.*

The lunch meal used was similar to that used in a previous study (Oldham-Cooper et al., 2010). All lunch items were manufactured by Tesco’s Ltd except for the potato chip snack (Hula Hoops; KP Snacks Ltd, Ashby-de-la-Zouch, United Kingdom). Nine foods were served one-by-one on separate plates in 90-second intervals. The foods were served in this way in order to match eating duration across foods and participants, and to measure how well participants remembered the order of the nine foods.

Table 1. Lunch items served, in presentation order.

|  |  |  |
| --- | --- | --- |
| **Food Item** | **Amount (grams)** | **Energy per portion (kcal)** |
| Cheese twists | 8 | 41 |
| Ham sandwicha | 35 | 94 |
| Carrot batons | 25 | 11 |
| Mini Cornish pasty | 30 | 104 |
| Cheese sandwichb | 35 | 125 |
| Sausage Roll | 11 | 34 |
| 8 Cherry tomatoes | 71 | 14 |
| Scotch egg | 12 | 28 |
| 15 Potato chip snacks | 13 | 64 |
| Total | 239 | 515 |

a Comprising half a slice of Tesco White Medium Bread (20 g), 5 g of Tesco Butterpak Spreadable Butter, 10 g of Tesco Everyday Value Cooked Ham.

b Comprising half a slice of Tesco White Medium Bread (20 g), 5 g of Tesco Butterpak Spreadable Butter, 10 g of Tesco Everyday Value Grated Cheddar.

*Taste Test Preparation***.** The test meal consisted of a 200 g serving of Maryland chocolate chip cookies (487 kcal/100g). The test meal was also served with a 250 g serving of water. Cookies were broken into smaller pieces so that participant could not easily monitor the amount consumed (Higgs & Woodward, 2009).

*Free recall task:* Participants were required to recall the nine food items they consumed during the lunch meal in no specific order. Using pen and paper, participants wrote down as many of the lunch items as they could remember. A list of accepted answers are included in the supplementary materials (Table S3). Two independent reviewers rated whether participants correctly recalled each of the nine lunch items, with an agreement of 94.45%. Disagreements in scoring was resolved by the lead author.

*Serial order recall task:* Participants were asked to recall the specific order in which the nine food items were presented.

*Meal vividness rating:* Participants were asked on a 100 mm VAS ‘How vividly can you remember the lunch meal you ate earlier?’ Anchored scores were ‘Not At All’ and ‘Extremely’.

*General Memory Measure:* General memory performance was also measured. Participants were shown a wordlist consisting of 6 capital cities and 6 countries to memorise.

*Dutch Eating Behavior Questionnaire.* The Dutch Eating Behaviour Questionnaire (DEBQ; van Strien, Frijters, Bergers, & Defares, 1986) is a 33-item questionnaire which measured eating styles associated with being overweight. The three subscales are restraint (ωt = .93), emotional eating (ωt = .96), and external eating (ωt = .90).

*Timeline Follow Back.* In the Timeline Follow Back (TLFB; Sobell & Sobell, 1992), participants estimated the number of alcohol units consumed over the past 7 days, measuring typical drinking habits.

*Alcohol Use Disorders Identification Test.*The Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, de la Fuente, & Grant, 1993) is a 10-item questionnaire assessing hazardous drinking. Scores range between 0 and 40, with scores of ≥ 8 indicating hazardous alcohol use (ωt = .84).

*Snack Urge Scale*.The Snack Urge Scale (SUS; Hardman et al., 2015) comprises four items which measured expected liking, desire to consume, craving, and difficulty to resist chocolate chip cookies. Each item was measured using a 100 mm VAS (‘Not at all’ – ‘Extremely’) and combined as a total snack urge score (maximum score of 400).

*Appetite Ratings.*(AR; Blundell et al., 2010) of hunger (I feel hungry) and fullness (My stomach feels full) were measured using a 100 mm VAS (‘Not at all’ – ‘Extremely’). These scores were combined (hunger added to the inverse score of fullness) and reported as a single appetite rating (maximum score of 200).

*Biphasic Alcohol Effects Scale.* (BAES; Martin et al., 1993). The BAES is a 14-item scale which is comprised of two 7- item sub-scales, measuring the sedative and stimulating effects of alcohol, respectively. Participants were required to rate the extent to which they are experiencing both sedative (e.g., down, inactive) and stimulatory feelings (e.g., elated, energized) at the present moment on a 10-point scale, anchored scores are ‘Not at all’ and ‘Extremely’.

2.1.4. Procedure

Test sessions took place between 12:00 – 18:00 on weekdays in the Department of Psychology on the University of Liverpool campus. Sessions lasted approximately 120 minutes. The study was advertised as a study investigating ‘alcohol’s effect on memory and taste perception’. Participants were told that memory performance would be measured but were not told that memory of the lunch meal would be assessed. Prior to the beginning of the session, all participants were asked to consume a light meal not high in fat approximately an hour before the beginning of the test session. Upon arrival, participants were presented with the information sheet and provided informed consent. Participants were asked to report when they had last eaten and what they had consumed, before being breathalysed (all had a BrAC of 0.00). Participants then completed a medical history questionnaire to assess whether they had any food allergies. Height and weight measurements were taken in order to calculate the alcohol dosage. Next, baseline appetite ratings and snack urge scale ratings were recorded, followed by completion of the DEBQ, AUDIT, TLFB and baseline BAES. Participants then consumed the test drink. They were required to consume the drink within 10 minutes, followed by a 10-minute absorption period where participants sat quietly. Next, a second breathalyser measure was taken, followed by a second set of BAES, appetite and snack urge ratings. Next, participants consumed their lunch meal. Afterwards, participants completed a third set of appetite, snack urge and BAES ratings. Participants were then presented with the word list for the general memory measure to memorise for one minute. This was measured in order to observe whether alcohol consumption successfully impaired general memory performance, as would be expected. Afterwards, participants took a 30-minute break during which, they were required to stay in the test room and to abstain from eating. Participants were offered light reading material during this time. After the break, participants were given one minute to recall items from the word list, before completing another breathalyser measure and appetite and snack urge ratings. Participants then completed the taste test for 10 minutes. During this period, participants were asked to taste the test food as much or as little as they wanted, and to provide ratings based on certain characteristics of the foods (data was not analysed). Afterwards, BAES ratings were taken again followed by a final breathalyser measure (see Table S2 of the supplementary materials for BrAc scores across both conditions). Participants were then given three minutes to complete the free recall lunch item task, followed by three minutes to complete the serial order recall task. The lunch memory measures were completed after the taste test to avoid cueing participants of their lunch meal. Participants then completed the vividness rating, and an awareness check. Finally, participants were fully debriefed and reimbursed for their time.

Table 2. Overview of the procedure. With approximate timings and durations of each task

|  |  |  |
| --- | --- | --- |
| Task/Measure | Start Time (Minutes Post-arrival) | Duration (in minutes) |
| Information Sheet | 0 | 1 |
| Consent Form | 1 | 2 |
| Baseline breathalyser measure | 3 | 1 |
| Medical History Questionnaire | 4 | 3 |
| Height and Weight Measurement | 7 | 2 |
| Baseline Appetite Ratings | 9 | 0.5 |
| First Snack Urge Questionnaire | 9.5 | 0.5 |
| Dutch Eating Behaviour Questionnaire | 10 | 3 |
| Alcohol Use Disorders Identification Test | 13 | 1 |
| Timeline Follow-back Questionnaire | 14 | 2 |
| Baseline BAES | 16 | 1 |
| Consumption of Drink | 17 | 10 |
| Absorption Period | 27 | 10 |
| Second Breathalyser Measure | 37 | 1 |
| Lunch Meal | 38 | 13.5 |
| Post-lunch Appetite Ratings | 51.5 | 0.5 |
| Second Snack Urge Questionnaire | 52 | 0.5 |
| Second BAES | 52.5 | 1 |
| Memorise Word List | 53.5 | 1 |
| Break | 54.5 | 30 |
| Word List Recall | 84.5 | 1 |
| Third Breathalyser Measure | 85.5 | 1 |
| Third Hunger, Fullness, & Thirst Ratings | 86.5 | 0.5 |
| Third Snack Urge Questionnaire | 87 | 1 |
| Third BAES | 88 | 1 |
| Taste Test | 89 | 10 |
| Fourth BAES | 99 | 1 |
| Fourth Breathalyser Measure | 100 | 1 |
| Free Recall Task | 101 | 3 |
| Serial Recall Task | 104 | 3 |
| Vividness Rating | 107 | 0.5 |
| Awareness Check | 107.5 | 2 |
| Debrief Sheet | 109.5 | 2 |
| Reimbursement | 111.5 | 2 |

2.1.5. Data Analysis

Analysis was performed using SPSS 25 (IBM Corporation, Armonk, NY, USA). We performed independent t-tests to test for any significant differences between conditions in the meal memory measures, general memory measure, food calorie intake and total calorie intake (cookie and drink calories combined). Mixed ANOVAs were conducted to observe differences between drink conditions and differences across time for appetite ratings, snack urge ratings and BAES stimulation and sedation ratings (see findings of snack urge ratings and BAES ratings in the supplementary materials). Four participants did not consume all of the lunch meal. A sensitivity analysis revealed that removing these participants from all analyses did not affect the statistical significance of the results. The method and analysis strategy for Study 1 was pre-registered on the Open Science Framework (see protocol here: osf.io/mbxs8/).

**2.2. Results**

2.2.1. Participant characteristics

Means and standard deviations are displayed in Table 3.

Table 3. Sample characteristics and baseline scores, split by drink condition (mean ± SD)

|  |  |  |
| --- | --- | --- |
|  | Alcoholic drink (N = 30) | Placebo-Alcohol (N = 30) |
| Age (y) | 23 ± 9.7 | 25.9 ± 10.5 |
| AUDIT (out of 40) | 10.8 ± 5 | 11 ± 5.4 |
| BMI (kg/m2) | 23.6 ± 3.8 | 25.8 ± 5.3 |
| DEBQ Emotional | 2.4 ± 0.9 | 2.5 ± 0.7 |
| DEBQ External | 3.4 ± 0.6 | 3.3 ± 0.7 |
| DEBQ Restraint | 2.4 ± 0.9 | 2.3 ± 0.7 |
| 7-day TLFB (alcohol units) | 17 ± 12 | 16 ± 11 |
| Baseline Appetite (out of 200) | 84 ± 42 | 73 ± 37 |
| Baseline Snack Urge (out of 400) | 205 ± 83 | 177 ± 64 |
| Baseline Sedation BAES (out of 49) | 18 ± 11 | 16 ± 12 |
| Baseline Stimulation BAES (out of 49) | 33 ± 11 | 34 ± 8 |

AUDIT = Alcohol Use Disorders Identification Test; BMI = Body Mass Index; DEBQ = Dutch Eating Behaviour Questionnaire; TLFB = Timeline Follow-back; BAES = Biphasic alcohol effects scale.

2.2.2 Memory Measures (Table 4)

Table 4. Scores on outcome measures, split by drink condition (mean ± SD)

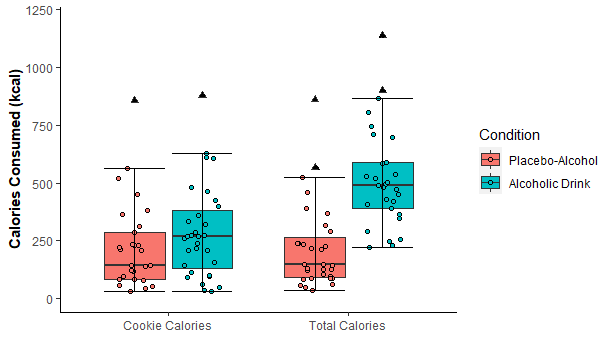
|  |  |  |
| --- | --- | --- |
|  | Alcoholic Drink | Placebo-Alcohol |
| Vividness Rating (out of 100) | 72 ± 15 | 73 ± 12 |
| General Memory Recall (out of 12) | 7.3 ± 2.1 | 8.2 ± 1.9 |
| Lunch Item Recall (out of 9) | 7.4 ± 1.6 | 8.1 ± 1.4 |
| Serial Order Recall (out of 9) | 4.6 ± 2.2 | 5.1 ± 2.3 |
| Cookie intake (kcal) | 285 ± 205 | 219 ± 186 |
| Total intake (drink and cookies combined; kcal) | 514 ± 218\* | 224 ± 186\* |

\*p < .05

There was no significant difference between drink conditions for vividness ratings *t*(58) = .34, *p* = .735, *d* = 0.09, general memory recall *t*(58) = 1.68, *p* = .098, *d* = 0.43, for serial-order recall *t*(58) = 0.92, *p* = .362, *d* = 0.24, or for the free-recall lunch item task *t*(58) = 1.66, *p*  = .103, *d* = 0.43.

2.2.3. Calorie Measures (Figure 1)

There was no significant difference between drink conditions on the amount of calories consumed during the taste test *t*(58) = 1.31, *p* = .196, *d* = 0.34. However participants in the alcohol drink condition consumed significantly more calories than the placebo-alcohol condition when combining calories from both the drink and cookies consumed *t*(58) = 5.55, *p* < .001, *d* = 1.43.



**\***

Figure 1. Boxplot displaying individual data points for number of calories consumed during the ad libitum taste test (cookie calories) and combined with calories consumed from the test drink (total calories), split by condition. \*p < .001. Triangles represent outliers.

2.2.4. Appetite Ratings

A 2 (drink; placebo-alcohol, alcoholic drink) x 4 (time; baseline, post-drink, post-lunch, pre-taste test) mixed ANOVA was conducted with drink as a between-subjects factor and time as a within-subjects. Mauchly’s test indicated that the assumption of sphericity had been violated for the main effect of time*χ*2 (5) = 23.25, *p* < .001, Greenhouse-Geisser corrected tests are reported (ε =.828). The analysis revealed a significant main effect of time *F*(2.48, 141.62) = 78.83, *p* < .001, *ηp2* = .58. Bonferroni pairwise comparisons revealed that baseline appetite ratings were significantly higher than post-lunch (*p* < .001; mean difference = 51; 95% CI [37, 66]) and pre-taste test ratings (*p* < .001; mean difference = 39; 95% CI [28, 51]). Post-drink ratings were also significantly higher than post-lunch (*p* < .001; mean difference = 63; 95% CI [49, 78]) and pre-taste test ratings (*p* < .001; mean difference = 51; 95% CI [36, 66]). Post-lunch ratings were shown to be significantly lower than pre-taste test ratings (*p* = .006; mean difference = 12; 95% CI [-21, -3]). The analysis also revealed a nonsignificant main effect of drink type *F*(1, 57) = 2.67, *p* = .108, *ηp2* = .05 and a nonsignificant drink type x time interaction *F*(3, 171) = .78, *p* = .504, *ηp2* = .01. See supplementary materials for full list of means and standard deviations of appetite ratings at each time point, split by condition.

**2.3. Interim discussion**

Study 1 found that consumption of an alcoholic drink did not significantly affect performance on the free-recall food memory task, serial-recall task or ratings of meal vividness, compared with those who consumed the placebo-alcohol. Therefore, our prediction that alcohol consumption can impair meal memory is rejected. Findings also revealed that consumption of the alcoholic drink did not significantly decrease performance on the general memory task, nor did it significantly alter *ad libitum* consumption of cookies. This goes against our prediction that alcohol consumption would decrease general recall and increase food intake.

One explanation for failing to find a significant difference in all memory measures may have been due to the alcohol dosage used. Previous studies investigating the effect of alcohol intoxication on delayed recall typically use higher doses than the one used in the present experiment (1 ml/kg - Söderlund et al., 2005; 0.66 ml/kg - Sutker et al., 1983). This is important because research has shown that memory impairment can occur in a dose-dependent manner (Bisby, Leitz, Morgan, & Curran, 2010). Furthermore, by minimising alcohol expectancy effects between the two conditions by using an alcohol-free placebo, the difference in recall may have been smaller than in a more naturalistic context where individuals are aware when a drink contains zero alcohol. However, previous research suggests alcohol expectancy has a small effect on information processing (Hull & Bond, 1986).

It is plausible that the aspects of meal memory measured in Study 1 may not be relevant to subsequent food intake. Other studies have used measures which focus on recalling the quantity of a lunch meal (Mittal et al. 2011; Whitelock et al., 2018; Whitelock et al., 2019) and recalling feelings relating to interoceptive states, such as hunger (Brunstrom et al., 2012; Whitelock et al., 2018; Whitelock et al., 2019). These may be more important and relevant components of meal memory which help guide subsequent eating episodes, as compared with the current measures used.

Study 1 has a few limitations. Firstly, participants in the alcohol condition completed the recall measures when they were still intoxicated (BrAc > 0, see Table S2 of the supplementary materials for BrAc ratings across both conditions). It is therefore not possible to confirm whether impairments of memory performance were the result of disruption during the encoding or the retrieval phase, this limitation could be overcome by incorporating a longer delay between alcohol consumption and subsequent recall. Furthermore, the present study was not able to isolate the effect of impaired memory on subsequent food intake. Alcohol intoxication influences many factors which can increase food consumption, such as inhibitory control (Christiansen et al., 2016) and reward processing (Schrieks et al., 2015). As participants were still intoxicated during the taste test, the two conditions were unmatched on a number of confounding factors. Given these issues, Study 2 looked to build upon the current findings and to address the mentioned limitations.

**3. Study 2**

Overview

Study 2 investigated whether other forms of meal memory may be disrupted by alcohol intoxication and alter later food intake. We chose to measure participants’ visual memory of the portion size of a meal consumed, vividness of a meal and memory of satiety experienced after a meal. Furthermore, we used a greater alcohol dosage - 0.6 g/kg - and told participants in the alcohol-free condition that they would be consuming a soft drink. This was done to produce a clearer measure of alcohol’s effect (combining both pharmacological and expectancy effects) on delayed recall. We also incorporated a longer interval between consumption of the test drink and subsequent recall in order to allow for participants to have a lower alcohol level at the point of recall.

An additional aim was to investigate whether alcohol consumed *after* a lunch meal may in fact *enhance* meal memory. The ability for alcohol to influence episodic memories may depend on whether information is presented before or after consuming alcohol. As previously mentioned, research has shown that alcohol impairs learning when intoxication occurs at the encoding stage (when alcohol is consumed before information is presented). However, alcohol can *enhance* learning when intoxication occurs *after* the encoding stage and during consolidation (when alcohol is consumed after information is presented; Knowles & Duka, 2004; Parker et al., 1980; Weafer, Gallo, & De Wit, 2016). For example, Weafer, Gallo and De Wit (2016) found that alcohol consumed after presentation of stimuli significantly improved recall compared with consumption of a placebo-alcohol, suggesting that alcohol consumption can aid consolidation of recent memories and boost later recall. This phenomenon, termed ‘retrograde facilitation’ is believed to occur due to the ability of alcohol intoxication to protect memories formed prior to alcohol consumption by impairing the ability to form new memories, and therefore reduce interference once alcohol has been consumed (Wixted, 2005). Alcohol consumed after a meal may therefore increase the quality of episodic memories relating to the meal, compared with when alcohol is consumed before the meal, and when alcohol is not consumed.

To investigate the effect of the timing of the alcoholic drink in relation to the meal, participants completed two sessions. In session one, all participants consumed a soft drink followed by a lunch meal. After a break, participants were given *ad libitum* access to chocolate chip cookies and completed a general memory task – these two tasks were used as baseline measures of food intake and general memory recall. This session was included in order to 1) provide a baseline score of food intake which was needed for the data analysis (see section 3.1.5), as this allowed us to control for between-subject variance of food intake when measuring differences in food intake between conditions, as has been done in previous research (e.g., Gadah, Brunstrom, & Rogers, 2016), and 2) to record baseline general memory performance in case differences between conditions may exist. In session two, participants were assigned to one of three conditions and either 1) consumed an alcohol-free drink before consuming a lunch meal (soft drink condition), 2) consumed an alcoholic drink before consuming a lunch meal (pre-meal drink condition), or 3) consumed an alcoholic drink after consuming a lunch meal (post-meal drink condition). After a break (2 hours long in the post-meal drink condition, 2.5 hours long in the soft-drink and pre-meal drink condition), participants were given *ad libitum* access to chocolate chip cookies and meal memory recall was measured. We predicted that meal memory would be greatest in condition three (i.e. post-meal drink condition)and lowest in condition two (pre-meal drink condition), and therefore we also predicted that *ad libitum* food intake would be lowest in condition three and greatest in condition two. We also tested for general memory performance of words and predicted that recall of words presented before the test drink would be greater in the two alcohol conditions as compared with the soft drink condition. Conversely, we predicted that recall of words presented after the test drink would be greater in the soft drink condition, compared with the two alcohol conditions.

**3.1 Method**

3.1.1 Participants

Sample size was calculated based from previous research examining the enhanced effect on memory consolidation after alcohol consumption. A previous study found that alcohol consumption after viewing neutral stimuli during consolidation produced a large effect on memory recall (Weafer, Gallo & De Wit, 2016; *d* = 0.79). In order to detect a comparable effect with 80% power, α = 0.05, 66 participants were required. We aimed to recruit 72 participants which would allow us to detect a large effect size (*d* = 0.76) at 80% power, α = 0.05. To power for food intake, the design controlled for between-subject differences in food intake by incorporating a baseline session, whereby *ad libitum* food intake was measured and included as a covariate when comparing differences in food intake. This analysis strategy has been used in previous research (e.g., Gadah, Brunstrom, & Rogers, 2016) and was used in the present study in order to reduce the between-subjects variance of food intake without implementing a within-subjects design. This is because the implementation of a within-subjects design would likely produce order effects relating to the meal memory recall measures. With 72 participants, we were powered to detect an effect size of *d* = 0.5 for differences in food intake at 80% power, α = 0.05. In total, 73 participants were recruited due to one participant failing to attend the second session. After excluding this participant, 72 (male = 36) participants aged between 18 and 60 y (M = 24.31, SD = 9.51) were included in all data analyses. Participants were recruited through online and email advertisement, and word-of-mouth. The inclusion criteria were the same as in Study 1, except participants were required to typically consume at least 15 UK alcohol units per week. This was increased due to the larger alcohol dosage implemented in Study 2. All participants provided written informed consent to participate in the experiment, which was approved by the University of Liverpool Health and Life Sciences Research Ethics Committee. Participants were reimbursed through either course credits or a £20 shopping voucher.

3.1.2. Design

The study used a between-subjects, single-blind randomised design with drink type (soft drink, pre-meal drink, and post-meal drink) as an independent variable. All participants attended two sessions. In the first (baseline) session, participants completed the same procedure and consumed a soft drink, followed by a lunch meal and then an *ad libitum* taste test. A week later, participants then completed the procedure in their randomly assigned condition. The dependent variables in session 2 were the number of calories consumed during the *ad libitum* taste test, total calories consumed (taste test calories and drink calories combined), meal vividness rating, memory for satiety, visual memory of the portion size of the lunch meal, and general memory recall.

3.1.3. Measures

*Beverage Preparation and Administration.* The present study used an alcohol dosage of 0.6 g/kg (42.96 grams of alcohol for a participant weighing 70 kg). The alcoholic drink contained vodka (Smirnoff Red, 37.5% ABV) up to a maximum of 200 ml of vodka (1 g of vodka = 2.08 kcal). The drink was mixed with chilled diet lemonade in the ratio one-part vodka to three parts diet lemonade. The soft drink consisted of diet lemonade only, and the volume was matched for body weight such that participants weighing the same would consume the same total volume of liquid in either condition. All participants were told that they were consuming an alcohol-free diet lemonade drink during the first session, as were participants who were in the soft drink condition in session two.

*Lunchtime meal.*

Due to a manufactory change in the caloric content of the lunch meal partway through the study, 13 participants consumed a lunch meal consisting of a 262.39 g serving of cheese and tomato pasta salad (Tesco UK). The remaining 59 participants consumed a 250.93 g serving of the same Tesco brand cheese and tomato pasta salad to ensure that all lunches were matched on caloric content (1.79 kcal per gram; 450 kcal per serving). The lunch meal was divided into six equicaloric portions, served one at a time in 90-second intervals to control for meal duration. A 250 g serving of water was provided with the lunch meal. The same lunch meal was served in both session 1 and 2.

*Taste Test Preparation***:** The same as in Study 1.

*Meal vividness rating (Session 2):* The same as in Study 1.

*Picture presentations (Sessions 1 and 2):* To bolster the cover story and to measure general memory performance, participants were required to provide visual ratings of different images in both sessions 1 and 2. Participants were exposed to one set of images in session 1, and two sets in session 2 (one before consumption of the test drink and one after). Pictorial stimuli were taken from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 1997). Images across the three picture sets consisted of objects, animals and people. Each presentation consisted of 24 images, each presented with a text label below which provided a name of the image (e.g., an image of an astronaut would have the text label ‘Astronaut’ displayed below it). All three presentations were matched on valence and arousal ratings (scored out of 9): session 1 picture set: valence = 5.91; arousal = 3.95, session 2 picture set A: valence = 5.99; arousal = 3.71, session 2 picture set B: valence = 5.95; arousal = 3.89. The order of picture sets in session 2 were counterbalanced. For each presentation, images were presented alone with the text label for 5 seconds. Afterwards, the image and text label were presented on the left hand-side of the screen, and three rating scales on the right-hand side, this stayed on screen for 15 seconds. Participants were asked to rate the content of the image on three scales – ‘calm/excited’, ‘unpleasant/pleasant’, ‘not dominant/dominant’ (data not analysed).

*General memory recall (Sessions 1 and 2):* A surprise free recall based on the picture presentation was implemented in both sessions. The surprise element ensured consistency between the general and meal memory recall tasks. Participants were given 5 minutes to recall as many of the picture text labels as they could remember from the session 1 picture set at the end of the first session, and from both the session 2 set A and B picture presentations at the end of the second session. Participants were told to recall the exact text of each label in any order they wished, and to avoid recalling any related words or synonyms. A response was marked as correct if it was the same text, with the exception of pluralising the word or recalling the text label correctly, but with incorrect spelling. The dependent variable was the number of text labels correctly recalled for each presentation set.

*Dutch Eating Behavior Questionnaire (Session 1):* The same as in Study 1. The three subscales are restraint (ωt = .96), emotional eating (ωt =.95), and external eating (ωt =.90).

*Expected Satiety Memory measure (Session 2):* To measure memory for satiety, participants completed a computerised task in which they were asked to select the portion size of 18 meal foods to indicate the amount of food that would be required to produce the sensation of fullness that they experienced after lunch; adapted from Brunstrom, Shakeshaft, and Scott-Samuel (2008). Food pictures started at 20 kcal and increased in 20 kcal increments up 1000 kcal. Participants completed this measure twice in session 2: once immediately after consuming their lunch meal and again at the end of the test session. The outcome measure for this task was the absolute score of the average of the kcal differences of the portion sizes selected between the two measures. A score of zero means there was no difference in portion size selection between the two time points, indicating perfect memory, larger scores indicate poorer memory. Participants were also asked whether they had consumed each of the food items to check for familiarity (referred to as the familiarity task in the procedure section).

*Visual memory for portion size (Session 2):* Participants were presented with a large bowl of pasta salad (twice the amount of the same pasta salad they were served for lunch). Participants were asked to self-serve the amount of food which they believe they were served earlier for lunch, from the bowl onto a plate. The outcome measure was the difference between the amount of pasta self-served and the actual amount of pasta served at lunch, converted into an error percentage (a percentage of zero indicating zero difference). A larger error percentage indicates a greater difference between the amount of pasta self-served and the actual amount served for lunch, indicating poorer memory for portion size.

*Timeline Follow Back (Session 1):* The same as in Study 1.

*Alcohol Use Disorders Identification Test (Session 1):*The same as in Study 1. (ωt = .82).

*Snack Urge Scale (Session 2):*The same as in Study 1

*Appetite Ratings (Session 2):*The same as in Study 1.

3.1.4. Procedure

Test sessions took place between 13:15 and 18:30 on weekdays in the Department of Psychology on the University of Liverpool campus. The study was advertised as investigating ‘alcohol’s effect on visual and taste perception’. Prior to both session 1 and 2, participants were told to consume a light meal approximately an hour before the beginning of each session. Upon arrival of session 1, participants were presented with the information sheet and provided informed consent. Participants were then asked to report when they had last eaten and what they had consumed. Participants then completed a medical history questionnaire to assess whether they had any food allergies. Height and weight measurements were then taken in order to calculate the volume of drink to be consumed. Next, participants consumed the test drink (a soft drink for all participants) in three separate servings in 5-minute intervals. Afterwards, a 10-minute absorption period was completed whereby participants sat quietly. Next, participants consumed the test meal, and then completed the picture presentation task. Afterwards, participants completed the AUDIT and TLFB. Next, there was an approximately 132-minute break during which participants were asked to abstain from eating. We incorporated a longer break in Study 2 in order to further reduce alcohol levels in session 2 which may otherwise confound subsequent recall. After the break, participants completed the taste test, general memory recall task and DEBQ.

After at least 1 week, participants completed session 2. Firstly, participants completed a baseline breathalyser measure (all had a BrAc of 0.00), and baseline appetite and snack urge ratings. For participants in the soft drink and pre-meal drink conditions, they were then shown the pre-drink picture presentation and consumed their test drink (served in the same way as in session 1), followed by a 10-minute absorption period. They were then shown the post-drink picture presentation. Afterwards, they consumed their lunch meal before completing the first expected satiety memory task, and a second set of appetite and snack urge ratings. After this, participants completed a 2.5-hour break where they were asked to stay in the building and to abstain from eating. Participants in the soft drink condition were given the option of staying in the building or leaving and coming back after the break due to there being no ethical requirement to stay. Participants in the post-meal drink condition, after completing the baseline ratings, were shown the pre-drink picture presentation, then consumed their lunch meal, followed by the first expected satiety memory task and ratings of appetite and snack urge. Next, they consumed their test drink, followed by an absorption period and were then shown the post-drink picture presentation, followed by a 2-hour break. The break duration was calculated such that the inter-meal interval between the lunch meal and taste test was the same across conditions. After the break, participants in all conditions completed a new set of appetite and snack urge ratings and then the taste test. This was followed by the general memory recall task, the second expected satiety memory task and its

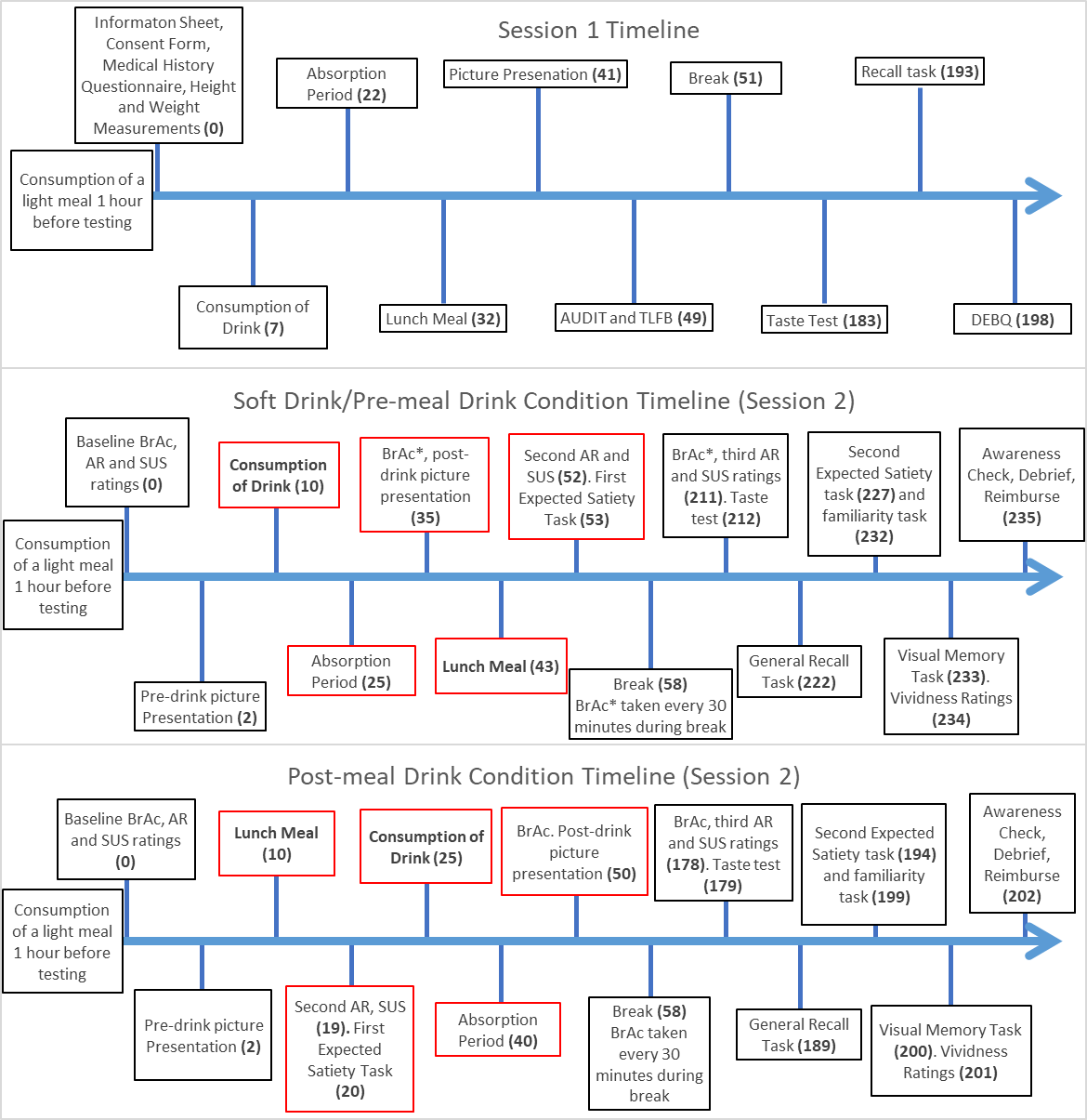
familiarity task, the visual memory for portion size task, vividness rating, awareness check, study debrief and reimbursement.

Figure 2. Schematic overview of the procedures for session 1 and session 2. Note. The procedure of session 1 was identical for all participants. Number in brackets represents the time (minutes) at which the task/measure was performed (relative to the start of the session). AR = Appetite Ratings; SUS = Snack Urge Scale ratings; BrAc = measure of breath alcohol concentration. \*The procedure in the soft drink condition was identical to the pre-meal condition, except no BrAc measures were taken apart from at baseline. Times are approximate. Boxes in red highlight where the order of tasks differs between the soft drink/pre-meal and post-meal drink condition.

3.1.5. Data Analysis

We analysed food intake using an ANCOVA with drink as the between-subjects factor and baseline (session 1) caloric cookie intake as a covariate. Performance on each meal memory measure was compared across drink conditions using one-way ANOVAs. For the expected satiety memory measure, foods which had been previously consumed by less than 50% of participants were excluded from this analysis, as has been done in previous research (Whitelock et al., 2018). Only 34% of participants had previously consumed grilled fish, therefore this item was excluded, leaving 17 food items for the analysis. For the general memory task, a mixed-design ANOVA was conducted to test for a drink by set interaction effect. Mixed ANOVAs were conducted to observe differences between drink conditions and differences across time for appetite ratings and snack urge ratings (see findings of snack urge ratings in the supplementary materials). Data for cookie intake from one participant from session 2 were lost due to human error, one participant did not complete the AUDIT questionnaire and one participant did not complete post-lunch snack urge ratings.

**3.2. Results**

3.2.1. Participant characteristics

Means and standard deviations are displayed in Table 5.

Table 5. Sample characteristics split by drink condition (mean ± SD).

|  |  |  |  |
| --- | --- | --- | --- |
|  | Soft Drink (N =24) | Pre-meal Drink (N = 24) | Post-meal Drink (N = 24) |
| BMI (kg/m2) | 25.2 ± 4 | 24.6 ± 4.9 | 23.9 ± 4 |
| Age (y) | 27.6 ± 13.2 | 23.5 ± 6.6 | 21.8 ± 6.5 |
| DEBQ Restraint | 2.7 ± 1.1 | 2.3 ± 0.8 | 2.6 ± 1 |
| DEBQ Emotional | 2.5 ± 0.8 | 2.3 ± 0.7 | 2.5 ± 0.8 |
| DEBQ External | 3.2 ± 0.5 | 3.1 ± 0.6 | 3.1 ± 0.6 |
| AUDIT (out of 40) | 10.4 ± 6.6 | 9.9 ± 3.9 | 11.7 ± 51 |
| 7-day TLFB (alcohol units) | 16 ± 12 | 19 ± 9 | 18 ± 8 |
| Baseline General Memory Recall (Session 1; out of 24) | 9 ± 2 | 9 ± 3 | 10 ± 2 |
| Baseline Appetite (out of 200; Session 2) | 112 ± 38 | 127 ± 36 | 106 ± 35 |
| Baseline Snack Urge (out of 400; Session 2) | 195 ± 67 | 202 ± 66 | 216 ± 55 |

Note. 1 = data missing from one participant. AUDIT = Alcohol Use Disorders Identification Test; BMI = Body Mass Index; DEBQ = Dutch Eating Behaviour Questionnaire; TLFB = Timeline Follow-back

3.2.2. Meal Memory measures (Table 6 and Figure 3)

There was a significant main effect of drink on expected satiety memory scores *F*(2, 69) = 4.67, *p* = .013, *ηp2* = .12. Bonferroni corrected pairwise comparisons revealed that the error score (higher scores indicating poorer memory) was significantly greater in the pre-meal drink condition, compared with the soft drink condition (*p* = .016; 95% CI [-81.52, -6.42]) (see Figure 3). No other significant main effects of drink condition were found for any other meal memory measure.

3.2.2.1 Sensitivity Analysis

With removal of outliers for the expected satiety error measure, the main effect remained statistically significant, as did the difference between the pre-meal and soft drink condition (*p* = .007; 95% CI [-67.58, -8.53]). Additionally, error scores were significantly greater in the pre-meal drink condition compared with the post-meal drink condition (*p* = .018; 95% CI [-63.54, -4.49]).

**\***

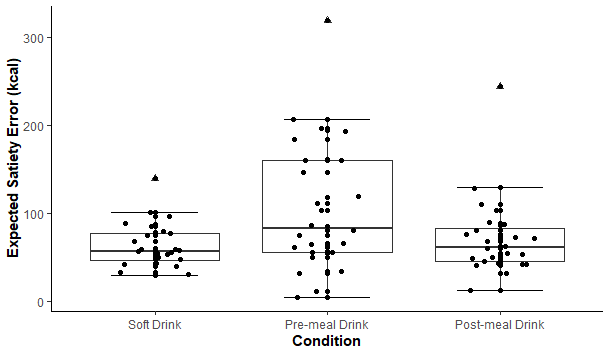


Figure 3. Boxplot displaying individual data points of expected satiety error scores, split by the three drink conditions. Note. \*p < .05. Triangles represent outliers.

3.2.3. Calorie intake (Table 6):

An ANCOVA with baseline cookie intake as a co-variate revealed a non-significant main effect of drink on cookie intake *F*(2, 67) = 0.49, *p* = .617, *ηp2* = .01. Using the same ANCOVA model, total calorie intake significantly differed between drink conditions *F*(2, 67) = 29.86, *p* < .001, *ηp2* = .47. Bonferroni corrected pairwise comparisons revealed that total caloric consumption was significantly lower in the soft drink condition compared with both the pre-meal drink (*p* < .001; mean difference = 324.83; 95% CI [-441.69, - 207.97]) and post-meal drink condition (*p* < .001; mean difference = 313.89; 95% CI [-443.05, -195.72]). Total calorie intake did not differ between the pre-meal and post-meal condition (*p* = 1.00; mean difference = 10.95; 95% CI [-107.27, 129.16]). See table 6 for means and standard deviations of caloric intake.

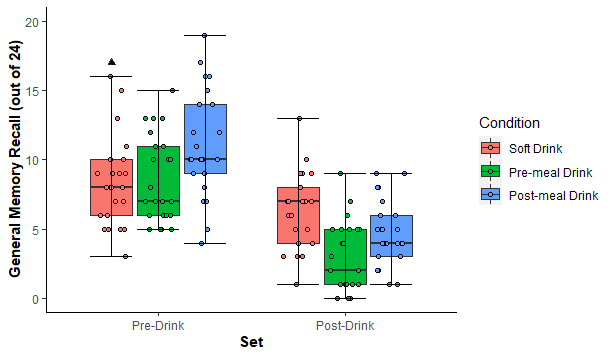
Table 6. Outcome measures, split by drink condition (mean ± SD)

|  |  |  |  |
| --- | --- | --- | --- |
|  | Soft Drink (N = 24) | Pre-meal Drink (N = 24) | Post-meal Drink (N = 24) |
| Vividness ratings (session 2; out of 100) | 80 ± 14 | 72 ± 19 | 79 ± 12 |
| Expected satiety error (kcal) | 64 ± 26a | 108 ± 76a | 72 ± 45 |
| Visual Memory (%) | 21.1 ± 14.3 | 14.8 ± 10.4 | 14.3 ± 10.3 |
| Baseline *ad libitum* food Intake (kcal; Session 1) | 293 ± 164 | 298 ± 135 | 281 ± 211 |
| *Ad libitum* food Intake (kcal; Session 2) | 358 ± 215 | 401 ± 197 | 384 ± 2161 |
| Drink and *ad libitum* intake combined (kcal; Session 2) | 364 ± 215d,e | 693 ± 212d | 667 ± 228e,1 |

Note. Means with the same letter indicate a significant difference between each other; p < .05, Bonferroni adjustment for multiple comparisons. 1 = data missing from one participant.

3.2.4. General memory recall (Figure 4):

For this analysis, we wanted to explore whether recall in the pre-drink set was greater in the two alcohol conditions relative to the soft drink condition, but greater in the soft drink condition relative to the two alcohol conditions in the post-drink set. Therefore, only the interaction effect is relevant. A 2 (set; pre-drink, post-drink) x 3 (drink; soft drink, pre-meal drink, post-meal drink) mixed ANOVA revealed a significant set by drink interaction *F*(2,69) = 8.26, *p* = .001, *ηp2* = .19. Univariate ANOVAs were conducted for each set separately (see figure 3 for general memory recall of the pre-drink and post-drink sets). A significant main effect of drink in the pre-drink set *F*(2,69) = 4.39, *p* = .016, *ηp2* = .11 was found, whereby recall in the post-meal drink condition was significantly greater than in the pre-meal drink condition (*p* = .029; mean difference = 2.71; 95% CI [0.21, 5.21]). This was unexpected, as, due to a predicted effect of retrograde facilitation, we expected recall in the pre-drink set to be significantly greater in both alcohol conditions (i.e. the pre-meal and post-meal conditions), compared with the soft drink condition. There was also a significant main effect of drink condition in the post-drink set *F*(2,69) = 11.03, *p* <.001, *ηp2* = .24, whereby recall in the pre-meal drink condition was significantly lower than in both the soft drink condition (*p* < .001; mean difference = 3.46; 95% CI [1.65, 5.27]) and the post-meal condition (*p* = .046; mean difference = 1.83; 95% CI [0.03, 3.64]). There was a nonsignificant difference between the soft drink and post-meal conditions (*p* = .092; mean difference = 1.63; 95% CI [-0.18, 3.43]).



**\***

**\***

**\***

Figure 4. Boxplot displaying individual data points of general memory recall split by the three drink conditions, and two set types in session 2. Note. \*p < .05. Triangles represent outliers.

3.2.5. Appetite Ratings

A 3 (drink; soft drink, pre-meal drink, post-meal drink) x 3 (time; baseline, post-lunch, post-break) mixed ANOVA was conducted with drink as a between-subjects factor and time as a within-subjects. This revealed a main effect of time *F*(2, 138) = 71.31, *p* < .001, *ηp2* = .51. Bonferroni corrected pairwise comparisons revealed that appetite ratings were significantly greater at baseline than post-lunch (*p* < .001; mean difference = 62; 95% CI [49, 75]) but did not differ from post-break ratings (*p* = .713; mean difference = 7; 95% CI [-8, 22]). Post-lunch appetite ratings were significantly lower than post-break ratings (*p* < .001; mean difference = 55; 95% CI [-68, -41]). The analysis also revealed a significant main effect of drink *F*(2,69) = 4.01, *p* = .023, *ηp2* = .10. Bonferroni corrected comparisons revealed that those in the soft drink condition had lower overall appetite ratings compared with the pre-meal drink condition (*p* = .029; mean difference = 22; 95% CI [-43, -2]) but did not significantly differ from the post-meal drink condition (*p* = 1.00; mean difference = 4; 95% CI [-25, 17]). Overall appetite ratings between the pre-meal and post-meal drink condition did not significantly differ (*p* = .100; mean difference = 18; 95% CI [-39, 2]). Lastly, there was a nonsignificant drink x time interaction effect *F*(4, 138) = 2.07, *p* = .088, *ηp2*= .06. See supplementary materials for full list of means and standard deviations of appetite ratings at each time point, split by condition.

**4. General Discussion**

Study 2 found that consumption of an alcoholic drink prior to consuming a lunch meal impaired meal memory when compared with consumption of a soft drink. In Study 2, this was evident for the measure of memory of satiety; participants in the pre-meal drink condition less accurately remembered the level of fullness experienced immediately after the lunch meal compared with those in the soft drink condition, as did those in the post-meal drink condition after removing outliers. However, this impairment was not evident for meal vividness ratings or visual memory of the portion size. Furthermore, the findings failed to show an enhanced recall of meal memory when the alcoholic drink was consumed after the lunch meal. There were also no significant differences in *ad libitum* food intake between the three conditions. Therefore, our hypothesis that meal memory would be lowest in the pre-meal drink condition is only partially supported, with no support to show that this increased food intake. Furthermore, our hypothesis predicting that those in the post-meal drink condition would show the greatest meal memory and lowest food intake is rejected.

Study 2, but not Study 1, showed evidence that consumption of an alcoholic drink before a lunch meal can impair certain forms of meal memory compared with memory performance after consumption of an alcohol-free drink. Altering episodic memories of a recent meal is therefore an additional factor which is both caused by acute alcohol consumption and which, in other studies, has been shown to increase food intake. However, in both Study 1 and 2 we found no significant difference in food intake between drink conditions, therefore this proposition remains unsupported.

The present findings add to the literature by implementing a novel form of meal memory disruption. By using alcohol intoxication as a tool to manipulate and disrupt the encoding phase of memory formation, findings revealed that this was successful in altering the quality of some meal memories. It also provides support for previous literature which has shown that different methods of disruptions to memory encoding impair meal recall (Higgs & Woodward, 2009; Mittal, Stevenson, Oaten, & Miller, 2011; Oldham-Cooper, Hardman, Nicoll, Rogers, & Brunstrom, 2010). The present findings also highlight the difficultly in identifying the components of meal memory which are important in determining later food intake, as although a meal memory impairment was observed in Study 2, food intake did not differ between conditions. However, this does not mean that meal memory is unimportant in determining food intake. Instead, it is possible that other components of meal memory, such as visual memory of portion size may be a more important determinant in food intake. The memory manipulation used in the present study did not appear to be strong enough in order to impair recall of all measured forms of meal memory, which may explain a lack of effect on food intake. Future research should continue to investigate which components of meal memory directly relate to subsequent food intake.

As discussed elsewhere (Whitelock et al., 2019), it is important to consider how motivated participants were to use recent memories of their lunch when deciding how much to eat in the taste test. One reason why memory of recent eating did not lead to a reduction in food intake could be due to the calorie content of the pre-load lunch meal. Pre-load meals in both Study 1 and 2 did not exceed 515 kcal. For some participants this may be considered a relatively small amount of food and therefore, after an inter-meal interval of 160 minutes (as was the case in Study 2), participants may not have felt motivated to restrict their food intake even when details of this lunch meal were well-remembered. There is some evidence to suggest sex differences may exist with regard to the effectiveness of manipulating meal memory on subsequent food intake. For example, the effect of focused attention has been established in female samples (Higgs & Donohoe, 2011; Robinson, Kersbergen, & Higgs, 2014), but is inconsistent in mixed sex samples (Seguias & Tapper, 2018; Whitelock et al., 2018) and has not been found in a male sample (Whitelock et al., 2019). One explanation for why Seguias and Tapper (2018) found a difference in food intake in a mixed-sex sample may be due to the caloric quantity of the pre-load used. In their study, participants were given *ad libitum* access to their lunch meal. This would have allowed participants to consume a personally ‘normal’ amount of food. This in turn may have resulted in the sample being more motivated to use episodic meal memory when deciding how much to consume at a subsequent eating episode. This suggestion is speculative, however future studies may wish to investigate how altering the personal appropriateness of a pre-load meal in terms of its caloric content, can moderate the effect of episodic memories on later food intake.

Findings of Study 2 failed to show evidence of enhanced meal memory recall when the meal was consumed prior to alcohol consumption. The magnitude of the retrograde facilitation effect may differ depending on the type of stimuli exposed to. For example, Weafer et al. (2016) found that the effect of consolidation was greatest for neutral stimuli (*d* = 0.79) compared with negative (*d* = 0.26) and positive (*d* = 0.31) stimuli. It is plausible to assume that food-related stimuli may not be considered neutral. Therefore, as Study 2 was powered to detect a large effect size, we may have been underpowered to detect consolidation effects of other, non-neutral stimuli. However, we also failed to find a consolidation effect for general memory recall, suggesting an overall failure in producing this effect.

Alternatively, a failure to detect enhanced meal memory may have resulted from the experimental design. By using the same test lunch in both the first and second session, participants may have already established a memory of the lunch meal from the first session, allowing participants to remember back to the previous session to recall details of their lunch meal, therefore minimising the importance of the effect of the drink on memory formation. Although we incorporated a 1-week washout period to counter this issue, some participants may still have remembered the quantity of the lunch meal. This may explain why no differences were found for the visual memory and vividness measures. However, in the case of the expected satiety memory measure, this was shown to be significantly impaired in the pre-meal drink condition relative to the soft drink condition. It may be that memory for fullness more difficult to remember between sessions, compared with other forms of meal memory.

There are some limitations with Study 2. Firstly, during the break in the second session, participants in the soft drink condition were not required to wait in a waiting room during the break. Although all participants were told to abstain from eating, some participants in this condition would have had a different experience during their break compared to participants in the other conditions, although no significant difference in food intake was found. A second limitation was that during the recall phase, participants in both alcohol conditions were on the descending limb of the blood alcohol curve (see supplementary materials for BrAc scores). The descending limb can produce sedation, negative mood (Babor et al., 1983; Lukas et al., 1986; Sukter et al., 1983) and impairment of certain forms of executive functioning (Pihl et al., 2003). One way to overcome this issue and to ensure participants were sober at the point of recall would have been to implement a longer delay of 24 or 48 hours after the exposure phase, which has been done in previous studies (Gawrylowicz et al., 2017; Weafer, Gallo & De Wit, 2016). However, we decided to implement a shorter period as this was essential in order to observe the effect of meal memory on food intake. This is because previous research has shown that cueing participants of their lunch consumed on the previous day does not affect food intake, but cueing lunch which has been consumed on the same day reduces subsequent food intake (Higgs, 2002). This suggests that memories relating to food consumed only very recently can alter food intake. Therefore, a greater delay may have failed to tap into the effect of meal memory on food intake. Despite this, a difference in mood and executive performance may have contributed to a lack of enhanced recall through retrograde facilitation which may have been observed otherwise with a longer delay.

In conclusion, there was some evidence to suggest that consuming a lunch meal whilst intoxicated can impair subsequent recall of certain lunch details. However, neither study provided evidence that meal memory predicted subsequent food intake. It therefore remains unclear whether alcohol induced changes in meal memory contribute towards alcohol-induced overeating.

**Competing interests:** CAH and PC receive research funding from the American Beverage Association for work outside of the submitted manuscript. CAH has also received speaker fees from International Sweeteners Association.

**Funding:** This research was not funded by any specific grant.

**Author contributions:** All authors contributed to designing the research. TG was responsible for data collection and conducted the analyses. TG drafted the manuscript, and all authors approved the final manuscript.

**Acknowledgements:** We would like to thank Professor Jeff Brunstrom for his contribution towards the design of Study 2. We would also like to thank Sarah Levy and Sarah Masterton for their assistance with scoring of the meal memory recall scoring in Study 1.

**Ethical approval and consent to participate**: Ethical approval was gained from the University of Liverpool Health and Life Sciences Research Ethics Committee.

**Consent for publication:** Not applicable.

**Availability of data and material:** The datasets generated and analysed for Study 1 and Study 2 are available on the Open Science Framework repository (osf.io/mbxs8/).

References

Babor, T. F., Berglas, S., Mendelson, J. H., Ellingboe, J., & Miller, K. (1983). Alcohol, affect, and the disinhibition of verbal behavior. *Psychopharmacology*, *80*(1), 53-60.

Bisby, J. A., Leitz, J. R., Morgan, C. J., & Curran, H. V. (2010). Decreases in recollective experience following acute alcohol: A dose–response study. *Psychopharmacology*, *208*(1), 67.

Blundell, J., De Graaf, C., Hulshof, T., Jebb, S., Livingstone, B., Lluch, et al. (2010). Appetite control: methodological aspects of the evaluation of foods. *Obesity reviews*, *11*(3), 251-270.

Brunstrom, J. M., Burn, J. F., Sell, N. R., Collingwood, J. M., Rogers, P. J., Wilkinson, L. L., ... & Ferriday, D. (2012). Episodic memory and appetite regulation in humans. *PloS one*, *7*(12), e50707.

Brunstrom, J. M., Shakeshaft, N. G., & Scott-Samuel, N. E. (2008). Measuring ‘expected satiety’ in a range of common foods using a method of constant stimuli. *Appetite*, *51*(3), 604-614.

Cains, S., Blomeley, C., Kollo, M., Rácz, R., & Burdakov, D. (2017). Agrp neuron activity is required for alcohol-induced overeating. *Nature communications*, *8*, 14014.

Caton, S. J., Ball, M., Ahern, A., & Hetherington, M. M. (2004). Dose-dependent effects of alcohol on appetite and food intake. *Physiology & behavior*, *81*(1), 51-58.

Caton, S. J., Marks, J. E., & Hetherington, M. M. (2005). Pleasure and alcohol: manipulating pleasantness and the acute effects of alcohol on food intake. *Physiology & behavior*, *84*(3), 371-377.

Christiansen, P., Rose, A., Randall-Smith, L., & Hardman, C. A. (2016). Alcohol’s acute effect on food intake is mediated by inhibitory control impairments. *Health Psychology*, *35*(5), 518.

Clifton, P. G., Vickers, S. P., & Somerville, E. M. (1998). Little and often: ingestive behavior patterns following hippocampal lesions in rats. *Behavioral neuroscience*, *112*(3), 502.

Davidson, T. L., Kanoski, S. E., Walls, E. K., & Jarrard, L. E. (2005). Memory inhibition and energy regulation. *Physiology & behavior*, *86*(5), 731-746.

Gadah, N. S., Brunstrom, J. M., & Rogers, P. J. (2016). Cross-over studies underestimate energy compensation: the example of sucrose-versus sucralose-containing drinks. *Appetite*, *107*, 398-405.

Gawrylowicz, J., Ridley, A. M., Albery, I. P., Barnoth, E., & Young, J. (2017). Alcohol-induced retrograde facilitation renders witnesses of crime less suggestible to misinformation. *Psychopharmacology*, *234*(8), 1267-1275.

Hannapel, R., Ramesh, J., Ross, A., LaLumiere, R. T., Roseberry, A. G., & Parent, M. B. (2019). Postmeal optogenetic inhibition of dorsal or ventral hippocampal pyramidal neurons increases future intake. *Eneuro*. ENEURO.0457-18.2018. <https://doi.org/10.1523/ENEURO.0457-18.2018>

Hardman, C. A., Rogers, P. J., Dallas, R., Scott, J., Ruddock, H. K., & Robinson, E. (2015). “Food addiction is real”. The effects of exposure to this message on self-diagnosed food addiction and eating behaviour. *Appetite*, *91*, 179-184.

Hashtroudi, S., Parker, E. S., DeLisi, L. E., Wyatt, R. J., & Mutter, S. A. (1984). Intact retention in acute alcohol amnesia. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *10*(1), 156.

Higgs, S. (2002). Memory for recent eating and its influence on subsequent food intake. *Appetite*, *39*(2), 159-166.

Higgs, S., & Donohoe, J. E. (2011). Focusing on food during lunch enhances lunch memory and decreases later snack intake. *Appetite*, *57*(1), 202-206.

Higgs, S., & Spetter, M. S. (2018). Cognitive control of eating: the role of memory in appetite and weight gain. *Current obesity reports*, *7*(1), 50-59.

Higgs, S., Williamson, A. C., & Attwood, A. S. (2008). Recall of recent lunch and its effect on subsequent snack intake. *Physiology & behavior*, *94*(3), 454-462.

Higgs, S., Williamson, A. C., Rotshtein, P., & Humphreys, G. W. (2008). Sensory-specific satiety is intact in amnesics who eat multiple meals. *Psychological Science*, *19*(7), 623-628.

Higgs, S., & Woodward, M. (2009). Television watching during lunch increases afternoon snack intake of young women. *Appetite*, *52*(1), 39-43.

Hull, J. G., & Bond, C. F. (1986). Social and behavioral consequences of alcohol consumption and expectancy: a meta-analysis. *Psychological bulletin*, *99*(3), 347.

Knowles, S. K. Z., & Duka, T. (2004). Does alcohol affect memory for emotional and non-emotional experiences in different ways?. *Behavioural pharmacology*, *15*(2), 111-121.

Kwok, A., Dordevic, A. L., Paton, G., Page, M. J., & Truby, H. (2019). Effect of alcohol consumption on food energy intake: A systematic review and meta-analysis. British Journal of Nutrition, 121(5), 481-495.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1997). International affective picture system (IAPS): digitized photographs, instruction manual and affective ratings. Technical Report A-6, *Gainesville, FL, University of Florida.*

Lukas, S. E., Mendelson, J. H., Benedikt, R. A., & Jones, B. (1986). EEG alpha activity increases during transient episodes of ethanol-induced euphoria. *Pharmacology Biochemistry and Behavior*, *25*(4), 889-895.

Martin, C. S., Earleywine, M., Musty, R. E., Perrine, M. W., & Swift, R. M. (1993). Development and validation of the biphasic alcohol effects scale. *Alcoholism: Clinical and Experimental Research*, *17*(1), 140-146.

Mittal, D., Stevenson, R. J., Oaten, M. J., & Miller, L. A. (2011). Snacking while watching TV impairs food recall and promotes food intake on a later TV free test meal. *Applied Cognitive Psychology*, *25*(6), 871-877.

Nilsson, L. G., Bäckman, L., & Karlsson, T. (1989). Priming and cued recall in elderly, alcohol intoxicated and sleep deprived subjects: A case of functionally similar memory deficits. *Psychological Medicine*, *19*(2), 423-433.

Oldham-Cooper, R. E., Hardman, C. A., Nicoll, C. E., Rogers, P. J., & Brunstrom, J. M. (2010). Playing a computer game during lunch affects fullness, memory for lunch, and later snack intake. *The American journal of clinical nutrition*, *93*(2), 308-313.

Parker, E. S., Birnbaum, I. M., Weingartner, H., Hartley, J. T., Stillman, R. C., & Wyatt, R. J. (1980). Retrograde enhancement of human memory with alcohol. *Psychopharmacology*, *69*(2), 219-222.

Pihl, R. O., Paylan, S. S., Gentes‐Hawn, A., & Hoaken, P. N. (2003). Alcohol affects executive cognitive functioning differentially on the ascending versus descending limb of the blood alcohol concentration curve. *Alcoholism: Clinical and Experimental Research*, *27*(5), 773-779.

Raben, A., Agerholm-Larsen, L., Flint, A., Holst, J. J., & Astrup, A. (2003). Meals with similar energy densities but rich in protein, fat, carbohydrate, or alcohol have different effects on energy expenditure and substrate metabolism but not on appetite and energy intake. *The American journal of clinical nutrition*, *77*(1), 91-100.

Robinson, E., Kersbergen, I., & Higgs, S. (2014). Eating ‘attentively’ reduces later energy consumption in overweight and obese females. *British Journal of Nutrition*, *112*(4), 657-661.

Röjdmark, S., Calissendorff, J., & Brismar, K. (2001). Alcohol ingestion decreases both diurnal and nocturnal secretion of leptin in healthy individuals. *Clinical endocrinology*, *55*(5), 639-647.

Saunders, J. B., Aasland, O. G., Babor, T. F., De la Fuente, J. R., & Grant, M. (1993). Development of the alcohol use disorders identification test (AUDIT): WHO collaborative project on early detection of persons with harmful alcohol consumption‐II. *Addiction*, *88*(6), 791-804.

Seguias, L., & Tapper, K. (2018). The effect of mindful eating on subsequent intake of a high calorie snack. *Appetite*, *121*, 93-100.

Schrieks, I. C., Stafleu, A., Griffioen-Roose, S., de Graaf, C., Witkamp, R. F., Boerrigter-Rijneveld, R., & Hendriks, H. F. (2015). Moderate alcohol consumption stimulates food intake and food reward of savoury foods. *Appetite*, *89*, 77-83.

Sobell, L. C., & Sobell, M. B. (1992). Timeline follow-back. In *Measuring alcohol consumption* (pp. 41-72). Humana Press, Totowa, NJ.

Söderlund, H., Parker, E. S., Schwartz, B. L., & Tulving, E. (2005). Memory encoding and retrieval on the ascending and descending limbs of the blood alcohol concentration curve. *Psychopharmacology*, *182*(2), 305-317.

Sutker, P. B., Tabakoff, B., Goist Jr, K. C., & Randall, C. L. (1983). Acute alcohol intoxication, mood states and alcohol metabolism in women and men. *Pharmacology Biochemistry and Behavior*, *18*, 349-354.

Tapper, K., & Seguias, L. (2020). The effects of mindful eating on food consumption over a half-day period. *Appetite*, *145*, 104495.

Van Strien, T., Frijters, J. E., Bergers, G. P., & Defares, P. B. (1986). The Dutch Eating Behavior Questionnaire (DEBQ) for assessment of restrained, emotional, and external eating behavior. *International journal of eating disorders*, *5*(2), 295-315.

Weafer, J., Gallo, D. A., & De Wit, H. (2016). Acute effects of alcohol on encoding and consolidation of memory for emotional stimuli. *Journal of studies on alcohol and drugs, 77*(1), 86-94.

White, A. M., Matthews, D. B., & Best, P. J. (2000). Ethanol, memory, and hippocampal function: a review of recent findings. *Hippocampus*, *10*(1), 88-93.

Whitelock, V., Gaglione, A., Davies-Owen, J., & Robinson, E. (2019). Focused attention during eating enhanced memory for meal satiety but did not reduce later snack intake in men: A randomised within-subjects laboratory experiment. *Appetite*, *136*, 124-129.

Whitelock, V., Higgs, S., Brunstrom, J. M., Halford, J. C., & Robinson, E. (2018). No effect of focused attention whilst eating on later snack food intake: Two laboratory experiments. *Appetite*, *128*, 188-196.

Wixted, J. T. (2005). A theory about why we forget what we once knew. *Current Directions in Psychological Science*, *14*(1), 6-9.

Yeomans, M. R. (2010). Alcohol, appetite and energy balance: is alcohol intake a risk factor for obesity?. *Physiology & behavior*, *100*(1), 82-89.

Zola-Morgan, S., Squire, L. R., & Amaral, D. G. (1986). Human amnesia and the medial temporal region: enduring memory impairment following a bilateral lesion limited to field CA1 of the hippocampus. *Journal of Neuroscience*, *6*(10), 2950-2967.