UNIVERSITY of STIRLING

THE IMPACT OF ALCOHOL-INDUCED BLACKOUTS ON MEMORY IN A SCOTTISH BASED STUDENT POPULATION

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Abstract

Rapid drinking of excessive quantities of alcohol disrupts normal memory functioning and can lead to the experience of an alcohol-induced memory blackout (MBO), meaning that events which occurred while intoxicated may not be recalled once sober. Although instances of extreme binge-drinking and MBOs are prevalent in student populations, there is a paucity of knowledge surrounding the topic. Critically, 1) we do not know what the wider influences on these behaviours may be, specifically for students studying in Scotland, and 2) we also do not know whether these events leave a lasting impact on memory formation and, if so, whether this deficit is temporary. This thesis addressed these issues firstly with a questionnaire which investigated student drinking behaviours, and then with a series of laboratory-based memory studies which participants carried out sober, after a scaled dose of alcohol, and within 20-hours of experiencing a blackout. We found that students in Scotland frequently binge-drink, with a high prevalence of MBOs, influenced by home country, year of study, and possibly by Scottish culture. We also found control and experimental participant groups performed recall and recognition memory tasks with similar behavioural accuracy while sober and that performance dropped but did not differ following alcohol. However, ERP evidence suggested a shift in neural strategy in MBO participants compared to controls. Further, *after-blackout* performance remained impaired in more cognitively demanding tasks. In sum, evidence suggests that our Scottish-based student population drink to excess, with a large proportion experiencing regular MBOs. Alcohol impairs behavioural memory performance for all, but underlying differences in memory strategy, and a lasting deficit following MBO, can be seen in those who frequently blackout. These findings highlight the importance of further investigating the trajectory of blackout experiences, and any damage they leave in their wake.

Ethical Approval Statement

Studies presented within this thesis received ethical approval from the General University Ethics Panel and the University of Stirling's NHS, Invasive and Clinical Research ethics committee.

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Table of Contents

Abstract *3*

Ethical Approval Statement *4*

Acknowledgements *5*

List of Figures & Tables *9*

Chapter One: *12*

Introduction **12**

What is an MBO? 13
History of blackout research 15
Binge-drinking 19
Binge-drinking and blackouts 21
Scotland's relationship with alcohol 23
Memory 25
Episodic Memory 26
Methods of testing memory: Behavioural 31
Methods of testing memory: Electroencephalography (EEG) 32
ERPs and Recognition Memory 35
ERPs and Alcohol 37
ERPs, Alcohol and Memory 38
Thesis Goals 39

Chapter Two: *42*

General Methods **42**

General Study Plan 42
Alcohol Protocol 45
After-Blackout Protocol 48

Chapter Three: *50*

Alcohol and the 'student experience': Influences on, and prevalence of, alcohol-induced memory blackouts in a Scottish based student population **50**

Abstract 51
Introduction 53
Materials and Methods 56
Results 66
Discussion 72

Chapter Four: *77*

The morning after the night before: Alcohol-induced blackouts impair next day recall in sober young adults **77**

Abstract 78
Introduction 79
Methods 83
Results 93
Discussion 101

Chapter Five: *108*

Am I sure that happened, or am I just making it up? False memories after alcohol-induced memory blackouts in sober young adults 108
Abstract 109
Introduction 110
Methods 113
Results 119
Discussion 124

Chapter Six: 131

ERP and Microstates of memory: Frequent alcohol-induced blackouts do not change behavioural performance but alter sober neural functioning 131
Abstract 132
Introduction 133
Materials and Methods 138
Results 143
Discussion 151

Chapter Seven: 156

Effects of alcohol and alcohol-induced memory blackouts on ERPs and recognition memory 156
Abstract 157
Introduction 158
Study One 162
Materials and Methods 162
Results 167
Discussion of Study One 177
Study Two 180
Materials and Methods 180
Results 181
Discussion of Study Two 184
General Discussion 185

Chapter Eight: 191

General Discussion 191
Review of Findings 191
What did our questionnaire tell us? 192
What can behavioural results tell us? 194
How does the ERP data fit with the behavioural data? 198
What were the lasting effects following blackout? 201
MBO vs Hangover – what are the differences, and can next day impairments be disassociated? 205
Fragmentary vs *en bloc* blackouts – are they really different? 208
Alcohol Expectancy Effects 211
The influence of bias 212
Impact of the Covid-19 Pandemic 215
Future directions 217
Conclusions 219

References 222

Appendices 262

Appendix 1.1 263

Between Group Analysis Model Output Tables (Chapter 4) 263

Appendix 2.1 267

Within MBO Group Analysis Model Output Tables (Chapter 4) 267

Appendix 3.1 269

ERPs across traditional time-windows (Chapter 6) 269

Appendix 3.2 272

Global variance explained by microstate 272

Appendix 4.1 273

Comparison of same MBO group individuals *before-alcohol*, *after-alcohol* and *after-MBO* (Chapter 7) 273

Results 273

Appendix 5.1 274

Questionnaire (Chapter 3) 274

List of Figures & Tables

Figure 1.1: Memory model……14

Figure 2.1: Participant recruitment plan……44

Figure 3.1: PISD model……70

Figure 3.2: Personal drinking model……71

Figure 4.1: *Free and serial recall task structure……85*

Figure 4.2: *Depth of encoding task structure……87*

Figure 4.3**:** Analysis structure……92

Figure 4.4: Free recall……94

Figure 4.5**:** Serial recall……95

Figure 4.6: Depth of encoding accuracy……97

Figure 4.7: Depth of encoding resampling and sleep data……100

Figure 5.1: *DRM task structure……117*

Figure 5.2: Analysis structure……119

Figure 5.3: dPrime and response bias……121

Figure 5.4: RKG hits and false alarms by relatedness……122

Figure 5.5: Resampling and sleep data within MBO group……124

Figure 6.1: *Source memory task structure……*140

Figure 6.2: Electrophysiological responses, difference waves, and difference topographies highlighting the mid-frontal familiarity effect……145

Figure 6.3: Electrophysiological responses, difference waves, and difference topographies highlighting the parietally distributed recollection effect.……146

Figure 6.4: Microstate segmentation analysis……150

Figure 6.5: Global Explained Variance……151

Figure 7.1: *Source memory task structure……*164

Figure 7.2: Electrophysiological responses and difference waves both before and after alcohol in the Control group……172

Figure 7.3: Electrophysiological responses and difference waves both before and after alcohol in the MBO group……173

Figure 7.4: Electrophysiological responses and difference waves both before and after alcohol in the Control group……176

Figure 7.5: Electrophysiological responses and difference waves both before and after alcohol in the MBO group……177

Figure 7.6: Electrophysiological responses and difference waves after alcohol-induced blackout in the MBO group……183

Figure 7.7: Behavioural accuracy across studies……186

Figure 8.1: Potential model of the blackout experience……210

Figure 8.2: Drinking patterns of questionnaire respondents……214

Figure S3.1: Electrophysiological responses and difference waves across 300-500ms……270

Figure S3.2: Difference topographies……271

Figure S3.3: Global explained variance……272

Figure S3.4: MBO group global explained variance……274

Table 3.1: Participant Demographics……57

Table 3.2: Family Drinking History……58

Table 3.3: Personal Drinking Behaviours……60

Table 3.4: Peer Influences on Student Drinking……62

Table 3.5: MBO Metric……64

Table 3.6: Descriptive statistics, Variance Inflation Factors and correlation matrix……68

Table 3.7: Regression Coefficients……68

Table 4.1: Self-reported frequency of drinking behaviours……84

Table 4.2: Alcohol dose, drinking time, and mean BrAC……89

Table 4.3: Self-reported frequency of drinking behaviours of MBO group……91

Table 5.1: Alcohol dose, drinking time, and mean BrAC……115

Table 5.2: MBO group drinking behaviour……116

Table 6.1: Behavioural Accuracy results……143

Table 7.1: Alcohol dose, drinking time and mean BrAC……165

Table 7.2: Behavioural Accuracy Results: Within Groups……168

Table 7.3: Behavioural Accuracy Results: Between Groups……169

Table 7.4: Behavioural Accuracy Results……181

Table 7.5: Comparison of HH & HM Accuracy……187

Table 8.1: Comparison of participants showing reduced performance after-MBO……204

Table S1.1: Free recall (between groups)……263

Table S1.2: Serial recall (between groups)……264

Table S1.3: Depth of encoding (between groups)……265

Table S2.1: Free recall (MBO group only)……267

Table S2.2: Serial recall (MBO group only)……267

Table S2.3: Depth of encoding (MBO group only)……268

Table S4.2: Behavioural mean accuracy……273

Table S4.2: Bonferroni corrected post hoc significant effects……274

Chapter One:

Introduction

You may be familiar, either through first or second-hand experience, with waking up and realising that your memory for the previous evening's alcohol-fuelled debauchery may not be a complete and accurate record of events. Colloquially in Scotland, this has been referred to as feeling 'the fear', the realisation of not knowing precisely what happened or what you did the night before. Anecdotally at least, alcohol-induced memory loss, and the accompanying fear, is a common occurrence for many undergraduate students. An alcohol-induced memory blackout (MBO) occurs when a rapid spike in blood alcohol content (BAC%) disrupts processing within the hippocampus (A. M. White, 2003), leading to transient anterograde amnesia. Although individuals are conscious and continue to operate within the world, memories for the period can be either fragmented, or simply not retrievable. The impact of alcohol consumption on health, behaviour and society has been extensively researched, however the effects of experiencing a memory blackout have not. Crucially, a number of important questions have been overlooked by researchers thus far. While the physiological mechanism underpinning the likely cause of blackouts is broadly understood (A. M. White, 2003), it is currently unknown (1) what the duration of a blackout effect is; (2) how repeated MBO events impact cognition; (3) whether there are differences in memory performance between those who do and do not experience MBOs. These unknowns are concerning since regular disruption of neural processing could plausibly leave a detriment in functioning which lasts beyond a return to sobriety, both acutely and through continued exposure to alcohol. Further, since binge-drinking is often seen in adolescents and young adults (Lees et al., 2019; Merrill & Carey, 2016; Torcaso et al., 2017), repeatedly exposing the child/young-adult brain to MBOs could impact its development.

Heavy alcohol consumption is ingrained within Scottish culture, with high rates of binge-drinking (Giles & Robinson, 2018), which is often enthusiastically practiced by those attending university. When school leavers and young adults go to university, the 'student experience' becomes as much about fresher's week and socialising

(usually involving heavy amounts of alcohol consumption), as it does about the opportunity to move away from parental influence and gain independence. It is arguably important that we conduct research focussed on the alcohol-experiences of students, their drinking behaviours and the prevalence and effects of MBOs in this population. This thesis recognises that experiencing alcohol-induced memory blackouts likely causes a range of harms, including to the brain, which may be specifically problematic in younger adults due to their stage in neural development. On that basis, I present a collection of studies and discussions on the theme of alcohol-induced harm and the prevalence and effects of MBOs in a student population. Firstly, the culture of alcohol binge-drinking within young adults attending Scottish universities is considered. The influence of peers and family relationships on drinking behaviours, and the prevalence of alcohol-induced memory blackouts, was investigated through a questionnaire distributed across several Scottish universities. Having established problematic rates of MBO experiences in Scottish-based students, I go on to explore how frequent MBO experiences impact memory performance across various episodic memory experiments, utilising a range of lab-based methodologies. Through comparing individuals who frequently blackout with those who do not drink, or who have never experienced a blackout, evidence for memory performance sober and after alcohol was collated and analysed. Further, a subset of participants repeated lab-based experiments within 24-hours of experiencing an MBO which provided indicators of lasting deficits to memory even once sober. Behavioural performance and neural imaging via electroencephalography (EEG) were combined to consider differences between control and blackout groups. In brief, the aim of this thesis is to develop a deeper understanding the factors influencing extreme binge-drinking events in students, and to investigate whether MBOs leave a lasting impact on cognitive and neural functioning.

What is an MBO?

An alcohol-induced memory blackout is a short-term anterograde amnesic event which occurs when excessive quantities of alcohol are consumed rapidly, leading

to disruption in normal cognitive processing (A. M. White, 2003). Alcohol impairs episodic memory, specifically by disrupting activity in the hippocampus (A. M. White & Best, 2000) and preventing the transfer of information from short to long term storage. Sometimes misinterpreted as loss of consciousness (A. M. White et al., 2002), individuals experiencing an MBO in fact remain awake and able to interact with others (Jellinek, 1952). While intoxicated, individuals retain aspects of long-term memory (Goodwin, Crane, et al., 1969b; A. M. White, 2003), and maintain immediate short-term memory, defined as up to/around 2-minutes (Goodwin et al., 1970). This means that, to those around, there would be no way of identifying that they were experiencing disruption to memory storage. The impairment becomes apparent when – once sober – individuals realise they cannot recall some or all of the time whilst they were drunk.

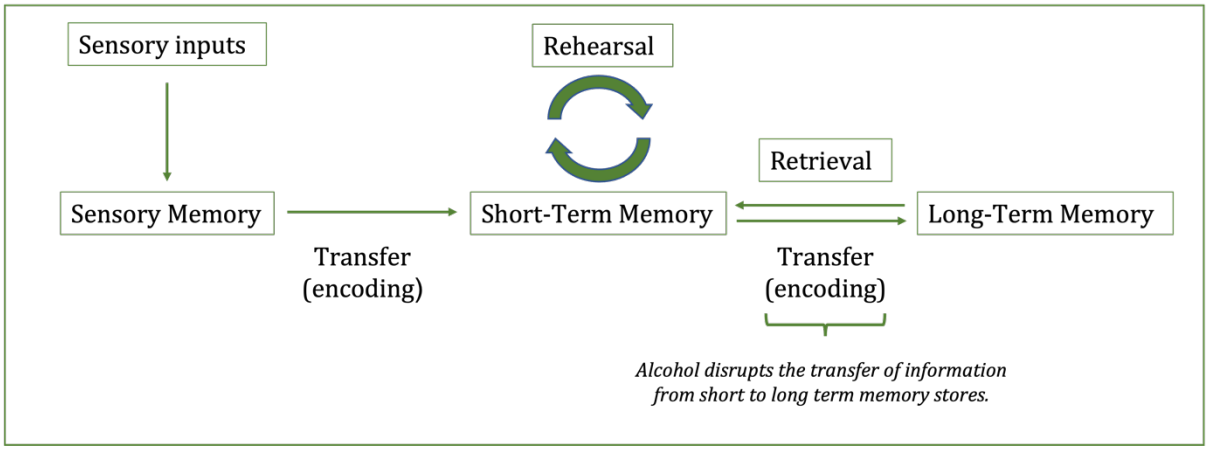

Figure 1.1: **Memory Model.** Based on the modal model of memory (Atkinson & Shiffrin, 1968), and adapted from White (2003), Figure 1.1 shows a simple depiction of the input from contextual information in the environment, through temporary short-term memory and on to long-term storage and retrieval. In memory processing, alcohol specifically acts on CA1 in the hippocampus to inhibit the transfer of information to long-term memory.

While the mechanism underpinning the onset of an MBO is well documented (for example, Hermens & Lagopoulos, 2018; Rose & Grant, 2010; A. M. White, 2003), less is known about long-term consequences for the brain and cognition which may occur as a result of frequent blackout experiences. It is already known that alcohol can cause enduring damage to the brain when given early in development, that is, in the womb (Charness et al., 2016; Granato & Dering, 2018; Riley et al., 2011). Further, since research suggests MBOs are prevalent in younger adults (Hingson et al., 2016; A. M.

White et al., 2002), understanding whether repeated assault from alcohol to the hippocampus during a period of ongoing neurodevelopment can cause lasting harm is a critical question.

History of blackout research

Published research examining the phenomena of alcohol-induced memory blackouts began in the United States of America in the 1940's when Alcoholics Anonymous (AA) issued a survey in their May 1945 magazine, The Grapevine. The purpose was to investigate patterns of past drinking behaviour, to look for commonalities, to advance understanding of what drives individuals to alcoholism, and to chart the progress of disease in their 1600 subscribers. Until this point, alcohol research had been focused on finding personality traits, or social indicators, which could predict a future potential for alcoholism, often with the intention of supporting psychological treatments and therapy. However, recovered alcoholics realised that a history of heavy drinking in itself influenced future drinking and behaviour, and progression towards alcoholism, and therefore any insight had to come from alcoholics themselves.

After distributing their survey, Alcoholics Anonymous invited Elvin Jellinek, a biometrician and professor of physiology at Yale University, to analyse their responses and provide a report on findings. Jellinek accepted their invitation, however complained at the lack of scientific rigour used in the development of the questions, and the subsequent lack of consideration for the statistical methods required to answer their overarching questions. He nevertheless presented his findings in his 1946 paper, "Phases in Drinking History of Alcoholics" (Jellinek, 1946). In these findings, he highlighted "blackouts" as a possible early symptom/phase of alcoholism. While he did not suggest that experiencing a blackout was only seen in alcoholics – indeed he acknowledged that a person could experience a blackout on the first and only occasion that they ever consumed alcohol - he did report that 90% of questionnaire respondents had experienced a blackout and therefore the experience was common within alcoholics and worthy of note.

Having delivered his results, Jellinek designed and distributed his own more detailed questionnaire to alcoholics in the years which followed, building on the

findings from the Grapevine survey and correcting some of the methodological issues. He shared results from this new work in the early 1950's through a series of lectures, and eventually published in 1952 (Jellinek, 1952). These findings claimed that experiencing regular blackouts indicated the beginning of a 'prodromal' phase of alcoholism, a period which he suggested lasted up to 5-years and from which the only effective 'cure' was complete abstinence. This sparked a number of publications on alcoholism, its symptoms and the progression of the disease, but few which focussed on blackouts themselves. The gap was addressed by Goodwin, Crane and Guze (1969a) who conducted structured interviews with 100 hospitalised, diagnosed alcoholics. Their conclusions somewhat differed from those of Jellinek in three ways. Firstly, they found that frequent, consistent blackouts occurred at a later stage in alcoholism than previously suggested; secondly, that they only occurred after large quantities of alcohol were consumed and did not happen after moderate amounts; and finally, that they did not happen to everyone who was an alcoholic. They put forward a number of possible explanations for these differences, importantly including the fact that it is difficult to describe an experience of which you have little or no memory and therefore may not even realise you have had. Further, the interpretation of the word 'blackout' could mean unconsciousness to some people, which is not the case in an alcohol-induced MBO. Therefore, an inability to recall experiencing a blackout, coupled with misunderstanding exactly what they are, highlighted both the difficulty of research in this area and the need for clearer definitions. Furthermore, the interviews also shed light on some of the possible circumstances required for a blackout to occur. It appeared that drinking or gulping large quantities of alcohol quickly resulted in "more severe" (Goodwin, 1995) blackouts, and that they could occur following consumption of either spirits or beer. Further commonalities included head injury or trauma, lack of food or sleep, and drinking over a prolonged period of time.

Two important facts became apparent from early blackout research. Firstly, for an individual to realise they had lost memory for a period something had to have happened during that period which was forgotten (Goodwin, 1995). If (for example) someone was at home alone, drinking alcohol and watching tv, it would be almost impossible to determine whether a blackout had occurred. Even if the MBO had lasted a number of hours, and the individual noticed that time had passed which could not be recalled, it is plausible that they would simply assume they had been asleep. Therefore,

acquiring details of blackouts – and if they had even occurred - after the event was extremely difficult. Secondly, the only real way to examine a memory blackout was to do so while it was happening - something easier said than done. Since people can continue to converse and interact with the world, observation alone cannot determine whether someone is experiencing a blackout. The solution was to supply large quantities of alcohol to individuals who could handle high levels of consumption without passing out, run regular tests during the drinking period, then test memory for the event afterwards. Ryback (1970) applied this method when he recruited seven volunteers from an alcohol rehabilitation facility to take part in a hospital-based study. The participants were monitored at all times by a nurse and a researcher, working in 8-hour shifts, and had access to alcohol which was dispensed when 'purchased' with tokens earned by using experimental apparatus. Blood alcohol levels were monitored, and records of behaviour kept which allowed participants to be questioned. Like the study by Goodwin and colleagues (1969a), participants who drank more slowly did not seem to experience blackouts as early in the study as those who drank more quickly. In fact, at times BAC% was higher for some individuals who drank more slowly and were not experiencing blackouts, than for those who were.

Following a second interview study, Goodwin, Crane and Guze (1969b) realised that reports of blackout experiences differed in an important way. Some blackouts appeared to feature a starting point, after which no memories were recoverable for a period of time. Others seemed less structured, with fragments of the event more easily remembered, and other memories recovered either following reminders or simply as time passed. To investigate this, and similar to Ryback (1970), Goodwin hospitalised a group of ten alcoholics and monitored their behaviour during a drinking session (Goodwin et al., 1970). He did this by presenting stimuli to the participants (either a children's' toy, or a clip from an adult film), asking them to describe the stimuli after 2-minutes (a test of short-term memory), after 30-minutes while still intoxicated, and again after 24-hours once sober. Five of the participants had reported experiencing regular blackouts outside of the experimental context, and these same individuals only displayed memory loss for stimuli after 30-minutes and were unable to either recall or recognise the items the following day. The other five participants did not regularly experience blackouts outside of the experiment and showed no impairments at any stage despite consuming the same quantity of alcohol. This confirmed that some

individuals had experienced a blackout, but that short-term memory was relatively unaffected. A follow-up study with male medical student social drinkers, and smaller quantities of alcohol, was designed to test memory loss in a variety of contexts, for example stimuli were studied sober, and then tested after alcohol, or studied after alcohol, tested after alcohol, etc. Their findings suggested there was a state-dependent influence on memory, but also urged caution as the differences between participants in the different studies (for example, personality variables) may have impacted on results. Nevertheless, Goodwin et al. concluded that it was likely that two types of blackout were possible, and that the hippocampus was probably implicated in their onset. This view was shared by Ryback (1971) who likened the two experiences to a 'grey-out', when memories were fuzzy but retrievable either spontaneously or after cueing, and to the amnesia seen in Wernicke-Korsakoff syndrome.

To the best of my knowledge, these different blackout 'types' were first introduced by Goodwin, Crane, et al. (1969b), and then described more fully by Goodwin in his 1970 book 'Blackouts and alcohol induced memory disfunction', no longer in print. Goodwin coined the phrases '*en bloc*' to describe the permanent missing chunks of time experienced in more extreme blackouts, and used '*fragmentary*' to distinguish the shorter, lost snippets of events which were often retrievable. However, the terms were not widely adopted in the literature until the 1990s. This failure to adopt new terminology may in part be due to a gap in the blackout literature, with a review by Sweeney (1989) complaining of a lack of meaningful new studies in the preceding seventeen years. In fact, there was very little interest in the topic until the early 21st century when (A. M. White et al., 2002) published details of an email survey conducted with college students. They found that around half of respondents who drank alcohol had experienced at least one blackout in their lives, and around 40% reported at least one MBO in the preceding year. Beyond prevalence, the authors were interested in factors related to blackouts. For example, they found an association between experiencing three or more blackouts, early onset of drinking, and heavy drinking. From there, interest in the topic revived. Progress since has been summarised in a series of review articles (H. Lee et al., 2009; Rose & Grant, 2010; Wetherill & Fromme, 2016; A. M. White, 2003) which have investigated the neural mechanisms underpinning the MBO experience, and the social consequences.

The patchy history of MBO research is puzzling for two reasons. Firstly, early theories of alcoholism highlighted alcohol-induced memory blackouts as central to the disease, viewing them as a strong predictor for, or symptom of, alcoholism. While there is no evidence of direct causality, MBOs likely have consequences for cognition and brain health which could exacerbate deficits seen in alcoholics. Since so much focus is placed on causes, intervention and treatments of alcoholics in the literature, it is surprising that MBOs appear to have been dropped from this area of study. Secondly, MBOs can and should be considered a marker of an extreme binge-drinking event. The binge-drinking literature is vast, yet concerningly, MBOs are mainly overlooked within the field or often relegated to a side issue (Courtney & Polich, 2009; Crews et al., 2000; Herring et al., 2008; Hunt, 1993; Kuntsche et al., 2004; J. W. Miller et al., 2007). While MBOs can happen to anyone given the right circumstances, frequent MBOs are arguably a sign of problematic drinking patterns, even if not alcoholism, and therefore should be considered as a trigger for instigating behavioural interventions. Further, as previously mentioned, we do not yet know whether repeated MBOs leave an enduring anatomical or cognitive deficit. Therefore, deepening understanding of their causes, as well as both acute and chronic damage, is a worthy endeavour in its own right.

In summary, MBOs are caused by a rapid spike in BAC% which leads the transfer of information from the hippocampus to long-term storage to be disrupted. Higher levels of BAC% are associated with *en bloc* MBOs, an apparent complete block of informational transfer, and lower levels with *fragmentary* MBOs, or partial blocks. They do not involve unconsciousness, are not exclusive to alcoholics, nor are they a predictor of developing alcoholism (although they are highly prevalent in alcoholics). They are associated with gulping drinks, and excessive consumption over a short period. In other words, binge-drinking.

Binge-drinking

Consumption of alcohol comes with a range of potential health harms and, as such, governments publish guidelines with suggested weekly drinking limits. These recommendations vary per country, but in the UK are currently no more than 14 units of alcohol per week both for men and women (Department of Health, 2016). It is further suggested that drinkers spread this consumption over at least three days across the week. This spread allows the body to process the alcohol in a more

controlled manner, reducing the likelihood of harms including some cancers (Sarich et al., 2021) and overall mortality (Jani et al., 2021). Binge-drinking is the term used to describe drinking more than the recommended average daily quantities in one single session (Courtney & Polich, 2009). Exactly how much alcohol per session, whatever length a "session" is supposed to be, is classed as binge-drinking also varies by country and gender but, for example, for males can range from 5 units in the United States (National Institute on Alcohol Abuse and Alcoholism, 2004) to 8 units in the UK (Department of Health, 2016). Although, within the UK, middle aged adults are the age group who consume the most alcohol spread across a week (Giles & Robinson, 2018), binge-drinking is particularly prevalent in younger drinkers (Office for National Statistics, 2018). This may reflect the difference in life stages between the under -25s, and the 55-64-year-old age groups. For example, it appears that older adults drink in smaller quantities per drinking session but perhaps enjoy a few drinks most evenings, whereas younger people may binge at the weekend but then abstain until the next weekend. Across a week, this can equate to older adults consuming more than younger binge-drinkers. Older adults may use alcohol as a stress reliever, a reward after a busy day, or simply a habitual accompaniment to a meal. On the other hand, younger people appear to associate drinking alcohol with intentionally getting drunk (Boekeloo et al., 2011; Kuntsche & Cooper, 2010) and social occasions (R. Brown & Murphy, 2020; Kuntsche et al., 2005). Indeed, evidence suggests that younger people 'mature out' of binge-drinking as they progress from studying and into the world of work or family responsibilities (M. R. Lee & Sher, 2018; Vaadal & Dahl, 2021). Additionally, as the brain matures, young adults are less likely to take part in risky behaviours, including substance use (Andrews et al., 2002; H. R. White et al., 2017), more able to engage inhibitory controls (Bedard et al., 2002; Christ et al., 2001), and less likely to be influenced by peers (Smith et al., 2015). This means that over the lifespan, the practice of regular binge-drinking may happen across a relatively short amount of time. However, since this time coincides with a period where young people are physically, emotionally and socially still developing, there are potential dangers attached. From our perspective, these would be to the development of the brain and cognitive functioning, but more broadly, these dangers could include an increased risk of sexually transmitted infection (H. L. Johnson et al., 2018), reduced academic

performance (Aertgeerts & Buntinx, 2002), and increased risk of injury (Sindelar et al., 2004).

Binge-drinking in adolescence and early adulthood is associated with a number of health-related harms. One particular concern surrounds brain health. The brain is known to continue developing until the mid-20s (Bava & Tapert, 2010; Blakemore, 2012; Crews et al., 2007), including dendritic pruning within the hippocampus (Selemon, 2013) and more general synaptic pruning across the pre-frontal cortex (Blakemore, 2008; Gogtay et al., 2004; Spear, 2013). This means that cortical development is occurring at the same time as a period when the brain is known to be sensitive to the effects of alcohol (Barron et al., 2005). Studies of adolescents who drink heavily show potentially long-lasting detrimental effects to the brain, including differences in grey and white matter volumes within the prefrontal cortex (De Bellis et al., 2005), and reduced performance in memory and learning tasks (Nguyen-Louie et al., 2016; Squeglia & Gray, 2016). Even drinking at levels which do not clinically meet the standards for alcohol use disorder (AUD) has been shown to produce changes to cerebellum volume (Kekkonen et al., 2021), suggesting that recreational binge-drinking could have irreparable consequences for the brain. Compared to control participants, individuals who binge-drink have also been shown to exhibit differences in ERP (Event Related Potential) component latency (Ehlers et al., 2007) and amplitude (Crego et al., 2012; Nichols & Martin, 1996) across various cognitive tasks. Evidence for both structural and cognitive performance deficits shows that binge-drinking therefore can impact normal brain development and functioning.

Binge-drinking and blackouts

Binge-drinking to extreme proportions can often lead to the experience of an alcohol-induced blackout (Hermens & Lagopoulos, 2018; Wetherill & Fromme, 2016). A number of factors contribute to the onset of an MBO, but the primary mechanism is the rapid consumption of large quantities of alcohol leading to a spike in blood alcohol levels (Goodwin, Crane, et al., 1969a; Rose & Grant, 2010; A. M. White, 2003), a mechanism facilitated by binge-drinking. There does not appear to be a specific BAC% which guarantees the onset of a blackout, this can vary based on a number of factors including speed of drinking, gender, body mass index, and time of last meal (Rose & Grant, 2010). However, there is a dose dependent relationship between BAC% and the

type of MBO experienced (A. M. White et al., 2004). Typically, *fragmentary* blackouts occur at lower levels of BAC% (Hartzler & Fromme, 2003; Peterson et al., 1990), with one study suggesting that *en bloc* MBOs require a BAC of around 0.16% (M. B. Miller et al., 2018). The body continually processes alcohol consumed via dispersion in body fluids (Cederbaum, 2012; Swift, 2003). Drinking, for example, a couple of glasses of wine with a meal and over the course of several hours is unlikely to lead to a blackout. However, drinking games, pre-drinking and bingeing all create circumstances where BAC% could rise rapidly. Since these behaviours are particularly prevalent in younger adults (Hermens & Lagopoulos, 2018; Lees et al., 2019), it follows that MBOs are experienced frequently in the same population. For example, many studies have shown a link between binge-drinking and MBOs (Hingson et al., 2016; Wombacher et al., 2019), particularly within student populations (A. M. White et al., 2004). It is possible that, beyond the social reasons for young adults wishing to drink in this way, that the sedative effects of alcohol have less impact on younger individuals (Silveri & Spear, 1998) which means that they have the capacity to continue drinking beyond a point when older adults may have fallen asleep. Anecdotally, other strategies include napping (colloquially termed a 'disco nap') before a night out in order to keep going for longer, or making themselves vomit after pre-drinking in order to handle continued consumption when out. Together, these behaviours could increase the likelihood of experiencing MBOs.

Since evidence suggests that binge-drinking is detrimental to brain development in young adulthood (Kekkonen et al., 2021; Petit et al., 2014; Spear, 2018), it is likely that frequent MBO experiences would exacerbate these effects. An MBO alters normal neural functioning by disrupting the processing and transfer of information from the hippocampus, specifically CA1, to cortical regions (A. M. White, 2003). One question not answered in the literature is whether frequent, repeated assault to hippocampal processing caused by the experience of an MBO leaves a lasting detriment to memory performance once sober, or in the future. We do know that individuals with Korsakoff Syndrome, a condition associated with alcoholism, experience anterograde memory amnesia (Fama et al., 2012; Kessels et al., 2008; Popa et al., 2021). This condition develops following a deficiency in Vitamin B1 (thiamine), often due to alcohol supplanting the calorie intake from food. Neuroimaging of Korsakoff's patients reveals structural changes to the brain in grey and white matter (Kril & Harper, 2012),

particularly in regions known to support episodic memory (Aggleton & Brown, 1999). It is likely that brain areas affected by alcohol in people who blackout and Korsakoff's patients show similar structural abnormalities, however, we must be cautious in comparing the acute amnesic episodes induced by alcohol we call MBOs with a more severe long-lasting amnesia caused by a thiamine deficiency.

In order to better understand MBOs and some of the experiments presented in the thesis, which examine memory performance before alcohol, after alcohol, and after MBO, it is necessary to take a closer look at current memory theories. Before we do this, let us briefly consider the factors influencing binge-drinking and blackout experiences in Scotland, and highlight the opportunities presented by conducting alcohol research in the UK.

Scotland's relationship with alcohol

Scotland has often been dubbed as the sick man of Europe (Mccartney et al., 2012). A high rate of excess deaths (Minton et al., 2017), caused by obesity levels (Tod et al., 2017), disease (Steel et al., 2018), drugs (Parkinson et al., 2018) and other lifestyle factors (Lu et al., 2018) contribute towards a low life expectancy when compared to other developed countries (Fenton et al., 2019). Additionally, deaths directly attributable to alcohol in Scotland continue to increase annually (Ramsay, 2021). Scottish culture seems to accept alcohol as a non-problematic tool for social bonding (Emslie et al., 2013, 2015; Leavy & Alexander, 1992) and - anecdotally at least – an integral factor of a 'good night out'. In response to these well documented problems, the Scottish Government introduced a minimum price per unit for alcohol in May 2018 (Scottish Government, 2018). This policy was designed to reduce affordability of cheaper alcohols thereby reducing consumption of alcohol overall and has seen success in reducing alcohol purchasing (Anderson et al., 2021; Robinson et al., 2021). Since alcohol-related deaths continued to rise in 2020 (Ramsay, 2021), it may yet be too early to fully measure the success of this policy. Arguably, with the recent Covid-19 pandemic affecting mental health and rates of alcohol consumption (decreased for the general population, increased in people with alcohol dependency (Alcohol Change UK, 2020; Jacob et al., 2021; J. U. Kim et al., 2020; Oldham et al., 2021), measuring how this policy would reduce alcohol-related harm will not be possible. Further, while alcohol consumption may be going down overall, problematic levels of

binge-drinking are still prevalent in younger adults (Office for National Statistics, 2018).

The majority of research surrounding binge-drinking and alcohol-induced blackouts has been conducted within the United States of America. This research has produced a wealth of understanding relating to the harms caused by alcohol, the mechanisms for MBOs, and the impact to the brain of binge-drinking (Hingson et al., 2009; Wetherill & Fromme, 2016; A. M. White, 2003). However, there are a number of differences between American culture and British culture. Firstly, the legal drinking age within America is 21 (CDC, 2020), whereas in the UK this is 18 (Gov.UK, n.d.). Underage drinking is a problem internationally (Inchley et al., 2018), yet this age limit means that alcohol cannot be legally given in lab-based studies within the USA. Therefore, even in studies where young adults/students have been recruited, the lowest possible age of participant would be 21. In the UK, young people typically begin their university education at 18 enabling research in a younger demographic. It is notable that in Scotland students may even be attending University at the age of 17. Therefore, there is a gap in the literature across a crucial period of neural development which can be addressed by UK based studies. Secondly, young people appear to have a different cultural relationship with alcohol in Scotland compared to America (Leavy & Alexander, 1992). For example, Deik & Meilman (1996) compared student drinking in Scotland to America and found that Scottish students drink more heavily than American students, that this is normalised in Scottish culture and accepted by the police. Further, they also showed that American students are more likely to drink-drive, and exhibit rowdy and aggressive behaviours, whereas Scottish students, whilst they started drinking at a younger age, had fewer incidences with authorities from their drinking behaviour. That said, Scottish students reported more hangovers, memory loss and regrettable behaviour than American students. On a broader European basis, alcohol is also viewed from different cultural standpoints than in the USA. For example, in France it is common for young people to have wine with a meal from a younger age (Ritchie & Valentin, 2011), and other southern European countries similarly have a higher tolerance of normal drinking levels (Nordlund & Østhus, 2013). Thus, much of the research on alcohol expectancies, norms and culture derived from US based studies is not entirely applicable to European, UK, or indeed Scottish people.

To the best of my knowledge, there have been no laboratory based experimental studies conducted in Scotland which investigate alcohol consumption, binge-drinking, or alcohol-induced memory blackouts. Further, in comparison to the majority of studies, which are US based, there is a 3-year gap during which young people are binge-drinking in the UK that is not addressed in the existing literature. The brain is still developing during this period, and alcohol consumption at age 18 may affect the brain differently than at age 21. It is therefore highly relevant to address these issues and to consider the effects of alcohol on memory.

Memory

In psychology the word 'memory' gives the impression of a single cognitive system. However, 'memory' is an umbrella term used to describe a collection of cognitive processes which together contribute to our perception and understanding of the world around us, and our place within it. Evidence from one of the most famous neuropsychological cases, patient H.M. (Corkin, 1968; Milner et al., 1968), whose hippocampus was removed during elective surgery for severe epilepsy, highlighted that memory is not a unitary system. Specifically, he developed a lifelong anterograde amnesia, but could remember events pre-surgery and critically, displayed an intact short-term memory (that is, passive retention of details lasting only a few minutes). Memory enables us to perform daily tasks, to learn and recall new information, to reflect on past experience, and to hold meaningful conversations. Competing memory theories have developed over time (see Ferbinteanu, 2019; or Squire, 2004, for reviews), as researchers attempted to define different types of memory mechanisms (for example, declarative memory, implicit or explicit memory). At its most simplistic level, memory is the collective term used for the cognitive processes which act to gather information received from the world around through sensory inputs, to store this information, and make it accessible for later use (Baddeley, 1997). Explicit memory refers to the ability to consciously recall knowledge or experiences, and implicit memory to the unconscious ability to perform learned behaviours (Schacter et al., 1993). Within the category of explicit memory comes further theories which include working memory (Baddeley & Hitch, 1974), semantic memory (Jones et al., 2015), and episodic memory (Tulving, 1983). In depth discussion of all short and long-term memory theories and models is beyond the scope of this thesis. Therefore,

discussion will focus on the memory system most affected by alcohol – episodic memory.

Episodic Memory

Often said to be a uniquely human trait (although see Clayton et al., 2001 for an opposing position), episodic memory allows individuals to "travel back in time and mentally relive events from their past" (Tulving, 2002; see Baddeley, 2020 for a more recent review of the field). To facilitate this ability, new information must be stored, readily accessible for future use. Further mechanisms must then enable efficient recovery of the required information at the appropriate time. This two-stage process relies on *encoding* (storage) and *retrieval.* Underpinning these theoretical processes, are the neurobiological mechanisms which support them. These two processes will be discussed in turn.

Encoding

Encoding could be described as the process during which information is gathered, sorted, and stored in the brain for future use. In brief, the hippocampus collates information gathered from sensory inputs, binds spatiotemporal details together (Ekstrom & Yonelinas, 2020), and transfers the bound events out towards long term storage in the cortex (Aggleton & Brown, 1999; Clewett et al., 2019). Binding and storing related contextual information together allows for the recall of details such as the who, what, when, why, and where of the event. This level of detail means people can not only recall facts surrounding the event but can imagine themselves being there. Other brain regions involved in this process include the frontal and medial temporal lobes (Squire & Zola-Morgan, 1991; Wheeler et al., 1995), and the entorhinal cortex and parahippocampal gyri (Davachi & Wagner, 2002). The literature suggests that encoding can be enhanced at the experimental level through various strategies, for example semantically linking word lists or including contextual information (see Lockhart, 2002). Adding 'depth' to encoding processes by requiring increased cognitive effort from participants during a task is a well-documented approach for strengthening a memory trace, and thereby improving chances of retrieval (Craik & Tulving, 1975). This theory is supported by more recent neuroimaging work which showed that greater engagement of the perirhinal cortex and hippocampus is

correlated with future ability to recollect stimuli (Davachi, 2006). Taken together, this evidence shows that contextual details surrounding an event can strengthen the bound memory, thereby improving chance of remembering later.

Alcohol is known to specifically impair the process of binding and storage by temporarily disrupting normal hippocampal functioning in the CA1 region (A. M. White, 2003; A. M. White et al., 2000), which can lead to an anterograde, alcohol-induced amnesia (MBO). Although alcohol easily passes through the blood brain barrier (Zeigler et al., 2005), the neurobiological mechanisms for this specific impairment vary depending on alcohol dosage (Rose & Grant, 2010). For example, lower doses appear to impair $GABA_A$ receptors whereas higher doses act upon NMDA glutamate receptors (Bisby et al., 2010; A. M. White et al., 2000). Interestingly, while alcohol impairs the encoding of new information (Söderlund et al., 2007; Weafer et al., 2016), it is also said to have a retroactive effect on information received immediately prior to alcohol consumption, that is, memory for information before alcohol is ingested is more easily recalled than during intoxication (Wixted, 2004). Further, it has been suggested that there may be a state-dependent effect of learning while intoxicated (Goodwin, 1995), although studies have not yet corroborated this claim (Weissenborn & Duka, 2000). A study by Söderlund et al. (2007) was the first to investigate the effects of alcohol during encoding on associated brain regions using functional neuroimaging. Participants in two groups (control and alcohol) were presented with different stimuli types (words, phrase-words, object-pairs and face-name pairs), chosen for the range of brain regions required for encoding. Those sober and intoxicated completed the encoding phase of the study and then returned the following day for recall testing. Within stimuli type comparison revealed that – even when recall performance was similar – encoding differed between groups across most tasks. In general, both groups showed activation in the left prefrontal regions during verbal encoding, but the alcohol group displayed decreased activation in parahippocampal (objects), and right inferior frontal gyrus (face-names) regions. The authors suggest that alcohol acts to impair activation of encoding neural structures for some stimuli but can spare others.

Participants in the Söderlund et al. (2007) study were moderate social drinkers and the level of BAC% aimed for was equivalent to that of an adult male drinking a

bottle of wine. While this amount would induce intoxicated effects, it is unlikely to instigate an MBO. Further, whether repeated MBO experiences leave a lasting impairment on hippocampal-related encoding while sober remains unknown. Evidence from a study of non-alcoholic clinical patients with a diagnosis of amnestic Mild Cognitive Impairment (MCI), a pre-clinical stage of Alzheimer's disease (Petersen, 2005), found that reduced grey matter in CA1 impaired encoding (Fouquet et al., 2012). Since MCI tends to increase as neural atrophy progresses, this suggests that the ability to encode declines over time, rather than an all or nothing threshold. Evidence also shows reduced grey matter in various brain regions in alcoholics (Jernigan et al., 1991), as well as younger heavy drinkers (El Marroun et al., 2021). It is therefore possible that, like the slow cognitive and neural decline in dementias, frequent assault from alcohol and binge-drinking may gradually impair encoding even while sober. More research is required to address the contribution of frequent MBOs to permanent neural damage.

Retrieval

Once encoded, information can be retrieved either by recognition of previously encountered stimuli, or by self-generated recollection, and an extensive literature supports both processes (for example, Bhatarah et al., 2008; Gardiner & Java, 1990; Jackson et al., 2021; Rugg & Yonelinas, 2003, etc.). Recognition of previously encountered stimuli could be accompanied by full recollection of the event (Tulving, 2002), or simply the knowledge that you have seen something before. Recognizing an item as having been previously encountered, that is, 'old', but failing to retrieve any supporting information (for example, where or when the item was first seen), is described as a *familiarity* response (Gardiner, 1988). Alternatively, an 'old' item remembered alongside accompanying contextual information, is a *recollection* response. These response types are often measured in laboratory studies by way of 'remember/know' paradigms (Tulving, 1985), a controversial method of asking participants to determine the quality of their recollection for a given stimuli. Dual-process models of recognition (Diana et al., 2007; Yonelinas, 2001, 2002) are pervasive in the memory literature, and suggest dissociable cognitive processes (however note the opposing single-process theory for example, Donaldson, 1996; J. C. Dunn, 2004; Slotnick & Dodson, 2005). Recollection is thought to be a threshold process (Murray et

al., 2015), where above threshold suggests that some qualitative information relating to the item has been retrieved. In contrast, below threshold leads to a sense of familiarity but with an absence of contextual details (see Yonelinas, 2002). Recollection and familiarity are believed to rely on separate brain regions, with the hippocampus supporting the transfer, storage and retrieval of details which are necessary for recollection, and the perirhinal cortex implicated in familiarity responses (Aggleton & Brown, 1999; Bisby et al., 2010; Brown & Aggleton, 2001). Additional support for this dissociation has been seen in clinical patients where hippocampal damage resulted in fewer recollection responses (for example, Turriziani et al., 2008).

Recall tasks are those where participants are asked to study stimuli then, unlike recognition, to recall without further cue. For example, free recall involves a list of words being presented to participants to memorise, and then recall without prompt later. Alternatively, a serial recall task would ask that in addition to recalling the words, that they be recalled in order of presentation. The episodic memory retrieval literature appears to have moved away from early recall studies (for example, Glanzer, 1968; Gruenewald & Lockhead, 1980; Tulving, 1967; Wixted & Rohrer, 1994) and is now disproportionally biased towards recognition studies both in humans (for example, Bird, 2017; Eichenbaum, 2017; Ratcliff et al., 2016; Roediger & Tekin, 2020) and also in non-humans (for example, Alvarado et al., 2017; Cruz-Sanchez et al., 2020; May et al., 2016; Toyoshima et al., 2018). Possible reasons why this could be the case are the advent of modern neuroimaging methods and also the existence of dual process theories of memory. Nevertheless, the ability to spontaneously recall events at will is central to human memory and worthy of study. This type of task can present a greater challenge than recognition studies due to the increased cognitive demand required with no cueing. However, stored events which have more detailed semantic, or contextual information are more easily recalled than events without (Craik & Tulving, 1975).

Recalling the details which surround an event are intrinsic to the ability of fully recollecting the experience. An influential theory surrounding the importance of contextual details was developed during the 1980s by Marcia Johnson and colleagues (1993). Their 'Source Monitoring' framework suggested that the 'source' (for example,

location, voice, smell) of an event provided the qualitative details required for people to understand that the event happened and was not imagined. Lack of source detail is said to reduce the chances of fully recalling events, for example, the experience of knowing someone told you something, but not remembering who that was, arguably induces a feeling of familiarity whereas retrieving the source of the information provides a full recollection of the event. Failure to bind contextual details at the time of the event can occur if people are distracted or stressed (M. K. Johnson et al., 1993), thereby making full retrieval difficult or even impossible. It has been suggested that fragmentary MBOs in part reflect source memory failings (Hartzler & Fromme, 2003), that is, impaired encoding of contextual detail while intoxicated which leads to retrieval of fragmented information. We know that, while intoxicated, individuals are less able to perceive environmental cues, and that their attentional focus narrows, a state known as 'alcohol myopia' (Steele & Josephs, 1990). Therefore, it appears more likely that source memory is a victim of excessive alcohol consumption rather than having a causal role in MBOs. Lack of encoded contextual detail is plausibly a factor in failing to fully recollect events once sober and, given that alcohol is known to impact episodic memory, any retrieval difficulties experienced following a fragmentary MBO would likely be caused by general encoding deficiencies rather than a specific source impairment.

As mentioned previously, alcohol impacts functioning within the hippocampus, including the ability to transfer information from short to long-term memory (A. M. White, 2003). Therefore, individuals who experience a high frequency of MBOs are repeatedly inflicting this disruption to normal functioning on neural structures. If lasting damage to the region is imparted, these individuals could exhibit poor recollection both when sober and after drinking alcohol. Recall that one way of quantifying the amount of recollection in a recognition memory task is to include a '*remember/know*' judgement. Curran and Hildebrandt (1999) claimed to be the first study which investigated recollection in social drinkers while under the influence of alcohol. They hypothesised, and indeed found, that alcohol would reduce *remember* responses (clear recollection of the earlier presentation of a stimulus) but have no effect on the familiarity response, *know* (a feeling of recognition for a stimulus but without the ability to recall its earlier presentation). Subsequent alcohol and recognition memory work has found mixed results, possibly due to the variation in

tasks (S. Ray & Bates, 2006). For example, some studies found no effect of alcohol on recognition tasks using a mix of old and new stimuli at test (Goodwin, Powell, et al., 1969; Hashtroudi et al., 1984), however it has been suggested that this may be because familiarity masked observable recollection deficits (S. Ray & Bates, 2006). Both aforementioned studies relied on a simple yes/no recognition judgment in response to stimuli therefore, it is impossible to separate the processes of recollection from familiarity or simply guessing. Unsurprisingly, the Goodwin, Powell, et al. (1969) study, being reflective of the times, was confounded by half the stimuli being classed as neutral, even though they were pictures of models from a mail order catalogue, while the other half were "emotional" physiologically arousing pictures collected from a nudist magazine. I leave it to reviewers to imagine how this may have affected the results. Studies which did find an effect of alcohol on recognition used stimuli where the old/new items were very similar, or where participants had consumed a high alcohol dose (Maylor et al., 1987; Wickelgren, 1975). Interestingly, neither Curran and Hildebrandt (1999) nor Ray and Bates (2006) found an effect of alcohol on false alarms, the false identification of previously unseen items. This is surprising as it could be expected that a decrease in accurate recollection may be accompanied by an increase in falsely recalled items, particularly when those items were similar to old stimuli.

In sum, alcohol detrimentally impacts on the encoding and storage of information by the hippocampus which, in turn, reduces potential recollection for previously experienced stimuli. While recognition memory is less impaired than free recall, this may be due to the reduced cognitive effort required following cueing, or that familiarity for an item masks true recollection. One question unanswered is why alcohol impairs episodic encoding related structures but spares other aspects of memory (procedural, declarative, etc.,) and cognition (for example, language comprehension). Whether this is an evolved preventative mechanism, or simply that these neural structures are more vulnerable to the effects of alcohol, is unknown.

Methods of testing memory: Behavioural

The study of cognition involves a wide range of experimental approaches, including eye-tracking, functional neuroimaging, electroencephalography, near-infrared spectroscopy and more. However, central to most methods – and a suitable

method in its own right – is behavioural testing. Behavioural measures rely on participant responses to stimuli, with analysis of reaction time or accuracy typically employed. In memory research, behavioural testing has a number of advantages. Firstly, from a technical standpoint, it can be relatively inexpensive and straightforward to conduct due to the lack of neuroimaging equipment required. In fact, online testing platforms (Rezlescu et al., 2020; Stoet, 2022) increasingly allow for wider participation beyond a laboratory, with the potential to increase replicability and reliability of findings. Secondly, and depending on the research question, testing accuracy of words recalled through either recall or recognition paradigms has been shown to provide a reliable measure of memory function without the need for neuroimaging (Bhatarah et al., 2008; Bisby et al., 2010; Cheke & Clayton, 2013). Much of the theorical framework relied upon by memory researchers today stems from behavioural studies conducted decades ago (for example, Baddeley & Hitch, 1974; Craik & Tulving, 1975; Tulving, 1967). However, while a behavioural experiment can offer clear evidence of quantity and quality of recollection it cannot uncover neuroanatomical structures involved in memory (see Yonelinas, 2002, for a review of behavioural and neuroimaging studies), or the latency of these memory processes.

Methods of testing memory: Electroencephalography (EEG)

One well established neuroimaging approach in the memory literature is the use of electroencephalography (EEG) and the Event Related Potential (ERP) method (Allan et al., 1998a; Wilding & Ranganath, 2012). We can measure the electrical activity of the brain through placement of electrodes on the scalp. The signal recorded is the summed activity of all inhibitory and excitatory post-synaptic potentials, and large-scale changes in this signal are thought to reflect the contributions of thousands of simultaneously firing neurons (Cohen, 2017; Olejniczak, 2006). While intracranial, single cell recording methods are suitable for some animal and clinical purposes, scalp recordings are non-invasive and therefore preferable for most psychological based research with humans. This method of investigation began in humans with psychiatrist Hans Berger in 1929, who used a single electrode to record electrical activity from the scalp, and then to plot the changes in voltage produced over time (Berger, 1931). While tools for analysis have moved on considerably in the intervening years with the development of computers and specialist software programmes, the basic principles

of recording, and the questions surrounding interpretation of the signal, still apply (Cohen, 2017; Kaiser, 2005).

Recording of EEG signal requires metal electrodes (usually tin, or silver/silver chloride) to be secured to the scalp either by direct adherence, or via tight fitting caps embedded with multiple electrodes. Regardless of the method of attachment, continuity of location of individual electrodes is important across participants to provide comparative recordings. However, while researchers can ensure that electrodes are approximately located in the same position on different participants, it should be noted that the signal detected does not necessarily reflect the cognitive processes occurring in the anatomical locations immediately beneath the electrode (Luck, 2014). The neocortex consists of columns of neurons (Mountcastle, 1997), providing an anatomical structure favourable for scalp recording of electrical signal (Luck, 2014). However, between neuron and scalp, the signal can be distorted and smeared by bone and dura mater (Kaiser, 2005). Further, what is a three-dimensional source (the brain) projects a two-dimensional signal (Olejniczak, 2006), creating what is known as the 'inverse problem'. This means that attributing an anatomical location to a recorded signal is problematic. It may sound like EEG is of limited use in furthering the understanding of cognitive processes however researchers have developed methods of isolating recorded waveforms from specific electrode locations which appear following presentation of certain stimuli, or task demands (see Handy, 2005; or Luck, 2014, for full descriptions and discussion of the technique), thereby making comparison between cognitive processes possible.

Despite the aforementioned problems, EEG is a particularly suitable method of neuroimaging for cognitive research for several reasons. Firstly, due to its high temporal resolution (Luck, 2014; Wilding & Ranganath, 2012), scalp recorded activity can be time-locked to experimental task demands with millisecond accuracy. Secondly, after recording, EEG output can be segmented by event (for example, stimulus presentation or participant task response). This allows the segmented EEG trace for similar events, for example accurate recognition of an item, '*hits*', to be averaged together. This is indeed the principle of the ERP technique: background noise is reduced by the averaging of many similar trials together, since averaging works like a filter, creating an epoch in response to the stimulus that potentially can isolate

cognitive processes. Alternative neuroimaging methods can provide a more visual picture of changes in the brain, for example using hemodynamic approaches such as functional magnetic resolution imaging (fMRI) or functional near infra-red spectroscopy (fNIRS). These approaches rely on detecting vascular changes based on the principle that cognitive activity in a brain region will be supported by additional blood flow providing oxygen. While extremely useful for some research [and combining functional imaging with EEG is becoming more popular with advances in technology (Freeman et al., 2009; Gotman et al., 2006; Scrivener, 2021)], each method remains valid independently of the other. EEG laboratory equipment is less expensive than fMRI and its high temporal resolution offers the ability to time-lock neural responses to experimental events. Therefore, this remains a useful tool for neuropsychological research.

One problem with EEG signal is that it includes electrical activity from a variety of sources. For example, in addition to recording the signal of interest, it could also include several other cognitive processes which were simultaneously occurring. For example, perception of sounds along with visual identification of a word, while also thinking about a mundane personal matter. To derive a 'process pure' signal, a technique of averaging time-locked waveforms was developed beginning in the 1930s and which enabled researchers to identify a specific ERP (H. Davis et al., 1939; P. A. Davis, 1939; cited in Luck, 2014). Over time, various ERP components were discovered, for example the P3 (Sutton et al., 1965) connected to decision making, the visual N1 (Haider et al., 1964) and the language based N400 (Kutas & Hillyard, 1980), and the method was therefore adopted by researchers in various neuropsychological fields. In particular, EEG and the ERP technique has been widely utilised for studies of memory processes (see Wilding & Ranganath, 2012, for a detailed review of ERP and episodic memory research). Careful study design allows episodic memory researchers to test both encoding (for example, Otten et al., 2001; Otten & Rugg, 2001) and retrieval (Friedman & Johnson, 2000) through either recognition (Addante, Ranganath, & Yonelinas, 2012; Rugg & Curran, 2007) or recall paradigms (B. R. Dunn et al., 1998; Wiswede et al., 2007).

ERPs and Recognition Memory

ERP studies of recognition memory have predominantly focused on the analysis of two components, commonly referred to as the 'mid-frontal effect', and the 'left parietal effect'. These components are suggested to be the neural indexes of *familiarity* and *recollection* respectively (see earlier discussion on p.28) and have been recorded in response to a variety of stimulus types including words (Wilding & Rugg, 1997), images (Ranganath & Paller, 2000), and faces (Johansson et al., 2004; MacKenzie & Donaldson, 2007). Although commonly referred to within recognition memory literature as stable across time windows (300-500ms; 500-800ms), and across electrode sites/scalp locations (frontal regions including F3; left parietal including P3), closer inspection of experimental methods show that many researchers apply slightly different measurement parameters; that is, when referring to either the mid-frontal or left parietal component, there can be differences in both the time-windows and electrode sites which are chosen for analysis across studies.

Typically, the mid-frontal effect is observable from around 300–500ms post stimulus presentation and is characterised by a difference in mean amplitude between previously studied old, and previously unseen new, items across mid to frontal electrode sites. However, the component has also been referred to as the as the FN400 (T. Curran, 2000), the medial frontal old/new effect (Friedman & Johnson, 2000) and the early frontal old/new effect (Mecklinger, 2000) due to both variations in the location in which effects are maximal, and functional interpretations. Although the component is suggested to reflect early stimulus recognition without additional contextual information (that is, *familiarity*), some researchers did posit an alternative hypothesis, suggesting that an N400 conceptual priming effect (that is, the semantic processing of meaningful stimuli; Olichney et al., 2000; Paller et al., 2007; Voss & Paller, 2006) may be required for familiarity-based recognition, and indeed be reliant on the same neural generators (Yonelinas, 2002). However, see Voss et al for a dissociation between the two (Voss et al., 2010).

The left parietal recollection effect is more consistent in location than the earlier mid-frontal effect and is normally present across parietal electrode sites (predominantly P3, although the other electrodes included in an analysis may vary) on the left hemisphere (although see Mecklinger, 2000; or Allan & Rugg, 1997, for

discussions of scalp location and variations resulting from specific task demands). This effect is characterised by an ERP showing more positive mean amplitude for old (previously studied) than new (previously unstudied) stimuli (Wilding & Ranganath, 2012). Despite this increased consistency in scalp location when compared to the mid-frontal effect, exact timings can vary beyond the broadly expected 500-800ms window; for example, some researchers describe the effect as beginning at around 400ms post-stimulus and lasting for up to a further 400-600ms (Allan et al., 1998b; Allan & Rugg, 1997). Other reported time windows have included 400-800ms (T. Curran, 2000), 600-900ms (Donaldson & Rugg, 1998), 500-700ms (Pilgrim et al., 2012) and 600-800ms (Addante, Ranganath, & Yonelinas, 2012), amongst others. These differences highlight that although the component is widely argued to be consistent, researchers regularly vary the time-windows and electrode locations based upon the data rather than restricting analysis to a conventional approach.

Although dual process theory positions familiarity and recollection as distinct memory processes and, by extension, the two ERP components previously discussed, this does not preclude the possibility that – to some extent - the processes operate in tandem rather than serially. For example, in a study of word recognition (T. Curran, 2000), an early FN400 effect was measured over 300-500ms, and the later parietal component across the overlapping 400-800ms window. Moreover, a review by Allan et al. (1998b) suggests that the onset of both components occurs at around 350-450ms post stimulus presentation but that the effects are dissociable due to their scalp location and duration. It is therefore possible that while neural generators for these two effects are different, the more frontal effect is reflective of a cognitive processing operation which then runs in parallel (or at least overlaps) with the process of recollection. While no memory researcher would suggest that the two processes run serially, they nevertheless attempt to ascribe cognitive functions to ERP waveforms in a serial fashion (300-500ms familiarity followed by 500-800ms recollection followed by 800-1200ms post retrieval monitoring) to explain memory operations.

Advancing understanding of whether these two memory processes (familiarity and recollection) are distinct may be achievable by combining multiple neuroimaging methods, or by applying a data driven approach to well-designed studies. For example, Hoppstädter et al. (2015) chose to analyse an early effect at electrode FCz across a 350-

550ms window, and later at electrode P5 between 580-750ms – both non-traditional time-windows and electrode sites - following inspection of data gathered on a joint EEG and fMRI recording study. A study by Addante, Ranganath and Yonelinas (2012) initially reported results from 400-600ms at Fz and 600-800ms at P3, but then reanalysed more central locations where effects were maximal (Cz and Cp5) following a data driven examination over the same time-windows. The same lead author (Addante et al., 2012) also reported results from 400-600ms at FC1, and 600-900ms at P3, in a word recognition study from the same year. Finally, by comparing effects over strings of electrodes, Wolk et al. (2004) found effects maximal over a central electrode cluster (Cz, C3, C4) between 300-550ms compared to frontal electrode clusters (FP1, FPz, FP2; and F3, Fz, F4). In the case of recognition memory ERP studies therefore, the application of a data driven approach, based on examination of data either by multiple recording methods or differing analysis techniques, rather than using a priori time-windows and electrodes, is an established method of analysis within the literature. In sum, although researchers broadly refer to these two components as dissociable and stable over both time (300-500ms; 500-800ms) and electrode sites (frontal and left parietal), this is an over-simplification based upon the accepted theoretical underpinning of the work (Rugg & Curran, 2007).

ERPs and Alcohol

The ERP method has been applied to a variety of alcohol research and has both experimental (for example, López-Caneda et al., 2014; Park & Kim, 2018; see Petit et al., 2014, for a review) and clinical utility (Coutin-Churchman et al., 2006; Krauss & Fisher, 1992; Mumtaz et al., 2018). Alcohol is known to inhibit normal neurotransmitter functioning within the brain, for example, glutamate (Dodd et al., 2000; Gonzales & Jaworski, 1997; P. S. S. Rao et al., 2015) and GABA, therefore altering both neuronal excitation and inhibition (Gonzales & Jaworski, 1997; Krauss & Fisher, 1992). These alterations change the pattern and strength of neuronal firing and in turn, the EEG signal observable at the scalp. There have been a variety of applications of EEG methods in the alcohol literature, for example it has been adopted as a complimentary method of screening individuals suspected to have alcohol use disorder (AUD), when partnered with standard screening methods (P. M. Miller et al., 2006). Work is ongoing

to develop the use of EEG as a tool to identify alcoholics at risk of relapse (Bauer, 2001; Wan et al., 2010), although this is not yet sufficiently reliable for clinical use (Mumtaz et al., 2018). Interestingly, ERP analysis has also been adopted as a method for investigating neural familial markers of alcoholism (Hill et al., 1998; Porjesz et al., 2005; Porjesz & Begleiter, 2003; Reese & Polich, 2003). Many of these clinical uses rely on the reductions in amplitude seen in standard ERP components following consumption (Courtney & Polich, 2009). In particular, markers of attention and inhibition have been shown to exhibit reliable differences following alcohol (Bartholow et al., 2003; Carbia et al., 2018; Curtin & Fairchild, 2003). These include the N1 (Campbell & Lowick, 1987; Jääskeläinen et al., 1995) and P3 (Grillon et al., 1995). This therefore provides an opportunity for experimental paradigms to target the same components to better understand the effects of alcohol on social drinkers, rather than just alcoholics.

ERPs, Alcohol and Memory

Given the vast literature on memory processes which use EEG, and the work on alcohol and ERPs, combining these areas is a valid approach for investigating the effects of alcohol on memory. While early use of EEG for alcohol and memory studies was not fruitful (Freemon et al., 1971), some later studies have had more success. For example, in the field of working memory, participants who were sober, but who were binge-drinkers displayed larger P3 amplitudes than controls in a study by Park and Kim (2018). Further, Zhang, Begleiter and Porjesz (1997) found differences in an ERP component approximately 250ms post-stimulus presentation in the right occipitotemporal region between alcoholics and controls, and in a separate study, a visual short-term memory task also found differences between alcoholic and control participants in occipitotemporal regions (Zhang, Begleiter, Porjesz, et al., 1997). There appears to be a surprising lack of EEG research investigating episodic memory and alcohol, and a complete lack of work which incorporates MBOs into the research question. While some authors interested in MBOs have employed fMRI (Berglund et al., 1989; Wetherill et al., 2012; Wetherill, Castro, et al., 2013), a review of the MBO literature by Wetherill and Fromme (2016) reported no EEG studies in the preceding 5-years. A slightly earlier review by Pressman and Caudill (2013) also reported no EEG studies with a search span reaching back to the 1960s. While conducting episodic

memory tests with participants who are in the process of experiencing an MBO would be a difficult task given ethical considerations, there is no such concern surrounding testing sober participants who experience frequent MBOs, or a short time after an MBO. These approaches could help uncover whether there are fundamental differences in ERP amplitudes or latencies between control and frequent MBO participants which would perhaps suggest underlying structural or cognitive differences in those who frequently blackout. It could also shed light on the duration of effects from an MBO, that is, how long it takes to recover to baseline cognitive functioning.

Thesis Goals

As demonstrated in this chapter, literature on memory performance of adolescents who binge-drink to the point of blacking out on a regular basis is sparse, and inconclusive. Further, studies which investigate the relationship between students and alcohol – both from a social and from an experimental perspective - within Scotland are either non-existent or out of date. The purpose of this thesis is therefore firstly to investigate the current relationship which young adults attending Scottish universities have with alcohol, specifically in relation to the influences on their drinking behaviours and the prevalence of MBOs. Secondly, the intention is to design and execute a series of simple episodic memory studies which could provide a baseline for future research. A series of related research questions provide the arch for this thesis. These are:

1. What are the psychological variables that influence young people's behaviour towards alcohol?
2. What is the frequency and prevalence of MBOs in a student population?
3. What is the impact to the brain of an MBO, and does alcohol impart any lasting effects on future memory formation?
4. Are people who experience a high frequency of MBOs from binge drinking more susceptible to the effects of alcohol than those who have never experienced a blackout?

These questions are addressed across a series of studies outlined in Chapters 3 – 7.

Chapter 3

The study presented in Chapter 3 was designed to answer the first two research questions via a detailed questionnaire which was distributed to a number of universities across Scotland, advertised and accessed via university portals. This ultimately resulted in a sample of 1144 students from predominantly three different establishments, the University of Stirling, the University of Glasgow, and the University of Edinburgh. Several other universities were represented by single individuals who were friends of participants. Their answers to questions relating to specific themes were scored and analysed in order to create a picture of drinking behaviours in students under the age of 25 across Scotland.

Chapters 4 and 5

In Chapters four and five, a series of lab-based behavioural experiments were conducted between two groups of participants – those who frequently experience MBOs and a control group who do not. The four short experiments included tasks of both recall and recognition memory and were all conducted first sober, and then after a lab appropriate scaled dose of alcohol, by the same individuals. Further, individuals from the MBO group were invited to return to the laboratory within 20-hours of experiencing a blackout (once sober) and repeat the studies. To the best of my knowledge, data which encompasses the same participants undertaking memory studies in all three alcohol conditions (sober/after alcohol/after blackout) does not exist within the literature.

Chapter 6 and 7

In order to examine the neural correlates of memory possibly affected by MBO experiences, Chapters 6 and 7 employed electroencephalography (EEG) measures. Participants completed a recognition memory study with added source judgement, and this paradigm was used in both Chapters 6 and 7. In Chapter 6, participants from a frequent blackout group and a control group all completed the study while sober, addressing the question of whether a history of frequent MBO experiences leads to observable neural changes between groups. In Chapter 7 the two new samples of MBO and control participants completed the first half of the study sober, and then the remaining section after a scaled dose of alcohol. Further, the MBO group also completed the same study less than 20 hours after experiencing an MBO. These studies

aimed to uncover what the impact to the brain of an MBO is, whether alcohol imparts any lasting effects on future memory formation, and whether people who experience a high frequency of MBOs resulting from binge-drinking are more susceptible to the effects of alcohol.

Overall, the studies presented in this thesis explore the current relationship between students, alcohol and the prevalence of MBOs within Scotland. Having established the frequency of MBO experiences in a student population, the thesis goes on to provide experimental evidence of differences in behavioural performance, and their neural correlates, between those who frequently experience blackouts and those who do not. Finally, my intention with this thesis is to provide the justification and foundation for future work investigating the potential harm caused to adolescent brains by frequent binge-drinking and alcohol-induced memory blackouts.

Chapter Two:

General Methods

The general methodology for data collection in this thesis required careful planning for a number of reasons. Firstly, a protocol had to be developed, trialled and receive ethical approval in order to dose participants with alcohol safely and accurately in the lab. Secondly, to capture potential after-blackout deficits, a plan which avoided asking participants to binge-drink, but which tapped into natural, unmanipulated behaviours, was required. Finally, there was an awareness that participants who met the required criteria were part of a special population and their willingness to take part in studies was unknown. A way to engage students with the studies and increase the likelihood of them participating in multiple lab experiments was therefore required to maximise the involvement of participants, without placing any pressure on individuals. The broad approach to data collection was therefore to simultaneously recruit participants to a number of experiments and to cross promote studies to these individuals, therefore collecting data which contributed to the whole thesis at the same time rather than taking each study in turn. This presented organisational and logistical challenges but proved to be the most efficient method of working with the participants. Specific experimental details, for example, memory tests and EEG recording procedure, are included in the corresponding experimental chapters.

General Study Plan

Two categorical groups of participants were required for all of the experimental studies. These were control participants (who had never experienced an alcohol-induced memory blackout, and who rarely - or never - drank alcohol) and MBO participants (who drank frequently and had experienced at least 9 MBO events in the preceding 12-months). Since these groups are effectively the tails of a normal distribution for a drinking population, it was predicted that they may be difficult to recruit due to smaller pools of potential participants. The plan developed to mitigate

for this is shown in Figure 2.1. Two online questionnaires were advertised to both drinkers and non-drinkers, with follow-up lab studies mentioned as an opt-in opportunity. The shorter questionnaire was advertised first and targeted participants specifically for the sober study presented in Chapter 6 (please see below for more details). Later, the more comprehensive of the two questionnaires gathered data for the study presented in Chapter 3, and was also used to identify participants for Chapters 4 and 5. While attending the lab, this group of individuals were invited to return and take part in the EEG study in Chapter 7. Further, as there was a period where both questionnaires were advertised simultaneously, lab studies were cross-promoted to participants in order to aid recruitment. Specifically, participants recruited for the behavioural study (Chapters 4 and 5) overlap (but not entirely) with those in the EEG study (Chapter 7). A small number of individuals from this participant pool also took part in the study presented in Chapter 6.

The larger questionnaire was developed over a number of months and following a literature review as well as focus group discussions. No previously published survey included the measures required for this project, therefore the questions were designed to answer the research questions specific to this thesis. To help refine the questionnaire, undergraduate students who were between 18-30 were asked to take part in discussions which included suitability of questions, interpreting their meaning, and the language used. This student population, being our target demographic, brought a modern perspective to the questionnaire, informing us of the range of current student drinking behaviours specifically related to MBOs. Firstly, a group of around 20 final year psychology students who were enrolled in an Alcohol and Psychology module were asked to proof-read and discuss the content. Following their helpful suggestions, two focus groups consisting of non-psychology students were conducted to further refine the questions. Finally, ethical approval was sought for the finalised questionnaire and then granted.

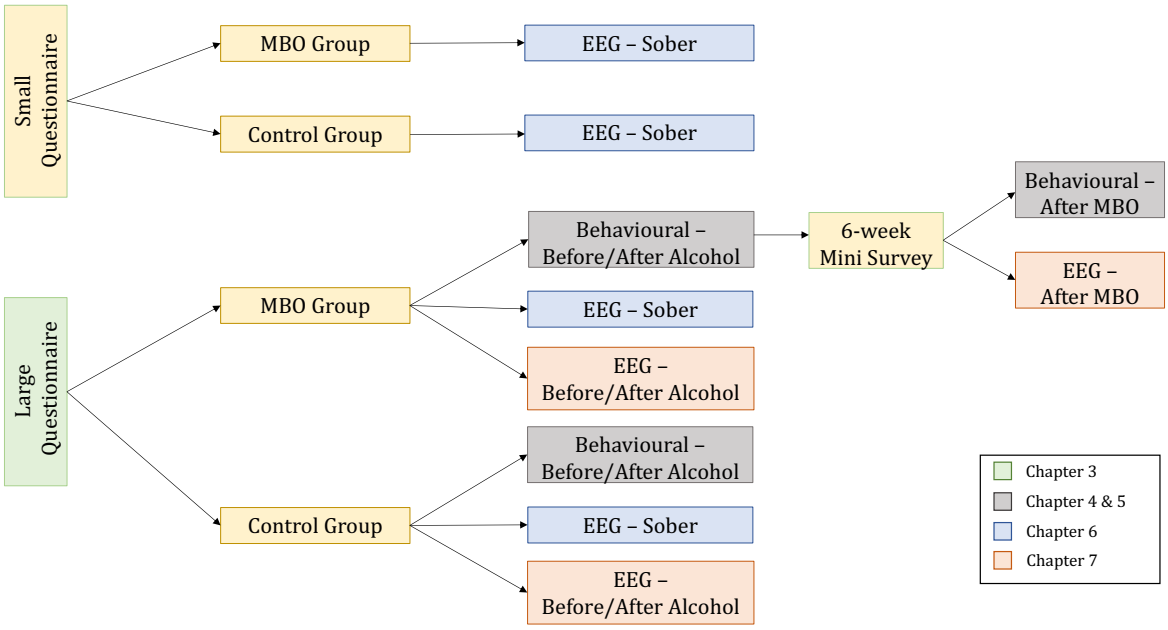

***Figure 2.1*: Participant recruitment plan.** The small questionnaire was used as a screening tool to identify eligible participants for the EEG sober study presented in Chapter 6. A small number of additional participants for this study were recruited via the large questionnaire. This means that the sample in Chapter 6 consists of a predominantly different group of individuals from the sample who took part in *all* other laboratory experiments. From the large questionnaire, eligible Control and MBO participants were invited to one of the three studies shown (Behavioural, EEG Sober, EEG Before/After alcohol). Each study was cross promoted at the first visit. The MBO group Behavioural testing arm of the strategy shows the two after-blackout studies, along with a mini-survey which recorded drinking behaviours across that time. The figure legend highlights the corresponding experimental chapters.

Working with the same MBO group participants over repeated sessions produced an unintended benefit. As people grew more comfortable in the lab setting, and with the research team, they began to get in touch if they had events planned where they predicted they were likely to drink heavily. This meant that after-MBO lab sessions could be arranged organically, and at the instigation of the participants, without any involvement or encouragement from the researchers.

It should be noted that the participant data included in Chapter 6 was collected at the start of the PhD with the help of undergraduate students as part of their dissertation projects. For this purpose, the aforementioned brief online questionnaire was used to screen for participants. Hence, the sample of participants in Chapter 6 is not the same as in Chapter 7, nor were they included in the behavioural studies (Chapters 4 and 5), making Chapter 6 a stand-alone study. This also explains the

difference in age ranges used in the behaviour studies reported in Chapter 4 and 5 (18 – 25-years old), and the EEG studies in Chapters 6 and 7 (18 – 30-years old).

Alcohol Protocol

Before participants attended at the laboratory, they were advised that they would be required to drink alcohol during the study, asked not to consume alcohol in the preceding 24-hours, nor food in the 3- 4 hours before arrival. They were also advised not to take part if there was a chance they could be pregnant or were on medication of any sort (other than the contraceptive pill). Upon arrival, photographic ID which included date of birth was presented to the researcher and written consent was obtained. A breathalyser test was also administered before commencing any experiment to ensure sobriety.

Methods for serving alcohol experimentally vary in the literature (for example, Conrod et al., 1997; H. V. Curran & Hildebrandt, 1999; Söderlund et al., 2007; Vinader-Caerols et al., 2017). While some researchers favour pure ethanol (Curtin & Fairchild, 2003; George et al., 2005; Söderlund et al., 2007), others have chosen vodka (Stock et al., 2017; Vinader-Caerols et al., 2017; Wetherill & Fromme, 2011). Spirits like vodka contain around 40% alcohol by volume (ABV), more than other types of alcoholic drink (for example, wine, beer, etc.). Choosing a high ABV drink meant that less liquid would have to be consumed by participants to reach the intended blood alcohol level. There was no experimental advantage to selecting one alcohol type (vodka/ethanol) over another however, due to the strong taste of ethanol and a desire to reduce discomfort for participants, vodka was chosen. This was also consistent with other MBO specific research (Wetherill & Fromme, 2009, 2011).

The next issue was how to deliver the vodka. It was preferable to serve the alcohol without a mixer to avoid both diluting the alcohol, and the confounding effects of additional substances, such as sugar, on cognition (Banoczi, 2005; Tryon et al., 2015). This meant serving the drink undiluted which had implications for taste. To reduce intensity, the vodka was served as quickly as possible following removal from a freezer, the cold minimising the taste and replicating the method outlined by Conrod et al. (1997). Participants were also offered the use of a glass straw with which to drink

the vodka, enabling them to swallow the drink without prolonged contact on the taste buds.

In order to dose participants with sufficient alcohol to enable BAC% to reach the experimentally required levels, a formula by Watson (1989; but see also Watson et al., 1981) (see below) was used. Target BAC% was calculated as a function of height, weight, age, gender, total body water (TBW), duration of expected drinking period (DDP), time to peak BAC% (TPB) and alcohol metabolism rate (MR). The required quantity of vodka was then calculated. It was assumed that peak BAC% would be reached around 30 mins following drinking.

$$Alcohol\ dose\ (g) = \frac{(10\ x\ BAC\%\ x\ TBW)}{0.8 + 10MR(DDP + TPB)\frac{TBW}{0.8}}$$

TBW varies between males and females therefore separate, gender-specific calculations were used to accommodate differences (Watson, 1989; Watson et al., 1980). These were:

Male TBW = 2.447 – 0.09516 x *age* + 0.1074 x *height (cm)* + 0.3362 x *weight(kg)*

Female TBW = -2.097 + 0.1069 x *height(cm)* + 0.2466 x *weight(kg)*

This meant that although absolute quantities of vodka differed between individuals, there was consistency of intended BAC% across the groups. Following consumption, participants were granted a 15-minute break to allow BAC to increase, after which they rinsed their mouths with water to remove any alcohol residue from the tongue, and breathalyser readings were taken to provide a baseline reading before the experiment was restarted. Since it was assumed that it would take 30 minutes to reach peak BAC%, testing therefore began 15 minutes after alcohol consumption and thereafter continually throughout regular breaks to measure the peak and fall of the BAC curve. Note that breath alcohol concentration (BrAC%) was recorded from a Dräger Alcotest® 3000 (Lübeck, Germany) professional breathalyser, and values then converted to BAC.

It should be noted that both females and males were included in this work, despite the potential harms which could be caused by alcohol to pregnant women. One solution would have been to ask female participants to conduct a pregnancy test before taking part. Resources available at the University of Stirling did not include the ability to perform blood tests therefore this would have required a home pregnancy test. Apart from the fact that subjecting all female participants to a urine sample pregnancy test before participation would suggest that we do not trust participants to exclude themselves from the study, this option presented several other ethical issues. Firstly, the accuracy of such tests is questioned in the literature (Cole et al., 2004; Tomlinson et al., 2008). Should a test provide a false positive, this could be an upsetting surprise for the participant and likely cause unnecessary stress to both the individual concerned, and the researcher who had to pass on the news. Further, on finding that the positive test was wrong, this would likely have compounded distress either because the participant has undergone an avoidable ordeal or because they had been happy at the news of the pregnancy and then were disappointed. Perhaps more worrying for the current thesis, if a possible test provided a false negative (often uncontrolled in the manufacturing of these tests), alcohol would have been administered with the potential for harm. Beyond the issues surrounding pregnancy, there were some practicalities to consider. Participants are mainly drawn from psychology undergraduates, of whom the majority are female. While the studies were advertised more broadly, only accepting male participants would have severely hampered data collection. Further, both males and females experience MBOs and take part in binge-drinking behaviours. It seems logical therefore to include both in the research. Taking these arguments into consideration, it was decided – and approved by the University of Stirling ethics panel – that female participants could take part if they confirmed that, to the best of their knowledge, they were not pregnant. This follows UK NHS guidelines for medical procedures which could be harmful to mother and baby.

In addition to inclusion criteria and mechanics of delivery, care for participants during the experiments was also paramount. Regular breathalyser readings were obtained during the experiments, and participants were offered further tests once finished and asked to remain in the laboratory until BrAC readings were at least below the Scottish drink drive limit of 0.22mg/l (0.05% BAC)(*Drink-Drive Limit in Scotland*,

2020), but preferably zero. This ensured that all participants were sufficiently sober to enable them to return home safely. They were also advised not to drive to the laboratory or, if they had, were asked to remain on the premises until their readings were 0.00mg/l. Soft drinks (tea/coffee/water) were offered during this time, and participants were advised to bring snacks for this period since they had been asked to fast prior to the study. The researcher remained in the laboratory with the participants until they left.

In practice, all participants were compliant with the requirements to stay in the laboratory following the studies until their alcohol levels were sufficiently reduced. Indeed, once some individuals recorded below safe limits on their BrAC tests, they chose to stay longer until they felt confident in their sobriety and ability to conduct themselves before leaving.

After-Blackout Protocol

To capture potential deficits which followed the experience of an MBO, even after alcohol was no longer present, participants in the experimental MBO group were invited to return to the laboratory the next day following a heavy drinking session. As described earlier, many participants instigated these visits by anticipating their own behaviours at planned drinking events. However, at the point of designing a recruitment strategy, this eventuality was not predicted and a method of identifying when participants would be likely to have experienced an MBO was therefore required. Individuals who agreed to take part in these visits received a weekly survey by email for six weeks. This questionnaire asked for details of frequency and quantity of drinking over the period in order to track average drinking behaviours across the days of the week. The intention was to look for patterns and to tentatively schedule lab visits for days where it was predicted the students may have experienced an alcohol-induced blackout the night before. No students were asked to binge-drink, or deliberately induce an MBO, for the purposes of these studies. In reality, this approach was relatively unused due to the engagement of the participants, and their willingness to instigate communication with the research team. It did, however, provide data to supplement group drinking norms and is detailed in Chapter 4.

All after-MBO visits were scheduled for the afternoon. This was to allow participants time to rest and to become sober. Upon arrival at the laboratory, participants were immediately breathalysed to ensure readings had returned to 0 mg/l. If this was not the case, they were invited to either return later that afternoon or to rest in the laboratory. All participants who took part in an after-MBO test session reported experiencing an MBO the previous evening and were sober again prior to taking part in the experiments. Informal conversations regarding start and stop time of drinking, what they could remember from the evening, and duration of sleep, took place with notes recorded for inclusion in analysis.

Chapter Three:

Alcohol and the 'student experience': Influences on, and prevalence of, alcohol-induced memory blackouts in a Scottish based student population

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Abstract

Background: Alcohol-induced memory blackouts (MBOs) are transient anterograde amnesic events where the encoding of episodic memories is disrupted, and can occur after extreme binge-drinking episodes. They are readily observed in western student populations, a time when young people may be more easily influenced by peers and culture. Furthermore, Scotland is renowned for its cultural acceptance of heavy-drinking behaviours, perpetuated by the student body. This study therefore investigates the prevalence of, and influences on, MBOs in a population of students attending Scottish universities.

Methods: Between 2018 and 2019, 1144 students attending 3 Scottish universities completed an online survey. Participants were asked to provide details of their typical drinking behaviours, their perception of peer influence on drinking behaviours, family history of drinking in the home, and the frequency and severity of any MBO experiences. Responses were grouped to form numeric metrics based on family drinking, peer influence, personal drinking behaviours, and blackout experiences.

Results: We found that 81% of our sample regularly drank alcohol, with a 12-month period prevalence of binge-drinking at 90%. Further, lifetime prevalence of MBOs was 75%, with a 12-month period prevalence of *fragmentary* blackouts at 61.5%, and *en bloc* blackouts at 38.7%. Regression analysis revealed that peer influence and personal drinking behaviours predicted blackout experiences, with Scottish students most influenced by peers, but that there was no association with family history. Home country of origin significantly predicted MBO scores, mediated by peer influence (partly) and personal drinking behaviours.

Conclusions: A high proportion of students attending Scottish universities report regular binge-drinking and MBOs, driven by both peer influence and personal drinking behaviours. MBOs should be considered a reliable marker of extreme binge-drinking. Critically, our findings suggest that Scotland's problematic relationship with alcohol is ingrained within our student populations, with potential consequences for future health.

Key words: *(alcohol-induced memory blackouts, binge-drinking, alcohol, peer-influences on student drinking behaviour, alcohol use in Scotland)*

Introduction

Binge-drinking is a common practice among young adults, especially those attending university, where alcohol-themed social activities are prevalent (Gambles et al., 2021). Students often assume that everyone drinks more than them (McAlaney & McMahon, 2007), creating an environment which fosters a binge-drinking culture (Balodis et al., 2009; Bhatti et al., 2020; Borsari et al., 2007; Davoren et al., 2016). One consequence of extreme binge-drinking, that is, drinking vastly more than the recommended guidelines, is the experience of an alcohol-induced memory blackout (MBO) (Wetherill & Fromme, 2016). This transient anterograde amnesic event following a period of rapid alcohol consumption (A. M. White, 2003) is a common experience within student populations (Hingson et al., 2016; Weitzman et al., 2003), suggesting another possible route for alcohol-induced harm to the brain. Most UK based students are aged between 18 and 25 (Mantle, 2019), an age of ongoing neural-development which can be negatively impacted by alcohol (Spear, 2018; Squeglia et al., 2009). It is therefore critical to understand what influences extreme binge-drinking behaviours, and the prevalence of alcohol-induced memory blackouts, within the student population, to be able to focus on diminishing instances of this specific alcohol-induced harm.

Excessive alcohol consumption is known to disrupt normal hippocampal functioning (A. M. White, 2003) and the transfer of episodic information from short to long-term memory stores. Therefore, details from events which occurred while intoxicated may be quickly forgotten leaving gaps in memory (*fragmentary* blackout), or may not even be stored at all (*en bloc* blackout). A dose dependant relationship between alcohol and MBOs exists, with *en bloc* blackouts (Goodwin et al., 1969b) being associated with a total lack of recall for events while intoxicated; *en bloc* blackouts are concomitant with high blood alcohol concentrations (BAC%), whereas a lower BAC% for *fragmentary* blackouts means that some details can be recalled (A. M. White et al., 2004). Critically, drinking alcohol during late adolescence and early adulthood is known to be detrimental for brain health and cortical development (Lees et al., 2019; Peeters et al., 2014; Squeglia et al., 2009). Alcohol alters the functioning of $GABA_A$ and NMDA receptors of neurons (A. M. White & Best, 2000), and it is known that exposure

to alcohol in the developing brain can lead to neuron death (Granato et al., 2012; Granato & Dering, 2018). Periods of sobriety interspersed with extreme binge-drinking episodes – a typical student pattern - can also be damaging (Duka et al., 2004). These potential neurobiological impairments stand alongside many other alcohol-induced harms often seen in younger drinkers, including risky behaviours (Melchior et al., 2008), and physical or sexual assaults (Hingson et al., 2009). Accidental injury is also prevalent (Sindelar et al., 2004), with the Global Drugs Survey 2020 (Winstock, 2021) finding that over 5% of under-25s in the UK, compared to an average of 2% globally, had required hospital treatment while drunk. Unfortunately, there is little focus on alcohol-induced MBOs in the literature, which are arguably caused by the most extreme binge-drinking episodes. A goal of the present paper is to identify the prevalence of blackout experiences.

The term 'binge-drinking' has, in recent years, come to mean single occasions of excessive alcohol consumption (Herring et al., 2008), typically an evening of heavy drinking interspersed with periods of abstinence. The Scottish government recommends 2 to 3 units of alcohol per day for women, and 3 to 4 for men, as a safe level of consumption; ingesting twice that amount they define as a binge-drinking session (Campbell-Jack et al., 2014). It should however be noted that there is no global consensus on what constitutes a binge-drinking event. Binge-drinking is highly prevalent in student populations worldwide (Davoren et al., 2016; McGee & Kypri, 2004; Tavolacci et al., 2016), and students themselves claim that alcohol forms an important part of the 'student experience' (Gambles et al., 2021). This can be observed from the practice of 'pre-drinking' in students, the act of drinking at home before going out in order to save money, extend drinking time across the evening, or as part of a 'getting ready' ritual with friends (Labrie et al., 2011; Wahl et al., 2013). Additionally, students view drinking games as a fun way to consume alcohol, and therefore get drunk quickly (A. E. Ray et al., 2014). Worryingly, studies have suggested that drinking game participants can reach BAC% levels which exceed the Scottish drink-drive limit of 0.05 BAC% (representing a point above which reduced cognitive control is typically observed) during the pre-drinking stage alone (LaBrie & Pedersen, 2008; Pedersen et al., 2009). If students regularly engage in pre-drinking and drinking games, this could be a significant precursor to a blackout event.

While binge-drinking occurs in student populations across the globe (for example, Balodis et al., 2009; Elgàn et al., 2019; McGee & Kypri, 2004), the impact of cultural norms and broader influences cannot be ignored. A systematic review by Davoren and colleagues (2016) found that alcohol consumption amongst UK-based students had steadily increased throughout the 1990s, becoming pervasive by the early 2000s. They further suggested that around two-thirds of students consumed alcohol at harmful levels, supporting previous work claiming that around 50% of students have experienced MBOs (A. M. White et al., 2002). Scots have also been found to buy more alcohol per person than drinkers in England and Wales (Giles & Robinson, 2018), and are more likely than individuals from other UK regions to binge-drink (Office for National Statistics, 2018). Although 55 to 64-year old adults consume the most alcohol across a week (Giles & Robinson, 2018), people under 25-years old are more likely to binge-drink (Office for National Statistics, 2018). Interestingly, although no recent work exists, Delk and Meilman (1996) found evidence suggesting that students in Scotland drink more than in other countries, particularly the USA, and that the Scottish culture accepts and normalises heavy drinking within their population.

Investigating the relationship between MBOs and students within Scotland is important for several reasons. Firstly, Scotland has a disproportionately adverse relationship with alcohol, and a family history of alcoholism leads to increased susceptibility for developing alcohol use disorders (Capone & Wood, 2008). It may be that MBOs are more prevalent in people who have family members with alcohol use disorders. Secondly, MBOs can be considered as markers of extreme binge-drinking, going beyond what would be defined as a binge, as they only occur at high levels of BAC%. Therefore, they should be used to identify individuals at risk from harms associated with binge-drinking (Hingson et al., 2016). Finally, the majority of our knowledge about the effects of alcohol-induced MBOs has so far been generated from the USA, where the legal drinking age is 21. In Scotland, we already know that 18-year olds are involved in heavy binge-drinking cultures, fostered by universities, meaning there is a paucity of literature across a critical 3-year period of development.

To begin addressing this gap, we asked students attending Scottish universities to complete an online survey related to drinking behaviours. We were specifically interested in the prevalence of binge-drinking and of MBO experiences in our Scottish-

based sample. We expected to find a large proportion of students reporting both binge-drinking and MBOs, and that there would be an influence of culture – measured through peer influence and country of home residence – on these behaviours.

Materials and Methods

Participants and Design

Participants aged 18 – 25 were recruited from three Scottish universities via internal online advertisements. Respondents who completed at least 50% of the questionnaire were included for analysis (see Table 3.1). Informed consent was obtained electronically from all participants, and the study was approved by the University of Stirling's NHS, Invasive and Clinical Research ethics committee.

The survey was created by the researchers to collect demographic, family, and drinking behaviour information. Early drafts of the questionnaire were discussed at three focus groups which each consisted of between 8-12 undergraduate students aged 18 – 25. Questions were each discussed in turn to ensure clarity and relevance. Wording of questions was then amended where appropriate, and the revised questionnaire discussed further with a group of 13 final year undergraduate students in a class setting. These students were also asked both to consider wording and clarity of questions and any relevant suggestions were then adopted. Changes made following these discussions were predominantly in phrasing, for example "night out" became "drinking session" as students reported binge-drinking at other times of day. The final questionnaire (see Appendix 5.1) was accessed via online software (Qualtrics, 2018). Participants from the University of Stirling who were Psychology students received research tokens for participation. Students who were ineligible for tokens, or attended other universities, did not receive any reward.

Procedure

Participants were advised that the questionnaire would ask a range of personal questions which they could skip if they felt uncomfortable. They were provided with a unique ID code to enable study withdrawal, and given contact details of relevant, local alcohol-support agencies. A total of 98 questions were included in the survey however,

since a number of those were 'qualifying questions' (that is, subsequent questions were displayed depending on the answer to the qualifying question), not all participants were presented with every question. Since we are concerned with the prevalence of MBOs and factors influencing these experiences, we focussed our analysis of the questionnaire on questions relating to personal drinking behaviours, peer influence, family history of drinking, and MBO experiences only.

Table 3.1:

Participant Demographics

		Year of Degree (Mean %)				
	Age	**1st**	**2nd**	**3rd**	**4th**	**Other**
	(mean months, SD)					
All, *n* = 1144	241.47 (20.95)	53.67	32.69	6.12	6.64	0.87
Male, *n* = 251	242.96 (21.15)	11.89	7.69	0.96	1.22	0.18
Female, *n* = 888	241.01 (20.85)	41.52	25	4.98	5.42	0.7
Other, *n* = 5	248.60 (28.47)	0.26	0	0.18	0	0

Alcohol Use Metrics

To measure prevalence of binge-drinking behaviour, we asked all participants how often they had consumed 6 or more units of alcohol in one drinking session in the past year. Further, topic related questions were grouped together, and responses scored per participant to create numerical metrics for analysis. These metrics represented family drinking history, personal drinking behaviours, peer influences on student drinking (PISD), and frequency and severity of MBOs, and are detailed below.

Family Drinking History

As an indicator of alcohol culture within the home, questions relating to family drinking history were presented. Firstly, "*Did any members of your immediate family regularly drink alcohol within your home(s) while you were growing up?"* acted as a classifying question (response options: '*Yes*', '*No*' or '*Not Sure*'). Follow-up questions were displayed if participants responded with either '*Yes*' or '*Not Sure*'. The first question was "*Which family members did this"* and responses were tallied as a count of adults who regularly drank in the home. Next, responses to *"Thinking of the family member who most frequently drinks currently, on how many days per week would they*

normally have an alcoholic drink" were scored from 1 to 7, with 1 being 1 day per week and 7 being 7 days. *"In a normal week, on how many of these days would you say this family member was drunk"* was scored from 0 to 14, with 0 being "*not as often as once a week, only now and then*" and 14 being 7 days, reflecting the impact that heavy drinking was liable to have on the family home environment. Finally, "*Do any other family members regularly get drunk when drinking in the home"* was scored 0 to 4, with 0 being "*never*" and 4 being "*always*". The combined maximum score was 25, with 1 being the minimum. The mean score was 5.49, median 4, and SD 4.46. Details of the number of participants who responded to each question, along with the percentage of total responses, per answer, are given in Table 3.2. Scores are further included as a percentage split by gender in brackets.

Table 3.2:

Family Drinking History (% responses per question, % within gender response in brackets)

Did any of your immediate family regularly drink alcohol within your home(s) while you were growing up?

	No	**Not sure**	**Yes**
All, *n* = 1144	31.82	3.06	65.12
Male, *n* = 251	6.03 (27.49)	0.87 (3.98)	15.03 (68.53)
Female, *n* = 888	25.61 (33)	2.1 (2.7)	49.91 (64.3)
Other, *n* = 5	0.17 (40)	0.09 (20)	0.17 (40)

Which of your family members currently drinks most frequently within the family home?

	Brother	**Father**	**Grand-father**	**Grand-mother**	**Mother**	**Sister**	**Other**	**None**
All, *n* = 1143	6.12	33.95	1.75	0.87	25.9	4.9	2.27	24.23
Male, *n* = 251	1.14 (5.18)	8.05 (36.65)	0.35 (1.59)	0.26 (1.2)	5.77 (26.29)	0.79 (3.59)	0.35 (1.59)	5.25 (23.9)
Female, *n* = 887	4.99 (6.43)	25.72 (33.15)	1.4 (1.8)	0.61 (0.79)	19.95 (25.7)	4.11 (5.3)	1.92 (2.48)	18.9 (24.35)

Other, *n* = 5	0	0.17 (40)	0	0	0.17 (40)	0	0	0.09 (20)

Thinking of this family member, on how many days per week would they normally have an alcohol drink?

	1	**2**	**3**	**4**	**5**	**6**	**7**
All, *n* = 866	20.55	24.13	17.9	11.43	8.31	5.31	12.36
Male, *n* = 191	4.85 (21.99)	6.58 (29.84)	3 (13.61)	2.89 (13.09)	1.62 (7.33)	1.15 (5.24)	1.96 (8.9)
Female, *n* = 671	15.36 (19.82)	27.44 (22.5)	14.9 (19.23)	8.55 (11.03)	6.7 (8.64)	4.16 (5.37)	10.39 (13.41)
Other, *n* = 4	0.35 (75)	0.12 (25)	0	0	0	0	0

In a normal week, on how many of these days would you say this family member was drunk?

	Not as often as 1	**1**	**2**	**3**	**4**	**5**	**6**	**7**
All, *n* = 858	70.63	10.61	9.67	3.61	2.21	1.28	0.58	1.4
Male, *n* = 191	16.43 (73.82)	1.86 (8.38)	2.1 (9.42)	0.58 (2.62)	0.7 (3.14)	0.23 (1.05)	0.23 (1.05)	0.12 (0.52)
Female, *n* = 663	53.73 (69.53)	8.74 (11.31)	7.58 (9.8)	3.03 (3.92)	1.52 (1.96)	1.05 (0.36)	0.35 (0.45)	1.28 (1.66)
Other, *n* = 4	0.35 (80)	0	0	0	0	0	0	0.12 (20)

Do any other family members regularly get drunk when drinking in the home?

	Always	**Usually**	**Sometimes**	**Rarely**	**Never**
All, *n* = 867	0.58	2.88	15.46	34.83	46.25
Male, *n* = 191	0.12 (0.52)	0.58 (2.62)	3 (13.61)	8.07 (36.65)	10.27 (46.6)
Female, *n* = 672	0.46 (0.6)	2.31 (2.98)	12.34 (15.92)	26.64 (34.38)	35.76 (46.13)
Other, *n* = 4	0	0	0.12 (25)	0.12 (25)	0.23 (50)

Personal Drinking Behaviours

To investigate individual drinking patterns, responses to six questions were scored to create a numerical scale of drinking behaviour. Included were "*Can you estimate how many drinks/shots you would have before you go out"* and *"How many drinks would you have when you go out"* which were scored from 0 ('*None*') to 7 (*'13 or more'*). Next, "*Do you continue drinking after you leave the club/bar"* had four response options which were scored from 0 (*'No, never'*) to 3 (*'Yes, every time'*). For qualifying participants who said that they drink alcohol regularly, we asked *"Roughly, how many times would you have a drink in a normal month"*, *"How many times have you been drunk in the past year?"* and *"During the past year, roughly how often have you drunk more than 6 units of alcohol on a single occasion?"*. These questions had the same response options (*'1-5 times'*, *'6-10 times'*, *'11-15 times'*, etc.) with scores from 1 (*'1-5 times'*) to 7 (*'More than 30 times'*). The final question included an additional '*Never*' option which was scored as 0. Response percentages per question are shown in Table 3.

Table 3.3:

Personal Drinking Behaviours (% responses per question, % within gender response in brackets)

Roughly, how many times would you have a drink in a normal month?

	1-5 times	**6-10 times**	**11-15 times**	**16–20 times**	**21–25 times**	**26–30 times**	**More than 30 times**
All, *n* = 918	48.47	28.54	11.76	6.75	2.51	1.42	0.54
Male, *n* = 212	8.5 (36.79)	6.64 (28.77)	3.81 (16.51)	2.18 (9.43)	1.09 (4.72)	0.65 (2.83)	0.22 (0.94)
Female, *n* = 703	39.87 (52.06)	21.79 (28.45)	7.95 (10.38)	4.58 (5.97)	1.42 (1.85)	0.65 (0.85)	0.33 (0.43)
Other, *n* = 3	0.11 (33.33)	0.11 (33.33)	0	0	0	0.11 (33.33)	0

How many times have you been drunk in the past year?

All, *n* = 999	28.13	14.91	9.71	9.61	6.21	6.11	25.33
Male, *n* = 226	6.01 (26.55)	2.5 (11.06)	1.3 (5.75)	1.4 (6.19)	0.9 (3.98)	1.6 (7.08)	8.91 (39.38)

Female, *n* = 769	22.02 (28.61)	12.41 (16.12)	8.41 (10.92)	8.21 (10.66)	5.31 (6.89)	4.4 (5.72)	16.22 (21.07)
Other, *n* = 4	0.1 (25)	0	0	0	0	0.1 (25)	0.2 (50)

During the past year, roughly how often have you drunk more than 6 units of alcohol on a single occasion?

	Never	**1- 5 times**	**6 – 10 times**	**11-15 times**	**16 – 20 times**	**21 – 25 times**	**26 – 30 times**	**More than 30 times**
All, *n* = 1106	9.4	23.6	13.38	11.12	9.86	6.42	5.7	20.52
Male, *n* =244	1.08 (4.92)	4.07 (18.44)	2.71 (12.3)	1.99 (9.02)	0.99 (4.51)	1.72 (7.79)	1.9 (8.61)	7.59 (34.43)
Female, *n* = 857	8.23 (10.62)	19.44 (25.09)	10.58 (13.65)	9.13 (11.79)	8.86 (11.44)	4.7 (6.07)	3.71 (4.78)	12.84 (16.57)
Other, *n* = 5	0.09 (20)	0.09 (20)	0.09 (20)	0	0	0	0.09 (20)	0.09 (20)

Can you estimate how many drinks/shots you would have before you go out

	None	**1 or 2**	**3 or 4**	**5 or 6**	**7 or 8**	**9 or 10**	**11 or 12**	**13 or more**
All, *n* = 999	2.3	25.83	45.35	17.12	5.51	2.1	0.9	0.9
Male, *n* = 224	0.2 (0.89)	3.9 (17.4)	8.31 (37.05)	5.6 (25)	2.2 (9.8)	1.3 (5.8)	0.7 (3.1)	0.2 (0.89)
Female, *n* = 770	2 (2.6)	21.72 (28.18)	36.9 (47.9)	11.5 (14.9)	3.3 (4.3)	0.7 (0.9)	0.2 (0.26)	0.7 (0.9)
Other, *n* = 5	0.1 (20)	0.2 (40)	0.1 (20)	0	0	0.1 (20)	0	0

How many drinks would you have when you go out?

All, *n* =1101	3.91	21.07	32.33	21.44	9.63	6.45	2.38	3.00
Male, *n* = 243	0.5 (2.5)	2.9 (13.2)	6.4 (28.8)	5.9 (26.8)	2.5 (11.1)	1.8 (8.2)	0.7 (3.3)	1.4 (6.2)
Female, *n* = 853	3.3 (4.2)	18.2 (23.5)	25.9 (33.4)	15.3 (19.8)	7.2 (9.3)	4.6 (5.3)	1.5 (1.9)	1.5 (2)

Other, *n* = 5	0.1 (40)	0	0.1(20)	0.2 (40)	0	0	0	0.1 (20)

Do you continue drinking after you leave the club/bar?

	No, never	**Now and again**	**Yes, most of the time**	**Yes, every time**
All, *n* =1097	43.57	48.4	6.11	1.91
Male, *n* = 241	7 (32)	12.5 (56.9)	2 (9.1)	0.5 (2.1)
Female, *n* = 851	36.4 (46.9)	35.7 (46.1)	4 (5.2)	1.5 (1.9)
Other, *n* = 5	0.2 (40)	0.2 (40)	0.1 (20)	0

Peer Influences on Student Drinking (PISD)

The PISD metric incorporates five questions focused on peer and cultural influences on perceptions of drinking in our student population. *"Do you ever feel pressured to drink by friends",* and *"Do you ever feel like you have to drink to fit in with friends"*, were both scored from 0 ("No, they don't pressure me"/ "No, never") to 4 ("Yes"). Next, *"Do you view drinking as something that everyone does at your age"* and *"Do you think being drunk, or binge-drinking, is something that everyone does at your age"* were scored from 0 *("I'm not sure")* to 4 *("Definitely yes").* Finally, participants were asked *"How does your current drinking compare to before you started university".* Responses of *"I drank more before Uni"* were scored -2, *"I drink more now I'm at Uni"* were scored 2, and *"I probably drink about the same"* were scored 0. Response details are shown in Table 3.4.

Table 3.4:

Peer Influences on Student *Drinking (% responses per question, % within gender response in brackets)*

Do you ever feel pressured to drink by friends?

	Yes, often	**Sometimes**	**Yes, but I ignore them**	**No**
All, *n* = 1142	8.7	12.8	34.3	44.2

Male, *n* = 251	2.5 (11.2)	3.2 (14.3)	7.6 (34.7)	8.8 (39.8)
Female, *n* = 886	6.2 (8)	9.5 (12.3)	26.6 (34.3)	35.2 (45.4)
Other, *n* = 4	0	0	0.09 (25)	0.3 (75)

Do you ever feel like you have to drink to fit in with friends?

All, *n* = 1143	7.3	33.4	7.1	52.2
Male, *n* = 251	1.5 (6.8)	8.1 (37.1)	0.7 (3.2)	11.6 (53)
Female, *n* = 888	5.8 (7.4)	25.2 (32.4)	6.3 (8.1)	40.4 (52)
Other, *n* = 4	0	0.09 (25)	0.09 (25)	0.2 (50)

Do you view drinking as something that everyone does at your age?

	Definitely yes	**Most people do**	**Some people do**	**Not everyone does**	**No, it's not common**	**I'm not sure**
All, *n* = 1140	28.7	61.2	2.8	6.7	0.26	0.35
Male, *n* = 251	5.8 (26.3)	14.6 (66.1)	0.04 (1.6)	1.3 (6)	0	0
Female, *n* = 885	22.8 (29.4)	46.5 (59.9)	2.5 (3.2)	5.3 (6.8)	0.26 (0.3)	0.35 (0.5)
Other, *n* = 4	0.09 (25)	0.18 (50)	0	0.09 (25)	0	0

Do you think being drunk, or binge-drinking, is something that everyone does at your age?

All, *n* = 1141	13.7	44.9	24.5	13.9	1.8	1.1
Male, *n* = 251	2.7 (12.4)	9.5 (43)	5.9 (26.7)	3.1 (13.9)	0.6 (2.8)	0.3 (1.2)
Female, *n* = 886	10.9 (14)	35.4 (45.6)	18.6 (23.9)	10.8 (13.9)	1.1 (1.5)	0.9 (1.1)
Other, *n* = 4	0.09 (25)	0	0.09 (25)	0.09 (25)	0.09 (25)	0

How does your current drinking compare to before you started University?

	I drank more before Uni	**I drink more now I'm at Uni**	**I probably drink about the same**
All, *n* = 1123	15.9	40.8	43.4
Male, *n* = 248	3.4 (15.3)	9.2 (41.5)	9.5 (43.1)
Female, *n* = 871	12.4 (16)	31.5 (40.6)	33.7 (43.4)

Other, *n* = 4	0.09 (25)	0.09 (25)	0.2 (50)

MBO Metric

In order to assess the severity of MBOs, we firstly split responses into the two types of MBO – *fragmentary* and *en bloc*. Data on blackout prevalence only partly gives an indication of frequency; the overall score of the MBO metric, however, incorporates a wider range of data covering both severity and frequency. The fragmentary MBO questions were "*Roughly how often [have you experienced an fMBO] in the past year*" which was scored from 1 ('*1-2 times*') to 7 ('*More than 12 times*'). Next, responses to "*If you selected more than 12 times in a year, roughly how often do you think this has happened*" were "*More than once a month, but not as much as every week*", "*More than once a week, but not as much as every time I drink*", and "*Every time I drink, on multiple occasions per week*" and were scored from 1 to 3. Wording and scoring was duplicated for the *en bloc* questions. Percentages of responses to each question are given in Table 3.5. To create an overall MBO metric, scores from the fragmentary and the *en bloc* MBO questions were combined.

Table 3.5:

MBO Metric (% responses per question, % within gender response in brackets)

How often have you experienced a fragmentary MBO in the past 12 months?

	1-2 times	**3-4 times**	**5-6 times**	**7-8 times**	**9-10 times**	**11-12 times**	**More than 12 times**
All, *n* = 696	28.88	22.27	15.37	7.47	6.9	4.6	14.51
Male, *n* = 160	5.32 (23.13)	3.88 (16.88)	4.17 (18.13)	1.72 (7.5)	2.16 (9.38)	1.15 (5)	4.6 (20)
Female, *n* = 535	23.56 (30.65)	18.39 (23.93)	11.21 (14.58)	5.75 (7.48)	4.6 (5.98	3.45 (4.49)	9.91 (12.9)
Other, *n* = 1	0	0	0	0	0.14 (100)	0	0

If you said 12 or more, how often has this happened in the last 12 months?

	Every time I drink, multiple times per week	**More than once a week, less than every time**	**More than once a month, less than every week**
All, *n* = 100	8	23	69
Male, *n* = 32	2 (6.25)	8 (25)	22 (68.75)
Female, *n* = 68	6 (8.82)	15 (22.06)	47 (69.12)
Other, *n* = 0	0	0	0

How often have you experienced an en bloc MBO in the past 12 months?

	1-2 times	**3-4 times**	**5-6 times**	**7-8 times**	**9-10 times**	**11-12 times**	**More than 12 times**
All, *n* = 412	44.66	23.06	12.86	4.13	5.58	3.4	6.31
Male, *n* = 112	9.71 (35.71)	6.31 (23.21)	3.64 (13.39)	1.21 (4.46)	1.46 (5.36)	1.7 (6.25)	3.16 (11.61)
Female, *n* = 299	34.95 (48.16)	16.75 (23.08)	9.22 (12.71)	2.91 (4.01)	3.88 (5.35)	1.7 (2.34)	3.16 (4.35)
Other, *n* = 1	0	0	0	0	0.24 (100)	0	0

If you said 12 or more, how often has this happened in the last 12 months?

	Every time I drink, multiple times per week	**More than once a week, less than every time**	**More than once a month, less than every week**
All, *n* = 26	3.85	69.23	26.92
Male, *n* = 13	0	34.62 (69.23)	15.38 (30.77)
Female, *n* = 13	3.85 (7.69)	34.62 (69.23)	11.54 (23.08)

Statistical Analysis

All analyses were completed using R version 4.1.1 (R Core Team, 2020), and R Studio (RStudio Team, 2020). Descriptive statistics quantified prevalence and proportions of key drinking behaviours, such as binge-drinking and MBOs. Next, a multiple linear regression assessed impact of age, family drinking history, personal

drinking behaviours, PISD, year of degree and gender on the MBO metric scores. Multicollinearity was assessed using Variance Inflation Factors (see Table 3.6) and robust bootstrapping analysis was applied.

A series of ANOVAs were run to investigate the contribution of year of degree and gender on all drinking metrics. Levene's test was used to confirm variance normality and, where significant, Welch's correction was applied. A MANOVA was conducted to consider whether location of family home predicted any of the drinking metrics. We report Pillai's trace, with separate univariate ANOVAs on outcome variables. We assessed normality using Shapiro-Wilk tests and, where significant, robust MANOVAs using Choi and Marden's (1997) extension of the Kruskal-Wallis test (conducted using the WRS package; Wilcox & Schonbrodt, 2014) are reported. Finally, categorical mediation analysis using the *MeMoBootR* package (Buchanan, 2018) explored whether personal or peer influenced drinking behaviours mediated the influence of family home on MBO scores.

Results

All results are based on the number of responses per question, which can differ from the total sample size as not all participants answered every question.

How frequently do students drink alcohol or binge-drink?

Firstly, we wanted to ascertain the frequency and quantity of alcohol consumed by our sample; 80.77% of respondents (total responses, n = 1139) reported drinking regularly, defined as at least once per month. Of those, 48.47% said they would drink between 1 and 5 occasions per month, 28.54% between 6 and 10 times, and 11.76% 11 to 15 times. A further 11.22% reported more than 16 drinking sessions, and 19.4% did not answer. Further, 90.6% of 1106 respondents reported binge-drinking at least once in the preceding 12-months. Of these, 20.52% reported at least 30 episodes of binge-drinking, and the largest group (23.6%) reported between 1 and 5 episodes. Participants were also asked if they had ever played a drinking game, 91% of 1100 respondents confirmed they had. Of these, 40.74% reported that drinking games happened on all, or a lot of, drinking occasions.

How prevalent are MBO experiences?

When asked if participants had ever experienced a ***fragmentary*** **blackout**, 849 of 1132 respondents (75%) reported knowingly having ever experienced an MBO (lifetime prevalence). The remaining 283 participants said they were either not sure (n = 40, 3.53%), that they did not drink (n = 64, 5.65%), or had never had a blackout (n = 179, 15.81%). Estimated period prevalence of *fragmentary* blackouts over the preceding 12-months (see Table 3.5) was 61.48%; calculated from the total number of participants (n = 1132) minus all participants who have no recollection of ever blacking out (n = 283) and non-responders (n = 153).

Additionally, from 1064 responses, lifetime prevalence of *en bloc* blackouts was 58.08% (n = 618). Remaining participants were either not sure (n = 60, 5.64%), or said it had never happened to them (n = 386, 36.28%). We estimated 1-year period prevalence for *en bloc* blackouts to be 38.72%, by contrasting those who were certain they experienced an *en bloc* blackout to those who were not.

There was a significant positive correlation between the amount of pre-drinking reported by students (n = 725), and our MBO Metric, r = 0.37, p < .001, r^2 = 13.69%. Unsurprisingly, pre-drinking before going out was associated with increased MBO experiences.

What other factors predict MBO experiences?

We ran a multiple linear regression to test whether age, year of degree, gender, family drinking history, PISD, or personal drinking behaviours predicted MBO scores (n = 724). Results are detailed in Table 3.7. In brief, the full model explained 30% of the variance and was a significant predictor of MBO experiences, $F(6,556) = 41.88$, p < .001. Only the personal drinking (β = 0.308, p < .001, $\eta^2 = 0.22$) and PISD (β = 0.149, p < .01, η^2 = 0.01) metrics significantly predicted MBO scores.

Family drinking history, year of degree, gender and age were therefore removed from the model and the regression was recalculated. The new, reduced model accounted for 33% of the variance and both personal drinking (β = 0.322, p < .001, η^2 = 0.27) and PISD(β = 0.109, p = .016, η^2 = 0.005) were again significant predictors of MBO scores, $F(2,721) = 175.2$, p < .001. Because of the violation of normality for MBO

scores, we ran a robust bootstrapped version of the reduced model, resampling 1000 times data for each partial regression coefficient. Bootstrapped results confirmed the analysis of the reduced model, where personal drinking (β = 0.51, 95% CI 0.28, 0.36) was more important than peer influenced student drinking (β = 0.11, 95% CI 0.07, 0.26*)* as a predictor of MBO experience (see Table 3.7).

Table 3.6:

Descriptive statistics, Variance Inflation Factors and correlation coefficients

Variable	VIF	Mean	SD	1	2	3	4	5	6
1. Gender	1.08			1.00					
2. Age	1.28	19.66	1.7	-0.1	1.00				
3. Family History	1.03	5.7	4.44	0.08	0.12	1.00			
4. Personal Drinking	1.16	15.75	6.89	-0.23	-0.05	0.02	1.00		
5. PISD	1.13	8.43	2.9	-0.04	-0.09	0.05	0.3	1.00	
6. Year of Degree	1.25	1.66	0.87	0.02	0.42	0.03	0.04	0.09	1.00

Table 3.7:

Regression Coefficients

	ΔR^2	B	SE B	ß	*p*	Lower Bound CI 95%	Upper Bound CI 95%
Full model	0.31				< .001		
Constant		-1.63	2.02		0.42	-5.6	2.33
Age		0.03	0.1	0.01	0.73	-0.16	0.23
Family History		0.03	0.03	0.03	0.44	-0.04	0.09
Personal Drinking		0.31	0.02	0.51	< .001*	0.26	0.35
PISD		0.15	0.05	0.1	0.01*	0.04	0.25
Year of Degree		-0.23	0.18	-0.05	0.21	-0.59	0.13
Gender		-0.34	0.36	-0.04	0.34	-1.04	0.36
Reduced Model	0.33				< .001		
Constant		-1.39	0.43		.001	-2.22	-0.55
Personal Drinking		0.32	0.02	0.54	< .001*	0.28	0.36
PISD		0.11	0.05	0.08	0.02*	0.02	0.2
Bootstrapped Model	0.3				< .001		
Constant		-1.92	0.45		< .001	-2.77	-1.07
Personal Drinking		0.34	0.02	0.51	< .001*	0.28	0.36
PISD		0.13	0.05	0.11	< .001*	0.07	0.26

*Note: * denotes significant differences at p < .05.*

Is drinking behaviour predicted by family drinking history, or location of family home?

We investigated whether family drinking history scores were associated with personal drinking and peer influenced student drinking. There was a positive but non-significant correlation between both personal drinking behaviour and family drinking history (n = 569, p = .580), and PISD and family history (p = .186).

To determine whether location of family home predicted drinking behaviours, respondents (n = 1073) were split into groups based on family home country - Scotland, England, Northern Ireland and Other (rest of the world; 58 countries were represented which, by continent, consisted of 8% of participants from Asia, 3% from Africa, 12% from North America, 3% from South America, 72% from Europe, and 2% from Australasia). Welsh participants were excluded due to small sample size (n = 3). A factorial MANOVA using Pillai's trace, with PISD and personal drinking metrics as outcomes, found a significant main effect of home country, V = 0.065, $F(6, 2138) = 12$, $p < .001$. Pairwise comparisons revealed significant effects of home country on both personal drinking behaviour, $F(3,1069) = 21.16$, $p < .001$, $\eta^2 = 0.06$, and PISD, $F(3,1069) = 8.84$, $p < .001$, $\eta^2 = 0.02$. We further applied a robust MANOVA, accounting for any violations of normality, which confirmed a significant main effect of country on drinking behaviours, $H(6) = 22.5$, p = .001. Compared to Other (rest of the world), England, Northern Ireland and Scotland all scored significantly more on the personal drinking behaviours metric ($p < .001$). Moreover, in comparison to the rest of the world, participants from Northern Ireland and Scotland were significantly more likely to be influenced by peer behaviour/attitudes ($p < .001$), than English participants (p = .051).

Having ascertained an effect of home country on drinking behaviours, a categorical mediation analysis was then used to investigate whether any effect of home country on MBO scores was mediated by personal or peer-influenced student drinking. England, Northern Ireland and Scotland were individually compared to the rest of the world (Other). The effect of home country on MBO scores was partially mediated by PISD scores. Figure 3.1 shows that the total effects regression coefficients between Northern Ireland and Scotland, and MBOs, were significantly different from the rest of the world, whereas England was not. The indirect effect was 0.32. We tested the

significance of this indirect effect using bootstrapping procedures, per country. Unstandardised indirect effects were computed for each of the 1000 bootstrapped samples. For England, this effect was 0.27, 95% CI [-0.047, 0.59], for Northern Ireland it was 0.64, 95% CI [0.24, 1.04], and for Scotland it was 0.3, 95% CI [0.1, 0.51]. Sobel tests of indirect effects were statistically significant for Northern Ireland (p = .001) and Scotland (p = .004), but not for England (p = .1) suggesting that partial mediation occurred. In sum, compared to the rest of the world and England, Scottish and NI students were influenced more by their peers, which in turn affected the frequency and severity of MBOs.

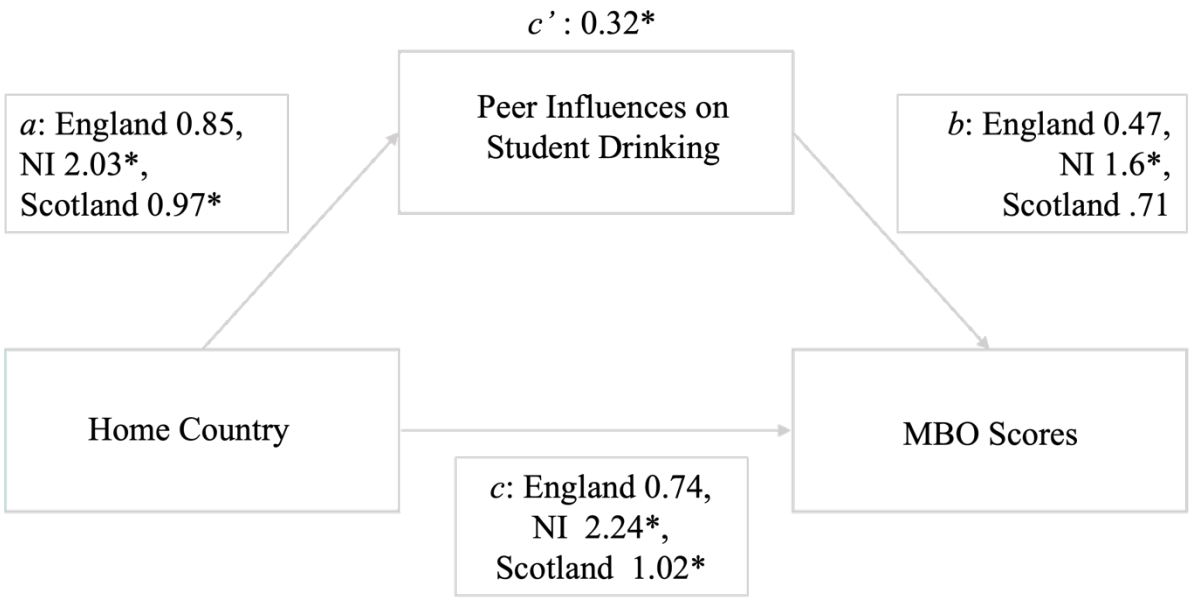

Figure 3.1: **PISD Model.** Hypothesised mediating relationship of peer influenced student drinking on home country and MBO scores.

Personal drinking behaviour scores also mediated the effect of home country on MBO scores. In comparison to the rest of the world, Figure 3.2 shows that the regression coefficients between England and MBO scores were not significant, but that being from Northern Ireland or Scotland did have a significant effect on MBO scores. The indirect effect was 0.34. Again, we tested the significance of this effect by bootstrapping procedures per country. For England, the bootstrapped unstandardised indirect effect was 0.95, 95% CI[0.15, 1.72], for Northern Ireland this was 1.82, 95%

CI[0.9, 2.73], and for Scotland was 1.19, 95% CI[0.7, 1.67]. All indirect effects were statistically significant and therefore mediation did occur. Note that the *b* pathway does not appear significant due to the strong correlation between personal drinking and MBO score [r (721) = 0.57, p < .001], that remains irrespective of country of origin [Rest of world: r (116) = 0.61, p < .001; England: r (47) = 0.4, p < .001; Scotland: r (515) = 0.55, p < .001; NI: r (37) = 0.69, p < .001].

Figure 3.2: **Personal Drinking Model.** Hypothesised mediating relationship of personal drinking behaviours on home country and MBO scores.

In summary, there was a significant effect of family home location on personal drinking behaviours. Mediation analysis showed that being from Northern Ireland and Scotland had a direct and significant effect on MBO scores but being from England did not. Personal drinking scores mediated this association for all three countries.

Is year of degree associated with drinking?

A series of ANOVAS were conducted to consider the effect of the year of undergraduate study on the MBO, PISD and personal drinking behaviour metrics. Levene's test was not significant for MBO scores (p = .98), for personal drinking behaviour scores (p = .562), or PISD scores (p = .737) per year group therefore

variances between cohorts did not differ. There was no main effect of year on MBO scores, $F(3, 715) = 0.25$, $p = .859$, $\eta^2 = 0.001$. There was also no main effect of year on the personal drinking metric, $F(3, 1066) = 1.3$, $p = .275$, $\eta^2 = 0.004$. We did however find a significant main effect of year of degree on the PISD metric, $F(3, 1124) = 4.44$, $p < .01$, $\eta^2 = 0.01$; paired t-tests revealed that second years ($M = 8.35$, $SD = 2.85$) were more influenced by peers than first years ($M = 7.75$, $SD = 2.85$), $t(3) = 3.26$, $p = .001$, but there was no difference between first and third ($M = 7.47$, $SD = 2.64$), or final year ($M = 8.21$, $SD = 2.79$) students.

Is there an influence of gender on drinking behaviour?

To test for differences between genders on the MBO, PISD and personal drinking behaviour metrics, a series of ANOVAs were conducted. Individuals identifying as other than male or female were removed from analysis due to the small number of participants. Levene's test was significant for both the MBO metric ($p < .001$), and the Drinking metric ($p < .001$) therefore Welch's correction was applied. There was no difference in variance for the PISD metric ($p = .23$). Males scored higher than females on all three metrics. In detail, there was a main effect of gender on MBO scores, Welch's $F(1, 233.21) = 13.196$, $p < .001$, $\eta^2 = 0.02$ (Males $M = 5.71$, $SD = 4.74$; Females $M = 4.26$, $SD = 3.79$). Gender also influenced personal drinking scores, Welch's $F(1, 348.2) = 37$, $p < .001$, $\eta^2 = 0.04$ (Males $M = 15.98$, $SD = 8.1$; Females $M = 12.49$, $SD = 6.95$). Finally, there was no effect of gender on PISD, $p = .632$.

Discussion

This study investigated the relationship between alcohol-induced memory blackouts, influences on drinking behaviour, and students attending Scottish universities. To start, one of our aims was to identify the lifetime prevalence of alcohol-induced MBOs, split by severity of MBO (*fragmentary* or *en bloc*), and the prevalence of MBOs in the past 12 months. A large proportion of students reported regular alcohol consumption and drinking to binge levels, with over half of all respondents reported having experienced an MBO, with lifetime prevalence of *fragmentary* and *en bloc* blackouts at 75% ($n = 1132$) and 58% ($n = 1064$) respectively. Period prevalence was 61.48% and 38.72%, highlighting the frequent occurrence of such events. A significant

proportion of our sample experienced regular MBO events over the past 12 months, highlighting the occurrence of regular binge-drinking to extreme episodes.

Unsurprisingly, scores on our personal drinking metric predicted MBO experiences, highlighting that individuals who drank more heavily experienced more blackouts. While this finding may seem unremarkable, two factors are critical in experiencing a blackout: the amount of alcohol consumed, and the time taken to consume it. Our personal drinking measure did not ask about the time taken to drink, yet we found a strong association between personal drinking behaviours and MBOs experienced. Further, binge-drinking is separated from sustained alcohol consumption by ingestion of excessive quantities in one session, which is more likely to lead to an MBO, followed by periods of abstinence. People who regularly drink alcohol are not necessarily binge-drinkers and would not necessarily experience frequent MBOs.

We expected to find an association between peer influences and drinking behaviours, partly reflecting the wider culture of drinking within Scotland. The PISD metric included questions which centred on the pressure to drink experienced by students from friends, and their perceptions of peer drinking behaviours influenced by cultural norms. This metric was found to predict scores on our MBO metric and was also associated with year of study. Those in their second year were more likely to be influenced by peers than those in the other three years of their Scottish degree programmes. It may be that by second year, students have settled into university life and have formed social friendship groups with whom they enjoy spending time, and likely have moved into accommodation with. In many (although not all) Scottish degrees, first and second-year grades do not count towards final degree classifications therefore while assessment is important for degree progression, there is less jeopardy attached to poor results. This increased academic demand may explain the reduction in peer influence seen as students enter third-year.

A regular feature of many student's drinking behaviours is pre-drinking with friends before a night out (Elgàn et al., 2019; Pedersen et al., 2009). We asked our participants to quantify the extent of their pre-drinking, although not whether this took place with friends or alone. We found that quantities of alcohol consumed when pre-drinking was correlated with MBOs, consistent with wider literature (Wahl et al., 2013; Wetherill & Fromme, 2016). Although students report pre-drinking to give them

confidence or to make the evening more enjoyable (Smit et al., 2021), more commonly the intent may be to get drunk cheaply (Pedersen et al., 2009). This pre-partying practice has been associated with increased risks for individuals, including aggression (Wahl et al., 2013), hangover, injury (Smit et al., 2021) and rape (Jaffe et al., 2022), in addition to increased memory blackouts. Further, 91% of our sample reported having played a drinking game with only 18.52% saying that this was a rare occurrence. Drinking games – a regular element of social pre-drinking events - often mean rapid consumption of alcohol, a practice which can spike BAC% and increase likelihood of MBOs (A. M. White et al., 2002). Future work should consider the impact of drinking games specifically on MBOs in our student population to investigate correlation between the practice and blackouts.

We predicted that family history of alcohol consumption would influence drinking behaviour and/or MBO experiences, yet this did not correlate with either metric. Parental influence is known to have a depressing effect on student drinking behaviours (Wood et al., 2004), particularly in the first year of study (Borsari et al., 2007) and has also been shown to weaken the influence of peers on drinking behaviours in adolescents (Balsa et al., 2018; Y. M. Kim & Neff, 2010). We cannot claim to have measured parental influence directly, since our family drinking history questions were focused more on indirect influence via drinking culture within the home. In this respect, there is mixed evidence for the extent to which parental drinking influences adolescent behaviour. For example, a longitudinal study by Van Damme et al. (2015) showed no direct relationship between parental drinking and that of their children at age 19, which is in contrast to other studies (for example, H. R. White et al., 2000). These differences could reflect whether parental drinking was measured from the parent or child's perspective, but could also be indicative of the wider positive influences on behaviour across adolescence, for example from sports clubs (Kuntsche et al., 2004). Finally, we did not explicitly recruit in our sample those with a family history of alcoholism/dependence since this was not the purpose of the study, thus, instances of familial alcoholism in our sample are likely to be relatively rare.

While our sample may not have shown direct family influence, there was an effect of home country on MBO scores which was mediated by personal and PISD scores. Being from Northern Ireland returned the highest average scores on both

metrics, with Scottish participants scoring second highest. Interestingly, we show that MBO scores for Scottish participants only were impacted more by peer influence than by personal drinking. This could indicate a Scottish cultural influence on social drinking behaviours which is more ingrained than other countries in our sample, despite Northern Irish respondents recording higher drinking scores. On the other hand, it may reflect an attachment to an in-group culture, that is, participants studying in Scotland identifying as being Scottish, compared to being from another country and studying in Scotland.

Overall, drinking in young adults has reduced over recent years (Oldham et al., 2018), and although males had previously reported drinking more than females, drinking patterns have been shown to be either converging (Davoren et al., 2016), or with no difference between genders (Northcote & Livingston, 2011; Stockwell et al., 2014). However, we found that males reported more MBOs and heavier drinking than females. It may be that our findings do not contradict those of Davoren and colleagues, as we have no way of knowing how big the difference would have been in the past, albeit with a different sample. It should also be noted that our participants may have either under, or over, estimated their drinking. For example, young males were found more likely to underestimate their drinking than young females in one study (Livingston & Callinan, 2015). However, overestimating may be an issue for students wishing to appear to drink more in front of peers (N. Schwartz et al., 1998). Self-report can be a reliable method of collecting alcohol consumption data (Del Boca & Darkes, 2003). Anecdotal conversations with students who had completed our survey indicated that some under-reporting occurred due to feelings of shame and embarrassment over their alcohol consumption and frequency of MBOs. Our data suggests that the more alcohol participants reported consuming, regardless of any under/over-estimation, the reported frequency of MBO experiences also increased. Thus, we suggest that MBO events should be considered reliable markers of extreme binge-drinking behaviour.

Our study was advertised explicitly to both drinkers and non-drinkers, nevertheless, the sample contained significantly more alcohol drinkers than those who abstain. This may be indicative of drinking within the Scottish university culture (Delk & Meilman, 1996), and the wider student population in general (Davoren et al., 2016;

Tavolacci et al., 2016). Nevertheless, it could be that participants in alcohol-related studies are already more likely to drink than those who do not. Further, the metrics we created could be considered unequally weighted. For example, in the personal drinking behaviours metric, whether reporting number of times drinking per month is equivalent to either number of times being drunk in a year, or number of episodes of binge drinking, is debatable and yet they were scored similarly. However, the question weightings were the same for all participants, and conservative, hence it is not possible that we have over-amplified effects which are not really present in the data; the reverse is actually true. The combination of questions included in each metric was designed to provide a reflection of overall drinking behaviour and therefore the effects of any discrepancies for individual items or participants would be minimised.

In summary, we report the period prevalence of binge-drinking in a Scottish student population to be 90.6%, which also allows for a high period prevalence of alcohol-induced memory blackout experiences (61.48% for *fragmentary* blackouts). We stress the point that alcohol-induced MBOs should be considered reliable markers of binge-drinking events – the more alcohol participants consumed, the more instances of MBOs they reported experiencing. Apart from their own personal drinking behaviour, participants were significantly influenced into drinking more by their peers and cultural expectations. Further, those from the UK reported drinking more than those from other parts of the world, with students from Scotland being most influenced by peers. Our findings highlight the scale of the binge-drinking problem in Scotland, with a focus on harmful binge-drinking events characterised by alcohol-induced memory loss, a problem we need to quickly address. Future work should establish how acute alcohol-induced MBO events affect everyday functioning, how prevalent MBOs are in the rest of the UK, and whether successive MBO events disrupt quality of life in any way.

Chapter Four:

The morning after the night before: Alcohol-induced blackouts impair next day recall in sober young adults

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Abstract

Binge-drinking in adolescents and young adults is a widespread problem, however, an often-unreported consequence of binge-drinking behaviour is an alcohol-induced memory blackout (MBO). An MBO is a transient amnesic event resulting from rapid, excessive alcohol consumption. Here, we examine the short-term impact of an alcohol-induced MBO event (testing < 20 hours after blackout) on memory performance in people who have experienced a high volume of MBOs. In addition, we aimed to test the hypothesis that people who experience a high volume of MBOs may have poorer recall than non-blackout controls in either sober or intoxicated states. Three episodic memory paradigms consisting of free recall, serial recall, and depth of encoding tasks, were conducted by a group of alcohol drinkers who had never experienced a memory blackout, and those who reported at least 9 in the preceding 12-months. Studies were completed sober and after alcohol by all participants, and sober but after blackout by the experimental group. Accuracy of recall was assessed with linear mixed effects modelling for all experiments and conditions. Recall rate both before and after alcohol consumption was similar between groups, with poorer recall after drinking alcohol by all participants in all three studies. After blackout, MBO participants showed no significant improvement from their intoxicated state in serial recall and depth of encoding tasks, but an improvement in free recall. Further analysis of these findings revealed that 10 out of 23 participants showed significantly impaired performance after blackout during free recall, extending up to 17 participants in serial recall. In general, alcohol reduced recall rate in both blackout and control participants similarly, but recall following MBO remained poor. Our evidence suggests that alcohol-induced blackouts impair memory functioning the next day, and future research should establish the duration of deficits after an acute alcohol-induced blackout episode.

Introduction

An alcohol-induced memory blackout (MBO) is a transient amnesic event during which the individual remains conscious in the environment but loses the capacity to form long term episodic memories (that is, memories for lived events and experiences). They are elicited by binge-drinking causing a rapid spike in blood alcohol content. Binge-drinking within adolescence and young adults is accepted as a global problem (Elgàn et al., 2019; Hingson & White, 2013; Intergovernmental Committee on Drugs, 2015; Johnston et al., 2015), yet the immediate consequences of binge-drinking, which can lead to an MBO, are rarely discussed. In sum, the long-term damage to people engaging in binge-drinking practices may in part be attributable to the frequency of MBOs experienced, that is, blackout events can be considered a marker of extreme alcohol binge-drinking, which in turn could inhibit memory and cognitive functioning more than average levels of alcohol consumption. Thus, the aims of the present paper are to (1) identify whether young adults who experience a high volume of MBOs are poorer in terms of episodic memory performance compared to non-blackout controls, either when sober or after ingesting alcohol, and (2) assess whether memory performance remains impaired the day after an alcohol-induced blackout, in sober young adults.

An MBO occurs when a rapid rise in blood alcohol levels disrupts processing within the hippocampus (A. M. White, 2003). As well as reducing cortical activity (through the known actions of alcohol on Glutamatergic and GABAergic neurons), alcohol leads to the inhibition of CA1 pyramidal neurons (A. M. White & Best, 2000), likely disrupting the transfer of information from short to long term storage, and as a result, the ability to retain new memories is restricted. Two types of MBO have been identified – *fragmentary* and *en bloc* (Goodwin, et al., 1969a). The term fragmentary blackout describes the more commonly experienced type of MBO, where episodic memory is punctuated by brief periods of memory loss. Some recovery of episodes has been observed in people after experiencing a fragmentary blackout, yet this often follows from cues by peers (Goodwin et al., 1969a). In contrast, an *en bloc* blackout could be described as a complete inability to form any new memories over an extended period of time, with no recovery of any episodes. There is a dose dependent

relationship between alcohol and MBOs, with fragmentary blackouts not normally reported in levels of less than 0.06% BAC, while *en bloc* blackouts are typically reported following higher blood alcohol levels than a fragmentary blackout (A. M. White et al., 2004).

Interestingly, not all heavy drinkers experience blackouts (Goodwin et al., 1969a; Nelson et al., 2004), and it is known that a wide range of factors influence when, or even if they occur. For example, the quantity of alcohol consumed and speed of drinking (Jennison & Johnson, 1994; A. M. White, 2003), gender (Marino & Fromme, 2015), physiological differences (Goodwin et al., 1969a; Nelson et al., 2004), environmental influences (A. M. White et al., 2002) and genetics (Marino & Fromme, 2015; Nelson et al., 2004), may all be indicators of blackout likelihood. It could be argued that these factors make adolescents particularly vulnerable, for example, in a university environment, students are often distanced from parental influence while at the same time they are encouraged to participate in binge-drinking culture (Balodis et al., 2009; Keeling, 2002). Common among students, both 'pre-drinking' (drinking large quantities of alcohol at home before going out with the purpose of getting drunk cheaply) and drinking games (typically involving quickly ingesting large quantities of spirits), are known to increase the chance of experiencing an MBO (Elgàn et al., 2019; A. E. Ray et al., 2014; Wahl et al., 2013). Alcohol blackouts can have serious detrimental effects for the individual experiencing them, for example they are associated with a higher risk of personal injury (Hingson et al., 2016), and increased likelihood of engaging in vandalism, physical aggression, and sexual activity with strangers (A. M. White et al., 2004). In addition, studies have shown that around 50% of university students have experienced an MBO within the preceding 12-months (N. P. Barnett et al., 2014; A. M. White et al., 2002), highlighting the endemic nature of the MBO event experienced by young adults.

Since alcohol-induced MBOs are endemic in some young adult populations, to what extent do the consequences of this extreme binge-drinking impart any damage to cognition or the brain? We already know that alcohol detrimentally affects plasticity in areas related to memory and learning, thereby altering cognitive processes and normal functioning (Zorumski et al., 2014). We also know that alcohol can cause harm to the developing brain through prenatal exposure (for example, Goodlett & Horn,

2001; Granato & Dering, 2018), and during early adolescence (Ferrini et al., 2018; Squeglia et al., 2009), thereby changing the developing brain. It is therefore plausible to expect that frequent blackout experiences, constituting extreme binge-drinking episodes, would potentially alter the structure of neural networks. In addition, some pre-existing neuroanatomical differences may be present between individuals who progress into heavy drinking, and therefore regularly experience MBOs, and those who do not (Wetherill et al., 2013), suggesting a predisposition towards heavy alcohol drinking. Indeed, longitudinal work by Squeglia and colleagues (2014) reported reduced grey matter volume in alcohol-naive adolescents who later transitioned to moderate binge drinking. Subsequent drinking by these individuals resulted in further abnormal reduction in the volume of subcortical and temporal brain structures (Squeglia et al., 2014) .

Since the brain continues to develop throughout adolescence, with cognitive and structural changes observable even in the mid-20s (de Graaf-Peters & Hadders-Algra, 2006; Spear, 2013), it is critical to understand whether or not an alcohol-induced MBO imparts any lasting damage to cognitive functioning during young adulthood. There has however been very little investigation of episodic memory in those who regularly experience MBOs. One recent review on alcohol related MBOs reported only two studies which included a test of memory (Wetherill & Fromme, 2016). These papers both showed that alcohol impaired memory for contextual details (that is, the context surrounding or embedded with a to be remembered item) in participants who experienced blackouts (Wetherill et al., 2012; Wetherill & Fromme, 2011). These findings suggest the possibility that the linking of context with an episodic memory is suppressed by the experience of memory blackouts. More simply, after an alcohol-induced blackout, newly created memories might be less rich in detail.

How does memory operate under the influence of alcohol? A number of studies have investigated episodic memory performance when intoxicated, for example, freely recalling previously studied words, in any order, has been shown to be impaired following low doses of alcohol (Hashtroudi et al., 1984; Peterson et al., 1990). Conversely, cued recall, where recall is prompted by the presentation of visually degraded words or word stems for example, is unaffected by the presence of alcohol (Hashtroudi et al., 1984). Weafer and colleagues (2016) showed reduced recall in a

cued recall task for emotional stimuli when alcohol was given prior to encoding, in comparison to a placebo control. In fact, alcohol disrupted performance in both cued recall and memory recognition tasks (for example, do you remember seeing this word before, yes or no?) for emotionally valanced stimuli when alcohol was given prior to encoding, compared to afterwards during consolidation (Weafer et al., 2016). This result implies that emotionally valanced stimuli may be more deeply encoded than neutral stimuli, and subsequently more affected during intoxicated states; Craik (1977) suggests that encoding can be improved by processing items more deeply, that is, encoding with meaningful analysis. Further, Curran and Hilderbrandt (1999) proposed that alcohol may impair encoding of contextual details - peripheral information which could assist in deeper processing. In sum, alcohol appears to significantly impair episodic memory when given prior to encoding, with associated details to the episode being most affected.

Towards our goal of understanding memory performance in the aftermath of an MBO event, we conducted a series of standard episodic memory paradigms on participants who reported experiencing at least 9 MBOs in the preceding 12-months (MBO group). We compared their performance with a control group who have never experienced memory loss as a result of binge-drinking. We employed a free recall task as a baseline for memory retrieval performance, and a serial recall task to assess memory for events in their order of occurrence (Long & Kahana, 2019). We also added a depth of encoding manipulation to an immediate and delayed free recall task which compared recall for items embedded within a sentence context (deep encoding condition) vs. orthographic changes in items (shallow encoding condition). We did this to investigate if recall for items embedded in a context is affected more by an alcohol-induced MBO compared to our shallow encoding manipulation. The delay component (three minutes) within the depth of encoding task was included to assess the impact of frequent MBO events on memory consolidation over time.

Across the three experiments we expected to find that, while sober, performance between both control and MBO groups would be comparable, as observed in previous literature (Hartzler & Fromme, 2003; Wetherill & Fromme, 2011). In line with Wetherill and Fromme (2011), and as suggested by Curran and Hildebrandt (1999), we hypothesised an increased detriment to recall for items

embedded within a context (deep compared to shallow encoding) after ingesting alcohol for our MBO group, compared to controls, in our depth of encoding experiment. The novelty of our studies concerns the subsequent testing of our MBO participants when sober and after experiencing a blackout (<20 hours), to examine if any deficits in memory performance remain. We predicted that our MBO participants would show significant reductions in recall compared to baseline (*before-alcohol*) in all three experiments, indicating a lack of recovery in memory performance after the MBO event.

Methods

Design

Participants from the University of Stirling were recruited via online advertisements (no specific exclusion criteria) and asked to complete a general questionnaire examining their alcohol use, behaviours, and familial/peer group relationships with alcohol. Individuals meeting the inclusion criteria for participation in the laboratory-based study then received a follow-up email invitation to take part. These criteria were: (1) either never having experienced an MBO or experiencing 9 or more MBOs in the past 12 months, (2) being aged between 18 and 25 years, and (3) being a fluent English language speaker. In total, 53 participants were recruited, consisting of a control group (n=24, 12 males, mean age = 20.17, SD 1.99), and experimental group (MBO group) (n=29, 11 males, mean age = 19.55, SD=1.38). Our control group reported either abstinence from alcohol or drinking alcohol only on very rare occasions, and responses from both groups are given in Table 4.1. All MBO and control participants in the laboratory experiments were students at the University of Stirling. At the first testing session, participants completed each of the 3 behavioural tasks sober, and then repeated the experiments following a scaled dose of alcohol. All participants were compensated for their time with either course credit tokens, or £15. The MBO group were invited to return to the laboratory following a blackout event. On this visit, they completed the 3 behavioural experiments for a third time when sober again, and received additional course credit or money. Of the invited MBO group, 23

participants returned for the additional testing session (10 males, mean age =19.43, *SD* = 1.2; mean number of days between testing sessions = 24.74 ±30.77). The reduced number of participants at this testing session reflects normal experimental drop-out rate of up to 20% (Bell et al., 2013). The studies, and protocol for administration of alcohol, were approved by the NHS, Invasive and Clinical Research committee at the University of Stirling.

Table 4.1:

Self-reported frequency of drinking behaviours between MBO (n = 29) and Control (n = 24) groups

	Group	Never	10 or less	11-20 times	21-30 times	Over 30 times	Chi Square (df)	p value	Fisher exact p values
Drinking sessions, per month	MBO	0	14	10	4	1	16.543 (4)	0.002**	0.0002424**
	Control	3	20	0	1	0			
Drunk instances, per year	MBO	0	1	1	7	20	49.188 (4)	<.001**	6.416E-14**
	Control	7	17	0	0	0			
Binge drinking episodes, per year *	MBO	0	1	2	6	20	45.555 (4)	<.001**	6.416E-14**
	Control	10	12	1	0	0			
		Never	1-4 times	5-8 times	9-12 times	Over 12 times			
Fragmentary MBOs, per year	MBO	0	0	0	14	15	53 (2)	<.001**	1.283E-15**
	Control	24	0	0	0	0			
En bloc MBOs, per year	MBO	0	13	3	7	6	53 (4)	<.001**	1.283E-15**
	Control	24	0	0	0	0			

Drinking characteristics by frequency of response, and with statistical comparison between groups. Note that chi-square tests for independence may be inappropriate if any expected frequencies are below 5, therefore we also provide Fisher exact p values.

** Defined as more than 6 units of alcohol in a single session*

Free and serial recall tasks

All experiments were presented using experimental software E-Prime 1.2 (Psychology Software Tools, Pittsburgh, PA). In both the Free and Serial tasks (Figure 4.1), participants were presented with 3 blocks of 15 study words on a computer screen and asked to remember them. Stimuli were word lists taken from Roediger and McDermott (1995), totalling 270 unique stimuli split into 18 blocks (9 blocks free recall task, 9 blocks serial recall task). Blocks for each individual task were presented pseudo-randomly, counterbalanced across participants. In study blocks, individual words were presented for 1000ms, followed by a blank inter-trial interval of 2000ms. Following each study block of 15 words in the free recall task, participants were asked to recall as many words as they could remember, in any order, by typing their response onto the screen using a keyboard. They were given as much time as they wanted to complete the recall component for each block. The procedure was identical for the serial recall task, except participants were explicitly asked to recall stimuli in the order in which they had been presented.

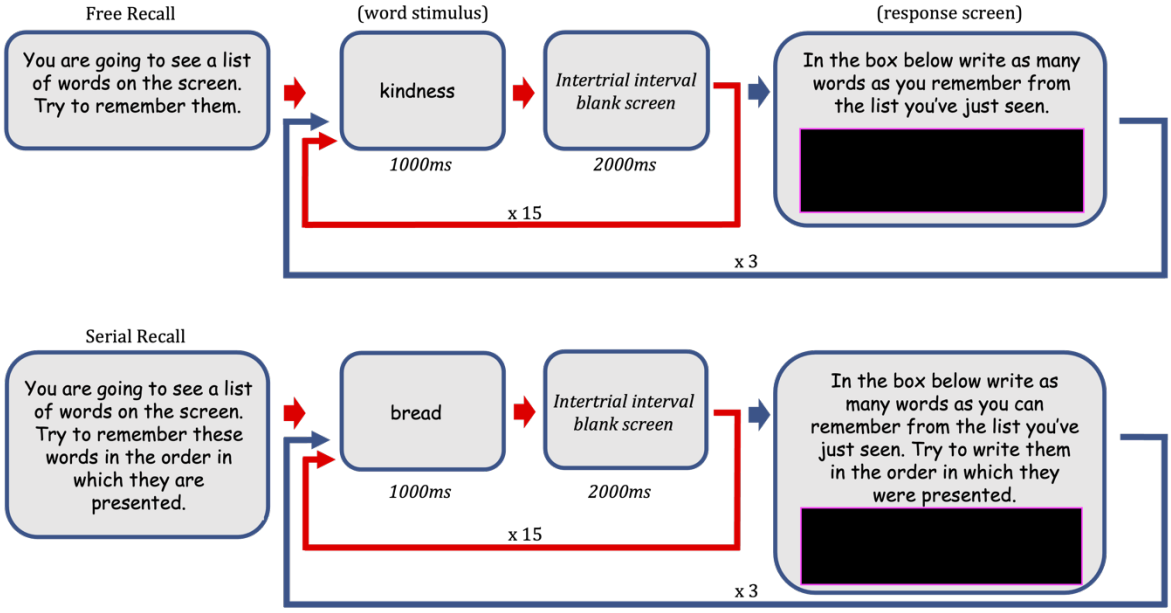

Figure 4.1: **Free and serial recall task structure.**

Depth of encoding task

The experiment consisted of 4 blocks of 15 randomly presented words; block order was also randomised, and blocks were split evenly between shallow and deep encoding manipulations (see Figure 4.2). All word stimuli were generated from the

MRC Psycholinguistic Database (Coltheart, 1981; M. Wilson, 1988) and were 5-9 letters in length, contained 2-4 syllables, and had a medium-high familiarity rating of 300-600. A total of 180 stimuli were used in the experiment, split into six blocks of deep and six of shallow stimuli, with the use of each individual block counterbalanced across all participants. In the shallow encoding blocks, stimuli were presented in either lowercase or capital letters for 3000ms. Participants were then asked if the word displayed had been in lowercase letters (yes/no judgement, response counterbalanced between participants, no time limit). In the deep encoding blocks, a sentence with a missing word appeared on-screen for 3000ms, followed by a target word below the sentence for an additional 3000ms. Participants were asked if the target word fitted the sentence (yes/no judgement, response counterbalanced between participants, no time limit to respond). Time taken between each trial for both shallow and deep encoded stimuli was 1000ms. Encoding manipulations (case judgement vs sentence) were based on methods from Craik and Tulving (1975). At test, for both shallow and deep conditions, participants were asked to freely recall as many words as possible, entering responses using a keyboard (immediate recall condition). They had unlimited time to do this. They were then given a distractor task for 3 minutes (Sudoku puzzles), followed by a repeated test session (delayed recall condition).

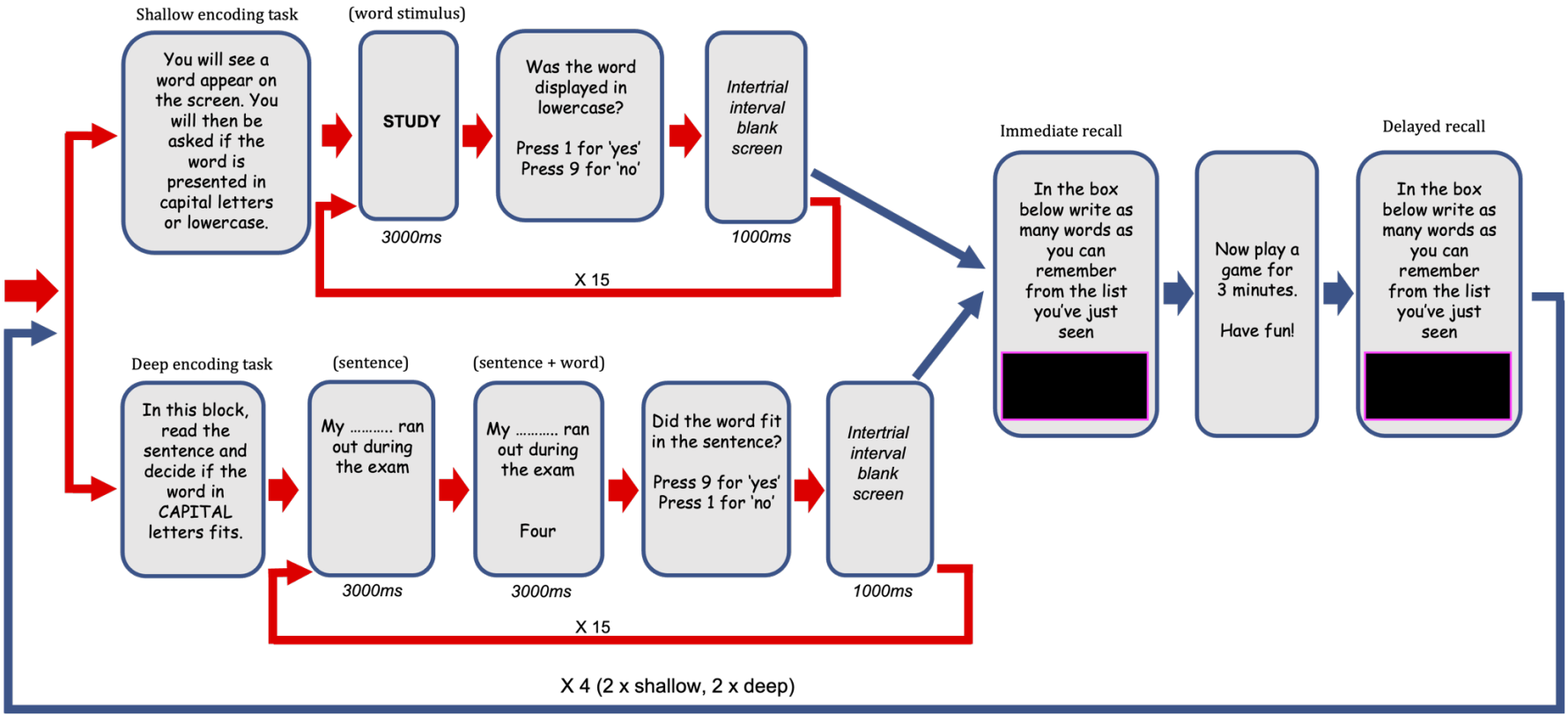

Figure 4.2: **Depth of encoding task structure.**

Procedure and alcohol protocol

Prior to attending the laboratory, participants were advised of exclusion criteria, and that they would be required to drink alcohol. Exclusion criteria included being under the age of 18, possibility of pregnancy, use of prescribed medication that may interact with alcohol (excluding the contraceptive pill), or previous substance abuse problems. Participants were asked to avoid alcohol for 24-hours and food for between 3 and 4 hours before the study.

Upon arrival, photographic identification, written consent and a breathalyser test (Dräger Alcotest® 3000; Lübeck, Germany) were provided by participants. Height and weight were recorded and entered into an alcohol-dose formula (Watson, 1989), along with gender and age. The formula was designed to dose each participant with enough alcohol to reach a Blood Alcohol Content percentage (BAC) of 0.06%, estimated at consistent intervals throughout testing from breath alcohol content (BrAC).

In the first lab visit participants completed all tasks when sober, before receiving undiluted 37.5% proof vodka in a glass tumbler with an optional glass straw. Prior to consumption, the vodka was kept in a freezer to minimise taste intensity. Participants were then asked to drink their vodka dose 'as quickly as was comfortable' to elicit a rapid spike in BAC. Fifteen minutes after alcohol consumption they gargled with water to remove any residue trace alcohol in the mouth, before being breathalysed. They then repeated the 3 tasks, submitting to additional breathalyser tests at regular intervals to measure the BAC spike and decline. In total, participants gave five BrAC recordings during the course of participation. Table 4.2 details the quantity of alcohol administered, the mean time taken to consume the alcohol, and subsequent mean BrAC readings across the duration of the tasks. Participants were asked to remain in the laboratory until their BrAC had dropped below the Scottish driving limit (BrAC 0.22mg/l, BAC 0.05%) during which time they were offered soft drinks.

Table 4.2:

Alcohol dose, drinking time, and mean BrAC

				Breath Alcohol (mg/l)		
	Vodka (ml)	Alcohol (g)	Drink Duration (secs.)	BrAC	Peak BrAC	Final BrAC
Whole Group	94.75 (22.1)	35.533 (8.29)	73.42 (83.76)	0.20 (0.06)	0.37 (0.07)	0.19 (0.04)
Controls (n=24)	97.75 (22.28)	36.656 (8.35)	72.82 (92.35)	0.22 (0.06)	0.36 (0.08)	0.20 (0.05)
MBO (n=29)	92.28 (22.03)	34.603 (8.26)	73.83 (79.15)	0.19 (0.05)	0.33 (0.04)	0.18 (0.04)

Means with standard deviations given in brackets

The studies were presented in two counterbalanced blocks – the free recall and serial recall tasks were combined into one block, and the depth of encoding task in another block. The free and serial study word lists were utilised in a DRM recognition memory task which was presented immediately following the serial task. Analysis of this recognition memory task was outside the scope of this manuscript focussing on recall and is therefore not reported. The free recall task always came before the serial recall task, to reduce influence of any memory strategy or heuristic employed in the serial recall task being applied to the free recall task. Presentation order of the two blocks was sequentially changed between participants, and also within participants when on returning visits (MBO group).

MBO protocol

The follow-up visit from the MBO group was timed to take place within 20 hours after experiencing an MBO. Participants were asked to keep a drinking diary over the course of six weeks, which consisted of their self-reported alcoholic beverage consumption on each day of the week, for six weeks, beginning at the onset of sign up to the study, used to track their average drinking behaviour. If the participant attended a drinking event which resulted in a blackout, they were asked to contact the researcher to arrange a testing session in the laboratory. To be clear, *no participants* were asked to binge-drink for the primary purpose of this experiment, their follow-up visits were voluntary and at their own instigation. The *after-MBO* laboratory sessions all took place in the afternoon, with no tests conducted before midday to allow

adequate time for the participant to sleep and recover and detoxify (Mean sleep duration = 6.55 hours ±2.05). On arrival all participants were breathalysed and only tested if their BrAC reading was 0.00 mg/l, signifying their return to a sober state. Participants were then asked to complete a consent form, verbally questioned on when they started and stopped drinking, duration and quality of sleep, and details of their blackout experience. All MBO participants confirmed having experienced a memory blackout prior to testing.

We report notable drinking characteristics given by MBO participants who returned for follow-up testing in Table 4.3. Participant's self-reported drinking behaviour is also given, recorded from participant's drinking diaries. One of the male participants' diary data was not filled in correctly, hence only data from 22 of the 23 are included in those figures. We also report the average number of drinking sessions per week, the amount of alcohol drank in one week, average amount of alcohol drank in any one session, and the participant's maximum alcohol drank for any one session. Note that these reports are likely to be underestimates due to the fact that reported values entail only what our participants remembered drinking at a particular event (see Devenney et al., 2019). Furthermore, UK definitions of binge-drinking suggest 6 or more units in any one session (for females, 8 units for males) constitutes a binge-drinking episode. Data from Table 4.3 suggests that our MBO participants engage in drinking alcohol 1.89 times per week, yet when they drink, they consume more than 6 (or 8) units for each session, that is, our MBO participants binge-drink heavily.

Table 4.3

Self-reported frequency of drinking behaviours of MBO group

	Never	10 or less	11-20 times	21-30 times	Over 30 times
Drinking sessions, per month (n = 23)	0	11	7	4	1
Drunk Instances, per year (n = 23)	0	1	4	5	16
Binge-drinking episodes, per year * (n = 23)	0	1	2	4	16

	Never	1-4 times	5-8 times	9-12 times	Over 12 times
Fragmentary MBOs, per year (n = 23)	0	0	0	12	11
En bloc MBOs, per year (n = 23)	0	11	3	5	4

	UK Units	Grams Ethanol (g)	Number of Sessions
Per week (n = 22)	26.99 (11.404)	215.918 (91.235)	1.89 (0.661)
Per session (n = 22)	13.364 (4.342)	106.912 (34.739)	
Max per session (n = 22)	21.225 (8.326)	169.8 (66.611)	

Frequency of responses to drinking behaviour questions, and quantity of alcohol consumed over a 6-week period given as mean scores with standard deviation in brackets. A drinking session refers to a single drinking event of unspecified duration.

** Defined as more than 6 units of alcohol in a single session*

Statistical analysis

We used linear mixed models (LMM) to analyse data from all experiments and to account for the difference in sample size between control and MBO participants, and multiple samples taken from the same participants at different timepoints (see Appendix 1.1 and 1.2 for full model outputs and structure). In the free and serial recall tasks we assessed the percentage of accurately recalled words, and frequency of false alarms, with fixed effects of alcohol (before and after alcohol), and group (control and

MBO). Random effects were not included in our modelling structure since our categorical variable of group (control vs. MBO) was central to our hypotheses, that is, there was no underlying categorical group structure where using random effects would improve the estimations of our models. We also did this for the MBO group only, looking at the impact of MBOs, compared to before and after drinking alcohol conditions (see Figure 4.3). To be clear, when we discuss an after-MBO effect, or a blackout effect, we are referring to any statistical difference between sober (*before-alcohol*) and *after-MBO* conditions. We used Bonferroni corrected paired t-tests, reporting Bonferroni adjusted *p* values, to compare the within-group means for the MBO group. In addition, in the serial recall task we further investigated sequence length (recalling 2 or more words in the correct order).

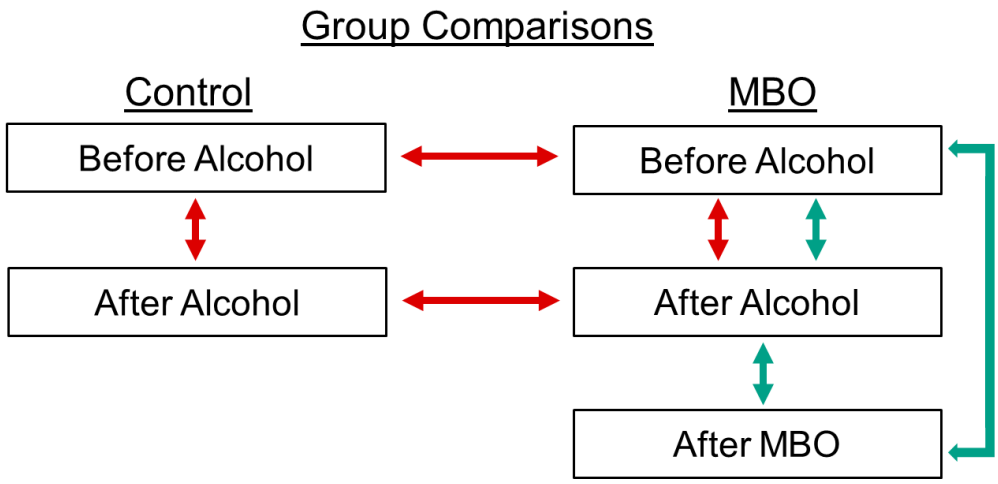

Figure 4.3: **Analysis structure.** Displays the design structure for all three experiments. Red arrows show the between group comparisons, comparing control and MBO participants before and after-alcohol. The green arrows highlight the design for the analysis of the MBO group data only.

For the depth of encoding task, models were conducted on accurately recalled words (%) and false alarms split by alcohol conditions, both between groups and within the MBO group only. We analysed fixed effects of group, alcohol, depth (shallow vs. deep) and delay (immediate vs. 3-minute delay), and also the interactions between these effects. All analysis was conducted using R (R Core Team, 2019) and *nlme*

(Pinheiro et al., 2019). Only results of interest are reported (all significant effects and selected non-significant results), and the effect sizes of planned contrasts, given by $r_{contrast} = \sqrt{\frac{t^2}{t^2+df}}$ (Rosenthal, 1991; Rosnow & Rosenthal, 2005).

Since we were interested in whether individual participants were significantly impaired after experiencing an MBO, we first ruled out the possibility that sleep impacted performance on the tasks, by correlating time slept with the difference in recall performance between *before-alcohol* and *after-MBO* conditions. Because we are interested in support for the null hypothesis, we include equivalent Bayes Factors (*K*) for all tests conducted (Morey & Rouder, 2018). Finally, to further quantify the differences between *before-alcohol* and *after-MBO* conditions in individuals, we resampled the ordering of *before-alcohol* and *after-MBO* conditions for each MBO participant 2000 times to build test distributions of possible mean differences (converted to z scores) between *before-alcohol* and *after-MBO* conditions (see Figure 4.3 for comparisons). For all three tasks we compared each individual participant's sampled mean difference (z scores) between *before-alcohol* and *after-MBO* conditions to our resampled test distributions to verify precisely how many participants showed significant memory deficits in each task.

Results

Between groups analysis: Control vs. MBO participants

Free recall

Comparing the free recall accuracy between groups, we found a significant main effect of alcohol, $X^2(1) = 63.96$, $p < .0001$. To summarise the model, the main effect of alcohol was a reduction *after-alcohol* in mean accuracy for both groups compared to *before-alcohol*, $b = -7.875$, $t(52) = -10.98$, $p < .0001$, $r = .84$ (see Figure 4.4A). No main effect of group was present, $p = .967$, nor did the factors of group and condition interact, $p = .637$. A Bayesian factorial ANOVA found weak evidence for a difference in accuracy between alcohol conditions, K = 1.207, moderate evidence for no difference between groups K = .244, and also for an interaction between group and condition, K = .289.

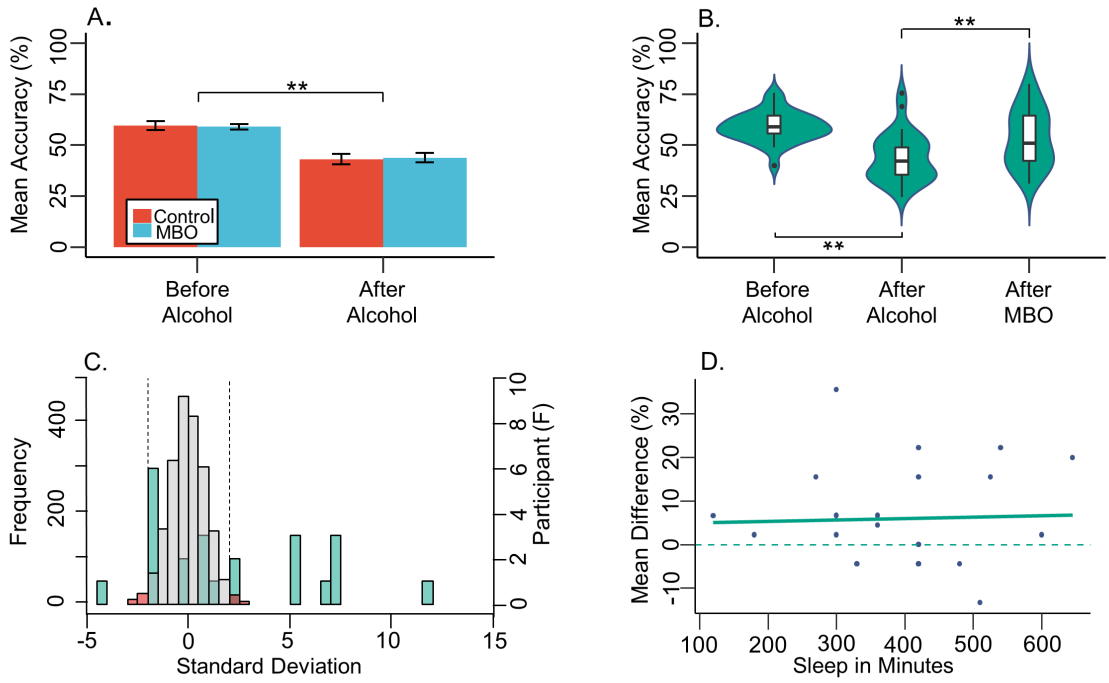

Figure 4.4: **Free recall.** (**A**) bar graph shows between control and MBO group mean accuracy (%) of freely recalled words, both before and after ingesting alcohol. Error bars depict standard mean error with ** denoting significance at p < .01, and * < .05. (**B**) violin plots displaying the distribution of MBO participant responses, with embedded box and whisker plots across all three test conditions (*before-alcohol, after-alcohol, after-MBO*). Outliers appear as dots above or below the box and whisker plots. Note the change in spread of the distribution for the *after-MBO* condition in comparison to before and *after-alcohol* conditions. (**C**) histograms depicting the resampling analysis for the free recall task in the MBO group. The left y axis shows the frequency of resampled mean differences, converted into z-scores, between *before-alcohol* minus *after-MBO* conditions. Bar width is 0.5 standard deviations. Grey bars depict roughly 95% of the resampled distribution, and the red bars show the 2.5% tails at either side, demarcated by vertical dashed lines. Overlaid green bars are a separate histogram (right y axis) showing the frequency of participants' mean differences (z-scores), with the same bar width of 0.5 standard deviations. Therefore, the figure displays how many participants are significantly different from our resampled distribution of mean differences, as these participants would be outside of the grey area on the resampled histogram. (**D**) scatterplot displays the difference between the mean accuracy (%) for freely recalled words *before-alcohol* minus *after-MBO*, correlated with reported minutes slept, within the MBO group.

Serial recall

Groups did not differ in the mean accuracy of recall within the serial recall task, however there was a significant main effect of alcohol, $X^2(1) = 42.08$, $p < .0001$ (see

Figure 4.5A). Like the free recall task, alcohol reduced recall similarly for both groups compared to *before-alcohol*, b = -5.53, $t(52)$ = -7.9, p < .0001, r = .74. When analysing the mean number of words recalled in sequence, we again found a main effect of alcohol, $X^2(1)$ = 7.2, p = .007 (see Figure 4.5B). After drinking alcohol, the sequence length was significantly reduced for both control and MBO groups, b = -0.27, $t(52)$ = -2.74, p = .009, r = .36. Bayes ACC - >100 suggesting extreme support for a difference between conditions (K=47607915), moderate evidence that there was no difference between groups, K= .281. For sequence length, there was moderate support for a difference between alcohol conditions, K = 5.397.

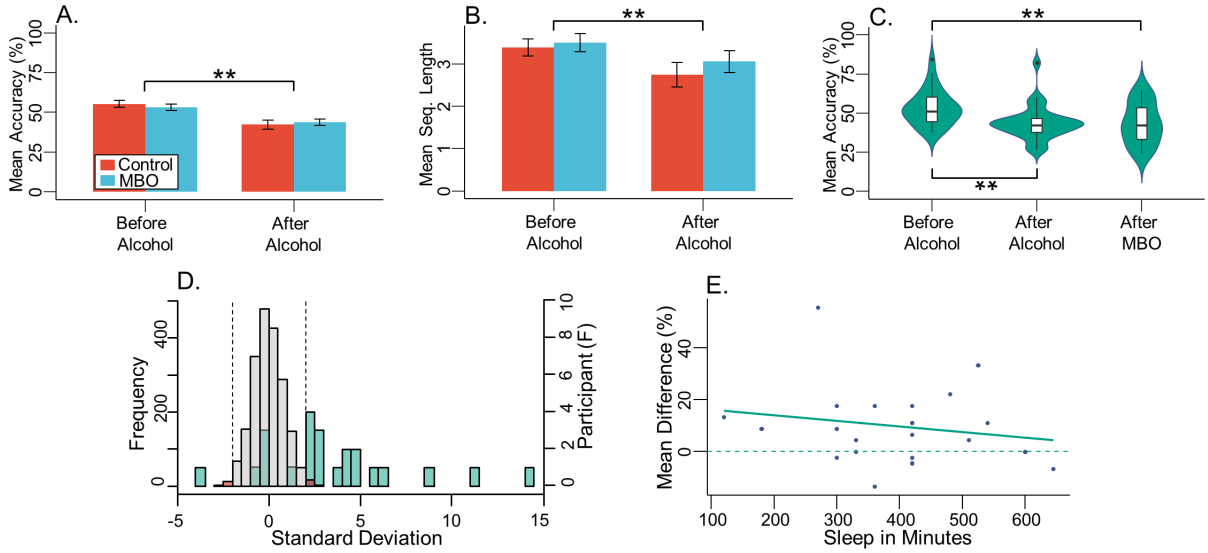

Figure 4.5: **Serial Recall.** (**A**) bar graph shows between control and MBO group mean accuracy (%) of serial recalled words, both before and after ingesting alcohol. Error bars depict standard error of the mean, with ** denoting significance at p < .01, and * < .05. (**B**) shows the same as (**A**), except that mean accuracy (%) for sequence length, i.e., the average number of words recalled in sequence, is plotted. (**C**) violin plots show the distribution of MBO participant responses in the serial recall task for overall recall accuracy, with embedded box and whisker plots across all three test conditions (*before-alcohol, after-alcohol, after-MBO*). Outliers appear as dots above or below the box and whisker plots. (**D**) histograms depicting the resampling analysis for mean accuracy % in the serial recall task for the MBO group. The left y axis shows the frequency of resampled mean differences, converted into z-scores, between *before-alcohol* minus *after-MBO* conditions. Bar width is 0.5 standard deviations. Grey bars depict roughly 95% of the resampled distribution, and the red bars show the 2.5% tails at either side, demarcated by vertical dashed lines. Overlaid green bars are a separate histogram (right y axis) showing the frequency of participants' mean differences (z-scores), with the same bar width of 0.5 standard deviations. (**E**) scatterplot displays

the difference between the mean accuracy (%) for serial recalled words *before-alcohol* minus *after-MBO*, correlated with reported minutes slept, within the MBO group.

Depth of encoding

For the depth of encoding task, our LMM analysis on the total number of words recalled (%) between groups highlighted a significant interaction effect between the alcohol (before vs after) and delay (immediate vs. 3-minute delay) conditions, and also alcohol and depth (shallow vs. deep), $X^2(1) = 6.32$, $p = .012$. To summarise the model, overall accuracy reduced after drinking alcohol, $b = -8.68$, $t(51) = -12.482$, $p < .0001$, K = 1.06E+41, $r = .868$. The 3 minute delay (with distraction task) also reduced performance compared to immediate recall, $b = -5.911$, $t(103) = -16.444$, $p < .0001$, K = 1.39E+16, $r = .851$. We also found that fewer words were recalled in shallow than in deep conditions, $b = .779$, $t(209) = 2.167$, $p = .031$, K = 0.211, $r = .148$. Further, we found an interaction between group and alcohol, such that alcohol had less of an effect on the MBO group than the control group, $b = 1.424$, $t(51) = 2.048$, $p = 0.046$, K = 4.177, $r = .276$; although the MBO group recalled fewer words to begin with, the control group showed a larger reduction in percentage words recalled *after-alcohol* (see Figures 4.4A and 4.4B). Drinking alcohol also interacted with the delay in test, $b = -1.077$, $t(103) = -3.009$, $p = .003$, K = 2.732, $r = .284$; there was a larger drop in recalled words following both the delay and after drinking alcohol compared to the reduction in accuracy *after-alcohol* but immediate recall (see Figure 4.6B). Finally, irrespective of group, *before-alcohol* more words were recalled in the deep than in the shallow condition, yet *after-alcohol* no differences were found between shallow and deep encoding conditions, $b = -0.893$, $t(209) = -2.497$, $p = 0.013$, K= 0.577, $r = 0.17$. To briefly summarise, alcohol reduced recall most for deep encoded conditions, and the drop in recall was largest for the control group.

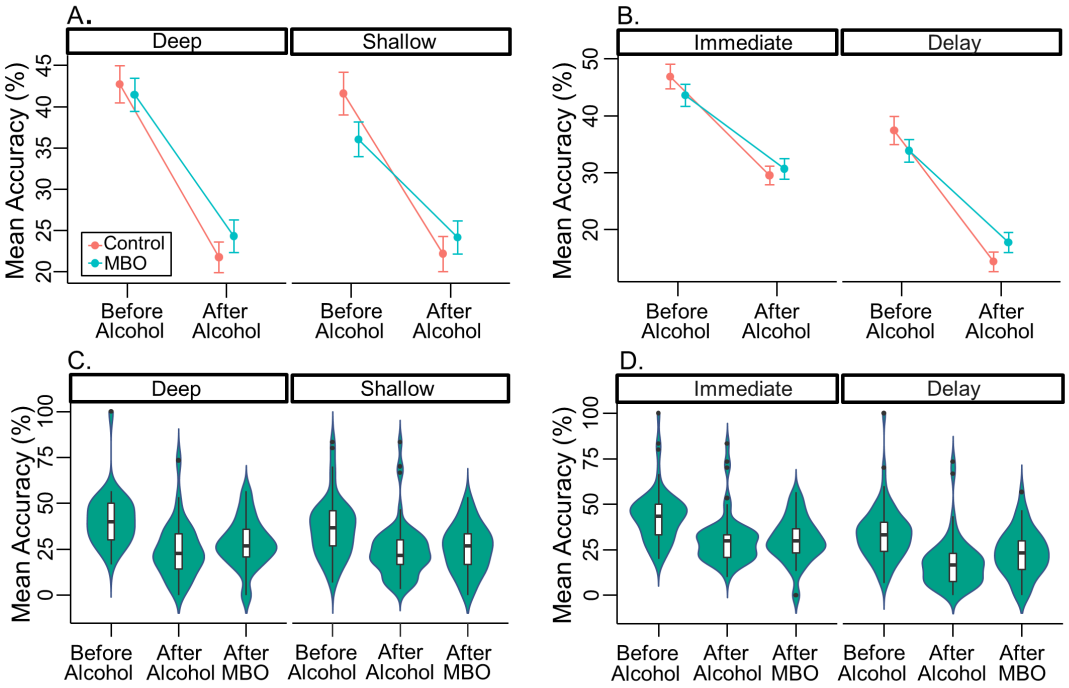

Figure 4.6: **Depth of encoding accuracy.** (**A, B**) line graphs showing between control and MBO group mean accuracy (%) for freely recalled words in the depth of encoding task, both before and after ingesting alcohol. (**A**) displays data for deep and shallow conditions collapsed across delay, whereas (**B**) shows the differences between immediate and delayed recall conditions, collapsed across deep and shallow. Error bars depict standard error of the mean. (**C, D**) violin plots show the distribution of MBO participant responses in the depth of encoding task for the shallow vs deep (**C**), and immediate vs delay recall (**D**) conditions, with embedded box and whisker plots across all three test conditions (*before-alcohol, after-alcohol, after-MBO*). Outliers appear as dots above or below the box and whisker plots.

In summary, alcohol impaired both groups of participants in free and serial recall tasks to a similar extent. In contrast, behavioural performance between groups differed in the depth of encoding task where control participants exhibited greater reduction in recall accuracy after alcohol than the MBO group.

Within MBO group analysis

Free recall

For the MBO group only, drinking alcohol significantly impaired recall, $X^2(2) = 33.79$, $p < .0001$. Specifically, *after-alcohol* there was a significant drop in recall

compared to *baseline* (*before-alcohol*) performance ($p < .0001$), yet after experiencing an MBO (*after-MBO*) we observed an improvement in recall performance compared to the *after-alcohol* condition ($p = .012$; see Figure 4.4B). There was no difference between the two sober conditions (*before-alcohol* and *after-MBO*) ($p = .068$). Furthermore, we found no difference in false alarms within the MBO group, or between alcohol conditions.

Serial recall

Within the MBO group there was a main effect of alcohol on total words recalled, $X^2(2) = 25.92$, $p < .0001$ (see Figure 4.5C). Bonferroni corrected pairwise comparisons revealed that alcohol significantly reduced recall compared to *before-alcohol*, $p < .0001$. *After-MBO* recall remained reduced compared to *baseline*, $p = .001$. Unlike the free recall task, recall between *after-alcohol* and the *after-MBO* conditions did not differ, $p = 1$, suggesting no recovery in recall after a blackout. There was no effect of alcohol on sequence length, suggesting that the average length of sequences held in memory was not affected by alcohol, only total recall, and therefore number of sequences. In addition, there was no significant difference in false alarm rates within the MBO group.

Depth of encoding

The LMM model which best fitted the MBO group data showed an interaction between the factors of alcohol (*Before-alcohol, after-alcohol, after-MBO*) and depth (shallow vs. deep), $X^2(2) = 7.321$, $p = .026$. We found that recall was reduced compared to *before-alcohol* both *after-alcohol* ($p < .0001$) and *after-MBO* ($p < .0001$), with no difference between recall *after-alcohol* vs. *after-MBO* ($p = 1$). Overall, fewer words were recalled in the shallow condition than in the deep condition, $b = -1.438$, $t(159) = -3.44$, $p = 0.001$, $r = .263$. Also, recall was reduced for delayed conditions compared to immediate recall, $b = -5.032$, $t(78) = -12.038$, $p < .0001$, $r = .806$. An interaction between alcohol and delay, $b = 1.288$, $t(78) = 2.096$, $p = .039$, $r = .231$, demonstrated that *after-alcohol* and *after-MBO* recall was similar for immediate recall conditions, but delayed recall conditions showed an improvement in performance *after-MBO* relative to *after-alcohol* (see Figures 4.6C and 4.6D). A final interaction between alcohol and depth revealed that *after-alcohol*, there was a drop in both shallow and deep encoding conditions, however this was greater for deeply encoded words, $b = -1.263$, $t(159) = -$

2.181, p = .031, r = .17. There was a small increase in recall of deeply encoded words *after-MBO* compared to *after-alcohol*, however no recovery in the recall of shallow encoded words.

In sum, we found evidence for reduced performance *after-MBO* compared to *before-alcohol* in our MBO group in two of the three tasks (serial recall and depth of encoding tasks).

Individual analysis of MBO effects

First of all, we investigated whether any blackout effects in any task within the MBO group could be attributable to a lack of sleep. No relationship between sleep quantity and performance after blackout was found for free recall (p = .876; adjusted R^2 = -0.046; K = 0.382; see Figure 4.4D), serial recall (ACC: p =.394; adjusted R^2 = -.011; K = 0.498; See Figure 4.5E; Sequence Length: p = .322; adjusted R^2 = .001; K = 0.548), or depth of encoding conditions. In more detail, immediate recall accuracy was not correlated with sleep, for either deep (p = .933, adjusted R^2 = -0.933, K = 0.38) or shallow (p = .777, adjusted R^2 = -0.044, K = 0.39) encoding measures. Likewise, delayed recall accuracy was unaffected (deep: p = .865, adjusted R^2 = -0.046, K = 0.383; shallow: p = .495, adjusted R^2 = -0.024, K = 0.451) (see Figures 4.7E and 4.7F). Taken together, these results suggest weak evidence favouring the null hypothesis (Jeffreys, 1961) and thus that individual blackout effects in any of the tasks may not be due to a lack of sleep.

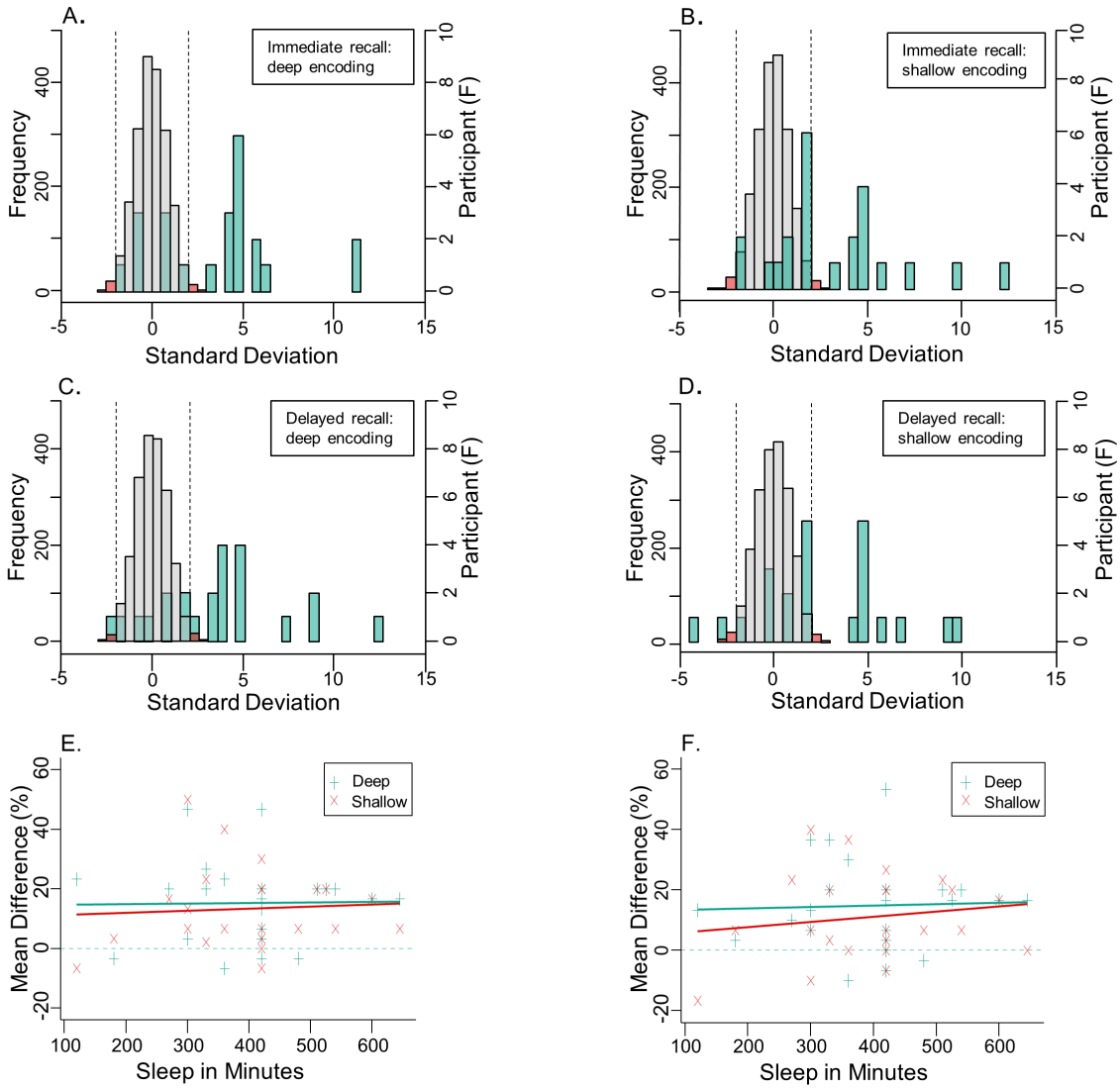

***Figure 4.7*: Depth of encoding resampling and sleep data**. Histograms (**A**, **B**, **C**, & **D**) depict the resampling analysis for the depth of encoding task in the MBO group. The left y axis' show the frequency of resampled mean differences, converted into z-scores, between *before-alcohol* minus *after-MBO* conditions for the immediate recall, deep encoding (**A**), immediate recall, shallow encoding (**B**), delayed recall, deep encoding (**C**), and delayed recall, shallow encoding (**D**) conditions. In all panels bar width is 0.5 standard deviations. Grey bars depict roughly 95% of the resampled distribution, and the red bars show the 2.5% tails at either side, demarcated by vertical dashed lines. Overlaid green bars are a separate histogram (right y axis) showing the frequency of participants' mean differences (z-scores), with the same bar width of 0.5 standard deviations. (**E**) scatterplot displays the difference between the mean accuracy (%) for immediately recalled words in the depth of encoding task, *before-alcohol* minus *after-MBO*, correlated with reported minutes slept, within the MBO group. (**F**) shows the same as (**E**), except for the delayed recall conditions.

In addition, we ran resampling analyses for each individual's performance between *before-alcohol* and *after-MBO* conditions in all the tasks to quantify the significance of blackout effects. For the free recall task, accuracy in 10 out of 23 participants (43.5%) was significantly impaired after experiencing an MBO (see Figure 4.4C). Twelve participants (52.2%) showed no difference between *before-alcohol* and *after-MBO* conditions, whereas 1 participant (4.3%) significantly improved after blackout. During the serial recall task, 17 out of 23 participants (73.9%) had significantly poorer recall accuracy *after-MBO* (see Figure 4.5D). Five participants (21.7%) showed no difference between *before-alcohol* and *after-MBO* conditions, whereas 1 participant (4.3%) significantly improved *after-MBO*. Finally, in the depth of encoding task, (see Figures 4.7A, 4.7B, 4.7C and 4.7D), for deeply encoded items at immediate recall, 15 (65.2%) showed significant differences between *before-alcohol* and *after-MBO* conditions, while 8 (34.8%) showed no effect. For deeply encoded items with delayed recall, 15 (65.2%) showed a significant *after-MBO* impairment, while 7 (30.4%) showed no effect and 1 (4.3%) an improvement after blackout. In the shallow encoding, immediate recall condition, 11 participants (47.8%) showed the effect and 12 (52.2%) did not. In the shallow encoding, delayed recall condition, 10 participants (43.5%) showed the effect, 11 (47.8%) did not, and 2 participants (8.7%) improved *after-MBO*. These results suggest that the deeply encoded conditions were most affected by binge-drinking until blackout.

Discussion

We aimed to examine whether young adults who experience a high volume of MBOs are poorer in terms of episodic memory performance compared to non-blackout controls, either when sober or after ingesting alcohol. Specifically, we hypothesised in line with other literature (Wetherill et al., 2012; Wetherill & Fromme, 2011) that our MBO participants would be most affected by the presence of alcohol when items would be presented in a context (sentence context, depth of encoding task). Against our hypothesis, we found that control participants showed increased recall when sober, and subsequently a larger fall in performance, compared to MBO participants after ingesting alcohol on the depth of encoding task. No significant differences between

control and MBO participants were found when sober, or after ingesting alcohol, on free and serial recall tasks.

We further aimed to determine whether an alcohol-induced MBO leads to impaired recall the next day which remains beyond the point of recovered sobriety. Examining individuals after an MBO we found delayed recovery of memory (that is, performance not returning to baseline levels) in serial recall and depth of encoding tasks, and variable recovery in the free recall task. Concerning the free recall task, group level statistics indicated no difference between *before-alcohol* and *after-MBO* conditions, however the data is variable and 43.5% of participants exhibited significantly poorer recall *after-MBO*. No evidence was found to suggest these blackout effects were impacted by a lack of sleep, in fact evidence from Bayes Factor Analysis favoured the null hypothesis that a lack of sleep had no effect on recall performance *after-MBO*. Taken together, these findings suggest that, even when sober, alcohol-induced blackout episodes impart some lasting damage on memory processes.

In our free recall experiment, both groups showed similar recall accuracy when sober and after drinking alcohol, where the amount of words recalled decreased at the same rate. It is known that alcohol spares short term memory in alcoholics, with spans of up to five minutes being reported as unaffected (Goodwin et al., 1970; Mello, 1973), therefore it is unsurprising that there would be few perceivable behavioural differences in immediate recall between groups, particularly at lab-appropriate levels of BrAC. Additionally, within the MBO group, recall *after-MBO* was variable across the group, with 10 participants showing a deficit in relation to sober conditions, while 12 showed no deficit. This pattern of variability may suggest a weak effect size within the overall population of individuals who blackout frequently for free recall, and mirrors findings across studies of hangovers in social drinkers. Some studies have shown no deficit in memory performance [see, for example, Chait & Perry, 1994; Verster et al., 2003), but others have found impaired performance during hangovers in free recall tasks (Devenney et al., 2019; McKinney & Coyle, 2004). Possibly the differences between findings reflects the design of experiments, either measuring in the laboratory or relying on self-reported drinking behaviour. It is probable that participants drink more in naturalistic studies, like the present investigation, than in lab-based experiments, leading to the increased performance deficits observed in

naturalistic studies. Note that a naturalistic design will also lead to variable reporting of MBO effects in the literature, due to the variability in each participant sampled. Time of testing after experiencing an MBO may also serve to weaken any *after-MBO* effects, that is, differences between baselines and after experiencing a blackout. In the present data sets, we tested all participants within 20 hours of experiencing an MBO, in an attempt to capture alcohol-induced MBO deficits before full recovery. However, the precise time when a blackout occurred is not possible to determine from participant self-report, nor did we examine the rate of recovery after blackout - our studies focussed on finding if any deficit was present after experiencing a blackout.

In comparison to the free recall task, the serial recall task increased cognitive load by asking participants to immediately recall words in the order of their presentation. We found again that alcohol impaired both the number of words recalled, and the length of sequences recalled, in both groups. Although we analysed total words recalled rather than considering just the number of words recalled in serial order, the additional load of trying to remember words in sequence appeared to disrupt recall regardless, in line with previous studies highlighting the effects of cognitive load on attention and memory (Barrouillet et al., 2007). In contrast to the free recall task, the MBO group displayed significantly reduced performance on the task after experiencing an MBO, similar to after ingesting alcohol. 73.9% of individuals exhibited consistently poor recall after experiencing an MBO, highlighting the severity of an alcohol-induced MBO on memory performance under demanding task constraints.

In the depth of encoding task, control participants showed a greater drop in performance after alcohol, suggesting that they were more impaired by the presence of alcohol than the MBO group in both immediate and delayed recall. The depth manipulation presented target words in a contextual sentence, or narrative, while the shallow presentation simply asked for a visual recognition judgment (upper- or lower-case letters). After alcohol, both groups performed similarly in deep and shallow conditions however, before alcohol, more words were recalled from the deep context than the shallow. Alcohol is known to affect encoding (Söderlund et al., 2007) therefore some may consider a greater drop in performance for deeply encoded items, compared to shallow, following alcohol consumption to be surprising. It may be that deeply encoded items, said to have a stronger memory trace (Lockhart, 2002), would be more

impervious to the effects of alcohol on free recall. However, it is also to be expected that at baseline deeply encoded items are recalled with greater frequency than shallow items, and therefore performance in this condition can fall further than the shallow condition after alcohol, as seems to be the case in the present experiment.

Moreover, our deeply encoded items were presented within a sentence context, which we did not test memory for. It is possible that the decay of memory for this sentence narrative could underlie the drop in performance for deeply encoded items, however, memory for contextual information, such as the sentence narrative in this present experiment, is not necessarily dependent on item recall (for discussions of source memory, see Dodson & Shimamura, 2000; M. K. Johnson et al., 1993; Mitchell & Johnson, 2009; Söderlund et al., 2007). Essentially, our deep (and shallow) encoded items could be said to contain source information which we tested at encoding but did not test at recall. Contextual details for events (what, where, when, etc.) are bound together with the event itself to create an episodic memory, and these contextual (source) details are hypothesised to aid recollection (Mitchell & Johnson, 2009). Alcohol is thought to impair this process; indeed, the loss of some contextual details, such as serial ordering of events, is thought to contribute in part to the experience of a fragmentary MBO (Hartzler & Fromme, 2003). Despite the fact we could not measure source recollection, it is conceivable that recall performance for deeply encoded items would drop to a similar level seen for shallow encoding, after ingesting alcohol.

In addition, our participants showed little overall difference between *after-alcohol* and *after-MBO* conditions in the depth of encoding experiment in terms of the number of words recalled. There was a small recovery in recall *after-MBO* for deep but not shallow encoded words, and for delayed but not immediately recalled words. This statistical "recovery" in delayed recall is not surprising as alcohol consumption reduced memory further for delayed than immediately recalled words, yet note that performance in delayed recall was always worse than immediate recall. Recovery in this context does not suggest that memory is operating as normal again for certain conditions such as deep encoding after the blackout event, it is minor, and relative to the impact of alcohol consumption on memory. Given that previous studies suggest that alcohol impairs encoding of contextual details (H. V. Curran & Hildebrandt, 1999) we speculate that alcohol-induced MBOs also affect encoding of associated details that

support recollection, for example, position in sequence of a word during the serial recall task or sentence narratives for deeply encoded items.

It is important to note that the variability in the *after-MBO* effects found across the three experiments can be explained by task demand differences and the additional cognitive processes these tasks engage in relation to free recall. For example, both our serial recall, and depth of encoding task are more cognitively demanding than simple free recall, involving an ordering of remembered episodes and also a delay to recall. Notwithstanding this, our findings in the three recall tasks are broadly in agreement with the small number of reported MBO studies (Hartzler & Fromme, 2003; Wetherill & Fromme, 2011). Neither Wetherill and Fromme (2011), nor Hartzler and Fromme (2003), found differences between control and blackout participants before alcohol in immediate recall tasks and across differing paradigms. Similar to our findings, Hartzler and Fromme (2003) also found no group differences following alcohol for immediate recall. In contrast to our results, both papers did report an increase in deficit after consuming alcohol for their blackout participants, specifically in delayed recall of narrative details. Although these results after ingesting alcohol were not replicated here, we did not use narrative recall tasks, nor did we administer such a high dose of alcohol to participants as the above-mentioned studies.

Our data for the high-volume blackout group relies on our participant's self-reporting of their own memory blackout behaviour. We acknowledge that in a naturalistic examination of blackouts it is not possible to identify the strength of the blackout, which introduces a measure of variability into results. Our investigation focussed on instances of extreme binge-drinking leading to MBOs, and whether they impact memory the day afterwards, yet it is important to highlight that blackout effects presented here may be influenced by the presence of hangover symptoms in our participants. Hangover symptoms have also been shown to negatively impact memory (Verster et al., 2003). However, note that hangovers and memory blackouts are not mutually inclusive; a blackout can occur with minor or no hangover symptoms, and similarly a hangover can occur without having also experienced a blackout. We have not found any work in the literature that has investigated both hangovers and MBOs concurrently. Critically, while a hangover can present with a multitude of physical symptoms, the experience of those symptoms is subjective. Van de Loo et al. (2020)

show that the most important determinant of hangover severity is a participant's own perceived levels of alcohol intoxication. It is important in the future to dissociate the study of hangovers and MBOs to determine the relative impact of both experiences on cognition. It is likely that both experiences impact memory performance when sober, but it is currently unknown whether this is caused by the multitude of physical symptoms experienced during hangover (for example, nausea, malaise, fatigue, etc.) or an enduring impact of the blackout (caused by alcohol) on hippocampal functioning.

Furthermore, Verster et al. (2003) suggested that a lack of sleep and detectable alcohol BAC% levels at time of testing could explain the mixed results in the literature (Chait & Perry, 1994; Devenney, Coyle, & Verster, 2019; McKinney & Coyle, 2004), since a lack of sleep may inflate the strength of after binge-drinking effects on cognition. We highlight these issues here, and note that we attempted to control where possible for average alcohol intake for our high volume MBO participants, and their estimated time slept after an MBO. All participants reported sleeping, all were tested when sober, and testing took place later in the day allowing time for detoxification. There were no correlations found between sleep and recall accuracy, in contrast we found weak evidence in support of the null hypothesis in all tests conducted. More importantly, we still observed performance deficits in the *after-MBO* condition. Alcohol is suggested to impact sleep quality (Devenney, Coyle, Roth, et al., 2019; Ebrahim et al., 2013), however, it is worth noting that measures of sleep quality are subjective, include qualitative components [see Devenney, Coyle, Roth, et al., 2019; A. D. Krystal & Edinger, 2008), and by their very nature are likely to strongly correlate with sleep quantity. Future work may focus on quantitative measures of sleep quality affected by alcohol.

We originally hypothesised that people who experience a high volume of MBOs may perform differently in recall tasks compared to people who have never experienced an alcohol-related memory blackout. Our data suggests that in general they do not perform differently, however, a lack of differences between controls and high frequency MBO participants here does not necessarily imply that the two groups of participants are equal. There is a paucity of neuroimaging work examining the impacts of memory blackouts, however, Squeglia et al. (2014) examined structural changes in the brains of low-moderate frequency binge drinkers, and highlighted

reduced grey matter volume in young adults compared to controls. Similarly, reduced event-related potential (ERP) amplitudes and delay of onset of early onsetting ERP components (for example, P1, N2, P300, P3b) have been observed in basic cognitive tasks in heavy binge drinkers (for example, Maurage et al., 2009; Nichols & Martin, 1996). In a meta-analysis of the binge-drinking literature, Lees and colleagues (2019) suggest that abnormal or delayed developmental of pre-frontal regions of the brain may be a consequence of binge-drinking in young adulthood, predisposing people to further alcohol-related harm. While caution is required when making assumptions about whether possible biomarkers would also be apparent within our blackout group during neuroimaging, our young adult participants displayed extreme binge-drinking behaviours showing behavioural deficits in memory after a single acute episode. It is reasonable to propose further examination of these performance differences using neuroimaging methods would constitute a more sensitive test of our hypothesis.

To conclude, the three experiments presented here examined episodic memory performance in people who experience alcohol-related memory blackouts. To the best of our knowledge, this is the first paper to compare frequent blackout participants when sober, after alcohol, and after blackout, and further, contrast their performance with a control group before and after alcohol. We hypothesised that in comparison to controls, MBO participants may show greater deficits in memory performance after drinking alcohol yet found limited group differences before and after alcohol. However, we show that after experiencing a blackout, deficits remained in all three experiments to varying degrees (individual participant data), and group data highlighted significant *after-MBO* effects in the serial recall and depth of encoding tasks. It remains possible that behavioural performance masks underlying differences in cognitive strategies between controls and frequent blackout participants observed in studies of binge-drinking (Campanella et al., 2013; Park & Kim, 2018). In sum, our data highlight a deficit in episodic memory performance after experiencing an alcohol-induced memory blackout, that does not correlate with time spent sleeping, and endures beyond the presence of alcohol in the body.

Chapter Five:

Am I sure that happened, or am I just making it up? False memories after alcohol-induced memory blackouts in sober young adults

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Abstract

Episodes of extreme binge-drinking can result in partial or whole memory loss for periods of time, transient anterograde amnesic experiences known as alcohol-induced memory blackouts. The current study investigates whether recognition memory, and receptiveness to falsely remembering details or events, differs in those who frequently experience MBOs compared to non-blackout controls. Based on work by Roediger and McDermott (1995), a recognition memory paradigm with a follow-up remember/know/guess judgement, was conducted on a Control group of participants who had never experienced an MBO, and an experimental (MBO) group who had experienced at least 9 in the preceding 12-months. The task was completed *before-alcohol* and *after-alcohol*, and the MBO group additionally completed the task for a third time within 20-hours of a blackout, but once sober. Both groups displayed reduced ability to discriminate between old and new words, and an increase in conservative response bias, *after-alcohol*. There were more false alarms to related lure words than unrelated words, and the frequency of false alarms dropped *after-alcohol* which reflected the more conservative response bias. Interestingly, the MBO group recorded more 'know' than 'remember' responses in comparison to controls, suggesting perhaps a greater reliance on the process of familiarity than recollection. *After-MBO*, a significant difference in response bias compared to *before-alcohol* remained, suggesting that participants retained a conservative bias *after-MBO*. We conclude, in contrast to our hypothesis, that uncertainty in memory led to more caution in this experimental context, that is, less acceptance of false memories. Future work on the fallibility of memory after drinking alcohol should focus on whether naturalistic studies of alcohol-induced memory blackouts could identify increased false memory acceptance rates.

Introduction

After episodes of memory loss following binge-drinking, people actively try to recall what happened, leading to the possibility that the recollection of events, if this occurs, could be open to suggestion or at the very least be an inaccurate record. Amnesia caused by binge-drinking is commonly known as an alcohol-induced memory blackout (MBO), a phenomenon where chunks of time cannot be recalled. We know that some cognitively demanding memory tasks are performed poorly while under the influence of alcohol (for example, Peterson et al., 1990), however, to the best of our knowledge, there has been no work looking at false memory specifically in those who frequently experience MBOs. We do not know how frequent blackout experiences affect memory in the long-term, but perhaps more worryingly, it could be that after an individual blackout experience people may falsely recall the events of the blackout period, perhaps by predicting what would have likely happened or relying upon knowledge from previous drinking occasions. We therefore begin to address these issues by considering whether those who experience a high volume of MBOs perform similarly in a recognition memory task to those who do not, both while sober and after consuming alcohol. We then compare the rate of false alarms between our MBO and control group before and after alcohol to investigate whether frequent MBO experiences lead to an increase in false recognition rates. Further, we examine whether those who frequently blackout show any remaining effect on memory of an acute blackout episode within 20-hours after experiencing an MBO.

A *fragmentary* blackout is an anterograde amnesic event during which individuals are unable to form new, recoverable, memories for short periods of time (Ryback, 1971). They typically occur when excessive quantities of alcohol are rapidly consumed, disrupting function within the hippocampus and interrupting the transfer of episodic information from short to long term memory (A. M. White, 2003). Goodwin and colleagues (1969b) suggested that people who have experienced a *fragmentary* blackout may only realise it occurred when reminded of events which they had forgotten, and that these reminders may then cue recollection of the forgotten event(s). What is less certain is whether any memories from the blackout period would have been retrieved without cue. Further, it is unclear whether recalled details from a

fragmentary MBO are a true representation of facts or are instead a plausible – but not necessarily accurate – account of the event. In other words, are memories of this type 'constructions' rather than 'reproductions' (Bartlett, 1932)?

A paradigm devised by Roediger and McDermott (1995), an extension of earlier work (Deese, 1959) and commonly known as the DRM, was developed to enable laboratory testing of false memory susceptibility. This method presents semantically related word lists to participants, for example 'bed, rest, awake, snooze', but omits one associated word, in our example this would be 'sleep'. Participants are then asked to recognise, or recall, these old previously studied words from a list including new semantically related words, new unrelated words, and the omitted lure (for example, 'sleep'). Replication studies have consistently found that the related lure will be falsely identified as an old word (for example, McDermott, 1997; Payne et al., 1997; I. Van Damme & d'Ydewalle, 2009). Further studies have investigated false memories in placebo-controlled alcohol designs (see, for example, Maylor et al., 1987; Mintzer & Griffiths, 2001; S. Ray & Bates, 2006) and - perhaps surprisingly - found that alcohol had little effect on total false alarms. Additionally, one DRM study by Milani and Curran (2000) found no change in false alarm rates, yet alcohol increased 'remember' responses to related lures compared to placebo. This result suggests a change in the subjective experience of false memories after drinking alcohol, that is, we may be more likely to accept false information as part of our memories when under the influence of alcohol.

While performance in a laboratory is informative, arguably this is removed from the real-world experience of an individual after blackout trying to recall events, or recognise items, previously encountered while drunk. Ethical constraints on the quantity of alcohol researchers can administer in a laboratory make controlled testing of true MBO memory loss difficult. We can, however, test whether people who frequently report experiencing blackouts are more likely to falsely identify stimuli at moderate levels of intoxication, and we can also look to the eyewitness literature, which provides some clues into memory performance within more ecologically valid contexts, that is, naturalistic settings or higher blood alcohol concentration levels (BAC%). For example, a recent meta-analysis of both laboratory and naturalistic eye-witness studies (Jores et al., 2019) found that higher levels of BAC% reduced quantity

of detailed recall compared to lower levels. Further, some studies showed a reduction in accuracy for details after heavy drinking (Crossland et al., 2016; Van Oorsouw et al., 2015), an increase in false alarms (Dysart et al., 2002; Yuille & Tollestrup, 1990) and suggestibility (Van Oorsouw et al., 2015), particularly in a cued recall condition (Altman et al., 2018).

Taken together, the laboratory evidence appears to contrast with the more consistent picture painted by the naturalistic eye-witness literature. This may be due to differing levels of alcohol in these two types of studies, therefore suggesting that recognition, and false memory suggestibility, are less likely to be impaired at lower alcohol doses whereas increased BAC reveals greater deficits. Alternatively, differences could be reflective of task designs, for example, where it is arguably less demanding to remember simple stimuli presented in standard memory paradigms (for example, words, faces, item images) than the more complex details included in witnessing a real crime, or one staged for experimental purposes (Yuille & Tollestrup, 1990). What is not clear, however, is whether participants in any of these studies frequently experienced MBOs (an index for repeated extreme binge-drinking) or if they were moderate, occasional drinkers. Repeated assault on cognitive function via alcohol may lead to performance differences and therefore the extent to which results can be extended to heavy binge-drinkers, or those who frequently blackout, is debatable.

We previously showed selective differences between MBO and control (who have never blacked out) participants, between sober and after alcohol conditions (Jackson et al., 2021) in a series of recall tasks. Further, reduced performance within the MBO group was observed *after-blackout* when compared to a sober baseline. Recall is said to be a more cognitively demanding process than recognition, and indeed deficits observed *after-blackout* were most visible in our more effortful recall tasks. However, since alcohol is known to inhibit normal hippocampal functioning (A. M. White, 2003), a brain region critical for recollection and context-based memory, it is plausible that differences between groups may be measurable in recognition memory tasks which feature *remember* (recollection) and *know* (familiarity) judgements. Additionally, it is conceivable that those who experience frequent MBOs may be more

likely to falsely identify items which are similar to 'old' stimuli, that is, accept plausible suggestions as true events.

To investigate this hypothesis, a recognition memory DRM study, with a remember/know/guess judgement, was conducted with participants who had never experienced an MBO, and with those who reported at least 9-10 blackouts in the preceding 12-months. Performance while sober, and after a scaled dose of alcohol, was compared between groups. We predicted that alcohol would have a deleterious effect on recognition memory in both groups equally with little differences between groups *before* or *after-alcohol*, as seen in the wider literature (Wetherill & Fromme, 2011), and we note evidenced in our earlier work (Jackson et al., 2021). Those in the MBO group were further invited to complete the study within 20-hours of experiencing a blackout. Here we expected to observe a detriment in performance *after-MBO* in comparison to *before-alcohol*, that is, for the two sober states. Finally, we hypothesise that a greater reduction in *remember* than *know* responses should remain *after-MBO* when compared to the *before-alcohol* condition, and that this would accompany an increase in related item false alarms.

Methods

Design

Participants from the University of Stirling were recruited via online advertisements for an alcohol use questionnaire. Following completion of this questionnaire, eligible individuals were invited to take part in a series of 4 behavioural studies. These consisted of a free recall, serial recall, depth of encoding, and a recognition memory paradigm. Three of these studies (the free, serial and depth tasks) are described in Jackson et al. (2021), and the fourth study is presented here. Eligibility criteria included: (1) either never having experienced an MBO or experiencing 9 or more MBOs in the preceding 12 months, (2) being aged between 18 and 25 years, and (3) being a fluent English language speaker. In total, 53 participants were recruited, consisting of a control group (n=24, 12 males, mean age = 20.17, SD=1.99), and experimental group (MBO group) (n=29, 11 males, mean age = 19.55, SD=1.38). Our

control group reported either abstinence from alcohol or drinking alcohol only on very rare occasions.

The current experiment utilised both the free and serial recall word lists presented in our previous work as study phases for the subsequent DRM recognition memory task. Participants firstly completed the four experiments while sober, and then repeated following a scaled dose of alcohol. All participants were compensated for their time with either course credit tokens, or £15. Additionally, the MBO group were invited to return to the laboratory within 20 hours of a blackout, but once sober. On this visit, they completed the experiment for a third time and received additional course credits or money. Of the invited MBO group, 23 participants returned for the additional testing session (10 males, 13 females, mean age =19.43, *SD* = 1.2). The study, and protocol for administration of alcohol, were approved by the NHS, Invasive and Clinical Research committee at the University of Stirling.

Procedure and Alcohol Protocol

The procedure for the current study and full alcohol protocol were detailed in our previous paper, Jackson et al. (2021). In brief, individuals were invited to take part if they were aged between 18 and 25, were fluent English speakers, and had either never experienced an MBO, or recorded at least 9 in the preceding 12-months. Invitations stipulated that participants should have no current possibility of pregnancy, that they should not be taking prescribed medication which could interact with alcohol, and that they should have no history of substance abuse. They were also asked not to drink alcohol in the 24-hours prior to the study, or eat in the 3-4 hours before arriving at the laboratory. Photographic identification to verify age was required, in addition to written consent. Height, weight and gender were recorded, and participants also provided an initial breathalyser test reading to ensure sobriety. Alcohol dose was calculated to achieve a Blood Alcohol Concentration percentage (BAC) of 0.06%, which was tested at regular intervals throughout the study from breath alcohol concentration (BrAC).

Participants completed the study while sober, and then received their individually calculated dose of vodka. To enable the alcohol to be absorbed and then distributed through the bloodstream, participants rested for 15-minutes before

gargling with water to remove any trace alcohol from the mouth, after which they provided a breathalyser reading. Regular breathalyser readings then tracked the ascent, and descent, of the BrAC curve while they repeated the study. Table 5.1 (reproduced from Jackson et al., 2021) provides details of the quantity of alcohol administered, the mean time taken to consume the alcohol, and subsequent mean BrAC reading immediately prior to, and following, the current experiment.

Table 5.1:

Alcohol dose, drinking time, and mean BrAC

				Breath Alcohol (mg/l)		
	Vodka (ml)	Alcohol (g)	Drink Duration (secs.)	BrAC	Peak BrAC	Final BrAC
Whole Group	94.75 (22.1)	35.533 (8.29)	73.42 (83.76)	0.20 (0.06)	0.37 (0.07)	0.19 (0.04)
Control (n=24)	97.75 (22.28)	36.656 (8.35)	72.82 (92.35)	0.22 (0.06)	0.36 (0.08)	0.20 (0.05)
MBO (n = 29)	92.28 (22.03)	34.603 (8.26)	73.83 (79.15)	0.19 (0.05)	0.33 (0.04)	0.18 (0.04)

Means with standard deviations given in brackets

MBO protocol

MBO group participants were invited to return to the laboratory within 20 hours of experiencing a blackout. Participants were asked to contact the researcher to arrange a suitable time if they were planning, or had attended, a drinking event which resulted in a blackout. No participants were asked to binge-drink, or induce an MBO, for the purposes of this study. Their self-reported average drinking behaviour over a 6-week period is given in Table 5.2 (reproduced here from Jackson et al., 2021). The follow-up *after-MBO* study sessions were all conducted after midday to allow adequate time for the participant to rest and recover (mean sleep duration = 6.55 hours ±2.05). On arrival all participants were breathalysed with testing commencing only if/when their BrAC reading was 0.00 mg/l, signifying their return to a sober state. All MBO participants confirmed having experienced a memory blackout prior to testing.

Table 5.2:

Self-reported frequency of drinking behaviours of MBO group

	Never	10 or less	11-20 times	21-30 times	Over 30 times
Drinking sessions, per month (n = 23)	0	11	7	4	1
Drunk Instances, per year (n = 23)	0	1	4	5	16
Binge-drinking episodes, per year * (n = 23)	0	1	2	4	16
	Never	1-4 times	5-8 times	9-12 times	Over 12 times
Fragmentary MBOs, per year (n = 23)	0	0	0	12	11
En bloc MBOs, per year (n = 23)	0	11	3	5	4

	UK Units	Grams Ethanol (g)	Number of Sessions
Per week (n = 22)	26.99 (11.404)	215.918 (91.235)	1.89 (0.661)
Per session (n = 22)	13.364 (4.342)	106.912 (34.739)	
Max per session (n = 22)	21.225 (8.326)	169.8 (66.611)	

Frequency of responses to drinking behaviour questions, and quantity of alcohol consumed over a 6-week period given as mean scores with standard deviation in brackets. A drinking session refers to a single drinking event of unspecified duration.

** Defined as more than 6 units of alcohol in a single session*

DRM Task

Words were presented on a computer screen for the free and serial recall tasks described elsewhere, and were used as study blocks for the current experiment. Stimuli were words taken from Roediger and McDermott (1995), totalling 270 unique words and split into 18 blocks of 15 words each. The 18 blocks were divided equally between the free and serial tasks (9 each), and each participant was presented with 3 pseudo-randomised blocks, per task, that is, 3 from the possible 9 free recall lists and the same for serial recall. In study blocks, individual words were presented for 1000ms, followed by a blank inter-trial interval of 2000ms. Following each study block of 15 words in the free recall task, participants were asked to recall as many words as they could remember by typing, in any order, and with no time constraint. An identical procedure was followed for the serial recall task, except participants were explicitly asked to recall stimuli in the order in which they had been presented.

At test for the current study, participants were presented with 3 blocks of 60 words, each containing 30 'old' words, and 30 'new' words. Of these 'new' words, 6 were related lures and 24 were unrelated to the study words, taken from unused lists in the Roediger and McDermott (1995) paper and supplemented with stimuli from the MRC Psycholinguistic database (Coltheart, 1981; M. Wilson, 1988) (search parameters of 5-9 letters, 2-4 syllables, familiarity of 300-600). Test stimuli were presented for 2000ms, following which participants made old/new judgements. To be clear, old stimuli presented in a DRM test block were not blocked by semantic relationship, they were mixed between categories to obscure semantic links. Therefore, responses to related lures were not driven by previous stimuli seen at test; any false recognition to a related lure came from a recognition of the item's semantic relatedness to themes presented at the encoding stage. We further required a remember-know-guess (RKG) judgement following an 'old' response as a way to separate recollection from familiarity memory processes. The experiment was presented using experimental software E-Prime 1.2 (Psychology Software Tools, Pittsburgh, PA).

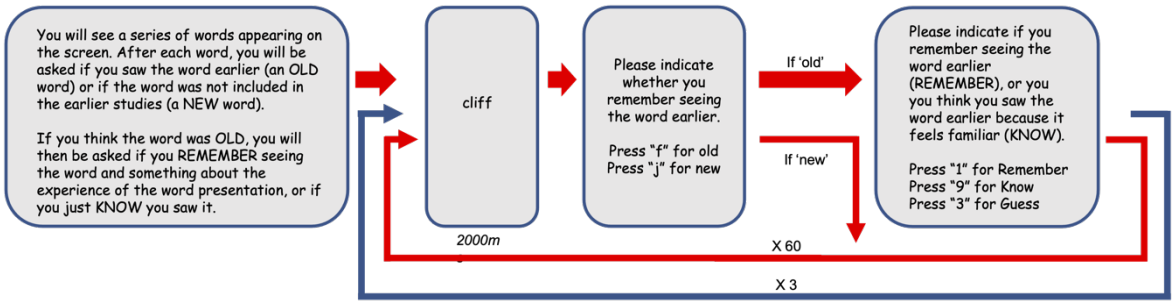

Figure 5.1: **DRM task structure.**

Statistical Analysis

We used linear mixed models (LMM) to analyse data from this experiment and to account for the difference in sample size between control and MBO participants. We assessed discrimination sensitivity (*d'*), response bias (*C*), percentage of accurately recognised words by RKG response, frequency of false alarms by related/unrelated lure type, with fixed effects of alcohol (*before* and *after-alcohol*), encoding category (free or serial; this was done for *d'* and *C* only), and group (control and MBO). We also

did this for the MBO group only, looking specifically at the effect of MBOs, compared to *before* and *after-alcohol* conditions (Figure 5.2). We used Bonferroni corrected paired t-tests to compare the within-group means for the MBO group. Analysis involved calculation of signal detection measures of sensitivity (*d'*), defined as:

$$d' = Z(H) - Z(FA)$$

where $Z(H)$ is the standardised hit rate (%), and $Z(FA)$ is the standardised false alarm rate (%) for total false alarms. We also calculated response bias (C):

$$C = -\frac{Z(H) + Z(FA)}{2}$$

a measure of the likelihood of conservative/liberal responding in conditions of uncertainty. Values lower than zero would indicate a liberal response where participants are likely to say 'yes' when uncertain; values greater than zero represent a conservative response strategy where participants are likely to say 'no' when they are uncertain. These measures were then taken as dependent variables for LMMs, with factors of group, encoding type, alcohol condition, and their interactions. Within the MBO group only, factors of alcohol condition and encoding type were used as dependent variables. We further assessed the likelihood of false alarming when new words were semantically related, compared to when new words were unrelated.

All analysis was conducted using R (R Core Team, 2019) and *nlme* (Pinheiro et al., 2019). We report all significant effects and a selection of non-significant results. Effect sizes of planned contrasts (Rosenthal, 1991; Rosnow & Rosenthal, 2005) are given by:

$$r_{contrast} = \sqrt{\frac{t^2}{t^2+df}}$$

For our MBO group only, we again ruled out the possibility that sleep impacted performance on the task during the *after-MBO* condition. We correlated time slept with the difference in *d',* and in total false alarms, between *before-alcohol* and *after-MBO* conditions. To determine support for the null hypothesis, we include equivalent Bayes Factors (K) for all correlations with sleep (Morey & Rouder, 2018). After ruling out the contribution of sleep to MBO effects, we aimed to identify individual participants who were significantly impaired after blackout. To do this, we resampled the ordering of

before-alcohol and *after-MBO* conditions for each MBO participant 2000 times to build test distributions of possible *d'* mean differences in between *before-alcohol* and *after-MBO* conditions. We then compared each individual participant's sampled mean difference in *d'* between *before-alcohol* and *after-MBO* conditions to our resampled test distributions to quantify the frequency and severity of blackout effects.

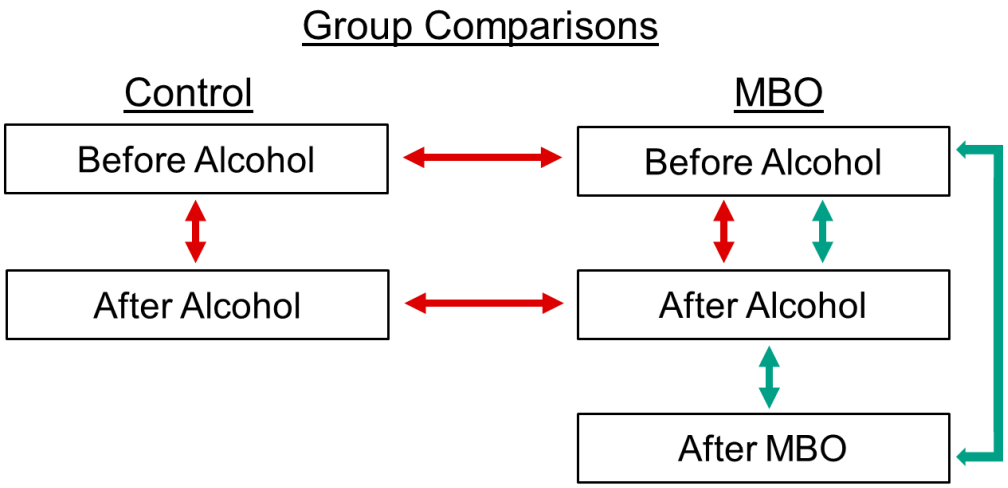

***Figure 5.2*: Analysis Structure.** Displays the design structure for the experiment. Red arrows highlight mixed design comparisons, with control and MBO participants in before and alcohol conditions. The green arrows signify the within MBO group comparisons only.

Results

Between Group Analysis: Control vs MBO participants

We measured discrimination sensitivity between old and new items using dPrime (*d'*), and found a significant main effect of alcohol, $X^2(1) = 15.78$, $p < .0001$, but no effects of group or encoding type (see Figure 5.3A). To summarise, the main effect of alcohol was that *after-alcohol*, *d'* reduced similarly for both groups compared to *before-alcohol*, $b = -0.238$, $t(52) = -4.257$, $p < .0001$, $r = .508$, reflecting poorer recognition memory *after-alcohol* in both groups. A main effect of alcohol, and an interaction between alcohol and encoding type (words recognised from either free or serial task encoding phase), was found in response bias *(C)*, $X^2(2) = 10.93$, $p < .001$ (see Figure 5.3B). The main effect showed that participants responded more conservatively *after-alcohol*, compared to *before-alcohol*, $p < .001$. This effect was driven by the

interaction between alcohol and words recognised from the two encoding phases (free and serial tasks). *Before-alcohol*, participants were more liberal in their responses to free than to serial task stimuli, but *after-alcohol* saw a greater increase in *C*, becoming more conservative towards free compared to serial stimuli, $p < .001$. To clarify, both free and serial stimuli were more liberally responded to *before-alcohol* and became more conservative *after-alcohol*, however there was a greater range of response bias values for free compared to serial stimuli between alcohol conditions.

We further assessed in an LMM model performance on the DRM task split by Remember, Know, and Guess responses, and by encoding type, for hit rate (%), to better understand the memory processes underlying performance. There was no effect of encoding type therefore this factor was removed and data collapsed back into RKG judgement only. The best fitting model found a main effect of RKG judgement, and interactions between RKG judgement and group, and RKG and alcohol, $X^2(2) = 13.52$, $p = .001$ (see Figure 5.4A). Overall, participants made more 'remember' responses than 'know', and more 'know' responses than 'guess' (all Bonferroni pairwise comparisons were significant, all $p < .0001$). Critically, we found an interaction between group and know vs. remember responses, $p = .003$, suggesting that the MBO group were significantly more likely than the control group to respond 'know'. In contrast, the control group made more 'remember' responses compared to the MBO group. *After-alcohol* there was a significant reduction in 'remember' responses for both groups, $p < .001$ compared to *before-alcohol*, and accompanied by non-significant increases in 'know' and 'guess' responses, which reflects less certainty in recognition, and an overall diminished contribution of recollection to completing the task.

We also examined the mean percentage of false alarm type, split between related and unrelated lures, and also between control and MBO groups. Our model suggested main effects of alcohol, relatedness, and an interaction between alcohol and relatedness, $X^2(1) = 9.62$, $p = .002$. Explaining these effects in more detail, alcohol reduced the total number of false alarms made, $p < .001$ (see Figure 5.4B). Across both groups and irrespective of alcohol condition, participants made fewer false alarms for unrelated compared to related lures, $p < .001$. Finally, the interaction between alcohol and relatedness suggested that for both groups of participants false alarm mean percentage for related lures dropped *after-alcohol* ($p = .003$), but unrelated false alarm

rate did not differ. We did not split the false alarm data by RKG judgements or encoding type, although such data may be theoretically of value, since the relatively low number of trials would make any interpretation of this data questionable.

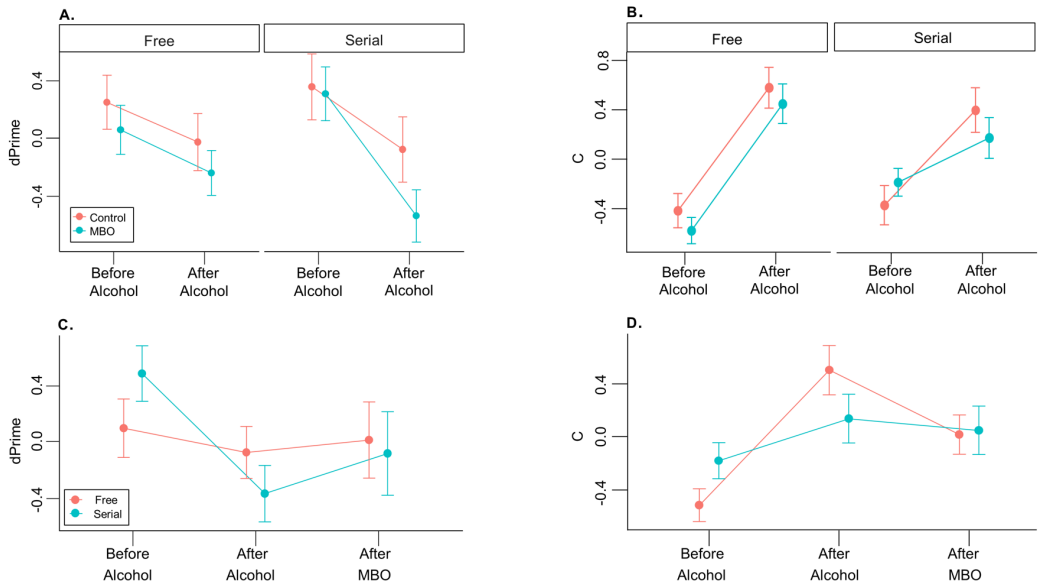

Figure 5.3: **dPrime and Response Bias (*C*). (A)** line graph shows control and MBO group discrimination sensitivity both before and after consuming alcohol, and split by free and serial recall task. Error bars show standard error of the mean. **(B)** depicts response bias (*C*) of control and MBO group participants. Again, error bars depict standard error of the mean, and distributions are split by free and serial recall phases. **(C)** line graph displays *d'* within the MBO group between *before-alcohol*, *after-alcohol* and *after-MBO* conditions. Discrimination sensitivity towards items encoded from the free recall task is depicted in red, and from the serial recall task in blue. **(D)** response bias within the MBO group across all three alcohol conditions. Again, bias towards freely recalled stimuli is displayed in red, and from serial recall in blue.

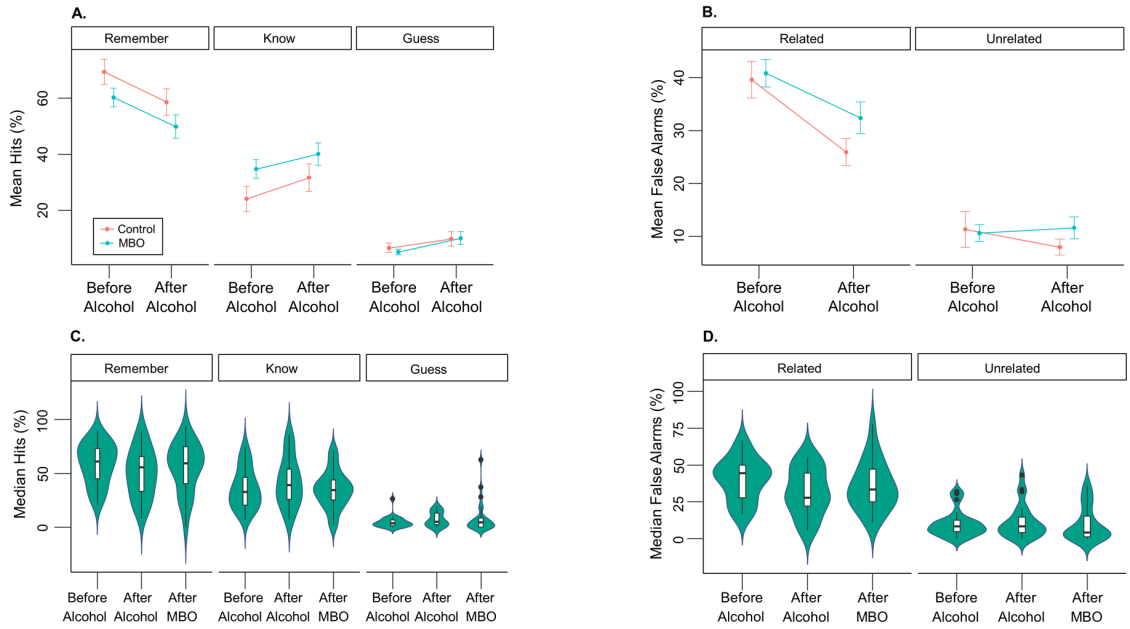

Figure 5.4: **RKG Hits and False Alarms by Relatedness. (A)** faceted line graph denotes control and MBO group mean percentage hits, split between *before-alcohol* and *after-alcohol* conditions and by RKG response. **(B)** between group false alarms are shown as mean percentages and displayed as semantically related lures, or words which were semantically unrelated to previously seen stimuli. Error bars again depict standard error means. **(C)** violin plots display the distribution of MBO group median percentage hits responses, split between alcohol conditions (*before-alcohol*, *after-alcohol*, and *after-MBO*) and by RKG response. **(D)** shows the distribution of false alarm responses within MBO group and across all three alcohol test conditions.

Within MBO group results

Assessing *after-MBO* performance within the MBO group, there was a significant main effect of alcohol on *d'*, $X^2(2) = 6.832$, $p = .033$ (see Figure 5.3C). Bonferroni corrected pairwise comparisons revealed a significant reduction in *d'* between *before-alcohol* and *after-alcohol* conditions, $p = .006$, and no difference between *after-alcohol* and *after-MBO* conditions, $p = 0.999$; however, we also found no difference between *before-alcohol* and *after-MBO* conditions, $p = .231$, which may indicate large individual variability in the *after-MBO* condition (see Figure 5.3C). Analysis of response bias data also showed a main effect of alcohol, and an interaction between alcohol and encoding type, on *C*, $X^2(2) = 11.598$, $p = .003$ (see Figure 5.3D), suggesting that participants became more conservative in their response strategy *after-alcohol* compared to *before-alcohol* ($p < .001$). This main effect was again driven

by the increased conservative bias for responses *after-alcohol*, compared to *before-alcohol*, to free recall stimuli, p = .006. Like *d'*, no difference between *after-alcohol* vs. *after-MBO* was found *(*p = .068*),* however there was a significant difference between *before-alcohol* and *after-MBO,* p = .008, suggesting participants retained a more conservative bias *after-MBO*.

We also analysed the proportion of RKG responses for hits only in the MBO group conditions. This showed a main effect of judgement type, $X^2(2) = 164.28$, $p <$ .001. MBO participants were more likely to make a 'remember' response compared to 'guess', p <.001, and also compared to 'know', p = .003 (see Figure 5.4C). This pattern of RKG judgements did not significantly change due to the presence of alcohol, nor after experiencing a blackout.

Next, we examined the percentage of false alarms for the MBO group, split between related and unrelated lures. Our model showed a significant main effect of false alarm type and an interaction between false alarm type and alcohol, $X^2(2) = 6.341$, p = 0.042. Overall, MBO participants made significantly fewer unrelated false alarms than related false alarms, b = -12.731, $t(66)$ = -16.153, p < .001, r = .893 (see Figure 5.4D). We also found an interaction effect which appeared to be driven by a drop in the related false alarms between *before-alcohol* and *after-alcohol* conditions, b = 2.496, $t(66)$ = 2.239, p = .029, r = .266. No other comparisons between alcohol conditions were significant for related or unrelated false alarm types.

We then considered whether lack of sleep may have affected performance in the DRM task for our MBO group. In terms of recall accuracy (*d'*) and total number of false alarms, neither measure correlated with minutes slept (*d'*: p = .729, adjusted R^2 = - .04149, K = .396; FA total: p = .757, adjusted R^2 = - .04275, K = .393), rather Bayes Factors show weak/moderate evidence in support of the null hypothesis that sleep does not impact task performance (See Figures 5.5B and 5.5C). Note that negative R^2 values can occur either when there are too many predictors in the model (not the case here in a simple linear regression) and not enough power, or when there is no relationship; these R^2 values should be interpreted as being equal to zero. Finally, we conducted a resampling analysis for individual performance between *before-alcohol* and *after-MBO* conditions. In this, we found that 10 out of 23 participants (43.5%) were significantly impaired in the *after-MBO* condition compared to the *before-alcohol*

condition (see Figure 5.5A). 11 participants (47.83%) showed no difference between the two conditions, while 2 participants (8.7%) actually improved *after-MBO*.

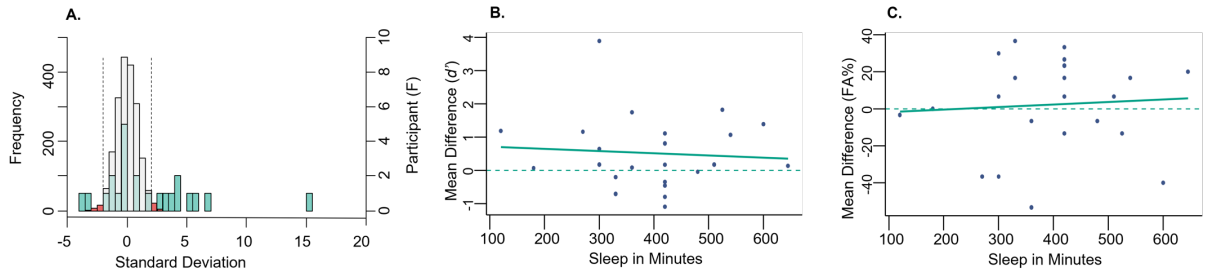

Figure 5.5: **Resampling and sleep data within MBO group. (A)** histogram depicts the resampling analysis of *d'* in the MBO group. The left y-axis shows the frequency of resampled mean differences between *before-alcohol* minus *after-MBO* conditions. Grey bars show around 95% of the resampled scores, with red highlighting the 2.5% tails and further highlighted by dashed lines. The overlaid green bars and corresponding right y-axis show the frequency of participants mean differences, converted to z-scores. **(B)** shows a scatterplot of individual participants' differences between *d' before-alcohol*, minus *after-MBO* and correlated with reported minutes slept. **(C)** as **(B)** but with differences in mean false alarms. Note that the data given in **(B)** & **(C)** do not show if an individual was significantly impacted by a blackout in these measures alone, only a significant difference between a regression line and the zero-difference horizontal line would show this.

Discussion

Our investigation centred on whether individuals who experience a high volume of alcohol-induced memory blackouts differ from control participants in recognition memory performance, and whether they exhibit more instances of false remembering. Overall, we found that both groups were significantly less able to discriminate between old and new items, shifted their response strategy to a more conservative bias, and displayed a reduction in remember responses *after-alcohol* compared to *before-alcohol*. MBO participants when sober or *after-alcohol*, were more likely to respond with a 'know' judgement than the control group, suggesting a reliance on recognition rather than recollection of the previously displayed stimuli. We further found that alcohol reduced the overall number of false alarms, specifically in related stimuli, and that groups did not differ in false alarm rate. Taken together, while recognition memory performance is broadly similar in control and experimental participants both *before* and *after-alcohol*, RKG data suggests that the groups may be reliant on different memory strategies to achieve similar performance.

Within the MBO group, we were interested in whether an episode of binge-drinking which resulted in an MBO left a deficit in recognition memory performance once participants were again sober. At the group level, this was only seen in response bias, *C*. Participants had exhibited a liberal response when sober which became more conservative *after-alcohol*. Following a blackout event, they retained a conservative response bias despite returning to a sober condition. Supplementary resampling analysis showed additional lasting detrimental effects in 10 (43%) of the 23 participants. Lack of sleep was unlikely to have contributed to any significant reduction in performance, as both R^2 values and Bayes Factors provided support for the null hypothesis. Evidence therefore suggests that remaining deficits after an MBO are highly variable within individuals but are unlikely to be related to lack of sleep following a night out. Instead, this variability likely reflects the severity of the MBO experienced, which in turn is affected by the speed and quantity of alcohol consumed. Although details regarding alcohol consumption (both quantity and timeframe) were provided by participants, we acknowledge both that participants are known to be somewhat unreliable in reporting alcohol consumption (Devenney, Coyle, & Verster, 2019), and that the time between participants stopping drinking and laboratory testing (once sober) varied, which may have influenced results. Controlling for these variables is ethically and practically challenging, however finding performance deficits in 43.5% of participants, despite the *after-MBO* condition being the third time which the group had completed the experiment, suggests that lasting effects on cognition are a probable consequence of a memory blackout for many individuals. Arguably, if we sampled from participants who only experienced an *en bloc* blackout, that is, a more severe blackout episode, we may have found a greater number of participants who displayed a blackout effect. While we may classify blackouts as *fragmentary* or *en bloc*, in a naturalistic setting it is likely that the transitions between these states is blurred, and further work is needed to examine the severity of blackout and their relationship with *after-MBO* effects when sober.

An MBO represents a period of time from which an individual will fail to recollect events, or these events may be remembered with great difficulty. It is unclear whether recalled events are genuine episodic recollections, or loose constructions from a mix of prompts, past experience, plausible scenarios, and partial recall. Indeed, events recalled from a blackout have been described as 'other-worldly', or not quite

real, at interview (Goodwin, et al., 1969a) suggesting a tenuous and malleable link to reality. False alarms in standard laboratory paradigms may give us a window into how accurately those who experience blackouts recall details, as frequency of false alarming to related lures could be indicative of suggestibility, that is, how easily influenced someone is. In the DRM task, we found no significant group difference in *d'* or false alarms, however, group averages suggested that the MBO group recorded both more unrelated and related false alarms than the control group. We found that alcohol reduced the total number of false alarms, which stands in contrast with previous work suggesting alcohol has no effect on false alarms (H. V. Curran & Hildebrandt, 1999; Maylor et al., 1987; Mintzer & Griffiths, 2001; S. Ray & Bates, 2006). Interestingly, both our study and the Milani and Curran (2000) study included a free recall test prior to recognition. It has been suggested that some false alarm responses to lures in recognition may in fact be recognitions of falsely recalled stimuli at an earlier recall state, a source memory failure (since participants fail to remember in which stage of the experiment they encountered the stimuli) which potentially increases false alarm rate (Mintzer & Griffiths, 2001). However, in studies where no recall test was given prior to recognition, for example, a standard recognition memory paradigm such as the DRM task, it would be impossible to attribute any false alarm responses to prior recall since recall is not tested. The fact that false alarms were reduced in the present study, suggests that alcohol has non-linear effects on cognition, that is, not all cognitive processes are affected equally, and negatively. In our case, with low levels of intoxication, participants became more cautious (greater control of the self?), false alarms decrease, but with a corresponding detriment in sensitivity/memory-performance. After alcohol ingestion both control and MBO participants showed a strong conservative response bias, indicating a shift in strategy towards response inhibition. Since alcohol is known to lead to decreased inhibition of oneself, in certain behaviours, our data suggest cognitive functions do not decrease in-line with behavioural functioning.

Individuals who experience a high frequency of MBOs repeatedly disrupt normal functioning within the hippocampus. Recollection and familiarity are believed to rely on separate brain regions, with the hippocampus supporting the transfer, storage and retrieval of details which are necessary for recollection, and the perirhinal cortex implicated in familiarity responses (Aggleton & Brown, 1999; Bisby et al., 2010;

Brown & Aggleton, 2001). Additional support for this dissociation has been seen in clinical patients where hippocampal damage resulted in fewer 'remember' responses (for example, Turriziani et al., 2008). Further, a review of recollection and familiarity studies by Yonelinas (2002) suggests that amnesiacs are more likely to show ERP correlates of familiarity than recollection, and are more likely to respond 'know' in an RKG task than 'remember'. Our blackout group were more likely to respond 'know', and less likely to respond 'remember', than our control group. They also falsely identified more stimuli as 'old' than the control group. If we accept the idea of dual memory processes, our findings suggest that the MBO group relied more on familiarity than recollection compared to the control group. Willingness to accept a false memory, but with a less convinced 'know' rather than a clear 'remember' response, may also mimic acceptance of memories 'retrieved' following cueing after a *fragmentary* blackout. Long-term frequent blackout experiences, affecting hippocampal functioning, may necessitate a reliance on familiarity in order to recall events, and therefore a plausible cue becomes accepted as a true past experience. Since no significant group differences in false alarm frequency were observed, our data suggests that our MBO group relied more upon familiarity/recognition memory to complete the recognition task compared to controls. To be clear, while the RKG task has been criticised as a poor indicator of the underlying memory processes driving participants responses (Wais et al., 2008), group differences in the task (given the same instructions and assuming both control and MBO groups understood these fully) must be indicative of a shift in cognitive strategy used to complete the task, regardless of no differences in behavioural memory performance. Future investigation of this hypothesis should be conducted with more sensitive measures of memory performance, for example, using electrophysiological indices of memory. It is further possible that any similarities in ERP's between amnesiacs and MBO individuals would become apparent, and differences in neural responses with our control group clearer.

Consistent with wider literature (Maylor et al., 1987; Mintzer & Griffiths, 2001), we found that alcohol decreased *d'* and increased conservative response bias, *C*. However, we were also interested in whether differences in encoding impacted either sensitivity or response bias. Maylor and colleagues (1987) suggested that processing semantically related items (deep encoding) versus making surface level judgements (shallow encoding) increased *d',* however we found no encoding effects on sensitivity.

This could be because our encoding manipulation always used semantically related groups of words whereas Maylor compared between one related and one unrelated set of stimuli. Therefore, their participants may have been more able to discriminate between related and unrelated items which led to the increase seen in *d'*, whereas our free and serial encoding contributions may have both been encoded at a deeper level than a shallow encoding manipulation, reducing the difference in *d'* from encoding type.

Unlike Maylor, we found that *after-alcohol*, there was an interaction between response bias and encoding type driven by an increased conservative bias towards free recall task stimuli. One possibility is that this result may be due to the order of the free and serial task blocks, which always presented the stimuli for free recall first. Therefore, more conservative performance for stimuli that were presented during the encoding phase for free recall could be a consequence of the time taken between free and serial stimuli encoding phases, and subsequent recognition. However, this account seems unlikely since any order effect should manifest as a main effect of encoding condition, that is, regardless of alcohol condition an order effect would be observable. A more likely explanation is that despite stimuli from both free and serial recall tasks being semantically related, our serial stimuli were more deeply encoded than free words due to the earlier task requirement of recalling the words in order of presentation. This task requirement necessitated either rehearsal or another cognitive strategy and therefore may have facilitated deeper processing, offering some protection against the effects of alcohol. Our previous work (Jackson et al., 2021), showed that recall for deeply encoded words was most impaired by alcohol, which appears to contradict findings described here. However, this contrast is likely due to task differences. Serial word list rehearsal only requires participants to focus on the stimuli to be remembered, whereas our previously reported depth task asked participants to place words in sentences, requiring processing of contextual information. The binding of contextual information into a single episode can be disrupted by alcohol which may lead to reduced recollection for details of events which occurred *after-alcohol*. In comparison, a simple strategy like rehearsal would encode words without unnecessary peripheral details and therefore may be more resistant to the effects of alcohol.

The MBO group results showed there was a significant recovery *after-MBO* compared to *after-alcohol* for *d'*, which moved closer to pre-alcohol consumption baseline levels. However, for response bias (*C*), no difference between *after-alcohol* and *after-MBO* was observed, such that participants' performance remained more conservative after drinking alcohol compared to sober states, and also after the experience of an MBO. The recovery of performance measured by *d'* suggests that MBOs in the current paradigm did not impart lasting deficits that affected our test period; however, our individual data suggests otherwise (43.5% of the sample showed a significant *after-MBO* effect). Note that the MBO group performance is still more conservative, that is, that when uncertain they were unlikely to say they remembered an item. Unlike the shift in cognitive strategy observed between *before-alcohol* and *after-alcohol* conditions in the MBO group, no significant alcohol effect or interaction was present when analysing RKG and alcohol, suggesting that MBO participants reverted back to a similar strategy *after-MBO* as used when sober. The reality is that after experiencing an MBO, and arriving at the laboratory for testing, the neural systems underpinning normal memory performance are active again, even if somewhat damaged, that is, they have access to long term memory stores. It remains unknown exactly how an MBO shuts down memory systems, or why people who experience a high volume of MBO's are more affected by alcohol, albeit in a brief number of tasks.

Recollection is said to be a process where some qualitative information relating to the item has been retrieved. In contrast, a sense of familiarity is argued to be support recognition when there is an absence of contextual details (see Yonelinas, 2002). The present study showed a change in RKG data *after-alcohol* away from remember responses, suggesting a reduction in recollection and therefore a proportional increase in know (familiarity) and guess responses. If a reliance on familiarity *after-alcohol* (Bisby et al., 2010) masks any potential behavioural differences either between conditions or groups, or if contextual information is unable to be encoded *after-alcohol*, this could be investigated with the addition of a source judgement. Further, it may be that behavioural similarities mask underlying neurological differences in memory strategy between control and MBO participants, hence neuroimaging could be employed to further clarify the neural underpinnings of performance.

To conclude, evidence reported here suggests that those who experience frequent memory blackouts, and those who do not, perform at a similar level in recognition memory tasks both *before* and *after-alcohol*, yet show differential response profiles for RKG tasks. All participants became less able to discriminate between old and new stimuli following alcohol and grew more conservative in their responses. Crucially, our data suggest a shift in cognitive strategy between controls and MBO participants, which may indicate increased reliance on familiarity processes in the experimental group compared to controls. With the exception of response bias, *after-MBO* participants performed no differently from baseline at the group level, although high variability *after-MBO* appeared to be the reason for a lack of difference, and individual instances of significant *after-MBO* memory impairment remained high. These results suggest that the MBO event does impart damage beyond the event itself, although, due to the naturalistic nature of the studied MBOs, this may be dependent upon the severity of the MBO experienced in individuals. Further, people who experience a high volume of MBOs when intoxicated show a definitive shift in cognitive strategy underlying their memory performance, which indicates greater uncertainty in their own memories. In contrast to our hypothesis, greater uncertainty in memory did not lead to an increase in false memories, rather uncertainty, in this experimental paradigm, led to more caution employed in completing the task. It remains to be known if, in more ecologically valid test settings, after experiencing a blackout, individuals are more susceptible to accepting false events as part of their memories.

Chapter Six:

ERP and Microstates of memory: Frequent alcohol-induced blackouts do not change behavioural performance but alter sober neural functioning

Judith Jackson, David I Donaldson, & Benjamin Dering

Abstract

It is widely accepted that excessive alcohol consumption has detrimental long-term effects on health and cognitive functioning. One notable but rarely studied phenomena is the experience of an alcohol-induced memory blackout (MBO), a short-term amnesic episode resulting from extreme binge-drinking. It is currently unknown whether the frequency of MBOs experienced in the past imparts any lasting changes in either cognition or at the neural level. Given that MBOs cause immediate impairments in memory, we reasoned that memory may also be affected in the long-term. To test this hypothesis, we examined neural activity associated with memory retrieval in (sober) participants who reported frequent alcohol-induced MBOs (9 or more in the past 12 months; *n*=20), and Control participants (*n*=21) who had never experienced an MBO. Memory was assessed using word recognition (discriminating old from new stimuli) with a secondary source judgement (remembering colour information), a task that both MBO and Control participants could easily perform. Neural activity was measured by recording electroencephalography (EEG) during the memory test, allowing the processes associated with memory retrieval to be characterised. The EEG data was examined using Event-Related Potentials (ERPs) and microstate segmentation, revealing clear differences in retrieval processing between MBO participants and Controls. Critically, the neural data revealed that Control and Blackout participants employed different retrieval strategies to achieve similar levels of memory performance. The present findings provide evidence that repeated alcohol-induced MBOs are associated with altered neural network functioning, highlighting the need for longitudinal studies examining the compound effects of MBOs and their impact on health, behaviour and quality of life.

Keywords: Alcohol-induced memory blackouts, episodic memory, binge-drinking, recognition memory, event related potentials, microstate segmentation

Introduction

Alcohol has many negative impacts on health and also cognition. Suffice to say, consistently drinking large quantities of alcohol is not good for you in the long term. One aspect of drinking culture which receives a lot of attention is binge drinking, yet conversely, there is a paucity of research on extreme binge drinking events, that is, events which lead to an alcohol-induced Memory Blackout (MBO[1]). Studies have shown high proportions of young adults reporting frequent episodes of binge-drinking (Elgàn et al., 2019; Tavolacci et al., 2016), with many also reporting alcohol-induced memory blackouts (A. M. White et al., 2002). While a clear link between alcohol and abnormal cortical development has been shown in animal studies (Crews et al., 2006, 2007), research in humans, which at best represents only a snapshot in time of brain structure, shows that binge-drinking can be associated with differences in cortical structures, including grey and white matter volumes when compared to non-binge drinkers (Lees et al., 2019; Squeglia et al., 2014; S. Wilson et al., 2015). Perversely, these snapshots from neuroimaging data suggest detrimental changes in the brain structure of binge-drinking populations which may be difficult to detect in cognitive assays. In short, as similar behavioural performance between groups can result from multiple strategies, any cognitive decline could be masked by the use of compensatory processes. In a recent investigation of the impacts of alcohol-induced MBOs on cognition, Jackson et al. (2021) highlighted the variability in memory performance between tasks in participants who had recently experienced an MBO but were sober again. Further, across studies behavioural performance in various cognitive tasks in young adults who frequently blackout is mixed (Jackson et al., 2021; Wetherill & Fromme, 2011, 2016). It is therefore likely that frequent MBO experiences, as markers of extreme alcohol binge-drinking, result in enduring negative changes to the brain. In the case of alcohol-induced MBOs, which disrupt the formation of new episodic memories, we are specifically interested in how frequent MBO experiences may alter future memory functioning, even when sober. We therefore examined electrophysiological indices of memory functioning, namely the old/new recognition

[1] An alcohol-induced memory blackout (MBO) is a transient amnesic event which occurs following a rapid spike in blood alcohol levels. During an MBO, an individual is conscious and continues to interact with the world, however is unable to form long-term memories and therefore cannot recall events which occurred during the period of the blackout.

effect, between two groups of participants, those who frequently blackout and people who have never experienced an alcohol-induced MBO.

An alcohol-induced MBO is caused by excessive and rapid consumption of alcohol leading to a spike in blood alcohol content (BAC%). This in turn interrupts normal hippocampal functioning (A. M. White, 2003), impairing the transfer of information from short to long term storage, and resulting in either a *fragmentary* or *en bloc* blackout experience (Goodwin 1969b). While both categories of blackout can be described as transient amnesic events, an *en bloc* blackout is typified by an inability to recall any details from a period of time, whereas the more common *fragmentary* blackout often allows either spontaneous or cued retrieval of fragments of events (H. Lee et al., 2009). Younger adults have a higher prevalence of both binge-drinking behaviour and of experiencing MBOs than older adults (McGee & Kypri, 2004; Merrill & Carey, 2016; A. M. White et al., 2004). This is of particular concern as MBOs are associated with a range of potential harms including physical injury (Hingson et al., 2016), aggression, and uncharacteristic sexual activity or substance use (A. M. White et al., 2004). Further, across late adolescence and young adulthood, it is known that the brain continues to mature (see Spear, 2018, for a review) and is primed to seek novel and rewarding events, while also remaining vulnerable to damage from frequent binge-drinking episodes (Luciana et al., 2013; Squeglia et al., 2015). Neuroimaging research has suggested that cortical volume differences between those who do and do not experience blackouts are measurable and consistent (for review, see Cservenka & Brumback, 2017), and that differences in neural-chemistry are pronounced in young binge-drinking blackout sufferers (Chitty et al., 2014; Silveri et al., 2014). Together, this suggests that extreme binge drinking, and the consequent MBO experiences, across a critical period of brain development may lead to permanent impairment or reorganisation of neural networks, with concomitant impacts on cognitive processing.

During the blackout experience procedural memory and working memory appear to remain intact, yet episodic memory is particularly vulnerable to the effects of alcohol (Söderlund et al., 2007; A. M. White, 2003). The mechanism underpinning episodic memory is a process which cohesively binds related information into one episode (Davachi et al., 2003; Davachi & Wagner, 2002; Ekstrom & Yonelinas, 2020), offering the chance to 'go back in time' and mentally re-experience the occasion

(Tulving, 1985, 2002). When recollecting an item, or event, the quality and quantity of associated information – often termed source details (M. K. Johnson et al., 1993) – enriches the retrieval process. To be clear, source details can be any associated context, such as the colour or location a stimulus was studied in. Dual-process recognition memory theories suggest that individuals either *recollect* an event including its source details, or have a feeling of *familiarity* where they know they have been previously exposed to the stimuli but cannot recover the context, that is, they do not recollect the source details (Jacoby & Dallas, 1981; Rugg & Curran, 2007; Yonelinas, 2002). If contextual information is not successfully bound into one episode, this may lead to reduced source memory retrieval (Davachi et al., 2003), where some recognition exists without source details. As noted above, fragmentary blackout experiences are punctuated by episodic details that can be recalled when cued (H. Lee et al., 2009), but rich contextual details may be missing.

How does alcohol impact brain regions involved in memory functioning? If hippocampal functioning is impaired by the presence of alcohol, it is plausible that this may reduce the quantity and quality of associated information available for transfer to long-term storage, with detrimental consequences for future recollection. The hippocampus communicates with neocortex, binding information received from sensory and perceptual networks and transferring back to long-term storage via the CA1 pyramidal network of neurons (A. M. White, 2003). Evidence from animal studies shows specific alcohol disruption to hippocampal region CA1 (Rose & Grant, 2010; A. M. White, 2003) in addition to wider cortical regions (Bava & Tapert, 2010; Ferrini et al., 2018; Tapert et al., 2005). It is known that atrophy in CA1 is linked to deficits in the encoding of memories (Fouquet et al., 2012), while multiple studies have also shown that amnesics, including alcoholic Korsakoff's patients, struggle with both encoding and retrieval in recognition memory tasks (for example, see Aggleton & Shaw, 1996; Oedekoven et al., 2019; Yonelinas et al., 1998). Schwartz et al. (2002) also found impairments in a source memory task between abstinent alcoholics and non-alcoholic controls suggesting that sustained heavy drinking behaviour leaves memory deficits even without the presence of alcohol at test. In sum, the hippocampus is strongly linked to encoding and retrieval, and is likely affected disproportionally by the presence of alcohol in comparison to other cognitive functions, leading to blackout experiences.

It is not a simple task to determine whether important memory structures are affected by frequent blackout experiences; within the context of alcohol binge drinking, the decline in cognitive functioning does not correspond exactly to a linear increase in blood alcohol concentration. For example, your ability to recognise a close friend is retained for relatively high levels of alcohol consumption, even if some blurring of vision may occur. Therefore, it is arguably necessary to examine neuroimaging data in binge-drinking populations, since, although it may be considered a criticism of the field, one benefit of neuroimaging is the ability to discriminate between underlying neural differences which may be masked at the level of overt behavioural performance (for example, in a memory task where different strategies could be used). For example, in a study examining neural activity using ERPs, Petit and colleagues (2012) found no differences in behavioural response latency between controls and an experimental group of binge-drinkers, yet binge-drinkers displayed a greater P100 amplitude than controls when shown alcohol-related stimuli. The authors suggest this P100 difference reflects increased attention towards alcohol-related stimuli. Similarly, Maurage et al. (2009) showed delayed latency of ERP components (P1, N2, P3b) when processing emotionally valanced audio cues following 9-months of frequent binge-drinking in an experimental group, but no differences compared to controls in behavioural responses. Further, an fMRI study by Bagga et al. (2013) showed that sober alcoholics employed broader neural networks (specifically, greater activations in the left supramarginal gyrus, precuneus bilaterally, left angular gyrus, and left middle temporal gyrus) than controls to perform a lexical task to similar levels of accuracy. Taken together, current evidence indicates that frequent binge-drinking may cause individuals to develop compensatory cognitive strategies, which would be indiscernible from the study of behavioural performance alone.

While a number of MBO studies examining source memory report behavioural measures (Hartzler & Fromme, 2003; Wetherill & Fromme, 2011), very few have employed neuroimaging. Indeed, a review of 26 studies published between 2010 and 2015 (Wetherill & Fromme, 2016) showed only 4 which utilised neuroimaging methods. To the best of our knowledge, the current study is the first to use Event-Related Potentials (ERPs) in a source memory paradigm with participants who frequently blackout. This method of neuroimaging is particularly appropriate for studies of memory deficits due to its high temporal resolution, which can produce

time-locked neural responses to encoding or recollection events (Wilding & Ranganath, 2012). Further, there is a robust body of research which highlights a parietal old/new ERP effect in recognition memory observable 500-800ms after stimulus onset (for example, see Rugg & Curran, 2007; Wilding & Rugg, 1996), associated with the process of episodic recollection (MacLeod & Donaldson, 2017; Murray et al., 2015; Wilding & Ranganath, 2012). In healthy young adults the old/new ERP effect is characterised by a graded increase in mean amplitude that is larger for correctly identified old items with sources (*Hit-Hits*) than for correctly identified old items without source (*Hit-Miss*), relative to a baseline provided by new, previously unstudied items (*Correct Rejections*). This effect is also preceded by a mid-frontal ERP component, occurring around 300-500ms following stimulus presentation, believed to reflect the rapid, automatic process of familiarity - item recognition without the added contextual details required for recollection (Addante, et al., 2012; T. Curran, 2000; Hintzman & Caulton, 1997).

If alcohol-induced memory blackouts cause disruption to the operations of the hippocampus, it is plausible that frequent MBOs may leave lasting damage to neural functioning, even while sober. To this end, the present study compared a sober Control group who either consumed alcohol infrequently or abstained completely, with a sober MBO group who reported experiencing a minimum of 9 blackouts in the preceding 12-months. Both groups completed a recognition memory task with a source judgement consisting of words displayed in either blue or green while both behavioural accuracy and ERP responses were recorded. Consistent with previous findings (Hartzler & Fromme, 2003), it was predicted that there would be few behavioural differences in recognition accuracy or source recall between groups. Further, since direct comparison of ERP amplitude between groups is often flawed, due to relative amplitude differences between samples leading to spurious effects (see MacLeod & Donaldson, 2017,for a discussion in relation to memory), we compared the presence – or absence – of ERP effects, predicting that the two groups would differ. Specifically, the Control group should show the classic old/new parietal ERP effect from 500ms onwards (*Hit-Hit* significantly greater in amplitude than *Correct Rejection* responses), with the magnitude of the ERP response for *Hit-Miss* (item correct, source incorrect) also larger than *Correct Rejection* responses. In contrast, since source memory is predicted to be the most likely aspect of episodic memory affected by frequent

blackout experiences, we predicted that our MBO group would display a parietal old/new effect with an altered pattern of activity for *Hit-Miss* responses. This could be either no differences in ERP amplitude between *Hit-Miss* and *Correct Rejection* responses, or a change in scalp topography reflecting the contribution of different neural sources to achieve the same behavioural performance as Controls. Note that if we are predicting an altered pattern of retrieval, it stands to reason that the response profile of the mid-frontal ERP effect would also be changed in our MBO group compared to Controls. Furthermore, since we are concerned with determining if our MBO participants employ alternative neural strategies for retrieval, this can not be discerned from ERP waveforms alone and topography must be analysed. We therefore employed microstate segmentation, a reference-independent data-driven analysis that highlights changes in information processing in the brain, to examine whether a shift in neural strategy is evidenced by different scalp topographies within each group separately.

Materials and Methods

Design

Participants aged 18-30 from the University of Stirling were invited to complete an online drinking behaviours questionnaire. From the 403 respondents, participants who met the inclusion criteria then received an email invitation to take part in the laboratory-based study. These criteria were: (1) being aged 18 to 30, (2) not currently suffering from any diagnosed mental health issues, (3) not taking prescribed medication (other than the contraceptive pill), (4) being a fluent or native English speaker, and (5) either having experienced at least 9 MBOs in the preceding 12-months, or never having experienced an alcohol induced MBO. All participants who took part in the laboratory study either received course credits as part of their course requirements or were reimbursed with £15. In total, 51 participants attended the laboratory, however 3 were excluded due to insufficient trial numbers (<16) in ERP conditions, and the remaining 7 due to incorrect eligibility criteria (n=1), participant discomfort (n=1), or lack of a discernible ERP signal (n=5). The remaining 41

individuals consisted of a Control group (n = 21, 9 males, mean age = 22.68, SD = 2.89) and experimental group (MBO group) (n = 20, 9 males, mean age = 22.8, SD = 2.8).

Informed consent was obtained from all participants in writing. The study was approved by Stirling University's General University Ethics Panel and their NHS, Invasive and Clinical Research committee.

Procedure

Upon arrival at the laboratory, participants firstly provided written consent and were then fitted with an EEG cap. The experiment was presented using experimental software E-Prime 1.2 (Psychology Software Tools, Pittsburgh, PA). Consisting of 8 blocks, each block contained a study phase, followed by an item recognition and source memory task (see Figure 6.1). Blocks were presented in a randomised order, and words within each study and test phase were also displayed in random order. Each block contained 30 study words selected from the MRC psycholinguistic database (Coltheart, 1981; M. Wilson, 1988), all nouns over 4 letters long with medium-high familiarity between 400-600, which were presented on a screen for 2000ms in either blue or green text with an interstimulus interval of 1000ms. The screen was positioned approximately 71cm in front of participants, with words at 6.047° of horizontal and 0.803° of vertical visual angle. Participants were asked to try and remember both the word and the colour in which it was presented. Following the study list, participants then viewed a test list of 60 words presented in black font. They were asked if each word had been previously displayed (an old word) or if it was a new word. If participants responded 'old' to an old word, we classified this as a hit, and a miss if they responded 'new'. Correspondingly, when new words were presented and the participant responded 'new', this was classified as a *Correct Rejection*. False alarms were when participants responded 'old' to the presentation of a new word. Old and new responses were recorded on a stimulus response box and response buttons counterbalanced across participants. If they selected 'old', they were then asked whether the word had previously been displayed in blue or green text, allowing us to separate out hit responses into successful source recollection (*Hit-Hit*) from unsuccessful (*Hit-Miss*). Again, button response (blue/green) was counterbalanced across participants. All responses (old/new and blue/green) were forced choice and binary. In total, the experiment included 480 trials.

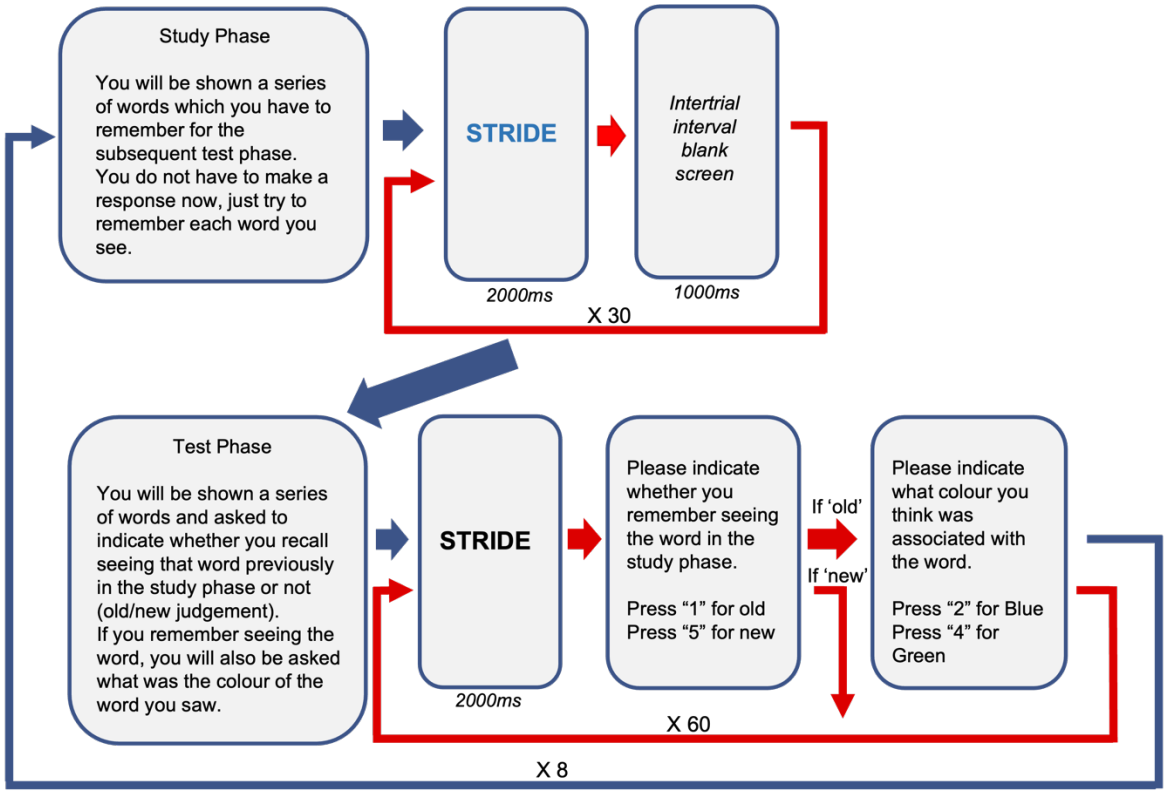

Figure 6.1: **Source memory task structure.**

Event-related potentials

Scalp activity was recorded using SynAmps^2 (Neuroscan, Inc., El Paso, TX, USA) amplifiers at a 1 kHz sampling rate from 64 Ag/AgCl electrodes mounted in an elastic cap and distributed across the scalp according to the extended 10-20 system, using CZ as a reference. Impedances were kept below 5kΩ. Individual electrodes were placed on both the left and right mastoid bones, above and below the left eye for recording vertical eye movements, and on both temples for horizontal eye movements. The electroencephalogram was filtered on-line between 0.01 and 200 Hz and off-line with a band-pass zero phase shift digital filter, comprising of a high pass filter at 0.1 Hz (12 dB/octave slope) and a low pass set to 30 Hz (48 dB/octave slope). Eye-blink artefacts were mathematically corrected using a model blink artefact computed for each individual ((Gratton et al., 1983). Signals exceeding ±75μV in any given epoch were automatically discarded. EEG recordings were cut into epochs ranging from -100ms to 1000ms after stimulus onset and averaged for each individual according to the category of participant response (*Hit-Hit, Hit-Miss, Correct Rejection)*. Grand averages for each response type were calculated for each group after re-referencing individual ERPs to the common average reference. Difference waves were calculated for old vs.

new items (*Hit-Hit* minus *Correct Rejection* and *Hit-Miss* minus *Correct Rejection*, which we abbreviate to *HH-CR* and *HM-CR*). Processed data are available at http://hdl.handle.net/11667/184.

Segmentation Analysis

EEG data from Control and MBO participants was also subjected, separately for each group, to topographical analyses looking for stable patterns of scalp activity, that is, functional microstates (Brunet et al., 2011). In brief, our use of microstates analysis is motivated by the fact that an analysis of ERP waveforms relying on amplitude changes between conditions cannot definitively distinguish between separable cognitive processes for each condition or a single cognitive process that differs only in response strength. Functional microstates allow the objective assessment of scalp topographic distributions which remain stable over time, where shifts between microstates represent a change in the underlying neural generators driving the EEG signal, and therefore indicate a shift in information processing (Lehmann et al., 1987).

We first conducted paired topographic ANOVA's (TANOVA) between *Hit-Hit* and *Correct Rejection* responses, and *Hit-Miss* and *Correct Rejection* responses for both groups. TANOVA determines topographic divergence between ERP responses for every millisecond of the waveform by measuring global dissimilarity, that is, highlighting each timepoint when topographic distributions change shape. To be clear, this refers to changes from one stable topographic distribution to another. We only considered stable periods of divergence greater than 10 ms. Next, we subjected grand averaged data from our two groups of participants to a segmentation procedure, which uses a hierarchical cluster analysis to produce a series of microstates in the form of topographic maps. The optimal number of microstates was determined by comparison of a Mean Criterion and a Meta Criterion, combining the best possible outcome metrics from a series of comparison statistics (see Bréchet et al., 2019). Finally, we assessed the statistical validity of our segmentation procedure through analysis of Global Explained Variance (GEV) for each microstate on individual participant data for both groups. Since our segmentation analysis revealed the presence of multiple microstates throughout the time windows for the mid-frontal and parietal ERP effects, which overlapped (400-600ms, 500-800ms traditionally), no microstate, for example, best fitted the whole 500-800ms period. We therefore assessed the change in GEV for each

microstate, when present, in 100ms bins (from 400-500ms, 500-600ms, 600-700ms, and 700-800ms).

Statistical Analysis

Behavioural accuracy analysis was conducted using t-tests between groups. We assessed differences between *Hit-Hit*, *Hit-Miss*, *Correct Rejection* and *False Alarm* mean accuracy (%). Additionally, we considered differences in d' (discrimination between old and new items) and C (response bias). Response time data was not analysed because they were recorded from a response screen rather than from stimulus onset. To be clear, in our experimental design, a stimulus was displayed on screen followed by a response screen from which response time was recorded. Since participants likely began the cognitive process of retrieving information about the stimulus following its presentation rather than waiting for the response screen, measuring RTs would not be an accurate reflection of the speed of the old/new decision.

ERP data was analysed using repeated measures ANOVAs conducted within each group separately. To be clear, we did not statistically compare ERP amplitudes between our MBO and Control participants. Between groups comparisons of ERP waveforms are not valid since fluctuations in amplitude or latency across independent samples can easily result in spurious significant effects arising. However, we can compare the presence or lack of statistical effects across samples, which is the approach we employ here. Previous literature (Rugg & Curran, 2007) highlights two time-windows of interest for examination: a mid-frontal effect between 300-500ms, and a left parietal distributed effect between 500 and 800ms, which purportedly index familiarity and recollection respectively. Note that these time windows and electrode sites where they are measured can vary across studies, (for example, Addante et al., 2012; Hoppstädter et al., 2015) but are in general accordance. In the case of our dataset, we inspected difference topographies (*HH-CR*, *HM-CR*) for both groups to determine time-periods and electrode sites where differences between responses arose. Using this approach, we identified (1) an early mid-frontal effect across a 400-600ms time window (see Figure 6.2) maximal over electrode sites C1, CZ, and C2; (2) parietally distributed differences onsetting from 500ms, which we analysed in a 500-700ms time-window (see Figure 6.3). Planned comparisons were made between difference waves of *HH-CR* and *HM-CR* responses for both groups over the 400-600ms

time window, and supplemented by ERP analysis of *HH*, *HM*, and *CR* responses where appropriate for clarity of reporting. We further analysed difference waves from left parietal electrode sites (P1, P3, and P5) across a 500-700ms time window, identified from inspection of difference topographies and confidence intervals. For convention, we have also included an analysis of the 500-800ms time window in supplementary data (see Appendix 1), in line with previous literature (Murray et al., 2015; Rugg & Curran, 2007). Again, for clarity we also complement our reporting with analysis of individual ERP responses (*HH*, *HM*, and *CR*), even though statistically the analysis of difference waves or individual responses are equivalent. All analysis was conducted using Jamovi version 1.6.23 (Jamovi, 2021; R Core Team, 2020).

Results

Behavioural Data Analysis

Independent sample t-tests were conducted to investigate differences in accuracy performance between groups. Means across all behavioural measures suggested that the Control group were more accurate than the MBO group, but critically no differences reached significance.

Table 6.1:

Behavioural Accuracy Results

	Control (n = 21)		**MBO (n = 20)**		
	Mean	S.D.[a]	Mean	S.D.	Difference
Hit-Hit (%)	61	0.17	57	0.18	$t(39) = 0.74$, $p = .463$
Hit-Miss (%)	16	0.07	19	0.07	$t(39) = 1.22$, $p = .231$
Source Accuracy (%)	72	0.14	65	0.15	$t(39) = 1.25$, $p = .219$
Correct Rejection (%)	92	0.08	89	0.1	$t(39) = 0.89$, $p = .377$
False Alarm (%)	8	0.08	11	0.1	$t(39) = -0.89$, $p = .377$
Miss (%)	23	0.14	24	0.15	$t(39) = -0.3$, $p = 1$
d'	0.18	1.19	-0.19	1.52	$t(39) = 0.88$, $p = .390$
C	0.05	0.62	-0.05	0.84	$t(39) = 0.402$, $p = .690$

[a] *standard deviation*

Event Related Potentials

Mid-frontal effect between 400-600ms

Figures 6.2A and 6.2B shows ERPs over central electrode sites (C1, CZ, C2) demonstrating a mid-frontal effect between 400-600ms, whereas Figures 6.2C and 6.2D highlight difference waves for *HH-CR* and *HM-CR* responses in both Control and MBO groups. Note the confidence intervals surrounding the difference waves in Figures 6.2C and 6.2D respectively; while confidence intervals suggest no differences between *HH-CR* and *HM-CR* in the Control group, in the MBO group they highlight a separation between *HH-CR* and *HM-CR* difference waves starting at 399 and lasting until 588ms (lower bound of *HH-CR* is greater than upper bound of *HM-CR*). We draw attention to the fact that an analysis of the mid-frontal effect as defined a priori (that is, 300 – 500ms time window, more frontally distributed), although visually conformed with previous literature, resulted in no significant differences between conditions (see Appendix 3.1).

Repeated Measures ANOVA analysis, with factors of Response Difference (*HH-CR*, vs. *HM-CR*) and electrode site (C1, CZ, and C2), confirmed our confidence interval analysis in the Control group; there was no significant difference between *HH-CR* and *HM-CR* responses (p = .233). However, the MBO group showed significantly greater mean amplitude for *HH-CR* (M = 0.941, SE = 0.185) than for *HM-CR* (M = 0.449, SE = 0.231), $F(1,19) = 5.841$, $p = 0.026$, $n^2_p = 0.235$. These results are easily understood by examining individual response amplitudes. As shown in Figure 6.2, the Control group did not differ between their *HH* and *HM* responses (p = .698), but both *HH* [$t(20) = 5.2$, $p < .001$] and *HM* [$t(20) = 3.65$, $p = .005$] were significantly greater than *CR*. Interestingly, the MBO group showed a different pattern with *HH* significantly greater than *CR*, $t(19) = 5.08$, $p < .001$, and a significant trend between *HH* and *HM*, $t(19) = 2.42$, $p = .078$, but critically, no difference between *HM* and *CR* (p = .202).

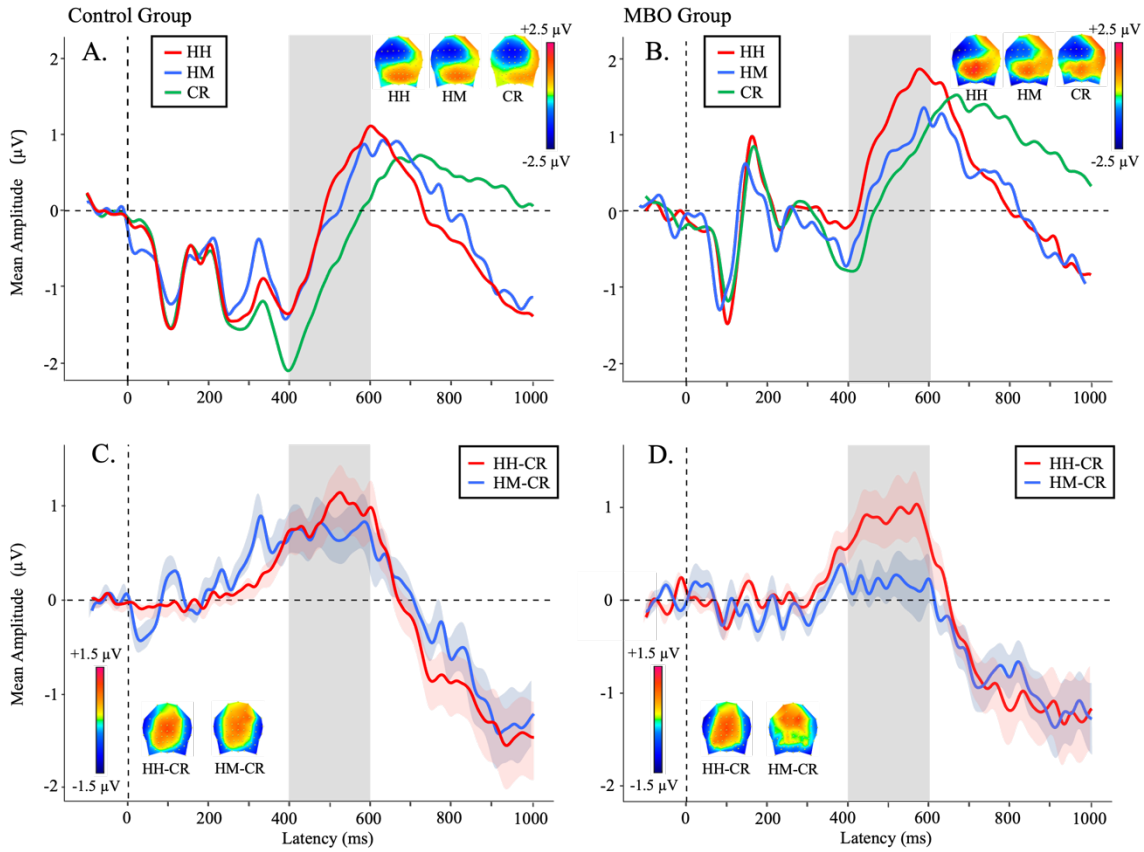

Figure 6.2: **Electrophysiological responses, response topographies, difference waves, and difference topographies highlighting the mid-frontal familiarity effect.** (A) & (B) display averages of electrode sites C1, CZ, and C2, for ERP responses from the Control and MBO groups respectively, where the grey shaded regions represent a 400-600ms time window of analysis and insets show response topographies (nasion at top of the maps); (C) & (D) show difference waves of *HH-CR* and *HM-CR* responses for both groups, with difference topographies highlighting maximal differences at central electrode sites between 400 and 600ms. Note the overlap between *HH* and *HM* conditions in the Control group (A), and *HM* and *CR* conditions in the MBO group (B), leading to the differences observed in (C) & (D).

Parietally distributed effect between 500 – 700ms

Figures 6.3A and 6.3B display average ERPs over left parietal electrode sites (P1, P3, P5) highlighting the 500-700ms time window. Figures 6.3C and 6.3D show the difference waves for *HH-CR* and *HM-CR* responses in both Control and MBO groups. However, the confidence interval differences in Figure 6.3D show a large separation between the difference wave effects beginning at 513ms and ending at 676ms in the

MBO group, but only trivial differences within the Control group (588-597ms, and 648-660ms).

In the Control group, a repeated measures ANOVA, with factors of response differences (*HH-CR* vs. *HM-CR*) and electrode site (P1, P3, P5), showed no significant difference between *HH-CR* and *HM-CR (p= .657)*. In contrast, the MBO group displayed a difference between *HH-CR* and *HM-CR*, $F(1,19) = 7.846$, $p = 0.011$, $n^2_p = 0.292$; mean amplitude for *HH-CR* ($M = 1.084$, $SE = 0.327$) was significantly greater than for *HM-CR* responses ($M = 0.302$, $SE = 0.236$). While the Control group display a graded pattern for responses (*HH>HM>CR*) over the left hemisphere, visible in Figure 6.3A, the statistics suggest no difference between *HH* and *HM* ($p = 1$), but both *HH* and *HM* are significantly greater in amplitude than *CR*, $t(20) = 2.762$, $p = .036$, and $t(20) = 2.711$, $p = .04$, respectively. Similarly, the MBO group also do not display a graded pattern of response (see Figure 6.3B); *HH* was statistically greater than *HM*, $t(19) = 2.8$, $p = .034$, and also *CR* responses, $t(19) = 3.31$, $p = .011$, but critically, no difference found between HM and CR responses ($p = .65$). See supplementary data for an analysis of the 500-800ms time window (Appendix 3.1)

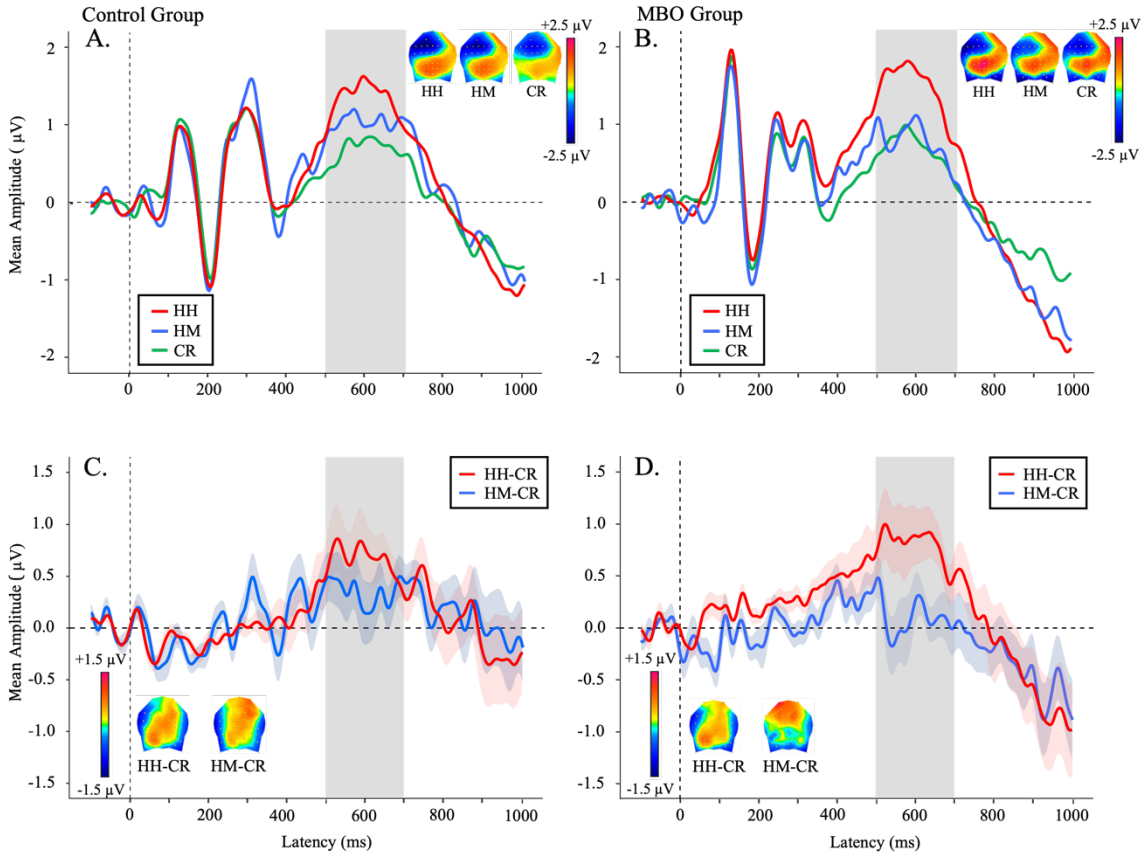

Figure 6.3: **Electrophysiological responses, response topographies, difference waves, and difference topographies highlighting the parietally distributed recollection effect.** (A) & (B) display averages of electrode sites P1, P3, and P5, for ERP responses from the Control and MBO groups respectively, where the grey shaded regions represent a 500-700ms time window of analysis and insets show response topographies (nasion at top of the maps); (C) & (D) show difference waves of *HH-CR* and *HM-CR* responses for both groups with difference topographies showing maximal differences at left parietal electrode sites between 500 and 700ms. Note the graded response pattern between *HH*, *HM* and *CR* conditions in the Control group (A), and the overlap between *HM* and *CR* conditions in the MBO group (B), leading to the differences observed in (C) & (D).

Topographical analysis and microstate segmentation

We conducted paired TANOVA comparisons between participant response differences (*HH vs. CR* and *HM vs. CR*) in both Control and MBO groups to map the point in time when topographies may diverge for our difference wave responses. This analysis revealed topographic differences between *HH* and *CR* in Controls over the 328-646ms time window, whereas the MBO group showed a sustained difference from 432ms to the end of the epoch (1000ms). Differences between *HM* and *CR* were evident from 363ms until 539ms, and from 562 – 635ms in Control participants, and from 504-

622ms, 635-end of epoch in the MBO group. These results highlight changes in topography that arise early in the ERP signal for both *HH* and *HM* conditions vs. *CR*, which consistently overlap our ERP findings and confidence interval data, however, they do not suggest how topographies may differ. We therefore subjected our grand averaged ERP data to microstate segmentation analysis. Since multiple possible topographic maps were present in the best fitting segmentation, we fitted our segmentation back to individual participants in 100ms periods, beginning from 400–800ms and encompassing the 400-600ms and 500-700ms time windows, for a clear interpretation. Global variance explained by each microstate, and across each 100ms time window, are detailed in Appendix 3.2, Figures 1C and 1D.

For clarity, we named the microstates from our analysis according to the ERP time windows with which they corresponded, and also numbered them according to onset time, with the Control group numbered before the MBO group (see Figure 6.4). For example, Mid-Frontal 1 (MF1) is the first microstate observable between 400-600ms, and subsequent microstates are labelled MF2, MF3, etc. In the Control group, we observed four microstates over the 400-500ms period, MF1, MF2, MF3, and Mid-Frontal/Parietal 1 (MF/P1), none of which explained the pattern of response across this time period. While these microstates fit the individual data, they did not separate out one response type compared to another. However, between 500 and 600ms, analysis of maps MF2, MF3, MF/P1 and MF/P2 revealed a main effect of map, $F(1.95, 39.05) = 3.34$, $p = 0.047$, $n^2_p = 0.143$, and an interaction between map and response, $F(3.53, 70.69) = 2.6$, $p = .05$, $n^2_p = 0.115$. Specifically, the interaction was driven by (1) the lack of explanatory power for MF2 and MF/P2 in this time period, and (2) the strength of fit for map MF3, fitting better the *CR* response, compared to map MF/P1, which appeared to fit *HH* and *HM* conditions better than *CR*. Post-hoc analysis suggested that map MF3 fitted the data better for *CR* than for *HH* or *HM* responses, $F(1.61,32.11) = 3.84$, $p = 0.04$, $n^2_p = 0.161$, however, for map MF/P1 no significant differences in fit were found ($p = 0.216$).

Across the 600-700ms time window, we analysed the fit of maps MF/P1,MF/P2, and Parietal 1 (P1), yet no map best fitted any one response over another $F(1.78,35.61) = 1.16$, $p = .319$, $n^2_p = .055$, that is, they all fitted the data equally during this period. However, during the 700-800ms time window, where maps MF/P2, P1, P2, and P3

fitted the data, only one microstate (P2) appeared to respond in a graded fashion, $F(2.86,57.28) = 5.051$, $p = .004$, $n^2_p = .202$. Microstate P2 explained more variance for *HH* responses than *CR*, $p = .017$, but did not separate *HH* from *HM*, $p = .306$, or *HM* from *CR*, $p = .164$. In short, in Control participants we found a distinct microstate (MF3) explaining *CR* responses over other response types between 500-600ms, and a further microstate (P2) which explained more of the variance in individual participants between a 700-800ms time window, in a seemingly graded fashion (Figure 6.4).

In the MBO group, fewer maps overall were present across the periods analysed (see Figure 6.4), highlighting that microstates involved in HH, HM, and CR responses are identical in the MBO group. The critical question is whether any one of the identified microstates best represents memory processes, such as recollection or familiarity. Between 400 and 500ms, we found two microstates, map MF4 and MF/P3. An interaction between map and response was found, $F(1.81,34.38) = 4.43$, $p = .022$, $n^2_p = 0.189$ suggesting a graded response (*HH*>*HM*>CR); however, post-hoc analysis of this interaction revealed that microstate MF/P3 explained more variance for *HH* than *CR*, $t(19) = -2.79$, $p = .035$, but was not different from *HM* ($p = .328$). Microstate MF/P3 remained present between 500-600ms, again producing a main effect of response, $F(1.7, 32.38) = 9.44$, $p < .001$, $n^2_p = 0.332$, which explained more variance for *HH* than for *HM* and *CR* (*HH*>*HM*, $t(19) = 2.83$, $p = .032$; *HH*>*CR*, $t(19) = -3.64$, $p = .005$), mirroring our ERP results across the mid-frontal effect and the parietal effect. Within the 600-700ms bin microstates MF/P3 and P4 were present, and we found overall an effect of condition, $F(2, 37.98) = 3.431$, $p = .043$, $n^2_p = 0.153$, but no interaction between map and condition, $p = .325$. Finally, between 700-800ms the same two microstates were present, however, map P4 now best fitted the data in this time period, $F(1, 19) = 7.854$, $p = .011$, $n^2_p = 0.292$, yet failed to separate out response types, $p = .86$.

To summarise the microstate findings, across the Control group we found evidence for a distinct microstate for *CR* responses compared to *HH* and *HM* during the 500-600ms time window (MF3). We also found one microstate that separated response type in a seemingly graded fashion during the 700-800ms time window (P2). In contrast, the MBO group showed no distinct microstate for *CR* responses, rather we found one microstate that separated out *HH* responses from others from 400-600ms (MF/P3). After this time period, no microstate separated out response condition.

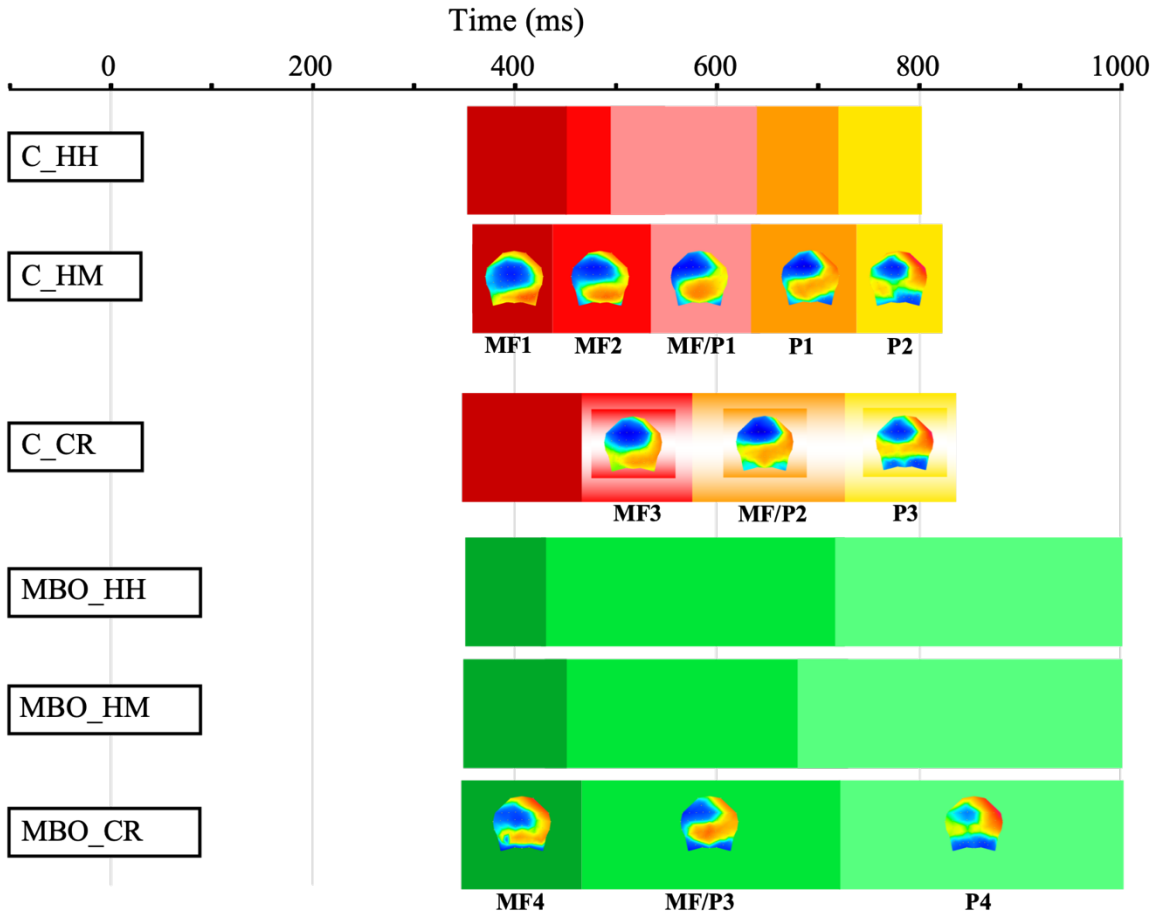

Figure 6.4: **Microstate segmentation analysis.** Rectangular coloured bars show onset and offset latencies of topographic microstates for both Control (in red tones) and MBO (in green tones) groups, separated by response type (HH, HM, CR). Where the same colour is shown across different responses (for example, the first green colour block for MBO HH, HM and CR responses) indicates where the same microstate was present for multiple response types. Map name abbreviations, alongside the change in colours, are given to dissociate each microstate (for example, MF1). Individual topographic images derived from the segmentation procedure for each microstate are embedded within the corresponding map. Note in the control group the presence of different microstates (denoted by the graded colour effect) for the Correct Rejection response type, indicating the employment of different neural generators for CR compared to HH and HM responses in Controls only.

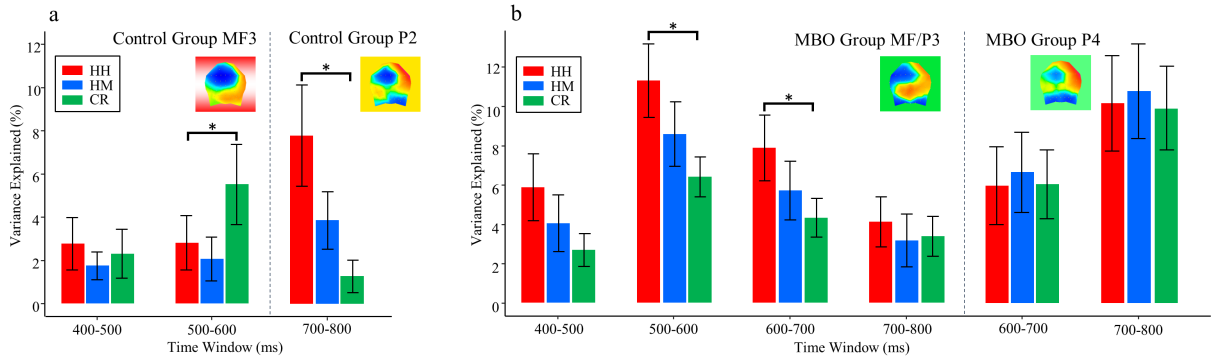

Figure 6.5: **Global Explained Variance.** Changes in global explained variance across time windows of microstate analysis for each response type in the Controls (a) and MBO groups (b), for the most relevant microstates (inset). Bars represent an average of the best fit for individual participants. Significance is indicated for microstates which better explain certain response types (for example, *HH* versus *CR*) by an asterisk.

Discussion

This study investigated differences in recognition and source memory between Control participants, and individuals who reported frequent alcohol-induced memory blackouts in the preceding 12 months. We predicted that behavioural performance was unlikely to differ between groups while both Blackout and Control participants were not intoxicated. Indeed, we found that behavioural performance between the two groups was comparable for item recognition and source recall – both groups performed above levels of chance, in line with previous literature (Hartzler & Fromme, 2003; Jackson et al., 2021; Wetherill & Fromme, 2011). However, we also hypothesised that the MBO group may employ alternative neural strategies compared to the Control group in order to complete the task; either no differences in ERP amplitude between *HM* and *CR* responses would be observed, or we could find a change in topographic shape during the time windows for the mid-frontal and parietal effects. Converging evidence from ERP, confidence intervals of difference waves, TANOVA, and microstate analysis highlighted differential patterns of effects between groups, supporting the prediction of no change in amplitude for *HM* and *CR* ERPs. We found an early mid-frontal ERP effect between 400-600ms in the Control group that separated *HH* and *HM* from *CR* responses, whereas in the MBO group *HM* was not different from *CR*. ERP

findings also exhibited a parietal ERP response across the 500-700ms time window, where *HH* and *HM* were larger than *CR* in the Control group, however in the MBO group *HM* mean amplitude again tracked the pattern of correctly rejected new items. Further, our microstate analysis built upon these ERP results: in the Control group we found a microstate specific to the *CR* response within the time window of the mid-frontal effect (500-600ms) suggesting that the *CR* judgement employed a different cognitive process to that for *HH* and *HM*. Moreover, we found a later microstate from 700-800ms that appeared to be graded in response (*HH* larger than HM, larger than CR). In the MBO group one microstate between 400-600ms separated out *HH* responses from *HM* and *CR*, with no other effects observable. Overall, both ERP and microstate data for the Control group show that *HH* and *HM* responses were clearly separated from *CR*, yet in the MBO group, only *HH* was different from *CR*, the *HM* response overlapped *CR* responses.

Why is behavioural performance identical across both groups and yet ERP and microstate analysis uncover a different pattern of response? Firstly, our participants completed a word recognition paradigm, a task which has been shown to be less cognitively demanding than recollection for Control participants as well as amnesics (Hirst et al., 1988), and after alcohol consumption (H. V. Curran & Hildebrandt, 1999). We also know that behavioural literature supporting differences between Control and alcohol-induced blackout participants is mixed (Jackson et al., 2021; Wetherill & Fromme, 2011). Secondly, it is clear from decades of research that functional neuroimaging offers insights not afforded by analysis of behaviour; in our case there is a history of research underlining a separation between memory processes with ERPs (familiarity and recollection, see Rugg & Curran, 2007), which may not be divisible in the current study by examining only behavioural responses. Arguably, multiple underlying cognitive processes can be mapped by neuroimaging, whereas the cognitive processes driving differences in behavioural data converge into one unitary measure and cannot be disentangled. Put simply, behavioural data (accuracy, RT's) may conceal the varied and differing cognitive processes operating at any one time.

The ERP data from our Control group, supported by the microstate analysis, follows an expected pattern observed in previous recognition memory studies (for example, Vilberg & Rugg, 2009; Wilding & Rugg, 1996) yet the pattern displayed in our

MBO group does not. To be more specific, an accurate item recognition response accompanied by successful source retrieval is purportedly indicative of the process of recollection (see MacLeod & Donaldson, 2017; Rugg & Curran, 2007) in our data our *HH* responses indicate accurate item recognition and also successful retrieval for the contextual detail during study, in this case, colour of the studied words. In contrast, the theorised memory process of familiarity is said to be devoid of any qualitative information that supports recollection, for example, source details in the present experiment, resulting in a *HM* response. Note that in actuality the *HM* and the *HH* ERP responses represent varying proportions of accurately recollected trials, trials where source memory was absent, and guesses, but the exact proportions of each are impossible to determine. Furthermore, our ERP and microstate data suggest that when source recollection fails, in our MBO group, the *HM* response appears identical to the *CR* response. When correctly rejecting new stimuli participants make the decision based upon knowledge that it was not present in the studied items, and this knowledge only (unless the decision was made by guessing, except that this would have increased our false alarm rate). It may be similar for items responded to as *HM* in the MBO group, in that they have no specific episodic memory for the item yet know that it must have been part of the previously studied material.

The present study showed no differences in source recollection between Controls and our sober MBO group, but interestingly, the *HM* response, representing a failure of source recall (correctly recognised word, no recall of colour source), differed between groups. It is unknown what the immediate impacts of an acute binge-drinking event are upon neural correlates of memory, however, we do know that memory is impaired when tested the next day after blackout, and participants are sober (Jackson et al., 2021). An MBO is a transient amnesic event, and there are similarities to be drawn between data from amnesics and individuals who experience frequent memory loss due to alcohol. For example, among other memory problems, deficits in source monitoring have been observed in individuals with an acquired amnesia, without being linked to item recall (Shimamura & Squire, 1987, 1991). However, it is noteworthy in these studies that many amnesic participants were patients with alcoholic Korsakoff's syndrome, who have been shown to have impaired source monitoring (Brion et al., 2017; Kessels et al., 2008).

The pattern of ERP effects observed in the mid-frontal familiarity time window (400-600ms) carry over into the parietal recollection effect time window (500-700ms), in both groups, suggesting that these processes are not necessarily separable in ERPs alone. Indeed, the separation of familiarity and recollection has always been driven by scalp topography at different time windows more than ERP differences (Rugg & Curran, 2007) and to this end we applied a data driven topographical approach (microstate segmentation) to recognition memory data with ERPs. Our microstate data from the Control group suggest that differences between *HH*, *HM*, and *CR* responses begin just before 500ms (onset of distinct microstates, see Figure 6.4), which fits within the reported time windows in the literature for the onset of a parietal effect linked to recollection (Wilding & Ranganath, 2012; Wilding & Rugg, 1996). Notably, while we found a specific microstate elicited by *CR* responses between 500-600ms suggesting recognition of new information, we also found no differences between microstates for *HH* and *HM* responses across the whole analysis window (400-800ms). This microstate data implies that the processes driving ERPs for *HH* and *HM* conditions, arguably recollection and familiarity respectively, come from the same neural source generators, that is, these processes are not distinct. Furthermore, our MBO group also show no distinct microstates for *HH*, *HM*, or *CR* responses, rather we found that our microstates differ in response strength only for *HH* responses compared to *CR*. Although we are reluctant to make any strong claims about theories of memory in this manuscript, the pattern of data here is noteworthy.

Conclusion

In conclusion, our ERP and Microstate data suggest that individuals with a history of frequent alcohol-induced MBOs exhibit an atypical pattern of neural functioning during recognition memory and source recollection, despite showing no obvious impairment in behaviour. This atypical neural pattern in young adults is concerning, given the known links between alcohol, deteriorating cognitive functioning, and health (Cao et al., 2015; Powell et al., 2021; Sarich et al., 2021).

CRediT author statement

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Chapter Seven:

Effects of alcohol and alcohol-induced memory blackouts on ERPs and recognition memory

Abstract

In previous chapters, our data showed that while sober, behavioural accuracy in episodic memory tasks does not differ between Controls and participants who frequently experience MBOs. However, Chapter 6 showed that event-related potential (ERP) activity differed, suggesting that behavioural performance alone may not reveal differences in memory strategies between groups. In an extension of the aforementioned study, we ask whether ERP response differences would differ between *before-alcohol* and *after-alcohol* conditions, or between groups. Further, we consider whether sober MBO group participants, who had recently experienced a blackout (<20-hours), would exhibit depressed neural functioning or memory performance.

EEG data was recorded from Control (n = 17) and MBO (n = 16) group participants, who took part in a recognition memory and source judgement (blue/green colour decision) task. Participants completed the first half of the study sober, and the remaining following a scaled dose of alcohol (Study 1). Another MBO group (n = 19) repeated the experiment sober, but within 20-hours after-blackout (Study 2). We predicted that groups would show no difference in behavioural performance while sober, which would reduce similarly *after-alcohol*. Like Chapter 6, we expect a difference in ERPs between the two groups, but in both conditions. Further, we predict some recovery in performance for our *after-MBO* group in comparison to *after-alcohol*, which could also be reflected in ERPs.

As expected, behavioural accuracy between groups did not differ, with a global reduction in accuracy *after-alcohol,* and a shift to a conservative response strategy. A*fter-MBO*, participant data showed continued impairment, even when sober. ERPs revealed that both groups appeared to change cognitive strategy *after-alcohol,* however, these changes differed between groups. *After-MBO* ERPs exhibited a significant shift in time for the left-parietal ERP effect, suggesting delays in recollection. In sum, ERP data suggest differing underlying memory strategies between individuals who blackout and those who do not, with delays to recollection *after-MBO* hinting at the enduring acute depression of memory processes after the MBO event.

Introduction

Alcohol is known to have deleterious consequences for health with a wide range of illnesses and harms directly attributable to its consumption (Castillo-Carniglia et al., 2019; Rehm, 2011; Spillane et al., 2020). Additionally, studies comparing alcoholics to controls have repeatedly shown differences in brain structures (Fortier et al., 2011; Jernigan et al., 1991; Moselhy et al., 2001) and in cognitive functioning (Maillard et al., 2020; Oscar-Berman et al., 2014; Sullivan et al., 2000). Participants in such studies have typically engaged in binge and heavy drinking patterns over years, and are predominantly middle-aged (for example, see Goodwin et al., 1973; Pfefferbaum et al., 2001; Wieben et al., 2018). However, heavier binge-drinking patterns are more frequently observed in younger adults, and adolescents, which is particularly concerning since adolescence and young adulthood coincide with periods of significant changes in brain development (for recent reviews, see de Goede et al., 2021; Lees et al., 2021). Indeed, neuroimaging studies with adolescents and young adults have highlighted structural differences in the brain between binge-drinkers and control participants (Petit et al., 2014; Squeglia et al., 2009, 2014). Further, extreme binge-drinking can be accompanied by an alcohol-induced memory blackout (MBO), a transient amnesic event linked to impaired functioning of the hippocampus due to alcohol (A. M. White, 2003). Since both binge-drinking and MBOs have been shown to frequently occur in late adolescence and early adulthood (Bhatti et al., 2020; Hingson et al., 2016), understanding their effects on brain function and cognition is critical. In Chapter 6, we described a recognition and source memory task completed by control and MBO participants while sober. Although behavioural results did not differ between groups, there were clear differences in the pattern of neural responses which require further investigation. Therefore, the aim of this chapter is to examine neural correlates of recognition and source memory performance after ingesting alcohol, and also within 20 hours after experiencing an MBO.

An alcohol-induced memory blackout occurs when the formation of episodic memories is impaired following rapid, excessive consumption of alcohol (A. M. White, 2003). Two types of blackout experience have been identified – *fragmentary* and *en bloc* (Goodwin et al., 1969a). Following the more common *fragmentary* blackout,

individuals may recall short sections of time from while they were intoxicated either spontaneously or after prompting, whereas no memories are typically recoverable following an *en bloc* blackout. Episodic memory – memory for previously experienced events – requires recollection of complex information, for example, location, context, and perceptual information (M. K. Johnson et al., 1993; Wilding & Rugg, 1996). These contextual details enrich recollected memories, invoking the subjective feeling of having 'lived' an event and providing the source context for target information (M. K. Johnson et al., 1993).

Studies of source memory have been utilised within the alcohol literature in an effort to increase understanding of how episodic memory is affected by MBOs (Hartzler & Fromme, 2003; B. L. Schwartz et al., 2002; Wetherill & Fromme, 2011). Hartzler and Fromme (2003) suggested that *fragmentary* blackouts are associated with poor retrieval of source detail and found that individuals prone to *fragmentary* MBOs showed increased deficits in recall tasks both under the influence of alcohol and during withdrawal, compared to controls. Wetherill and Fromme (2011) also found significant differences in recollection of source information between participants who experienced MBOs and those who did not following a moderate dose of alcohol. Interestingly, while sober, they found no difference in memory performance between the two groups. These findings suggest that those who experience MBOs may be more susceptible to memory deficits after consuming alcohol than those who do not, even when the quantity of alcohol is much smaller than would be required to induce an MBO. Further, they imply that deficits in memory may remain for a period of time even after cessation of drinking despite no discernible behavioural differences between groups.

The cognitive processes which underpin episodic memory are known to be disrupted by alcohol (H. V. Curran & Hildebrandt, 1999; Mintzer, 2007; Söderlund et al., 2007). The hippocampus communicates with sensory and perceptual networks, receiving information which is bound together and then transferred out to neocortical regions via the CA1 pyramidal network of neurons (White, 2003). If normal hippocampal functioning is impaired by alcohol, this may adversely affect information transfer and storage. It could be possible to recall fragments of an event, or have a sense of familiarity towards some stimuli, but complete, contextual source information may not be accessible due to incomplete encoding and storage. Interestingly, events

can be comprehended while they unfold, but not recalled later in some stroke related amnesic patients (Oedekoven et al., 2019), hippocampal amnesics (Milner et al., 1968), and also in heavy drinkers experiencing an alcohol-induced MBO (Ryback, 1970; Tamerin et al., 1971). This suggests that either working memory or the ability to employ alternative memory strategies, may facilitate awareness of current events despite hippocampal damage. Moreover, alcohol may not impair short-term memory, at least for durations of up to/around 2 minutes (Ryback, 1970). This ability to function 'normally', albeit within the context of having consumed a large quantity of alcohol, means that the drinker, and those around them, would be unaware of the resulting alcohol-induced blackout occurring at that time. Typically, studies of heavy drinkers do not include adolescent or young adult binge-drinkers and therefore whether those populations display adverse memory functioning akin to long-term alcoholics remains unknown. Further, the point in time when binge-drinking and frequent MBOs may begin to cause observable neural damage is also unclear. It has been suggested that binge-drinking in adolescent mice may not have immediate consequences, except in recognition memory performance (Van Hees et al., 2022), however work in humans has suggested that neural differences can already be seen in young people who drink heavily (Hermens & Lagopoulos, 2018; Nguyen-Louie et al., 2016; Squeglia et al., 2009). It could be that deficits seen in older alcoholics are partly a consequence of the cumulative impact to memory caused by heavy drinking over time, which is therefore not yet visible in younger binge-drinkers. Further, young binge-drinkers may adopt alternative cognitive or neural strategies that can mask differences in behaviour. Regardless, developing a greater understanding of the impact of alcohol-induced memory blackouts on brain functioning and cognition in younger drinkers is of obvious importance.

Laboratory based behavioural studies have advanced our understanding of the effects of alcohol on memory, but it is arguably necessary to employ neuroimaging methods to fully assess its impact. The Event-Related Potential technique (ERP) measures neuronal-based electrical changes at scalp level and is widely adopted in the study of memory. As discussed in the previous chapter, specific ERP components have been identified which are believed to index episodic memory processes, for example, the old/new effect seen in recognition memory tasks over left parietal electrode regions at around 500-800ms following stimulus onset (Rugg & Curran, 2007),

associated with the process of recollection (MacLeod & Donaldson, 2017; Murray et al., 2015). In Chapter 6, ERP findings suggested that Control and MBO groups displayed distinctive response and difference wave patterns, in addition to processing differences evidenced via microstate analysis. If demonstrable variations exist when sober, it is therefore of interest to investigate how/whether these contrasts change after a moderate dose of alcohol has been consumed, and more importantly, if these contrasts remain altered, when sober again, closely following a blackout event.

Here, we investigate episodic memory performance after consuming a scaled dose of alcohol, and after experiencing a blackout event. A Control group who had never experienced a blackout, and an MBO group who reported frequent alcohol-induced blackouts in the preceding 12-months, completed the first half of the present study sober. Participants then received a scaled dose of alcohol before completing the remainder of the experiment (Study 1). Participants in the MBO group were invited to return to the laboratory and repeat the study (Study 2) when sober, but within 20 hours of experiencing an MBO. Consistent with Chapter 6, we did not expect behavioural performance to differ between groups *before-alcohol*. We further expected that *after-alcohol*, both groups would show a similar reduction in accuracy. Again, in replication of findings in Chapter 6, we predicted that *before-alcohol* ERP mean amplitudes within the MBO group would be similar for *hit-miss* (correct identification of an old item but failure to recollect the colour source) and *correct rejection* (correctly identifying a new item) responses across both a mid-frontal and parietal distribution, whereas the Control group would display similar amplitudes for *hit-hit* (correctly identified old item and source) and *hit-miss* responses across both scalp distributions. *After-alcohol*, we hypothesised that the MBO group would show greater evidence of ERP differences compared to *before-alcohol* than the Control group, despite no group behavioural differences. These differences could be related to either global reductions in mean amplitude, or changes in response patterns.

In Chapters 4 and 5 participants showed varying degrees of impairment in both recall and recognition tasks after blackout. Specifically, we observed that blackout effects arose as task difficulty increased. Recognition memory is a less demanding process than recollection, arguably it is a reactive response to a stimulus whereas recollection requires conscious effort. In Chapter 5, where we presented a recognition

memory study, no differences in dprime were observable between *after-alcohol* and *after-MBO* conditions, nor between *before-alcohol* and *after-MBO*, suggesting greater variability *after-MBO* and partial recovery (in a subset of participants). Therefore, in Study 2, we expect similar results, with some behavioural and ERP recovery towards earlier sober baseline measures in line with our previous findings.

Study One

Materials and Methods

Design

The recruitment methods outlined in Chapter 6 were broadly replicated for the present study. In brief, students attending the University of Stirling between 2018-2019 were invited to complete an online general drinking behaviours questionnaire in return for course credits. Responses were screened and individuals meeting the inclusion criteria outlined below were invited via email to take part in a follow-up laboratory-based study. As before, inclusion criteria were (1) being aged 18 to 30, (2) not currently suffering from any diagnosed mental health issues, (3) not taking prescribed medication (other than the contraceptive pill), (4) being a fluent or native English speaker, and (5) having either experienced at least 9 MBOs, or none at all, in the previous 12-months. Additionally, for the current study participants were also excluded if they were taking medication which could interact with alcohol, or if they were at risk of pregnancy. 758 participants completed at least 60% of the questionnaire and a total of 62 eligible participants agreed to take part in the laboratory study following email invitation. Of these, 2 individuals were initially excluded (one due to technical issues, the other did not complete the study). A further 27 participants did not reach 16 trials in all response types (e.g., due to poor EEG recording, or lack of a certain response type), necessary for segmenting into ERP averages, either before or after drinking alcohol and were therefore not included for analysis.

Of the remaining 33, the control group consisted of 17 participants (10 male, mean age = 21.71, *SD* = 2.89), and 16 in the MBO group (5 male, mean age 19.75, *SD* = 1.29). All participants were compensated for their time with either course credits or £22.50. The study was approved by the General University Ethics Panel, while

protocols for both electrophysiological (EEG) recordings and alcohol administration received additional approval from the NHS, Invasive and Clinical Research ethics committee at the University of Stirling.

Source Memory Task

The experiment consisted of an item recognition and source memory paradigm which was presented using E-Prime 1.2 (Psychology Software Tools, Pittsburgh, PA). Each of the 8 blocks within the experiment contained a study phase, followed by a recognition and source memory judgement task (see Figure 7.1). The block presentation order was randomised, and words within each block (both study and test) were also randomly displayed. Each study block consisted of 30 words retrieved from the MRC psycholinguistic database (Coltheart, 1981; Wilson, 1988), all nouns with 4 of more letters with medium-high familiarity between 400-600. Words were presented on screen in either blue or green text for 2000ms with an interstimulus interval of 1000ms and the screen was positioned approximately 71cm in front of participants, with words at 6.047° of horizontal and 0.803° of vertical visual angle. In the study phase, participants were asked to memorise both the word and its colour. Corresponding test blocks contained a list of 60 words presented in black font, including both the 30 previously displayed words plus 30 new words. Participants were asked to identify whether a word was old (previously studied) or if it was new by using a stimulus response box, and response buttons counterbalanced across participants. If 'old' was selected, participants were then asked whether the word had originally been displayed in blue or green text (source judgement). Correct recognition of an old word plus colour was classed as a '*Hit-Hit*', while correct word recognition but incorrect colour source was a '*Hit-Miss'*. Incorrectly identifying a new word as old was classified as a '*False Alarm'*, and new words correctly identified were '*Correct Rejections*'. All responses were forced choice and binary. In total, the experiment included 480 trials. Participants completed first 4 blocks of the study sober (240 trials), and then the remaining 4 blocks following a scaled dose of alcohol.

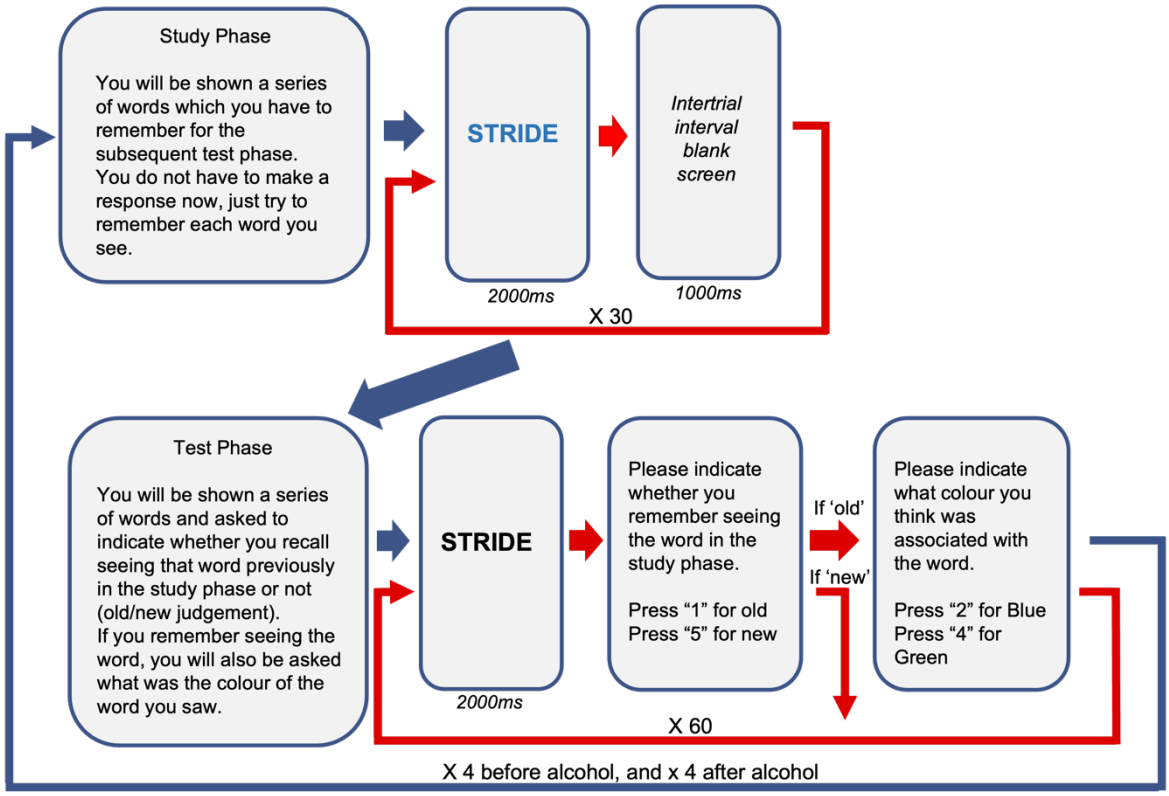

Figure 7.1: **Source memory task structure.**

Procedure

Participants received written information prior to their attendance at the laboratory which asked that they avoid alcohol for 24-hours, and food for 3-4 hours, before their visit, and that they bring photographic ID. They were reminded again of exclusion criteria and that they would be required to drink alcohol during their visit. Upon arrival, all participants presented photographic identification as proof of age, submitted to a breathalyser test (Dräger Alcotest ® 3000; Lübeck, Germany), and provided written consent. Height and weight were recorded and entered into an alcohol-dose formula (Watson, 1989; Watson et al., 1981) along with gender and age. The formula was designed to dose individual participants with sufficient alcohol to achieve a Blood Alcohol Concentration (BAC) of 0.06%, estimated through regular breathalyser testing (BrAC).

Participants completed the first 4 blocks of the study sober before receiving undiluted 37.5% proof vodka in a glass tumbler with optional glass straw. Prior to consumption, the vodka was stored in a freezer to minimise taste intensity. Participants were asked to consume their drink 'as quickly as was comfortable' to elicit a rapid spike in BAC. After 15-minutes, they gargled with water to remove any alcohol

residue from their mouths, and then provided a breathalyser reading. The remaining 4 blocks were then completed, with breathalyser tests between each block. Blocks were completed at participants own pace, with an average time taken per block *after-alcohol* of 7.11 minutes (SD = 0.72). Table 7.1 details the alcohol dose quantities, mean drinking time, and BrAC readings throughout the study. Upon completion of the study, participants were asked to remain in the laboratory until their BrAC had fallen below the Scottish drink drive limit (BrAC 0.22mg/l, BAC 0.05%). During this time, the EEG cap was removed, participants were invited to wash their hair, and soft drinks were offered.

Table 7.1:

Alcohol dose, mean drinking time, and mean BrACC

				Breath Alcohol (mg/l)				
	Vodka (ml)	Alcohol (g)	Drink Duration (secs)	15 mins	Break 1	Break 2	Break 3	End
All (n=33)	96.48 (25.53)	36.18 (9.57)	67.49 (101.76)	0.18 (0.07)	0.23 (0.08)	0.24 (0.07)	0.23 (0.07)	0.23 (0.07)
Controls (n = 17)	103.82 (27.64)	38.93 (10.36)	57.72 (100.54)	0.18 (0.07)	0.23 (0.09)	0.24 (0.08)	0.24 (0.09)	0.24 (0.08)
MBOs (n = 16)	88.69 (21.2)	33.26 (7.95)	76.61 (105.55)	0.19 (0.08)	0.23 (0.09)	0.23 (0.07)	0.22 (0.05)	0.22 (0.05)

Means with standard deviations given in brackets

Event-related potentials

The procedure for recording scalp activity mirrors that described in Chapter 6. To summarise, using CZ as a recording reference, we recorded scalp EEG activity from 64 channels using a SynAmps^2 (Neuroscan, Inc., El Paso, TX, USA) amplifier at a 1 kHz sampling rate. Ag/AgCl electrodes were mounted in an elastic cap and distributed across the scalp according to the extended 10-20 system and using CZ as a reference. On-line data collection was filtered between 0.01 and 200 Hz, whereas off-line we

applied a low-pass zero phase shift digital filter set to 30 Hz (48 dB/octave slope). Eye-blink artefacts were mathematically transformed as previously outlined. Epochs ranging from -100ms to 1000ms after stimulus onset were created from the EEG signal, and averaged per experimental condition, per participant. Grand averages for each response type (*Hit-Hit*, *Hit-Miss*, *Correct Rejection*) were calculated for each group after re-referencing individual ERPs to the common average reference, and difference waves for old/new effects (*Hit-Hit – Correct Rejection*, *Hit-Miss – Correct Rejection*) produced for analysis.

Statistical Analysis

Behavioural accuracy was assessed using independent t-tests between groups, and paired t-tests within groups. We assessed differences between *Hit-Hit* (*HH*), *Hit-Miss* (*HM*), *Correct Rejection* (*CR*) and *False Alarm* (*FA*) mean accuracy (%) *before-alcohol*, and *after-alcohol*. Additionally, we assessed differences in *d'* (signal detection measure of sensitivity, or the ability to discriminate between old and new items) and *C* (response bias). These were defined as:

$$d' = z(HH + HM) - z(FA)$$

where z(HH+HM) is the standardised *HH* plus *HM* rate (%), and *zFA* is the standardised false alarm rate (%); and

$$C = -\frac{z(HH + HM) + z(FA)}{2}$$

which was a measure of the likelihood of a conservative or liberal response. Response time was not analysed as this was not recorded from stimulus onset but from a response screen.

ERP data was analysed within each group separately using repeated measures ANOVAS. Between group analysis was not conducted as fluctuations or differences in amplitude may simply be the result of two independent samples, and not a meaningful difference in variables of interest. Instead, the presence or lack of effects within each group is compared. In order for any meaningful comparisons between the previous experiment presented in Chapter 6 and the current study to be drawn, the time windows and electrode clusters for analysis are replicated here. To reiterate in brief, we inspected a mid-frontal effect between 400-600ms, maximal at electrodes C1, C2

and Cz, and a parietal effect between 500-700ms, maximal at electrodes P1, P3 and P5. Planned comparisons of difference waves (*HH-CR* and *HM-CR*) were conducted for each group, with follow-up analysis of individual responses (*HH, HM, CR*) to support and clarify findings. All analysis was conducted using Jamovi version 1.6.23 (Jamovi, 2021; R Core Team, 2020).

Results

Behavioural Data Analysis

Independent t-tests of accuracy data between Control and MBO group were conducted for the *before-alcohol* condition, and then repeated for the *after-alcohol* condition. Paired t-tests carried out within groups showed reduced sensitivity (*d'*) *after-alcohol* for both the Control and MBO groups (*p*'s ≤ .005), and changes in *C* such that participant's responses in both groups became significantly more conservative (*p*'s ≤ .015) *after-alcohol*, that is, when uncertain about whether they had seen the stimulus before, they became more likely to respond no after drinking (see Table 7.2). Participants in the Control group significantly reduced their *HH* accuracy *after-alcohol* compared to *before-alcohol, t*(16) = 3.533, *p* = .003, n^2_p = .857, and were more likely to *Miss* an old word response *after-alcohol*, *t*(16) = - 3.93, *p* = .001, n^2_p = - .952. Further, source accuracy was also reduced *after-alcohol* in our controls, *t*(16) = 2.23, *p* = .04, n^2_p = .541. Like the Control group, the MBO group also significantly reduced their *HH* responses *after-alcohol*, *t*(15) = 2.442, *p* = .027, n^2_p = .611, and recorded more *Miss* responses *after-alcohol*, *t*(15) = - 4.966 *p* < .001, n^2_p = - 1.241. Additionally, the MBO group recorded fewer *HM* responses *after-alcohol*, *t*(15) = 3.519, *p* = .003, n^2_p = .88. In contrast to Controls, the MBO group showed no change in their source accuracy recall *after-alcohol* (*p* = .81). Mean accuracy (%) suggests slight differences between controls and our MBO group in terms of the pattern of *HH*, *Misses*, and *FA*, yet no significant differences between groups in any metric were observed (see Table 7.3).

Table 7.2:

Behavioural Accuracy Results: Within Groups

	Before Alcohol		After Alcohol		
Control (*n* = 17)	**Mean**	**SD**	**Mean**	**SD**	**Difference**
Hit Hit (%)	49.66	12.12	37.79	14.56	$t(16) = 3.53$, $p = .003$*
Hit Miss (%)	24.71	6.31	22.84	6.21	$t(16) = 1.63$, $p = .122$
Source Accuracy (%)	66.09	10.21	60.82	11.49	$t(16) = 2.23$, $p = .04$*
Correct Rejection (%)	84.66	12.86	84.69	12.52	$t(16) = 0.02$, $p = .986$
False Alarm (%)	15.34	12.86	15.39	12.52	$t(16) = -0.01$, $p = .986$
Miss (%)	25.64	8.27	39.36	14.59	$t(16) = -3.93$, $p = .001$*
d'	0.69	0.997	-0.23	1.31	$t(16) = 3.29$, $p = .005$*
C	-0.22	0.59	0.23	0.68	$t(16) = -2.72$, $p = .015$*
MBO (*n* = 16)					
Hit Hit (%)	44	13.79	35.9	13.91	$t(15) = 2.44$, $p = .027$*
Hit Miss (%)	26.9	6.67	21.3	6.28	$t(15) = 3.52$, $p = .003$*
Source Accuracy (%)	60.91	11.08	61.66	9.02	$t(15) = -0.24$, $p = .81$
Correct Rejection (%)	81.9	13.58	80.6	16.64	$t(15) = 0.81$, $p = .43$
False Alarm (%)	18.1	13.58	19.4	16.64	$t(15) = -0.81$, $p = .43$
Miss (%)	29.1	13.3	42.8	16.49	$t(15) = -4.97$, $p < .001$*
d'	0.26	1.09	-0.75	0.91	$t(15) = 9.27$, $p < .001$*
C	-0.21	0.77	0.20	1.07	$t(15) = -2.84$, $p = .012$*

Table 7.3:

Behavioural Accuracy Results: Between Groups

	Before Alcohol	After Alcohol
Hit Hit (%)	$t(31) = 1.25$, $p = .22$	$t(31) = 0.37$, $p = .711$
Hit Miss (%)	$t(31) = -0.98$, $p = .333$	$t(31) = 0.71$, $p = .484$
Source Accuracy (%)	$t(31) = 1.4$, $p = .17$	$t(31) = -0.23$, $p = .82$
Correct Rejection (%)	$t(31) = 0.6$, p .55	$t(31) = 0.79$, $p = .435$
False Alarm (%)	$t(31) = -0.6$, $p = .55$	$t(31) = -0.79$, $p = .435$
Miss (%)	$t(31) = -0.89$, $p = .378$	$t(31) = -0.63$, $p = .535$
d'	$t(31) = 1.19$, $p = .24$	$t(31) = 1.32$, $p = .198$
C	$t(31) = -0.06$, $p = .96$	$t(31) = 0.11$, $p = .92$

Event Related Potentials Analysis: Mid-frontal effect between 400-600ms

Figure 7.2A shows ERPs for the Control group averaged across electrode sites C1, C2 and Cz *before-alcohol*, while Figure 7.2B displays response difference waves for *HH-CR* and *HM-CR.* Confidence intervals surrounding response difference waves show no difference between *HH-CR* and *HM-CR* across the epoch. *After-alcohol*, Figure 7.2C shows response ERPs, and Figure 7.2D highlights difference waves; note the separation of confidence intervals *after-alcohol*, indicative of a significant difference later than the 400-600ms time window. Figure 7.3 denotes the same details for the MBO group; again confidence intervals highlight a significant period of difference within the 400-600ms time window at Figure 7.3B, which disappears in Figure 7.3D.

In the Control group, repeated measures ANOVAs with factors of alcohol condition (*before-alcohol, after-alcohol*), electrode (C1, C2, Cz), and response difference (*HH-CR*, *HM-CR*) found a main effect of response difference, $F(1,16) = 6.9$, $p = .018$, $n^2_p = .301$, such that the *HH-CR* difference was on average larger in amplitude than *HM-CR*. No main effect of alcohol condition ($p = .775$), or interaction between alcohol condition and response difference ($p = .566$), was found. Note that CI's showed differences above zero from 378 to 613ms in *HH-CR*, and 355 to 594ms (*HM-CR*) for *before-alcohol* conditions, while *after-alcohol* this window was extended for *HH-CR* (285 to 687ms), but slightly reduced from 395 to 580ms for *HM-CR* (see Figure 7.2B,D).

Between response differences, CI's highlighted a period of significant difference *after-alcohol* only, from 503 to 677ms, suggesting a graded pattern of ERP response with largest amplitudes for *HH*, then *HM* followed by *CR after-alcohol*.

A further repeated measures ANOVA, this time with factors of individual response types (*HH*, *HM*, *CR*), electrodes (C1, C2, Cz), and alcohol conditions (*before-alcohol, after-alcohol*) found a main effect of alcohol, $F(1,16) = 12.78$, $p = .003$, $n^2_p = .444$, and a main effect of response type, $F(2,32) = 16.13$, $p < .001$, $n^2_p = .502$. Again, there was no interaction between factors ($p = .8$). Bonferroni corrected post-hoc tests revealed an overall reduction in amplitude for responses *after-alcohol*, $t(16) = 3.58$, $p = .003$, and a significant reduction in mean amplitude for *CR* responses between *before-alcohol* ($M = 1.152$, $SE = 0.431$) and *after-alcohol* ($M = -0.207$, $SE = 0.444$), $t(16) = 5.201$, $p = .001$. Additionally, *before-alcohol*, *HH* responses were significantly greater in mean amplitude than *CR* responses, $t(16) = 3.642$, $p = .033$, and this pattern was repeated *after-alcohol*, $t(16) = 5.231$, $p = .001$.

Since we planned to investigate changes in the presence of effects *before-alcohol* and *after-alcohol*, we further separated our analyses by alcohol condition. This also allows clearer comparison with the previously reported experiment in Chapter 6. Following this approach, two repeated measures ANOVA models with factors of response difference (*HH-CR, HM-CR*) and electrode (C1, C2, Cz) were conducted on *before-alcohol* and *after-alcohol* conditions separately. There were no significant effects in the *before-alcohol* model, however there was a significant effect of response difference *after-alcohol*, $F(1,16) = 6.56$, $p = .021$, driven by the differences in *HH-CR* being significantly greater than differences between *HM-CR*. This is supported separately by our CI analysis, which highlighted a separation extending beyond the epoch (285 to 687ms for *HH-CR*).

Next, a repeated measures ANOVA with factors of response (*HH, HM, CR*) and electrode site (C1, C2, Cz) was conducted on *before-alcohol* and *after-alcohol* conditions separately. We found a main effect of response type *before-alcohol*, $F(2,32) = 7.377$, $p = .002$, $n^2_p = .316$. Bonferroni corrected post-hocs showed a graded response pattern, with no significant difference between *HH* ($M = 2.52$, $SE = 0.618$) and *HM* ($p = .396$, $M = 1.98$, $SE = 0.594$), nor between *HM* and *CR* ($p = .106$, $M = 1.15$, $SE = 0.431$). However, *HH* did show a significantly greater mean amplitude than *CR*, $t(16) = 3.64$, p

= .007. Similarly, *after-alcohol* there was a main effect of response type, $F(2,32) = 15.26$, $p < .001$, $n^2_p = .488$. This again reflected a significant difference between *HH* and *CR* responses, $t(16) = 5.23$, $p < .001$, but critically, also between *HM* and *CR* responses, $t(16) = 3.22$, $p = .016$. The difference between *HH* and *HM* did not reach significance ($p = .063$).

MBO Group

We next analysed MBO group differences using a repeated measures ANOVA with factors of condition (*before-alcohol*, *after-alcohol*), electrode (C1, C2, Cz), and response difference (*HH-CR*, *HM-CR*). There were no main effects, or interactions, between alcohol conditions. A further ANOVA with factors of response type (*HH*, *HM*, *CR*), electrode (C1, C2, Cz), and condition (*before-alcohol*, *after-alcohol*) revealed a main effect of condition, $F(1,15) = 13.59$, $p = .002$, $n^2_p = .475$, and a main effect of response type, $F(2,30) = 3.66$, $p = .038$, $n^2_p = .196$, but no interaction between factors ($p = .261$). Like the Control group, Bonferroni corrected post-hoc tests showed a significant reduction in mean amplitude for *CR* responses between *before-alcohol* ($M = 1.321$, $SE = 0.39$) and *after-alcohol* ($M = 0.238$, $SE = 0.433$, $t(15) = 5.9$, $p = < .001$). There were no other relevant significant differences, although *HH* responses *before-alcohol* ($M = 2.259$, $SE = 0.571$) and *after-alcohol* ($M = 1.03$, $SE = 0.605$) trended towards a significant difference ($p = .067$).

Again, the data was separated into before and after alcohol conditions, but we failed to find significant differences between responses in either the *before-alcohol* or *after-alcohol* conditions. However, CI analysis suggested differences above zero, that is, differences from the CR condition, were only reliably present *before-alcohol* for *HH-CR* (306 to 595ms). After-alcohol, *HH-CR* (184 to 665 ms) and *HM-CR* (90-680ms) showed large differences from zero for sustained periods, highlighting the change in *CR* response (see Figure 7.3B,D). Comparing across conditions, the difference between *HH-CR* and *HM-CR* was significant from 488 to 663ms for *before-alcohol* conditions only.

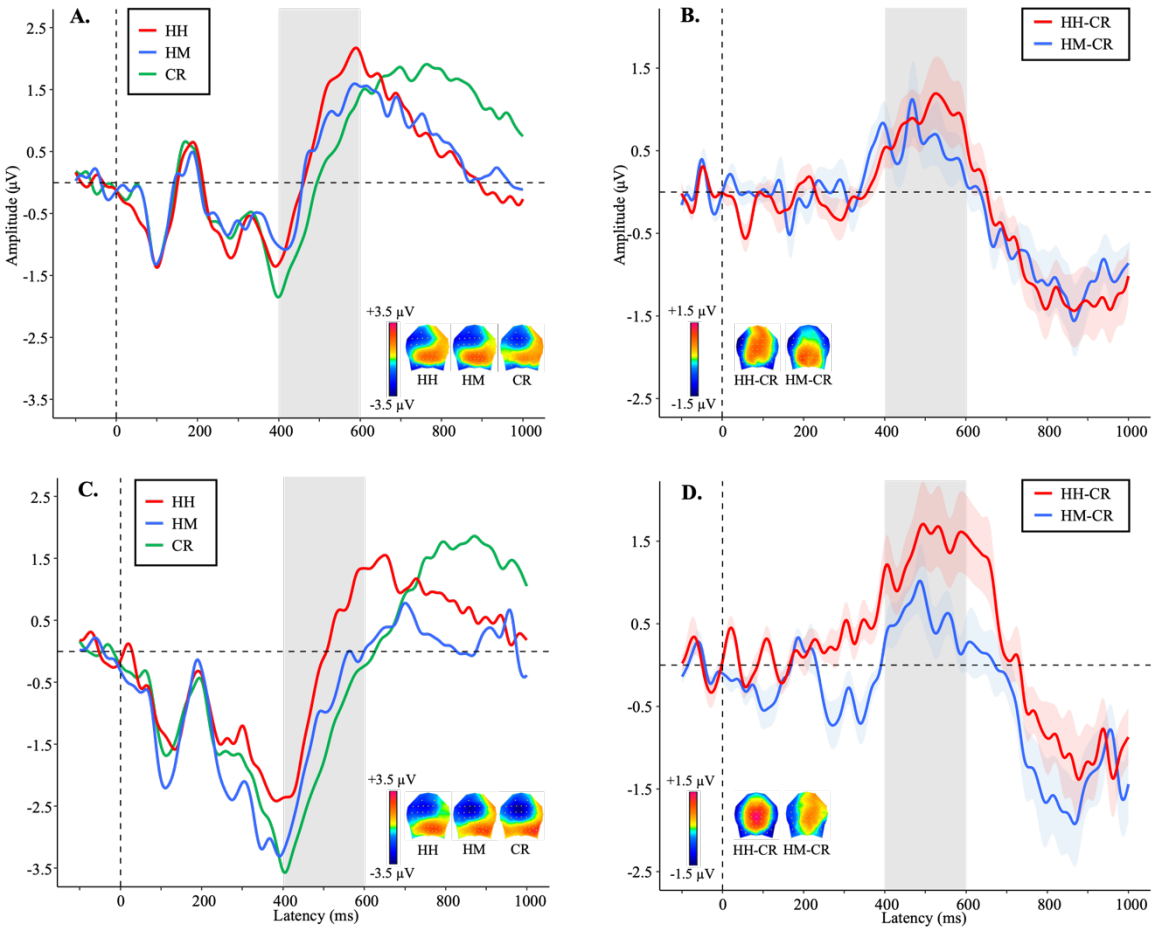

Figure 7.2: **Electrophysiological responses and difference waves both before and after alcohol in the Control group**. (A) and (C) display averages of electrode sites C1, C2 and Cz, for ERP responses, with *before-alcohol* shown in (A), and *after-alcohol* in (C). Insets show topographic maps of individual response (nasion at top) and grey shaded areas represent the 400-600ms time window used in analysis. (B) and (D) show difference waves of *HH-CR* and *HM-CR* responses, with confidence intervals highlighted surrounding the wave forms and difference topographies representing *HH-CR* and *HM-CR*. (B) represents differences *before-alcohol,* with (D) *after-alcohol*.

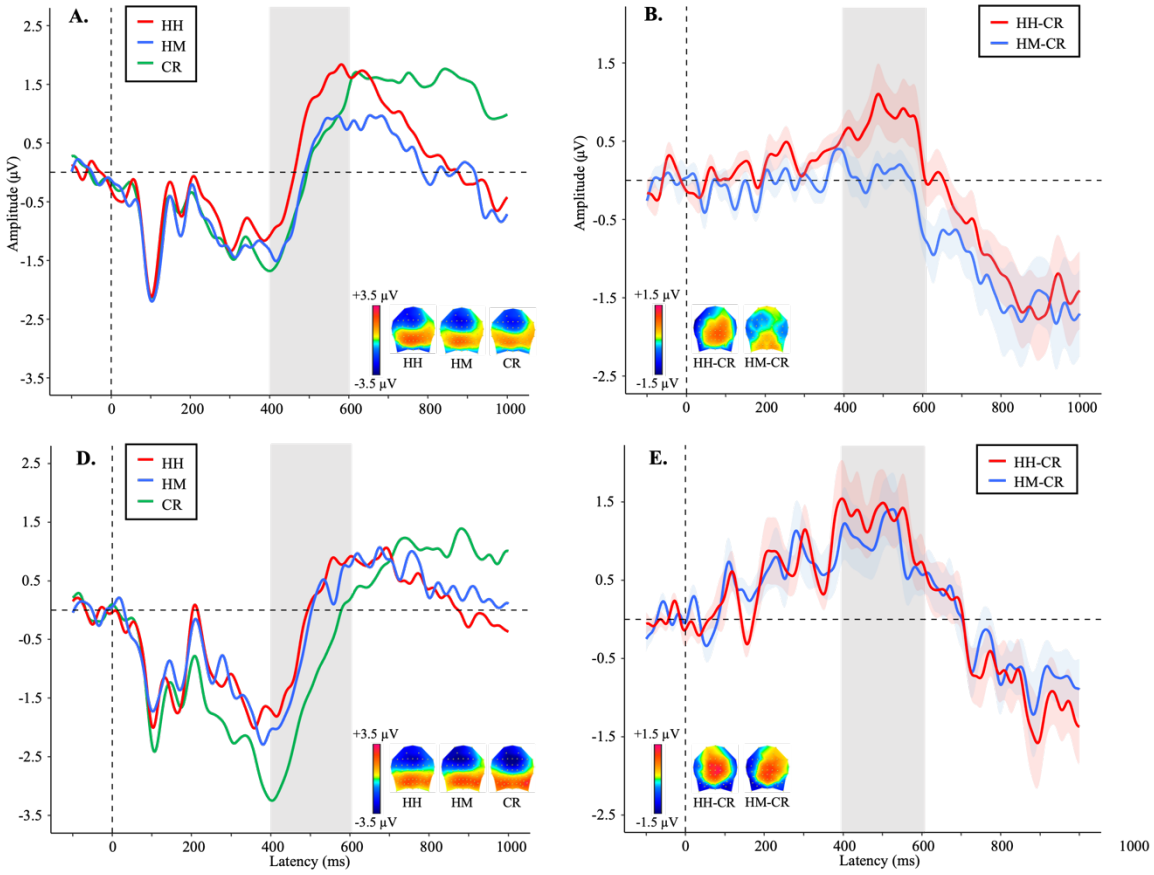

Figure 7.3: **Electrophysiological responses and difference waves, before and after alcohol, in the MBO group.** (A) and (C) display averages of electrode sites C1, C2 and Cz for ERP responses, with *before-alcohol* shown in (A), and *after-alcohol* in (C). Grey shaded areas represent the 400-600ms time-window used for analysis and response topographies depicting *HH*, *HM* and *CR* are embedded (nasion at the top of the maps). (B) and (D) display difference waves and difference topographies (*HH-CR, HM-CR*) both *before-alcohol* (B), and *after-alcohol* (D).

Parietal effect between 500-700ms

To study the impact of alcohol on the parietal effect, we conducted a repeated measures ANOVA on difference wave data with factors of alcohol condition (*before-alcohol*, *after-alcohol*) and response difference (*HH-CR*, *HM-CR*), and electrode (P1, P3, and P5) on mean amplitudes separately for each group. Firstly, the Control group showed no main effect of alcohol condition, $F(1,16) = 0.147$, $p = .706$, $n^2_p = .009$. Also, no main effect of response difference could be found, $F(1,16) = 0.364$, $p < .555$, $n^2_p = .022$. Critically, we found an interaction between both factors, $F(1,16) = 9.764$, $p = .007$, $n^2_p = .379$, suggesting that while *before-alcohol* no differences between *HM-CR* and *HH-*

CR in the strength of the effect were observed, *after-alcohol* only the *HH* signal was different from the *CR* signal. Confidence Interval analysis supported these findings, with no separation between *HH-CR* and *HM-CR* across the epoch *before-alcohol,* but short periods of difference were present in the *after-alcohol* condition (603-636ms, 670-698ms), likely driven by noise in the signal. Differences above zero were visible in the *before-alcohol* condition for *HH-CR* (518-673ms) and *HM-CR* (242-866ms) showing sustained periods where mean amplitude for both *HH* and *HM* was greater than for *CR* (see Figure 7.4B). However, *after-alcohol*, differences from zero were shorter, with *HH-CR* being different from 510-642ms, and also from 665-1000ms, and *HM-CR* from 570-589, and 689–728ms, reflecting the change predominantly in the *HM* signal.

Next, repeated measures ANOVAs with factors of response type (*HH*, *HM*, *CR*), electrode (P1, P3, P5), and alcohol condition (*before-alcohol*, *after-alcohol*) were conducted, again on each group separately. The Control group showed a main effect of response type, $F(2,32) = 12.24$, $p < .001$, $n^2_p = .433$, and an interaction between response and condition, $F(2,32) = 4.61$, $p = .017$, $n^2_p = .224$. Bonferroni corrected post-hocs showed mean amplitude for *HH* responses ($M = 2.228$, $SE = 0.523$) was significantly greater than *CR* responses *after-alcohol* ($M = 0.587$, $SE = 0.352$; $t(16) = 4.315$, $p = .008$).

Following the planned strategy of analysing *before-alcohol* and *after-alcohol* data separately, with factors of response difference (HH-CR, HM-CR) and electrode (C1, C2, Cz), we found no difference *before-alcohol*, and a trend towards a significant difference *after-alcohol* ($p = .053$). Two further repeated measures ANOVAs with response type (*HH, HM, CR*) and electrode sites as factors showed a main effect of response *before-alcohol*, $F(2,32) = 8.02$, $p = .001$, $n^2_p = .334$. Bonferroni corrected post-hoc tests showed no difference between *HH* and *HM* responses ($p = .217$), a trend towards a significant difference between *HH* and *CR* ($p = .058$). However, *HM* was significantly greater than *CR*, $t(16) = 3.23$, $p = .016$. In the *after-alcohol* condition, there was again a main effect of response, $F(2,32) = 10.57$, $p < .001$, $n^2_p = .398$. Like *before-alcohol*, this reflected a difference between *HM* ($M = 1.433$, $SE = .515$) and *CR* ($M = .587$, $SE = .352$), $t(16) = 2.79$, $p = .039$, but also a difference between *HH* ($M = 2.228$, $SE = .523$) and *CR, t*(16) = 4.32, $p = .002$. Again, there was no difference between *HH* and *HM* ($p = .161$).

MBO Group

In the MBO group, a repeated measures ANOVA with factors of alcohol condition, electrode and response differences found no main effects of response difference (p = .597), or alcohol condition (p = .697), and no interaction between factors (p = .129). Next, we analysed response types (*HH, HM, CR*) and alcohol condition (*before-alcohol, after-alcohol*), however a main effect of response, $F(2,30) = 5.75$, p = .008, n^2_p = .277, was found at post-hocs to reflect a meaningless difference between *HH* responses *before-alcohol*, and *CR after-alcohol* (p = .05). No other differences between alcohol conditions were present.

When treating each alcohol condition separately, the MBO group also did not differ meaningfully in any comparison. ANOVA showed no differences in mean amplitude between *HH-CR* and *HM-CR* either *before-alcohol* (p = .258), or *after-alcohol* (p = .721). Two final repeated measures ANOVAs also showed no main effects of response type *before-alcohol* (p = .107). However, in the *after-alcohol* condition, there was a main effect of response, $F(2,30) = 4.52$, p = .019, n^2_p = .232. This difference appeared to be driven by a trend towards significance between *HH* and *CR*, $t(15) = 2.664$, p = .053, with no significant differences between *HH* and *HM* (p = 1), nor between *HM* and *CR* (p = .099).

Confidence Interval analysis confirmed the lack of difference between *HH-CR* and *HM-CR* across the 500-700ms time-window in both *before-alcohol* and *after-alcohol* conditions. However, note that continuous differences from zero were present *before-alcohol* for *HH-CR* (513-826ms), whereas the difference between *HM* and *CR* occurred for shorter periods (534-581ms, 635-843ms). After alcohol, this pattern reversed with a more sustained difference present for *HM-CR* from 519-953ms, whereas differences between *HH* and *CR* were more variable (463-542ms, 574-583ms, 604-919ms).

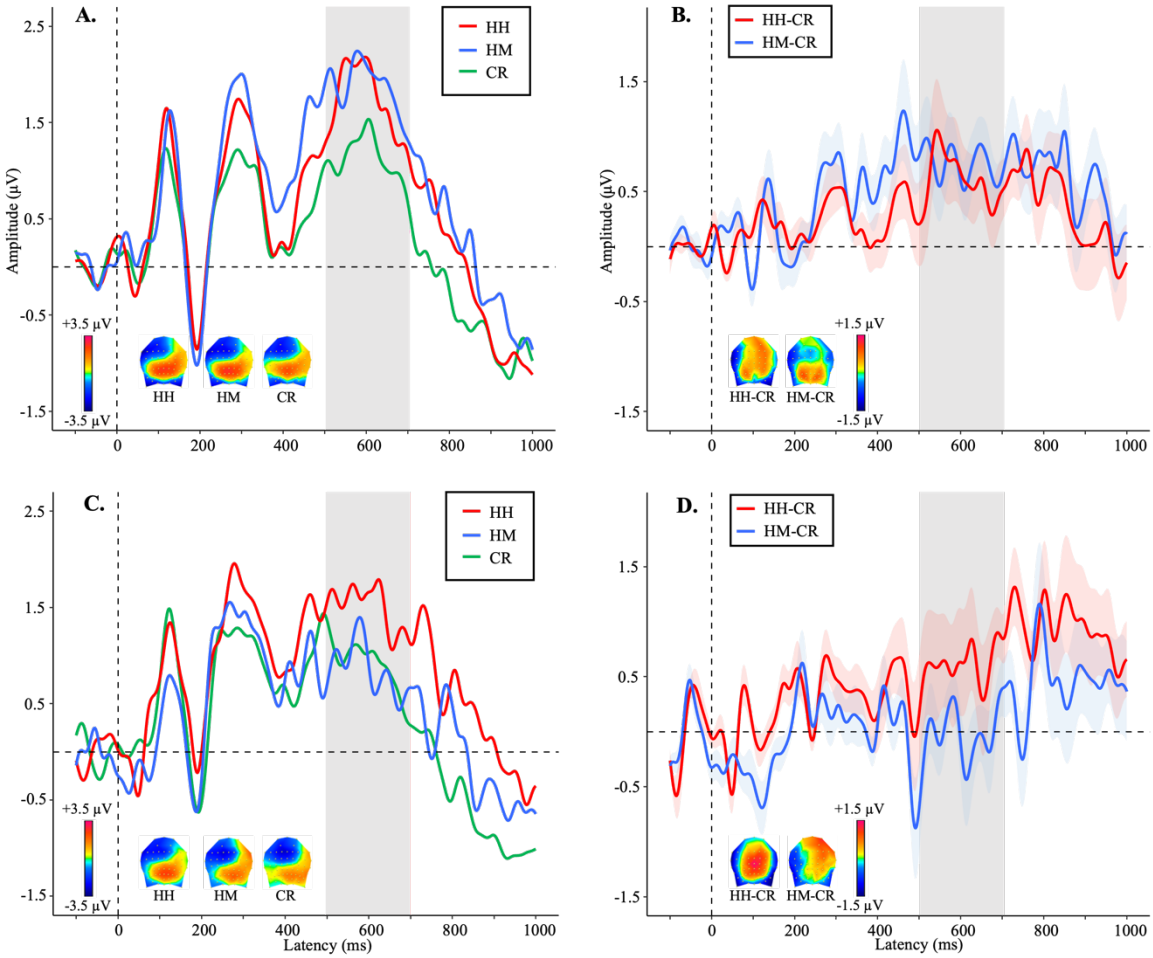

Figure 7.4: **Electrophysiological responses and difference waves, before and after alcohol, in the Control group.** (A) and (C) display averages of electrode sites P1, P3 and P5 for ERP responses, with *before-alcohol* shown in (A), and *after-alcohol* in (C). Grey shaded areas represent the 500-700ms time-window used for analysis, and topographic maps highlight *HH*, *HM* and *CR* responses (nasion at the top of the maps). (B) and (D) display difference waves both *before-alcohol* (B), and *after-alcohol* (D). Insets show difference topographies (*HH-CR, HM-CR*).

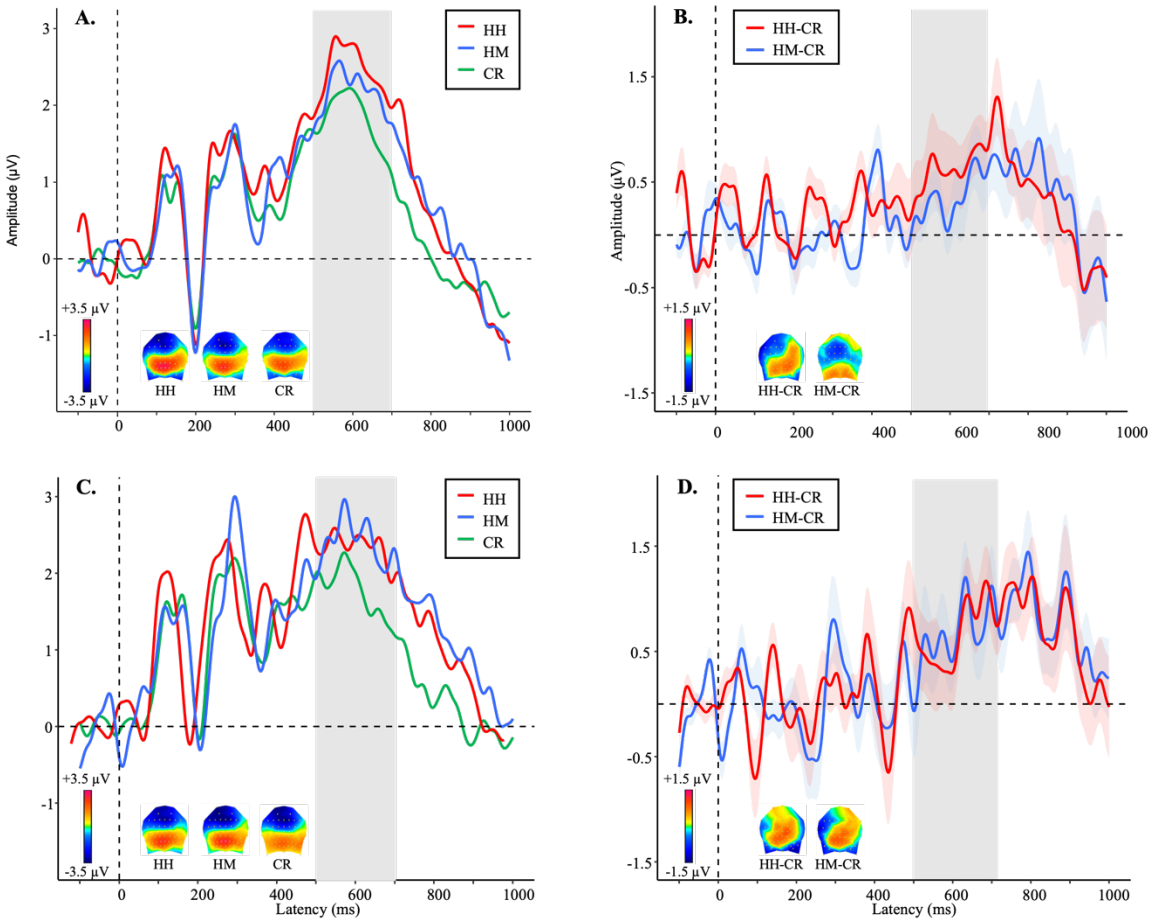

Figure 7.5: **Electrophysiological responses and difference waves, before and after alcohol, in the MBO group.** (A) and (C) display averages of electrode sites P1, P3 and P5 for ERP responses, with *before-alcohol* shown in (A), and *after-alcohol* in (C). Grey shaded areas represent the 500-700ms time-window used for analysis and insets highlighting topographic response maps (HH, HM, CR). (B) and (D) display difference waves both *before-alcohol* (B), and *after-alcohol* (D). Embedded difference topographies show *HH-CR*, and *HM-CR*, nasion at the top of the maps.

Discussion of Study One

Consistent with the findings reported in Chapter 6, analysis of behavioural results showed no difference in accuracy performance between groups *before-alcohol.* There were also no group differences in the *after-alcohol* condition. However, within group analysis showed that both Control and MBO participants became less accurate in their responses *after-alcohol*. The Control group recorded more *HH* responses in general than the MBO group and were less likely to record a *HM* response in either

condition in comparison to the experimental group. Both groups showed a drop in *d' after-alcohol*, and an increase in *C* which means groups were less able to discriminate between old and new items after alcohol, and also that they became more conservative in their response bias.

In the 400-600ms time-window, the Control group ERP results in the *before-alcohol* condition mirror those seen in Chapter 6 with no difference between *HH-CR* and *HM-CR* response difference, and a separation between *HH* and *CR.* Unlike the previous chapter, there was no overall statistical difference between and *HM* and *CR* responses, although analysis of Confidence Intervals suggested that differences were present at points across the time-window. In contrast, the MBO group deviated from the previous findings with no statistical separation between *HH-CR* and *HM-CR*, and also no statistical difference between individual response types. Despite this, visual inspection of waveforms and Confidence Interval analysis suggested differences were present for over half of the time-window, and also that the *HM* signal tracked closer to *CR* than to *HH before-alcohol*, consistent with the experiment in Chapter 6. The lack of statistical evidence for replication within the MBO group was surprising when considering the observed pattern and may reflect fewer trial numbers and participants, coupled with greater individual variability.

After alcohol, the Control group appear to alter strategy, with *HH* and *HM* response ERPs both significantly differing from *CR,* in contrast to *before-alcohol*. This could reflect a reduction in certainty for *HH* responses, reducing mean amplitude to mirror that of the *HM* signal. To be clear, certainty is a synonym for confidence, however we did not measure confidence. We note that being confident in making a decision is not the same as making an accurate decision. One interesting point to note was that in the *after-alcohol* condition, the ERP pattern displayed by the Control group (see Figure 7.4D) was visually similar to that seen in the MBO group while sober in Chapter 6 (see Figure 6.3B). Within the MBO group in the present study, there are fewer differences between the two alcohol conditions, however they also show a sustained difference from zero for *HH-CR* and *HM-CR*, like the Control group and which was not present *before-alcohol.* Despite this similarity, difference waves for the MBO group *after-alcohol* overlapped, suggesting that *HH* and *HM* responses were equally different to *CR*, unlike the graded pattern seen in the Control group.

Across the parietal region, the Control group response difference wave pattern *before-alcohol* again mirrored the findings in Chapter 6, with no significant difference between *HH-CR* and *HM-CR*. However, unlike the previous chapter, this time only *HM* mean amplitude was greater than *CR*. Confidence Intervals (CI) offer a more detailed picture, with differences above zero also present for *HH-CR* across a sustained period, which is more consistent with findings in Chapter 6. In contrast, the MBO group *before-alcohol* differed from the previous study with no difference between *HH-CR* and *HM-CR* visible statistically, or in CI analysis. However, CI differences from zero again suggested a pattern of responses similar to that seen in the last chapter, where *HH* was significantly greater than *CR*, and *HM* was not consistently different to *CR*. In the *after-alcohol* condition, visually the MBO group ERPs show an overlap between *HH* and *HM* which could reflect a deliberate focusing on the task by the more experienced alcohol drinkers, a phenomenon known as alcohol myopia (Steele & Josephs, 1990). On the other hand, ERPs suggest the opposite pattern in the Control group, with the *HM* response ERP tracking *CR* across much of the 500-700ms time-window, similar to the visual – but not statistical - pattern seen in the MBO group in the previous study.

In terms of dual process theory, these findings suggest that *before-alcohol*, the two groups employed both the processes of familiarity and recollection when judging whether a stimulus was old or new. However, *after-alcohol,* the MBO group altered strategy placing a greater reliance on recollection than on familiarity when making a decision, whereas Control participants appeared to make accurate familiarity-based judgements for *HM* stimuli without recollection of having previously seen the stimulus.

To summarise succinctly, in both groups' ERPs changed after ingesting alcohol, yet both groups also appeared to show different patterns of activity despite no differences in behavioural performance. This perhaps suggests that frequent blackout experiences could change neural correlates of memory, however, further replication of these effects are needed, especially given the loss of trial numbers after drinking alcohol.

Study Two

Materials and Methods

Design

Participants from the MBO group in Study 1 were invited to return to the laboratory following an alcohol-induced memory blackout (<20 hours) and to repeat the study while sober, but still recovering from the blackout event. In total, 21 participants returned to complete the study, however one participant was excluded due to recording fewer than 16 trials in relevant response categories, and another because they did not complete the task correctly. Therefore, data from 19 participants is presented here (7 males, mean age = 19.26, *SD* = 1.1). Participants in the MBO group who completed Study 2 received a further £22.50. Study 2 was approved by the General University Ethics Panel, and the NHS, Invasive and Clinical Research committee at the University of Stirling.

Procedure

Participants were asked to informally notify the researchers if they either had plans to attend a drinking event which was likely to result in a blackout, or if they found themselves in that situation spontaneously. To be clear, *no participants* were asked to binge-drink for the purpose of this experiment. Follow-up visits were initiated by the participants and entirely voluntary. All *after-MBO* sessions took place in the afternoon to allow time for participants to rest and recover from their drinking event. Upon arrival at the laboratory, participants were breathalysed and only completed the study if BrAC readings were 0.00mg/l. Participants were informally asked for details of their drinking event, including when they started and stopped drinking, and the duration and quality of sleep. All MBO participants advised that they had experienced a blackout.

The recognition memory with source judgement experiment detailed above in Study 1 (see Figure 7.1) was repeated, however this time participants completed all 8 blocks (480 trials) sober. Again, scalp activity was recorded using SynAmps^2 (Neuroscan, Inc., El Paso, TX, USA) amplifiers and all EEG recording protocols were as previously outlined.

Statistical Analysis

As before, behavioural performance was recorded as percentages of *Hit-Hit, Hit-Miss, Correct-Rejection, False Alarm* and *Source* mean accuracy. Additionally, we again report *d'* (discrimination between old and new items) and *C* (response bias). ERP data analysis followed the strategy previously outlined, with difference waves (*HH-CR* and *HM-CR*) and responses (*HH, HM, CR*) compared using ANOVAs, over 400-600ms at electrodes C1, C2, and Cz, and between 500-700ms at P1, P3, and P5.

Data from individual MBO participants who completed both Study 1 and 2 was inspected to determine whether analysis of the same participants across all three conditions (*before-alcohol*, *after-alcohol*, *after-blackout*) could reasonably be analysed together. There were 12 participants who recorded sufficient trial numbers in each relevant condition, which was considered insufficient to produce a meaningful analysis. Further, since the *before-alcohol* and *after-alcohol* conditions each had 240 trials whereas the *after-MBO* study had 480, it was decided that this analysis would lack power. It is however included in Appendix 4.1for completeness.

Results

Behavioural Data

Behavioural accuracy is presented in Table 7.4.

Table 7.4:

Behavioural Accuracy (n = 19)

	Mean	**SD**
Hit-Hit (%)	41.8	14.34
Hit-Miss (%)	20.75	7.85
Source Accuracy (%)	65.99	9.59
Correct Rejection (%)	81.38	18.05
False Alarm (%)	18.62	18.05
Miss (%)	37.46	17.67

Event Related Potentials: Mid-Frontal effect between 400-600ms

Figure 7.6A shows ERP mean amplitudes for responses across clustered electrode sites (C1, C2, Cz), and Figure 7.6B shows corresponding difference waves for *HH-CR* and *HM-CR*. Confidence Interval analysis showed short periods of separation between response differences (444-450ms, 474-493ms, 517-547ms, 583-609ms; see Figure 7.6B), however repeated measures ANOVA analysis, with factors of response difference *HH-CR* (M = 0.628, SE = 0.221) and *HM-CR* (M = 0.377, SE = 0.199), and electrode site (C1, C2, Cz), was not significant (p = .263), suggesting that separation between the two was not sustained across the epoch. A further repeated measures ANOVA with factors of response type (*HH, HM, CR*) and electrode site (C1, C2, Cz) revealed a main effect of response, $F(2,36) = 4.4$, $p = .019$, $n^2_p = .197$, which was driven by a difference in mean amplitude between *HH* (M = -1.55, SE = 0.489) and *CR* (M = -2.17, SE = 0.475), $t(18) = 2.83$, $p = .033$. There were no significant differences in mean amplitude between *HH* and *HM* (M = -1.8, SE = 0.475), nor between *HM* and *CR*, suggesting a graded pattern of response. This was supported by CI analysis which found a sustained period where differences between *HH* and *CR* were above zero (317-661ms). Differences between *HM* and *CR* were present between 381-455ms, and again from 465-589ms, suggestive of a graded pattern of mean amplitude between responses.

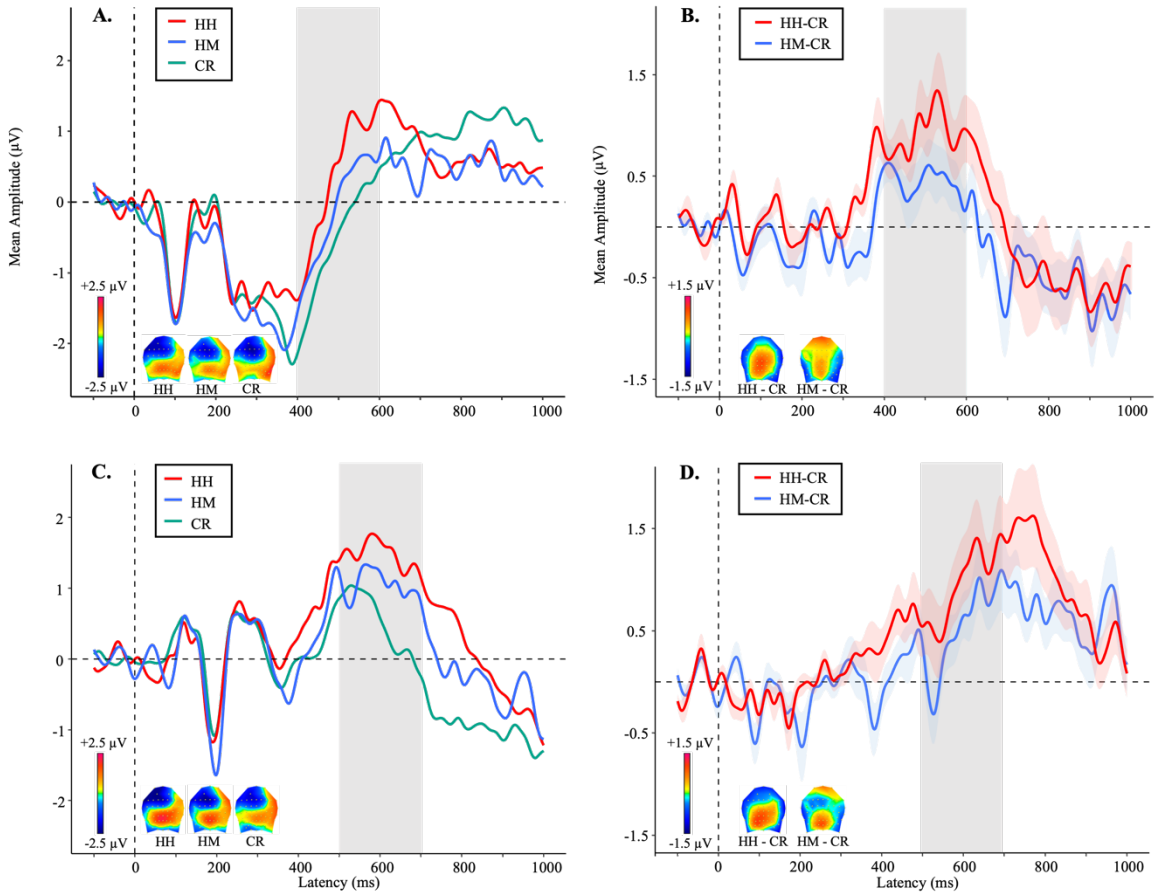

Figure 7.6: **Electrophysiological responses and difference waves, after alcohol-induced blackout in the MBO group.** (A) displays an average of electrode sites C1, C2 and Cz for ERP responses *HH, HM* and *CR*. The grey shaded area highlights the time-window 400-600ms used for analysis. (C) also displays ERP responses, but across an average of electrode sites P1, P3 and P5 and with the grey shaded region representing the 500-700ms time-window. (B) displays difference waves of *HH-CR* and *HM-CR*, averaged over C1, C2 and Cz, and with the 400-600ms time-window highlighted in grey. (D) displays difference waves from averaged electrode sites P1, P3 and P5, and highlights the 500-700ms time window. Confidence intervals are shaded to match difference waves in both (B) and (D). Topographic maps (nasion at top) in (A) and (C) show responses (*HH, HM, CR*), while (B) and (D) highlight difference topographies (*HH-CR, HM-CR*).

Parietal effect between 500-700ms

Figures 7.6C and 7.6D show average ERPs and difference waves across left parietal electrode sites P1, P3 and P5 with the 500-700ms time-period of interest highlighted. A repeated measures ANOVA with factors of response difference waves (*HH-CR* vs *HM-CR*) and electrode site (P1, P3, P5) did not find any significant difference

between response differences (p = .399), however a short period of separation between Confidence Intervals is visible from 510-534ms. A repeated measures ANOVA with factors of response type (*HH, HM, CR*) and electrode (P1, P3, P5) also found no main effect of difference between responses (p = .075), however post-hocs showed a difference between *HH* (M = 0.862, SE = 0.403) and *CR* (M = 0.23, SE = 0.381), t(18) = 2.702, p = 0.04. There was no difference between *HH* and *HM* (M = 0.642, SE = 0.359, p = 1), nor between *HM* and *CR* (p = 0.643). The CI analysis showed a stable time period (390-924ms) where the *HH-CR* difference was greater than zero, supporting the significant difference previously reported between the two responses. Note that differences between *HM-CR* were also present from 551-900ms suggesting that *HH* and *HM* mean amplitude were both different to *CR* for much of the 500-700ms epoch, and that these ERP responses are delayed in comparison to standard time windows of analysis of these effects.

Discussion of Study Two

To summarise the findings from study two, the mid-frontal familiarity effect showed differences between *HH* and *CR* conditions, but no differences between *HM* and *HH* or *CR*. Later, in the parietal region and time window, this finding was also replicated. Moreover, our CI analysis highlighted extended periods of differences between *HH* and *CR* and *HM* and *CR,* lasting almost until the end of the epoch (*HH* compared to *CR*, 390 to 924ms; *HM* to *CR*, 551-900ms). It is noteworthy that after experiencing an alcohol-induced MBO, neural correlates of memory appear to be significantly delayed beyond the traditionally analysed time window of the left-parietal recollection effect (500-800ms).

General Discussion

The studies presented in this chapter were designed to address three aims. Firstly, to compare behavioural and neural correlates of memory performance within a Control group to our MBO group while sober and after drinking alcohol. This also provides a replication of the study outlined in Chapter 6. Secondly, to consider whether individuals who reported experiencing frequent blackouts were more susceptible to the effects of alcohol than those who drank rarely, if at all. We did not find any evidence in support of this aim, or vice versa. Finally, to investigate any acute impact to episodic memory performance following an MBO experience, behaviourally or at the neural level. In brief, while we found changes in behavioural performance within groups before and *after-alcohol*, between groups did not differ while sober in either Chapter 6, or Study 1, supporting our first aim, nor *after-alcohol*. Note that a reduction in source recall accuracy *after-alcohol* was found in the Control group, but not the MBO group. Control group ERP data in Study 1 also reflected findings in Chapter 6, and similar patterns were observed between MBO group ERPs in the two sober studies (Chapter 6 and Study 1) for the mid-frontal effect, but not the parietal effect. For the parietal effect, the *HM* response did significantly differ from *CR's* for brief periods of time (534-581ms; 635-843ms) in Study 1 here.

To assess the acute impact of a blackout event on episodic memory performance, we can now compare data from three studies assessing source memory accuracy with ERPs. In relation to the ERP data however, we can only discuss the presence or absence of effects within each study - which we will come to in due course - since any statistical differences found between groups may just be a result of differing absolute amplitudes. We can though compare behavioural performance across studies. This is especially interesting in the case of the MBO participants, who completed the study when sober (Chapter 6), when sober and *after-alcohol* in the same session (Study 1 here), and also the next day after experiencing an MBO (Study 2 here). Treating these groups as independent samples, we note that behavioural performance, measured as the total percentage recognition of *HH* and *HM*, does not change when sober yet drops after consuming alcohol compared to sober. Critically, performance also drops compared to sober after experiencing a blackout (see Table 7.5, and Figure 7.7), with

no differences between *after-alcohol* and *after-MBO* conditions. This data reproduces our findings from Chapters 4 and 5, suggesting that blackout events impact measurable behavioural indicators of memory performance (recall and recognition).

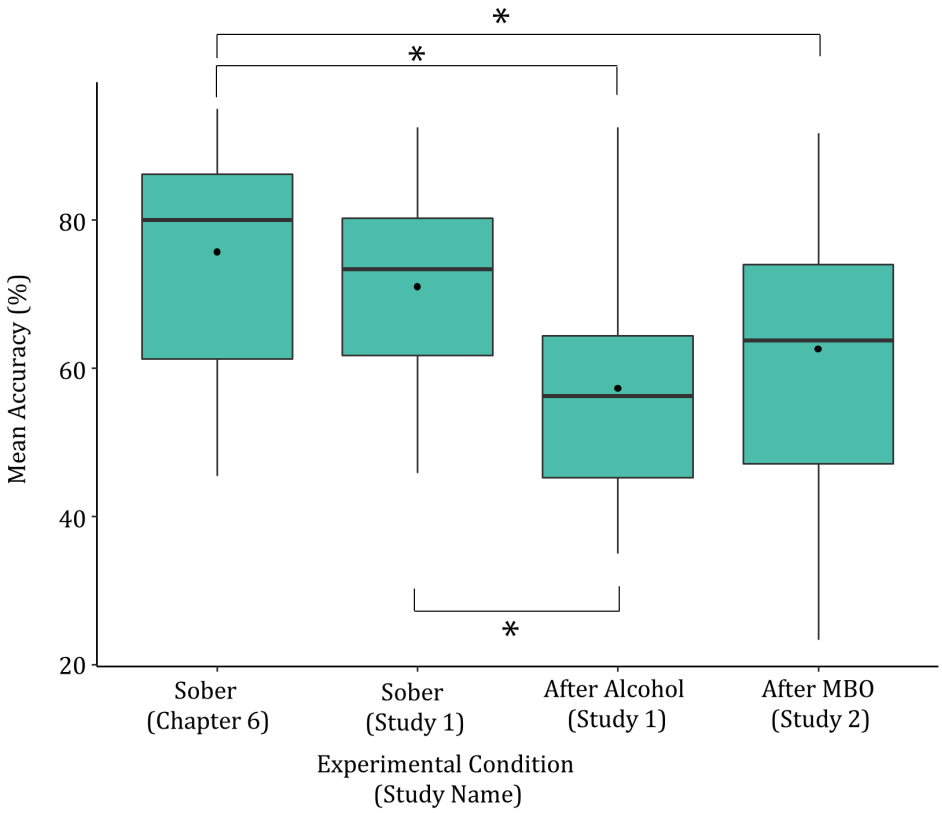

Figure 7.7: **Behavioural accuracy across studies.** Boxplots showing median and mean accuracy (black dots), calculated as percentage of combined *HH & HM* scores, shown for MBO group participants across 4 studies (two sober, one *after-alcohol*, and one *after-MBO*). Statistical differences between studies denoted by *. Note that the MBO groups differed per study in terms of size and makeup of each sample.

Table 7.5:

Comparison of HH and HM Accuracy (Mean %) in MBO participants

Conditions	Comparison	Mean Difference
Sober (Chapter 6) – Sober (Study 1)	$t(33.47) = 1.001$, $p = .324$, $r = 0.17$	4.6875
Sober (Chapter 6) – After Alcohol (Study 1)	$t(30.484) = 3.482$, $p = 0.015$*, $r = 0.533$	18.385
Sober (Chapter 6) – Sober (Study 2)	$t(35.132) = 2.503$, $p = 0.017$*, $r = 0.389$	13.081
After Alcohol (Study 1) – Sober (Study 2)	$t(32.618) = -0.917$, $p = 0.366$, $r = 1$	-5.30428

In more detail, behavioural results in this Chapter mirrored the results seen in our previously reported recognition study (Chapter 5), where both groups showed a reduction in their ability to discriminate between old and new items *after-alcohol*, and an increase in conservative response bias reflecting an unwillingness to risk an incorrect answer. While performance did not differ either before or *after-alcohol* between groups, some variations within groups were found, with significant reductions for MBO participants in *HM* responses, and Control participants in source accuracy, suggesting that groups may have differed in the cognitive strategies employed to complete the task. An increase in *C after-alcohol* is consistent with wider research (Maylor et al., 1987; Mintzer & Griffiths, 2001), and explained by the lack of difference in false alarms between alcohol conditions. Alcohol myopia theory suggests that *after-alcohol*, available cognitive resources, and specifically attention, are reduced leading to an intentional focus on the stimuli most salient at any given time (Steele & Josephs, 1990). Before alcohol, it is likely that participants will attend to a laboratory task with some reasonable care, however they may employ additional focus *after-alcohol* in a bid to maintain accuracy which prevents reductions in performance and increases conservative responses.

Why do the results from Study 1 not closely replicate the findings of Chapter 6? The decision was taken to half the trial numbers in this study into separate *before-alcohol* and *after-alcohol* conditions, in order to reduce the time burden on participants. Data from 62 individuals was collected to ensure reasonably sized groups and mitigate for any reduction in power. However, this did not take into account the effects alcohol would have on participants. Almost half of participants did not reach the designated threshold of 16 trials per response type *after-alcohol* and were excluded from analysis in both the alcohol conditions. Even though we predicted similarities, it was still surprising that the Control group results in the *before-alcohol* condition did mirror those found in Chapter 6, with visible differences *after-alcohol*. Likewise, while not reaching significance, MBO group difference waves in the *before-alcohol* condition were visually similar to those seen in Chapter 6. These findings broadly support evidence which suggests that even when sober, and behavioural performance is similar (Hartzler & Fromme, 2003), the two groups may diverge in memory strategies. It is important to keep in mind how the response profile changes in Study 1 due to the ingestion of alcohol, and therefore how this subsequently affects the recorded ERPs, all of which are a consequence of these changes. *After-alcohol* all participants became more uncertain, and conservative in their response, and therefore only likely to hit if they were certain. For the MBO group specifically, it appears they did this regardless of source information. Hence, it could be that the ERPs of this group reflect a lack of any other memory process but recollection after alcohol.

Employing confidence interval analysis can somewhat mitigate the loss in statistical power caused by too few trials. CI analysis considers differences across the epoch of interest at each millisecond, rather than using data averaged over a specific time window, which therefore can objectively identify differences at a more granular level. While we are not suggesting that this approach is a substitute for traditional ERP analysis, it does provide more clarity with challenging datasets. Using this approach, the 400-600ms *before-alcohol* period replicates the pattern reported in Chapter 6. The pattern again replicates for Controls in the 500-700ms window across parietal sites, but this time differs for MBOs. In Chapter 6, the experimental group showed a separation between response differences (*HH-CR, HM-CR*) which was not present in the current chapter. However, small CI differences were present in Study 2 therefore it is possible that this marked a return towards the pattern seen in the sober study in

Chapter 6 compared to the *after-alcohol* condition. Further clarity could be achieved by repeating the MBO group *before-alcohol* and *after-alcohol* conditions with equivalent numbers of trials to Study 2. Alternatively, and considering that the focus of the research is on the consequences of experiencing MBOs and not on performance under the influence of alcohol, eliminating the *after-alcohol* condition would afford a cleaner comparison of the two sober conditions in the MBO group, and offer a more direct replication of the experiment presented in Chapter 6.

There are several additional factors to consider when comparing the *after-MBO* condition to previous findings. Firstly, it could be that despite having a breath alcohol concentration of zero, the pattern observed *after-MBO* reflected a transitive state between being drunk and normal cognitive functioning. This would suggest an unknown period of impairment following blackout, where the participant is sober yet cognition is still affected. Secondly, in Figure 7.6D, a separation between difference waves peaking at around 800ms is visible, beyond the 500-700ms time window analysed here. Therefore, it may be that following an MBO, the process of recollection is delayed or more variable and we have therefore considered the wrong time window for this current analysis in a bid to provide consistency across studies. Neither the sober condition in Study 1, nor the findings presented in Chapter 6, show this later difference, which further provide evidence for delayed processing *after-MBO*. Arguably, the parietal memory effect observed in the literature reflects the combined signal of when recollection occurs, which can be variable within an individual. This latency variability has the potential to smear the parietal effect, for example, when comparing memory performance in young and older adults (Murray et al., 2019). It is undetermined whether recollection is consistently delayed or more variable *after-MBO*, but it would be interesting to see if Study 2's findings replicate in another sample *after-MBO*, and if other analysis techniques (such as residual iteration decomposition, RIDE) would clarify this point.

To conclude, we show that in Study 1 acute alcohol ingestion affected the behavioural performance of both control and MBO groups equally. Changes in neural data were observed too, and while we acknowledge that trial numbers may change between before and *after-alcohol* conditions, this may be indicative of perhaps a shift in memory strategy when appraising items – remember that the ERP signal constitutes

all trials for which participants were accurate (that is, *HH*, *HM*, *CR*). Critically, in Study 2 after participants had experienced an MBO, behavioural performance was still depressed when compared to equivalent data from Chapter 6, and ERPs demonstrated an enduring parietal effect that appeared to extend significantly beyond the traditional time windows for analysis of the parietal effect. This indicates that, even if behavioural performance was on a par to sober conditions, the speed of recollection may be impacted by acute blackout experiences, and future work should examine whether this extended parietal effect *after-MBO* is attributable to the variability, and therefore vulnerability, of memory after extreme binge-drinking episodes.

Chapter Eight:

General Discussion

Alcohol-induced memory blackouts are an indicator of extreme binge-drinking events and are clearly common occurrences in our Scottish based student-population. This thesis has presented evidence surrounding the prevalence of these events, and an investigation into some of the consequences for memory both acutely and in the longer term. Firstly, this chapter will offer a very brief review of some key points from the preceding 5 chapters, before going on to discuss the similarities and findings between the studies in more detail. The topic and project more broadly will then be considered, including the implications for our understanding of alcohol-related harm and future directions of research.

Review of Findings

In Chapter 3, the prevalence of binge-drinking and alcohol-induced memory blackouts in our Scottish based student population was assessed via a questionnaire, distributed to universities across Scotland. Extreme binge-drinking which leads to blackout was found to be associated with home country, mediated by personal drinking habits and – for Scottish students – by peer influenced drinking. Moreover, if you are male or in the earlier stages of academic study, this had a significant negative impact on drinking behaviours. Having established a 61.48% 12-month period prevalence of *fragmentary* MBOs within our sample, we moved to investigate any effects these amnesic events would have on episodic memory performance in a series of laboratory-based studies.

Chapters 4 - 7 showed that a Control group of participants who had never experienced an MBO, and a group of individuals who reported at least 9 MBOs in the preceding 12-months, displayed few differences in their behavioural accuracy across a selection of recall and recognition memory tasks while sober. However, converging evidence from ERP studies across Chapters 6 and 7 suggested that the groups may employ divergent neural strategies to achieve similar levels of memory performance. This finding implies an enduring negative consequence for cognitive functioning

resultant from frequent MBO experiences, even in younger drinkers, which requires an adaptation from normal processing during memory tasks in order to achieve the same performance accuracy as Control participants.

Chapters 4, 5 and 7 also included an *after-alcohol* condition where both Control and MBO group participants repeated the studies following a scaled dose of alcohol. Alcohol impacted upon behavioural performance across studies similarly for both groups, with a global reduction in accuracy and increase in conservative response bias. Further, participants in the MBO group were invited to return to the laboratory when sober but within 20-hours of experiencing an MBO and repeat the studies. Behavioural results showed that following a blackout, participants failed to achieve baseline sober levels of behavioural performance in some more cognitively demanding measures, retaining their conservative bias within their responses which suggests less willingness to risk an incorrect answer, or a lack of confidence in their ability to perform the task correctly. These chapters highlight evidence of the short-term effects to the brain and on memory formation which remain following an MBO.

What did our questionnaire tell us?

Consistent with wider literature (Davoren et al., 2016; McGee & Kypri, 2004; Tavolacci et al., 2016), we showed that our student population experienced frequent episodes of binge-drinking, and of MBOs. This is unsurprising, but nonetheless concerning within a young population due to the wide array of potential associated harms. Patterns of drinking were found to somewhat change across the undergraduate experience, with an increase in socially influenced drinking in 2nd year compared to 1st year, which might reflect students moving from university managed accommodation into privately rented property with friends. In later years, there was a reduction in drinking patterns which could be associated with increasing academic demands. Socially influenced drinking was the same for both males and females, although being male predicted a higher score on our blackout metric. Heavy alcohol consumption appears to be embedded within university culture and is a much-anticipated feature of the 'student experience' (Davies et al., 2018; Gambles et al., 2021; Tarrant et al., 2019), a suggestion which is consistent with the behaviours reported by our Scottish-based students and in-line with predictions.

Our findings of prevalence of binge drinking and blackouts is particularly concerning given that binge-drinking behaviour leads to increased chances of physical injury from accident or aggression and increased risk taking in general (Melchior et al., 2008; Sindelar et al., 2004). It is unlikely that any acute cognitive harm would be visible, beyond the hangover feelings the next day – peers engaging in similar drinking behaviour will not notice someone experiencing blackout conditions, and it appears that the memory loss is only transient. While binge-drinking has been shown to impact upon executive function in students (Salas-Gomez et al., 2016), would anyone think that this is any worse than your speech being slurred after drinking alcohol? This could lead to students dismissing any concerns regarding the long-term consequences of their binge-drinking due to a lack of immediate, and obvious, evidence. However, differences in brain matter are observable in those who binge-drink in adolescence and early adulthood (Bava & Tapert, 2010), compared to those who do not. The experimental studies in this thesis suggest that frequent MBO individuals employ divergent memory strategies - even while sober - to reach similar levels of performance as controls. Taken together, it appears that large numbers of students' binge-drink frequently, in correspondence with their own expectations of student life. In general, there is a lack of awareness of the potential consequences of prolonged binge-drinking behaviour, and any concerns young people may have seem to be outweighed by social norms. Arguably, this thesis highlights some of the acute consequences of extreme binge-drinking behaviour, which have received little attention so far, which could have repercussions far into the future.

Although drinking patterns in students are influenced by some wider factors (for example, home country/peers, etc.), there is a decline in drinking as students' progress towards leaving university. In the over-25s, research suggest that alcohol consumption rates stabilise, with fewer binge-drinking episodes recorded (Giles & Robinson, 2018; Office for National Statistics, 2018). Beyond university, increased levels of responsibility accompany career progression, mortgages, relationships, and starting a family, which can alter social priorities and financial circumstances. This change from the unstructured life of a student, coupled with a reduction in tolerance due to fewer drinking episodes (Elvig et al., 2021), likely explain this shift from binge-drinking. However, middle-aged adults are known to consume the most alcohol overall (Giles & Robinson, 2018). This could indicate a 'grazing' attitude to alcohol where

perhaps only a few drinks are consumed across an evening instead of bingeing larger quantities, but the pattern is repeated frequently across a week. It is unknown if the reduction in binge-drinking, and the following period of reduced alcohol drinking, mitigates for any harms caused to the brain during early adulthood and adolescence. It is also unknown whether those who progress to higher levels of diffused drinking across a week in middle age are the same individuals who reported more instances of binge-drinking while younger, or if there is no relationship between these drinking behaviours.

What can behavioural results tell us?

The episodic memory studies selected for this thesis spanned both recall and recognition tasks. Since very few neuropsychological studies of individuals who frequently blackout have been published (Wetherill & Fromme, 2016), this series of memory experiments were designed as a baseline, providing some initial findings and suggesting directions for future work. As such, they included a variety of complexities, and demands. For example, the three recall tasks differed in cognitive load, beginning with a simple free recall, the addition of a serial recall component, and finally a depth of encoding manipulation with delayed recall. In general, we found that behavioural performance across this series of tasks was impaired by alcohol similarly in both groups, and that deficits following an MBO increased with task complexity.

The word lists from the Free and Serial recall tasks were used as study phase for the recognition memory DRM experiment. Typically, and as seen in the design of the studies in Chapters 6 and 7, recognition memory paradigms do not always include a recall task prior to recognition. Further, recognition tasks should be easier than recall tasks for participants to perform due to the reduced cognitive effort involved in passively viewing a stimulus to prompt memory (recognition) compared to consciously and actively trying to conjure from the ether the word you were trying to remember. It was a concern that by adding a recall task prior to recognition, we made the DRM task too simple for our groups. However, including recall prior to recognition is not a new method, and has been utilised widely within the literature (for example, Maylor et al., 1987; Milani & Curran, 2000; Roediger & McDermott, 1995). If recalling words prior to the recognition task had simplified the study, it might have been expected that mean accuracy would have been at ceiling levels, particularly when

considering the semantic relationships between those studied words. This was not the case while sober, and alcohol further impacted upon performance for both groups, suggesting that the methods employed did not have a detrimental effect on results. In general, behavioural patterns of performance across the DRM and the later recognition tasks which did not include a recall phase, were similar for both groups.

One benefit of the experimental designs employed throughout this thesis is the ease of which comparisons can be made across chapters. For example, in Chapter 4, the MBO group recalled more words in the less cognitively demanding Free recall task (58.98%) than in the more challenging Serial recall task (53.26%) while sober. In Chapter 5, they also recognised more words from the earlier Free recall word lists (82.84%), than the Serial word lists (73.41%). The same pattern applied to the Control group. The simplicity of the Free recall phase prior to the recognition task clearly increased the number of words initially recalled, and therefore the likelihood of recognition of those same words in the later DRM study, compared to words from the Serial recall task. Interestingly, words from the Free and Serial conditions were recalled by the MBO group (Free 43.83%, Serial 43.68%), and Controls (Free 43.06%, Serial 42.41%) with almost identical mean accuracy *after-alco*hol in the recall tasks. In the DRM study, Free words were no more likely to be recognised than Serial *after-alcohol* (Free 60.15%. Serial 58.77% for MBOs; 59.72% and 58.89% respectively for Controls), suggesting that while recognition performance accuracy may be slightly better than recall *after-alcohol*, the level of impairment did not differ between groups, or by encoding type. Given the scientific rigour and volume of evidence accumulated in this thesis, in contrast to the literature (Hartzler & Fromme, 2003; Wetherill & Fromme, 2011) people who experience frequent blackouts are not more affected by the presence of alcohol than those who do not blackout, at least in terms of behavioural memory performance. The suggestion that drug tolerance, that is, any argument that refers to the amount/frequency of previous drinking, would lead to some kind of advantage/disadvantage in behavioural performance is not supported by our findings.

The mechanisms of encoding, binding and transfer of information from the hippocampus to long term storage are reportedly impacted by alcohol (A. M. White, 2003), resulting in an MBO experience where events cannot be recalled due to inefficient or non-existent storage. Therefore, we hypothesised that individuals who regularly experience MBOs may display differences in encoding strategies compared

to controls, and as a result, included several encoding manipulations to investigate this (free and serial recall; shallow and deep encoding conditions). There was no effect of encoding type on *d'* in the DRM task for either group. This means that participants' ability to discriminate between old and new words was not impacted by the differing demands in the recall element of the encoding phase (free/serial recall). Serial words were recalled and recognised slightly less than Free words in both sets of studies when sober and *after-alcohol*, although there was a larger drop in accuracy for words from the Free recall word lists than the Serial lists in both recall and recognition tasks for both groups. The additional cognitive complexity required to recall words in serial order might have been expected to strengthen encoding and therefore protect performance *after-alcohol* in both the recall and recognition phases of the studies, but the drop in accuracy for both sets of words suggests this did not happen. It is likely instead that recalling fewer words in serial order instead meant fewer words were recognised later.

The encoding manipulation in the depth task involved participants making judgements about presented words, either shallow (upper or lowercase lettering) or deep (did the word fit in a sentence). Again, total words recalled decreased *after-alcohol*, but it was the Control group who showed the greatest impact. It could be that Controls, who were not practiced at consuming alcohol to the same degree as the MBO group, had not developed the drug tolerance or memory strategies to perform more complex tasks *after-alcohol*. Alternatively, the reverse could be true, and it was the frequency of drinking by the MBO group which protected their performance and therefore prevented a greater drop in accuracy. For both groups, words from the deep encoding manipulation were recalled with greater accuracy than shallow words *before-alcohol*, and yet there was no difference between deep and shallow *after-alcohol*. This is somewhat reflective of the results in the Free and Serial recall tasks, where *after-alcohol* words from each task were recalled similarly, despite the differing encoding manipulation. It may be that, after ingesting alcohol, encoding of new information is impacted despite manipulating how much attention is focussed on encoding for both groups, perhaps implying that the levels of processing memory framework is not accurate.

While we were limited by the quantity of alcohol we could ask participants to safely consume, we were able to repeat our studies the day following a blackout event.

We found sustained behavioural impairment in performance across most of the studies. Chapter 4 showed that only in the Free recall task did performance accuracy improve *after-MBO*, compared to *after-alcohol.* In the Serial task, there was no difference in mean accuracy between *after-alcohol* and *after-MBO,* and within the DRM task, there was no difference in *d*' *after-MBO*. However, in this task, there was a more nuanced result, with also no difference between *before-alcohol* and *after-MBO*. This speaks to the individual variability between participants but may also be further explained by the different analysis measures. In the recall tasks, participants simply recalled as many words as they could which were then summed and averaged across groups to provide a measure of mean accuracy. In the recognition tasks however, participants were asked to identify words as old (previously seen/recognised) or new. In Chapter 5 (and later in 6 and 7), accuracy is therefore discussed as *d'*, a signal detection measure of sensitivity between accurately identified old and new words, rather than as simple mean accuracy. While ability to discriminate between old and new words decreased *after-alcohol* and showed slight (although not significant) improvement *after-MBO* using *d'*, mean accuracy remained impaired. The difference between the two results is easily understood when considering *C*, response bias. After blackout, participants retained a conservative bias when responding 'old' or 'new' to displayed words, which reduced their willingness to say 'old' and risk an incorrect answer. Therefore, their mean accuracy was impacted due to responding 'new' when they were less certain. However, when they did respond 'old', they were more likely to be correct. This meant more chance of responding 'old' correctly and preserving *d'*, but also more likelihood of a 'miss', reducing mean accuracy.

In addition to the DRM study in Chapter 5, we also conducted recognition and source memory experiments (Chapters 6 and 7). We again found that group behavioural performance did not differ while sober across a range of measures (for example, mean accuracy/*d'*), and that *after-alcohol*, performance dropped similarly for both groups, in each of the studies. The *after-MBO* conditions also consistently showed continued impairment compared to baseline. The DRM study showed no difference in *d'* between *after-alcohol* and *after-MBO*, and behavioural accuracy in the source memory studies (see Table 7.5 on p.187) also show no difference between *after-alcohol*, and *after-MBO*. Whilst I acknowledge that this statement somewhat generalises the findings, which are more nuanced in actuality, there is evidence of

continued impairment in recognition *after-MBO* which can be measured across these studies.

It is interesting to note the difference in Remember/Know/Guess (RKG) responses in Chapter 5 and the *Hit-Hit* (*HH*) and *Hit-Miss* (*HM*) responses in Chapters 6 and 7, between Controls and MBO group participants. In the DRM task, participants were asked to make an RKG response which would indicate their strength of memory for the word identified as 'old'. *Remember* responses are suggested to reflect recollection, whereas *Know* shows less certainty and perhaps a reliance on familiarity (Tulving, 1985). Notwithstanding the disputed nature of these judgements, let us accept for now that this is a valid framework to understand the contribution of different memory processes (attested to by the volume of literature on this topic). Although both groups recorded more *Remember* responses overall, the MBO group responded *Know* more often than Controls in all conditions. In Chapters 6 and 7, while again recording a *HH* response most frequently, MBOs were also more likely than Controls to record *HM* response, where the word was correctly identified as 'old' but the colour source was incorrect. A *HM* response is believed to reflect a recognition of the stimulus but without contextual detail, similar to the memory process of familiarity. Taken together, this could be behavioural evidence that the groups relied on different cognitive strategies while performing recognition memory tasks to similar levels of accuracy. Memory literature suggests that familiarity is a process which is not hippocampal dependent, unlike recollection (Aggleton & Brown, 1999; Bisby et al., 2010) and, although alcohol causes global impairments, it is the hippocampus which is acutely impacted in memory processing. Therefore, it is perhaps unsurprising that the MBO group rely on strategies which do not necessarily require hippocampal involvement for recall. In other words, if the MBO group relied more on familiarity than Controls – even while sober – this could be an indication of underlying neural shifts in processing in order to complete tasks.

How does the ERP data fit with the behavioural data?

Studies presented here and in the wider literature with participants who frequently blackout have shown that behavioural performance does not necessarily differ from that seen in controls, when sober. This could mean that the groups are not different and if so, ERP data should also be similar. Alternatively, the groups may rely

on different neural strategies to perform tasks to similar levels, which would reveal differences in ERPs. As discussed, most behavioural findings were similar, however there was some suggestion that there may be differences in the way our groups approached the tasks. Although it is not good practice to statistically compare ERP amplitudes between groups consisting of different individuals, it is possible to consider the presence or absence of effects more generally. Further, most ERP research looks for evidence which compliments, or explains, behavioural activity rather than looking for a dissociation, which may be more common in clinical research. For example, a statistical difference between stimulus types could be expected to be reflected in ERPs in neurotypical participants. Yet in the studies presented here, some comparisons showed no behavioural differences between groups, or within groups, and yet ERP differences were present. In neuropsychological studies, this could be indicative of damaged neural structures which are not necessarily required for task performance (Rugg, 1992). Alternatively, individuals in one group may have evolved alternative neural strategies for performing tasks to the same level of accuracy, as could be the case with our MBO group.

Differences and similarities between the EEG studies, and groups, have been discussed in some detail in Chapter 7 and shall therefore only be summarised here. In brief, Chapter 6 revealed distinctively different neural response ERP patterns between groups across both a 400-600ms, and 500-700ms time-window, overlapping with the mid-frontal familiarity effect and the left parietal recollection effect, despite similar behavioural performance. This suggested that the groups differed in strategy in order to achieve the same end while sober. These findings were broadly replicated by new groups of Control and MBO participants in Chapter 7, also sober. It is therefore plausible that experiencing frequent MBOs leads to a shift in neural memory processes for recognition tasks in order to achieve the same accuracy as Controls. While recognition is arguably a simpler process than recollection, we found no differences between controls and MBOs during pure recollection tasks while sober (see Chapter 4). To confirm our interpretation that memory processes are affected by previous blackout experiences, future work should attempt to move beyond recognition tasks with ERPs, which do not quantify the amount of recollection (for example, the *HH* signal contains instances of accurately recollected items and guesses), and isolate the process of recollection with ERPs.

In the *after-alcohol* condition, we again showed that group accuracy was similar, but neural responses in both time-windows differed. For example, the Control group's *HM* signal across the left parietal region did not differ to *CR*, whereas it overlapped with *HH* responses in the MBO group. This difference is reflected in behavioural data when examining source accuracy. Although no statistical difference between groups existed, Controls showed a significant reduction in source accuracy *after-alcohol*, which was not present in the MBO group. However, the MBOs significantly reduced their *HM* responses *after-alcohol*, whereas there was no difference in Controls. The ERPs suggest that the MBO group had the same neural response for *HH* and *HM*, therefore a response for either of these was believed by participants to be a correct answer. This is supported by the reduction in *HM* behavioural accuracy. It appears that the MBO group were less willing to risk an incorrect answer *after-alcohol* (therefore only responding 'old' if they were sure) which reduced the total number of *HM* responses, but not source accuracy. In other words, when they thought they were right, they were likely to be right. This pattern *after-alcohol* is similar to that seen in the DRM recognition paradigm. The reverse appeared true for Controls – either they knew they were right (*HH*), or they were not sure, and the ERP mapped the *CR* signal. In terms of dual process theory, while recollection and familiarity were diminished by alcohol consumption in both groups, the MBO group make no decisions based purely upon the sense of familiarity, which is argued to support the process of recollection.

In Chapter 6, we found that our frequent MBO and Control participants are comparable in behavioural memory performance yet the underlying neural data suggests that they use different strategies to achieve the same result. There are a number of possible explanations for this; they are not mutually exclusive and further research would be needed to disentangle their individual contributions. Firstly, it may be that the two samples are quantitatively different from each other in the way that memory operates; that is, that memory in some people operates differently due to structural or functional differences in the brain, which could lead to them being predisposed towards experiencing alcohol-induced memory blackouts (Wetherill et al., 2013; Wetherill & Fromme, 2016). Another possibility is that persistent short-term damage to the brain caused by binge drinking alcohol to extreme levels, and perhaps consequently blacking out, has altered the structure and therefore functioning of

neural networks. Such change need not be immediately observable in behavioural data, or indeed everyday behaviour, if the participants can compensate for this loss of functioning. However, while neural plasticity and reorganisation can be compensatory, they can never fully account for the loss of functioning attributable to abnormal cell death (Bennet et al., 2013; Crews et al., 2006; Dorszewska, 2013; Granato & Dering, 2018). At some future time-point, the damage done by extreme binge drinking may become more evident, akin to the cognitive decline observable in alcoholism over time (Oscar-Berman et al., 2014; Perry, 2016; Stavro et al., 2013).

When planning these studies, we did not account for the impact of alcohol on EEG recordings seen in Chapter 7. Participants from both groups responded very differently to the *after-alcohol* conditions; the behaviour in some seemed unaffected despite BrAC readings, others became noticeably less focused or able to sit still – crucial for recording interpretable EEG data. Unfortunately, this meant that almost half of participants had to be removed from analysis due to noisy data, or insufficient trial numbers. In hindsight, more trials from individuals could have been collected in order to mitigate for these exclusions which, eventually, would have provided a more valid sample size. This was not possible due to Covid-19 pandemic restrictions. Despite these difficulties, we did find that while behavioural results were again similar between groups, ERPs differed. Further, *after-MBO,* there appeared to be a delay in processing beyond the window of analysis. This may not be surprising, as repeated detoxification in alcohol abuse has been shown to increase time taken across a range of cognitive tasks (Duka et al., 2003). While we are not suggesting that our MBO participants were alcoholics, the pattern of extreme binge-drinking and detoxification may have similar effects on cognition (Duka et al., 2004), even in a young adult population. Further, although this thesis is concerned with the effects of MBOs on episodic memory, it may be that impairments *after-MBO* could be found in executive functioning more broadly, consistent with binge-drinking studies (Townshend & Duka, 2002). This could be easily investigated with laboratory-based paradigms which follow a similar structure (*before-alcohol*, *after-alcohol*, *after-MBO*).

What were the lasting effects following blackout?

The continued reduction in recognition memory performance, compared to sober baseline, may be explained by neurotransmitter functioning in the hippocampus.

A high dose of alcohol (such as in an extreme binge-drinking event) acts as an antagonist on NMDA receptors (A. M. White & Best, 2000), which in turn has been shown to reduce recognition memory performance (Bisby et al., 2010; J. H. Krystal et al., 2003). It is unknown exactly how long it would take for the balance of neurotransmitters in the brain, for example, glutamate and GABA (Granato & Dering, 2018), to return to pre-alcohol levels after an MBO as this depends on the quantity of alcohol consumed, and if any imbalance persists after the blackout event. Interestingly, we do know that individuals with substance abuse conditions go through a period of withdrawal, when (in the case of alcohol) they may be sober but are experiencing cravings for alcohol linked to reductions in dopamine, or feel negative emotions (Volkow & Morales, 2015) associated with increased stress-related hormones (Koob & Le Moal, 2005). Taken together, a possible neurotransmitter imbalance which extends beyond the point of sobriety, and perhaps withdrawal symptoms, could impact on memory performance even in those who may not reach the threshold for a substance use disorder diagnosis. While this is speculative, there is enough evidence accumulated to suggest that this is a valid hypothesis, albeit difficult to test in humans.

It is unclear whether MBO experiences constitute a temporary 'injury' to the brain which is quickly recovered from once alcohol is metabolised and excreted from the body, or if the process of blacking out evolved as a neural defence mechanism, working to protect neurons and synapses from high concentrations of ethanol. Regardless of the neurobiological considerations, the focus here was on whether the experience of an MBO left a lasting deficit in memory performance. The results presented across the chapters in this thesis suggest that deficits in memory performance remain, even when sober, after experiencing a blackout. However, what is less clear is how this cognitive impairment is alleviated, that is, the time taken for 'recovery' of functioning to baseline sober levels of performance. When drinking alcohol, cognitive functioning in general becomes impaired in a non-linear fashion, where not all abilities decline at the same rate or time. For example, speech fluency improves initially with weak doses of alcohol, as people become less inhibited, before declining later at high concentrations. Therefore, as people revert towards sobriety, it could be that a host of cognitive abilities return to baseline functioning at different rates (or gradients of linear slopes). Indeed, the evidence presented in this thesis suggests this. To investigate lasting effects, we resampled mean differences between

before-alcohol and *after-MBO* accuracy scores across a range of 7 possible measures in our behavioural studies (see Chapters 4 and 5) and compared these normalised distributions to the differences in recorded mean scores of our MBO group. We found evidence of continued impairment in all studies, although in varying numbers of participants. Moreover, as cognitive demand increased within tasks, so did the number of participants who showed a significant performance deficit. Therefore, while basic cognitive functionality returns rapidly once sober, our evidence shows that an unspecified amount of additional time is required to fully regain pre-alcohol performance levels. Note that differing levels of alcohol intoxication were not controlled due to the naturalistic nature of the alcohol consumption in our MBO group but, in the future, we aim to account for this variability.

We discounted the role of sleep as impacting negatively in these calculations, with no evidence found for any correlation between duration of sleep and differences between *before-alcohol* and *after-MBO* scores. However, it was not possible to quantify exactly the amount of alcohol consumed by participants which led to their MBO experiences, nor the time which passed between the MBO starting/ending and testing the next-day. These factors likely contributed to the range of variability seen in our participant scores. It is known that an MBO occurs following rapid, extreme binge-drinking (Rose & Grant, 2010; A. M. White, 2003) and that there is a dose dependant relationship between types of blackout (A. M. White et al., 2004), and therefore level of cognitive impairment. Therefore, it is possible that those who displayed the greatest impairment in our studies consumed the most alcohol and/or experienced the most severe MBOs. This can be seen when viewing Table 8.1, displaying the number of individual participants from our resampling analyses who show deficits *after-MBO* in Chapters 4 and 5. On average, participants displayed a reduction in performance *after-MBO*, compared to *before-alcohol*, on 3.83 occasions of a possible 7, or 54.66% times. Note that 3 participants showed reduced performance in all 7 measures, while a further 7 remained impaired in 5.

Table 8.1:
Number of participants showing reduced performance after-MBO compared to sober (from resampling analyses)

Participant	Free ACC	Serial ACC	Imm. Deep	Imm. Shallow	Delay Deep	Delay Shallow	DRM *d'*	Total	Frequency of effect%
1	1	1	1		1		1	5	71.43
2		1	1		1		1	4	57.14
3		1	1		1			3	42.86
4	1	1					1	3	42.86
5	1	1	1	1		1		5	71.43
6		1						1	14.29
7		1						1	14.29
8		1	1	1	1	1		5	71.43
9	1	1	1		1			4	57.14
10 *	1	1	1	1	1	1	1	7	100
11			1	1	1	1		4	57.14
12			1	1	1	1	1	5	71.43
13	1	1		1			1	4	57.14
14		1						1	14.29
15		1	1	1	1	1		5	71.43
16 *	1	1	1	1	1	1	1	7	100
17 *	1	1	1	1	1	1	1	7	100
18								0	0
19	1			1		1	1	4	57.14
20			1		1			2	28.57
21		1	1	1	1	1		5	71.43
22					1			1	14.29
23	1	1	1		1		1	5	71.43
Total | Mean	10	17	15	11	15	10	10	3.83	54.66

Difference between total number of participants who showed impairment in deeper encoding conditions (serial, immediate deep, delay deep) compared to shallow encoding conditions (free, immediate shallow, delay shallow), $t(2.30) = 21$, $p = .032$, $r = .489$.

*Tasks in which individual participants displayed a lasting impairment are indicated above, with each participants' total number of measure impairments, and the percentage of occasions on which this occurred, given. Note that individuals who showed deficits in all tasks are denoted by *.*

Interestingly, the task in which the largest number of participants (n = 17, 73.91%) showed an *after-MBO* deficit was the serial recall task (Chapter 4). This task required participants to study, and then recall word lists in the order of stimuli presentation. Analogous to recalling events across time, and compared to simpler tasks, the increased focus and cognitive processing required to complete this task could have meant that even *after-MBO*, participants would have been able to recall the stimuli, but this was not the case. Further, the deep encoding condition within the depth of processing task (described in Chapter 4) also required additional cognitive resources which may have been expected to provide a protective effect on word recall, yet 15 participants (65.22%) displayed an *after-MBO* effect in both the immediate and

delayed recall conditions. It appears the tasks which had increased cognitive load were performed most poorly by participants after blackout, a suggestion supported by the statistical difference between impairment recorded in deep compared to shallow tasks.

In sum, our *after-MBO* participants were all sober, yet individual performance varied across tasks, and remained impaired compared to *before-alcohol.* This suggests that BAC sobriety does not necessarily mean an immediate return to pre-alcohol levels of functioning. Further, we know that the body processes alcohol at around the rate of one unit per hour, but we can be less certain of the time taken after detoxification to return all cognitive functions to baseline performance. Therefore, while some basic neural functionality may quickly return once sober (for example, you are less likely to slur your words), the ability to process more cognitively demanding tasks appears to take additional time to fully come back online. The evidence presented here would suggest that a lasting deficit remains for more complex memory functioning for an undetermined period beyond the point of sobriety, and that recovery of functions is non-linear. This may extend to some other cognitive processes; however, this question has not yet been addressed in the literature.

MBO vs Hangover – what are the differences, and can next day impairments be disassociated?

A criticism of MBO research is that the experience of an MBO is subjective, therefore do they really occur? They cannot be observed and studied while they are happening, and we have to rely on self-report from people who experience them. However, in order to do so means that people must realise that they have forgotten events from while they were drinking, and be willing to talk about it. Remembering that you've forgotten is a circular argument – how can you remember what has been forgotten? And if something is forgotten, how do you know to try and remember? This basic issue is compounded by the fact that we all forget (Popov et al., 2019; D. G. Ray et al., 2019). For example, during a social occasion, not every comment made by every guest is recalled with clarity, or even at all. Therefore, it could even be argued that *fragmentary* MBOs are simply examples of ordinary forgetting which appear exaggerated by the addition of alcohol. However, there is substantial anecdotal

evidence surrounding *en bloc* MBOs where no details spanning large chunks of time, even after cueing, can be recalled by individuals. Moreover, most adults who drink alcohol can describe from personal experience the realisation that they have periods of memory loss from while intoxicated, or they know others who have. Therefore, while descriptions may be subjective and reliant on self-report, substantial evidence for the occurrence of MBOs is easy to find. Further, there is consistence in the descriptions of the experience across time and research groups (for example, Goodwin et al., 1970; Jellinek, 1946; Jennison & Johnson, 1994; Ward et al., 2021; A. M. White et al., 2004), which suggests a similar experience in diverse participants.

I postulate that perhaps an alcohol-induced MBO may only be recognised by the individual who had the blackout if there is a significant shift in the context of where the blackout began, for example, if an individual changes location during the course of the blackout, when memory functions come back online, this may give the individual a shock and indicate that they must have lost some memory of events for a period of time. Naturally, if the location and context remain the same, uncovering whether you had a blackout or not becomes more difficult. There is a large enough body of research suggesting that to deny the blackout phenomena is real would be naive. Early alcohol research reported the existence of blackouts, and this was obtained from individuals who had no reason to deny recognising the stimuli presented to them. For example, Goodwin et al., (1970) showed that although highly salient stimuli could be recalled after 2-minutes, they were not always recognised or recalled by participants after 30-minutes, or the next day. The phenomenon appears to be real and therefore developing a greater understanding of its effects is important. Moreover, since it appears that MBOs only occur at high levels of BAC, conducting experiments with participants who self-report experiencing frequent MBOs at the very least provides evidence for how cognitive functioning differs in extreme binge-drinkers compared to controls. Given the harms associated with binge-drinking, this factor alone deems research of those who frequently claim to blackout a worthwhile pursuit.

Accepting that MBOs are a 'real' experience, can we definitively say that any lasting cognitive deficits seen following a blackout are caused by the experience itself rather than their accompanying hangover? At present, literature makes this a difficult question to answer definitively, and firstly requires discussion of hangovers more

generally. The Alcohol Hangover Group, along with van Schrojenstein Lantman and colleagues (2016), defined a hangover as "the collection of mental and physical symptoms, experienced the day after a single episode of heavy drinking, starting when blood alcohol concentration approaches zero"; arguably these would be conflated with any lasting blackout effects after such an experience when intoxicated. Further, the suggested quantity of alcohol which is required in order to invoke this circumstance has been changed from 0.11% BAC (Verster et al., 2010), to an unspecified amount. This reflects the interesting fact that hangovers can be reported after a single drink in some people. It may be that a hangover occurs when an individual drinks more than their personal norm (Verster et al., 2020), regardless of how small the quantity consumed or BAC reached. Conversely, to invoke a blackout requires a large amount of alcohol to be consumed rapidly. Therefore, a hangover can occur with or without a preceding MBO, the two are not mutually inclusive, and deficits recorded after one phenomenon cannot therefore be assumed to be present after the other.

Studies conducted within the hangover literature typically do not ask about MBOs, and instead recruitment of participants ranges from self-identified 'social drinkers' (Verster et al., 2014) to experimenter instigated opportunity sampling (Scholey et al., 2019; see Gunn et al., 2018 for a review of the field). Our sample, on the other hand, specifically reported frequent extreme binge-drinking *and* MBO experiences. A review of hangover literature by Stephens et al. (2008) suggested that laboratory-based studies may produce some physiological symptoms of hangover but that for cognitive deficits to be seen, participants would have to drink at more naturalistic levels. However, they also pointed to problematic methodological factors in some naturalistic studies. For example, Stephens and colleagues showed that BAC was not measured before hangover task commencement in one study included in their review, therefore it was unknown if participants were still intoxicated; and levels of BAC reached during intoxication were simply averaged across groups in others. This is problematic because in a laboratory study, alcohol consumed by participants can be controlled to ensure similar levels of BAC within groups, however in naturalistic studies there is likely to be much greater variation. This means that an average BAC from a naturalistic study will consist of greater individual variation than the more tightly controlled laboratory study, and therefore the two should not be directly compared. Moreover, they also suggest that individuals who take part in naturalistic

studies are typically aware that their next-day performance will be assessed for impairment while hungover, and that this expectation could therefore introduce confounding factors. For example, they may drink more than normal in order to ensure a hangover occurs. However, it could also be argued that participants in any study of hangover would realise that they are being tested for hangover impairment, and therefore the expectation is not reserved for naturalistic studies, even if there is more control on alcohol consumed in the laboratory.

Like some hangover literature, the work presented in this thesis could not provide accurate BAC levels for participants in the *after-MBO* conditions. However, we highlight some strengths of our approach which differs from many of the published studies. Firstly, we did not begin testing until the afternoon and we ensured that participants were sober, as measured by BrAC; and secondly, all participants confirmed that they had experienced an MBO which is indicative of an extreme binge-drinking event. It could be that there is no perfect way to measure either hangover, or blackout effects, as it is impossible to create placebo conditions for either experience with which to compare performance. Therefore, work in this area may be open to criticism of expectancy effects, and of lack of control over quantities of alcohol consumed. While it may not be possible to eliminate expectancy effects from MBO research (if indeed these are relevant; see below for further discussion), other variables could be controlled, or documented, more carefully. For example, technology which recorded drinking quantities and speed, or confederates who monitor behaviour, offer the possibility of mapping drinking sessions. Of course, the presence of a confederate may in itself alter behaviour and could present ethical challenges. Nonetheless, since it is not possible to responsibly induce an MBO in a laboratory, researchers must work within ethical constraints by taking advantage of naturally occurring behaviour and new technologies, and by seeking to reduce as many confounding factors as is practically possible. It may be that simply increasing details recorded from a naturalistic drinking session would allow for more accurate comparison of behaviour and performance than is currently seen in the literature.

Fragmentary vs *en bloc* blackouts – are they really different?

Two types of alcohol-induced memory blackout are believed to exist – *fragmentary* and *en bloc*. These were differentiated by Goodwin (1971) and defined as

the loss of snippets of time which may be recalled at some future point (*fragmentary*), or longer periods for which no memory can be recovered (*en bloc*). The difference is suggested to stem from the levels of BAC at any given time, with *en bloc* blackouts resulting from greater alcohol consumption than *fragmentary*, leading to a seemingly total 'shut down' of information transfer and encoding. This differentiation is widely accepted in the literature, and indeed discussed in this thesis. However, beyond this accepted model little has been done to further dissociate or understand the differences between MBOs. Indeed, it is likely that instead of an oversimplified dichotomy between fragmentary and en bloc MBOs, alcohol-induced memory loss operates on a continuum.

To further the idea of blackouts operating on a continuum, I propose that a simple model of blackout behaviour, taking a threshold BAC for when memory transfer begins to shut down, explains both *fragmentary* and *en bloc* MBO phenomena (see Figure 8.1). As BAC increases rapidly over time, drinkers experience a series of fleeting *en bloc* blackouts, that is, the *fragmentary* experience. Further, it may be that memories recoverable from this period could be from the points in between, rather than actually from during the impairment, which appear whole due to the brief nature of the blackouts. If drinking continues at pace, these *fragmentary* MBOs may then become a more enduring *en bloc* event, arguably which would only be recognised by the individual when a significant duration has passed in a blackout state. If alcohol ingestion slows down, it is likely that *fragmentary* blackout experiences occur with only minimal ingestion needed to bridge the threshold again. In other words, the blackout mechanism is the same in each, but the duration of the experience differs based on levels of BAC. The speed of alcohol metabolism is constant (0.015g/l/hr) but much slower than the pace of binge drinking, which arguably would lead to increasing time periods of blackout proceeding an en bloc experience. Following an *en bloc*, individuals may not recall that these shorter interludes also occurred due to the greater impact from the more severe blackout. This suggested model is speculative, however there is no evidence of differing routes to experiencing the two types of blackout, other than the quantity and speed of alcohol consumed. Therefore, it seems likely that they are the same, with only duration differing.

It has been suggested that *fragmentary* blackouts occur at lower BAC thresholds than *en bloc* blackouts do (Rose & Grant, 2010), highlighting the difference in memory loss and severity between the two. Yet this idea cannot be accurate; if the route towards an *en bloc* MBO must pass first through *fragmentary* blackouts (that is, reaching a higher BAC threshold than the one for *fragmentary* blackout experiences), then what happens on the descending limb of the alcohol curve? Do people also pass through brief *fragmentary* moments until cognitive functioning begins to operate more normally, or (once BAC has decreased sufficiently) is there a moment where memory comes 'back online'? Memory, and wider cognitive functioning, appear to be more greatly impaired on the ascending limb of the BAC curve than the descending limb (Schweizer et al., 2006; Söderlund et al., 2005). Therefore, the proposed model of a single BAC threshold for blackout experiences seems most accurate. Under this model, when detoxification has reached the point where memory systems are once again operational, they remain so rather than tipping back into short-term, *fragmentary* impairment. To be clear, this model is not directly based on the findings presented in this thesis, but is instead a suggestion based on knowledge of the wider literature and recognition of gaps in the current understanding of blackout experiences.

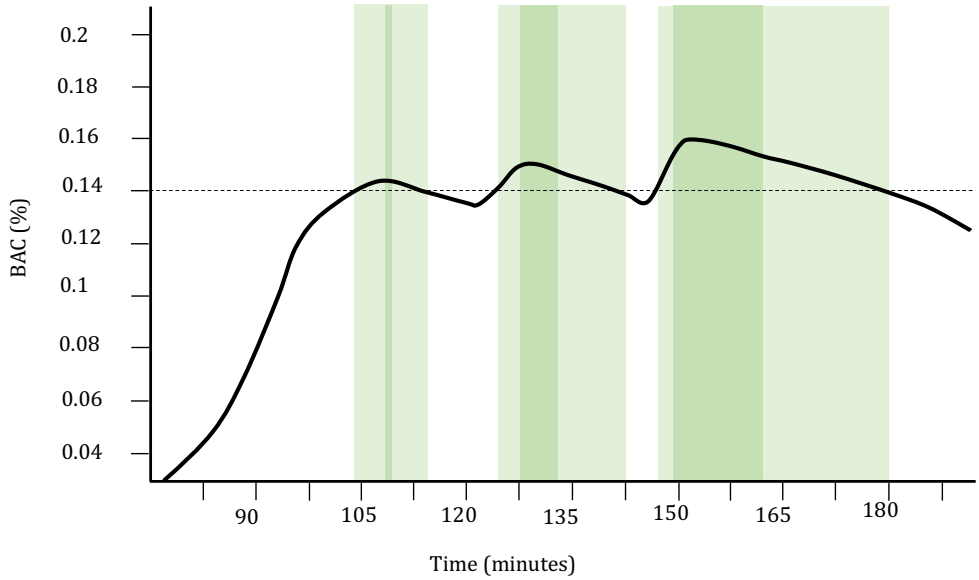

Figure 8.1: **Potential model of the progression of blackout experiences.**
Horizontal line denotes the point at which this fictional individual would begin to experience memory loss, a hypothetical threshold for when memory loss could occur. The rapid rise and steady fall of BAC means that this person would experience 2 short

blackouts, before progressing to a sustained period of memory loss. Note that the BAC point at which this occurs above is just for illustration, there is no current consensus in literature of the BAC level which instigates an MBO. The light green bars represent a period of time where potential memory loss occurs, i.e., shut down of memory transfer begins, whereas the darker green bars suggest periods which are highly likely that memory loss happens.

Alcohol Expectancy Effects

In our laboratory-based studies, participants knew that they were going to be asked to consume alcohol before arrival, and were aware of it happening, that is, there was no attempt to mask the alcohol, or offer a placebo. Did this impact upon our findings *after-alcohol* by reducing performance in line with the expectation of participants? In theory, this is a possibility. However, literature suggests that expectancy effects apply more to social than cognitive consequences (Hull & Bond, 1986; Lyvers & Maltzman, 1991; Peterson et al., 1990). Further, I would argue that while in the laboratory, the MBO group showed little obvious effects of the alcohol consumed in their demeanour or apparent attention to the tasks, regardless of BrAC readings and performance accuracy, suggesting that they did not consider themselves intoxicated. Additionally, for the *after-alcohol* testing, they were asked to drink substantially less alcohol than they reported drinking outside the laboratory in their own time, and sometimes seemed surprised (and concerned) by their BrAC readings. This suggests that given the quantities of vodka they consumed, they did not expect to feel drunk. On the other hand, the Control group were less used to drinking alcohol and some participants appeared apprehensive prior to drinking. This could imply a negative expectation of how they would feel or behave, which could have impacted performance. However, between before and *after-alcohol* conditions, both groups showed similar drops in performance results in the behavioural studies presented in Chapters 4 and 5, and in the behavioural measures in Chapter 7. If either group had expected to feel acute alcohol intoxication which impacted on their behaviour, it is likely that one of the groups would have shown a greater decrease in performance than the other. It is also likely that this would have been seen in the Control group due to their inexperience with drinking alcohol, and yet in some measures, they performed better than the MBO group *after-alcohol*. This appears to be consistent with wider literature which suggests that expectancy is restricted to the social anticipation

surrounding drinking events, rather than the physiological feelings which accompany alcohol consumption.

In theory, the *after-MBO* condition could also have been impacted negatively by expectancy effects. If participants expected to feel extreme hangover symptoms, then this may have confounded their task performance. On the other hand, participants in this group were selected due to the frequency of their MBO experiences, and therefore familiarity with the consequences of their drinking behaviour. Anecdotally, individuals in this group reported a mix of next-day symptoms when they arrived in the laboratory, but all had instigated their next-day testing session in advance, suggesting that they believed they would be capable of attending the laboratory and completing the studies. This could be said to point to expectancy of a return to normal functioning, rather than an expectation of impairment. We can only venture this as a possibility, but it could in part explain some of the variability between individuals in *after-MBO* effects.

The influence of bias

Social norms and public health campaigns suggest that alcohol is harmful, and therefore hypotheses and results presented in this thesis reflect this starting position. Indeed, the position presented here is no different from the majority of alcohol research. Studying the effects of alcohol with the expectation of finding alcohol-related harm is arguably less concerning than some other positions of bias. There is an extensive literature, from a variety of fields, which converge to link the consumption of alcohol to detrimental health and social consequences. For example, cancer (LoConte et al., 2018), Korsakoff's syndrome, alcohol related dementia (R. Rao & Topiwala, 2020), liver cirrhosis (Ohashi et al., 2018), and foetal alcohol spectrum disorders (Roozen et al., 2018) are examples of physiological harms; while family violence (Dostanic et al., 2022) and unemployment (Jørgensen et al., 2017) are instances of societal consequences. While there have been arguments which suggest a specific quantity or type of alcohol can offer protective effects (Hamaguchi et al., 2012; Li et al., 2022; Scott et al., 2013), and careful marketing tactics from the alcohol industry which adopts marketing strategies designed to minimise or manipulate the availability and accessibility of information surrounding health-related harms (Petticrew et al., 2020), these are arguably outweighed by the substantial body evidence to the contrary (for example, Chosy et al., 2022; Goel et al., 2018; Yoon et al.,

2020). In sum, beginning from a position that alcohol will have negative consequences is justifiable based upon the vast literature highlighting alcohol harms. Nonetheless, the studies presented were all looking for differences between control and experimental participant groups. Finding no differences may suggest that, at least during young adulthood, there are measures in which frequently experiencing MBOs may not cause noticeable harm. However, arguably when a difference between groups is found, we have to be careful with interpretations of the cause of the effects – is it always due to the negative impacts of alcohol? Throughout the thesis care has been taken to interpret findings in the light of previous evidence, rather than from an inherently biased position, even though I acknowledge that these biases are unavoidable.

It could be suggested that the work itself is inherently biased due to self-selection of participants, that is, people who are willing to take part in alcohol studies are more likely to drink alcohol in the first place, thereby producing a biased sample of drinkers. The specific design and questions being asked in this thesis demanded the inclusion of non-drinkers, or infrequent social drinkers, in addition to participants who drank heavily enough to elicit blackouts. Therefore, our population of interest did self-select, yet it is unknown whether these samples are a fair representation of the population of people who blackout heavily, or on the other hand, do not drink. Clearly, the ability to recruit individuals to our control group, and the range and volume of responses given by participants in the questionnaire more generally, speaks to the initial success of the recruitment strategy. Our questionnaire appeared to capture a full range of drinking behaviours from tee-total to people who may be alcohol dependent (see Figure 8.2). Interestingly, in order to undertake the laboratory studies, there was a requirement to recruit from the group of non, or infrequent, drinkers which posed challenges. For example, some control participants who took part in the sober study presented in Chapter 6 then refused invitations to take part in one of the other experiments as they were unwilling to drink alcohol for religious or health reasons. Nevertheless, the experimental chapters do include groups of participants who either did not drink, or drank infrequently, therefore the comparisons made with frequent binge-drinkers were reliable.

Alcohol consumption is the norm in many cultures around the world, meaning that drinking to excess, that is, until blackout, also becomes normalised in society. Therefore, the baseline for assessing normal cognitive functioning in western society is derived from people (an unknown proportion) who may have in the past engaged in extreme binge drinking practices. Our data speak more to the extremes of a western population: we sampled from the tails of a normal population to find people who do not (or rarely) drink in comparison to people who blackout frequently, not from the average person in the middle of the distribution. Would we still find differences between our MBO group and the 'normal', a sample of people from the middle of the distribution (for example, who consume alcohol regularly and who may have experienced a low volume of MBO events)? While it is important to keep in mind that cultural and societal norms may be affecting what we consider to be normal (behaviour, cognition, brain health) in relation to alcohol, our research highlights a need for longitudinal examinations of the impacts of extreme binge-drinking events and MBOs.

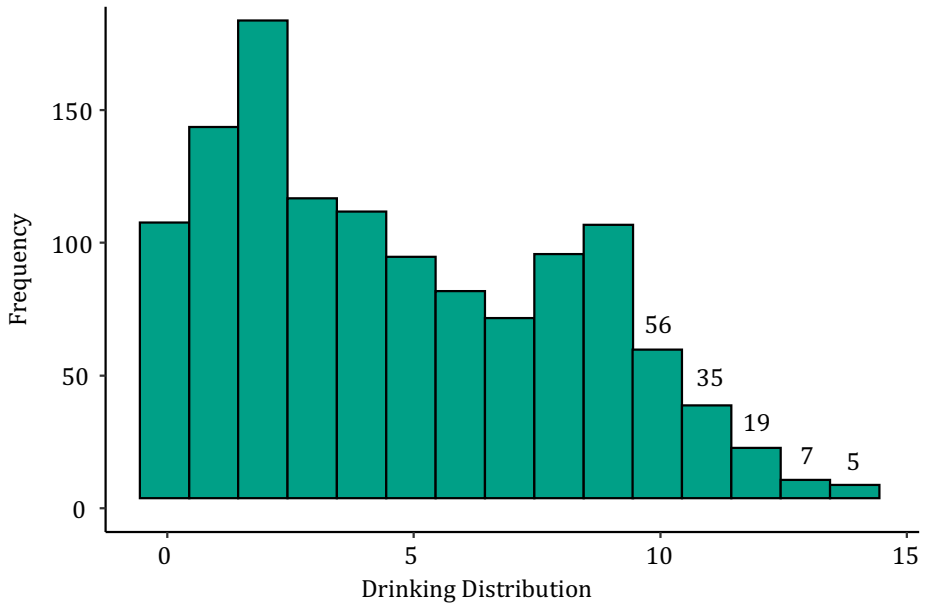

Figure 8.2: **Drinking patterns of questionnaire respondents.** Participants were asked to indicate how many times they would have a drink in a normal month, how often they had been drunk in the past year, or if they drank at all. Responses were summed to create a metric of drinking frequency, with scores ranging from 0 to 14 (*n* = 1199). Numbers above bars indicate frequency of participants in the top third of possible scores; for a score of 14, participants would have answered that they were

drinking on more than 30 occasions in a normal month, and were drunk more than 30 times in the past year. While data is positively skewed and bimodal, it does suggest that our participants spanned the full range of possible drinking behaviours from abstinence to heavy episodic drinking.

Impact of the Covid-19 Pandemic

The data for this thesis was collected across 2018 and 2019, prior to the Covid-19 pandemic. Similar to many other research projects, this has shaped the final presentation of this thesis. For example, and as previously discussed, it would have been advantageous to collect additional participants for the study presented in Chapter 7 in order to build a more powerful, useable dataset, however government restrictions made this impossible within the timeframe. Further, how the pandemic has altered drinking behaviour patterns is still being investigated, and indeed the full extent of any lasting influences will not be known for years to come. While the Covid-19 pandemic may alter our perception of the findings from Chapter 3, arguably these patterns of behaviour are beginning to return. Nor does this alter the results presented in Chapters 4 – 7, data included here reflects the differences in performance between those who frequently blackout, and those who do not.

During government mandated stay-at-home periods, some interesting yet contradictory findings relating to binge-drinking and MBOs emerged. Questionnaire studies which investigated changes in alcohol use behaviours within student populations worldwide found an overall decrease in binge-drinking (Bonar et al., 2021; Evans et al., 2021; Tavolacci et al., 2021; van Hooijdonk et al., 2022; Vasconcelos et al., 2021). However, some other researchers found evidence which suggested a general increase in alcohol consumption. For example, an analysis of tweets which referenced MBOs during March and April 2020 found an increase in mentions of alcohol-induced blackouts (Ward et al., 2021). Studies of middle-aged adults in the UK (Daly & Robinson, 2021), and a review of Covid-19 alcohol consumption literature (Xu et al., 2021), both found evidence for increased drinking within certain groups. Differences between the frequent and/or extreme binge-drinking noted in student populations, and drinking patterns in other adult groups, may reflect the way alcohol is used with university and college settings. As a social bonding tool, students rely heavily on alcohol. However, when opportunities for gathering in a social setting were

removed, so was the binge-drinking behaviour (Valente et al., 2021). At time of writing, although most restrictions have been lifted within the UK, many general patterns of behaviour from before the pandemic have not fully returned, that is, working and studying from home is still common, and social gatherings are less frequent. We cannot predict with certainty that there will be no lasting changes to drinking behaviour, or student drinking behaviour, post-pandemic. Arguably, student drinking behaviour is likely to continue in a similar vein as before, inspired by the expectations of the 'student experience' and the increased opportunities for socialising. Further, it is possible that those who endured restrictions on movement at influential life stages, for example leaving school, may respond by embracing student life more enthusiastically than they might otherwise have done in response to earlier missed experiences. These potential outcomes remain to be seen.

Monitoring drinking behaviour in all age groups remains crucial both for informing public health policies, and for health service planning. Large-scale shock, or disruptions, to life have been associated with problematic drinking several years later. For example, evidence following both the 9/11 attack (Welch et al., 2014) and the 2003 SARS outbreak (Wu et al., 2008) showed an increase in problematic alcohol consumption in individuals associated with those events up to six years later. While both events were internationally publicised and hugely shocking, arguably they would have directly impacted on fewer individuals than the global COVID-19 pandemic. Feelings of extreme trauma have been recorded worldwide by millions of healthcare workers since 2020. These people faced a deadly, novel virus, which they could not avoid due to their career choices. Other essential sectors similarly had to continue to work throughout, and these groups are in addition to the unimaginable numbers of people who have lost friends and family members, or who are suffering the effects of long-COVID. The potential for an unprecedented and enduring mental-health crisis as we move beyond the pandemic should not be underestimated. Furthermore, since we know that alcohol is a tool often used by those coping with trauma (Back & Jones, 2018; Guinle & Sinha, 2019; Moustafa et al., 2021), there is a very real danger of an alcohol misuse crisis emerging in the next few years. These dangers emphasise the importance of continued research into the cognitive effects of extreme binge-drinking and MBOs on cognition, in young adults and also across the lifespan.

Future directions

Research into the potential cognitive harms caused by alcohol-induced memory blackouts has been scarce, despite academic interest stretching back to the 1940s. This lack of available literature to build upon, and the ethical constraints rightly imposed on 21st century research design, necessitated a creative yet back-to-basics approach for this thesis. The research questions posed, and experimental methods developed, were therefore intended to address some important gaps in knowledge, while creating a platform for future research to expand from. The deceptively simple experiments did indeed address the initial questions, but also opened avenues for future investigation. For example, it is important to try and separate any lasting, if temporary, memory deficits seen after an MBO from general hangover effects. If these repeated, self-inflicted amnesic events do indeed leave a lasting neural mark or vulnerability on normal memory processing beyond what may be expected with a hangover, then it is critical to fully understand what this harm means for both immediate cognitive functioning, and future brain health. Does a single acute MBO experience cause a permanent scar? How frequently do individuals have to experience MBOs before lasting harm is visible? It may be that, without lifetime longitudinal studies, answers to such questions are not possible. Or, potentially inferences may be made by examining data from existing large datasets, such as the ABCD study in the USA, or the UK Biobank. Using datasets with include neuroimaging acquired from the same individuals over time may also help to uncover whether MBOs which only occur during late adolescence and early adulthood are less damaging than those which happen later in life due to potential protective effects offered by neural plasticity. Alternatively, it may be that greater impairments are caused than if the behaviours happened later. Of course, without accompanying behavioural testing, we may only be able to note changes in grey or white matter and extrapolate potential consequences of structural differences from there.

Innovative wearable tools which can track the rise and fall of the BAC curve are in development (Tehrani et al., 2022), and offer a range of new investigative and experimental options. Enabling individuals to wear devices, as they would a smartwatch or health tracker, means a stream of available data in real time while people are engaging in drinking behaviours naturally. Data of this type could offer a more detailed picture of the alcohol blackout, and answer some of the questions raised

in this thesis. For example, at what BAC do blackouts typically occur, is it the same in all individuals, do *fragmentary* and *en bloc* blackouts have different thresholds or are they the same (see Figure 8.1)? Further, they would allow the tracking of the descending BAC limb, helping to pinpoint the time when memory processing comes back online. Coupling technological approaches, for example regular prompting from a smartwatch to attend to a behavioural task on your phone, alongside wearable trackers, offers many opportunities to help understand the course of MBOs outside of the laboratory.

Analysis of biomarkers may help explain the chemical underpinnings of blackouts, and also why memory remains impaired following a blackout once the drinker is sober. For example, there may be an increased quantity of glutamate which continues to act as an antagonist on NMDA receptors in the hippocampus (Banerjee, 2014; J. H. Krystal et al., 2003). Investigations into the neurobiology of blackouts is becoming of increasing interest due to recent findings of the effects of binge drinking in adolescence. For example, binge-drinking in adolescence has been shown to reduce brain glucose metabolism with adverse consequences for multiple brain regions (Rapp et al., 2022), and may increase the chance of developing Alzheimer's disease in later life (A. Barnett et al., 2022), measured by the increase in proinflammatory signalling and proteins in mice. Understanding the neurobiological effects of MBOs may therefore offer both a method to track the trajectory of a blackout, and the potential longer lasting consequences for health.

If memory processing remains compromised following a blackout, this raises the question of how, or whether, other cognitive functions may show enduring impairments. Although the design of our studies did not support reaction time measures, our EEG data in Chapter 7 did suggest a delay in memory processing following blackout. It is also possible that attention remains either delayed or reduced following blackout, as can be seen in hangover studies of sober individuals (McKinney et al., 2012). The law relies on BAC, usually converted from BrAC readings, to assess whether someone is capable of sober functioning. If deficits endure even after the point of sobriety, this has consequences for a range of human activities, for example, driving and witness testimony. Clarification of the point at which cognition returns to baseline performance is therefore essential for multiple purposes.

Finally, it must be acknowledged that our student population, along with other young drinkers, may also use other substances while drinking alcohol. An investigation into the relationships between alcohol and drugs was well beyond the scope for this thesis, the scale of the question could indeed be the whole focus for a future PhD project. However, it is important for future work to consider how certain classes of drugs interact with alcohol and whether they may increase the likelihood of experiencing an MBO. It may be that, for example, ketamine, because of its hypnotic properties, increases the severity of blackouts whereas nicotine, marijuana, or ecstasy have no effect (no hypnotic effects, or are known anti-hypnotics). The study of these associations could work in tandem with wider biomarker research to further develop models of the mechanisms underpinning blackouts, in addition to the immediate and lingering effects from MBOs.

Conclusions

This thesis began with a selection of research questions**,** and we will conclude by very briefly answering these directly.

1. **What are the psychological variables that influence young people's behaviour towards alcohol?**

Our questionnaire suggested that extreme binge-drinking which led to blackouts is influenced by home country but mediated by peers and personal drinking habits. Further, being in the second year of an undergraduate degree was associated with increased peer influenced social drinking, and being male increased both the number of blackouts experienced, and personal drinking behaviours in general.

2. **What is the frequency and prevalence of MBOs in a student population?**

Within our Scottish based student population, 81% regularly drank alcohol, with a 90% 12-month period prevalence of binge-drinking. Lifetime prevalence of MBOs was 75% within our sample. In more detail, this reflected a 61.5% 12-month period prevalence of *fragmentary* blackouts, and 38.7% of *en bloc* blackouts.

3. **What is the impact to the brain of an MBO, and does alcohol impart any lasting effects on future memory formation?**

After-blackout, some deficits in memory functioning remain even once sober, specifically in tasks which require greater focus and cognitive effort. ERPs suggest that there may be a delay in neural processing compared to *before-alcohol* conditions. Further, and in contrast to Controls, blackout individuals show divergent neural indices of memory, suggesting that memory may be operating fundamentally differently after multiple blackout events. This coincided with no significant behavioural differences in memory performance between our Control and MBO groups when sober (assuming no blackout the preceding evening).

4. Are people who experience a high frequency of MBOs from binge drinking more susceptible to the effects of alcohol than those who don't experience blackouts?

We found little evidence to suggest that our experimental group were more susceptible to alcohol effects in our *after-alcohol* conditions as between group analyses did not differ across behavioural measures. However, we did find that neural strategies may differ between groups in memory processing tasks.

In sum, this thesis has shown a high prevalence of binge-drinking in students who attend Scottish universities, and that a substantial proportion of these individuals regularly experience alcohol-induced memory blackouts following extreme binge-drinking events. This behaviour may be impacted by Scottish culture, but also reflects the home country of individuals. While alcohol consumption impairs performance in both recall and recognition memory tasks, there is a delayed recovery to baseline memory performance in the aftermath of an MBO, even once sober. This becomes more pronounced as task demands increase. Finally, there is a shift in neural memory strategy between individuals who experience frequent memory blackouts and Controls, despite behavioural performance before and *after-alcohol* reaching similar levels. The results of this thesis form a solid foundation for future research into alcohol-induced memory loss, potentially allowing us to establish (1) the duration of *after-MBO* effects, (2) how blackouts and hangovers interact, (3) a more in-depth insight of the trajectory of blackouts through possible biomarker use, and (4) the long-term impacts of a lifetime of blackout experiences. Memory defines our whole being,

our conscious self, and to lose even a part of our memories therefore changes who we are; in the case of alcohol, this change is rarely for the better.

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Appendices

Appendix 1.1

Between Group Analysis Model Output Tables (Chapter 4)

Table S1:1 Free Recall

Model specification	Model name	Nested Model	Fixed Effects	Model Comparison	Model fit					
					df	AIC	BIC	LogLik	L. Ratio	p-value
Mean Accuracy (%)	Baseline				5	856.101	869.4182	-423.0505		
	freel1	Baseline	+ Group	free1 to Baseline	6	858.0992	874.0799	-423.0496	0.00175	0.9666
	free2	free1	+ Condition	free2 to free1	7	796.1364	814.7804	-391.0682	63.9629	<.0001
	free3	free2	+ Group:Condition	free3 to free2	8	797.9142	819.2217	-390.9571	0.22212	0.6374

free2 model equation: ***MeanACC ~ Group + Condition, random = ~1|Participant/Group/Condition***

Model Summary	df	Beta	SE	t-value	p-value
Intercept	52	51.35	1.27	40.29	0
Group	51	0.05	1.27	0.042	0.9667
Condition	52	-7.87	0.72	-10.976	0

Table S1:2b Serial Recall – Mean Sequence Length

Model specification	Model name	Nested Model	Fixed Effects	Model Comparison	Model fit					
					df	AIC	BIC	LogLik	L. Ratio	p-value
Mean Sequence Length (%)	Baseline				5	355.6559	368.9731	-172.8279		
	serialseq1	Baseline	+ Group	serialseq1 to Baseline	6	357.084	373.0646	-172.542	0.571946	0.4495
	serialseq2	serialseq1	+ Condition	serialseq2 to serialseq1	7	351.8829	370.527	-168.9415	7.201037	0.0073
	seriaseql3	serialseq2	+ Group:Condition	serialseq3 to serialseq2	8	353.6076	374.9151	-168.8038	0.275324	0.5998

serialseq2 equation: ***MeanSEQ ~ Group + Condition, random ~1\|Participant/Group/Condition***

Model Summary	df	Beta	SE	t-value	p-value
Intercept	52	3.176331	0.14166303	22.421734	0
Group	51	0.105894	0.14166303	0.747508	0.4582
Condition	52	-0.267363	0.09766057	-2.737676	0.0085

Table S1:3 Depth of Encoding

Model specification	Model name	Nested Model	Fixed Effects	Model Comparison	Model fit				
					df	AIC	BIC	LogLik	L. Ratio	p-value
Mean Accuracy (%)	Baseline				7	3381.735	3410.083	-1683.868		
	depth1	Baseline	+ Group	depth1 to Baseline	8	3383.703	3416.101	-1683.851	0.03222	0.8576
	depth2	depth1	+ Condition	depth2 to depth1	9	3316.024	3352.472	-1649.012	69.6783	<.0001
	depth3	depth2	+ Delay	depth3 to depth2	10	3162.999	3203.497	-1571.5	155.02521	<.0001
	depth4	depth3	+ Depth	depth4 to depth3	11	3159.768	3204.315	-1568.884	5.23107	0.0222
	depth5	depth4	+ Group:Condition	depth5 to depth4	12	3157.638	3206.234	-1566.819	4.13057	0.0421
	depth6	depth5	+ Group:Delay	depth6 to depth5	13	3159.223	3211.87	-1566.612	0.41433	0.5198
	depth7	depth6	+ Group:Depth	depth7 to depth6	14	3158.468	3215.164	-1565.234	2.75568	0.0969
	depth8	depth7	+ Condition:Delay	depth8 to depth7	15	3151.499	3212.245	-1560.75	8.96828	0.0027
	depth9	depth8	+ Condition:Depth	depth9 to depth8	16	3147.175	3211.971	-1557.588	6.32388	0.0119
	depth10	depth9	+ Delay:Depth	depth10 to depth9	17	3148.883	3217.728	-1557.441	0.29285	0.5884
	depth11	depth10	+ Group:Delay:Depth	depth11 to depth10	18	3150.72	3223.615	-1557.36	0.16248	0.6869
	depth12	depth11	+ Group:Condition:Delay	depth12 to depth11	19	3151.923	3228.868	-1556.961	0.79721	0.3719
	depth13	depth12	+ Group:Condition:Depth	depth13 to depth12	20	3152.178	3233.173	-1556.089	1.7446	0.1866
	depth14	depth13	+ Condition:Delay:Depth	depth14 to depth13	21	3154.169	3239.213	-1556.084	0.00937	0.9229
	depth15	depth14	+ Group:Condition:Delay:Depth	depth15 to depth14	22	3155.359	3244.453	-1555.68	0.80973	0.3682

depth9 equation: ***MeanACC ~ Group + Condition + Delay + Depth + Group:Condition + Group:Delay + Group:Depth + Condition:Delay + Condition:Depth, random = ~1|Participant/Group/Condition/Delay/Depth***

Model Summary	df	Beta	SE	t-value	p-value
(Intercept)	209	31.76425	1.603	19.815326	0
Group	51	-0.28436	1.603	-0.177393	0.8599
Condition	51	-8.67996	0.6954	-12.482079	0
Delay	103	-5.91056	0.3594	-16.444148	0
Depth	209	0.77886	0.3594	2.166903	0.0314
Group:Condition	51	1.42421	0.6954	2.048063	0.0457
Group:Delay	103	0.23527	0.3594	0.654568	0.5142
Group:Depth	209	0.60524	0.3594	1.683889	0.0937
Condition:Delay	103	-1.07704	0.3578	-3.009937	0.0033
Condition:Depth	209	-0.89361	0.3578	-2.497298	0.0133

Appendix 2.1

Within MBO Group Analysis Model Output Tables (Chapter 4)

Table S2:1 Free Recall

Model specification	Model name	Nested Model	Fixed Effects	Model Comparison	Model fit					
					df	AIC	BIC	LogLik	L. Ratio	p-value
Mean Accuracy (%)	Baseline				4	648.7356	658.3134	-320.3678		
	freel1	Baseline	+ Condition	free1 to Baseline	6	618.9457	633.3124	-303.4728	33.78991	<.0001

free1 model equation: ***MeanACC ~ Condition, random = ~1|Participant/Condition***

Model Summary	df	Beta	SE	t-value	p-value
Intercept	50	52.00168	1.649031	31.534697	0
Condition - Before/After	50	-8.17027	1.345111	-6.074048	0
Condition - Before/AfterMBO	50	1.19685	1.448267	0.826405	0.4125

Table S2:2 Serial Recall: Mean Accuracy

Model specification	Model name	Nested Model	Fixed Effects	Model Comparison	Model fit					
					df	AIC	BIC	LogLik	L. Ratio	p-value
Mean Accuracy (%)	Baseline				4	633.8919	643.4697	-312.946		
	serial1	Baseline	+ Condition	serial1 to Baseline	6	611.9743	626.341	-299.9872	25.9176	<.0001

serial2 equation: ***MeanACC ~ Condition, random = ~1|Participant/Condition***

Model Summary	df	Beta	SE	t-value	p-value
Intercept	50	46.47487	1.834484	25.334027	0
Condition - Before/After	50	-6.78183	1.186782	-5.714473	0
Condition - After/AfterMBO	50	-3.98512	1.282463	-3.107396	0.0031

Table S2: 3 Depth of Encoding

Model specification	Model name	Nested Model	Fixed Effects	Model Comparison	Model fit					
					df	AIC	BIC	LogLik	L. Ratio	p-value
Mean Accuracy (%)	Baseline				6	2557.885	2580.569	-1272.942		
	depth1	Baseline	+ Condition	depth1 to Baseline	8	2518.357	2548.602	-1251.178	43.52814	<.0001
	depth2	depth1	+ Delay	depth2 to depth1	9	2417.606	2451.633	-1199.803	102.75014	<.0001
	depth3	depth2	+ Depth	depth3 to depth2	10	2408.377	2446.184	-1194.188	11.22965	0.0008
	depth4	depth3	+ Condition:Delay	depth4 to depth3	12	2405.505	2450.874	-1190.753	6.8715	0.0322
	depth5	depth4	+ Condition:Depth	depth5 to depth4	14	2402.185	2455.115	-1187.092	7.32071	0.0257
	depth6	depth5	+ Delay:Depth	depth6 to depth5	15	2403.769	2460.48	-1186.884	0.4158	0.519
	depth7	depth6	+ Condition:Delay:Depth	depth7 to depth6	17	2407.445	2471.717	-1186.722	0.32416	0.8504

depth5 equation: ***MeanACC ~ Condition + Delay + Depth + Condition:Delay + Condition:Depth, random = ~1|Participant/Condition/Delay/Depth***

Model Summary	df	Beta	SE	t-value	p-value
(Intercept)	159	29.528623	1.9802001	14.911939	0
Condition - After/Before	50	9.207009	1.1433623	8.052574	0
Condition - After/AfterMBO	50	-3.902524	1.2371711	-3.154393	0.0027
Delay - Delay/Immediate	78	-5.031512	0.4179765	-12.037786	0
Depth - Deep/Shallow	159	-1.438031	0.4179765	-3.440459	0.0007
Condition After/Before:Delay	78	0.146455	0.5791653	0.252872	0.801
Condition After/AfterMBO: Delay	78	1.287551	0.6142975	2.095972	00393
Condition After/Before:Depth	159	-1.263118	0.5791653	-2.180929	0.307
Condition After/AfterMBO:Depth	159	-0.107863	0.6142975	-0.175587	080

Appendix 3.1

ERPs across traditional time-windows (Chapter 6)

Our data driven strategy for ERP analysis in Chapters 6 was informed by the distribution of the ERP effects in both time window and electrode site, which differed from a number of studies of recognition memory. Therefore, for consistency, we include here an analysis of the mid-frontal effect using a 300-500ms time window at electrode sites F1, F3, & Fz, and of the parietally distributed effect between 500 and 800ms at P1, P3 and P5, which mirrors previously published literature in the field.

Mid-frontal effect between 300-500ms.

A repeated measures ANOVA with factors of Response Difference (*HH-CR, HM-CR*) and electrode site (F1, F3, Fz) was conducted on Control group mean amplitudes over the 300-500ms time-window. No effect of difference was found (p = .430). A follow-up repeated measures ANOVA with factors of Response Type (*HH, HM* and *CR*) and electrode site (F1, F3, Fz) also found no effect of response (p = .617).

The analysis was repeated for the MBO group data. A repeated measures ANOVA with factors of Response Difference (*HH-CR, HM-CR*) and electrode site (F1, F3, Fz) failed to find an effect of difference (p = .390). A final ANOVA conducted on Response Type (HH, HM, CR) and electrode site (F1, F3, Fz), again did not find a difference between responses (p =.296).

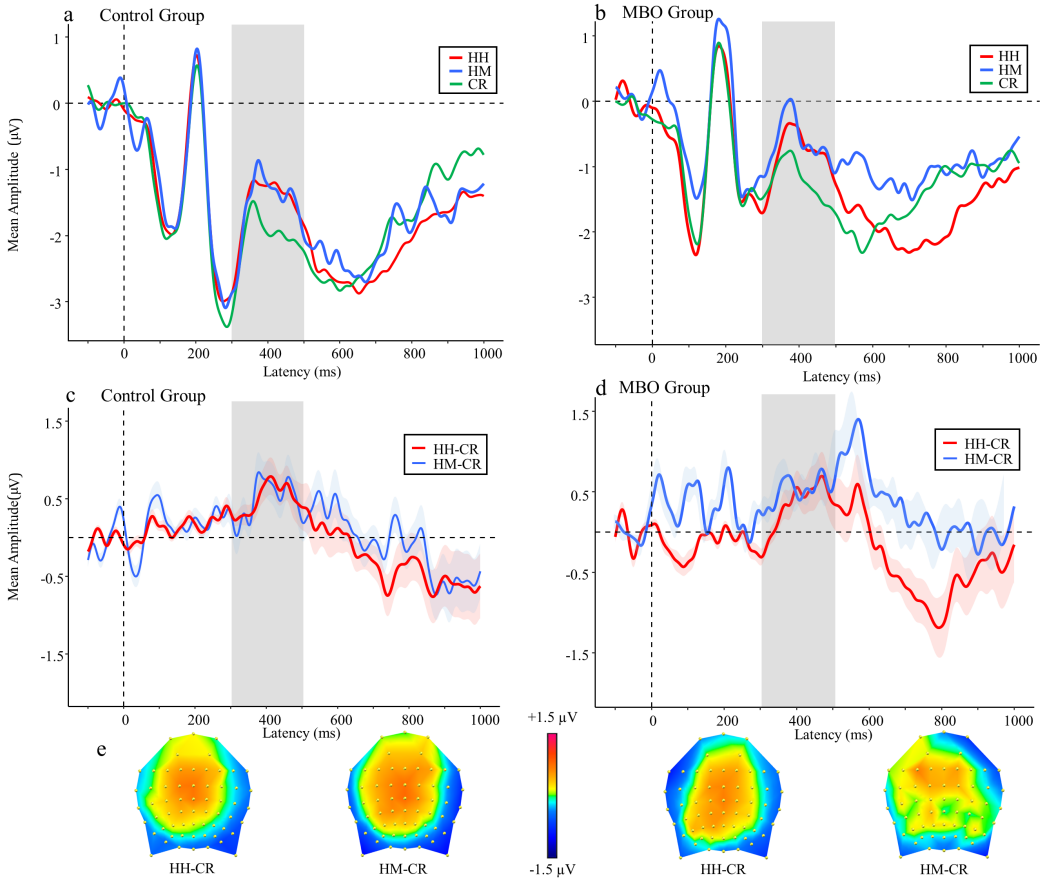

Figure S3.1: **Electrophysiological responses, difference waves, and difference topographies highlighting the 300-500ms time window.** (a) & (b) display averages of electrode sites F1, F3 and FZ, for ERP responses from the Control and MBO groups respectively. Grey shaded regions represent the 300-500ms time window of analysis; (c) & (d) show difference waves of *HH-CR* and *HM-CR* responses for both groups; and (e) shows difference topographies (nasion at top of maps).

Parietally distributed effect between 500-800ms.

Figure 6.3A&B in Chapter 6 displays ERPs over left parietal electrode sites (P1, P3, P5) whereas Figure S3.2 below shows the difference topographies for *HH-CR* and *HM-CR* responses in both Control and MBO groups across the 500-800ms time window. In the Control group, a repeated measures ANOVA, with factors of response differences (*HH-CR* vs. *HM-CR*) and electrode site (P1, P3, P5), showed no significant difference between *HH-CR* and *HM-CR (p*= .154*)*. In contrast, the MBO group displayed a difference between *HH-CR* and *HM-CR*, $F(1,19) = 6.233$, $p = 0.022$, $n^2_p = 0.247$; mean amplitude for *HH-CR* ($M = 0.616$, $SE = 0.267$) was significantly greater than for *HM-CR* responses ($M = 0.026$, $SE = 0.249$). The Control group show a graded pattern for responses (*HH>HM>CR*) over the left hemisphere, confirmed by statistical analysis

which found no difference between *HH* and *HM* (p = .463), nor between *HM* and *CR* (p = .504). *HH* was, however, significantly greater in amplitude than *CR*, $t(20) = 2.7$, p = .042. The MBO group did not display a similar graded pattern; *HH* trended towards a statistical difference to *HM* (p = .066), and also towards *CR* responses (p = .098), but critically, no difference found between *HM* and *CR* responses (p = 1). Mean amplitudes showed that while *HM* (M = 0.451, SE = 0.357) and *CR* (M = 0.426, SE = 0.283) were similar, *HH* was greater (M = 1.042, SE = 0.366) suggesting that individual variability across the time window masked differences in response amplitude.

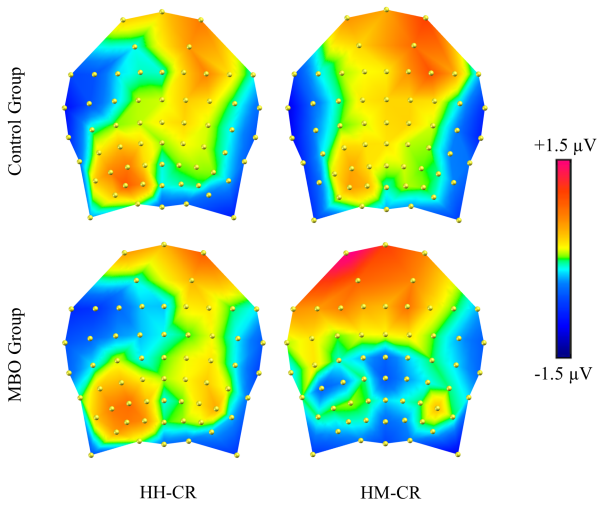

Figure S3.2: **Difference Topographies.** Across 500-800ms, topographies (nasion at the top of the map) showing Maximal differences at left parietal electrodes (*HH-CR*, and *HM-CR*) for both Control and MBO group.

Appendix 3.2

Global variance explained by microstate

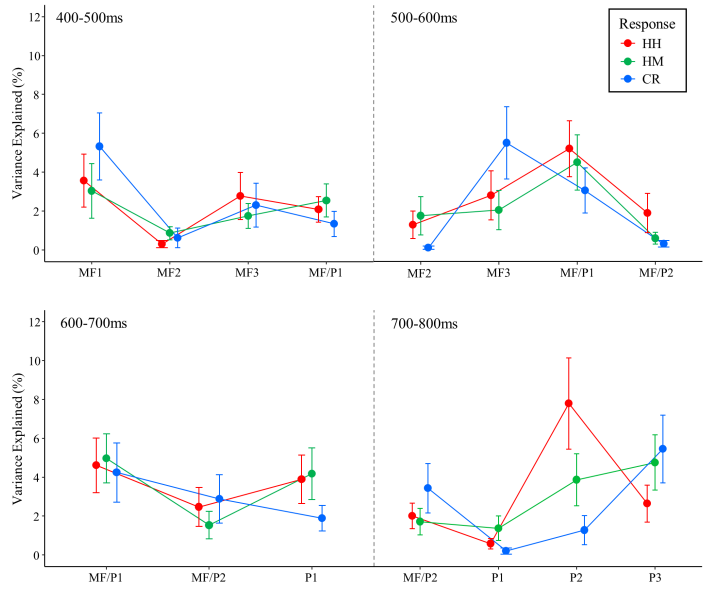

Figure S3.3: **Global Explained Variance.** Within the Control group, changes in global explained variance in 100ms periods, across 400 – 800ms. Variance split by response type and relevant microstate.

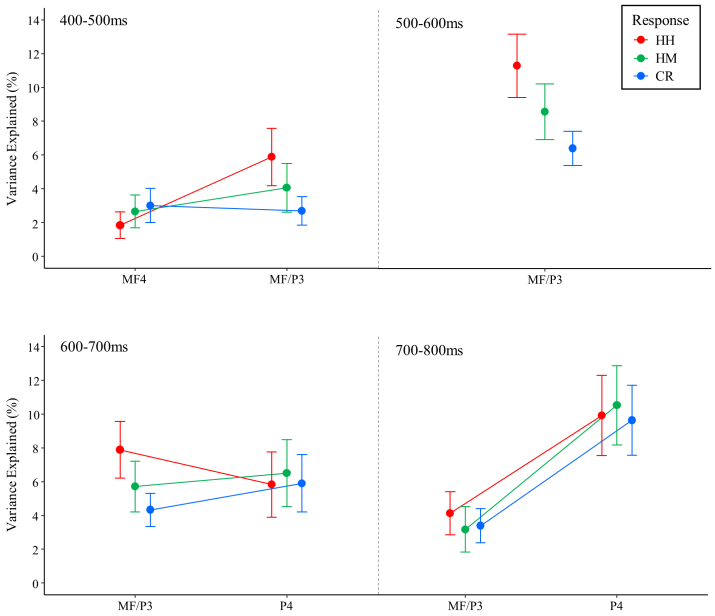

Figure S3.4: **MBO Group Global Explained Variance.** As for Figure S3.3, but for the MBO group.

Appendix 4.1

Comparison of same MBO group individuals *before-alcohol*, *after-alcohol* and *after-MBO* (Chapter 7)

Participants from the MBO group in Chapter 7 were invited to return to the lab following an alcohol-induced memory blackout (<20 hours) and to repeat the study while sober but still recovering from a blackout event. Of the 17 in the initial blackout group, all returned to complete the study again. Of these individuals, data from 12 are presented here (3 males, mean age = 19.67, SD = 1.15; mean number of days between testing sessions = 43.92, SD = 33.21). Please note, 3 participants completed Study 2 in Chapter 7 (*after-MBO* condition) before completing Study 1 (*before* and *after-alcohol*). Remaining participants were excluded due to insufficient trial numbers (>16).

Results

Behavioural Results

Repeated measures ANOVAs were conducted on percentage accuracy for *Hit Hit, Hit Miss,* and *False Alarm* scores between alcohol conditions. A main effect of alcohol condition, $F(2,22) = 5.82$, $p = .009$, $n^2_p = .346$, and of response type, $F(2,22) = 43.23$, $p < .001$, $n^2_p = .797$, was found with no interaction ($p = .390$). Means for each behavioural measure, per alcohol condition, are presented in Table S4.1, and differences between responses within alcohol conditions are given in Table S4.2.

Table S4.1:

Behavioural Mean Accuracy (standard error)

	Before Alcohol	After Alcohol	After MBO
Hit Hit %	42.43 (4.23)	36 (4.33)	42.5 (3.79)
Hit Miss %	27.9 (1.87)	22.6 (1.87)	24.2 (1.93
Correct Rejection%	80.2 (4.34)	78.5 (5.39)	76.4 (5.9)
Source Accuracy %	59.16 (2.92)	60.24 (2.35)	62.90 (2.18)
dPrime	0.41 (0.28)	-0.38 (0.22)	-0.02 (0.29)
C	-0.1 (0.22)	0.1 (0.29)	-0.1 (0.27)

Table S4.2:

Bonferroni corrected post hoc significant effects

	Before Alcohol	After Alcohol	After MBO
HH – CR	$t(11) = -5.57$, $p = .006$	$t(11) = -4.76$, $p = .02$	$t(11) = -4.37$, $p = .04$
HM - CR	$t(11) = -9.56$, $p < .001$	$t(11) = -8.48$, $p < .001$	$t(11) = -6.78$, $p = .001$
HH - HM	n/a	n/a	$t(11) = 4.93$, $p = .02$

Participants did not differ in their source accuracy between any of the alcohol conditions, nor in response bias *C*. Ability to discriminate between old and new words (*dPrime*) did drop between the *before-alcohol* and *after-alcohol* conditions, $t(11) = 5.74$, $p = .005$, however there was no difference between *before-alcohol* and *after-MBO* ($p = 1$), nor *after-alcohol* and *after-MBO* ($p = 1$) suggesting variability within the group.

ERP Results

Across the 400-600ms time window, repeated measures ANOVAs were conducted on both the factors of response (*HH, HM, CR*), alcohol condition, and electrode (C1, C2, Cz). No meaningful main effects, nor interactions were, found. A further ANOVA this time with *HH-CR* and *HM-CR* included also found no meaningful differences. This was replicated for the 500-700ms time window, with electrodes P1, P3 and P5. No meaningful differences either between responses, or difference waves, were found.

In summary, analysis of MBO group behavioural and ERP data showed that response accuracy dropped *after-alcohol* but improved *after-MBO*. There were no significant differences in ERP mean amplitudes.

Appendix 5.1

Questionnaire (Chapter 3)

Your unique ID number is ${q://QID152/ChoiceTextEntryValue} Consent

Alcohol and Students – Drinking Behaviours and the Consequences for Memory You are invited to participate in a two-part online survey.Firstly, a comprehensive questionnaire investigates alcohol behaviours, family history of drinking behaviours and memory loss due to drinking.Secondly, there is a shorter, follow-up survey which asks you to record your drinking behaviours over the past 7 days. You have been invited to participate because you are an undergraduate student attending a Scottish university, and are within the age range of interest.The project is being conducted by Judith Jackson, a PhD Researcher at the University of Stirling. You will be asked to answer some multiple-choice questions, and a few short answer questions, which should take approximately 20 – 25 minutes to complete.

Please read through these terms before agreeing to participate below.

1. Background, aims of project

This survey is a first step to studying the attitudes and behaviours towards alcohol within undergraduate students attending Scottish universities.We are particularly interested in what factors influence your relationship with alcohol, and the typical drinking behaviours which form part of the 'student experience'.Following this survey, we plan to invite a small group of participants from the University of Stirling to take part in some laboratory based studies which will be designed to deepen our understanding of the effects of alcohol on memory.

2. Do I have to take part?

No. Your participation in this survey is voluntary. You may refuse to take part in the research or exit the survey at any time without penalty by pressing the 'Exit' button or closing the browser. You are free to decline to answer any particular question for any reason, no questions will be mandatory.

3. Are there any potential risks in taking part?

Some of the survey questions ask about your family history of drinking behaviours and may be distressing to you as you consider your own experiences.Please remember that you can decline to respond to any question by simply leaving it unanswered and moving on.You can also exit the study at any time before completion.If you would like to talk to someone about any of the topics included in the questionnaire, there are contact details provided below.

4. Are there any benefits in taking part?

No, there are no benefits other than the knowledge that you are providing a hugely valuable contribution to our research.

5. Expenses/Payments

If you are a Psychology student in your first or second year of study at the University of Stirling, and you have entered this survey via Psychweb, you will receive 0.5 course credit tokens.Otherwise, you may be entered into a prize draw to win £50 Amazon voucher. If you would like to be entered into the draw, please leave a contact email address at the appropriate place in the survey.Email addresses will be downloaded separately from your survey responses and one will be selected at random on Friday 14th December 2018 .

6. Legal basis for processing personal data.

As part of the project, we will be recording personal data relating to you.This will be processed in accordance with the General Data Protection Regulations (GDPR).Under GDPR the legal basis for processing your personal data will be public interest.

7. What happens to the data I provide?

Your answers will be completely anonymous, and we will use all reasonable endeavours to keep them confidential. Your data will be stored in a passwordprotected file. Your IP address will not be stored.

Only the research team (Judith Jackson and Dr Benjamin Dering) will have access to research data.

If you wish to withdraw your data, you can do this for up to one month following the date you complete the survey.To do this, please contact the researchers with the randomly generated identifying number which will be displayed for you at the start of the survey.All your responses will then be destroyed. Your personal data will be kept until October 2020 and then will be stored on the University secure research server for up to 10 years.

8. Will the research be published?

The methods used to conduct this research, and any group-level results, will be published in a doctoral thesis and may also be published in an academic journal or conference paper. There will be no analysis of responses at the individual level, therefore you will not be individually identifiable from any published research.

9. Who has reviewed this research project?

This project has been ethically approved via The NHS, Invasive or Clinical Research Committee (NICR) at the University of Stirling.

10. Your rights.

You have the right to request to see a copy of the information we hold about you and to request corrections of deletions of the information that is no longer required. You have the right to withdraw from this project at any time without giving reasons and without consequences to you.You also have the right to object to us processing relevant personal data however, please note that after one month, or once the data are being analysed, and/or results published, it may not be possible to remove your data from the study.

11. Whom do I contact if I have concerns about this study or I wish to complain?

If you would like to discuss the research with someone, please contact:

Judith JacksonJudith.Jackson1@stir.ac.uk
Dr Benjamin Deringb.r.dering@stir.ac.uk
Professor Peter Hancockp.j.hancock@stir.ac.uk

You have the right to lodge a complaint against the University regarding data protection issues with the Information Commissioner's Office (https://ico.org.uk/concerns/). The University's Data Protection Officer is Joanna Morrow, Deputy Secretary. If you have any questions relating to data protection these can be addressed to data.protections@stir.ac.uk in the first instance.

Should you wish to speak to someone about the issues raised, please contact any of the researchers above or your University counselling service. For University of Stirling students, you can email student.counselling@stir.ac.uk.

Alternatively, you can contact:

Alcoholics Anonymous	help@aamail.org
Scottish Families Against Drugs	08080 10 10 11
Breathing Space	0800 83 85 87

Thank you for your participation. Your responses are exceptionally valuable to our research.

12. Electronic Consent

Please select your choice below. Clicking on the “Agree” button indicates that:

- I have been informed about the aims and procedures involved in the experiment I am about to participate in.
- I know that my data will be securely stored, and any identifying information will be removed from the data after collection.
- I reserve the right to withdraw at any stage in the proceedings. If I do so, I understand that any information that I have provided as part of the study will be destroyed unless I agree otherwise.
- I can leave unanswered any question which I feel uncomfortable answering, although I understand that my responses are valuable.
- I understand that I may be contacted and invited to take part in a laboratory based experiment. I am aware that there is no obligation to agree to do this.
- I voluntarily agree to participate
- I am between 18 and 25 years of age and consent to taking part in this survey.

◯ Agree
◯ Disagree

Identify

Thank you for agreeing to take part. Your randomly assigned participant number is 922612.

Please take a note of this number as you will need to quote this to the researchers should you wish to contact them in the future.

Please write the number provided in the box below.

Thank you!

If you are a University of Stirling student, and you would be happy to receive a followup email with an invitation to take part in a laboratory based study, please enter your email address here.

If you would like to be entered for the prize draw of £50 Amazon voucher, please enter your email address here. The prize draw will take place on Friday 14th December 2018 and the winner will be notified by email.

(n.b. University of Stirling Psychology students in 1st or 2nd year will receive 0.5 course credit tokens if the survey is accessed via Psychweb and are not eligible to be entered into the draw)

Block 3: Demographics

Please read carefully!

Part One

Please complete as many of the following questions as possible. Your responses will be confidential and securely stored spearately from any identifying information. While it would assist our research greatly for you to answer every question, please leave blank any that you are uncomfortable answering. No questions are compulsory.

Definition of drunk

When answering, think of being drunk as having lost some (but not necessarily all) control over behaviour following drinking alcohol. This could be noticed by someone saying things which they would not normally, by someone slurring words, by being unsteady on their feet or unusually clumsy. If it was you, you might feel ok, but notice you are unable to physically move or verbally respond in your normal fashion. You may also feel more emotional than usual or more confident than usual.

Date of Birth (Month and Year)

Gender

Ethnicity (i.e. Scottish, Asian British, White, African etc.)

Nationality (i.e. Scottish, British, Chinese etc.)

Who lives in your family home (i.e. the home you grew up in/lived in before coming to University, which may not be the household you live in during semester). Please

select all that apply.

- [ ] Mum
- [ ] Dad
- [ ] Step mum / Dad's partner
- [ ] Step dad / Mum's partner One
- [ ] brother
- [ ] One sister
- [ ] Two or more brothers
- [ ] Two or more sisters
- [ ] Grandmother
- [ ] Grandfather
- [ ] Other family member

If you selected other family member, can you tell us who?

If your family home is in the UK, can you tell us the first part of your postcode? (i.e. FK9, or SW19)

Where do you live during semester?

- ◯ In my family home (my permanent home) and I commute to Uni
- ◯ In halls/student accommodation on campus
- ◯ In a flat/house nearby the University
- ◯ Other

If you selected other, can you tell us where you live?

Are you a member of a sports team or sports society?

- ◯ Yes
- ◯ No
- ◯ Sometimes

If so, can you tell us which one(s)?

Which year of study are you in?

- ◯ 1st3rd 2nd4th
- ◯
- ◯
- ◯

What is your main degree?

Which university do you attend?

Block 4: Family History

Did any members of you immediate family (i.e. parents/step

parents/grandparents/siblings) regularly drink alcohol within your home(s) while you were growing up? (i.e. most weeks, they would have at least one alcoholic drink)?

- ◯ Yes
- ◯ No
- ◯ Not sure

If yes, which members did this? (Please select as many as apply)

- ☐ DadGrandmother
- ☐ MumGrandfather
- ☐ SisterOther family member
- ☐ Brother

Can you tell us who the family member is?

If one (or more) of these family members is a biological grandparent, which one(s) are they? (Please select as many as apply)

- ☐ Dad's dadMum's dad
- ☐ Dad's mumMum's mum

Which of your family members currently drinks most frequently within the family home?

- ◯ None of themBrother(s)
- ◯ DadGrandmother
- ◯ MumGrandfather
- ◯ Sister(s)Other

If you said other family member, can you tell us who?

Thinking of this family member, on how many days per week would they normally have an alcoholic drink?

- 15
- 26
- 37
- 4

In a normal week, on how many of these days would you say this family member was drunk?

- 1
- 26
- 37
- 4Not as often as once a week, only now and again
- 5

Do any other family members regularly get drunk when drinking in the home?

- NeverUsually RarelyAlways
- Sometimes

Would you say that anyone in your family has a problem with alcohol? (i.e. may be an alcoholic, or someone who drinks heavily on a regular basis)

- Definitely yes
- Possibly yes
- No
- Don't know

If so, can you tell us who that is?

- [ ] DadGrandmother
- [ ] MumGrandfather
- [ ] SisterOther family member
- [ ] Brother

Can you estimate how old you were when they started drinking heavily? (If more than one family member drinks heavily, pick the person who has been drinking heavily for the longest).

- ◯ Before I was born16 - 20 years old
- ◯ 0 - 5 years old21 + years old
- ◯ 6 - 10 years oldI don't know
- ◯ 11 - 15 years old

Do you any of your immediate family (over the age of 18) NEVER drink alcohol?

- ◯ None of them ever drink
- ◯ Some drink, but some don't
- ◯ All of them drink
- ◯ Not sure

If so, who doesn't drink at all? (select all that apply)

- [ ] DadGrandmother
- [ ] MumGrandfather
- [ ] SisterOther family member
- [ ] Brother

Have any of your immediate family members ever complained that they were so drunk that they couldn't remember things?

- ◯ Yes
- ◯ No
- ◯ Don't know

If so, can you tell us who? (select all that apply)

- ◯ DadGrandfather
- ◯ MumGrandmother
- ◯ SisterOther family member
- ◯ Brother
- ◯
- ◯
- ◯

If other, which family member?

Do any of your immediate family members get violent, verbally aggressive or psychologically abusive when drunk?

- ◯ UsuallyNever
- ◯ SometimesDon't know
- ◯ Rarely
- ◯
- ◯

If so, can you tell us who? (select all that apply)

- ☐ DadGrandfather
- ☐ MumGrandmother
- ☐ BrotherOther family member
- ☐ Sister
- ☐
- ☐
- ☐

Do any of your immediate family members have a history of psychiatric problems (i.e. depression, anxiety, bulimia, schizophrenia etc.)

- ◯ Yes
- ◯ No
- ◯ Don't know

If so, can you tell us who and what?

To the best of your knowledge, do any of your immediate family use other substances regularly? (i.e. cigarettes, marijuana, cocaine, anti-depressants etc.)

- ◯ Yes
- ◯ No
- ◯ Don't know

If so, can you tell us who and what?

Do you know if your mother drank at all while pregnant with you, even a little?

- ◯ Definitely yes
- ◯ Probably not
- ◯ Probably yes
- ◯ Definitely not
- ◯ Might or might not

Block 5: Peers and Culture

At what age do you think it's socially acceptable to start drinking alcohol?

- ◯ Under 12 years
- ◯ 18
- ◯ 13
- ◯ 19
- ◯ 14
- ◯ 20
- ◯ 15
- ◯ 21
- ◯ 16
- ◯ 22 or over
- ◯ 17

Do you think your friends would agree with this age?

- ◯ Definitely yesProbably not
- ◯ Probably yesDefinitely not
- ◯ Don't know

If not, how old do you think they would say is acceptable?

- ◯ Under 12 years18
- ◯ 1319
- ◯ 1420
- ◯ 1521
- ◯ 1622 or over

- ◯ 17

From a health perspective, at what age do you think it is acceptable to drink alcohol?

- ◯ Under 12 years18
- ◯ 1319
- ◯ 1420
- ◯ 1521
- ◯ 1622 or over
- ◯ 17
- ◯
- ◯
- ◯
- ◯
- ◯

Do you think it is acceptable to drink until you're drunk, rather than stopping after a couple?

- ◯ All the time, yes
- ◯ Sometimes, yes
- ◯ Not really, you should be able to control your drinking

How do you feel if one of your friends get really drunk? (select all that apply)

- ☐ I think it's funnyI feel scared for them
- ☐ I think they're coolI feel embarrassed
- ☐ I feel uncomfortableI don't mind, it
- ☐ I feel responsible for themOther I think
- ☐ disgusting
- ☐
- ☐
- ☐ doesn't bother me
- ☐ it's

If you said other, can you tell us how it makes you feel?

Do you feel like you <u>have</u> to drink alcohol in order to relax, or to have fun?

- ◯ Definitely yes, always
- ◯ Sometimes I do
- ◯ Yes, but I don't do it
- ◯ No, I don't

Do you ever feel pressured to drink by immediate family members?

- Yes, oftenYes, but I don't do it Only
- sometimesNo, never

Do you ever feel pressured to drink by friends?

- Yes, and I usually then have a drinkYes,
- but I ignore them if I don't want to
- Sometimes they pressure me more others
- thanNo, they don't pressure me

Do you ever feel like you have to drink to fit in with friends?

- Yes, all the timeYes, but I don't do it
- Yes, quite a lotNo, never
- Now and again

Do you ever drink alcohol when you're by yourself?

- Yes
- Sometimes
- No

What is the one most common reason you decide to have a drink?

- Social occasionHabit
- To celebrateFor confidence on a night out
- BoredomStress relief
- To forget or escape from negativeI like the taste of alcohol emotions
- My friends are drinking so I join inI don't want to miss out
- It's just something to do
- Other reason

If you selected other reason, can you tell us what that reason is?

Do you view drinking as something that everyone does at your age?

- Definitely yes
- Most people do
- Some people do
- Not everyone does
- No, it's not common
- I'm not sure

Do you think being drunk, or binge-drinking, is something that everyone does at your age?

- Definitely yes
- Most people do
- Some people do
- Not everyone does
- No, it's not common
- I'm not sure

How does your current drinking compare to before you started University?

- I drank more before Uni
- I drink more now I'm at Uni
- I probably drink about the same

Block 6: Drinking Habits

A unit of alcohol is the measure used in the UK to define the actual alcohol content of a drink. Roughly, one unit equates to 25ml of a spirit. Here are some approximate examples, but it can vary depending on strength of individual drink.

- Small 125ml glass of wine 1.5 units
- Large 250ml glass of wine 3 units
- 440ml can of lager/beer/cider 2 units
- 275ml bottle of alcopop 1.5 units
- Single shot of vodka/bacardi/gin 1 unit

Do you know how many units of alcohol are recommended by the government as the safe limit?

- ◯ Yes, I know
- ◯ I'm not sure, I might know
- ◯ No, I don't know

What is the maximum units per week recommended for males?

- ◯ 2012
- ◯ 1810
- ◯ 168
- ◯ 14Don't know

What is the maximum units per week recommended for females?

- ◯ 2012
- ◯ 1810
- ◯ 168
- ◯ 14Don't know

Have you ever tried alcohol, even just a small sip or taste?

- ◯ Yes
- ◯ No
- ◯ Don't know

If you have had tried alcohol, how old were you when you had that first sip/taste?

- ◯ Less than 10 years
- ◯ 11
- ◯ 1218
- ◯ 1319
- ◯ 1420 or over
- ◯ 15
- ◯ 16
- ◯ 17

Would you say you currently drink alcohol regularly? (i.e. normally more than once per month)

- Yes
- Sometimes
- No

Roughly, how many times would you have a drink in a normal month?

- 1-5 times
- 6-10 times
- 11-15 times
- 16-20 times
- 21-25 times
- 26-30 times
- More than 30 times

When did you last have a drink?

- Within the last 24 hours
- 1-2 days ago
- 2-4 days ago
- 5-7 days ago
- More than a week ago
- More than a month ago
- More than 6 months ago

Have you ever been drunk?

- Yes
- I'm not sure
- No
- I have never drunk alcohol

How many times have you been drunk in the past year?

- 1-5 times
- 6-10 times
- 11-15 times
- 16-20 times
- 21-25 times
- 26-30 times
- More than 30 times

How old were you the first time you got drunk?

- ◯ Less than 10 years old
- ◯ 11
- ◯ 12
- ◯ 13
- ◯ 14
- ◯ 15
- ◯ 16
- ◯ 17
- ◯ 18
- ◯ 19
- ◯ 20
- ◯ 21
- ◯ 22 or over

Reminder

A unit of alcohol is the measure used in the UK to define the actual alcohol content of a drink. Roughly, one unit equates to 25ml of a spirit. Here are some approximate examples, but it can vary depending on strength of individual drink.

- Small 125ml glass of wine 1.5 units
- Large 250ml glass of wine 3 units
- 440ml can of lager/beer/cider 2 units
- 275ml bottle of alcopop 1.5 units
- Single shot of vodka/bacardi/gin 1 unit

During the past year, roughly how often have you drunk more than 6 units of alcohol on a single occasion?

- ◯ Never
- ◯ 1-5 times
- ◯ 6-10 times
- ◯ 11-15 times
- ◯ 16-20 times
- ◯ 21-25 times
- ◯ 26-30 times
- ◯ More than 30 times

If you're going on a night out to a club or bar, do you drink before you go out?

- ◯ Yes, every time
- ◯ Yes, a lot of the time
- ◯ Occasionally
- ◯ No, I wait until I'm out

Why do you drink before you go out? (select all that apply)

- [ ] It's cheaper to drink more before you change outour minds
- [ ] It gets me in the mood to go outTo get
- [ ] It makes the evening longer (we can out later)
- [ ] It's something to do while we're getting ready
- [ ] goWe don't always plan to go out, but
- [ ] drunk before we go out
- [ ] goOther

If other, can you tell us why?

Can you estimate how many drinks/shots you would have before you go out?

- ◯ None7-8
- ◯ 1-29-10
- ◯ 3-411-12
- ◯ 5-613 or more

Roughly, how many more drinks would you have when you go out?

- ◯ No more7-8
- ◯ 1-29-10
- ◯ 3-411-12
- ◯ 5-6
- ◯ 13 or more

Would the type of drinks you choose change? (i.e. would you drink beer before going out, then switch to shots?)

- ◯ Yes, all the timeOccasionally
- ◯ Yes, sometimesNo, I try not to mix drinks
- ◯ in an evening

What type of drinks would you most commonly have before going out?

- [ ] BeerRum
- [ ] LagerBrandy
- [ ] CiderLiqueurs
- [ ] WineAlcopops
- [ ] Sparkling wineCocktails
- [ ] VodkaStrong shots (i.e. Jagerbomb)
- [ ] GinPre-mixed drinks
- [ ] TequilaOther
- [ ] Whisky

If other, please can you tell us what?

What kind of drink would you most commonly have while out?

- [ ] BeerWhisky
- [ ] LagerRum
- [ ] CiderBrandy
- [ ] WineLiqueurs
- [ ] Sparkling wineAlcopops
- [ ] VodkaCocktails
- [ ] GinShots (i.e. Jagerbomb)
- [ ] TequilaOther

If other, what would you drink?

How many drinks would you have when you go out?

- None7-8
- 1-29-10
- 3-411-12
- 5-613 or more

Do you continue drinking after you leave the club/bar? (i.e. do you go to a flat party/after party)

- Yes, every timeNow and again Yes, most of the timeNo,
- never

Have you ever played a drinking game?

- Yes
- No
- Not sure

Is this a regular part of your socialising? (i.e. happens at least one time in every three drinking occasions)

- Yes, it happens every timeIt only happens now and again
- Yes, it happens quite a lotNo, it's rare

Block 7: Memory Blackouts

This section asks questions about memory blackouts. A blackout doesn't mean you drank so much that you passed out, but refers to forgetting small events or chunks of time while you were drinking. There are two types of blackout. If you have a fragmentary blackout, you might not realise you've forgotten anything but then events will start to come back to you, or a friend will remind you of something which you

maybe can't immediately recall happening. An en-block blackout occurs when you forget a chunk of time. For example, you may remember up until a certain point in the evening, but then remember nothing until you got home, or got in a taxi, or woke up the next day. Typically, these memories don't come back, even when you're reminded of things which happened.

When you sober up after a drinking session (night out/full day/multiple days), have you ever realised that you've forgotten some things that happened while you were drinking?

- Yes, frequently
- Sometimes
- It's only happened once
- No, it's never happened
- I'm not sure
- I don't drink

Roughly, how often has this happened in the past year?

- 1-2 times
- 3-4 times
- 5-6 times
- 7-8 times
- 9-10 times
- 11-12 times
- More than 12 times

If you selected more than 12 times in a year, roughly how often do you think this has happened?

- More than once a month, but not as much as every week
- More than once a week, but not as much as every time I drink
- Every time I drink, on multiple occasions per week

If you did forget some things, did you remember the events all by yourself, with no help from others?

- Yes
- No
- I'm not sure

How often did you remember the events only after other people reminded you?

- Every time
- Very often
- Sometimes
- Rarely
- Always remembered by myself

Once you were sober, how long did it take to remember all the events?

- 1-2 hours
- 3-5 hours
- 6-8 hours
- 9-24 hours
- 1-2 days
- Within a week
- More than a week
- Still don't remember everything

When you sober up after a drinking session (night out/full day/multiple days etc.), have you ever realised that you've forgotten everything from a chunk of time while you were drinking? (i.e. a large part of the evening?)

- Yes, frequently
- Sometimes
- It's only happened once
- No, it's never happened
- I'm not sure

How many times has this happened in the past year?

- 1-2 times
- 3-4 times
- 5-6 times
- 7-8 times
- 9-10 times
- 11-12 times
- More than 12 times

If you selected more than 12 times in a year, roughly how often do you think this has happened?

- More than once a month, but not as much as every week
- More than once a week, but not as much as every time I drink
- Every time I drink, on multiple occasions per week

Have you ever been able to remember parts of the event later?

- ◯ Yes, all of it
- ◯ Yes, bits of it
- ◯ No, I don't remember anything

Can you describe your feelings towards losing part of your memories due to drinking alcohol?

- ◯ It frightened me, it won't happen againI
- ◯ I don't like it, trying not to let it happenIt
- ◯ I've cut back my drinkingI quite like it
- ◯ I don't like it, but it won't stop me
- ◯ didn't like it the first time, but don't mind now
- ◯ has never bothered me at all again
- ◯
- ◯ drinkingOther

If other, how do you feel about losing part of your memories due to drinking alcohol?

Do you ever use any other substances when you're drinking? (i.e. cigarettes, marijuana, cocaine, anti-depressants etc.)

- ◯ Yes, every timeOnly done so once Yes,
- ◯ ◯Only occasionally
- ◯ sometimesNo, never
- ◯

If you do, can you tell us what?

Block 8: Additional Questions

Have you ever been assessed for a neurological disorder? (i.e. ADHD, epilepsy, dyslexia etc.)

- ◯ Yes
- ◯ No
- ◯ Not sure

Can you tell us what that assessment was for?

Have you ever received a head injury which required medical attention? (i.e. the result of an accident or childhood illness)

- ◯ Yes
- ◯ No
- ◯ Not sure

The Scottish Government introduced a minimum per unit price for alcohol on 1st May 2018. This may have increased the cost of your regular drinks. On a typical week, how has this affected your drinking?

- ◯ I drink as often, but I drink a bit less than used tofewer occasions
- ◯ II drink as much as I used to, but I do it on
- ◯ It hasn't changed my behaviour, I drink as much - and as often - as I used to

Block 9: thank you and useful info

Block 10: Small Questionnaire

Part Two

Please complete as many of the following questions as possible. Your responses will be confidential and securely stored spearately from any identifying information. While it would assist our research greatly for you to answer every question, please leave blank any that you are uncomfortable answering. No questions are compulsory.

Definition of drunk

When answering, think of being drunk as having lost some (but not necessarily all) control over behaviour following drinking alcohol. This could be noticed by someone saying things which they would not normally, by someone slurring words, by being unsteady on their feet or unusually clumsy. If it was you, you might feel ok, but notice you are unable to physically move or verbally respond in your normal fashion. You may also feel more emotional than usual or more confident than usual.

Counting back from midnight last night, have you consumed alcohol in the last 7 days?

- ◯ Yes
- ◯ No

If yes, can you select the days on which you had a drink?

- ☐ MondayFriday
- ☐ TuesdaySaturday WednesdaySunday
- ☐ Thursday
- ☐ Would you say you were drunk on any of these occasions?

- ◯ Yes
- ◯ Maybe
- ◯ No

To the best of your ability, can you tell us what combination, quantity and brands of drinks you were drinking on which days? (i.e. 3 x 330ml bottles Budweiser, 2 large glasses Barefoot Pinot Grigio. Please give as much detail as you can)

Monday -

Tuesday -

Wednesday -

Thursday -

Friday -

Saturday -

Sunday -

When you were drinking during the past week, where were you? (select all that apply)

- [ ] At homeRestaurant
- [ ] Friend's home/flatParty (i.e. in a
- [ ] Bar/pubOther
- [ ] Club
- [ ]
- [ ] venue/hall/hotel)
- [ ]

If you were drinking somewhere other than at home or in a friends home (i.e. if you went to a bar/pub/club/party), did you pre-drink before you went out?

- ◯ Yes, every timeNo, I just drank while I was
- ◯ Yes, sometimesNo, I was only drinking at
- ◯ out
- ◯ home

Did you drink more before you went out than when you were out?

- ◯ Yes, every timeMaybe I drank about the
- ◯ Most timesNo, I drank more when I was
- ◯ same
- ◯ out than

before I went out

If you did drink before you went out, why did you do this? (select all that apply)

- [ ] It's cheaper to drink more before you change outour minds
- [ ] It gets me in the mood to go outTo get
- [ ] It makes the evening longer (we can out later)
- [ ] It's something to do while we're getting ready goWe don't always plan to go out, but
- [ ] drunk before we go out
- [ ] goOther

If you pre-drank for another reason, can you tell us what?

When you were drinking, did you play any drinking games during the last week?

- ◯ Yes
- ◯ Don't remember
- ◯ No

Did you drink more than you intended during the game?

- ◯ Yes
- ◯ Not sure
- ◯ No

The day after drinking, did you notice you'd forgotten some things that happened while you were drinking?

- ◯ Yes
- ◯ Not sure
- ◯ No

If so, on which nights out did this happen?

- [ ] MondayFriday
- [ ] TuesdaySaturday
- [ ] WednesdaySunday Thursday
- [ ]
- [ ]
- [ ]
- [ ]

If you did forget some things, did you remember all the events by yourself?

- ◯ Yes
- ◯ Not sure
- ◯ No

How often did you remember some of the events only after other people reminded you?

- ◯ Every time
- ◯ Usually needed reminding
- ◯ Sometimes needed reminding
- ◯ Usually remembered myself
- ◯ Always remembered myself

Once you were sober, how long did it normally take to remember all the events?

- ◯ 1-2 hours1-2 days
- ◯ 3-5 hours2-7 days
- ◯ 6-8 hoursDon't remember everything yet
- ◯ 8-24 hours
- ◯
- ◯
- ◯

The day after drinking, did you realise that you'd forgotten everything that happened from a chunk of time while you were drinking? (i.e. maybe a few hours)

- ◯ Yes
- ◯ Not sure
- ◯ No

If so, on which nights out did this happen?

- [ ] MondayFriday
- [ ] TuesdaySaturday
- [ ] WednesdaySunday Thursday
- [ ]

Have you since been able to remember any bits of time from that occasion?

- ◯ Yes
- ◯ Not sure
- ◯ No

Did you use any other substances while you were drinking in the last week? (i.e. cigarettes, marijuana, cocaine, prescription drugs etc.)

- ◯ Yes - every time
- ◯ Yes - sometimes
- ◯ Only once
- ◯ No

If so, can you tell us what?

Thank you for taking part in our research!

Please remember that your responses are confidential and will be stored with no identifying information attached.

Your responses are invaluable to us.

Researchers: Judith Jackson Judith.Jackson1@stir.ac.uk
Dr Benjamin Dering b.r.dering@stir.ac.uk

Debrief

Participant Debrief Information

Alcohol and Students - Drinking behaviours and the consequences for memory Thank you for participating in this study.

1. Background, aims of project

Thank you for taking part in the survey – your data is invaluable to us! The questionnaires measure the drinking behaviour of university undergraduates, family history of drinking behaviour, and instances of memory loss due to drinking.Specifically, we are interested in the prevalence of memory black-outs caused by alcohol, and the environmental influences which may affect them.Based upon your responses, we may invite you to take part in a further laboratory based study, however you are under no obligation to do this.

2. Legal basis for processing personal data.

As part of the project we collected personal data relating to you.This will be processed in accordance with the General Data Protection Regulation (GDPR).Under GDPR the legal basis for processing your personal data will be the public interest.

3. What happens to the data I provide?

Your answers will be completely anonymous, and we will use all reasonable endeavours to keep them confidential.Your data will be stored in a passwordprotected file. Your IP address will not be stored.

Only the research team (Judith Jackson and Dr Benjamin Dering) will have access to research data.

If you wish to withdraw your data, you can do this for up to one month following the date you complete the survey.To do this, please contact the researchers with the identifying number which you entered at the start of the survey.All your responses will then be destroyed.

4. Will the research be published?

The methods used to conduct this research, and any group-level results, will be published in a doctoral thesis and may also be published in an academic journal or

conference paper.There will be no analysis of responses at the individual level, therefore you will not be individually identifiable from any published research.

5. Your rights.

You have the right to request to see a copy of the information we hold about you and to request corrections or deletions of the information that is no longer required.

You have the right to withdraw from this project at any time.However, after one month or once the data are being analysed, it may not be possible to remove your individual responses from the group analyses.

6. Contact Details

Again, thank you for taking part. If you have any further queries about the study, please contact one of the principal researchers.

Judith.Jackson1 @stir.ac.uk	Judith Jackson
b.r.dering@stir.ac.uk	Dr. Benjamin Dering
p.j.hancock@stir.ac.uk	Professor Peter Hancock

You have the right to lodge a complaint against the University of Stirling regarding data protection issues with the Information Commissioner's Office (https://ico.org.uk/concerns/).

The University of Stirling's Data Protection Officer is Joanna Morrow, Deputy Secretary. If you have any questions relating to data protection these can be addressed to data.protection@stir.ac.uk in the first instance.

If you have been affected by any of the issues raised in this study, then please contact your University counselling service. For University of Stirling students, you can email student.counselling@stir.ac.uk.

Alcoholics Anonymous	help@aamail.org
Scottish Families Against Drugs	08080 10 10 11
Breathing Space	0800 83 85 87