THE INFLUENCE OF COMORBID NEGATIVE MOOD ON CRAVING’S RELATIONSHIP TO POST-TREATMENT ALCOHOL USE

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ABSTRACT

Alcohol is ranked as the third highest burden of disease worldwide and the eighth highest leading cause of death. An estimated 19.5% of Australians consume alcohol in quantities that place them at risk of alcohol-related injury or disease over their lifetime. Alcohol misuse is also highly problematic, being associated with a range of negative physical, psychological and social consequences. While treatments are effective in helping people to achieve reductions, relapse rates are high, with up to 80% of treated alcohol users eventually relapsing. The ability to identify which treatment seekers may be at greater risk for relapse would enable appropriate tailoring of interventions and planning of aftercare.

Craving has been widely studied as a potential predictor of relapse, but has performed inconsistently. The effect of comorbid depression on craving’s predictive performance however, has been largely neglected, despite demonstrated associations between negative affect and craving, and between negative affect and substance use. The aim of this thesis was to explore the performance of craving as a predictor of post-treatment alcohol use outcomes in the presence of comorbid depressed mood, under the hypothesis that presence of negative affect would augment effects of craving, strengthening its predictive power and increasing vulnerability to post-treatment relapse. Two studies were conducted, one with a sample of drinkers with comorbid depression, and the other with a sample of drinkers with a range of depression severity.

Study 1 included 284 males and females who self-referred for a randomised controlled trial of treatments for comorbidity of depression with alcohol use. Participants scored 17 or greater on the Beck Depression Inventory-II and reported at
Abstract

least two occasions in the previous month of greater than 6 standard drinks (6 x 10g ethanol) for men, or greater than 4 for women. Craving was measured prior to treatment using the obsessive subscale of the Obsessive Compulsive Drinking Scale. Two drinking outcomes, average weekly drinks and frequency of alcohol binges, were assessed 18 weeks and 12 months post-baseline. Interaction effects between craving and depression were examined by including an interaction term in the analyses. Craving was found to be a significant predictor of 18-week average weekly drinking and of higher frequency of binges at 18 weeks and 12 months. Neither depression by itself, nor the interaction of depression with craving were significant, although this may have been due to lack of sufficient spread in depression scores to detect an effect. Item analysis suggested different influences of craving over time, with items pertaining to interference from drinking thoughts and success in diverting thoughts being related to 18-week outcomes, and items pertaining to frequency of thoughts and efforts to resist thoughts being related to 12-month outcomes.

Study 2 included 242 males and females who self-referred for a randomised controlled trial of treatments for alcohol use disorder. Participants were consuming more than 28 standard drinks per week for men, or more than 14 per week for women, and met diagnostic criteria for an alcohol use disorder. Craving was measured prior to treatment using the obsessive subscale of the Obsessive Compulsive Drinking Scale and a newly developed craving measure, the Alcohol Craving Experience questionnaire. Depression was measured using the Depression, Anxiety and Stress Scale-21. Outcome assessments, measuring the same outcomes as in Study 1, were conducted 3 and 12 months post-baseline. Identical analyses were conducted as in Study 1, including an interaction term between craving and depression. Unlike Study 1, craving by itself was not a significant predictor of either of the outcomes at any time point, although the
interaction between craving and depression was a predictor of average weekly drinking at 12 months. Consistent with Study 1, depression was not related to outcomes. Results were also consistent with the item analyses of Study 1, with items pertaining to intrusiveness of alcohol thoughts and efforts to not think about alcohol performing most strongly in the prediction of 12-month drinking.

The finding that craving was predictive of treatment outcomes in the high depression sample of Study 1, and interacted with depression to predict outcomes in Study 2, appears to provide preliminary support for a potential moderating effect of depression on the relationship between pre-treatment craving and post-treatment alcohol use. Furthermore, results suggest that risk from craving may come from how alcohol thoughts are managed, rather than from the thoughts themselves.
STATEMENT OF ORIGINALITY

This work has not previously been submitted for a degree or diploma in any university. To the best of my knowledge and belief, the thesis contains no material previously published or written by another person except where due reference is made in the thesis itself. The data reported in the studies of this thesis were collected by myself and a team of research assistants during the course of two randomised controlled trials.

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Jennifer Connolly
# TABLE OF CONTENTS

ABSTRACT................................................................. II

STATEMENT OF ORIGINALITY........................................ V

TABLE OF CONTENTS...................................................... VI

LIST OF TABLES........................................................... IX

LIST OF FIGURES........................................................ XII

LIST OF APPENDICES.................................................. XIII

LIST OF PUBLICATIONS................................................ XIV

ACKNOWLEDGEMENTS.................................................. XV

CHAPTER 1  OVERVIEW..................................................1

CHAPTER 2  WHY WORRY ABOUT ALCOHOL? ..................5
  2.1 High prevalence..................................................5
  2.2 Consequences of alcohol misuse ..............................6
  2.3 Treatment and relapse .........................................9
     2.3.1 Common treatment approaches ...........................9
     2.3.2 Recent advances in treatment ............................14
     2.3.3 Treatment impact ........................................16

CHAPTER 3  ROLE OF CRAVING IN ADDICTION................21
  3.1 What is craving?..................................................21
  3.2 Why craving is important .....................................23
  3.3 Models of craving in addiction theory .....................24
     3.3.1 Cognitive model .......................................25
     3.3.2 Negative reinforcement theory and affective model of drug motivation .......................................................26
     3.3.3 Positive reinforcement and opponent-process theory of motivation .........................................................29
     3.3.4 Biological theories of addiction and the incentive-sensitisation model .........................................................................................................................31
     3.3.5 Elaborated intrusion theory of craving ..................35
  3.4 Implications of craving ...........................................38

CHAPTER 4  CRAVING AS A PREDICTOR OF SUBSTANCE TREATMENT OUTCOMES ..................40
  4.1 High variability between studies ..............................48
CHAPTER 5  ROLE OF NEGATIVE AFFECT IN ADDICTION ..........66

5.1 Relationship between negative affect and substance use ..........67
  5.1.1 Evidence of proximal associations .....................67
  5.1.2 Relationship to substance treatment outcomes ..........70

5.2 Relationship between negative affect and craving ..........73
  5.2.1 Negative affect and substance cue reactivity ..........73
  5.2.2 Negative affect and craving \textit{in situ} ..........77

5.3 Summary of role of negative affect in addiction ..........78

5.4 Dynamic association between craving, negative affect and substance use ..........80

5.5 Craving as a predictor of outcomes in the context of negative affect ..........83

CHAPTER 6  AIMS OF THIS THESIS ..................................86

6.1 Studying craving as a predictor in drinkers with comorbid depressed mood ..........86

6.2 Using context and construct relevant measures ..........87

6.3 Use of continuous outcome measures ..........88

CHAPTER 7  STUDY 1: CRAVING AS A PREDICTOR OF TREATMENT OUTCOMES IN HEAVY DRINKERS WITH COMORBID DEPRESSED MOOD ........................................89

7.1 Introduction ..........................................................89

7.2 Method .................................................................93
  7.2.1 Participants .....................................................93
  7.2.2 Measures .......................................................95
  7.2.3 Interventions ...................................................99
  7.2.4 Procedure .....................................................100
  7.2.5 Statistical analysis ..........................................100

7.3 Results .................................................................102
  7.3.1 Data adjustments .............................................104
  7.3.2 Missing data ..................................................105
  7.3.3 Average weekly drinks ....................................106
  7.3.4 Frequency of binges .......................................110
  7.3.5 OCDS-O items ...............................................114
  7.3.6 Sensitivity analyses .......................................117

7.4 Discussion ...........................................................118

7.5 Conclusion ...........................................................125
# Table of contents

## CHAPTER 8  STUDY 2: THE INFLUENCE OF MOOD-CRAVING RELATIONSHIPS ON ALCOHOL TREATMENT OUTCOMES

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>8.1 Introduction</td>
<td>127</td>
</tr>
<tr>
<td>8.2 Method</td>
<td>131</td>
</tr>
<tr>
<td>8.2.1 Participants</td>
<td>131</td>
</tr>
<tr>
<td>8.2.2 Measures</td>
<td>134</td>
</tr>
<tr>
<td>8.2.3 Interventions</td>
<td>137</td>
</tr>
<tr>
<td>8.2.4 Procedure</td>
<td>139</td>
</tr>
<tr>
<td>8.2.5 Statistical analysis</td>
<td>140</td>
</tr>
<tr>
<td>8.3 Results</td>
<td>142</td>
</tr>
<tr>
<td>8.3.1 Data adjustments</td>
<td>145</td>
</tr>
<tr>
<td>8.3.2 Missing data</td>
<td>146</td>
</tr>
<tr>
<td>8.3.3 Average weekly drinks</td>
<td>147</td>
</tr>
<tr>
<td>8.3.4 Frequency of binges</td>
<td>156</td>
</tr>
<tr>
<td>8.3.5 Sensitivity analyses</td>
<td>164</td>
</tr>
<tr>
<td>8.4 Discussion</td>
<td>166</td>
</tr>
<tr>
<td>8.5 Conclusion</td>
<td>176</td>
</tr>
</tbody>
</table>

## CHAPTER 9  GENERAL DISCUSSION

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>9.1 Evidence for moderating effect of depression</td>
<td>180</td>
</tr>
<tr>
<td>9.2 Inconsistencies and implications</td>
<td>182</td>
</tr>
<tr>
<td>9.2.1 Prediction of average weekly drinks</td>
<td>183</td>
</tr>
<tr>
<td>9.2.2 Prediction of alcohol binges</td>
<td>185</td>
</tr>
<tr>
<td>9.3 Consistencies and implications</td>
<td>186</td>
</tr>
<tr>
<td>9.3.1 Variance explained by craving</td>
<td>186</td>
</tr>
<tr>
<td>9.3.2 Nature of significant craving items</td>
<td>188</td>
</tr>
<tr>
<td>9.3.3 Lack of prediction by depression</td>
<td>189</td>
</tr>
<tr>
<td>9.4 Clinical implications</td>
<td>191</td>
</tr>
<tr>
<td>9.5 Limitations of this body of work</td>
<td>193</td>
</tr>
<tr>
<td>9.6 Future directions</td>
<td>195</td>
</tr>
<tr>
<td>9.7 Conclusions</td>
<td>197</td>
</tr>
</tbody>
</table>

## REFERENCES  199

## APPENDICES  222
LIST OF TABLES

Table 4.1 Studies predicting post-treatment substance use outcomes from pre-treatment craving measurements ................................................................. 41

Table 7.1 Demographic characteristics of the full sample (n=284) ......................... 95

Table 7.2 Baseline characteristics of the analysed sample (N = 260) ..................... 102

Table 7.3 Correlation matrix of baseline variables .................................................. 104

Table 7.4 Results of correlations (r) and ANOVAs (F) of baseline and treatment variables with average weekly drinks at each follow-up time point........... 107

Table 7.5 Results of partial correlations (r) and ANCOVAs (F) of baseline and treatment variables with average weekly drinks at each follow-up time point, controlling for baseline consumption......................................................... 108

Table 7.6 Coefficients of baseline and treatment variables predicting average weekly drinks at 18 weeks at each step of hierarchical linear regression (n = 205) ............................................................... 109

Table 7.7 Results of ANOVAs (F) and chi-squares (χ²) of baseline and treatment variables with frequency of binges at each follow-up time point ............. 111

Table 7.8 Results of ANCOVAs (F) and likelihood ratio tests (χ²) of baseline variables with frequency of binges at each follow-up time point, controlling for baseline binge frequency .......................................................... 112

Table 7.9 Parameter estimates for baseline variables predicting binge frequency at each follow-up time point (n = 207). Reference category is ‘half or more of the week’ ........................................................................................................ 114
List of tables

Table 7.10 Regression coefficients and semi-partial correlations for OCDS-O items predicting 18-week average weekly drinks (n = 205) .......................... 115

Table 7.11 Likelihood ratio tests ($\chi^2$) and parameter estimates (B) for OCDS-O items predicting post-treatment binge frequency (n = 206). Reference category is ‘half or more of the week’ ................................................................. 116

Table 8.1 Demographics characteristics of the full sample (N = 242) .......................... 134

Table 8.2 Baseline characteristics of the full sample (N = 242) .................................. 143

Table 8.3 Correlation matrix of baseline variables ......................................................... 144

Table 8.4 Results of correlations (r) and ANOVAs (F) of baseline and treatment variables with average weekly drinks at each follow-up time point .......... 149

Table 8.5 Results of partial correlations (r) and ANCOVs (F) of baseline and treatment variables with average weekly drinks at each follow-up time point, controlling for baseline consumption ........................................ 150

Table 8.6 Coefficients of baseline variables, with craving measured using the OCDS-O, predicting average weekly drinks at 12 months (N = 239) ....................... 152

Table 8.7 Coefficients of baseline variables, with craving measured using the ACE-Intrusions, predicting average weekly drinks at 12 months (N = 229) ...... 154

Table 8.8 Coefficients of baseline variables at step 4 of the linear regressions predicting 12-month average weekly drinks from the interactions of depression with the OCDS-O and ACE-Intrusions (N = 226) ................................................. 156

Table 8.9 Results of ANOVAs (F) and chi-squares ($\chi^2$) of baseline and treatment variables with frequency of binges at each follow-up time point ............ 158

Table 8.10 Results of ANCOVs (F) and likelihood ratio tests ($\chi^2$) of baseline variables with frequency of binges at each follow-up time point, controlling for baseline binge frequency ......................................................... 160
Table 8.11 *Parameter estimates for baseline variables predicting frequency of binges at 3-month follow-up (N = 227). Reference category is ‘half or more of the week’* ................................................................. 162

Table 8.12 *Likelihood ratio tests ($\chi^2$) and parameter estimates (B) for baseline variables predicting frequency of binges at 12 months (N = 237). Reference category is ‘half or more of the week’* ................................................................. 163

Table 8.13 *Likelihood ratio tests ($\chi^2$) and parameter estimates (B) for baseline and treatment variables predicting frequency of binges 3 months (N = 237). Reference category is ‘half or more of the week’* ................................................................. 164
List of figures

LIST OF FIGURES

Figure 7.1. CONSORT diagram showing flow of participants through Study 1........... 94

Figure 8.1. CONSORT diagram showing flow of participants through Study 2........ 133

Figure 8.2. Predicted values of 12-month average weekly drinks for people with low or high depression, at low craving (1SD below the standardised mean of OCDS-O) and high craving (1SD above)............................................ 153

Figure 8.3. Predicted values of 12-month average weekly drinks for people with low or high depression, at low craving (1SD below the standardised mean of ACE-Intrusion) and high craving (1SD above)........................................... 155
LIST OF APPENDICES

Appendix A Study 1 assessment measures ................................................................. 223
Appendix B Study 1 demographic characteristics of reduced sample ....................... 242
Appendix C Results of Study 1 missing value analysis and imputation ...................... 243
Appendix D Alcohol Craving Experience Questionnaire ........................................... 244
Appendix E Alcohol Craving Experience Questionnaire after item reduction .......... 248
Appendix F Study 2 assessment measures ................................................................. 250
Appendix G Results of Study 2 missing value analysis and imputation ..................... 261
Appendix H Baseline and demographic differences between the populations of Study 1 and Study 2 ........................................................................................................ 262
LIST OF PUBLICATIONS

Publications arising from this thesis


Publications related to this thesis

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CHAPTER 1 OVERVIEW

This research attempts to explore the influence of comorbid negative mood on craving’s relationship to post-treatment alcohol use. After a review of the relevant literature, two clinical experimental studies are described that directly test the proposition central to this research. In the final chapter, results are amalgamated and their implications discussed. Supplementary material is provided in appendices.

Chapters 2 to 5 provide the background rationale for the thesis with an overview of relevant literature. Chapter 2 begins by setting the scene for the focus on alcohol, with illustration of its high prevalence and high personal and societal impact. Effective treatments are briefly reviewed, but their failure to achieve high rates of sustained recovery is acknowledged, with review of the high relapse rates often observed in substance using populations. The benefit of being able to identify people most at risk of relapse is raised, and craving introduced as a potential indicator of outcomes.

Chapter 3 justifies why craving is important to consider in addiction, and why it could be expected to predict likely treatment response. Craving is firstly defined, pulling together definitions and descriptions from multiple sources, acknowledging the lack of a single accepted definition of the concept. Various models of craving, as described by leading addiction theories, are reviewed, with brief review of the evidence supporting or contradicting each. Evidence linking craving with substance use is presented, leading into Chapter 4 which presents a comprehensive methodological review of studies examining craving as a predictor of outcomes following treatment for substance use. Twenty-one studies are reviewed in detail and the variability in measures used, timing
Overview

of assessment and outcomes examined is highlighted. The inconsistent performance of craving as a predictor is acknowledged, and the possibility that craving’s relationship to post-treatment substance use may be influenced by other variables is raised. Negative affect is put forward as one such possible variable of influence.

The role of negative affect in addiction is reviewed in Chapter 5, with evidence demonstrating its association with both craving and substance use. Negative affect is also acknowledged as a predictor of substance use outcomes following treatment, although, like craving, it does not always perform consistently. The value of examining negative affect as a potential influence on the relationship between craving and post-treatment outcomes is justified, and recent evidence supporting dynamic associations between the two is presented. Chapter 6 introduces the aims of the present research, which are to study the dynamic relationship between craving and negative affect and how this relationship influences outcomes following treatment, and to overcome several key limitations of previous research.

Chapter 7 introduces the first experimental study, which is an examination of the predictive performance of craving in a sample of heavy drinkers with comorbid depressed mood. Epidemiological statistics relating to comorbidity of alcohol with depression are provided, along with a brief review of the impact of comorbidity including factors associated with poorer clinical outcomes. The only other identified study investigating craving as a predictor in comorbid depressed drinkers is reviewed, with identification of limitations that warrant further study. Chapter 7 hypothesises that craving will perform significantly as a predictor of post-treatment alcohol consumption in the presence of comorbid depressed mood, and that an interaction between mood and craving will be evident, with the combination of high levels of depression with high levels of craving being associated with greater drinking post-treatment. The method and
procedure for the study are described, along with full description of the statistical procedures adopted. Univariate analyses are initially conducted to identify potential predictors, with all significant univariate predictors entered into subsequent regressions. Results are reviewed in reference to the hypotheses, and interpreted in the context of other relevant literature.

Chapter 8 introduces the second experimental study, which is an examination of the influence of mood and craving relationships on post-treatment alcohol use. This second study extends on the first by targeting a sample of heavy drinkers with a range of depression severity, enabling more robust testing of interaction effects between mood and craving. Limitations from the first study are reviewed, with description of how these are addressed in the second study. More detail on the background development of the craving measures used is provided, leading into description of how the second study addresses limitations of previous research to investigate the influence of mood and craving on post-treatment outcomes. Like Chapter 7, Chapter 8 hypothesises that craving will be predictive of post-treatment drinking, and that the interaction between mood and craving will be predictive of outcomes also, such that high depression with high craving will be associated with worse post-treatment outcomes. The study method and procedures are described in detail, along with description of the analytic approach, which is identical to that of Chapter 7. Results are reviewed in reference to the hypotheses, and interpreted in the context of other relevant literature.

The ninth and final chapter brings together the results of both studies and interprets their meaning in the context of current literature on craving, affect and addictions. The implications of the results for the major premise of this research, that craving will interact with high levels of negative mood to predict greater substance use following treatment, is discussed first. This is followed by review of the inconsistencies
Overview

between the two studies and discussion of the likely causes and implications. Consistencies across the two studies are then reviewed, with discussion of likely meaning and implications. The clinical implications of the results are briefly discussed, followed by acknowledgement of the limitations and recommendations for further research. A final conclusion summarises the major findings and their implications.

Supplementary material that is not directly pertinent but may be of interest or use to the reader is provided in appendices. This includes copies of the assessment instruments, results of the missing value analyses and imputations, and comparison statistics of the demographic and baseline characteristics of the two study samples.
CHAPTER 2 WHY WORRY ABOUT ALCOHOL?

2.1 High prevalence

Alcohol is the most widely used psychoactive substance of all licit and illicit drug types (excluding caffeine and nicotine), with 78.3% of the Australian population consuming at least one standard alcoholic drink\(^1\) in a 12 month period, compared to much lower rates of use of other drugs (cannabis: 10.3%; ecstasy: 3.0%; meth/amphetamine: 2.1%; cocaine: 2.1%; heroin: 0.2%; Australian Institute of Health and Welfare, 2011). While the majority of people who drink alcohol do so infrequently or at low levels, a substantial proportion consume it in quantities that place them at increased risk for harm. A person who consumes more than four standard drinks on a single occasion is at least 5.6 times more likely to sustain an alcohol-related injury on that occasion than someone who had not been drinking (National Health and Medical Research Council, 2009). Approximately 10.8% of Australians drink more than four drinks per occasion weekly, and 4.6% drink that much daily (Australian Institute of Health and Welfare, 2011). The proportion drinking at levels risky for lifetime harm are even higher. Consumption of more than two standard drinks per day on a regular basis over a lifetime is associated with increased risk for alcohol-related disease and death (National Health and Medical Research Council, 2009). According to recent survey data, in a 12-month period, approximately 19.5% of Australians are drinking at a level placing them at high risk of such harm.

\(^1\) 1 standard drink = 10g of ethanol.
Why worry about alcohol?

The population prevalence of alcohol use disorder is also high, with 18.3% of Australians meeting DSM-IV-TR (Association, 2000) criteria for lifetime alcohol abuse\(^2\) and 2.9% meeting criteria in the previous 12 months (Teesson et al., 2010). Prevalence of alcohol dependence\(^3\) is not quite as high, but is still substantial, with 3.9% of Australians meeting lifetime criteria, and 1.4% meeting criteria in the previous 12 months (Teesson et al., 2010). Rates in US populations have been observed to be even higher, with 12 month alcohol use disorder prevalence estimated at 8.5%, and lifetime at 30.3% (Hasin, Stinson, Ogburn, & Grant, 2007).

Use of alcohol is very common in the population. Especially concerning is the high prevalence of consumption at levels considered risky for short-term and/or long-term harm, and the high population prevalence of diagnosable alcohol use disorders. These high prevalence rates are of particular concern because it has been well documented that alcohol misuse (i.e., drinking alcohol at levels considered to be high risk) and alcohol use disorders are associated with a range of negative health and social consequences, causing significant cost and burden to health systems and society.

### 2.2 Consequences of alcohol misuse

Alcohol misuse is associated with a range of negative physical, social and psychological consequences. The physical health risks are amongst the most well documented (Cargiulo, 2007). Risks for accident and injury increase substantially with increasing levels of consumption. Borges et al. (2006) reported the relative risk of acute, non-fatal injury following alcohol use within the previous 6 hours as being 2.97 in

\(^2\) One or more of: i) recurrent failure to fulfil role obligations; ii) recurrent use in physically hazardous situations; iii) recurrent legal problems; iv) continued use despite persistent interpersonal problems.

\(^3\) Three or more of: i) tolerance; ii) withdrawal; iii) used more or for longer than intended; iv) persistent desire or unsuccessful efforts to control; v) great deal of time spent to obtain, use or recover; vi) important activities given up or reduced; vii) continued use despite persistent physical or psychological problems.
Australia, and the risk was much higher in other countries such as Mexico (17.92-23.17), India (33.46) and South Africa (35.00). Globally, the relative risk of injury was found to be 5.18 (Borges et al., 2006). The risk increases substantially with increasing levels of consumption. In Australian standard drinks (10g ethanol), the odds ratio for admission to an emergency department for injury following alcohol use increases from 1.9 after one drink to 3.8 after three, 5.6 after five and 10.1 after seven or more drinks are consumed on a single occasion (National Health and Medical Research Council, 2009).

Lifetime alcohol misuse is associated with increased risk for a variety of cancers, but is particularly high for cancers of the oral cavity, pharynx, esophagus, larynx, stomach, colon, rectum, liver, breast and ovaries (Bagnardi, Blangiardo, La Vecchia, & Corrao, 2001). Cognitive impairments become evident even after low doses of alcohol that would, in an average person, place blood alcohol levels in a range that remains legal for driving (Kerr et al., 1992; Molnár, Boha, Czigler, & Gaál, 2010). In the long term, chronic alcohol misuse is associated with cognitive impairments in a number of domains including memory, problem solving ability, attention span and reaction time (Fals-Stewart, Schafer, Lucente, & Rustine, 1994; Mintzer, 2007; Molnár et al., 2010; Tomberg, 2010b). Chronic alcohol use is also associated with a range of psychosocial impairments and consequences, including lower employment and education levels, more frequent trouble with the law and high levels of relationship discord (Kunz & Graham, 1998; Nolen-Hoeksema, 2004; Perkins, 2002). Intimate partner violence is also strongly associated with high levels of alcohol consumption in the home (Foran & O’Leary, 2008).

Additionally, alcohol use disorders are associated with high rates of mental health and other drug comorbidity. Results from Australia’s 2007 National Survey of
Why worry about alcohol?

Mental Health and Wellbeing revealed that one third of people with a 12-month alcohol use disorder also met criteria for at least one comorbid anxiety, affective or drug use disorder (Burns, Teesson, & Lynskey, 2001). They were 10 times more likely than people without an alcohol use disorder to have a drug use disorder, 4 times more likely to have an affective disorder and 3 times more likely to have an anxiety disorder. US data give odds ratios of 2.6 for 12-month mood disorder comorbid with an alcohol use disorder and 1.7 for an anxiety disorder (Grant et al., 2006).

Given the significant impact of alcohol misuse on most domains of health and functioning, it is not surprising that alcohol misuse imposes significant cost and burden on individuals, families, communities, health systems and governments at every level. The World Health Organization places alcohol as the third highest burden of disease globally (as measured by disability adjusted life years or DALYs) and the eighth highest leading cause of death, with alcohol contributing to more than 60 types of disease and injury (World Health Organization, 2009). The estimated total societal cost of alcohol in Australia in 2004/05 was in excess of $15 billion (Collins & Lapsley, 2008). This included costs associated with death and disease, accidents and injury, lost productivity and absenteeism, and judicial system costs.

Due to the high prevalence of alcohol misuse and the enormous costs to society, significant government resources are invested in trying to prevent and reduce incidence of alcohol use disorders and high risk drinking in the community. Australia’s National Alcohol Strategy, an initiative of the federal government, commenced in 2006 and outlines priority areas for coordinated action to develop drinking cultures that support a reduction in alcohol-related harm in Australia. In 2008, the Australian government announced investment of $53.5 million in a National Binge Drinking Strategy, with the aim of reducing binge drinking among young people. Significant resources are being
spent on trying to reduce population levels of harmful drinking, and this includes intense interest in, and research on, effective interventions to treat alcohol abuse and dependence, though no intervention has yet been found to be effective for all people.

2.3 Treatment and relapse

A wide variety of treatments have been evaluated for their efficacy in achieving alcohol reductions, with many targeting sustained abstinence as the primary goal. Research supports the efficacy of many of these interventions, some more so than others, yet none have achieved consistently high rates of recovery. For every treatment there is always a proportion of recipients who do not respond, and who will relapse either during or after treatment.

2.3.1 Common treatment approaches

2.3.1.1 Twelve-step facilitated interventions

Twelve-step programs, Alcoholics Anonymous (AA) being the most widely known example, stem from disease models of addiction, have a strong basis in spirituality and draw heavily on social support networks, most notably through use of a ‘sponsor’. Consistent with the disease model, recovery is viewed as a lifelong commitment, with complete abstinence viewed as the only truly viable goal. Kassel and Wagner (1993) provide an overview of the principles and steps of AA, which include admitting powerlessness over alcohol, giving over one’s life to a ‘Higher Power’ and making amends to people harmed. Other 12-step programs are highly consistent with the principles and approach of AA, and include admitting having a problem, seeking help from a higher power and enhancing social connectedness (Moos, 2007).
Why worry about alcohol?

Twelve-step programs, including AA, have been found to be beneficial and to have positive outcomes for people seeking help for alcohol dependence (Kaskutas, 2009; Ouimette, Finney, & Moos, 1997). The mechanisms of its effectiveness are believed to lie in the spirituality and social components, the promotion of abstinence and in coping skills and confidence gained throughout the process (Kelly, Magill, & Stout, 2009; Moos, 2007).

2.3.1.2 Cognitive-behavioural interventions

Cognitive-behavioural therapies (CBT) are also widely utilised in addiction treatment and intensely researched. These interventions are based in cognitive social learning theory which views alcohol use as a learned behaviour mediated to a large extent by self-efficacy and expectancies (Scaturo, 1987; Wilson, 1987). CBT interventions take many forms, but typically have the same core elements of psychoeducation, social skills training, risk situation management, adoption of non-alcohol activities, drink refusal skills, challenging of expectancies, craving management and relapse prevention (Magill & Ray, 2009; Moos, 2007). CBT is often considered the treatment of choice in most clinical and health settings, due largely to the substantial body of literature supporting its efficacy in the treatment of alcohol use disorder, as well as substance use disorders more generally (Magill & Ray, 2009; Miller et al., 1995), though the precise mechanisms of its effect on change are still difficult to determine (Morgenstern & Longabaugh, 2000).

2.3.1.3 Cue-exposure treatments

Cue-exposure treatments are based in learning theory with the basic premise that substance cues elicit conditioned appetitive responses (cravings), and that these responses can therefore be unconditioned (Marlatt, 1990). Cues can be anything paired
with the substance or its use, including the sight, smell or taste of the substance, or environmental cues related to its use or effects. From a classical learning theory perspective, exposure to substance cues gives rise to a conditioned response that may include a sense of withdrawal if the substance itself is absent, a conditioned physiological response, or an appetitive response similar to that produced by use of the substance (Monti & Rohsenow, 1999). From this learning perspective, the cue-exposure treatment involves habituation or extinction of the response by repeated exposure to the cue with prevention of the usual response, i.e., prevention of use of the substance. From a social learning theory perspective, exposure to substance cues increases the salience of the positive effects of use of the substance (Monti & Rohsenow, 1999), and the cue-exposure treatment involves training of social or coping skills to better manage craving in the presence of the cue.

Support for the effectiveness of cue-exposure treatment has been mixed. Conklin and Tiffany (2002) performed a meta-analysis on available cue-exposure treatment studies, reporting a non-significant effect size of $d = .0868$. However, only 9 studies were included in the meta-analysis and these were highly heterogeneous. Monti and MacKillop (2007) reviewed the same 9 studies and noted opposing effects in the studies focused on smoking and alcohol use, with cue-exposure treatments yielding positive effects in alcohol studies but negative effects in smoking studies. Cue-exposure treatment may therefore be most effective in treating alcohol disorders, although recent studies have not found it to add extra benefit over the effects of CBT (Kavanagh et al., 2006).

2.3.1.4 Motivational interviewing

Motivational interviewing was first described by William Miller in 1983 as a technique for overcoming ambivalence and helping clients to get ‘unstuck’ (Miller,
Why worry about alcohol?

1983). Motivational interviewing was further refined by William Miller and Stephen Rollnick in 1991, and put forward as a technique for priming addictions patients for change (Miller & Rollnick, 1991), but has since broadened in scope and definition to be conceptualised as more of a client-centered counselling style aimed at amplifying intrinsic motivation to help clients change problem behaviours by assisting them to explore and resolve ambivalence about change (Rollnick & Miller, 1995). The popularity and use of motivational interviewing in addiction treatment settings and for the treatment of alcohol misuse has grown exponentially, fueled by consistent evidence supporting its effectiveness in soliciting change (Branscum & Sharma, 2010; Vasilaki, Hosier, & Cox, 2006).

2.3.1.5 Brief interventions

Brief interventions arose from the need to deliver opportunistic intervention to people presenting with alcohol-related problems within primary health care settings such as hospital emergency rooms (Bien, Miller, & Tonigan, 1993). Brief interventions typically include the basic elements of psychoeducation and assessment feedback and may also include basic skills training and, more recently, motivation enhancement. Evidence shows not only that brief interventions are effective in the treatment of alcohol misuse (Ballesteros, Duffy, Querejeta, Ariño, & González-Pinto, 2004; Kaner et al., 2009), but also that in many cases they are as effective as longer CBT-based treatments (Bien et al., 1993; Moyer, Finney, Swearingen, & Vergun, 2002).

2.3.1.6 Pharmacotherapies

A variety of pharmacotherapies for alcohol dependence have also been studied. Pharmacotherapies act at a neurochemical level to block the effect of alcohol or to induce unpleasant side effects. The three most commonly utilised pharmacotherapies
are: disulfiram, which impedes the metabolism of alcohol leading to a buildup of acetaldehyde which causes a range of unpleasant physical side effects; naltrexone, which binds with opioid receptors, blocking the pleasurable effects of alcohol and reducing craving, and; acamprosate, which depresses the elevated glutamatergic transmission and NMDA \((N\text{-}methyl~D\text{-}aspartate)\) receptor activation that occur in alcohol dependence and withdrawal, making abstinence from alcohol more easily tolerated (Mann, 2004). These therapies have all been found to be more effective than placebo or controls in achieving alcohol reductions (Boothby & Doering, 2005; Mann, 2004; Pettinati, Anton, & Willenbring, 2006; Roozen et al., 2006), and evidence suggests the combination of psychological intervention with both acamprosate and naltrexone together, may yield even better outcomes than psychological intervention combined with only one or the other (Boothby & Doering, 2005; Feeney, Connor, Young, Tucker, & McPherson, 2006).

2.3.1.7 Combination therapies

There has been some debate as to the additive benefit of combining pharmacotherapy with psychological therapies, and this question was addressed on a large scale in the COMBINE study (Combining Medications and Behavioral Interventions; The COMBINE Study Research Group, 2003). The COMBINE study was a large scale, multi-site randomised placebo controlled trial investigating if the combination of pharmacotherapy with behavioural interventions resulted in enhanced outcomes following treatment for alcohol dependence. The research randomised 1375 alcohol dependent treatment seekers to one of nine treatment conditions: placebo pill + placebo pill; placebo pill + acamprosate; naltrexone + placebo pill; naltrexone + acamprosate; placebo pill + placebo pill + behavioural intervention; placebo pill + acamprosate + behavioural intervention; naltrexone + placebo pill + behavioural intervention.
Why worry about alcohol?

intervention; naltrexone + acamprosate + behavioural intervention; and behavioural intervention + no pills. The results of the study revealed that after 16 weeks of treatment all nine groups showed significant reductions in days drinking, however, the best results were observed for the naltrexone + placebo pill group and the placebo pill + placebo pill + behavioural intervention group, suggesting either approach is efficacious for treating alcohol dependence, and that there is no additive benefit of combining therapies (Anton et al., 2006). Of interest, though, was the finding that the worst outcomes were observed for the behavioural intervention + no pills group, suggesting a placebo effect contributed to the enhanced outcomes for the behavioural intervention + placebo pill group, even though behavioural intervention + real pharmacotherapy did not result in enhanced outcomes. These results are in contrast with other studies that have found that combining therapies results in the best outcomes (Boothby & Doering, 2005; Feeney et al., 2006; Walters, Connor, Feeney, & Young, 2009), so this is clearly an area requiring further research.

2.3.2 Recent advances in treatment

2.3.2.1 Mindfulness and acceptance-based interventions

Mindfulness and acceptance-based interventions are also gaining momentum in treatment of addiction disorders. These approaches are derived from Buddhist philosophies and, unlike disease models that posit that the causes of addiction lie in biological origins, they hold that the cause of addiction is based in the mind (Marlatt, 2002). Unlike CBT which targets and challenges thoughts directly, mindfulness aims “not to change the content of the thoughts (as in cognitive therapy), but to develop a different attitude or relationship to thoughts and feelings as they occur in the mind.” (Marlatt, 2002, p. 47). In both mindfulness and acceptance-based therapies, negative
cognitive and emotional states are handled by approaching them with detached observation and ‘accepting’ their existence, but ‘letting go’ of the need to engage the thoughts or act on them. Khong (2009) summarises the approach eloquently:

Mindfulness practice encourages us to be aware of feelings (experiences, thoughts, beliefs, etc.) and experience them as they are (letting it be) without needing to change or do something about them (accepting things as they are). In short, to allow ourselves to just be with the feeling, by making space for it, and not identifying with it (nonattachment). In so doing, we can learn to let the feeling go. (p. 131)

Evidence supporting its effectiveness as a treatment for alcohol misuse is still building and more research needs to be done, but so far the results look promising (Vieten, Astin, Buscemi, & Galloway, 2010; Witkiewitz, Marlatt, & Walker, 2005; Zgierska et al., 2009).

2.3.2.2 Computerised psychological interventions

Remotely delivered psychotherapies are also becoming more popular and more widely available, partly in response to statistics revealing that the majority of people with substance use disorders do not access any kind of treatment (Australian Bureau of Statistics, 2007), and partly as a potential solution to overburdened health systems. While the research investigating efficacy of computerised and web-based interventions for alcohol use is not prolific, the available evidence shows promise for these forms of therapeutic delivery and their effectiveness at achieving positive alcohol reductions (Bewick et al., 2008; Khadjesari, Murray, Hewitt, Hartley, & Godfrey, 2011; Riper et al., 2011; Rooke, Thorsteinsson, Karpin, Copeland, & Allsop, 2010; White et al., 2010).
Why worry about alcohol?

2.3.3 Treatment impact

2.3.3.1 Relative efficacy

There is an extensive amount of research indicating that all of the interventions discussed above are effective in helping people to reduce their alcohol intake and achieve higher recovery rates than the estimated 11-44% rate of natural recovery (recovery achieved without treatment) observed in alcohol users (Hasin, Liu, & Paykin, 2001; Moos & Moos, 2005; Moos & Moos, 2006; Timko, Moos, Finney, & Lesar, 2000). This indicates these treatments are more effective than no treatment. For example, abstinence rates one year after AA or 12-step facilitated treatment range from 15-50% (Galaif & Sussman, 1995; Kaskutas, 2009; Kelly, Stout, Magill, Tonigan, & Pagano, 2010). CBT studies have yielded high proportions of abstinent days (70-80%) throughout treatments lasting 16 and 26 weeks (Anton et al., 2006; Kadden, Litt, Cooney, Kabela, & Getter, 2001) and post-treatment (12-26 weeks) abstinence rates of 30-40% (Anton et al., 2006; Burtscheidt, Wölwer, Schwarz, Strauss, & Gaebel, 2002; Feeney et al., 2006). Treatment effects are well maintained with abstinence rates between 20 and 40% observed 6 to 12 months following treatment (Burtscheidt et al., 2002; The Project MATCH Research Group, 1997). Although CBT is not always found to outperform other treatments such as motivation enhancement therapy or Twelve-Step Facilitation (The Project MATCH Research Group, 1997) or pharmacotherapies (Anton et al., 2006; Feeney et al., 2006), a meta-analytic review concluded that CBT yielded a small but statistically significant treatment effect ($g=0.154$; Magill & Ray, 2009).

A review of brief interventions reported substantial reductions in alcohol use ranging from 29-78% that were maintained throughout follow-ups of 3-24 months (Bien et al., 1993), with an average of 38g of alcohol per week difference at 1-year follow-up between people receiving brief interventions and people receiving control treatment.
(Kaner et al., 2009). Treatments combining cue-exposure with coping skills training have yielded reductions in drinks per drinking day of 57-79% (Monti et al., 1993; Rohsenow et al., 2001) and an increase of percentage of 3-month abstinent days of 83% (Monti et al., 1993). Pharmacotherapies achieve an approximate 35% rate of continuous abstinence throughout active treatment (Mann, 2004), and higher rates of 70% have been observed when combined with behavioural interventions (Feeney et al., 2006).

The literature supporting the efficacy of a broad range of addiction treatments is quite substantial. Miller and Wilbourne (2002) compared the relative efficacy of the most common treatment approaches for alcohol use, reviewing 361 controlled trials published since 1970. Cumulative evidence scores were calculated for each treatment type based on the outcomes of trials (positive effect in favour of the intervention or negative effect) and methodological quality of the study. Out of 46 treatment approaches examined, Brief Interventions yielded the highest cumulative evidence score due to the large number of studies examining this treatment, the high proportion reporting positive effects, and the high methodological quality of the studies. Motivational Interviewing interventions were ranked second out of 46, and acamprosate and naltrexone were ranked third and fourth respectively. Cognitive therapy was ranked 11 out of 46, ahead of self-help which was ranked at 18, 12-step facilitation treatments which were ranked 24 and Alcoholics Anonymous which was ranked 39 out of 46.

This evidence base highlights that psychological and pharmacological interventions can be more effective than no or limited treatment in reducing alcohol use. However, the abstinence rates discussed above convey that there are still substantial portions of people who do not appear to respond to treatment, or who respond only partially, or who relapse during the post-treatment period, regardless of the type of intervention received.
Why worry about alcohol?

2.3.3.2 Relapse

In the large scale Project MATCH treatment study (Matching Alcoholism Treatments to Client Heterogeneity), which matched treatment seeking alcoholics to CBT, 12-step facilitated or motivation enhancement therapy based on a number of matching rules and criteria, only 29.4% of the whole study sample reported complete abstinence in months 37-39 following initial intake into the study (The Project MATCH Research Group, 1998). In the COMBINE study, 79% of patients who received behavioural intervention with no pills had a relapse to heavy drinking before treatment had even been completed, and across all study groups, approximately 80% reported at least one day of heavy drinking at the 12-month follow-up assessment. Feeney et al. (2006) showed similarly high rates of relapse in their study of combined CBT + pharmacotherapy. The CBT only group showed 30% relapse within 2 weeks of starting the program, and by the end of treatment 10 weeks later, a total of 60% had relapsed. While relapse was lower in the groups who received adjunctive pharmacotherapy, rates were still high, with approximately 50% of the CBT + acamprosate group relapsing by the end of the 12 week treatment, and approximately 35% of the CBT + naltrexone and CBT + naltrexone + acamprosate groups relapsing by 12 weeks.

As would be expected, rate of relapse varies across studies depending on how relapse is defined, the type and intensity of treatment administered and individual factors, but in the short term, rates have been observed to range between 20 and 50% and in the long term between 20 and 80% (Moos & Moos, 2006). Being able to identify which individuals might be most at risk for relapse would be very advantageous for treatment and follow-up planning. Addition of adjunctive therapies such as naltrexone and acamprosate to psychological therapies have been found in numerous studies to result in lower relapse rates than psychological therapy alone (Boothby & Doering,
combined therapies could be considered as one avenue to enhance treatment for people who may be at risk for relapse. High social engagement achieved through participation in self-help groups has been identified as an important factor maintaining recovery in people who have reduced drinking without the help of formal intervention (Bischof, Rumpf, Hapke, Meyer, & John, 2000), and this could be another avenue to supplement or enhance treatment for people at higher relapse risk. Scott, Dennis and Foss (2005) propose a Recovery Management Checkup model, where patients are assessed for regular, brief checkups and re-engaged in treatment if required, to reduce transition from recovery to relapse. This would be a costly model to employ within a health service on a large scale, but could conceivably be implemented on a smaller scale with people identified as being more likely to relapse within the first year of treatment.

There has been much research interest in factors associated with treatment outcomes and relapse, and in the prediction of outcomes following treatment (Adamson, Sellman, & Frampton, 2009; McKay & Weiss, 2001; Witkiewitz, 2011). Adamson et al. (2009) reviewed studies of patient predictors of alcohol treatment outcome and concluded that of all variables examined across studies, baseline consumption and severity of dependence were the two variables that accounted for most variance in prediction models. Witkiewitz (2011) also acknowledged that most of the variance in drinking outcomes in the COMBINE data set was explained by drinking variables rather than other predictors. Baseline drinking should therefore always be controlled for when studying outcome predictors. While there is no doubting that past drinking behaviour is the best predictor of future drinking behaviour, when everyone presenting to a treatment service is consuming large quantities of alcohol and reporting high levels of dependence, these variables alone are unlikely to be enough to differentiate those people...
Why worry about alcohol?

who are at the highest risk of relapsing to heavy drinking following treatment.

Examining other variables, aside from the amount of alcohol already being consumed, may provide the additional information needed to best identify those at greatest risk for relapse. One variable that has potential to provide this additional information is craving.
CHAPTER 3  ROLE OF CRAVING IN ADDICTION

3.1 What is craving?

In the addiction literature, there is much debate about the nature of craving and how the concept should be defined. The terms ‘craving’, ‘urge’ and ‘desire’ tend to be used interchangeably, despite arguments that the term craving is ambiguous and can lead to confusion because some believe it to mean a strong urge to use a drug, and others believe it to mean any urge (Kozlowski, Mann, Wilkinson, & Poulos, 1989; Kozlowski & Wilkinson, 1987). Some agree with the recommendation of Kozlowski and colleagues that the term ‘urge’ be used instead of ‘craving’ as it is less ambiguous and a better descriptor of the construct being measured (Hughes, 1987). Others argue that the terms low-craving and high-craving can be used meaningfully to describe varying intensities of the same construct, and that the concept of craving does not have to be limited to the most severe end of the desire spectrum (Stockwell, 1987). Marlatt (1987) attempted to differentiate the terms by defining craving as a motivational state and urge as representing behavioural intention. West (1987) argues that attempts to define the term craving are unproductive and lose the fundamental sense of craving as being a subjective state that covers the whole motivational continuum.

Despite these arguments, the word craving is often used interchangeably with urge and desire, although its definition and description vary depending on the theoretical framework in question (Skinner & Aubin, 2010). Franken (2003) asserted that craving is an emotion, since desire is the emotion that accompanies approach behaviour, just as fear is the emotion that accompanies avoidance. He defines craving as “the accompanied emotional state that is produced by conditioned stimuli that are
Role of craving in addiction

associated with the reward effects of substances or behaviour.” (p. 565). Others argue, that while craving is affective in nature (Kavanagh, Andrade, & May, 2005), it also involves discrete cognitive processes (Nosen & Woody, 2009; Witkiewitz & Marlatt, 2004). More generally, craving is described as a motivational state that is often accompanied with affective and/or cognitive elements (Kavanagh et al., 2005; Monti, Rohsenow, & Hutchison, 2000; Tiffany, 1990). Drawing these observations together, craving is generally considered to be a subjective motivational state (Shiffman, 2000) representing a wanting or desire for the target substance (Monti et al., 2000) that may be affectively laden, but is primarily cognitively driven (Kavanagh et al., 2005).

In efforts to better understand the phenomenology of craving, Merikle (1999) and Westerberg (2000) both qualitatively examined substance users’ self-reported experiences of craving. Merikle (1999) asked 23 substance misusers to provide a definition of craving. The word ‘want’ appeared in the definitions of 74% of participants and the wanting was described as powerful and intense. The definitions also evinced obsessional and appetitive qualities, and craving was viewed as being distinct from the ‘need’ associated with physical withdrawal. Participants were also asked about their subjective experience of craving, and responses were found to fit largely consistently within eight hypothesised dimensions (specificity; strength; positive outcomes; behavioural intention; thoughts; physical symptoms; affect; cues), but to also have a high degree of heterogeneity between individuals (Merikle, 1999).

Westerberg (2000) sought to ‘profile’ the experience of craving by investigating the symptoms and features that differentiated between people who report experiencing craving and those who do not. Adults presenting for alcohol treatment were administered a checklist and asked to rate the frequency in the past 30 days of 37 items that described both physiological and psychological events reported in other studies to
relate to craving. Participants were also asked if they had craved alcohol, had an urge for alcohol and had a drink. Westerberg found that 16 of the 37 items reliably discriminated between people who reported high craving versus low. These items pertained to physical events (feelings in body, queasy feeling, feeling tense or jittery), psychological events (thoughts about alcohol, feelings of defeat, alcohol thoughts staying in mind, not able to get rid of alcohol thoughts, thoughts about taste of a drink, feelings of failure, images of taste, feeling alcohol is the most important thing, feeling ‘blue’, feeling depressed, thoughts of taste of alcohol) and environmental cues (seeing alcohol, seeing advertisements for alcohol). Westerberg’s (2000) study clearly demonstrates that whatever definition of craving is adopted, from an individual’s perspective, the subjective, consciously perceived experience of craving is multidimensional and encompasses a broad range of physiological, cognitive, affective and cue-based elements.

3.2 Why craving is important

Craving is one of the most extensively studied and debated variables in addiction research, in large part because it is one of the most reliably observed phenomenon. Most addiction patients report experiencing craving and there is substantial evidence demonstrating a link between craving and substance use.

In Westerberg’s (2000) study, the majority of the sample of alcohol users reported having experienced cravings or urges in the past 30 days. Only 28% of the sample reported having none. Studies have consistently observed high rates of self-reported craving prior to periods of use for most substances including alcohol, cigarettes and cocaine (Chandra, Scharf, & Shiffman, 2011; Cooney et al., 2007; Dunbar, Scharf, Kirchner, & Shiffman, 2010; Rankin, Hodgson, & Stockwell, 1979; Shiffman et al.,
Role of craving in addiction

2002; Weiss et al., 2003; Yoon, Kim, Thuras, Grant, & Westermeyer, 2006), with higher craving levels being associated with higher rates of subsequent drinking and smoking, and with increased drinking speed (Rankin et al., 1979). High subjective craving has also been observed as a common antecedent to relapse (Allen, Bade, Hatsukami, & Center, 2008; Cooney et al., 2007; McKay, 1999; McRobbie, Hajek, & Locker, 2008; Oslin, Cary, Slaymaker, Colleran, & Blow, 2009; Waters et al., 2004).

Craving has been reliably observed to increase following substance cessation and during acute withdrawal (Krahn, Bohn, Henk, Grossman, & Gosnell, 2005; Sherman, Morse, & Baker, 1986) and is frequently reported as one of the most predominant characteristics of the withdrawal experience (Cornelius, Chung, Martin, Wood, & Clark, 2008; Van Zundert, Boogerd, Vermulst, & Engels, 2009; Zorick et al., 2010).

It is clear that craving is an integral part of the experience of addicted substance users, and plays a key role in the maintenance and reinstatement of substance use behaviour. For this reason, craving is an integral component of most theories of addiction.

3.3 Models of craving in addiction theory

Numerous theoretical models have been put forward to explain the initiation and maintenance of addictive behaviours. These theories all have a unique point of view, but also share a number of common elements, the role of craving being one of them. Substantial research has been devoted to trying to understand the nature of cravings and how they influence substance use behaviours. A number of models and theories have been put forward that attempt to describe how cravings originate, what perpetuates them and how they influence drug seeking and using behaviour. These theories continue to be developed over time, as research expands and understanding of the addiction process
grows richer. The summary in this section is not an exhaustive overview of every craving or addiction theory, but covers most of the major theories that drive addiction research and for which craving is an important component.

3.3.1 Cognitive model

Tiffany’s (1990) cognitive model of drug use behaviour is the leading cognitive-based addiction theory. Tiffany’s theory proposes that the skills and behaviours required to acquire and consume substances become stored in memory in the form of automatised action schemata. Over time, with repeated practice, drug seeking and using behaviour becomes an automatic cognitive process that can occur without intention or full awareness. The more readily available and accessible the drug is to the person and the fewer the obstacles that have to be overcome to acquire and use it, the more readily the process of automatisation will occur. The action schemata then become triggered by exposure to external cues (e.g., environment, time of day) and/or internal cues (e.g., moods, withdrawal), leading to activation of the automatic cognitive processes and engagement in drug seeking behaviour and use. In this model of addictive behaviour, craving is epiphenomenal in that it is a non-automatic process only generated when the automatic drug seeking behaviour is blocked, either because of an external (e.g., running out of cigarettes in the middle of the night) or internal obstruction (e.g., quit attempt). This triggers a need for active cognitive processes to problem solve to overcome the encountered obstacle. If the automatic process is not blocked, cravings are not generated. Tiffany (1990) separates cravings into two categories; abstinence-avoidance and abstinence-promotion.
Role of craving in addiction

Abstinence-avoidance urges

Abstinence-avoidance urges (urges to use substances) are generated when automatic drug use is blocked, and when the drug user desires to enact the usual drug seeking behaviour and achieve drug use. This urge relates to a desire to avoid abstinence, that is, a desire to use the substance.

Abstinence-promotion urges

Abstinence-promotion urges are generated when cues that activate the automatic process are present, but the user seeks to remain abstinent. Tiffany argues that this is a particularly difficult state, as the user has to inhibit previous responding, and also has to deal with reinforcement contingencies that were also activated by the cue.

Support for Tiffany’s model has been found in laboratory based studies that have demonstrated diminished cognitive performance when drug urges are cued (Sayette & Hufford, 1994; Zwaan, Stanfield, & Madden, 2000), suggesting cognitive resources are being devoted to processing of the urge response. Evidence for automatised lapses has also been found in ecological momentary assessment of smokers attempting to quit, although, in contrast to Tiffany’s model, cravings were not rated as being absent prior to these lapses (Catley, O’Connell, & Shiffman, 2000). The authors acknowledge though, that these ratings may have been due to the assumption in retrospect that if a lapse had occurred, it must have been triggered by craving.

3.3.2 Negative reinforcement theory and affective model of drug motivation

Negative reinforcement theory is one of the oldest addiction theories, having been conceptualised more than half a decade ago (Wikler, 1948). In its earliest days, the theory centred around the physiological sequelae following cessation of a substance
after a period of prolonged use. Prolonged substance use results in physiological adaptations at a cellular level (Koob & Bloom, 1988), the neurochemical consequences of which cause the substance to have diminished effect over time (Koob et al., 1998). When substance use is suddenly stopped or dramatically reduced, the neurochemical balance, or homeostasis, is disturbed (Littleton, 1998), resulting in a series of highly unpleasant physical and psychological symptoms that constitute the withdrawal syndrome (West & Gossop, 1994). According to negative reinforcement theory, drug seeking behaviour is driven by a desire to avoid the unpleasant symptoms associated with withdrawal (Wikler, 1948).

Negative reinforcement models are not just restricted to relief from the adverse biological effects of withdrawal. The self-medication hypothesis asserts that people use substances as a coping mechanism to relieve or alter negative affective states and psychological distress (Khantzian, 1985). Research exploring the notion of self-medication has yielded mixed results, with some finding support for the hypothesis (Colder, 2001; Dixon, Leen-Feldner, Ham, Feldner, & Lewis, 2009; Leeies, Pagura, Sareen, & Bolton, 2010; Ouimette, Read, Wade, & Tirone, 2010; Weiss, Griffin, & Mirin, 1992), and others not (Gregg, Barrowclough, & Haddock, 2007; Hall & Queener, 2007). Research evidence showing support for negative reinforcement expectancies predicting and influencing later substance use does exist (Carey & Correia, 1997), but most studies find it is just one of a number of factors.

In negative reinforcement models, craving is elicited in the face of aversive physical or affective states, and is a manifestation of the desire to relieve that negative state. It is a response based on conditioning and prior learning that acquisition of the substance will alleviate the negative condition. Baker, Piper, McCarthy, Majeskie and Fiore (2004) present a model of craving (described as drug motivation) based on
Role of craving in addiction

negative reinforcement theory. Their model shares concepts of preconscious and conscious cognitive processing with Tiffany’s (1990) model of urges, but where Tiffany’s model describes craving as a cognitive event, Baker et al. describe it as an affective one. According to Baker et al.’s model, drug users become sensitive over time to internal cues that signal withdrawal from a substance and to the association of these cues with ensuing negative affect. Over time, detection of these cues becomes preconscious and prompts drug use routines. The authors argue that while the individual may be aware of wanting to use drugs and of using them, they are typically unaware of what drove the motivation. Like Tiffany’s model, when access to drugs is readily available, drug use tends to occur without awareness, but in Baker et al.’s model, this is to escape and avoid negative affect caused by falling levels of the drug.

Higher levels of negative affect, caused either by interruptions in drug use or significant stressors, enter consciousness and influence information processing in ways that promote drug seeking and use. This is called ‘hot information processing’, which biases the drug user’s attention to focus on perceived threats and on negative affect, making avoidance of or escape from the negative affect the primary motivational urge. This hot information processing also decreases ‘cool information processing’, interfering with employment of cognitive control and implementation of coping strategies. When negative affective levels are moderate or mild, it is possible for cool processing to prevail and to interrupt the motivational processing, enabling engagement of cognitive control resources.

Unlike Tiffany’s model where urge and consciousness are only generated when automatic responding is blocked, in Baker et al.’s model, motivational urge and consciousness are generated when negative affect is high. When negative affect is low,
drug use remains proceduralised. In this model, craving itself is not an affect, but is driven by affective conditions.

Baker et al.’s proposition that craving and drug use are generated in the context of negative affect is supported by evidence that negative mood is reliably found to precede substance use episodes and to induce drug cravings (Cleveland & Harris, 2010; Fucito & Juliano, 2009; Perkins et al., 2008). Negative affect instigated by abstinence has also been found to be a stronger predictor of smoking cessation outcomes than general psychological distress (Kenford et al., 2002). However, cravings can occur long after withdrawal from a substance has passed (Robinson & Berridge, 1993) and lapses have also been found to occur in the context of positive affect situations (Shiffman, 1982; Swendsen et al., 2000).

3.3.3 Positive reinforcement and opponent-process theory of motivation

de Wit and Phan (2010) provide an overview of the positive reinforcement theory of addiction and the evidence supporting it. The basic premise of positive reinforcement theory is that drug use results in positive and pleasurable outcomes that the drug user seeks to reproduce. Drug-seeking behaviour is driven by a desire, or craving, for these positive effects of the substance. This theory is supported by evidence that alcohol produces pleasurable and euphoric effects, evidenced in both animal and human research (Amit & Smith, 1985; Gilman, Ramchandani, Davis, Bjork, & Hommer, 2008), and by findings that alcohol consumption is often associated with expectancies for positive outcomes (Colder & O'Connor, 2002; Palfai, Ralston, & Wright, 2011) and that positive expectancies predict subsequent alcohol consumption (Kuntsche & Cooper, 2010; Yusko, Buckman, White, & Pandina, 2008). However, the theory fails to account for observations that the motivation to take drugs is not always
Role of craving in addiction

linked to subjective pleasurable effects (Robinson & Berridge, 2000), leading to
distinction between drug ‘wanting’ from drug ‘liking’ (Berridge & Robinson, 1995).

The opponent-process theory of motivation (Solomon & Corbit, 1973) draws on elements of positive reinforcement theory, as well as negative reinforcement, to describe and explain the cycle of addiction. It proposes that substance use is initially positive and pleasurable (stage A), and its absence then becomes associated with a negative sense of deprivation (stage B) that drives further drug seeking behaviour and forms the basis of a feedback loop where substance use is sought both for the positive effects it yields, but also to relieve the negative state associated with its absence (A → B → A’ → B’). In this theory, craving is elicited in stage B’ in the same way as in negative reinforcement theory; in response to perception of an aversive physical or psychological state. But in opponent-process theory, the motivation behind the craving is not only to relieve the negative state as it is in negative reinforcement theory, but also to achieve the pleasurable effects of the substance encountered in stage A’, as per positive reinforcement theory. Over time, as stage A’ fails to produce the level of pleasure it once did due to development of tolerance to the drug’s effect, the state B’ becomes increasingly aversive, and craving consequently becomes more intense over time, driving the process in a feedback loop.

While studies testing the principles of opponent-process theory have found some support for the premises of affect habituation and affect reversal (Mauro, 1988; Sandvik, Diener, & Larsen, 1985), the premise that the withdrawal effect associated with state B’ should be greater with greater habituation to state A’ has failed to be supported, suggesting that affect habituation is not due to opponent processes (Sandvik et al., 1985). Observations that substance use and craving are not always associated with
positive expectancies (Robinson & Berridge, 2000) also contradicts the premise that substances are sought for their pleasurable effects.

### 3.3.4 Biological theories of addiction and the incentive-sensitisation model

Changes in the form of reinforcement following repeated substance use that were highlighted in opponent process theory (from positive to negative reinforcement) can also be explained by changes in neural sensitisation. The mechanisms of drug effects lie in biological pathways, so it is logical that addiction theory should have at least some basis in neurophysiology. Incentive-sensitisation theory is one of the leading biological theories of addiction. This theory, proposed by Robinson and Berridge (1993), posits that “the defining characteristics of addiction (craving and relapse) are due directly to drug-induced changes in those functions normally subserved by a neural system that undergoes sensitization-related neuroadaptations.” (p. 249). In this model, ‘sensitisation’ refers to “a progressive increase in a drug effect with repeated treatments” (p. 249). According to the incentive-sensitisation theory, drug use activates the dopamine system and incentive salience is attributed to stimuli associated with this activation, causing them to become highly attractive and wanted. Repeated drug use causes the system to become hypersensitive (sensitised) and enhanced incentive salience is then attributed to the act of drug taking and to stimuli associated with it. Over time, the drugs come to be pathologically wanted, or craved. The sensitisation responsible for incentive salience motivates compulsive drug seeking behaviour, regardless of other motivating factors or disincentives, and the drug craving is related neither to pleasure nor withdrawal.

This biological model of addiction has strong roots in animal research (Robinson & Berridge, 1993, 2000) and is supported by observations in human addiction also. The
Role of craving in addiction

neurobiological systems proposed to give rise to incentive salience are found to become activated on exposure to alcohol cues (Filbey et al., 2008), and preliminary evidence exists that disrupting these systems decreases craving and facilitates abstinence (Heinze et al., 2009). Further evidence is found in observations that stimuli associated with substance use can trigger craving (Winkler et al., 2011) and activate dopamine systems (Boileau et al., 2007) in the absence of the drug cue itself.

In Robinson and Berridge’s (1993) model, craving is presented as synonymous with addiction. They describe it as one of the defining characteristics of addiction and state that craving is “manifest behaviorally as compulsive drug seeking and drug taking.” (p. 249). Unlike other models of craving, the process is entirely biologically driven, and the model does not attribute affective qualities to the drug use experience. Craving is defined as a “subjective experience that accompanies the attribution of excessive levels of incentive salience to drug-related stimuli (or their mental representations), due to sensitization of dopamine systems.” (p. 266). They describe craving as a psychological process and as a state of pathological ‘wanting’. It is argued to be distinct from both the pleasures associated with drug use and from the unpleasant symptoms of withdrawal. It is due solely to “excessive activity in a separate and sensitized neuronal system that mediates the attribution of salience to incentives. This is a neuronal system that normally mediates the ‘wanting’ of things in the environment.” (p. 266). Because subjective pleasure is not required for the attribution of salience, incentive salience (or craving), can be high even when pleasure is low. Much of the salience attribution process is implicit, often leading to an unawareness of why the drug is being craved, and explains why craving persists even when the individual views drug use as being negative and undesirable.
Robinson and Berridge have published extensive evidence supporting the incentive-sensitisation model of addiction and craving (Berridge & Robinson, 1995; Robinson & Berridge, 1993, 2000, 2003), highlighting that addicted drug users continue to seek drugs even in doses insufficient to produce pleasure, that craving frequently occurs in the absence of withdrawal, and that manipulations of the dopamine system alter ‘wanting’ more than ‘liking’.

Franken (2003) takes biological theories of addiction a step further by integrating components of psychological based theories. Franken proposes a cognitive psychopharmacological model, where perception of drug related stimuli results in activation in the dopamine system which triggers attentional bias to drug related cues and an increase in drug related cognitions. This attentional bias elicits craving, which further enhances the attentional bias. When the cognitive load is high, it diminishes resources for alternative cues (e.g., coping strategies), and drug use and relapse ensues.

### 3.3.4.1 Role of genetics

Alcohol dependence is estimated to be based at least 50% on genetic factors (Müller, Likhodi, & Heinz, 2010). Linkage studies, which examine markers that are inherited and are linked to substance use disorder, have implicated regions on numerous chromosomes, but findings have not been consistent across studies, and linkage analyses are only sensitive to genes of major effect (Ball, 2008; Müller et al., 2010). Association analyses compare variations in DNA sequences between unrelated cases and nonaffected controls, and are capable of detecting genes of relatively small effect (Ball, 2008). Of particular interest, and most widely researched, are polymorphisms of receptor, transport and metabolic genes associated with the neurotransmission and metabolism of substances.
Role of craving in addiction

The most robust finding in genetic research relates to variation in the enzyme gene aldehyde dehydrogenase 2 (ALDH2). ALDH2 breaks down acetaldehyde, a toxic byproduct of alcohol metabolism associated with unpleasant physiological effects (Edenberg, 2007). People who have an inactive variant of this gene do not break down acetaldehyde as readily, making consumption of alcohol aversive, reducing likelihood of developing an alcohol disorder (Ball, 2008; Edenberg, 2007; Kohnke, 2008). Some support has also been found for an effect of the D2 dopamine receptor gene (DRD2) (Noble, 2012). The mesolimbic dopaminergic system plays a key role in the rewarding properties of substances (Tomberg, 2010a). Alcohol is proposed to be more reinforcing in people with the DRD2 A1+ allele compared to people with the A1- allele, due to reduced glucose metabolism in the mesocortico-limbic dopamine reward system in people with the A1+ polymorphism (Noble, 2012). Consumption of alcohol increases dopamine levels, activating brain glucose metabolism in areas associated with pleasure, making alcohol use rewarding and reinforcing (Noble, 2012).

Genetic influences are not only of interest for the development and maintenance of substance use disorder. They may also underlie individual variability in response to substance cues, explaining some of the subjectivity observed in craving (MacKillop & Monti, 2007) and representing important phenotypes for alcohol misuse (Monti & Ray, 2012; Ray et al., 2010). Evidence supporting this notion is emerging. Ray et al. (2010) found the long allele of the dopamine D4 receptor gene (DRD4) to be associated with greater self-reported urge to drink following alcohol consumption, compared to people with the short allele. Agrawal et al. (2013) examined a number of genes implicated in past research as being associated with craving. These included dopamine receptor and transporter genes as well as alpha-synuclein (SNCA), which regulates dopamine D2 synthesis. The authors found that multiple single nucleotide polymorphisms in the
dopamine D3 receptor (DRD3) and SNCA were associated with craving, and that this association appeared to be independent of alcohol dependence. Some evidence for stress-induced and cue-induced endophenotypes has also been found, with Ray (2011) finding variation in polymorphisms of the corticotrophin-releasing hormone binding protein (CRH-BP) gene being associated with stress-induced craving, while polymorphisms of the µ-opioid receptor (OPRM1) gene were associated with cue-induced craving following presentation of neutral imagery compared to stressful imagery.

Genetic research has investigated a wide range of genes associated with multiple neuronal and metabolic systems in a quest to identify endophenotypes of substance use disorder. In addition to those discussed above, numerous genes associated with the dopamine, γ-aminobutyric-acid (GABA), glutamate, opioid, cholinergic and serotonin systems (Kohnke, 2008) have been studied, but findings have been inconsistent and studies have not been able to conclusively identify genes involved in addiction (Ball, 2008; Kohnke, 2008). The field is also hampered by the high false positive rates of association studies (Ball, 2008). More research is needed to be able to draw definitive conclusions about the role of genetics in addiction and craving.

3.3.5 Elaborated intrusion theory of craving

Elaborated intrusion theory (Kavanagh et al., 2005) is a relative new-comer to the field of craving research, and aims to consolidate some of the important elements of addiction and craving models into a single coherent theory. One of the biggest obstacles with craving theories to date is the failure of craving to perform consistently in the predicted ways (Tiffany & Wray, 2009), implying a failure of existing theories to describe the construct adequately. For example, models based in negative reinforcement
Role of craving in addiction

theory which argue that craving represents a desire to avoid aversive physical and psychological states such as withdrawal and negative affect, are contradicted by findings that substance use and craving can also occur in the presence of positive affective states (Baer & Lichtenstein, 1988; Shiffman, 1982, 1986; Swendsen et al., 2000), are also appetitively driven, and that drug use can be reinstated long after the withdrawal syndrome has extinguished (Baker et al., 2004; Robinson & Berridge, 1993). A model of craving that can encompass the various ways in which craving presents and relates to actual drug use behaviour is needed. Elaborated intrusion theory is presently the closest to achieving this.

Like Tiffany’s (1990) cognitive model of drug urges, elaborated intrusion theory is a cognitive craving theory, where cravings are the product of both automatic associative events and effortful cognitive processes. Consistent with Baker et al.’s (2004) affective model of craving, elaborated intrusion theory posits that affect is a key aspect of the craving process, but where Baker et al. argue that craving is generated in response to negative affective states and cues, elaborated intrusion theory argues that affective cues can be either positive or negative, and that negative affect is also a consequence of the craving process, and not just a cue. Unlike other craving models which relate specifically to psychoactive substances, elaborated intrusion theory is applicable to all desires, and the authors argue that “such a broad applicability is in fact essential to an adequate theory of desires and craving.” (p. 447).

The authors present a theoretical model where a variety of factors can trigger intrusive cognitions (which can also take the form of mental images) about the appetitive target. These triggers can include external cues that have previously been associated with the target (learned associations), physiological deficit states associated with deprivation and withdrawal, negative affect, cognitions pertaining to the target
(e.g., availability, anticipated outcomes) and involuntary anticipatory responses (e.g., salivation, increased heart rate). When these intrusive cognitions reach awareness, a process of elaboration ensues, where stored information pertaining to acquisition and consumption of the target is sought and retrieved, then manipulated and embellished in working memory.

Imagery is described in the theory as a cognitive event, and is argued to play a key role in the elaboration process. In fact, the authors argue that imagery lies at the very heart of desire. The intrusive cognitions triggered by target related stimuli may take the form of mental images, but it is their role in the elaboration process that places them at the heart of intense desire. Once intrusive cognitions related to the target have entered consciousness, an active process of embellishment and elaboration commences, driving a growing desire for the target. The authors argue that the form of this elaborative process is primarily based in mental imagery, which may involve visualisation of the target itself, actions required to obtain the target, or senses associated with consumption of the target such as tastes, smells or physical sensations. The more vivid the imagery is during this elaborative process, the stronger the desire for the target will be, and the more effective the process will be in activating emotional and motivational pathways.

The authors argue that the predominant emotional reaction associated with extended episodes of craving is in fact negative. While initial images and thoughts relating to the target may be pleasurable, the cognitive elaborative process heightens awareness of the absence of the target, and gives rise to a sense of deprivation. During attempts to control or limit the target, the process can give rise to feelings of guilt or shame. These emotional consequences interact with other factors such as availability of the target, balance of other incentives, and skills and self-efficacy to acquire or resist the
Role of craving in addiction

target. The balance of these interacting factors influences the response to the craving and whether the target is actively sought and obtained, or if it is successfully resisted.

Although elaborated intrusion theory is relatively new, a series of laboratory studies have yielded consistent results supporting its basic tenets. Strong cravings have been observed to be characterised by a greater degree of imagery than weaker cravings (May, Andrade, Kavanagh, & Penfound, 2008), and active engagement in directed visual and olfactory imagery tasks has been found to reliably reduce craving for cigarettes (May, Andrade, Penabokke, & Kavanagh, 2010; Versland & Rosenberg, 2007), food (Kemps & Tiggemann, 2007) and coffee (Kemps & Tiggemann, 2009). 

Imagery was also found to be a key feature of craving in a clinical sample of alcohol users, with image frequency correlating with stronger craving (Kavanagh, May, & Andrade, 2009). Intrusive thoughts were also found to be commonly reported (Kavanagh et al., 2009; May et al., 2008).

3.4 **Implications of craving**

Across all of these theories and models of addiction and craving, there is a common theme and assumption that there is a causal (though not necessarily inevitable) link between craving and drug seeking, albeit modified by a number of variables (Tiffany, 1990). Craving has already been shown to be an important antecedent to episodes of substance use and to relapse, and it has been shown also to be predictive of ad libitum drinking (Leeman, Corbin, & Fromme, 2009) and smoking (Carpenter et al., 2009), although the correlations are often observed to be small to moderate (Tiffany, 1990). Of real clinical interest however, is whether craving could act as a possible predictor of outcomes following substance use intervention. In theory, it would seem
that high levels of craving should predict greater substance use and relapse risk following treatment. In reality this is not always found to be the case.
A significant amount of research has been conducted over the last several decades examining the utility of craving in predicting outcomes following substance use intervention. As emphasised earlier, the ability to predict who is more likely to respond poorly to treatment and is at greatest risk of relapse would be a significant advantage in treatment and resource planning. Craving’s performance as a predictor has been studied in the context of multiple substances of abuse, varying interventions and treatment settings, and using a wide variety of assessment methods and defined outcomes. It is therefore perhaps not surprising that the results have been quite mixed.

The selection of studies for this brief review was limited to those that investigated prediction of treatment outcomes from craving measured before treatment commenced. The review was restricted in this way because the focus of the present research is on studying craving as a potential variable that could be examined in patients when they first present for treatment to identify those people who may be at greater risk for relapse after treatment, and that this information could be used to assist treatment planning. Studies that predicted outcome using craving measured during or after treatment were therefore excluded, as were studies that predicted craving as the outcome. Studies were identified primarily through database searches using the PsycInfo database search engine, and were also indentified from citations in other relevant papers. Table 4.1 provides an overview of the studies included in this review.
### Table 4.1

*Studies predicting post-treatment substance use outcomes from pre-treatment craving measurements*

<table>
<thead>
<tr>
<th>Authors</th>
<th>Target substance</th>
<th>Sample</th>
<th>Craving measure</th>
<th>When craving measured</th>
<th>Intervention</th>
<th>Outcome</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ahmadi et al. (2009)</td>
<td>Cocaine</td>
<td>164 cocaine and alcohol dependent outpatients</td>
<td>100mm scale for strength of cocaine and alcohol craving over past week</td>
<td>Treatment entry, after detoxification</td>
<td>12 weeks of naltrexone</td>
<td>4 weeks of sustained abstinence during 12 weeks of treatment</td>
<td>Craving was not a predictor</td>
</tr>
<tr>
<td>Alterman et al. (2000)</td>
<td>Cocaine</td>
<td>160 cocaine dependent inpatients</td>
<td>Five point analog scale of current cocaine craving</td>
<td>At application for treatment</td>
<td>4 weeks inpatient residential treatment</td>
<td>Relapse 6 months post-discharge</td>
<td>Craving was not a predictor</td>
</tr>
<tr>
<td>Bottlender &amp; Soyka (2004)</td>
<td>Alcohol</td>
<td>103 alcohol dependent outpatients</td>
<td>Obsessive Compulsive Drinking Scale</td>
<td>Prior to treatment, after abstinence ranging from 1-8 months</td>
<td>80-120 therapy sessions over 9-12 months</td>
<td>Relapse during 9-12 months of treatment</td>
<td>Pre-treatment OCDS scores were higher in people who relapsed during treatment</td>
</tr>
<tr>
<td>Crits-Christoph et al. (2007)</td>
<td>Cocaine</td>
<td>487 cocaine dependent outpatients</td>
<td>3-item craving cocaine scale rating urge 0-9</td>
<td>At intake</td>
<td>6 months psychosocial therapy</td>
<td>Sustained abstinence during 6 months of treatment</td>
<td>Greater craving was predictive of fewer months of consecutive abstinence</td>
</tr>
</tbody>
</table>
Table 4.1 Continued

*Studies predicting post-treatment substance use outcomes from pre-treatment craving measurements*

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<tbody>
<tr>
<td>Fidler et al. (2011)</td>
<td>Nicotine</td>
<td>2593 smokers</td>
<td>2 items measuring frequency and strength of urge in last 24 hours</td>
<td>Baseline</td>
<td>Nil. Quit attempts were self-initiated</td>
<td>Relapse following a quit attempt 6 months post-baseline</td>
<td>Strength of urges was the strongest predictor of relapse following a quit attempt</td>
</tr>
<tr>
<td>Garbutt et al. (2009)</td>
<td>Alcohol</td>
<td>40 alcohol dependent outpatients</td>
<td>Penn Alcohol Craving Scale</td>
<td>At least 3 days before treatment</td>
<td>12 weeks of naltrexone</td>
<td>Percent heavy drinking days during 12 weeks of treatment</td>
<td>Craving predicted % abstinent days, though effect was moderated by sweet liking phenotype</td>
</tr>
<tr>
<td>Hillhouse et al. (2007)</td>
<td>Methamphetamine</td>
<td>420 methamphetamine dependent outpatients</td>
<td>Craving Frequency, Intensity and Duration Estimate</td>
<td>1 week before treatment entry</td>
<td>16 weeks of psychosocial therapy</td>
<td>Relapse status 6 and 12 months post-treatment</td>
<td>Craving was not a predictor</td>
</tr>
</tbody>
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Table 4.1 Continued

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<tr>
<td>Kampman et al. (2004)</td>
<td>Alcohol and cocaine</td>
<td>84 alcohol and cocaine dependent outpatients</td>
<td>100mm scale for strength of cocaine and alcohol craving over past week</td>
<td>Baseline – unclear if before detoxification</td>
<td>30 days naltrexone following detoxification</td>
<td>At least 5 days consecutive abstinence from both alcohol and cocaine during 30 days of naltrexone treatment</td>
<td>Craving did not predict outcomes with other predictors in the model</td>
</tr>
<tr>
<td>Kiefer et al. (2005)</td>
<td>Alcohol</td>
<td>160 alcohol dependent inpatients</td>
<td>Obsessive Compulsive Drinking Scale</td>
<td>12-15 days after start of detox but prior to pharmacological intervention</td>
<td>12 weeks of acamprosate and/or naltrexone</td>
<td>Time to first drink during 12 weeks of treatment</td>
<td>Craving was not a predictor</td>
</tr>
<tr>
<td>Killen et al. (1999)</td>
<td>Nicotine</td>
<td>408 smokers</td>
<td>Average of 2 items measuring frequency and strength of urges</td>
<td>Baseline</td>
<td>Nicotine patch plus self-help treatment manual over 6 weeks</td>
<td>Abstinence (no smoking for 7 days prior to assessment) at 2, 6 and 12 months</td>
<td>Craving was predictive of relapse through the 12 months of follow-up</td>
</tr>
</tbody>
</table>
Table 4.1 Continued

*Studies predicting post-treatment substance use outcomes from pre-treatment craving measurements*

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<tr>
<td>Kranzler et al. (1999)</td>
<td>Alcohol</td>
<td>127 alcohol dependent outpatients</td>
<td>Obsessive Compulsive Drinking Scale</td>
<td>Prior to treatment, after minimum 3 days abstinence</td>
<td>12-week placebo controlled trial of nefazadone and naltrexone</td>
<td>Relapse 3 months after treatment</td>
<td>Neither subscale of the OCDS predicted drinking during follow-up</td>
</tr>
<tr>
<td>Paliwal et al. (2008)</td>
<td>Cocaine</td>
<td>132 cocaine dependent inpatients</td>
<td>Cocaine Craving Questionnaire – Now and Brief versions</td>
<td>At treatment entry</td>
<td>Average of 3 weeks inpatient treatment</td>
<td>Time to relapse, frequency and amount of use, 90 days post-discharge</td>
<td>Craving predicted time to relapse but not frequency or amount of use</td>
</tr>
<tr>
<td>Ray et al. (2006)</td>
<td>Alcohol</td>
<td>72 alcohol dependent outpatients</td>
<td>Obsessive Compulsive Drinking Scale</td>
<td>Prior to first treatment session</td>
<td>2 sessions of motivation-based psychosocial intervention plus 12 weeks of olanzapine</td>
<td>Percent days abstinent during 12 weeks post-treatment</td>
<td>Higher craving on OCDS was associated with lower % days abstinent during follow-up</td>
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<tr>
<td>Roberts et al. (1999)</td>
<td>Alcohol</td>
<td>132 alcohol dependent outpatients</td>
<td>Obsessive Compulsive Drinking Scale</td>
<td>Baseline</td>
<td>12 sessions of CBT plus naltrexone</td>
<td>Time to first heavy drinking up to 26 weeks after baseline</td>
<td>Resistance/ control impairment subscale greatest predictor of time to first heavy drinking, but this subscale contains consumption items</td>
</tr>
<tr>
<td>Rohsenow et al. (2007)</td>
<td>Cocaine</td>
<td>163 cocaine dependent residential inpatients</td>
<td>Cocaine-Related Assessment of Coping Skills (cue-elicited measure)</td>
<td>In the first week of residential treatment</td>
<td>8-85 days of residential rehabilitation</td>
<td>Days of cocaine use and $ spent on cocaine 0-3 months and 4-6 months post-discharge</td>
<td>Craving predicted amount spent on cocaine in the 3 months following discharge</td>
</tr>
<tr>
<td>Rohsenow et al. (1994)</td>
<td>Alcohol</td>
<td>45 alcohol dependent inpatients</td>
<td>Cue-reactivity (salivation) and self-report of urge strength from 1-10</td>
<td>After admission, during withdrawal</td>
<td>Inpatient detoxification</td>
<td>Return to drinking in 3 months post-treatment</td>
<td>Cue-reactivity was predictive of fewer abstinent days during follow-up</td>
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<tr>
<td>Toll et al. (2007)</td>
<td>Nicotine</td>
<td>207 smokers</td>
<td>Questionnaire on Smoking Urges-Brief</td>
<td>In week before quit, before and after first cigarette</td>
<td>7 weeks of bupropion (1 week before quit and 6 weeks after)</td>
<td>Relapse at end of treatment and 3 months</td>
<td>Larger decreases in craving following first cigarette of day predicted relapse at end of treatment and at 3 months</td>
</tr>
<tr>
<td>Tunis et al. (1994)</td>
<td>Cocaine</td>
<td>57 cocaine dependent inpatients</td>
<td>Intrusive cocaine thoughts using Impact of Event Scale and craving in last week using 10-point scale</td>
<td>1-3 days after entry to the inpatient unit</td>
<td>8 weeks of desipramine hydrochloride</td>
<td>Use of cocaine 1, 8, 12 and 26 weeks post-discharge</td>
<td>Baseline intrusive cocaine thoughts predicted use of cocaine 1-week post-discharge for placebo group only</td>
</tr>
<tr>
<td>Walton et al. (2003)</td>
<td>Alcohol</td>
<td>241 alcohol and other drug user inpatients and outpatients</td>
<td>Ratings of frequency and strength of cravings in 8 contexts</td>
<td>Month before treatment</td>
<td>3-7 day inpatient detox or outpatient treatment of varying intensity</td>
<td>Graded relapse 2 years post-treatment</td>
<td>Craving was not a predictor</td>
</tr>
</tbody>
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<tr>
<td>Weiss et al. (1995)</td>
<td>Cocaine</td>
<td>73 cocaine dependent inpatients</td>
<td>5 items rating strength and frequency of urge from 0-9</td>
<td>Daily from day 1 of admission</td>
<td>6-30 days of inpatient rehabilitation</td>
<td>Graded relapse 3 months post-discharge</td>
<td>Craving was not a predictor</td>
</tr>
<tr>
<td>Zhou et al. (2009)</td>
<td>Nicotine</td>
<td>2431 smokers recruited from Internet panel who indicated intention to stop smoking within 3 months</td>
<td>Desire or craving to smoke taken from Minnesota Nicotine Withdrawal Scale</td>
<td>Baseline</td>
<td>Nil. Quit attempts were self-initiated and aids could be used if desired e.g., medications, support programs</td>
<td>Relapse in a 3-month period where a quit attempt was made</td>
<td>Craving was not a predictor</td>
</tr>
</tbody>
</table>
Craving as a predictor

Some empirical support for the prediction of treatment outcomes from pre-treatment craving has been found for alcohol (Bottlender & Soyka, 2004; Garbutt et al., 2009; Ray, Hutchison, & Bryan, 2006; Roberts, Anton, Latham, & Moak, 1999; Rohsenow et al., 1994), smoking (Fidler, Shahab, & West, 2011; Killen, Fortmann, Davis, Strausberg, & Varady, 1999; Toll, Schepis, O'Malley, McKee, & Krishnan-Sarin, 2007) and cocaine (Crits-Christoph et al., 2007; Paliwal, Hyman, & Sinha, 2008; Rohsenow, Martin, Eaton, & Monti, 2007; Tunis, Delucchi, & Hall, 1994). Yet there are also many studies that have not found craving to perform significantly in prediction models, for alcohol (Ahmadi et al., 2009; Kampman et al., 2004; Kiefer et al., 2005; Kranzler, Mulgrew, Modesto-Lowe, & Burleson, 1999; Walton, Blow, Bingham, & Chermack, 2003), smoking (Zhou et al., 2009), cocaine (Ahmadi et al., 2009; Alterman et al., 2000; Kampman et al., 2004; Weiss, Griffin, & Hufford, 1995) and methamphetamine (Hillhouse, Marinelli-Casey, Gonzales, Ang, & Rawson, 2007).

However, these studies all used various methods to measure craving, assessed different outcomes at different time points, and have methodological weaknesses, making it difficult to draw definitive conclusions about pre-treatment craving as a predictor of substance treatment outcomes.

4.1 High variability between studies

As an example of the high variability between studies, Rohsenow et al. (1994) and Ray et al. (2006) both measured cue-elicited craving at the start of treatment, and both measured drinking outcomes at 3 months, but they studied different populations, used different methods to assess craving and found different, even contrasting, results. Rohsenow et al. studied an inpatient sample of male alcoholics, and assessed them for craving within a week of their last drink, but after they had ceased alcohol use. Ray et
al. studied an outpatient community sample and assessed craving before alcohol reduction had commenced. Where Rohsenow et al. measured craving in response to alcohol cues using both a physiological (salivation) and self-report measure (single item on a 10-point Likert scale), Ray et al. measured cue-elicited craving using only a self-report measure; the Alcohol Urge Questionnaire (Bohn, Krahn, & Staehler, 1995), and this was measured in the second week of treatment in the context of a cue-exposure treatment session. Ray et al. also obtained a pre-treatment measure of craving using the Obsessive Compulsive Drinking Scale (OCDS; Anton, Moak, & Latham, 1995) at study intake.

Rohsenow et al. (1994) found that the physiological craving measure, salivation, was predictive of a higher frequency of drinking days at follow-up, but that the self-reported craving measure was not. This contrasts with the results of Ray et al. (2006), who did find that the self-reported craving measure taken after the cue-exposure session was predictive of total number of drinks being consumed at 3-month follow-up. Ray et al. also found that the pre-treatment measure of craving performed slightly better than the cue-elicited measure, in that it predicted both the total number of drinks being consumed at follow-up as well as the percentage of days abstinent.

These two studies were chosen to exemplify the myriad ways in which the study of craving’s performance as an outcome predictor can vary, and how those variations can lead to different results. Similar variation is observed among other studies, particularly in relation to how craving is measured and how outcomes are defined.
4.1.1 Measurement limitations

4.1.1.1 Measures used

Different theoretical conceptualisations of the concept of craving have led to development of a variety of methods that attempt to measure the state or condition of ‘craving’, leading to substantial variability in how craving is measured across studies. Rosenberg (2009) provides a comprehensive overview of the most commonly utilised clinical and laboratory craving assessments. In its simplest form, craving is assessed using a single item, rated on a Likert or visual analog scale. These items may ask how much the substance is wanted or desired at a particular point in time or how often it was desired. Assessments of physiological reaction to substance cues are popular in laboratory based studies and involve capturing evidence of autonomic arousal such as salivation, startle eye blink, heart rate, sweat gland activity and skin temperature. These assessments are based on the premise that such arousal in response to substance cues is analogous with craving. The most commonly utilised and most prolific of all craving measures are self-report questionnaires. Rosenberg reviewed 24 such scales in his review of craving assessment measures, highlighting the substantial breadth and diversity in this area of craving assessment. It is perhaps therefore not surprising that so much variability in craving measurement is observed in prediction studies.

Single-item measures are popular due to their quick and easy administration and seemingly high face validity. However, single-item assessments have been widely criticised for their lack of reliability, high susceptibility to measurement error, wide variability in anchors and end-points and for failing to capture the multi-dimensional nature of craving (Anton & Drobes, 1998; Sayette et al., 2000; Sinha & O'Malley, 1999; Tiffany, Carter, & Singleton, 2000). Single-item measures remain popular despite these limitations, although they are generally found to perform poorly as predictors. For
example, Ahmadi et al. (2009) and Kampman et al. (2004) both used a single-item 100mm visual analog scale, adapted from the Minnesota Cocaine Craving Scale (Halikas, Kuhn, Crosby, & Carlson, 1991) to measure alcohol and cocaine craving in patients with comorbid alcohol and cocaine dependence. They both found this measure of craving to not be a significant predictor of sustained abstinence following treatment for either alcohol or cocaine. Alterman et al. (2000) also failed to predict cocaine treatment outcomes using a single-item measure of current craving at treatment intake. Fidler et al. (2011) did manage to predict smoking relapse following a quit attempt from a single-item measuring strength of smoking urge, but their focal period was on the previous 24 hours. The importance of the focal point of measurement is discussed further on.

Averaging of scores over a number of assessment items is considered a better alternative to single-item assessment as this improves reliability and reduces measurement error (Sayette et al., 2000; Tiffany et al., 2000), which should improve statistical ability to detect associations with substance use. Crits-Christoph et al. (2007) and Killen et al. (1999) averaged scores over 3 and 2 items respectively and both found these averaged scores to be predictive of abstinence from cocaine and cigarettes (respectively) during follow-up.

Multiple item measures are generally favoured as unique error implicit in each item is cancelled out by the other items (Sayette et al., 2000) and multiple items allow for assessment of the various dimensions and experiences of craving (Anton & Drobes, 1998; Tiffany et al., 2000). This has led to development of a wide range of standardised self-report craving instruments (Rosenberg, 2009). Despite the advantages of multi-item self-report questionnaires, association with substance use, in particular outcomes following treatment, remains inconsistent. Out of 12 studies reviewed that used a self-
Craving as a predictor

report measure to assess craving, seven (58%) found it to be a significant predictor of
treatment outcome (Bottlender & Soyka, 2004; Garbutt et al., 2009; Paliwal et al., 2008;
Ray et al., 2006; Roberts et al., 1999; Toll et al., 2007; Tunis et al., 1994), while five
(42%) did not (Hillhouse et al., 2007; Kiefer et al., 2005; Kranzler et al., 1999; Weiss et
al., 1995; Zhou et al., 2009).

There is substantial variability in measures used among studies, and this
impedes direct comparison of results. For example, in studies examining predictors of
relapse to cocaine use, Paliwal et al. (2008) measured craving using the brief 10-item
version of the Cocaine Craving Questionnaire (Sussner et al., 2006) and found that high
craving was associated with shorter time to relapse, but was not predictive of either
amount or frequency of use. Weiss et al. (1995) used a five-item scale which they
developed and validated and found scores on this scale were not predictive of relapse
status at 3 months. Even among studies using the same measure, inconsistent results are
observed. Five of the studies examining alcohol outcomes utilised the Obsessive
Compulsive Drinking Scale to measure craving. Of these, three (60%) found it to be a
predictor of treatment outcome (Bottlender & Soyka, 2004; Ray et al., 2006; Roberts et
al., 1999) while two (40%) did not (Kiefer et al., 2005; Kranzler et al., 1999). There was
substantial variability between the studies in when the assessment was administered and
what outcome was being assessed. However, two of the studies that were most similar
on these factors both found discrepant results. Bottlender and Soyka (2004) and Kiefer
et al. (2005) both measured craving after their participants were already abstaining from
alcohol and both measured relapse to drinking during treatment as their outcome. Where
Bottlender and Soyka found that craving was predictive of relapse during treatment,
Kiefer et al. did not. The treatment period was much longer in the study of Bottlender
and Soyka, however, extending over 9 to 12 months, while Kiefer et al.’s intervention ran for 12 weeks.

While considered better measures of craving than single items, multi-item self-report measures are not without limitations. Some scales confound measurement of craving by inclusion of items that are not focused on craving but measure related phenomena such as consumption, intentions, plans, self-efficacy or arousal (Kavanagh et al., 2013). Additionally, all self-report measures of craving, whether single item or multi-item, are subject to potential bias from differential interpretation of items and anchors and from social desirability (Sayette et al., 2000). In multi-item measures, earlier items can also influence responses on later items. Of particular concern in self-report measurement of craving is the bias inherent in retrospective recall. When asked to judge frequency of events, people have been demonstrated to rely on availability of incidents of the event in recall (Tversky & Kahneman, 1973). Availability is affected by a number of factors, and this introduces bias into the frequency judgment (Tversky & Kahneman, 1973). Highly salient experiences are therefore likely to be more readily recalled and will contribute greater influence in averaging over a time period (Shiffman, 2000).

Rosenberg (2009) raises the very good point that with measures requiring averaging it is difficult to really know what timeframe is actually being captured, and whether it is the person’s general sense of their craving or an indication of peak craving at its most intense. Additionally, averaging craving over a timeframe does not reflect the variability and dynamics inherent in craving patterns (Ferguson & Shiffman, 2009; Sinha & O’Malley, 1999), and fails to reflect the potentially important distinction between background craving and episodes of acute, intense craving. The longer the period of time over which craving is averaged, the worse this problem.
Craving as a predictor

Distinctions are made between what is termed tonic and phasic craving. Tonic craving is considered a stable, or only slightly varying, background level of craving (Munafò & Hitsman, 2010; Shiffman, 2000). Ferguson and Shiffman (2009) define it as “slowly varying phenomena that are experienced as relatively steady, tonic states over the course of days or at least hours, fluctuating slowly.” (p. 253). Overlaid against this background craving are phasic cravings, which Ferguson and Shiffman describe as “experienced as acute episodes of intense craving overlaid on background craving. These episodes occur in and are triggered by particular situational stimuli…” (p. 236). Munafò and Hitsman (2010) distinguish between tonic craving as being triggered by withdrawal states and phasic craving as being triggered by situational and environmental cues, and argue that the difference between these states and what they represent is important for prediction of substance use. Measures detecting intensity of acute phasic cravings, such as cue-elicited craving (Ferguson & Shiffman, 2009), are argued to hold more value in prediction of later substance use than measures of more stable, background craving (Ferguson & Shiffman, 2009; Munafò & Hitsman, 2010; Witkiewitz & Marlatt, 2004), although this is not necessarily supported by the research.

Perkins (2009) conducted a review of the field of cue-elicited craving and nicotine dependence and observed a lack of evidence supporting the performance of self-reported craving in response to smoking cues in predicting relapse to smoking. Cofka-Woerpel et al. (2011) found that self-reported background urge, collected by ecological momentary assessment, was actually a better predictor of smoking relapse during the first week of a quit attempt than phasic urge. How cue-elicited craving is measured seems to be important, however, with Heinz, Beck, Grusser, Grace, and Wrase (2008) arguing that a non-self-report measure, such as cue-elicited brain activation, is a better predictor of outcomes than self-reported craving. Physiological
measures such as heart rate, perspiration and temperature are other non-self-report alternatives, but can be criticised on the grounds that these autonomic reactions can be triggered by other states aside from craving, such as fear and anxiety (Lang, Davis, & Öhman, 2000). These cue-elicited responses may therefore be detecting other reactions aside from craving (Shiffman, 2000).

Of the studies reviewed here, Rohsenow et al. (1994) found that a physiological craving measure, salivation, was predictive of a higher frequency of drinking days at follow-up, but that a self-reported measure was not. This contrasts with the results of Ray et al. (2006) who did find that the self-reported measure taken after the cue-exposure session was predictive of total number of drinks being consumed at 3-month follow-up. Rohsenow et al. (2007) measured cue-reactivity in a different way using the Cocaine-Related Assessment of Coping Skills (Rohsenow et al., 2004). This assessment involved presentation of audiotapes conveying high risk situations for cocaine use, after which participants were asked to rate how strong their urge to use cocaine would be if they were not in hospital. This measure was found to be a significant predictor of the amount of money spent on cocaine in the first 3 months following treatment, but not during the subsequent 3 months (4-6 months post-treatment). The finding that a context-relevant measure of cue-related craving was predictive of later substance use highlights a point raised by Sinha and O'Malley (1999) that craving measures are likely to be more sensitive if taken at times and in contexts that are relevant to those when greater craving might be expected. The timing of administration of assessments is therefore an important consideration.

4.1.1.2 Timing of administration

Just as there is great variability in choice of assessments used to measure craving, there is also considerable variability in when those assessments are
Craving as a predictor

administered, and this could have substantial impact on the results obtained. For example, Kranzler et al. (1999) and Ray et al. (2006) both measured craving using the Obsessive Compulsive Drinking Scale, both administered pharmacotherapy interventions, and both examined alcohol use outcomes 12 weeks after commencing treatment. Kranzler et al. measured craving after a minimum of 3 days of alcohol abstinence and found craving was not a predictor of relapse at 12 weeks, while Ray et al. measured craving prior to the first treatment session while alcohol was still being consumed and found that craving was a significant predictor of percentage of abstinent days during the 12 weeks of treatment.

Differences in timing of administration are often associated with differences in populations studied. When patients of inpatient facilities are recruited, baseline assessment for the research typically occurs after some degree of stabilisation of the patient, which may or may not include pharmacotherapy. Even within this timeframe, there can be substantial variation in when assessment occurs. Bottlender and Soyka’s (2004) participants had been abstinent from alcohol for a minimum of 30 days before they completed the craving assessment, with some participants having been alcohol free for up to 8 months. Walton et al. (2003) measured craving in the first month of inpatient substance abuse treatment, but admit that assessment occurred anywhere within that timeframe, with it occurring earlier in the month for some and later in the month for others. Rohsenow et al. (1994; 2007) attempted to minimise this problem by ensuring assessment occurred within the first week of admission and within a week of last substance use, but participants had still already commenced abstinence at the time of assessment.

Timing of administration has important implications for the context of the craving measurement, which may be important for its applicability to the outcome being
measured. As Sinha and O'Malley (1999) point out, the state qualities of craving are important, and the circumstances under which it is measured will influence its sensitivity as a predictor. Craving measurements obtained in an inpatient context where substance use is unavailable, cues are minimal, and medications are commonly administered to assist detoxification, may have limited contextual relevance to the post-treatment environment where the person is once again exposed to substance cues and is likely to experience episodic spikes in craving. That is not to say that craving measured after abstinence or reduction has commenced is not important or useful to consider in the prediction of outcomes following treatment. In fact, from the eight studies reviewed which measured craving after abstinence from alcohol or cocaine had commenced, four (50%) found craving to be a significant predictor of treatment outcomes (Bottlender & Soyka, 2004; Rohsenow et al., 2007; Rohsenow et al., 1994; Tunis et al., 1994), suggesting craving measured in the context of treatment-supported withdrawal does hold significance for vulnerability to relapse. On the other hand, the other four studies did not find craving measured post-abstinence to be predictive of outcomes (Ahmadi et al., 2009; Kiefer et al., 2005; Kranzler et al., 1999; Weiss et al., 1995). The picture is no clearer looking at studies that measured craving prior to commencement of alcohol or cocaine abstinence, with five out of eight studies (63%) finding craving measured before reduction was predictive of outcomes (Crits-Christoph et al., 2007; Garbutt et al., 2009; Paliwal et al., 2008; Ray et al., 2006; Roberts et al., 1999) and three finding it was not (Alterman et al., 2000; Hillhouse et al., 2007; Walton et al., 2003). Timing of assessment however, is not the only source of variability between craving studies.
Craving as a predictor

4.1.2 Outcome variability

4.1.2.1 Outcomes defined

In addition to differences in how and when craving is measured, studies also differ in the way in which outcomes are measured and defined. Not all studies examine the same outcome variables when investigating craving’s predictive performance. Some study relapse as an outcome simply in terms of whether sustained abstinence had been achieved or not at the time the follow-up was assessed. Any consumption of the substance is considered a violation and is coded as a relapse (e.g., Crits-Christoph et al., 2007; Fidler et al., 2011; Toll et al., 2007). Of the three studies that assessed sustained abstinence as the outcome, all three (100%) found that craving was a significant predictor, one measuring cocaine relapse (Crits-Christoph et al., 2007) and two measuring smoking relapse (Fidler et al., 2011; Toll et al., 2007).

Other studies that assess relapse as the outcome place thresholds on a quantity or frequency of use that must be reached for the person to be considered relapsed. For example, Weiss et al. (1995), in their study of cocaine users, defined relapse as having used cocaine on average more often than monthly in the 3 months following treatment. Kranzler et al. (1999), in their study of alcohol treatment outcomes, defined relapse as having 5 or more drinks for 2 consecutive days. Kiefer et al. (2005) set the same quantity as Kranzler et al., but required a greater frequency of drinking to be considered a relapse, requiring consumption of 5 or more drinks on at least 5 days in a single week. Kampman et al. (2004) measured outcomes following outpatient detoxification for alcohol and cocaine, and measured achievement of 5 consecutive days of abstinence from both as the primary outcome of interest. Killen et al. (1999) required abstinence from smoking for 7 days prior to assessment to be coded as abstinent at that assessment point. Of the 11 studies that measured outcomes according to degrees of relapse...
(Ahmadi et al., 2009; Alterman et al., 2000; Bottlender & Soyka, 2004; Hillhouse et al., 2007; Kampman et al., 2004; Kiefer et al., 2005; Killen et al., 1999; Kranzler et al., 1999; Walton et al., 2003; Weiss et al., 1995; Zhou et al., 2009), only two (18%) found craving to be a significant predictor (Bottlender & Soyka, 2004; Killen et al., 1999). This might suggest that craving is better at predicting whether any breach of abstinence will occur than it is at predicting whether a threshold of use will be reached. Other studies examine time to lapse or relapse as the outcome. This has also been associated with inconsistent results, with three of five studies (60%) finding craving did not predict this outcome (Garbutt et al., 2009; Kiefer et al., 2005; Rohsenow et al., 1994) and two (40%) finding it did (Paliwal et al., 2008; Roberts et al., 1999).

Prediction of indices of consumption measured on continuous scales (e.g., percentage of days abstinence throughout follow-up, number of drinks consumed throughout follow-up, number of days of heavy drinking) seems a little more promising. Four out of six studies (67%) that investigated outcomes of consumption found craving to be a significant predictor, though most of those studies investigated multiple indices of consumption and found that craving was only predictive of some and not others. Where Garbutt et al. (2009) found craving to not be predictive of time to alcohol relapse, they did find it to be predictive of percent abstinent days at the end of treatment. Ray et al. (2006) similarly found that craving was a significant predictor of percent abstinent days from alcohol following treatment, and also found it was a predictor of the number of drinks consumed. Like Garbutt et al., Rohsenow et al. (1994) found craving did not predict days to first drink, but that it did predict a higher frequency of heavy drinking. Rohsenow et al. (2007) found cocaine craving to be predictive of amount of money spent on cocaine at follow-up, but not of number of days of cocaine use. Contrasting with studies that found craving did not predict time to relapse but did
predict continuous outcomes, Paliwal et al. (2008) found the opposite. In their study, craving was predictive of time to relapse but was not predictive of frequency or quantity of use. Similarly, Walton et al. (2003) found that craving was not a significant predictor of number of drinking days post-treatment or of drinks per drinking day. So while four out of the six studies found that craving did predict continuous outcome measures, this was not consistent across studies and the particular outcomes that were predicted varied from study to study. The substantial variability in the outcomes investigated makes it difficult to draw definitive conclusions about the type of outcomes pre-treatment craving seems best suited to predicting. Couple this with the variability in measures used and timing of administration, and the picture becomes even cloudier. Complicating this further is the need to also consider when the outcomes being investigated were measured.

### 4.1.2.2 Timing of outcome measurement

When studying prediction of treatment outcomes there are multiple time points that might be of interest. Substance use status immediately upon completing the treatment is of interest in assessing whether the participant responded successfully to the treatment. Substance use status in the long term is of interest in assessing whether the participant was successful in maintaining treatment gains. Craving has been investigated as a predictor of both, with no consistent picture of a temporal effect emerging.

Of the studies that investigated treatment outcomes either during treatment or immediately post-treatment, four of seven (57%) found craving to be a significant predictor (Bottlender & Soyka, 2004; Crits-Christoph et al., 2007; Garbutt et al., 2009; Toll et al., 2007), while three (43%) did not (Ahmadi et al., 2009; Kampman et al., 2004; Kiefer et al., 2005). There was substantial variability however, in the timeframes of these assessments. For example, Kampman et al.’s (2004) treatment lasted only 30
days, while Bottlender and Soyka’s (2004) extended over 9 to 12 months. The picture for longer term outcomes is not any clearer.

Of studies that examined prediction of outcomes 3 months post-treatment, six out of seven (86%) found baseline craving to be predictive of relapse or of the frequency or amount of use of alcohol (Ray et al., 2006; Roberts et al., 1999; Rohsenow et al., 1994), cocaine (Paliwal et al., 2008; Rohsenow et al., 2007) and cigarettes (Toll et al., 2007). Weiss et al. (1995) did not find craving to be a predictor of 3-month post-treatment relapse to cocaine use. Evidence for prediction of longer term outcomes is not as strong, with three out of four studies (75%) finding baseline craving was not a predictor of substance use relapse 6 months post-treatment (Alterman et al., 2000; Hillhouse et al., 2007; Rohsenow et al., 2007) and only one (25%) finding it was (Killen et al., 1999). Walton et al. (2003) studied relapse to alcohol use 2 years following completion of an inpatient or outpatient treatment program, but did not find that craving measured prior to treatment was predictive of relapse after 2 years.

It would seem that craving performs better at predicting shorter term outcomes than outcomes over more extended periods. This is perhaps not surprising given the issues discussed above relating to the nature of craving and the limitations of the ways in which it is measured. To predict future substance use from craving measured at a much earlier time point assumes a stability in craving that would make it relate evenly to substance use across time. This is not how craving tends to behave, with ecological momentary assessments showing cravings can fluctuate wildly throughout a given day (Piper et al., 2011; Shiffman et al., 1997) and be highly reactive to external and internal cues (Shiffman, Paty, Gnys, Kassel, & Hickcox, 1996). Therefore, the means by which craving is measured, and the timing of its assessment, are highly important factors to consider when endeavouring to predict long-term substance use from earlier craving.
Craving as a predictor

measures. There are also a range of methodological issues to consider, some of which may be contributing to the discrepant results observed between studies here, and which make amalgamation of results across studies difficult.

4.2 Methodological issues

One of the more important methodological considerations is whether other baseline variables, such as baseline consumption, were controlled for in the prediction model. Craving has already been established as being strongly associated with proximal substance use, and prediction studies need to rule out this association as being the source of craving’s relationship with outcomes. If craving is associated with post-treatment outcomes because people who consume higher quantities of substances at baseline are also more likely to consume more at follow-up, and tend to also have higher baseline craving, then a substantial portion of the predictive variance is likely due to baseline consumption rather than craving. It is therefore important to control for baseline consumption in the prediction models to disentangle the effects of baseline consumption from those of craving.

Kampman et al. (2004) illustrate this point in their finding that cocaine craving was initially a significant predictor of abstinence success following treatment, but that this effect disappeared when craving was entered into a stepwise regression with the other significant predictors, leaving only days of cocaine use at baseline and cocaine withdrawal symptom severity as significant in the model. Most studies are explicit about controlling for other predictors and, having done this, some find that craving is still significant in the prediction model (Crits-Christoph et al., 2007; Paliwal et al., 2008; Ray et al., 2006; Rohsenow et al., 2007; Rohsenow et al., 1994), while others find that it is not (Ahmadi et al., 2009; Alterman et al., 2000; Hillhouse et al., 2007; Kranzler
et al., 1999; Walton et al., 2003; Zhou et al., 2009). Other studies do not control for baseline variables or do not explicitly state if they do (Kiefer et al., 2005; Weiss et al., 1995).

Also of concern is when craving is found to be a significant predictor, but no further analysis controlling for other predictors is reported. Bottlender and Soyka (2004) did not mention if baseline consumption and/or severity were controlled for in their prediction analyses. This casts doubt on the results of this study and means the results must be interpreted with caution. This study also failed to specify if the consumption items of the OCDS were removed for the analysis. The OCDS contains two items measuring frequency and quantity of alcohol use, and these items are therefore likely to confound with other baseline measurements and with the fact that baseline consumption tends to be a strong predictor of post-treatment outcomes, as already discussed. In fact, in Kranzler et al.’s (1999) study of the ability of the OCDS to predict outcomes following treatment, only the consumption factor, made up of these two frequency and quantity items, was found to be predictive. Similarly, in Roberts et al.’s (1999) study, the only factor that was related to outcomes was one that contained these consumption items. Inclusion of these items confounds baseline alcohol consumption with craving and likely inflates the predictive power of the scale.

Another major methodological issue with studies to date is the very small sample sizes of some studies, limiting generalisability of results and suggesting possible underpowering of regression analyses. For example, Garbutt et al. (2009) had a sample size of only 40 participants, and Rohsenow et al. (1994) had only 45, and they were also all male, further limiting the generalisability of their results.
4.3 Implications of variability and methodological issues

All of these studies provide important information about the relationship between craving and substance use following treatment, but with so much variability in the methodological approaches of each study, it is difficult to derive decisive conclusions about the value of craving as a predictor of outcomes. As the literature on craving’s performance as a predictor stands, it neither supports nor dismisses craving as being a strong or useful predictor in its own right, over and above the influence of other baseline variables. The divergent results could be due to the different populations studied, the various measures of craving used, the different points in time at which craving was measured, the nature of the outcomes that were studied or when the outcomes were measured. It is not expected that all studies should employ the same methodologies and study the same populations. This would hamper the field far more than it would help it, but clearly these differences need to be acknowledged and highlighted, particularly in relation to how they may affect the results and deliver different results from other studies.

It is also highly plausible that the divergent results observed in prediction studies could also be due to other variables not measured or reported, and there is a growing body of research evidence suggesting that many potential moderators and/or mediators could be influencing substance use outcomes or acting on the relationship between craving and outcomes. For example, Garbutt et al. (2009) examined whether craving measured prior to naltrexone treatment would predict drinking outcomes at 12 weeks. At first analysis, craving did not predict treatment outcome either in percent heavy drinking days or percent days abstinent. But when the group was separated according to sweet liking or sweet disliking phenotypes (reflecting a hedonic response to sweet taste which is believed to be related to activation of the opioid system), a highly significant
interaction effect between craving, sweet liking or disliking status and percent abstinent days emerged, such that for people with the sweet liking phenotype, craving was positively correlated with percent abstinent days, and for people with the sweet disliking phenotype it was negatively correlated. These results suggest a moderating effect of sweet liking status on the relationship between craving and treatment outcomes.

It may be the case that there are certain conditions under which craving is more influential, and therefore more predictive, than in others. This is a notion that has been raised by craving researchers before (Drummond, Litten, Lowman, & Hunt, 2000; Tiffany, 2000; Tracy, 1994), but continues to be insufficiently addressed in craving prediction research. One variable that is highly likely to be engaged in a relationship of influence with craving is negative affect. There is substantial evidence linking negative affect to elevated craving and to substance use. It is therefore important to investigate how negative affect and craving may work together to influence substance use outcomes.
CHAPTER 5    ROLE OF NEGATIVE AFFECT IN ADDICTION

It has long been recognised that negative affect has a relationship of mutual influence with substance use. Mood disorder prevalence is higher in substance using populations than in the general population, with 24.1% of Australians with a 12-month substance use disorder also reporting symptoms of an affective disorder (Teesson, Slade, & Mills, 2009). This is substantially higher than the 6.2% prevalence of 12-month affective disorders observed in the general Australian population (Slade, Johnston, Oakley Browne, Andrews, & Whiteford, 2009).

Negative affect is the driving force behind negative reinforcement theories, which postulate that addiction is driven by attempts to relieve negative affective states caused either by withdrawal from the substance or by the psychological sequelae of mental disorders or stressful life events. In cognitive theories it is one of the cues that trigger activation of automatic drug seeking behaviour (Tiffany, 1990). It is the backbone of Baker et al.’s (2004) affective model of drug motivation, which proposes that substance use becomes conditioned to negative affect over time, through the association of negative affect with withdrawal states, so that eventually the presence of negative affect alone, regardless of its source, will prompt drug use routines. According to elaborated intrusion theory (Kavanagh et al., 2005), negative affect is the predominant emotional reaction associated with urges, initiated by a sense of deprivation. The associations between negative affect, substance use and cravings have been well documented in both laboratory and clinical studies.
5.1 Relationship between negative affect and substance use

5.1.1 Evidence of proximal associations

Numerous studies have evinced proximal associations between acute negative affective states and subsequent substance use, including evidence that the relationship of influence works in both directions. A substantial proportion of this research has been done in the field of smoking. Perkins et al. (2008) found a positive association between negative affect and smoking reinforcement, with higher levels of negative mood in response to mood induction tasks being associated with shorter latency to smoke and a higher number of puffs, independent of actual nicotine intake or dose expectancy. The association was found to work in both directions, with exposure to smoking cues being found to attenuate negative mood, regardless of whether nicotine was expected or received, suggesting a conditioned response. Kassel et al. (2007) also found evidence of conditioned responding among adolescent smokers who experienced a decline in negative affect after they smoked either a low yield or high yield nicotine cigarette. The reduction in negative affect was observed to be greater for the high yield cigarette, but reductions were observed in both groups.

Fucito and Juliano (2009) also found that a negative mood induction led to increased cigarette consumption, but found the effect was strongest for people who also had high concurrent depressive symptomatology, measured using the Beck Depression Inventory II (Beck, Steer, & Brown, 1996). Those who scored in the clinical range on the depression inventory and were exposed to the sad mood induction had more puffs and smoked for a longer duration than did people who scored below the clinical cut off.

Perkins, Karelitz, Giedgowd, Conklin, and Sayette (2010) studied the effect of negative affect, induced through a variety of methods, on cigarette liking and ad libitum
Negative affect in addiction

smoking according to history of depression, levels of distress tolerance and anxiety sensitivity. They found that high anxiety sensitivity was associated with greater reported cigarette liking in response to a speech preparation task, suggesting that people with high anxiety sensitivity could be motivated by greater smoking reward and negative affect relief. However, the result did not generalise to the other affect induction tasks, which included exposure to negative mood slides, a computer challenge and a 12-hour abstinence from smoking. So the effect seemed limited to anxiety provoking situations rather than negative mood more generally. Lower level of distress tolerance, measured using the Distress Tolerance Scale (Simons & Gaher, 2005), was found to be associated with greater smoking reinforcement (measured by puff volume) after 12 hours of abstinence than for people with higher distress tolerance, but again, this result did not generalise to other negative affect inducing tasks. While Perkins, Karelitz, Giedgowd, et al. (2010) did find that a self-reported history of depression was associated with higher smoking reinforcement in general (across all mood induction sessions including neutral) compared to people without a depression history, they did not find that it was associated with enhanced reactivity to negative affect induction, with both groups responding similarly to the induction tasks.

Berkman, Dickenson, Falk, and Lieberman (2011) used text messages to collect ecological momentary assessments of smoking, craving and mood for 3 weeks starting from the day prior to a quit attempt. Up to eight measurements were collected each day from within 15 minutes of rising to 15 minutes before bed. The only significant prediction of smoking lapse by negative mood was from the mood measurement recorded immediately prior to the smoking episode. Mood records from the day before or at the same time as the smoking lapse were not predictive. Elevated negative mood was thus found to be an immediate antecedent to smoking lapse.
Similar proximal associations between negative affect and drinking have also been observed. Doumas (2011) collected 28 days of diary records from moderate to heavy drinkers who were not alcohol dependent and not engaged in alcohol treatment. Alcohol use, mood, perceived stress and drinking-related consequences were measured twice per day at 4pm and just prior to bed. Elevated ratings of depression and anxiety during the day were found to be predictive of greater alcohol consumption the same evening. Armeli, Tennen, Affleck, and Kranzler (2000) also found that daily negative affect associated with experience of negative life events was predictive of greater drinking on the same day.

Some studies find evidence of a gender difference in how negative affect relates to substance use. Swendsen et al. (2000) found that while an increase in subjective nervousness was associated with higher levels of later alcohol consumption, this effect was more pronounced for men than for women. Weinberger and McKee (2012) found that women displayed greater vulnerability to the effects of negative affect on subsequent smoking, with women displaying shorter latency to smoking than men following a negative mood induction, although there were no differences in number of cigarettes smoked. Detection of gender differences appears inconsistent, however, with Doumas (2011) finding no evidence of gender effects on the relationship between daily mood and later drinking. Furthermore, a meta-analytic review by Conner, Pinquart, and Gamble (2009) failed to find evidence of a moderating effect of gender on the relationship between alcohol or drug use and depressive symptoms. They suggest instead that findings of gender differences in depression amongst people with alcohol use disorder reflects a greater propensity for women to show higher levels of depression in general.
5.1.2 Relationship to substance treatment outcomes

There are numerous ways in which depression might increase relapse vulnerability, such as reducing self-efficacy (Rabois & Haaga, 2003; Ralston & Palfai, 2010), diminishing cognitive capacity to engage effective coping strategies (Drobes, Meier, & Tiffany, 1994; Levens, Muhtadie, & Gotlib, 2009) or activating expectancies oriented to alleviation of the negative affective state (Birch et al., 2004). Negative affect has been found in numerous studies to relate to substance use outcomes following treatment, demonstrating that its relationship with substance use goes beyond just proximal associations.

Kodl et al. (2008) investigated whether depression symptoms, measured using the Beck Depression Inventory II, would predict either alcohol or cigarette consumption following treatment for dependence on both. They found that clinically significant depressive symptoms at a given assessment point significantly predicted having relapsed to alcohol use at the next assessment point after controlling for other covariates and time trends. They estimated that depressed individuals were 12% more likely to be drinking at follow-up than non-depressed individuals. This prediction value was limited to alcohol outcomes however, with depression not showing significant predictive power for smoking abstinence after taking account of covariates and time. Gamble et al. (2010) found similar results when they examined whether pre-treatment depression symptoms, also measured using the Beck Depression Inventory II, would predict alcohol consumption up to 1 year following treatment in Project MATCH. Like Kodl et al. (2008), they found that after controlling for other covariates, pre-treatment depression scores were significantly associated with drinking outcomes in the year following treatment. Patients with greater depression pre-treatment drank more days and higher amounts in the year following treatment than patients with lower depression scores.
Witkiewitz and Villarroel (2009) also examined Project MATCH data to investigate the dynamics of the relationship between negative affect and alcohol lapses. Associative latent transition analysis was used to examine transitions in latent class of negative affect and drinking over the course of treatment and follow-up. Individuals were assigned to latent classes (categories representing unmeasured latent variables) at post-treatment, 3, 6, 9 and 12 months, based on observed measures of drinking and negative affect. Patterns of transition from one class to another were examined over the multiple time points. Results supported the presence of a dynamic relationship of mutual influence, with people classed as high in negative affect or transitioning to high negative affect over time, being more likely to be classed as heavy, frequent drinkers post-treatment, and people who were heavy and frequent drinkers over time, showing an increased tendency to high negative affect. The authors propose that negative affect and alcohol use may function within a feedback loop where increases in one generate increases in the other, which further generates increases in the other and so on.

Relationships between depression and smoking outcomes have also been reported, with Zhou et al. (2009) concluding that people who presented with elevated anxiety or depression prior to a smoking quit attempt were 30% more likely to return to smoking within 3 months of their quit attempt. Pre-treatment depression has also been reported as a significant predictor of 6- and 12-month post-treatment methamphetamine use (Hillhouse et al., 2007).

The results of Suter, Strik and Moggi’s (2011) study suggest the risk from depression is from active negative mood symptoms rather than from comorbid depression per se. They studied prediction of 1-year post-treatment alcohol relapse in residential treatment patients who had entered treatment with alcohol use disorder comorbid with either major depression or just elevated depressive symptoms, as well as
Negative affect in addiction

a group with no depression comorbidity. They found no difference in time to lapse in
the 12 months following discharge between patients with and without a comorbid major
depressive disorder, but did find that patients who had higher depression severity
measured using the German version of the Brief Symptom Inventory (Derogatis, 1993;
Franke, 2000) experienced a shorter time to lapse. Severe depression symptoms,
regardless of whether they were experienced in the context of a depressive disorder,
were associated with shorter time to relapse following alcohol treatment.

Other studies have failed to find an association between depression and
substance treatment outcomes. For example, Oslin et al. (2009) found contradicting
results to those of Witkiewitz and Villarroel (2009). They also used latent class analysis
to examine affective levels during the course of alcohol treatment, but did not find any
significant associations between positive or negative affect class during treatment and
any of the outcome measures. Crits-Christoph et al. (2007) examined whether a
composite measure of psychiatric severity was predictive of sustained abstinence during
treatment for cocaine dependence. The composite was composed of an average of
standardised scores on the Hamilton Rating Scale for Depression (Hamilton, 1960), the
Beck Anxiety Inventory (Beck, Epstein, & Brown, 1988), the Brief Symptom Inventory
(Derogatis, 1992), and the Addiction Severity Index ( McLellan, Luborsky, Woody, &
O'Brien, 1980). While it was not purely a measure of negative affect, it was composed
of scales that were or that included elements of negative affectivity. The composite
created from these scales was not found to be a significant predictor, although it should
be noted this was under the stringent condition of a Bonferroni correction. Tunis et al.
(1994) similarly found that pre-treatment negative mood was not related to cocaine use
post-discharge, as did Alterman et al. (2000). Numerous smoking studies have also
found no association between baseline negative affect and relapse to smoking following treatment (Killen et al., 1999; Toll et al., 2007)

While these studies do not represent a comprehensive review of the prediction of substance treatment outcomes from depression, they serve to illustrate that relationships of mutual influence do appear to exist between negative affect and substance use following treatment. Results have not been consistent across studies however, suggesting that other factors may also be involved. An interaction with craving may be one such dynamic, and there is substantial evidence linking negative affect with craving.

5.2 Relationship between negative affect and craving

A large number of studies have demonstrated a strong association between negative affect and substance craving, with negative affect acting as a cue to trigger craving, but also arising as a consequence of it. Despite substantial variability in methodologies, reasonably consistent results have been reported across studies. Significant associations have been observed in laboratory based cue-reactivity studies as well as in situ clinical studies, and using a variety of methods to assess affect and craving.

5.2.1 Negative affect and substance cue reactivity

Negative affect has consistently demonstrated a strong association with enhanced reactivity to alcohol related cues. Neuroimaging studies have shown differential brain activation in response to alcohol cues for people scoring high on depression scales compared to people who score low. Feldstein Ewing, Filbey, Chandler and Hutchison (2010) found greater activation in the insula, cingulate, ventral tegmental area, striatum and thalamus in response to alcohol cues in people scoring high on the
Negative affect in addiction

Beck Depression Inventory compared to people scoring low. These areas are linked to emotion, reward perception and motivation, behaviour planning and execution, and processing of sensory information. The authors suggest an interpretation that for people experiencing high levels of negative affect, alcohol cues activate areas of the brain associated with reward and pleasure seeking. It is proposed that the alcohol cues generate positive expectancies of the ameliorative effect alcohol will have on the negative mood.

Similarly, Gilman and Hommer (2008) found differential brain activation in alcoholics compared to controls in response to presentation of negative versus positive images, suggesting alcoholics have a higher sensitivity to negative stimuli. Of interest is the finding that this differential was attenuated by the concurrent presentation of alcohol cues, such that the reactivity to the negative images was not as great compared to positive images when alcohol related images were also presented. This could have been simply due to the effects of the alcohol cues being given cognitive processing priority, ameliorating the effect of the negative images, but this result might also mean that positive associations generated by the alcohol cues moderated the effect of the negative images.

Laboratory based studies measuring self-reported reactivity to cues show similar patterns of heightened urge in the presence of negative affective stimuli. Tiffany and Drobes (1990) presented smokers with imagery scripts that portrayed negative affect + smoking urge; positive affect + smoking urge; negative affect alone; positive affect alone; or neutral affect. After reading each script and being asked to spend some time imagining it, participants were asked to rate their maximum urge and craving during the trial. Self-reported cravings were strongest for the scripts that portrayed explicit smoking urges, regardless of the affective content, but of the affect only scripts, the
negative affect script induced significantly stronger cravings than either the positive or neutral affect scripts.

Coffey, Stasiewicz, Hughes, and Brimo (2006) examined cue-elicited craving in a sample of patients with comorbid post-traumatic stress disorder and alcohol dependence, in the context of an exposure-based, trauma-focused treatment. They found that, as expected, participants reported increased craving for alcohol, measured using a visual analog scale, and higher levels of emotional distress, in response to exposure to both trauma and alcohol cues. This demonstrates the ability of negative affect to elicit urges in the same way that alcohol cues do. Further supporting the evidence of a relationship between negative affect and craving, the study also found that individuals who received the exposure-based treatment, compared to a relaxation only treatment, showed decreased alcohol craving and decreased emotional distress at the post-treatment testing session, suggesting that attenuation of negative affect through effective treatment resulted in accompanying attenuation of craving.

Fox, Bergquist, Hong, and Sinha (2007) replicated this finding of similar reactivity to negative affective cues and alcohol cues when they investigated the effect of emotional and alcohol related cues on alcohol craving and emotion indices. The cues took the form of personalised scripts, based on each participant’s own experiences in relation to a stressful event, an alcohol-related event and a neutral/relaxing event. These scripts were then recorded onto a tape and played to participants in a laboratory session during which subjective and physiological measures of emotional state and alcohol craving were taken. Results revealed that both the stress and alcohol conditions increased subjective reports of craving and measures of physiological arousal, compared to the neutral/relaxing script. Exposure to these scripts also increased reported strength of negative emotions. These findings demonstrate the ability of a stressful, negative
Negative affect in addiction

emotion-provoking event to elicit alcohol cravings, but also provide some support for the ability of craving, induced through exposure to alcohol-related cues, to elicit negative emotion.

Similar evidence of the ability of negative affect alone to induce heightened craving is provided by Cooney, Litt, Morse, Bauer and Gaupp (1997), who conducted mood inductions followed by cue-exposure with alcohol dependent males currently engaged in inpatient treatment. They presented participants with either a neutral or negative mood script followed by either a neutral (spring water) or alcohol cue. Self-reported craving for alcohol increased significantly in the negative affect group who were subsequently exposed to the neutral beverage cue, demonstrating that induction of a negative mood state is sufficient to trigger a craving response, even in the absence of an alcohol cue. Addition of the alcohol cue enhanced the effect further. Comparable effects have also been observed with cannabis users, with McRae-Clark et al. (2011) finding that self-reported craving for cannabis increased following a stress induction task compared to the non-stress task. Unlike Cooney et al.’s (1997) study, they did not find added effect of a cannabis cue over and above the response to the stress cue.

Contrary to the findings above, Kambouropoulos and Staiger (2009) did not find an association between exposure to alcohol cues and subsequent positive or negative affect, even though self-reported urge to drink did increase. However, their study was with a sample of relatively young social drinkers (mean age of 25 years) who only had to be drinking five or more standard drinks a session (for males) or four per session (for females) at least once per week. Such a group is less likely to be motivated by negative reinforcement contingencies and so is less likely to have an affective response to alcohol cues. In fact, the authors conclude that the increase in craving was for the pleasant effects of alcohol.
5.2.2 Negative affect and craving in situ

Of greater ecological validity are studies examining the relationship between negative affect and craving in real life situations. This typically involves momentary administration of state measures of affect, followed by subsequent analyses that focus on comparison of people who are high on negative affect versus low. Studies of this nature could be argued to have greater ecological validity, as the affective states measured represent a genuine affective condition that is likely characteristic of the individual in other ‘real-life’ circumstances, and therefore likely to be a more realistic representation of the relationship between affect and craving in situ. A number of studies have examined negative affect/craving relationships in this way and have found associations consistent with laboratory based research.

Cleveland and Harris (2010) examined daily moods and cravings amongst college students engaged in a 12-step recovery program. They found that negative affect predicted same day craving, although this prediction was moderated by avoidance coping and to a lesser extent, problem-solving coping. Negative social experiences were also found to predict same day cravings. These results suggest that negative experiences and emotions in a given day will elicit alcohol craving, particularly if an individual uses avoidance coping strategies.

Rohsenow et al. (2007) studied the interaction between mood and craving prior to first relapse to cocaine use following treatment, as well as prior to situations where relapse almost occurred but was able to be resisted. Craving was not related to either positive or negative mood prior to an actual lapse, but was in the situations where a lapse almost occurred but did not. In these situations, craving was significantly positively correlated with negative mood and negatively correlated with positive mood. The authors do not offer an explanation of this effect, but it is possible that in the group
Negative affect in addiction

that resisted the urge, negative affect was generated due to a sense of deprivation (Kavanagh et al., 2005). This effect was not seen in the group that actually lapsed because their craving led to substance use, and so there was little or no sense of deprivation.

Andersohn and Kiefer (2004) examined the relationship between depressed mood and craving during alcohol withdrawal, taking daily ratings of each for 14 days from the day of admission. Strong positive correlations were observed between the two measures throughout withdrawal, with the greatest magnitudes in the earlier stages. The relationship was found to be specific to depression, with anxiety scores not correlating with craving. The correlations also persisted when other demographic and drinking history variables were controlled for.

Osli et al. (2009) found evidence for reverse directionality in the relationship between affect and craving, finding that higher craving during treatment was associated with higher reported levels of negative affect. This is consistent with the results of Witkiewitz and Villarroel (2009), suggesting the relationship between affect, craving and substance use is a dynamic one, where each can influence the other, possibly within a feedback loop as suggested by Witkiewitz and Villarroel.

5.3 Summary of role of negative affect in addiction

In summarising the research on the relationship of negative affect to substance use and craving, the same methodological issues that are encountered in the field of craving research must be considered. Craving research is limited by substantial variation in the methods and timing of assessment of both craving and of the outcomes it attempts to predict. Similarly in research on negative affect, there is substantial variability in the way in which affect and substance use are defined, measured and tested. Conner et al.
(2009) noted in their meta-analytic review that continuous measures of depression were more strongly associated with concurrent alcohol use and impairment compared to categorical measures, suggesting the way in which affect is measured can have a significant impact on results. Perkins, Karelitz, Conklin, Sayette, & Giedgowd (2010) conclude this also, noting that within their own study, relationships between negative affect and ad libitum smoking differed depending on the affect measure used, with some measures showing significant associations and others showing no relationship at all. They also highlight the fact that results differ depending on the source of the negative affect, with relationships being seen in some situations (after 12 hours of abstinence and after preparation and delivery of two speeches about bodily likes and dislikes), but not in others (such as a challenging computer task or exposure to negatively valenced slides). Additionally, many studies examine negative affect as a composite of a variety of negative mood states such as depression, anxiety, stress and irritability; or they examine only one of these, making it difficult to generalise results to the overarching construct of ‘negative affect’. For example, Swendsen et al. (2000) found that increases in ratings of nervousness was associated with higher alcohol intake later in the same day, but did not find the same effect for sadness, suggesting effects may be limited to certain emotional states only, or that emotional states relate differentially to substance use.

Despite these methodological shortcomings and differences across studies, the weight of evidence indicates that, in general, negative affect is highly associated with increased risk for substance use and elevated craving (Conner et al., 2009). Given the strength and direction of these associations, it seems highly plausible that negative affect could be one variable that interacts with craving to influence substance use outcomes. Evidence suggesting that this is the case has recently been emerging.
5.4 Dynamic association between craving, negative affect and substance use

Evidence suggesting a dynamic relationship between negative affect and craving, and potential interactions in the way they relate to influence outcomes has been steadily mounting. For example, Gordon et al. (2006) found that actively craving alcohol in the week prior to discharge from inpatient treatment was highly predictive of alcohol use in the 3 months following discharge. They also found that patients coded as being active cravers in the week before discharge had significantly higher Beck Depression Inventory scores on entering treatment than the patients who reported no craving in the week before discharge. Elevated depression appeared to be associated with vulnerability for higher craving, and this association may have contributed to poorer outcomes following treatment.

Berkman et al. (2011) found evidence for such a dynamic between mood and craving in their study of factors influencing smoking lapse during a quit attempt. They found that negative mood and craving at a given time point predicted smoking lapse at the next time point (2 hours later), and that in this relationship, craving acted as a mediator between negative mood and smoking. The dynamic relationship between depression and craving in influencing substance treatment outcomes is further evidenced by studies demonstrating that addressing one or the other in treatment results in changes in the other, which subsequently influences the substance use outcomes.

Witkiewitz and Bowen (2010) demonstrated this in their finding of a moderated mediation relationship between negative affect, craving and substance outcomes following treatment. The authors propose that treatment targeting the effect of depression-related negative cognitions on substance use and relapse risk can have an indirect effect on craving, which then mediates the relationship between depression and substance use following treatment. They delivered mindfulness-based relapse
prevention or treatment as usual to 168 substance abusers (45.2% alcohol; 36.2% cocaine/crack; 13.7% methamphetamine; 4.9% other) who had already completed a private intensive inpatient or outpatient treatment within the previous 2 weeks. Mindfulness-based relapse prevention consisted of eight weekly 2-hour sessions that included guided meditations and experiential exercises. Treatment as usual continued in standard outpatient aftercare.

The goal of mindfulness-based relapse prevention interventions is to teach clients to “observe physical cognitive, emotional, or craving states without ‘automatically’ reacting.” (Witkiewitz & Bowen, 2010, p. 363). By training clients to better manage the negative cognitive and mood states that are associated with depression, mindfulness-based relapse prevention should reduce the likelihood of these negative states leading to substance use and relapse. Results showed that craving at 2 months post-treatment, depression scores at immediate post-treatment, and total treatment hours were all significant predictors of 4-month post-treatment days of substance use. Furthermore, craving at 2 months post-treatment was significantly predicted by treatment allocation and by depression scores immediately post-treatment. These relationships were further explored in a series of mediation and moderation analyses. The relationship between depressive symptoms immediately following treatment, and treatment outcomes 4 months later, was partially mediated by craving measured 2 months post-treatment. Depressive symptoms at immediate post-treatment moderated the relation between treatment allocation and 2-month post-treatment craving. The relation between depressive symptoms immediately post-treatment and craving 2 months post-treatment was mediated by craving in the treatment-as-usual group, but not in the mindfulness-based relapse prevention group. Witkiewitz and Bowen (2010) conclude that the mindfulness-based relapse prevention intervention
Negative affect in addiction attenuated the impact of depressive symptoms on craving, thereby reducing post-intervention substance use.

To further test this finding that intervention-attenuated craving mediates the relationship between depression and substance use, Witkiewitz, Bowen and Donovan (2011) examined the moderating effect of a craving-focused treatment module between negative mood and heavy drinking following treatment for alcohol dependence. Data from the COMBINE study were used, as one of the combined behavioural interventions included a module specifically targeting craving. Witkiewitz et al. (2011) used the subsample of participants who received this module to examine if craving attenuation through treatment moderates the relationship between negative mood and drinking outcomes, comparing them to participants who did not receive the module.

A series of analyses supported the model of moderated mediation proposed by Witkiewitz and Bowen (2010). They found that decreases in negative mood during the course of treatment were associated with decreases in frequency of heavy drinking and that this effect was moderated by the Coping with Craving and Urges module, such that people who received the module showed a weaker relationship between negative mood and heavy drinking during treatment and 1 year later than people who did not receive the module. Furthermore, changes in craving during treatment explained the effect of the craving module on the relation between negative mood and heavy drinking during the course of treatment. Consistent with the results of Witkiewitz and Bowen (2010), a model of moderated mediation was supported, where the effects of negative mood on drinking were mediated by changes in craving following the craving module.

The results of these two studies are consistent with negative reinforcement models that postulate that negative affective states elicit urges to use with the aim of relieving the negative state, and that these urges then lead to substance seeking and use.
These results imply that the combination of high levels of negative affect with high levels of craving leave an individual more vulnerable to substance use, and other research has supported this. Carpenter et al. (2009) found that higher craving scores in response to stressful imagery cues were significantly and positively correlated with increased smoking in the following week, while reactivity to smoking-only cues was not. This suggests that craving generated by negative affective events may be a more powerful antecedent of substance use than craving that has not been generated by negative affect. It is possible that affectively driven craving may be a stronger predictor of longer-term substance use, and of substance use outcomes following treatment than non-affectively driven craving. Of particular clinical interest is whether this high affect with high craving combination could be measured before treatment to determine who may be more at risk for poor treatment response or for relapse.

5.5 Craving as a predictor of outcomes in the context of negative affect

After reviewing the literature, only one study was found that investigated the predictive performance of pre-treatment craving in the context of high negative affectivity. Farren and McElroy (2010) examined predictors of relapse following inpatient integrated treatment for comorbid alcohol dependence with unipolar or bipolar depression. Of the 187 participants recruited, 30.1% had bipolar I disorder, 16.7% had bipolar II disorder and 53.1% had major depressive disorder. Participants completed the baseline assessment prior to starting a 4-week inpatient CBT-based program, but after detoxification, which was at least 7 days after their last drink. Assessments were repeated 3 and 6 months after discharge from the 4-week program. Baseline craving was measured using the OCDS and sustained abstinence was measured as the outcome variable. It was not explicitly stated in the study, but it is assumed that any drinking in
Negative affect in addiction

the follow-up period constituted a relapse. Depression was measured at baseline using the Beck Depression Inventory, and the mean score for both the unipolar and bipolar depressed participants was within the moderate range, confirming that participants were experiencing elevated levels of negative mood.

Regression analyses revealed that baseline craving was not a significant predictor of relapse in the 6 months post-discharge, seemingly discounting an effect of increased risk from the combination of high craving with comorbid depression. However, there were a number of methodological limitations with the study that restrict generalisability of results. Firstly, craving was measured at least a week after the participants’ last drink, after they had completed detoxification. Craving is known to fluctuate in response to salience of cues (Kavanagh et al., 2005; Monti et al., 2000) and has been documented to decline steadily during withdrawal (Shiffman et al., 1997). The experience of craving after admission to treatment and after abstinence has commenced could be influenced by these factors, and so is unlikely to be a true representative of pre-treatment craving. Even though participants had not started the research treatment at the time of assessment, intervention began the moment they were admitted and commenced detoxification and mood stabilisation. This could also have attenuated depressive symptoms, further reducing the potency of the effect of the negative affect on craving. As discussed in Chapter 4, the context in which craving is measured is important for the context for which one is trying to predict, and craving is likely to perform more robustly as a predictor if it is measured in a context and state relevant to that being predicted (Sinha & O'Malley, 1999).

Another limitation of Farren and McElroy’s study is the outcome measure they employed. As discussed earlier, evidence suggests that craving may perform more consistently in prediction of continuous outcome measures that convey level of
consumption rather than simply whether a relapse occurred or not (Garbutt et al., 2009; Ray et al., 2006; Rohsenow et al., 2007; Rohsenow et al., 1994). Farren and McElroy did measure continuous outcomes (number of drinking days and average units per drinking day), but did not report on prediction of these outcomes from baseline variables. Also, Farren and McElroy were interested in identifying which of a range of variables predict treatment outcomes, and so did not explicitly study potential interactions between level of depression and craving and how these interactions may influence outcomes. For all of these reasons, the results of Farren and McElroy’s (2010) study are not able to answer the question of whether pre-treatment levels of craving experienced in the context of elevated levels of negative affect are a stronger predictor of outcomes than craving that is not paired with high levels of negative affect.
CHAPTER 6  AIMS OF THIS THESIS

This thesis attempts to address some of the limitations of past research to explore the dynamic relationship between negative affect and craving and how this impacts on outcomes following treatment for alcohol use. As discussed earlier, being able to predict likely outcomes following treatment for alcohol use disorder would be highly advantageous for treatment and follow-up planning. Craving has long been touted as a potential indicator of treatment outcomes and has been extensively studied. However, the picture of how it relates to post-treatment alcohol use remains unclear. This is partly due to wide variability in measurements used, outcomes defined and assessment points targeted, but may also be due to the influence of other variables that are not being measured consistently in other studies. It is plausible that craving’s relationship to post-treatment drinking could be moderated by a number of other variables, negative affect being one likely candidate. This thesis attempts to explore this and to also overcome limitations of previous research in a number of important ways.

6.1 Studying craving as a predictor in drinkers with comorbid depressed mood

Drawing on the observation of Suter et al. (2011) that active depression symptoms appear to be more strongly associated with relapse vulnerability than depressive disorder per se, an inclusive approach to depression comorbidity will be taken, with inclusion being based on current level of depression severity rather than a diagnosis of major depression. This will ensure that a certain level of active depressive symptoms is present in the sample being studied.
This thesis will also extend on previous research by examining interaction effects between craving and depression. Although evidence has been found that craving acts as a mediator between negative affect and later substance use (Berkman et al., 2011; Witkiewitz & Bowen, 2010; Witkiewitz et al., 2011), two of these studies examined this relationship as an effect of treatment and the other examined proximal relationships only. The current research is focused on prediction of post-treatment outcomes from pre-treatment craving and mood states to investigate whether the combination of the two have more than an additive effect in predicting post-treatment drinking.

6.2 Using context and construct relevant measures

Craving and depression will be measured in an outpatient setting, prior to delivery of any intervention and while alcohol reduction is being contemplated. This is a context most directly comparable to that being predicted and measures at such a time are likely to be representative of craving and mood states the participant is likely to experience following treatment, after support is withdrawn. Such state-relevant measures are likely to have greater predictive potential (Sinha & O'Malley, 1999).

To remove the confounding effects of factors that may be associated with craving but are not truly representative of the construct, only the obsessive subscale of the OCDS will be used as a measure of craving. The obsessive subscale captures cognitive aspects of craving, such as frequency of drinking thoughts, interference and distress caused by drinking thoughts and efforts to resist those thoughts. The compulsive subscale of the OCDS reflects consequences of craving rather than being a characteristic of craving itself (Modell, Glaser, Cyr, & Mountz, 1992), and the two consumption items that usually form part of the compulsive subscale confound craving
Aims of this thesis

with current alcohol use. For this reason, Nakovics, Diehl, Croissant, & Mann (2008) recommend that these items be excluded when using the OCDS as a predictor.

6.3 Use of continuous outcome measures

As noted earlier, craving appears to perform more consistently when predicting outcomes measured on a continuous rather than dichotomous scale. Additionally, focusing only on binary outcomes of relapse does not allow for detection of partial success. Therefore, the present research will employ continuous outcome measures with the intention of improving sensitivity in the analyses.
CHAPTER 7 STUDY 1: CRAVING AS A PREDICTOR OF TREATMENT OUTCOMES IN HEAVY DRINKERS WITH COMORBID DEPRESSED MOOD

7.1 Introduction

Incidence of depression and alcohol use disorder comorbidity is high, in both the general population and in treatment settings (Burns & Teesson, 2002; Frisher, Collins, Millson, Crome, & Croft, 2004; Grant et al., 2006; Kessler, Chiu, Demler, & Walters, 2005; Weaver et al., 2003). Population surveys suggest a 12-month prevalence of 14-17% of major depressive disorder in people with an alcohol use disorder, and a 2-3% prevalence of dysthymic disorder (Burns & Teesson, 2002; Grant et al., 2006). Survey respondents with 12-month alcohol use disorder were 2-3 times more likely to have a 12-month major depressive or dysthymic disorder than respondents who did not have an alcohol use disorder (Burns & Teesson, 2002; Grant et al., 2006). Rates are even higher in treatment-seeking populations, with around 40% of patients presenting for alcohol treatment also having a concurrent depressive disorder (Grant et al., 2006; Weaver et al., 2003).

Comorbidity of depression with substance use disorder is associated with a range of poor outcomes following treatment. Patients with comorbid disorders tend to have lower rates of remission, longer time to remission, greater levels of mental distress, higher risk of psychiatric adverse events and hospitalisation, higher incidence of physical comorbidities and poorer social functioning (Davis et al., 2010; Howland et al., 2009; Kirchner et al., 2002; Landheim, Bakken, & Vaglum, 2006). Comorbid
conditions cost more to the health system than single disorders (Mihalpopoulos, Meadows, Stiller, Pirkis, & Burgess, 2005). In the US, it is estimated that comorbid major depression with substance use disorder requires 61% greater mental health and substance abuse treatment costs, and 44% greater medical care costs, compared with depression alone (Mark, 2003). The ability to predict the likely treatment response of patients with depression and substance use comorbidity would facilitate identification of those most at risk of relapse and would enable treatment plans to be tailored accordingly. This might allow for more efficient and cost-effective distribution of resources, and potentially, better outcomes for clients.

A number of factors have been found to be associated with poorer clinical outcomes in depression and substance use comorbidity. Being younger and single is more likely to be associated with poor outcomes, as is earlier onset of the substance use disorder and longer history of mood disorder (Landheim et al., 2006), though people with a primary substance use disorder rather than mood disorder tend to have better outcomes (Najt, Fuser-Poly, & Brambilla, 2011). Farren and McElroy (2010) found baseline anxiety, illegal drug history and high score on the Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, de le Fuente, & Grant, 1993) to be predictive of poorer drinking outcomes in comorbidity of depression with alcohol. Similarly, Kirchner et al. (2002) found comorbid drug use disorder on top of alcohol use disorder to be predictive of poor outcomes and Xie, Drake, McHugo, Xie and Mohandas (2010) found alcohol dependence, rather than abuse, to be associated with lower remission rates. Findings with regards to gender have been mixed, with some finding males do worse (Najt et al., 2011) and others finding females do (Kirchner et al., 2002).

Craving, or the subjective desire or need for a substance, has been touted as a potential indicator of treatment outcomes in substance using populations. Some
empirical support for the prediction of treatment outcomes from pre-treatment craving has been found for alcohol (Bottlender & Soyka, 2004; Garbutt et al., 2009; Ray et al., 2006; Rohsenow et al., 1994), smoking (Fidler et al., 2011; Killen et al., 1999; Toll et al., 2007) and cocaine (Crits-Christoph et al., 2007; Paliwal et al., 2008). However, other studies have found craving to perform poorly in prediction models (Ahmadi et al., 2009; Kampman et al., 2004; Kiefer et al., 2005; Kranzler et al., 1999; Zhou et al., 2009) suggesting there may be certain conditions under which craving will act as a predictor.

The impact of comorbid depressed mood on craving’s predictive performance has been largely neglected, despite demonstrated associations between negative affect and craving. For example, negative affect has been found to predict same-day craving for alcohol (Cleveland & Harris, 2010) and to be correlated with cocaine craving that was elicited by highly tempting situations but did not result in relapse (Rohsenow et al., 2007). Additionally, elevated depression and negative affective stimuli have been found to be associated with heightened reactivity to alcohol cues (Feldstein Ewing et al., 2010; Fox et al., 2007). The presence of comorbid negative affect, such as experienced in depression, may augment the relationship between craving and substance use, improving craving’s predictive ability.

Despite these clear relationships, Farren and McElroy’s (2010) was the only study identified that examined pre-treatment craving as a predictor of alcohol relapse in a population with comorbid depression. They reported that baseline craving, measured using the OCDS (Anton et al., 1995), was not a significant predictor of relapse to alcohol during the 6 months following discharge. However, as previously discussed in Chapter 5, key limitations existed with this study, the most important of which included the temporal sequencing of mood, alcohol and craving measurements, with baseline
craving and mood measurements taken after alcohol detoxification and mood stabilisation had already occurred. Since state measurements of craving show strong associations with subsequent drinking (Oslin et al., 2009) and smoking (Allen et al., 2008; Chandra et al., 2011; Shiffman et al., 2002), craving measured in a state that most closely resembles that experienced after disengagement from treatment is likely to hold the greatest predictive potential. A further limitation of the study by Farren and McElroy (2010) was the use of dichotomous outcomes (abstinent versus relapsed), which restricts the power of predictive analyses. Also, relapse was not clearly defined.

The present study examined the ability of craving to predict alcohol use outcomes in a sample of depressed heavy drinkers who were taking part in a randomised controlled trial comparing four psychological treatments for comorbidity of depression with alcohol misuse. Limitations of previous research were addressed in a number of important ways: (i) measuring craving in a state relevant to the context being predicted (i.e., measuring craving prior to treatment and while contemplating control of drinking); (ii) measuring drinking outcomes on a continuous scale to enable examination of the strength and degree of relationships; (iii) including participants with moderate levels of depression and alcohol use, regardless of whether they met diagnostic criteria for major depression or alcohol dependence. This ensured a broader representation of the range of comorbidity experienced by people with depression and alcohol use problems.

It was hypothesised that, in the presence of comorbid depressed mood, pre-treatment craving would significantly predict post-treatment alcohol consumption, controlling for treatment condition and the baseline measure of the dependent variable. It was also hypothesised that there would be an interaction effect between mood and craving, with the combination of high depressed mood and high craving at pre-treatment predicting higher alcohol consumption post-treatment.
7.2 Method

7.2.1 Participants

Data for the present study were collected during a randomised controlled trial investigating integrated and single-focused treatments for comorbid alcohol use and depression (Baker et al., 2010). Participants for the study were recruited via television and print advertising and through promotion of the research to health, government and non-government agencies. The study was conducted at two Australian locations: Newcastle, New South Wales and Brisbane, Queensland. The study held ethical clearance from The University of Newcastle, The University of Queensland and Queensland University of Technology.

Inclusion criteria were: (i) aged over 16 years; (ii) Beck Depression Inventory-II (Beck et al., 1996) score ≥ 17 (higher end of mild range); and (iii) at least two occasions in the previous month of > 6 x 10g ethanol drinks for men or > 4 for women. Exclusion criteria were: (i) positive screen on The Screening Instrument for Psychosis (White & Chant, 2006); (ii) self-reported history of traumatic brain injury; (iii) lack of English fluency evidenced by difficulty comprehending study information; and (iv) inability to travel to treatment sessions. Concurrent pharmacotherapy was not excluded, but a stable dose for ≥ 4 weeks was required before participation.

A total of 284 participants (53% male) were recruited and randomly allocated to the four treatment conditions (Baker et al., 2010). The CONSORT\(^4\) diagram outlining recruitment and attrition is presented in Figure 7.1. Demographic characteristics of the sample are presented in Table 7.1.

\(^4\) The CONSORT (Consolidated Standards of Reporting Trials) statement was developed to improve the reporting of clinical trials. It comprises a checklist of essential items that should be included in reports of randomised controlled trials and guidelines for documenting the flow of participants through a trial.
Figure 7.1. CONSORT diagram showing flow of participants through Study 1.
Table 7.1

Demographic characteristics of the full sample (n=284)

<table>
<thead>
<tr>
<th>Demographic Characteristics</th>
<th>M (SD, range) or n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age (years)</td>
<td>45.51 (10.93, 20–73)</td>
</tr>
<tr>
<td>Male</td>
<td>149/284 (52.5%)</td>
</tr>
<tr>
<td>Married/partnered</td>
<td>95/284 (33.5%)</td>
</tr>
<tr>
<td>Mean age left school (years) (n=281)</td>
<td>16.09 (1.40, 11–21)</td>
</tr>
<tr>
<td>Post-school qualification</td>
<td>134/277 (48.4%)</td>
</tr>
<tr>
<td>Receiving welfare support</td>
<td>111/281 (39.5%)</td>
</tr>
</tbody>
</table>

7.2.2 Measures

Only a subset of the data collected in the study were used for the present research. These are the measures described below and provided in Appendix A (excluding copyright measures). A full description of all measures used in the original study can be viewed in Baker et al. (2010). Assessments were administered in face-to-face interviews by postgraduate-trained psychologists, with the exception of the obsessive subscale of the OCDS, which was provided in written form with other self-report questionnaires to be completed by the participant at home. This minimised assessment contact time.

7.2.2.1 Alcohol

Consumption

The Timeline follow-back (TLFB; Sobell & Sobell, 1992) was used to assess alcohol consumption in the preceding 2 weeks to obtain a point estimate of alcohol use at the time of assessment. The TLFB uses a calendar-based approach to collect retrospective substance use data using event-based cues to aid recall. Two outcome
variables were calculated from the TLFB data; average weekly consumption and average number of binge days (> 6 x 10g ethanol/occasion for men; > 4 x 10g ethanol for women) per week.

The TLFB has demonstrated sound temporal stability in substance using groups who have comorbid psychiatric diagnoses, with 30-day test-retest reliability coefficients ranging from .73 to 1.00 (Carey, Carey, Maisto, & Henson, 2004). Convergent validity in comorbid samples has also been demonstrated, with correlations of .78 between the TLFB and Addiction Severity Index (McLellan et al., 1980) in measuring number of drinking days in the last 30 days, and .52 between the TLFB and collateral reports in measuring number of drinking days in the last 90 days (DeMarce, Burden, Lash, Stephens, & Grambow, 2007). Drinking data derived from shorter assessment timeframes of 7 and 14 days has also been found to correlate strongly with data derived from longer timeframes of 30 and 60 days (Toll, Leeman, McKee, & O'Malley, 2008).

**Craving**

The obsessive subscale of the OCDS (OCDS-O; Anton et al., 1995) was used to measure alcohol craving. The compulsive subscale was not administered as the items focus on compulsive drinking behavior which is more reflective of the consequences of craving rather than a fundamental characteristic of craving itself (Kavanagh et al., 2013; Modell, Glaser, Cyr, et al., 1992). The OCDS-O is comprised of six items assessing drinking-related thoughts and the distress and preoccupation caused by those thoughts (Kranzler et al., 1999; Nakovics et al., 2008). Items are scored from 0 to 4, giving a maximum possible total score of 24, with higher scores representing greater craving.

This subscale has good internal consistency (alpha = .83; Kranzler et al., 1999) and is significantly correlated with the Alcohol Dependence Scale and Addiction
Severity Index (Kranzler et al., 1999), the Penn Alcohol Craving Survey (Flannery, Volpicelli, & Pettinati, 1999) and the Alcohol Urge Questionnaire (Bohn et al., 1995; Rosenberg & Mazzola, 2007). Internal consistency (Cronbach’s alpha) in the present study was .90.

Severity of dependence

Alcohol dependence severity was included in the current study only to describe the characteristics of the sample. It was not investigated as an outcome or predictor as alcohol consumption was the primary outcome of interest. A 6-month version of the Alcohol Use Disorders Identification Test (AUDIT; Saunders et al., 1993) was used as an index of severity. The AUDIT is a 10-item self-report scale assessing consumption and indices of dependence.

Reinert and Allen (2007) provide an overview of the psychometric properties of the AUDIT, noting a median internal reliability of .83 across studies and test-retest reliabilities over 1 to 4 weeks ranging from .70 to .89. Criterion validity is also noted to be high with a median sensitivity of .78 and median specificity of .89. Concurrent validity has been demonstrated with significant correlations between the AUDIT and other measures of dependence such as the Alcohol Dependence Scale, as well as with indices of alcohol consumption (Maisto, Conigliaro, McNeil, Kraemer, & Kelley, 2000). Predictive validity has also been demonstrated, with baseline AUDIT scores correlating significantly with measures of consumption and dependence 12 months later (Maisto, Conigliaro, et al., 2000), and with various indices of alcohol-related harm after 2-3 years (Conigrave, Saunders, & Reznik, 1995). Its psychometric performance in

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5 A measure of the proportion of people with alcohol dependence who were correctly classified as such.

6 A measure of the proportion of people who do not have alcohol dependence who were correctly classified as such.
comorbid psychiatric populations has been established to be consistent with its strong performance in the general population (Dawe, Seinen, & Kavanagh, 2000; Maisto, Carey, Carey, Gordon, & Gleason, 2000; O'Hare, Sherrer, LaButti, & Emrick, 2004). Cronbach’s alpha in the present study was .69.

Alcohol use disorder diagnosis

This variable was used in the current study only to describe the sample, and was not used as an outcome or predictor. Clinical diagnosis of current and lifetime alcohol use disorder was determined using the Structured Clinical Interview for DSM-IV (SCID; First, Gibbon, Spitzer, & Williams, 1995).

7.2.2.2 Depression

Severity of depression

Severity of depression symptoms was measured using the Beck Depression Inventory, version 2 (BDI-II; Beck et al., 1996). A short version of the BDI (the Beck Depression Inventory – Fast Screen; Beck, Steer, & Brown, 2000) was used at screening to determine initial eligibility. The BDI-II is a 21 item self-report questionnaire used to screen for the presence of depressive symptoms over the previous two weeks. The BDI-II has demonstrated high internal consistency ranging from .87 to .91 (Dozois, Dobson, & Ahnberg, 1998; Storch, Roberti, & Roth, 2004; Titov et al., 2011) and excellent test-retest reliability of .96 over 1 to 12 days (Sprinkle et al., 2002). Convergent validity has been supported by significant correlations with the Patient Health Questionnaire 9-Item version (Kroenke, Spitzer, & Williams, 2001; Titov et al., 2011) and the State-Trait Anxiety Inventory-Trait Version-Depression subscale (Bieling, Antony, & Swinson, 1998; Storch et al., 2004). The BDI-II’s criterion validity has also been supported, with scores successfully discriminating between college students with and without a
diagnosed affective disorder (Sprinkle et al., 2002). A sensitivity of .71 and specificity of .88 were documented by Dozois et al. (1998).

Use of the BDI-II as a depression screen in addiction treatment settings has been supported, with similarly high internal consistencies over .90 observed in substance using populations (Buckley, Parker, & Heggie, 2001; Dum, Pickren, Sobell, & Sobell, 2008; Hepner, Hunter, Edelen, Zhou, & Watkins, 2009). Cronbach’s alpha in the present study was .83.

Depression diagnosis

Depression diagnosis was used only to describe the sample. Clinical diagnosis of current, 12-month and lifetime major depressive episode was assessed with the Structured Clinical Interview for DSM-IV (SCID; First et al., 1995).

7.2.3 Interventions

Interventions were fully manualised, and are described in detail by Baker et al. (2010). Following assessment, all participants attended a 90-minute session providing assessment feedback, psychoeducation and motivation enhancement for both depression and alcohol misuse. At the end of this session, the therapist opened a sealed envelope, revealing the random allocation that had been independently assigned to that participant. This procedure ensured consistency in delivery of the first session across conditions.

Participants were allocated to one of four treatment conditions: (i) Brief Intervention, where participants did not receive further treatment after the initial session; (ii) Alcohol Intervention, comprising nine additional 1-hour sessions that delivered cognitive-behavioural therapy (CBT) incorporating brief mindfulness exercises, focused on alcohol reduction; (iii) Depression Intervention, comprising nine
additional 1-hour sessions that delivered CBT and mindfulness exercises focused on depressed mood; or (iv) *Integrated Intervention*, where the additional nine 1-hour sessions provided integrated CBT and mindfulness exercises targeting both alcohol use and depressed mood.

### 7.2.4 Procedure

After self-referral, potential participants were screened by telephone for initial eligibility. This included assessment of age, English language fluency, ability to travel to appointments, recent alcohol use, history of psychosis and administration of the BDI-Fast Screen. Participants who passed this initial screening were invited to attend a face-to-face assessment interview during which final eligibility was determined. If the participant was eligible, he or she was invited to enter the study and to complete the remainder of the baseline assessment, consisting of a take-home self-report assessment package and a neuropsychological assessment. Approximately one week after completion of the baseline assessment, participants attended the first treatment session. Assessments were repeated by blind assessors 18 weeks, 6 months and 12 months post-baseline. Participants were paid $20 for each completed assessment, including baseline.

### 7.2.5 Statistical analysis

Data were analysed with SPSS Statistics 19. An intention-to-treat approach was used. Missing data were substituted with estimations generated using the Expectation-Maximisation method in SPSS Missing Value Analysis. Variable characteristics including normality and extreme values were examined using descriptive statistics.

Consistent with previous reports of this study (Baker et al., 2010), intervention effects were examined using three orthogonal contrasts: (i) Brief versus Long; (ii)
Integrated versus Single-focused, and; (iii) Alcohol versus Depression. The effect of an interaction between baseline depression and craving was examined by creating an interaction term from the standardised, mean centered scores and entering this as a variable in the analyses. Aiken and West (1991) recommend using standardised scores to test interactions as this sets the mean of both variables to 0, enabling more meaningful interpretation of the effect of each variable on the other and on the dependent variable.

Two key outcome variables were examined in the analyses: average weekly consumption and average weekly binges (> 6 x 10g ethanol/occasion for men; > 4 for women). Relationships of baseline and treatment variables with outcome variables were examined using Pearson bivariate correlations (where the predictor and outcome variables were both continuous), univariate analysis of variance (ANOVA; where the predictor was categorical and the outcome was continuous or vice versa) or Pearson chi-square (where both the predictor and outcome were categorical). Uncontrolled analyses were run in the first instance to examine raw relationships, followed by a repeat analysis that controlled for the baseline measure of the outcome being predicted (partial correlations, analysis of covariance; ANCOVA or logistic regressions) to partial out covariance attributable to baseline drinking.

The unique prediction from key variables was examined in linear and multinomial logistic regressions. The focus of this study was on prediction of short-term post-treatment (18-week) and long-term post-treatment (12-month) follow-up outcomes from baseline measures. For all analyses, an alpha cut-off of .05 was adopted.
Study 1

7.3 Results

A total of 284 participants (53% male) were recruited and randomly allocated. However, the TLFB was not collected for 24 participants at baseline. As this was a key predictor, these participants were excluded, producing a sample of 260. The demographic characteristics of the sample did not alter substantially after removal of these participants (see Appendix B). Baseline characteristics of the reduced sample ($N = 260$) are presented in Table 7.2.

Table 7.2

Baseline characteristics of the analysed sample ($N = 260$)

<table>
<thead>
<tr>
<th>Continuous Variables</th>
<th>$M$</th>
<th>($SD$, range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TLFB mean drinks per week</td>
<td>61.60</td>
<td>(42.95, 6.3-280)</td>
</tr>
<tr>
<td>TLFB mean binge days per week</td>
<td>4.39</td>
<td>(2.31, 0-7)</td>
</tr>
<tr>
<td>TLFB mean abstinent days per week</td>
<td>1.58</td>
<td>(1.88, 0-6.5)</td>
</tr>
<tr>
<td>TLFB mean drinks per drinking day</td>
<td>11.40</td>
<td>(6.70, 2.4-53)</td>
</tr>
<tr>
<td>Mean BDI-II total</td>
<td>31.54</td>
<td>(8.95, 17-55)</td>
</tr>
<tr>
<td>Mean AUDIT total</td>
<td>25.70</td>
<td>(6.59, 7-40)</td>
</tr>
<tr>
<td>Mean OCDS-O total ($n = 207^a$)</td>
<td>9.17</td>
<td>(5.75, 0-23)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Categorical Variables</th>
<th>$n$</th>
<th>(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major depressive episode: current</td>
<td>180/257</td>
<td>(70.0%)</td>
</tr>
<tr>
<td>Alcohol abuse diagnosis</td>
<td>177/260</td>
<td>(68.1%)</td>
</tr>
<tr>
<td>Alcohol dependence diagnosis</td>
<td>218/260</td>
<td>(83.8%)</td>
</tr>
<tr>
<td>Current antidepressant medication</td>
<td>132/257</td>
<td>(51.4%)</td>
</tr>
<tr>
<td>Current anticraving medication</td>
<td>15/257</td>
<td>(5.8%)</td>
</tr>
</tbody>
</table>

$^a$ The OCDS-O was added to the assessment battery after recruitment had commenced, and was not collected from the first 52 participants. Data was incomplete for one participant.

Participants had a mean weekly alcohol consumption well in excess of Australian guidelines for low risk drinking (> 28 standard drinks per week for men; >
14 standard drinks per week for women; National Health and Medical Research Council, 2009), but wide variations were observed, ranging from a minimum of just 6.3 standard drinks per week to a maximum of 280. The mean depression score of the sample fell within the lower end of the severe range, indicating that recruitment targeting alcohol misuse comorbid with high levels of depression had been successful. Over two thirds met criteria for a current major depressive episode and just over half were on depression pharmacotherapy at baseline. The high average AUDIT score was consistent with the majority of the sample meeting diagnostic criteria for alcohol dependence.

At baseline, 15 participants reported taking pharmacotherapy to manage alcohol craving (7 on acamprosate, 4 on naltrexone, 3 on disulfiram and 1 on acamprosate + disulfiram). This raised concerns that use of such medication might blunt their craving experience, resulting in poor predictive value of their scores. However, a univariate ANOVA revealed that OCDS-O scores were significantly higher in people on anticraving medication versus those who were not \((M = 14.78, SD = 5.83\) and \(M = 8.89, SD = 5.67\) respectively), \(F(1, 204) = 9.27, p = .003\). In view of this result, these participants were included in all analyses. Relationships between baseline variables were not a focus of this research, but are provided in Table 7.3 for reference.
### Table 7.3

*Correlation matrix of baseline variables*

<table>
<thead>
<tr>
<th></th>
<th>Average weekly drinks</th>
<th>Average weekly binges</th>
<th>OCDS-O</th>
<th>BDI-II</th>
<th>Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average weekly drinks</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Average weekly binges</td>
<td>.682***</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>OCDS-O</td>
<td>.394***</td>
<td>.160*</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>BDI-II</td>
<td>.148*</td>
<td>.089</td>
<td>.267***</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Age</td>
<td>-.025</td>
<td>.080</td>
<td>-.192**</td>
<td>-.141*</td>
<td>-</td>
</tr>
<tr>
<td>Gender(^a)</td>
<td>-.275***</td>
<td>-.034</td>
<td>-.060</td>
<td>.113</td>
<td>-.067</td>
</tr>
<tr>
<td>Alcohol abuse Dx(^b,c)</td>
<td>.210**</td>
<td>.083</td>
<td>.380***</td>
<td>.138*</td>
<td>-.183**</td>
</tr>
<tr>
<td>Alcohol dependence Dx(^b)</td>
<td>.213**</td>
<td>.208**</td>
<td>.237**</td>
<td>.174*</td>
<td>-.117</td>
</tr>
<tr>
<td>Depression Dx(^b)</td>
<td>.094</td>
<td>.053</td>
<td>.145*</td>
<td>.166**</td>
<td>-.057</td>
</tr>
<tr>
<td>Antidepressant medication(^b)</td>
<td>-.092</td>
<td>-.018</td>
<td>.065</td>
<td>.031</td>
<td>.081</td>
</tr>
</tbody>
</table>

* \( p < .05 \) ** \( p < .01 \) *** \( p < .001 \); Note. Where one of the variables is dichotomous, the coefficient depicts the Point-Biserial correlation. Cells that cross at two dichotomous variable are therefore excluded; \(^a1 = \text{male}, 2 = \text{female}; \(^b0 = \text{absent}, 1 = \text{present}; \(^c\text{Dx = diagnosis}\)

#### 7.3.1 Data adjustments

Variables were examined for normality and extreme cases to determine if the data were appropriate for parametric testing. Extreme cases were identified using the rule of > 3 standard deviations from the mean with application of an additional rule of also needing to be > 1 standard deviation from the next most extreme case. This additional rule was applied to minimise mislabelling of values as extreme when they may actually represent the tail end of a genuinely skewed distribution. Only one extreme value was identified in average weekly drinks at 18 weeks. This value was trimmed down to the next most extreme value (Howell, 2007a).
Average binge days per week at baseline were heavily left-skewed, with many participants bingeing daily. Due to the violation of the assumption of normality, this variable was recoded into dichotomous categories representing ‘less than half of the week’ and ‘half or more of the week’ for all assessment periods. Other baseline and follow-up variables were suitable for parametric testing.

7.3.2 Missing data

Where item responses were missing within a scale but did not exceed >25% of the total scale, the missing response values were substituted by the mean of the other items for that scale, enabling a total score to be calculated. Missing total scores on outcome measures were substituted with estimations generated using the Expectation-Maximisation method in SPSS Missing Value Analysis.

Baseline variables associated with follow-up attrition were examined. The results of these analyses are provided in Appendix C. There were no differences in baseline levels of depression or craving between follow-up completers and non-completers. Weekly drinking at baseline was significantly higher in people who were missing at 18 weeks compared to people who were not ($M = 74.13$ vs. $M = 58.91$), $F(1, 258) = 4.83$, $p = .029$, but was not different between people missing or not at 12 months. Number of treatment sessions attended was related to attrition during follow-up, but treatment allocation was not, suggesting that being missing at follow-up was related to withdrawing from treatment rather than being allocated to a shorter intervention. Baseline drinking and number of treatment sessions were included in the EM estimation model to adjust for this bias.

Estimations for post-treatment assessments of weekly drinks and binges were imputed using baseline weekly drinks, binges, abstinent days and number of sessions
attended. Although some of those predictors were non-normal, the Expectation-Maximisation algorithm is robust to violations of this assumption (Howell, 2007b). Inclusion of the baseline measure of the predicted variable in the estimation of missing data ensured a conservative estimation of unique variance accounted for by craving and other predictors. Covariance-based statistics using these imputed values can result in underestimation of the associated parameter values, also contributing to conservative estimation. Results of the imputations are presented in Appendix C. All analyses were repeated using the non-imputed data (sensitivity analyses) to determine the robustness of the results. The results of the sensitivity analyses are reported at the end of the results section.

7.3.3 Average weekly drinks

Across the whole sample, there was a significant effect of time on average weekly alcohol consumption with consumption at 18 weeks \( (M = 40.66, SD = 39.92) \) and 12 months \( (M = 39.29, SD = 33.69) \) being significantly reduced from baseline, \( F(2, 259) = 56.01, p < .001 \). There was no difference between consumption at 18 weeks and 12 months, \( F(1, 259) = 0.33, p = .567 \). The majority of participants continued to drink at some level, with only 7.7% abstinent at 18 weeks and 9.6% abstinent at 12 months.

7.3.3.1 Univariate predictors

Table 7.4 summarises the uncontrolled univariate analyses. The OCDS-O was significantly correlated with average weekly drinks at both 18 weeks and 12 months, though the association was stronger at the 18-week assessment. BDI-II score was not significant at either time point, and neither was the interaction between depression and craving. Of the demographic variables, there was a significant main effect of gender at 18 weeks and 12 months, with males consuming on average a higher number of
alcoholic drinks per week than females. There were also main effects of both relationship and welfare status at 12 months, with partnered people drinking significantly less per week than non-partnered people, and people receiving welfare support drinking significantly more than people who were not. Of the treatment contrasts, only Brief versus Long (in favour of the longer interventions) and Integrated versus Single-focused (in favour of the integrated intervention) were significant, and that was only at the 18-week assessment. These treatment effects are discussed in detail in (Baker et al., 2010).

Table 7.4

Results of correlations (r) and ANOVAs (F) of baseline and treatment variables with average weekly drinks at each follow-up time point

<table>
<thead>
<tr>
<th></th>
<th>18 weeks</th>
<th></th>
<th>12 Months</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>r</td>
<td></td>
<td>n</td>
</tr>
<tr>
<td>Age</td>
<td>260</td>
<td>-.059</td>
<td></td>
<td>260</td>
</tr>
<tr>
<td>Education</td>
<td>258</td>
<td>-.001</td>
<td></td>
<td>258</td>
</tr>
<tr>
<td>Baseline average weekly drinks</td>
<td>260</td>
<td>.600***</td>
<td>.527***</td>
<td>260</td>
</tr>
<tr>
<td>OCDS-O score</td>
<td>207</td>
<td>.406***</td>
<td>.247***</td>
<td>207</td>
</tr>
<tr>
<td>BDI-II score</td>
<td>260</td>
<td>.097</td>
<td>.093</td>
<td>260</td>
</tr>
<tr>
<td>Interaction BDI-II and OCDS-O</td>
<td>207</td>
<td>.122</td>
<td>.073</td>
<td>207</td>
</tr>
<tr>
<td></td>
<td>df</td>
<td>F</td>
<td></td>
<td>df</td>
</tr>
<tr>
<td>Gender</td>
<td>1, 258</td>
<td>12.73***</td>
<td>8.33**</td>
<td>1, 258</td>
</tr>
<tr>
<td>Relationship status</td>
<td>1, 258</td>
<td>3.72</td>
<td>6.79*</td>
<td>1, 258</td>
</tr>
<tr>
<td>Welfare status</td>
<td>1, 257</td>
<td>1.97</td>
<td>4.84*</td>
<td>1, 257</td>
</tr>
<tr>
<td>Current depressive episode</td>
<td>1, 255</td>
<td>0.62</td>
<td>0.52</td>
<td>1, 255</td>
</tr>
<tr>
<td>Current antidepressants</td>
<td>1, 255</td>
<td>2.58</td>
<td>2.58</td>
<td>1, 255</td>
</tr>
<tr>
<td>Brief vs. Long</td>
<td>1, 258</td>
<td>6.61*</td>
<td>0.30</td>
<td>1, 258</td>
</tr>
<tr>
<td>Integrated vs. Single-focused</td>
<td>2, 257</td>
<td>3.45*</td>
<td>0.20</td>
<td>2, 257</td>
</tr>
<tr>
<td>Alcohol vs. Depression</td>
<td>2, 257</td>
<td>0.56</td>
<td>0.71</td>
<td>2, 257</td>
</tr>
</tbody>
</table>

* p < .05  ** p < .01  *** p < .001
Study 1

As expected, average weekly drinks at baseline correlated strongly with average weekly drinks at 18 weeks and at 12 months. To control for variance contributed by baseline consumption (Table 7.3 shows that baseline drinking was also significantly correlated with other baseline variables of interest), the analyses were repeated with average weekly drinks at baseline entered as a covariate. The results of those analyses are presented in Table 7.5.

Table 7.5

*Results of partial correlations (r) and ANCOVAs (F) of baseline and treatment variables with average weekly drinks at each follow-up time point, controlling for baseline consumption*

<table>
<thead>
<tr>
<th></th>
<th>18 weeks</th>
<th></th>
<th>12 Months</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>df</td>
<td>r</td>
<td>df</td>
<td>r</td>
</tr>
<tr>
<td>Age</td>
<td>257</td>
<td>-.054</td>
<td>20</td>
<td>.020</td>
</tr>
<tr>
<td>Education</td>
<td>255</td>
<td>.079</td>
<td>29</td>
<td>-.029</td>
</tr>
<tr>
<td>OCDS-O score</td>
<td>204</td>
<td>.221**</td>
<td>8</td>
<td>.028</td>
</tr>
<tr>
<td>BDI-II score</td>
<td>257</td>
<td>.010</td>
<td>18</td>
<td>.018</td>
</tr>
<tr>
<td>Interaction BDI-II and OCDS-O</td>
<td>204</td>
<td>.102</td>
<td>42</td>
<td>.042</td>
</tr>
<tr>
<td></td>
<td>df</td>
<td>F</td>
<td>df</td>
<td>F</td>
</tr>
<tr>
<td>Gender</td>
<td>1, 257</td>
<td>1.16</td>
<td>0.39</td>
<td></td>
</tr>
<tr>
<td>Relationship status</td>
<td>1, 257</td>
<td>0.49</td>
<td>2.70</td>
<td></td>
</tr>
<tr>
<td>Welfare status</td>
<td>1, 256</td>
<td>0.09</td>
<td>1.90</td>
<td></td>
</tr>
<tr>
<td>Current depressive episode</td>
<td>1, 254</td>
<td>0.02</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>Current antidepressants</td>
<td>1, 254</td>
<td>0.80</td>
<td>0.80</td>
<td></td>
</tr>
<tr>
<td>Brief vs. Long</td>
<td>1, 257</td>
<td>8.55**</td>
<td>0.16</td>
<td></td>
</tr>
<tr>
<td>Integrated vs. Single-focused</td>
<td>2, 256</td>
<td>5.88**</td>
<td>0.22</td>
<td></td>
</tr>
<tr>
<td>Alcohol vs. Depression</td>
<td>2, 256</td>
<td>0.01</td>
<td>0.36</td>
<td></td>
</tr>
</tbody>
</table>

* p < .05  ** p < .01  *** p < .001

After controlling for baseline drinking, many of the previously significant associations were no longer significant. The correlation between the OCDS-O and
average weekly drinks at 18 weeks remained significant, although the magnitude of the association was lower. Aside from the OCDS-O, only the two treatment contrasts remained significant after controlling for baseline drinking.

### 7.3.3.2 Craving as a predictor of short-term post-treatment alcohol consumption

The strength of pre-treatment OCDS-O scores to predict average weekly drinking immediately post-treatment was examined using hierarchical linear regression. Baseline consumption was entered at the first step, followed by the two significant treatment contrasts at the second step, with baseline OCDS-O score entered at the final step. The results of the regression are presented in Table 7.6.

**Table 7.6**

*Coefficients of baseline and treatment variables predicting average weekly drinks at 18 weeks at each step of hierarchical linear regression (n = 205)*

<table>
<thead>
<tr>
<th>Step</th>
<th>Variables</th>
<th>$R^2$ change</th>
<th>$F$ (df)</th>
<th>$p$</th>
<th>$B$</th>
<th>Std Err</th>
<th>t</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>BL average drinks</td>
<td><strong>.360</strong></td>
<td>115.56 (1, 205)</td>
<td><strong>.000</strong></td>
<td>0.56</td>
<td>0.05</td>
<td>10.75</td>
<td><strong>.000</strong></td>
</tr>
<tr>
<td></td>
<td>BL avg drinks</td>
<td>0.56</td>
<td>0.05</td>
<td>10.97</td>
<td>0.000</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Brief vs. Long</td>
<td><strong>.028</strong></td>
<td>4.67 (2, 203)</td>
<td><strong>.010</strong></td>
<td>-3.32</td>
<td>1.28</td>
<td>-2.59</td>
<td><strong>.010</strong></td>
</tr>
<tr>
<td></td>
<td>Int vs. Sing Foc</td>
<td>-2.78</td>
<td>1.76</td>
<td>-1.58</td>
<td>0.116</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>BL average drinks</td>
<td><strong>.034</strong></td>
<td>11.87 (1, 202)</td>
<td><strong>.001</strong></td>
<td>-3.37</td>
<td>1.25</td>
<td>-2.29</td>
<td><strong>.008</strong></td>
</tr>
<tr>
<td></td>
<td>Brief vs. Long</td>
<td>-2.68</td>
<td>1.72</td>
<td>-1.56</td>
<td>0.119</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Int vs. Sing-foc</td>
<td>1.39</td>
<td>0.40</td>
<td>3.45</td>
<td><strong>.001</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>OCDS-O</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Addition of the OCDS-O to the model at step 3 resulted in a significant increase in $R^2$ from .389 to .423, meaning craving accounted for a small but still significant 3.4% of the total variance in 18-week drinking, even after other significant baseline and
Study 1

treatment variables had been accounted for. The OCDS-O $B$-weight indicates that for every one point rise in OCDS-O score at baseline, there was a rise of 1.39 drinks per week in average weekly consumption immediately after treatment, when other univariate predictors were held constant. Average weekly drinks at baseline remained a strong and significant predictor when all variables were in the model, as did the Brief versus Long treatment contrast, in favour of the longer interventions.

7.3.4 Frequency of binges

Across the whole sample, significantly fewer participants were bingeing (> 6 x 10g ethanol/occasion for men; > 4 for women) half or more of the week at 18 weeks (36.5%) and 12 months (40.4%) than at baseline (63.5%), $\chi^2(1, N = 260) = 52.90, p < .001$ and $\chi^2(1, N = 260) = 38.68, p < .001$ respectively.

7.3.4.1 Univariate predictors

Table 7.7 summarises the uncontrolled univariate analyses. The OCDS-O was a significant univariate predictor of frequency of both 18-week and 12-month weekly binges. People bingeing half or more of the week at 18 weeks and 12 months had significantly higher OCDS-O scores at baseline than people who were bingeing less than half of the week. The effect was stronger at the 18-week assessment. Consistent with the prediction of weekly drinks, depression and the depression with craving interaction were not significant at either time point. Of demographic variables, gender was significant at the 18-week assessment only, with a greater proportion of females bingeing less than half the week (70.9%) than males (59.1%), and a greater proportion of males bingeing half or more of the week (40.9%) compared to females (29.1%). Relationship status was also significant, but only at 12 months, with a greater proportion of partnered people bingeing less than half the week (69.4%) than non-partnered people
(56.3%), and a greater proportion of non-partnered people bingeing half or more of the week (43.7%) compared to partnered people (30.6%). None of the treatment contrasts were significant in differentiating people based on frequency of binge drinking post-treatment.

Table 7.7

Results of ANOVAs (F) and chi-squares ($\chi^2$) of baseline and treatment variables with frequency of binges at each follow-up time point

<table>
<thead>
<tr>
<th></th>
<th>18 weeks</th>
<th>12 Months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>df</td>
<td>F</td>
</tr>
<tr>
<td>Age</td>
<td>1,258</td>
<td>0.00</td>
</tr>
<tr>
<td>Education</td>
<td>1,256</td>
<td>0.33</td>
</tr>
<tr>
<td>OCDS-O score</td>
<td>1,205</td>
<td>16.09***</td>
</tr>
<tr>
<td>BDI-II score</td>
<td>1,258</td>
<td>0.10</td>
</tr>
<tr>
<td>Interaction BDI-II and OCDS-O</td>
<td>1,205</td>
<td>0.60</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>df, N</th>
<th>$\chi^2$</th>
<th>$\chi^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline binge frequency</td>
<td>1,260</td>
<td>43.68***</td>
<td>37.61***</td>
</tr>
<tr>
<td>Gender</td>
<td>1,260</td>
<td>3.04</td>
<td>2.07</td>
</tr>
<tr>
<td>Relationship status</td>
<td>1,260</td>
<td>2.20</td>
<td>4.31*</td>
</tr>
<tr>
<td>Welfare status</td>
<td>1,259</td>
<td>0.46</td>
<td>1.00</td>
</tr>
<tr>
<td>Current depressive episode</td>
<td>1,257</td>
<td>1.59</td>
<td>0.16</td>
</tr>
<tr>
<td>Current antidepressants</td>
<td>1,257</td>
<td>1.57</td>
<td>1.66</td>
</tr>
<tr>
<td>Brief vs. Long</td>
<td>1,260</td>
<td>1.73</td>
<td>0.77</td>
</tr>
<tr>
<td>Integrated vs. Single-focused</td>
<td>2,260</td>
<td>2.42</td>
<td>2.38</td>
</tr>
<tr>
<td>Alcohol vs. Depression</td>
<td>2,260</td>
<td>2.82</td>
<td>2.53</td>
</tr>
</tbody>
</table>

* p < .05  ** p < .01  *** p < .001

As expected, frequency of binges at baseline significantly differentiated people based on frequency of bingeing at follow-up, with people who were bingeing less than half of the week at baseline being more likely to also be bingeing less than half of the week at 18 weeks (89.5%) and at 12 months (84.2%). People who were bingeing half or
Study 1

more of the week at 18 weeks and 12 months were more likely to have also been
bingeing at that frequency pre-treatment (89.0% and 85.3% respectively). To examine
relationships with the effect of baseline drinking removed, univariate analyses were
repeated to partial out the effects of baseline binge frequency. The OCDS-O main effect
remained significant after controlling for baseline binge frequency. All other effects
dropped out. The results of the controlled analyses are presented in Table 7.8.

Table 7.8

Results of ANCOVAs (F) and likelihood ratio tests ($\chi^2$) of baseline variables with
frequency of binges at each follow-up time point, controlling for baseline binge
frequency

<table>
<thead>
<tr>
<th></th>
<th>18 weeks</th>
<th></th>
<th>12 Months</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>df</td>
<td>F</td>
<td>F</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>1, 257</td>
<td>0.21</td>
<td>0.47</td>
<td></td>
</tr>
<tr>
<td>Education</td>
<td>1, 255</td>
<td>1.36</td>
<td>0.02</td>
<td></td>
</tr>
<tr>
<td>OCDS-O score</td>
<td>1, 204</td>
<td>13.46***</td>
<td>6.34*</td>
<td></td>
</tr>
<tr>
<td>BDI-II score</td>
<td>1, 257</td>
<td>0.02</td>
<td>0.25</td>
<td></td>
</tr>
<tr>
<td>Interaction BDI-II and OCDS-O</td>
<td>1, 204</td>
<td>2.19</td>
<td>3.43</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>df, N</th>
<th>$\chi^2$</th>
<th>$\chi^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>1, 260</td>
<td>3.14</td>
<td>2.04</td>
</tr>
<tr>
<td>Relationship status</td>
<td>1, 260</td>
<td>1.17</td>
<td>3.08</td>
</tr>
<tr>
<td>Welfare status</td>
<td>1, 259</td>
<td>0.40</td>
<td>0.97</td>
</tr>
<tr>
<td>Current depressive episode</td>
<td>1, 257</td>
<td>0.99</td>
<td>0.01</td>
</tr>
<tr>
<td>Current antidepressants</td>
<td>1, 257</td>
<td>1.77</td>
<td>2.01</td>
</tr>
<tr>
<td>Brief vs. Long</td>
<td>1, 260</td>
<td>2.21</td>
<td>0.99</td>
</tr>
<tr>
<td>Integrated vs. Single-focused</td>
<td>2, 260</td>
<td>3.66</td>
<td>2.21</td>
</tr>
<tr>
<td>Alcohol vs. Depression</td>
<td>2, 260</td>
<td>5.27</td>
<td>2.81</td>
</tr>
</tbody>
</table>

* $p < .05$  ** $p < .01$  *** $p < .001$
7.3.4.2  Craving as a predictor of short-term and long-term post-treatment binge frequency

The strength of pre-treatment OCDS-O scores in predicting post-treatment binge frequency was examined using multinomial logistic regressions. Separate regressions were conducted for 18-week and 12-month outcomes, with the OCDS-O and baseline binges entered as predictors. The model fit was significant for both 18 weeks and 12 months, $\chi^2(2, N = 207) = 56.20, p < .001$ and $\chi^2(2, N = 207) = 37.99, p < .001$ respectively, with a Nagelkerke pseudo $R^2$ of .323 at 18 weeks and .227 at 12 months, meaning the set of predictors were accounting for an estimated 32.3% of the variance at 18 weeks and 22.7% of the variance at 12 months. For the 18-week regression, the likelihood ratio tests were significant for baseline binge frequency, $\chi^2(1, N = 207) = 40.85, p < .001$, and the OCDS-O, $\chi^2(1, N = 207) = 12.77, p < .001$. Similar results were found for 12 months, with baseline binges and OCDS-O both emerging as significant in the likelihood ratio tests, $\chi^2(1, N = 207) = 29.58, p < .001$ and $\chi^2(1, N = 207) = 6.18, p = .013$ respectively. The parameter coefficients show that for every one point increase in pre-treatment OCDS-O score, the log odds of bingeing less than half the week (versus half or more) decrease by 0.10 at 18 weeks and by 0.07 at 12 months. The parameter estimates for each regression are displayed in Table 7.9.
Table 7.9

Parameter estimates for baseline variables predicting binge frequency at each follow-up time point (n = 207). Reference category is ‘half or more of the week’

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>Std Error</th>
<th>Wald</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>18-week</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BL binge frequency (ref = less than half of week)</td>
<td><strong>2.32</strong></td>
<td>0.42</td>
<td>29.85</td>
<td>1</td>
<td><strong>.000</strong></td>
</tr>
<tr>
<td>OCDS-O</td>
<td>-0.10</td>
<td>0.03</td>
<td>11.85</td>
<td>1</td>
<td><strong>.001</strong></td>
</tr>
<tr>
<td><strong>12-month</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BL binge frequency (ref = less than half of week)</td>
<td><strong>1.81</strong></td>
<td>0.37</td>
<td>24.63</td>
<td>1</td>
<td><strong>.000</strong></td>
</tr>
<tr>
<td>OCDS-O</td>
<td>-0.07</td>
<td>0.03</td>
<td>5.98</td>
<td>1</td>
<td><strong>.014</strong></td>
</tr>
</tbody>
</table>

7.3.5 OCDS-O items

To investigate if particular items of the OCDS-O were more strongly associated with 18-week average weekly drinks and 18-week and 12-month frequency of binges than others, the regression analyses were repeated entering the individual items of the OCDS-O instead of the total score. Table 7.10 presents the results of the item analysis for 18-week average weekly drinks and Table 7.11 presents the results of the item analysis for 18-week and 12-month binge frequency.
Table 7.10

Regression coefficients and semi-partial correlations for OCDS-O items predicting 18-week average weekly drinks (n = 205)

<table>
<thead>
<tr>
<th>Variables</th>
<th>B</th>
<th>Std Err</th>
<th>t</th>
<th>p</th>
<th>Semi-partial corr.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline average weekly drinks</td>
<td>0.54</td>
<td>0.06</td>
<td>8.86</td>
<td>.000</td>
<td>.458</td>
</tr>
<tr>
<td>Brief vs. Long</td>
<td>-3.89</td>
<td>1.25</td>
<td>-3.12</td>
<td>.002</td>
<td>-.161</td>
</tr>
<tr>
<td>OCDS-O 1 – preoccupation with drinking thoughts</td>
<td>1.17</td>
<td>3.52</td>
<td>0.33</td>
<td>.740</td>
<td>.017</td>
</tr>
<tr>
<td>OCDS-O 2 – frequency of drinking thoughts</td>
<td>-0.45</td>
<td>3.22</td>
<td>-0.14</td>
<td>.888</td>
<td>-.007</td>
</tr>
<tr>
<td>OCDS-O 3 – interference from drinking thoughts</td>
<td>6.94</td>
<td>2.86</td>
<td>2.43</td>
<td>.016</td>
<td>.126</td>
</tr>
<tr>
<td>OCDS-O 4 – distress from drinking thoughts</td>
<td>-1.13</td>
<td>3.53</td>
<td>-0.32</td>
<td>.749</td>
<td>-.017</td>
</tr>
<tr>
<td>OCDS-O 5 – effort to resist thoughts</td>
<td>-4.61</td>
<td>2.87</td>
<td>-1.61</td>
<td>.110</td>
<td>-.083</td>
</tr>
<tr>
<td>OCDS-O 6 – success in diverting thoughts</td>
<td>5.64</td>
<td>2.93</td>
<td>1.92</td>
<td>.056</td>
<td>.099</td>
</tr>
</tbody>
</table>
Study 1

Table 7.11

*Likelihood ratio tests ($\chi^2$) and parameter estimates ($B$) for OCDS-O items predicting post-treatment binge frequency (n = 206). Reference category is ‘half or more of the week’*

<table>
<thead>
<tr>
<th>Predictor</th>
<th>$\chi^2$</th>
<th>df</th>
<th>p</th>
<th>B</th>
<th>Std Error</th>
<th>Wald</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>18 weeks</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BL weekly binges (ref = less than half of week)</td>
<td>49.90</td>
<td>1</td>
<td><strong>.000</strong></td>
<td><strong>2.91</strong></td>
<td>0.51</td>
<td>32.84</td>
<td>1</td>
<td><strong>.000</strong></td>
</tr>
<tr>
<td>OCDS-O 1</td>
<td>0.17</td>
<td>1</td>
<td>.681</td>
<td>0.11</td>
<td>0.28</td>
<td>0.17</td>
<td>1</td>
<td>.682</td>
</tr>
<tr>
<td>OCDS-O 2</td>
<td>0.03</td>
<td>1</td>
<td>.871</td>
<td>0.04</td>
<td>0.25</td>
<td>0.03</td>
<td>1</td>
<td>.871</td>
</tr>
<tr>
<td>OCDS-O 3</td>
<td>10.24</td>
<td>1</td>
<td><strong>.001</strong></td>
<td><strong>-0.68</strong></td>
<td>0.22</td>
<td>9.41</td>
<td>1</td>
<td><strong>.002</strong></td>
</tr>
<tr>
<td>OCDS-O 4</td>
<td>0.25</td>
<td>1</td>
<td>.620</td>
<td>0.14</td>
<td>0.28</td>
<td>0.25</td>
<td>1</td>
<td>.621</td>
</tr>
<tr>
<td>OCDS-O 5</td>
<td>2.19</td>
<td>1</td>
<td>.139</td>
<td>0.35</td>
<td>0.240</td>
<td>2.10</td>
<td>1</td>
<td>.147</td>
</tr>
<tr>
<td>OCDS-O 6</td>
<td>4.24</td>
<td>1</td>
<td><strong>.039</strong></td>
<td><strong>-0.51</strong></td>
<td>0.250</td>
<td>4.14</td>
<td>1</td>
<td><strong>.042</strong></td>
</tr>
<tr>
<td><strong>12 months</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BL weekly binges (ref = less than half of week)</td>
<td>32.48</td>
<td>1</td>
<td><strong>.000</strong></td>
<td><strong>2.16</strong></td>
<td>0.43</td>
<td>23.86</td>
<td>1</td>
<td><strong>.000</strong></td>
</tr>
<tr>
<td>OCDS-O 1</td>
<td>1.75</td>
<td>1</td>
<td>.186</td>
<td>0.36</td>
<td>0.28</td>
<td>1.96</td>
<td>1</td>
<td>.195</td>
</tr>
<tr>
<td>OCDS-O 2</td>
<td>12.21</td>
<td>1</td>
<td><strong>.000</strong></td>
<td><strong>-0.86</strong></td>
<td>0.26</td>
<td>11.74</td>
<td>1</td>
<td><strong>.001</strong></td>
</tr>
<tr>
<td>OCDS-O 3</td>
<td>2.85</td>
<td>1</td>
<td>.091</td>
<td>-0.36</td>
<td>0.21</td>
<td>3.17</td>
<td>1</td>
<td>.095</td>
</tr>
<tr>
<td>OCDS-O 4</td>
<td>0.72</td>
<td>1</td>
<td>.395</td>
<td>0.23</td>
<td>0.27</td>
<td>1.12</td>
<td>1</td>
<td>.397</td>
</tr>
<tr>
<td>OCDS-O 5</td>
<td>7.08</td>
<td>1</td>
<td><strong>.008</strong></td>
<td><strong>0.63</strong></td>
<td>0.25</td>
<td>6.39</td>
<td>1</td>
<td><strong>.011</strong></td>
</tr>
<tr>
<td>OCDS-O 6</td>
<td>2.52</td>
<td>1</td>
<td>.113</td>
<td>-0.38</td>
<td>0.25</td>
<td>3.47</td>
<td>1</td>
<td>.117</td>
</tr>
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</table>

The 18-week results for average weekly drinks and frequency of binges were largely consistent, with item 3 of the OCDS-O (how much ideas, thoughts, impulses, or images related to drinking interfere with social or work functioning) being a significant predictor in the regression models of both. The direction was such that higher scores on this item (reflecting greater degrees of interference) predicted higher levels of
consumption and greater frequency of binges at 18 weeks. In the linear regression of average weekly consumption, item 3 yielded the largest semi-partial correlation of all the OCDS-O items, suggesting it contributes the largest portion of unique variance to the prediction of 18-week average weekly drinks at 1.6%. Item 6 (success in stopping or diverting these thoughts) was also significant in the prediction of 18-week frequency of binges, but fell just short of significance in the regression of average weekly drinks ($p = .056$). As for item 3, the direction was such that higher scores on item 6 (reflecting greater difficulty diverting thoughts) predicted greater drinking at 18 weeks.

A different picture emerged in the prediction of frequency of binges at 12 months where items 2 (frequency of drinking thoughts) and 5 (effort to resist thoughts) were significant predictors, but with opposite effects. Higher scores on item 2 (reflecting greater frequency of thoughts) were associated with decreased odds of bingeing less than half of the week at 12 months. That is, greater frequency of alcohol thoughts at baseline was associated with greater frequency of bingeing at 12 months. Higher scores on item 5 (reflecting no effort to resist thoughts) were associated with increased odds of bingeing less than half of the week. That is, the less effort invested in resisting drinking thoughts at baseline, the lower the frequency of binges at 12 months.

### 7.3.6 Sensitivity analyses

To determine the robustness of the results, all analyses were repeated using the non-imputed data (complete cases). Results were highly consistent with those of the imputed data set. Only minor differences emerged. In the complete case analysis, the depression with craving interaction had a small but significant correlation with 18-week average weekly drinking, $r = .153, p = .042$ (cf. Table 7.4), however, this dropped out in the subsequent controlled analysis, $r = .089, p = .241$ (cf. Table 7.5). Relationship
Study 1

status was significant in the controlled univariate prediction of 12-month frequency of
binges, $F(1, 189) = 6.03, p = .015$ (cf. Table 7.8).

The overall $R^2$ of the 18-week average weekly drinking regression was slightly
lower in the complete case analysis at .253, which was still significant, $F(1, 174) =
59.04, p < .001$ (cf. Table 7.6). A similar reduction in pseudo $R^2$ was observed in the
multinomial logistic regressions, which were .305 at 18 weeks and .169 at 12 months.
The higher variance explained in the imputed analyses is likely due to the use of
baseline drinking variables in the estimation imputation. The regression coefficients of
the OCDS-O all remained significant, with only minor differences in coefficient
magnitudes. In the 18-week average weekly drinks regression, $B = 1.64, t(173) = 3.41, p
= .001$ (cf. Table 7.6). In the 18-week regression of frequency of binges, $B = -0.11, \chi^2(1, N = 176) = 12.43, p < .001$ (cf. Table 7.9). In the 12-month regression of frequency of
binges, $B = -0.08, \chi^2(1, N = 155) = 6.15, p = .013$ (cf. Table 7.9).

Two key differences emerged in the item regression results. Item 6 became
significant in the prediction of 18-week average weekly drinking, $B = 6.97, t(173) =
2.00 p = .047$ (cf. Table 7.10) but was not significant in the prediction of 18-week
binges, $\chi^2(1, N = 174) = 2.24 p = .134$ (cf. Table 7.11).

7.4 Discussion

This study sought to examine whether pre-treatment craving that is experienced
in the context of comorbid depressed mood would be correlated with post-treatment
alcohol consumption in heavy drinkers, based on the rationale that the combination of
elevated craving with high levels of depressed mood may be particularly potent and
associated with poor prognosis. This study also examined whether an interaction
between mood and craving influenced outcomes following treatment.
Greater craving on the OCDS-O significantly predicted greater weekly alcohol consumption at 18 weeks, and greater frequency of binges at both 18 weeks and 12 months. Even with baseline consumption and other significant treatment variables controlled for in the model, craving remained a significant predictor. The directions of the associations were such that higher scores on the OCDS-O pre-treatment were associated with higher levels of alcohol consumption at 18 weeks, and greater frequency of binges at both 18 weeks and 12 months.

Although craving was a significant predictor of frequency of binges at 12 months, it did not predict weekly consumption at that time point. These results indicate that baseline levels of craving and overall consumption became less closely associated over time. This is not surprising given the strong proximal associations often observed between craving and substance use (Allen et al., 2008; Oslin et al., 2009; Shiffman et al., 2002), supporting the notion that craving functions as a state rather than a trait (Kavanagh et al., 2013), and therefore relates most strongly to substance use measured in the same context. From this perspective, it is remarkable that a significant prediction was observed in the current study at all.

The association between pre-treatment craving and frequency of binges throughout follow-up could represent an ongoing struggle to exercise control over alcohol use. People with high craving before treatment could be more vulnerable to the control-inhibiting effects of alcohol, making it more difficult for them to maintain controlled drinking (Field, Wiers, Christiansen, Fillmore, & Verster, 2010; Weafer & Fillmore, 2008). This interpretation is consistent with the elaborated intrusion theory of craving (Kavanagh et al., 2005), which argues that craving is heightened by cognitive elaboration that captures working memory resources, reducing the capacity of the individual to engage appropriate coping responses. If the person is also prone to loss of
inhibitory control at low levels of alcohol consumption, this would further exacerbate difficulties in forming and implementing effective coping plans. This idea is also supported by the results of the item analyses where higher scores on items 2 and 5, reflecting high frequency of drinking thoughts and greater effort invested in resisting those thoughts, were predictive of a higher frequency of binges at 12 months.

A similar yet slightly different mechanism of influence seems to be at work for 18-week outcomes, reflected in the divergent item results for this time point compared to 12 months. Item 3 was a significant predictor in the regressions of both average weekly drinks and frequency of binges. Item 6 was also significant in predicting frequency of binges and fell just short of significance in predicting average weekly drinks (though it was significant in the complete case analysis). These items respectively relate to how much thoughts of drinking interfere with functioning and success in stopping or diverting alcohol related thoughts. These results are also consistent with the notion that craving depletes cognitive resources thereby reducing capacity to effectively engage coping strategies. People who experience high levels of interference from drinking thoughts pre-treatment, and who have little success in diverting such thoughts, appear to consume more drinks and binge more frequently after treatment. Contributing further to this vulnerability, it has been observed that deprivation enhances the cognitive interference experienced during exposure to substance cues (Sayette & Hufford, 1994). People who already experience a high degree of cognitive interference from drinking thoughts may experience further depletion when faced with alcohol cues, increasing their vulnerability to lapses.

The finding that different items were significant in the predictions of 18-week and 12-month outcomes, coupled with the consistency of the item results across the two 18-week regressions, suggests that different cognitive aspects of craving have
differential influences on drinking behaviour over time following treatment. These results seem to suggest that experiencing a high degree of pre-treatment cognitive interference from drinking thoughts may be an indicator for poor short-term outcomes following treatment, while having a high pre-treatment frequency of drinking thoughts and investing great effort to resist those thoughts may be prognostic of ongoing, long-term impaired control over alcohol use.

The finding that pre-treatment craving was predictive of alcohol consumption after treatment is consistent with results of several other previous studies (e.g., Bottlender & Soyka, 2004; Garbutt et al., 2009; Ray et al., 2006; Rohsenow et al., 1994). However, the results differed from those of Farren and McElroy (2010), who did not find that craving differentiated alcoholics with unipolar or bipolar depression who relapsed or remained abstinent at 6 months. As discussed in the introduction, there were a number of important differences between that study and the current one, which may explain these discrepant results.

Firstly, Farren and McElroy (2010) measured craving after participants had already undergone detoxification and had been abstinent from alcohol for at least a week. Craving has been documented to decline steadily following initiation of abstinence (Cutler, 2005; Galloway et al., 2010; Kranzler et al., 1999; Zorick et al., 2010), and to be moderated by factors such as perceived availability (Wertz & Sayette, 2001; Wilson, Sayette, Delgado, & Fiez, 2005). Craving measured after detoxification and while still in an inpatient setting is unlikely to be representative of craving that is experienced in the real world when faced with real cues, incentives and opportunity. Such a measure is therefore unlikely to hold value in predicting outcomes following treatment, when individuals return to their usual environments. Craving in the current study was measured prior to treatment commencement after a decision to address
Study 1

alcohol use, but prior to receipt of support. This is a context that closely resembles the one subsequently experienced during recurrences of craving that occur in their natural environment after treatment is completed. A measure of craving at that time was likely to be a better indication of the strength and pattern of urges the individual would experience post-treatment, and therefore may explain why craving was related to outcomes in the current study.

The second important difference between Farren and McElroy’s (2010) study and the current one was the type of outcome measured. Farren and McElroy studied the ability of craving to predict relapse at a 6-month follow-up. Relapse was not clearly defined, but presumably people were coded according to whether they had experienced a lapse to drinking during the 6 months following baseline assessment, or if they had remained entirely abstinent from alcohol. Such a definition of outcome is limited, because not all treatment seekers view abstinence as their goal, even if this is prescribed in treatment, and such a definition of outcome fails to detect partial success. The results of the present study suggest that craving may perform better in predicting how much people are likely to be drinking following treatment, rather than simply whether they will be drinking or not. This conclusion is consistent with the work of Rohsenow et al. (2007), who found that cocaine craving measured before treatment was predictive of how much cocaine was used post-treatment, measured as amount of money spent on cocaine, but was not predictive of whether they used or not, measured as number of days of use.

Results of the current study supported the hypothesis that pre-treatment craving would be a significant predictor of later alcohol consumption, although the effect was small and the results across outcomes mixed. Neither depression nor the depression with craving interaction were related to either outcome measure at either time point,
indicating that the relationship between pre-treatment craving and post-treatment drinking was not related to the level of comorbid depressed mood.

The lack of a relationship between baseline depression and later drinking was inconsistent with other studies that have shown depression to be predictive of later alcohol use (Bottlender & Soyka, 2005; Conner et al., 2009; Gamble et al., 2010; Kodl et al., 2008). However, it was consistent with the results of Farren and McElroy (2010) who also found that baseline BDI scores were not related to relapse at 3 or 6 months, and with other studies that also found that pre-treatment depression was not related to post-treatment drinking outcomes (Oslin et al., 2009; Zhou et al., 2009). Even though associations between depression and alcohol use tend to be stronger when depression is measured using a continuous scale (Conner et al., 2009), the presence of substantial levels of depressed mood across the present sample may have resulted in insufficient spread in depression scores to enable detection of associations. While the presence of a depressive disorder was not a requirement of the present study, over half of the sample met criteria for a current major depressive episode (70%), and inclusion cut-off of 17 on the BDI-II was still slightly higher than the cut-off for mild depression. The BDI-II scores may have been condensed too much in the top end of the scale. This restriction in the range of scores may have precluded detection of interaction effects between depression and craving. Inclusion of people without significant depressive mood may be necessary to detect effects of depression on outcomes, whether through direct effects or through an interaction with craving.

It must also be noted that half of the sample (52%) were on antidepressant pharmacotherapy at entry to the study. It is possible that subsequent recovery by some of these participants may have reduced the ability of baseline depression levels to be predictive. However, there was no significant association between baseline
Study 1

antidepressant status and concurrent BDI-II scores, $F(1, 255) = 0.24, p = .624$, and participants had typically been on antidepressants for some time ($M = 90$ weeks), making it unlikely that this factor explained the results.

The lack of relationship between pre-treatment depression and post-treatment drinking outcomes leads to the question of whether depression was related to drinking at all in this sample. Table 7.3 shows that at baseline, BDI-II scores were modestly but significantly correlated with average weekly drinks, but not with average weekly binges. The current study did therefore detect a small but significant association between higher levels of depressed mood and greater weekly alcohol consumption. It is possible that levels of depressed mood are most strongly associated with proximal drinking behaviour, and indeed, many of the studies that have shown links between negative affect and substance use have been laboratory-based and have shown links between induced negative affect and subsequent substance seeking or use for very proximal time points, such as the same day or week (Fucito & Juliano, 2009; Perkins et al., 2008). Depressive mood tends to be substantially higher at entry to treatment than at subsequent assessments (Brown & Schuckit, 1988), and post-treatment measures may provide better estimates of subsequent depressed mood (Curran, Flynn, Kirchner, & Booth, 2000; Witkiewitz & Villarreal, 2009). Furthermore, as with craving, day-to-day fluctuations in mood are common, and measures of mood on a specific day are likely to offer more powerful predictions of outcomes than a measure taken at a distant time point.

Further research is clearly needed to elucidate the conditions and circumstances under which depression, craving and drinking are related. Even within the current study, craving accounted for a very modest $3.4\%$ of unique variance in 18-week alcohol consumption, suggesting it is just one of a number of variables that are influencing post-
treatment drinking. Research suggests complex associations between affect, craving and substance use that are moderated or mediated by myriad factors such as gender (Boykoff et al., 2010), treatment seeking status (Thomas, Randall, Brady, See, & Drobes, 2011) and the nature of the treatment intervention administered (Witkiewitz & Bowen, 2010). How these relationships and influences can be best modeled to enhance prediction of treatment outcomes requires further investigation.

In addition to the restricted range of depression scores and the absence of a group of participants with low levels of depressed mood, a further limitation of the current study is the restriction of craving measurement to only conscious, cognitive craving. Craving may also be experienced as a physiological reaction to the presence of certain cues (Elash, Tiffany, & Vrana, 1995; Rohsenow et al., 1992) or a sudden intrusive image that is so fleeting as to barely register on a conscious level, but may still sometimes be sufficient to trigger a behavioral response (Kavanagh et al., 2005). The obsessive items of the OCDS tap into intrusive alcohol thoughts experienced on a fully conscious level. However, craving is not only experienced in this way. The results of this study need to be replicated using other craving measures, to determine if the relationships observed are specific to intrusive cognitive aspects of craving, or if they relate to the craving construct more generally.

7.5 Conclusion

This study demonstrated that craving was a significant predictor of post-treatment drinking outcomes in a sample of drinkers with comorbid depressed mood, although no evidence of moderating or mediating influences by depression on craving were detected. This suggests the prediction of outcome by craving was not related to the level of depressed mood at baseline, although there were limitations within the current
Study 1

study that may have precluded detection of such an association. Examination of the individual OCDS-O items that contributed most variance to the predictions suggested that different cognitive aspects of craving may have differential impacts on alcohol use following treatment, and that the influence of these cognitive experiences may vary over time. Pre-treatment depression was not related to post-treatment drinking outcomes at all, suggesting that depression at baseline may not be the best indicator of later drinking, and that more proximal measures of depression may be needed to clarify the true extent that mood influences relapse risk. Further research is needed to elucidate the nature of the relationship between depression and craving, and how the two may interact to influence outcomes following treatment.
CHAPTER 8 STUDY 2: THE INFLUENCE OF MOOD-CRAVING RELATIONSHIPS ON ALCOHOL TREATMENT OUTCOMES

8.1 Introduction

The relationship and meaning of pre-treatment craving to post-treatment substance use outcomes remains unclear despite extensive investigation, as outlined in Chapter 4. Some studies find support for pre-treatment craving being predictive of post-treatment alcohol use (e.g., Bottlender & Soyka, 2004; Ray et al., 2006), while others find it is not (e.g., Kiefer et al., 2005; Kranzler et al., 1999; Walton et al., 2003). These discrepant findings suggest there may be other factors, as yet unidentified, that are influencing the relationship between craving and substance use, impacting on its performance as an outcome predictor. As discussed in Chapter 5, negative affect is one such possible factor, based on its demonstrated associations with higher levels of substance use and relapse (Gamble et al., 2010; Kodl et al., 2008; Witkiewitz & Villarroel, 2009) and greater intensity of self-reported craving (Chaplin, Hong, Bergquist, & Sinha, 2008; Cleveland & Harris, 2010). The presence of elevated negative mood may augment craving intensity, strengthening its relationship with subsequent substance use.

Farren and McElroy (2010) did not find support for this notion when they examined predictors of relapse in alcohol abusers with comorbid mood disorders. In their study, baseline craving was not related to relapse in the 6 months following discharge from a 4-week inpatient treatment. Study 1 was designed to overcome some of the limitations of Farren and McElroy’s (2010) study, such as the timing of baseline
Study 2

assessments and the drinking outcome predicted. With these adaptations, Study 1 did find relationships between baseline craving and post-treatment alcohol consumption, although the lack of apparent moderating or mediating relationships by depression on craving suggests this relationship was not associated with the level of comorbid depressed mood. Study 1 was limited however by a lack of spread in depression scores resulting from the absence of drinkers with low levels of depression. Although the study did not require presence of a diagnostic mood disorder, it did require at least a mild to moderate level of depression severity as measured using the BDI-II (Beck et al., 1996). This may have hampered detection of an interaction effect between depression and craving in influencing drinking outcomes following treatment.

A second limitation of Study 1 that needs to be addressed to further explore the relationships between mood, craving and substance use outcomes is the measure of craving used. Study 1 used only the OCDS as the measure of craving. The OCDS was developed based on observations that drinking thoughts and behaviours appear to resemble the obsessions and compulsions of obsessive compulsive disorder, namely, recurrent and persistent thoughts, inability to resist thoughts, compulsive drive to consume alcohol and loss of control of that drive (Modell, Glaser, Cyr, et al., 1992; Modell, Glaser, Mountz, & Schmaltz, 1992). The OCDS was developed by adapting items of the Yale-Brown Obsessive Compulsive Scale (Goodman, Price, Rasmussen, & Mazure, 1989) to apply to desires to consume alcohol. Early validation studies did demonstrate that scores on the adapted scale were significantly positively correlated with subjective ratings of craving in alcoholic samples (Modell, Glaser, Cyr, et al., 1992) and were successful in distinguishing between alcoholic and non-alcoholic samples (Modell, Glaser, Mountz, et al., 1992). Nevertheless, the scale was developed based on clinical observation rather than craving or addiction theory and its authors
acknowledge that it only seems “intuitively probable, that this obsessive-compulsive dimension is associated with the concept of craving” (Anton et al., 1995, p. 95). It is also acknowledged that the OCDS likely measures only one possible dimension of craving (Anton, 2000).

As discussed in Chapter 3, craving is a complex multidimensional construct which can be comprised of cognitive (Kavanagh et al., 2005; Nosen & Woody, 2009; Tiffany, 1990), emotional (Baker et al., 2004; Monti et al., 2000) and physiological (Heinz et al., 2008; Rohsenow et al., 1994) processes. While the obsessive subscale of the OCDS (OCDS-O) captures particular aspects of the cognitive experience of craving, such as frequency of drinking thoughts and interference and distress caused by such thoughts, there are other cognitive processes it does not capture.

For example, elaborated intrusion theory (Kavanagh et al., 2005) distinguishes between automatic associative processes and higher level elaborative processes, both of which are experienced on a conscious cognitive level. Intrusive thoughts are triggered by learned associations to internal and external cues and under certain conditions can lead to deliberate and effortful cognitive elaboration, where information relevant to the target stimuli is actively sought and manipulated in working memory. Elaborated intrusion theory differs from other craving theories in its emphasis on sensory imagery as a key type of desire cognition, arguing that it is central to the craving experience. Intrusive thoughts may be experienced in the form of fleeting images, but the greatest impact of imagery is during the elaborative process where sensory images are constructed and played out in vivid detail. Whether this leads to seeking and acquisition of the target depends on a number of other factors, such as competing desires, skills and availability. The frequency and strength of these associative intrusions and imagery-based elaborations may be useful indicators of vulnerability to relapse.
Study 2

The Alcohol Craving Experience Questionnaire (ACE; Statham et al., 2011) was developed to measure the key constructs of elaborated intrusion theory, namely, cognitive intrusions and sensory imagery. Twenty-nine items were developed assessing the frequency of these two dimensions in the past week (ACE-Frequency; “over the last week how often did you...”) and their intensity at the time during the past week when craving was at its strongest (ACE-Strength; “think about the time you most wanted an alcoholic drink...”). Exploratory factor analysis yielded three factors: Intensity (e.g., how strong was the urge to drink, how hard was it to think about anything else); Imagery (e.g., how vividly did you picture alcohol or drinking, how vividly did you imagine what it would taste like); and Intrusion (e.g., how hard were you trying not to think about alcohol, how intrusive were the thoughts). Based on confirmatory factor analysis, the authors removed 2 items from the ACE-Strength scale (ACE-S) and 5 items from the ACE-Frequency scale (ACE-F) to achieve a stable factor structure, leaving 22 items in the final scale.

A subset of data from the present study contributed to examination of the reliability, validity and factor structure of the scale (Statham et al., 2011). Internal consistencies of the three factors of the ACE-S and ACE-F were good to excellent, ranging from .80 to .94. Concurrent validity was supported through significant associations between total AUDIT scores and the Intrusion factor of the ACE-S and all three factors of the ACE-F. Additionally, all ACE subscales correlated significantly with subscales of the OCDS. Both the ACE-S and ACE-F discriminated between a University student sample and a clinical sample of treatment seeking alcohol abusers, with the ACE-S accounting for 37% of the between group variance and the ACE-F accounting for 50.2%. The performance of the ACE as a potential predictive tool for post-treatment alcohol use has not yet been investigated.
The present study sought to further investigate the influence of pre-treatment depression and craving on post-treatment alcohol use by addressing the limitations of Study 1 in two important ways: i) inclusion of non-depressed drinkers to capture a range of depression severity, enabling more robust examination of the effects of high versus low depression, and; ii) inclusion of an additional measure of craving (the ACE) to enable interpretation of findings in relation to the construct of craving more generally as well as identification of particular facets of craving that may be more influential for outcomes than others.

It was hypothesised that, consistent with the results of Study 1, craving would be a significant predictor of post-treatment alcohol consumption and frequency of alcohol binges. It was further hypothesised that greater spread of depression scores would result in significant prediction of outcomes by depression as well as a significant interaction effect between depression and craving, such that high levels of pre-treatment depression combined with high levels of craving would predict greater alcohol consumption and more frequent bingeing post-treatment.

8.2 Method

8.2.1 Participants

Data for the present study were collected during the course of a randomised controlled trial investigating a new intervention for alcohol craving (Kavanagh, Connor, Sitharthan, & Ross, unpublished). Participants were recruited through newspaper adverts and articles, health practitioner referral, and referral from another research study. The study was conducted at two Australian locations: Brisbane, Queensland and Sydney, New South Wales. The study held ethical clearance with The University of
Study 2

Queensland, Queensland University of Technology and the Sydney West Area Health Service.

A total of 242 participants (61% male) were recruited into the study over 2 years. A CONSORT flow diagram summarising recruitment and retention is depicted in Figure 8.1. Descriptive demographics of the group are provided in Table 8.1.
Figure 8.1. CONSORT diagram showing flow of participants through Study 2.

- Screened for eligibility = 451 (initial phone screen)
- Ineligible = 67
  - Not interested = 28
  - Seeking information only = 41
  - Screening incomplete = 1
- Eligible = 314
- Assessed for eligibility = 255 (face-to-face assessment interview)
- Ineligible = 13
- Randomised = 242
- Withdrawn = 59
- Allocated to CBT* = 81
  - Received any intervention = 76 (94%)
  - Received no intervention = 5
  - Withdrawn = 5
  - Discontinued intervention = 29 (36%)
  - Follow-up completed
    - 3 month = 67 (83%)
    - 6 month = 59 (73%)
    - 9 month = 62 (77%)
    - 12 month = 55 (68%)
  - Analysed = 81
- Allocated to CBT + CARM† = 80
  - Received any intervention = 77 (96%)
  - Received no intervention = 3
  - Withdrawn = 3
  - Discontinued intervention = 28 (35%)
  - Follow-up completed
    - 3 month = 72 (90%)
    - 6 month = 64 (80%)
    - 9 month = 54 (68%)
    - 12 month = 54 (68%)
  - Analysed = 80
- Allocated to Brief = 81
  - Received any intervention = 77 (95%)
  - Received no intervention = 4
  - Withdrawn = 4
  - Discontinued intervention = 0
  - Follow-up completed
    - 3 month = 65 (80%)
    - 6 month = 58 (72%)
    - 9 month = 47 (58%)
    - 12 month = 46 (57%)
  - Analysed = 81

* Cognitive-Behavioural Therapy
† Craving for Alcohol: Reduction and Management
Study 2

Table 8.1

Demographics characteristics of the full sample (N = 242)

<table>
<thead>
<tr>
<th>Demographic Characteristics</th>
<th>M (SD, range) or n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age (years)</td>
<td>48.82 (10.90, 21–76)</td>
</tr>
<tr>
<td>Male</td>
<td>147 (60.7%)</td>
</tr>
<tr>
<td>Married/partnered</td>
<td>166 (68.6%)</td>
</tr>
<tr>
<td>Mean years of education</td>
<td>13.70 (3.24, 7–27)</td>
</tr>
<tr>
<td>Post-high school qualification</td>
<td>153 (63.2%)</td>
</tr>
</tbody>
</table>

Inclusion criteria were: (i) aged 18 years or above; (ii) hazardous alcohol consumption in at least 1 of the 2 weeks prior to baseline (> 28 x 10g ethanol drinks per week for men; > 14 for women); (iii) presence of either alcohol abuse or alcohol dependence. Exclusion criteria were: (i) concurrent psychological or pharmacological treatment for alcohol use; (ii) self-reported diagnosis of concurrent psychotic or bipolar disorder; (iii) concurrent illicit substance use disorder; (iv) lack of English fluency; (v) inability to travel to treatment sessions; (vi) self-reported or interviewer-detected presence of intellectual disability or organic brain disease; (vii) other member of household already participating. Comorbid disorders aside from psychosis and bipolar were not excluded. Concurrent treatment for comorbid disorders was also not excluded.

8.2.2 Measures

Only a subset of the collected data was used for the present study. These are the measures described below and provided in Appendix F. Measures were administered in interviews by postgraduate-trained psychologists, with the exception of the OCDS-O which was provided in written form to be completed by the participant at home.
8.2.2.1 Alcohol

Consumption

The Timeline Follow-Back (TLFB; Sobell & Sobell, 1992) was used to record daily alcohol intake in the preceding two weeks to obtain a point estimate of alcohol use at the time of assessment. The psychometric properties of the TLFB were reported in Chapter 7. For consistency between the two studies, the same two indices of consumption that were derived for Study 1 were also derived for the present study; average weekly drinks and average number of binge days per week (> 6 per day for men and > 4 per day for women).

Craving

The OCDS (Anton et al., 1995) and Alcohol Craving Experience (ACE) (Statham et al., 2011) questionnaires were used to measure alcohol craving. As per Study 1 and for the reasons outlined in Chapter 7, only the obsessive subscale of the OCDS (OCDS-O) was administered. The psychometric properties of this scale were reported in Chapter 7. Internal consistency (Cronbach’s alpha) in the present study was .87.

The ACE questionnaire (Statham et al., 2011) is a new addition to the field of craving assessment and was developed to measure aspects of craving described by elaborated intrusion theory (Kavanagh et al., 2005). The ACE measures three dimensions of the cognitive experience of craving: Intensity (e.g., “how strong was the urge to drink?”); Intrusion (e.g., “how hard were you trying not to think about alcohol?”); and Imagery (e.g., “how vividly did you imagine what it would taste like?”). As reported in the introduction, the ACE assesses these experiences for the time when...
Study 2

craving was strongest (ACE-Strength; ACE-S) and for frequency over the last week (ACE-Frequency; ACE-F).

Participants in the present study were administered the 35-item development version of the ACE as shown in Appendix D. Scores for the analyses of this study however, were calculated based on the reduced scale described by Statham et al. (2011) (Appendix E), as this is the version that has demonstrated sound psychometric properties, as detailed in the introduction. Internal consistencies of the Intensity, Intrusion and Imagery scales of the ACE-S and ACE-F in the present sample were good, ranging between .80 and .91.

Severity of dependence

As per Study 1, a 6-month version of the Alcohol Use Disorders Identification Test (AUDIT; Saunders et al., 1993) was administered as a measure of dependence severity for the purpose of describing the sample. It was not used as an outcome or predictor in the present study. Abundant research has demonstrated the AUDIT has internal consistency, test-retest reliability and construct and criterion validity (see Reinert & Allen, 2007 for a recent review).

Alcohol use disorder diagnosis

The Structured Clinical Interview for DSM-IV (SCID; First et al., 1995) was used to assess for presence of alcohol use disorder. In the current study this variable was not assessed as an outcome or a predictor.
8.2.2 Depression

Severity of depression

Severity of depression symptomatology was assessed using the Depression, Anxiety, Stress Scale – 21-item version (DASS21; Lovibond & Lovibond, 1995b). The DASS21 is comprised of 21 items that measure how often particular symptoms occurred over the last week. It is composed of 3 subscales, each with 7 items, that measure symptoms of depression (e.g., dysphoria, hopelessness, anhedonia, inertia), anxiety (e.g., autonomic arousal, skeletal muscle effects) and stress (e.g., difficulty relaxing, nervous arousal). The 21-item version of the DASS was derived from an original 42-item version, so derived scores are doubled for interpretation. Only the depression subscale of the DASS21 (DASS-D) was used in this study.

The DASS-D has been found to have good internal consistency, with Cronbach’s alphas ranging between .88 and .94 (Antony, Bieling, Cox, Enns, & Swinson, 1998; Henry & Crawford, 2005; Tully, Zajac, & Venning, 2009). Concurrent and convergent validity have been supported through significant correlations with the Beck Depression Inventory (Antony et al., 1998), the Mental Health Questionnaire (Ng et al., 2007) and the positive and negative affect scales of the Positive and Negative Affect Schedule (Henry & Crawford, 2005). Internal consistency of the DASS-D in the current study was .92.

8.2.3 Interventions

Following assessment, all participants attended an initial brief intervention treatment session (120 minutes). This session included assessment feedback, motivation enhancement, goal setting, building self-efficacy and basic planning. Random allocation occurred at the end of this session, with the therapist opening the allocation envelope in
the presence of the participant. The therapist was blind to treatment allocation until this point.

Randomisations, stratified by gender, were generated before commencement of the study in permutations of 6 and 9. The allocations were generated and sealed in envelopes by a researcher not connected to the study. Envelopes were assigned to participants immediately after the completion of the baseline assessment (the next envelope in the sequence was taken and placed in the participant’s file) so that allocations were assigned consecutively based on order of entry into the research.

8.2.3.1 Brief intervention

This comprised the initial session that all participants attended. Those assigned to this intervention did not receive any further face-to-face treatment after this initial session. To maintain engagement with the study, these participants were phoned fortnightly for 10 weeks for a brief assessment of their alcohol consumption. These calls were made by the therapist who conducted the initial session, but were kept as brief as possible (5-10 minutes) and did not involve any further counselling or advice. Where counselling or advice could not be avoided, this extra contact was recorded and counted as a treatment dose.

8.2.3.2 Cognitive-behavioural therapy (CBT) intervention

This comprised an additional 10 hours of therapy that covered risk situation planning, problem solving, craving education, cognitive restructuring, drink refusal, pleasant non-alcohol activities, future goals, relapse prevention and relaxation. Sessions 2, 3, and 5 were face-to-face for 120 minutes each. Sessions 4 and 6 were half an hour each and were conducted over the phone, ideally at a time of day when the participant was most at risk for drinking. This phone call guided the participant through application
Chapter 8

of therapeutic techniques in vivo. Sessions 7-9 were face-to-face for 60 minutes each. Sessions 1-7 were held weekly, with the exception of session 4 which occurred during the week in between sessions 3 and 5. Sessions 8 and 9 were fortnightly.

8.2.3.3 CBT + Craving for Alcohol: Reduction and Management (CARM)

This comprised an additional 10 hours of therapy that covered all the content of the CBT intervention, substituting mindfulness meditation for relaxation, and included additional modules in sessions 2, 3 and 5 on craving reduction and management. These additional modules represent a new approach to craving intervention based on elaborated intrusion theory (Kavanagh et al., 2005). They included education about the nature and origins of cravings according to the theory, and guided the participant through various strategies to reduce and manage cravings, primarily based on disruption and prevention of cognitive elaboration.

As per the CBT treatment, sessions 2, 3 and 5 were face-to-face for 120 minutes each. Sessions 4 and 6 were half an hour phone sessions, ideally at a time of day when the participant tended to experience their strongest urge to drink. This phone call focused on guiding the participant through application of the CARM strategies in vivo, as well as application of CBT principles. Sessions 7-9 were face-to-face for 60 minutes each.

8.2.4 Procedure

Participants self-referred by phone and were screened initially for age, English language fluency, weekly alcohol consumption, concurrent treatment, psychosis or bipolar disorder, injected drug use in last month, and whether other members of their household were already involved in the research. If initial eligibility criteria were met, potential participants were invited to attend a face-to-face assessment interview during
Study 2

which eligibility based on the remaining inclusion criteria was determined. Prior to the face-to-face assessment, participants were posted the study information sheet and consent form and some paper based self-report assessments to complete and bring to their interview.

If participants were assessed to be eligible, they were invited to enter the study and the first treatment session was scheduled. This was the point at which participants were considered to have entered the study. Their treatment allocation was assigned at this point, but not opened until the end of the first treatment session, as described above.

Follow-up assessments were conducted by blind assessors 3, 6, 9 and 12 months after baseline. Follow-up assessments comprised a face-to-face interview in addition to some postal questionnaires. There were occasions when participants would complete one of the elements of the follow-up assessment, but then not complete the other, resulting in partial missing data for some cases. Participants were reimbursed with a $20 gift voucher for each completed assessment.

8.2.5 Statistical analysis

To enable direct comparison of results between Study 1 and Study 2, the same set of statistical procedures were adopted. Data were analysed with SPSS Statistics 19 using an intention-to-treat approach. Missing post-treatment data were substituted with estimations generated using the Expectation-Maximisation (EM) method in SPSS Missing Value Analysis. Baseline variables related to missing post-treatment assessment were examined using analysis of variance (ANOVA) and Pearson chi-squares, with significant variables included as predictors in the EM estimation. As per Study 1, sensitivity analyses using the non-imputed data were run for comparison and are reported at the end of the results section.
Treatment effects were examined by including the following orthogonal contrasts: (i) Brief versus Long treatment; and (ii) CBT versus CBT + CARM. Interaction effects between depression and craving were examined in a similar fashion as for Study 1, but with a categorical version of the depression variable (see data adjustments section in results for explanation of why depression was coded categorically). The interaction term was created by multiplying mean-centered craving scores by a dummy coded version of the depression variable which represented no depression versus at least moderate depression. A further interaction term between craving and a contrast of CARM versus Brief or CBT was created to assess the influence of having received a craving-focused intervention on the relationship between pre-treatment craving scores and post-treatment drinking outcomes.

Consistent with Study 1, two key outcome variables were examined in the analyses: average weekly consumption and average weekly binges (> 6 x 10g ethanol/occasion for men; > 4 for women). Uncontrolled relationships between baseline and treatment variables and outcome variables were examined first using Pearson bivariate correlations, ANOVA or Pearson chi-squares, followed by controlled analyses to remove the effects of the outcome being predicted (partial correlations, analysis of covariance; ANCOVA, or logistic regressions). Linear regressions (for continuous outcomes) or multinomial logistic regressions (for non-continuous outcomes) were conducted only where either craving or the interaction of depression with craving were significant in the controlled analyses. To maintain consistency with Study 1, only short-term post-treatment (3-month) and long-term post-treatment (12-month) outcomes were examined. For all analyses, an alpha cut-off of .05 was adopted.
8.3 Results

Baseline characteristics (see Table 8.2) show the study was successful in recruiting the targeted population of high risk, dependent drinkers. Participants had a mean weekly alcohol intake well in excess of government guidelines with a high incidence of alcohol dependence. Frequency of abstinence days was very low, with a mean of less than 1 day a week over the whole sample. These characteristics were consistent with those of the Study 1 sample. A history of depression was reported in almost half of the sample (100 of depression, 2 of bipolar disorder), though only a small proportion reported currently experiencing an episode. The mean DASS-D score for the sample was in the mild severity range, with a good spread of high and low scores. Correlations between baseline variables are provided in Table 8.3.
Table 8.2

**Baseline characteristics of the full sample (N = 242)**

<table>
<thead>
<tr>
<th>Continuous variables</th>
<th>$M$</th>
<th>(SD, range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TLFB mean drinks per week</td>
<td>63.13</td>
<td>(30.56, 7.5-209)</td>
</tr>
<tr>
<td>TLFB mean binge days per week</td>
<td>5.12</td>
<td>(1.90, 0-7)</td>
</tr>
<tr>
<td>TLFB mean abstinent days per week</td>
<td>0.78</td>
<td>(1.16, 0-6)</td>
</tr>
<tr>
<td>Mean DASS-D score</td>
<td>10.60</td>
<td>(9.43, 0-42)</td>
</tr>
<tr>
<td>Mean AUDIT total ($n = 241$)</td>
<td>24.50</td>
<td>(5.32, 12-38)</td>
</tr>
<tr>
<td>Mean OCDS-O total ($n = 239$)</td>
<td>6.89</td>
<td>(4.61, 0-23)</td>
</tr>
<tr>
<td>Mean ACE-S Intensity score ($n = 229$)</td>
<td>24.47</td>
<td>(8.78, 0-40)</td>
</tr>
<tr>
<td>Mean ACE-S Intrusion score ($n = 229$)</td>
<td>6.69</td>
<td>(5.88, 0-20)</td>
</tr>
<tr>
<td>Mean ACE-S Imagery score ($n = 229$)</td>
<td>19.16</td>
<td>(13.12, 0-50)</td>
</tr>
<tr>
<td>Mean ACE-F Intensity score ($n = 227$)</td>
<td>18.89</td>
<td>(8.42, 2-40)</td>
</tr>
<tr>
<td>Mean ACE-F Intrusion score ($n = 227$)</td>
<td>5.97</td>
<td>(5.60, 0-20)</td>
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<tr>
<td>Mean ACE-F Imagery score ($n = 227$)</td>
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<td>(11.63, 0-50)</td>
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<table>
<thead>
<tr>
<th>Categorical Variables</th>
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<th>(%)</th>
</tr>
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<tr>
<td>Current alcohol abuse diagnosis</td>
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<td>(55.4%)</td>
</tr>
<tr>
<td>Current alcohol dependence diagnosis</td>
<td>228/242</td>
<td>(94.2%)</td>
</tr>
<tr>
<td>History of mood disorder</td>
<td>102/242</td>
<td>(42.1%)</td>
</tr>
<tr>
<td>Current depressive episode</td>
<td>15/242</td>
<td>(6.2%)</td>
</tr>
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Table 8.3

Correlation matrix of baseline variables

<table>
<thead>
<tr>
<th></th>
<th>Average weekly drinks</th>
<th>Average weekly binges</th>
<th>OCDS-O</th>
<th>ACE-S Intensity</th>
<th>ACE-S Intrusion</th>
<th>ACE-S Imagery</th>
<th>ACE-F Intensity</th>
<th>ACE-F Intrusion</th>
<th>ACE-F Imagery</th>
<th>DASS-D</th>
<th>Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average weekly drinks</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Average weekly binges</td>
<td>.609***</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>OCDS-O</td>
<td>.170**</td>
<td>.088</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>ACE-S Intensity</td>
<td>.010</td>
<td>.0013</td>
<td>.359***</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>ACE-S Intrusion</td>
<td>.020</td>
<td>-.074</td>
<td>.508***</td>
<td>.438***</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
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<td>-</td>
</tr>
<tr>
<td>ACE-S Imagery</td>
<td>-.071</td>
<td>-.109</td>
<td>.427***</td>
<td>.484***</td>
<td>.493***</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>ACE-F Intensity</td>
<td>.160*</td>
<td>.104</td>
<td>.447***</td>
<td>.613***</td>
<td>.496***</td>
<td>.501***</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>ACE-F Intrusion</td>
<td>-.024</td>
<td>-.101</td>
<td>.493***</td>
<td>.432***</td>
<td>.761***</td>
<td>.475***</td>
<td>.522***</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>ACE-F Imagery</td>
<td>.050</td>
<td>-.083</td>
<td>.445***</td>
<td>.428***</td>
<td>.530***</td>
<td>.776***</td>
<td>.658***</td>
<td>.592***</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>DASS-D</td>
<td>.167**</td>
<td>.027</td>
<td>.469***</td>
<td>.203***</td>
<td>.373***</td>
<td>.250***</td>
<td>.363***</td>
<td>.366***</td>
<td>.327***</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Age</td>
<td>-.071</td>
<td>.045</td>
<td>-.249***</td>
<td>-.191***</td>
<td>-.147*</td>
<td>-.235***</td>
<td>-.198*</td>
<td>-.246***</td>
<td>-.250***</td>
<td>-.072</td>
<td>-</td>
</tr>
<tr>
<td>Gender*</td>
<td>-.399***</td>
<td>-.119</td>
<td>.062</td>
<td>.098</td>
<td>.156*</td>
<td>.032</td>
<td>.046</td>
<td>.159*</td>
<td>.020</td>
<td>.014</td>
<td>-.064</td>
</tr>
<tr>
<td>Alcohol abuse Dx</td>
<td>.091</td>
<td>.047</td>
<td>.172**</td>
<td>.227**</td>
<td>.269***</td>
<td>.173**</td>
<td>.276***</td>
<td>.299***</td>
<td>.239***</td>
<td>.274</td>
<td>-.050</td>
</tr>
<tr>
<td>Alcohol dependence Dx</td>
<td>.034</td>
<td>-.077</td>
<td>.078</td>
<td>.025</td>
<td>.103</td>
<td>.038</td>
<td>.008</td>
<td>.073</td>
<td>.045</td>
<td>.076</td>
<td>-.125</td>
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<td>History of depression</td>
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<td>.009</td>
<td>.037</td>
<td>-.032</td>
<td>.102</td>
<td>-.083</td>
<td>-.003</td>
<td>.116</td>
<td>-.014</td>
<td>.289***</td>
<td>.116</td>
</tr>
</tbody>
</table>

* p < .05  ** p < .01  *** p < .001; Note. Where one of the variables is dichotomous, the coefficient depicts the Point-Biserial correlation. Cells that cross at two dichotomous variables are therefore excluded; a1 = male, 2 = female; b0 = absent, 1 = present; cDx = diagnosis.
8.3.1 Data adjustments

Variables were examined for normality and extreme cases, to determine if the data were appropriate for parametric testing. Extreme cases were identified and handled as per Study 1, using the rule of > 3 standard deviations from the mean and > 1 standard deviation from the next most extreme case. Adjustments for extremity were required for 5 cases. One was a baseline variable (education in years), 2 were from 3-month follow-up (average weekly drinks and OCDS-O score) and 2 were from 12-month follow-up (average weekly drinks and OCDS-O score). These outliers were trimmed back to the next most extreme value (Howell, 2007a), reducing the degree of skew and allowing for their inclusion in parametric testing.

The same problem with the distribution of the average weekly binges variable that was observed in Study 1 was also observed in the current study, with the variable being heavily skewed to the left (i.e., most cases bingeing 7 days per week). Consequently, this variable was recoded into the same dichotomous categories that were used in Study 1; ‘less than half of the week’ and ‘half or more of the week’. DASS-D scores were also skewed, and were coded as < 14 (low depression) or ≥ 14 (high depression). A score of 14 on DASS-D is the cut off for moderate depression (Lovibond & Lovibond, 1995b), which was considered a suitable cut off to enable examination of the core research question of whether there is an effect of high levels of depression on the relationship between craving and post-treatment drinking. Proportion of treatment attended also required dichotomous coding due to being heavily left skewed. This variable was recoded to ‘100% attended’ and ‘< 100% attended’, due to the majority of participants having attending all treatment sessions (68%).

Strong correlations were observed between matching subscales of the ACE-S and ACE-F. This suggested a high degree of shared variance between the subscales of
the two scales. Subscale scores were therefore averaged across the two scales to create single scores for Intensity, Intrusion and Imagery.

8.3.2 Missing data

Missing data were handled using the same procedures as in Study 1. Where item responses within a scale did not exceed > 25% of the total scale, missing response values were substituted with the mean of the other items for the scale, enabling calculation of a total scale score. Missing outcome values were substituted with estimations generated using the Expectation-Maximisation method in SPSS Missing Value Analysis.

As per Study 1, baseline variables associated with follow-up non-completion were examined. The results of these analyses are provided in Appendix G. There were no differences in baseline demographics or levels of alcohol consumption, craving or depression for people missing or not at either 3-month or 12-month assessment. Similar to Study 1, number of treatment sessions attended was significantly associated with non-completion at both time points, but treatment allocation was not, suggesting attrition was due to having withdrawn from treatment, rather than the particular type of treatment received. Predictor variables for the estimation of missing values included the baseline measures of drinking (average weekly drinks, binges and abstinent days) and number of treatment sessions attended. Other baseline variables were not included as predictors in the estimation as this would have exaggerated their relationship with the outcome measures in later prediction analyses. Sensitivity analyses using the non-imputed complete case data were run for comparison. The results of the imputations are presented in Appendix G.
8.3.3 Average weekly drinks

There was an overall significant effect of time on average weekly drinking, with consumption at 3 months ($M = 28.43, SD = 28.08$) and 12 months ($M = 31.12, SD = 24.06$) being significantly reduced from baseline, $F(2, 240) = 253.36$, $p < .001$. Abstinence rates at 3 months were slightly higher in this sample than in Study 1, with 12.4% abstinent compared to 7.7% in Study 1. Rates at 12 months were comparable, with 8.3% of the current sample abstinent at 12 months, compared to 9.6% at 12 months in Study 1.

8.3.3.1 Univariate predictors

Results of the univariate analyses are summarised in Table 8.4. Of the craving measures, only the OCDS-O correlated significantly with average weekly alcohol consumption, and that was at both the 3-month and 12-month assessments. Depression classification (low versus high) was associated with significant differences in average weekly drinking at 3 and 12 months, with those with higher depression drinking more at post-treatment assessment ($M = 33.87, SD = 37.73$ at 3 months; $M = 35.82, SD = 30.82$ at 12 months) than those with low depression ($M = 25.43, SD = 20.48$ at 3 months; $M = 28.54, SD = 18.98$ at 12 months). The interaction between depression and OCDS-O scores was significantly correlated with drinking at 12 months. The interaction between depression and ACE-Intensity scores was significant at 12 months but not at 3 months, while the interaction between depression and ACE-Intrusion scores was significant at both time points. Of the demographic variables, only education and gender were significant univariate predictors of drinking at both 3 and 12 months, with higher education levels being associated with lower levels of post-treatment drinking and males drinking more on average than females. Treatment-related variables were
Study 2

significant at 3 months, but not at 12 months, with people who received Brief treatment tending to drink more at 3 months than people who received a longer treatment, and people who received CBT treatment tending to drink slightly less at 3 months than those who received CBT + CARM treatment. Interactions between craving and having received the CBT + CARM intervention or not were also not significant.
Table 8.4

Results of correlations (r) and ANOVAs (F) of baseline and treatment variables with average weekly drinks at each follow-up time point

<table>
<thead>
<tr>
<th></th>
<th>3 Months</th>
<th></th>
<th>12 Months</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>r</td>
<td>n</td>
<td>r</td>
</tr>
<tr>
<td>Age</td>
<td>242</td>
<td>-.112</td>
<td>242</td>
<td>-.060</td>
</tr>
<tr>
<td>Education years</td>
<td>242</td>
<td>-.139*</td>
<td>242</td>
<td>-1.87**</td>
</tr>
<tr>
<td>Baseline average weekly drinks</td>
<td>242</td>
<td>.493***</td>
<td>242</td>
<td>.551***</td>
</tr>
<tr>
<td>OCDS-O score</td>
<td>239</td>
<td>.134*</td>
<td>239</td>
<td>.179**</td>
</tr>
<tr>
<td>ACE-Intensity score</td>
<td>229</td>
<td>.082</td>
<td>229</td>
<td>.084</td>
</tr>
<tr>
<td>ACE-Intrusion score</td>
<td>229</td>
<td>.086</td>
<td>229</td>
<td>.043</td>
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<td>ACE-Imagery score</td>
<td>229</td>
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<td>229</td>
<td>.011</td>
</tr>
<tr>
<td>Depression x OCDS-O</td>
<td>239</td>
<td>.098</td>
<td>239</td>
<td>.260***</td>
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<tr>
<td>Depression x ACE-Intensity</td>
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<td>.075</td>
<td>229</td>
<td>.137*</td>
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<tr>
<td>Depression x ACE-Intrusion</td>
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<td>.145*</td>
<td>229</td>
<td>.201**</td>
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<td>.091</td>
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<tr>
<td>CARM vs. Brief/CBT x OCDS-O</td>
<td>239</td>
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<td>CARM vs. Brief/CBT x ACE total</td>
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<td>227</td>
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<td>13.58***</td>
<td>1, 240</td>
<td>10.47**</td>
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<td>0.10</td>
<td>1, 239</td>
<td>0.64</td>
</tr>
<tr>
<td>Mental health diagnosis</td>
<td>1, 238</td>
<td>1.99</td>
<td>1, 238</td>
<td>0.61</td>
</tr>
<tr>
<td>Current mental health episode</td>
<td>1, 237</td>
<td>0.12</td>
<td>1, 237</td>
<td>0.07</td>
</tr>
<tr>
<td>DASS-D</td>
<td>1, 240</td>
<td>5.10*</td>
<td>1, 240</td>
<td>5.16*</td>
</tr>
<tr>
<td>Proportion of treatment attended</td>
<td>1, 240</td>
<td>1.01</td>
<td>1, 240</td>
<td>2.57</td>
</tr>
<tr>
<td>Brief vs. Long</td>
<td>1, 240</td>
<td>6.36*</td>
<td>1, 240</td>
<td>0.05</td>
</tr>
<tr>
<td>CBT vs. CARM</td>
<td>2, 239</td>
<td>3.33*</td>
<td>2, 239</td>
<td>0.03</td>
</tr>
</tbody>
</table>

* p < .05  ** p < .01  *** p < .001

As expected, average weekly drinks at baseline was strongly correlated with average weekly drinks at both post-treatment assessment points and with other baseline variables. As such, these relationships were explored again, controlling for the effect of
Study 2

baseline drinking, to examine whether these variables were independently related to outcomes. The results of the controlled analyses are presented in Table 8.5.

Table 8.5

Results of partial correlations (r) and ANCOVAs (F) of baseline and treatment variables with average weekly drinks at each follow-up time point, controlling for baseline consumption

<table>
<thead>
<tr>
<th></th>
<th>3 Months</th>
<th></th>
<th>12 Months</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>df</td>
<td>r</td>
<td>df</td>
<td>r</td>
</tr>
<tr>
<td>Age</td>
<td>239</td>
<td>-.089</td>
<td>239</td>
<td>-.025</td>
</tr>
<tr>
<td>Education years</td>
<td>239</td>
<td>-.079</td>
<td>236</td>
<td>-.130*</td>
</tr>
<tr>
<td>OCDS-O score</td>
<td>236</td>
<td>.058</td>
<td>236</td>
<td>.104</td>
</tr>
<tr>
<td>ACE-Intensity score</td>
<td>226</td>
<td>.044</td>
<td>226</td>
<td>.042</td>
</tr>
<tr>
<td>ACE-Intrusion score</td>
<td>226</td>
<td>.099</td>
<td>226</td>
<td>.053</td>
</tr>
<tr>
<td>ACE-Imagery score</td>
<td>226</td>
<td>.095</td>
<td>226</td>
<td>.025</td>
</tr>
<tr>
<td>Depression x OCDS-O</td>
<td>236</td>
<td>.008</td>
<td>236</td>
<td>.193**</td>
</tr>
<tr>
<td>Depression x ACE-Intensity</td>
<td>226</td>
<td>.028</td>
<td>226</td>
<td>.095</td>
</tr>
<tr>
<td>Depression x ACE-Intrusion</td>
<td>226</td>
<td>.118</td>
<td>226</td>
<td>.184**</td>
</tr>
<tr>
<td>Depression x ACE-Imagery</td>
<td>226</td>
<td>.027</td>
<td>226</td>
<td>.109</td>
</tr>
<tr>
<td>CARM vs. Brief/CBT x OCDS-O</td>
<td>236</td>
<td>-.049</td>
<td>236</td>
<td>-.113</td>
</tr>
<tr>
<td>CARM vs. Brief/CBT x ACE total</td>
<td>224</td>
<td>-.050</td>
<td>224</td>
<td>-.084</td>
</tr>
<tr>
<td>Gender</td>
<td>2, 239</td>
<td>0.45</td>
<td>2, 239</td>
<td>0.10</td>
</tr>
<tr>
<td>Relationship status</td>
<td>2, 239</td>
<td>0.03</td>
<td>2, 239</td>
<td>0.00</td>
</tr>
<tr>
<td>Employment status</td>
<td>2, 238</td>
<td>0.09</td>
<td>2, 238</td>
<td>1.07</td>
</tr>
<tr>
<td>Mental health diagnosis</td>
<td>2, 237</td>
<td>4.11*</td>
<td>2, 237</td>
<td>0.23</td>
</tr>
<tr>
<td>Current mental health episode</td>
<td>2, 236</td>
<td>0.24</td>
<td>2, 236</td>
<td>0.04</td>
</tr>
<tr>
<td>DASS-D</td>
<td>2, 239</td>
<td>2.00</td>
<td>2, 239</td>
<td>1.80</td>
</tr>
<tr>
<td>Proportion of treatment attended</td>
<td>2, 239</td>
<td>0.26</td>
<td>2, 239</td>
<td>1.35</td>
</tr>
<tr>
<td>Brief vs. Long</td>
<td>2, 239</td>
<td>7.66**</td>
<td>2, 239</td>
<td>0.17</td>
</tr>
<tr>
<td>CBT vs. CARM</td>
<td>3, 238</td>
<td>4.04*</td>
<td>3, 238</td>
<td>0.09</td>
</tr>
</tbody>
</table>

*p < .05  **p < .01  ***p < .001
Few of the baseline variables were significant univariate predictors of post-treatment average weekly alcohol intake after baseline drinking had been controlled for. Education remained significant only at 12 months and past mental health diagnosis emerged as significant at 3 months, with people with a past mental health diagnosis drinking more at the 3-month post-treatment assessment than people without a history of mental health disorder. The relationship between DASS-D classification and post-treatment drinking dropped out, suggesting the significant effect observed in the uncontrolled analyses was a reflection of people with high depression having a tendency to drink more in general, rather than an effect of level of depression being predictive of outcomes. Correlations between craving and post-treatment drinking also dropped out. However, the interaction terms of depression with the OCDS-O and depression with the ACE-Intrusion remained significant at 12 months.

### 8.3.3.2 Interaction testing

**Interaction between depression and the OCDS-O**

To determine if there was a significant interaction effect between baseline depression level and OCDS-O scores in predicting 12-month average weekly drinking, the interaction term was entered into a linear regression along with the mean-centered OCDS-O scores and the binary DASS-D variable (Aiken & West, 1991). Baseline average weekly drinks were entered at step 1, education, depression and craving at step 2 and the interaction separately at step 3. This enabled determination of the unique variance explained by the interaction. The regression results are presented in Table 8.6.
Study 2

Table 8.6

*Coefficients of baseline variables, with craving measured using the OCDS-O, predicting average weekly drinks at 12 months (N = 239)*

<table>
<thead>
<tr>
<th>Step</th>
<th>Variables</th>
<th>R² change</th>
<th>F (df)</th>
<th>p</th>
<th>B</th>
<th>Std Err</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>BL average weekly drinks</td>
<td><strong>.304</strong></td>
<td>103.41</td>
<td><strong>.000</strong></td>
<td><strong>0.43</strong></td>
<td>0.04</td>
<td>10.17</td>
<td><strong>.000</strong></td>
</tr>
<tr>
<td></td>
<td>BL avg drinks</td>
<td></td>
<td></td>
<td></td>
<td><strong>0.41</strong></td>
<td>0.04</td>
<td>9.41</td>
<td><strong>.000</strong></td>
</tr>
<tr>
<td></td>
<td>Education</td>
<td><strong>.020</strong></td>
<td>2.37</td>
<td><strong>.076</strong></td>
<td>-0.81</td>
<td>0.42</td>
<td>-1.92</td>
<td>.056</td>
</tr>
<tr>
<td></td>
<td>DASS-D</td>
<td></td>
<td></td>
<td></td>
<td>1.44</td>
<td>2.98</td>
<td>0.48</td>
<td>.630</td>
</tr>
<tr>
<td></td>
<td>zOCDS-O</td>
<td></td>
<td></td>
<td></td>
<td>1.90</td>
<td>1.42</td>
<td>1.34</td>
<td>.183</td>
</tr>
<tr>
<td>2</td>
<td>BL avg drinks</td>
<td><strong>.40</strong></td>
<td></td>
<td></td>
<td><strong>0.40</strong></td>
<td>0.04</td>
<td>9.28</td>
<td><strong>.000</strong></td>
</tr>
<tr>
<td></td>
<td>Education</td>
<td></td>
<td></td>
<td></td>
<td>-0.79</td>
<td>0.41</td>
<td>-1.90</td>
<td>.059</td>
</tr>
<tr>
<td></td>
<td>DASS-D</td>
<td><strong>.018</strong></td>
<td>6.36</td>
<td><strong>.012</strong></td>
<td>0.46</td>
<td>2.97</td>
<td>0.16</td>
<td>.877</td>
</tr>
<tr>
<td></td>
<td>zOCDS-O</td>
<td></td>
<td></td>
<td></td>
<td>-1.26</td>
<td>1.88</td>
<td>-0.67</td>
<td>.503</td>
</tr>
<tr>
<td></td>
<td>Dep x OCDS-O</td>
<td><strong>7.07</strong></td>
<td></td>
<td></td>
<td>7.07</td>
<td>2.80</td>
<td>2.52</td>
<td><strong>.012</strong></td>
</tr>
</tbody>
</table>

The addition of the depression with craving interaction at step 3 resulted in a significant increase in R² of .018, meaning the interaction accounted for a small but significant 1.8% of unique variance in 12-month drinking when the effects of baseline drinking and education were held constant. The craving coefficient represents the effect of baseline craving on 12-month drinking at low levels of depression. For people with low baseline depression on the DASS-D, a 1 standard deviation increase in OCDS-O score was associated with drinking 1.26 fewer drinks at 12 months. The sum of the craving and interaction coefficients indicates the effect of craving at high levels of depression. For people with high depression on the DASS-D at baseline, a 1 standard deviation increase in baseline OCDS-O score was associated with drinking 5.81 more drinks at 12 months.
The significant regression coefficient of the interaction represents a significant difference in the slopes of craving predicting 12-month average weekly drinking between people coded as having low depression at baseline, and people coded as having high depression. Figure 8.2 depicts these regression slopes.

![Figure 8.2](image_url)  
*Figure 8.2. Predicted values of 12-month average weekly drinks for people with low or high depression, at low craving (1SD below the standardised mean of OCDS-O) and high craving (1SD above).*

The regression slope of craving predicting 12-month drinking in people with high depression was significantly different from zero, $t(233) = 2.78$, $p = .006$. The slope of the low depression group was not significantly different from zero $t(233) = -0.67$, $p = .504$, indicating no effect of low depression on the relationship between baseline craving and 12-month average weekly drinks.

*Interaction between depression and ACE-Intrusion*

The significance of the interaction between depression and ACE-Intrusion in predicting 12-month drinking was also examined in a linear regression. Similarly to the OCDS-O regression, baseline average weekly drinks was entered at step 1, followed by education, depression and craving at step 2, and the interaction at step 3. The regression results are presented in Table 8.7.
Study 2

Table 8.7

Coefficients of baseline variables, with craving measured using the ACE-Intrusions, predicting average weekly drinks at 12 months ($N = 229$)

<table>
<thead>
<tr>
<th>Step</th>
<th>Variables</th>
<th>R² change</th>
<th>$F$ (df)</th>
<th>$p$</th>
<th>B</th>
<th>Std Err</th>
<th>$t$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>BL average weekly drinks</td>
<td><strong>.292</strong></td>
<td><strong>93.46</strong> (1, 227)</td>
<td><strong>.000</strong></td>
<td><strong>0.41</strong></td>
<td>0.04</td>
<td>9.67</td>
<td><strong>.000</strong></td>
</tr>
<tr>
<td></td>
<td>BL avg drinks</td>
<td><strong>0.39</strong></td>
<td>0.04</td>
<td>9.27</td>
<td>.000</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Education</td>
<td><strong>.018</strong></td>
<td><strong>1.90</strong> (3, 224)</td>
<td>.130</td>
<td>-0.79</td>
<td>0.40</td>
<td>-1.95</td>
<td>.052</td>
</tr>
<tr>
<td></td>
<td>Dep x ACE-Intrusion</td>
<td><strong>0.66</strong></td>
<td>1.33</td>
<td>0.50</td>
<td>.620</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>BL avg drinks</td>
<td><strong>0.38</strong></td>
<td>0.04</td>
<td>9.13</td>
<td>.000</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Education</td>
<td>-0.66</td>
<td>0.40</td>
<td>-1.64</td>
<td>.103</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>DASS-D</td>
<td><strong>.021</strong></td>
<td><strong>7.06</strong> (1, 223)</td>
<td><strong>.008</strong></td>
<td>1.35</td>
<td>2.83</td>
<td>0.48</td>
<td>.634</td>
</tr>
<tr>
<td></td>
<td>Dep x ACE-Intrusion</td>
<td><strong>7.25</strong></td>
<td>2.73</td>
<td>2.66</td>
<td><strong>.008</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The addition of the interaction term at step 3 resulted in a significant increase in $R^2$ of .021, meaning the interaction accounted for an estimated 2.1% of unique variance in 12-month drinking with the effects of baseline drinking and education held constant.

A 1 standard deviation increase in ACE-Intrusion score at baseline is associated with drinking 2.11 fewer drinks at 12 months for people with low depression, and drinking 5.14 more drinks for people with high depression. The significant interaction coefficient indicates differences in the slopes of craving predicting 12-month average weekly drinking in people with low versus high depression. Figure 8.3 depicts the regression slopes.
Consistent with the OCDS-O interaction results, the regression slope of the low depression group was not significantly different from zero, $t(223) = -1.26, p = .209$, while the slope of the high depression group was, $t(223) = 2.41, p = .008$.

**Comparative prediction by OCDS-O and ACE-Intrusion**

To investigate if one of the craving measures contributed unique variance over and above that of the other, the two linear regressions predicting 12-month average weekly drinks were repeated with an additional fourth step. In step 4, the interaction of the alternative measure was included along with its mean centred craving score (e.g., the depression with ACE-Intrusion interaction was entered in a step following the OCDS-O interaction). The regression coefficients resulting from the fourth step are presented in Table 8.8.
Study 2

Table 8.8

Coefficients of baseline variables at step 4 of the linear regressions predicting 12-month average weekly drinks from the interactions of depression with the OCDS-O and ACE-Intrusions (N = 226)

<table>
<thead>
<tr>
<th>Variables</th>
<th>B</th>
<th>Std Err</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline average weekly drinks</td>
<td>0.38</td>
<td>0.04</td>
<td>8.79</td>
<td>.000</td>
</tr>
<tr>
<td>Education</td>
<td>-0.71</td>
<td>0.41</td>
<td>-1.75</td>
<td>.082</td>
</tr>
<tr>
<td>DASS-D</td>
<td>0.51</td>
<td>2.95</td>
<td>0.17</td>
<td>.864</td>
</tr>
<tr>
<td>zACE-Intrusion</td>
<td>-2.01</td>
<td>1.91</td>
<td>-1.05</td>
<td>.295</td>
</tr>
<tr>
<td>Depression x ACE-Intrude</td>
<td>5.38</td>
<td>3.13</td>
<td>1.72</td>
<td>.088</td>
</tr>
<tr>
<td>zOCDS-O</td>
<td>-0.14</td>
<td>2.05</td>
<td>-0.07</td>
<td>.947</td>
</tr>
<tr>
<td>Depression x OCDS-O</td>
<td>3.62</td>
<td>3.11</td>
<td>1.17</td>
<td>.245</td>
</tr>
</tbody>
</table>

Including the interaction between depression and ACE-Intrusion in the prediction model, the interaction between depression and the OCDS-O was no longer significant in predicting alcohol consumption at 12 months. Similarly, with the interaction of depression with OCDS-O in the prediction model, the interaction of depression with the ACE-Intrusion had a significance level of \( p < .10 \). Neither interaction added a substantial amount of unique variance over and above that provided by the other interaction. Addition of the OCDS-O interaction to that of the ACE-Intrusion added only 0.7% unique variance to the multivariate prediction, \( F(2, 218) = 1.09, p = .338 \), and addition of the ACE-Intrusion interaction to that of the OCDS-O added only 0.9% unique variance, \( F(2, 218) = 1.47, p = .232 \).

8.3.4 Frequency of binges

Consistent with the time effects observed for average weekly drinking, there was a significant overall reduction in the numbers of people bingeing half or more the week
at 3 months (24.4%) and 12 months (19.4%) compared to baseline (80.6%), \( \chi^2(1, N = 242) = 12.81, p < .001 \) and \( \chi^2(1, N = 242) = 4.44, p = .035 \) respectively.

8.3.4.1 Univariate predictors

Results of the univariate analyses are summarised in Table 8.9. The OCDS-O was a significant univariate predictor of 12-month frequency of binges, as was the interaction between depression and OCDS-O. ACE-Intensity scores were associated with 3-month binges only. Depression classification on the DASS-D did not significantly differentiate people based on frequency of binges at follow-up. Of demographic variables, education was a significant univariate predictor of 12-month binges, with people bingeing less than half the week having a higher number of years of education. History of mental health disorder was also a univariate predictor of 3-month binges, with people reporting a lifetime diagnosis of a mental health disorder being more likely to be bingeing half or more of the week at 12 months than people reporting no lifetime diagnosis. Neither of the treatment variables were significant, although the interaction between baseline OCDS-O and type of treatment was significant at 3 months, and the interaction between baseline ACE and treatment was significant at 12 months.
Table 8.9

Results of ANOVAs (F) and chi-squares ($\chi^2$) of baseline and treatment variables with frequency of binges at each follow-up time point

<table>
<thead>
<tr>
<th></th>
<th>3 Months</th>
<th>12 Months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>df</td>
<td>F</td>
</tr>
<tr>
<td>Age</td>
<td>240</td>
<td>3.25</td>
</tr>
<tr>
<td>Education years</td>
<td>240</td>
<td>1.52</td>
</tr>
<tr>
<td>OCDS-O score</td>
<td>237</td>
<td>3.34</td>
</tr>
<tr>
<td>ACE-Intensity score</td>
<td>227</td>
<td>4.38*</td>
</tr>
<tr>
<td>ACE-Intrusion score</td>
<td>227</td>
<td>2.66</td>
</tr>
<tr>
<td>ACE-Imagery score</td>
<td>227</td>
<td>3.64</td>
</tr>
<tr>
<td>Depression x OCDS-O</td>
<td>239</td>
<td>1.57</td>
</tr>
<tr>
<td>Depression x ACE-Intensity</td>
<td>227</td>
<td>0.45</td>
</tr>
<tr>
<td>Depression x ACE-Intrusion</td>
<td>227</td>
<td>2.19</td>
</tr>
<tr>
<td>Depression x ACE-Imagery</td>
<td>227</td>
<td>0.96</td>
</tr>
<tr>
<td>CARM vs. Brief/CBT x OCDS-O</td>
<td>237</td>
<td>3.99*</td>
</tr>
<tr>
<td>CARM vs. Brief/CBT x ACE total</td>
<td>225</td>
<td>0.92</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>df, N</th>
<th>$\chi^2$</th>
<th>$\chi^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline frequency of binges</td>
<td>1, 242</td>
<td>12.81***</td>
<td>4.44*</td>
</tr>
<tr>
<td>Gender</td>
<td>1, 242</td>
<td>2.50</td>
<td>1.32</td>
</tr>
<tr>
<td>Relationship status</td>
<td>1, 242</td>
<td>0.64</td>
<td>0.19</td>
</tr>
<tr>
<td>Employment status</td>
<td>1, 241</td>
<td>0.01</td>
<td>0.04</td>
</tr>
<tr>
<td>Mental health diagnosis</td>
<td>1, 240</td>
<td>3.89*</td>
<td>0.66</td>
</tr>
<tr>
<td>Current mental health episode</td>
<td>1, 239</td>
<td>0.17</td>
<td>0.91</td>
</tr>
<tr>
<td>DASS-D</td>
<td>1, 242</td>
<td>0.90</td>
<td>3.24</td>
</tr>
<tr>
<td>Proportion of treatment attended</td>
<td>1, 242</td>
<td>0.11</td>
<td>0.09</td>
</tr>
<tr>
<td>Brief vs. Long</td>
<td>1, 242</td>
<td>2.78</td>
<td>0.89</td>
</tr>
<tr>
<td>CBT vs. CARM</td>
<td>2, 242</td>
<td>2.94</td>
<td>1.01</td>
</tr>
</tbody>
</table>

* $p < .05$  ** $p < .01$  *** $p < .001$

As expected, frequency of binges at baseline was strongly associated with frequency of binges following treatment. People who were binging less than half of the week at baseline were more likely to also bingeing less than half of the week at 3
months (95.7%) and at 12 months (91.5%). The univariate analyses were therefore repeated, controlling for baseline binge frequency. The results of the controlled analyses are presented in Table 8.10.
Study 2

Table 8.10

Results of ANCOVAs ($F$) and likelihood ratio tests ($\chi^2$) of baseline variables with frequency of binges at each follow-up time point, controlling for baseline binge frequency

<table>
<thead>
<tr>
<th></th>
<th>3 Months</th>
<th>12 Months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$df$</td>
<td>$F$</td>
</tr>
<tr>
<td>Age</td>
<td>239</td>
<td>4.44*</td>
</tr>
<tr>
<td>Education years</td>
<td>239</td>
<td>1.21</td>
</tr>
<tr>
<td>OCDS-O score</td>
<td>236</td>
<td>2.59</td>
</tr>
<tr>
<td>ACE-Intensity score</td>
<td>226</td>
<td>3.95*</td>
</tr>
<tr>
<td>ACE-Intrusion score</td>
<td>226</td>
<td>3.00</td>
</tr>
<tr>
<td>ACE-Imagery score</td>
<td>226</td>
<td>4.45*</td>
</tr>
<tr>
<td>Depression x OCDS-O</td>
<td>238</td>
<td>1.48</td>
</tr>
<tr>
<td>Depression x ACE-Intensity</td>
<td>226</td>
<td>0.28</td>
</tr>
<tr>
<td>Depression x ACE-Intrusion</td>
<td>226</td>
<td>2.40</td>
</tr>
<tr>
<td>Depression x ACE-Imagery</td>
<td>226</td>
<td>1.20</td>
</tr>
<tr>
<td>CARM vs. Brief/CBT x OCDS-O</td>
<td>236</td>
<td>4.51*</td>
</tr>
<tr>
<td>CARM vs. Brief/CBT x ACE total</td>
<td>224</td>
<td>2.03</td>
</tr>
<tr>
<td>Gender</td>
<td>1, 242</td>
<td>1.48</td>
</tr>
<tr>
<td>Relationship status</td>
<td>1, 242</td>
<td>0.44</td>
</tr>
<tr>
<td>Employment status</td>
<td>1, 241</td>
<td>0.02</td>
</tr>
<tr>
<td>Mental health diagnosis</td>
<td>1, 240</td>
<td>4.28*</td>
</tr>
<tr>
<td>Current mental health episode</td>
<td>1, 239</td>
<td>0.23</td>
</tr>
<tr>
<td>DASS-D</td>
<td>1, 242</td>
<td>0.69</td>
</tr>
<tr>
<td>Proportion of treatment attended</td>
<td>1, 242</td>
<td>0.07</td>
</tr>
<tr>
<td>Brief vs. Long</td>
<td>1, 242</td>
<td>2.40</td>
</tr>
<tr>
<td>CBT vs. CARM</td>
<td>2, 242</td>
<td>2.73</td>
</tr>
</tbody>
</table>

* $p < .05$  ** $p < .01$  *** $p < .001$

After controlling for baseline binge frequency, the OCDS-O was no longer significantly related to 12-month binges. However, its interaction with depression
remained significant. The significant association between ACE-Intensity scores and 3-month binges remained, and a significant association between ACE-Imagery scores and 3-month binges emerged. Education remained a significant univariate predictor of 12-month binges, and age emerged as being significantly associated with 3-month frequency of binges, with people bingeing less than half the week being older than people bingeing half or more of the week. History of mental health disorder remained significant also, as did the interaction between type of treatment and the OCDS-O at 3 months. The interaction between treatment and ACE scores was no longer significant.

8.3.4.2 Prediction by ACE subscales

The contribution of the ACE-Intensity and ACE-Imagery subscales to the prediction of 3-month frequency of binges was examined in a multinomial logistic regression. Craving scores for both subscales were entered along with baseline binge frequency, age, mental health diagnosis status and the interaction between treatment and OCDS-O. The parameter estimates are displayed in Table 8.11. The model fit was significant, $\chi^2(6, N = 224) = 28.14, p < .001$, with a Nagelkerke pseudo $R^2$ of .179, meaning the combination of the predictors accounted for an estimated 17.9% of the variance in 3-month frequency of binges. The likelihood ratio tests were significant for baseline weekly binges, $\chi^2(1, N = 224) = 14.12, p < .001$ and mental health diagnosis, $\chi^2(1, N = 224) = 5.25, p = .022$, but were not for age, $\chi^2(1, N = 224) = 1.87, p = .171$, ACE-Intensity, $\chi^2(1, N = 224) = 0.61, p = .434$, or ACE-Imagery, $\chi^2(1, N = 224) = 0.50, p = .481$, or the treatment with OCDS-O interaction, $\chi^2(1, N = 224) = 2.69, p = .101$. 
### Study 2

#### Table 8.11

*Parameter estimates for baseline variables predicting frequency of binges at 3-month follow-up (N = 227). Reference category is ‘half or more of the week’*

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>Std Error</th>
<th>Wald</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>BL Weekly binges (ref = less than half of week)</td>
<td>2.19</td>
<td>0.76</td>
<td>8.32</td>
<td>1</td>
<td>.004</td>
</tr>
<tr>
<td>Mental health diagnosis (ref = none)</td>
<td>0.79</td>
<td>0.35</td>
<td>5.08</td>
<td>1</td>
<td>.024</td>
</tr>
<tr>
<td>Age</td>
<td>0.02</td>
<td>0.02</td>
<td>1.85</td>
<td>1</td>
<td>.173</td>
</tr>
<tr>
<td>CARM vs. Brief/CTB x OCDS-O</td>
<td>0.19</td>
<td>0.12</td>
<td>2.55</td>
<td>1</td>
<td>.110</td>
</tr>
<tr>
<td>ACE-Intensity</td>
<td>-0.02</td>
<td>0.03</td>
<td>0.61</td>
<td>1</td>
<td>.435</td>
</tr>
<tr>
<td>ACE-Imagery</td>
<td>-0.01</td>
<td>0.02</td>
<td>0.49</td>
<td>1</td>
<td>.482</td>
</tr>
</tbody>
</table>

Neither of the ACE subscales were significant predictors with other baseline variables in the model. Only baseline frequency of binges and lifetime history of a mental health disorder were significant predictors of frequency of alcohol binges at 3 months.

### 8.3.4.3 Interaction testing

*Interaction between depression and the OCDS-O*

To determine if the interaction effect between baseline depression and OCDS-O scores was significant in predicting 12-month frequency of binges, the interaction term was entered into a multinomial logistic regression along with the mean-centered OCDS-O scores and the binary DASS-D variable (Jaccard, 2001). Baseline binge frequency and education were also entered. The model fit was significant, \( \chi^2(5, N = 238) = 19.96, p = .001 \), with a Nagelkerke pseudo \( R^2 \) of .128, however none of the craving,
depression, or interaction variables were significant in the model. The regression results are presented in Table 8.12.

Table 8.12

Likelihood ratio tests ($\chi^2$) and parameter estimates (B) for baseline variables predicting frequency of binges at 12 months ($N = 237$). Reference category is ‘half or more of the week’

<table>
<thead>
<tr>
<th>Predictor</th>
<th>$\chi^2$</th>
<th>df</th>
<th>$p$</th>
<th>B</th>
<th>Std Error</th>
<th>Wald</th>
<th>df</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>BL weekly binges (ref = less than half of week)</td>
<td>4.35</td>
<td>1</td>
<td>.037</td>
<td>1.06</td>
<td>0.56</td>
<td>3.56</td>
<td>1</td>
<td>.059</td>
</tr>
<tr>
<td>Education</td>
<td>9.87</td>
<td>1</td>
<td>.002</td>
<td>0.18</td>
<td>0.06</td>
<td>8.59</td>
<td>1</td>
<td>.003</td>
</tr>
<tr>
<td>DASS-D (ref = low depression)</td>
<td>0.12</td>
<td>1</td>
<td>.726</td>
<td>0.14</td>
<td>0.39</td>
<td>0.12</td>
<td>1</td>
<td>.725</td>
</tr>
<tr>
<td>zOCDS-O</td>
<td>0.47</td>
<td>1</td>
<td>.492</td>
<td>-0.18</td>
<td>0.25</td>
<td>0.48</td>
<td>1</td>
<td>.488</td>
</tr>
<tr>
<td>Depression x OCDS-O</td>
<td>0.34</td>
<td>1</td>
<td>.561</td>
<td>-0.21</td>
<td>0.36</td>
<td>0.34</td>
<td>1</td>
<td>.563</td>
</tr>
</tbody>
</table>

Treatment with craving interaction

To test whether the interaction between baseline OCDS-O scores and receiving a craving-focused treatment was significant in predicting frequency of binges at 3 months, the interaction term was entered into a multinomial logistic regression along with the mean centred OCDS-O, binary code of treatment received (CARM vs. Brief or CBT) and other significant baseline variables (Jaccard, 2001). Regression results are presented in Table 8.13. The interaction term fell short of significance, ruling out a moderating effect of type of treatment on the relationship between baseline OCDS-O and frequency of post-treatment binges.
Study 2

Table 8.13

Likelihood ratio tests (\(\chi^2\)) and parameter estimates (B) for baseline and treatment variables predicting frequency of binges 3 months (N = 237). Reference category is ‘half or more of the week’

<table>
<thead>
<tr>
<th>Predictor</th>
<th>(\chi^2)</th>
<th>df</th>
<th>(p)</th>
<th>B</th>
<th>std Error</th>
<th>Wald</th>
<th>df</th>
<th>(p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BL weekly binges (ref = less than half of week)</td>
<td>17.59</td>
<td>1</td>
<td>.000</td>
<td>2.36</td>
<td>0.75</td>
<td>9.78</td>
<td>1</td>
<td>.002</td>
</tr>
<tr>
<td>Mental health diagnosis (ref = none)</td>
<td>3.39</td>
<td>1</td>
<td>.066</td>
<td>0.60</td>
<td>0.33</td>
<td>3.33</td>
<td>1</td>
<td>.068</td>
</tr>
<tr>
<td>Age</td>
<td>3.95</td>
<td>1</td>
<td>.047</td>
<td>0.03</td>
<td>0.02</td>
<td>3.86</td>
<td>1</td>
<td>.050</td>
</tr>
<tr>
<td>CARM vs. Brief/CBT (ref = CARM)</td>
<td>0.68</td>
<td>1</td>
<td>.410</td>
<td>0.31</td>
<td>0.38</td>
<td>0.659</td>
<td>1</td>
<td>.417</td>
</tr>
<tr>
<td>zOCDS-O</td>
<td>0.23</td>
<td>1</td>
<td>.628</td>
<td>-0.08</td>
<td>0.17</td>
<td>0.235</td>
<td>1</td>
<td>.628</td>
</tr>
<tr>
<td>CARM vs. Brief/CBT x OCDS-O</td>
<td>3.16</td>
<td>1</td>
<td>.076</td>
<td>0.20</td>
<td>0.11</td>
<td>3.03</td>
<td>1</td>
<td>.082</td>
</tr>
</tbody>
</table>

8.3.5 Sensitivity analyses

To determine the robustness of the results, all analyses were repeated using the non-imputed data. Results with the complete cases were largely consistent with those of the imputed data set and did not alter the major findings of the study. In the uncontrolled analyses, the OCDS-O correlation with 3-month average weekly drinks fell just short of significance, \(r = .137, p = .051\) (cf. Table 8.4) and the correlation between the interaction of depression with ACE-Intensity and 12-month consumption was also not significant, \(r = .121, p = .140\) (cf. Table 8.4). Depression was not a significant univariate predictor of 12-month weekly drinking in the uncontrolled complete case analyses, \(F(1, 153) = 2.52, p = .114\) (cf. Table 8.4). In the uncontrolled univariate prediction of 3-month binges, none of ACE-Intensity, craving treatment with
OCDS-O interaction, or mental health diagnosis were significant at \( p < .05 \); \( F(1, 194) = 2.97, p = .086 \), \( F(1, 203) = 2.85, p = .093 \), and \( \chi^2(1, N = 205) = 3.53, p = .060 \) respectively (cf. Table 8.9), while the Brief versus Long treatment contrast was, \( \chi^2(1, N = 207) = 5.86, p = .015 \) (cf. Table 8.9).

Results of the controlled univariate analyses of average weekly drinks were all consistent between the complete case and imputed cases data sets. Some differences in the controlled 3-month binge frequency analyses were found. Age, ACE-Intensity, ACE-Imagery and the interaction of craving treatment with OCDS-O were not significant, \( F(1, 203) = 2.64, p = .106 \), \( F(1, 192) = 1.93, p = .167 \), \( F(1, 192) = 1.41, p = .236 \) and \( F(1, 201) = 1.66, p = .199 \) respectively (cf. Table 8.10). As in the uncontrolled analyses, the Brief versus Long interaction also emerged as significant in the controlled 3-month binges analysis, \( \chi^2(1, N = 206) = 5.88, p = .015 \) (cf. Table 8.10). This was not explored further as treatment effects were not a primary focus of the current study.

Regression results remained unchanged except for differences in the magnitude of coefficients. The variance of 12-month average weekly drinking explained by the regression model encompassing the depression by OCDS-O interaction decreased from .304 to .220 and in the model encompassing the depression with ACE-Intrusion interaction decreased from .292 to .190. The higher \( R^2 \) explained in the imputed regression is likely due to use of baseline drinking variables in the imputation model. The unique variance explained by the interactions was higher however, rising from .018 to .036 for the depression with OCDS-O interaction and from .021 to .034 for the depression with ACE-Intrusion interaction. The \( B \)-weight for the depression with OCDS-O interaction in the complete case analysis was 13.12, \( t(150) = 2.77, p = .006 \) (cf. Table 8.6) and was 10.82 for the depression with ACE-Intrusion interaction, \( t(141) = 2.54, p = .012 \) (cf. Table 8.7). The regression coefficient for the OCDS-O was -1.19
Study 2

(cf. Table 8.6) in the complete case analysis and was -2.61 for the ACE-Intrusion (cf. Table 8.7). Results of the slopes tests were consistent between the two sets of analyses.

The 3-month binge frequency regression was not repeated as the ACE variables were not significant univariate predictors in the complete case analysis. This was consistent with the finding that they were not significant predictors in the imputed data set regression. Results of the 12-month binge frequency regression were also consistent with the interaction not being a significant predictor, although baseline binges did emerge as significant, $\chi^2(1, N = 159) = 5.13, p = .024$ (cf. Table 8.12).

**8.4 Discussion**

This study aimed to examine whether craving and depression measured prior to delivery of an alcohol intervention would be predictive of short-term (3-month) and long-term (12-month) post-treatment level of alcohol consumption and frequency of alcohol binges. This study also examined the hypothesis that the combination of experiencing high levels of depression and high levels of craving would be associated with poorer post-treatment prognosis.

The hypothesis that craving would be predictive of post-treatment outcomes was not supported in the current study. Neither the OCDS-O nor the ACE were significant predictors of weekly alcohol consumption or frequency of binges at either 3 or 12 months. Even though the Intensity and Imagery subscales of the ACE were significant univariate predictors of 3-month binge frequency when baseline binges were held constant, these variables failed to remain significant in the logistic regression when other significant baseline variables were also controlled for. The finding that craving was not related to post-treatment outcomes contrasts with other studies, including Study 1, that have found baseline craving to be a significant predictor of alcohol use following
treatment (Bottlender & Soyka, 2004; Garbutt et al., 2009; Ray et al., 2006), but is consistent with others that have not (Kiefer et al., 2005; Kranzler et al., 1999). While craving itself was not found to be a significant predictor, some support for the hypothesis of an interaction effect between craving and depression in influencing drinking outcomes was found, supporting the notion that craving may be associated with post-treatment drinking in the context of high levels of comorbid depressed mood.

The interaction terms between baseline depression with craving measured using the OCDS-O and the ACE-Intrusion were significant in the prediction of 12-month weekly alcohol consumption, supporting a moderating effect of depression on the relationship between craving and long-term post-treatment drinking. Post-hoc interaction probing revealed a significant effect of craving at high levels of depression, with the regression slope for high depression differing significantly from zero. The slope for low depression did not differ from zero, suggesting no effect of low depression on the relationship between craving and 12-month alcohol use. Weekly alcohol consumption at 12 months was significantly greater for people who had high depression and high craving at baseline compared to people who had high depression but low craving.

One obvious interpretation of this interaction is that people who experience high levels of depressive symptoms experience strong urges to drink to relieve their negative affective state. This is the basis of Baker et al.’s (2004) affective processing model of negative reinforcement in which craving is predominantly affectively driven and negative affect is the prototypic context for substance use and relapse. While there is support for this pattern of influence in observations of associations between negative affect and proximal substance use (Fucito & Juliano, 2009; Perkins et al., 2008), in the current study, baseline depression by itself was not associated with post-treatment
Study 2

drinking. This suggests that the interaction of depression with craving was not accounted for primarily by the depression component. An alternative relationship is suggested by elaborated intrusion theory (Kavanagh et al., 2005), which posits that craving can give rise to a sense of deprivation, which can then generate negative affect. Presence of comorbid depression may exacerbate this effect, augmenting the negative affect generated by craving, amplifying the effect of craving.

Another possible mechanism by which high depression levels may interact with high craving to lead to greater levels of alcohol consumption is through diminished self-efficacy. Negative mood has been found to be associated with lower reported self-efficacy to resist drinking (Moss, Kirisci, & Mezzich, 1994; Ralston & Palfai, 2010), smoking (Rabois & Haaga, 2003) and substance use (Greenfield, Venner, Kelly, Slaymaker, & Bryan, 2012) in negative affect situations as well as other high risk substance use situations. Couple this with the reductions in drinking refusal self-efficacy often observed following exposure to alcohol cues (Cooney, Baker, Pomerleau, & Josephy, 1984; Jansma, Breteler, Schippers, De Jong, & Van Der Staak, 2000), and confidence to control drinking is likely diminished to levels where alcohol use can no longer be resisted.

A mechanism of diminished self-efficacy may also explain why the interaction was only predictive of 12-month drinking and not at 3 months. Self-efficacy is observed to increase in response to substance intervention (Greenfield et al., 2012; Kavanagh et al., 2006; Wong et al., 2004). While improvements in self-efficacy tend to be well maintained following treatment (McKellar, Ilgen, Moos, & Moos, 2008), they are generally observed to be at their highest immediately post-treatment (Kavanagh et al., 2006; Witkiewitz, Donovan, & Hartzler, 2012), and levels after 1 year have been found to be associated with degree of improvement in drinking and depression, with greater
improvement predicting greater self-efficacy (McKellar et al., 2008). If depression interacts with craving to diminish drinking control self-efficacy, the effect is likely to be weakest immediately following treatment when self-efficacy is highest and effective coping strategies are being optimally applied. Over time, if depression and craving remain high, some reversion back to baseline may occur, making conditions at 12 months more likely to resemble those of baseline, resulting in an association between baseline measures and 12-month drinking.

Another possible mechanism of interaction is suggested by the nature of the ACE items that were found to interact with depression to influence later drinking. Of all the ACE subscales, only the interaction of depression with the Intrusion subscale was significant. The Intrusion items are cognitive in nature and measure intrusion of alcohol thoughts and attempts to suppress those thoughts (“How hard were you trying not to think about alcohol?” and “How intrusive were the thoughts?”). Thought suppression is a generally unhelpful strategy that usually results in an increase of thoughts about the suppression target as soon as active attempts to suppress have ceased (Wegner, Schneider, Carter, & White, 1987; Wenzlaff & Wegner, 2000). Furthermore, any addition to cognitive load during suppression attempts, such as might be encountered if cognitively demanding distraction techniques were employed as a coping strategy, undermines the cognitive processes involved in suppression, increasing accessibility of the suppression target (Wegner, 2011; Wenzlaff & Bates, 2000). Therefore, the more effort that is put into suppressing thoughts about drinking, the more likely the person is to think about drinking, resulting in increased risk for drinking. Erskine, Georgiou and Kvavilashvili (2010) demonstrated this in smokers, finding that a group instructed to suppress thoughts about smoking smoked more in the following week than a group who were instructed to express their thoughts.
Study 2

The detrimental effect of thought suppression efforts can also be explained by ego depletion, as described by the Strength Model of self-control (Baumeister, Bratslavsky, Muraven, & Tice, 1998). According to the Strength Model, self-control is a limited resource that can become depleted with repeated use. Efforts at self-control, which extend to any self-regulation process including controlling thoughts, managing emotions, overcoming unwanted impulses, fixing attention, guiding behavior and making choices (Baumeister, Vohs, & Tice, 2007), drain the global resource, making subsequent efforts at control less effective. The more effort that is put into resisting and suppressing thoughts about drinking, the less resources that are left available to resist impulses to drink, leaving the person vulnerable to a lapse. An ego depletion effect on alcohol use has been demonstrated experimentally, with people exposed to an ego depleting task drinking more beer afterwards than people who engaged in a non-ego depleting control task (Christiansen, Cole, & Field, 2012). Presence of negative affect places additional demands on the pool of self-control resources, as regulating emotion requires suppression of innate tendencies to display and express emotional states (Hagger, Wood, Stiff, & Chatzisarantis, 2010). People who exhibit a tendency to exert effort on suppression at baseline may be more prone to revert back to this strategy after treatment has ended, resulting in greater difficulty controlling drinking at later time points.

Cognitive deficits associated with depression may further contribute to the failure of thought suppression efforts. Depression has been well documented to be associated with a range of cognitive deficits including diminished attention, reduced working memory and executive function deficits such as reduced ability to plan and inability to manage multiple tasks (Castaneda, Tuulio-Henriksson, Marttunen, Suvisaari, & Lönnqvist, 2008; Doumas, Smolders, Brunfaut, Bouckaert, & Krampe, 2012;
It is possible that the diminished cognitive capabilities associated with depression, when combined with intrusive and distracting drinking thoughts and cognitively demanding efforts invested in resisting those thoughts, reduces the person’s ability to engage the cognitive resources required to effectively problem solve and plan to avoid drinking, resulting in greater vulnerability to drinking lapses.

The moderating effect of depression on the relationship between baseline craving and post-treatment drinking was limited to the prediction of average weekly drinks and did not extend to frequency of binges. Alcohol binges represent a particular pattern of alcohol use associated with impaired control (Field et al., 2010; Weafer & Fillmore, 2008) and are likely driven by different factors from those that drive alcohol use more generally, as proposed in Study 1. The lack of consistency in the prediction of binges compared to weekly consumption may be a reflection of different influences. It is also possible that binges are related more strongly to much more proximal measures of craving, although no association between craving and binges was observed at baseline either. The proportion of people bingeing 6 or 7 days per week at baseline was slightly higher in the current study than in Study 1 (41.3% compared to 33.1%) and it could be that the reduced variability in binge frequencies in the current study was of sufficient magnitude to preclude detection of effects.

Consistent with Study 1, depression was not independently associated with any of the post-treatment outcomes (cf. Tables 8.5 and 8.10). The only influence of depression was through its interaction with craving in predicting 12-month drinking. This makes it unlikely that the absence of association observed in Study 1 was due to lack of variability in depression scores, as the current study had a sufficient spread of scores to enable detection of effects. Depression scores on the DASS21 ranged from 0
Study 2

to 42 with a mean of 10.6 and a standard deviation of 9.43. While the two studies did utilise different depression measures, the depression subscale of the DASS has been found to correlate strongly with the BDI (Lovibond & Lovibond, 1995a), the depression scale of the Hospital Anxiety and Depression Scale (Crawford & Henry, 2003; Zigmon & Snaith, 1983), the negative affect scale of the Positive and Negative Affect Scale (Crawford & Henry, 2003; Watson, Clark, & Tellegen, 1988) and to differentiate clinical populations and measure clinical change (Ng et al., 2007). Additionally, the DASS-D correlated significantly with baseline average weekly drinks at a magnitude equivalent to the OCDS-O. The lack of prediction from depression is therefore not believed to be related to the type of measure used.

The lack of prediction from baseline depression is consistent with other studies that have also found depression to not be related to post-treatment alcohol use or abstinence (Oslin et al., 2009), including the study of Farren and McElroy (2010), but is inconsistent with other studies that have (Gamble et al., 2010; Kodl et al., 2008; Witkiewitz & Villarroel, 2009). The consistency of the results across Study 1 and the current study lends some credence to the argument that depression may be a stronger proximal associate of drinking. Alternatively, it may also be possible that, like craving in the current study, the relationship between depression and later alcohol use is moderated by other factors, and therefore only emerges under certain conditions. Further research is needed to explore this possibility.

Another finding of interest in the current study is the highly consistent results between the OCDS-O and ACE-Intrusion scales. Both scales interacted significantly with depression to influence 12-month drinking and the regression results of the two scales were close to equivalent. The interaction of depression with OCDS-O accounted for 1.8% of unique variance in 12-month average weekly drinking while the depression
with ACE-Intrusion interaction accounted for 2.1%. For people with high baseline depression, a one standard deviation increase in OCDS-O score was associated with drinking 5.81 more drinks at 12 months, and a one standard deviation increase in ACE-Intrusion score was associated with drinking 5.14 more. The ACE-Intrusion scales also had the highest baseline correlations with the OCDS-O, suggesting a higher degree of shared variance between these scales than between the OCDS-O and other subscales of the ACE. The fact that neither the interaction of depression with OCDS-O nor the interaction of depression with ACE-Intrusion were statistically significant when both were entered into the same regression also suggests that the two measures are capturing overlapping predictive variance. Examining the items of the two scales, both measure preoccupation with drinking thoughts and efforts to resist and control those thoughts. While these cognitive experiences represent only one dimension of the experience of craving (Anton, 2000; Kavanagh et al., 2005), in the current study they held the most value in predicting long-term treatment prognosis.

Other subscales of the ACE (Intensity and Imagery) did not predict post-treatment drinking. The Intensity subscale measures strength of desires for alcohol, need for a drink and difficulty directing thoughts away from alcohol. The Imagery subscale measures frequency and intensity of images pertaining to alcohol and drinking. These subscales tap into the cognitive elaborative process of craving, as described by elaborated intrusion theory which argues that these processes underpin the experience of desire (Kavanagh et al., 2005). The results of the current study appear to suggest that these aspects of craving may not be as strongly associated with alcohol use measured at a more distant time point as the degree of intrusiveness of alcohol thoughts is. When concurrent measures were examined, only the ACE-Intensity subscale was related to baseline drinking, correlating with average weekly drinks to a similar magnitude as the
OCDS-O. Intensity of alcohol thoughts may be more closely related to proximal alcohol use, while intrusiveness of thoughts may be a stronger indicator of longer term prognosis. This may be because a high level of intrusion from alcohol thoughts at baseline conveys a high degree of availability of alcohol thoughts, which may present an ongoing risk following treatment. Elaborative processes on the other hand, may change over time, possibly as a consequence of treatment effects. This may make them associate poorly with alcohol use measured at a later time.

The Imagery subscale was not associated with either baseline or post-treatment drinking, suggesting it may lack sensitivity as a measure of craving. However, all subscales of the ACE, including Imagery, were moderately to strongly correlated with the OCDS-O, hinting at a high degree of shared variance between the scales. All ACE subscales also correlated significantly with depression at baseline and with alcohol abuse diagnosis. There may be other factors influencing how alcohol-related imagery relates to drinking behaviour. Further research testing the propositions of elaborated intrusion theory and how they translate to drinking behaviour is needed.

Study 1 found that particular items of the OCDS-O related more strongly to post-treatment drinking than others, indicating that not all aspects of craving are related to post-treatment outcomes. The same appears to be true of the ACE, with the Intrusion subscale being more important in the prediction of later drinking than the Intensity and Imagery subscales. This suggests that it may not be the measurement of craving per se that is important for prediction, but what certain experiences with aspects of craving might represent about a person’s potential coping skills, and what that may mean for their prognosis. The predictive value of scoring high on the OCDS-O and ACE-Intrusion may derive from these scores representing a tendency to engage in maladaptive coping strategies to deal with drinking thoughts, such as suppression.
Having a pre-disposition to use such strategies may make a person more vulnerable to later relapse. This could have clinical implications and suggests potential benefit in providing education about thought suppression not being a useful drinking control strategy and instruction in more effective alternative strategies.

One limitation of the current study was the use of a different depression scale from Study 1, limiting direct comparison of results between the two studies with regards to depression and the interactions. While there is reasonable convergence between the DASS and BDI (Antony et al., 1998; Lovibond & Lovibond, 1995a), replication of these results using the BDI and other depression scales is necessary to establish generalisability of the results. Another limitation was the lack of more extreme cases of depression. While the current study achieved the desired wider spread of depression severity than in Study 1, only 14% of the current sample scored in the severe or extremely severe range of depression, making secondary analyses with a subset of participants with depression levels comparable to Study 1 inadvisable. This further limited the ability to directly compare results with Study 1. Also a limitation was the low follow-up rate for the 12-month assessment, with only 64% of participants able to be contacted. This is not believed to have altered the results of the study however, as a high degree of consistency was obtained between the imputed and complete case analyses, suggesting the results obtained were robust and not influenced by variables related to attrition.

The results of the current study suggest numerous avenues for future research. Replication of the interaction effect using different depression, craving and outcome measures and with different substances of abuse is required to establish the robustness and generalisability of the effect. Examination of other variables such as self-efficacy and coping styles will shed light on how depression interacts with craving to affect
outcomes. Finally, further investigation of the prediction by depression is required to explore if, similar to craving in the current study, the effect of depression on later substance use is moderated by other variables.

8.5 Conclusion

This study found that high levels of depressive symptoms pre-treatment interacted significantly with craving such that high depression with high craving was associated with greater drinking at 12 months. These results provide preliminary evidence of a moderating effect of depression on the prediction of 12-month alcohol consumption by craving, although the effect was admittedly small and the results mixed. There was no effect of low depression. Further research is needed to determine the robustness and source of this effect, but possible mechanisms include reduced self-efficacy or diminished cognitive capacity to engage effective coping strategies. Baseline depression was not related to any of the outcome measures, suggesting its influence may be stronger proximally, or that its effect may also be moderated by other variables. This study suggests some benefit in considering pre-treatment presentations of both depression and craving as reporting high symptoms on both may be associated with poorer long-term prognosis following treatment.
CHAPTER 9 GENERAL DISCUSSION

This thesis aimed to explore the performance of craving as a predictor of post-treatment alcohol use outcomes in the presence of comorbid depressed mood, under the hypothesis that presence of negative affect would augment effects of craving, strengthening its predictive power and increasing vulnerability to post-treatment relapse.

Craving has performed inconsistently in past prediction studies, emerging as a predictor in some (e.g., Bottlender & Soyka, 2004; Crits-Christoph et al., 2007; Ray et al., 2006; Toll et al., 2007), but not in others (e.g., Ahmadi et al., 2009; Kampman et al., 2004; Weiss et al., 1995; Zhou et al., 2009). It was hypothesised that some of these inconsistencies may be due to unmeasured moderator variables, and that the relationship between craving and later substance use would be stronger under certain conditions and in the presence of certain other variables. Negative affect was proposed as one such variable, given positive associations observed between negative affect and craving (e.g., Andersohn & Kiefer, 2004; Cleveland & Harris, 2010; Cooney et al., 1997), and between negative affect and substance use (e.g., Doumas, 2011; Fucito & Juliano, 2009; Kodl et al., 2008). It was also hypothesised that the combination of high levels of depression with high levels of craving would be predictive of poorer post-treatment outcomes. Some support for this hypothesis was found in the present research.

Study 1 investigated the predictive performance of craving in a sample of heavy drinkers (> 28 x 10g ethanol per week for men; > 14 for women) who were also reporting elevated levels of depressed mood (≥ 17 on BDI-II). Participants completed baseline assessments prior to commencement of their randomly assigned intervention, and were assessed again after 18 weeks and 12 months. Study 2 examined craving as a
General discussion

predictor in a sample of heavy drinkers with alcohol use disorder, with no specific inclusion or exclusion based on depression. Depression scores (on DASS21 depression scale) in this sample ranged from none to extremely severe. As per Study 1, baseline assessments were conducted prior to delivery of any intervention, and were repeated 3 and 12 months later. Before comparing the results of these two studies, it is important to consider the similarities and differences between the two populations.

Appendix H provides statistical comparisons on the key baseline and demographic variables. The two samples were well matched on age and gender. While the full sample of Study 2 participants was significantly older ($M = 48.82$ vs. $M = 45.42$ years) and had a higher proportion of males (60.7% vs. 50.8%) than that of Study 1, there was no difference when the Study 2 sample was restricted to those with moderate or higher depression ($M = 46.89$ years and 58.8% male). The Study 2 sample had a significantly higher number of years of education than Study 1 ($M = 13.68$ vs. $M = 11.10$ years) and a higher proportion of partnered participants (68.6% vs. 33.1%). These differences remained when the Study 2 sample was restricted to those with high depression ($M = 12.96$ years and 56.5% partnered).

Average weekly alcohol consumption was not different between the groups for either the full Study 2 sample ($M = 63.13$ vs. $M = 61.61$) or the high depression subsample ($M = 69.02$). The Study 2 population did however have a significantly greater proportion of people bingeing half or more of the week than in Study 1 (80.6% vs. 63.5%). This difference was also significant in the high depression subgroup (83.5%). Severity of alcohol dependence measured using the AUDIT was significantly lower in the overall Study 2 sample ($M = 24.50$ vs. $M = 25.70$), but was not different between the studies when only the high depression subgroup of Study 2 were examined ($M = 26.52$). Study 2 had a significantly greater proportion of alcohol dependent
participants in both the overall (94.2% vs. 83.8%) and high depression (94.1%) samples. Prevalence of alcohol abuse diagnosis was not different when only the high depression subsample were examined (69.4% vs. 68.1%), but was significantly lower in the overall Study 2 sample (55.4%) than in Study 1. Total craving score on the OCDS-O was significantly lower in the Study 2 group when examining the overall sample ($M = 6.89$ vs. $M = 9.17$) but was not different when the Study 2 group was restricted to those with higher depression ($M = 9.19$).

Depression scores could not be directly compared as different measures were used between the studies. The proportion of participants categorised according to severity of depressive symptoms was compared instead, combining the severe and extremely severe categories of the DASS-D to create a four category severity coding across both studies (none or minimal; mild; moderate; severe). As Study 1 specifically excluded people with low depression, there was a significantly higher proportion of people in Study 2 with no or minimal depression than in Study 1 (50.8% vs. 0%). Even when restricting the Study 2 sample to those with moderate or greater depression, the Study 2 sample still had a significantly lower prevalence of severe depression than in Study 1 (38.8% vs. 57.1%). Presence of a current depressive episode was also significantly lower in Study 2 than in Study 1 (5.8% vs. 70%), even when the Study 2 sample was restricted to those with high depression (14.1%), although it must be noted that depression diagnosis in Study 2 was based on self-report and not diagnostic interview.

The higher levels of education and partnering in Study 2 suggest it was potentially a higher functioning sample than in Study 1 with higher levels of social support and engagement. This may have been associated with enhanced coping abilities in the Study 2 group than in Study 1. On the other hand, Study 2 had a higher incidence
General discussion

of alcohol dependence and a higher frequency of alcohol binges, suggesting it was a population of more problematic drinkers. This could imply diminished coping ability compared to participants in Study 1, or alternatively, represent a greater scope for change in the Study 2 group than in Study 1. The potential implications of the group differences between studies are considered in the comparison and discussion of the results.

9.1 Evidence for moderating effect of depression

While the pattern of results across Studies 1 and 2 showed consistencies and inconsistencies, the pattern of the inconsistencies appears to provide preliminary support for the overarching hypothesis of this thesis. Two key points of difference between the two studies were: a) craving on its own was not a significant predictor of any of the outcome measures at either of the follow-up points in Study 2, but it was in Study 1; and b) the interaction between depression and craving was not a significant predictor of any of the outcomes in Study 1, but it was a predictor of 12-month average weekly drinks in Study 2. When taken together, it could be argued that these two results appear to be telling the same story, that high craving plus high depression may be associated with greater risk for higher post-treatment drinking.

When Study 1 failed to find evidence for a moderating effect of depression on the relationship between baseline craving and post-treatment drinking, it was argued that this may have been due to lack of sufficient variation in depression scores to detect an interaction effect. To test this, Study 2 targeted a broader range of depression severity, ranging from no symptoms to severe symptoms. With adequate variation in depression scores, Study 2 was able to detect a significant interaction effect, with the source of the interaction arising from high depression. Craving was predictive of post-
treatment drinking only in the presence of elevated levels of depressed mood, supporting the hypothesis that people with high levels of craving and high levels of depressed mood will be at higher risk for greater alcohol consumption, and that this combination will be predictive of post-treatment outcomes. In Study 1, this same effect was expressed in the performance of the OCDS-O as a significant predictor, because it was reacting with the high levels of depression present in the whole sample. Supporting this notion is the near identical craving scores in the Study 1 sample and the subset of Study 2 participants who had moderate or greater depression ($M = 9.17$ and 9.19 respectively), compared to the significantly lower mean craving score observed in the full Study 2 sample ($M = 6.89$; Appendix H).

Chapter 8 discussed a number of possible mechanisms by which depression may interact with craving to influence later alcohol use. One of the mechanisms proposed was an effect on self-efficacy. Since both negative mood and alcohol cues are associated with lower reported self-efficacy to refuse drinking (Cooney et al., 1984; Jansma et al., 2000; Moss et al., 1994; Ralston & Palfai, 2010), it was argued that the combination of high levels of depressed mood with high cravings generated by exposure to alcohol would substantially reduce self-efficacy to resist drinking, resulting in greater levels of post-treatment alcohol use. Supporting this argument is evidence that self-efficacy is negatively associated with alcohol consumption in community and clinical populations (Hasking & Oei, 2002; Oei, Hasking, & Phillips, 2007) and that self-efficacy measured prior to treatment is predictive of post-treatment drinking (Kavanagh et al., 2006; Solomon & Annis, 1990; Witkiewitz et al., 2012).

Another possible mechanism of the interaction effect discussed in Chapter 8 related to cognitive processes. Of the three subscales of the ACE, only the Intrusion subscale interacted significantly with depression to predict 12-month drinking. The
General discussion

items of the Intrusion subscale relate to intrusiveness of alcohol-related thoughts and efforts made to resist those thoughts. It was argued that the cognitive demands associated with attempts to suppress thoughts about alcohol during times of craving, paired with the cognitive impairments observed to accompany depression (Castaneda et al., 2008; Hasselbalch et al., 2011; Porter et al., 2007), may reduce an individual’s capacity to engage effective coping strategies, making them more vulnerable to drinking. The effect of ego depletion caused by efforts to resist and suppress drinking thoughts was also discussed. Ego depletion refers to the “state of diminished resources following exertion of self-control” (Baumeister et al., 2007, p. 352). If great efforts are invested in trying to control drinking-related thoughts through suppression, fewer resources are left for controlling and resisting drinking behaviour, leading to higher levels of alcohol use than in people who have not depleted their ego resources through attempts at suppression. Comorbid depressed mood contributes further to depletion through efforts required to regulate negative affect (Hagger et al., 2010).

The results of Studies 1 and 2 appear to provide preliminary support for a potential moderating effect of depression on the relationship between pre-treatment craving and post-treatment alcohol use. However, the effects observed were small and the results mixed, with effects not extending to all outcomes measured or to all time points assessed. The results must therefore be interpreted cautiously, and further research is needed to investigate if the effect is replicated in other samples and with other measures.

9.2 Inconsistencies and implications

The results of this research exemplify the state of the field of craving prediction research in general, with some findings replicating across the two studies and others not.
Just as past research has found certain outcomes to be significantly predicted by craving in some studies and not others (e.g., time to relapse; Garbutt et al., 2009; Kiefer et al., 2005; Paliwal et al., 2008; Roberts et al., 1999; Rohsenow et al., 1994), the present research also found such differences across Studies 1 and 2, with craving in Study 1 predicting frequency of post-treatment alcohol binges, while in Study 2 it did not.

Similarly, just as past research has found variation in the timeframe of outcome assessment that craving predicts (e.g., 3-month outcomes predicted in some studies but not others; Ray et al., 2006; Weiss et al., 1995), such differences were also found in the present research, with craving predicting average weekly consumption at the short-term follow-up assessment in Study 1, but not in Study 2, and 12-month consumption being predicted by craving in Study 2 but not in Study 1. As raised in Chapter 4, these inconsistencies may be attributable, at least in part, to issues relating to the nature and timing of assessment. The implications of the inconsistencies between Studies 1 and 2 are therefore considered with the issues raised in Chapter 4 in mind.

### 9.2.1 Prediction of average weekly drinks

Study 1 found craving to be a significant predictor of average weekly drinks at 18 weeks but not at 12 months. It was argued that this may reflect a weakening association between craving measured at one point and overall consumption measured at a more distant time, as the contextual state in which the alcohol use was measured becomes further and further removed from the contextual state in which the craving was measured. However, Study 2 found the interaction of depression with craving was predictive of only 12-month average weekly drinks and not 3-month drinking, the opposite of the effect observed in Study 1. This was also interpreted as a reflection of the context in which craving is measured, such that conditions 9 months following completion of treatment are more likely to resemble those of baseline, when the person
is attempting to control their alcohol use without support and in the presence of their usual cues and triggers.

One possible explanation for this discrepancy may lie in the timeframes of the first follow-up assessment of each study. The first post-treatment assessment in Study 1 was conducted 18 weeks after the baseline assessment. The long treatment arms of Study 1 were only 10 sessions, designed to be conducted 1 week apart. If participants adhered to the prescribed treatment regimen, treatment should have been finished within 11 weeks of when the baseline assessment was conducted. Even allowing for delayed appointments, the immediate post-treatment assessment typically occurred several weeks after treatment had been completed. In Study 2, the immediate post-treatment assessment was conducted at 13 weeks. If participants adhered to the prescribed treatment schedule of Study 2, treatment was completed in week 11, leaving only 2 weeks before the immediate post-treatment assessment. This difference of 5 weeks, while small, may have been large enough to make the contexts of the two post-treatment assessments sufficiently dissimilar to affect their comparability. It may be that in the early weeks following treatment, motivation, skills and self-efficacy remain high, but begin to diminish over time.

Witkiewitz, Donovan, et al. (2012) examined drink-refusal self-efficacy at baseline, end of treatment and 10 weeks post-treatment. A significant increase in self-efficacy from baseline to the end of treatment was observed, and while self-efficacy scores remained higher than baseline at 10 weeks, they had declined from the level measured at the end of treatment. Ratner, Johnson, Bottorff, Dahinten and Hall (2000) documented a decline in smoking cessation self-efficacy in the 12 months following a postpartum smoking relapse prevention intervention, with the steepest decline occurring in the first 6 months. If craving is a stronger predictor of alcohol use occurring in a
context similar to that in which the craving was measured, it is possible that craving was predictive of 18-week alcohol consumption in Study 1 and of 12-month alcohol use in Study 2 because enough time had lapsed between the end of treatment and those assessment points for some reversion back to baseline to occur, creating contextual environments similar to those of baseline when the craving was measured.

It is more difficult to explain why craving was not related to 12-month outcomes in Study 1 when it was in Study 2. This may relate to variables not examined or reported here, such as differences in treatment seeking outside of the research study during follow-up. The fact that the Study 1 sample had more severe depression and more demographic risk factors may have made them more likely to seek outside treatment if they relapsed.

9.2.2 Prediction of alcohol binges

The second major point of difference between the two studies relates to the prediction of frequency of binges. In Study 1, craving was a significant predictor of both 18-week and 12-month frequency of alcohol binges, but in Study 2 it was not, either singly or through an interaction. One possible reason for this is the extreme skew in binge frequencies observed in Study 2 relative to Study 1. A significantly higher proportion of the Study 2 participants were bingeing half or more of the week at baseline (80.6%) than in Study 1 (63.5%). This may have been related to the greater prevalence of alcohol dependence in the Study 2 population. Binge frequencies also differed between the studies at follow-up, with fewer participants in Study 2 bingeing half or more of the week at the follow-up assessments than in Study 1 (24.4% vs. 36.5% at 18 weeks/3 months, and 19.4% vs. 40.4% at 12 months). The large imbalance in the binge frequency group sizes in Study 2 (numbers bingeing less than half the week
General discussion

versus numbers bingeing half or more of the week) may have made it harder to detect an effect in Study 2 than in Study 1.

9.3 Consistencies and implications

9.3.1 Variance explained by craving

A somewhat consistent finding across both studies is the amount of unique variance accounted for by craving, or the depression with craving interaction. In Study 1, the OCDS-O accounted for 3.5% of unique variance in 18-week alcohol consumption. In Study 2, the depression with OCDS-O interaction accounted for 1.8% of unique variance in 12-month alcohol use, while the depression with ACE-Intrusion interaction accounted for 2.1%. It is difficult to compare the variance explained by craving in the current research with that of other studies, as most studies examine craving as just one of a set of predictors and do not report its unique contribution in the prediction model. Rohsenow et al. (2007) did report on the unique contribution of craving, documenting that urge accounted for 5% of unique variance in amount spent on cocaine in the 3 months following treatment. This is highly comparable with the results of the present research.

The amount of variance explained by craving, while significant, was still very small. The largest portion of unique variance was attributable to baseline consumption. This is highly consistent with the review of Adamson et al. (2009), which concluded that baseline alcohol consumption and severity of alcohol dependence accounted for most of the post-treatment drinking variance in prediction models. Yet even with baseline consumption in the model, the full set of predictors still only accounted for less than 40% of the variance in post-treatment drinking (less than 30% in the analyses on
the non-substituted data). This suggests that there are still other factors influencing post-treatment drinking that were not measured or examined in the present research. These could include self-efficacy (Kavanagh et al., 2006; Solomon & Annis, 1990; Witkiewitz et al., 2012), coping styles (Bricker, Schiff, & Comstock, 2011; Hasking & Oei, 2002), personality factors (Gullo, Dawe, Kambouropoulos, Staiger, & Jackson, 2010; Schuckit, 2009), expectancies (Hasking & Oei, 2002; Heinz, Kassel, Berbaum, & Mermelstein, 2010; Young, Connor, Ricciardelli, & Saunders, 2006) or motives (Kuntsche & Cooper, 2010; Kuntsche & Kuentig, 2012; Palfai et al., 2011), to name just a few. Furthermore, research is increasingly demonstrating that these variables do not influence substance use in isolation, but interact with each other to increase or decrease substance use risk. For example, Gullo et al. (2010) found that positive alcohol expectancy fully mediated the association between reward sensitivity and hazardous alcohol use in young adults and treatment-seeking substance users. They also found that drinking refusal self-efficacy fully mediated the association between rash impulsiveness and hazardous drinking amongst the treatment seeking group.

There are clearly innumerable variables that can influence substance use at any given time, and myriad pathways through which they may act to make a person more or less likely to engage in substance use either in the present moment or at some distant time point. The present research demonstrates an influence of depression on the relationship between craving and some measures of post-treatment alcohol use, though the small amount of variance explained highlights the need to continue examining other factors, including how they may work together to influence outcomes.
9.3.2 Nature of significant craving items

Also consistent across the two studies is the nature of the craving items contributing to the predictions. In Study 1, regressions with the individual items of the OCDS-O were examined to determine if particular items were more important in the prediction of post-treatment drinking than others. The results suggested different mechanisms of influence of craving over time following treatment, with items pertaining to interference from drinking thoughts and success in diverting such thoughts being related to short-term outcomes, and items pertaining to frequency of drinking thoughts and efforts to resist those thoughts being related to long-term outcomes. The results of Study 2 are largely consistent with those of Study 1, in the finding that the ACE-Intrusion subscale was the only one of the ACE subscales to significantly predict 12-month drinking (through its interaction with depression). The ACE-Intrusion items pertain to intrusiveness of drinking thoughts and efforts made to not think about alcohol. These align very closely with the OCDS-O items that were significant in the prediction of 12-month drinking in Study 1 (frequency of drinking thoughts and efforts to resist thoughts). Prediction by individual OCDS-O items was not examined in Study 2 as the OCDS-O was not independently predictive of outcomes, but the fact that both the OCDS-O and ACE-Intrusion interactions cancelled out each other’s prediction when entered into the same regression suggests that they are parallel measures tapping into the same construct.

It was hypothesised in Chapter 8, and again earlier in the current chapter, that efforts to resist and control drinking-related thoughts may drain cognitive resources, leaving the person vulnerable to a lapse. People demonstrating a tendency to manage their cravings in this way at baseline appear to be at greater risk of higher levels of
alcohol consumption following treatment. This suggests that it may not be craving per se that is important and meaningful for relapse, but how people manage those cravings.

Thoughts about alcohol do not necessarily constitute craving, and this may be why scale items pertaining to frequency and intensity of alcohol-related thoughts were not predictive of drinking in the present research. Craving is generally considered to be synonymous with desire, and this implies that by nature it is affectively laden. Elaborated intrusion theory (Kavanagh et al., 2005) describes this as imaginary relish and exquisite torture. Thoughts about the substance may initially be pleasurable as the positive aspects of obtaining or using the substance are elaborated, but soon become unpleasant as a sense of deficit arises (Kavanagh et al., 2005). When motivation to abstain is high, attempts may be made to resist or suppress these thoughts, placing additional demands on already stretched cognitive resources, leaving diminished capacity to problem solve and implement appropriate coping strategies. It appears to be use of such strategies that is predictive of poorer post-treatment prognosis, rather than simply whether craving occurred or not. This is consistent with the assertions of Ferguson and Shiffman (2009) and Miller, Westerberg, Harris and Tonigan (1996), both of whom argue for the importance of coping strategies in decreasing relapse risk.

9.3.3 Lack of prediction by depression

A third point of consistency between the two studies is the failure of baseline depression to predict any of the alcohol use variables at any of the post-treatment time points. In Chapter 7 it was proposed that this might have been due to limited variability in depression scores, due to the sample being restricted to people scoring 17 or above on the BDI-II. However, Study 2 also found depression to not be predictive of post-treatment drinking, despite adequate spread in depression scores ranging from none to extremely severe on the DASS-D. Taken together, along with the observation that
General discussion

Baseline depression was significantly correlated with baseline average weekly drinks, these results seem to suggest that the influence of depression on alcohol use may be limited in proximity, as discussed in Chapter 8. While reliable proximal associations between negative mood and substance use have been demonstrated in experimental studies (e.g., Fucito & Juliano, 2009; Perkins et al., 2008) and ecological momentary research (e.g., Armeli et al., 2000; Berkman et al., 2011; Doumas, 2011), this explanation is contradicted by numerous other studies that have found pre-treatment depression to be a predictor of post-treatment substance use (e.g., Gamble et al., 2010; Suter et al., 2011; Zhou et al., 2009). As discussed in Chapter 8, this suggests there may be other factors, not measured or examined in the present research, influencing the relationship between pre-treatment depression and post-treatment substance use.

The context in which depression is measured does appear to be important. For example, Zhou et al. (2009) found that smokers who endorsed experiencing withdrawal as anxiety/depression were 30% more likely to relapse in the 3 months after making their quit attempt than those who did not. Mood disturbance that was unrelated to withdrawal, and so an indication of a substance independent affective condition, was not associated with greater risk of relapse. These results suggest that negative affect in the context of withdrawal may be more important in predicting later relapse to smoking than depression measured more generally. Alternatively, McCarthy et al. (2006) looked at the importance of mood changes leading up to a quit attempt in smokers and found that pre-quit increases in negative affect were significantly and inversely related to abstinence at 3 months, suggesting measures of reactive mood states may be better predictors.
The primary aim of this thesis was to investigate whether there were certain conditions, such as high levels of comorbid negative mood, under which pre-treatment craving may perform more strongly as a predictor of post-treatment substance use, allowing for identification of people who may be at a greater risk for relapse after treatment. The results of this research provide some preliminary support suggesting a potential moderating effect of depression on the relationship between pre-treatment craving and post-treatment alcohol use. If this effect is replicated, this would suggest that people presenting for alcohol treatment who are reporting high levels of craving and high levels of depressed mood may be at greater risk for higher levels of alcohol use post-treatment than people reporting lower levels of depression or low levels of craving. Additional support in the post-treatment period may be of particular importance for these people, and this knowledge could assist in prioritising distribution of resources. Numerous studies have demonstrated the efficacy of aftercare support in achieving greater rates of abstinence and longer time to relapse than when no aftercare is provided (Burleson, Kaminer, & Burke, 2012; Dennis & Scott, 2012; Sannibale et al., 2003), however aftercare programs can be expensive to run, and are often not a routine part of service delivery.

The results of the present research also provide further support for the need to provide alcohol users with training in the effective management of alcohol-related thoughts and cravings. The results of Studies 1 and 2 suggest that people reporting tendencies to invest considerable effort in resisting and controlling alcohol thoughts are more at risk for greater drinking following treatment than people who do not make substantial efforts to resist or control their thoughts. It is recognised that suppression is an ineffective strategy to manage unwanted thoughts (Wegner et al., 1987; Wenzlaff &
General discussion

Bates, 2000). Acceptance-based therapies, such as mindfulness meditation, teach skills in detaching from alcohol thoughts and learning to ‘let them be’ without needing to engage with them or control them (Khong, 2009). Evidence supports the efficacy of acceptance-based interventions in the treatment of addictions (Vieten et al., 2010; Witkiewitz et al., 2005; Zgierska et al., 2009), and acceptance of one’s experience has been found to reduce the strength of association between alcohol-approach associations and hazardous drinking (Ostafin & Marlatt, 2008). Studies of the mechanism of action have demonstrated changes in how substance-related thoughts are engaged and processed, and that this consequently results in lower reported levels of craving (Witkiewitz, Bowen, Douglas, & Hsu, in press). These changes in how craving is engaged and managed have been found to mediate the relationship between treatment and outcomes (Bowen, Witkiewitz, Dillworth, & Marlatt, 2007) and between negative affect and relapses (Witkiewitz & Bowen, 2010; Witkiewitz et al., 2011). The current results suggest that provision of acceptance-based training may be of particular benefit to people reporting high baseline tendencies to resist and control drinking thoughts.

Study 2 did provide an intervention targeted specifically at craving management, including mindfulness meditation exercises (CBT + CARM), but this was not found to be more effective in reducing alcohol use than standard CBT alone. However, the strategies taught in the craving intervention were designed to target the cognitive elaborative process of craving (Kavanagh et al., 2005). The results of this research suggest that phasic peaks of craving, captured by degree of intrusiveness of alcohol thoughts, may be a more important indicator of relapse risk, and may therefore be a more important target for interventions. This is especially relevant for cue-exposure treatments which focus on training skills to manage episodic cravings that arise in response to substance cues. People who experience a high degree of intrusion from their
alcohol thoughts may benefit to a greater extent from cue-exposure treatments than people who do not experience a high level of intrusion from alcohol thoughts.

It must also be noted that no investigations of possible mediators or moderators of the effect of the CBT + CARM treatment were conducted in the present research as treatment effects were not a primary focus of this thesis. It is possible that the treatment may be most effective for those experiencing a higher degree of cognitive elaboration during craving.

9.5 Limitations of this body of work

One of the aims of the present research was to overcome limitations of previous research in the craving field by studying continuous outcomes. While this was possible for average weekly drinks, the distribution of responses for average weekly binges precluded use of the continuous version of this variable. This variable was highly left skewed at baseline, with the majority of participants bingeing 6 or 7 days out of the week, and was highly right skewed at follow-up, with the majority of participants not bingeing, or bingeing only 1 day per week. It was therefore not possible to study this variable in any other way except for a binary coding. This resulted in substantial loss of variability, particularly in Study 2 where the distribution of binges was even more extremely skewed than in Study 1, and may explain why craving was not predictive of binges in that study when it had been in Study 1.

Another limitation of the present research is the lack of inclusion of other potential variables of influence. A number of variables have been found to influence both cue-elicited and self-reported urges, including perceived access to the substance (Wilson et al., 2005), expectancies (Perkins, Grottenthaler, et al., 2010), self-efficacy (Shadel & Cervone, 2006), and a number of personality traits (Kambouropoulos &
General discussion

Rock, 2010; Litvin & Brandon, 2010; Reuter & Netter, 2001; VanderVeen, Cohen, Cukrowicz, & Trotter, 2008; Zilberman, Tavares, & el-Guebaly, 2003). Factors influencing relapse risk are numerous, and are described in detail by the Cognitive-Behavioral Model of Relapse (Witkiewitz & Marlatt, 2007). These factors include negative and positive emotional states, coping skills, outcome expectancies, self-efficacy, abstinence-violation effect (guilt or shame about lapsing), craving and social support. Attentional bias has also been found to predict relapse risk in heroin users (Marissen et al., 2006). Lack of measures of additional variables such as these prevented exploration of other potential mechanisms of influence.

The generalisability of the results is also limited to the population studied and the measures used. Both studies were conducted on treatment-seeking outpatients who self-referred from the community predominantly in response to advertising. This was therefore a sample of drinkers who were presumably motivated for change, and potentially higher functioning than participants in other studies recruited from inpatient facilities. The findings and conclusions are also limited to the particular measures used in these studies. The BDI-II was used to measure depression in Study 1 while the DASS-D was used in Study 2. The use of two different measures of depression across studies makes direct comparison of the results difficult. Replication with other depression measures is required to ascertain if the results found here can be generalised to depression or are limited to the particular measures used. This is true of the craving measures also. While the OCDS-O was used across both studies, the ACE was used in only Study 2. As discussed in Chapter 4, craving is measured in many different ways, and further investigation is required with other craving measures to determine if the moderating effect of depression applies to craving in general, or only craving measured using particular scales and in particular contexts.
A final limitation of the present research is analysis using SPSS imputed EM-generated estimations in place of missing values. It has been observed that analyses using these imputed values can be biased (von Hippel, 2004), and the sensitivity analyses found the parameter estimates calculated from the imputed data set to be lower than those generated by the non-imputed data, resulting in likely underestimation of the unique variance accounted for by craving in the predictions. However, in both studies, the overall results were highly consistent between the analyses using imputed values and those using non-imputed data, suggesting that the imputation method did not affect the overall study outcomes or conclusions.

9.6 Future directions

There are numerous possibilities for future research based on the results of these studies. The moderating effect of depression observed in the current research was small and the results were mixed. Further research to replicate the effect is therefore needed. Investigation using different measures of both depression and craving is also required, as well as testing of the effect with other substances aside from alcohol, and in other populations, such as inpatients or patients mandated to treatment. Other potential variables of influence, such as self-efficacy, coping and expectancies also need to be examined.

If the effect is found to replicate, further research is needed to dissect the mechanisms by which craving and depression interact to influence outcomes. Chapter 8 put forward a number of hypotheses including actions on self-efficacy and cognitive resources. Further research could explore these possibilities by examining these factors as mediators of the relationship between the interaction at baseline and post-treatment drinking. If high depression and high craving lead to greater alcohol use post-treatment
General discussion

because they work together to diminish self-efficacy, self-efficacy measured at follow-up should mediate the relationship between the interaction at baseline and drinking at the follow-up assessment. The possibility that high depression and high craving place additional demands on cognitive resources, leaving fewer resources to devote to problem solving and coping, could be tested experimentally by comparing the cognitive performance of people reporting clinically high levels of depressed mood and craving with the performance of people reporting non-clinical depression and craving. If the mechanism of influence is through additional burden on cognitive resources, performance between the two groups should differ and should be a mediator of the relationship between the interaction and post-treatment drinking.

The present research also postulated that the context of craving and depression measurement may be an important determinant of their predictive utility. Because craving tends to function as a state rather than a trait (Kavanagh et al., 2013), measuring it in a state that is relevant to the context being predicted, for example in the person’s usual environment and when efforts at control are being made, should result in improved predictive ability. Preliminary support for this notion was found in the present research, but these results need to be replicated in other populations, with other craving measures and with other outcomes. Just as the context of craving measurement appears to be important, the context of depression measurement may also matter. It may be reactive depressive symptoms, or negative affect triggered by deprivation, that is more important for prediction of later substance use than trait depression. Further research exploring the importance and influence of context of measurement is needed.
9.7 Conclusions

This thesis was designed to examine the influence of negative affect on the relationship between craving and treatment outcomes under the hypothesis that negative affect would augment the effects of craving, amplifying its predictive power and resulting in greater levels of post-treatment alcohol use. Craving was found to be predictive of post-treatment alcohol use in the presence of high levels of depressed mood, providing the first preliminary evidence of a possible moderating effect of depression on the relationship between pre-treatment craving and post-treatment alcohol use. The effects observed were however small and mixed, requiring further research to replicate the effect and to elucidate the mechanisms by which depression and craving may interact to influence later drinking. Possible mechanisms include an adverse effect on self-efficacy or excessive demand on cognitive resources.

The results of this research also emphasise the need to consider the context and timing of measurements. Craving’s ability to perform as a predictor in the present research may have been due in part to the fact that it was measured in a context that was relevant and similar to that of the outcome being predicted; that is, in the person’s usual environment and when attempting to control drinking in the absence of treatment support. This is relevant for the timing of outcome measurement also. The context of an immediate post-treatment assessment, when motivation, self-efficacy and coping skills are still high, is going to be different from that of later post-treatment assessment, when skills and motivations may become more challenged. This may explain why craving was predictive of 18-week (7 weeks post-treatment) drinking in Study 1 but not of 3-month (2 weeks post-treatment) drinking in Study 2.

The nature of the craving items emerging as significant in the predictions of both studies suggest that risk from craving may be linked to phasic episodes in particular,
General discussion

triggered by intrusive thoughts about alcohol. The frequency of these episodes, and the degree to which they are experienced as intrusive, may be a more important prognostic indicator of relapse than other aspects of craving. The nature of the significant items also suggest that risk from craving does not come just from the thoughts about alcohol, but from how those thoughts are managed. OCDS-O items pertaining to frequency of drinking thoughts and efforts to resist those thoughts were predictive of 12-month alcohol use in Study 1, while the ACE-Intrusion subscale, which assesses intrusiveness of alcohol thoughts and efforts to not think about drinking, was related to 12-month alcohol use in Study 2. These results appear to suggest that risk from craving may be heightened by adoption of maladaptive coping strategies, in particular, efforts to resist or suppress alcohol thoughts.

Both studies showed no prediction of outcomes from baseline depression. This could mean that the direct influence of depression on alcohol use is strongest proximally rather than distally. Alternatively, the influence of depression on post-treatment alcohol use, like craving, could be moderated or mediated by other variables not measured in the present research. Or perhaps the context of measurement is important, as appears to be the case for craving. Further research is needed to explore these possibilities.

Clinically, the present research suggests benefit in considering both craving and depression levels in patients presenting for treatment, as people reporting high levels of both may be at greater risk for relapse. The results also lend further support to the clinical practice of providing training in skills to effectively manage alcohol-related thoughts.
REFERENCES


References


References


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References


References


References


References


References


APPENDICES
APPENDIX A  STUDY 1 ASSESSMENT MEASURES

SECTION A: DEMOGRAPHICS

A1. Date of birth

A2. Age (years)

A3. Sex
1 = Male
2 = Female

A4. Country of birth – What country were you born in?
1 = Australia
2 = UK and Ireland
3 = Europe (including former USSR)
4 = Central and South America
5 = NZ, Pacific islands, PNG
6 = South East Asia
7 = Indian subcontinent and other Asia
8 = Middle East
9 = North Africa
10 = Central and Southern Africa
11 = North America
12 = Other

A5. Aboriginal / Torres Strait islander descent
Are you of Aboriginal or Torres straight Islander descent?
0 = No
1 = Yes

A7. Present Marital Status
What is your marital status? Have you been living with a partner for 6 months or more?
0 = Single, never married
1 = Married
2 = Defacto
3 = Separated
4 = Divorced
5 = Widowed
88 = NK

A8. Number of children
How many living children do you have? (include step-children)
00 = No children

A9. Children living with subject
How many dependent children under the age of 18 do you have living with you?
(Include step-children)
00 = No children

A10. Main carer for the children or not
Have you been the main carer for the children in the last 12 months?
0 = No
1 = Yes
88 = NK
99 = NA
Appendix A

A11. **Who do you live with?**
1=Parent(s)  
2=Spouse +/- children  
3=Defacto partner +/- children  
4=Friend(s)  
5=Alone  
6=Children without partner  
7=Relatives  
8=Other (specify____________________)  
9=No fixed address  
10=Institution

A12. **Accommodation during last month**

*Where have you been living during the last month?*

*How long have you lived there/been homeless?*

Code up to 3 types of accommodation in past month, if applicable

Code number of weeks in each accommodation in last month (01=<1 week)

<table>
<thead>
<tr>
<th>Accommodation #1</th>
<th>N. Wks</th>
<th>Accommodation #2</th>
<th>N. Wks</th>
<th>Accommodation #3</th>
<th>N. Wks</th>
</tr>
</thead>
<tbody>
<tr>
<td>01= Homeless / NFA</td>
<td></td>
<td>05= Institution: nursing home, lodge</td>
<td></td>
<td>10=Renting (private)</td>
<td></td>
</tr>
<tr>
<td>02= Crisis shelter or rooming house</td>
<td></td>
<td>06=Group home</td>
<td></td>
<td>11=Own home</td>
<td></td>
</tr>
<tr>
<td>03= Hostel</td>
<td></td>
<td>07=Supported housing</td>
<td></td>
<td>12=Family home</td>
<td></td>
</tr>
<tr>
<td>04= Institution: hospital</td>
<td></td>
<td>08=Hotel/rented room</td>
<td></td>
<td>88=Other (Specify________)</td>
<td></td>
</tr>
<tr>
<td>09=Renting (public - e.g. public housing)</td>
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</tbody>
</table>

A13. **Accommodation during the last 12 months** (excluding the past one month already rated)

*Where have you lived for more than a week during the last 12 months?*

*How long have you lived there/been homeless?*

Code up to 3 types of accommodation longest held (if applicable)

Code number of weeks in each type of accommodation during the previous 12 months (01=<1 week)

<table>
<thead>
<tr>
<th>Accommodation #1</th>
<th>N. Wks</th>
<th>Accommodation #2</th>
<th>N. Wks</th>
<th>Accommodation #3</th>
<th>N. Wks</th>
</tr>
</thead>
<tbody>
<tr>
<td>01= Homeless/NFA</td>
<td></td>
<td>05= Institution: nursing home, lodge</td>
<td></td>
<td>10=Renting (private)</td>
<td></td>
</tr>
<tr>
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<td></td>
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<td>03= Hostel</td>
<td></td>
<td>07=Supported housing</td>
<td></td>
<td>12=Family home</td>
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<tr>
<td>04= Institution: hospital</td>
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<td></td>
<td>88=Other (Specify________)</td>
<td></td>
</tr>
<tr>
<td>09=Renting (public - e.g. public housing)</td>
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<td></td>
<td></td>
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</tbody>
</table>

A14. **Age at leaving school**

*How old were you when you left school?*

00=Never went to school  
88=Still at school

A15. **Secondary school completion**

*Did you complete the highest year of secondary school available?*

0=No  
1=Yes  
99=NA
Appendix A

A16. Highest qualification obtained
What is the highest qualification you obtained?
1=Secondary school qualification  8=Undergraduate diploma
2=Nursing qualification      9=Bachelor degree
3=Teaching qualification    11=Masters degree/doctorate
4=Trade certificate/apprenticeship 12=Left school, no qualifications
5=Technician’s/ advanced certificate  88=Other
6=Certificate other than above   99=NA
7=Associate diploma

A17. During the past month, how frequently have you been taking part in any of the following jobs around the home? Would you say frequently, occasionally or not at all?
If living alone adapt questions to own activity, ie cooking for self.
0=Not at all
1=Occasionally
2=Frequently
88=NK
99= Not Applicable

Cooking for others……………………………………………………………………………………………………
Cleaning or washing up………………………………………………………………………………………………
Gardening………………………………………………………………………………………………………………
Shopping for household………………………………………………………………………………………………
Having meals together…………………………………………………………………………………………………
Watching TV program together……………………………………………………………………………………
Playing games…………………………………………………………………………………………………………
Doing Chores/Errands……………………………………………………………………………………………………
Other Activities (specify: _________________________________)……………………………………………………

A18. Participation in Household Activities
Over the past 12 months, have you been unable to do things that your family (or household) would normally expect of you?

What have you been unable to do?
Do others not let you do things? Why?
Is it that you lack interest in it?
Or have you been unable to do things because of physical/mental health or forgetfulness?

0= No dysfunction; has participated about as much as an average person of same sex/age group would under similar circumstances
1= Obvious dysfunction; household participation significantly reduced, due to lack of interest or incompetence
2= Severe dysfunction; no participation, self-alienated or excluded by others from daily household routine, or disruptive
88= Uncertain or impossible to assess
99= NA; does not share a household
Appendix A

A19. Availability of Friends

*How many people do you regard as friends?*

Ask the name of friend/s. Only count people outside the family. Some form of contact (face to face or phone conversation) over the last 12 months is required for considering a person a friend.

*How often have you been seeing them over the past month?*

*And over the past year?*

*What do you do together?*

0=None 3=Many
1=One 88=NK
2=A few 99=NA

A20. Perceived Need for Friends

*Do you feel that you have as many good friends as you need or would you like to have more?*

0=Does not need good friends at all
1=Needs and would like more friends
2=Has as many friends as needed
88=NK
99=NA

A21. Overall Socialising during past 12 months

*How have you been getting on with other people at work, neighbours, family members during the last 12 months?*

Did you go out to any social activities?
Did you meet any friends, or would you say that you are a bit reserved?
Did you make any phone calls to friends or other people you knew?
How much of the time did you spend alone, in your room, or just walking around on your own?
Did you feel lonely?

Rate overall socialising/isolation over past 12 months – rate isolation on its own merits, regardless of self imposed (eg. avoidance).
0= No dysfunction; has been socialising during the period as much as could be expected of an average person of same sex/age group and social background
1= Obvious dysfunction; may regard some people as friends but actual socialising with them is minimal, has been significantly reduced, sporadic participation in any organised activity
2= Severe dysfunction; no friends and no organised social activities, extremely restricted social relationships outside the household
88= Uncertain or impossible to assess
99=NA

A22. Social Withdrawal during last 12 months

*Would you say that over the past 12 months you enjoyed company a lot or preferred to be on your own?*

Did you find it difficult to mix or communicate with people?
Did you prefer to be left alone?
About how much of the time did you spend doing things by yourself?
Would you join in the company of others if encouraged to do so, or would you normally refuse even if asked?
Did the presence of other people annoy you?

Rate social withdrawal (ie. isolation which is not imposed by others or by the circumstances, but results mainly from subject’s active avoidance of social contacts).
0= No dysfunction; mixes and generally interacts with people as much or more than the average person of the same sex/age group would under similar circumstances
1= Obvious dysfunction; maintains a very restricted range of social contacts, generally avoids being with other people, but would mix with people if encouraged or pressured
2= Severe dysfunction; marked tendency to self-isolation, not responsive to encouragement, inaccessible, may frequently lock him/herself up or wander aimlessly
88= Uncertain or impossible to assess
99=NA
Appendix A

A23. Deterioration in Interpersonal Relationships
If you compare the past 12 months with previous years, do you think that your relations with friends, workmates or other persons may have gotten worse?
Did this happen because of your health or nervous problems?
Or because you lost interest or motivation?
Or because others have lost interest in maintaining a relationship with you?

0= No deterioration perceived in the past year compared to previous years
1= Deterioration perceived mainly attributed to subject’s own health/nervous problems or loss of interest
2= Deterioration perceived mainly attributed to other people’s loss of interest
3= Improvement perceived in past year compared to previous years
88=NK
98=NA

A24. Intimate Relationships
During the past 12 months have you had a close female/male friend – someone that you would share your thoughts and feelings with or think of as a best friend, or someone you might rely on for support when you need it?
Have you ever had such a special relationship?
How often do you see this special friend?

0= Not dysfunctional: has close and/or intimate affective relationship during the past 12 months
1= Obvious dysfunction: has had close friends or intimate relationship in the past but not during the last 12 months
2= Severe dysfunction: never had close friend or intimate relationship
88=Uncertain or impossible to assess
99=NA

(1) A25. Currently Employed
Do you have a job at present?
0=No job at present
1=Employment outside the home (full time job)
2=Employment outside the home (part time job)
3=Household
4=Studying
5=Retired
6=Volunteer
88=NK
99=NA

A26. If Unemployed, looking for work (past month)
At any time in the last 4 weeks have you been looking for full time or part time work?
0=No
1=Yes; looking for a full time job
2=Yes; looking for a part time job
88=NK
99=NA

A27. Participation in rehabilitation or day programme in last 12 months
When you were not in a psychiatric hospital, have you been involved in a rehabilitation or day program?(not including AOD)
0=No
1=Yes
88=NK
99=NA

Skip to A30 if 0

A28. Number of weeks in rehabilitation or day program in last 12 months
How many weeks did you attend rehab/day program at ________________?
(Range=0-52)
88=NK
99=NA

A29. Frequency of attendance of rehab/day program
How many days per week did you attend the rehab/day program at ________?
(Range=0-7)
88=NK
99=NA
Appendix A

A30. **Current Source of Income**

*What are your main sources of income in the past month?* Code up to 3 sources.

Source of current income #1
Source of current income #2
Source of current income #3

1=Wage/salary from employer  
2=Own business  
3=Family/spouse payment  
4=Government pension/cash benefit  
5=Maintenance/child support  
6=Superannuation/annuity  
7=Workers compensation/accident or sickness insurance  
8=other income (specify ___________________)  
88=NK  
99=NA

A31. **Pension/other benefits**

*Have you received any of the following pensions or benefits in the past month?*  
Read out the items below as a checklist. Code up to 3 types of benefit.  
Present=past month

Benefit #1
Benefit #2
Benefit #3

1=Age pension  
2=Service pension  
3=Disability support/invalid Pension  
4=Widow’s pension or wife’s pension  
5=Carer’s pension  
6=Sole parent’s pension  
7=Sickness allowance/benefit  
8=New start/job search/mature age allowance  
9=Unemployed benefit  
10=Special benefit  
11=Other (specify_______)  
88=NK  
99=NA

A32. **Interests**

*How have you been keeping up with what is happening in the world in the past month?*  
*Did you watch TV, or keep up with the news in other ways?*  
*Would you say that you have been trying to keep up with the national/international news? Can you give examples?*  
*Did you follow the football teams?*  
*Have you been involved in any particular interests over the past four weeks?*  
*Did you read any books, buy newspapers or magazines? Which ones?*  
*Have you developed any interests or hobbies?*

0= No dysfunction; seeks information, talks with people about local and world events, has a ‘world map’ as appropriate to sociocultural context  
1= Obvious dysfunction; less than average interest, no special efforts to obtain information, never reads anything, does not listen to radio or watch news on TV  
88=Uncertain or unable to assess  
99=NA (eg. moderate to severe intellectual handicap)
DEPRESSIVE DISORDERS

CODES:
? = unclear or inadequate information
1 = absent or false (symptom did not occur)
2 = subthreshold (e.g. symptom did occur but not for a 2-week period)
3 = threshold or true (symptom did occur)

A. Five or more of the following symptoms have been present during the same 2-week period and represent a change from previous functioning. At least one of these symptoms is (1) depressed mood or (2) loss of interest or pleasure.

<table>
<thead>
<tr>
<th>Symptom</th>
<th>1-6 months</th>
<th>7-12 months</th>
<th>Ever</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) Depressed mood</td>
<td></td>
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<tr>
<td>Has there been a period of time when you were feeling depressed or down most of the day, nearly every day? What was that like?</td>
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<tr>
<td>If YES, How long did it last (as long as 2 weeks)?</td>
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<tr>
<td>(2) Loss of interest or pleasure</td>
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</tr>
<tr>
<td>What about losing interest or pleasure in things you usually enjoyed?</td>
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<tr>
<td>If YES, was it nearly every day? How long did it last (as long as 2 weeks)?</td>
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</tbody>
</table>

If neither (1) nor (2) above is coded 3 for any time period, go on to D1 (Duration of illness)

<table>
<thead>
<tr>
<th>Symptom</th>
<th>1-6 months</th>
<th>7-12 months</th>
<th>Ever</th>
</tr>
</thead>
<tbody>
<tr>
<td>(3) Significant change of more than 5% in weight or change in appetite</td>
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<tr>
<td>During the time when you felt depressed, did you lose or gain any weight? How much? Were you trying to lose weight?</td>
<td></td>
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<tr>
<td>If NO, How was your appetite during this time? What about compared to your usual appetite? Did you have to force yourself to eat (more or less than usual)? Was that nearly every day?</td>
<td></td>
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<tr>
<td>(4) Insomnia or hypersomnia</td>
<td></td>
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<tr>
<td>How were you sleeping during this time? Did you have trouble falling asleep, waking frequently, trouble staying asleep, waking too early, OR sleeping too much during this time? How many hours a night compared to usual? Was that nearly every night?</td>
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<tr>
<td>(5) Psychomotor agitation or retardation</td>
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<tr>
<td>Were you so fidgety or restless during this time that you were unable to sit still? Was it so bad that other people noticed it? What did they notice? Was that nearly every day?</td>
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<tr>
<td>If NO, what about the opposite…talking or moving more slowly than is normal for you? Was that so bad that other people noticed? What did they notice?</td>
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</tbody>
</table>
Appendix A

<table>
<thead>
<tr>
<th></th>
<th>1-6 months</th>
<th>7-12 months</th>
<th>Ever</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>(6) Fatigue or loss of energy</strong>&lt;br&gt;During this time, what was your energy like?&lt;br&gt;Were you tired all the time? Was this nearly every day?</td>
<td></td>
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<td></td>
</tr>
<tr>
<td><strong>(7) Feelings of worthlessness or excessive, inappropriate guilt</strong>&lt;br&gt;During this time, how did you feel about yourself?&lt;br&gt;Worthless?&lt;br&gt;Was this nearly every day?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>If NO, what about feeling guilty about things you had done or not done?&lt;br&gt;Was this nearly every day?</td>
<td></td>
<td></td>
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</tr>
<tr>
<td><strong>NOTE:</strong> only code “1” or “2” for low self-esteem</td>
<td></td>
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<tr>
<td><strong>(8) Diminished ability to concentrate or make decisions</strong>&lt;br&gt;During this time, did you have trouble thinking or concentrating?&lt;br&gt;What kinds of things did it interfere with?&lt;br&gt;Was this nearly every day?</td>
<td></td>
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<tr>
<td>If NO, was it hard to make decisions about everyday things?&lt;br&gt;Was this nearly every day?</td>
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<tr>
<td><strong>(9) Reccurrent thoughts of death</strong>&lt;br&gt;During this time, were things so bad that you were thinking about death or that you would be better off dead?&lt;br&gt;What about thinking of hurting yourself?</td>
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<tr>
<td>If YES, did you do anything to hurt yourself?</td>
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**Criteria met for A?**

1=absent or false (no)<br>2=subthreshold<br>3=threshold or true (criteria met for A)

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<tr>
<th></th>
<th>1-6 months</th>
<th>7-12 months</th>
<th>Ever</th>
</tr>
</thead>
<tbody>
<tr>
<td>Score “3” for each time period where at least 5 of the above 9 criteria are coded “3”, and at least one of these is item (1) or (2).</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**NOTE** – Criterion B has been omitted from the SCID

**C. Symptoms cause clinically significant distress or impairment in social, occupational or other important areas of functioning.**

<table>
<thead>
<tr>
<th></th>
<th>1-6 months</th>
<th>7-12 months</th>
<th>Ever</th>
</tr>
</thead>
<tbody>
<tr>
<td>When you were feeling depressed, did it make it hard for you to do your work, take care of things at home or get along with other people?</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
D. Symptoms are not due to the direct physiological effects of a substance (e.g. drug abuse or medication).

NOTE: a score of 3 in this section indicates that symptoms are not due to physiological effects

<table>
<thead>
<tr>
<th></th>
<th>1-6 months</th>
<th>7-12 months</th>
<th>Ever</th>
</tr>
</thead>
<tbody>
<tr>
<td>Just before you began to feel depressed, were you physically ill?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>If YES, what did the doctor say?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>If NO, just before this began, were you using any medications? Had you made any changes in the amount you were taking?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>If STILL NO, what about drinking or using any street drugs? Has your depression occurred at time when you weren't using these substances?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NOTE: only score 1 if the symptoms are related to physiological effects</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

E. Symptoms are not better accounted for by bereavement.

NOTE: a score of 3 in this section indicates that symptoms are not due to bereavement

<table>
<thead>
<tr>
<th></th>
<th>1-6 months</th>
<th>7-12 months</th>
<th>Ever</th>
</tr>
</thead>
<tbody>
<tr>
<td>Did all this begin soon after someone close to you died?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NOTE: only score 1 if the symptoms are related to bereavement issues</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

SCID Criteria met for Major Depressive Episode?

1= absent or false (no major depressive episode)
2= subthreshold
3= threshold or true (criteria met for major depressive episode)

<table>
<thead>
<tr>
<th></th>
<th>1-6 months</th>
<th>7-12 months</th>
<th>Ever</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major Depressive Episode Criteria: Criteria A, C, D, and E are coded “3”</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(2) D1. Duration of Illness in Weeks (since first episode till now)
Max=99

(3) D2. Impairment/Incapacity During Disorder
Rate of the basis of worst episode
0= No impairment
1= SUBJECTIVE impairment only (at work, school or in social functioning)
2= Evidence of OBJECTIVE impairment in major life role with definite reduction in productivity and/or criticism has been received
3= INPATIENT treatment (any duration) has been received or no function at all in major life role for more than 2 days.
Appendix A

D3. Current medication
In the past month, have you been taking any medication or injection that had been prescribed by a doctor for your mental health or nerves or alcohol/other drug use?
0=No
1=Yes

In the past month, have you been taking any non-prescription medication or supplements for your mental health or nerves or alcohol/other drug use? (e.g. St John's Wort, vitamins etc.)
0=No
1=Yes

Skip to QUESTION D10 if 0

D4. What medication are you currently taking?
Show person the CHART. If person is unable to identify drug(s) on the chart, read out the names of the drugs. Write drug code R (from chart) into the boxes provided below. If only “red pills” identified, code 88=NK. Code up to 5 drugs. Code only if person has been on a given drug for >1 month. (77= drug code if not on list)

<table>
<thead>
<tr>
<th>Drug Name (specify drug name, and dose)</th>
<th>Drug Code</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug #1: ____________________________</td>
<td></td>
</tr>
<tr>
<td>Drug #2: ____________________________</td>
<td></td>
</tr>
<tr>
<td>Drug #3: ____________________________</td>
<td></td>
</tr>
<tr>
<td>Drug #4: ____________________________</td>
<td></td>
</tr>
<tr>
<td>Drug #5: ____________________________</td>
<td></td>
</tr>
</tbody>
</table>

D5. Perceived benefits
Would you say that [quote each drug identified and coded below] was helpful?
What would happen if you stopped [quote ‘helpful’ drug]?

0=Not helpful at all
1=Helpful
2=Very helpful
88=Impossible to assess
99=NA

<table>
<thead>
<tr>
<th>Drug Name (specify)</th>
<th>Benefit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug #1</td>
<td></td>
</tr>
<tr>
<td>Drug #2</td>
<td></td>
</tr>
<tr>
<td>Drug #3</td>
<td></td>
</tr>
<tr>
<td>Drug #4</td>
<td></td>
</tr>
<tr>
<td>Drug #5</td>
<td></td>
</tr>
</tbody>
</table>
SECTION R: TLFB (Time Line Follow Back)

What I would like to do now is to write down all your drinking over the past two weeks. I want to get an idea of how much alcohol you had on each day during this time. The idea is to write down then number of drinks you had each day (on the calendar). On days when you did not drink any alcohol, you write “0”. For days when you had something drink, use the table below to calculate the number of standard drinks you had, and write that on the calendar.

Make sure that something is written in for each day on the calendar. If something happens every week, e.g. you go to the pub every Friday night or you go to watch a game every Saturday, then use that to help you remember. If you can’t remember exactly what happened then GIVE IT YOUR BEST GUESS. Start with what you had yesterday and then fill out any other days that you can remember easily, then try to fill out the rest.

Use the following as a guide to the number of standard drinks consumed - Ask about all categories.

<table>
<thead>
<tr>
<th>Wine</th>
<th>Spirits</th>
<th>Full Strength Beer</th>
<th>Light Beer</th>
<th>Fortified Wine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glass (100mL)</td>
<td>30ml nips</td>
<td>Schooner (15oz/425mL)</td>
<td>Schooner (15oz/425mL)</td>
<td>Port Glass (60mL)</td>
</tr>
<tr>
<td>[1]</td>
<td>[1]</td>
<td>[1.5]</td>
<td>[0.75]</td>
<td>[1]</td>
</tr>
<tr>
<td>750ml bottles</td>
<td>750ml bottles</td>
<td>Can</td>
<td>Can</td>
<td>750ml bottles</td>
</tr>
<tr>
<td>[7.5]</td>
<td>[25]</td>
<td>[1.3]</td>
<td>[0.7]</td>
<td>[10]</td>
</tr>
<tr>
<td>Flagon (2 Litres)</td>
<td>UDL (cans)</td>
<td>Stubby</td>
<td>Stubby</td>
<td>2 lt. flagons</td>
</tr>
<tr>
<td>[20]</td>
<td>[1.3]</td>
<td>[1.3]</td>
<td>[0.7]</td>
<td>[32]</td>
</tr>
<tr>
<td>___lt. casks</td>
<td>___lt. casks</td>
<td>750ml bottles (longneck)</td>
<td>750ml bottles (longneck)</td>
<td></td>
</tr>
<tr>
<td>[10 per litre]</td>
<td>[10 per litre]</td>
<td>[2.5]</td>
<td>[2]</td>
<td></td>
</tr>
</tbody>
</table>

N.B. Figures in square brackets are numbers of standard drinks in one unit

<table>
<thead>
<tr>
<th>Enter the days/dates of the fortnight prior to assessment Day</th>
<th>What Happened that Day?</th>
<th>How Many Standard Drinks Were Consumed?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sunday</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Saturday</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Friday</td>
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<td></td>
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<tr>
<td>Thursday</td>
<td></td>
<td></td>
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<tr>
<td>Wednesday</td>
<td></td>
<td></td>
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<tr>
<td>Tuesday</td>
<td></td>
<td></td>
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<tr>
<td>Monday</td>
<td></td>
<td></td>
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<tr>
<td>Sunday</td>
<td></td>
<td></td>
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<tr>
<td>Saturday</td>
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<td>Friday</td>
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<td>Wednesday</td>
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<td>Monday</td>
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<td>Sunday</td>
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<td>Saturday</td>
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<td>Wednesday</td>
<td></td>
<td></td>
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<tr>
<td>Tuesday</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Monday</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

233
Appendix A

SECTION S: AUDIT

Please circle the answer that is correct for you for the last 6 months.

1. How often do you have a drink containing alcohol?

Specify exact frequency: ______________________________________________

Then, code according to following:
0 = never
1 = monthly or less
2 = 2-4 times a month
3 = 2-3 times a week
4 = 4 or more times a week

2. How many drinks containing alcohol do you have on a typical day when you are drinking?

Specify exact number of standard drinks:
______________________________________________

Then, code according to following:
0 = 1 to 2
1 = 3 to 4
2 = 5 to 6
3 = 7 to 9
4 = 10 or more

3. How often do you have six or more drinks on one occasion?

Specify exact frequency: ______________________________________________

Then, code according to following:
0 = never
1 = less than monthly
2 = monthly
3 = weekly
4 = daily or almost daily

4. How often during the last 6 months have you found that you were not able to stop drinking once you had started?

0 = never
1 = less than monthly
2 = monthly
3 = weekly
4 = daily or almost daily

5. How often during the last 6 months have you failed to do what was normally expected from you because of drinking?

0 = never
(a) 1 = less than monthly
(b) 2 = monthly
(c) 3 = weekly
(d) 4 = daily or almost daily
Appendix A

6. How often during the last 6 months have you needed a first drink in the morning to get yourself going after a heavy drinking session?
   0 = never
   1 = less than monthly
   2 = monthly
   3 = weekly
   4 = daily or almost daily

7. How often during the last 6 months have you had a feeling of guilt or remorse after drinking?
   0 = never
   1 = less than monthly
   2 = monthly
   3 = weekly
   4 = daily or almost daily

8. How often during the last 6 months have you been unable to remember what happened the night before because you had been drinking?
   0 = never
   1 = less than monthly
   2 = monthly
   3 = weekly
   4 = daily or almost daily

9. Have you or someone else been injured as a result of your drinking?
   0 = no
   2 = yes, but not in the last 6 months
   4 = yes, during the last 6 months

10. Has a relative or friend or a doctor or other health worker, been concerned about your drinking or suggested you cut down?
    0 = no
    2 = yes, but not in the last 6 months
    4 = yes, during the last 6 months

    AUDIT TOTAL =


## Appendix A

<table>
<thead>
<tr>
<th>Section W: SCID (Alcohol Use)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CODING FOR THIS SECTION...</strong></td>
</tr>
<tr>
<td>? = Inadequate information</td>
</tr>
<tr>
<td>1 = Absent or false</td>
</tr>
<tr>
<td>2 = Subthreshold</td>
</tr>
<tr>
<td>3 = Threshold or true</td>
</tr>
</tbody>
</table>

### Alcohol Abuse Criteria

A maladaptive pattern substance use leading to clinically significant impairment or distress, as manifested by 1 or more of the following occurring within a 12 month period:

1. **Recurrent alcohol use resulting in a failure to fulfill major role obligations at work, school, or home (e.g. repeated absence or poor work performance related to alcohol use; alcohol-related absences, suspensions, or expulsions from school; neglect of children or household)**

<table>
<thead>
<tr>
<th>1-6 months</th>
<th>7-12 months</th>
<th>Ever</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Have you ever missed work or school because you were intoxicated, high or very hung over? (How often? What about doing a bad job at work or failing courses at school because of your drinking?)

**If NO:** What about not keeping your house clean or not taking proper care for your children because of your drinking? (How often?)

**If YES to either of above:** How often? (Over what period of time?)

2. **Recurrent alcohol use in situations in which it is physically hazardous (e.g. driving a car, operating a machine when impaired by alcohol)**

<table>
<thead>
<tr>
<th>1-6 months</th>
<th>7-12 months</th>
<th>Ever</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Did you ever drink in a situation in which it might have been dangerous to drink at all? (Did you ever drive while you were really too drunk to drive?)

**If YES and UNKNOWN:** How many times?

3. **Recurrent alcohol-related legal problems (e.g. arrests for alcohol-related disorderly conduct)**

<table>
<thead>
<tr>
<th>1-6 months</th>
<th>7-12 months</th>
<th>Ever</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Has your drinking gotten you into trouble with the law?

**If YES and UNKNOWN:** How often? (Over what period of time?)
(4) Continued alcohol use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of alcohol (e.g. arguments with spouse about consequences of intoxication, physical fights)

<table>
<thead>
<tr>
<th>If not already known: Has your drinking caused problems with other people, such as with family members, friends or people at work? (Have you ever gotten into physical fights when you were drinking? What about having bad arguments about your drinking?)</th>
<th>1-6 months</th>
<th>7-12 months</th>
<th>Ever</th>
</tr>
</thead>
<tbody>
<tr>
<td>If YES: Did you keep on drinking anyway? (Over what period of time?)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Alcohol Abuse Present?

1 = Absent or False
2 = Subthreshold
3 = Threshold or True

<table>
<thead>
<tr>
<th>1-6 months</th>
<th>7-12 months</th>
<th>Ever</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Appendix A

**Alcohol Dependence Criteria**

A maladaptive pattern of alcohol use, leading to clinically significant impairment or distress, as manifested by 3 or more of the following occurring at any time in the same 12 month period:

1. **Alcohol is often taken in larger amounts OR over a longer period than was intended**

<table>
<thead>
<tr>
<th>1-6 months</th>
<th>7-12 months</th>
<th>Ever</th>
</tr>
</thead>
<tbody>
<tr>
<td>Have you often found that when you started drinking you ended up drinking much more than you were planning to?</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>If NO:</strong> What about drinking for a much longer period of time than you were planning to?</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

2. **There is a persistent desire OR unsuccessful efforts to cut down or control substance use**

<table>
<thead>
<tr>
<th>1-6 months</th>
<th>7-12 months</th>
<th>Ever</th>
</tr>
</thead>
<tbody>
<tr>
<td>Have you tried to cut down or stop drinking alcohol?</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>If YES:</strong> Did you ever actually stop drinking altogether? (How many times did you try to cut down or stop drinking altogether?)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>If NO:</strong> Did you want to stop or cut down? (Is this something you kept worrying about?)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

3. **A great deal of time is spent on activities necessary to obtain alcohol, use alcohol, or recover from its effects**

<table>
<thead>
<tr>
<th>1-6 months</th>
<th>7-12 months</th>
<th>Ever</th>
</tr>
</thead>
<tbody>
<tr>
<td>Have you spent a lot of time drinking, being high, or hung over?</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

4. **Important social, occupational, or recreational activities given up or reduced because of alcohol use**

<table>
<thead>
<tr>
<th>1-6 months</th>
<th>7-12 months</th>
<th>Ever</th>
</tr>
</thead>
<tbody>
<tr>
<td>Have you had times when you would drink so often that you started to drink instead of working or spending time with your family or friends or engaging in other important activities, such as sports, gardening, or playing music?</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

5. **Alcohol use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by alcohol (e.g. continued drinking despite recognition that an ulcer was made worse by alcohol consumption)**

<table>
<thead>
<tr>
<th>1-6 months</th>
<th>7-12 months</th>
<th>Ever</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>If no already known:</strong> Has your drinking ever caused any psychological problems like making you depressed or anxious, making it difficult to sleep, or causing “blackouts”?</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>If not already known:</strong> Has your drinking ever caused significant physical problems or made a physical problem worse?</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>If YES to either of above:</strong> Did you keep on drinking anyway?</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Appendix A

(6) Tolerance, as defined by either of the following:
   (a) A need for markedly increased amounts of alcohol to achieve intoxication or desired effect,
   (b) Markedly diminished effect with continued use of the same amount of alcohol.

<table>
<thead>
<tr>
<th>Have you found that you needed to drink a lot more in order to get the feeling you wanted than you did when you first started drinking?</th>
<th>1-6 months</th>
<th>7-12 months</th>
<th>Ever</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>If YES:</strong> How much more?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>If NO:</strong> What about finding that when you drank the same amount, it had much less effect than before?</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(7) Withdrawal, as manifested by either (a) or (b):
   (a) at least 2 of the following: automatic hyperactivity (e.g. sweating or pulse rate greater than 100); increased hand tremor; insomnia; nausea or vomiting; psychomotor agitation; anxiety; grand mal seizures; transient visual, tactile or auditory hallucinations or illusions
   (b) Alcohol (or a substance from the sedative/hypnotic/anxiolytic class) taken to relieve or avoid withdrawal symptoms

<table>
<thead>
<tr>
<th>Have you ever had any withdrawal symptoms when you cut down or stopped drinking like....</th>
<th>1-6 months</th>
<th>7-12 months</th>
<th>Ever</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sweating or racing heart?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hand shakes?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trouble sleeping?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Feeling nauseated or vomiting?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Feeling agitated?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Feeling anxious?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>How about a seizure or seeing, feeling, or hearing things that weren’t really there?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>If NO:</strong> Have you ever started the day with a drink, or did you often drink or take some other drug or medication to keep yourself from getting the shakes or becoming sick?</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Alcohol dependence with physiological dependence present?**
At least three dependence items coded “3” and items occurred within the same 12 month period

1 = Absent or False
2 = Subthreshold
3 = Threshold or True

<table>
<thead>
<tr>
<th>1-6 months</th>
<th>7-12 months</th>
<th>Ever</th>
</tr>
</thead>
</table>

**NOTE:** If No, diagnose Alcohol Dependence without physiological dependence
Appendix A

SR5 OC Drinking Scale

The questions below ask you about your drinking alcohol and your attempts to control your drinking. Please enter the number of the statement that best applies to you in the space provided:

1. **How much of your time when you’re not drinking is occupied by ideas, thoughts, impulses, or images related to drinking?**
   - 0=none
   - 1=less than 1 hour a day
   - 2=1-3 hours a day
   - 3=4-8 hours a day
   - 4=Greater than 8 hours a day

2. **How frequently do these thoughts occur?**
   - 0=never
   - 1=No more than 8 times a day
   - 2=More than 8 times a day, but most hours of the day are free of those thoughts
   - 3=More than 8 times a day, and during most hours of the day
   - 4=Thoughts are too numerous to count, and an hour rarely passes without several such thoughts

   INSERT THE HIGHER SCORE OF QUESTIONS 1 and 2 HERE:

3. **How much do these ideas, thoughts, impulses or images related to drinking interfere with your social or work (or role) functioning? Is there anything you don’t or can’t do because of them?** [If you are not currently working, how much of your performance would be affected if you were working?]
   - 0=Thoughts of drinking never interfere – I can function normally
   - 1=Thoughts of drinking slightly interfere with my social or occupational activities, but my overall performance is not impaired
   - 2=Thoughts of drinking definitely interfere with my social or occupational performance, but I can still manage
   - 3=Thoughts of drinking cause substantial impairment in my social or occupational performance
   - 4=Thoughts of drinking interfere completely with my social or work performance

4. **How much distress or disturbance do these ideas, thoughts, impulses or images related to drinking cause you when you are not drinking?**
   - 0=none
   - 1=Mild, infrequent, and not too disturbing
   - 2=Moderate, frequent, and disturbing, but still manageable
   - 3=Severe, very frequent, and very disturbing
   - 4=Extreme, nearly constant, and disabling distress
5. **How much of an effort do you make to resist these thoughts or try to disregard or turn your attention away from these thoughts as they enter your mind when you are not drinking?** [rate your efforts made to resist these thoughts, not your success or failure in actually controlling them]

   - 0=My thoughts are so minimal, I don’t need to actively resist. If I have thoughts, I make an effort to always resist
   - 1=I try to resist most of the time
   - 2=I make some effort to resist
   - 3=I give in to all such thoughts without attempting to control them, but I do so with some reluctance
   - 4=I completely and willingly give in to such thoughts

6. **How successful are you in stopping or diverting these thoughts when you are not drinking?**

   - 0=I am completely successful in stopping or diverting such thoughts
   - 1=I am usually able to stop or divert such thoughts with some effort and concentration
   - 2=I am sometimes able to stop or divert such thoughts
   - 3=I am rarely successful in stopping such thoughts and can only divert such thoughts with difficulty
   - 4=I am rarely able to divert such thoughts even momentarily

7. **How many drinks do you drink each day?**

   - 0=none
   - 1=less than 1 drink per day
   - 2=1-2 drinks per day
   - 3=3-7 drinks per day
   - 4=8 or more drinks per day

8. **How many days each week do you drink?**

   - 0=none
   - 1=No more than 1 day per week
   - 2=2-3 days per week
   - 3=4-5 days per week
   - 4=6-7 days per week

**INSERT THE HIGHER SCORE OF QUESTIONS 7 and 8 HERE:**

__________________________________________________________________________________________
Demographic characteristics of the analysed sample ($N = 260$).

<table>
<thead>
<tr>
<th>Demographic Characteristics</th>
<th>$M (SD, range)$ or $n$ (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age (years)</td>
<td>45.42 (10.86, 20–73)</td>
</tr>
<tr>
<td>Male</td>
<td>132/260 (51.0%)</td>
</tr>
<tr>
<td>Married/Partnered</td>
<td>86/260 (33.1%)</td>
</tr>
<tr>
<td>Mean age left school (years) (n=258)</td>
<td>16.10 (1.41, 11–21)</td>
</tr>
<tr>
<td>Post-school qualification</td>
<td>123/254 (48.4%)</td>
</tr>
<tr>
<td>Receiving welfare support</td>
<td>121/259 (46.5%)</td>
</tr>
</tbody>
</table>
# Appendix C

## Results of Study 1 Missing Value Analysis and Imputation

### Results of follow-up attrition tests

<table>
<thead>
<tr>
<th>Variable</th>
<th>18 weeks</th>
<th>12 Months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>df</td>
<td>$F$</td>
</tr>
<tr>
<td>Number of treatment sessions</td>
<td>1, 258</td>
<td>10.04**</td>
</tr>
<tr>
<td>Age</td>
<td>1, 258</td>
<td>1.14</td>
</tr>
<tr>
<td>Education</td>
<td>1, 256</td>
<td>0.44</td>
</tr>
<tr>
<td>OCDS-O score</td>
<td>1, 205</td>
<td>0.40</td>
</tr>
<tr>
<td>BDI-II score</td>
<td>1, 258</td>
<td>0.66</td>
</tr>
<tr>
<td>Average weekly drinking</td>
<td>1, 258</td>
<td>4.83*</td>
</tr>
</tbody>
</table>

|                                | df, N    | $\chi^2$  | $\chi^2$  |
| Treatment allocation           | 3, 260   | 5.98      | 1.79      |
| Gender                         | 1, 260   | 0.01      | 0.33      |
| Relationship status            | 1, 260   | 0.07      | 0.12      |
| Current depressive episode     | 1, 257   | 0.18      | 0.91      |
| Current antidepressants        | 1, 257   | 1.42      | 3.32      |

* $p < .05$ ** $p < .01$ *** $p < .001$

### Results of Expectation-Maximisation Estimation Imputation

<table>
<thead>
<tr>
<th>Variable</th>
<th>18-week mean</th>
<th>std. dev</th>
<th>12-month mean</th>
<th>std. dev</th>
</tr>
</thead>
<tbody>
<tr>
<td>Original Average weekly drinks</td>
<td>38.56</td>
<td>40.59</td>
<td>37.91</td>
<td>37.50</td>
</tr>
<tr>
<td>Imputed Average weekly drinks</td>
<td>40.66</td>
<td>42.47</td>
<td>39.29</td>
<td>37.54</td>
</tr>
<tr>
<td>Original Average weekly binges</td>
<td>2.74</td>
<td>2.64</td>
<td>2.86</td>
<td>2.76</td>
</tr>
<tr>
<td>Imputed Average weekly binges</td>
<td>2.81</td>
<td>2.67</td>
<td>2.92</td>
<td>2.76</td>
</tr>
</tbody>
</table>
APPENDIX D  ALCOHOL CRAVING EXPERIENCE QUESTIONNAIRE

ACE-Strength scale shown on next page
## Think about the time you MOST WANTED an alcoholic drink during the LAST WEEK.

When was that? ____ day at ___am/pm. How long did it last? Write a number here: ____ minutes (OR ___secs)

For each item, **CIRCLE A MARK** to make your rating.

<table>
<thead>
<tr>
<th>At that time…</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. …how strongly did you want a drink?</td>
</tr>
<tr>
<td>2. …how much did you feel you needed a drink?</td>
</tr>
<tr>
<td>3. …how strong was the urge to drink?</td>
</tr>
<tr>
<td>4. …how hard was it to think about anything else?</td>
</tr>
<tr>
<td>5. …how hard was it to get other things done?</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>At that time, how vividly did you…</th>
</tr>
</thead>
<tbody>
<tr>
<td>6. …imagine a drink?</td>
</tr>
<tr>
<td>7. …picture alcohol or drinking?</td>
</tr>
<tr>
<td>8. …imagine what it would taste like?</td>
</tr>
<tr>
<td>9. …imagine what it would smell like?</td>
</tr>
<tr>
<td>10. …imagine what it would feel like in your mouth or throat?</td>
</tr>
<tr>
<td>11. …imagine how your body would feel if you had a drink?</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>At that time, when you thought about alcohol…</th>
</tr>
</thead>
<tbody>
<tr>
<td>12. …how unpleasant or distressing were the thoughts?</td>
</tr>
<tr>
<td>13. …how guilty or worried were you about the thoughts?</td>
</tr>
<tr>
<td>14. …how much worse did you think things would be if you had a drink?</td>
</tr>
<tr>
<td>15. …how hard were you trying not to think about alcohol?</td>
</tr>
<tr>
<td>16. …how intrusive were the thoughts?</td>
</tr>
</tbody>
</table>
Appendix D

ACE-Frequency scale shown on next page
Appendix D

\textbf{Now, we want you to answer some similar questions. But this time, please answer HOW OFTEN these things happened over the LAST WEEK.}

\textbf{Over the last week, HOW OFTEN did you …}

<table>
<thead>
<tr>
<th>Question</th>
<th>Not at all</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th>Constantly</th>
</tr>
</thead>
<tbody>
<tr>
<td>17. …want a drink?</td>
<td>Not at all</td>
<td></td>
<td></td>
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<td>Constantly</td>
</tr>
<tr>
<td>18. …think about needing a drink?</td>
<td>Not at all</td>
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<td>Constantly</td>
</tr>
<tr>
<td>19. …have an urge to drink?</td>
<td>Not at all</td>
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<td>Constantly</td>
</tr>
<tr>
<td>20. …find it hard to think about anything else?</td>
<td>Not at all</td>
<td></td>
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<td>Constantly</td>
</tr>
<tr>
<td>21. …find it hard to get other things done?</td>
<td>Not at all</td>
<td></td>
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<td>Constantly</td>
</tr>
<tr>
<td>22. …imagine a drink?</td>
<td>Not at all</td>
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<td>Constantly</td>
</tr>
<tr>
<td>23. …picture alcohol or drinking?</td>
<td>Not at all</td>
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<td>Constantly</td>
</tr>
<tr>
<td>24. …imagine what it would taste like?</td>
<td>Not at all</td>
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<td>Constantly</td>
</tr>
<tr>
<td>25. …imagine what it would smell like?</td>
<td>Not at all</td>
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<td></td>
<td>Constantly</td>
</tr>
<tr>
<td>26. …imagine what it would feel like in your mouth or throat?</td>
<td>Not at all</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td>Constantly</td>
</tr>
<tr>
<td>27. …imagine how your body would feel if you had a drink?</td>
<td>Not at all</td>
<td></td>
<td></td>
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<td>Constantly</td>
</tr>
</tbody>
</table>

\textbf{Over the last week, how often did you…}

<table>
<thead>
<tr>
<th>Question</th>
<th>Not at all</th>
<th></th>
<th></th>
<th></th>
<th></th>
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<th>Constantly</th>
</tr>
</thead>
<tbody>
<tr>
<td>28. …were the thoughts unpleasant or distressing?</td>
<td>Not at all</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td>Constantly</td>
</tr>
<tr>
<td>29. …were you guilty or worried about the thoughts?</td>
<td>Not at all</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td>Constantly</td>
</tr>
<tr>
<td>30. …did you think things would be worse if you had a drink?</td>
<td>Not at all</td>
<td></td>
<td></td>
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<td>Constantly</td>
</tr>
<tr>
<td>31. …were you trying not to think about alcohol?</td>
<td>Not at all</td>
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<td>Constantly</td>
</tr>
<tr>
<td>32. …were the thoughts intrusive?</td>
<td>Not at all</td>
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<td>Constantly</td>
</tr>
<tr>
<td>33. …did thoughts about alcohol seem to pop into your head?</td>
<td>Not at all</td>
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<td></td>
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<td>Constantly</td>
</tr>
<tr>
<td>34. …did you strongly want or need a drink?</td>
<td>Not at all</td>
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<td></td>
<td>Constantly</td>
</tr>
<tr>
<td>35. …did you have a strong urge to have a drink?</td>
<td>Not at all</td>
<td></td>
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<td>Constantly</td>
</tr>
</tbody>
</table>

\textbf{When you thought about alcohol over the last week, how often…}

<table>
<thead>
<tr>
<th>Question</th>
<th>Not at all</th>
<th></th>
<th></th>
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<th></th>
<th></th>
<th></th>
<th></th>
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<th></th>
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<td>30. …did you think things would be worse if you had a drink?</td>
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<td></td>
<td>Constantly</td>
</tr>
<tr>
<td>31. …were you trying not to think about alcohol?</td>
<td>Not at all</td>
<td></td>
<td></td>
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<td>Constantly</td>
</tr>
<tr>
<td>32. …were the thoughts intrusive?</td>
<td>Not at all</td>
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<td></td>
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<td>Constantly</td>
</tr>
<tr>
<td>34. …did you strongly want or need a drink?</td>
<td>Not at all</td>
<td></td>
<td></td>
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<td>Constantly</td>
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</tbody>
</table>
APPENDIX E  ALCOHOL CRAVING EXPERIENCE QUESTIONNAIRE
AFTER ITEM REDUCTION

ACE-Strength
(with original question numbers)

Intensity factor items

1. …how strongly did you want a drink?
2. …how much did you feel you needed a drink?
3. …how strong was the urge to drink?
4. …how hard was it to think about anything else?

Imagery factor items

7. …picture alcohol or drinking?
8. …imagine what it would taste like?
9. …imagine what it would smell like?
10. …imagine what it would feel like in your mouth or throat?
11. …imagine how your body would feel if you had a drink?

Intrusion factor items

15. …how hard were you trying not to think about alcohol?
16. …how intrusive were the thoughts?
ACE-Frequency
(with original question numbers)

Intensity factor items

17. …want a drink?
18. …think about needing a drink?
20. …find it hard to think about anything else?
35. …did you have a strong urge to have a drink?

Imagery factor items

23. …picture alcohol or drinking?
24. …imagine what it would taste like?
25. …imagine what it would smell like?
26. …imagine what it would feel like in your mouth or throat?
27. …imagine how your body would feel if you had a drink?

Intrusion factor items

31. …were you trying not to think about alcohol?
32. …were the thoughts intrusive?
Please note: This is only to be used in Session 1 if the client has not completed their drinking record card from pre-test assessment.

Drinking In The Past 2 –3 Weeks

Time-line Followback

- What we would like you to do is write down all your drinking over the past couple of weeks
- We want to get an idea of how much alcohol you had on each day during this time
- The idea is to write down the number of drinks you had each day (on the calendar over the page)
- On days when you did not drink any alcohol you write “0”
- For days when you had something to drink, you write in the number of standard drinks you had
- Make sure something is written in for each day on the calendar
- If you have a diary you can use it to help you remember the day
- If something happens every week – eg you go to the pub every Friday night or you go to watch a game every Saturday, use that to help you remember
- If you can’t remember exactly what you had to drink, GIVE IT YOUR BEST GUESS
- Start with what you had yesterday, and then fill out any other days that you can remember easily, then try to fill in the rest
<table>
<thead>
<tr>
<th>Date:</th>
<th>Monday</th>
<th>Tuesday</th>
<th>Wednesday</th>
<th>Thursday</th>
<th>Friday</th>
<th>Saturday</th>
<th>Sunday</th>
</tr>
</thead>
<tbody>
<tr>
<td>What Happened</td>
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<td></td>
</tr>
<tr>
<td>How Many Drinks</td>
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</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Date:</th>
<th>Monday</th>
<th>Tuesday</th>
<th>Wednesday</th>
<th>Thursday</th>
<th>Friday</th>
<th>Saturday</th>
<th>Sunday</th>
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</thead>
<tbody>
<tr>
<td>What Happened</td>
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<tr>
<td>How Many Drinks</td>
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</table>

<table>
<thead>
<tr>
<th>Date:</th>
<th>Monday</th>
<th>Tuesday</th>
<th>Wednesday</th>
<th>Thursday</th>
<th>Friday</th>
<th>Saturday</th>
<th>Sunday</th>
</tr>
</thead>
<tbody>
<tr>
<td>What Happened</td>
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</tr>
<tr>
<td>How Many Drinks</td>
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</tr>
</tbody>
</table>

Type and brand of usual drink/s (e.g., VB stubbies, Johnny Walker UDLs): ____________________________________________________________

Favourite drink/s: ____________________________________________________________
Appendix F

**OCDS**

1. How much of your time when you’re not drinking is occupied by ideas, thoughts, impulses, or images related to drinking?
   0 None
   1 Less than 1 hour a day
   2 1-3 hours a day
   3 4-8 hours a day
   4 Greater than 8 hours a day

2. How frequently do these thoughts occur?
   0 Never
   1 No more than 8 times a day.
   2 More than 8 times a day, but most hours of the day are free of those thoughts.
   3 More than 8 times a day and during most hours of the day.
   4 Thoughts are too numerous to count and an hour rarely passes without several such thoughts occurring.

3. How much do these ideas, thoughts, impulses, or images related to drinking interfere with your social or work (or role) functioning? Is there anything you don’t or can’t do because of them? [If you are not currently working, how much of your performance would be affected if you were working?]
   0 Thoughts of drinking never interfere – I can function normally.
   1 Thoughts of drinking slightly interfere with my social or occupational activities, but my overall performance is not impaired.
   2 Thoughts of drinking definitely interfere with my social or occupational performance, but I can still manage.
   3 Thoughts of drinking cause substantial impairment in my social or occupational performance.
   4 Thoughts of drinking interfere completely with my social or work performance.

4. How much distress or disturbance do these ideas, thoughts, impulses, or images related to drinking cause you when you’re not drinking?
   0 None.
   1 Mild, infrequent, and not too disturbing.
   2 Moderate, frequent, and disturbing, but still manageable.
   3 Severe, very frequent, and very disturbing.
   4 Extreme, nearly constant, and disabling distress.
5. How much of an effort do you make to resist these thoughts or try to disregard or turn your attention away from these thoughts as they enter your mind when you’re not drinking? (Rate your effort made to resist these thoughts, NOT your success or failure in actually controlling them.)

0  My thoughts are so minimal, I don’t need to actively resist. If I have thoughts, I make an effort to always resist.
1  I try to resist most of the time.
2  I make some effort to resist.
3  I give in to all such thoughts without attempting to control them, but I do so with some reluctance.
4  I completely and willingly give in to all such thoughts.

6. How successful are you in stopping or diverting these thoughts when you’re not drinking?

0  I am completely successful in stopping or diverting such thoughts.
1  I am usually able to stop or divert such thoughts with some effort and concentration.
2  I am sometimes able to stop or divert such thoughts.
3  I am rarely successful in stopping such thoughts and can only divert such thoughts with difficulty.
4  I am rarely able to divert such thoughts even momentarily.

7. How many drinks do you drink each day?

0  None
1  Less than 1 drink per day
2  1 - 2 drinks per day
3  3 - 7 drinks per day
4  8 or more drinks per day

8. How many days each week do you drink?

0  None
1  No more than 1 day per week
2  2 – 3 days per week
3  4 - 5 days per week
4  6 – 7 days per week
Appendix F

9. How much does your drinking interfere with your work functioning? Is there anything that you don’t or can’t do because of your drinking? (If you are not currently working, how much of your performance would be affected if you were working?)

   0 Drinking never interferes – I can function normally.
   1 Drinking slightly interferes with my occupational activities, but my overall performance is not impaired.
   2 Drinking definitely interferes with my occupational performance, but I can still manage.
   3 Drinking causes substantial impairment in my occupational performance.
   4 Drinking problems interfere completely with my work performance.

10. How much does your drinking interfere with your social functioning? Is there anything that you don’t or can’t do because of your drinking?

   0 Drinking never interferes – I can function normally.
   1 Drinking slightly interferes with my social activities, but my overall performance is not impaired.
   2 Drinking definitely interferes with my social performance, but I can still manage.
   3 Drinking causes substantial impairment in my social performance.
   4 Drinking problems interfere completely with my social performance.

11. If you were prevented from drinking alcohol when you desired a drink, how anxious or upset would you become?

   0 I would not experience any anxiety or irritation.
   1 I would become only slightly anxious or irritated.
   2 The anxiety or irritation would mount but remain manageable.
   3 I would experience a prominent and very disturbing increase in anxiety or irritation.
   4 I would experience incapacitating anxiety or irritation.

12. How much of an effort do you make to resist consumption of alcoholic beverages? (Only rate your effort to resist, not your success or failure in actually controlling the drinking).

   0 My drinking is so minimal, I don’t need to actively resist. If I drink, I make an effort to always resist.
   1 I try to resist most of the time.
   2 I make some effort to resist.
   3 I give in to almost all drinking without attempting to control it, but I do so with some reluctance.
   4 I completely and willingly give in to all drinking.
13. How strong is the drive to consume alcoholic beverages?
   0  No drive
   1  Some pressure to drink
   2  Strong pressure to drink
   3  Very strong drive to drink
   4  The drive to drink is completely involuntary and overpowering.

14. How much control do you have over the drinking?
   0  I have complete control.
   1  I am usually able to exercise voluntary control over it.
   2  I can control it only with difficulty.
   3  I must drink and can only delay drinking with difficulty.
   4  I am rarely able to delay drinking even momentarily.
Appendix F

**AUDIT**

*Please place a mark in the box next to your answer*

1. How often do you have a drink containing alcohol?
   - □ never  □ monthly or less  □ once a week  □ 2 – 4 times a week  □ 5 or more times a week

2. How many ‘standard drinks’ (see below) containing alcohol do you have on a typical day when you are drinking?
   - □ 1  □ 2  □ 3 or 4  □ 5 or 6  □ 7 or more

3. How often do you have six or more drinks on one occasion?
   - □ never  □ less than monthly  □ monthly  □ weekly  □ daily or almost daily

4. How often during the last 6 months have you found that you were not able to stop drinking once you had started?
   - □ never  □ less than monthly  □ monthly  □ weekly  □ daily or almost daily

5. How often during the last 6 months have you failed to do what was normally expected from you because of your drinking?
   - □ never  □ less than monthly  □ monthly  □ weekly  □ daily or almost daily

6. How often during the last 6 months have you needed an alcoholic drink in the morning to get yourself going after a heavy drinking session?
   - □ never  □ less than monthly  □ monthly  □ weekly  □ daily or almost daily

7. How often during the last 6 months have you had a feeling of guilt or remorse after drinking?
   - □ never  □ less than monthly  □ monthly  □ weekly  □ daily or almost daily

8. How often during the last 6 months have you been unable to remember what happened the night before because you had been drinking?
   - □ never  □ less than monthly  □ monthly  □ weekly  □ daily or almost daily

9. Have you or someone else been injured as a result of your drinking?
   - □ no  □ Yes, but not in the last 6 months  □ Yes, during the last 6 months

10. Has a relative or friend, a doctor or other health worker been concerned about your drinking or suggested you cut down?
    - □ no  □ Yes, but not in the last 6 months  □ Yes, during the last 6 months
<table>
<thead>
<tr>
<th>SCID-IV FOR ALCOHOL ABUSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Have you ever missed work or school because you were intoxicated, high or very hung over? (How often? What about doing a bad job at work or failing courses at school because of your drinking?)</td>
</tr>
<tr>
<td>IF NO: What about not keeping your house clean or not taking proper care of your children because of your drinking? (How often?)</td>
</tr>
<tr>
<td>IF YES TO EITHER OF ABOVE: How often? (Over what period of time?)</td>
</tr>
<tr>
<td>1 NO (Absent or false) 2 INFREQUENTLY (subthreshold) 3 OFTEN (threshold or true)</td>
</tr>
<tr>
<td>☐ current in last 3 mths ☐ current in last 3 mths ☐ current in last 3 mths</td>
</tr>
<tr>
<td>☐ lifetime ☐ lifetime ☐ lifetime</td>
</tr>
<tr>
<td>Recurrent alcohol use resulting in a failure to fulfill major role obligations at work, school or home (e.g., repeated absences or poor work performance related to alcohol use; alcohol-related absences, suspensions, or expulsions from school; neglect of children or household)</td>
</tr>
</tbody>
</table>

| Did you ever drink in a situation in which it might have been dangerous to drink at all? (Did you ever drive while you were really too drunk to drive?) |
| IF YES: How many times? (When?) |
| 1 NO (Absent or false) 2 INFREQUENTLY (subthreshold) 3 OFTEN (threshold or true) |
| ☐ current in last 3 mths ☐ current in last 3 mths ☐ current in last 3 mths |
| ☐ lifetime ☐ lifetime ☐ lifetime |
| Recurrent alcohol use in situation in which it is physically hazardous (e.g., driving an automobile or operating a machine when impaired by alcohol use) |

| Has your drinking gotten you into trouble with the law? |
| IF YES: How often? (Over what period of time?) |
| 1 NO (Absent or false) 2 INFREQUENTLY (subthreshold) 3 OFTEN (threshold or true) |
| ☐ current in last 3 mths ☐ current in last 3 mths ☐ current in last 3 mths |
| ☐ lifetime ☐ lifetime ☐ lifetime |
| Recurrent alcohol related legal problems (e.g., arrests for alcohol-related disorderly conduct) |

| Has your drinking caused problems with other people, such as with family members, friends or people at work? (Have you ever gotten into physical fights when you were drinking? What about have bad arguments about your drinking?) |
| IF YES: Did you keep on drinking anyway? |
| 1 NO (Absent or false) 2 INFREQUENTLY (subthreshold) 3 OFTEN (threshold or true) |
| ☐ current in last 3 mths ☐ current in last 3 mths ☐ current in last 3 mths |
| ☐ lifetime ☐ lifetime ☐ lifetime |
| Continued substance use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance (e.g., arguments with spouse about consequences of intoxication, physical fights) |
### SCID-IV FOR ALCOHOL DEPENDENCE

<table>
<thead>
<tr>
<th>Question</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>Alcohol is often taken in larger amounts OR over a longer period than was intended.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Have you often find that when you started drinking you ended up drinking much more than you were planning to?</td>
<td>NO (Absent or false)</td>
<td>INFREQUENTLY (subthreshold)</td>
<td>OFTEN (threshold or true)</td>
<td>□ current in last 3 mths</td>
</tr>
<tr>
<td>IF NO: What about drinking for a much longer period of time than you were planning to.</td>
<td>NO</td>
<td>INFREQUENTLY</td>
<td>OFTEN</td>
<td>□ current in last 3 mths</td>
</tr>
<tr>
<td>Have you tried to cut down or stop drinking alcohol?</td>
<td>NO (Absent or false)</td>
<td>INFREQUENTLY (subthreshold)</td>
<td>OFTEN (threshold or true)</td>
<td>□ current in last 3 mths</td>
</tr>
<tr>
<td>IF YES: Did you ever actually stop drinking altogether?</td>
<td>NO</td>
<td>INFREQUENTLY</td>
<td>OFTEN</td>
<td>□ current in last 3 mths</td>
</tr>
<tr>
<td>(How many times did you try to cut down or stop altogether?)</td>
<td>NO</td>
<td>INFREQUENTLY</td>
<td>OFTEN</td>
<td>□ current in last 3 mths</td>
</tr>
<tr>
<td>IF NO: Did you want to stop or cut down? (Is this something you kept worrying about?)</td>
<td>NO</td>
<td>INFREQUENTLY</td>
<td>OFTEN</td>
<td>□ current in last 3 mths</td>
</tr>
<tr>
<td>Have you spent a lot of time drinking, being high, or hung over?</td>
<td>NO (Absent or false)</td>
<td>INFREQUENTLY (subthreshold)</td>
<td>OFTEN (threshold or true)</td>
<td>□ current in last 3 mths</td>
</tr>
<tr>
<td>A great deal of time is spent in activities necessary to obtain alcohol, use alcohol or recover from its effects.</td>
<td>NO</td>
<td>INFREQUENTLY</td>
<td>OFTEN</td>
<td>□ current in last 3 mths</td>
</tr>
<tr>
<td>Have you had times when you would drink so often that you started to drink instead of working or spending time at hobbies or with your family or friends or engaging in other important activities, such as sports, gardening, or playing music?</td>
<td>NO (Absent or false)</td>
<td>INFREQUENTLY (subthreshold)</td>
<td>OFTEN (threshold or true)</td>
<td>□ current in last 3 mths</td>
</tr>
<tr>
<td>Important social, occupational, or recreational activities given up or reduced because of alcohol use.</td>
<td>NO</td>
<td>INFREQUENTLY</td>
<td>OFTEN</td>
<td>□ current in last 3 mths</td>
</tr>
<tr>
<td>Appendix F</td>
<td></td>
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<tr>
<td>Has your drinking ever caused any psychological problems like making you depressed or anxious, making it difficult to sleep, or causing “blackouts?”</td>
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<tr>
<td>Has your drinking caused significant physical problems or make a physical problem worse?</td>
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<tr>
<td>IF YES to either: Did you keep on drinking anyway?</td>
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<td></td>
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<td>(subthreshold)</td>
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<td>lifetime</td>
<td>lifetime</td>
<td>lifetime</td>
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</tbody>
</table>

Tolerance as defined by either of the following:
(a) a need for markedly increased amounts of alcohol to achieve intoxication or desired effect
(b) markedly diminished effect with continued use of the same amount of alcohol

Withdrawal as manifested by either of the following:
(a) the characteristic withdrawal syndrome for alcohol
(b) alcohol (or a substance from the sedative/hypnotic/anxiolytic class) taken to relieve or avoid withdrawal symptoms.
Appendix F

**DASS21 (Depression items in bold)**

Please read each statement and circle a number 0, 1, 2 or 3 which indicates how much the statement applied to you over the past week. There are no right or wrong answers. Do not spend too much time on any statement. *The rating scale is as follows:*

<table>
<thead>
<tr>
<th>Did not apply to me at all</th>
<th>Some of the time</th>
<th>Good part of the time</th>
<th>Most of the time</th>
</tr>
</thead>
</table>

1. I found it hard to wind down
   - 0
   - 1
   - 2
   - 3

2. I was aware of dryness in my mouth
   - 0
   - 1
   - 2
   - 3

3. I couldn’t seem to experience any positive feeling at all
   - 0
   - 1
   - 2
   - 3

4. I experienced breathing difficulty (e.g. excessively rapid breathing, breathlessness in the absence of physical exertion)
   - 0
   - 1
   - 2
   - 3

5. I found it difficult to work up the initiative to do things
   - 0
   - 1
   - 2
   - 3

6. I tended to over-react to situations
   - 0
   - 1
   - 2
   - 3

7. I experienced trembling (e.g. in the hands)
   - 0
   - 1
   - 2
   - 3

8. I felt that I was using a lot of nervous energy
   - 0
   - 1
   - 2
   - 3

9. I was worried about situations in which I might panic and make a fool of myself
   - 0
   - 1
   - 2
   - 3

10. I felt that I had nothing to look forward to
    - 0
    - 1
    - 2
    - 3

11. I found myself getting agitated
    - 0
    - 1
    - 2
    - 3

12. I found it difficult to relax
    - 0
    - 1
    - 2
    - 3

13. I felt down-hearted and blue
    - 0
    - 1
    - 2
    - 3

14. I was intolerant of anything that kept me from getting on with what I was doing
    - 0
    - 1
    - 2
    - 3

15. I felt I was close to panic
    - 0
    - 1
    - 2
    - 3

16. I was unable to become enthusiastic about anything
    - 0
    - 1
    - 2
    - 3

17. I felt I wasn’t worth much as a person
    - 0
    - 1
    - 2
    - 3

18. I felt that I was rather touchy
    - 0
    - 1
    - 2
    - 3

19. I was aware of the action of my heart in the absence of physical exertion (e.g. sense of heart rate increase, heart missing a beat)
    - 0
    - 1
    - 2
    - 3

20. I felt scared without any good reason
    - 0
    - 1
    - 2
    - 3

21. I felt that life was meaningless
    - 0
    - 1
    - 2
    - 3
Results of follow-up attrition tests

<table>
<thead>
<tr>
<th>Variable</th>
<th>3 Months</th>
<th></th>
<th>12 Months</th>
<th></th>
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<tbody>
<tr>
<td></td>
<td>df</td>
<td>F</td>
<td>df</td>
<td>F</td>
</tr>
<tr>
<td>Number of treatment sessions</td>
<td>1, 240</td>
<td>24.65***</td>
<td>1, 240</td>
<td>30.34***</td>
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<td>Age</td>
<td>1, 240</td>
<td>2.76</td>
<td>1, 240</td>
<td>0.04</td>
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<td>Education</td>
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<tr>
<td>Baseline OCDS-O score</td>
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<td>Baseline ACE-Intensity score</td>
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<td>Baseline ACE-Intrusion score</td>
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<td>1, 227</td>
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<td>0.42</td>
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<td>Treatment allocation</td>
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<td>3.12</td>
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<td>3.38</td>
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<td>1, 242</td>
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<td>0.17</td>
<td>1, 242</td>
<td>0.01</td>
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<td>Mental health diagnosis</td>
<td>1, 240</td>
<td>1.13</td>
<td>1, 240</td>
<td>0.16</td>
</tr>
</tbody>
</table>

* p < .05  ** p < .01  *** p < .001

Results of Expectation-Maximisation Estimation Imputation

<table>
<thead>
<tr>
<th>Variable</th>
<th>3-Month mean</th>
<th>3-Month std dev</th>
<th>12-Month mean</th>
<th>12-Month std dev</th>
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</thead>
<tbody>
<tr>
<td>Original Average weekly drinks</td>
<td>28.37</td>
<td>30.08</td>
<td>29.43</td>
<td>28.23</td>
</tr>
<tr>
<td>Imputed Average weekly drinks</td>
<td>28.43</td>
<td>29.98</td>
<td>31.12</td>
<td>28.31</td>
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<tr>
<td>Original Average weekly binges</td>
<td>1.93</td>
<td>2.37</td>
<td>2.08</td>
<td>2.34</td>
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<tr>
<td>Imputed Average weekly binges</td>
<td>1.95</td>
<td>2.36</td>
<td>2.26</td>
<td>2.36</td>
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</tbody>
</table>
APPENDIX H  BASELINE AND DEMOGRAPHIC DIFFERENCES BETWEEN THE POPULATIONS OF STUDY 1 AND STUDY 2

<table>
<thead>
<tr>
<th></th>
<th>Study 1 (N = 260)</th>
<th>Study 2 All participants (N = 242)</th>
<th>Study 2 High depression$^a$ (N = 85)</th>
</tr>
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<tbody>
<tr>
<td>Age</td>
<td>45.42</td>
<td>48.82**</td>
<td>46.89</td>
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<tr>
<td></td>
<td>(10.86)</td>
<td>(10.90)</td>
<td>(11.96)</td>
</tr>
<tr>
<td>Gender (male)</td>
<td>50.8%</td>
<td>60.7%*</td>
<td>58.8%</td>
</tr>
<tr>
<td>Years of education</td>
<td>11.10</td>
<td>13.68***</td>
<td>12.96***</td>
</tr>
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<td>(1.41)</td>
<td>(3.17)</td>
<td>(2.96)</td>
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<td>Relationship status (partnered)</td>
<td>33.1%</td>
<td>68.6%***</td>
<td>56.5%***</td>
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<tr>
<td>Baseline average weekly drinks</td>
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<td>63.13</td>
<td>69.02</td>
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<td>(42.95)</td>
<td>(30.56)</td>
<td>(34.49)</td>
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<td>Baseline binges (half or more of week)</td>
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<td>80.6%***</td>
<td>83.5%**</td>
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<tr>
<td>Baseline AUDIT total</td>
<td>25.70</td>
<td>24.50*</td>
<td>26.52</td>
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<td>(6.59)</td>
<td>(5.32)</td>
<td>(5.48)</td>
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<td>Baseline alcohol abuse (present)</td>
<td>68.1%</td>
<td>55.4%**</td>
<td>69.4%</td>
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<tr>
<td>Baseline alcohol dependence (present)</td>
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<td>94.2%***</td>
<td>94.1%*</td>
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<td>Baseline OCDS-O total</td>
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<td>6.89***</td>
<td>9.19</td>
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<td></td>
<td>(5.75)</td>
<td>(4.61)</td>
<td>(4.82)</td>
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<tr>
<td>Baseline depression (none or minimal)</td>
<td>0%</td>
<td>50.8%***</td>
<td>0%</td>
</tr>
<tr>
<td>Baseline depression (mild)</td>
<td>6.9%</td>
<td>13.6%***</td>
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<tr>
<td>Baseline depression (moderate)</td>
<td>35.9%</td>
<td>21.5%***</td>
<td>61.2%***</td>
</tr>
<tr>
<td>Baseline depression (severe)</td>
<td>57.1%</td>
<td>14.0%***</td>
<td>38.8%***</td>
</tr>
<tr>
<td>Baseline current depression (present)</td>
<td>70.0%</td>
<td>5.8%***</td>
<td>14.1%***</td>
</tr>
</tbody>
</table>

* p < .05  ** p < .01  *** p < .001; $^a$Moderate or greater depression on the DASS21