Craving as a Predictor of Treatment Outcomes in Heavy Drinkers with Comorbid Depressed Mood

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Word count: 5412 plus abstract, references and tables.

Abstract

Alcohol and depression comorbidity is high and is associated with poorer outcomes following treatment. The ability to predict likely treatment response would be advantageous for treatment planning. Craving has been widely studied as a potential predictor, 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. The current study examined the performance of craving, measured pre-treatment using the Obsessive subscale of the Obsessive Compulsive Drinking Scale, in predicting 18-week and 12-month post-treatment alcohol use outcomes in a sample of depressed drinkers. Data for the current study were collected during a randomized controlled trial (Baker, Kavanagh, Kay-Lambkin, Hunt, Lewin, Carr, & Connolly, 2010) comparing treatments for comorbid alcohol and depression. A subset of 260 participants from that trial with a Timeline Followback measure of alcohol consumption were analyzed. Pre-treatment craving was a significant predictor of average weekly alcohol consumption at 18 weeks and of frequency of alcohol binges at 18 weeks and 12 months, but pre-treatment depressive mood was not predictive, and effects of Baseline craving were independent of depressive mood. Results suggest a greater ongoing risk from craving than from depressive mood at Baseline.

Keywords: Craving, Prediction, Post-treatment, Alcohol, Depression, Comorbidity

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 (MDD) in people with an alcohol use disorder (AUD), and a 2-3% prevalence of Dysthymic 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, including lower remission rates, longer time to remission and higher risk of adverse events and hospitalization (Davis et al., 2010; Howland et al., 2009; Landheim, Bakken, & Vaglum, 2006). 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 enable identification of those most at risk of relapse and would enable treatment plans to be tailored accordingly. This would enable more efficient and cost-effective distribution of resources, and potentially, better outcomes for clients.

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, Hutchison, & Bryan, 2006; Rohsenow et al., 1994; Soyka, Helten, & Schmidt, 2010), smoking (Zelman, Brandon, Jorenby, & Baker, 1992) and cocaine (Crits-Christoph et al., 2007; Paliwal, Hyman, & Sinha, 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, Mulgrew, Modesto-Lowe, & Burleson, 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 poorly investigated, 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, Martin, Eaton, & Monti, 2007). Additionally, elevated depression and negative affective stimuli have been found to be associated with heightened reactivity to alcohol cues (Feldstein Ewing, Filbey, Chandler, & Hutchison, 2010; Fox, Bergquist, Hong, & Sinha, 2007). The presence of comorbid negative affect, such as experienced in depression, may augment the relationship between craving and substance use, improving its predictive ability.

We could identify only one study examining pre-treatment craving as a predictor of alcohol relapse in a population with comorbid depression (Farren & McElroy, 2010). This study reported that baseline craving, measured using the Obsessive Compulsive Drinking Scale (OCDS; Anton, Moak, & Latham, 1995), was not a significant predictor of relapse to alcohol during the 6 months following discharge. However, key limitations existed with this study, the most important of which included

the temporal sequencing of mood, alcohol and craving measurements, with baseline craving measurements taken after alcohol detoxification and mood stabilization had already occurred. Since state measurements of craving show strong associations with subsequent drinking (Oslin, Cary, Slaymaker, Colleran, & Blow, 2009) and smoking (Allen, Bade, Hatsukami, & Center, 2008; Chandra, Scharf, & Shiffman, in press; 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. (Farren & McElroy, 2010) was the use of dichotomous outcomes (abstinent versus relapsed), which restricts the power of predictive analyses. 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. We aimed to overcome the limitations of previous research in a number of important ways: (i) measuring craving in a state relevant to the context being predicted, i.e. prior to treatment and while contemplating control of drinking control; (ii) to measure outcomes on a continuous scale to enable examination of the strength and direction of relationships; (iii) to take an inclusive approach to eligibility for the study, encompassing people with moderate levels of depression and alcohol use who did not meet diagnostic criteria for Major Depression or Alcohol Dependence, to ensure adequate representation of the range of comorbidity experienced by people with depression and alcohol use problems.

We hypothesized 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. We also examined whether an interaction effect between mood and craving

was present, with the combination of depressed mood and craving at pre-treatment predicting higher subsequent alcohol consumption.

2. Materials and method

2.1 Participants

Data for the present study were collected during a randomized 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.

Inclusion criteria were: (i) aged over 16 years; (ii) Beck Depression Inventory-II (Beck, Steer, & Brown, 1996) score ≥ 17; and (iii) at least two occasions in the previous month of >6x10g ethanol drinks for men, or >4 for women. Exclusion criteria were: (i) concurrent psychotic disorder; (ii) self-reported history of traumatic brain injury; (iii) lack of English fluency; and (iv) inability to travel to treatment sessions. Concurrent pharmacotherapy was not excluded, but a stable dose for ≥ 4 weeks was required before participation.

2.2 Measures

Only a subset of the data collected in the study was used for the present research. A full description of measures can be viewed in Baker et al. (2010).

2.2.1 Craving

The Obsessive subscale of the Obsessive Compulsive Drinking Scale (OCDS-O; Anton, et al., 1995) was used to measure alcohol craving. The Compulsions 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 (Modell, Glaser, Cyr, & Mountz, 1992; Statham et al., in submission). 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, Diehl, Croissant, & Mann, 2008). This subscale has good internal consistency (alpha = .83) and is significantly correlated with the Alcohol Dependence Scale and Addiction Severity Index (Kranzler, et al., 1999). Internal consistency (Cronbach's alpha) in the present study was .90.

2.2.2 Alcohol consumption and severity

The Timeline Follow-Back (TLFB; Sobell & Sobell, 1992) assessed alcohol use in the preceding 2 weeks, using event-based cues to aid recall. The TLFB allowed calculation of average weekly consumption and average number of binge days (>6x10g ethanol/occasion for men; >4x10g ethanol for women) per week. The TLFB has demonstrated sound temporal stability and concurrent validity for 30 day assessment in respondents with comorbid psychiatric diagnoses (Carey, 1997; Carey, Carey, Maisto, & Henson, 2004). Validity using shorter assessment timeframes of 7 and 14 days has also been demonstrated (Toll, Leeman, McKee, & O'Malley, 2008).

A 6-month version of the Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, de le Fuente, & Grant, 1993) was used as an index of

severity to describe the sample. The psychometric performance of the AUDIT in comorbid psychiatric populations is consistent with its strong characteristics in the general population (Dawe, Seinen, & Kavanagh, 2000; Maisto, Carey, Carey, Gordon, & Gleason, 2000; O'Hare, Sherrer, LaButti, & Emrick, 2004).

2.2.3 Diagnoses

Diagnoses of current and lifetime Alcohol Use Disorder and Major Depression or Dysthymia were made using the Structured Clinical Interview for DSM-IV (SCID; First, Gibbon, Spitzer, & Williams, 1995).

2.2.4 Depression severity

The Beck Depression Inventory Version 2 (BDI-II; Beck, et al., 1996) was used to determine depression severity. The BDI-II is a 21-item self-report questionnaire used to screen for the presence of depressive symptoms over the previous two-week period. The questionnaire has been validated with both adult and adolescent populations (age range 13-80 years), and is commonly used to screen for depressive symptoms among people with drug and alcohol use problems (Dawe, Loxton, Hides, Kavanagh, & Mattick, 2002). A short version of the BDI (the Beck Depression Inventory – Fast Screen; Beck, Steer, & Brown, 2000) was used at screening to determine initial eligibility.

2.3 Interventions

Interventions were fully manualized, and were 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 comparability of the 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-behavioral therapy (CBT) incorporating brief mindfulness exercises, focused on alcohol reduction; (iii) *Depression Intervention*—involving a further nine 1-hour sessions, with 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.

2.4 Procedure

After self-referral, potential participants were screened by telephone for initial eligibility. Those who passed this initial screening were invited to an interview where final eligibility was determined. Eligible participants were invited to complete the remainder of the baseline assessment, comprising a face-to-face interview, 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 reimbursed with $20 for each completed assessment including Baseline.

2.5 Statistical analysis

Data were analyzed with SPSS Statistics 19. An intention-to-treat approach was used. 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-Maximization method in SPSS Missing Value Analysis. Estimations for post-baseline assessments of weekly drinks and binges were imputed using Baseline weekly drinks, binges, abstinent days and number of sessions attended. Inclusion of the Baseline measure of the predicted variable in the estimation of later 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. Sensitivity analyses using the complete case data were run for comparison.

Variables were examined for normality and extreme cases (>3 SDs from the mean and >1 SD from the next most extreme case). Two extreme values were identified (1 each for average weekly drinks at 18 weeks and 12 months): these were Winsorized. Average binge days per week at Baseline were heavily left-skewed, with many cases bingeing daily. Accordingly, this variable was recoded into dichotomous categories (< and ≥ half the week) for all assessment periods. Other Baseline and follow-up variables were suitable for parametric testing.

Consistent with previous reports of this study, intervention effects were examined using three orthogonal contrasts: (i) Brief versus Long; (ii) Integrated versus Single-focused, and; (iii) Alcohol versus Depression. The prediction of an interaction

between Baseline depression and craving was examined by multiplying standardized, mean-centered scores (Aiken & West, 1991).

Relationships of Baseline and treatment variables with outcome variables were examined using Pearson bivariate correlations, univariate ANOVAs and Chi-Squares. The unique prediction from key variables was examined in linear and logistic regressions. The focus of this study was on predictions of post-treatment (18-week) and 12-month follow-up outcomes from Baseline measures. For all analyses, an alpha of .05 was adopted.

3. Results

3.1 Participants

A total of 284 participants (53% male) were recruited and randomly allocated (Baker, et al., 2010). However, the TLFB was not collected for 24 participants at Baseline. As this was a key prediction variable, these participants were excluded, producing a sample of 260. Demographic characteristics of this sample are shown in Table 1.

INSERT TABLE 1 AROUND HERE

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 Reserach Council, 2009), but wide variations in consumption were observed. The mean depression score of the sample fell in the lower end of the severe range. 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 meeting diagnostic criteria for Alcohol Dependence.

At Baseline, 15 participants reported taking pharmacotherapy to manage alcohol craving. However, a univariate ANOVA revealed that OCDS-O scores remained significantly higher in people on anticraving medication ($F(1, 203) = 9.31$, $p = .003$). In view of this result, these participants were retained in analyses. Craving at Baseline was significantly correlated with depression severity ($r = .267$, $p < .001$).

Of the 260 analyzed participants, 15% did not commence their allocated treatment, while 86% of the Brief group and 39% of the extended intervention groups completed all treatment sessions. Follow-up retention across all groups was 82% at 18 weeks and 73% at 12 months. Further detail on retention and attrition is provided in Baker et al. (2010). 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. Including baseline drinking in the EM estimation model provided adequate adjustment for this difference when imputing the missing outcome values. Sensitivity analyses yielded consistent outcomes between the imputed and complete case data sets.

3.2 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 ($F(2,259) = 56.01$, $p < .001$). The majority of participants continued to drink at some level, with only 7.7% abstinent at 18 weeks and 9.6% at 12 months.

Univariate predictors of post-treatment average weekly drinks are displayed in Table 2. Treatment effects have been reported elsewhere (Baker, et al., 2010). Apart from differential treatment effects and the predictions from baseline weekly drinks, the only significant univariate predictors of greater weekly consumption at both 18 weeks and 12 months were higher baseline OCDS-O score and male gender. Not having a partner and being a recipient of welfare payments predicted weekly drinking at 12 months. Neither depression nor the depression-craving interaction was significantly associated with weekly consumption at either time point.

INSERT TABLE 2 AROUND HERE

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, gender, relationship status and welfare status, with baseline OCDS-O score entered at the final step. Results of the 18-week and 12-month regressions are presented in Tables 3 and 4.

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Addition of the OCDS-O to the model at Step 3 only resulted in a significant increase in predicted variance in the prediction of 18-week outcomes. At 18 weeks, R^2 increased from .393 to .427, indicating that baseline craving accounted for a modest

but still significant 3.5% of the total variance in 18-week drinking, after other significant Baseline and 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 1.44 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 was the Brief vs. Long treatment contrast, in favor of the longer interventions.

3.3 Frequency of binges

Across the whole sample, significantly fewer participants were bingeing (drinking > 6x10g ethanol/occasion for men or > 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). Baseline univariate predictors of post-treatment binges are displayed in Table 5. Along with the baseline frequency of binges, greater craving on the OCDS-O was a significant univariate predictor of a higher binge frequency at both 18 weeks and 12 months. Lack of a partner was associated with more binges at 12 months, but not at 18 weeks. Neither welfare status nor any treatment contrast was a significant univariate predictor. Consistent with the prediction of weekly drinks, depression and the depression-craving interaction also failed to reach significance.

INSERT TABLE 5 AROUND HERE

The unique prediction of post-treatment binges from the pre-treatment OCDS-O was examined using logistic regressions. Baseline binge frequency and relationship

status were entered along with the OCDS-O. Results for the 18-week and 12-month regressions are presented in Table 6.

INSERT TABLE 6 AROUND HERE

The model fit was significant for both 18 weeks and 12 months ($\chi^2(3, N=207) = 57.50$, $p < .001$ and $\chi^2(3, N=207) = 39.75$, $p < .001$ respectively), with a Nagelkerke pseudo R^2 of .330 at 18 weeks and .236 at 12 months, meaning the set of predictors accounted for an estimated 33.0% of the variance at 18 weeks and 23.6% of the variance at 12 months. For the 18-week regression, likelihood ratio tests were only significant for baseline binge frequency ($\chi^2(1, N=207) = 40.50$, $p < .001$) and OCDS-O ($\chi^2(1, N=207) = 12.05$, $p = .001$). Similar results were found for the 12-month regression, with baseline binges and OCDS-O emerging as the only significant contributors to the prediction ($\chi^2(1, N=207) = 29.19$, $p < .001$ and $\chi^2(1, N=207) = 5.61$, $p = .018$ respectively).

The parameter coefficients show that for every one point pre-treatment increase in OCDS-O score, the log odds of bingeing less than half the week (versus half or more) decrease by .099 at 18 weeks and by .064 at 12 months.

3.4 Sensitivity analyses

Results from the complete case analyses were highly consistent with those of the imputed data set. Only minor differences emerged. In the complete case analysis, the depression by craving interaction had a small but significant correlation with 18-week average weekly drinking ($r = .153$, $p = .042$). However it was not a significant predictor in the later regression ($B = 3.57$, $t = 1.56$, $p = .121$), where the OCDS-O

accounted for a slightly higher proportion of variance than in the imputed data analysis (4.3% compared to 3.5%).

In tests predicting the frequency of binges, relationship status was not a significant univariate predictor at either time point. Pseudo R^2 was lower in the multinomial logistic regressions (.305 at 18 weeks and .169 at 12 months), which may have been partly because baseline drinking was used as a predictor of missing data during imputation. The log odds of bingeing less than half the week compared to half or more of the week decreased to .144 at 18 weeks and to .081 at 12 months in the complete case analysis.

4. Discussion

This study sought to examine whether pre-treatment craving that is experienced in the context of comorbid depressed mood, would predict post-treatment and follow-up alcohol consumption, based on the rationale that the combination of elevated craving with high levels of depressed mood may be particularly potent, and may be 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 binge frequencies at both 18 weeks and 12 months. Even with baseline consumption and other significant demographic and treatment variables in the model, the significant predictions from craving remained. These results suggest 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 (Statham, et al., in submission), 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 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, Andrade, & May, 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.

The finding that pre-treatment craving was predictive of alcohol consumption after treatment is consistent with results of several previous studies (e.g. Evren, Cetin, Durkaya, & Dalbudak, 2010; Ray, et al., 2006). However, our 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 alcohol use, but prior to receipt of support--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 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 amount of money spent on cocaine post-treatment, but not of 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, though no evidence of moderating or mediating effects by either depression or craving were detected. Neither depression nor the depression by craving interaction were related to either outcome measure at either time point, suggesting 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, Pinquart, & Gamble, 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 sample may result 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 the inclusion cut-off of 17 on the BDI-II was slightly higher than the cut-off for mild depression. Inclusion of people without significant depressive mood may be necessary to detect the effect that it has on subsequent outcomes.

Half of the current sample was on depression pharmacotherapy at entry to the study (52%). 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 antidepressant status and concurrent BDI-II scores ($F(1,255) = .240$, $p = .624$), 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. Examination of baseline correlations showed that BDI-II scores were modestly but significantly correlated with baseline average weekly drinks ($r = .148$, $p = .017$), but not with frequency of binges ($r = .065$, $p = .293$). The current study did therefore detect a small but significant association between higher levels of depressed mood and greater weekly consumption. It is likely that levels of depressed mood are most strongly associated with proximal drinking behavior, 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 (e.g. 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, allowing them to emerge as a significant predictor of follow-up outcomes (Curran, Flynn, Kirchner, & Booth, 2000; Witkiewitz & Villarroel, 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.

Gender was also found to not perform as a predictor of treatment outcomes, suggesting the significant bivariate associations between gender and post-treatment drinking reflected a tendency for men to drink larger quantities than women rather than a tendency for them to have poorer outcomes. This contrasts with the findings of Farren, Snee, and McElroy (2011) that more women than men remained abstinent 2 years following treatment. It is not clear whether the focus on abstinence rather than drinking levels was responsible for the difference in results between the two studies.

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 3.5% 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.

Limitations of the current study included the absence of a group of participants without depressive mood, which may have allowed for a stronger test of predictions from depression, and its restriction 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 a cognitive elaborative process, as described in Elaborated Intrusion theory (Kavanagh, et al., 2005), that is processed and manipulated on a fully conscious level. 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 cognitive aspects of craving, or if they relate to the craving construct more generally.

A further limitation 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 complete case data, resulting in likely underestimation of the unique variance accounted for by craving in the predictions. However, the results were highly consistent between the two sets of analyses, suggesting that the imputation method did not affect the study outcomes.

5. Conclusions

This study provides preliminary evidence that craving acts as a significant predictor of post-treatment drinking outcomes in the presence of comorbid depressed mood, although no evidence of moderating or mediating influences by either depression or craving were detected. This suggests the prediction by craving was not related to the level of depressed mood at Baseline, although there were limitations within the current study that may have precluded detection of such an association. 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.

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Table 1. *Characteristics of the analysed sample (n = 260).*

Continuous Variables	M	(SD, range)
TLFB mean drinks per week	61.60	(42.95, 6.3-280)
TLFB mean binge[a] days per week	4.39	(2.31, 0–7)
TLFB mean abstinent days per week	1.58	(1.88, 0-6.5)
TLFB mean drinks per drinking day	11.40	(6.70, 2.4-53)
Mean BDI-II total	31.54	(8.95, 17-55)
Mean AUDIT total	25.70	(6.59, 7-40)
Mean OCDS-O total (n=206)	9.17	(5.75, 0–23)
Categorical Variables	**n**	**(%)**
Major Depressive Episode: current	180/257	(70.0%)
Alcohol Abuse diagnosis	177/260	(68.1%)
Alcohol Dependence diagnosis	218/260	(83.8%)
Current antidepressant medication	132/257	(51.4%)
Current anticraving medication	15/257	(5.8%)

[a] >6x10g ethanol/occasion for men; >4 for women

Table 2. *Univariate predictions of average weekly drinks.*

		18 weeks	**12 Months**
	n	r	r
Age	260	-.059	.003
Education	258	-.001	-.080
Baseline average weekly drinks	260	.600***	.527***
OCDS-O score	207	.406***	.247***
BDI-II score	260	.097	.093
Interaction BDI-II and OCDS-O	207	.122	.073
	df	F	F
Gender	1, 258	12.73***	8.33**
Relationship status	1, 258	3.72	6.79*
Welfare status	1, 257	1.97	4.84*
Current depressive episode	1, 255	.615	.521
Current antidepressants	1, 255	2.58	2.58
Brief vs Long	1, 258	6.61*	.304
Integrated vs Single focused	2, 257	3.45*	.196
Alcohol vs Depression	2, 257	.558	.706

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

Table 3. *Coefficients of Baseline and treatment variables predicting average weekly drinks at 18 weeks (n=207).*

Variables	R^2 change	F (df)	p	B	Std Err	t	p
BL Average weekly drinks	.360	115.00 (1, 204)	.000	.558	.052	10.72	.000
BL Avg drinks	.032	2.10 (5, 199)	.067	.543	.054	9.98	.000
Brief vs Long				-3.34	1.30	-2.57	.011
Integ vs Single				-2.72	1.78	-1.53	.128
Gender				-3.72	4.61	-.807	.421
Relationship				-3.58	4.93	-.725	.469
Welfare				.990	4.62	.214	.831
BL Avg drinks	.035	12.06 (1, 198)	.001	.467	.057	8.16	.000
Brief vs Long				-3.33	1.27	-2.63	.009
Integ vs Single				-2.54	1.73	-1.47	.144
Gender				-4.78	4.50	-1.06	.290
Relationship				-3.85	4.83	-.801	.424
Welfare				-2.20	4.59	-.480	.632
OCDS-O				1.44	.415	3.47	.001

Table 4. *Coefficients of Baseline and treatment variables predicting average weekly drinks at 12 months (n=207).*

Step	Variables	R^2 change	F (df)	p	B	Std Err	t	p
1	BL Average weekly drinks	.277	78.35 (1, 204)	.000	.413	.047	8.85	.000
2	BL Avg drinks	.013	.726 (5, 199)	.605	.393	.050	7.93	.000
	Brief vs Long				-.541	1.19	-.456	.649
	Integ vs Single				-.701	1.63	-.432	.666
	Gender				-2.32	4.21	-.551	.582
	Relationship				-5.46	4.50	-1.21	.226
	Welfare				3.70	4.22	.878	.381
3	BL Avg drinks	.001	.268 (1, 198)	.605	.383	.054	7.12	.000
	Brief vs Long				-.541	1.19	-.455	.650
	Integ vs Single				-.677	1.63	-.416	.678
	Gender				-2.47	4.23	-.584	.560
	Relationship				-5.50	4.51	-1.22	.224
	Welfare				3.26	4.31	.755	.451
	OCDS-O				.202	.390	.518	.605

Table 5. *Univariate predictions of frequency of binges.*

		18 weeks	**12 Months**
	df	F	F
Age	1, 258	.001	.049
Education	1, 256	.327	.110
OCDS-O score	1, 205	16.09***	8.60**
BDI-II score	1, 258	.099	.004
Interaction BDI-II and OCDS-O	1, 205	.601	2.14
	df, N	χ^2	χ^2
Baseline binge frequency	1, 260	43.68***	37.61***
Gender	1, 260	3.04	2.07
Relationship status	1, 260	2.20	4.31*
Welfare status	1, 259	.458	1.00
Current depressive episode	1, 257	1.59	.163
Current antidepressants	1, 257	1.57	1.66
Brief vs Long	1, 260	1.73	.772
Integrated vs Single focused	2, 260	2.42	2.38
Alcohol vs Depression	2, 260	2.82	2.53

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

Table 6. *Parameter estimates for pre-treatment variables predicting post-treatment binge frequency (n=207). Reference category is 'half or more of the week'.*

Predictor	B	Std Error	Wald	df	p
18 week					
BL binge frequency (ref = less than half of week)	2.32	.426	29.62	1	.000
Relationship status (ref = single)	-.405	.357	1.29	1	.257
OCDS-O	-.099	.030	11.22	1	.001
12 month					
BL binge frequency (ref = less than half of week)	1.81	.366	24.35	1	.000
Relationship status (ref = single)	-.446	.339	1.74	1	.188
OCDS-O	-.064	.027	5.45	1	.020