Published in final edited form as:

Addiction. 2015 August ; 110(8): 1262–1271. doi:10.1111/add.12964.

Pain as a Predictor of Heavy Drinking and Any Drinking Lapses in the COMBINE Study and the United Kingdom Alcohol Treatment Trial

Katie Witkiewitz^{1,2}, Kevin E. Vowles¹, Elizabeth McCallion¹, Tessa Frohe^{1,2}, Megan Kirouac^{1,2}, and Stephen A. Maisto³

¹Department of Psychology, University of New Mexico, MSC 03-2220, Albuquerque NM, 87131

²Center on Alcoholism, Substance Abuse, and Addictions, University of New Mexico, 2650 Yale Blvd SE, Albuquerque NM, 87106

³Department of Psychology, Syracuse University, 430 Huntington Hall, Syracuse NY, 13244

Abstract

Aims—To test the association between pain and heavy drinking lapses during and following treatment for alcohol use disorders (AUD).

Design—Secondary data analysis of data from two clinical trials for AUD.

Setting and participants—Participants included 1383 individuals from the COMBINE Study in the United States [31% female, 23% ethnic minorities, average age=44.4 (SD=10.2)] and 742 individuals from the United Kingdom Alcohol Treatment Trial (UKATT) in the United Kingdom [25.9% female, 4.4% ethnic minorities, average age=41.6 (SD=10.1)].

Measurements—Form-90 (a structured assessment interview) was used to assess the primary outcome: time-to-first heavy drinking day. The Short Form Health Survey and Quality of Life measures were used to assess pain interference and pain intensity.

Findings—Pain was a significant predictor of heavy drinking lapses during treatment in UKATT (OR=1.19 (95% CI: 1.08, 1.32), $p=0.0003$) and COMBINE (OR=1.12 (95% CI: 1.03, 1.21), $p=0.009$) and was a significant predictor of heavy drinking lapses following treatment in COMBINE (OR=1.16 (95% CI: 1.15, 1.17), $p<.00001$). After controlling for other relapse risk factors (e.g., dependence severity, self-efficacy, temptation, psychiatric distress), pain remained a significant predictor of heavy drinking lapses during treatment in UKATT (OR=1.19 (95% CI: 1.06, 1.34), $p=.004$) and following treatment in COMBINE (OR=1.44 (95% CI: 1.07, 1.92, $p=.01$).

Conclusions—Among people treated for alcohol use disorder, being in physical pain appears to predict heavy drinking lapses during or after treatment.

Introduction

Treatment for alcohol use disorder (AUD) is often followed by high rates of lapses [6], defined as drinking beyond one's limit after either a period of abstinence, or maintenance of a moderate drinking goal. To better understand the process of returning to heavy drinking following treatment, which is often defined as a “relapse” [7], researchers have developed

numerous models for characterizing the relapse process [8, 9]. Recent models have focused on the importance of biological, psychological, environmental, and social factors that may influence relapse, yet few models have incorporated physical health, particularly experiences of pain, as potential predictors.

Alcohol and the Experience of Pain

Pain is a frequent human experience. In Europe and the United States (US), for example, prevalence surveys indicate approximately 20%-30% of individuals in the general population and 50% of medical patients experience chronic pain, typically defined as persistent pain lasting three months or longer [10–12]. Despite the high prevalence of pain and the fact that historically alcohol has been used as an analgesic in the absence of other methods [13, 14], there is limited research on pain as a predictor of alcohol use or AUD [15].

Multiple studies have indicated pain experience and alcohol use to be closely related. Caldeiro and colleagues [16] found the prevalence of chronic pain among substance users (not including opioid-dependent patients) to be 33%. Patients in substance use disorder treatment with severe chronic pain reported higher alcohol/drug craving than those without severe chronic pain, and those reporting lower levels of pain were twice as likely to remain abstinent at 12-months than those with higher levels of pain [16]. Among patients recruited from a residential detoxification program for heroin, alcohol, or cocaine use, persistent pain significantly increased the risk of returning to any substance use (heroin/opiate use, and heavy alcohol use) within two-years following detoxification [17]. Older adults with symptoms of AUD were more likely to experience pain, more likely to treat their pain with alcohol than those without AUD, and had higher levels of pain intensity that were found to be associated with greater alcohol consumption [19].

The association between chronic pain and opiate use disorders has been more widely researched. Among individuals with prescription opiate use disorder, over 42% met criteria for current chronic pain, and 83.2% of those with chronic pain reported that pain relief was the initial reason for using opiates [20]. Individuals with comorbid opiate use disorder and chronic pain are twice as likely to use prescription opiates in order to cope with physical pain [20]. Interestingly, individuals in treatment for chronic pain who have comorbid SUDs are more often treated with prescription opiates and are often prescribed higher doses, than those in treatment for chronic pain without a current SUD [21]. Individuals with SUDs in chronic pain treatment are also more likely to report both aberrant prescription opiate use behaviors and worse pain outcomes than patients without a SUD [21, 22].

The findings on the association between SUD and pain raise the question of whether pain may be related to AUD treatment outcomes. Moreover, if alcohol use is positively associated with pain, examining pain may be an important component of interventions for individuals with pain and AUD. Clinically, we have treated patients who have attributed the primary cause of their alcohol lapses to the experience of physical pain; nevertheless, there has been no published report of data on the prospective association between pain and AUD treatment outcomes. The current study involved secondary analyses of data from two randomized clinical trials for alcohol dependence: the COMBINE Study [1], a large

multisite study for alcohol dependence conducted in the US, and the United Kingdom Alcohol Treatment Trial (UKATT; [2]), a large multisite study conducted in the United Kingdom (UK). The primary goals of this study were to (1) examine whether pain interference or intensity is related to AUD treatment outcomes, and (2) whether pain remains related to AUD treatment outcomes after controlling for other relapse risk factors. Given the recent focus in the AUD literature on any heavy drinking as a primary endpoint [23], we tested pain as a predictor of the time-to-first heavy drinking day as the primary outcome. Time-to-first drinking day (any drinking) was tested as the secondary outcome measure.

Methods

COMBINE Participants and Procedures

In the COMBINE study, a total of 1383 participants across 11 research sites were randomized into 9 treatment groups, consisting of a combination of medical management (MM) or combined behavioral intervention (CBI) and medications (acamprosate, naltrexone, or placebo [1]). Participants received treatment for a total of 16 weeks; participants receiving study medication were offered 9 MM visits, and those who received CBI had a maximum of 20 sessions. Participants completed assessments at 10, 36, and 52 weeks following treatment. The sample was recruited from treatment referrals at study sites and from the community (see Table 1 for demographic information and eligibility criteria) [24].

UKATT Participants and Procedures

In UKATT, 742 participants across seven treatment sites in three UK cities (Birmingham, Cardiff, Leeds) were randomized into two treatment groups: motivation enhancement therapy (MET) or social behavior and network therapy (SBNT; [25]). Participants receiving SBNT received up to 8 sessions and those who received MET received up to 3 sessions. Participants were followed for assessments at 3- and 12-months following entry into the trial. The study participants were recruited if they were seeking alcohol treatment from one of the treatment sites (see Table 1 for demographic information and eligibility criteria) [2, 25].

Measures

Alcohol use—In both studies, alcohol consumption was assessed using a calendar method via the Form-90 [3]. In COMBINE, alcohol consumption measured in United States standard drinks (unit of alcohol=11.358 grams) was assessed for 485 days (120 days during treatment and 365 days following treatment). In UKATT, alcohol consumption measured in United Kingdom standard drinks (unit of alcohol=8 grams of ethanol) was assessed for the prior 90 days at baseline, 3-month follow-up (corresponding to the post-treatment assessment), and the 12-month follow-up.

Pain Interference and Intensity—In COMBINE, pain interference was assessed via items from the 26-item World Health Organization Quality of Life (WHO-QOL) assessment [26] and the 12-item Short Form Health Survey (SF-12; [4]). The WHO-QOL measure was administered at baseline, 26 weeks, and 52 weeks; while the SF-12 was administered at

baseline, 16 weeks, and 52 weeks. The WHO-QOL [26] item “To what extent do you feel that physical pain prevents you from doing what you need to do?” with response options ranging from 1 “not at all” to 5 “an extreme amount” and the SF-12 item “During the past four weeks, how much did pain interfere with your normal work including both work outside the home and housework?” with response options ranging from 1 “not at all” to 5 “extremely” were included as indicators in graded response models at baseline, 16 weeks, 26 weeks, and 52 weeks. For both the WHO-QOL and SF-12 items, we truncated the ordered categories by collapsing the two categories indicating greatest amount of pain interference due to low responding (<1%) in the most extreme category on both items. The recoded response options were set at 1 “not at all” to 4 “very much or extreme amount” for the WHO-QOL and 1 “not at all” to 4 “quite a bit or extremely” for the SF-12. Internal consistency of the two items exceeded $\alpha=0.81$ at all time points.

In UKATT, pain interference and pain intensity were assessed via items from the European Quality of Life Group EQ-5D [5], a general measure of health status on five dimensions, and the 36-item Short Form Health Survey (SF-36 [27]). The EQ-5D and SF-36 were administered at baseline, 3-month, and 12-month follow-ups. We included one item for pain intensity from the EQ-5D: “Please indicate which statements best describes your health today...” with options of “I have no pain or discomfort,” “I have moderate pain or discomfort,” or “I have extreme pain or discomfort,” and one item of pain intensity from the SF-36: “How much bodily pain have you had during the past 4 weeks” with response options ranging from “none” to “very severe.” We also included the SF-36 item of pain interference, “During the past four weeks, how much did pain interfere with your normal work including both work outside the home and housework?” with response options ranging from “not at all” to “extremely.” All three items were used as indicators in graded response models at baseline, 3-months, and 12-months. Internal consistency reliability of the three items was $\alpha=0.77$ at baseline and exceeded $\alpha=0.81$ at 3- and 12-month time points.

Covariate measures—To adjust for potential confounding variables that could explain the association between pain and alcohol treatment outcomes, we selected covariate measures that were available in both datasets and that have been shown to be associated with alcohol treatment outcomes in prior studies [28–30], including treatment assignment, alcohol dependence severity, self-efficacy, motivation to change, temptation to drink when experiencing urges, gender, and psychiatric distress. In COMBINE, covariates included treatment assignments (behavioral intervention versus medication management and naltrexone versus placebo naltrexone), scores on the Alcohol Dependence Scale (ADS) [31], the University of Rhode Island Change Assessment (URICA) readiness scale [32], the Alcohol Abstinence Self-Efficacy (AASE) scale [33], and the Brief Symptom Inventory Global Severity Index (BSI) [34]. In UKATT, covariates included treatment assignment (social behavior network therapy versus motivation enhancement therapy), scores on the Leeds Dependence Questionnaire (LDQ) [35], the Readiness to Change Questionnaire (RCQ) [36], the AASE, and the General Health Questionnaire (GHQ) total score [37]. For both studies, we included motivation to change (URICA or RCQ), dependence severity (ADS or LDQ), gender, and treatment assignment as assessed at baseline. In COMBINE, we included self-efficacy (AASE), temptation (AASE), and psychiatric distress (BSI) as

assessed at baseline and end of treatment. In UKATT, we included psychiatric distress (GHQ), dependence severity (LDQ), and motivation (RCQ) as assessed at baseline and end of treatment.

Statistical Analyses

Measurement Model of Pain Interference and Intensity—All measurement models were estimated separately in the COMBINE and UKATT datasets using Mplus version 7.2 [38]. The fit of all models was evaluated by χ^2 values, the Root Mean Square Error of Approximation [RMSEA; [39]], and the Comparative Fit Index [CFI; [40]]. Models with RMSEA < 0.05 and CFI > 0.95 were considered a good fit to the observed data, [41] and models with RMSEA < 0.08 and CFI > 0.90 were considered a reasonable fit. Given the multiple indicators for pain in both studies, we created measurement models of pain within each dataset. Given that the pain items were ordered categorical, we used longitudinal graded response models [42–44] to create study-independent graded response scores for each individual at each time point. The graded response model is an extension of the two-parameter logistic item response theory (IRT) model to accommodate ordered categorical data [42]. IRT is used to characterize the association between the probability of endorsing an individual item with an underlying latent factor (i.e., trait; [45]). Graded response models were estimated separately in the COMBINE and UKATT studies in order to generate graded response scores for the pain latent factors at each time point (COMBINE: baseline, 16-, 26-, and 52-weeks; UKATT: baseline, 3-, and 12-months). Graded response scores were estimated using the expected a posteriori approach [46].

In the COMBINE study we used a planned missingness approach [47, 48] to accommodate all available data across the WHO-QOL and SF-12 measures. Using a latent variable approach with varying model constraints (to establish measurement invariance across time [49]), we estimated the latent pain graded response model at baseline (items from the WHO-QOL and SF-12), 16 weeks (item from the SF-12), 26 weeks (item from WHO-QOL), and 52 weeks (items from the WHO-QOL and SF-12).

Analyses of Pain Predicting Alcohol Treatment Outcomes—Cox proportional hazard models were used to estimate pain scores as a predictor of time-to-first heavy drinking lapse and time-to-first any drinking lapse. We conducted two series of Cox proportional hazard models in each dataset. First we examined the direct effect of baseline pain scores on the time-to-first heavy drinking day and time-to-first drinking day during the treatment period (COMBINE: 120 days; UKATT: 90 days). Second, we examined the direct effect of end-of-treatment pain scores (COMBINE: 16 week scores; UKATT: 3-month scores) on the time-to-first heavy drinking day and time-to-first drinking day during the follow-up (COMBINE: 365 days; UKATT: 90 days preceding the 12-month follow-up). We then repeated the Cox proportional hazard models with gender, dependence severity, motivation, self-efficacy, temptation, treatment assignment, and psychiatric distress as additional predictors of time-to-first heavy drinking day and time-to-first drinking day.

Results

Descriptive statistics of all continuous measures and the frequency (%) of item endorsement on the WHO-QOL and SF-12 items (COMBINE) as well as EQ-5D and SF-36 items (UKATT) are provided in Tables 2a (COMBINE) and 2b (UKATT).

Measurement Model of Pain

The longitudinal graded response models of pain indicators provided an adequate fit to the observed data in the COMBINE data (χ^2 (13)=70.04, RMSEA=0.06 (90% CI: 0.044, 0.070), CFI=0.99) and a reasonable fit in the UKATT data (χ^2 (43)=279.97, RMSEA=0.08 (90% CI: 0.077, 0.096), CFI=0.99). In UKATT a graded response model with only the pain intensity items had unacceptable model fit, thus the model including items of intensity and interference was retained. From these models we generated pain scores using the expected a posteriori approach (see Tables 2a and 2b for descriptive statistics and Table 3 for correlations with covariates).

Pain predicting time-to-first heavy drinking lapse and first any drinking lapse

Results from the Cox proportional hazards models of time-to-first heavy drinking day and time-to-first drinking day during treatment indicated significant effects of pain scores at baseline in predicting the odds of lapsing to heavy drinking during treatment in both the COMBINE and UKATT datasets. As seen in Table 4, in the COMBINE study, each unit increase in baseline pain scores (corresponding to a 1.37 SD increase) predicted an 11.6% increase in the hazard odds of lapsing to heavy drinking during treatment. For the UKATT study, each unit increase in baseline pain scores (corresponding to a 1.24 SD increase) predicted a 10% increase in the hazard odds of lapsing to any drinking and a 19% increase in the hazard odds of lapsing to heavy drinking during treatment.

Also shown in Table 4, results indicated significant effects of pain scores assessed at end-of-treatment in predicting the odds of lapsing and lapsing to heavy drinking following treatment in COMBINE, but not in UKATT. For COMBINE, each unit increase in end-of-treatment pain scores (1.57 SD increase) predicted a 10.7% increase in the hazard odds of lapsing to any drinking and a 16.3% increase in the hazard odds of lapsing to heavy drinking. For UKATT, pain scores at the end of treatment did not increase the hazard odds of lapsing following treatment. Importantly, there was only a small proportion of initial lapses that occurred in the 90 days prior to the 12-month follow-up in UKATT (3.7% for any drinking and 6.1% for heavy drinking lapses), reducing variability to predict the hazard probabilities of lapsing following treatment.

Pain predicting time-to-first lapse with covariate adjustment

The Cox proportional hazard models were then repeated with additional predictors of time-to-first heavy drinking day and time-to-first drinking day in both studies. For the models of lapses during treatment, pain scores at baseline remained a significant predictor of time-to-first heavy drinking day in UKATT, but were no longer significantly associated with time-to-first heavy drinking lapse in COMBINE or time-to-first drinking day in UKATT or

COMBINE. As shown in Table 5, dependence severity, motivation, self-efficacy, and psychiatric distress were all significant predictors of lapses during treatment in both studies.

For the models of lapses following treatment, pain scores at the end of treatment remained significant in predicting the odds of lapsing and lapsing to heavy drinking following treatment in COMBINE, but not in UKATT. Temptation and gender were also significantly associated with the odds of lapsing in COMBINE, whereas baseline self-efficacy, psychiatric health, and gender were associated with the odds of lapsing in UKATT (Table 6).

Discussion

This study provides evidence that pain latent factor scores, defined by both pain interference (COMBINE and UKATT) and pain intensity (UKATT only), significantly predicted time-to-first heavy drinking lapse and time-to-first lapse during or following AUD treatment in two separate clinical trials. Pain assessed at baseline was a significant predictor of lapses during treatment in both UKATT and COMBINE. Likewise, pain assessed at the end of treatment was a significant predictor of lapses following treatment in COMBINE. After covariate adjustment, pain remained a significant predictor of heavy drinking lapses during treatment in UKATT and a significant predictor of both heavy drinking lapses and any drinking lapses following treatment in COMBINE.

The lack of an association between pain, assessed at the end of treatment, and post-treatment lapses in UKATT in both unadjusted and covariate-adjusted analyses might be explained by the low frequency of initial lapses during the 12-month follow-up assessment, as well as the time lag from the end of treatment pain assessment (month 3) to the first assessment of follow-up drinking (month 9). The finding that pain was associated with lapses during treatment in the COMBINE study in the unadjusted analyses, but was not significant in the covariate-adjusted analyses, would suggest that other factors are more pertinent to lapses that occur during treatment. It may also be the case that some of the COMBINE patients who received naltrexone, which at low doses has recently been shown to be an effective treatment for chronic pain [50], may have experienced less pain during the course of treatment and receiving naltrexone might have also decreased the risk of lapsing in response to pain. As shown in Table 5, assignment to the naltrexone condition predicted a significantly lower risk of lapsing during treatment in COMBINE. The timing of the assessment of pain might also be important in predicting AUD treatment outcomes. Specifically, the experience of pain was much higher at baseline (as shown in Tables 2a and 2b) and in the unadjusted analyses the baseline assessment of pain was a significant predictor of lapses during treatment in UKATT and COMBINE, whereas end of treatment pain only predicted lapses following treatment in COMBINE.

Previous studies suggest that pain assessment and management could potentially lead to better pain and alcohol or substance use outcomes [52, 53]. The results from this study suggest that pain interference, in addition to pain intensity, may be an important target for treatment. Measurement of pain intensity was limited to only the UKATT dataset, thus it is impossible to know whether pain interference, pain intensity, or both are the most relevant

predictors. Although pain intensity and interference are arguably different aspects of the pain experience, multiple studies have indicated that they tend to be closely related [54–57]. In UKATT the correlation between the two SF-36 items that assessed pain interference and intensity exceeded 0.81 at all time-points, and the correlation between the pain interference item from the SF-36 and pain intensity item from the EQ-5D ranged from 0.37–0.62. Future research should continue to examine the association between pain and alcohol treatment outcomes, ideally using multidimensional assessments of pain intensity and interference (e.g., [54, 56, 58]).

The lack of multidimensional measurement of pain is a limitation of this study, and using self-report measures of pain and drinking outcomes is an additional limitation. COMBINE and UKATT collected biomarkers of alcohol use to corroborate self-reported data, but biomarkers of alcohol consumption only provide some incremental validity to self-reported consumption [59]. Furthermore, self-report has been accepted as an accurate method for measuring alcohol consumption [60]. The amount of follow-up data in the UKATT study, which only covered the 90 days prior to the 12-month follow-up assessment, limited the variability in lapses following treatment and increased the time lag between the end of treatment pain assessment and drinking outcomes in UKATT.

The results of this study have important implications for AUD treatment. Notably, clinicians and researchers should assess for pain interference and intensity as potential triggers for relapse. It may also be important to consider pain as a potential clinical target in behavioral interventions for AUD. For example, acceptance and commitment therapy, a gold standard treatment for chronic pain [63], has also been adapted for the treatment of addiction [64]. Likewise, pain management combined with cognitive behavioral treatment, relaxation and mindful walking was shown to be both feasible and acceptable among patients in a methadone maintenance program [65]. An integrated treatment incorporating both pain management and relapse prevention was shown to be effective in a small sample of patients with concurrent chronic pain and AUD [66]. Finally, mindfulness-based relapse prevention [67, 68], established from mindfulness-based stress reduction for chronic pain [69], has been shown to be efficacious in the treatment of AUD and substance use disorders [70, 71].

Acknowledgments

This research was supported by grants funded by the National Institute on Alcohol Abuse and Alcoholism, including support for KW, EM, TF (R01 AA022328, Witkiewitz, PI), MK was supported by a T32 training grant (T32 AA018108, McCrady, PI), and SAM was supported by a K05 award (K05 AA016928, Maisto, PI).

References

1. COMBINE Study Group. Testing combined pharmacotherapies and behavioral interventions in alcohol dependence: rationale and methods. *Alcohol Clin Exp Res*. 2003; 27:1107–22. [PubMed: 12878917]
2. UKATT Research Team. United Kingdom Alcohol Treatment Trial (UKATT): Hypotheses, design and methods. *Alcohol Alcohol*. 2001; 36:11–21. [PubMed: 11139410]
3. Miller, WR. Form 90: A Structured Assessment Interview for Drinking and Related Behaviors. Bethesda, MD: National Institute on Alcohol Abuse and Alcoholism; 1996. Project MA
4. Ware J, Kosinski M, Keller SD. A 12-Item Short-Form Health Survey: construction of scales and preliminary tests of reliability and validity. *Med Care*. 1996; 34:220–33. [PubMed: 8628042]

5. European Quality of Life Group. EuroQol--a new facility for the measurement of health-related quality of life. *Health Policy*. 1990; 16:199–208. [PubMed: 10109801]
6. Sutton SR. Interpreting relapse curves. *J Consult Clin Psychol*. 1979; 47:96–8. [PubMed: 429669]
7. Witkiewitz K, Marlatt GA. Relapse Prevention for Alcohol and Drug Problems: That Was Zen, This Is Tao. *Am Psychol*. 2004; 59:224–235. [PubMed: 15149263]
8. Connors GJ, Maisto SA, Donovan DM. Conceptualizations of relapse: A summary of psychological and psychobiological models. *Addiction*. 1996; 91:S5–S13. [PubMed: 8997777]
9. Moos RH, Moos BS. Rates and predictors of relapse after natural and treated remission from alcohol use disorders. *Addiction*. 2006; 101:212–22. [PubMed: 16445550]
10. Breivik, H.; Campbell, WL.; Nicholas, MK. *Clinical Pain Management: Practice and Procedures*. 2nd. London: Hodder Arnold; 2008.
11. Hoy D, Bain C, Williams G, March L, Brooks P, Blyth F, et al. A systematic review of the global prevalence of low back pain. *Arthritis Rheum*. 2012; 64:2028–2037. [PubMed: 22231424]
12. Johannes CB, Le TK, Zhou X, Johnston Ja, Dworkin RH. The prevalence of chronic pain in United States adults: results of an Internet-based survey. *J Pain*. 2010; 11:1230–1239. [PubMed: 20797916]
13. Woodrow KM, Eltherington LG. Feeling no pain: alcohol as an analgesic. *Pain*. 1988; 32:159–63. [PubMed: 3362554]
14. Trafton JA, Oliva EM, Horst DA, Minkel JD, Humphreys K. Treatment needs associated with pain in substance use disorder patients: implications for concurrent treatment. *Drug Alcohol Depend*. 2004; 73:23–31. [PubMed: 14687956]
15. Booker EA, Haig AJ, Geisser ME, Yamakawa K. Alcohol use self report in chronic back pain--relationships to psychosocial factors, function performance, and medication use. *Disabil Rehabil*. 2003; 25:1271–7. [PubMed: 14617444]
16. Caldeiro RM, Malte CA, Calsyn DA, Baer JS, Nichol P, Kivlahan DR, et al. The association of persistent pain with out-patient addiction treatment outcomes and service utilization. *Addiction*. 2008; 103:1996–2005. [PubMed: 18855809]
17. Larson MJ, Paasche-Orlow M, Cheng DM, Lloyd-Travaglini C, Saitz R, Samet JH. Persistent pain is associated with substance use after detoxification: a prospective cohort analysis. *Addiction*. 2007; 102:752–60. [PubMed: 17506152]
18. Sheu R, Lussier D, Rosenblum A, Fong C, Portenoy J, Joseph H, et al. Prevalence and characteristics of chronic pain in patients admitted to an outpatient drug and alcohol treatment program. *Pain Med*. 2008; 9:911–7. [PubMed: 18346064]
19. Brennan PL, Schutte KK, Moos RH. Pain and use of alcohol to manage pain: prevalence and 3-year outcomes among older problem and non-problem drinkers. *Addiction*. 2005; 100:777–86. [PubMed: 15918808]
20. Weiss RD, Potter JS, Griffin ML, McHugh RK, Haller D, Jacobs P, Gardin J, Fischer D, Rosen KD. Reasons for opioid use among patients with dependence on prescription opioids: the role of chronic pain. *J Subst Abuse Treat*. 2014; 47:140–5. [PubMed: 24814051]
21. Morasco BJ, Gritzner S, Lewis L, Oldham R, Turk DC, Dobscha SK. Systematic review of prevalence, correlates, and treatment outcomes for chronic non-cancer pain in patients with comorbid substance use disorder. *Pain*. 2011; 152:488–97. [PubMed: 21185119]
22. Morasco BJ, Corson K, Turk DC, Dobscha SK. Association between substance use disorder status and pain-related function following 12 months of treatment in primary care patients with musculoskeletal pain. *J Pain*. 2011; 12:352–9. [PubMed: 20851057]
23. Falk D, Wang XQ, Liu L, Fertig J, Mattson M, Ryan M, Johnson B, Stout R, Litten RZ. Percentage of subjects with no heavy drinking days: evaluation as an efficacy endpoint for alcohol clinical trials. *Alcohol Clin Exp Res*. 2010; 34:2022–34. [PubMed: 20659066]
24. Anton RF, O'Malley SS, Ciraulo DA, Cisler RA, Couper D, Donovan DM, et al. Combined pharmacotherapies and behavioral interventions for alcohol dependence: the COMBINE study: a randomized controlled trial. *JAMA*. 2006; 295:2003–17. [PubMed: 16670409]
25. UKATT Research Team. Effectiveness of treatment for alcohol problems: findings of the randomised UK alcohol treatment trial (UKATT). *BMJ*. 2005; 331:541. [PubMed: 16150764]

26. World Health Organization. World Health Organization Quality of Life. Geneva, Switzerland: World Health Organization; 1998.
27. Ruta D, Garratt A, Abdalla M, Buckingham K, Russell I. The SF 36 health survey questionnaire. A valid measure of health status. *BMJ*. 1993; 307:448–9. [PubMed: 8374477]
28. Witkiewitz K. Predictors of heavy drinking during and following treatment. *Psychol Addict Behav*. 2011; 25:426–38. [PubMed: 21480681]
29. Lowman C, Allen J, Stout RL. Replication and extension of Marlatt's taxonomy of relapse precipitants: overview of procedures and results. The Relapse Research Group. *Addiction*. 1996; 91 Suppl:S51–71. [PubMed: 8997781]
30. Hendershot CS, Witkiewitz K, George WH, Marlatt GA. Relapse prevention for addictive behaviors. *Subst Abuse Treat Prev Policy*. 2011; 6:17. [PubMed: 21771314]
31. Skinner, HA.; Horn, JL. Alcohol Dependence Scale (ADS) User's Guide. Toronto: Addiction Research Foundation; 1984.
32. DiClemente CC, Hughes SO. Stages of change profiles in outpatient alcoholism treatment. *J Subst Abuse*. 1990; 2:217–35. [PubMed: 2136111]
33. DiClemente CC, Carbonari JP, Montgomery RP, Hughes SO. The Alcohol Abstinence Self-Efficacy scale. *J Stud Alcohol*. 1994; 55:141–8. [PubMed: 8189734]
34. Derogatis LR. The Brief Symptom Inventory: An introductory report. *Psychol Med*. 1983; 13:595–605. [PubMed: 6622612]
35. Raistrick D, Bradshaw J, Tober G, Weiner J, Allison J, Healey C. Development of the Leeds Dependence Questionnaire (LDQ): a questionnaire to measure alcohol and opiate dependence in the context of a treatment evaluation package. *Addiction*. 1994; 89:563–72. [PubMed: 8044122]
36. Heather N, Honekopp J. A revised edition of the Readiness to Change Questionnaire. *Addict Res Theory*. 2008; 16:421–433.
37. Goldberg DP, Hillier VF. A scaled version of the General Health Questionnaire. *Psychol Med*. 1979; 9:139–45. [PubMed: 424481]
38. Muthén LK, Muthén BO. Mplus users guide (Version 7). 2012
39. Browne, MW.; Cudeck, R. Alternative ways of assessing model fit. In: Bollen, KA.; Long, JS., editors. *Test Struct Equ Model*. Beverly Hills, CA: Sage; 1993. p. 136-162.
40. Bentler PM. Comparative fit indexes in structural models. *Psychol Bull*. 1990; 107:238–46. [PubMed: 2320703]
41. Hu LT, Bentler PM. Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Struct Equ Model*. 1999; 6:1–55.
42. Samejima F. Estimation of latent ability using a response pattern of graded scores. *Psychom Monogr Supplement*. 1969; 34
43. Samejima, F. Graded response model. In: van der Linden, WJ.; Hambleton, R., editors. *Handbook of Modern Item Response Theory*. New York, NY: Springer-Verlag; 1997.
44. Skrondal, A.; Rabe-Hesketh, S. *Generalized Latent Variable Modeling: Multilevel, Longitudinal and Structural Equation Models*. Boca Raton, FL: Chapman & Hall; 2004.
45. Embretson, SE.; Reise, S. *Item Response Theory for Psychologists*. Mahwah, NJ: Erlbaum Publishers; 2000.
46. Uebersax JS, Grove WM. A latent trait finite mixture model for the analysis of rating agreement. *Biometrics*. 1993; 49:823–35. [PubMed: 10798855]
47. Enders, CK. *Applied Missing Data Analysis*. New York, NY: Guilford Press; 2010.
48. Graham JW. Missing data analysis: making it work in the real world. *Annu Rev Psychol*. 2009; 60:549–76. [PubMed: 18652544]
49. Byrne BM, Shavelson RJ, Muthén BO. Testing for the equivalence of factor covariance and mean structures: The issue of partial measurement invariance. *Psychol Bull*. 1989; 105:456–466.
50. Younger J, Parkitny L, McLain D. The use of low-dose naltrexone (LDN) as a novel anti-inflammatory treatment for chronic pain. *Clin Rheumatol*. 2014; 33:451–9. [PubMed: 24526250]
51. Riley JL, King C. Self-report of alcohol use for pain in a multi-ethnic community sample. *J Pain*. 2009; 10:944–52. [PubMed: 19712901]

52. Savage SR. Addiction in the treatment of pain: significance, recognition, and management. *J Pain Symptom Manage.* 1993; 8:265–78. [PubMed: 7525743]
53. Ziegler PP. Addiction and the treatment of pain. *Subst Use Misuse.* 2005; 40:1945–54. [PubMed: 16282087]
54. Tan G, Jensen MP, Thornby JI, Shanti BF. Validation of the Brief Pain Inventory for chronic nonmalignant pain. *J Pain.* 2004; 5:133–137. [PubMed: 15042521]
55. Wells N. Pain intensity and pain interference in hospitalized patients with cancer. *Oncol Nurs Forum.* 2000; 27:985–991. [PubMed: 10920837]
56. Yamashiro K, Arimura T, Iwaki R, Jensen MP, Kubo C, Hosoi M. A multidimensional measure of pain interference: Reliability and validity of the pain disability assessment scale. *Clin J Pain.* 2011; 27:338–343. [PubMed: 21178590]
57. Campbell, WI.; Vowles, KE. Practical methods for pain intensity measurements. In: Rice, A.; Breivik, H.; Campbell, WI.; Nicholas, MK., editors. *Clin pain Manag Pract Proced.* 2nd. New York: Hodder Arnold; 2008. p. 12-27.
58. Cleeland CS, Ryan KM. Pain assessment: global use of the Brief Pain Inventory. *Ann Acad Med.* 1994; 23:129–38.
59. Bertholet N, Winter MR, Cheng DM, Samet JH, Saitz R. How accurate are blood (or breath) tests for identifying self-reported heavy drinking among people with alcohol dependence? *Alcohol Alcohol.* 49:423–9. [PubMed: 24740846]
60. Del Boca FK, Darkes J. The validity of self-reports of alcohol consumption: state of the science and challenges for research. *Addiction.* 2003; 98 Suppl 2:1–12. [PubMed: 14984237]
61. Højsted J, Sjøgren P. Addiction to opioids in chronic pain patients: a literature review. *Eur Jounal Pain.* 2007; 11:490–518.
62. Ballantyne JC, LaForge KS. Opioid dependence and addiction during opioid treatment of chronic pain. *Pain.* 2007; 129:235–55. [PubMed: 17482363]
63. McCracken LM, Vowles KE. Acceptance and Commitment Therapy and mindfulness for chronic pain: Model, process, and progress. *Am Psychol.* 2014; 69:178–187. [PubMed: 24547803]
64. Hayes, SC.; Levin, M. *Mindfulness and Acceptance for Addictive Behaviors: Applying Contextual CBT to Substance Abuse and Behavioral Addictions.* Oakland, CA: New Harbinger Publications; 2012.
65. Barry DT, Savant JD, Beitel M, Cutter CJ, Schottenfeld RS, Kerns RD, et al. The feasibility and acceptability of groups for pain management in methadone maintenance treatment. *J Addict Med.* 8:338–44. [PubMed: 25100310]
66. Currie SR, Hodgins DC, Crabtree A, Jacobi J, Armstrong S. Outcome from integrated pain management treatment for recovering substance abusers. *J Pain.* 2003; 4:91–100. [PubMed: 14622720]
67. Witkiewitz K, Marlatt GA, Walker D. Mindfulness-Based Relapse Prevention for Alcohol and Substance Use Disorders. *J Cogn Psychother.* 2005; 19:211–228.
68. Bowen, S.; Chawla, N.; Marlatt, GA. *Mindfulness-Based Relapse Prevention for Addictive Behaviors: A Clinician's Guide.* New York, NY: Guilford Press; 2010. p. 179
69. Kabat-Zinn J. An outpatient program in behavioral medicine for chronic pain patients based on the practice of mindfulness meditation: theoretical considerations and preliminary results. *Gen Hosp Psychiatry.* 1982; 4:33–47. [PubMed: 7042457]
70. Bowen S, Chawla N, Collins SE, Witkiewitz K, Hsu S, Grow J, et al. Mindfulness-based relapse prevention for substance use disorders: a pilot efficacy trial. *Subst Abus.* 2009; 30:295–305. [PubMed: 19904665]
71. Bowen S, Witkiewitz K, Clifasefi SL, Grow J, Chawla N, Hsu SH, et al. Relative efficacy of mindfulness-based relapse prevention, standard relapse prevention, and treatment as usual for substance use disorders: a randomized clinical trial. *JAMA Psychiatry.* 2014; 71:547–56. [PubMed: 24647726]

Table 1
Demographic Characteristics, Design, and Eligibility Criteria for COMBINE and UKATT

Demographic Characteristics	COMBINE	UKATT
Sample size	1383	742
Gender Male N (%)	955 (69.0%)	550 (74.1%)
Age Mean (SD)	44.4 (10.2)	41.6 (10.1)
Race and Ethnicity		
White N (%)	1062 (76.8%)	709 (95.6%)
Hispanic American N (%)	155 (11.2%)	-
Black N(%)	109 (7.9%)	10 (1.3%)
American Indian/Alaska Native N (%)	18 (1.3%)	--
Multi-racial	18 (1.3%)	--
Asian American/Pacific Islander N (%)	4 (0.3%)	--
Indian or other Asian N(%)	--	15 (2.0%)
Pakistani N(%)	--	1 (0.1%)
"Other" N(%)	17 (1.2%)	7 (0.9%)
Married or cohabitating N (%)	618 (44.7%)	290 (39.1)%
Full or part-time employment N (%)	982 (71.0%)	257 (34.8%)
Higher education or equivalent N (%)	958 (70.6%)	143 (19.3%)
Design		
Randomization to treatment	9 groups	2 groups
Length of treatment	16 weeks	8 to 12 weeks
Follow-up assessments	12 months	12 months
Exclusion criteria		
Age	<18	<16
Reading level	Literate	Literate
Comorbid psychiatric diagnoses	X	X
Unable to identify collateral informant		X
Severe cognitive impairment		X
Residential instability		X
Other illicit drug dependence	X	

Table 2a
Descriptive Statistics, N (%) or Mean (Standard Deviation), for COMBINE Study Variables

	Baseline	16-weeks	26-weeks
WHO-QOL pain interference			
1-Not at all	845 (62%)	--	705 (66%)
2-A little	335 (25%)	--	221 (21%)
3-A moderate amount	120 (9%)	--	95 (9%)
4-Very much/extremely	51 (4%)	--	40 (4%)
SF-12 pain interference			
1-Not at all	796 (59%)	762 (69%)	--
2-A little bit	342 (25%)	237 (22%)	--
3-Moderately	137 (10%)	70 (6%)	--
4-Quite a bit/extremely	81 (6%)	33 (3%)	--
Pain graded response scores (range -0.5 – 2.5)	0.13 (0.73)	0.11 (0.64)	0.13 (0.77)
Alcohol dependence severity (ADS)	16.68 (7.32)	--	--
Motivation to change (URICA)	10.61 (1.53)	--	--
Self-efficacy (AASE-Confidence)	2.61 (0.74)	3.50 (0.92)	3.40 (1.06)
Temptation (AASE-Temptation urge)	3.24 (0.91)	2.35 (0.95)	2.38 (1.06)
Psychiatric distress (BSI-GSI)	60.29 (10.91)	52.32 (12.11)	52.45 (12.99)

Note. WHO-QOL=World Health Organization Quality of Life survey; SF-12=Short Form Health Survey; BSI=Brief Symptom Inventory; ADS = Alcohol Dependence Scale; URICA = University of Rhode Island Change Assessment, AASE = Alcohol Abstinence Self-Efficacy Scale.

Table 2b
Descriptive Statistics, N (%) or Mean (Standard Deviation), for UKATT Study Variables

	Baseline	3-months
EQ-5D pain intensity		
1-No pain or discomfort	265 (36%)	284 (44%)
2-Some pain or discomfort	419 (57%)	332 (51%)
3-Extreme pain or discomfort	47 (7%)	34 (5%)
SF-36 pain interference		
1-Not at all	207 (28%)	273 (42%)
2-A little bit	178 (24%)	150 (23%)
3-Moderately	152 (21%)	92 (14%)
4-Quite a bit	122 (17%)	93 (15%)
5-Extremely	72 (10%)	39 (6%)
SF-36 pain intensity		
1-None	109 (14.9%)	169 (26%)
2-Very mild	95 (13.0%)	105 (16%)
3-Mild	133 (18.2%)	102 (16%)
4-Moderate	241 (32.9%)	174 (27%)
5-Severe	110 (15.0%)	74 (11%)
6-Very severe	44 (6.0%)	24 (4%)
Pain graded response scores (range -1.49 – 2.49)	0.06 (0.81)	-0.05 (0.82)
Alcohol dependence severity (LDQ)	15.68 (8.06)	10.62 (8.55)
Motivation to change (RCQ)	18.15 (7.31)	14.75 (8.36)
Self-efficacy (AASE-Confidence)	51.13 (17.56)	--
Temptation (AASE-Temptation urge)	62.49 (17.00)	--
Psychiatric health (GHQ Total)	37.91 (18.02)	28.29 (18.94)

Note. EQ-5D=European Quality of Life survey; SF-36=Short Form Health Survey; GHQ=General Health Questionnaire; AASE = Alcohol Abstinence Self-Efficacy Scale; LDQ = Leeds Dependence Questionnaire; RCQ = Readiness to Change Questionnaire; GHQ = General Health Questionnaire.

Correlations between Pain Scores and Covariates in the COMBINE study (Below Diagonal) and UKATT Study (Above Diagonal)**Table 3**

	1	2	3	4	5	6	7	8	9	10	11	12
1. Baseline Pain Scores		.89*	.26*	.27*	.03	.01	-.23*	--	.19*	--	.38*	.44*
2. End-of treatment Pain	.81*		.23*	.36*	.02	.08	-.24*	--	.19*	--	.34*	.53*
3. Baseline LDQ/ADS	.20*	.18*		.42*	.10*	.07	-.42*	--	.56*	--	.53*	.27*
4. End-of-treatment LDQ	--	--	--	--	-.03	.15*	-.32*	--	.33*	--	.26*	.67*
5. Baseline URICA/RCQ	.03	.05	.24*	--	--	.31*	.09*	--	.08*	--	.07	-.02
6. End-of-treatment RCQ	--	--	--	--	--	--	.001	--	.10*	--	.06	.10*
7. Baseline self-efficacy	-.10*	-.06*	-.15*	--	.02	--	--	--	-.60*	--	-.35*	-.25*
8. End-of-treatment self-efficacy	-.13*	-.21*	-.14*	--	.004	--	.19*	--	--	--	--	.25*
9. Baseline temptation	.07*	.06*	.28*	--	.06*	--	-.48*	-.10*	--	--	.42*	.25*
10. End-of-treatment temptation	.07*	.15*	.16*	--	.02	--	-.17*	-.64*	.23*	--	--	--
11. Baseline BSI/GHQ	.36*	.33*	.46*	--	.14*	--	-.24*	-.20*	.25*	.16*	--	.44*
12. End-of-treatment BSI/GHQ	.30*	.41*	.29*	--	.03	--	-.11*	-.49*	.15*	.48*	.55*	--

Note.

*
 $p < 0.05$;

-- = measure that was not assessed in COMBINE/UKATT;

End-of-treatment = Week 16 (COMBINE) and Month 3 (UKATT); ADS = Alcohol Dependence Scale; URICA = University of Rhode Island Change Assessment; BSI = Brief Symptom Inventory Global Severity Index; LDQ = Leeds Dependence Questionnaire; RCQ = Readiness to Change Questionnaire; GHQ = General Health Questionnaire.

Table 4
Results from Cox Regression Models of Pain Scores Predicting Time-to-first Drinking
Day and Time-to-first Heavy Drinking Day

COMBINE	OR (95% CI)	B (SE)
During Treatment Lapses		
Baseline Pain → Lapse to any drinking	1.04 (0.96, 1.13)	0.04 (0.04)
Baseline Pain → Lapse to heavy drinking	1.12 (1.03, 1.21)	0.11 (0.04) **
Post Treatment Lapses		
Week 16 Pain → Lapse to any drinking	1.107 (1.10, 1.12)	0.10 (0.004) **
Week 16 Pain → Lapse to heavy drinking	1.163 (1.15, 1.17)	0.15 (0.004) **
UKATT	OR	B (SE)
During Treatment Lapses		
Baseline Pain → Lapse to any drinking	1.10 (1.01, 1.21)	0.10 (0.05)*
Baseline Pain → Lapse to heavy drinking	1.19 (1.08, 1.32)	0.18 (0.05) **
Post Treatment Lapses		
Month 3 Pain → Lapse to any drinking	0.97 (0.69, 1.36)	-0.03 (0.17)
Month 3 Pain → Lapse to heavy drinking	1.04 (0.78, 1.37)	0.03 (0.14)

**
 $p < 0.01$;

OR (95% CI)=odds ratio (95% Confidence Interval); B=unstandardized regression coefficient (SE=Standard Error)

Table 5
Results from Cox Regression Models during Treatment with Covariates

	Time-to-first heavy drinking day		Time-to-first drinking day	
	OR (95% CI)	B (SE)	OR (95% CI)	B (SE)
COMBINE				
Baseline pain scores	1.08 (.98, 1.18)	.07 (.05)	1.01 (.92, 1.11)	.01 (.05)
Baseline Percent drinking days	0.99 (.994, .999)	-.004 (.001)*	0.99 (.994, .999)	-.004 (.001)*
Alcohol dependence severity (ADS)	1.01 (1.003, 1.02)	.01 (.005)*	0.99 (.979, .999)	-.01 (.005)*
Motivation to change (URICA)	0.91 (.87, .95)	-.10 (.02)**	0.89 (.86, .94)	-.11 (.02)**
Self-efficacy (AASE-Confidence)	0.84 (.76, .94)	-.17 (.05)**	0.89 (.80, .98)	-.12 (.05)*
Temptation (AASE-Temptation urge)	1.06 (.97, 1.2)	.06 (.04)	1.08 (1.001, 1.2)	.08 (.04)*
Psychiatric distress (BSI GSI)	1.002 (.99, 1.01)	.002 (.004)*	1.01 (1.003, 1.02)	.01 (.004)**
Treatment (Combined behavioral intervention = 1)	1.02 (.89, 1.17)	.02 (.07)	0.92 (.80, 1.04)	-.09 (.07)
Treatment (Naltrexone=1)	0.86 (.75, .98)	-.16 (.07)*	0.86 (.75, .98)	-.16 (.07)*
Gender (Male=1)	0.82 (.70, .96)	-.20 (.08)*	0.69 (.60, .79)	-.37 (.07)**;
UKATT				
Baseline pain scores	1.19 (1.06, 1.34)	.18 (.06)**	1.10 (.98, 1.23)	.10 (.06)
Baseline Percent drinking days	1.00 (.99, 1.004)	.001 (.002)	1.00 (1.00, 1.01)	.004 (.002)
Alcohol dependence severity (LDQ)	1.03 (1.02, 1.05)	.03 (.006)**	1.02 (1.01, 1.04)	.02 (.006)**
Motivation to change (RCQ)	0.97(.96, .98)	-.03 (.01)**	0.96 (.95, .97)	-.04 (.005)**
Self-efficacy (AASE-Confidence)	0.98 (.97, .982)	-.02 (.003)**	0.98 (.97, .98)	-.03 (.003)**
Temptation (AASE-Temptation urge)	1.05 (.94, 1.2)	.05 (.06)	1.04 (.94, 1.15)	.04 (.05)
Psychiatric health (GHQ Total)	0.99 (.98, 0.992)	-.01 (.003)**	0.99 (.979, .991)	-.02 (.003)**
Treatment (Social behavior network therapy = 1)	0.85 (.71, 1.01)	-.17 (.09)	1.01 (.84, 1.20)	.01 (.09)
Gender (Male = 1)	1.15 (.94, 1.41)	.14 (.10)	1.11 (.91, 1.34)	.10 (.10)

* $p < 0.05$;

** $p < 0.01$;

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

OR (95% CI)=odds ratio (95% Confidence Interval); B=unstandardized regression coefficient (SE=Standard Error); ADS = Alcohol Dependence Scale; URICA = University of Rhode Island Change Assessment, AASE = Alcohol Abstinence Self-Efficacy Scale; BSI GSI = Brief Symptom Inventory Global Severity Index; LDQ = Leeds Dependence Questionnaire; RCQ = Readiness to Change Questionnaire; GHQ = General Health Questionnaire.

Table 6
Results from Cox Regression Models following Treatment with Covariates

	Time-to-first heavy drinking day		Time-to-first drinking day	
	OR (95% CI)	B (SE)	OR (95% CI)	B (SE)
COMBINE				
End of treatment pain scores	1.44 (1.07, 1.92)	.36 (.15) *	1.54 (1.10, 2.15)	.43 (.17) *
Alcohol dependence severity (ADS)	1.01 (.98, 1.03)	.01 (.01)	1.01 (.98, 1.04)	.01 (.02)
Motivation to change (URICA)	1.09 (.97, 1.21)	.08 (.06)	1.06 (.93, 1.21)	.06 (.07)
End of treatment self-efficacy (AASE-Confidence)	0.95 (.77, 1.18)	-.05 (.11)	0.89 (.70, 1.15)	-.11 (.13)
End of treatment temptation (AASE-Temptation)	1.99 (1.61, 2.48)	.69 (.11) **	1.73 (1.29, 2.33)	.55 (.15) **
End of treatment psychiatric distress (BSI GSI)	0.99 (.97, 1.01)	-.01 (.01)	0.97 (0.95, 0.99)	-.03 (.01) *
Treatment (Combined behavioral intervention = 1)	0.82 (.58, 1.17)	-.20 (.18)	0.97 (.65, 1.43)	-.04 (.20)
Treatment (Naltrexone=1)	0.83 (.59, 1.18)	-.19 (.18)	0.77 (.51, 1.14)	-.27 (.21)
Gender (Male=1)	0.57 (.39, .84)	-.56 (.20) **	0.50 (.33, .77)	-.69 (.21) **
UKATT				
Time-to-first heavy drinking day		Time-to-first drinking day		
	OR (95% CI)	B (SE)	OR (95% CI)	B (SE)
End of treatment pain scores	0.81 (0.51, 1.28)	-.22 (.24)	0.60 (.34, 1.06)	-.51 (.27)
End of treatment alcohol dependence severity	1.04 (0.98, 1.11)	.04 (.03)	1.01 (.95, 1.08)	.01 (.03)
End of treatment motivation to change (RCQ)	0.98 (0.95, 1.01)	-.02 (.02)	0.98 (.95, 1.02)	-.02 (.02)
Self-efficacy (AASE-Confidence)	0.98 (.96, .99)	-.02 (.01) *	0.99 (.97, 1.03)	-.002 (.01)
Temptation (AASE-Temptation urge)	0.89 (.63, 1.25)	-.12 (.18)	0.73 (.43, 1.24)	-.31 (.27)
End of treatment psychiatric health (GHQ Total)	0.99 (.97, 1.03)	-.001 (.02)	1.04 (1.01, 1.08)	.04 (.02) *
Treatment (Social behavior network therapy = 1)	0.88 (.46, 1.68)	-.13 (.33)	1.94 (.82, 4.60)	.66 (.42)
Gender (Male = 1)	1.41 (.67, 3.01)	.34 (.39)	0.35 (.12, .98)	-1.06 (.53) *

* $p < 0.05$;

** $p < 0.01$;

OR (95% CI)=odds ratio (95% Confidence Interval); B=unstandardized regression coefficient (SE=Standard Error); ADS = Alcohol Dependence Scale; URICA = University of Rhode Island Change Assessment; AASE = Alcohol Abstinence Self-Efficacy Scale; BSI GSI = Brief Symptom Inventory Global Severity Index; LDQ = Leeds Dependence Questionnaire; RCQ = Readiness to Change Questionnaire; GHQ = General Health Questionnaire.