Published in final edited form as:
Addiction. 2010 April ; 105(4): 626–636. doi:10.1111/j.1360-0443.2009.02820.x.

Mechanisms of Behavior Change in Alcoholics Anonymous: Does AA lead to better alcohol use outcomes by reducing depression symptoms?

John F. Kelly,
Center for Addiction Medicine, Department of Psychiatry, Massachusetts General Hospital & Harvard Medical School, 60 Staniford St., Suite 120, Boston, MA 02114

Robert L. Stout,
Decision Sciences Institute/PIRE, 1005 Main St., Pawtucket, RI 02860-7802

Molly Magill,
Brown University, Center for Alcohol and Addiction Studies, Providence RI 02912

J. Scott Tonigan, and
Center on Alcoholism, Substance Abuse and Addiction (CASAA), 2650 Yale SE., Suite 243, Albuquerque, NM 87106

Maria E. Pagano
Case Western Reserve University School of Medicine, Department of Psychiatry, Division of Child Psychiatry, W.O. Walker Center, 10524 Euclid Avenue

Abstract

Rationale—Indices of negative affect, such as depression, have been implicated in stress-induced pathways to alcohol relapse. Empirically-supported continuing care resources, such as Alcoholics Anonymous (AA), emphasize reducing negative affect to reduce relapse risk, but little research has been conducted to examine putative affective mechanisms of AA's effects.

Method—Using lagged, controlled, hierarchical linear modeling and mediational analyses this study investigated whether AA participation mobilized changes in depression symptoms and whether such changes explained subsequent reductions in alcohol use. Alcohol dependent adults (N = 1,706), receiving treatment as part of a clinical trial, were assessed at intake, 3, 6, 9, 12, and 15 months.

Results—Findings revealed elevated levels of depression compared to the general population, which decreased during treatment and then remained stable over follow-up. Greater AA attendance was associated with better subsequent alcohol use outcomes and decreased depression. Greater depression was associated with heavier and more frequent drinking. Lagged, mediation analyses revealed that the effects of AA on alcohol use was partially mediated by reductions in depression symptoms. However, this salutary effect on depression itself appeared to be explained by AA's proximal effect on reducing concurrent drinking.

Conclusions—AA attendance was both concurrently and predictively associated with improved alcohol outcomes. Although AA attendance was additionally associated with subsequent improvements in depression, it did not predict such improvements over and above concurrent

Correspondence: John F. Kelly, Massachusetts General Hospital, Department of Psychiatry, Center for Addiction Medicine, 60 Staniford St., Suite 120, Boston, MA 02114. Tel: 617-643-1980. Fax: 617-643-1998. jkelly11@partners.org..

Conflict of Interest Declaration: This study was funded by NIAAA R21AA016762. The authors have no financial interests, relationships, or affiliations relevant to this manuscript.

alcohol use. AA appears to lead both to improvements in alcohol use and psychological and emotional well-being, which, in turn, may reinforce further abstinence and recovery-related change.

Keywords

Alcoholics Anonymous; mechanisms; alcohol dependence; depression; self-help groups; mutual-help groups

Introduction

Affect regulation problems are common among individuals with alcohol use disorders (AUD [1-3]). Indices of negative affect, such as depression, have also been implicated in stress-induced pathways to relapse [4-6]. Depression symptoms may represent difficulties that predate problems with alcohol or drug use. Alternatively, symptoms may emerge later either independent of, or resulting from, neurophysiologic and psychosocial consequences of alcohol misuse [7-8]. Chronic alcohol misuse, for example, can cause direct neurotoxic effects that compromise mood-regulating serotonergic and dopaminergic systems [9]. Alcohol misuse also can cause mood problems indirectly, through inadequate nutritional intake or absorption of certain vitamins (e.g., thiamin) that may compound the deleterious effects of heavy consumption [10-12]. From a psychosocial perspective, the disinhibiting pharmacological properties of alcohol can lead to behaviors that result in psychological distress often characterized by feelings of sadness, remorse, self-blame, and self-loathing [13]. While many of these symptoms may remit with alcohol abstinence (e.g., [14]), affective distress clearly presents early recovery challenges for many individuals with AUDs.

Alcohol and other drug treatment programs often seek to enhance outcomes by alleviating affective symptoms through medical stabilization, pharmacotherapy, and psychosocial interventions (e.g., [15]). A primary goal is to reduce the intensity and duration of negative affect, and to enhance affect tolerance and subjective well-being, which in turn should reduce stress-related drug cravings and decrease relapse risk. Evidence-based adjunctive continuing care resources [16-17], such as Alcoholics Anonymous (AA), also emphasize the importance of affect regulation in relapse prevention [18-19]. While AA does not make as explicit a case for the role of depression in alcohol relapse as it does for anger (e.g., [19], pg 64), there are allusions to it throughout the AA literature (e.g., “...we couldn’t control our emotional natures, we were a prey to misery and depression, we couldn’t make a living, we had a feeling of uselessness, we were full of fear, we were unhappy...”; ([19], pg 52).

Because of the robust relationship between attendance at AA meetings and improved drinking outcomes [20-22], common social therapeutic group elements may operate. These include a feeling of social support and belonging captured in the group therapy concepts of “universality” and “instillation of hope” [23]. The attitudinal and behavioral shifts from AA participation are also intended to produce a “joy of living” ([18], pg 106). Thus, AA participation may serve as an antidote to the negative affect associated with early recovery and relapse. Howard and colleagues’ [24] general model of therapeutic change emphasizes a sequential mediational process beginning with “remoralization” (enhancement of subjective well-being), followed by “remediation” (symptomatic relief), and “rehabilitation” (replacing maladaptive patterns of functioning with more adaptive approaches). Since AA is likely to operate through similar therapeutic principles as more formal interventions [13,25], AA group meeting attendance might not only enhance subjective well-being as argued in the AA literature, but also remediate negative affect through psychotherapeutic processes.

Research has shown that AA is effective as a stand-alone or adjunctive treatment [20,26-28,22], but these studies have typically had small samples and have not employed longitudinal designs that enhance causal inference [29]. Also, the mechanisms of AA's beneficial effects are only beginning to be revealed. While many studies have examined the relationship between AA attendance and drinking outcome (e.g., [30-33]), few have considered how AA may actually facilitate recovery. In this emerging area, studies that have performed formal mediation tests suggest that coping, motivation, self-efficacy, social network changes, and spirituality may be some of the mechanisms through which AA facilitates salutary behavior change [13]. Yet, despite the importance of negative affect in the AA literature and in well-known theories of relapse (e.g., [5]), amelioration of negative affect as a mediator of AA's effects has seldom been investigated.

In previous work, we examined the role of anger in recovery through AA. Despite an explicit focus on reducing anger and resentment in AA's core literature [34,19], anger was not found to mediate alcohol outcomes among AA attendees [35]. Currently, we examine how AA may affect depression symptoms and whether such changes lead to subsequent changes in alcohol use using a large alcohol dependent sample (Project MATCH; [36]).

Since negative affect is broadly implicated in theories of relapse (e.g., [37,5]), we first considered levels of depression in this treatment seeking sample compared to those in the general population. Second, we examined the clinical course of depression over the 15 months of study follow-up. Third, we tested the nature of the relationship between AA attendance and depression over time, and whether any beneficial effects of AA on drinking might be mediated by reductions in depression symptoms. Importantly, given that depression symptoms often remit following cessation of drinking and continue to diminish with abstinence [37-38], we sought to determine whether AA was associated with reductions in depression symptoms over and above the effects of abstinence from alcohol. In line with criteria for enhancing cause-effect conclusions in mechanisms of behavior change [39,29], this study also used lagged hierarchical linear modeling to test depression as a purported affective mediator over time controlling for important static and time-varying covariates.

Method

Subjects

The current study is based on a sample of 1,706 Project MATCH participants followed through 15 months (n = 764 aftercare subjects [80% male]; n = 942 outpatient subjects [72% male). Of the two samples, outpatient participants were significantly younger, more residentially stable, and less dependent on alcohol than aftercare patients [40]. Patients in both trial arms (95% outpatient, 98% aftercare) primarily met criteria for alcohol dependence (SCID for DSM-III-R; [41]). See Table 1 for additional sample characteristics.

Procedures

Subjects were randomly assigned to one of three individually-delivered, psychosocial interventions: Cognitive Behavioral Therapy (CBT; [42]), Motivational Enhancement Therapy (MET; [43]), and Twelve-Step Facilitation Therapy (TSF; [44]). TSF and CBT consisted of 12 weekly sessions while MET included only four sessions (weeks 1, 2, 6 and 12). Participants were reassessed 3, 6, 9, 12 and 15 months following treatment. In both study arms, follow-up rates remained over 90%. More complete details regarding this trial are available elsewhere (e.g., [45]).

Measures

Alcohol use—Estimates of alcohol consumption were assessed using the Form 90 [46-47]), an interview procedure combining calendar-based Timeline Followback [48] and drinking pattern estimation [49] methods. The Form 90 also elicits information about drug use, treatment, incarceration, and AA involvement during the past 90 days.

Alcoholics Anonymous attendance—AA attendance was assessed using the Form 90, which captured the number of AA meetings attended during the past 90 days at intake and 3, 6, 9, 12, and 15 months. Percent of days attending AA was created and used for all analyses.

Depression measure—Depression symptoms were assessed using the Beck Depression Inventory (BDI; [50]). This 21-item measure assesses depression symptom severity in the past week with a score range of 0 to 63, and higher values indicate greater depression severity. The measure is well-established psychometrically with good internal consistency, test-retest stability, and construct validity (see [51] for review).

Statistical methods

Data preparation and descriptive analyses—The dependent variables (percent days abstinent [PDA], drinks per drinking day [DDD]) and the independent AA attendance variable required transformation. PDA received an arcsine transformation, and DDD was given a square root transformation [45,52]. AA attendance was log transformed. We ran the models with and without log transformation of AA attendance; no differences in findings were demonstrated. For easier interpretation, we report descriptive statistics using the untransformed variables. Due to the large sample size, we set the threshold for statistical significance at .01.

Lagged, controlled mediation analyses—To examine the relationship between AA attendance, depression symptoms, and alcohol use, we employed lagged hierarchical linear models (HLM) with both static and time-varying covariates. Separate HLM models were run for the aftercare and outpatient samples and for each alcohol use variable (PDA, DDD) to examine: (1) the independent (AA attendance) to dependent variable path, (2) the independent to mediator (depression) path, and (3) the mediator to dependent variable path. To make the tests prospective (lagged), we used paired time points in which time-varying predictors were used for time-varying outcomes (i.e., AA attendance in the past 90 days at month 3 to predict alcohol use outcomes at month 9, and AA at month 9 to predict alcohol use outcomes at month 15; similarly, we used depression at months 3 and 9 to predict drinking at months 9 and 15). Figure 1 shows the conceptual lagged model. The static covariates were age, ethnicity, gender, a gender by time interaction, marital status, employment status, number of prior alcohol-related treatments, treatment assignment, treatment site, the relevant baseline level of the dependent variable (depression, PDA, DDD), and an interaction between AA attendance and the baseline dependent variable.

Missing data—To address missing data, we used multiple imputation [53]. Missing data for key variables ranged from 0.05% for baseline AA attendance to 7.8% for drinking data at months 13–15. Since missing data patterns were non-monotone (i.e., many data were intermittently missing), the Markov Chain Monte Carlo (MCMC) method for multiple imputation was used [54]. We performed ten imputations using MI and MIANALYZE in SAS 9.1.3, and reported statistics are averaged across imputations. The degrees of freedom for the reported t-statistics vary by analysis due to adjustment [see 55].

Tests of statistical mediation—Mediation analyses included methods described by MacKinnon and colleagues [56-57]. The MacKinnon method tests directly for the existence

of a significant path from the independent variable (A) through the mediator (B) to the outcome (C) by computing a product of the regression coefficients for the A-B and B-C associations. This approach has been shown to maximize power in testing mediated effects [57].

Results

Sample characteristics

Demographic characteristics are shown in Table 1.

Table 2 provides descriptive data on the proportion of patients attending any AA meetings at each follow-up point, and the average number of meetings attended. Table 2 also shows drinking outcomes and depression scores over time and by treatment arm. As noted in prior research on this sample [58], rates of AA attendance were high even among patients assigned to treatments that did not explicitly endorse AA. As shown in Table 2, the proportion of patients who attended AA and the rates of attendance were higher in the aftercare compared to the outpatient sample over time. However, substantial numbers from both patient groups discontinued or declined attendance during the first year following treatment.

Depression symptoms across time

Repeated measures ANOVA revealed a significant main effect for change in depression over time for both aftercare ($F = 20.58$, $p < .0001$) and outpatient ($F = 58.55$, $p < .0001$) participants. Reductions in depression did not interact with treatment assignment (ps > .48). Figure 1 shows levels of depression symptoms at intake and across follow-up by gender and study arm. Depression levels in the sample were in the mild-moderate range ($M = 10.19$; $SD = 8.26$), were approximately two standard deviations higher than levels observed in general population samples (e.g., [59-60]), but were similar to alcohol dependent inpatient samples (e.g., [61]; $M = 10.03$ $SD = 8.39$). Women ($M = 11.6$, $SD = 9.14$) reported significantly more depression symptoms than men ($M = 9.8$, $SD = 7.91$) at intake only ($t = -4.48$, $p<.001$). Conversely, aftercare patients ($M = 10.6$, $SD = 8.6$) did not significantly differ from outpatients ($M = 9.8$, $SD = 7.9$) at the start of the MATCH treatment ($F = 3.70$, $p = .054$), but the outpatients' depression symptoms dropped more rapidly and stayed significantly lower over follow-up compared to aftercare patients (ps < .001 at each subsequent follow-up; see Figure 2).

Relation between AA, depression symptoms, and alcohol use

The Relationship between AA and alcohol use outcomes—Table 3 shows the relationship between AA and subsequent (lagged) alcohol use (PDA, DDD) for the outpatient and aftercare samples. As anticipated, there were robust associations between more frequent AA attendance and less frequent and less intense subsequent alcohol use across time in both study arms. However, there was also an AA x time interaction observed for aftercare patients for both PDA ($p=.05$) and DDD ($p=.007$). Follow-up tests of these interactions revealed that the relation between AA attendance during MATCH treatment (months 1–3) and subsequent past 90-day alcohol use at 9-month follow-up (months 7–9) was comparatively weaker than the relationship between AA at 9 months and alcohol use at 15 months. There was also one covariate that made an independent contribution in the aftercare and outpatient samples (not shown in Table 3): alcohol use in the prior period (ps < .0001). Older age was also independently associated with fewer PDA, but only among outpatients ($p =.004$).

The relationship between AA and depression symptoms—AA attendance was significantly and independently associated with reductions in depression symptoms for outpatient ($t = -2.93$, $p = .004$) and aftercare patients ($t = -2.45$, $p = .014$) when concurrent alcohol use was not in the model. However, when concurrent alcohol use was controlled to test the independent effect of AA on depression symptoms, the contribution of AA attendance in predicting depression symptoms was rendered non-significant. This suggests that the salutary influence of AA on reducing depression is related to, and may be explained by, AA's proximal effect on reducing alcohol use. Table 4 shows the full model with concurrent alcohol use controlled. Two additional covariates made significant contributions among aftercare patients: single/unmarried patients reported more depression symptoms ($F = 7.08$, $p = .008$), and greater concurrent PDA and DDD predicted greater subsequent depression (ps < .0001). Among outpatients, Whites had significantly higher depression scores than non-Whites ($F = 12.34$, $p = .0005$), as did those who were unemployed ($F = 13.35$, $p = .0003$).

The relationship between depression symptoms and alcohol use outcomes—Depression symptoms were independently associated with lower PDA ($t = -5.00$, $p < .0001$) and greater DDD ($t = 5.13$, $p < .0001$) among outpatients, and aftercare patients (PDA $t = -4.01$, $p < .0001$; DDD $t = 3.68$, $p = .0003$). However, the relationship between depression symptoms and subsequent frequency (PDA) and intensity (DDD) of alcohol use was no longer significant when concurrent alcohol use was controlled (see Table 5). Two covariates made independent contributions to these models. Among aftercare patients prior PDA predicted later PDA ($p < .0001$). For DDD among aftercare patients, greater prior PDA and fewer DDD predicted fewer DDD (ps >.0001). Similarly for outpatients, prior PDA predicted later PDA ($p < .0001$). Older age was also associated with greater PDA ($p = .009$). Finally and consistent with the aftercare sample, greater prior PDA and fewer DDD predicted fewer DDD (ps > .0001).

Lagged Tests of Mediation

Given that all predicted paths of the lagged models in both study arms were significant when concurrent alcohol use was not in the model, we conducted mediational tests [57]. Results indicated that for aftercare patients, the effect of AA on frequency (PDA; Mack $Z = 1.91$, $p<.01$, % direct effect mediated by depression = 6%) and intensity (DDD; Mack $Z = -1.86$, $p<.01$, % direct effect mediated by depression = 6%) of alcohol use was partially mediated by reductions in depression symptoms. Similarly, among outpatients, the effect of AA attendance on frequency (PDA; Mack $Z = 2.36$, $p<.01$, % direct effect mediated by depression = 7%) and intensity (DDD; Mack $Z = -2.41$, $p<.01$, % direct effect mediated by depression = 9%) of subsequent alcohol use was partially mediated by reductions in depression.

As noted previously, depression symptoms often remit with abstinence or reductions in alcohol use. Thus, we sought to determine whether AA was associated with reductions in depression symptoms over and above the effects of abstinence from alcohol. Because one of the mediational paths was not supported (i.e., AA to depression), when concurrent alcohol use was in the model, we did not conduct these mediational analyses.

Discussion

This study employed controlled, lagged, longitudinal modeling to examine the relationships among AA attendance, depression symptoms, and alcohol use in the year following alcohol treatment with the principal aim of testing reduced depression as a mechanism of AA's salutary effect on drinking. The sample initially exhibited substantially higher depression

compared to the general population, but was similar to other alcohol dependent clinical samples. Lagged models revealed that AA attendance was robustly associated with subsequent reductions in the frequency and intensity of alcohol consumption. Greater AA attendance was also associated with greater reductions in depression symptoms, and greater depression symptoms were associated with more subsequent drinking. However, these effects were rendered non-significant, when concurrent alcohol use was entered into the model. This suggests that AA's beneficial effect on depression and on subsequent alcohol use may be accounted for, in part, by AA's proximal effect on drinking.

Levels of depression within a large alcohol dependent sample

Elevated levels of depression were consistent with other clinical samples and were substantially higher than in general population samples. On average, these levels dropped significantly, but covaried with alcohol use over time. Compared to outpatients, aftercare patients maintained a consistently higher level of depression, although these groups were not different initially. This difference may reflect greater depression psychopathology among the more severe aftercare patients. Alternatively, it may be indicative of more pervasive alcohol-induced mood regulation problems. Although the direction of the relationship between depression and alcohol use could not be definitively determined, it is likely to be reciprocal. However, the pattern of findings suggests that heavy alcohol use may cause or exacerbate depression symptoms rather than depression symptoms exacerbating drinking - a pattern similar to that found in the same sample when examining anger [35]. Consequently, helping individuals with alcohol dependence to reduce or cease drinking may concurrently improve depression symptoms, since some may be due to post-acute withdrawal phenomena. Although women reported significantly higher levels of depression than men, they dropped to, and remained at, a level similar to men over the course of follow-up. Initially higher depression may reflect a greater deleterious impact of drinking on mood among women, which is consistent with the pattern of more serious sequelae ("telescoping") suffered by women at similar or lower levels of use [62-64].

Reductions in depression symptoms as a mechanism of AA's beneficial effect on alcohol use

AA attendance was independently associated with reductions in depression symptoms over time in the outpatient and aftercare samples when concurrent alcohol use was not in the model. In fact, we found that AA's beneficial effect on subsequent alcohol use was partially mediated by reductions in depression symptoms. It is likely that AA mobilizes salutary change through several mechanisms [13], hence, the magnitude of any single mechanism's effect is likely to be modest, as was the case here. However, it was consistent across the outpatient and aftercare arms and across both alcohol use measures. Thus, AA attendance appears to help individuals to increase abstinence and to reduce the intensity of drinking when lapses do occur, partially by reducing symptoms of negative affect. When concurrent alcohol use was controlled, however, the effect of AA attendance on depression was rendered non-significant. This pattern of findings suggests that AA is associated with reduced drinking concurrently and predictively, and also with improvements in depression symptoms.

AA's association with reduced drinking and reduced depression is encouraging, since AA's emphases on "powerlessness", "surrender", and "character defects" could be conceived as perpetuating a pessimistic world view cultivated by subjugation of will. On the contrary, in keeping with AA's emphasis on reducing negative affect and increasing subjective well-being, attending AA appears to relate not only to improved drinking outcomes, but also to improved psychological adjustment. It is possible that AA attendance alleviates depression

through the group therapy principles of instilling hope, universality, group cohesiveness, and catharsis [13].

Noteworthy too, was the interaction between AA and alcohol use. This relative increase in covariance over time may be due to the earlier protective effect of the MATCH treatments that helped offset potentially worse outcomes among non-AA attendees. Alternatively, it could be due to motivational self-selection as those attending longer may have been more strongly motivated to abstain and therefore had better longer-term outcomes.

Limitations

The temporal sequencing of assessments does not lend itself to an ideal test of these mediational relationships, since AA attendance and depression were measured well in advance of the alcohol use outcome measures. A finer time resolution might reveal a different picture and should be explored. It is likely, for example, that the social interaction and support derived from attending an AA group meeting may have an immediate impact on enhancing mood and well-being that may proximally decrease the chance of drinking that day. Such an effect may quickly decay, however, without re-attending (perhaps the next day or later the same week), indicating a short-term "half-life" of meeting attendance. The sample consists mostly of employed, White, males; generalizability to other groups is not known. Also, we examined depression as a mediator of AA attendance and not AA involvement or affiliation. This is because most prior studies that show a relationship between AA and drinking have examined AA attendance, but additional measures of active involvement (e.g., having a sponsor, step work) may reveal different findings. There are many potential "third" variables not specified in these models that also might influence depression symptoms (e.g., other psychiatric and physical co-morbidities, other drug use disorders and other drug use etc.). Consequently, although we have conducted a rigorous set of analyses, caution should nevertheless be taken when drawing conclusions from these correlational results. Further replications with other samples are needed to confirm the robustness of this pattern of findings.

Conclusions

Prior studies have shown a relationship between greater AA participation and improvements in alcohol use and related problems. However, most have not used lagged designs that enhance causal conclusions [29] and many have not controlled for multiple confounds due to smaller samples [21]. Very few have employed HLM or similar methods that test for averaged effects over multiple time points. Using such rigorous methodology and employing a large clinical sample, we found that more frequent AA attendance was robustly associated with less intense and less frequent alcohol use both concurrently and predictively. Although AA attendance was additionally associated with subsequent reductions in depression symptoms, it did not predict such reductions over and above concurrent alcohol use. Consequently, AA appears to lead both to improvements in alcohol use and psychological and emotional well-being, which, in turn, may reinforce further abstinence and recovery-related change.

Acknowledgments

Support for this project was provided by the National Institute on Alcohol Abuse and Alcoholism (NIAAA) through an R21 award (R21AA016762) "Mechanisms and Moderators of Behavior Change In Alcoholics Anonymous". Content is solely the responsibility of the authors, and does not necessarily represent the official views of the NIAAA or the NIH.

References

1. Kessler RC, Crum RM, Warner LA, Nelson CB, Schulenberg J, Anthony JC. Lifetime co-occurrence of DSM-III-R alcohol abuse and dependence with other psychiatric disorders in the National Comorbidity Survey. Archives of General Psychiatry. 1997; 54(4):313–321. [PubMed: 9107147]
2. Regier DA, Narrow WE, Rae DS. The epidemiology of anxiety disorders: The Epidemiologic Catchment Area (ECA) Experience. Journal of Psychiatric Research. 1990; 24(Suppl. 2):3–14. [PubMed: 2280373]
3. Schuckit M, Tipp J, Bucholz K, Nurnberger J, Hesselbrock V, Crowe R, et al. The life-time rates of three major mood disorders and four major anxiety disorders in alcoholics and controls. Addiction. 1997; 92(10):1289–1304. [PubMed: 9489046]
4. Greenfield S, Weiss R, Muenz L, Vagge L, Kelly J, Bello L, et al. The effect of depression on return to drinking: A prospective study. Archives of General Psychiatry. 1998; 55(3):259–265. [PubMed: 9510220]
5. Marlatt, GA.; Gordon, JR., editors. Relapse prevention: Maintenance strategies in the treatment of addictive behaviors. Guilford Press; New York: 1985.
6. Zywiak W, Connors G, Maisto S, Westerberg V. Relapse research and the Reasons for Drinking Questionnaire: A factor analysis of Marlatt's relapse taxonomy. Addiction. 1996; 91(12):s121–s130. [PubMed: 8997786]
7. Fergusson DM, Boden JM, Horwood LJ. Tests of causal links between alcohol abuse or dependence and major depression. Archives of General Psychiatry. 2009; 66(3):260–266. [PubMed: 19255375]
8. Schuckit MA. Comorbidity between substance use disorders and psychiatric conditions. Addiction. 2006; 101:76–88. [PubMed: 16930163]
9. Heinz A, Ragan P, Jones D, Hommer D, Williams W, Knable M, et al. Reduced central serotonin transporters in alcoholism. American Journal of Psychiatry. 1998; 155(11):1544–1549. [PubMed: 9812115]
10. Harper C. The neuropathology of alcohol-related brain damage. Alcohol & Alcoholism. 2009; 44(2):136–140. [PubMed: 19147798]
11. He X, Sullivan E, Stankovic R, Harper C, Pfefferbaum A. Interaction of thiamine deficiency and voluntary alcohol consumption disrupts rat corpus callosum ultrastructure. Neuropsychopharmacology. 2007; 32(10):2207–2216. [PubMed: 17299515]
12. Schuckit MA. Alcohol-use disorders. The Lancet. 2009; 373:492–501.
13. Kelly JF, Magill M, Stout RL. How do people recover from alcohol dependence? A systematic review of the research on mechanisms of behavior change in Alcoholics Anonymous. Addiction Research & Theory. 2009; 17(3):236–259.
14. Brown S, Inaba R, Gillin J, Schuckit M. Alcoholism and affective disorder: Clinical course of depressive symptoms. American Journal of Psychiatry. 1995; 152(1):45–52. [PubMed: 7802119]
15. Witkiewitz K, Marlatt G. Relapse prevention for alcohol and drug problems: That was Zen, this is Tao. American Psychologist. 2004; 59(4):224–235. [PubMed: 15149263]
16. Humphreys, K. Circles of recovery: Self-help organizations for addictions. Cambridge University Press; Cambridge, UK: 2004.
17. Kelly, JF.; Yeterian, JD. Mutual-help groups. In: O'Donohue, W.; Cunningham, JR., editors. Evidence-Based Adjunctive Treatments. Elsevier; New York: 2008. p. 61-105.
18. Alcoholics Anonymous. Twelve Steps and Twelve Traditions. Alcoholics Anonymous World Services; New York: 1953.
19. Alcoholics Anonymous. Alcoholics Anonymous: The Story of How Thousands of Men and Women Have Recovered from Alcoholism. 4th ed.. Alcoholics Anonymous World Services; New York: 2001.
20. Emrick, CD.; Tonigan, JS.; Montgomery, H.; Little, L. Alcoholics Anonymous: What is currently known?. In: McCrady, BS.; Miller, WR., editors. Research on alcoholics anonymous: Opportunities and alternatives. Rutgers Center of Alcohol Studies; Piscataway, NJ: 1993. p. 41-76.
21. Kelly JF, Yeterian JD. The role of mutual-help groups in extending the framework of treatment. Alcohol Research & Health. in press.

22. Tonigan JS, Toscova R, Miller WR. Meta-analysis of the literature on Alcoholics Anonymous: Sample and study characteristics moderate findings. Journal of Studies on Alcohol. 1996; 57(1): 65–72. [PubMed: 8747503]
23. Yalom, ID.; Leszcz, M. The Theory and Practice of Group Psychotherapy. 5th ed.. Basic Books; New York: 2005.
24. Howard K, Lueger R, Maling M, Martinovich Z. A phase model of psychotherapy outcome: Causal mediation of change. Journal of Consulting and Clinical Psychology. 1993; 61(4):678–685. [PubMed: 8370864]
25. Moos RH. Active ingredients of substance use-focused self-help groups. Addiction. 2008; 103(3): 387–396. [PubMed: 18269361]
26. Ferri M, Amato L, Davoli M. Alcoholics Anonymous and other 12-step programmes for alcohol dependence. Cochrane Database of Systematic Reviews 2006. 2006; (3) Art. No.: CD005032. DOI: 10.1002/14651858.CD005032.pub2.
27. Moos RH, Moos BS. Participation in treatment and Alcoholics Anonymous: A 16-year follow-up of initially untreated individuals. Journal of Clinical Psychology. 2006; 62(6):735–750. [PubMed: 16538654]
28. Timko C, Moos RH, Finney JW, Lesar MD. Long-term outcomes of alcohol use disorders: Comparing untreated individuals with those in Alcoholics Anonymous and formal treatment. Journal of Studies on Alcohol. 2000; 6(4):529–540. [PubMed: 10928723]
29. Nock M. Conceptual and design essentials for evaluating mechanisms of change. Alcoholism: Clinical and Experimental Research. 2007; 31(3):4s–12s.
30. Kaskutas L, Ammon L, Delucchi K, Room R, Bond J, Weisner C. Alcoholics Anonymous careers: Patterns of AA involvement five years after treatment entry. Alcoholism: Clinical and Experimental Research. 2005; 29(11):1983–1990.
31. Kelly JF, Stout R, Zywiak W, Schneider R. A 3-year study of addiction mutual-help group participation following intensive outpatient treatment. Alcoholism: Clinical and Experimental Research. 2006; 30(8):1381–1392.
32. Laffaye C, McKellar J, Ilgen M, Moos R. Predictors of 4-year outcome of community residential treatment for patients with substance use disorders. Addiction. April; 2008 103(4):671–680. [PubMed: 18339113]
33. McCrady B, Epstein E, Kahler C. Alcoholics Anonymous and relapse prevention as maintenance strategies after conjoint behavioral alcohol treatment for men: 18-month outcomes. Journal of Consulting and Clinical Psychology. 2004; 72(5):870–878. [PubMed: 15482044]
34. Alcoholics Anonymous. Alcoholics Anonymous: The story of how thousands of men and women have recovered from alcoholism. 3rd ed.. Alcoholics Anonymous World Services; New York: 1939.
35. Kelly JF, Stout RL, Tonigan JS, Magill M, Pagano M. Negative Affect, Relapse, and Alcoholics Anonymous: Does AA work by reducing Anger? Journal of Studies on Alcohol and Drugs. in press.
36. Project MATCH Research Group. Project MATCH (Matching Alcoholism Treatment to Client Heterogeneity): Rationale and methods for a multisite clinical trial matching patients to alcoholism treatment. Alcoholism: Clinical Experimental Research. 1993; 17(6):1130–1145.
37. Brown SA, Vik PW, Patterson TL, Grant I, Schuckit MA. Stress, vulnerability, and adult alcohol relapse. Journal of Studies on Alcohol. 1995; 56:538–545. [PubMed: 7475034]
38. Brown S, Schuckit M. Changes in depression among abstinent alcoholics. Journal of Studies on Alcohol. 1988; 49(5):412–417. [PubMed: 3216643]
39. Kazdin AE, Nock MK. Delineating mechanisms of change in child and adolescent therapy: Methodological issues and research recommendations. Journal of Child Psychology and Psychiatry. 2003; 44(8):1116–1129. [PubMed: 14626454]
40. Timko C, Finney J, Moos R, Moos B. The process of treatment selection among previously untreated help-seeking problem drinkers. Journal of Substance Abuse. 1993; 5(3):203–220. [PubMed: 8312728]
41. Spitzer, RL.; Williams, JBW. Manual for the Structured Clinical Interview for DSM-III. New York State Psychiatric Institute, Biometrics Research Department; New York: 1985.

42. Kadden, R.; Carroll, K.; Donovan, D.; Cooney, N.; Monti, P.; Abrams, D., et al. NIAAA Project MATCH Monograph Series. National Institute on Alcohol Abuse and Alcoholism; Rockville, MD: 1992. Cognitive-Behavioral Coping Skills Therapy manual: A clinical research guide for therapists treating individuals with alcohol abuse and dependence.
43. Miller, WR.; Zweben, A.; DiClemente, CC.; Rychtarik, RG. NIAAA Project MATCH Monograph Series. National Institute on Alcohol Abuse and Alcoholism; Rockville, MD: 1992. Motivational Enhancement Therapy manual: A clinical research guide for therapists treating individuals with alcohol abuse and dependence.
44. Nowinski, J.; Baker, S.; Carroll, KM. NIAAA Project MATCH Monograph Series. National Institute on Alcohol Abuse and Alcoholism; Rockville, MD: 1992. Twelve-Step Facilitation Therapy manual: A clinical research guide for therapists treating individuals with alcohol abuse and dependence.
45. Project MATCH Research Group. Matching alcoholism treatments to client heterogeneity: Project MATCH posttreatment drinking outcomes. Journal of Studies on Alcohol. 1997; 58(1):7–29. [PubMed: 8979210]
46. Miller, WR. NIAAA Project MATCH Monograph Series. Vol. 5. National Institute of Alcohol Abuse and Alcoholism; Rockville, MD: 1996. Form 90: A structured assessment interview for drinking and related behaviors. NIH Publication No. 96-4004
47. Miller WR, Del Boca FK. Measurement of drinking behavior using the Form 90 family of instruments. Journal of Studies on Alcoholism. 1994; (suppl. 12):112–118.
48. Sobell, LC.; Sobell, MB. Timeline follow-back: A technique for assessing self-reported alcohol consumption. In: Litten, RZ.; Allen, JP., editors. Measuring alcohol consumption: Psychosocial and biochemical methods. Humana Press; Totowa, NJ: 1992. p. 41-72.
49. Miller, WR.; Marlatt, GA. Manual for the Comprehensive Drinker Profile. Psychological Assessment Resources; Odessa, FL: 1984.
50. Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. Archives of General Psychiatry. 1961; 4:561–571. [PubMed: 13688369]
51. Beck A, Steer R, Garbin M. Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. Clinical Psychology Review. 1988; 8(1):77–100.
52. Tabachnick, BG.; Fidell, LS. Using Multivariate Statistics. 3rd Ed.. Harper Collins; New York: 1996.
53. Little, RJA.; Rubin, DB. Statistical Analysis with Missing Data. 2nd Ed.. John Wiley; New York: 2002.
54. Gilks, WR.; Richardson, S.; Spiegelhalter, D. Markov Chain Monte Carlo in Practice: Interdisciplinary Statistics. Chapman & Hall; London: 1996.
55. Barnard J, Rubin DB. Small-sample degrees of freedom with multiple imputation. Biometrika. 1999; 86:948–955.
56. MacKinnon DP, Dwyer JH. Estimating mediated effects in prevention studies. Evaluation Review. 1993; 17:144–158.
57. MacKinnon DP, Lockwood CM, Hoffman JM. A comparison of methods to test mediation and other intervening variable effects. Psychological Methods. 2002; 7(1):83–104. [PubMed: 11928892]
58. Tonigan, JS.; Connors, GJ.; Miller, WR. Participation and involvement in Alcoholics Anonymous. In: Babor, TF.; DelBoca, FK., editors. Treatment Matching in Alcoholism. Cambridge University Press; New York: 2003. p. 184-204.
59. Knight R. Some general population norms for the short form Beck Depression Inventory. Journal of Clinical Psychology. 1984; 40(3):751–753. [PubMed: 6746984]
60. Michalak EE, Murray G, Wilkinson C, Dowrick C, Lasa L, Lehtinen V, et al. Estimating depression prevalence from the Beck Depression Inventory: Is season of administration a moderator? Psychiatry Research. 2004; 129:99–106. [PubMed: 15572189]
61. Choquette K. Assessing depression in alcoholics with the BDI, SCL-90R, and DIS criteria. Journal of Substance Abuse. 1994; 6(3):295–304. [PubMed: 7703706]
62. Greenfield S. Women and alcohol use disorders. Harvard Review of Psychiatry. 2002; 10(2):76–85. [PubMed: 11897748]

63. Hernandez-Avila C, Rounsaville B, Kranzler H. Opioid-, cannabis- and alcohol-dependent women show more rapid progression to substance abuse treatment. Drug and Alcohol Dependence. 2004; 74(3):265–272. [PubMed: 15194204]

64. Schuckit M, Daeppen J, Tipp J, Hesselbrock M, Bucholz K. The clinical course of alcohol-related problems in alcohol dependent and nonalcohol dependent drinking women and men. Journal of Studies on Alcohol. 1998; 59(5):581–590. [PubMed: 9718111]

Figure 1.
Lagged Model Conceptualization[a]
a The HLM model represents averaged, lagged, effects across the two time points noted. (Conceptually, this is akin to running two separate lagged models simultaneously at the two different time points and averaging their effects).
b Models were run with, and without, this variable specified in the model
* Past week

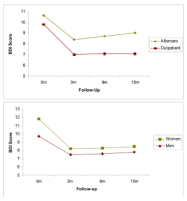

Figure 2.
Change in BDI Depression Symptoms Scores by Over Time by Gender and Study Arm

Table 1

Demographic characteristics by study arm

	Aftercare ***n*** **= 764**	**Outpatient** ***n*** **= 942**
Age	41.9(11.1)	38.8(10.7)
Years Education	13.1(2.0)	13.4(2.1)
Gender		
Female	20.3%	27.3%
Ethnicity		
White	80.5%	79.8%
Hispanic	14.8%	5.6%
Black	3.5%	12.3%
Other	1.2%	2.2%
Married/Non-Married		
Married/Cohabiting	33.8%	35.7%
Employment		
Full-time	47.5%	51.06%

Table 2

AA Attendance, Depression, and Alcohol use Outcomes by study Arm

	Aftercare *n* = 764				Outpatient *n* = 942			
	0m	**3m**	**9m**	**15m**	**0m**	**3m**	**9m**	**15m**
Any AA	81.3%	84.9%	56.5%	53.6%	38.8%	46.2%	29.4%	30.2%
AA M (SD)	23.2(42.2)	32.4(32.3)	19.0(27.3)	17.4(26.1)	9.8(33.6)	11.0(20.3)	7.8(18.9)	7.7(18.5)
Depression M (SD)	10.6(8.6)	8.4(8.5)	8.7(8.6)	8.9(9.1)	9.8(7.9)	7.0(7.3)	7.1(7.5)	7.1(7.6)
PDA M (SD)	26.7(29.6)	90.0(21.3)	80.9(29.9)	79.5(31.9)	34.2(29.9)	79.9(27.3)	73.9(31.2)	72.9(33.3)
DDD M (SD)	20.3(11.9)	6.0(9.7)	6.9(9.9)	6.3(9.4)	13.5(8.0)	7.5(8.0)	6.3(6.6)	5.8(6.2)

Table 3

HLM Results for AA attendance predicting transformed PDA and DDD for the Outpatient and Aftercare samples [a]

Variable	*b* (*se*)	F (*df*)	*p*
Outpatient Sample	.0017(.0003)	37.09(494)	<.0001
baseline PDA	.0025(.0006)	17.39(486)	<.0001
Lagged AA attendance	−.0013(.0008)	2.22(469)	.1359
Lagged AA attendance X time			
Aftercare Sample	.0014(.0003)	17.39(405)	<.0001
baseline PDA	.0011(.0004)	6.35(491)	.0121
Lagged AA attendance	.0013(.0006)	3.72(430)	.0541
Lagged AA attendance X time			
Outpatient Sample	.0089(.0039)	5.15(411)	.0240
baseline DDD	−.0076(.0021)	12.89(469)	.0004
Lagged AA attendance	.0007(.0029)	0.06(366)	.8134
Lagged AA attendance X time			
Aftercare Sample	.0140(.0034)	17.22(467)	<.0001
baseline DDD	−.0047(.0017)	7.84(491)	.0053
Lagged AA attendance	−.0068(.0025)	7.40(454)	.0068
Lagged AA attendance X time			

[a] Control and other variables included in models but not shown above include age, gender, ethnicity, marital status, employment status, number of prior alcohol treatments, treatment assignment, treatment site, lagged PDA and lagged DDD.

Table 4

HLM Results for AA attendance predicting depression for the Outpatient and Aftercare samples [a]

Variable	*b* (*se*)	F (*df*)	*p*
Outpatient Sample	.4683(.0382)	150.06(113)	<.0001
baseline Depression	−.0056(.0109)	0.26(390)	.6083
Lagged AA attendance	−.0002(.0010)	0.04(235)	.8417
Lagged AA attendance X Depression	−.0107(.0152)	0.49(387)	.4838
Lagged AA attendance X time			
Aftercare Sample	.4300(.0402)	114.28(353)	<.0001
baseline Depression	.0031(.0110)	0.08(197)	.7763
Lagged AA attendance	−.0010(.0007)	11.42(358)	.1690
Lagged AA attendance X Depression	.0156(.0133)	1.90(340)	.2441
Lagged AA attendance X time			

[a] Control and other variables included in these tested models but not shown above include age, gender, ethnicity, marital status, employment status, number of prior alcohol treatments, treatment assignment, treatment site, gender X time, concurrent PDA and concurrent DDD.

Table 5

HLM Results for depression predicting PDA and DDD for the Outpatient and Aftercare samples [a]

Variable	*b* (*se*)	F (*df*)	*p*
Outpatient Sample	.0017(.0003)	32.60(490)	<.0001
baseline PDA	−.0020(.0017)	1.44(217)	.2313
Lagged Depression	−.0019(.0022)	0.71(357)	.3997
Lagged Depression X time			
Aftercare Sample	.0014(.0003)	16.97(409)	<.0001
baseline PDA	−.0013(.0017)	0.59(314)	.4417
Lagged Depression	.0050(.0024)	4.49(245)	.0346
Lagged Depression X time			
Outpatient Sample	.0066(.0039)	2.79(441)	.0957
baseline DDD	.0097(.0057)	2.86(277)	.0929
Lagged Depression	.0026(.0079)	0.11(235)	.7421
Lagged Depression X time			
Aftercare Sample	.0132(.0034)	14.75(466)	.0001
baseline DDD	.0043(.0066)	0.436(390)	.5114
Lagged Depression	.0010(.0088)	1.82(428)	.9050
Lagged Depression X time			

[a] Control and other variables included in these tested models but not shown above include age, gender, ethnicity, marital status, employment status, number of prior alcohol treatments, treatment assignment, treatment site, gender x time, concurrent PDA and concurrent DDD.