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Changes in depression mediate the effects of AA attendance on alcohol use outcomes

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Abstract

Background—Depression may contribute to increased drinking in individuals with alcohol use disorder. Although Alcoholics Anonymous (AA) attendance predicts drinking reductions, there is conflicting information regarding the intermediary role played by reductions in depression.

Objectives—We explored whether AA attendance reduces depressive symptoms, the degree to which improvement in depression results in reductions in drinking, and in which subgroups these effects occur.

Methods—253 early AA affiliates (63% male) were recruited and assessed at baseline 3, 6, 9, 12, 18, and 24 months. Depression was measured using the Beck Depression Inventory (BDI) and was administered at baseline 3, 6, 12, 18, and 24 months. AA attendance and alcohol use outcomes were obtained with the Form 90. Mediation analyses were performed at early (3, 6, and 9 months) and late (12, 18, and 24 months) follow-up to investigate the degree to which reductions in depression mediated the effect of AA attendance on drinking, controlling for concurrent drinking. In addition, a series of moderated mediation analyses were performed using baseline depression severity as a moderator.

Results—At early follow-up, reductions in depression (6 months) mediated the effects of AA attendance (3 months) on later drinking (drinks per drinking day) (9 months) ($b = -0.02$, boot CI $[-0.055, -0.0004]$), controlling for drinking at 6 months. Baseline depression severity did not moderate the degree to which BDI mediated the effects of AA attendance on alcohol use ($ps > .05$).

Conclusion—These findings provide further evidence that depression reduction is a mechanism by which AA attendance leads to reductions in alcohol use. Improving depression may help reduce alcohol use in individuals with AUD, and AA attendance may be an effective way to achieve that goal.

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Disclosure statement

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Keywords

12-step; alcoholics anonymous; depression; alcohol use disorders; negative affect

Background

Relatively consistent evidence has accumulated, indicating that community-based Alcoholics Anonymous (AA) attendance is predictive of decreased alcohol use for many, but not all, problem drinkers (1–5). Importantly, self-selection bias does not appear to account for AA-related benefit (6–8), and the salutary effects of AA have been observed in diverse populations including urban Native Americans (9), dually diagnosed adults (10,11), and ethnic minorities (12,13). Factors that do appear to account for AA-related benefit during early AA affiliation include acquiring an AA sponsor (14–16) social support for abstinence (17,18), gains in spiritual practices (19,20), and increased abstinence self-efficacy (21–24).

Central in the core AA literature (25) is the proposition that negative affect is a leading precipitant to relapse and much of the prescribed step work in 12-step programs is therefore directed at reducing the unbridled expression of anger, depression, and selfishness. The degree to which AA attendance influences AA member depression and, in turn, how changes in depression among AA members actually explains the beneficial effect of AA on drinking, remains unclear, however. Specifically, three rigorous longitudinal studies have investigated changes in depression among AA members (26–28) (hereafter referred to as Wilcox et al. (26), Worley et al. (27) and Kelly et al. (28), respectively). Found in each of these studies, AA exposed adults reported significant reductions in depressive symptoms over time, with such pre-post reductions observed at 6-month follow-up among veterans with substance use and major depressive disorder [Worley et al. (27); Hedges g (g , Cohen's d adjusted for small sample bias) = 0.55], at 9 months in both outpatient and aftercare treatment seeking Project MATCH samples (Kelly et al. (28); g = 0.29), and over the course of 2 years in a community-recruited sample (Wilcox et al. (26); g = 0.18). Lagged analyses in each aforementioned study showed that frequency of AA attendance was associated with later reductions in depression.

Findings diverge, however, when changes in depressive symptoms were examined after statistically controlling for concurrent drinking. In the Kelly et al. (28) study, for example, the inclusion of current drinking in the lagged models eliminated the direct and independent effect of AA attendance on levels of depression symptoms, so reductions in depressive symptoms were attributed to the direct effect of AA attendance on reducing drinking. Furthermore, Kelly et al. (28) determined that changes in depressive symptoms did not mediate that association between frequency of AA attendance and later drinking in either the aftercare or outpatient MATCH samples. A different relationship between AA attendance and changes in depression was presented in Worley et al. (27) and Wilcox et al. (26). Specifically, these studies found that the independent effect of AA on later levels of depression symptoms remained even after controlling for concurrent alcohol and substance use, suggesting that reductions in levels of depression symptoms may be attributable to the influence of AA beyond that explained by reductions in alcohol or substance use. Also

noteworthy, Worley et al. (27) reported that reductions in depression significantly mediated 15% of the direct effect of AA on drinking. Wilcox et al. (26) did not examine whether reductions in depression mediated the effect of AA on drinking.

There are some possible explanations for the conflicting results between these three studies. Foremost, at baseline, the absolute severity of depressive symptoms was significantly different across the three studies, with the MATCH aftercare and outpatient samples for Kelly et al. (28) reporting significantly lower levels of depression. Combining the MATCH aftercare and outpatient samples, for example, yielded a mean Beck Depression Inventory-II (BDI) (29) score at intake of 10.16 (SD 8.25). The mean score for the community-based AA sample for Wilcox et al. (26) was 19.79 (SD 11.35), almost twice as depressed as the Kelly et al. (28) sample, and approached the BDI cutoff score of 20 for moderate depression. The most depressed sample studied was that of Worley et al. (27) which had a mean Hamilton Depression Scale score of 28.55 (SD 10.82), well above the cutoff of 17 for moderate depression (30), and all subjects had a diagnosis of major depressive disorder. We therefore suspected that adults with major depressive disorders were disproportionately represented in Worley et al. (27) and Wilcox et al. (26), and that, to some extent, the significantly lower levels of depression symptoms found in the MATCH sample from Kelly et al. (28) could have resulted in lower effects of AA on depression. Other related work, using multiple mediator analyses, has also found a stronger relationship between AA, depression, and later drinking (mediation) in individuals with more severe depression (19).

Objectives

The objective of this study was to shed light on the reasons for conflicting findings in studies of the relationship between AA exposure, changes in depression, and drinking outcomes by performing additional analyses on the dataset used in the Wilcox et al. (26) study, with a particular focus on whether or not changes in depression mediated the reductions in drinking observed AA attendance. To do so, we investigated whether changes in levels of depression could explain the effects of AA exposure on later drinking in our sample, using similar approaches to those utilized by Kelly et al. (28) and Worley et al. (27). Furthermore, we investigated whether or not the presence or absence of a clinically relevant depression score at study entry moderated the strength of these associations. We hypothesized that since our sample was more depressed than that of Kelly et al. (28), and therefore more similar to that of Worley et al. (27), changes in depression would mediate the effects of AA on drinking (Aim 1), similar to the results in Worley et al. (27), but unlike those in Kelly et al. (28). Furthermore, if differences in baseline depression levels between the Worley et al. (27) and Kelly et al. (28) studies were driving the differences in findings, we hypothesized that baseline depression would moderate the mediational effect, and that mediation would be more pronounced (larger indirect effect) in those with greater baseline depression (Aim 2).

Methods

Participants and procedure

This is a follow-up report to a previously published paper (26), and details about the community-based AA sample can be obtained from the original paper. In brief, participants

were early AA affiliates with little previous exposure to 12-step programs (participants were excluded for more than 16 weeks of lifetime AA exposure and if they reported ever having had a period of alcohol abstinence of at least 12 months at any time in their life after their alcohol use had become a problem). Two hundred and fifty-three adults with alcohol use problems were recruited from AA groups; from outpatient substance abuse treatment facilities; and from community sources including homeless shelters, advertisement in local newspapers, and flyers (68 were recruited from community-based AA, 87 were recruited from outpatient treatment abuse treatment centers, and 98 at shelters or through advertisements and flyers). Participants were also required to have attended one or more AA meetings in the prior 3 months, to have consumed alcohol in the prior 90 days, and to meet Diagnostic and Statistical Manual of Mental Disorders (31) criteria for alcohol dependence or abuse (32). All procedures were approved by the institutional review board at the University of New Mexico (UNM Protocol No. 24028).

Breathalyzers were used to ensure that participants' blood alcohol concentration did not exceed 0.05 prior to the consent process, or at any subsequent interview. Once consented, participants were administered a baseline interview that included 15 self-report questionnaires, 3 semi-structured interviews, and a urine toxicology screen. Follow-up interviews were conducted in 3-month increments for one year and then at 18 and 24 months. More than 85% of the original sample provided follow-up data at 24 months. No intervention was offered in this assessment-only study, although clinical referrals were made upon participant request, or when deemed warranted by clinical staff.

Measures

Alcohol use—Alcohol use data were obtained using the Form 90 (33), which is a calendar-based semi-structured interview. Two alcohol use measures from the Form 90 were computed. Proportion of days abstinent from alcohol (PDA) was defined as the number of alcohol-abstinent days in an assessment period divided by the total number of days in the period. Drinks per drinking day (DPDD) was defined as number of drinks consumed per drinking (i.e., non-abstinent) days in the assessment period.

12-step meeting attendance—A single item from the Form 90 interview documented frequency of 12-step meeting attendance during an interview period. The proportion days of 12-step attendance for each participant was calculated as a ratio of days 12-step meeting attendance divided by the number of days in an interview period.

Depression—The Beck Depression Inventory (BDI) (29), commonly used in both clinical and research settings to screen for and establish severity of depression, was used as a measure of depressive symptomatology. The BDI is composed of 21 questions each scored on a scale of 0 to 3 asking about symptoms over the past 2 weeks such as hopelessness, irritability, guilt, feelings of being punished, fatigue, weight loss, and lack of interest in sex. Total scores on the BDI of 0–13 indicate minimal depression, 14–19 mild depression, 20–28 moderate depression, and 29–63 severe depression. For mediation analyses, we used the BDI as a continuous variable to mark the degree of depressive symptomatology. When used as a moderator, we used a dichotomous moderator variable by grouping individuals with minimal

or mild depression into one group, and those with moderate or severe depression into the other group as a marker of the presence or absence of clinically relevant depression.

Statistical analyses

To examine whether the effect of AA attendance on drinking (DPDD, PDA) could be partially or fully accounted for by changes in BDI scores, we conducted mediation tests using the bias-corrected bootstrap (34) as implemented in the PROCESS macro for SPSS (35), which uses ordinary least squares regression. In all models, we bootstrapped 1000 samples and significant effects were determined by 95% bias-corrected confidence intervals that do not contain zero. We conducted two single-mediation models (Aim 1: Analysis 1 for PDA, Analysis 2 for DPDD) in which we examined the total, direct, and indirect effects of AA attendance on alcohol use via BDI scores at early follow-up (3-month AA; 6-month BDI; 9-month alcohol use; $n = 194$). For these two models, drinking and BDI at study entry (baseline) were entered as covariates, and concurrent drinking (6 months) was entered as a mediator operating in parallel [Figure 1; Model 4 in PROCESS Macro (36)]. We then conducted two tests of moderated mediation (Aim 2: Analysis 3 for PDA, Analysis 4 for DPDD) by entering baseline categorical depression as a moderator into the two single-mediation models defined above [Figure 2; Model 58 in PROCESS Macro (36)]. Although we did not formally test for heteroskedasticity, we used the heteroskedasticity-consistent standard errors option in the process macro (37). For our post hoc late follow-up analyses, we used exactly the same methods described above, except that we used 12 month AA, 18 month BDI and 24 month alcohol use ($n = 183$) instead of the 3-, 6-, and 9-month time points described above (Figures 1 and 2; Analyses 5, 6, 7, 8). The outcome variables were transformed prior to the analyses using a transformation that eliminated extreme outliers (DPDD was square root transformed; PDA was arc sin of square root transformed), although this may not have been necessary, as bootstrapping (as opposed to the Sobel test for indirect effects) significantly minimizes the need for normally distributed outcome variables (38). We note for the reader that p values are appropriate for all of the reported parameters except the indirect effects. In this case, we are interested in whether the CI contains 0 or not.

Results

Participant information

Of the 253 participants, 194 participants had complete data for the planned analyses at early follow-up. Of these 194 participants, 63% of the sample was male ($n=123$) and the mean age of participants was 39.14 years (SD 9.84). A majority of the reduced sample had a high school degree (22.2% no degree, 12.4% GED, 38.1% high school diploma), were unemployed (63.9%), and were single or divorced (48.1%, 34.5%); 16.0% of the participants reported being homeless. Of the 194 participants, 42.9% were Hispanic, 32.1% were non-Hispanic White, 18.7% were Native American, and the remaining participants were of African, Asian, or unspecified ancestry. Finally, at study entry, participants reported that they were attending AA meetings 16.0% of the days (SD 18.5%) and had a mean BDI score of 20.1 (SD 11.3), a mean proportion of days abstinent of alcohol of 52.7 (SD 30.5) and a mean drinks per drinking day of 17.60 (12.81).

Simple correlations between primary measures [alcohol use (baseline), BDI (baseline), AA (3 month), BDI (6 month), alcohol use (9 month)] were in the expected directions such that greater AA attendance was associated with lower BDI scores and lower alcohol consumption, and lower BDI scores were associated with lower alcohol consumption (Table 1). Of note, the full sample ($n = 253$) and the complete data sample for the early follow-up analyses ($n = 194$) had very similar mean values for the key variables in our analyses (Supplementary Table 1).

Mediation analyses—Aim 1/early follow-up (Analysis 1, 2)—There was a significant direct effect of AA (3 months) on drinking (6 months) for both PDA ($p = 0.000$, $b = 0.441$, $t = 5.88$) and DPDD ($p = 0.009$, $b = -1.482$, $t = -3.388$). The direct effect of AA (3 months) on drinking (9 months) was not significant, although the total effect was highly significant for both PDA ($p = 0.006$, $b = 0.102$, $t = 2.757$) and DPDD ($p = 0.008$, $b = -1.177$, $t = -2.694$). Furthermore, in support of previous findings (26), there were significant effects of AA (3 months) on BDI (6 months) for PDA ($p = 0.040$, $b = -5.043$, $t = -2.073$) and DPDD ($p = 0.050$, $b = -4.723$, $t = -1.973$) (path A; Figure 1). For DPDD, but not PDA, BDI (6 month) was associated with DPDD (9 months) ($p = 0.041$, $b = 0.023$, $t = 2.060$) (path B; Figure 1), and the indirect effect of AA (3 months) on alcohol use at 9 months via BDI (6 months) was also significant. Not surprisingly, alcohol use at 6 months (PDA and DPDD) was a significant mediator of the effects of AA (3 months) on alcohol use (9 months) (Table 2).

Moderated mediation—Aim 2/early follow-up (Analysis 3, 4)—We then entered a BDI categorical moderator variable (as a marker of the likely presence or absence of a depressive disorder diagnosis) into the mediation models (Figure 2). When we did so, none of the indirect effects via BDI for either category of depression and neither of the indices of moderated mediation were statistically significant for the two models tested, indicating that baseline depression severity did not moderate the degree to which BDI (6 months) mediated the relationship between AA attendance (3 months) and alcohol use (9 months). The interaction terms were not significant for either model for either path A or path B, indicating that the baseline BDI categorical variable did not moderate either the relationship between AA (3 months) and alcohol use 6 months or BDI (6 months) or the relationship between BDI (6 months) and alcohol use (9 months), respectively (Table 4).

Post hoc mediation Analysis—late follow-up (Analysis 5, 6)—In the aforementioned analyses looking at the 3-, 6-, and 9-month follow-up time points, we supported our findings from previous work in Wilcox et al. (26), showing that AA attendance was associated with reductions in later depression, controlling for concurrent drinking (26). Since our previous analysis included all time points (baseline to 24 months) and did not look at early and late time points separately, we wanted to explore whether or not changes in depression mediated the effect of AA on alcohol use.

As seen at early follow-up, at late follow-up, AA (12 month) was significantly associated with alcohol use (18 months) for PDA ($p = 0.009$, $b = 0.397$, $t = 2.627$) and at a trend level for DPDD ($p = 0.064$, $b = -1.478$, $t = -1.864$). In addition, AA (12 months) was significantly associated with BDI (18 months) for PDA ($p = 0.047$, $b = -7.947$, $t = -2.004$)

and at a trend level for DPDD ($p = 0.053$, $b = 7.801$, $t = -1.945$) (path A; Figure 1), supporting previous findings from Wilcox et al. (26). However, BDI (18 months) was not significantly associated with alcohol use (24 months) (path B; Figure 1) for either DPDD or PDA, and the indirect effects via BDI were not significant, indicating that at late follow-up, BDI (18 months) did not mediate the effects of AA (12 month) on alcohol use (24 months) (Table 3).

Post hoc moderated mediation analysis—late follow-up (Analysis 7, 8)—When the baseline BDI categorical variable was entered into the model, none of the indirect effects via BDI for either category of depression and neither of the indices of moderated mediation were statistically significant for the two models tested, indicating that baseline depression severity as a categorical variable did not moderate the degree to which BDI (18 months) mediated the relationship between AA (12 months) and alcohol use (24 months). For path A for the PDA analysis, both the BDI moderator variables and the interaction terms in the moderated mediation analysis were significant for AA (12 months) to both BDI (18 months) and PDA (18 months) (Table 4, Figure 2). For path A for the DPDD analysis, the BDI moderator variable and the interaction term for AA (12 months) to BDI (18 months) were significant (Table 4, Figure 2). Plotting slopes by group indicated that those with greater depression levels at baseline were more likely to show a decrease in BDI scores (18 months) with higher levels of AA attendance (12 months).

Discussion

This study investigated the relationship between AA attendance, depression, and alcohol consumption in individuals new to AA. In particular, our primary aim was to see whether the beneficial effects of AA attendance on drinking were mediated by effects on depression. Previous work has shown conflicting results using similar approaches at similar time points (26–28), and our findings help clarify things, somewhat. First, and most interestingly, we found that changes in depression mediated the beneficial effects of AA attendance at 3 months on drinking (DPDD) at 9 months controlling for concurrent drinking, consistent with Worley et al. (27), but in contrast to Kelly et al. (28). Our results were in support of our hypothesis, given that our sample was more similar to that of Worley et al. (27) than that of Kelly et al. (28) in terms of baseline depression levels. By contrast, in our sample, for PDA, the indirect effects were not significant, although they were still in the expected directions; changes in depression appeared to have a greater effect on the quantity consumed, rather than the decision to drink or not drink on a particular day. Other related work has also found that drinking mediated the relationship between AA and later drinking (19), and that the effect was stronger for DPDD.

We also hypothesized that baseline depression severity would moderate the indirect effect, but this hypothesis was not supported. Had it been supported, this would have indicated that differences in depression levels were driving the differences in results between the three studies. The absence of an observed effect could have resulted from the fact that BDI scores are fluid in this population and that a single time point measurement of BDI (our dichotomous moderator variable) is not a reliable marker of the presence or absence of a depressive syndrome. An alternative explanation for the differences lies in the variability in

rates of AA attendance. Namely, our sample and that of Worley et al. (27) had higher AA attendance frequency than that of Kelly et al. (28); in our sample, rates were 29%, 19%, and 15% of days attending AA meetings during the preceding assessment periods at 3, 6, and 9 months, respectively (26), whereas in Kelly et al. (28) participants had minimal AA attendance. Moreover, a clear majority of the participants in Wilcox et al. (26) attended AA throughout the 24 months and, in the Worley et al. (27) study, participants reported, on average, attending more than 10 AA meetings prior to the 3-month follow-up. By contrast, in Kelly et al. (28) study, a majority of the outpatient participants did not attend AA during any follow-up and, by the 9-month follow-up, the percent of outpatient MATCH participants reporting any AA attendance had declined to 29%. The absence of mediation in the Kelly et al. (28) study may have occurred because participants had significantly less overall exposure to AA.

Although we used different methods than we had used in our previous paper (26), we still observed that AA attendance was associated with reductions in later depressive symptoms, controlling for concurrent drinking (path A; Figures 1 and 2). Moreover, we obtained further detail about the time points at which these effects were stronger (early follow-up) and in which populations (greater baseline depression levels for late follow-up). One possible reason that baseline depression severity did not moderate the relationship between AA attendance and later depression at early follow-up is that there was more room for improvement in all individuals, and due to the powerful effect of novelty, otherwise known as the “pink cloud” by AA attendees early in their AA exposure. At late follow-up, there may have been a stronger effect for those with greater depression at baseline, perhaps because they were the individuals for whom depression was not tightly linked with alcohol consumption, and who took longer to improve in their depressive symptom levels. Finally, that those with moderate or severe depression scores at baseline benefited even more at late follow-up than those with minimal or mild depression also supports findings in the literature that individuals with co-occurring psychiatric issues are just as likely to benefit from AA (11,39), and that drinking reduction improves chances of recovery from psychiatric illness.

Negative affect has long been considered a trigger for drinking and is considered a reasonable target for AUD treatment both within the AA literature (25) and treatment literature at large (40). This is supported by studies showing that compulsive alcohol use is driven by negative reinforcement (41), that people with AUD report drinking to relieve negative affect (42–44), that depression precedes relapse and is associated with greater drinking in individuals with AUD seeking treatment (45–49), and that treatments targeting negative affect and emotion regulation result in improvements in drinking (42,50). On the other hand, previous work has also shown that antidepressants in individuals with AUD but without depression are not effective at reducing drinking (51), and has shown a minimal role of depression in predicting later drinking (52). Our results supported the possibility that changes in negative affect (depression) do indeed mediate the effects of AA on later drinking, that this effect occurs above and beyond the effect of AA on drinking (for DPDD at early follow-up), and that improving negative affect is a reasonable treatment target.

One limitation of our study has to do with the fact that we only had a single measurement tool, the BDI, for negative affect. Although anger has been explored as a mediator of AA’s

effect on drinking (53), to our knowledge, neither anxiety nor irritability has been. Furthermore, we had some limitations to our analysis methods. For one, we only analyzed subjects with complete data, which could have limited our power to detect effects and may have caused us to have null effects at late follow-up, for example. Second, although lagging the time points in our analyses for the independent, mediator, and dependent variables was a strength in our study in terms of being able to make inferences about causality, latent growth curve modeling would have allowed us to include all time points and may have had more sensitivity to detect effects (54). Finally, this is an observational cohort study rather than, say, a randomized trial targeting depression or assigning individuals to degrees of AA attendance frequency, and therefore, causality cannot be definitively attributed to the independent variable or mediator in such a design; associations could have been driven by unmeasured variables.

Conclusions

In conclusion, we observed that changes in depression mediated the effects of AA attendance on later drinking (DPDD) at early follow-up consistent with some but not all studies. However, baseline depression severity did not moderate the indirect effect and therefore did not provide support to the hypothesis that baseline depression differences were driving the differences between studies. Importantly, however, these findings have clinical significance by increasing support for models that ameliorating negative affect may help to reduce alcohol use in individuals with AUD and that encouraging AA attendance may be one way to achieve drinking reduction via this mechanism.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Pictorial representation of statistical mediation model used for Aim 1/Analysis 1 and 2 (early follow-up for PDA and DPDD) and Analysis 5 and 6 (late follow-up for PDA and DPDD). Definitions: AA = AA attendance; PDA = percent days abstinent; DPDD = drinks per drinking day; BDI = Beck Depression Inventory; Early = early follow-up; Late = late follow-up; A = path A, B = path B, C = path C representing total effect before mediator is entered into model, C' = path C' representing direct effect after mediator is entered into model. Coefficients and significance values for Analysis 1 and 2 are in Table 2, and for Analysis 5 and 6 are in Table 3.

Figure 2.

Pictorial representation of statistical moderated mediation model used for Aim 2/Analysis 3 and 4 (early follow-up for PDA and DPDD) and Analysis 7 and 8 (late follow-up for PDA and DPDD). Definitions: AA = AA attendance; PDA = percent days abstinent; DPDD = drinks per drinking day; BDI = Beck Depression Inventory; Early = early follow-up; Late = late follow-up; A = path A, B = path B, C = path C representing total effect before mediator is entered into model, C' = path C' representing direct effect after mediator is entered into model. Coefficients and significance values for Analysis 7 and 8 are in Table 4.

Table 1

Correlations between AA attendance, depression, and alcohol use (early).

	PDA BL	DPDD BL	BDI BL	AA 3 months	PDA 6 months	DPDD 6 months	BDI 6 months	PDA 9 months
DPDD BL	-0.046, 253							
BDI BL	-0.247 ^{**} , 227	-0.014, 227						
AA 3 months	0.196 ^{**} , 239	0.238 ^{**} , 239	-0.201 ^{**} , 215					
PDA 6 months	0.269 ^{**} , 239	0.077, 239	-0.227 ^{**} , 214	0.438 ^{**} , 236				
DPDD 6 months	-0.113, 239	0.020, 239	0.202 [*] , 214	-0.282 ^{**} , 236	-0.817 ^{**} , 239			
BDI 6 months	-0.097, 215	-0.133, 215	0.455 ^{**} , 199	-0.235 ^{**} , 215	-0.381 ^{**} , 214	0.327 ^{**} , 214		
PDA 9 months	0.278 ^{**} , 237	0.072, 237	-0.212 ^{**} , 213	0.370 ^{**} , 233	0.705 ^{**} , 234	-0.532 ^{**} , 234	-0.362 ^{**} , 211	
DPDD 9 months	-0.142 [*] , 237	0.076, 237	0.172 [*] , 213	-0.258 ^{**} , 233	-0.573 ^{**} , 234	0.575 ^{**} , 234	0.305 ^{**} , 211	-0.837 ^{**} , 237

*
 $p < 0.05$,**
 $p < 0.01$.

All values are reported in the format: Spearman's rho, number of participants; BL = baseline, BDI = Beck Depression Inventory, PDA = percent days abstinent, DPDD = drinks per drinking day (transformed variables for PDA and DPDD).

Table 2

Results from mediation analyses to examine whether changes in depression mediate the effect of AA attendance on alcohol use outcomes at early follow-up (3, 6, 9 months; Fig.1; Aim 1; Analysis 1, 2).

PDA early (n = 194)	Coeff	SE	t	p
Path A (to BDI 6 months)				
AA 3 months	-5.043	2.432	-2.073	0.040
PDA baseline	-0.009	2.336	-0.004	0.997
Path A (to PDA 6 months)				
AA 3 months	0.441	0.075	5.875	0.000
BDI baseline	-0.004	0.002	-1.751	0.082
Path B (to PDA 9 months)				
BDI 6 months	-0.004	0.003	-1.415	0.159
AA 3 months	-0.059	0.088	-0.669	0.505
BDI baseline	0.001	0.003	0.526	0.599
Total Effect (to PDA 9 months)				
AA 3 months	0.282	0.102	2.757	0.006
BDI baseline	-0.003	0.003	-1.195	0.234
	Coeff	Boot SE	Boot LLCI	Boot ULCI
Indirect effect (BDI 6 months is mediator)	0.013	0.012	-0.002	0.051
Indirect Effect (PDA 6 months is mediator)	0.221	0.043	0.141	0.310
DPDD early (n=194)	Coeff	SE	t	p
Path A (to BDI 6 months)				
AA 3 months	-4.723	2.394	-1.973	0.050
DPDD baseline	-0.293	0.507	-0.579	0.563
Path A (to DPDD 6 months)				
AA 3 months	-1.482	0.437	-3.388	0.001
BDI baseline	0.023	0.010	2.204	0.029
Path B (to DPDD 9 months)				
BDI 6 months	0.023	0.011	2.060	0.041
AA 3 months	-0.293	0.347	-0.845	0.399
BDI baseline	-0.0001	0.010	0.008	0.994
Total Effect (to DPDD 9 months)				
AA 3 months	-1.177	0.437	-2.694	0.008
BDI baseline	0.022	0.011	2.023	0.044
	Coeff	Boot SE	Boot LLCI	Boot ULCI
Indirect effect (BDI 6 months is mediator)	-0.019	0.013	-0.055	-0.0004
Indirect effect (DPDD 6 months is mediator)	-0.138	0.043	-0.234	-0.065

PDA = percent days abstinent, DPDD = drinks per drinking day, BDI = beck depression inventory, AA = AA attendance, Coeff = coefficient, SE = standard error, LLCI = lower level of confidence interval, ULCI = upper level of confidence interval. All indirect effect values are completely standardized.

Table 3

Results from analyses to examine whether changes in depression mediate the effect of AA attendance on alcohol use outcomes at late follow-up (12, 18, 24 months; Fig. 1; Analysis 5, 6).

PDA late (<i>n</i> = 183)	Coeff	SE	<i>t</i>	<i>p</i>
Path A (to BDI 18 months)				
AA 12 months	−7.947	3.966	−2.004	0.047
PDA baseline	0.134	2.114	0.064	0.949
Path A (to PDA 18 months)				
AA 12 months	0.397	0.151	2.627	0.009
BDI baseline	−0.004	0.003	−1.779	0.077
Path B (to PDA 24 months)				
BDI 18 months	0.000	0.003	0.080	0.937
AA 12 months	0.067	0.097	0.691	0.490
BDI baseline	0.001	0.003	0.169	0.866
Total effect (to PDA 24 months)				
AA 12 months	0.351	0.131	2.680	0.008
BDI baseline	−0.003	0.003	−0.839	0.403
	Coeff	Boot SE	Boot LLCI	Boot ULCI
Indirect Effect (BDI 18 months is mediator)	−0.006	0.103	−0.293	0.159
Indirect effect (PDA 18 months is mediator)	0.137	0.050	0.033	0.234
DPDD late (<i>n</i>=183)	Coeff	SE	<i>t</i>	<i>p</i>
Path A (to BDI 18 months)				
AA 12 months	7.801	4.010	−1.945	0.053
DPDD baseline	−0.109	0.486	−0.225	0.823
Path A (to DPDD 18 months)				
AA 12 months	−1.478	0.793	−1.864	0.064
BDI baseline	0.029	0.012	2.417	0.017
Path B (to DPDD 24 months)				
BDI 18 months	−0.004	0.012	−0.362	0.718
AA 12 months	−0.356	0.807	−0.441	0.660
BDI baseline	0.006	0.011	0.570	0.570
Total effect (to DPDD 24 months)				
AA 12 months	−1.358	0.730	−1.860	0.065
BDI baseline	0.024	0.012	2.021	0.045
	Coeff	Boot SE	Boot LLCI	Boot ULCI
Indirect effect (BDI 18 months is mediator)	−0.004	0.012	−0.015	0.034
Indirect effect (DPDD 18 months is mediator)	−0.122	0.066	−0.257	0.001

PDA = percent days abstinent, DPDD = drinks per drinking day, BDI = beck depression inventory, AA = AA attendance, Coeff = coefficient, SE = standard error, LLCI = lower level of confidence interval, ULCI = upper level of confidence interval. All indirect effect values are completely standardized.

Table 4

Results from moderated mediation analyses to examine whether baseline depression severity moderates the degree to which changes in depression mediate the effect of AA attendance on alcohol use outcomes at late follow-up (12, 18, 24 months; Fig. 2; Analysis 7, 8).

PDA late with baseline depression severity moderator variable (n=194)	Coeff	SE	t	p
Path A (to BDI 18 months)				
BDI mod	6.227	3.108	2.004	0.047
BDI mod × AA 12 months	-15.811	7.644	-2.068	0.040
Path A (to PDA 18 months)				
BDI mod	-0.403	0.124	-3.242	0.001
BDI mod × AA 12 months	0.712	0.284	2.507	0.013
Path B (to PDA 24 months)				
BDI mod	0.080	0.227	0.351	0.726
BDI mod × BDI 18 months	0.001	0.006	0.209	0.835
BDI mod × PDA 18 months	-0.055	0.139	-0.396	0.692
	Coeff	Boot SE	Boot LLCI	Boot ULCI
Indirect effect (BDI 18 months is mediator); minimal/mild sepression	0.001	0.042	-0.036	0.065
Indirect effect (BDI 18 months is mediator); moderate/severe depression	-0.009	0.067	-0.139	0.135
Indirect effect (PDA 18 months is mediator); minimal/mild depression	0.073	0.157	-0.254	0.346
Indirect effect (PDA 18 months is mediator); moderate/severe depression	0.570	0.129	0.326	0.844
Index of moderated mediation (BDI 18 months is mediator)	-0.009	0.072	-0.155	0.132
Index of moderated mediation (PDA 18 months is mediator)	0.497	0.204	0.137	0.922
DPDD late with baseline depression severity moderator variable (n=183)	Coeff	SE	t	p
Path A (to BDI 18 months)				
BDI mod	6.157	3.115	1.977	0.050
BDI mod × AA 12 months	-15.868	7.628	2.080	0.039
Path A (to DPDD 18 months)				
BDI mod	0.447	0.475	0.942	0.602
BDI mod × AA 12 months	-0.169	1.680	-0.101	0.920
Path B (to DPDD 24 months)				
BDI mod	0.667	0.606	1.100	0.273
BDI mod × BDI 18 months	0.014	0.029	0.476	0.635
BDI mod × DPDD 18 months	0.117	0.192	0.610	0.542
	Coeff	Boot SE	Boot LLCI	Boot ULCI
Indirect effect (BDI 18 months is mediator); minimal/mild depression	-0.0042	0.119	-0.412	0.172
Indirect effect (BDI 18 months is mediator); moderate/severe depression	0.129	0.238	-0.266	0.687
Indirect effect (DPDD 18 months is mediator); minimal/mild depression	-1.094	0.758	-2.806	0.156
Indirect effect (DPDD 18 months is mediator); moderate/severe depression	-1.028	0.945	-2.954	0.487
Index of moderated mediation (BDI 18 months is mediator)	0.133	0.262	-0.301	0.733

PDA late with baseline depression severity moderator variable (n=194)	Coeff	SE	<i>t</i>	<i>p</i>
Index of moderated mediation (DPDD 18 months is mediator)	0.066	1.180	-2.282	2.389

BDI mod = Categorical BDI moderator variable, DPDD = drinks per drinking day, BDI = beck depression inventory, AA = AA attendance, Coeff = coefficient, SE = standard error, LLCI = lower level of confidence interval, ULCI = upper level of confidence interval; none of the paths for the analysis using percent days abstinent as the outcome were significant so these results are not reported in a table.

There were no significant interaction terms nor were the indices of moderated mediation significant for PDA or DPDD at early follow-up (Analyses 3,4) and so no table was created.