

Social networks as mediators of the effect of Alcoholics Anonymous

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ABSTRACT

Aims This study tested the hypothesis that the relationship between Alcoholics Anonymous (AA) involvement and reduced substance use is partially explained (or 'mediated') by changes in social networks.

Design This is a naturalistic longitudinal study of the course of alcohol problems.

Setting Study sites were the 10 largest public and private alcohol treatment programs in a northern California county.

Participants Three hundred and seventy-seven men and 277 women were recruited upon seeking treatment at study sites.

Measurements At baseline and 1-year follow-up, we assessed alcohol consequences and dependence symptoms, consumption, social support for abstinence, pro-drinking social influences and AA involvement.

Findings In the structural equation model, AA involvement was a significant predictor of lower alcohol consumption and fewer related problems. The size of this effect decreased by 36% when network size and support for drinking were included as mediators. In logistic regression models predicting abstinence at follow-up, AA remained highly significant after including social network variables but was again reduced in magnitude. Thirty-day abstinence was predicted by AA involvement ($OR = 2.9$), not having pro-drinking influences in one's network ($OR = 0.7$) and having support for reducing consumption from people met in AA (versus no support; $OR = 3.4$). In contrast, having support from non-AA members was not a significant predictor of abstinence. For alcohol-related outcomes other than abstinence, significant relationships were found for both AA-based and non-AA-based support.

Conclusions The type of social support specifically given by AA members, such as 24-hour availability, role modeling and experientially based advice for staying sober, may help to explain AA's mechanism of action. Results highlight the value of focusing on outcomes reflective of AA's goals (such as abstinence) when studying how AA works.

KEYWORDS AA, mediation, social support.

INTRODUCTION

In the United States, Alcoholics Anonymous (AA) is the most commonly sought source of help for alcohol-related problems (Room & Greenfield 1993; Weisner *et al.* 1995), and most alcohol treatment programs introduce clients to AA and rely on it as a form of aftercare (Institute of Medicine 1990; Donovan & Mattson 1994). A positive

association between involvement in AA and better drinking outcomes has been established (Emrick *et al.* 1993; Tonigan *et al.* 1996; Tonigan *et al.* 2000), but the mechanisms underlying this relationship are not well understood. One partial explanation for AA's effect may be the changes in social networks that accompany AA involvement. Such changes include newcomers to AA replacing their substance using friends with new abstinent friends

who also attend 12-Step self-help groups (Humphreys & Nock 1997), those currently involved in AA being more likely to seek out helping relationships and places where others are not drinking (Humphreys *et al.* 1994; Snow *et al.* 1994), and AA involvement leading to greater trust and support within social networks (Humphreys *et al.* 1994, 1999).

Two recent longitudinal studies have examined social networks as a potential link in the causal path involving AA affiliation and improved substance abuse-related outcomes. The first studied 2337 male in-patients in US Veterans Affairs (VA) programs, and found that the positive relationship between AA/NA involvement and less frequent substance use at 1-year follow-up was partially mediated (i.e. explained) by two aspects of friendship networks: general friendship quality and support for abstinence (Humphreys & Nock 1997). We do not know whether these findings generalize beyond male VA inpatients, beyond that part of social networks made up of friends, or to other outcomes.

Project MATCH also modeled AA's mechanism of action and found that AA-orientated treatment (Twelve-Step facilitation or TSF) appeared to 'inoculate' against a social network saturated with frequent drinkers (Project MATCH Research Group 1998), which was explained in part by the higher AA involvement among the TSF subjects (Longabaugh *et al.* 1998). Somewhat limiting the generalizability of these findings, none of the subjects in that clinical trial were recent intravenous drug users, nor had current diagnoses for sedatives/hypnotic drugs, stimulants, cocaine or opiates; none had legal or probation problems that could prevent protocol compliance; none were likely to remain residentially unstable; and all had contacts to help the study locate them later (Project MATCH Research Group 1997).

This paper integrates the strength of these studies and attempts to build upon them. We consider alcohol problem measures (e.g. dependence symptoms and social consequences) in addition to alcohol consumption. We use a broader definition of social networks than friendship, and consider network function (general and abstinence-specific support) and structure (network size and drinking status) (Beattie & Longabaugh 1997) as well as the source of support. Our study population is a mixed gender sample entering treatment in heterogeneous public and private programs, who were recruited with minimal exclusion criteria and were re-interviewed 1 year later. Thus, they are more broadly representative of alcohol treatment seekers than either the VA or Project MATCH samples.

We hypothesized that the relationship between AA involvement, and alcohol problem severity and use, is

partially explained by an increase in the number of people regularly available for emotional and instrumental support, and by a reduction in the number of people in the social network who drink heavily and who are supportive of the respondent's drinking. We also hypothesized higher rates of abstinence (an AA goal) among respondents whose support for cutting down comes from AA members. In contrast, we hypothesized that the very presence of 'functional' (Beattie & Longabaugh 1997) support for cutting down—regardless of source—will be associated with other (non-AA-specific) outcomes such as social consequences.

METHODOLOGY

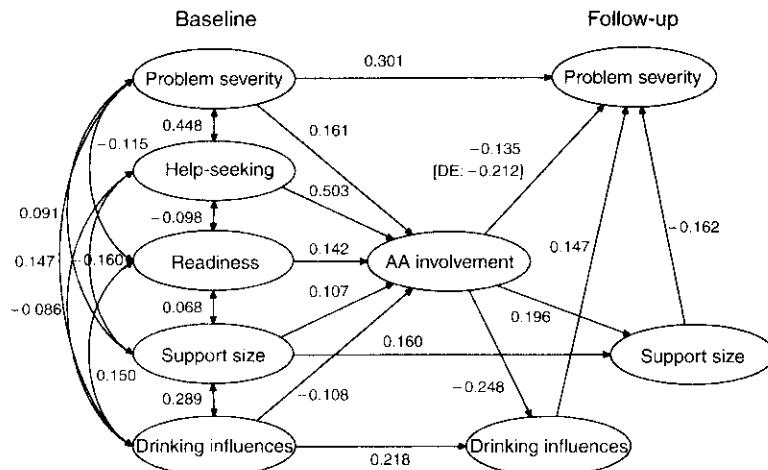
Sample

The study was conducted in a Northern California County of 900 000 residents that was selected on the basis of its diverse population characteristics, mix of rural and urban areas and generalizability (Weisner & Schmidt 1992; Weisner & Schmidt 1995; Greenfield & Weisner 1995). Baseline data were collected in 1995 and 1996 on individuals representative of people entering public and private treatment programs in the county. Recruitment covered consecutive admissions at all programs in the county which had at least one admission per week: these included public and private detoxification programs, private in-patient and day hospital programs, public out-patient and residential services, and the out-patient clinics at a large HMO (health maintenance organization) (Kaskutas *et al.* 1997). Trained research staff who were not employees of the treatment agencies administered a structured survey interview to all consenting participants by the end of their third day of residential treatment or third out-patient visit. The overall baseline participation rate was 80% ($n = 927$).

One-year follow-up interviews were successfully conducted by telephone (or in person when unreachable by telephone) with 78% of the baseline sample ($n = 722$). Attrition analysis found no differences in income, psychiatric or alcohol problem severity; however, males and African Americans were under-represented at follow-up (77% male and 33% African American sample at baseline, 57% male and 26% African American sample at follow-up; $p < 0.001$).

Because of our focus on how AA involvement affects alcohol problems, respondents who did not report drinking in the 12 months prior to the baseline interview (33 men and 35 women) were excluded from the analysis presented here. The resultant sample ($n = 654$) was 42% female, 26% African American and 7% Hispanic; 8.3%

Figure 1 Path model and coefficients of social influences mediating AA's influence on outcome.



had at least a high school degree, half were single or divorced and the mean age was 38 years.

Measures

Variables used in our structural equation models have been grouped conceptually into five baseline and four follow-up constructs (Fig. 1), as follows.

Severity of alcohol problems (baseline and follow-up)

This was the study's central outcome variable. Three composite measures and one single-item variable represent problem severity: dependence symptoms (based on nine items; e.g. got drunk when should not; blacked-out; had eye-opener; had shakes) (American Psychiatric Association 1994; Caetano & Weisner 1995); alcohol-related consequences (based on eight items, e.g. being arrested when drinking, having an accident or close call when drinking) Hilton 1987; Weisner *et al.* 1995); number of drinks in the past year (based on the graduated frequency series) (Clark & Hilton 1991).

Readiness to change (baseline)

This control variable was included as it seemed likely to predict both AA affiliation and drinking outcome. Four subscales represent readiness to change (Prochaska & DiClemente 1984): precontemplation, contemplation, action and maintenance.

Alcohol-related help-seeking (baseline)

This variable was used as a baseline predictor because it predicts AA affiliation (Emrick *et al.* 1993). Three composite measures were used: AA meeting attendance (number attended, life-time and last 12 months); AA

involvement, using seven items from the AA affiliation scale (Humphreys *et al.* 1998), e.g. do respondents identify as a member; have a sponsor; read literature, etc.; and formal alcohol service utilization (number of times in past 12 months that respondent received some form of alcohol treatment).

Social network size (baseline and follow-up)

This was the first hypothesized mediator. The size of the respondents' social network was distinguished using three questions developed for this study: number of people you 'had to talk to when you are worry about personal problems, such as family or work'; number of people who have 'helped you with practical things when you needed it, such as giving you rides, helping with babysitting, a quick loan, and so forth'; and number of 'family members (including spouse, children, stepchildren and parents) and friends (including partner or lover and roommates) who you have regular contact with. By regular, we mean you see them or talk on the phone with them once or more every couple of weeks'.

Social network support for drinking (baseline and follow-up)

This was the second hypothesized mediator, assessed by two questions about the group of people with whom the respondent had regular contact: how many 'are heavy or problem drinkers'; and how many 'encourage you to drink or use drugs'.

AA involvement (follow-up)

This was assessed at the follow-up interview as the number of AA meetings attended and the number of AA activities engaged in (from a list of four: had a sponsor; sponsored someone; read literature; did service) in the last 12 months.

Variables developed for regression analyses

Five of the conceptual areas used in the path models were retained in the linear regression models predicting follow-up problem severity: baseline and follow-up problem severity; and AA involvement, social network size and drinking influences at follow-up. Composite measures representing each area were constructed by factor analyzing the correlation matrices of the manifest variables for the retained latent constructs.

Four of the five composite measures (all but the outcome variable, follow-up problem severity) were also used in the logistic regression analysis. To enable a focus on the AA-specific goal of total abstinence, two dichotomous outcome variables were created, respectively, indicating short- (30-day) and longer-term (90-day) abstinence prior to the follow-up interview. Two other variables (also inappropriate for use in the structural equation model) were constructed that allowed us to look more directly at mechanisms and outcomes specific to AA. For example, we had asked respondents how many of their family and close friends 'actively support your effort to reduce your alcohol or drug use?' and 'how many [of these] did you meet at AA or NA?' This variable was not used in the structural equation model, as it did not fit conceptually with the other social network constructs and adding it would have further increased the instability of the model. To capture simultaneously the presence and the source of a respondent's social network supportive of their effort to cut down, a three-level composite variable was created: no regular contacts who are supportive of their effort to cut down; regular contacts who are supportive of their effort, none of whom were met at AA; and regular contacts who are supportive of their effort, some (or all) of whom were met at AA. In the logistic regression, the latter two groups are each compared to the first, using an SPSS indicator variable.

Data analysis

Preparatory to the structural equation modeling, Pearson's correlations and regression analysis were used to assess the simple (bivariate) relationships between baseline and follow-up measures. EQS (EQuationS, Bentler & Weeks 1980) was then used to construct and test a more complex, simultaneous structural equation model hypothesizing specific paths of influence among baseline and follow-up measures. To relieve the non-normality in the measures used in our analysis, tetrachoric and polychoric correlations (for dichotomous variables) were estimated and standard Box-Cox transformations (for continuous variables) were applied (West *et al.* 1995) where appropriate. Path coefficients dis-

cussed here are derived from the standardized solution provided in EQS (Bentler 1989).

The five baseline latent factors are shown along the left in Fig. 1. In our model, each of these baseline factors are predicted to influence AA involvement at follow-up (shown in the center of the figure). In turn, AA involvement at follow-up is modeled as relating to social network size and pro-drinking influences at follow-up (the proposed mediators), and ultimately to alcohol problem severity at follow-up (shown along the top right of the figure). The model also accounts for the baseline influences of problem severity and pro-drinking influences on those respective measures at follow-up.

The arrow between drinking influences at follow-up and problem severity at follow-up represents a path that is estimated only in the mediational model. This approach was suggested by MacKinnon & Dwyer (1993) and was employed in Humphreys *et al.* (1999). Two coefficients are shown for the path between AA involvement at follow-up and problem severity at follow-up—one indicating the relationship between AA involvement and problem severity without considering the mediating effect of drinking influences (labeled 'DE' for 'direct effects'), the other presenting the relationship under the mediational model (in which the influence of social networks on problem severity are also considered). The path models were estimated with and without the mediating paths; and the beta coefficients between AA involvement and problem severity were compared between the two models. The magnitude of change in the beta coefficient in the presence of mediation was calculated as the percentage decrease in the direct effect of AA on problem severity in the mediational model:

$$[(\text{Beta AA}_{\text{pl}}) - (\text{Beta AA}_{\text{mediation}})] / \text{Beta AA}_{\text{DE}}$$

The larger the percentage, the stronger the support for mediation. *T*-tests of the ratios of parameter estimates (over their standard errors) were also conducted.

One method of establishing model fit in latent path models is a non-significant χ^2 statistic, which is based on normality assumptions of the measured variables. However, because this statistic is not asymptotically correct in cases of non-normal underlying data distributions, we instead employ EQS (Bentler & Weeks 1980) fit indices (NFI, NNFI, CFI) which are based on comparisons of residual sums of squares (Bentler & Bonett 1980; Bentler 1990). Indication of a 'reasonable model fit' is an average fit index close to 1.

As a second approach to studying the robustness of the mediational model, linear regression models were tested for the presence of mediation. Problem severity at follow-up is first studied as a function of AA involvement

Table 1 Baseline and follow-up scores for key measures of structural equation model ($n = 654$).

	Baseline (\pm SE)	Follow-up (\pm SE)	Correlation ¹
Problem severity			
mean # dependence symptoms	4.6 (\pm 0.1)	2.8 (\pm 0.1)**	0.49
mean # alcohol-related consequences	1.2 (\pm 0.1)	0.8 (\pm 0.1)**	0.40
no. drinks in past year	1851 (\pm 71)	930 (\pm 73)**	0.38
no. usual drinks consumed	7.3 (\pm 0.3)	3.3 (\pm 0.2)**	0.19
AA involvement			
no. AA meetings in past year	34.6 (\pm 3.2)	106.8 (\pm 6.6)**	0.07
% who have a sponsor	14%	26%*	0.26
% who read AA literature in past year	54%	55%	0.39
% who did AA service in past year	28%	24%	0.30
% who sponsor someone now	0.5%	4.5%**	0.21
no. of times in treatment in past year	3.4 (\pm 0.7)	8.0 (\pm 1.5)**	-0.01
Social network size			
no. people you can talk to	3.8 (\pm 0.2)	4.8 (\pm 0.3)*	0.09
no. people you can get help from	3.9 (\pm 0.2)	3.0 (\pm 0.1)*	0.23
no. people regularly in contact with	6.0 (\pm 0.3)	11.1 (\pm 0.5)**	0.25
Pro-drinking influences w/regular contacts			
no. heavy or problem drinkers	0.6 (\pm 0.04)	1.3 (\pm 0.1)**	0.11
% who are heavy or problem drinkers	14%	12%	0.26
no. encourage you to drink	0.2 (\pm 0.03)	0.5 (\pm 0.07)*	0.12
% who encourage you to drink	4%	4%	0.17

All correlations between baseline and follow-up are significant at $p < 0.05$ except for # of times in treatment in past year.

* Significant increase/decrease from baseline to follow-up at $p < 0.05$; ** significant increase/decrease from baseline to follow-up at $p < 0.01$.

without social influences included in the equation (the direct effects model); next, a second regression is conducted which includes drinking influences and size of the support network (the mediational model). In both models, baseline problem severity is included as a control variable. Evidence of a mediating effect is observed as above, by assessing the percentage of change in beta coefficients when mediation is modeled. We also conducted a *t*-test of the means of the estimated beta coefficients between the two models to judge further whether the change in beta coefficients (with versus without social networks included in the regression model) was significant. Such a test assumes independence of estimates which probably overestimates the resulting standard errors and is thus a conservative estimate of the effect. In addition, we studied the significance of the nested model (using the difference in the $-2\log$ likelihood).

Next, logistic regression models were used to study the odds of 30- and 90-day abstinence. Here we introduce in the model our measure indicating the presence and source of social support. Similar tests of mediation as those used in the linear regression were used to judge significance of the hypothesized mediating effect.

One last set of bivariate (χ^2 and ANOVA) analyses looks specifically at the presence and source of social

support, for each of the six potential drinking-related outcomes studied here.

RESULTS

Table 1 shows baseline and follow-up values for measures in five content areas from which our latent constructs are drawn. Pearson's correlation coefficients indicate a moderate to strong relationship between the same variable at each timepoint. Three measures of AA-specific involvement were significantly higher at follow-up: number of AA meetings, and the percentages who have sponsors and who are sponsors. The size of respondents' social networks is also higher at follow-up, as are the absolute number of pro-drinking influences. However, the proportion of heavy drinkers and the proportion who encourage respondents to drink in the social network is similar at baseline and follow-up.

As recommended by Baron & Kenny (1986), prior to model testing the presence of significant relationships at follow-up between AA involvement and outcome, between AA involvement and the hypothesized mediators and between the mediators and outcome, were each verified. Pearson's correlation coefficients ranged from

0.09 (network size with problem severity) to 0.15 (drinking influences with problem severity) and were significant in all cases.

An important preliminary step in the construction of more complex latent models is the extent to which individual marginal factors account for variability among its measured variables; in studying this, correlation matrices were used to remove the effects of individual variables' location and scale parameters. The proportion of variance explained by the respective marginal factors ranged from 6.2% to 74%. Cronbach's alpha for manifest variable groups ranged from 0.31 to 0.56.

The coefficients obtained in the path model with mediation are shown in Fig. 1. The path model χ^2 was significant, as is often the case with non-normal data (Humphreys & Noke 1997) ($\chi^2 = 1151$, $p < 0.001$, $df = 435$). However, the average value of the three fit indices produced by EQS (NFI = 0.913, NNFI = 0.917, CFI = 0.899) reflecting the difference in the sum of squares between the observed data and an independent model, and the observed data and our proposed model was 0.909 (± 0.009), indicating a reasonable fit for the path model which included the hypothesized mediators.

Relative magnitude of *t*-test statistics obtained from ratios of parameter estimates over their standard errors were used to gauge the relative size of the mediating effect. Using this liberal criterion, path coefficients in the estimation of AA involvement from the baseline factors for help-seeking and stage of change were both relatively large, and would be considered significant at the 5% level if model fit were adequate (problem severity at the 10% level). Correcting significance level for multiple comparisons, correlations between baseline factors were all significant at the 5% level, with some (problem severity and help-seeking; support size and drinking influences) significant at the 1% level. The *t*-statistics for the paths at follow-up between AA involvement and problem severity ($\beta = -0.135$), and between drinking influences and problem severity ($\beta = 0.147$), would also be significant at the 5% and 1% level if model fit were adequate.

Also shown in Fig. 1 is the estimated path coefficient for the prediction of problem severity from AA involvement, when the mediating path from social network pro-drinking influences to problem severity is not included (this is designated 'DE' on the figure). In the direct effects model, the average of the fit indices (NFI = 0.858, NNFI = 0.884, CFI = 0.870) was somewhat lower at 0.871 (± 0.013); and the coefficient for AA involvement was somewhat larger ($\beta = -0.212$) than in the mediating model ($\beta = -0.135$). The difference between the betas in the competing models is 0.077; thus the influence of AA involvement on problem severity is lower by 36% ($0.077/0.212$) when the mediating path is included.

These results are consistent with the hypothesis that part of the effect of AA involvement on follow-up problem severity is due to the effect of friends' drinking influences at follow-up.

Replication with linear regression

A more parsimonious approach was next pursued, using mediational and direct effects linear regression models involving a reduced set of constructs. First, individual factors were estimated from observed variables for five constructs in the path model: baseline and follow-up problem severity and follow-up AA involvement, network size and support for drinking. The resulting variables were entered into linear regression models, in which follow-up problem severity was predicted by baseline problem severity and follow-up AA involvement (in the direct effects model) and additionally by network size and support for drinking (in the mediational model). Controlling for baseline problem severity, the coefficient for AA involvement as a predictor of problem severity at follow-up was -0.10 (± 0.06) in the mediational model (in which the two types of social network influences were included) and -0.15 (± 0.05) in the direct effects regression (without social networks in the model). This difference in the beta coefficients (via a *t*-test comparing the means) was not statistically significant at $p < 0.05$; but the ratio of the change in coefficients equalled 0.33, very close to that found in the path models (0.36). In the regression model that included the hypothesized mediators, the beta coefficient for network support size was not significant, but the coefficient for drinking influences was ($\beta = 0.16$ (0.04), $p < 0.001$). The percentage of variance explained in the mediational model was 46% and for the direct effects model it was 43%, and the difference ($\ln -2^* \log \text{likelihood}$) between the models was not significant ($p = 0.07$).

Odds of being abstinent at follow-up

Five independent variables were used in the two logistic regression models predicting the likelihood of being abstinent for the 30 days prior to the follow-up interview and also for the last 90 days (Table 2). In studying abstinence, the presence and source of support for cutting down is introduced into the model. It has three values: no support (13%, $n = 85$), support but not from people met in AA (61%, $n = 394$) and support which includes people met in AA (26%, $n = 167$). Baseline problem severity and AA involvement at follow-up were entered into the first step, prior to considering the effect of social networks on abstinence. Controlling for baseline problem severity, the odds ratio (OR) for AA involvement as a predictor of 30-day

Table 2 Logistic regression predicting 30- and 90-day abstinence at follow-up ($n = 534$).

	Odds ratio of 30-day abstinence Step 1	Odds ratio of 30-day abstinence Step 2	Odds ratio of 90-day abstinence Step 1	Odds ratio of 90-day abstinence Step 2
Baseline problem severity	0.78***	0.87	0.80***	0.90
AA involvement at follow-up	3.50*	2.94*	3.20*	2.75*
Size of social network at follow-up		0.97		1.14
Pro-drinking influences at follow-up		0.70***		0.56****
Presence and source of support for cutting down:				
Support but not from AA members (versus no support)		1.71		1.55
Support, from AA members (versus no support)		3.40**		2.79***

* $p < 0.00001$; * $p < 0.001$; ** $p < 0.005$; *** $p < 0.01$.**Table 3** The association between support for abstinence, AA-based support for abstinence, and six drinking outcomes ($N = 654$).

	No. drinks last year (mean)	Usual no. drinks (mean)	No dependence symptoms (mean)	No. social consequences (mean)	% sober, past 30 days	% sober, past 90 days
Rates overall	981	3.4	2.8	0.8	56%	51%
Based on support for cutting down:						
No support	1465	4.7	3.6	0.9	37%	33%
Support, none AA-based	937	3.2	2.7	0.7	52%	45%
Support, some AA-based significance	847	3.4	2.9	0.8	78%	72%
	$p = 0.04$	$p = 0.03$	$p = 0.05$	$p = 0.34$	$p < 0.00001$	$p < 0.00001$

abstinence at follow-up was 3.50 ($p < 0.00001$). When social network influences were included in the next step of the equation, the OR for AA involvement decreased to 2.94 but remained highly significant ($p < 0.00001$). The odds of being abstinent were not significantly predicted by the size of the respondent's social network, but the magnitude of pro-drinking influences in the social networks significantly reduced the likelihood of abstinence at follow-up (OR = 0.70, $p < 0.01$). In addition, compared to respondents with no social support for their effort to cut down, those with support from people they met at AA were at 3.4 times the odds of being abstinent for the 30 days preceding the follow-up interview ($p < 0.001$). Those with support for cutting down from non-AA members were not at a significantly higher odds of abstinence, as compared to those with no such support. The percentage of variance explained in the first step was 21%, increasing to 27% in the second step, and the difference in $-2\log$ likelihood from the nested model was significant at $p = 0.05$. The t -test comparing the means of the difference in the beta coefficients (in the mediation versus direct effects models) was not significant; the ratio of change in coefficients equalled 0.14.

The same approach was used to model 90-day abstinence as the outcome, with a similar pattern of results.

Drinking outcomes and source of social support

Finally, we studied the relationships between having a particular type of social support, and our six different drinking-related outcomes; overall values for each outcome are also shown (Table 3). As hypothesized, the rate for 30-day continuous abstinence is significantly higher among the respondents whose support for cutting down on drinking and drug use comes, at least in part, from people they had met in AA. About a third of the respondents with no support for their effort to cut down, about half of those with non-AA members' support and over three-quarters of those with AA members' support had been abstinent for the 30 days before the follow-up interview ($p < 0.00001$); similar rates obtained for 90-day abstinence. The number of drinks in the past year and the usual number of drinks consumed at a sitting were highest among those with no social support for abstinence ($p < 0.05$). Those with no support had, on average, one more symptom of dependence ($p < 0.05$) as compared to those with support (be it from AA members

or not), but there was no significant difference in alcohol-related social consequences among the groups.

DISCUSSION

These results from a heterogeneous, mixed-gender sample are consistent with the mediating influence that Humphreys *et al.* (1999) identified in their study of male veterans. Here, 36% of the influence of AA involvement on alcohol problem severity was explained by AA's effect on social networks. This is similar to the 47% decrease seen in Humphreys *et al.*'s (1999) study. In both studies, the fit of the path model was imperfect (due in part to the distributions of variables in the model, as well as the number of variables used in the model). However, the parallel result from our simple linear regression models (of a 33% decrease in the AA beta coefficient when the mediating variables were included) provides evidence of a mediating effect in our sample. Further, the consistency of findings across different statistical methods, samples and measures of social networks and drinking outcomes also suggests a certain robustness to the theoretical model itself.

Although the analyses presented here produced results consistent with mediation, we cannot judge the statistical significance of the mediational effect in our path model. Another limitation is the magnitude of effects: for example, our social network variables only reduced AA's influence on problems at follow-up by 36%, suggesting that other variables must be involved in AA's mechanism of action. Thirdly, in studying the relationship between abstinence and support from people met in AA, we looked at support for 'reducing your alcohol or drug use' rather than support for 'quitting altogether.' The latter would have better reflected the type of advice and support most likely to be forthcoming from AA members, and would have represented a stronger test of our hypothesis about the source of support mattering when it came to outcomes such as abstinence.

Despite these limitations we believe two implications follow, one methodological and the other potentially informative for treatment goals. The availability of sophisticated software for structural equation analysis has made it possible to test complex simultaneous models consisting of latent constructs with many paths between them. This approach is especially attractive because of the multiple influences at work in determining whether a problem drinker will remain so or will improve. However, variables representing some of these influences will not be normally distributed, and the resulting model fit will be statistically unsupported. As shown here, the use of parallel regression models, especially with a reduced variable set, provides a valuable window to further judge

the presence of mediation. The result from our complex structural equation models in support of mediation is strikingly similar to that obtained from much simpler linear regression models, with both indicating a decrease in the magnitude of the effect of AA on alcohol problems of about a third, when social networks are taken into account.

Turning to clinical implications, in their insightful discussion of the inoculating effect of AA involvement on drinkers who have wet social networks, Longabaugh *et al.* (1998) make several points that bear repeating. First, treatment providers and referral agencies should take extra steps to encourage AA participation for patients with networks supportive of drinking. They should help clients realize the value of changing their social network in the direction of support for abstinence, even when clients are reluctant to go to AA. In fact, focusing on other places that the drinker might go (besides AA) to develop a network supportive of abstinence may represent a welcome alternative for clients with wet drinking environments who are unwilling to become involved in AA. New therapies and strategies for helping patients learn how to change their social network, in the direction of greater support for abstinence, are needed; and existing programs are likely to offer direction for this effort. Several approaches come to mind, such as relapse prevention (Marlatt & Gordon 1985), the social model of recovery (Barrows 1998; Borkman *et al.* 1998; Kaskutas *et al.* 1998), AA-alternatives women for sobriety (WFS) (Kaskutas 1989, 1994) and SMART Recovery in the United States and Canada, the Links in Scandinavia, clubs for treated alcoholics in Croatia and Italy and Vie Libre in France (see Room 1998).

With this sample and an untreated sample of problem drinkers from the same county, Weisner found that those with fewer heavy drinkers in their social network were less likely to have remained a problem drinker at follow-up (Weisner & Matzger 2002). This result was not dependent on AA involvement and was found even among those who had not gone to AA in the past 12 months, supporting the hypothesis that social network changes represent a fundamental component not only of AA's effectiveness but of the recovery process at its most basic. These findings demonstrate the powerful influence of enduring environmental features in shaping drinking behavior, which exceeds that of transitory features (e.g. most treatments) (Humphreys & Tucker 2002).

That said, having people from AA in one's supportive social network appears to be especially important when abstinence is the outcome under study. For four of the six outcomes under study here, there was little difference between those whose support came from AA members versus from others—what made the difference was having support for one's effort to cut down. However,

rates for 30- and 90-day abstinence were highest among those having a support network that included people from AA, and the odds of 30- and 90- day abstinence were twice as high if some of that support came from people they had met at AA. This suggests that AA members offer types of social support that differ from those typically offered by non-members. Probable examples include role modeling of drink refusal skills, specific suggestions for avoiding situations in which relapse is likely ('slippery places and people' in AA parlance), offering to be available at all hours, sponsorship and experientially grounded practical advice for staying sober (including learning how to have fun in sobriety; Kaskutas *et al.* 1998). A full cataloging of AA-specific support behaviors and their impact on drinking remains an important objective for future research.

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