Association Between Social Influences and Drinking Outcomes Across Three Years

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ABSTRACT. Objective: Multiple studies have shown social network variables to mediate and predict drinking outcome, but, because of self-selection biases, these studies cannot reliably determine whether the influence is causal or correlational. The goal of this study was to evaluate evidence for a causal role for social network characteristics in determining long-term outcomes using state-of-the-art statistical methods. **Method:** Outpatient and aftercare clients enrolled in Project MATCH (N = 1,726) were assessed at intake and at 3, 6, 9, 12, and 15 months; the outpatient sample was also followed to 39 months. Generalized linear modeling with propensity stratification tested whether changes in social network ties (i.e., number of pro-abstainers and pro-drinkers) at Month 9 predicted percentage of days abstinent and drinks per drinking day at 15 and 39 months, covarying for Alcoholics Anonymous (AA) attendance at Month 9. **Results:** An increase in the number of pro-drinkers predicted worse drinking outcomes, measured by percentage of days abstinent and drinks per drinking day, at Months 15 and 39 ($p < .0001$). An increase in the number of pro-abstainers predicted more percentage of days abstinent for both time periods ($p < .01$). The social network variables uniquely predicted 5%–12% of the outcome variance; AA attendance predicted an additional 1%–6%. **Conclusions:** Network composition following treatment is an important and plausibly causal predictor of alcohol outcome across 3 years, adjusting for multiple confounders. The effects are consistent across patients exhibiting a broad range of alcohol-related impairment. Results support the further development of treatments that promote positive social changes and highlight the need for additional research on the determinants of social network changes. (*J. Stud. Alcohol Drugs, 73,* 489–497, 2012)

SOCIAL VARIABLES PLAY A CENTRAL ROLE in theories of the etiology, resolution of substance-related problems, and relapse in substance use disorder (Brown et al., 1989; Marlatt and Gordon, 1985; Tarter and Vanyukov, 2002). Individuals suffering from such disorders exist in a complex network of social forces that, in contrast to the short-term effects of formal treatment, exert a more enduring influence on behavior (Moos, 2003). Research of increasing sophistication shows these forces affect behavioral health trajectories and outcomes, including alcohol use (Beattie, 2001; Beattie and Longabaugh, 1999; Rosenquist et al., 2010; Valente, 2010). For example, a large, prospective, social network study found that less drinking among a person's close social ties led to reduced subsequent alcohol use (Rosenquist et al., 2010).

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Recognizing social influences on behavior change, both positive and negative, interventions have been developed that specifically target social resources to help individuals sustain adaptive social changes (Henggeler et al., 2002; Litt et al., 2009; McCrady et al., 2006; Nichols and Schwartz, 2006; United Kingdom Alcohol Treatment Trial [UKATT] Research Team, 2001). In terms of continuing care and recovery management, social contexts are also believed to play a central role in sustaining long-term change (McKay, 2011; Moos and Moos, 2004; Stout et al., 1999; White, 2009). The link between participation in recovery-oriented mutual-help organizations—such as Alcoholics Anonymous (AA) and Narcotics Anonymous—and better substance use outcomes may be due in large part to social changes in the networks of attendees that support abstinence (Bond et al., 2003; Kaskutas et al., 2002; Kelly et al., 2011). Changes in AA attendance linked to social networks may affect outcome as much as 3 years later (Longabaugh et al., 1998).

However, evidence to date that supports the potential importance of social mechanisms suffers from important methodological limitations. These limitations include cross-sectional rather than prospective measurement, failure to test for alternative explanations, and self-selection biases. Of these methodological flaws, self-selection biases are among the most difficult to detect and/or overcome and pose a potential threat to the validity of findings. Specifically,

a researcher can randomly assign research participants to conditions designed to facilitate changes in social structure (UKATT Research Team, 2001), but the researcher cannot directly compel the desired change(s). Participants who make desired (or undesired) changes are choosing to do so and are, therefore, self-selected (Cook and Campbell, 1979). That being the case, any subsequent improvement or deterioration in outcome attributed to social change could be because of factors merely correlated with the social change rather than being because of the social change itself. This is the first study to address self-selection biases within this framework.

"Self-selection" is construed broadly to include situations in which the change in the participant's status is associated with environmental forces as well as the client's own initiative. It would be a mistake, however, to think of modeling of the self-selection processes solely in terms of removing bias. If we were to find, for example, a variable that predicts a network measure and also predicts outcome, a new step would have been revealed in the probably lengthy causal chain that leads through social change to outcome.

In the present study, we used propensity score stratification (Dehejia and Wahba, 2002), which is designed to minimize the impact of selection biases attributable to measured covariates. No statistical adjustment can completely eliminate the chance that an unknown factor is responsible for improvement or deterioration that appears to be correlated with a change in social network characteristics. However, propensity stratification methods represent the state of the statistical art in this domain (Rubin, 2006) and have been rarely used in substance use disorder research. Traditional methods, such as covariate adjustment, can be helpful, but their weaknesses, such as failure to handle nonoverlapping distributions across groups, have been widely discussed in the statistical literature (Alemayehu, 2011; Hernández et al., 2005).

The method of matching or stratifying individuals on the basis of a propensity score allows a measure of control over multiple variables simultaneously and allows a test for an interaction between the predictor variable of interest and the propensity score. The latter capacity is valuable because intervention effects may vary by severity score (Thornton et al., 1998). The process of constructing a propensity score for the analysis additionally elucidates key predictor variables associated with social network changes, which can help us understand the mechanisms that lead to changes, for better or for worse, in social networks. In this study, we used propensity score stratification to inform our substantive knowledge of the causal status of social network changes. Furthermore, we evaluated the extent of these connections across two different clinical subsamples representing a range of alcohol-related impairment and involvement (i.e., a more severe aftercare sample and a less severe outpatient sample) from a large treatment trial for alcohol use disorder (i.e., Project MATCH [Matching Alcoholism Treatments to Client Heterogeneity]; Project MATCH Research Group, 1993). We examined the effects also across two alcohol use outcomes representing frequency (percentage of days abstinent [PDA]) and intensity (drinks per drinking day [DDD]) of alcohol use.

Method

Subjects

Two samples were recruited for the Project MATCH trial (N = 1,726): an outpatient sample (n = 952) and an aftercare sample (n = 774). The aftercare sample was recruited directly following residential treatment. Overall, outpatients were significantly younger, more residentially stable, and less dependent on alcohol than the aftercare patients (Goodman et al., 1992; Timko et al., 1993). A smaller proportion of outpatients (45%) than aftercare patients (62%) reported prior treatment for alcohol use disorder. The majority of patients in each trial arm (95% in outpatient, 98% in aftercare) met the criteria for alcohol dependence, as assessed using the Structured Clinical Interview for DSM-III-R (Spitzer and Williams, 1985). Although individuals dependent on other drugs (except marijuana) were excluded, a sizable minority of subjects reported some illicit drug use before recruitment (44% in the outpatient arm, n = 417, and 32% in the aftercare arm, n = 247). All participants signed informed consent, and the protocol was approved by all relevant institutional review boards.

Inclusion/exclusion criteria

Inclusion criteria were current *Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised* (DSM-III-R; American Psychiatric Association, 1987), diagnosis of alcohol abuse or dependence; alcohol was the principal drug of misuse; use of alcohol during the 3 months before study entry; minimum age of 18; and minimum sixth-grade reading level. Exclusion criteria were a DSM-III-R diagnosis of current dependence on sedative/hypnotic drugs, stimulants, cocaine, or opiates; any intravenous drug use in the prior 6 months; currently a danger to self or others; existing probation/parole requirements that might interfere with protocol attendance; lack of clear prospects for residential stability; inability to identify at least one "locator" person to assist in tracking for follow-up assessments; and acute psychosis or severe organic impairment.

Procedures

Following recruitment, subjects in both trial arms were randomly assigned to one of three individually delivered, psychosocial intervention conditions: cognitive–behavioral

therapy (Kadden et al., 1992), motivational enhancement therapy (Miller et al., 1992), and 12-step facilitation therapy (Nowinski et al., 1995). Twelve-step facilitation therapy and cognitive–behavioral therapy consisted of 12 sessions over a period of 12 weeks. Motivational enhancement therapy consisted of four sessions, also delivered over 12 weeks.

Completeness and accuracy of data

In both study arms, the follow-up rates remained greater than 90% over the first year after treatment; participation was 85% at Month 39. The outpatient sample was chosen to receive follow-up at Month 39 because it was more representative than the aftercare sample of clinical practice at the time. More complete details regarding this trial are available elsewhere (Project MATCH Research Group, 1997, 1998).

Measures

Alcohol use. Alcohol consumption was assessed using Form 90 (Miller, 1996; Miller and del Boca, 1994). Two dependent variables were derived from this measure: the percentage of days in the past 90 days a patient was abstinent from alcohol (PDA) and the number of DDD, on average, a patient consumed on days when there was any consumption.

Alcoholics Anonymous attendance. AA attendance was assessed using Form 90, which captured the number of AA meetings attended at intake as well as the 9- and 15-month follow-up time points. The percentage of days attending AA was the variable constructed to examine AA participation.

Social support variables. Social support variables were assessed using the Important People and Activities (IPA) Instrument (Clifford and Longabaugh, 1991). The forms of the IPA used at baseline and at Month 9 were similar; it was administered as an interview. We derived from the IPA two social support indices widely used, with some variations, in the literature. To limit participant burden, the IPA collected detailed data on a maximum of four "important" people, described as those who "have been important to you and with whom you've had contact (face to face or by phone) during the past 6 months. . . . anyone that you see as having had a significant impact on your life, regardless of whether you liked them." For these people, patients were asked how each reacted to their abstinence or drinking. The response options were the same for each: 1 = *left, or made you leave when you were not drinking/drinking;* 2 = *didn't accept;* 3 = *neutral;* 4 = *accepted;* 5 = *encouraged;* 8 = *person did not know about not drinking/drinking;* and 9 = *not applicable.* A person was recorded as allowing/encouraging abstinence/drinking if he or she endorsed either code 4 or code 5. A network member was recorded as "discouraging" abstinence/drinking if he or she endorsed either code 1 or code 2. Because preliminary analyses revealed a strong correlation between encouraging abstinence and discouraging drinking, and vice versa, we combined these into paired items, pro-abstinence and pro-drinking. A person was coded as "pro-abstinence" if he or she either encouraged abstinence or discouraged drinking, or both. A person was coded as "pro-drinking" if he or she either encouraged drinking or discouraged abstinence, or both. The number of each type of network member was summed to produce a scale range from 0 to 4. We used the raw number of such persons rather than a percentage of such persons in the network because we wanted to use measures that would be simple to use clinically and easy to interpret for researchers as well. The correlation between pro-abstainers and pro-drinkers was low in both arms (aftercare: $r = -.15$, $N = 698$, $p < .0001$; outpatient: $r = -.06$, $N = 864$, N.S.); therefore, we analyzed each index separately.

Statistical methods

Descriptives and data transformation. Means, standard deviations, and frequencies were calculated for descriptive statistics. Variables were examined for skewness and kurtosis. Both of the dependent variables (PDA, DDD) and AA attendance variable required transformations. The PDA variable received an arcsine transformation, and the DDD variable was given a square root transformation, as was done in the primary MATCH outcome analyses (Project MATCH Research Group, 1997; Tabachnick and Fidell, 2001). AA attendance was also positively skewed and was log transformed after adding 1.

For the current analyses, we used the following time points: baseline, Month 9 (social network variables [IPA]), and Months 15 and 39 (alcohol outcomes). These time points were chosen because the IPA was only given at baseline and Month 9, and to permit fully prospective analyses.

Multiple imputation. We used multiple imputation (Little and Rubin, 2002) for both propensity score estimation and the outcome analyses to take into account missingness possibly related to the independent and/or dependent variables in the primary analyses. Imputation was done for each arm separately, but the same set of imputations was used for the propensity and the outcome analyses to assure consistency. The amount of data missing for key variables in our analyses ranged from 0.05% to 7.8%. Because the missing data patterns were nonmonotone (i.e., some data were intermittently missing), we used the Markov Chain Monte Carlo method for the multiple imputation (Gilks et al., 1996). We performed 50 imputations using the procedures MI and MIANALYZE of SAS Version 9.2 (SAS Institute Inc., Cary, NC). The statistics we report for the variables in our analyses are the averaged results across the 50 imputations.

Propensity score stratification. Propensity score stratification is a multivariate matching technique pioneered by Donald Rubin and colleagues (Rubin, 2006, contains a collection of papers) for use in situations where group

equivalence cannot be guaranteed by randomization. A propensity analysis uses a regression model in which the dependent variable is the "exposure" of interest, and the predictors are variables that are confounded with the exposure. In our case, the exposure is measured by social network characteristics at Month 9, and the confounders are baseline variables known or suspected to be related to the social network characteristics. The predicted values from the regression are used to create groups, or strata, that are similar on the confounding variables. The outcome analysis then tests whether, within these matched groups, the exposure variable affects outcome.

Calculation of the propensity score. A separate propensity score was calculated for each social support variable because different variables would be expected to predict the number of network members supportive of drinking versus abstinence. However, the same pool of candidate variables was used across both network measures. The predictor variables were selected on the basis of prior research or theoretical considerations indicative of an association between each proposed predictor variable and at least one of the social network measures. Linear regression was used to calculate the propensity scores. This is an extension of classic propensity methods, which usually have dealt with binary exposure measures; it assumes linearity of the relationship between the propensity predictors and the exposure variables. Propensity models are usually overloaded to avoid overlooking potential strong confounders. The predictors (all measured at baseline except for AA attendance at Month 3) were the baseline values of IPA network measures (total network size, number of heavy drinkers and number of abstainers in the network, number of pro-abstinence and pro-drinking network members, and number of abstinent and drinking activities); demographics (age, gender, ethnicity [dichotomized European American vs. minority]); marital status; work status; whether the participant lives with family; general social support from family and friends (Procidano and Heller, 1983); diagnoses for agoraphobia, social phobia, and antisocial personality disorder; lifetime interpersonal consequences of drinking (Drinker Inventory of Consequences [DrInC]); prior treatment for alcoholism; AA attendance at baseline and Month 3; readiness for change; and baseline PDA and percentage of heavy drinking days. These predictors were selected because of empirical and/or theoretical considerations, suggesting they might be strong predictors of social network composition. The propensity score is the predicted value for each Month 9 social support measure.

Evaluation of the propensity score. We chose to use quintiles to stratify the propensity scores, based on simulation studies by Rubin (2006).

Propensity-stratified outcome analyses. The primary analysis is for an effect of the exposure variables (social network variables) on future drinking outcome, nested within propensity strata. This tests for a main effect of the social support variables conditional on the stratification. If the confounding variables are responsible for the apparent effect of the exposure variable on outcome, then the different strata will be related to outcome, but the exposure variable will have no effect. If, however, the exposure variable predicts outcome within the matched groups, the case that the exposure variable has causal effects independent of the confounding variables is strengthened. A test that pools across the five strata is used to test for overall significance for the exposure variable. The procedure is multivariate in that it takes into account a set of potential confounding variables simultaneously. Propensity stratification was used, for example, to demonstrate that cancer can be caused by smoking and not by a host of variables—such as gender, age, or access to medical care—that are at least somewhat correlated with smoking (Foody et al., 2001; Rubin, 2006).

Each social support predictor was tested independently because the stratification models were different for each. After testing for the main effect of each variable, we tested for an interaction between the propensity stratification and the social network predictor. This tests whether there is a larger/smaller effect on drinking for the social support measure at high versus low predicted levels for each social support variable. For example, we wanted to ascertain whether improvements in support for abstinence have a larger effect on drinking for those who have drinking-supportive versus abstinence-supportive predicted social networks. In these analyses, we included AA attendance measured at Month 9 as a covariate to determine whether the social network predictors add to the amount of variance accounted for by contemporaneously measured AA. We also did follow-up tests to estimate the unique variance attributable to network variables and AA.

Results

Descriptive data

Patients were, on average, 40 years old; 20.5% were female, 34.8% were married, and 49.3% were employed full time. Of the aftercare patients, 80.5% were White, 14.8% were Hispanic, 3.5% were Black, and 1.2% were of another race/ethnicity. Of the outpatients, 79.8% were White, 5.6% were Hispanic, 12.3% were Black, and 2.2% were of another race/ethnicity.

Changes in the average number of pro-drinkers and pro-abstainers between baseline and Month 9 were small. In aftercare, the number of pro-abstainers increased from 3.11 to 3.16, whereas the number of pro-drinkers dropped from 1.06 to 0.50. In outpatients, pro-abstainers increased from 2.70 to 2.83, and pro-drinkers declined from 1.18 to 0.89. However, although the overall number of relationships was relatively stable, which specific individuals were listed often changed from baseline to Month 9.

Table 1. Effects of social network predictors and propensity stratification on drinking outcomes at study Month 15, aftercare arm

	Percentage of days abstinent			Drinks per drinking day		
Effect	*F*	*df*	*p*	*F*	*df*	*p*
Pro-abstainer main effect	3.96	5, 15977	.0014	1.52	5, 12283	.1794
Pro-abstainer by strata interaction	0.09	1, 1346	.7662	0.02	1, 712.49	.8873
Stratification main effect	0.83	4, 37649	.5070	0.69	4, 29724	.6009
Pro-drinker main effect	10.08	5, 13001	<.0001	5.22	5, 13774	<.0001
Pro-drinker by strata interaction	2.38	1, 3326.4	.1229	0.80	1, 2407.9	.3708
Stratification main effect	1.83	4, 68711	.1200	1.10	4, 74775	.3533

Table 2. Effects of social network predictors and propensity stratification on drinking outcomes at study Month 15, outpatient arm

	Percentage of days abstinent			Drinks per drinking day		
Effect	*F*	*df*	*p*	*F*	*df*	*p*
Pro-abstainer main effect	7.27	5, 30809	<.0001	2.86	5, 32311	.0139
Pro-abstainer by strata interaction	2.71	1, 7941.1	.0996	3.61	4, 4149.5	.0577
Stratification main effect	1.68	4, 44782	.1511	0.97	4, 36686	.4239
Pro-drinker main effect	13.77	5, 25022	<.0001	10.07	5, 29718	<.0001
Pro-drinker by strata interaction	0.01	1, 3088.2	.9029	0.00	1, 2448.8	.9880
Stratification main effect	0.25	4, 43675	.9088	0.26	4, 32391	.9047

Table 3. Effects of social network predictors and propensity stratification on drinking outcomes at study Month 39, outpatient arm

	Percentage of days abstinent			Drinks per drinking day		
Effect	*F*	*df*	*p*	*F*	*df*	*p*
Pro-abstainer main effect	4.13	5, 126601	.0010	2.32	5, 94846	.0404
Pro-abstainer by strata interaction	2.58	1, 27362	.1083	0.88	1, 12006	.3492
Stratification main effect	2.56	4, 1.77E6	.0366	1.45	4, 1.42E6	.2146
Pro-drinker main effect	12.00	4, 141161	<.0001	7.05	4, 115657	<.0001
Pro-drinker by strata interaction	2.53	4, 7357.8	.1115	0.29	4, 7658.7	.5876
Stratification main effect	1.35	4, 143000	.2482	1.31	4, 1.87E6	.2618

Propensity score

The propensity models accounted for 20% of the variance of the number of pro-abstainers in aftercare and 21% in outpatients. The significant predictors at the .01 level or better, in descending order of effect size, were (a "+" after the predictor indicates a positive relationship and a "–" a negative one): baseline number of pro-abstainers (+), AA attendance at Month 3 (+), baseline network size (+), and social support from family (+) (Procidano and Heller, 1983) for aftercare. In the outpatient arm, the predictors were baseline number of pro-abstainers (+), AA attendance (+), DrInC interpersonal consequences (+), and number of prior treatments (+).

For the number of pro-drinkers, the propensity models explained 11% of the variance in aftercare and 14% in the outpatient arm. The significant predictors in aftercare were AA attendance at Month 3 (−), number of pro-drinkers (+), and AA attendance at baseline (−). In outpatients, the only predictor significant at $p < .01$ was the number of pro-drinkers at baseline (+).

Outcome analyses

Primary results of the outcome analyses (Tables 1–3). The number of pro-drinkers was a strong and consistent predictor of both 15-month outcome measures in both arms and in the outpatient arm at Month 39 ($p < .0001$). The number of pro-abstainers was a consistent predictor of PDA in both arms at Month 15 and at Month 39 in the outpatient arm. It was a weak and nonsignificant predictor of DDD because it did not achieve our alpha level of .01. There were no significant main effects for the propensity stratification, suggesting that the biases corrected by the propensity score were weakly correlated with outcome. There was also no propensity stratification by network variable interactions, which suggests that the impact of adding a pro-drinker or pro-abstainer to one's network was comparable for persons having either good or poor networks at Month 9. In supplementary analyses, we found that the baseline number of pro-abstainers or pro-drinkers were not significant predictors of drinking outcomes at any time point.

Table 4. Effects of covariates on drinking outcomes at study Month 15, aftercare arm[a]

	PDA				DDD			
Covariate	Coefficient	[95% CI]	*df*	*p*	Coefficient	[95% CI]	*df*	*p*
Baseline PDA/DDD	.158941	[.07747, .240408]	478.7	.0001	.293958	[.20132, .38660]	440.92	<.0001
12-step facilitation therapy	.000553	[-.07927, .080375]	468.3	.9891	.056625	[-.22347, .33672]	479.68	.6914
Cognitive–behavioral therapy	.034723	[-.04313, .112580]	476.2	.3813	.015910	[-.25989, .29171]	474.41	.9098
AA attendance	.004539	[.00330, .005779]	482.4	<.0001	-.015670	[-.02005, -.01129]	483.26	<.0001

Notes: PDA = percentage of days abstinent (arcsin transformed); DDD = drinks per drinking day (square root transformed); AA = Alcoholics Anonymous.
[a]From analyses in which the number of pro-abstainers was the predictor. Results from analyses where the number of pro-drinkers was the predictor are similar.

Table 5. Effects of covariates on drinking outcomes at study Month 15, outpatient arm[a]

	PDA				DDD			
Covariate	Coefficient	[95% CI]	*df*	*p*	Coefficient	[95% CI]	*df*	*p*
Baseline PDA/DDD	.358533	[.28139, .435672]	468.2	<.0001	.258555	[.16180, .35531]	446.4	<.0001
12-step facilitation therapy	.046327	[-.02428, .116931]	459.2	.1979	-.038693	[-.26181, .18442]	446.7	.7334
Cognitive–behavioral therapy	.011648	[-.06006, .083354]	470.8	.7497	.053476	[-.17342, .28037]	457.0	.6435
AA attendance	.003280	[.00166, .004898]	447.8	<.0001	-.012440	[-.01754, -.00734]	449.8	<.0001

Notes: PDA = percentage of days abstinent (arcsin transformed); DDD = drinks per drinking day (square root transformed); AA = Alcoholics Anonymous.
[a]From analyses in which the number of pro-abstainers was the predictor. Results from analyses where the number of pro-drinkers was the predictor are similar.

Table 6. Effects of covariates on drinking outcomes at study Month 39, outpatient arm[a]

	PDA				DDD			
Covariate	Coefficient	[95% CI]	*df*	*p*	Coefficient	[95% CI]	*df*	*p*
Baseline PDA/DDD	.327	[.239, .414]	496.7	<.0001	.136	[.030, .241]	497.0	.0117
12-step facilitation therapy	.0721	[-.0087, .1529]	496.4	.0805	-.171	[-.418, .075]	496.7	.1727
Cognitive–behavioral therapy	-.0101	[-.0920, .07183]	497.0	.8086	.089	[-.161, .339]	496.6	.4834
AA attendance, Month 9	.00324	[.00143, .00505]	493.0	.0005	-.0086	[-.0141, -.0030]	493.9	.0024

Notes: PDA = percentage of days abstinent (arcsin transformed); DDD = drinks per drinking day (square root transformed); AA = Alcoholics Anonymous.
[a]From analyses in which the number of pro-abstainers was the predictor. Results from analyses where the number of pro-drinkers was the predictor are similar.

Results for the covariates in the propensity models (Tables 4–6). The baseline value of the dependent variable predicted drinking outcome, except for DDD at Month 39. AA attendance was a significant predictor of both drinking outcomes for both arms at Month 15 and for the outpatient arm at Month 39. As in the primary Project MATCH outcome reports (Project MATCH Research Group, 1997, 1998), there were no treatment main effects.

Effect size estimates for the social network measures and AA (Table 7). The effect sizes for the number of pro-drinkers were strong and relatively consistent across samples, time, and dependent measures. The number of pro-abstainers was a more consistent predictor of abstinence than of drinking intensity. The effect size for AA was strong in the aftercare arm but weaker and less consistent in the outpatient arm.

Who are the "black hats"? Because of the strong negative effects of pro-drinkers, we conducted analyses to describe those who either discouraged abstinence or encouraged drinking. We call these people "black hats" after the bad guys in old cowboy movies. Across both study arms, we have

Table 7. Amount of variance of drinking outcome explained by social network predictors and AA[a]

Study month	Arm	Dep. var.	Pro-abstainers	Pro-drinkers	Pro-abstainers + Pro-drinkers	AA attendance
15	AC	PDA	3.3%	9.2%	10.9%	5.8%
15	AC	DDD	1.6%	4.4%	5.2%	6.0%
15	OP	PDA	3.6%	9.1%	11.8%	0.9%
15	OP	DDD	0.8%	6.9%	7.4%	1.8%
39	OP	PDA	2.5%	8.1%	10.0%	0.9%
39	OP	DDD	1.2%	5.8%	6.8%	0.7%

Notes: AA = Alcoholics Anonymous; dep. var. = dependent variable; AC = aftercare; OP = outpatient; PDA = percentage of days abstinent (arcsin transformed); DDD = drinks per drinking day (square root transformed). [a]Partial eta-squared values were estimated from Type III sums of squares, averaged over multiple imputation samples.

data on 1,120 black hats. The majority encouraged drinking; only 80 persons (7%) were described as discouraging abstinence. Forty percent were friends, 38% were family members, 14% were spouses or current intimates, and 8% were other relationships; 85% were people the index subject liked. Fifty-nine percent were the same gender as the index subject; this percentage was close to the same for both male and female subjects. Eighty-four percent had at least weekly contact with the index subject. Forty-seven percent were described by the index subject as "moderate" to "heavy" drinkers, with 23% being described as abstainers or recovering. Fifty-three percent were described as at least "somewhat" supportive of treatment, with only 7% "mixed" or opposed; however, 21% were not aware that the subject had been in Project MATCH treatment.

Discussion

The results provide multiple reasons in support of the notion that the effects of social network changes on alcohol use outcomes are causal in nature. The first is the use of propensity methods; there was, in most instances, a significant main effect for the network variables in the nested analyses, with strong effect sizes. Another reason is the consistency of results across both dependent variables and across two independent samples. The broad consistency of the network effects stands in contrast to the weak and inconsistent effects reported in the main Project MATCH publications (Project MATCH Research Group, 1997, 1998). In addition, the effects of social support are over and above those attributable to contemporaneously measured AA attendance. This finding implies that multiple distinct causal mechanisms are at work, although there is some shared variance in predicting outcome. The effect size for social network measures on drinking is substantial. Therefore, both AA and social support should be assessed separately in future studies of alcohol prognosis.

Having an advocate for abstinence in your social network is beneficial for recovery. Having a pro-drinker in your network, however, seems to more than offset the positive influence of a pro-abstainer. Therefore, social network interventions should prioritize minimizing negative influences. The predictors of salutary changes in high-risk social ties were greater AA attendance and a smaller number of high-risk pro-drinkers at baseline. This suggests that promoting AA is an important way to help someone with a pro-drinker in their network and is consistent with recent work showing that AA appears to mobilize effectively such specific recovery-supportive social network changes (Kelly et al., 2011). Besides AA attendance, only the baseline values of the social network measures were consistent predictors of the network measures at Month 9. Other propensity model predictors of pro-drinkers/abstainers had weak and inconsistent effects. Given the wide array of predictors we had in our propensity models, these findings underline how little we understand about the processes that lead people to change their social networks.

Most of us have an image of a "pro-drinker" as someone likely to be a heavy drinker dismissive of treatment, and the sample of "black hats" in the MATCH data contains a number of people who roughly fit that description. However, more than half are abstainers or light drinkers who may not believe that the subject has a drinking problem that requires treatment. This may be because, in the eyes of these social network members, these particular subjects were not perceived to be stereotypically alcoholic. Thus, there may be an important opportunity to intervene with these individuals to see if they can be converted to "white hats"; broader public health message initiatives might need to focus on dispelling the commonly held "skid row" alcoholic stereotypes (Litt et al., 2009; Longabaugh et al., 1995, 2010).

Because our models for predicting Month 9 social network status, despite incorporating a wide range of variables, had only a few significant predictors, more research is warranted. Although some predictors—such as the role of AA involvement—provide some guidance as to why and how people change their networks, the best predictor overall of your network at Month 9 is your baseline network. Thus, although the causal chain from network change to drinking is strong, we understand little about why someone adds or subtracts either a sobriety or drinking champion between baseline and Month 9. We can advise people that they should add a sobriety champion to their network or avoid a drinking champion, but we lack research guidance to help them meaningfully in this task. This highlights the importance of research to understand what leads people to change their networks. It is likely that there are different predictors for entirely new relationships versus renewal or rejuvenation of prior relationships and probably also different predictors for family, acquaintances, and other kinds of relationships. Thus, to move to the next level in our ability to help clients use social resources wisely, we must study the predictors of the success or failure of individual relationships. Future social network research should explore the impact of different kinds of supportive/negative roles, separating family (parents, spouse, children), long-term friendships, new associations, and re-initiated associations. Future intervention research should prioritize finding ways to eliminate or minimize the influence of pro-drinkers.

Limitations

The propensity stratification cannot directly take into account unmeasured confounding variables; however, unmeasured confounders that are correlated with measures we did use would have been partially adjusted. The propensity regression also assumes linearity. The significant effects found with the propensity stratification do not demonstrate

causality but do rule out the variables included in the propensity model as alternative explanations for the effects of the social variables, subject to the assumptions of the propensity analysis. The nature of the data gathered on the important people and activities measure, including its limit of four people with extensive data, restricted the social variables available for our analyses. That AA participation was primarily assessed by AA attendance is a limitation; but, in analyses not reported here, similar results were obtained when AA involvement was used. We chose AA attendance over involvement because of the larger *N* for attendance. Generalizability is also limited by the nature of the Project MATCH treatment-seeking sample and by the fact that alcohol treatment has changed in many ways since the inception of Project MATCH.

Conclusions

Social variables play a prominent role in theoretical explications of onset, remission from, and relapse to alcohol use disorders. Given the nature of these variables, experimental intervention studies, although used in an attempt to influence social network change, are limited in their ability to estimate the causal effects of social variables. Individuals choose relationships or choose the degree of exposure to such relationships themselves. This social self-selection can create a bias when one attempts to estimate the causal influence of social networks on behavior change. While it is not a perfect substitute for experimentation, propensity analysis is a rigorous method for minimizing this bias, although not the only one (Heckman, 1997). Many behavioral scientists have been trained in the tradition that randomized clinical trials are the best path to truth, a tradition that tends to deprecate observational research. Our colleagues in astronomy and geology, however, would be surprised to hear that their observational research is anything less than first rate. Methods like propensity stratification can help us make the best use of data that may be affected by selection processes, such as treatment compliance, non-ignorable missing data, stressful life events, and natural changes over time in psychopathology. Many important topics can only be studied outside the context of randomization, and behavioral scientists should embrace methods that help us make the best use of such data.

Having employed such methods using a fully prospective design, our findings provide support for the notion that close social ties causally influence subsequent changes in alcohol use behavior. Importantly, these pro-drinking and pro-abstinent network members were found to exert enduring influence across a 3-year period, over and above that of other influential social organizations, such as AA, which are specifically intended to help individuals achieve and maintain sobriety. Given the compelling magnitude of the effects of these social variables on drinking behavior and the often intransigent nature of social contexts, interventions that place relatively greater emphasis on effectively facilitating changes in individuals' high-risk social contexts are likely to achieve better long-term patient outcomes.

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