Published in final edited form as:

Alcohol Clin Exp Res. 2016 January ; 40(1): 170–177. doi:10.1111/acer.12925.

Association of Reduced Social Network Drinking with Improved Response Inhibition in Women During Early Recovery from Alcohol Use Disorders: A Pilot Study

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Abstract

BACKGROUND AND AIMS: Social support for recovery from alcohol use disorders (AUDs) is associated with better drinking outcomes and with improvements in self-reported impulsive behavior in individuals treated for AUDs. We build on these findings by using a behavioral task-based measure of response inhibition, a well-defined component of impulsivity, to examine the association of disinhibition with alcohol-specific social network characteristics during early recovery.

METHODS: Women (n=28) were recruited from treatment for AUD within 3–4 weeks of their last drink and were assessed at baseline and again 3 months later. Outcome measures were level of disinhibition at baseline and change in disinhibition from baseline to follow-up, measured using a computer-based continuous performance test. The primary independent variables were level of drinking in the social network at baseline, and change in network drinking from baseline to follow-up. Control variables included participants' recent alcohol consumption, IQ, and self-reported negative urgency.

RESULTS: The sample (50% black, age M(SD)=42.3 (9.5)) reported high rates of physical and sexual abuse before age 13 (43%), psychiatric disorder (60%), and previous treatment (71%); these variables were not associated with disinhibition. More drinking in participants' social networks was associated with greater disinhibition at baseline ($\beta = 11.8$, 95% CI= 6.0, 17.6). A reduction in network drinking from baseline to follow-up was associated with reduced disinhibition (interaction of network drinking x time on disinhibition: $\beta = -5.4$, 95% CI=-10.1, -.59), adjusted for control variables.

CONCLUSIONS: This study extends previous findings of an association between social networks and self-reported impulsivity to a neurobehavioral phenotype, response inhibition. Replication of these findings would support the potential role of social network drinking in cognitive change during early recovery from AUDs.

Keywords

social networks; response inhibition; impulsivity; alcohol use disorder

INTRODUCTION

Efforts to improve the behavioral treatment of alcohol use disorders (AUDs) during the last two decades have met with limited success. Project MATCH, a multi-site randomized clinical trial whose goal was to improve patient-treatment matching by assessing the relative efficacy of Motivational Enhancement Therapy (MET), Cognitive Behavioral Therapy (CBT), and Twelve-Step Facilitation (TSF) for clients with different sets of characteristics (Project MATCH Research Group, 1993) found little evidence for treatment-specific effects on drinking outcomes (Project MATCH Research Group, 1997). A similar multi-site trial in the United Kingdom which compared MET with a network therapy designed to help clients build supportive social networks found both treatments equally effective (UKATT Research Team, 2005). Numerous clinical trials in the decade following Project MATCH have also yielded little evidence for treatment-specific effects (Morgenstern and McKay, 2007). The lack of compelling evidence for treatment-specific effects, combined with findings from population-based samples that more than 50% of individuals with AUDs remit without any form of treatment (Cunningham et al., 2000; Sobell et al., 1996), and that remission from AUDs is largely influenced by individual-specific factors (McCutcheon et al., 2012), suggest that extra-treatment factors, or the context of recovery from AUDs, is at least as important as treatment (Humphreys et al., 1997; Moos, 2003; Moos and Moos, 2005).

Social networks are one factor outside of treatment that can potentially be changed by the individual (as opposed to, for example, residential neighborhood, socioeconomic status, or family of origin), and their role in recovery from AUDs has been documented since the 1980s (Favazza and Thompson, 1984; Logan, 1983). In 1982, a naturalistic study of 110 inner-city men followed from adolescence to age 47 found that increased religious involvement and Alcoholics Anonymous (AA) attendance were associated with remission from AUDs, while variables predictive of AUD development, including family history of alcoholism, premorbid antisocial behavior, and childhood adversity, were not (Vaillant and Milofsky, 1982). More recent longitudinal studies confirm the importance of social networks to recovery. A recent analysis of Project MATCH data found that treatment type had no association with drinking outcomes 15 and 39 months after treatment, but that AA attendance and the number of individuals in the network 9 months after treatment who were pro-abstinence or pro-drinking did, accounting for unique amounts of variance in drinking outcome (5–12% and 1–6%, respectively) (Stout et al., 2012). Similarly, a multisite study which assessed the efficacy of combinations of behavioral and medication treatments for alcohol dependence (Anton et al., 2006) found that more drinking in the social network was associated with fewer abstinent days during and after treatment (Longabaugh et al., 2010). A treatment designed specifically to change the composition of social networks to include more people supportive of abstinence resulted in 20% more abstinent days 2 years after treatment than 2 comparison behavioral interventions, demonstrating that changing network support is in itself an effective treatment (Litt et al., 2007; Litt et al., 2009). Consistent with

the above findings regarding AA attendance and social networks more generally, a recent analysis of Project MATCH data suggests that social factors are the primary drivers of behavior change in remission from AUDs, and that AA participation improves recovery by facilitating social changes (Kelly et al., 2012).

In addition to its association with improved drinking outcomes, AA attendance is associated with reductions in impulsivity. A study of previously untreated individuals with alcohol problems who were followed over 16 years found that AA participation was associated with reduced self-reported impulsivity in the first year after treatment, which in turn was associated with fewer alcohol problems; at 16 years, AA (but not professional treatment) was associated with decreased impulsivity (Blonigen et al., 2011a; Blonigen et al., 2009; Blonigen et al., 2013). In addition, although higher levels of impulsivity at year 1 were associated with greater mortality risk at year 16, this risk was reduced among individuals with high levels of support from friends and peers (Blonigen et al., 2011b).

Impulsivity is a broadly-defined behavioral characteristic underpinned by executive function (Dick et al., 2010). The self-report measures of impulsivity used in the studies mentioned above reflect an individual's interpretation of the environment and his or her responses to it. Task-based measures of impulsivity, by contrast, reflect more specific cognitive processes (Dick et al., 2010), such as response inhibition, a core construct of executive function (Miyake et al., 2000) and a well-defined component of impulsivity (Dick et al., 2010). Response inhibition is represented by the ability to deliberately inhibit a prepotent or dominant response. Deficits in response inhibition are found in unaffected individuals from families with parental alcohol problems (Acheson et al., 2011), and predict alcohol problems in youth from high-risk families, independent of IQ, parental alcoholism, and externalizing disorders (Nigg et al., 2006). Deficits in response inhibition may also result from the heavy alcohol use characteristic of severe AUDs. Individuals with AUDs commit more errors on behavioral tasks of response inhibition (Bjork et al., 2004; Goudriaan et al., 2011), and this deficit contributes to the maintenance of disorder despite negative consequences (Adinoff et al., 2007; Koob, 2009; Volkow et al., 2011). Executive functions, including response inhibition, can improve with abstinence (Drake et al., 1995; Duka et al., 2003; Loeber et al., 2010; Pitel et al., 2009). However, improvement takes time, and during early recovery individuals with AUD may require supplements to their own internal inhibition in order to tolerate the discomforts of early sobriety and behavioral change. In the current report, we build on previous findings that social networks are associated with AUD recovery and, more specifically, with decreases in impulsivity. We examine the association of alcohol-specific network characteristics with respondent's level of disinhibition during early recovery using a task-based measure of response inhibition. Our broad hypothesis is that social networks can act as an external source of executive control during early recovery from AUD. This is an observational study focused on the social context of recovery and not on specific treatment protocols. Thus, an underlying assumption is that type of treatment is not as important as the context in which it occurs.

MATERIALS AND METHODS

Sample

Women were recruited from 2 local alcohol treatment facilities in the central city and 1 treatment center in a nearby county via posters briefly describing the study, with contact information so that interested women could call for more information. The PI and study staff also attended group meetings at the facilities, where they described the study to clients and enrolled those who were willing to participate. Inclusion criteria were female gender, age 18 or older, in treatment for alcohol problems at enrollment, within 4 weeks of their last drink or drug, expect to be living in the area in 4 months, and willing to provide name and contact information of at least one person who could provide help in locating them for follow-up. Informed consent documents were reviewed and signed by all subjects prior to participating in the study. Of 75 women who were screened for the study, 24 had been sober for longer than 4 weeks and were excluded, and 51 women were enrolled. Of these, 29 completed baseline interviews, 1 completed only part of the baseline interview and was excluded from further assessments, 18 never showed up for baseline interviews despite repeated scheduling and reminders, and 3 left treatment before they were scheduled. Of the 29 who completed baseline interviews, 1 was excluded from further assessment because, although she had reported being in treatment for alcohol problems during screening, during her interview she reported no alcohol problems. Notably, of 9 women who were homeless, 7 were located for follow-up interviews. One woman lost to follow-up was incarcerated and thus unavailable for interview. Of 28 women eligible for follow-up, 18 completed follow-up interviews (64%). Follow-up rates at the treatment center in the county whose residents were younger than those in the city treatment sites ($M (SD) = 37.8 (10.2)$ versus $44.5 (8.5)$, $t_{(26)} = 1.8$, $p = .04$) were much lower, at 20% ($n=2 / 10$). The follow-up rate for women recruited from city treatment centers, by contrast, was 88% ($n=16 / 18$).

Assessment and measures

Face-to-face interviews were conducted at our research facilities in private interview rooms or, when participants were unable to travel to our facility, at their treatment facility. Private rooms were provided by the staff at each facility when available. If no private room was available, interviews were conducted in the cafeteria during unoccupied periods. At the baseline interview, an abbreviated version of the Semi-Structured Assessment for the Genetics of Alcoholism (Bucholz et al., 1994; Hesselbrock et al., 1999) was used to assess demographic information, medical history, lifetime alcohol and drug use disorders, history of psychiatric disorders and treatment, and trauma history; this information was updated at follow-up. Drinking quantity and frequency and drug use data for the previous 90 days were collected using the Form 90 (Tonigan et al., 1997) at the baseline interview, and for the intervening time at the follow-up interview. The Wechsler Abbreviated Scale of Intelligence (Wechsler, 2008) was administered to obtain an estimate of IQ for use as a control variable. Urgency, a self-report measure of disinhibition, was assessed using the UPPS Impulsive Behavior Scale (Whiteside and Lynam, 2001, 2003). The UPPS (urgency, [lack of] perseverance, [lack of] premeditation, sensation-seeking) is based on careful analysis of existing self-report measures of impulsivity (Whiteside and Lynam, 2001). Urgency, or the tendency to act impulsively in response to negative emotional states, is not measured in other

commonly-used self-report measures, and may be a particularly important construct during early recovery. In the current study, controlling for self-reported urgency allowed a clearer interpretation of the task-based measure of behavioral disinhibition described below.

Disinhibition—Disinhibition was measured using the *Conners Continuous Performance Test* (CPT-II) (Conners et al., 2003; Conners, 2010), a computerized assessment of sustained attention and response inhibition. The CPT-II produces measures related to inattentiveness, response inhibition, and vigilance. The test is performed on a computer, where letters appear on the screen at varying intervals of 1, 2, or 4 seconds. Participants are asked to depress the space bar for any letter that is not an “X”, and to withhold their response when they see an “X.” Commission errors are the number of times a participant responds to the “X”. Hit reaction time is the mean response time for all correct responses. Unusually fast reaction time when combined with a large number of commission errors indicates high levels of disinhibition. Disinhibition for this study was calculated as the sum of commission errors plus hit reaction time (based on age-adjusted T-scores). The measures have good test-retest reliability and little evidence for practice effects (Conners, 2010).

Network drinking score—Network drinking score was based on respondent answers to a version of the Important People Inventory, an instrument designed specifically to assess support for drinking or not drinking and general social support in clinical populations (Longabaugh et al., 2010). Participants were asked to name up to 15 people who were important in their lives and with whom they had had contact over the preceding 3 months. For each nominated network member, they were asked to report their relationship to them, frequency of contact, importance, and general and alcohol-specific support. Specific to the current report, participants were asked to quantify network member’s drinking as one of five possible choices (heavy, moderate, light, abstainer, recovering), with heavy drinking represented by “5” and recovering as “1”. Heavy, moderate, and light drinking were based on respondent’s interpretation of those terms. “Abstainer” was used only for lifetime abstainers, and “recovering” for individuals who had a history of alcohol problems but had not had a drink in at least 3 months. Recovering network members who had had a drink in the last 3 months were classified as light drinkers. The network drinking score was calculated by dividing the sum of all scores by the number of individuals in the network whose drinking status was reported. Other network measures tested but not associated with disinhibition were average frequency of drinking in the network, network drinking quantity-frequency score, network support for drinking, network support for treatment, and network support for recovery.

Statistical analyses

A mixed-effects within-subject design using a multilevel model was used to test the association between disinhibition score and network drinking score. The interaction of time with network drinking score was modeled to determine whether a change in network drinking was associated with a change in respondent’s level of disinhibition. Multilevel models use maximum likelihood estimation to accommodate missing data due to subject attrition (Luke, 2004). Control variables included in the equation were IQ, relapse (defined as any heavy drinking days between baseline and follow-up assessments; all drinking

between assessments met criteria for heavy drinking, > 3 drinks per day or 7 per week (Abuse, 2010)), self-reported urgency, and a dummy variable representing individuals lost to follow-up. Potential control variables tested in the multivariate equation which were not included due to lack of statistical significance, and whose exclusion produced an improvement in model fit, based on comparison of Akaike (AIC) and Bayes Information Criterion (BIC) statistics, were previous treatment for alcohol problems, number of lifetime AUD symptoms, and days since last drink. Disinhibition, network drinking score, and self-reported urgency were modeled as time-varying covariates. Data from one subject whose change in disinhibition score was an influential outlier was removed from the multivariate equation (her score increased 33% from baseline to follow-up, with a residual variance of 4.7 in its association with change in network drinking score; by contrast, the remaining subjects' scores changed less than 10% on average with residuals ranging from -1.3 to 1.0).

RESULTS

Sample characteristics

Participants ranged in age from 26 to 56, with a mean of 42.3 (SD=9.5, Table 1). Half the sample was African American, a majority was unemployed, and one-third had been homeless within the previous 90 days. Forty-three percent of the sample endorsed sexual or physical abuse before age 13; 21.4% reported both types of abuse. All women met criteria for AUD; drug use and psychiatric disorders were also common. All but 4 women had previously been treated for substance use or psychiatric problems. Variables associated with lower levels of disinhibition at baseline were higher estimated IQ, previous treatment for alcohol problems, greater number of lifetime AUD symptoms, and more days since last drink. Higher self-reported urgency was associated with a higher level of disinhibition at baseline. Women lost to follow-up had higher mean network drinking score than women who completed both baseline and follow-up interviews (M(SD)=2.8 (.41) versus 2.3 (.65), $t_{(26)}=2.2$, $p=.04$), but their mean level of disinhibition at baseline did not differ (M(SD)=113.8 (13.5) versus 110.3 (11.9), $t_{(26)}=.69$, $p=.49$), nor did IQ, demographic characteristics, trauma, substance use, psychiatric disorder, treatment experiences, or self-reported urgency.

Network characteristics

The average network drinking score was 2.5 (minimum: 1.2, maximum: 3.7) out of a possible range of 1 to 5 (Table 1). Its positive association with disinhibition indicated that women with heavier-drinking networks had higher levels of disinhibition (disinhibition scores for above and below-median levels of network drinking, respectively: M(SD) = 116.5 (3.1); M(SD) = 106.7 (3.1), $t_{(26)}=-2.24$, $p=.03$). The presence of recovering alcoholics and friends (regardless of drinking status) in the network was associated with lower levels of disinhibition. The presence of heavy drinkers and family members (regardless of drinking status) in the network was associated with higher levels of disinhibition. Ninety-three percent of women with an above-median proportion of recovering alcoholics in their networks had at least one network member who had attended AA in the previous year (M (SD, minimum, maximum) = 4.1 (3.1, 0, 20)), and 71.4% of women with a below-median

proportion of recovering alcoholics in their network had at least one network member who attended AA in the last year (M (SD , minimum, maximum) = 1.5 (1.2, 0, 3)).

Network drinking and respondent drinking outcomes

Network drinking score was not associated with proportion of heavy drinking days ($\beta = .04$, 95% $CI = -.11, .18$, $p=.63$) or with days since last drink ($\beta = -3.0$, 95 % $CI = -21.7, 15.6$, $p=.75$) before or after adjustment for lifetime AUD symptoms and previous treatment.

Network drinking and respondent response inhibition

In the first model with no interaction, a higher network drinking score was associated with higher levels of disinhibition (Table 2). The level of disinhibition decreased from baseline to follow-up, as indicated by the significant main effect of time. In model 2, the interaction of network drinking score with time was significant, and this model provided a better fit to the data as indicated by smaller AIC and BIC statistics. The interaction is graphically displayed in Figure 1. Women whose network drinking scores increased had a lower mean level of disinhibition at baseline than did women whose network drinking decreased. However, women whose network drinking scores increased also had an increase in disinhibition between baseline and follow-up, while women whose network drinking scores decreased had a slight decrease in disinhibition.

DISCUSSION

This study examined the association of social network drinking characteristics with disinhibition, measured using a computer-based behavioral task, in women early in their recovery from AUDs and again three months later. Two main findings were that the level of drinking among members of the social network was associated with the level of respondent disinhibition at baseline, and that a decrease in the level of network drinking over the follow-up period was associated with a decrease in respondent disinhibition, after adjustment for IQ, relapse during follow-up, and self-reported urgency. This is the first study to our knowledge to examine the association of social network drinking with a behavioral measure of response inhibition in a clinical sample.

These findings extend research that documents decreases in self-reported impulsivity as a function of duration of AA attendance across 16 years (Blonigen et al., 2009). Our use of a task-based measure of impulsivity, rather than self-reports of situational impulsivity, and our assessment of social networks directly, rather than using AA attendance as a measure of social support, has extended previous findings in ways that may help guide research on brain function in recovery from AUD. First, use of a task-based measure to detect change in response inhibition is more closely aligned with research showing improvements in neuropsychological abilities and executive function with abstinence than are studies using self-report measures. Second, the association of a broad measure of network drinking with improvement in response inhibition is consistent with evidence that the benefits of AA attendance are due largely to its facilitation of changes in the social network outside of AA to include more pro-abstinence members and reduce the number of members who support drinking (Kelly et al., 2012). Finally, the fact that network drinking was associated with

response inhibition during early recovery, but not with drinking outcomes, may help explain previous findings that the association of social network measures with drinking outcomes does not emerge until later in recovery. In Project MATCH, for example, the drinking habits of the social network at baseline had no association with drinking outcomes at 12, 15, or 39-months, but network drinking 9 months after treatment did (Stout et al., 2012). It is possible that response inhibition may be a more proximate measure of social influence, which is later translated to drinking behavior, consistent with the finding that AA participation was associated with reduced impulsivity 1 year after treatment, which in turn was associated with fewer alcohol problems, in individuals who were untreated at the beginning of study (Blonigen et al., 2011a).

The array of variables that were not associated with disinhibition, including childhood trauma, antisocial behavior, and illicit drug use, also contribute to the importance of these findings. Replication of these findings in a larger sample would support the important role of social networks in recovery from AUDs. This would be consistent with evidence that the same factors that contribute to the development of AUD have little agency in its remission (Knop et al., 2007;McCutcheon et al., 2012;Penick et al., 2010;Vaillant and Milofsky, 1982). The possibility that social networks are capable of enhancing early cognitive recovery from AUDs even among individuals with a host of risk factors predating the development of AUD, such as childhood abuse or early antisocial behavior, suggests that treatment for AUD might be modified to emphasize social support, while the skills of the therapist might best be used to address issues underlying the development and course of AUD.

Limitations

This study had numerous limitations which should be remembered when interpreting results. No specific treatment information was recorded, so whether treatment itself contributed to change in response inhibition could not be determined. However, the fact that no treatment main effects on drinking outcomes were found in a large, randomized trial (Group, 1998) and in subsequent randomized trials (Morgenstern and McKay, 2007) suggests that treatment effects on change in response inhibition are unlikely. The sample came from several different treatment settings and is not generalizable to larger treatment samples or to non-treatment samples. Follow-up rates were low in the group recruited from county treatment centers, but were 88% in the city sample. Women lost to follow-up reported higher levels of drinking in their social networks than did women who returned for follow-up, but their levels of disinhibition were similar to those of women who were followed up. The use of a multilevel model which accounts for missing data, as well as the lack of significance of a dummy variable representing women lost to follow-up, provide some reassurance that the missing data points do not unduly influence results; nonetheless, cautious interpretation is warranted.

Conclusion

This study extends previous findings of an association between social networks and self-reported impulsivity to a neurobehavioral phenotype, response inhibition. This innovation can help to guide research on brain function in recovery from alcohol use disorders (AUDs).

Replication of these findings in a larger sample including both men and women would support the important role of social network drinking in recovery from AUDs.

Acknowledgements

This study was funded by the University Research Strategic Alliance (URSA), Washington University in Saint Louis (McCutcheon and Luke, Co-PIs), and by the National Institute on Alcohol Abuse and Alcoholism (AA018146, McCutcheon, PI) and the National Institute on Drug Abuse (DA027046, Lessov-Schlaggar, PI). We thank the women who participated in this study for sharing their experiences and the treatment center directors and staff who made their participation possible. We also thank Nicole Werner, PhD, for her assistance with neuropsychological testing and Rose Denness, MFA, for her patient assistance with tracking, scheduling, and interviewing.

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Figure 1.

Illustration of interaction in Table 2. Plotted lines show change in disinhibition from baseline to follow-up as a function of change in network drinking score, and markers at time 1 and time 2 represent individual levels of disinhibition at each time point, adjusted for IQ, relapse, self-reported urgency, and follow-up status. Women whose network drinking score increased showed an increase in disinhibition on average. Women whose network drinking score decreased showed a decrease in disinhibition, on average.

Table 1.

Sample and social network characteristics and univariate associations with baseline disinhibition.

	Association with baseline disinhibition	
	M (SD) or %	B (95% CI)
Sample characteristics		
Demographics		
Age, M (SD)	42.3 (9.5)	-.09 (-.59, .40)
IQ, M (SD) *	91.7 (15.7)	-.30 (-.58, -.02)
African American, %	50.0	1.4 (-7.9, 10.8)
Years Education, M (SD)	12.2 (2.2)	-.02 (-2.3, 2.2)
Marital status		
Married	10.7	13.6 (-4.3, 31.6)
Separated/Divorced	42.9	3.7 (-5.7, 13.1)
Never married ^R	46.4	1.0
Number biological children, M (SD)	2.4 (1.6)	1.6 (-1.3, 4.6)
Unemployed	85.7	-6.12 (-19.0, 6.8)
Any days homeless in last 90 days, %	32.1	-6.5 (-16.4, 3.4)
Childhood trauma		
Childhood rape or molestation before age 13, %	28.6	8.9 (-.64, 18.5)
Childhood physical abuse before age 13, %	35.7	4.3 (-5.2, 13.8)
Substance Use		
Alcohol Use Disorder, lifetime, %	100.0	inestimable
AUD symptoms (range 0–11), M (SD) *	9.1 (2.2)	-2.6 (-4.4, -.83)
Marijuana Use Disorder, lifetime, %	50.0	4.8 (-4.3, 14.0)
Cocaine Dependence, lifetime, %	64.3	-4.1 (-13.7, 5.4)
Opiate Dependence, lifetime, %	17.9	1.8 (-10.2, 13.8)
Daily nicotine use, last 90 days, %	67.9	-6.2 (-16.1, 3.7)
Any illicit drug use, last 90 days, %	60.7	-1.4 (-11.1, 8.2)
Proportion heavy drinking days, last 90 days, M (SD)	.56 (.36)	-3.6 (-16.8, 9.5)
Days since last drink, M (SD) *	27.7 (10.2)	-.55 (-.95, -.15)
Psychiatric Disorders		
Major Depression, lifetime, %	60.8	-.67 (-10.3, 9.0)
Major Depression, current, %	50.0	-.19 (-9.5, 9.2)
PTSD, lifetime, %	35.7	4.3 (-5.2, 13.8)
Panic Disorder, lifetime, %	10.7	12.6 (-1.3, 26.6)
ASPD symptoms (range 1–7), M (SD)	5.4 (1.1)	-1.9 (-6.3, 2.4)
Treatment		
Number inpatient treatments for substance or psychiatric problems, M (SD)	6.6 (6.0)	.35 (-.43, 1.1)
Treated for drinking problem before now, % *	71.4	-10.5 (-19.8, -1.2)
Self-reported urgency, M (SD) ^{I*}	25.7 (6.5)	.95 (.28, 1.6)

Association with baseline disinhibition		
	M (SD) or %	B (95% CI)
Sample characteristics		
Network characteristics		
Network drinking score (range 1–5), M (SD) *	2.5 (.62)	11.8 (6.0, 17.6)
Drinking status of network members, as percent of network, (%)		
Recovering *	25.9	–22.8 (–39.2, –6.4)
Lifetime abstainer	31.1	1.9 (–17.0, 20.9)
Light drinker	22.1	6.8 (–17.2, 31.0)
Moderate drinker	10.8	16.4 (–19.2, 51.9)
Heavy drinker *	9.7	33.0 (3.6, 62.5)
Relationship of network members, as percent of network, (%)		
Family members *	53.5	30.1 (13.0, 47.3)
Friends *	26.3	–21.1 (–37.9, –4.3)
Professionals	20.2	–7.5 (–37.7, 22.6)

NOTE:

* variable associated with baseline disinhibition at $p < .05$;

R reference category

I urgency had possible range of 12 to 48, with higher scores representing greater urgency.

Table 2.

Mixed model regression of disinhibition on network drinking score

	Model 1			Model 2		
	β (95% CI)	SE	p-value	β (95% CI)	SE	p-value
Network drinking score	6.7 (3.3, 10.1)	1.7	.000	9.1 (5.2, 13.0)	2.0	.000
Time	-4.0 (-6.9, -1.1)	1.5	.007	8.6 (-3.0, 20.2)	5.9	.145
IQ	-.37 (-.55, -.21)	.08	.000	-.37 (-.53, -.21)	.08	.000
Relapsed	8.3 (1.6, 15.1)	3.4	.016	8.3 (1.9, 14.7)	3.3	.011
Self-reported urgency	.32 (-.03, .66)	.17	.070	.37 (.04, .70)	.17	.030
Lost to follow-up	-3.6 (-9.8, 2.6)	3.2	.256	-2.5 (-8.5, 3.4)	3.0	.408
Network drinking score X Time				-5.4 (-10.1, -.59)	2.4	.028
Model Fit	Parameters	AIC	BIC	Parameters	AIC	BIC
	9	307.21	323.27	10	304.61	322.46