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Drinking motives as prospective predictors of outcome in an intervention trial among heavily drinking HIV patients

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Abstract

BACKGROUND—Heavy alcohol consumption in HIV patients is an increasing health concern. Applying the drinking motivational model to HIV primary care patients, drinking motives (drinking to cope with negative affect, for social facilitation, and in response to social pressure) were associated with alcohol consumption at a baseline interview. However, whether these motives predict continued heavy drinking or alcohol dependence in this population is unknown.

METHODS—Participants were 254 heavy-drinking urban HIV primary care patients (78.0% male; 94.5% African American or Hispanic) participating in a randomized trial of brief drinking-reduction interventions. Drinking motive scales, as well as measures of alcohol consumption and alcohol dependence, were administered at baseline. Consumption and dependence measures were re-administered at the end of treatment two months later. Regression analyses tested whether baseline drinking motive scale scores predicted continued heavy drinking and alcohol dependence status at the end of treatment, and whether motives interacted with treatment condition.

RESULTS—Baseline drinking to cope with negative affect predicted continued heavy drinking (p<0.05) and alcohol dependence, the latter in both in the full sample (adjusted odds ratio [AOR]=2.14) and among those with baseline dependence (AOR=2.52). Motives did not interact with treatment condition in predicting alcohol outcomes.

CONCLUSIONS—Drinking to cope with negative affect may identify HIV patients needing targeted intervention to reduce drinking, and may inform development of more effective interventions addressing ways other than heavy drinking to cope with negative affect.

Keywords

HIV; AIDS; drinking; alcohol; prediction; motives

1. Introduction

Over 1.1 million individuals in the US are infected with HIV (Centers for Disease Control and Prevention, 2012). Maintaining low levels of alcohol consumption is highly relevant to

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maintaining the health of these HIV-infected individuals, for several reasons. HIV-infected individuals with heavy drinking or alcohol dependence may experience lowered immune functioning (Shuper et al., 2010) and higher viral loads (Hahn and Samet, 2010). Liver disease is also a significant contributor to mortality among HIV-infected individuals (Antiretroviral Therapy Cohort, 2010; Palella et al., 2006), due to high rates of Hepatitis B and C, antiretroviral (ART) medication hepatotoxicity, and alcohol consumption (Barve et al., 2010). However, high rates of alcohol use disorders among HIV-infected individuals dying of liver disease suggest considerable contribution of alcohol use to liver disease in this population (DeLorenze et al., 2011). Additionally, providers are sometimes reluctant to provide ART medication to patients with alcohol use disorders (Loughlin et al., 2004), and when they are prescribed, these patients have lower adherence (Azar et al., 2010). Due to these and other consequences of heavy alcohol involvement among those with HIV, better understanding of who is most at risk for prolonged heavy drinking and alcohol dependence in this population is important.

Relatively little is known about predictors of heavy alcohol involvement among individuals with HIV. Clinical trials with drinking-reduction outcomes in HIV populations show varied results (Meade et al., 2010; Papas et al., 2011; Parsons et al., 2007; Rotheram-Borus et al., 2009; Samet et al., 2005; Velasquez et al., 2009; Wong et al., 2008), with no consistent relationship between study design and drinking outcome, demonstrating that we do not yet understand what precedes changes in drinking. In cross-sectional observational studies, correlates of drinking include stressful experiences, depressive symptoms, poor coping strategies, and low self-efficacy (Longmire-Avital et al., 2012; Pence et al., 2008). A longitudinal study of women with HIV replicated depression as a prospective predictor of drinking (Cook et al., 2009; Cook et al., 2012). However, given the limited knowledge base on predictors of heavy drinking and alcohol dependence status over time in HIV samples, more information is needed, particularly from samples with both genders.

The motivational model of alcohol use may help identify predictors of persistent heavy alcohol involvement among individuals with HIV. This theory posits that alcohol consumption is driven by the anticipated benefits of drinking, which may include decreasing negative states or achieving positive effects (Cox and Klinger, 1988). Commonly studied motives address coping with unpleasant emotions (e.g., depressed affect, anxiety), enhancement of positive feelings, and responding to social situations (Kuntsche et al., 2005). Drinking motives (or reasons for drinking) predict alcohol consumption in general population samples, as shown by cross-sectional (Abbey et al., 1993; Carpenter and Hasin, 1998c; Mezquita et al., 2011; Tragesser et al., 2007; Trocki and Drabble, 2008) and prospective (Beseler et al., 2011; Crutzen et al., 2012; Tragesser et al., 2007) studies. Drinking motives also predict alcohol dependence status/symptoms, in both cross-sectional (Tragesser et al., 2007; Trocki and Drabble, 2008) and prospective (Beseler et al., 2007; Trocki and Drabble, 2008) and prospective (Beseler et al., 2007; Trocki and Drabble, 2008) and prospective (Beseler et al., 2007; Trocki and Drabble, 2008) and prospective (Beseler et al., 2007; Trocki and Drabble, 2008) and prospective (Beseler et al., 2007; Trocki and Drabble, 2008) and prospective (Beseler et al., 2007; Trocki and Drabble, 2008) and prospective (Beseler et al., 2007; Trocki and Drabble, 2008) and prospective (Beseler et al., 2008; Carpenter and Hasin, 1998b; Tragesser et al., 2007) community samples.

Recently, we evaluated *cross-sectional* associations between drinking motives and alcohol consumption in a heavily drinking HIV primary care sample recruited to participate in a drinking-reduction randomized trial (Elliott et al., under review). Three of the four original subscales from the Reasons for Drinking Scale (Carpenter and Hasin, 1998c) had clear factor structure and good internal consistency in this sample: (a) drinking to cope with negative affect (e.g., sadness, boredom, irritability), (b) drinking for social facilitation, and (c) drinking in response to social pressure. These motives were associated with many aspects of past-year drinking measured at baseline: coping with negative affect was associated with higher levels of all drinking and heavy drinking measures, drinking for social facilitation was associated with more frequent drinking to intoxication, and drinking in response to social pressure was associated with lower drinking quantity and binge

frequency. Although identifying cross-sectional correlates of heavy drinking in HIV patients is informative, identifying predictors of *continued*, *persistent* heavy drinking or alcohol dependence may be particularly important to improve clinical practice. Determining predictors of *drinking despite intervention* may help identify the most robust drinking predictors. Given the cross-sectional associations we found, we hypothesized that drinking motives would also predict *continued* heavy drinking and alcohol dependence symptoms in this heavily drinking HIV-infected sample, even despite participation in a drinking reduction trial. Drinking to cope with negative affect was a drinking motive of particular interest, given elevated rates of depression among individuals with HIV (Bing et al., 2001; Ciesla and Roberts, 2001; Zanjani et al., 2007), and studies showing that depression itself predicts later drinking in HIV-infected women (Cook et al., 2009; Cook et al., 2012). An additional question of interest was whether drinking motives would be differentially predictive among patients receiving different types of drinking-reduction interventions.

The present study therefore aimed to determine if drinking motives predict continued heavy alcohol consumption and alcohol dependence despite intervention among heavy-drinking HIV patients, and to determine if the effects of motives differed by treatment type. To do this, we conducted additional analysis of data from a randomized trial of brief drinkingreduction interventions with urban minority HIV primary care patients (Hasin et al., in press), also used for the cross-sectional validation of the scale (Elliott et al., under review). In this study, patients were randomized to one of three conditions, all involving brief (20–25 min) sessions at baseline and briefer (5–10 min) sessions at 30 and 60 days. One condition was a Motivational Interview (MI) session. Another was the MI session plus HealthCall (MI +HealthCall), which involved daily self-monitoring (2–3 min) via automated telephone interactive voice response technology with personalized feedback from the self-monitoring data provided at 30 and 60 days. The third, an attentional control condition, consisted of advice to reduce drinking and a video on HIV self-care without alcohol content. Although all groups reduced drinking, patients receiving MI+HealthCall had significantly greater drinking reduction than others (Hasin et al., in press). In the current study, we examined three questions. First, do baseline motives predict whether these heavily drinking patients continued to drink heavily at end-of-treatment (i.e., do drinking motives predict continued heavy drinking despite intervention)? Second, do baseline motives predict alcohol dependence status at end-of-treatment? Third, do the effects of motives differ by treatment condition?

2. Methods

2.1. Participants

Participants were 254 HIV-infected patients recruited between 2007 and 2010 from a large urban HIV primary care clinic for a randomized trial of the comparative efficacy of brief alcohol interventions (Hasin et al., in press). Eligibility required at least one heavy drinking occasion (four or more drinks on one occasion) in the prior month. As described elsewhere (Elliott et al., under review), patients ranged in age from 22 to 68 (M = 45.7; SD = 8.1); most were male (78.0%), African American (49.6%) or Hispanic (44.9%), and had completed at least high school or a graduate equivalency degree (58.1%). Patients first received their HIV diagnosis on average 12.8 (SD = 7.6) years prior, and 77.1% were on ART medication.

Of the 254 patients completing a baseline assessment, 240 also provided data on drinking at the end of treatment two months later (including 51 who missed the 60-day assessment but provided data retrospectively), and 189 provided 60-day information on alcohol dependence (no retrospective data available). Completion of on-time follow-up data did not differ by age, ethnicity, gender, education, language of study completion, HIV medication status, or

years since HIV diagnosis (ps>0.20). Completion of on-time follow-up was also unrelated to baseline and 60-day drinking quantity and frequency (ps>0.05).

2.2. Measures

Drinking motives—Drinking motives were assessed at baseline using the Reasons for Drinking Scale (RDS) (Carpenter and Hasin, 1998c). In the previous study of drinking motives in this sample (Elliott et al., under review), a three-factor model was found. The subscales demonstrated good internal consistency and concurrent validity with drinking measures: coping with negative affect ("COPE"; 6 items; e.g., "I drink to avoid sadness or depression"; α =0.78;), drinking for social facilitation ("SOCIAL-FACILITATION"; 7 items; e.g., "Drinking makes me more outgoing with other people"; α =0.88), and drinking in response to social pressure ("SOCIAL-PRESSURE"; 6 items; e.g., "I drink because my friends expect me to drink when we get together"; α =0.85) (Elliott et al., under review). Participants rated the items from these subscales on a five-point scale, scored as agree strongly to disagree strongly (in presented results, 1=disagree strongly; 5=agree strongly). We utilized average (as opposed to sum) scale scores for ease of interpretation. As reported previously, correlations between subscales ranged from r = 0.32-0.47 (Elliott et al., under review).

Alcohol consumption—Patients reported on alcohol consumption at baseline and endof-treatment (60-days later) using 30-day TimeLine FollowBacks (TLFB) (Sobell, 1995). With these data, quantity and frequency of drinking were summarized as the mean number of drinks per drinking day, and percentage of days abstinent, as examined in the comparative efficacy study (Hasin et al, in press). If patients missed the 60-day appointment but attended later follow-ups, drinking data provided retrospectively were used, as extensive examination of on-time and retrospectively-reported 60-day data showed virtually no differences (Hasin et al., in press).

Alcohol dependence—Alcohol dependence was assessed using the Alcohol Use Disorder and Associated Disabilities Interview Schedule (AUDADIS), a reliable and valid instrument (Canino et al., 1999; Grant et al., 2003; Grant et al., 1995; Hasin et al., 1997; Hasin et al., 2006; Hasin et al., 2007) that assesses dependence symptoms from the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) (American Psychiatric Association, 1994). Patients were assessed for past-year symptoms at baseline, and pastmonth symptoms at 60 days (end-of-intervention). The main dependence outcome in the present study was dependence status, considered positive when three criteria in the assessed time frame were endorsed (American Psychiatric Association, 1994). Supplemental analyses were also done on the number of dependence criteria endorsed.

Demographic and HIV information—Consistent with previous research (Elliott et al., under review), we controlled for age, ethnicity, gender, highest level of education, preferred language for participation (Spanish or English), HIV medication status, and number of years since HIV diagnosis.

Treatment condition—Treatment condition was used as a control variable and as a potential moderator of motive effects on outcome.

2.3. Data Analysis

First, we assessed whether drinking motives predicted continued heavy drinking. Generalized linear models tested whether each motive predicted 60-day drinking quantity (drinks per drinking day), using SAS "proc genmod" (SAS Institute Inc, 2011). A separate model was run for each motive, specifying a negative binomial distribution to suit the

distribution of the drinking outcome, and including relevant control variables (intervention condition, baseline drinking quantity, age, ethnicity, gender, education, preferred language, HIV medication status, years since HIV diagnosis). A similar procedure was conducted for 60-day drinking frequency (percent days abstinent), again using negative binomial regression models, and including the same control variables (except changing baseline quantity to baseline frequency). For descriptive purposes, baseline and 60-day drinking (quantity and frequency) are summarized for individuals scoring (a) at or below the median and (b) above the median on each motive scale.

Second, we assessed predictive models for alcohol dependence. Logistic regressions were run to determine if the drinking motives predicted alcohol dependence status at 60 days, using SAS "proc logistic" (SAS Institute Inc, 2011). We tested a separate model for each of the three motives, controlling for the same variables listed above except that baseline past-year dependence status was controlled rather than consumption. Baseline and 60-day dependence prevalence is presented for those (a) at or below the median and (b) above the median on all motive scales. We also examined whether motives predicted the number of dependence symptoms at follow-up using SAS "proc genmod," specifying a negative binomial distribution. In these models, we used the same control variables as in the logistic regressions except that we controlled for number of baseline symptoms instead of baseline diagnosis. Next, we repeated the logistic regressions among the subgroup of patients with baseline past-year dependence to determine whether motives predicted dependence *persistence*. In these models, all control variables except baseline diagnosis were retained.

Third, we ran analyses to determine whether motives were differentially predictive for the different intervention conditions. To do this, we added a motive-by-condition interaction term to the above models and re-ran them to assess interaction.

3. Results

3.1. Drinking patterns

At baseline, participants drank a mean of 6.98 (SD = 3.83) drinks per drinking day in the prior 30 days. Their percentage of days abstinent ranged from 0% to 96.67% (M = 68.10, SD = 24.31). Almost half of the sample (48.22%) met criteria for past-year alcohol dependence at baseline. Scores on the COPE scale ranged from 1.00-5.00, with a mean score of 3.06 (SD=0.99) and a median score of 3.17 (for analyses contrasting low and high scorers, 134 participants scored at or below the median; 119 above). Scores on the SOCIAL-FACILITATION scale ranged from 1.00-5.00, with a mean score of 3.57 (131 at or below the median; 122 above). Scores on the SOCIAL-PRESSURE scale ranged from 1.00-5.00, with a mean score of 2.00 (SD=0.87) and a median score of 2.00 (I57 at or below the median; 96 above).

At 60 days (end-of-treatment), 172 of the 240 responding participants (71.67%) drank in the prior 30 days, and they drank an average of 4.12 (SD = 2.76) drinks per drinking day. The percent days abstinent ranged from 0% to 100% (M = 83.60, SD = 21.08). When assessed for past-month dependence criteria at end-of-treatment, 13.23% met full criteria.

3.2. Predictive models

Baseline COPE predicted number of drinks per drinking day at 60 days (end-of-treatment) (B=0.14, SE=0.05, 95% CI: 0.05, 0.23). Individuals scoring above the median on COPE reported drinking over one drink more per drinking day than those at or below the median (Table 1). Drinks per drinking day was not predicted by SOCIAL-FACILITATION (B=0.01, SE=0.04, 95% CI=-0.07, 0.09) or SOCIAL-PRESSURE (B=-0.01, SE=0.05, 95%

CI: -0.11, 0.09). None of the drinking motives predicted percent days abstinent at end-ofintervention (COPE: B=-0.03, SE=0.02, 95% CI=-0.08, 0.01; SOCIAL-FACILITATION: B=-0.02, SE=0.02, 95% CI: -0.06, 0.02; SOCIAL-PRESSURE: B=-0.01, SE=0.03, 95% CI: -0.06, 0.04).

Of the 189 patients with full 60-day data, 25 met criteria for past-month dependence. Baseline COPE predicted alcohol dependence at 60 days (Adjusted Odds Ratio [AOR] = 2.14, 95% CI: 1.21, 3.77). Individuals scoring above the median on COPE had rates of dependence more than three times as great as those at or below the median (Table 2). Neither SOCIAL-FACILITATION (AOR=1.11; 95% CI: 0.72, 1.72) nor SOCIAL-PRESSURE (AOR=1.38; 95% CI: 0.86, 2.23) predicted alcohol dependence status. No motive variables predicted the number of dependence symptoms (COPE: B=0.14, SE=0.16, 95% CI=-0.17, 0.45; SOCIAL-FACILITATION: B=-0.02, SE=0.13, 95% CI: -0.27, 0.24; SOCIAL-PRESSURE: B=0.18, SE=0.16, 95% CI: -0.13, 0.48).

Among the 94 patients with baseline past-year dependence and full 60-day data, 19 met past-month dependence criteria at end-of-treatment. Continued alcohol dependence was predicted by baseline values on COPE (AOR=2.52, 95% CI: 1.23, 5.15), but not by SOCIAL-FACILITATION (AOR: 1.27; 95% CI: 0.73, 2.22) or SOCIAL-PRESSURE (AOR:1.43, 95% CI: 0.82, 2.49). Individuals scoring above the median on COPE were more than three times as likely to continue meeting criteria for alcohol dependence (27.1%) at end-of-treatment than those scoring at or below the median on COPE (8.3%).

Analyses adding a motive-by-condition interaction term yielded no significant interactions for alcohol consumption or alcohol dependence outcomes (ps>0.05).

4. Discussion

In this HIV primary care sample, we tested whether drinking motives assessed at baseline predicted continued heavy drinking and alcohol dependence status two months later, at the end of treatment in a randomized trial. Drinking to cope with negative affect predicted drinking quantity and DSM-IV alcohol dependence status, but not drinking frequency; drinking for social facilitation and in response to social pressure did not predict alcohol outcomes.

Results for the COPE motive are consistent with prospective community studies for drinking quantity (Beseler et al., 2011; Crutzen et al., 2012; Tragesser et al., 2007) and alcohol dependence status (Beseler et al., 2008; Carpenter and Hasin, 1998b; Tragesser et al., 2007). Although previous research on this HIV-infected sample supported associations of coping motives with alcohol consumption cross-sectionally (Elliott et al., under review), this is the first study that established prediction over time in an HIV-infected sample. When interpreted along with literature showing high rates of depression among those with HIV (Bing et al., 2001; Ciesla and Roberts, 2001; Zanjani et al., 2007), and associations between drinking and stressful experiences, depression, and poor coping strategies among individuals with HIV (Longmire-Avital et al., 2012; Pence et al., 2008), drinking to regulate varied negative psychological experiences clearly emerges as an important factor in understanding alcohol involvement in this population. However, it should be noted that COPE did not predict the number of dependence symptoms, only dependence status. This suggests that drinking to cope makes individuals more likely to cross the threshold into dependence, but does not necessarily account for incremental differences in the number of symptoms of those above or below the diagnostic threshold.

In the current study, SOCIAL-FACILITATION did not predict continued heavy alcohol involvement, in contrast with community studies that found this drinking motive to be

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predictive of later drinking (Beseler et al., 2011; Beseler et al., 2008; Crutzen et al., 2012). HIV-infected individuals may be more likely to use alcohol on an ongoing basis (and subsequently develop alcohol problems) in an effort to cope with negative emotions than in an effort to socialize, whereas alcohol involvement in the general population may stem from either motive. SOCIAL-PRESSURE also did not predict continued heavy drinking or dependence status in the current sample. This motive prospectively predicted alcohol involvement in one community study (Beseler et al., 2011) but not others (Beseler et al., 2008; Crutzen et al., 2012). Thus, although drinking in response to social pressure is associated with current drinking patterns among individuals with HIV (Elliott et al., under review), this may be because social pressure is a *consequence* of drinking less. This hypothesis, raised in the previous study (Elliott et al., under review), is consistent with the *lack of* prediction of subsequent drinking in the current study.

Drinking motives did not interact with treatment condition. This suggests that drinking to cope was related to continued heavy drinking and alcohol dependence status similarly across varying levels of intervention. The consistency of results is noteworthy given that patients in the different treatment conditions received interventions with varying relevance to drinking (patients in the control condition received drinking-reduction advice but no further alcohol content) and dose/intervention exposure (patients receiving MI+HealthCall made brief daily self-monitoring calls). Patients in the three treatment conditions *were* equivalent in demographics, HIV characteristics, and baseline alcohol involvement (Hasin et al., in press), consistent with successful randomization, allowing us to look across treatment groups. The consistent findings across conditions highlight the strength of the coping/drinking association.

Limitations of the study are noted. The sample is from one urban HIV primary care clinic in the northeastern United States, with primarily low-SES, male, and minority patients; further studies should determine if results would generalize to HIV-infected patients who are high-SES, female, White, and/or rural. However, our focus on patients from minority groups over-represented in the HIV epidemic supports generalizability of these findings. Also related to generalizability, the heavy drinking nature of the sample makes results most applicable to prediction of *continued* heavy alcohol involvement in already-heavy drinkers, and may not apply equally to the *development* of heavy drinking in lighter drinkers. However, heavily drinking adult samples have been used in previous research on the associations between motives and later drinking (Beseler et al., 2011; Beseler et al., 2008; Carpenter and Hasin, 1998a), and are arguably the most appropriate samples, as they have already begun the risky behavior in question. Further, this study tested prediction during an interval in which patients received interventions of varying dose and relevance; however, the consistency of the results across intervention conditions supports the robustness and generalizability of the present results. Other issues should also be considered in addition to generalizability. Some participants with missing drinking data at end-of-treatment provided drinking data retrospectively (n = 51). These data were examined extensively, and no significant differences were found between on-time and retrospective data, or the patients providing data at these two timepoints (Hasin et al., in press). Thus, including all data maximized the information available about alcohol consumption. Alcohol dependence information was not available retrospectively, leading to missing data. However, lack of significant associations between attrition and demographic, HIV, and drinking variables supported the assumption that this data was missing at random. Additionally, the sample size of the current study precluded some analyses. For example, new cases of dependence at 60 days were too few to test whether motives predicted development of alcohol dependence among those without baseline dependence. This question should be studied in future larger studies of drinking motives among individuals with HIV with longer follow-ups, which may help determine whether coping with negative affect is important in the development of

alcohol dependence. Finally, the much lower prevalence of dependence at end-of-treatment almost certainly reflects the different timeframes used to assess dependence at baseline and 60 days in addition to success of treatment. Rates of dependence should be considered in light of the one-year timeframe at baseline, and one-month timeframe at 60 days.

The present study also has several strengths. These include (a) the use of a theoretical model of drinking with considerable empirical evidence, (b) the use of well-validated measures, including the AUDADIS and the Reasons for Drinking Scale, and (c) the prediction of alcohol involvement at a later time, which provides important evidence of the temporality of associations that were previously only shown cross-sectionally in this population (Elliott et al., under review). This study also assessed interaction with treatment, suggesting robustness of the motive/drinking associations across different conditions. Also, findings of the current study contribute to two separate areas of research, including (a) the study of drinking motives, and (b) the study of drinking among individuals with HIV, a high-risk sample in which drinking can have serious medical consequences. Finally, the motives supported in the current study are in many ways good practical options for identifying patients at high risk for ongoing heavy alcohol involvement. These constructs could be easily incorporated into brief discussions about drinking (or even brief questionnaires); such content would be consistent with motivational interviewing approaches to intervening with substance use (i.e., decisional balance) as well as cognitive-behavioral approaches (e.g., identification of triggers). Motive assessment could help providers understand patients' behavior, avoid incorrect assumptions about patients' relationship with alcohol, and provide empathy. Further, if providers are aware that patients reporting coping motives are most at risk for ongoing heavy alcohol involvement, they could provide additional intervention to patients reporting high levels of these motives.

HIV-infected individuals are at high risk for medical consequences of alcohol use, making identification of factors underlying continued heavy drinking and alcohol dependence for these individuals of particular importance. This study showed that social motives were not predictive, but drinking to cope with negative affect was predictive of drinking quantity and alcohol dependence two months later. Drinking to alleviate psychological distress appears to be a powerful motivator that is closely linked to behavior, regardless of intervention administered. These findings highlight the importance of working with heavy-drinking HIV patients to help them develop strategies other than drinking to cope with distress and negative affect. In addition, studies of evidence-based interventions that add content specifically addressing coping with negative affect could lead to development of more effective treatments. Further, used in conjunction with a measure of current drinking or alcohol dependence, the COPE subscale of the RDS may be a useful tool to identify those at risk for chronic heavy drinking and alcohol dependence. Understanding the role of drinking motives among HIV-infected individuals is a promising area of research that may benefit the clinical care of HIV patients, and also the development of better interventions for those drinking in a manner that jeopardizes their health and survival.

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Table 1

Drinking quantity and frequency at baseline and 60-day appointment by level of baseline motive variables: Data from a large primary care clinic in the urban northeast, 2007–2010 (N=240).

	Drin	Drinks per drinking day: Unadjusted mean (SD)	lay: Unadjustet	1 mean (SD)		Per	Percent days abstinent: Unadjusted mean (SD)	nt: Unadjusted	mean (SD)	
Drinking motive	At/below media	At/below median motive score Above median motive score IRR	Above media	1 motive score	IRR		At/below median motive score Above median motive score	Above media	n motive score	IRR
	Baseline	60 days	Baseline	60 days		Baseline	60 days	Baseline	60 days	
To cope with negative affect	6.37 (3.50)	3.50 (2.00)	7.67 (4.09)	4.74 (3.25)	1.15^{*}	70.49 (22.69)	$7.67\ (4.09) \qquad 4.74\ (3.25) \qquad 1.15^* \qquad 70.49\ (22.69) \qquad 86.22\ (16.51) \qquad 65.38\ (25.85) \qquad 80.70\ (24.95) \qquad 0.97$	65.38 (25.85)	80.70 (24.95)	0.97
For social facilitation	6.57 (3.57)	4.13 (2.85)	7.43 (4.07)	4.11 (2.69)	1.01	4.11 (2.69) 1.01 68.51 (24.28)	86.05 (17.94) 67.65 (24.43) 80.98 (23.79) 0.98	67.65 (24.43)	80.98 (23.79)	0.98
In response to social pressure	6.95 (3.77)	4.14 (2.74)	7.04 (3.96)	4.10 (2.81)	0.99	66.33 (25.31)	7.04 (3.96) 4.10 (2.81) 0.99 66.33 (25.31) 83.06 (20.79) 71.01 (22.38) 84.47 (21.64) 0.99	71.01 (22.38)	84.47 (21.64)	0.99

els testing whether (continuous) baseline motives predict drinking at 60 days, using generalized linear models controlling for intervention condition, baseline drinking, age, ethnicity, gender, education, preferred language, HIV medication status, and the number of years since HIV diagnosis.

* indicates significance at p < 0.01; the risk ratio of 1.15 can be interpreted such that each unit increase in the cope motive is associated with a 15% increase in drinking rate.

Table 2

Alcohol dependence rates at baseline and 60-day appointment by level of baseline motive variables: Data from a large primary care clinic in the urban northeast, 2007-2010 (N=187).

	Percent of the sample			
Drinking motive	At/below median motive score		Above median motive score	
	Baseline	60 days	Baseline	60 days
To cope with negative affect	33.6	5.9	64.7	21.8*
For social facilitation	41.2	10.9	55.7	15.5
In response to social pressure	44.6	12.3	54.2	14.7

Note. Higher values indicate higher rates of dependence.

indicates that (continuous) baseline motives predict dependence at 60 days, using logistic regressions controlling for intervention condition, baseline dependence, age, ethnicity, gender, education, preferred language, HIV medication status, and the number of years since HIV diagnosis (p < 0.05).