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Post-treatment drinking among HIV patients: Relationship to pre-treatment marijuana and cocaine use

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Abstract

BACKGROUND—For individuals with HIV, heavy drinking can pose serious threats to health. Some interventions are effective at reducing drinking in this population, but many HIV-infected heavy drinkers also use marijuana or cocaine. Although these drugs have predicted poor alcohol outcomes in other treatment studies, whether this occurs among HIV patients who drink heavily is unknown.

METHODS—Participants were binge-drinking HIV primary care patients (N=254) enrolled in a randomized trial of three brief drinking interventions over 60 days that varied in intensity. We investigated the relationship of baseline past-year drug use (marijuana-only, cocaine-only, both, neither) to end-of-treatment drinking quantity and frequency. We also evaluated whether the relationship between intervention type and end-of-treatment drinking varied by baseline drug use. Final models incorporated control for patients' demographic and HIV characteristics.

RESULTS—In final models, drinking frequency at the end of treatment did not vary by baseline drug use, but drinking quantity did (X^2 [3] = 13.87, $p < 0.01$), with individuals using cocaine-only drinking significantly more per occasion ($B = 0.32$, $p < 0.01$). Baseline drug use also interacted with intervention condition in predicting end-of-treatment drinking quantity (X^2 [6] = 13.98, $p < 0.05$), but not frequency, with the largest discrepancies in end-of-treatment drinks per drinking day by intervention intensity among cocaine-only patients.

CONCLUSIONS—In general, HIV patients using cocaine evidenced the highest levels of drinking after alcohol intervention. However, these individuals also evidenced the most pronounced differences in end-of-treatment drinking by intervention intensity. These results suggest the importance of more intensive intervention for individuals using alcohol and cocaine.

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Keywords

HIV; alcohol; drinking; cocaine; marijuana; brief intervention

1. Introduction

Approximately one in five (18.5%) individuals with HIV report heavy or frequent heavy drinking within the past month (Bing et al., 2001). Heavy drinking is dangerous among individuals infected with HIV, as it can contribute to liver damage (Barve et al., 2010), interfere with medication adherence (Azar et al., 2010), and may compromise immune function (Hahn and Samet, 2010). A wide range of outpatient interventions (targeting alcohol alone or in conjunction with other health behaviors) have been designed to reduce the drinking of individuals with HIV (as reviewed by Brown et al., 2013; Samet and Walley, 2010). Of interventions designed for heavy drinkers in particular, some (targeting alcohol alone or with sexual risk or medication adherence; consisting of outpatient motivational interviewing or cognitive-behavioral treatment) have shown efficacy (Aharonovich et al., 2006; Hasin et al., 2013; Papas et al., 2011; Velasquez et al., 2009). However, half of HIV-infected individuals also report past-year illicit drug use (Bing et al., 2001), and no studies known to us have evaluated whether drug use relates to drinking after alcohol intervention for individuals with HIV. Given the increasing recognition of the need to reduce alcohol use due to alcohol's serious threats to health among HIV patients (Bryant et al., 2010), whether drug use affects the efficacy of these alcohol interventions in this group is an important question.

Some previous studies in *non-HIV* populations suggest that individuals using cocaine and marijuana have worse drinking outcomes after alcohol or combined alcohol/drug treatment. For example, when compared with drinkers not using cocaine, comorbid cocaine users show poorer drinking outcome following outpatient alcohol treatment (Aguilar et al., 2012). Regarding marijuana's effects, Aharonovich et al. (2005) showed that marijuana use after discharge from inpatient substance abuse treatment was associated with higher likelihood of relapse to alcohol use. Similarly, Mojarrad et al. (2014) found that baseline marijuana use reduced substance abstinence after outpatient substance abuse treatment, with no difference in effect by dependence type (i.e., alcohol, drugs, both). That comorbid drug use is associated with less successful alcohol outcomes following treatment in several studies may reflect drug interference with engagement in treatment (Mitchell and Selmes, 2007), or drug-related impairments in ability to manage drinking behavior (e.g., impairment in self-regulation of impulses and cravings; Pani et al., 2010). Alternately, comorbid alcohol and drug users may simply represent more severe and treatment resistant substance abusers. Regardless of the reason, comorbid drug use appears to be a prognostic indicator of poor alcohol treatment outcomes. Yet, no studies to date have evaluated these associations among individuals with HIV, who warrant separate study due to their high rates of drug involvement (Bing et al., 2001), increased harmful medical consequences of drinking (Azar et al., 2010; Barve et al., 2010; Hahn and Samet, 2010), and increased likelihood of using marijuana for non-recreational (i.e., medical) purposes (Cinti, 2009).

We therefore investigated the association between baseline drug use and drinking after brief alcohol intervention among heavy drinkers with HIV, using data from a recent alcohol intervention trial in HIV primary care. This large, randomized clinical trial compared the efficacy of a brief educational intervention, a brief motivational interviewing (MI) intervention, and an intervention consisting of MI plus HealthCall. HealthCall involved patient daily use of interactive voice response (IVR) technology to self-monitor alcohol- and other health- related behaviors, with call data summarized in personalized feedback graphs presented to patients by their MI counselors at 30 and 60 days to facilitate brief discussions of patient drinking (Hasin et al., 2013). The trial showed that although participants in all conditions reduced drinking, those in the MI+HealthCall condition demonstrated the largest reduction (Hasin et al., 2013). However, many patients in this trial reported comorbid drug use (cocaine: 33%; marijuana: 32%; Elliott et al., 2014a), whose relationship to drinking outcomes was unknown. Thus, we examined the relationship of baseline drug use to end-of-treatment drinking in two sets of analyses. First, we assessed whether baseline past-year drug use was generally associated with differences in drinking at end-of-treatment, regardless of intervention condition. Second, we determined whether the use of these drugs interacted with intervention condition in predicting end-of-treatment drinking.

2. Method

2.1 Participants and procedures

Participants were 254 HIV-infected patients referred by their HIV primary care providers for a randomized intervention trial to reduce drinking (Hasin et al., 2013). The interventions focused on drinking and HIV self-care more generally, and did not specifically target drug use. Inclusion criteria required at least one episode of past month binge drinking (four or more drinks on an occasion) (Hasin et al., 2013). All patients provided informed consent, and the study protocol was approved by institutional review boards at Columbia University, St. Vincent's Hospital, and Mt. Sinai Medical Center. Of the 254 participants, 88 patients completed the DVD educational control condition, 82 completed MI-only, and 84 completed MI+HealthCall (Hasin et al., 2013).

As reported previously, the sample was on average 45.7 years of age (s.d. = 8.1; range = 22–68), and mostly male (78%), minority (94.5%, including 49.6% African American and 44.9% Hispanic), and high school graduates (58.1%) (Elliott et al., 2014b). At baseline, patients drank on an average of 31.90% (SD=24.31) of the prior 30 days, consuming an average of 6.98 (SD = 3.83) drinks per drinking day (Elliott et al., 2014c).

2.2. Intervention conditions

2.2.1. Motivational Interviewing (MI) only—At baseline, patients completed a 20–25 minute individual MI session with a study counselor regarding their alcohol use. Patients were encouraged to set a drinking-reduction goal by the end of the session. Patients then received brief (~10 minute) booster sessions 30 and 60 days later, where progress was reviewed and drinking-reduction goals were re-evaluated.

2.2.2. MI+HealthCall—At baseline, patients completed the same MI session as the MI-only group. Following this session, they were introduced to HealthCall, and asked to use this program daily for 60 days. HealthCall consisted of 60 days of patient IVR calls (1–3 minutes each) to self-monitor alcohol- and other health-related behaviors. Call data were summarized in personalized feedback graphs presented to patients by their MI counselors at 30 and 60 days to facilitate brief (~10 minute) discussions of patients' drinking.

2.2.3. Educational session (control)—At baseline, patients were told by the study counselor that they drank more than was medically advisable, and were shown a 30-minute DVD on HIV self-care that did not address alcohol. At the end of the session, the study counselor provided patients with a pamphlet on drinking reduction. At 30 and 60 days, patients met with the study counselor again briefly, at which time patients reported on their drinking, and received continued advice to decrease drinking.

2.3. Measures

2.3.1. Alcohol consumption—At baseline and end-of-treatment (60 days post-baseline), drinking quantity and frequency in the last 30 days were measured with the TimeLine FollowBack (TLFB) (Sobell, 1995). This measure has shown good reliability in past research (quantity: $r_s=0.95-0.98$; frequency: $r_s=0.79-0.96$; Sobell et al., 1979; Sobell et al., 1988; Sobell et al., 1986). For the current study, we analyzed drinks per drinking day (quantity) and percent days abstinent (frequency) variables calculated from the TLFB, consistent with prior work with this sample (Elliott et al., 2014c; Hasin et al., 2013). If patients missed the 60-day appointment but attended later follow-ups, a retrospective report of drinking for this timeframe was used, as such data did not differ from on-time data (Hasin et al., 2013).

2.3.2. Drug use—Past-year use of marijuana and cocaine (each considered positive or negative) was assessed at baseline, using the Alcohol Use Disorder and Associated Disabilities Interview Schedule (AUDADIS). This measure has demonstrated good reliability in past research ($r_s=0.77-0.86$; Grant et al., 1995; Hasin et al., 1997). To assess the effects of these substances alone and in combination, we created a combined four-category variable representing drug use status (categories: neither substance, marijuana only, cocaine only, both marijuana and cocaine).

2.3.3. Demographic and HIV characteristics—Patients reported their age (in years), gender (male, female), race (African American / Black, Hispanic, White; others not endorsed), and education (fourteen ordered levels ranging from “no formal schooling” to “completed graduate or professional degree”). The language in which participants chose to complete the study was recorded (English, Spanish). Participants also reported their current HIV medication status (positive or negative) and time since HIV diagnosis (in years). These demographic and HIV variables were used as control covariates as specified below, consistent with prior work in this sample (Elliott et al., 2014a; Elliott et al., 2014b, c; Elliott et al., in press) and due to previously documented associations between many of these variables with alcohol consumption (Elliott et al., 2014b).

2.4. Analysis plan

First, we tabulated the number of participants with past year marijuana-only use, cocaine-only use, use of both substances, and use of neither substance. We tested whether drug use distribution differed by intervention condition using chi-squared tests.

Second, a generalized linear model was used to determine the association between past-year drug use and end-of-treatment drinks per drinking day. The model specified drug use as the predictor, end-of-treatment drinks per drinking day as the outcome, and intervention condition and baseline drinks per drinking day as control covariates. An additional model was then conducted that also included demographic and HIV control covariates: gender, age, ethnicity, education, language of study participation, HIV medication status, and years since HIV diagnosis. We conducted similar models for drinking frequency, replacing drinks per drinking day with percent days abstinent. All models specified the drinking variables' negative binomial distributions.

Third, we evaluated potential interactions between intervention condition and past-year drug use status in predicting end-of-treatment drinking. Specifically, the above models were modified to specify drug use, intervention condition, and their (additive) interaction as primary predictors, end-of-treatment drinking as the outcome, and baseline drinking as a control covariate. Models were conducted with and without control for demographic and HIV covariates. In the event of statistical difficulties with additive models (e.g., convergence problems), we replaced negative binomial models with normal linear regression models, using Huber white heteroskedastic error estimation.

3. Results

Of the 254 total participants, 41 reported past-year marijuana use only at baseline, 42 reported past-year cocaine use only, 41 reported past-year use of both, and 130 reported neither. Past-year drug use did not differ by intervention condition, $X^2(6) = 2.23, p=0.90$.

Past-year drug use predicted drinks per drinking day at end-of-treatment across intervention conditions, $X^2(3) = 14.07, p<0.01$ (Table 1). Specifically, compared with those who used neither drug, those who used cocaine only drank more at end-of-treatment ($B=0.32$ [presented Betas are in log scale], $p<0.01$). Those using marijuana only drank the least at end-of-treatment (see Figure 1), but their drinking level was not significantly lower than those using neither substance ($B=-0.20, p=0.11$). When models were repeated incorporating additional control for demographic and HIV covariates, past-year drug use remained a significant predictor of end-of-treatment drinks per drinking day, $X^2(3) = 13.87, p<0.01$, again with past-year cocaine-only users drinking more ($B=0.32, p<0.01$). Past-year drug use did not predict percent days abstinent at end-of-treatment in the initial model, $X^2(3) = 4.60, p=0.20$, or when all control covariates were included, $X^2(3) = 3.14, p=0.37$.

Past-year drug use status evidenced a marginal interaction with intervention condition in predicting drinks per drinking day at end-of-treatment ($X^2[6] = 11.46, p=0.08$). Graphical representation (Figure 2) suggested that this was a meaningful differential response, and when demographic and HIV covariates were controlled, this interaction became significant

(X^2 [6] = 13.98, $p < 0.05$). The model indicated that, compared with those who did not use either drug, those who used cocaine only evidenced a more substantial response to MI+HealthCall versus educational control ($p < 0.05$; Table 2). The percent days abstinent variable necessitated use of linear regression models, which indicated no interaction between past-year drug use and intervention condition in the initial model (X^2 [6] = 6.63, $p = 0.36$) or in the model with demographic and HIV control covariates included (X^2 [6] = 6.93, $p = 0.33$).

4. Discussion

In this sample of heavily drinking HIV patients, baseline use of cocaine was generally associated with higher drinking after alcohol intervention. Baseline drug use status also interacted with intervention condition such that the largest reductions in response to MI-HealthCall (as compared the educational control condition) were also found among those using cocaine only. These findings were specific to drinking quantity, not frequency, and controlled for baseline drinking. In contrast, marijuana use was not a risk factor for continued heavy drinking.

Individuals who used cocaine-only in the past year reported the highest drinking at end-of-treatment, over two drinks higher than those in all other drug use categories, and significantly higher than the neither-drug comparison group. This relatively large discrepancy is likely to be meaningful in terms of higher medical consequences (e.g., liver damage) and more risk behaviors (e.g., sexual risk-taking, medication nonadherence) among those using cocaine. This finding is consistent with prior research showing worse drinking outcomes (Aguilar et al., 2012) and more craving (Fox et al., 2008) among alcohol-abusing patients who also abused cocaine. Although cocaine use has been shown to be generally associated with risky drinking in prior research (Borders and Booth, 2012), the relationship between cocaine use and end-of-treatment drinking was significant despite controlling for baseline drinking differences. The difficulty that cocaine-using patients had in *reducing* alcohol use could be due to continued use of alcohol to counteract cocaine effects (Magura and Rosenblum, 2000), to cocaine's interference with patients' involvement in the alcohol intervention, or to common co-factors (e.g., addiction severity, poor executive control) influencing both cocaine use and the ability to reduce drinking. These speculations should be investigated in future research, which would require a different design than the present study. In any case, cocaine users' continued heavy drinking despite intervention indicates the importance of screening for cocaine use during alcohol intervention and discussing how cocaine use may influence drinking. This finding also highlights the need for more intensive interventions (such as MI+HealthCall) for HIV patients using both alcohol and cocaine. However, interestingly, individuals using *both marijuana and cocaine* reported similar drinking at end-of-treatment to those who used neither substance, and at levels intermediate between the marijuana-only and cocaine-only groups.

Individuals who used marijuana-only in the past year did not differ from those who used neither marijuana or cocaine. That we did *not* find marijuana to be a risk factor for continued heavy drinking despite intervention is worth noting, as two other studies found marijuana to be associated with worse drinking outcomes (Aharonovich et al., 2005; Mojarrad et al., 2014). Several explanations may account for our finding. First, this study is unique in its

focus on HIV patients. As these patients are medically ill, some of them may have used marijuana in the last year to alleviate medical symptoms, as opposed to using marijuana for recreational purposes. Such patients may have been more responsive to the alcohol interventions, which emphasize health benefits of drinking reduction, potentially diminishing marijuana's role as a risk factor in this group. Although this study did not measure marijuana use for medical reasons, future researchers could assess whether medical users in particular are more responsive to alcohol intervention. Second, HIV patients who have histories of both alcohol and marijuana use may have begun to use marijuana in lieu of alcohol after the medical risks of drinking with HIV were brought to their attention. This is consistent with research suggesting that substitution may occur between these two substances (Allsop et al., 2014; Peters and Hughes, 2010), although not all research supports substitution (Blanco et al., 2014; Kadden et al., 2009). While our study focused on *baseline* drug use as a predictor of intervention outcome (preventing investigation of substitution during treatment), future research specifically designed to assess during-treatment use could examine substitution. Finally, it is worth noting that although the current study suggests that marijuana is not a risk factor for heavy post-treatment drinking, marijuana may still carry other health risks for individuals with HIV. For example, especially for individuals co-infected with Hepatitis C, marijuana may negatively impact liver function (Abramovici, 2013).

Interactions between drug use and intervention condition indicated that the benefit of MI +HealthCall was most pronounced among individuals using cocaine only. In the main trial, differences in drinking quantity by intervention condition were concentrated among alcohol-dependent patients (Hasin et al., 2013). Taken together, these findings suggest that there are subtypes of HIV-infected drinkers (non-dependent, marijuana-using) who are just as likely to reduce the amount they drink after brief education as they are after more extensive, tailored intervention. Drinking reduction after even brief interventions in less severe drinkers is consistent with findings from general primary care (Saitz, 2010). Therefore, for busy clinics without resources to implement MI or MI+HealthCall, providing even brief education to HIV-infected drinkers may yield noticeable benefit for certain subgroups of patients. However, other subtypes of HIV-infected patients (alcohol dependent, those using cocaine but not marijuana) do reduce drinking to a greater degree after they receive more extensive intervention (i.e., MI+HealthCall). This is important, as such groups are likely most in need of alcohol intervention.

This study is subject to certain limitations. First, only marijuana and cocaine use were examined, as only these substances were used by substantial proportions of patients in the current sample (>30%) (Elliott et al., 2014a). Future research, if conducted with HIV-infected samples with different drug use profiles, could examine whether methamphetamine, heroin, or other drugs are associated with differences in drinking after alcohol intervention. Second, the current study was conducted in one large HIV primary care clinic in the urban northeast, with primarily disadvantaged, minority patients; replication is needed to determine if these findings apply to HIV patients in other regions or with different demographic characteristics. Third, whether these findings apply to HIV patients receiving other types of treatment, such as cognitive-behavioral therapy or pharmacotherapy, is also unknown. Fourth, although the results are prospective, marijuana and cocaine use were

clearly not randomly assigned, precluding causal inference about the effects of drug use on alcohol treatment outcomes. Instead, the current study identifies patients' characteristics that serve as prognostic indicators of treatment outcome, which could be due to either direct effects of the substances or to other currently unidentified factors also associated with use. Fifth, drug use was measured for the past-year at baseline, and therefore does not indicate concurrent use over the course of the study, which could be viewed as a limitation. However, the analyses we conducted provide important information on prediction of patient outcome and choice of appropriate intervention type based on baseline presentation, which is when clinicians must make treatment plans and decisions. Future studies should investigate concurrent drug use and its effect on intervention, a different but also important research question. Sixth, the dichotomous nature of drug use assessment (i.e., positive or negative for each substance) does not allow for consideration of drug use intensity. Although analyses of the influence of drug use quantity and frequency were not possible in our study, investigation of this association in future studies could provide information on whether the magnitude of drug use predicts post-intervention drinking in a dose-response manner. Despite these limitations, the study demonstrates several strengths, notably: the importance of identifying patients' attributes associated with drinking after alcohol intervention, the large sample (n=254), and the use of psychometrically strong assessment measures.

This study suggests that among HIV-infected heavy drinkers, those also using cocaine may generally respond less to alcohol interventions. However, the cocaine users who received MI +HealthCall did reduce drinking, demonstrating potential utility of this particular intervention for this challenging patient group. Our findings indicate the need for enhanced clinical attention to HIV-infected heavy drinkers using cocaine, as well as the need for larger studies to replicate the benefit of MI+HealthCall for this group. More research should determine whether these findings are replicable across HIV-infected populations of differing demographic and geographic characteristics, and with use of different interventions.

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Highlights

- We evaluated effects of drug use on post-intervention drinking in a sample with HIV.
- Baseline cocaine use predicted heavier drinking after alcohol intervention.
- Baseline marijuana use was not a risk factor for heavy post-intervention drinking.
- Cocaine users showed the largest differences in drinking by intervention intensity.

Figure 1.
Drinks per drinking day before and after intervention by drug use status (unadjusted means).

Figure 2.
End of treatment drinks per drinking day by intervention type and drug use status (unadjusted means).

Table 1

Drinks per drinking day (unadjusted M[s.d.]) and percent days drinking (unadjusted M[s.d.]) at baseline and end-of-treatment by drug use status.

	No drug use	Marijuana-only	Cocaine-only	Both Marijuana and cocaine
Drinks per drinking day				
Baseline	6.76 (3.55)	6.51 (3.27)	8.51 (5.15)	6.58 (3.38)
End-of-treatment	3.92 (2.69)	3.11 (1.37)	5.99 (3.96)	3.74 (1.38) *
Percent days drinking				
Baseline	29.82 (23.90)	40.89 (29.40)	26.75 (17.97)	34.80 (23.79)
End-of-treatment	12.93 (17.58)	18.95 (23.55)	16.92 (20.19)	24.44 (27.09)

Note. Statistical modeling uses *percent days abstinent* as a frequency measure, whereas *percent days drinking* is used in this table for ease of interpretation.

* Indicates significant differences in end-of-treatment drinking by drug use status, controlling for intervention condition and baseline drinking, as well as demographic and HIV characteristics (age, sex, race, education, language, HIV medication status, years since HIV diagnosis), $p < 0.01$.

Table 2

Additive interaction of past-year drug use and intervention condition for drinks per drinking day outcome.

Condition	Adjusted mean differences (SE)			
	Marijuana-only vs. No drug	Cocaine-only vs. No drug	Both drugs vs. No drug	
Educational control	-1.43 (0.73)	2.97 (1.05)**	-0.11 (0.77)	
MI only	-0.62 (0.71)	1.53 (1.03)	-0.07 (0.81)	
MI+HealthCall	0.51 (0.74)	-0.23 (0.75)	0.63 (0.81)	
Interaction contrast MI – vs. control	0.81 (1.02)	-1.43 (1.49)	0.04 (1.12)	
Interaction contrast MI – HealthCall vs. control	1.93 (1.04)	-3.19 (1.28)*	0.74 (1.11)	

* p<0.05,

** p<0.01.

Mean difference values are adjusted for baseline drinking, as well as demographic and HIV characteristics (age, sex, race, education, language, HIV medication status, years since HIV diagnosis).