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Increased Drinking in a Trial of Treatments for Marijuana Dependence: Substance Substitution?

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Abstract

This report examines whether participants in a study of treatments for marijuana dependence may have increased their use of alcohol when they reduced or ceased marijuana use. Participants were randomly assigned to one of four psychosocial treatments and followed at 3-month intervals for one-year. Findings are from 207 cases with data at posttreatment and at least one other follow-up. 73% of cases reported an increase of at least 10% in drinking days over their level at intake, and 65% reported an increase of at least 10% in drinks per drinking day. Drinking increases were not related to treatment condition nor to change in marijuana use, but were related to baseline drinking: those with less baseline drinking tended to increase their drinking during treatment and those with more baseline drinking reported less drinking during treatment. Thereafter, drinking levels remained fairly stable throughout the follow-up year. The results are most likely reflective of a regression to the mean effect, and indicate that use of alcohol and marijuana are independent of one another.

Keywords

Marijuana dependence; alcohol; substitution; contingency management; CBT; MET

1. Introduction

Substance substitution is a potential concern in treating substance use disorders. Clinicians frequently caution patients about the danger of substituting a ‘safe’ substance (one that has not been a problem) after they cut down or stop using the one(s) for which they sought treatment. Researchers have been advised to include multiple dependent measures in assessment batteries

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to assess for the possibility of substance substitution (Jeffrey, 1975). In a recent study of treatments for marijuana dependence (Kadden et al., 2007), we noted anecdotally that some participants increased use of alcohol as marijuana use declined.

Two studies of treated marijuana-dependent individuals, and two studies of untreated marijuana users, reported that reductions in marijuana use were not accompanied by increased drinking (Hammer & Vaglum, 1992; Hughes et al., 2008; Marijuana Treatment Project Research Group, 2004; Stephens et al., 2000). However, one study (Copersino et al. 2006) reported that about one-third of untreated daily marijuana users who quit, increased use of alcohol.

The present report provides a systematic analysis of the extent to which participants without current alcohol dependence increased drinking when they reduced or ceased marijuana use during psychosocial treatment (Kadden et al., 2007).

2. Methods

2.1 Participants

People who wanted help stopping or decreasing marijuana use were recruited through print and radio advertisements. Participants met DSM-IV criteria for marijuana dependence (American Psychiatric Association, 1994) and used marijuana on ≥ 40 of the prior 90 days. People were excluded if they currently met dependence criteria for other illicit substances or alcohol, or had severe medical or psychiatric problems that precluded participation. The sample included 240 participants ≥ 18 years, average age 32.7 ($SD=9.6$), 29% female, 38% non-Caucasian, 60% unmarried, and 37% unemployed. This paper is based on 207 cases with data at posttreatment and at least one other follow-up.

Participants provided IRB-approved informed consent, and were randomly assigned among four conditions: (1) two sessions of Motivational Enhancement Therapy plus seven of Cognitive Behavior Therapy (MET/CBT); (2) contingency management (ContM), with vouchers provided for drug-free urines; (3) MET/CBT plus ContM; or (4) case management (CaseM). All interventions were provided in nine individual sessions with a therapist, for 1 hour each, or with a research assistant for 20 minutes (ContM). The MET/CBT and CaseM interventions were similar to those in the Marijuana Treatment Project (Marijuana Treatment Project Research Group, 2004). ContM was similar to that described by Petry et al. (2005).

2.2 Measures

The Time-Line Follow-Back (TLFB) was used to gather marijuana frequency-of-use data during the 90 days before intake and each follow-up (Sobell & Sobell, 1992). Alcohol use was assessed by point estimates of number of days of use, average quantity per day, and days of heavy drinking over the preceding 90 days. Additional assessments included the Addiction Severity Index (ASI; McLellan et al., 1992), Beck Depression Inventory (Beck et al., 1979), Marijuana Problem Scale (Stephens et al., 1993), Coping Strategies Scale (Litt et al., 2003), and a measure of self-efficacy (Stephens et al., 1995). Assessments were conducted at study intake, posttreatment (approximately 2 months later), and at 3-month intervals for 1 year (5, 8, 11, and 14 months post-intake). Further information can be found in Kadden et al. (2007).

2.3 Data Analysis

The two primary drinking variables were frequency, the proportion of days using alcohol (PDU) in the previous 90 days, and quantity, drinks per drinking day (DDD). Participants were considered to have increased drinking if their PDU or DDD at any follow-up point was $\geq 10\%$ over baseline. A 10% increase is a small change, given the low levels of baseline

drinking; it provided a sensitive criterion for detecting change. Logistic regressions evaluated predictors of increased v. non-increased drinking (PDUA and DDD).

3. Results

3.1 Drinking Frequencies and Quantities

Drinking levels were quite low in this sample. Mean PDUA at intake was .12 ($SD=.18$), with 33 (16%) cases reporting no drinking, and 139 (67%) reporting drinking <3 days per month. Mean DDD at intake was 2.8 drinks ($SD=2.8$), with 178 (86%) reporting no heavy drinking days. Alcohol dependence was an exclusion criterion, minimizing heavy drinking at intake.

3.2 Occurrence of Increased Drinking

Examination of PDUA indicated that 73% (151 cases) reported an increase in drinking days of $\geq 10\%$ over intake at some point after treatment began. Logistic regression indicated that increased drinking days were not related to treatment condition, but to baseline drinking frequency. Those with higher frequencies of baseline drinking were *less* likely to increase drinking at later time points ($B=-3.87$; $se=1.06$; Wald $\chi^2=13.32$; $p<.001$; OR=0.02; 95% CI: .01-.17). This effect is illustrated in Panel A of Figure 1, which depicts mean drinking frequency across the trial by drinking change status. Those who did not increase drinking had reported more frequent drinking at baseline, and actually decreased drinking during treatment [within group $F_{pre-post}(1,55)=27.32$; $p<.001$]. Conversely, those who drank more frequently after starting treatment, began with lower levels which increased significantly at posttreatment [within-group $F_{pre-post}(1, 150)=20.64$; $p<.001$]. Following these initial changes, the average frequency of drinking remained fairly stable, at an average level of approximately 0.12 (about 3.6 days/month) across all participants throughout the follow-up year.

Results were similar for DDD: 65% (135 cases) increased DDD $\geq 10\%$ after starting treatment. As with PDUA, treatment condition was not a factor in who increased drinking, but pretreatment drinking intensity was ($B=-0.22$; $se=0.06$; Wald $\chi^2=12.12$; $p<.001$; OR=0.80; 95% CI: .71-.91). Those with higher baseline DDD were less likely to report increases in DDD after starting treatment (Figure 1, Panel B), and this remained relatively stable throughout follow-up.

An initial impression is that changes in drinking during treatment might be characterized as a regression to the mean effect (Finney, 2007), in which the distribution narrows over time. Due to the absence of a treatment condition effect on drinking, treatment was not considered in any subsequent analyses.

3.3 Effects of Reduced Marijuana Use on Increased Drinking

Marijuana use (proportion days of use, PDUM) decreased sharply from pre to posttreatment in all conditions, and remained at a relatively low level through the 14-month follow-up (Kadden et al., 2007). A marijuana use change score was computed (intake PDUM minus posttreatment PDUM) to represent the reduction for each person.

With PDUA increase as the criterion, and marijuana change score and intake PDUA as predictors in a logistic regression analysis, significant effects occurred only for intake PDUA ($B=-3.83$; $se=1.04$; Wald $\chi^2=13.58$; $p<.001$; OR=0.02; 95% CI: .01-.16). The marijuana change score failed to show an effect on drinking. Similarly, when drinking was defined in terms of DDD, marijuana change score had no effect but baseline DDD was a significant predictor ($B=-0.21$; $se=0.06$; Wald $\chi^2=11.82$; $p<.001$; OR=0.80; 95% CI: .71-.91).

For illustrative purposes, participants were classified into three drinking groups, based on frequency tertiles of PDUA at intake: 34% were classified as low drinkers (intake PDUA=.00-.03), 33% as mid drinkers (PDU=.03-.11), and 33% as high drinkers (PDUA \geq .11). Figure 1, panel C, shows the relationship between marijuana use and drinking rates (PDUA) over time, for the three levels of initial drinking. Frequency of marijuana use did not differ among drinking levels: light, mid, and heavy drinkers all smoked at equal rates, and reduced smoking by equivalent amounts. Panel C also shows that drinking did not increase as marijuana use decreased, suggesting that participants did not compensate for decreased marijuana use by increasing drinking.

3.4 Determining Increased Drinking

It appears that reduced marijuana use was not a factor in the increased drinking that occurred. Logistic regressions assessed if increased drinking could be explained by other variables. For the first analysis, PDUA increase of $\geq 10\%$ was the criterion variable. Predictors included demographics (age, sex, ethnic group, education, employment, and marital status), psychiatric status indicators, substance use severity, and measures of coping and self-efficacy for changing marijuana use. With these variables in the model the only significant predictor of increase in PDUA was baseline drinking (B=-4.36; se=1.21; Wald $\chi^2=13.05$; $p<.001$; OR=0.01; 95% CI:.01-.14).

The same set of variables was used to predict a DDD increase of $\geq 10\%$. The only significant predictors were baseline coping score and baseline DDD. As seen previously, those with higher baseline drinking were less likely to increase drinking over the study period. (B=-0.30; se=0.08; Wald $\chi^2=13.58$; $p<.001$; OR=0.74; 95% CI:.63-.87). Additionally, those with higher coping scores were less likely to drink above baseline levels (B=-1.01; se=0.40; Wald $\chi^2=6.26$; $p<.05$; OR=0.37; 95% CI:.17-.80).

3.5 Progression to Dangerous Drinking

To evaluate the possibility that reduction of marijuana use might prompt drinking at dangerous levels, we examined the rate of heavy drinking days. At each follow-up, participants were asked how many times they consumed ≥ 6 drinks a day. Response options were: (0) never; (1) <weekly; (2) weekly (1-1.9 times/week); (3) <daily (2-4 times); and (4) daily or almost daily (5-7 times).

The median value of the Heavy Drinking Days variable was 0 at all time points, and the mean was below 0.55 (between 'never' and '<weekly'). Correlations of marijuana use with the heavy drinking frequency categories were non-significant. Thus, dangerous drinking in response to reduction or cessation of marijuana use did not occur in this study.

4. Discussion

On average, participants cut their marijuana use at least in half. At the same time, a considerable number (73%) increased their drinking by at least a small amount. Nevertheless, there was no relationship between drinking and change in marijuana use. The only variable consistently related to increased drinking was a low level of baseline drinking.

We utilized a very liberal criterion for detecting increased drinking. A change of 10% above baseline represents an increase of only 1.08 in number of days out of 90 on which drinking occurred, and an increase of only 0.28 drinks per drinking day. Although 73% of cases increased PDUA and 65% increased DDD, these upward shifts were quite small, as seen in Figure 1. None of the analyses provided any indication that these shifts were related to changes in marijuana use, treatment type, or any other predictor variables tested, leaving us unable to

account for the shifts. One may conclude that reduced marijuana use does not substantially enhance drinking in a non alcohol-dependent sample. It would nevertheless be prudent in clinical settings to monitor drinking to detect cases that do increase.

In this study there appeared to be a regression to the mean effect, with heavier drinkers at baseline reducing drinking at the follow-ups, and lighter drinkers showing an increase. The reason for this is unknown. The extent to which it may have involved a change in participants' reporting of drinking across the trial cannot be determined.

There was an inverse relationship between total coping score and the DDD measure: participants with higher coping scores were less likely to increase drinking. Although this makes intuitive sense, it is an isolated finding that cannot be given too much credence.

Some limitations ought to be noted. Because the underlying study was an evaluation of treatments for marijuana dependence, drinking data were not collected with the same precision as marijuana data. Whereas the TLFB was used to assess daily marijuana use, drinking was estimated as an average over the preceding 90 days, which is less accurate than the daily assessment procedure, and there was no verification of drinking self-reports. Finally, to focus the study on marijuana dependence, people who were also dependent on alcohol or other drugs were excluded, thereby limiting the upper range of drinking.

We are left with a mystery: 73% of participants increased their PDUA slightly in this study of marijuana treatment, but there were no indications that this was due to treatment, decreased marijuana use, or other variables. These results most likely reflect regression to the mean. This study may help allay concerns of treatment providers, in that treatment will likely not result in increased drinking in non alcohol-dependent individuals.

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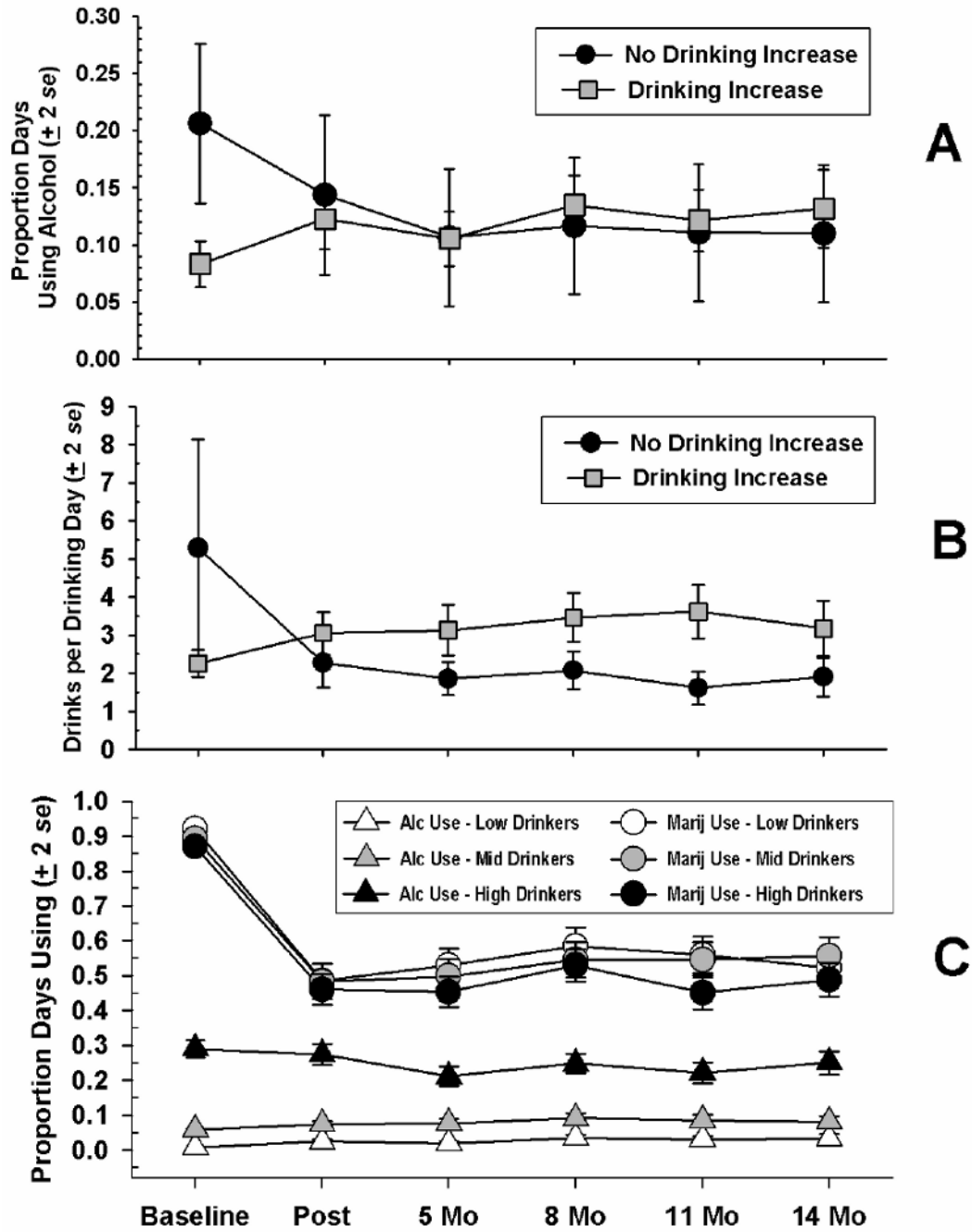


Figure 1. Panels A and B: mean drinking during the 90 days preceding baseline and each of the five follow-up points, subdivided according to those who increased their drinking above the level recorded at intake, and those who did not increase their drinking. Panel A: drinking frequency data; Panel B: drinking quantity per drinking day. Panel C: frequency of alcohol and marijuana use across the trial, subdivided into three groups based on level of drinking during the 90 days prior to intake: least drinking prior to intake; moderate level of drinking; and highest level of drinking.