Alcohol and cocaine interact both behaviorally and biologically, and there is a high rate of comorbidity for abuse of both substances. \(^1\)\(^-\)\(^3\) In most treatment settings, abstinence from all drugs including alcohol is encouraged. It is not clear, however, whether concurrent alcohol use affects baseline clinical characteristics and treatment outcome for cocaine abuse. Carroll and colleagues have reported an association between concurrent alcohol abuse and more severe cocaine dependence and increased polysubstance abuse. \(^4\) Other studies have found an association between comorbid alcohol and cocaine use and higher baseline scores on the family and alcohol sub-scales of the Addiction Severity Index, along with higher scores on baseline depression and global severity measures. \(^1\)\(^,\)\(^5\)\(^,\)\(^6\) Research also indicates concurrent cocaine and alcohol use results in a prolonged euphoria and increased heart rate. Both the euphoria and increased heart rate may be associated with cocaethylene, a metabolite formed from combined cocaine and alcohol ingestion. \(^2\) Higgins

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**Alcohol Use Affects the Outcome of Treatment for Cocaine Abuse**

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This study investigated whether alcohol use affects baseline characteristics and treatment outcome in 128 adults who participated in a randomized trial of cognitive behavioral vs. 12-step treatment for crack cocaine abuse. Assessments were taken at baseline and weeks 4, 8, 12, and 26 on biologically-verified cocaine abstinence and psychometric measures. Alcohol use was measured at intake and subsequent assessments using the Addiction Severity Index (ASI) and self-reported frequency of alcohol consumption. Results indicate alcohol use at baseline was associated with increased baseline cocaine use and ASI drug severity but was not associated with ASI psychiatric severity, psychiatric diagnoses, or other baseline variables. Alcohol use at baseline did not predict worse treatment outcome for cocaine abstinence. However, alcohol use after four weeks of treatment did predict ability to achieve cocaine abstinence at assessment points during and after treatment. (Am J Addict 2002;11:219-227)

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and colleagues also reported that alcohol can increase preference for cocaine over an alternative monetary reinforcer.\(^7\)

Research on the effect of comorbid alcohol use on treatment outcome for cocaine abuse has yielded mixed results. One year following inpatient treatment, Brower and colleagues reported similar outcomes in subjects with comorbid alcohol and cocaine use disorders compared to subjects with a single cocaine or alcohol use disorder.\(^8\) In a sample of 303 cocaine abusing clients, Hoffman and colleagues found that recent alcohol use did not predict retention in outpatient treatment or treatment exposure rates.\(^9\) Khalsa and colleagues compared participants with pretreatment, comorbid alcohol and cocaine dependence to those with cocaine dependence alone, and they found no difference in cocaine use at one year follow-up.\(^10\) Carroll and colleagues reported that cocaine abusers who were alcoholic at baseline were more likely to remain alcoholic at follow-up, but did not differ significantly on other outcome measures one year after treatment.\(^4\)

In contrast, Schmitz and colleagues reported that participants with both alcohol and cocaine dependence showed less improvement in drug and psychiatric symptomatology at follow-up than participants with cocaine dependence alone.\(^11\)

In a second study assessing predictors of treatment outcome, Carroll found that concurrent alcoholism was associated with poorer outcome among treated cocaine abusers in the domains of substance abuse and psychiatric functioning.\(^12\) McKay and colleagues evaluated cocaine dependent males in the 6 month follow-up period after intensive outpatient treatment and found that though alcohol dependence status before treatment did not predict treatment outcome for cocaine use, any alcohol use in the four of the first five follow-up months significantly predicted cocaine relapse status in the next month.\(^13\)

Based on the studies described above, it is still unclear how alcohol use may affect treatment outcome for cocaine abuse. The difference in results of these studies may be based on several variables, including whether a diagnosis of alcohol dependence was obtained, when alcohol and cocaine use was assessed, and the type of treatment under study. Many studies to date have used a baseline diagnosis of alcohol abuse or dependence to predict cocaine abstinence after treatment but did not examine how alcohol use during treatment might affect outcome. The purpose of this study was to investigate how alcohol use at baseline and during treatment affected treatment outcome of crack cocaine-abusing adults who participated in a randomized trial of cognitive behavioral vs. 12-step treatment.\(^12\)

The following hypotheses were proposed:

1) Greater alcohol use at baseline would predict poorer baseline clinical functioning as measured by greater cocaine use and higher drug and psychiatric severity. This hypothesis is based on the findings from earlier studies that indicate that alcohol abuse is associated with increased polysubstance abuse, addiction severity, and depression.\(^1,4,6\)

2) Greater alcohol use at baseline would predict lower rates of cocaine abstinence during and after treatment.

3) Greater alcohol use during treatment would also predict poorer outcome for cocaine abstinence.

The second and third hypotheses are based on the findings from Schmitz and Carroll that comorbid alcohol dependence resulted in poorer treatment outcomes for cocaine abusers, and the findings from McKay that alcohol use during follow-up predicted cocaine relapse.\(^11-13\) They are also based on data that alcohol may act to reinforce cocaine use and enhance euphoria.\(^2\)
METHOD

Participants

A detailed description of participants and assessments in the main study is provided in an earlier paper by Maude-Griffin et al. Only relevant information is repeated here. Participants (n = 128) were recruited from three San Francisco, California Veterans Affairs Medical Center (VAMC) programs: the Substance Abuse Inpatient Unit, the Substance Abuse Outpatient Programs, and the Health Care for Homeless Veterans Program. Participants who met DSM-III-R criteria for current cocaine abuse were included. Exclusion criteria included current or historical opioid dependence, current or historical diagnosis of schizophrenia, or a current medical or psychiatric condition that would contraindicate outpatient treatment. Participants were predominantly male (99%) and African-American (80%) with the remainder identifying as Caucasian (13%), Hispanic (2%), and Other/Mixed (5%). Age ranged from 29 to 62 years (M = 42.35; SD = 7.20). At intake, 84% of participants were unemployed and 15% were homeless.

Assessments

Participants were assessed by research assistants at intake and weeks 4, 8, 12, and 26 after intake for demographics, alcohol use, cocaine and other drug use, HIV risk behaviors, and general psychosocial functioning. Urine samples were collected at every research assessment and once each week during treatment. Samples were analyzed qualitatively for all drugs of abuse.

A battery of psychometric measures was used in the assessments; a full description of all the measures used are described by Maude-Griffin et al. The specific measures relevant to this study are listed below.

Diagnostic Interview Schedule (DIS). The computerized version of the DIS (CDIS) was used for which acceptable kappas have been demonstrated (.57-.64). At baseline, the CDIS was used to assess participants for Major Depressive Disorder (MDD), Post-Traumatic Stress Disorder (PTSD), and Antisocial Personality Disorder (ASPD).

Addiction Severity Index (ASI). The ASI was used to assess alcohol and drug severity and psychosocial functioning at intake and 4, 8, 12, and 26 weeks after intake. The ASI yields composite severity scores for medical status, employment, drug use, alcohol use, legal status, family and social relationships, and psychiatric functioning. These scores are weighted combinations of individual items that have been shown to provide reliable, valid, and sensitive measures of problem severity during 30 days prior to the interview.

Categorical Frequency of Alcohol Consumption. We also used a questionnaire developed for an earlier multi-site study to categorize participants based on their self-reported frequency of alcohol consumption in the thirty days before intake and weeks 4, 8, 12, and 26. Subjects were grouped into three categories: 1) Non-drinkers, who denied any alcohol consumption in the thirty days prior to assessment; 2) Less than Daily drinkers, who reported drinking at least once a week and as often as 2-6 times per week; 3) Daily drinkers, who reported drinking at least daily and as often as 4 or more times per day.

Treatment

Participants were randomly assigned to cognitive behavioral or 12-step facilitation group treatment. Treatment lasted 12 weeks and participants in each treatment condition attended three group therapy sessions per week.
sessions (90 min each) and one individual counseling session each week. Treatment conditions were standardized using detailed treatment manuals. Both treatment conditions strongly encouraged abstinence from all drugs, including alcohol.

Outcome Variables

The main outcome variables were: 1) four consecutive weeks of abstinence from cocaine any time during treatment as measured by self report and weekly urine toxicology, and 2) point prevalence cocaine abstinence as measured by self-reported cocaine use in the past 30 days and verified by urinalysis. Point prevalence abstinence was measured at intake and 4, 8, 12, and 26 weeks after intake. Urine samples were collected once each week on a random schedule and qualitatively analyzed for all drugs of abuse.

Data Analysis

To test the relationship between a dichotomous measure (such as cocaine abstinence) and a continuous measure (such as ASI alcohol severity), we compared the mean scores by computing a point-biserial correlation coefficient. Test for change among continuous measures was calculated using a one-way ANOVA based on likelihood estimation. A Bowker’s test was used to test for change in the categorical variables. Pearson’s chi-square test was used for testing homogeneity among categorical measures. A significance level of $p = 0.05$ or less was used.

RESULTS

Sample Pretreatment Characteristics

Participants were crack cocaine users who had a mean history of cocaine use of 19 years and were using cocaine a mean of 13.80 days ($SD = 11.83$) out of the 30 days before intake. Mean number of days of alcohol use in the past thirty days before intake was 10.26 ($SD = 10.92$). Seventeen percent of participants reported no alcohol consumption in the past 30 days, 48 percent reported that they drank less than daily, and 37 percent reported drinking daily or more. A diagnosis of alcohol abuse or dependence was not an exclusion or inclusion criterion. Other drug use was relatively minimal. Participants reported a mean of three days of marijuana use and one day of heroin use in the thirty days before intake.

Eighty-two percent of the participants had a lifetime diagnosis of at least one of the three psychiatric disorders assessed at baseline in the main study Major Depressive Disorder, Post Traumatic Stress Disorder, and Antisocial Personality Disorder. Eighty-six percent of participants with a lifetime diagnosis of major depression also met criteria for current major depression. Additionally, a small number of participants ($n = 6$) had a lifetime diagnosis of Bipolar Disorder.

Baseline Alcohol Use and Clinical Severity

With respect to the first hypothesis, that greater alcohol use at baseline would correlate with worse baseline clinical functioning as measured by greater cocaine use and greater ASI drug and psychiatric severity, analyses indicated that both measures of alcohol use the ASI and the categorical frequency of alcohol consumption correlated with greater drug severity and cocaine use but not with greater psychiatric severity. The ASI alcohol severity score correlated with the ASI drug severity score and the number of days of cocaine use before intake (Pearson $r(df) = 0.56$ (126), $p = 0.0001$; $r(df) = 0.44$ (126), $p = 0.0001$, respectively). Similarly, participants who fit into the more
severe category of alcohol consumption had more days of cocaine use and greater ASI drug severity ($p = 0.0026$ and 0.0001, respectively). There was no significant relationship between baseline alcohol use and ASI psychiatric severity or the presence of a psychiatric diagnosis. There also was no significant relationship between baseline alcohol use and baseline demographic variables or other baseline ASI severity scores (medical, employment, family, and legal).

The second hypothesis, that greater alcohol use at baseline would portend overall worse outcome for cocaine abstinence, was not supported. Baseline ASI alcohol severity and categorical frequency of alcohol consumption did not predict point prevalence cocaine abstinence at future assessment points, nor did either variable predict four consecutive weeks of cocaine abstinence during treatment.

Changes in Alcohol Use During Treatment

Before testing the third hypothesis, that greater alcohol use during treatment would also predict poorer outcome for cocaine abstinence, changes in ASI alcohol severity and categorical frequency of alcohol consumption during treatment were evaluated. The ASI alcohol severity decreased significantly between intake and Week 4 of treatment ($F(df) = 13.5(4,446), p = 0.0001$), with no significant change thereafter. The mean ASI alcohol severity score at intake was 0.237 (SD = 0.222). The Week 4 score was 0.125 (SD = 0.158).

There was also a significant change from Intake to Week 4 in the frequency of alcohol consumption (Bowker’s Test ($df = 40.159(3), p = 0.001$). At Week 4, five participants reported a higher frequency of drinking compared to baseline and 52 reported a lower frequency of drinking. The remaining 57 reported no change. The changes in frequency of alcohol consumption are presented in Table 1. There was no significant change in

| TABLE 1. Change in Categorical Alcohol Consumption in the First Four Weeks of Treatment |
|-----------------------------------------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Intake No alcohol                            | Week 4 No Alcohol | Week 4 Less than Daily | Week 4 Daily or More | Intake Categorical Total |
| Intake No alcohol                            | Week 4 No Alcohol | Week 4 Less than Daily | Week 4 Daily or More | Intake Categorical Total |
| n                                             | 17               | 4               | 0               | 21               |
| %Total n (114)                                 | 15%             | 4%              | 0%              | 18%              |
| % Categorical n (21)                          | 81%             | 19%             | 0%              | 81%              |
| Intake Less than daily                        | Week 4 No Alcohol | Week 4 Less than Daily | Week 4 Daily or More | Intake Categorical Total |
| n                                             | 18               | 35              | 1               | 54               |
| %Total n (114)                                 | 16%             | 31%             | 1%              | 47%              |
| % Categorical n (54)                          | 33%             | 65%             | 2%              | 33%              |
| Intake Daily or more                          | Week 4 No Alcohol | Week 4 Less than Daily | Week 4 Daily or More | Intake Categorical Total |
| n                                             | 19               | 15              | 5               | 39               |
| %Total n (114)                                 | 17%             | 13%             | 4%              | 34%              |
| % Categorical n (39)                          | 49%             | 38%             | 13%             | 49%              |
| Week 4 Categorical total                      | 54               | 54              | 6               | Total n = 114* |
| %Total n (114)                                 | 47%             | 47%             | 5%              | 100%             |

*14 subjects failed to provide data at one or more occasions.
categorical frequency of alcohol consumption after Week 4.

Predicting Cocaine Abstinence from Alcohol Use During Treatment

In testing the third hypothesis, we found that less alcohol use at Week 4 predicted improved outcome for cocaine abstinence. A lowerASI alcohol severity at Week 4 predicted cocaine abstinence at both Weeks 8 and 12 as indexed by a Pearson correlation coefficient ($r$ (df) = −.33(112), $p = 0.0003$; $r$ (df) = −.23(112), $p = 0.0013$, respectively), indicating less alcohol severity at one assessment meant a greater probability of cocaine abstinence at future assessments during treatment. Similarly, Week 8 alcohol severity predicted Week 12 cocaine abstinence ($r$ (df) = −.26(106), $p = 0.0066$), and Week 12 predicted Week 26 ($r$ (df) = −.23(117), $p = 0.0133$). Lower ASI alcohol severity at Week 4 also predicted achievement of four consecutive weeks of cocaine abstinence during treatment ($r$ (df) = −.25(112), $p = 0.0073$).

Parallel and slightly stronger results were obtained with the categorical frequency of alcohol consumption predicting future cocaine abstinence. A lower frequency of alcohol consumption at Week 4 predicted point prevalent cocaine abstinence at Weeks 8, 12, and 26 (chisquare(df) = 25.98(2), $p = 0.001$; chisquare(df) = 19.69(2), $p = 0.001$; chisquare(df) = 6.96(2), $p = 0.031$, respectively). Less alcohol consumption at Week 8 also predicted cocaine abstinence at Weeks 12 and 26 (chisquare(df) = 19.50(2), $p = 0.001$; chisquare(df) = 6.23(2), $p = 0.044$, respectively), and Week 12 predicted Week 26 (chisquare (df) = 8.639(2), $p = 0.013$). In addition, a lower frequency of alcohol consumption at Week 4 of treatment predicted achievement of four consecutive weeks of cocaine abstinence during treatment (chisquare (df) = 31.55(2), $p = 0.001$).

Interaction Between Alcohol Use and Treatment Condition

Since results from the main study found that participants in the cognitive behavioral treatment condition were significantly more likely to achieve four consecutive weeks of abstinence from cocaine than participants in the 12-step treatment, we examined whether there was any interaction between response to cognitive behavioral versus 12-step treatment and alcohol use at baseline and during treatment. In general, neither ASI alcohol severity nor frequency of alcohol consumption interacted with treatment condition to predict cocaine abstinence.

DISCUSSION

This study investigated whether alcohol use was associated with baseline clinical severity or negative treatment outcome in a randomized trial of cognitive behavioral versus 12-step therapy for cocaine abuse. We found a significant correlation between baseline alcohol use and baseline cocaine use and ASI drug severity. There was no significant association between alcohol and psychiatric severity or diagnoses. Alcohol use at baseline did not predict cocaine abstinence at future assessments but alcohol use during treatment did predict decreased cocaine abstinence. We found that there was a significant reduction in ASI alcohol severity and frequency of alcohol consumption in the first four weeks of treatment. There was no interaction found between alcohol use and the cognitive behavioral or 12-step treatment condition.

Results of this study support previous research which found that comorbid alcohol and cocaine abuse are associated with increased drug use and severity, though we did not find an increased association with depression or other psychiatric diagnoses. These differences may have
resulted in differences in how alcohol and psychiatric problems were assessed and diagnosed.

This study confirms earlier research which found that baseline alcohol comorbidity had no effect on treatment outcome for cocaine abuse. However, we found that alcohol use changed significantly during the first four weeks of treatment, and that a lower level of alcohol use both at and after Week 4 predicted future cocaine abstinence, implying that the ability to reduce alcohol use during early treatment may be an important prognostic factor. This finding is supported by data indicating that concurrent alcohol and cocaine use is common and may enhance the euphoric effects of both drugs. Such data along with the findings of this study suggest that alcohol may be a trigger or positive reinforcer for cocaine and vice versa. This information also supports the common treatment strategy of encouraging abstinence from all drugs during treatment, including alcohol.

This study was a retrospective analysis of a completed study and thus was limited specifically in its measures of alcohol use. An objective diagnosis of alcohol dependence or abuse was not obtained at baseline, which made it difficult to compare to previous studies. The study also measured frequency of alcohol consumption and not the amount of alcohol per drinking episode, which may have given a clearer picture of total alcohol consumption. The strength of this study was that it looked at two measures of alcohol use frequency and the ASI and examined the predictive value of these measures not only at baseline but during treatment. Suggestions for future research include investigating how variables of concurrent alcohol and cocaine use, such as cocaethylene and enhanced euphoria, interact with each other to influence treatment outcome. Several studies have already investigated how pharmacotherapy aimed at decreasing alcohol use may enhance treatment outcome for cocaine use disorders. In a randomized trial of disulfiram and three forms of psychotherapy for comorbid cocaine and alcohol abuse, Carroll and colleagues discovered disulfiram was associated with significantly longer periods of consecutive abstinence for cocaine, alcohol, and concurrent alcohol and cocaine use. Higgins and colleagues also reported a significant decrease in alcohol and cocaine use in 16 cocaine-dependent and alcohol-abusing patients treated with disulfiram. Hersh and colleagues found no advantage of naltrexone over placebo in subjects with comorbid alcohol and cocaine disorders, though the study was limited to a single naltrexone dosage of 50 mg. Oslin and colleagues found a reduction in both alcohol and cocaine usage in alcohol- and cocaine-dependent subjects treated with a naltrexone dose of 150 mg.

Psychosocial interventions are also a topic for ongoing research. Though no interaction was found between alcohol use and either 12-step or cognitive behavioral therapy in the main study, these treatments were not compared to a control psychotherapy condition. In the treatment of comorbid alcohol and cocaine abuse, Carroll and colleagues also found no difference in effectiveness between cognitive behavioral and 12-step therapy but found that these treatments were both more effective than supportive psychotherapy. They also concluded that the combination of pharmacotherapy and psychotherapy was the most promising treatment strategy.

Findings from this study indicate that a reduction in alcohol use early in cocaine abuse treatment is an important prognostic factor. Continuing research and development of targeted pharmacological and psychosocial interventions that facilitate this reduction in alcohol use may lead to significantly improved treatment outcome for cocaine use disorders.
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REFERENCES

