Effects of Pretreatment and Posttreatment Depressive Symptoms on Alcohol Consumption Following Treatment in Project MATCH*

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ABSTRACT. Objective: This study examined the associations of pretreatment and posttreatment depressive symptoms with drinking outcomes in the year following treatment in Project MATCH (Matching Alcoholism Treatments to Client Heterogeneity), a multisite clinical trial of behavioral treatments for alcohol-use disorders. **Method:** Data from 1,726 participants were modeled using generalized estimating equations to examine drinking frequency and intensity, as reflected by percentage days abstinent (PDA) and average drinks per drinking day (DDD). We predicted that patients who reported more pretreatment and posttreatment depressive symptoms would report greater drinking frequency (PDA) and more intense drinking (DDD) across the 12-month follow-up period. **Results:** Pretreatment Beck Depression Inventory (BDI) scores predicted more frequent and intense drinking in the year following treatment, although not after accounting for posttreatment BDI scores, which were associated with the drinking outcomes as hypothesized. Patients who reported more depressive symptoms in the year following treatment reported less abstinence (PDA) and more intense drinking (DDD) than patients with fewer posttreatment depressive symptoms. **Conclusions:** Our findings underscore the importance of obtaining repeated assessments of depression during the course of substance use treatment. Moreover, the data suggest the potential utility of augmenting standard chemical dependency care with depression-focused interventions for alcohol-dependent patients whose depressive symptoms do not subside during treatment. (*J. Stud. Alcohol Drugs, 71,* 71-77, 2010)

INDEPENDENTLY, ALCOHOL DEPENDENCE and major depressive disorder constitute major public health problems in the United States (National Institute on Alcohol Abuse and Alcoholism, 2000). However, alcohol dependence and major depression often co-occur, compounding the impact of each individual disorder (Conway et al., 2006; Hasin et al., 2007; Kessler et al., 1997; Regier et al., 1990). Estimates from the National Co-morbidity Survey-Replication study (Kessler et al., 1997) indicate nearly one quarter (24.3%) of men and nearly one half (48.5%) of women with alcohol dependence experience a lifetime episode of major depression. Among those in treatment settings, the rates of co-occurrence are even higher, with estimates ranging from 50% to 70% (Cornelius et al., 1995; Curran and Booth, 1999).

Understanding the relationship between drinking behavior and depression, and how that relationship influences alcohol treatment outcomes, is crucial for the development of effective interventions for these patients (Schuckit, 2006). For example, recent evidence that untreated depression increases risk for drinking following alcohol treatment suggests that adjunctive depression-specific interventions might be helpful to improve drinking outcomes (Kodl et al., 2008). Knowing *when* to assess depressive symptoms and intervene accordingly with an adjunctive treatment is yet another crucial question to address to improve care.

Among individuals with alcohol-use disorders, co-occurring depression is associated with greater alcohol-related impairment and with greater participation in substance abuse treatment (Conner et al., 2009). Despite increasing the likelihood that alcohol-dependent patients may seek care, depression can interfere with patients' attempts at sobriety in a number of ways at various time-points in the treatment process. *Pretreatment* depressive symptoms are associated with higher rates of treatment drop-out (Curran et al., 2002),

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shorter time to first drink (Greenfield et al., 1998), lower rates of abstinence following treatment completion (Ilgen and Moos, 2005), and greater risk for suicide attempts (Ilgen et al., 2004). In addition, findings among treated alcohol-dependent patients point to the relationship between *post-treatment* depression and more rapid relapse for drinking problems following treatment (Glenn and Parsons, 1991; Curran et al., 2000; Curran et al., 2002; Svanum and McAdoo, 1989), greater nonabstinence, more drinking days per month, and more drinks per drinking day (Kodl et al., 2008). Longitudinal analyses conducted by Kodl and colleagues (2008) revealed that alcohol-dependent patients who reported at least mild to moderate levels of depressive symptoms (characterized by Beck Depression Inventory [BDI] scores ≥ 14) following treatment for alcohol and nicotine dependence were almost twice (1.67) as likely to report drinking at subsequent time points over an 18-month follow-up period.

But not all research has found an association between depressive symptoms and adverse drinking-related outcomes among individuals with alcohol-use disorders (Hesselbrock et al., 1985; Kranzler et al., 1996; Randall et al., 2001). In one study that investigated the extent to which depression diagnoses at pre-admission versus posttreatment predicted drinking outcomes following detoxification, no significant differences were found between patients with a diagnosis of depression at either time point and those with alcohol dependence alone (Davidson and Blackburn, 1998). Likewise, Conner and colleagues (2005) found that depressive symptoms at treatment entry were unrelated to drinking outcomes at the conclusion of treatment in Project MATCH (Matching Alcoholism Treatments to Client Heterogeneity). Interestingly, pretreatment depressive symptoms were, however, associated with drinking intensity and frequency during the first month of treatment (Conner et al., 2005).

A recent meta-analysis of published reports of depression and substance use and impairment among individuals with alcohol-use disorders (Conner et al., 2009) helps to integrate conflicting findings in the literature. The meta-analysis showed that depression is prospectively associated with subsequent alcohol use and impairment. However, when measures of alcohol use (e.g., relapse to drinking, drinking frequency) and alcohol-related impairment (e.g., alcohol withdrawal, alcohol-related consequences) were disaggregated, the results indicated that depression was predictive only of the latter. Thus, among individuals with alcohol-use disorders, depression predicts alcohol-related impairments but may not predict drinking per se. However, cautious interpretation is needed because insufficient data were available to examine different drinking outcomes (e.g., relapse to drinking, drinking frequency, drinking intensity), and it may be that depressive symptoms are relevant to some aspects of drinking and not others.

Indeed, the disaggregated results of the meta-analysis highlight the possibility that some of the discrepant findings that exist across studies may be due in part to the numerous ways in which drinking-related outcomes are defined (e.g., relapse to drinking, drinking frequency, drinking intensity, drinking-related consequences). Conflicting results in the literature may also be due in part to the various timeframes when depressive symptoms and drinking behaviors are assessed (e.g., pretreatment vs. posttreatment) or whether depressive symptoms emerge in the context of alcohol dependence, originate before an episode of alcohol dependence, or occur during a period of prolonged abstinence. Patient characteristics such as age and sex may also influence the relationship between depression and drinking behaviors (Marmorstein, 2009). Another possibility is that the relationship between depression and drinking may vary depending on the setting where patients are recruited (e.g., inpatient vs. outpatient; addiction vs. mental health treatment programs), the types of treatment investigated (i.e., medication, detoxication, cognitive-behavioral therapy), and the extent to which these and other potential confounding factors (e.g., demographics, lifetime depression, previous drinking) are controlled in analyses.

In this study, we examine the influence of pretreatment and posttreatment depressive symptoms in predicting drinking behavior in the year following treatment in Project MATCH, and we conservatively account for the influence of various demographic and treatment-related factors, worst lifetime depressive symptoms, and recent drinking behavior. Although a prior examination of Project MATCH data investigated the short-term relationship between patients' pretreatment depressive symptoms and their immediate drinking behavior at the conclusion of treatment (Conner et al., 2005), this is the first study to investigate the influence of pretreatment and posttreatment depressive symptoms on drinking outcomes for an extended year-long period following one of three empirically supported behavioral treatments for alcohol abuse and dependence. Unlike the Kodl et al. (2008) study, which compared pretreatment and posttreatment depressive symptoms among alcohol-dependent smokers receiving treatment for co-occurring alcohol and nicotine dependence, this study squarely focuses on drinking frequency and intensity following alcohol treatment while rigorously controlling for potential confounding factors such as lifetime depression and prior drinking behavior. We hypothesize that patients who report more depressive symptoms at treatment entry will report greater drinking frequency (lower percentage days abstinent [PDA]) and more intense drinking (DDD) across the 12-month follow-up period. We expect a similar pattern will emerge for those who report greater posttreatment depressive symptoms.

Method

Sample

The data are from Project MATCH (Project MATCH Research Group, 1993), a multisite, randomized clinical trial

of three behavioral treatments for patients with alcohol-use disorders. Participants were eligible for Project MATCH if they met Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised (DSM-III-R), criteria for either alcohol dependence or alcohol abuse (American Psychiatric Association, 1987), reported alcohol as their primary substance of use, were 18 years of age or older, and were capable of reading at a sixth-grade level. Exclusion criteria were intravenous drug use; dependence on sedative/hypnotic drugs, stimulants, cocaine, or opiates; acute psychosis or severe organic impairment; and high-risk for suicidal behavior (Project MATCH Research Group, 1993). The total sample size for Project MATCH was 1,726. All participants were included in the current study.

Procedure

Detailed descriptions of Project MATCH procedures have previously been reported (Project MATCH Research Group, 1993). In brief, Project MATCH was designed to investigate the extent to which patient characteristics and treatment type interact to predict outcomes following a behavioral treatment for alcohol problems. Patients were drawn from two samples: an outpatient arm (n = 952), composed of patients who had not recently completed any inpatient treatment, and an aftercare arm (n = 774), composed of patients who had received 7 days or more of inpatient or intensive day treatment immediately before assignment to outpatient care. Participants completed a structured diagnostic interview, an in-depth interview about drinking behavior, and a battery of self-report measures at study entry. They were then randomly assigned to one of three treatments conducted over a 3-month period: 4 sessions of motivational enhancement therapy (Miller et al., 1992), 12 sessions of cognitive-behavioral therapy (Kadden et al., 1992), or 12 sessions of twelve-step facilitation (Nowinski et al., 1992). Participants were re-assessed at the end of the active treatment phase and again at multiple time points in the year following discharge from care (Babor and Del Boca, 2003). This study uses all available drinking and depression data from the baseline interview through the 12-month follow-up period.

Measures

Covariates. Demographic information including participant age, sex, and race/ethnicity was obtained from all patients before treatment assignment. At baseline, participants also completed the Computerized Diagnostic Interview Schedule–Revised (CDIS-R; Robins et al., 1989) to establish a DSM-III-R diagnosis of alcohol abuse or dependence and to assess other current and lifetime mental disorders, including worst lifetime major depression. For the purposes of this study, worst lifetime depression was calculated by the sum of clinically significant depressive symptoms that patients endorsed during the worst depressive episode of their lives. Scores on the CDIS-R worst lifetime depression scale ranged from 0 to 9.

Pretreatment and posttreatment depression symptoms. The 21-item Beck Depression Inventory (BDI; Beck, 1978) was used to measure depression symptom severity throughout the study period. The BDI is a reliable and valid way to assess depression severity among patients with alcohol-use disorders (Brown et al., 1997).

Drinking intensity and frequency. We calculated average DDD and PDA to quantify drinking intensity and frequency respectively (Babor et al., 1994; Project MATCH Research Group, 1997). DDD and PDA were derived from data collected on the Form 90 (Miller, 1996; Miller and Del Boca, 1994). Baseline drinking behavior was based on a summary assessment of DDD and PDA in the 90 days before study entry. DDD and PDA for each of the follow-up assessments were calculated using the data of the last interview as the starting point.

Data analysis

Preliminary analyses using generalized estimating equations (GEE) were first conducted to assess whether the associations among pretreatment and posttreatment depressive symptoms and drinking behaviors (PDA, DDD) differed between men and women. To test the potential moderating effects of sex, we included Sex × Depression interaction terms (i.e., Sex × Pretreatment Baseline BDI and Sex × Posttreatment BDI) in each of the models detailed below to determine whether subsequent model testing should be conducted separately for women and men. Because results from these preliminary analyses indicated that sex did not moderate the association between depression and either of the drinking outcomes (p values ranged from .4706 to .988), we proceeded with analyses that combined men and women in one sample.

GEE approaches were used to assess the effects of pretreatment and posttreatment depressive symptoms on drinking behaviors (PDA, DDD) in the year following treatment in Project MATCH. To test our hypotheses, we first specified a model where PDA and DDD at each of the follow-up interviews were modeled as conditional on pretreatment (baseline) BDI scores. Next, we specified a model where PDA and DDD at each of the follow-up interviews were analyzed as conditional on both pretreatment and posttreatment BDI scores. Additional lagged analyses were conducted to investigate whether BDI scores assessed at the conclusion of treatment predicted PDA and DDD 6 and 12 months later. In each these models, sex, age (continuous), race (White vs. non-White), study arm (outpatient vs. aftercare), treatment group (motivational enhancement therapy, cognitive-behavioral therapy, twelve-step facilitation), baseline drinking (PDA, DDD), and worst lifetime depressive symptoms were

included as covariates. Finally, in PDA models concurrent DDD was controlled and in DDD models concurrent PDA was controlled. All variables were entered simultaneously and all tests were two-tailed with α set at .05.

Use of GEE is an appropriate approach for our purposes given the nonnormal distribution of the drinking data and the significant correlations between drinking behaviors from one assessment period to the next (Kowalski and Tu, 2007). The increased likelihood of drinking (i.e., PDA or DDD) per 1-unit increase in BDI score was quantified by exponentiating the beta estimates and standard errors from the GEE models. The potential effect of missing data was investigated by modeling participants' missing data under the missing-at-random assumption (Little and Rubin, 1987) using logistic regression. Our results indicated that the probability of patients' missing data did not depend on observed responses, making GEE appropriate. The SAS GENMOD procedure was used to estimate and test all models (SAS Institute, 2003).

Results

Study sample

Project MATCH participant characteristics have been described in detail in previous reports (Project MATCH Research Group, 1997). In brief, our sample of 1,726 participants had a mean (SD) age of 40.3 (11.0) years; 1,391 (80.6%) were White and 1,310 (75.9%) were male. The majority of patients met diagnostic criteria for alcohol dependence (95% in the outpatient arm, 98% in the aftercare arm) as opposed to alcohol abuse according to DSM-III-R guidelines.

Mean (*SD*) depression symptom severity and drinking behavior (nontransformed) at each of the study time points are presented in Table 1. Comparing pretreatment and posttreatment scores of the overall sample, PDA increased nearly threefold (271%), and DDD decreased by more than one half (59%). Data collected 6 and 12 months after treatment suggest these gains were maintained for the overall sample. Mean BDI scores declined more than a quarter (26%) from before treatment to after treatment and were significantly correlated ($r = .55$, $p < .0001$).

Table 1. Descriptive data on drinking and depression variables (n = 1,726)

Measure	Pre-tx *M* (*SD*)	Post-tx *M* (*SD*)	6-mo. follow-up *M* (*SD*)	12-mo. follow-up *M* (*SD*)
BDI scores	10.17 (8.24)	7.57 (7.87)	7.78 (7.96)	7.94 (8.40)
PDA	0.31 (0.30)	0.84 (0.25)	0.77 (0.31)	0.76 (0.33)
DDD	16.62 (10.62)	6.85 (8.80)	6.60 (8.29)	6.09 (7.88)

Notes: Pre-tx = pretreatment; post-tx = posttreatment; mo. = month; BDI = Beck Depression Inventory; PDA = percentage days abstinent; DDD = drinks per drinking day.

Results from the GEE analyses are presented in Table 2. The top half of the table presents parameter estimates for our initial model examining the independent effects of pretreatment BDI on PDA and DDD in the year following treatment, after adjusting for covariates. The bottom half of the table includes parameter estimates for PDA and DDD once posttreatment depression scores were included in the multivariate model.

Covariates

The effects of sex, age, race, study arm, treatment group, worst lifetime depressive symptom scores, and drinking behavior at the previous assessment were controlled in all analyses. Of these variables, study arm ($b = -0.1156$, $p < .001$), baseline PDA ($b = 0.3423$, $p < .001$), baseline DDD ($b = 0.0028$, $p < .001$), and concurrent DDD ($b = -0.0128$, $p < .001$) significantly predicted PDA during the year-long follow-up period. Aftercare patients reported less abstinence during the follow-up period, as did those who reported less abstinence and more intense drinking at baseline and who drank more intensely during the follow-up period. In models that examined DDD, age ($b = -0.0051$, $p < .05$), study arm ($b = 0.1305$, $p < .01$), sex ($b = 0.2081$, $p < .001$), baseline PDA ($b = 0.5634$, $p < .001$), baseline DDD ($b = 0.0319$, $p < .001$), and concurrent PDA ($b = -1.0858$, $p < .001$) were significant. Aftercare patients drank more intensely during the follow-up period, as did those who were older and male. Individuals who reported less abstinence and more intense drinking at baseline and who were less abstinent during the follow-up period consumed more alcohol when they did drink. Worst lifetime depressive symptom scores were not significantly associated with either PDA or DDD.

Pretreatment depressive symptoms and drinking outcomes

Consistent with our hypothesis, initial models indicated that, after adjustment for covariates, pretreatment BDI scores were significantly associated with PDA and DDD in the year following treatment. Patients with greater baseline depressive symptoms drank more frequently and intensely in the year following treatment than those with fewer baseline depressive symptoms. However, once posttreatment depressive symptoms were added to the model, the associations between pretreatment BDI, PDA, and DDD became nonsignificant.

Posttreatment depressive symptoms and drinking outcomes

As the results in the bottom half of Table 2 indicate, findings supported our hypothesis. Posttreatment depressive symptoms were associated with drinking frequency and intensity following behavioral treatment for alcohol-use disorders. Patients who experienced greater depressive symptoms

TABLE 2. Predictors of drinking behaviors following alcoholism treatment (n = 1,726)

	PDA Model 1			DDD Model 2		
Parameters	Coefficient	Standard error	*Z* score	Coefficient	Standard error	*Z* score
Initial model of pretreatment variables						
Pre-tx BDI	-0.0024	0.0011	-2.16*	0.0082	0.0033	2.48*
Pre-tx PDA	0.3453	0.0253	13.65***	0.5304	0.0895	5.93***
Pre-tx DDD	0.0025	0.0008	2.98***	0.0319	0.0026	12.44***
Model with posttreatment variables added						
Pre-tx BDI	0.0000	0.0011	0.01	-0.0006	0.0035	-0.17
Pre-tx PDA	0.3423	0.0249	13.75***	0.5634	0.0899	6.27***
Pre-tx DDD	0.0028	0.0008	3.51***	0.0319	0.0026	12.18***
Post-tx BDI	-0.0069	0.0009	-7.49***	0.0212	0.0023	9.43***
Post-tx PDA	–	–	–	-1.0858	0.0574	-18.93***
Post-tx DDD	-0.0128	0.0011	-11.41***	–	–	–

Notes: Unstandardized coefficients with standard errors are shown. All models are adjusted for the following baseline variables: race/ethnicity, age, sex, study arm, treatment assignment, and worst lifetime depressive symptoms. PDA = percentage days abstinent; DDD = drinks per drinking day; pre-tx = pretreatment; BDI = Beck Depression Inventory; post-tx = posttreatment.
*$p < .05$; ***$p < .001$.

in the year following treatment reported fewer days abstinent and consumed more drinks on those nonabstinent days than those with fewer depressive symptoms.

Using the parameter estimates derived from each of the models tested, we calculated the change in PDA and DDD associated with each 1-unit increase in BDI by exponentiating its estimate. Specifically, a 1-unit increase in posttreatment BDI is associated with a 0.993-unit decrease in PDA ($p < .0001$) during the year-long follow-up period and a 1.021-unit ($p < .0001$) increase in DDD ($p < .0001$). Lagged analyses were conducted to evaluate the extent to which depressive symptoms reported at posttreatment assessment predicted drinking at the 6- and 12-month follow-up points. Results from these analyses showed that patients who reported a one-unit increase in depressive symptoms immediately following treatment reported an average of 1.014 more drinks per day 6 and 12 months later ($p <.0001$).

Discussion

Among patients with alcohol-use disorders, pretreatment and posttreatment depressive symptoms decrease abstinence and increase drinking intensity in the year following specialized treatment for alcohol-use disorders. When compared, posttreatment depressive symptoms are more predictive of drinking behavior in the year following treatment than the depressive symptoms that patients report before beginning alcohol treatment. This suggests that when concluding treatment with patients with alcohol-use disorders, clinicians should include an assessment of depression as an important component of relapse prevention. Moreover, the findings highlight the potential value of augmenting standard chemical dependency care with depression-focused interventions for alcohol-dependent patients with co-occurring depression.

The data indicate that individuals who conclude treatment with higher levels of depressive symptoms drink more often and more intensively in the year following treatment than patients with fewer symptoms. These results replicate and extend findings from previous reports (Curran et al., 2000; Curran et al., 2002; Kodl et al., 2008) in an ethnically diverse group of men and women who have undergone one of three empirically supported behavioral treatments for their alcohol-use disorders. It also bears considering that for the majority of patients, treatment (regardless of the type) was successful and greatly reduced their drinking at the conclusion of therapy and over the 12-month follow-up period. Nevertheless, when lapses occurred, posttreatment depressive symptoms were associated with them, but pretreatment BDI scores and worst lifetime depressive symptoms were not.

In contrast to prior reports that have not found a causal relationship between depression and drinking (Hodgins et al., 1999), results from our lagged analyses confirm that depressive symptoms at the conclusion of treatment predict subsequent drinking 6 and 12 months later. Taken together, these findings point to a role that posttreatment depressive symptoms may play in increasing risk for drinking behavior and vulnerability to relapse following treatment for substance use—even when the treatment has been largely successful.

What is unclear from the present findings is *why* posttreatment depressive symptoms are related to drinking intensity

and frequency in the year following treatment. Some theorists propose that depressed chemically dependent patients use alcohol and other substances to "self-medicate" their depressive symptoms and regulate their emotions (Khantzian, 1985). However, the majority of research indicates that depressive symptoms do not improve when patients begin drinking again following substance use treatment (Hodgins et al., 1999). Instead, a "rebound effect" may occur in which drinking produces or incites psychiatric symptoms rather than diminishing them (Blume et al., 2000; Tomlinson et al., 2006). Another possible explanation may be that a third factor, such as interpersonal difficulties, underlies the relationship between drinking problems and depressive symptoms. Given that relationship disruptions and conflicts are one of the most often-cited reasons for drinking relapse (Connors et al., 1998) and are also common triggers for depression (Brown and Harris, 1978), it is conceivable that as the newly abstinent patient attempts to re-establish a social network that supports his/her sobriety, the conflicts and tensions that arise could trigger drinking, depression, or both. Additional studies are needed to understand this association and clarify the mechanisms through which depression and chronic drinking problems are related (Schuckit, 2006).

Limitations

This study does not explore the possibility of reciprocal effects of drinking on depressive symptoms. Although well validated, the measures of drinking and depressive symptoms relied on participants' self-reports. In addition, no provision was made to assess whether the depressive symptoms endorsed on the BDI were the result of the expected effects of intoxication or withdrawal (Hasin et al., 1996). Moreover, the BDI does not inquire about the onset of depressive symptoms in relation to the onset of one's alcohol-use disorder—an important area for future research. Strengths of the study include a large sample of diverse patients with alcohol-use disorders who received one of three empirically supported treatments for substance use, a year-long follow-up period of repeated standardized assessments of drinking behavior and depressive symptoms, and complex statistical modeling procedures that were robust to issues of missing data and skewed distributions. It should also be emphasized that the effects were observed using a conservative statistical approach that controlled patients' previous levels of drinking, which are by far the strongest predictor of subsequent drinking. Additionally the models controlled for lifetime depression, treatment condition, study arm, and demographic variables known to be associated with drinking problems following treatment. This conservative analytical approach lends added support for our finding linking posttreatment depressive symptoms to increased risk for drinking relapse following treatment.

Summary

Given our findings that posttreatment depressive symptoms are associated with increased drinking following alcohol treatment, clinicians may consider including an assessment of depression before discharging patients from care. By targeting co-occurring depression among those with alcohol-use disorders, we will likely be better positioned to improve longer-term abstinence and overall functioning.

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