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Psychosocial treatments for cocaine dependence: The role of depressive symptoms

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ABSTRACT

Background: The association between cocaine use and depression has been frequently observed. However, less is known about the significance of depression in the treatment of cocaine use disorders. This study examined possible interrelations between drug use and depression severity among cocaine-dependent patients in psychosocial treatments for cocaine dependence.
Methods: Monthly assessed drug use and depression severity scores of $N = 487$ patients during 6-month psychosocial treatments for cocaine dependence were analyzed using hybrid latent growth models.
Results: Results indicated a moderate but statistically significant ($z = 3.13$, $p < .01$) influence of depression severity on increased drug use in the upcoming month, whereas drug use did not affect future depression severity.
Conclusions: Findings suggest that depression symptoms are an important predictor of drug use outcomes during psychosocial treatments for cocaine dependence and, hence, underline the importance of adequately addressing depression symptoms to improve treatment outcomes.

1. Introduction

Cocaine use is often associated with depression (Falck et al., 2004). Among treatment-seeking cocaine users, depression is one of the most common non-substance use disorders (Kleinman et al., 1990; Rounsaville et al., 1991) and even more of them are likely to suffer from subclinical levels of depressive symptomatology that do not justify diagnosis of depression but that might be clinically important nevertheless. Furthermore, there seems to be a specific trend for treatment-seeking cocaine abusers to have higher rates of depression than untreated community control subjects with cocaine abuse (Carroll and Rounsaville, 1992; Ford et al., 2009). Therefore, clinicians frequently encounter patients presenting with both cocaine abuse or dependence and depressive psychopathology.

Effective psychosocial treatments for cocaine dependence are available (Carroll, 2005; Dutra et al., 2008; Woody, 2003). For example, in the largest study done to date, in the National Institute on Drug Abuse (NIDA) Collaborative Cocaine Treatment Study (CCTS), all four treatments under examination–individual drug counseling, group drug counseling, and two types of professional psychotherapy–produced statistically and clinically significant improvements in terms of reducing cocaine use as well as overall drug use, with a pattern of greater clinical benefit for the group randomized to receive both group and individual drug counseling (Crits-Christoph et al., 1999). However, with a few exceptions (e.g., Brown et al., 1998), previous research has also shown the clinical significance of concurrent depression for the treatment of cocaine abuse or dependence (e.g., Hasin et al., 2002; Ziedonis and Kosten, 1991). Some of these studies found concurrent depression to be associated with positive (e.g., McKay et al., 2002), others with negative (e.g., Carroll et al., 1993) treatment outcomes. Whatever may apply, these findings suggest that the frequently high acute levels of depression in cocaine users when entering treatment warrant clinical attention and point to the need to better understand the role of depression symptoms in the treatment of cocaine dependence.

To explain the common association between substance use disorders and depression, different hypotheses have been formulated (Kosten et al., 1998; Mueser et al., 1998; Rounsaville, 2004). These models include: (a) depression as a cause of cocaine use (e.g., a high sensitivity of depressed patients to even small amounts of drugs may lead to protracted drug use and drug use disorders, or use of cocaine may represent as a self-medication coping response to manage depression symptoms) (Khantzian, 1985; Markou et al., 1998; Uslander et al., 1999); (b) cocaine use as a cause of depression (e.g., use of cocaine may lead to stressful

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life events that in turn promote depression, or pharmacological effects of cocaine may depress mood after prolonged or excessive use) (Kendler et al., 2003; Kosten et al., 1998; Weiss et al., 1992); (c) common risk factors (e.g., neuroticism, genetics, or antisocial personality traits) that represent shared vulnerabilities for both substance use and depression (Khan et al., 2005); and (d) bidirectional models hypothesizing that ongoing interactional effects account for the common association between substance use and depression (Mueser et al., 1998). There is at least partial support for all of these explanatory models, but none of them has unequivocal support for explaining all cases pointing to the need to further explore the relationship between the causes and course of cocaine use and depression during treatment (Mueser et al., 1998; Rounsaville, 2004). Following the recommendation of Mueser et al. (1998) that multiple assessments of substance use and affect over time (e.g., monthly) would provide much richer data for evaluating models of comorbidity, we used new statistical methods, so-called hybrid latent growth (HLG) models (Bollen and Curran, 2004), to carefully examine the relationships and the reciprocal influences of monthly assessed drug use and depression severity during 6-month psychosocial treatments for cocaine dependence.

1.1. Hybrid latent growth models

Traditionally there have been two broad classes of models for the analysis of longitudinal panel data: autoregressive (AR) models assume time-lagged effects of a variable on itself and examine these effects by the regression of a variable on its earlier value. Whereas these AR models allow the prior value to determine the current value of the same variable, they do not account for person-specific change trajectories (i.e., individual differences in change). In contrast, latent growth curve (LGC) models account for person-specific change trajectories by allowing each case in the sample to have a different time trend as marked by a different intercept or slope. However, such LGC models do not simultaneously allow the prior value to determine the current value of the same variable or cross-lagged effects between multiple outcome variables.

In the past, the AR and the LGC models have often been perceived as competing and mutually exclusive methodologies for the analysis of change. However, recently introduced HLG models take advantages of both traditions through the incorporation of AR and LGC parameters into a more flexible structural equation modeling (SEM) framework (Bollen and Curran, 2004). Moreover, if there are multiple parallel processes, these HLG models may also include cross-lagged effects between different repeatedly assessed outcome variables. Conceptually these more encompassing HLG models estimate the AR, cross-lagged and LGC models simultaneously with one set of fit indices to evaluate the adequacy of the entire model. Furthermore, like LGC models, these HLG models can also include time-invariant variables (e.g., treatment type) as covariates to estimate their effects on developmental trajectories (e.g., change in drug use or depression severity).

In this study, we therefore used HLG models in a reanalysis of the NIDA CCTS data set to carefully examine the relationships and the reciprocal influences of cocaine use and depressive symptoms during psychosocial treatments for cocaine dependence.

2. Methods

2.1. Design and procedures

The design and procedures of the NIDA CCTS are detailed elsewhere (Crits-Christoph et al., 1997, 1999). Briefly, the NIDA CCTS was a multi-site randomized clinical trial that compared the efficacy of four psychosocial treatments for cocaine dependence: In two of these treatments, professional psychotherapy, either cognitive therapy (CT) or supportive-expressive psychodynamic therapy (SE), was added to group drug counseling (GDC). A third treatment combined individual drug counseling (IDC) with GDC, and the fourth consisted of GDC alone. All treatments were planned to include 6 months of active phase treatment and a 3-month booster phase. Cognitive therapy followed a detailed manual for CT of substance abuse or dependence (Beck et al., 1993). This treatment is based on the assumption that substance use disorders are related to individual's maladaptive beliefs and related thought processes. Among the treatment techniques used are Socratic questioning, advantages-disadvantages analysis, monitoring of drug-related beliefs and activities, behavioral experiments, and role playing. Brief SE psychodynamic therapy followed the general SE treatment manual by Luborsky (1984) with modifications for cocaine dependence (Mark and Luborsky, 1992). In this treatment, the problems associated with the use of cocaine and with its cessation are viewed in the context of understanding the person's interpersonal and intrapsychic functioning and they are addressed by supportive and interpretive techniques. Individual drug counseling followed a manual with specific stages, tasks, and goals based on the 12-step philosophy (Mercer and Woody, 1992). It focuses primarily on helping the patient achieve and maintain abstinence by encouraging behavioral changes, such as avoiding drug triggers, structuring one's life, and engaging in healthy behaviors (e.g., exercise). Group drug counseling followed a manual designed to educate patients about the stages of recovery from addiction, to strongly encourage participation in 12-step programs, and to provide a supportive group atmosphere for initiating abstinence and an alternative lifestyle (Mercer et al., 1994). Individual treatment sessions were held twice per week during the first 12 weeks, weekly during weeks 13–24, and monthly during the booster phase. Group drug counseling sessions were held weekly during the active phase treatment and patients in the GDC alone condition met with the group counselor individually once per month during the booster phase.

2.2. Patients

A total of $N = 487$ outpatients, all of them having a principal diagnosis of cocaine dependence according to the 4th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994) and all of them using cocaine during the past 30 days, were randomly assigned to one of the four treatment conditions. Characteristics of these patients are found in Table 1. As shown there, more than half (55%) of the cocaine-dependent patients in the sample reported at least mild-moderate depression at treatment entry. 147 patients were diagnosed with mood disorders, most of them ($n = 139$) with a cocaine-induced mood disorder. Exclusion criteria are reported in detail elsewhere (Crits-Christoph et al., 1999). Relevant to the current analyses is that history of Bipolar I disorder, imminent suicide risk, and need to be maintained on a psychotropic medication were exclusion criteria (thus, no patients were receiving antidepressant medication).

The average number of individual treatment sessions attended differed significantly between treatment conditions, $F(2,361) = 5.7$, $p < .01$. Patients in IDC + GDC attended significantly fewer sessions ($M = 11.9$, SD = 10.5) than patients in CT + GDC ($M = 15.5$, SD = 10.6) and SE + GDC ($M = 15.7$, SD = 11.3). The mean number of group treatment sessions attended was 8.6 (7.2) in IDC + GDC, 9.5 (7.2) in CT + GDC, 8.8 (6.8) in SE + GDC, and 8.6 (7.2) in GDC alone, $F(3,483) = 0.6$, n.s. Further details on therapies and therapists can be found in previous publications on the study (Crits-Christoph et al., 1997, 1999).