Contingency Management Targeting Abstinence is effective in Reducing Depressive and Anxiety Symptoms among Crack Cocaine-Dependent Individuals

André Q. C. Miguel, Ph.D[1,2,*], **Clarice S. Madruga, Ph.D.**[1,2], **Hugo Cogo-Moreira, Ph.D.**[2], **Rodolfo Yamauchi**[1], **Viviane Simões**[1], **Ariadne Ribeiro, MSc**[1,2], **Claudio J. da Silva, Ph.D.**[1], **Andrew Fruci**[3], **Michael McDonell, Ph.D.**[3], **Sterling McPherson, Ph.D.**[3], **John M. Roll, Ph.D.**[3], and **Ronaldo R. Laranjeira, Ph.D.**[1,2]

[1]National Institute of Policies on Alcohol and Drugs (INPAD) of the Department Psychiatry and Medical Psychology of the Federal University of São Paulo (UNIFESP), Brazil

[2]Department of Psychiatry and Medical Psychology of the Federal University of São Paulo-UNIFESP, Brazil

[3]Program of Excellence in Addictions Research, Washington State University

Abstract

Although contingency management (CM) is effective in promoting abstinence and treatment retention among crack cocaine users who meet the criteria for cocaine dependence, less is known about its off-target effects. In this secondary analysis, we evaluated the impact of CM on depressive and anxiety symptoms in a sample of cocaine-dependent individuals under treatment.

Sixty-five crack cocaine users who met the criteria for cocaine dependence were randomly assigned to receive 12 weeks of standard treatment alone (STA; $n = 32$) or 12 weeks of standard treatment plus CM (STCM; $n = 33$). The outcome measures of the secondary analysis were depressive and anxiety symptoms assessed with the Beck Depression Inventory-II (BDI-II) and the Beck Anxiety Inventory (BAI).

At baseline, 59 (90.8%) of the participants reported at least mild depressive symptoms and 47 (72.5%) reported at least mild anxiety symptoms. The mean BDI-II (24.5 ± 12.1) and BAI (20.7 ± 13.5) scores in the sample as a whole was moderate. After treatment, the reported levels of depressive symptoms ($\beta = -9.6$, $p < .05$) and anxiety symptoms ($\beta = -9.9$, $p < .05$) were lower among the individuals receiving STCM than among those receiving STA.

This study provides evidence that an STCM intervention targeting crack cocaine abstinence also produces significant reductions in depressive and anxiety symptoms. This low cost intervention also demonstrated significant promise and optimization potential for crack cocaine users in a setting of scarce resources and high mental health comorbidity.

*Corresponding concerning this article should be addressed to: André Q. C. Miguel at Rua Fransisco Leitão 469, conj 612, Pinheiros, CEP 05414-020; São Paulo, SP, Brasil. Tel. (0055) 11 96588-4111. aqcmiguel@gmail.com.

Keywords

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Mood and anxiety disorders are highly prevalent among cocaine users (Grant et al., 2004; Tang, Kranzler, Gelernter, Farrer, & Cubells, 2007). It is estimated that 36% of adults with cocaine use disorders and 48% of adults with cocaine dependence will suffer from major depression during their lifetime (Conway, Compton, Stinson, & Grant, 2006). Similarly, 32% of adults with cocaine use disorders and 45% of adults with cocaine dependence will suffer from an anxiety disorder (Conway et al., 2006). A cross-sectional study of 430 active crack cocaine users found that 80% had symptoms of depression, and that 55% had either moderate or severe depression (Falck, Wang, Carlson, Eddy, & Siegal, 2002). Likewise, a cross-sectional study of crack cocaine users admitted to a psychiatric hospital in Brazil found that 84% had ether moderate or severe levels of anxiety symptoms (Zubaran, Foresti, Thorell, & Franceschini, 2013).

Contingency management (CM) is among the most effective behavioral treatments for dependence on cocaine and other stimulants. It has also been demonstrated to be highly effective at promoting treatment retention and continuous abstinence among cocaine users (Dutra et al., 2008; Lussier, Heil, Mongeon, Badger, & Higgins, 2006). In a recent clinical trial conducted by (Miguel et al., 2016), CM was found to be associated with abstinence from crack cocaine use (OR = 18.6, $p < .05$) and treatment retention (OR = 68.9, $p < .05$) in a population of crack cocaine users in the city of Sao Paulo, Brazil. It has consistently been shown that CM is associated with secondary benefits such as reduced substance use and amelioration of mental health symptomatology (Higgins et al., 2003; McDonell et al., 2014; McDonell et al., 2013; Miguel et al., 2016; Petry, Alessi, & Rash, 2013).

In a sample of 176 adult cocaine users with concomitant serious mental illness, our research group observed that a CM intervention was associated with reductions in psychiatric symptoms, and that CM-treated subjects were five times less likely to be hospitalized for a psychiatric condition than were controls (McDonell et al., 2013). In a population of individuals without mental illness (393 participants in three randomized trials), (Petry et al., 2013) observed that CM targeting cocaine use was associated with overall reductions in psychiatric symptoms, specifically depressive and anxiety symptoms. Although those studies suggest that CM is effective in reducing psychiatric symptoms among cocaine users, they were both conducted in the United States and it is unknown whether these effects would be replicated in low-income countries. In Brazil, crack cocaine use represents a major public health problem and is strongly associated with school dropout, unemployment, homelessness, exposure to violence, psychiatric comorbidities, prostitution, HIV infection, hepatitis B, hepatitis C, syphilis, engagement in illegal activities, incarceration, and mortality (Dunn & Laranjeira, 1999; Narvaez et al., 2014; Ribeiro, Dunn, Laranjeira, & Sesso, 2004; Santos Cruz M et al., 2013). In addition, data on treatment effectiveness in this population are scarce, coming from a small number of studies with major methodological limitations. It is noteworthy that the (Miguel et al., 2016) study was the first randomized

controlled trial to evaluate the impact of a psychosocial intervention for crack cocaine dependence in Brazil. Therefore, studies addressing the impact of evidence-based psychosocial interventions for crack cocaine-dependent individuals in Brazil are crucial to the development of effective treatments for this population. Although there have been few studies comparing the treatment response of crack cocaine users among countries with different economic, social, and treatment resources, it is important to determine whether treatments proven effective in high-income countries can also be effective in low-income countries.

Although the prevalence rates of lifetime depressive and anxiety disorders are comparable between users of powder cocaine and users of crack cocaine (Kiluk, Babuscio, Nich, & Carroll, 2013), the latter show poorer treatment responses, with higher dropout rates and lower post-treatment abstinence rates (Ferri, Gossop, Rabe-Hesketh, & Laranjeira, 2002; Gossop, Marsden, Stewart, & Kidd, 2002; Hser, Joshi, Anglin, & Fletcher, 1999; Kiluk et al., 2013; Rowan-Szal, 2000). This evidence underscores the need to investigate the effect of CM on depressive and anxiety symptoms in crack cocaine users.

The purpose of this study, conducted in a low-resource setting in the city of Sao Paulo, was to determine whether the post-treatment severity of depressive and anxiety symptoms was lower among adult crack cocaine users receiving the standard treatment plus CM than among those receiving the standard treatment alone.

Method

Design

The present study utilized data collected in a randomized clinical trial designed to evaluate the effect of CM on crack cocaine use, the duration of abstinence from crack cocaine, treatment attendance, and treatment retention in a population of treatment-seeking crack cocaine-dependent individuals in the city of Sao Paulo (Miguel et al., 2016).

Participants

Individuals were screened for eligibility only after having been diagnosed with cocaine dependence—according to the Diagnostic and Statistical Manual of Mental Disorders, Fourth edition (DSM-IV), as assessed with the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I) (First, 1997). All screened individuals were between 18 and 65 years of age and were seeking treatment at the Vila Maria Specialized Medical Outpatient Clinic for Alcohol and Drug Treatment, in the city of Sao Paulo. Polydrug users were eligible for enrollment if crack cocaine was their drug of choice. Individuals who had not used crack cocaine in the last 4 weeks were excluded, as were those who had a SCID-I-confirmed diagnosis of schizophrenia and those who were unable to attend treatment sessions at least three times a week. Sixty-five individuals met the inclusion criteria and were enrolled in the study. The study was approved by the Research Ethics Committee of the Federal University of Sao Paulo and by the Ethics Committee of the Brazilian National Ministry of Health (CAAE no: 00745912.4.0000.5505). All participants provided written informed consent.

Procedures

The baseline assessment included the following instruments: the SCID-I, to screen for a DSM-IV diagnosis of substance abuse/dependence or schizophrenia (no other psychopathologies were assessed with the SCID-I); the Minnesota Cocaine Craving Scale (Focchi GRA, 2001); the Cocaine Craving Questionnaire-Brief (Araujo, 2010); the Alcohol, Smoking, and Substance Involvement Screening Test (Group, 2002); the Barratt Impulsiveness Scale (von Diemen, Szobot, Kessler, & Pechansky, 2007); the Beck Depression Inventory-II (Gorenstein C, 2011); and the Beck Anxiety Inventory (Cunha, 2001). After completing the initial intake assessments, each participant provided a urine sample (to assess cocaine and marijuana use) and a breath sample (to assess recent alcohol use). Post-treatment assessments were conducted at week 12 of treatment, with the same instruments listed above.

Treatment

We used permuted-block randomization to allocate participants to one of two treatments: 12 weeks of standard treatment alone (STA, $n = 32$), or 12 weeks of standard treatment plus CM (STCM group, $n = 33$). Participants were stratified by a baseline diagnosis of alcohol dependence.

The STA approach consisted of weekly, 90-min group therapy sessions focusing on relapse prevention and coping skills training; weekly, 90-min group occupational therapy sessions; at least one individual session per month with a psychiatrist; and no more than one individual psychotherapy session per week. Participants were asked to submit urine and breath samples three times per week (Mondays, Wednesdays, and Fridays). After submitting a biological sample, participants were immediately informed of the results. No form of monetary incentive was offered to the STA group participants.

The STCM approach was identical to the STA approach except that STCM group participants could earn vouchers for submitting samples that tested negative for crack cocaine. For the first crack cocaine-negative sample, STCM participants earned US$1.25. Voucher values increased by US$0.50 for each consecutive crack cocaine-negative sample, up to a maximum of US$3.75. An extra US$5.00 was given if a participant was abstinent from crack cocaine for an entire week. If testing negative for crack cocaine, participants could earn an additional US$0.50 for submitting a alcohol-negative breath sample. Participants who were abstinent from crack cocaine, alcohol, and marijuana for an entire week earned an additional US$2.50 in vouchers. Participants testing positive for crack cocaine earned no vouchers for testing negative for alcohol or marijuana. If abstinent from all substances for the entire 12 weeks of treatment, STCM participants could earn a total of US$235.50. Participants could exchange vouchers for goods immediately after submitting a crack cocaine-negative sample. Vouchers could be exchanged for any goods available in the surrounding community, with the exceptions of tobacco products and alcohol. A full description of the methodology employed is available elsewhere (Miguel et al., 2016).

Statistical Analyses

For all baseline assessments, between-group comparisons were conducted with chi-square tests for dichotomous variables and *t*-tests for continuous variables. To assess the fully adjusted impact of the STCM intervention on depressive symptoms, we used multiple linear regression with group assignment as the primary predictor of interest, controlling for age, gender, alcohol dependence, and baseline BDI-II score as covariates, together with post-treatment BDI-II score as the primary outcome.

To determine the impact of the STCM intervention on anxiety symptoms, we conducted a multiple linear regression with group assignment as the primary predictor of interest, controlling for age, gender, alcohol dependence, and baseline BAI score as covariates. Post-treatment BAI score was the primary outcome.

We also explored the impact of the total numbers of crack cocaine-negative samples, alcohol-negative samples, and marijuana-negative samples submitted during the treatment period as additional predictors. However, when we included those in our model, there was a problem with multicollinearity, such that each of these predictors had a variance inflation factor of 16, none of them were significant, and the next largest variance inflation factor was 1.6. Therefore, we opted not to include the additional predictors in our final analysis.

The proportion of missing data was considerable (35.4%): 34.4% in the STA group and 36.4% in the STCM group. To comply with the intention-to-treat principle and based on current expert recommendations, we used multiple imputation to handle missing data in the final multiple linear regression model. Using Rubin's rules, we created and pooled 50 imputed datasets to produce aggregate covariate estimates (D. B. Rubin, 1996). The missing data were assumed to be more consistent with the missing-at-random assumption than with the missing-not-at-random assumption, because the reasons for missing data were most likely related to the variables in our analytic model (McPherson, Barbosa-Leiker, Burns, Howell, & Roll, 2012; McPherson et al., 2015): group assignment, age, sex, alcohol dependence, and baseline BDI-II and BAI scores.

To assess crude group differences (i.e., without covariate adjustment) in post-treatment depressive symptoms, between-group comparisons of the mean post-treatment BDI-II scores were compared by *t*-test. To assess the unadjusted impact of STA and STCM on depressive symptoms over time, the mean baseline and post-treatment BDI-II scores in each group were compared separately by *t*-tests. The same analytical procedures were conducted using the baseline and post-treatment BAI scores. Only observed post-treatment BDI-II and BAI scores were included in the *t*-test analyses.

For all analyses, the level of significance was set at $p < .05$. Statistical analyses were performed with SPSS Statistics software package, version 22.0 (IBM Corporation, Armonk, NY) and Stata, version 14.2 (StataCorp LP, College Station, TX).

Results

Baseline Characteristics

As shown in Table 1, our sample was composed primarily of individuals who were male (87.7%), unemployed, (83.1%) and had a low level of education (9.3 years; SD 3.6). Nearly a quarter of the participants (23.1%) were homeless at enrollment, and more than 60% reported having slept on the streets at least once because of their crack cocaine use. The mean reported age of crack cocaine use onset was 22 years, and the mean duration of use was 12 years. Nearly half (49.2%) of the participants submitted a crack cocaine-positive sample at baseline. Drug-related comorbidities were high, 69.2% and 66.1% of the participants meeting the criteria for polysubstance dependence and alcohol dependence, respectively. Psychotic symptoms were also common, nearly half (46.1%) of the participants exhibiting at least one symptom of psychosis at enrollment. At baseline, the mean BDI-II score was in the moderate (24.5 ± 12.1) range, with over 90% of the participants showing at least mild depressive symptomatology and a third having scores that were consistent with severe depression. None of the above covariates differed statistically between the groups at baseline.

The mean baseline BAI score was in the moderate range (20.7 ± 13.5), with 72% of the participants having at least mild anxiety symptomatology. The mean BAI score and the proportion of participants having severe anxiety symptomatology at baseline were statistically different among groups. The mean BAI score was significantly higher in the STCM group than in the STA group.

Post-Treatment Depressive Symptoms

The post-treatment levels of depressive symptomatology were significantly lower in the STCM group than in the STA group ($\beta = -9.59$; 95% CI: -18.39 to -0.78; $p = .03$; Table 2). There were no significant differences in the post-treatment BDI-II score regarding age ($\beta = 0.12$; 95% CI: -0.52 to 0.76; $p = .70$), sex ($\beta = -8.22$; 95% CI: -22.53 to 6.08; $p = .25$), alcohol dependence ($\beta = 0.46$; 95% CI: -7.25 to 8.17; $p = .90$), or baseline BDI-II score ($\beta = 0.07$; 95% CI: -0.33 to 0.47; $p = .73$). Table 2 also provides the standardized coefficients for the predictors of the post-treatment BDI-II score, in addition to the effect size (R^2) reported for each effect. The variance accounted for by treatment assignment to the CM group was approximately 14%. Lastly, the R^2 for the overall model, inclusive of the entire predictor set was approximately 24% (adjusted for overlap in variance accounted for by more than 1 predictor).

The mean post-treatment BDI-II score was lower in the STCM group than in the STA group; (9.9 ± 10.0 vs. 21.2 ± 12.6), and the difference was statistically significant ($t = -3.20$; $p < .01$). In the STCM group, the mean BDI-II score was also significantly lower after treatment than at baseline (9.9 ± 10.0 vs. 27.1 ± 10.8; $t = -5.85$, $p < .01$), whereas no difference was observed in the STA group (21.2 ± 12.6 vs. 21.9 ± 12.8; $t = -0.19$; $p = .85$).

Post-Treatment Anxiety Symptoms

As seen in Table 2, the post-treatment levels of anxiety symptomatology were lower in the STCM group than in the STA group ($\beta = -9.95$; 95% CI: -19.29 to -0.61; $p = .04$). Post-treatment BAI scores were not associated with age ($\beta = 0.10$; 95% CI: -0.54 to 0.74; $p = .75$), sex ($\beta = -7.50$; 95% CI: -21.12 to 6.13; $p = .27$), alcohol dependence ($\beta = 0.41$; 95% CI: -5.47 to 13.31; $p = .39$), or baseline BAI score ($\beta = 0.06$; 95% CI: -0.30 to 0.40; $p = .76$). The effect size (R^2) reported for the STCM group on post-treatment BAI score outcome was approximately 12%. Lastly, the R^2 for the overall model, inclusive of the entire predictor set was approximately 14%.

The mean post-treatment BAI score was significantly lower in the STCM group when compared to the STA group (9.8 ± 12.4 vs. 19.5 ± 15.1; $t = -2.26$; $p < .01$). In the STCM group, the mean BAI score was also significantly lower at post-treatment than at baseline (9.8 ± 12.4 vs. 25.6 ± 12.2; $t = -4.59$, $p < .01$). However, no statistical difference was observed when baseline and post-treatment mean BAI scores were compared for the STA group (19.5 ± 15.1 vs. 15.8 ± 13.1; $t = 0.94$; $p = .35$).

Discussion

In Brazil, crack cocaine addiction has become a major public health problem and studies on treatment effectiveness in crack cocaine-dependent individuals are extremely scarce. To our knowledge, ours was the first randomized clinical trial of a psychosocial intervention involving a population of crack cocaine-dependent individuals in Brazil. The main purpose of this secondary analysis was to determine whether CM targeting crack cocaine abstinence in a high-risk, crack cocaine-using population in a low-resource setting is effective in reducing off-target depressive and anxiety symptoms. As has previously been reported (Conway et al., 2006; Falck et al., 2002; Kiluk et al., 2013), we found depressive and anxiety symptoms to be common among crack cocaine users, with mean BDI-II and BAI scores considered moderate. Additionally, nearly all (90%) of the participants suffered from at least mild depressive symptoms and over 70% suffered from at least mild anxiety symptoms. As hypothesized, we found that the CM intervention was associated with significantly greater reductions in depressive and anxiety symptoms among crack cocaine users than those obtained with the standard treatment alone. A medium to large effect size in favor of the CM intervention for both outcomes was observed after adjustments for age, sex, alcohol dependence, and baseline BDI-II and BAI scores. These effects are also clinically significant, as the mean BDI-II and BAI scores were 10 points lower in the STCM group when compared to the STA group. These findings are consistent with those of Petry and colleagues, who found CM to be more effective than standard treatment in reducing psychiatric symptoms (including depression and anxiety) in a substance-abusing population (Petry et al., 2013). It is also in agreement with findings from our previous work demonstrating similar reductions in psychiatric symptoms in stimulant users with concomitant disorders (McDonell et al., 2013). This provides additional support for the idea that the therapeutic effects of CM on depressive symptoms are not limited to high-income countries but can be generalized to crack cocaine users receiving treatment in countries with limited resources and an insufficient welfare system. Also of note is the significant

reductions in post-treatment means of BDI-II and BAI scores observed in the STCM group, were not observed in the STA group, suggesting that only CM promoted a substantial reduction in depressive and anxiety symptoms.

Our data suggests that CM aimed at achieving crack cocaine abstinence has the additional benefit of reducing depressive and anxiety symptoms, without pharmacotherapy. This is of special importance because pharmacological treatment targeting depression and anxiety in individuals with substance use disorders have shown little efficacy, with small overall effects at best (Nunes & Levin, 2004; Torrens, Fonseca, Mateu, & Farre, 2005; Zhou et al., 2015). However, it is likely that the observed decrease in BDI-II and BAI scores could have been greater if CM had been combined with evidence-based pharmacotherapy. Therefore, there is a need for further trials that combine CM and pharmacological interventions to target depression and anxiety in substance-abusing populations.

Finally, although our analysis does not include mediation analyses, in part due to statistical power considerations, it is likely that the post-treatment reductions in depressive and anxiety symptoms are a function of the differential rates of drug abstinence produced by the CM intervention. This hypothesis is in agreement with the findings of others (Petry et al., 2013), who found that a reduction in drug use mediated the effects of CM on psychiatric symptoms.

Limitations

Our study has several limitations. First, the sample was relatively small and comprised individuals receiving treatment at a single center. The baseline prevalence rates and treatment outcomes might have been different if the study had involved a larger sample or had been a multicenter study. In addition, our sample was primarily (87.7%) male, and sex effects might therefore have been missed. On the basis of data in the literature (Chen et al., 2011; Rounsaville et al., 1991), we can assume that the BDI-II scores were lower than they would have been if the sample had included more females. Furthermore, we observed significant group differences for the mean BAI score and proportion of participants with severe anxiety symptomatology during the baseline assessments. Such baseline differences represent an important bias, rendering these comparisons problematic and limiting the interpretation of our findings. It is also important to disclose that, at the three- and six-month follow-up assessments, a considerable proportion of the data were missing (62.0% and 70.7%, respectively), which precluded any analysis of the long-term effects of CM on depressive symptoms. Therefore, we were able to evaluate the effects of CM on depressive and anxiety symptoms only during the 12 weeks of treatment. Moreover, the SCID-I was used only to assess substance use and schizophrenia, preventing us from examining the impact of CM on other psychopathologies. Finally, our study was conducted in a low-resource setting, which reduces the generalizability of our results.

Conclusions

The present study represents a first wave of studies with rigorous methodology designed to investigate an evidence-based psychosocial intervention in a hard-to-treat, high-risk crack cocaine-dependent population in South America. Our results demonstrate the high prevalence of depression and anxiety among treatment-seeking crack cocaine-dependent

individuals and highlight the therapeutic benefits of adding CM to the standard treatment for crack cocaine users. The high prevalence of depression and anxiety among treatment-seeking crack cocaine-dependent individuals underscores the importance of assessing and treating comorbid common mental disorders in this population, in which CM appears to improve depressive and anxiety symptoms. Future studies should involve larger samples and should investigate the mechanisms whereby CM improves common mental disorders, as well as potentially combining CM with other treatments for comorbid psychiatric disorders and addiction, especially in low-resource and underserved populations.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Relevance Statement

We found that the prevalence of depressive and anxiety symptoms were extremely high among crack cocaine users, and that, among such individuals, contingency management (CM) reduced depressive and anxiety symptomatology to a greater degree than did standard treatment. Our results suggest that CM targeting crack cocaine abuse can have off-target effects on psychiatric symptomatology.

Table 1

Demographic Characteristics, Comorbid Psychiatric Disorders, and Patterns of Drug Use Among Crack Cocaine Users Receiving Outpatient Treatment

Variable	Sample as a whole ($n = 65$)	STA group ($n = 32$)	STCM group ($n = 33$)	p-value
Age (years), mean ± SD	35.3 ± 8.5	35.4 ± 8.5	35.3 ± 8.7	.85
Male sex, n (%)	57 (87.7)	26 (81.3)	30 (90.0)	.26
Education (years), mean ± SD	9.3 ± 3.6	9.8 ± 3.7	8.9 ± 3.4	.34
Unemployment, n (%)	54 (83.1)	27 (84.4)	28 (84.8)	.96
Homelessness, n (%)	15 (23.1)	9 (28.1)	6 (18.2)	.34
Age at onset of crack use (years), mean ± SD	22.7 ± 6.9	23.6 ± 7.3	21.8 ± 6.6	.40
Duration of crack use (years), mean ± SD	12.6 ± 7.4	11.8 ± 7.5	13.5 ± 7.4	.32
Has slept on the streets due to crack use, n (%)	42 (64.6)	19 (59.4)	23 (69.7)	.38
History of inpatient treatment, n (%)	55 (84.6)	26 (81.1)	29 (87.5)	.73
Number of previous treatments, mean ± SD	2.8 ± 4.0	2.5 ± 3.1	3.2 ± 4.8	.60
Crack cocaine-positive urine sample at baseline, n (%)	32 (49.2)	15 (46.9)	17 (51.5)	.71
THC-positive urine sample at baseline, n (%)	2 (3.1)	1 (3.1)	1 (3)	.78
Alcohol-positive breath sample at baseline, n (%)	11 (16.9)	5 (15.6)	6 (18.2)	.98
Marijuana dependence, n (%)	8 (12.3)	3 (9.4)	5 (15.2)	.48
Alcohol dependence, n (%)	43 (66.1)	21 (65.6)	22 (66.7)	.95
Polysubstance dependence, n (%)	45 (69.2)	24 (75.0)	21 (63.6)	.32
Psychotic symptoms, n (%)	30 (46.1)	15 (46.9)	15 (45.5)	.89
Pre-treatment BDI-II score, mean ± SD	24.5 ± 12.1	21.9 ± 12.8	27.1 ± 10.8	.08
Mild pre-treatment depression, n (%)	18 (27.7)	9 (28.1)	9 (27.3)	.94
Moderate pre-treatment depression, n (%)	19 (29.2)	10 (31.2)	10 (30.3)	.93
Severe pre-treatment depression, n (%)	22 (33.8)	8 (25.0)	14 (42.4)	.14
Pre-treatment BAI-II score, mean ± SD	20.7 ± 13.5	15.8 ± 13.1	25.6 ± 12.2	.01
Mild pre-treatment anxiety, n (%)	14 (21.5)	5 (15.6)	9 (27.3)	.25
Moderate pre-treatment anxiety, n (%)	16 (24.6)	8 (25.0)	8 (24.2)	.94
Severe pre-treatment anxiety, n (%)	17 (26.2)	4 (12.5)	13 (39.4)	.01
Post-treatment BDI-II score, mean ± SD	15.6 ± 12.6	21.2 ± 12.6	9.9 ± 10	.01
Mild post-treatment depression, n (%)	6 (14.3)	4 (19.0)	2 (9.5)	.39
Moderate post-treatment depression, n (%)	8 (19.0)	6 (28.6)	2 (9.5)	.13

Variable	Sample as a whole (*n* = 65)	STA group (*n* = 32)	STCM group (*n* = 33)	*p*-value
Severe post-treatment depression, n (%)	6 (14.3)	5 (23.8)	1 (4.8)	.11
Post-treatment BAI-II score, mean ± SD	14.7 ± 14.5	19.5. ± 15.1	9.8 ± 12.4	.03
Mild post-treatment anxiety, n (%)	10 (23.8)	6 (28.6)	4 (19.0)	.47
Moderate post-treatment anxiety, n (%)	5 (11.9)	4 (19.0)	1 (4.8)	.15
Severe post-treatment anxiety, n (%)	6 (14.3)	4 (19.0)	2 (9.5)	.38

Note. STA = standard treatment alone; STCM = standard treatment plus contingency management; THC = tetrahydrocannabinol; BDI-II = Beck Depression Inventory II; BAI-II = Beck Anxiety Inventory.

Table 2

Contingency Management and other Covariates of the Post-Treatment Beck Depression Inventory-II score and the Post-Treatment Beck Anxiety Inventory score

Outcome	Covariates	Multiple imputation (pooled datasets)				
		Estimate	β(R²)	95% CI	S.E.	*p*-value
Post-treatment BDI-II Score	CM group	−9.59	−0.38 (14.4%)	−18.39 to − 0.78	4.28	0.03
	Age	0.12	0.08 (0.64%)	−0.52 to 0.76	0.30	0.70
	Sex	−8.22	−0.22 (4.84%)	−22.53 to 6.08	6.93	0.25
	Alcohol dependence	0.46	0.02 (0.04%)	−7.22 to 8.17	3.80	0.90
	BDI-II score at baseline	0.07	0.06 (0.36%)	−0.33 to 0.47	0.19	0.73
Post-treatment BAI Score	CM group	−9.95	−0.35 (12.2%)	−19.29 to − 0.61	4.62	0.04
	Age	0.10	0.04 (0.16%)	−0.54 to 0.74	0.31	0.75
	Sex	−7.50	−0.17 (2.89%)	−21.12 to 6.13	6.76	0.27
	Alcohol dependence	0.41	−0.12 (1.44%)	−5.47 to 13.31	4.69	0.39
	BAI score at baseline	0.54	0.06 (0.36%)	−0.30 to 0.40	0.17	0.76

Note. CI = confidence interval; SE = standard error; CM = contingency management; BDI-II = Beck Depression Inventory II; BAI = Beck Anxiety Inventory.