Meta-analysis of depression and substance use and impairment among cocaine users[*]

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Abstract

Background—The study evaluated, among cocaine users, the hypothesized positive association of depression and concurrent cocaine use and impairment, alcohol use and impairment, and general drug use and impairment. The hypothesis that gender would moderate these associations, with women showing a stronger correlation between depression and measures of substance use and impairment, was also tested. Also examined was the association of depression with future cocaine use and impairment and substance use treatment participation.

Methods—Empirical reports on adult cocaine users published in English in peer-reviewed journals since 1986 that contained data on depression and substance use outcome(s) were obtained using a systematic search. Studies that placed restrictions on range of depression scores to select the sample, experiments that administered cocaine to subjects, and trials of antidepressant medications were excluded. The search yielded 60 studies for the analysis including 53 reports that collected data from clinical venues and 7 that were community-based.

Results—As hypothesized, the analyses showed that depression is associated with concurrent cocaine-, alcohol-, and general drug use and impairment. Effect sizes were small. Hypothesized moderating effects of gender were not supported. Depression was not associated, at a statistically significant level, with treatment participation or future cocaine use and impairment.

Conclusions—Depression is consistently but modestly associated with measures of cocaine-, alcohol-, and general drug use and impairment among cocaine users. Associations of depression with treatment participation and with future cocaine use and impairment are not immediately evident, although limitations of data warrant cautious interpretation.

Keywords

depression; cocaine; alcohol abuse; drug abuse

1. Introduction

Cocaine users show high levels of depression (Brienza et al., 2000; Kidorf et al., 2004; Kilbey et al., 1992; Falck et al., 2002; Wild et al., 2005). Cocaine use may be associated with depression in a variety of ways (Markou et al., 1998; Rounsaville, 2004). Cocaine users experience a "crash" that includes depressive symptoms following cessation of use (Dackis & Gold, 1987; Gawin & Kleber, 1986). Experimental studies of cocaine administration have shown that such symptoms are more pronounced in cocaine users who experience a greater subjective "high" from cocaine (Sofuoglu et al., 2001; Uslaner et al., 1999), and so individuals reporting a greater depressive crash may also be those who are most strongly reinforced for using cocaine. Experimental data also suggest an association of depressive symptoms and craving following cocaine administration (Elman et al., 2002) and such craving may lead to further substance use. Correlational studies also support a link between depression and more chronic and severe cocaine use and impairment (Booth et al., 2005; Kilbey et al., 1992; Kasarabada et al., 1998). Potential "causal" explanations for such findings are many (Markou et al., 1998; Rounsaville, 2004) and include that recurrent cocaine use may cause chronic overstimulation of brain reward circuitry, leading to neuroadaptation and consequent depression and anhedonia when use is interrupted. Chronic cocaine use also produces stressful life circumstances that may in turn lead to depression. The depression literature indicates that stressful events characterized by loss and/or humiliation, types of events to which chronic cocaine users are vulnerable (e.g., rejection from a partner, assault victimization), may be especially pathogenic (Kendler et al., 2003). Causality may also flow in the other direction, for example use of cocaine or other stimulants may be a "self-medication" coping response to manage depression (Khantzian, 1985; Markou et al., 1998). There are also shared "third variable" influences on both substance use disorders and depression to consider, for example neuroticism is independently associated with each (Khan et al., 2005).

Overall, research and theory make a compelling case for an association of depression and cocaine use and impairment. The association is not merely a clinical artifact, as levels of depression obtained from non-clinical samples of cocaine users are also elevated (Falck et al., 2002; Kilbey et al., 1992; Shaffer & Eber, 2002). However, *within the population of cocaine users*, it is unclear whether or not greater depression is associated with higher levels of cocaine use and impairment, with several reports failing to show a statistically significant association, particularly prospective reports (Alterman et al., 2000; Bobo et al., 1998; Brown et al., 1998; Carroll et al., 1993a). Studies using experimental designs (Dudish-Poulsen & Hatsukami, 2000; Foltin & Fischman, 1998) also suggest that the "crash" experience may be less pronounced than first described (Gawin & Kleber, 1986). Moreover, the self-medication model of substance use disorder liability is not as well supported as etiological models emphasizing the contribution of externalizing features such as aggression, conduct problems, and disinhibition (Krueger et al., 2002; Vanyukov et al., 2003) particularly in the development of illicit drug misuse as opposed to misuse of alcohol (McGue et al., 1999). Overall, as substance use and impairment as well as depression are multiply caused, it may be that other difficulties confronting cocaine users overwhelm any influence of depression on substance use and impairment or vice-versa. On the other hand, a strong link between depression and substance use and impairment in this population would suggest that depression should be addressed in the context of mainstream substance abuse treatment and/or that depressed substance users may require enhanced interventions targeting substance use and related problems. Data showing an association of depression and treatment participation among cocaine users have also been reported, including reports showing depression is associated with greater (Joe et al., 1999) and lesser treatment involvement (Williams & Roberts, 1991), indicating the need for an integration of research findings to clarify any association.

There is also a need to examine potential moderators of the association of depression and substance use and impairment among cocaine users. In particular data suggest gender differences in depression scores (Falck et al., 2002; Griffin et al., 1989; McCance-Katz et al., 1999). Such differences may merely reflect the higher rate of depression observed among women in the general population (Kessler et al., 2003). Alternatively, the association may be moderated by gender such that the link between depression and substance use and impairment is stronger among women than men, for example due to a greater tendency among women to use psychoactive substances as a coping response (Rubonis et al., 1994). If so, female cocaine users may be expected to show not only higher depression scores but also a higher correlation between depression and measures of substance use and impairment, suggesting a moderating effect. Meta-analysis (Lipsey & Wilson, 2001; Rosenthal, 1991) is a tool that can integrate the relevant data and examine moderating effects.

Conner, Pinquart and Duberstein (2008) performed a meta-analysis of intravenous drug users that examined the levels of association of depression and various measures of substance use and impairment and treatment involvement, as well as potential moderators of these associations. Results are reported in detail in that report. We are carrying out a separate study of cocaine users because the meta-analysis on intravenous drug users is based primarily on opiate users, a drug class that shows differing levels of depression (Malow et al., 1990; Rounsaville et al., 1991) and dissimilar pharmacological properties compared to cocaine that may affect the nature of the association (Khantzian, 1985). We had four aims. 1) We tested the hypothesized positive association of depression with concurrent cocaine use and impairment, alcohol use and impairment, general drug use and impairment, and treatment participation, and estimated the magnitude of the associations. 2) We tested the hypothesized positive association of depression with future cocaine use and impairment. 3) We analyzed associations between depression and sociodemographic characteristics. 4) We analyzed change in depressive symptoms over time. 5) We examined moderating effects of gender, age, and race/ethnicity, as well as methodological features of studies that may act as moderators including the nature of the depression measure used (categorical or continuous) and the recruitment venue (inpatient or outpatient). We hypothesized that associations of depression and the substance-related variables are greater among women.

2. Methods

2.1. Sample

The search was conducted in 2007 and included use of MEDLINE (search terms: depression AND cocaine-related disorders/ or crack cocaine/ or cocaine/ or opioid-related disorders/ or substance abuse, intravenous/ or amphetamine-related disorders) and PsychINFO databases (search terms: depression AND cocaine-related disorders/ or crack cocaine/ or cocaine/ or intravenous drug usage/ or intravenous injections), limited to the years 1986–2007, English language, and humans. Reference sections of relevant reports were also reviewed. This search was also used to identify reports for the meta-analysis on intravenous drug users (Conner et al., 2008).

Inclusion criteria for the current report were: 1. Studies of samples that are exclusively or predominantly composed of cocaine users or studies that present relevant data on a subgroup of cocaine users. 2. Studies containing at least one assessment of depression using a multi-item published scale or published diagnostic interview. 3. Association(s) of depressive symptoms with drug use and/or drug use impairment, other substance-related behaviors (e.g., alcohol use, substance use treatment drop out), or change in depression, that were reported as correlations or as other effect size measures. Studies were excluded from the meta-analysis if: 1. Cocaine was supplied or administered by the experimenter. 2. Depression cutoffs or diagnoses were used to create the sample, resulting in restriction of range on depression. 3. They were trials

of antidepressant medications. 4. They were unpublished. 5. Mean age of the sample was less than 21 years. If more than one study from the same research group was available, we checked whether these papers referred to different data sets, and omitted duplicate results.

Based on the search, we reviewed 367 full-length reports that yielded 60 studies for the current analysis (Alterman et al., 2000; Araujo et al., 1996; Avants et al., 2000; Bobo et al., 1998; Booth et al., 2005; Booth et al., 2006; Brady et al., 1995; Brienza et al., 2000; Brown et al., 1998; Brown et al., 1994; Burkett et al., 2005; Carroll et al., 1993a; Carroll et al., 1993b; Compton et al., 2000; Cunningham et al., 1993; de los Cobos et al., 2001; Falck et al., 2002; Focchi et al., 2005; Gawin & Kleber, 1986; Gillin et al., 1994; Griffin et al., 1989; Hasin et al., 2002; Herbeck et al., 2006; Hser et al., 2006; Husband et al., 1996; Joe et al., 1999; Kampman et al., 2001; Kasarabada et al., 1998; Kasarabada et al., 1999; Kilbey et al., 1992; Kleinman et al., 1992; Knowlton et al., 2001; Kosten et al., 1987; Kowatch et al., 1992; Kranzler et al., 1995; Latkin & Mandell, 1993; Lee et al., 1990; Leventhal et al., 2006; Margolin et al., 1993; Margolin et al., 2005; McCance-Katz et al., 1999; McKay et al., 1997; McKay et al., 2002; McMahon et al., 1999; Montoya et al., 1995; Nishimoto & Gordon, 1997; Nunes et al., 1989; Nunes et al., 1993; Pathiraja et al., 1995; Riehman et al., 2002; Satel et al., 1991; Schmitz et al., 2000; Stahler et al., 2005; Strain et al., 1991; Torrens et al., 1991; Weddington et al., 1990; Weiss et al., 1986; Weiss et al., 1989; Weiss et al., 1996; Williams & Roberts, 1991) with 42 papers identified in the electronic search and 18 papers identified through reference sections. A comprehensive list of these studies is provided in Appendix 1. Four additional studies meeting eligibility criteria were identified in the search (Boyd, 1993; Carroll & Rounsaville, 1992; Dolan et al., 1991; Williamson et al., 2006) but were not used in the analysis because they did not contribute an effect size that was used in five or more reports, the minimum required for analysis.

2.2. Measures

2.2.1. Depression—Studies basing depression data on structured clinical interviews used various versions of the Diagnostic Interview Schedule, DIS (Robins et al., 1981); Psychiatric Research Interview for Substance Use and Mental Disorders, PRISM (Hasin et al., 1998); Schedule for Affective Disorders and Schizophrenia, SADS (Endicott & Sptizer, 1978); and Structured Clinical Interview for DSM, SCID (Spitzer et al., 1988). Studies basing depression data on self-report scales used various versions of the Beck Depression Inventory, BDI (Beck et al., 1961); Brief Symptom Inventory, BSI (Derogatis, 1993); Center for Epidemiological Studies – Depression Scale, CES-D (Radloff, 1977); General Health Questionnaire, GHQ (Goldberg & Williams, 1988); Hamilton Rating Scale for Depression, HRSD (Hamilton, 1960); Hopkins Symptoms Checklist, HSCL (Derogatis, 1974); Millon Clinical Multiaxial Inventory, MCMI (Millon, 1987); Minnesota Multiphasic Personality Inventory, MMPI (Hathaway & McKinley, 1940); and Physician's Health Questionnaire–9, PHQ-9 (Spitzer et al., 1999). Investigators quantified the depression data using continuous indexes (e.g., BDI total score), categorical determinations (e.g., major depression diagnosis), or both. Several studies used two measures for assessment of depression. Additional information about the depression measures used in the individual studies and the manner in which depression data were quantified are presented in the Appendix.

2.2.2. Cocaine use and impairment—Information in this domain was collected via participant self-report and structured interviews and urine toxicology screens.

2.2.3—General drug use and impairment not specific to cocaine or alcohol was assessed via the composite drug use scale of the ASI (McLellan et al., 1992) and self-report of use of other substances (e.g., marijuana).

2.2.4. Alcohol use and impairment—Data on frequency of alcohol use, alcohol use status (e.g., abstinent, relapsed), and alcohol use disorder diagnoses were used to assess this domain.

2.2.5. Treatment involvement—Data on the length of stay in drug treatment and on treatment completion were analyzed.

2.2.6. Change in depressive symptoms—Thirty longitudinal samples provided data on the level of depressive symptoms for more than one time of measurement so that the level of change in these symptoms could be computed.

3. Statistical integration of the findings

Computations were based on random-effects models and the noniterative method of moments (Hedges & Vevea, 1998). 1. We computed effect sizes (*d*) for each study by transforming correlation coefficients, *t* values, *F* values, and exact *p* values (Rosenthal, 1991). Effect size estimates were adjusted for bias due to overestimation of the population effect size in small samples. If a study variable was assessed with more than one measure, we included the average effect size in our analysis. 2. Studies were weighted by the inverse of their variances, and weighted mean effect sizes *d* and their confidence intervals (*C.I.*) that include 95% of the effects were computed. Because readers may be more familiar with interpreting correlation coefficients than effect sizes *d* as indicators of the size of association between variables, we converted the effects sizes and their confidence intervals back into the metric of correlation coefficients (Rosenthal, 1991). 3. The significance of the mean was tested by dividing the weighted mean effect size by the estimated standard error of the mean effect size. 4. Homogeneity of effect sizes was tested by using the homogeneity statistics (*Q*). 5. Weighted multiple linear regression analysis was used for the search for moderating effects of study characteristics on the size of associations between depression with concurrent and prospective cocaine use, alcohol use, and for the size of change in substance use. Thus, four weighted multiple ordinary least squares regression analyses were computed, following the random-effects approach and the method of moments (Raudenbush, 1994). Independent variables were mean age of the participants, percentage of men, percentage of whites, the percentage of inpatients, whether a continuous or dummy variable was used for assessing depression, and length of study interval (for analysis of change in depressive symptoms only). Moderator analysis could not be conducted for the association of depression with general drug use or treatment participation due to the lack of a sufficient number of available studies. 6. As a tool for interpreting the practical significance of correlation coefficients, we used the Binomial Effect Size Display, BESD (Rosenthal, 1991). For example, after the median split of the level of depressive symptoms and of substance-related behavior, the percentage of persons with above-average depressive symptoms and above-average level of substance-related behavior is computed by $0.5+r/2$, and the percentage of above-average behavior level in the less depressed group is $0.5−r/2$.

Funnel plots were used as an indicator of potential publication bias (Lipsey & Wilson, 2001). For estimating the number of of nonsignificant studies that would be necessary to reduce the effect size to a nonsignificant value, we computed the fail-safe *N* for the significant correlation coefficients (Rosenthal, 1991). In some studies the majority of subjects are both cocaine users and intravenous drug users given comorbidity, and so our prior meta-analysis on intravenous drug use and depression (Conner et al., 2008) contained twelve reports also analyzed for the current study. Therefore, we re-analyzed the data with the common studies excluded in order to clearly distinguish the findings of the current study.

3. Results

3.1. Sample description

Fifty-three studies collected data from clinical venues and seven were community-based. The sampling venue (clinical, community) and, for clinic-based samples, information on whether inpatients and/or outpatients were assessed are listed in the appendix. The participants had a mean age of 33.8 years ($SD = 4.4$ years) and about 68% were men and 46% were White. The participants had, on average, completed about 12.6 ($SD = 0.6$) years of education.

3.2. Associations of depressive symptoms with substance-related variables

We found a positive association of depression with concurrent cocaine use and impairment (see Table 1). According to standard criteria (Cohen, 1992), the size of the association is small. Nonetheless, according to the BESD, 57% of persons with above-average levels of depressive symptoms show above-average levels of current cocaine use, as compared to 43% of persons with below-average levels of depressive symptoms, suggesting that the result is of practical significance. Interestingly, longitudinal studies found no significant prospective association of depressive symptoms with cocaine use and impairment.

Our results further showed a significant, but small, concurrent relationship between depressive symptoms and alcohol use and impairment. According to the BESD, 59% of individuals with above-average levels of depressive symptoms show above-average levels of alcohol use, as compared to 41% of individuals with below-average levels of depressive symptoms. A small, but statistically significant positive relationship between depression and general drug use and impairment was found. According to the BESD, 55% of persons with above-average levels of depression show above-average levels of drug-related problems, as do 45% of the individuals with below-average levels of depression. No significant association of depression with treatment involvement was found.

We next analyzed whether the level of depressive symptoms would vary by sample characteristics. Women showed higher levels of depressive symptoms than men, and the size of the association was small. No significant associations of depression with age, ethnicity, and educational attainment were found.

On average, longitudinal studies (30 samples) showed a decline of depressive symptoms of $d = 0.71$ (95% $CI = .57, .81$) standard deviation units over time. The change is highly significant ($t = 9.99$, $p<.001$) and, according to Cohen's criteria, the size of decline is interpreted as moderate. The test for heterogeneity of effect sizes was also highly significant ($Q = 56.04$, $p<.001$).

3.3. Analysis of moderating effects

With regard to the association of depressive symptoms and concurrent cocaine use, we found a moderating effect of the method for assessing depression: associations between depression and cocaine use were stronger in studies that had measured depression with a continuous variable than in studies that had used a dummy variable (Table 2). In addition, we found a stronger decline in depressive symptoms in samples with a larger percentage of white cocaine users. No significant moderating effects were found for associations of depression with future cocaine use and alcohol use, probably due to the restricted number of available studies.

3.4 Analyses to evaluate publication bias and to distinguish the current findings

Funnel plots (available by accessing the on-line version of this paper at http://dx.doi.org by entering doi:xxxxxxxx) indicated no publication bias with the exception that associations between depression and alcohol use and impairment were less positive in smaller studies which

may indicate a publication bias. Nonetheless, as only one large study found a negative association between depression and alcohol use and impairment, publication bias had probably no strong effect on the observed positive average association between depression and alcohol use and impairment. The fail-safe N indicates that between 68 and 646 studies with null results would be needed in order to reduce the significant effects to a nonsignificant level (Table 1). After excluding the twelve reports contained in our prior meta-analysis, the results were comparable with the exception that in the reduced data set, there was a negative association between depression and treatment participation. Because this analysis was only based on three studies, this result was less robust and no longer significant after inclusion of the full number of available studies.

4. Discussion

4.1. Tests of hypotheses

Results support the hypothesized positive association of depression and concurrent cocaine use and impairment, alcohol use and impairment, and general drug use and impairment, suggesting the relevance of depression to a range of substance use and impairment among cocaine users. There are many potential explanations for the findings that include: the cessation of cocaine use resulting in a "crash" marked by depressive symptoms; use of cocaine, alcohol, and other substances to cope with crashing and/or to cope with a more longstanding mood disorder; use of these substances leading to stressful life events that in turn promote depression; and shared underlying causes of depression and substance use and impairment. Although the current report substantiates that there is a concurrent association between depression and substance use and impairment among cocaine users, the data do not inform which, if any, of these mechanism(s) is responsible. Unlike the results of cross-sectional reports, prospective studies did not support an association between depression and future cocaine use and impairment.

Results showed that cocaine using women have higher depressive symptoms than men but tests of moderation did not indicate that the size of the association between depression and the various measures of substance use and impairment are greater among women. The results do not support the notion that depression is more inextricably linked with substance use and impairment among women, for example due to greater reliance on substance use to cope. Gender differences in depression among male and female cocaine users may reflect a general propensity for women to show higher levels of depression, although limitations of the data warrant cautious interpretation including that the lack of significant moderating effects of gender on the association between depression and cocaine use may have been based on the restricted variance in gender composition of the samples that were composed mostly of men.

4.2. Additional findings

Prospective studies indicated that cocaine users had a strong decline in depressive symptoms. Interestingly, moderator analysis of clinical reports did not show that the decline was stronger in inpatient venues compared to outpatient treatment settings, despite a greater capacity to promote full abstinence in the former. Most of the prospective studies were based on clinical samples and so the strong decline in depressive symptoms is probably attributable, at least in part, to effects of substance use intervention and/or concomitant treatments for depressive symptoms such as antidepressant medications, psychotherapy, and dual-diagnosis education. Because treatment seeking may be increased during peaks in substance abuse and depressive symptoms (Brienza et al., 2000; Rounsaville & Kleber, 1985), inherent motivation and other natural recovery processes independent of treatment as well as regression to the mean (Finney, 2007) are likely also contributing factors to the decline in depression. Whites showed a greater decline in depressive symptoms than minority subjects. The explanation for such a finding is

unclear. Poorer recognition of depression by clinicians treating racial/ethnic minority individuals, lower rates of referral for such treatment, and/or poorer access to such care (Fiscella et al., 2000; Wells et al., 2002) may be among the reasons that whites showed greater desistance in depressive symptoms.

Analysis of prospective reports suggests that depression is not of prognostic significance in predicting substance use and impairment among cocaine users. The data also did not support that depression is associated with the level of treatment involvement. Cautious interpretation of these non-significant findings is necessary because rarely were reports explicitly designed to examine these questions and because there was significant between-study heterogeneity of effect sizes so that significant associations would be found under particular conditions.

Continuous measures of depression were more strongly associated with concurrent cocaine use and impairment compared to categorical measures, generally based on diagnostic interviews. This finding is probably attributable to the statistical advantages of the use of continuous measures for correlational analyses. Shared method variance, such that individuals reporting greater substance use and impairment on self-report scales could also be inclined to report higher depression, may also contribute to this result.

4.3. Limitations

There were limitations of the study. Different measures of cocaine use and impairment, alcohol use and impairment, depression, and treatment participation had to be combined into single summary measures, producing between-study heterogeneity. An exception is that studies of general drug use and impairment did not show significant heterogeneity, likely because the ASI composite drug scale was used in most of these reports. There were insufficient data for more refined analysis of substance-related measures, for example to disentangle associations of depression with measures of substance use versus impairment. Studies did not consistently report route of administration (smoking, intravenous, intranasal) and so its association with depression could not be examined. We were also unable to examine the potential moderating influences of socioeconomic status (e.g., education level), social instability (e.g., housing status), or race/ethnicity beyond a general comparison of white and non-white subjects because comparable measures of such information was not consistently available. Depression was assessed typically by self-report measures that are sensitive to transient substance intoxication and withdrawal effects. Sufficient data were not available to distinguish substance-induced and independent depressive symptoms. Data were not available to disentangle drug use impairment attributable to the illicit status of cocaine and other psychoactive substances (with the exception of alcohol) as opposed to that attributable to the pharmacological properties of the drugs themselves. Similarly, due to the lack of sufficient data we were not able to analyze whether substance use at the first time of measurement predicts change in depressive symptoms over time. Of 60 studies analyzed, only seven used community samples, three of which had longitudinal designs, and so the relevance of the results to untreated cocaine users, particularly in regards to the prospective association of cocaine use and depression, is unclear. Reports were overwhelmingly from the U.S., which may limit generalizability. Correlations do not imply causation.

4.4. Conclusion

To our knowledge this study represents the first published meta-analysis of depression and substance use and impairment among cocaine users. Depression is relevant across measures of substance use and impairment in this population including assessments of cocaine, alcohol, and general drug use. Effect sizes were small however, and we uncovered no evidence to support the idea that depression is associated with future substance use and impairment. Because depression shows only weak associations with measures of substance use and

impairment, and does not appear to portend a poorer substance abuse treatment outcome, the targeting of depression during the course of mainstream substance abuse treatment may not be an efficient way to improve substance-related outcomes, and depressed cocaine users may not require enhanced interventions targeting substance use and impairment per se, although additional interventions targeting depression in these clients may be beneficial in many cases. In our meta-analysis of intravenous drug users (Conner et al., 2008) we also obtained small effect sizes in concurrent analyses of depression and measures of substance use and impairment, and the absence of a prospective association, and so this pattern of results may apply to illicit drug users broadly. However, unlike the current report, the study of intravenous drug users uncovered several moderating effects, particularly as concerns gender, suggesting the presence of population-specific relationships.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Correlates of depression in cocaine users

Correlate	k	r	95%-CI		Z	Q	Fail-safe N
Sociodemographic variables							
Age in years	9	−.02	−.10	.06	−0.53	22.63 ***	-
Female gender (1=yes, 0=no)	13	.14	.11	.16	10.04 ***	14.50	451
Caucasian (1=yes, 0=no)	9	.08	−.00	.15	1.90	32.82 ***	-
Educational attainment	9	−.03	−.10	.04	−0.88	26.73 ***	-
Substance-related variables							
Cocaine use and impairment (concurrent relationship)	18	.14	.05	.23	3.00 **	166.98 ***	646
Cocaine use and impairment (prospective relationship)	10	.04	−.04	.12	0.97	29.94 ***	-
Alcohol use and impairment (concurrent relationship)	12	.18	.07	.29	3.04 **	88.19 ***	208
General drug use and impairment	8	.10	.07	.13	5.96 ***	12.78	68
Treatment involvement	7	−.07	−.24	.10	−0.83	48.75 ***	-

Note. k = number of samples, r = weighted mean correlation coefficient, 95%-CI = 95%-confidence interval, Z = test for significance of the mean, Q = homogeneity statistics (significant values indicate heterogeneity of effect sizes),

**
$p < .01$,

***
$p < .001$.

Table 2

Test for moderating effects of study characteristics (weighted multiple linear regression analysis)

Variable	Association. with concurrent cocaine use		Association with prospective cocaine use		Association with alcohol use		Change in depressive symptoms	
	B	β	B	β	B	β	B	β
Age	.02	.34	−.03	−.40	.00	.04	−.02	−.30
% men	−.01	−.49	−.00	−.08	.01	.45	.00	.06
% white	.00	.17	−.00	−.26	.00	.31	−.01*	−.53
% inpatients	−.00	−.04	−.00	−.01	.00	.17	−.00	−.01
Depression measure (1=dummy variable, 2= contin. variable)	.37*	.56	.22	.51	−.25	−.29	−.59	−.37
Length of study interval							.03	.25
Constant	−.29		.94		−.65		1.25	
R^2	.36		.52		.32		.39	
k	18		10		12		30	

Note. B ($β$) = unstandardized (standardized) regression coefficient, k = number of samples, R^2 = explained variance,

*
$p < .05$,

**
$p < .01$

Appendix 1

Articles included in the meta-analysis.

Report (country)	Depression Measure (type)	Sample (type if clinical)	Type of report (duration if longitudinal)	N	Mean age	% men	% white	yrs educ.
Alterman et al. 2000 (U.S.)	DIS (categorical)	clinical (in/outpatient)	longitudinal (7 months)	160	34.4	100	5.0	mean 12.2
Araujo et al., 1996 (U.S.)	HRSD (continuous)	clinical (inpatient)	longitudinal (5 days)	55	33.4	73	36.5	mean 11.2
Avants et al., 2000 (U.S.)	BDI (continuous)	clinical (outpatient)	longitudinal (12 weeks)	302	36.7	72	60	63% ≥ 12yr
Bobo et al., 1998 (U.S.)	CESD (continuous)	clinical (in/outpatient)	longitudinal (1 year)	575	33	67	67	nr
Booth et al., 2005 (U.S.)	BSI, PHQ9 (continuous)	clinical (inpatient)	cross-sectional	145	39	67	23	61% ≥ 12yr
Booth et al., 2006 (U.S.)	BSI (continuous)	community	cross-sectional	613	33	63.0	66	58% ≥ 12 yr
Brady et al., 1995 (U.S.)	HRSD (continuous)	clinical (outpatient)	cross-sectional	74	32.4	72	27	nr
Brienza et al., 2000 (U.S.)	SCID (categorical)	community	cross-sectional	251	35	67	85.3	67% ≥ 12yr
Brown et al., 1998 (U.S.)	SCID, HRSD (categorical, continuous)	clinical (in/outpatient)	longitudinal (3 months)	89	28.6	73	87.6	nr
Brown et al., 1994 (Canada)	SCL-90 (continuous)	clinical (inpatient)	longitudinal (4 weeks)	82	32.1	72	94	mean 11.2
Burkett et al., 2005 (U.S.)	BDI (continuous)	clinical (in/outpatient)	longitudinal (12 months)	178	40.5	100	15.3	mean 11.5
Carroll et al., 1993a (U.S.)	SADS, BDI (categorical)	clinical (outpatient)	longitudinal (12 months)	94	26.5	66	61	mean 12.5
Carroll et al., 1993b (U.S.)	SADS (categorical)	clinical (outpatient)	longitudinal (12 months)	298, 94	27.7	69	64	mean 12.3
Compton et al., 2000 (U.S.)	DIS (categorical)	community	longitudinal (3 months)	966	39.1	61	92	53% ≥ 12yr
Cunningham et al. 1993 (U.S.)	BDI, MMPI (continuous)	clinical (inpatient)	cross-sectional	144	32.4	100	0	mean 12.7 yr
de los Cobos et al., 2001 (Spain)	BDI (continuous)	clinical (inpatient)	longitudinal (2 weeks)	40	31.4	77.6	0	mean 7.9 yr
Falck et al., 2002 (U.S.)	BDI (categorical)	community	cross-sectional	430	37.2	60.9	38.1	50.3% ≥ 12yr
Focchi et al., 2005 (Brazil)	HRSD (continuous)	clinical (outpatient)	longitudinal (3 months)	42	27.1	100	83.4	14.3% ≥ 12yr
Gawin & Kleber, 1986 (U.S.)	DIS (categorical)	clinical (outpatient)	cross-sectional	30	28.6	73	80	12.8
Gillin et al., 1994 (U.S.)	BDI, HRSD (continuous)	clinical (inpatient)	longitudinal (18 days)	28	nr	100	18	nr
Griffin et al., 1989 (U.S.)	HRSD (continuous)	clinical (inpatient)	longitudinal (4 weeks)	129	29.5	73.4	92	nr
Hasin et al., 2002 (U.S.)	PRISM (categorical)	clinical (in/outpatient)	longitudinal (18 months)	250	36.9	66	57	85% ≥ 12yr
Herbeck et al., 2006 (U.S.)	HSCL (continuous)	clinical (in/outpatient)	longitudinal (12 years)	266	48.6	100	25	95% ≥ 12 yrs
Hser et al., 2006 (U.S.)	HSCL (continuous)	clinical (outpatient)	longitudinal (12 years)	266	48.2	100	25	86% ≥ 12 yrs
Husband et al., 1996 (U.S.)	BDI (continuous)	clinical (inpatient)	longitudinal (4 weeks)	82	32.0	71	8	nr

Report (country)	Depression Measure (type)	Sample (type if clinical)	Type of report (duration if longitudinal)	N	Mean age	% men	% white	yrs educ.
Joe et al., 1999 (U.S.)	SCL-90 (continuous)	clinical (outpatient)	longitudinal (360 days)	3209	33	65	37	64% ≥12yr
Kampman et al., 2001 (U.S.)	BDI (continuous)	clinical (outpatient)	longitudinal (8 weeks)	108	37	82	23	12.6
Kasarabada et al., 1998 (U.S.)	HRSD (continuous)	clinical (in/outpatient)	cross-sectional	226	33	100	27	12.8
Kasarabada et al., 1999 (U.S.)	BDI, SCL-90 (continuous)	clinical (in/outpatient)	longitudinal (2 years)	216	35	100	26	46%> 12 yrs
Kilbey et al., 1992 (U.S.)	DIS (categorical)	community	cross-sectional	124	26	49	94	≥12 94%
Kleinman et al., 1992 (U.S.)	BDI (continuous)	clinical (outpatient)	longitudinal (24 sessions "time" nr)	148	30.5	87	16	≥12 72%
Knowlton et al., 2001 (U.S.)	CESD (categorical)	community	longitudinal (1 year)	393	nr	63.9	5.6	≥12 44.5%
Kosten et al., 1987 (U.S.)	SADS (categorical)	clinical (outpatient)	longitudinal (2.5 years)	260	27.6	76	48	nr
Kowatch et al., 1992 (U.S.)	HRSD (continuous)	clinical (inpatient)	longitudinal (3 weeks)	9	28.5	55.5	nr	11.8
Kranzler et al., 1995 (U.S.)	BDI (continuous)	clinical (outpatient)	longitudinal (3 months)	40	34	100	65.0	13.9
Latkin & Mandell, 1993 (U.S.)	GHQ (categorical, continuous)	community	longitudinal (6 months)	91	34	85.7	8.9	57.1% ≥12yrs
Lee et al., 1990 (U.S.)	SADS, HRSD (categorical, continuous)	clinical (inpatient)	cross-sectional	16	37.6	100	nr	nr
Leventhal et al., 2006 (U.S.)	SCID (categorical)	clinical (outpatient)	cross-sectional	339	39	79	31	12.6 yrs
Margolin et al., 1993 (U.S.)	BDI (continuous)	clinical (outpatient)	longitudinal (8 weeks)	32	34	43.8	53.1	
Margolin et al., 2005 (U.S.)	BDI (continuous)	clinical (outpatient)	longitudinal (8 weeks)	40	42.8	60	35	52% ≥ 12 yrs
McCance-Katz et al., 1999 (U.S.)	SADS, BDI (categorical, continuous)	clinical (outpatient)	longitudinal (1 year)	298, 94	27.7	69	61.7	12.3 yr
McKay et al., 1997 (U.S.)	SCID (categorical)	clinical (outpatient)	longitudinal (6 months)	98	40	100	13	nr
McKay et al., 2002 (U.S.)	SCID (categorical)	clinical (outpatient)	longitudinal (2 years)	132	41	100	nr	12.5 yr
McMahon et al., 1999 (U.S.)	MCMI (continuous)	clinical (inpatient)	cross-sectional	304	29.3	100	34	12.2 yr
Montoya et al., 1995 (U.S.)	BDI (continuous)	clinical (outpatient)	longitudinal (8 weeks)	62	33.2	79.0	30.7	12.7 yr
Nishimoto & Gordon, 1997 (U.S.)	BDI (continuous)	clinical (outpatient)	longitudinal (1 year)	84	31.8	0	1.2	64% ≥12 yrs
Nunes et al., 1989 (U.S.)	SCID,HRSD (categorical, continuous)	clinical (outpatient)	cross-sectional	30	34	77	87	nr
Nunes et al., 1993 (U.S.)	HRSD (continuous)	clinical (outpatient)	longitudinal (6 weeks)	7	34	100	43	100
Pathiraja et al., 1995 (U.S.)	HRSD (continuous)	clinical (inpatient)	longitudinal (6 weeks)	27	36	100	nr	nr
Riehman et al., 2002 (U.S.)	SCL-90 (continuous)	clinical (outpatient)	longitudinal (1 year)	2176	33	61	36	67% ≥12 yrs
Satel et al., 1991 (U.S.)	BDI (continuous)	clinical (inpatient)	longitudinal (3 weeks)	22	33.8	86.4	45.5	nr

Report (country)	Depression Measure (type)	Sample (type if clinical)	Type of report (duration if longitudinal)	N	Mean age	% men	% white	yrs educ.
Schmitz et al., 2000 (U.S.)	SCID, SCL–90 (categorical, continuous)	clinical (outpatient)	cross-sectional	151	36.1	67.5	48	12.9 yrs
Stahler et al., 2005 (U.S.)	BDI (continuous)	clinical (in/outpatient)	longitudinal (18 months)	111	32.7	0	4	11.2 yrs
Strain et al., 1991 (U.S.)	BDI (continuous)	clinical (outpatient)	longitudinal (4 weeks)	58	34.3	67	41	11 yrs
Torrens et al., 1991 (Spain)	BDI (continuous)	clinical (inpatient)	longitudinal (11 days)	30	25.1	73	0	nr
Weddington et al., 1990 (U.S.)	BDI (continuous)	clinical (inpatient)	longitudinal (28 days)	12	28.1	100	92	11.5 yrs
Weiss et al., 1986 (U.S.)	HRSD (continuous)	clinical (inpatient)	longitudinal (4 weeks)	30	29.7	63.3	93.3	nr
Weiss et al., 1989 (U.S.)	HRSD (continuous)	clinical (inpatient)	longitudinal (4 weeks)	149	29.5	74	95	nr
Weiss et al., 1996 (U.S.)	SCID (categorical)	clinical (inpatient)	cross-sectional	90	32.0	60.0	84.4	nr
Williams & Roberts, 1991 (U.S.)	BDI (continuous)	clinical (inpatient)	cross-sectional	136	27.3	0	65.4	11.7 yrs

Note. nr = not reported.