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Meta-analysis of depression and substance use and impairment among intravenous drug users (IDUs)

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Abstract

Aims—To evaluate, among intravenous drug users (IDUs), the hypothesized positive association of depression with substance-related behaviors including concurrent drug use and impairment, future drug use and impairment, alcohol use and impairment, needle sharing and substance use treatment participation, and to identify moderators of these associations.

Design—Meta-analysis of reports on IDUs published in English in peer-reviewed journals since 1986 that contained data on depression and substance use outcome(s) with no restrictions on range of depression scores to select the sample.

Setting—Fifty-five reports containing 55 samples met criteria, including 42 (76%) samples from clinical venues and 13 (24%) that were community-based.

Participants—Mean age was 34.3 (standard deviation = 4.5) years, comprising approximately 68% men and 43% white, non-Hispanic subjects.

Measurements—Depression was assessed with the Beck Depression Inventory, Center for Epidemiological Studies Depression Scale (CES-D) and other validated scales or diagnostic interviews. The Addiction Severity Index was the most frequently used measure of substance-related outcomes.

Findings—A priori hypotheses pertaining to depression and the substance-related variables were supported, with the exception of the predicted association of depression and future drug use and impairment. Effect sizes were small. Moderating effects of gender were identified, including greater associations of depression with substance use treatment participation and needle sharing among women and a greater association of depression with future drug use and impairment among men. Effect sizes of moderators were large.

Conclusions—Depression is associated with several substance-related behaviors, and select associations are stronger according to gender. Prospective associations of depression with future drug use and impairment are not immediately evident, but could be examined in subsequent research.

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Depression; intravenous drug use; meta-analysis; substance-related disorders

INTRODUCTION

Although high rates of depression among intravenous drug users (IDUs) have been documented [1–3], the implications of depression for drug use and impairment remain unclear. Also unknown are the effects of depression on other substance-related behaviors including alcohol use and impairment, participation in substance use treatment and high-risk forms of intravenous drug use such as needle sharing. Effects of depressive symptoms on drug use and impairment might be minimal compared to the effects of the many other concerns that may confront IDUs (e.g. incarceration, poverty, homelessness, infectious disease). Alternatively, if depressive symptoms are associated independently with drug use and impairment among IDUs, then it is essential to consider depression in formulating general strategies to address addiction.

Research on the associations of depressive symptoms with drug use and impairment among general samples of IDUs has yielded contradictory conclusions. For example, depression has been shown to be associated both with greater [4-6] and lesser [7,8] drug use. The apparently contradictory findings may be explained by differences among study characteristics, including sampling (i.e. clinic- versus community-based), design (i.e. crosssectional versus longitudinal) and subject composition (i.e. gender distribution). A further complication is that single reports rarely contain samples of sufficient size or heterogeneity to examine potential moderators of the relationship between depressive symptoms and drug use. Moderators are critical to examine, as they can identify subpopulations for which a treatment may be particularly effective or especially ineffective. For example, although there are exceptions [6] most studies show that, among IDUs, women have higher levels of depression than men [3,9,10] and may be more likely to use substances to cope with negative affect [11]. These findings suggest that the association of depression and drug use may be higher in women. Moreover, depression may promote entry into treatment for substance use disorders [1,12] and is generally higher in clinical compared to community samples [3,10]. These findings suggest a stronger association of depression and drug use in IDUs recruited from clinics as opposed to those recruited from community settings. Taken together, the data on differences in gender and recruitment site, if confirmed in moderator analyses, suggest the value of addressing depressive symptoms routinely as part of addiction treatment for female IDUs, but only in particular subgroups of male IDUs.

Examinations of depression among IDUs have also yielded several reports on other widely studied behaviors including alcohol use and impairment [13,14], substance use treatment participation [5,15], changes in depression over time [5,16,17] and needle sharing [3,18,19]. A straightforward association of depression with worse outcomes cannot be presumed, as depression may *increase* treatment participation [15]. Again, moderating effects are important to explore. Studies have shown, for example, that the association of depression

Using meta-analysis [22,23], we examined the association of depression with drug use and impairment and other substance-related behaviors among IDUs and examined changes in depression over time. We had five aims: (i) we tested the hypothesized positive association of depression with concurrent drug use and impairment and future drug use and impairment, and estimated the magnitude of the association; (ii) we examined whether gender, age, race/ ethnicity or clinical status moderate the associations of depression with drug use and impairment. Clinical status was defined in terms of recruitment venue. We hypothesized stronger associations of depression and drug use and impairment among IDUs recruited from clinics and among women; (iii) we analyzed change in depressive symptoms over time; (iv) we estimated the magnitude of the associations of depression with alcohol use and impairment, needle sharing and substance use treatment participation; and (v) we conducted exploratory analyses designed to identify moderators of depression and needle sharing and substance use treatment participation, and designed to identify moderators of changes in depression and alcohol use and impairment.

METHODS

Sample

The search included use of MEDLINE (search terms: depression and opioid-related disorders/or substance abuse, intravenous/or amphetamine-related disorders/or cocaine-related disorders/or crack cocaine/or cocaine) and PsychINFO databases (search terms: depression and intravenous drug usage/or intravenous injections/or cocaine-related disorders/or cocaine), limited to the years 1986–2007, English language and humans. Reference sections of relevant reports were also reviewed.

Inclusion criteria were: (i) studies of samples that are comprised exclusively or predominantly of intravenous users of illicit drugs or studies that present relevant data on a subgroup of IDUs; (ii) studies containing at least one assessment of depression using a multi-item published scale or published diagnostic interview; and (iii) association(s) of depressive symptoms with drug use and/or drug use impairment, other substance-related behaviors (e.g. alcohol use, substance use treatment dropout), or change in depression, that were reported as correlations or as other effect size measures. Studies were excluded from the meta-analysis if: (i) they were conducted on intravenous (i.v.) users of prescribed drugs (e.g. for diabetes) or steroids; (ii) depression cut-offs or diagnoses were used to create the sample, resulting in restriction of range on depression; (iii) they were trials of antidepressant medications; (iv) they were unpublished; and (v) 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 55 eligible papers [1,3–10,13–21,24–60] for the current investigation based on 55 samples. Thirty papers had been identified in the electronic search and 25 papers were identified through reference

sections. Forty-two (76%) samples were from clinical venues and 13 (24%) were community-based.

A comprehensive list of the studies is provided in Appendix I. We entered the following variables: sample size; sampling venue (1 = clinical sample if all or most participants were enrolled in intervention programs, 0 = community sample); socio-demographic characteristics (mean age, percentage of men, percentage of whites); measurements of depression; assessments of substance-related variables (drug use and impairment, alcohol use and impairment, substance use treatment participation, needle sharing); correlations between depression and substance-related variables, distinguishing associations of depression and concurrent drug use and impairment versus future drug use and impairment; and the level of change in depressive symptoms with a median interval of 12 months' follow-up [mean \pm standard deviation (SD) = 17 [15], range = 0.5–54]. If data on race and ethnicity were available we coded white, non-Hispanic in the 'white' category and white Hispanic in 'other'. Note that there were insufficient data on other socio-demographic characteristics, for example education, marital status, or income, for their inclusion in the analyses.

Measures

Depression—Studies basing depression data on structured clinical interviews used various versions of the Composite International Diagnostic Interview (CIDI) [61]; Diagnostic Interview Schedule (DIS) [62]; Schedule for Affective Disorders and Schizophrenia (SADS) [63]; and Structured Clinical Interview for DSM (SCID) [64]. Studies basing depression data on self-report scales used various versions of the Beck Depression Inventory (BDI) [65]; Brief Symptom Inventory (BSI) [66]; Center for Epidemiological Studies—Depression Scale (CES-D) [67]; General Health Questionnaire (GHQ) [68]; Hamilton Rating Scale for Depression (HRSD) [69]; Millon Clinical Multiaxial Inventory (MCMI) [70]; PERI Depression Scale (PERI) [71]; Symptom Checklist—90, SCL-90 [72]; TCU Scale, TCU [55]; and Zung Depression Inventory (Zung) [73]. Investigators quantified the depression data using continuous indexes (e.g. BDI total score), categorical determinations (e.g. major depression diagnosis) or both. Details about the depression measure(s) used in each study and the manner in which depression data were quantified in the original reports are presented in Appendix I.

Drug use and impairment—Information in this domain was collected via participant self-reports and structured interviews, most often using a version of the Addiction Severity Index (ASI) [74]. Specific measures of drug use and impairment were percentage of time on drugs, frequency of drug use (non-prescribed drug use, heroin use, cocaine use, injecting), drug use status (presence or absence of any non-prescribed drug use, heroin use, cocaine use, injecting), number of classes of drugs used, number of classes of drugs dependent on, ASI drug related-impairment scale and classes of drug use disorder diagnoses. In addition, urine toxicology screening was used in one study to derive assessments of cocaine use and heroin use [26].

Alcohol use and impairment—Data on frequency of alcohol use, alcohol use status (e.g. abstinent, relapsed), alcohol-related impairment and alcohol use disorder diagnoses were based on seven studies.

Substance use treatment participation—In clinical studies, this variable was defined operationally in terms of the frequency or duration of treatment (hours of treatment attended, number of days in treatment, completing at least 90 days of treatment; 14 samples). Community-based studies assessed whether the participants were currently in drug-related treatment or have recently been in treatment (four studies).

Needle sharing—The information on whether the respondents had shared their needles with other drug users was reported in 13 studies. With few exceptions [18,20], data on the associations of depressive symptoms and needle sharing with cleaning versus without cleaning were unavailable.

Change in depressive symptoms—Eighteen 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.

Statistical integration of the findings

Computations were based on random-effects models [75]. (i) We computed effect sizes (d) for each study by transforming correlation coefficients, t-values, F-values and exact P-values [23]. Effect size estimates were adjusted for bias due to overestimation of the population effect size in small samples. If more than one depression measure was related to an outcome variable, we included the average effect size in our analysis. (ii) Studies were weighted by the inverse of their variances, and weighted mean effect sizes d and their confidence intervals (CI) 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 [23]. (iii) The significance of the mean was tested by dividing the weighted mean effect size by the estimated standard error of the mean effect size. (iv) Homogeneity of effect sizes was tested by using the homogeneity statistics (Q). (v) The overall goal of the multivariate analyses was to analyze whether the associations of depression with (a) concurrent drug use and impairment; (b) future drug use and impairment; (c) needle sharing; (d) treatment participation, varies by study characteristics; and (e) whether the amount of change in depressive symptoms varies by study features. In other words, can the between-study heterogeneity of the association of depression with the variables a-d, and the between-study heterogeneity in the amount of change in depressive symptoms (e), be explained by cross-study differences in participant demographic characteristics or recruitment venue? Thus, five weighted multiple ordinary least squares regression analyses were computed, following the random-effects approach and the method of moments [76]. The variables a-e (the effect sizes of the individual studies) were the dependent variables. Independent variables were mean age of the participants, percentage of men and sample status (clinical versus community-based) of the participants of these studies. Because an identification of significant moderating effects is difficult when

few studies are available, multivariate analyses were computed if at least 10 studies were available for individual research questions. Given that many studies did not report the percentage of white or racial/ethnic minority participants, univariate regression analyses were used to test for the moderating effects of race/ethnicity on the association between depression and substance-related behavior. (vi) As a tool for interpreting the practical significance of correlation coefficients, we used the Binomial Effect Size Display (BESD) [23]. For example, after the median split of the level of depressive symptoms and of substance-related behavior, the percentage of people with above-average depressive symptoms and above-average level of substance-related behavior is computed by 0.5 + t/2, and the percentage of above-average behavior level in the less depressed group is 0.5 - t/2. Studies providing longitudinal data on depression and future drug use and impairment and/or on change in depressive symptoms were distinguished from the remaining studies that provided cross-sectional data (see Appendix I).

RESULTS

Sample description

Forty-two samples were obtained from clinical venues and 13 were community-based samples in which none or a minority of participants were involved in substance use treatment. The sampling venue (clinical, community) and, for clinic-based samples, the nature of the service provided at the venue are listed in Appendix I. Overwhelmingly, clinic-based studies either used a purely substitution treatment sample (methadone and/or buprenorphine) or a mixed sample that combined subjects recruited from non-opioid substitution and substitution treatment venues. The participants had a mean age of 34.3 years (SD = 4.5 years); approximately 68% were men and 43% were white. Studies reporting data on marital status, education and employment suggest that approximately 21% of participants were married, 47% had graduated from high school and 23% were employed.

Associations of depressive symptoms with substance-related variables

We found a positive association of depression with concurrent drug use and impairment (Table 1). According to Cohen's [77] criteria the size of the association is small and according to the BESD, 55% of people with above-average levels of depressive symptoms show above-average levels of current drug use and impairment, compared to 45% of people with below-average levels of depressive symptoms. Interestingly, longitudinal studies found no significant prospective association of depressive symptoms with drug use and impairment.

Our results further showed a significant, but small, concurrent relationship between depressive symptoms and alcohol use and impairment. Higher levels of depression were associated with a small increase in the probability of needle sharing. People with higher levels of depressive symptoms also showed higher substance use treatment participation, but the size of the association was small. For example, according to the BESD, 53% of patients with above-average levels of depressive symptoms would show above-average levels of treatment participation, compared to 47% of patients with below-average levels of depressive symptoms.

On average, longitudinal studies (n = 18) showed a decline of depressive symptoms of d = 0.34 (95% CI = 0.21, 0.47) standard deviation units over time. The change is highly significant (t = 5.20, P < 0.001) although, according to Cohen's criteria, the size of decline is interpreted as small. The test for heterogeneity of effect sizes was also highly significant (Q = 39.10, P < 0.001). An additional analysis showed smaller improvement of depressive symptoms in studies with longer intervals (B = -0.01, beta = -0.56, t = -2.60, P < 0.02).

Analysis of moderating effects

With regard to the association of depressive symptoms and future drug use, we found a moderating effect of gender: Studies with a higher percentage of men were more likely to show a positive relationship between depression and future drug use and impairment (Table 2). Gender also moderated the relationship between depressive symptoms and substance use treatment participation: as shown in Table 2, there was a stronger relationship between these variables in samples with a lower percentage of men; in other words, samples with more women. Similarly, higher levels of depressive symptoms showed a stronger association with needle sharing in samples with a lower percentage of men. Two moderating effects emerged on the level of change in depressive symptoms. A stronger decline of these symptoms was observed in younger samples, and a marginally stronger decline in clinical samples. Clinical venue did not moderate the association of depression and the measures of substance use and impairment, contrary to hypotheses.

Two moderating effects of race/ethnicity appeared (Table 3): depressive symptoms were associated with greater substance use treatment participation and lower levels of future drug use in samples with larger percentages of white, non-Hispanic participants.

DISCUSSION

Results support the hypothesized positive association of depression and current drug use and impairment among IDUs. An association with alcohol use and impairment was also identified, broadly suggesting the relevance of depression in substance use and impairment. There are many potential explanations for the association [78] that include: pharmacological properties of opiates, alcohol and other substances inducing depressive symptoms; mood disturbance following substance withdrawal; a role of substance use in promoting or exacerbating stressors, for example interpersonal disruptions, that in turn influence mood; and the use of substances to cope with depressed mood.

Results show a significant association of depression with *greater* substance use treatment participation among IDUs. Perhaps depression serves as a motivator for seeking [1,12] and engaging actively in substance use treatment [15]. Another possibility is that depression serves as a counterweight to other characteristics, for example antisocial personality features, that could otherwise undermine treatment engagement among IDUs [79]. A role of depression in greater treatment participation may also help to explain the non-significant association of depression with future drug use and impairment, in so far as IDUs with higher treatment engagement may also be expected to show better drug-related outcomes.

Importantly, results indicate a significant association of depression with needle sharing, a key infectious disease risk behavior [80,81], suggesting the value of targeting depression to reduce the dissemination of HIV and other infectious diseases among IDUs. Needle sharing might be more common among IDUs with higher levels of depression, as it may represent an effort to cope with negative affect by affiliating with others through injecting. It is also possible that depression may promote hopelessness about the future, leading to more risk taking [18,21]. Moderator analyses supported an overall stronger association of depression and needle sharing among women, in line with some previous reports [20,21]. Given limited data we were unable to test additional factors that may explain these findings, including gender differences in the use of clean versus unclean needles that may, in turn, affect the magnitude of association of depression with needle sharing [20] and gender patterns of affiliation with other IDUs; for example, women may be more likely to share needles with a cohabitating partner [21].

Treated IDUs showed greater improvement in depression. This may be attributable to the beneficial effects of treatment, but treated samples may be expected to show a greater decline on account of their higher baseline levels of depression and lower vulnerability to show 'floor' effects. None the less, these findings are potentially important given that the current analysis contained general samples of IDUs, not those receiving specialized treatment for depression. Moderation analyses did not support a greater association of depression and concurrent drug use and impairment among female IDUs. Although this seems inconsistent with the notion that depression is more strongly intertwined with substance use among women, the lack of significance may have been based on the restricted variance in gender composition of the samples (55% to 86% men). Data were insufficient to test moderation associated with concurrent alcohol use and impairment as only seven studies were available, which is unfortunate because much of the evidence for gender differences in the association of depression and substance use are based on studies of alcohol abuse and dependence [78]. Other statistically significant moderating effects included higher associations of depression with substance use treatment participation among women and white non-Hispanics, and stronger associations of depression with future drug use and impairment among men and racial/ethnic minorities. Gender differences in treatmentseeking for mental disorders and physical disease have been well established [82]. As male and black or Hispanic IDUs tend to report lower levels of depression on self-report scales than women and white non-Hispanics [55,58], these effects may have been influenced by lesser variance in depressive symptoms in male and minority drug users. Younger IDUs also showed somewhat greater improvement in depressive symptoms, which may indicate that depressive symptoms become more chronic with increasing age and are therefore more difficult to change [83].

There were limitations of the study. Different measures of drug use and impairment, alcohol use and impairment, depression, treatment participation and needle-sharing, respectively, had to be combined into single summary measures. None the less, associations of depression with substance use treatment participation, alcohol use and impairment, and needle-sharing showed no significant between-study heterogeneity, and so differences in measurement of study variables did not play a role in these analyses. Depression was assessed typically by self-report measures that are sensitive to transient substance intoxication and withdrawal

effects. Data were not available to distinguish substance-induced and independent depressive symptoms. Reporting bias cannot be firmly ruled out; those who report more depression on self-report questionnaires may also report higher levels of drug use and impairment. Although the clinical-community categorization generally fit well, a small number of studies were well represented by clinical and community samples, and these border cases had to be forced into one or the other category. The meta-analysis focused squarely on depression and substance-related behaviors and impairment, along with changes in depression, but did not address other important correlates of depression (e.g. suicide, HIV progression, etc.). We were unable to examine the potential moderating influences of socioeconomic status or social instability because comparable measures of these data were not available across reports. Data were not available to disentangle drug use impairment attributable to the illicit status of psychoactive substances as opposed to that attributable to the pharmacological properties of the drugs themselves, although it may be hypothesized that depression is associated more strongly with the latter, an issue that should be examined in future studies. Reports were from western countries, most often the United States, which may limit generalizability. Some analyses were exploratory and warrant further replication. Correlations do not imply causation.

Although summary data on IDUs with clinical levels of depression are available [84], to our knowledge this study represents the first published meta-analysis of depression and substance-related behaviors among the general population of IDUs. Depression is relevant to several substance-related behaviors including current drug use and impairment, alcohol use and impairment, and needle sharing. However, to the investigators' surprise, effect sizes were small, and we also uncovered no evidence to support the idea that depression is associated with future drug use and impairment. Moderator analyses revealed that it is critical to consider the effects of socio-demographic characteristics, particularly gender, in order to evaluate the association of depression and substance-related outcomes. Indeed, when gender, age or race/ethnicity served as moderators, the effects were universally large in magnitude, illustrating the complexity of associations of depression and substance-related outcomes and of changes in depression over time. For example, the association of depression and treatment participation was stronger among women, suggesting that depression may serve as a motivator for treatment engagement and retention among female IDUs in particular. The mechanisms that relate depression to substance-related outcomes among IDUs remain unclear, including explanations for moderating effects, and require further study.

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APPENDIX I

Articles included in the meta-analysis

Report, (country of origin)	Recruitment setting (treatment venue if clinical)	n	Mean age	% men	% white	Type of report	Depression measure(s) (quantification of depression data)
Abbott <i>et al.</i> [24] (US)	Clinical (methadone)	144	35.0	71	15	Cross-s.	BDI, SCID (categorical, continuous)
Araujo <i>et al.</i> [25] (US)	Clinical (detoxification)	68	33.4	73	37	Cross-s.	HRSD (continuous)
Avants <i>et al.</i> [26] (US)	Clinical (methadone)	302	36.7	72	60	Longit.	BDI (continuous)
Avants <i>et al.</i> [27] (US)	Clinical (methadone)	106	34.0	43	60	Cross-s.	BDI (continuous)

Report, (country of origin)	Recruitment setting (treatment venue if clinical)	n	Mean age	% men	% white	Type of report	Depression measure(s) (quantification of depression data)
Bouhnik <i>et al.</i> [16] (France)	Clinical (buprenorphine)	243	35.0	72	n.r.	Longit.	CES-D (categorical, continuous
Brienza <i>et al.</i> [1] (US)	Community	528	36.0	76	78	Cross-s.	SCID (categorical)
Campbell <i>et</i> <i>al.</i> [28] (US)	Clinical (behavioral risk reduction)	598	26.0	77	60	Cross-s.	BDI (continuous)
Carrieri <i>et al.</i> [4] (France)	Clinical (buprenorphine)	114	33.6	67	n.r.	Cross-s.	CES-D (continuous)
Darke & Ross [29] (Australia)	Clinical (methadone)	222	29.8	59	n.r.	Cross-s.	CIDI (categorical)
Davis <i>et al.</i> [30] (US)	Clinical (methadone)	97	39.9	100	24	Longit.	BDI (continuous)
Dean <i>et al.</i> [31] (Australia)	Clinical (buprenorphine, methadone	54	29.5	62	n.r.	Longit.	BDI (continuous)
de los Cobos <i>et al.</i> [32] (Spain)	Clinical (detoxification)	40 sample #1	31.4	77	n.r.	Longit.	BDI (continuous)
de los Cobos <i>et al.</i> [32] (Spain)	Clinical (detoxification)	40 sample #2	29.9	85	n.r.	Longit.	BDI (continuous)
Dinwiddie et al. [33] (US)	Community	158	36.5	68	25	Cross-s.	DIS (categorical)
El-Bassel <i>et</i> <i>al.</i> [34] (US)	Clinical (methadone)	201	38.0	50	10	Cross-s	BSI (continuous)
Golub <i>et al.</i> [35] (US)	Community	193	25.8	76	65	Cross-s.	BDI, CES-D (categorical, continuous)
Gossop <i>et al.</i> [36] (UK)	Clinical (mixed)	753	29.0	73	92	Longit.	BSI (continuous)
Grella <i>et al.</i> [37] (US)	Clinical (methadone)	409	39.0	51	24	Cross-s.	CES-D (continuous)
Grella <i>et al.</i> [38] (US)	Clinical (methadone)	427	39.0	51	24	Longit.	CES-D (continuous)
Havard <i>et al</i> . [5] (Australia)	Clinical (mixed)	495	29.2	65	n.r.	Longit.	CIDI (categorical)
Hawkins <i>et</i> <i>al.</i> [20] (US)	Community	514	n.r.	80	21	Cross-s.	GHQ (continuous)
Joe <i>et al.</i> [15] (US)	Clinical (methadone)	981	37.0	61	48	Cross-s.	SCL-90 (continuous)
Johnson <i>et</i> <i>al.</i> [39] (US)	Clinical (mixed)	187	38.7	65	13	Longit.	SCID (categorical)
Johnson <i>et</i> <i>al.</i> [21] (US)	Community	513	38.5	76	55	Cross-s.	BDI (continuous)
Kang & DeLeon [40] (US)	Clinical (methadone)	152	33.0	61	37	Cross-s.	SCL-90 (continuous)
Knowlton <i>et</i> <i>al.</i> [13] (US)	Community	503	39.0	62	4	Cross-s.	CES-D (categorical, continuous
Knowlton <i>et</i> <i>al.</i> [6] (US)	Community	393	n.r.	64	6	Longit.	CES-D (categorical, continuous

Report, (country of origin)	Recruitment setting (treatment venue if clinical)	n	Mean age	% men	% white	Type of report	Depression measure(s) (quantification of depression data)
Kosten <i>et al.</i> [41,42] (US)	Clinical (mixed)	268	27.6	76	48	Longit.	SADS (categorical)
Kosten <i>et al.</i> [43] (US)	Clinical (buprenorphine)	40	30.7	76	93	Longit.	BDI (continuous)
Latkin & Mandell [44] (US)	Community	91	34.0	86	10	Longit.	GHQ (categorical, continuous
Maddux <i>et</i> <i>al.</i> [14] (US)	Clinical (mixed)	173	49.0	n.r.	<20	Longit.	Zung (continuous)
Mandell <i>et</i> <i>al.</i> [18] (US)	Community	499	36.0	70	2	Cross-s.	GHQ (categorical)
Margolin <i>et</i> al. [45] (US)	Clinical (methadone)	32	34.0	44	53	Longit.	BDI (continuous)
Margolin <i>et</i> al. [46] (US)	Clinical (methadone)	40	42.8	60	35	Longit.	BDI (continuous)
McCusker et al. [47] (US)	Clinical (residential)	162	29.2	71	n.r.	Longit.	DIS, BDI (categorical, continuous)
Metzger et al. [48] (US)	Clinical (methadone)	323	38.0	70	47	Cross-s.	BDI, SCL-90 (continuous)
Mino <i>et al.</i> [49] (Switzerland)	Clinical (methadone)	149	28.1	77	n.r.	Longit.	BDI (categorical)
Musselman & Kell [50] (US)	Clinical (methadone)	71	39.8	61	94	Longit.	MCMI (categorical, continuo
Nemoto & Foster [51] (US)	Clinical (methadone)	262	33.7	62	13	Cross-s.	PERI (continuous)
Pani <i>et al.</i> [52] (Italy)	Clinical (buprenorphine, methadone)	72	28.0	86	n.r.	Longit.	SCL-90 (continuous)
Perdue <i>et al.</i> [19] (US)	Clinical (mixed)	1228	37.0	66	59	Cross-s.	CES-D (categorical)
Rabkin <i>et al.</i> [9] (US)	Clinical (mixed)	187	38.7	65	13	Longit.	SCID, SCL-90 (categorical, continuous)
Rao <i>et al.</i> [7] (US)	Clinical (methadone) sample 1	727	37.2	60	47	Longit.	SCL-90 (continuous)
Rao <i>et al.</i> [7] (US)	Clinical (methadone) sample 2	432	37.2	59	48	Longit.	SCL-90 (continuous)
Rounsaville <i>et al.</i> [17,53] (US)	Clinical (mixed)	268	27.6	76	48	Longit.	BDI, SADS (categorical, continuous)
Schottenfeld et al. [8] (US)	Clinical (buprenorphine, methadone)	116	32.6	69	78	Longit.	SCID (categorical)
Schottenfeld et al. [54] (US)	Clinical (mixed)	120	32.5	74	64	Cross-s.	BDI (continuous)
Simpson et al. [55] (US)	Community	194	35.0	89	15	Cross-s.	TCU (continuous)
Steer <i>et al.</i> [56] (US)	Community	1290	35.3	76	18	Cross-s	BDI (continuous)
Strain <i>et al.</i> [57] (US)	Clinical (methadone)	66	36.0	55	79	Cross-s.	SADS (categorical)

Report, (country of origin)	Recruitment setting (treatment venue if clinical)	n	Mean age	% men	% white	Type of report	Depression measure(s) (quantification of depression data)
Strain <i>et al.</i> [58] (US)	Clinical (methadone)	58	34.3	67	41	Longit.	BDI (continuous)
Strathdee <i>et</i> <i>al.</i> [59] (Canada)	Community	281	34.9	68	58	Cross-s.	CES-D (categorical)
Teesson <i>et al.</i> [10] (Australia)	Clinical (mixed)	615	29.3	66	n.r.	Cross-s.	CIDI (categorical)
Torrens <i>et al.</i> [60] (Spain)	Clinical (detoxification)	62	25.9	76	n.r.	Longit.	BDI (continuous)
Wild <i>et al</i> . [3] (Canada)	Community	679	34.7	67	68	Cross-s.	CIDI (categorical)

n.r. = not reported. For the purpose of this meta-analysis, reports described as longitudinal (longit.) contributed data on change in depressive symptoms and/or depression and future drug use/impairment. Abbreviations for the depression measures are explained in the text along with the relevant citation.

Association of depression with substance-related behaviors.

0	52.29*	6.66	43.27*	22.96	15.86
t	$0.10 0.07 0.14 5.91^{*} 52.29^{*}$	$0.08 0.04 0.12 4.22^{*} 6.66$	0.48	4.59* 22.96	$0.11 0.08 0.14 8.23^{*} 15.86$
	0.14	0.12	0.07	0.09	0.14
CI	0.07	0.04	0.01 -0.05 0.07	0.06 0.04 0.09	0.08
r	0.10	0.08	0.01	0.06	0.11
k	25	٢	15	18	13
	Depression—current drug use and impairment	Depression—current alcohol use and impairment	Depression—future drug use and impairment	Depression-treatment participation	Depression—needle sharing

 $^{*}_{P<\,0.001.}$

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Table 2

Moderator effect of age, gender, and clinical status on the association of depressive symptoms with substance-related behavior and on change of denressive symptoms over time (multiple linear reoression analysis)

Conner et al.

VariableBBBBBBBBBBBBAge 0.01 0.32 0.04 0.73 -0.01 -0.15 0.00 0.06 -0.02^{**} -0.53^{*} $-0.53^{$		Association current dru	Association of depression with current drug use/impairment	Association of future drug us	Association of depression with future drug use/impairment	Association of depressio treatment participation	Association of depression with treatment participation	Association of d needle sharing	Association of depression with needle sharing	Improvement symptoms	Improvement of depressive symptoms
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	Variable	в	β	в	đ	в	β	в	β	в	β
$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$	Age	0.01	0.32	0.04	0.73	-0.01	- 0.15	0.00	0.06	-0.02	-0.53
0.11 0.28 0.03 0.05 -0.46 -0.09 -0.41 0.29^{***} 0.20 0.30 0.54 0.38 0.42 0.20 15 18 13 18	% men	-0.00	-0.02	0.03 *	0.95	-0.02	- 0.86	-0.00	-0.55 *	00.00	0.02
0.20 0.30 0.54 0.38 25 15 18 13	Clinical status		0.28	0.03	0.05	-0.05	- 0.46	-0.09	-0.41	0.29^{***}	0.37
15 18 13	R^2	0.20		0.30		0.54		0.38		0.42	
	u	25		15		18		13		18	
	$^{*}_{P<0.05},$										
$F_{<0.05}^{*}$	$^{**}_{P<0.01}$,										
$ \begin{array}{c} {}^{*} \\ P < 0.05, \\ \\ \end{array} \\ \end{array} \\ \begin{array}{c} {}^{**} \\ P < 0.01, \end{array} \end{array} $											

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 $^{***}_{P<0.07.}$

Clinical status (1 = clinical sample, 0 = others). Moderator analyses not performed on current alcohol use and impairment given too few reports.

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Table 3

Moderator effect of race/ethnicity on the association of depressive symptoms with substance-related behavior and on change of depressive symptoms over time (univariate linear regression analysis).

Variable B β B β B B B B B B β			nebLessi	depressive symptoms
-0.15 -0.02^{**} -0.82 0.68	В	В β	в	β
0.68	0.01^{*} 0.56	-0.00 -0.13	00.00	0.49
	0.31	0.09	0.24	
<i>n</i> 18 12 13	13	12	11	

P < 0.001.

Moderator analyses not performed on current alcohol abuse/dependence given too few reports.