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Meta-analysis of depression and substance use among individuals with alcohol use disorders

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Abstract

The relationships of depression with alcohol and drug use and impairment were examined. Additional analyses were conducted to examine moderators of these associations. Empirical reports on adults with alcohol abuse or dependence published in English in peer-reviewed journals since 1986 that contained data on depression and substance use variable(s) were obtained using a systematic search. The search yielded 74 studies including 58 reports from clinical venues, 10 that were community-based, and 6 with subjects from both settings. As hypothesized, the analyses showed that depression is associated with concurrent alcohol use and impairment and drug use and impairment. Effect sizes were small. Depression was also related to future alcohol use and impairment, an earlier age of onset of an AUD, and higher treatment participation. Age moderated the association between depression and alcohol use and impairment such that the association was stronger in older samples.

Keywords

depression; alcohol abuse; alcohol dependence; meta-analysis

1. Introduction

High rates of depression are common among individuals with alcohol use disorders (AUD), particularly alcohol dependence. Data from the National Comorbidity Survey estimated the lifetime prevalence of major depression to be nearly one quarter (24.3 %) among alcohol dependent men and nearly one half (48.5%) among alcohol dependent women, exceeding the prevalence rates among individuals without AUD. In clinical samples, the lifetime rates of co-occurrence are greater still, ranging from 50 to 70% (Cornelius et al., 1995; Curran & Booth, 1999; Hesselbrock et al., 1985).

Researchers have attempted to explain the association between AUD and depressive symptoms in a variety of ways. The pharmacologic effects of alcohol may produce symptoms of depression more or less directly during periods of intoxication and/or withdrawal (Brown et al., 1995). Relatedly, laboratory studies have shown that depressive symptoms can spontaneously emerge in the context of heavy drinking and abate with abstinence (Isbell et al., 1955; Tamerin et al., 1970). Chronic drinking and related symptoms may promote depression indirectly as well, for example by contributing to stressful life circumstances (e.g., partner-relationship disruptions) that in turn are known to promote depression (Sullivan et al., 2005). Other research supports the idea that depressed individuals are motivated to drink in an effort to cope with negative affect, a potential mechanism for development of AUD (Cooper et al., 1995; Schuckit et al., 2006). Although these and other mechanisms of co-morbidity have been investigated, no *single* definitive causal or shared etiological risk factor has been found to underlie both disorders (Swendsen & Merikangas, 2000; Schuckit, 2006).

Regardless of the mechanisms for elevated depression among individuals with AUD, depression poses a variety of risks to this population, the most grave of which include non-lethal suicide attempts (Preuss et al., 2002) and suicide deaths (Schneider et al., 2006). Interestingly however, data on the relevance of co-occurring depression to drinking and other substance use outcomes among AUD individuals are less definitive. Some studies show that the co-occurrence of depression is associated with poorer drinking outcomes including higher rates of treatment drop-out (Curran et al., 2002), greater risk for drinking relapse (Curran et al., 2000; Greenfield et al., 1998; Hodgins et al., 1999) and more rapid relapse following treatment (Glenn & Parsons, 1991; Svanum & McAdoo, 1989), but not all studies show poorer alcoholism outcomes (Turnbull & Gombert, 1988). A strong link of depression with substance use and impairment would suggest that depressed substance users may require enhanced treatment interventions. Data also suggest that depression is associated with greater treatment involvement (Burns et al., 2005) and use of more treatment services (O'Sullivan et al., 1988; Schuckit, 1985).

There is also a need to examine potential moderators of the association of depression and substance use and impairment among those with AUD. In particular, among individuals with AUD, data suggest gender differences in both rates of depression (Brady, Grice, Dustan, & Randall, 1993; Compton et al., 2006; Kessler et al., 1997) and depressive symptoms (Glenn & Parsons, 1991; King, Bernardy, & Hauner, 2003) with women showing more depression than men. Such differences may merely reflect the higher rate of depression observed among women in the general population (Hasin et al., 2005; 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, women with AUD 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. No meta-analyses to date have examined whether gender moderates the association between depression and measures of substance use and impairment among those with AUD. We hypothesize that this association will be stronger among women compared to men. Although they are not as large

as sex differences, rates of comorbid depression among alcoholics also vary by age (Schuckit et al., 1997) and race/ethnicity (Hesselbrock et al., 2003), and so we examined potentially moderating effects of age and race/ethnicity. We tested moderating effects of methodological features of studies that may act as moderators including the nature of the depression measure used (categorical or continuous) and the sample venue (clinical or community). These moderator tests were conducted because the high prevalence rates of depression in clinical samples of AUD (Cornelius et al., 1995; Curran & Booth, 1999; Hesselbrock et al., 1985) suggests that drinking and other substance use may be more heavily intertwined with depression in clinical populations, and because differences between categorical and continuous scales, including the statistical advantages of continuous measurement, may affect the size of associations between depression and substance-related measures in substance use populations (Conner et al., 2008).

In summary, the aims of the study were to: 1) Test the hypothesized positive associations of depression with concurrent alcohol use and impairment, general drug use and impairment, and treatment participation, and estimate the magnitude of the associations for each. 2) Test the hypothesized positive association of depression with future alcohol use and impairment. 3) Analyze the associations between depression and socio-demographic characteristics. 4) Describe changes in depressive symptoms over time. 5) Test the moderating effects of gender, age, race/ethnicity, nature of the depression measure used, and the type of sample venue.

2. Materials and methods

2.1. Sample

Studies were located by searching electronic databases using MEDLINE and PsychINFO in September, 2007 (search terms: depression AND [alcohol dependence, alcohol abuse, alcohol use disorders, or alcohol-related disorders]), limited to 1986 to 2007, English language, and Humans. Studies that became known to the authors through the remainder of 2007 were also included. We also examined the reference sections of relevant studies and review articles. 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.

Criteria for inclusion were: 1. Studies published in peer reviewed journals. 2. Studies that are exclusively or predominantly composed of adults with an AUD diagnosis of alcohol abuse and/or dependence, that present relevant data on subgroup(s) of adults with AUD diagnoses, or that recruited subjects from adult alcoholism treatment venues whether or not AUD diagnosis documentation was provided in the report (because extant data support that such settings overwhelmingly contain patients who meet criteria for AUD). 3. Studies containing at least one assessment of depression using a published, multi-item scale or a published, standardized diagnostic interview. 4. Association(s) of depression with measure(s) of substance use/impairment or other substance-related behavior (e.g., substance use treatment drop out) amongst individuals with AUD, or data on change in depression scores within this population, that were reported as correlations or as other effect size measures. 5. Mean age of the sample was 21 years or greater. Studies were excluded from the meta-analysis if: 1. Depression cutoffs, diagnoses, or subgroups were used as inclusionary or exclusionary

criteria to compose the sample. 2. Unpublished studies and reports that may not have been subject to peer review (e.g., book chapters, letters to editor, monographs). 3. They were trials of antidepressant medication(s) and/or psychosocial treatment(s) for depression. 4. Alcohol consumption data or referrals for drinking-related incidents (e.g., drinker driver program, college counseling clinic) alone were used to form the sample or subgroups, without supporting evidence to indicate AUD. 5. Studies that did not contain relevant comparisons amongst AUD individuals including reports in which data on depression and alcohol use and impairment were limited to comparisons between individuals with AUD and one or more groups without AUD, and general samples examining associations between depressive disorders or symptoms and AUD diagnosis or symptoms.

2.2. Measures

Depression—Studies basing depression data on structured clinical interviews used various versions of the Alcohol Use Disorders and Associated Disabilities Schedule (Grant & Hasin, 1992), Diagnostic Interview Schedule (Robins et al., 1981), Psychiatric Diagnostic Interview (Powell et al., 1985), Schedule for Affective Disorders and Schizophrenia (Endicott & Spitzer, 1978), Semi-Structured Assessment for the Genetics of Alcoholism (Bucholz et al., 1994), and Structured Clinical Interview for DSM (Spitzer et al., 1988). Studies basing depression data on rating scales used various versions of the Beck Depression Inventory (Beck et al., 1961), Brief Symptom Inventory (Derogatis & Melisaratos, 1983), Center for Epidemiological Studies – Depression Scale (Radloff, 1977), Hamilton Rating Scale for Depression (Hamilton, 1960), Health and Daily Living scale (Moos et al., 1992), Inventory to Diagnose Depression (Zimmerman, 1994), Initial Evaluation Form (Mezzich et al., 1981), Montgomery-Asberg Depression Rating Scale (Montgomery & Asberg, 1979), Millon Clinical Multiaxial Inventory (Millon, 1987), Personal Attributes Questionnaire (Spence et al., 1974), Hopkins Symptom Checklist (Derogatis et al., 1973), Symptom Checklist-90 Revised (Derogatis, 1977), and Zung Depression scale (Zung, 1965). 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 more than one measure for the assessment of depression.

Alcohol use and impairment—Data on frequency and/or intensity of alcohol use (27 studies), alcohol use status (e.g., abstinent, relapsed; 15 studies), and alcohol-related symptoms (e.g., withdrawal; 25 studies) were used to assess this domain.

Age of onset of alcohol-related problems (14 studies), number of years of these problems (5 studies), and family history of alcohol abuse or dependence (7 studies) were assessed with single-item indicators.

Treatment involvement—Data on the length of stay in alcohol treatment, on number of received treatments, and on treatment completion were analyzed (15 studies).

General drug use and impairment not specific to alcohol was assessed via the composite drug use scale of the ASI (McLellan et al., 1992), lifetime diagnosis of drug dependence, and self-report of use of other substances (e.g., marijuana) (7 studies).

Change in depressive symptoms—Thirty longitudinal samples contained in 28 studies 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.

2.3. Statistical integration of the findings

The analytic methods are comparable to those used in our prior meta-analyses of depression and substance use and impairment among cocaine users (Conner et al., in press) and intravenous drug users (Conner et al., 2008). For additional details, see those reports. 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 (Rosenthal, 1991) that were adjusted for bias due to overestimation of population effect size in small samples and included the average effect size if a study contained more than one measure. 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. Effect sizes were converted 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 alcohol use, age of onset of alcohol-related problems, treatment involvement, of alcohol use with prospective levels of depressive symptoms, and for the size of change in alcohol use. Thus, 6 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, whether the study used a clinical sample, 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 prospective association of alcohol use with depression, and for associations of depression with length of alcohol-related problems and family history of alcoholism 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).

3. Results

A total of 74 studies were included in the present meta-analysis (Andersohn & Kiefer, 2004; Benishek et al., 1992; Bickel & Amass, 1992; Blume et al., 2001; Bobo et al., 1998; Brown & Schuckit, 1988; Buydens-Branchey et al., 1989; Conner et al., 2005; Cornelius et al., 1995; Curran & Booth, 1999; Currie et al., 2001; Davidson, 1995; Davidson & Blackburn, 1998; Dick et al., 2007; Dorus et al., 1987; Driessen et al., 2001; Golding et al., 1993; Goldman & Bander, 1990; Grant, 1996; Greenfield et al., 1998; Hanna & Grant, 1997; Hasin et al., 1988a; Hasin et al., 1988b; Haver & Gjestaad, 2005; Heh et al., 1990; Heinz et al., 1996; Hesselbrock, 1991; Hodgins et al., 1999; Holdcraft et al., 1998; Ilgen & Moos, 2005; Karno & Longabough, 2003; Kirchner et al., 2002; Kranzler et al., 1996; Laine et al., 1999; Leibenluft et al., 1993; Litt et al., 2001; MacMurray et al., 1987; Maharaj, 1990; Markianos

et al., 2001; McMahon & Davidson, 1986; Miller et al., 1996; Moos & Moos, 2006; Oslin et al., 1999; Overall et al., 1985; Penick et al., 1988; Penick et al., 1994; Pettinati et al., 1997; Pomerleau et al., 1997; Powell et al., 1992; Roggla & Uhl, 1995; Rounsaville et al., 1987; Roy et al., 1991; Rudolf & Priebe, 2002; Salloum et al., 1995; Schuckit et al., 2003; Schuckit et al., 1998; Schuckit et al., 1997; Schutte et al., 1997; Schutte et al., 2006; Sellman & Joyce, 1996; Shields & Hufford, 2005; Singh et al., 1997; Sitharthan et al., 2001; Skaff et al., 1999; Stetter et al., 1991; Stewart et al., 2006; Timko et al., 1999; Turnbull & Gomburg, 1988; Vaglum et al., 1987; Wetterling & Junghanns, 2000; Windle & Miller, 1989; Windle & Miller, 1990). Fifty-eight studies collected data from clinical venues, 10 were community-based, and 6 studies combined clinical and community-based data. Participants had a mean age of 38.1 years ($SD = 6.2$ years) and about 70% were men and 82% were White. Over three quarters (79%) of the sample had completed high school, almost half (49%) were employed, and 38% were currently married. An additional six studies that met inclusion/exclusion criteria were identified (Booth et al., 1991; Braggio et al., 1991; Curran et al., 2000; Daepfen et al., 2000; Lis-Turlejska & Polak, 2002; Mattson et al., 1998) but they were excluded from the analysis because they provided no data for our research questions or because it was not possible to compute effect sizes.

3.1. Association of depression with substance-use variables

We found a positive association of depression with concurrent alcohol use and impairment. According to standard criteria (Cohen, 1992), the size of the association is small, and according to the BESD, 60.5% of persons with above-average levels of depressive symptoms show above-average levels of current alcohol use and impairment, as compared to 39.5% of persons with below-average levels of depressive symptoms (Table 1). The association of depression with alcohol-related impairment seemed to be slightly stronger than the association of depression with measures of frequency and intensity of alcohol use, but the overlap of the 95% confidence interval indicates that the difference was not statistically significant.

Longitudinal studies found that depressive symptoms at the first time of measurement predicted higher levels of alcohol-related impairment at the follow-up, and a stronger increase of alcohol use and impairment over time. In addition, higher levels of alcohol use and impairment at time one were associated with higher levels of depressive symptoms at the follow-up. No sufficient data were available for testing whether alcohol use and impairment at the first time of measurement would predict change in depressive symptoms over time. As shown by the non-overlap of the 95% confidence intervals, we found stronger concurrent than prospective associations of depression with alcohol use and impairment. According to Cohen's criteria, most prospective associations were small.

With regard to other substance-related variables, we found that depression was associated with an earlier age of onset of AUD. However, the association between depression and length of AUD was not significant, probably due to the smaller number of available studies ($N = 5$). Individuals with a family history of AUD had higher levels of depression, but the association was again small. Cross-sectional analyses also found that depression was positively related to general drug-use and impairment. In addition, we observed a small

positive association of depression with treatment involvement. According to the BESD, 56% of the individuals with above-average depressive symptoms showed above-average levels of treatment involvement, as compared to 44% of individuals with below-average levels of depressive symptoms.

Finally, we observed that the level of depressive symptoms declined over time by about 0.6 standard deviation units ($k = 28$, $d = -.62$, $Z = -7.47$, $p < .001$). According to Cohen's criteria, improvement of depressive symptoms was of medium size.

3.2. Association of depression with sociodemographic variables

Associations of depressive symptoms with socio-demographic variables are shown in Table 1. Women showed higher levels of depressive symptoms than men and whites showed higher levels of depressive symptoms than individuals of racial/ethnic minority. Levels of depressive symptoms did not vary by age, marital status, educational attainment, and employment status.

3.3. Analysis of moderating effects

Hypothesized moderating effects of gender on the associations between depression and substance use and impairment were not supported. With regard to moderator analyses of the association of depressive symptoms and concurrent alcohol use and impairment, we found a moderating effect of the method for assessing depression: associations between depression and alcohol use/impairment 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 weaker association of depression with future alcohol use and impairment in studies with a larger percentage of White individuals. In addition, associations of depression with increase in alcohol use/impairment were stronger in older samples. Finally, we found a stronger decline of depressive symptoms in older samples, in samples with a larger percentage of non-white participants, and in studies with longer time interval. No significant moderating effects were found for associations of depression with age of onset of alcohol misuse/dependence and treatment involvement, probably due to the restricted number of available studies.

4. Discussion

Results support the hypothesized positive association of depression and concurrent alcohol use and impairment and general drug use and impairment. The prospective studies included in the analysis also support an association between depression and future alcohol use and impairment. The data further indicate that more depressed individuals participated in more treatment than less depressed individuals. As we analyzed general samples of individuals with AUD, and excluded from the analysis studies that selected subjects using depression cutoffs, the findings suggest the relevance of depression to substance use and impairment and treatment participation in mainstream AUD samples, indicating that depressive symptoms may be an important consideration in the routine treatment of this population.

Contrary to our hypotheses, tests of moderation did not indicate that the size of the association between depression and the various measures of substance use and impairment is greater among women. As expected, results showed that among those with AUD, women have higher depressive symptoms than men. The magnitude of the difference between men and women is also generally consistent with extant data including a meta-analysis of depression and stressful life events in the general population (Davis et al., 1999) that showed gender differences ($r=.14$) that were similar to the gender differences found in the current meta-analysis ($r=.12$). Together with the absence of moderating effects of gender, the data suggest that gender differences in depression among those with AUD reflect a general propensity for women to show higher levels of depression, rather than a stronger link among women between depression and measures of substance use and impairment. Cautious interpretation is warranted however because in the present meta-analysis, 50% of the studies had more than 72% male participants, and only 8% were exclusively on women, making the identification of moderating effects of gender difficult.

Prospective studies indicated that those with AUD had a modest decline in depressive symptoms over time. Interestingly, moderator analysis indicated that the decline was stronger in older samples and in samples with a larger percentage of non-white participants, novel findings that require replication. Moderator analyses also indicated a stronger decline in studies with longer time intervals; a straightforward conclusion is that longer intervals offer more time for change. Continuous measures of depression were also more strongly associated with concurrent alcohol use and impairment compared to categorical measures, generally based on diagnostic interviews. This finding may be 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.

4.1. Limitations

There were limitations of the study. Different measures of 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. 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 AUD individuals with substance-induced and independent depressive symptoms, a limitation because these groups show differences on several relevant domains including substance use severity (Schuckit et al., 1997). Data were not available to disentangle whether degree of substance use at the first time of measurement predicts change in depressive symptoms over time. Of the 74 studies analyzed, only 10 used community samples, 3 of which had longitudinal designs, and so the relevance of the results to untreated individuals with AUDs, particularly in regards to the prospective association of alcohol use and

depression, is unclear. This limitation is significant because only about one-quarter of individuals with alcohol dependence ever receive treatment (Hasin et al., 2007). Reports were overwhelmingly from the U.S., which may limit generalizability. Correlations do not imply causation. The mechanism(s) for the associations of depression and measures of substance use and impairment and treatment participation could not be evaluated. Statistically significant moderator results obtained require further study and replication. The absence of statistically significant moderating effects of gender may be attributable to the restricted variance in gender composition of the samples that were composed mostly of men. Few studies were designed a-priori to examine the association of depression and substance use and impairment, and in many instances the data were not well suited to address this question. Finally, the current analyses concern the associations between depressive symptoms and drinking and other substance-related variables only, with unclear relevance to the proximal influence of more transient affective states (e.g., sadness, anger, anxiety) on drinking. Explicating the proximal influence of negative affective states on drinking requires studies that use more closely spaced assessments of affect and drinking (Hussong, 2007) or the use of alternative research designs, for example retrospective reports by AUDs of their emotional state immediately prior to a drinking occasion (Miller & Marlatt, 1996).

4.2. Future Directions

This study represents the first published meta-analysis of depression and substance use and impairment among individuals with AUD. Our findings indicate that depression is relevant both cross-sectionally and prospectively to measures of alcohol use and impairment. Findings also indicate that depression is associated with greater treatment participation. Effect sizes are small however, and moderating effects of gender were not identified. Although the current findings represent the best available summary analysis of these questions, cautious interpretation is warranted given limitations of available data. More definitive results can be obtained with studies designed a-priori to examine the relationship between depressive symptoms, substance-related variables, and treatment participation, as well as potential gender patterns of these associations. In particular, studies are needed that use prospective designs, assessments that are spaced closely enough to allow for the rigorous evaluation of the reciprocal influence of depressive symptoms and substance use and impairment, and well-chosen instruments including those designed to differentiate individuals experience substance-induced versus independent depressive episodes. Such studies should also test hypothesized mechanisms of these associations, for example, whether or not depression increases treatment participation by increasing motivation to seek care, by promoting treatment failure and recidivism, or by mobilizing greater services.

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Table 1**Correlates of depression in individuals with AUD**

Correlate	k	r	95%-CI	Z	Q
Sociodemographic variables					
Age in years	16	-.02	-.08 .03	-0.78	91.52***
Female gender (1=yes, 0=no)	25	.12	.08 .15	6.44***	108.18***
Being married (1=yes, 0=no)	12	-.04	-.09 .00	-1.86	33.89***
White (1=yes, 0=no)	10	.08	.01 .15	2.17*	94.13***
Educational attainment	13	-.01	-.05 .03	-0.55	26.60**
Employed	8	-.00	-.08 .08	-0.04	19.38**
Substance abuse/dependence					
Alcohol consumption/alcohol-related impairments (concurrent relationship)	49	.21	.16 .25	8.80***	526.54***
Alcohol use (concurrent relationship)	33	.15	.09 .21	5.08***	398.69***
Alcohol-related impairments (concurrent relationship)	27	.26	.20 .31	8.39***	156.20***
Depression (T1) – alcohol use/impairments (T2) (prospective relationship)	18	.08	.01 .15	2.26*	95.24***
Depression (T1) – use (T2)	13	.04	-.04 .12	0.93	48.89***
Depression (T1) – alcohol-related impairments (T2)	5	.19	.08 .28	3.51***	12.44*
Depression (T1) – increase of alcohol use/impairments (T1 – T2)	10	.08	.05 .11	4.71***	14.17
Alcohol use/impairments (T1) – depression (T2) (prospective relationship)	9	.14	.08 .19	4.95***	17.08*
Age of onset of alcohol misuse/dependence	14	-.08	-.14 -.03	-3.21**	42.77***
Years of misuse/dependence	5	.08	-.12 .27	0.77	44.59***
Family history of alcohol abuse/dependence	7	.09	.02 .16	2.51*	19.85**
Treatment involvement	15	.12	.08 .16	6.25***	27.48*
Drug use	7	.08	.01 .15	2.10*	58.93***

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,

.100) > d

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

Variable	Association. with concurrent alcohol use and impairment		Association with prospective alcohol use and impairment		Association of depression with age of onset of alcohol misuse/dependence		Association of depression (T1) with increase in alcohol use/impairment (T1 – T2)		Association of depression with treatment involvement		Decline of depressive symptoms ^I	
	B	β	B	β	B	β	B	β	B	β	B	β
Age	.00	.09	.01	.25	-.01	-.41	.04	1.20***	.01	.39	.05***	.47
% men	-.00	-.12	-.00	-.07	-.00	-.39	-.00	-.52	-.00	-.45	.00	.13
% white	-.00	-.04	-.01	-.51*	-.01	-.34	-.00	-.26	-.00	-.10	-.02***	-.52
Clinical sample (1=yes, 0=no)	-.08	-.08	.21	.17	-.06	-.09	.22	.23	-.07	-.21	.32	.11
Depression measure (1=dummy variable, 0=continuous variable)	-.33	-.47***	-.26	-.37	.48	.64	.28	.87	-.06	-.20	-.32	-.20
Length of study interval												
Constant	.74		.90		1.05		-1.14		.09		.92	
R ²	.19		.43		.45		.81		.28		.43	
k	49		18		14		10		14		27	

Note. B (β) = unstandardized (standardized) regression coefficient, k = number of samples, R² = explained variance,

^I positive regression coefficients indicate that higher scores of the independent variable are associated with stronger decline of depression.

* p < .05,

** p < .01,

*** p < .001.