Published in final edited form as:

Compr Psychiatry. 2015 August ; 61: 64–71. doi:10.1016/j.comppsy.2015.05.010.

Proximal vs. distal predictors of alcohol use disorders and treatment utilization in at-risk men in early middle age

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Abstract

Understanding factors that predict both development and treatment of alcohol misuse and its consequences can inform prevention and treatment efforts. This study used measures of both proximal (e.g., behaviors) and distal (e.g., traits) risk factors that were predicted to relate to both an alcohol use disorder (AUD) and to treatment utilization among AUD-diagnosed men to test the hypothesis that both type of factors predict AUDs but only proximal factors influence treatment-seeking. Analyses of variance with contrasts were used to compare the means for risk factors between men with an AUD and AUD-free men and—given an AUD diagnosis—between men who were treated for an AUD and untreated men ($n = 181$). As predicted, men with AUDs differed on a broad range of proximal and distal factors, including number of alcohol problems, alcohol-related influences of peers and partners, alcohol expectancies, familial factors, and psychopathology. As hypothesized, only proximal risk factors predicted treatment-seeking among the AUD men, particularly alcohol problems and related consequences.

1. Introduction

Research on the etiology of problematic drinking and alcohol use disorders (AUDs), particularly on the roles of risk and protective factors, is needed to understand the dynamics of addiction and to inform prevention approaches [1,2]. Known risk factors for problematic drinking include influences of peers and romantic partners, such as drinking by significant others [3–7], preexisting or co-occurring psychopathology [1], other substance abuse disorders [8], and socioeconomic status (SES) [9].

A consistent finding is that most people meeting diagnostic criteria for an AUD never receive the needed treatment [10]. For example, analysis of data from a large national sample (National Epidemiologic Survey on Alcohol and Related Conditions) [11] found that only 24% of respondents diagnosed with an AUD ever sought professional help [8]. Another study found that few college students with any type of substance use disorders had ever entered treatment [12]. Accordingly, a separate line of alcohol research has addressed determinants of treatment utilization among individuals who need treatment [13].

Studies of individuals with AUDs that have compared people treated for their disorder with those who have never been in treatment have identified several discriminating variables.

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Treatment utilization for AUDs was associated with severity of alcohol abuse [14,15], social influence of peers and partners [12,16], low SES [15,17], problems with other substances [14], and recognition of the need for outside help [12]. Thus, consistent with life-span developmental models for risk behaviors and associated factors [18], a number of factors are related to treatment utilization. However, there appears to be an important distinction between factors that predict AUDs and determinants of treatment-seeking. Specifically, AUDs appear to be more strongly associated with distal factors (e.g., traits) associated with antecedents of psychopathology, including genetic predisposition and inappropriate parenting, whereas treatment-seeking seems to be more influenced by proximal factors (e.g., current drinking behaviors, social influence, and alcohol-related arrests).

This hypothesis could be tested by examining factors that predict AUDs and treatment utilization in the same sample with the same variables in order to compare patterns of prediction for the two types of AUD outcomes (AUD diagnostic status vs. treatment utilization, given an AUD diagnosis), but such a study has yet to be conducted. In the present study, we are predicting that AUDs will be associated with a broader range of univariate predictors than will treatment-seeking, and the difference will be qualitative as well as quantitative. That is, treatment-seeking is expected to be associated only with proximal factors, particularly consequences of problematic drinking.

Thus, this study examines risk factors for AUD diagnoses and alcohol treatment participation in the longitudinal Oregon Youth Study (OYS), comprising a community sample of men who had lived in relatively disadvantaged neighborhoods in childhood and deemed at-risk for development of problems. The OYS men were assessed regularly over 3 decades for risk and protective factors for AUDs, and for treatment for an AUD in the past year from the preteen years until early middle age. For this study, the OYS men were divided into three groups: (1) men who never had met diagnostic criteria for an AUD by ages 37–38 years (when last examined), (2) men who had an AUD history but never reported receiving treatment, and (3) men diagnosed with AUDs who also had a history of treatment utilization. Because all men received a comprehensive assessment approximately every year or two over the course of the study, comparisons among the three groups on the measures were conducted to determine whether the variables that predicted AUDs also influenced treatment utilization among the subgroup of men diagnosed with an AUD. This study complements and extends our earlier work with the OYS men that had examined risk factors associated with heavy alcohol consumption, heavy episodic drinking (HED), and alcohol problems [4,6,12,19] but did not investigate prediction to AUDs or to treatment-seeking in the subsample of OYS men.

For this study, predictor variables were first categorized as proximal or distal. The proximal variables were then classified into one of two categories: drinking behaviors (and its consequences) and social influence factors related to alcohol use (e.g., heavy drinking by one's friends or romantic partners). The distal variables were sorted into one of three categories: SES (education, verbal intelligence, and income), psychopathology (internalizing and externalizing behaviors), and alcohol expectancies/drug use. Of most importance, we included measures of alcohol expectancies because such expectancies may affect treatment utilization but this association has largely gone unexamined in the research on treatment-

seeking. Although the alcohol expectancy construct could be seen as proximal or distal, current operationalizations of it are essentially personality measures that would fall into the distal measures category and thus be predicted to be more strongly associated with AUD diagnoses rather than treatment utilization.

2. Methods

2.1. Sample

A total of 206 9–10-year-old boys and their families were recruited for participation in the long-term OYS from schools in neighborhoods in the Pacific Northwest characterized by higher rates of juvenile delinquency for the area. The boys were assessed annually or biennially from 1984 through 2013 [20,21]. Past year substance use measures were first collected when the boys were aged 11–12 years, but some of the predictors used in the current study were not administered until later waves. Most substance use measures were the same at every OYS assessment. The current study included the men who had participated in the OYS through the last wave of data collection, when they were 37–38 years of age, and for whom AUD and treatment data were both available. The men were mostly non-Hispanic Whites (90%), and 75% were of low SES. Starting when the men were late adolescents (ages 17–18 years), they were invited to participate in the OYS-Couples Study, approximately biennially, with a current romantic partner, and substance use-related measures were obtained from partners. All procedures were approved by the Institutional Review Board of the Oregon Social Learning Center.

From the original sample of 206 OYS boys, 5 died prior to participation in the last two assessments, 1 reported treatment but was lacking diagnostic data, and a further 16 did not participate when the final treatment instrument was administered. This resulted in 183 participants, 2 of whom reported treatment but no AUD and were thus eliminated from the current study, resulting in a final N of 181.

2.2. Measures

Each predictor used in this study was assigned to one of five categories, three of which were deemed distal and two proximal.

2.3. Distal measures

2.3.1. Socioeconomic/Cognitive

2.3.1.1. Verbal intelligence: Verbal intelligence was measured by the Vocabulary Subscale of the Wechsler Intelligence Scale for Children-Revised (WISC-R) [22] or the Wechsler Adult Intelligence Scale-Revised (WAIS-R) [23] on three occasions (WISC-R at ages 13–14 years; WAIS-R at ages 20–21 and 31–32 years).

2.3.1.2. Income: From ages 9 through 20 years, the parents were asked in the demographics segment of the Parent Interview [24] to describe their annual household income, using an eight category answering scale (1 = 0–\$4999; 8 = \$40,000) until age 15 years, after which the scale added a category (9 = \$50,000+). From age 21 years on, the OYS man reported gross personal income in dollars during the interview. To limit the effect of outliers, these

raw scores were recoded to the 1–9 scale used by the parents, and then all reports were recoded to the z scale prior to calculation of the variables.

2.3.1.3. Education: The men's educational attainment was measured as the last grade the men reported completing using a 10-category scale (grade 8 or less = 1, master's degree or higher = 10). Parental education for each parent was measured with a 7-point scale (grade 7 or less = 1, and some graduate training or more = 7) and the average of the two measures defined this variable.

2.3.2. Psychopathology

2.3.2.1 Antisocial behavior: Antisocial behavior was measured with the Elliott Delinquency Scale [25], with first administration $\alpha = .94$. Arrest data were used as an additional indicant of antisocial behavior. Each year the male participated, he was asked about all the places he had lived since his last participation. Several times during the 29 years of the study, staff wrote to every jurisdiction for the area the man had reported living in, asking for arrest records on the individual. The number of criminal arrests, as well as subscores of number of arrests for alcohol (e.g., DUIs) and drug offenses, were then calculated for each year of the study. Total number of arrests and a number of arrests for drug offenses were considered as behavioral measures of externalizing behavior. (Number of alcohol-related arrests was included in the proximal category.)

2.3.2.2. Depressive symptoms: The men were administered the Center for Epidemiologic Studies Depression Scale (CES-D) [26] on 20 occasions from ages 14–15 to 37–38 years, with first administration $\alpha = .85$.

2.3.3. Drug-related

2.3.3.1. Marijuana and other illicit drug use frequency: At each assessment from ages 11–12 to 37–38 years, men were asked about their frequency of marijuana use in the past year on a 9-point rating scale ranging from never “0” to 2–3 times a day “8”. The men were also asked about their frequency of use of substances other than tobacco, alcohol, or marijuana to “get high,” as providing ratings for frequency of use of other drugs on the same 9-point scale.

2.3.3.2. Parental alcohol use: Parental alcohol use was measured by the mean frequency of use by the men's mothers and fathers across nine measurement occasions (see Ref [19]), from the OYS men's ages 9–10 to 17–18 years. At each time, each parent provided a report of their own drinking and that of their partner. The parental alcohol use measure at each time was an average of the two reports for each parent.

2.3.3.3. Age at first intoxication: Starting at ages 9–10 years, the participants were asked at each wave whether they had even been drunk, and the age was recorded for the first positive response [19].

2.3.3.4. Alcohol expectancies: Expectations about the effects of alcohol were measured using nine self-report items (e.g., “alcohol makes people worry less”) when the children

were aged 9–10 and 11–12 years (α s = .61–.65), and when the men were aged 35–36 and 37–38 years using the Modified Drinking Motive Questionnaire-Revised [27]; α s = 0.98 at both assessments).

2.4. Proximal measures

2.4.1. Social influence

2.4.1.1. Partner and peer heavy drinking: Male peer heavy drinking (HD) was assessed twice when the OYS men were aged 20–23 and 37–38 years using the OYS men's reports of “How many of your friends get drunk once in a while?” Responses were on a 5-point Likert scale, ranging from 0 (‘none of them’) to 4 (‘all of them’), and scores were summed to generate the peer drinking measure for each assessment wave.

Romantic partners' HD was assessed up to eight times from the men's ages 20–23 to 37–38 years. The open-ended answers provided by the partner were recoded to a 4-point scale as follows; 0 = ‘0 drinks’, 1 = ‘1–2 drinks’, 2 = ‘3–4 drinks’, and 3 = ‘5 or more drinks’, and then summed to yield the Partner HD score for each wave at which the man participated with a partner.

2.4.1.2. Romantic partner and male peer substance use influence: At each of the OYS-Couples Study assessments from ages 27 to 38 years (up to five participations), the romantic partner completed a social influence questionnaire. A male peer was administered the questionnaire on two occasions (OYS men's ages 23–24 and 27–28 years). The items from each questionnaire formed seven subscales (OYS men's substance use, partner/peers' facilitation of men's substance use, partner/peer substance use, partner/peers' avoidance of substance use situations, partner/peer organize/enjoy socializing without substances, partners/peer disapproval of substance use, and partner/peers' attempts to quit substances) that were combined to yield a substance use influence score.

2.4.2. Alcohol related—Predictors included three measures of men's alcohol behavior: heavy episodic drinking (HED), alcohol consumption (based on volume and frequency of use) [6,19], and alcohol problems [19]. These three measures were assessed 21 times from ages 11–12 to 37–38 years. For the alcohol problems scale, first administration α = .72. (Because the MAST was used as one of the measures of an AUD, it could not be used in the analysis as a predictor of that outcome.)

The men's HED was assessed by the question, “In the last 2 weeks, how many times did you have 5 drinks or more in a row?” Responses ranged from 0 (‘never’) to 3 (‘more than twice’), resulting in a 4-point scale. Alcohol consumption was measured by alcohol volume and frequency, as described in our prior work [19]. Number of alcohol-related arrests also fell into this category.

2.5. Outcome measures

The two outcome measures were lifetime diagnoses of AUDs and self-reported receipt of treatment for alcohol problems by ages 37–38 years for men who had an AUD. A structured psychiatric interview was administered to determine lifetime DSM-IV diagnoses for an

AUD at three times, when the men were aged 16–17 years, 25–26 years, and 35–36 years. The Diagnostic Interview Schedule for Children (DISC) [28] was administered once when the men were late adolescents, and the Composite International Diagnostic Interview (CIDI) [29] was administered twice when the men were adults. In addition, the men completed the Michigan Alcohol Screening Test (MAST) [30] at each wave; a score exceeding 12 on that scale indicates a diagnosis of an AUD. An AUD history was defined as either being diagnosed with an AUD at least once with the DSM-IV [31] criteria using the DISC or the CIDI, or scoring above 12 on the MAST at least 1 time. Given that the MAST was administered at 7 time points from ages 20–21 to 37–38 years, including it likely gave a more accurate score of meeting AUD criteria because there were substantial gaps between the diagnostic interview assessments. At first administration, $\alpha = .72$.

At each time from adolescence, men were asked to report whether they had received treatment for substance use problems in the previous year. At ages 35–36 years, an interviewer went through each of these answers with the men to check if the treatment was for alcohol use or for use of other substances (or both). At ages 37–38 years, the men were specifically asked about whether they had received treatment for alcohol use since the prior assessment. If a man with an AUD diagnosis had ever reported participation in treatment, he was assigned to the treatment category and the remaining AUD men were assigned to the no-treatment category. Thus, all 181 men were assigned to one of three mutually exclusive and exhaustive groups according to their diagnostic and treatment histories: AUD-free ($n = 55$), AUD without treatment ($n = 85$), and AUD with treatment ($n = 41$).

2.6. Data analytic plan

Analysis of variance (ANOVA) with contrasts [32] was first used to compare the means of the AUD-free with the AUD men for each predictor variable, and also between the treatment and no-treatment subgroups among men with an AUD diagnosis. For repeatedly measured variables, the groups were compared on each measure based on (a) initial administration (to determine the prognostic value of the variable) and (b) a time-averaged measure (based on all waves at which the measure was administered). Effect sizes (standardized mean differences) were expressed in the d metric [33], and the effect size and its 95% confidence interval were calculated for each contrast using established equations [34].

Next, the mean d s were calculated for each subcategory of measures, separately for predicting AUDs and treatment-seeking among the subsample of men with a lifetime diagnosis of AUDs. The key tests of our hypotheses were based by comparing mean effect sizes for AUDs and treatment utilization for each of the give categories of the predictors.

Finally, in order to determine the validity of lifetime measures of AUD based on retrospective reports in middle age, a 2×2 cross-tabulation of the AUD frequency data was used to compare AUD diagnosis on the CIDI at two times. If the men were perfect reporters at each time point, every man who met lifetime criteria at the first assessment with the CIDI should be similarly diagnosed at the second assessment when the CIDI was re-administered to them about 10 years later.

3. Results

3.1. Descriptive statistics

Table 1 reports the descriptive statistics for the measures at three time points: first assessments, and when the CIDI was administered at 25–26 and 35–36 years.

3.2. Prevalence of AUDs on the CIDI at ages 25–26 and 35–36 years

The prevalence of lifetime AUD diagnoses from the CIDI for the 181 OYS men at ages 25–26 and 35–36 years included in the present analyses is reported in Table 2. At both times, the prevalence of AUDs was about 50%. However, of the 94 men who did not have an AUD in their 20s, 27 (29%) were newly diagnosed with an AUD in their 30s. All men who had a lifetime diagnoses of an AUD in their 20s should have been similarly diagnosed when reexamined 10 years later. However, of the 89 men with such a diagnosis in their 20s, 24 (27%) were not so diagnosed when in their 30s, indicating that men may either forget or choose not to report symptoms of abuse and dependence experienced substantially earlier in their lives. Thus, when lifetime diagnosis was based on a diagnosis at either age, 118 of the men, or 64%, had a lifetime diagnosis of an AUD based on the CIDI. That number was increased to 70% when AUD diagnoses based on the DISC and/or MAST were also used to determine lifetime AUD diagnoses.

3.3. Prediction of AUDs and treatment utilization for men with AUDs

Table 3 reports the findings from the contrast analysis for all the predictors, as grouped by category, for each of the alcohol-related outcomes. The top half of the table reports the comparisons among means for the three categories distal predictors and the bottom half reports the results for the two categories of proximal variables. The left hand side of the table reports the findings for AUD prediction and the right hand side gives the respective results for treatment utilization. The within-domain mean effect sizes for each outcome are reported in the row following the last variable in each category.

As hypothesized, the mean effect sizes indicated that AUD and AUD-free men differed in the variables in all five domains that included both proximal and distal classes of predictors. These average effect sizes for the group differences all exceeded .20, which is considered to be the minimum value for a mean difference to be meaningful. Note that it was high rather than low SES that was more common among the men with AUDs. However, the SES difference was the results of AUD men having come from more educated families and attaining more education. Verbal intelligence and income were not associated with AUD diagnoses. For all variables for which comparisons were made separately for first assessed and time-averaged measures, a larger number of findings were statistically significant for the latter than the former, with effect sizes consistently larger for the time-averaged variables. This pattern suggests that individuals whose status on the predictor variables changed for the worse as they matured were more likely to have developed an AUD by early middle age.

As hypothesized, none of the mean effect sizes for the three proximal variables indicated a meaningful association between these variables and AUD status, as all three mean ds were less than .20. Moreover, the tests of individual variables indicated that only 2 of the 21

proximal predictors significantly differentiated between those who did and did not receive treatment, which is about what would be expected by chance.

The hypothesis that treatment-seeking would be associated with the proximal predictors received only partial support. The alcohol-related proximal measures, particularly alcohol-related problems, did discriminate between the two subgroups of AUD men as predicted, with a meaningful mean effect size of .28. In addition, although the MAST could not be treated as a predictor of AUDs because the MAST was one of the measures used to determine AUDs, it could be used as a predictor of treatment among the men with AUDs. The AUD men who had received treatment had much higher time-averaged MAST scores than untreated AUD men, $t = 5.93$ ($p < .001$) $d = .89$ (CI = .50, 1.27). However, contrary to prediction, the social influence measures, which were predictive of AUDs, were not at all associated with treatment-seeking.

4. Discussion

As hypothesized, comparisons between the means of AUD and AUD-free men during early middle age found differences between the groups on a broad range of proximal and distal factors, including educational attainment, psycho-pathology, heavy drinking by peers and romantic partners, alcohol expectancies, alcohol-related social influence measures, and alcohol behavior. Also as predicted, fewer differences were observed between the treated and untreated men among the subgroup of men with a history of an AUD, with the discriminating variables being limited to proximal factors, especially alcohol problems. However, our prediction that social influence factors would affect treatment utilization was not supported.

Interestingly, HED was not associated with treatment utilization, indicating that problematic alcohol use only results in treatment involvement when it causes noticeable symptoms. Thus, it is the consequences of drinking that appear to drive treatment utilization. The finding that alcohol-related arrests (but not arrests for drug use or other infractions) predicted treatment involvement is consistent with this explanation, especially as only alcohol-related arrests are likely to result in mandated treatment for alcohol abuse [35,36].

One important issue raised by the findings of this study regards the validity of one-time only self-reports for estimating lifetime diagnoses of AUDs. When a psychiatric interview is administered to assess lifetime AUDs, as is standard practice in epidemiological research [8], there is an assumption that respondents accurately reported their history of AUD symptoms. Yet, the present study found that a single diagnostic assessment of lifetime AUD appears to involve underreporting of the prevalence of AUDs. There were an appreciable number of OYS men who had a late onset of an AUD, which was offset by about the same number of men who had a lifetime AUD diagnosis on the CIDI in the mid-20s but not when interviewed when they were in their mid-30s. Thus, when a lifetime diagnosis on the CIDI at ages 35–36 years was based on whether the men had a diagnosis of an AUD at either of the two ages, the prevalence rate increased from about one half to about two thirds. Moreover, when diagnostic information from the DISC and MAST collected from other years during the course of the study were also considered, the prevalence rate for a lifetime AUD

increased to 70%. These results indicate that prevalence of a lifetime AUD will be underestimated at middle age if based on a single interview conducted when respondents are in their 30s, and that attempts to determine predictors of AUDs in middle age will be biased if examinations use a cross-sectional design based on retrospective reports of AUD symptoms. In addition, because treatment experience was assessed throughout the course of the 3-decade study and a treatment history was based on any reports of treatment at any wave, the assessment of treatment did not have the underestimation bias that also may have been found had we only assessed the men's treatment history in their 30s. Thus, our ability to determine accurately whether middle-aged men had a lifetime history of AUDs and, if so, whether they ever received treatment for an AUD is an important strength of this study of predictors of AUDs and related treatment.

Although the study was unusual in the availability of regular prospective assessments from late childhood to the late 30s, it also had several limitations. Time-averaged predictors were used to predict both AUDs and treatment-seeking. Given the complexity of timing of the AUDs and treatment occasions, the possibility that each could involve prolonged or multiple episodes, and the varying times when some of the predictors were assessed (e.g., ranging from childhood until adulthood), time-averaged predictors were considered a robust measure because the measures were correlated over time. However, time sequences (e.g., peer drinking prior to an AUD or to treatment) cannot be assumed. For example, having an AUD could cause depressive symptoms rather than depressive symptoms being a predictor of an AUD. Because our primary diagnostic measures of AUDs were infrequently administereded, it was not possible to determine precisely when each OYS man first met the criteria for an AUD, which made it impossible to determine sequencing of behaviors. Second, we did not assess recognition of the need for treatment, which is a key predictor of help seeking among men with alcohol problems [12]. Third, not all possible antecedents (e.g., anxiety symptoms, emotional regulation) for AUDs and treatment utilization were examined. Finally, the study involved predominantly White men from lower SES backgrounds, and findings may not be generalizable to minorities, women, and professionals.

These limitations notwithstanding, the finding that only alcohol problems and involvement in the justice system seemed to affect treatment involvement, as opposed to the broader range of factors that discriminated AUD from AUD-free men, suggests a need for identifying men with an AUD and providing needed treatment to them to forestall development of alcohol problems.

Acknowledgments

Funding for this study was provided by awards from the National Institutes of Health (NIH), U.S. PHS Award Number 1R01AA018669 from the National Institute on Alcohol Abuse and Alcoholism (NIAAA); R01 DA 015485 from the National Institute of Drug Abuse (NIDA); and HD 46364 from the National Institute of Child Health and Development (NICHD). The content is solely the responsibility of the authors and does not necessarily represent the official views of the NIH, NIDA, NIAAA, or NICHD. NIH, NIDA, NIAAA, or NICHD had no further role in study design; in the collection, analysis, and interpretation of data; in the writing of the report; or in the decision to submit the paper for publication.

We thank Jane Wilson and the data collection staff for their commitment to high-quality data, and Sally Schwader for editorial assistance.

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Table 1

Means and percentage for demographic characteristics and time-varying measures for at-risk men at first assessment and Composite International Diagnostic Interview (CIDI) assessment times.

Measure	Age first assessed for present study (in years)	First assessment	Ages 25–26 years	Ages 35–36 years
Age	11–12	11.9 (.48)	25.9 (.49)	36.2 (.56)
% Married		0	30.5	53.0
% with AUD	16–17	4.97	54.1	69.6
HED	11–12	.01 (.14)	.80 (1.35)	.62 (1.23)
MAST	20–21	3.81 (4.69)	4.48 (5.60)	4.43 (8.15)
Partners' heavy drinking	20–23	1.89 (1.11)	1.84 (1.14)	1.65 (1.05)
Peer heavy drinking	21–22	2.72 (1.13)	2.41 (1.24)	1.94 (1.34)
Vocabulary	13–14	9.8 (2.7)	9.2 (2.6)	10.9 (3.3)
Elliott Delinquency Scale	12–13	23.2 (75)	11.7 (37)	21.9 (196)
Total arrests (past year)	11–12	.13 (.66)	.21 (.61)	0 (0)
Depressive symptoms	14–15	11.33 (8.09)	7.67 (7.58)	8.8 (8.38)
Alcohol-related arrests	9–10	0	.02 (.18)	.03 (.23)
Drug use-related arrests	9–10	0	.03 (.23)	.02 (.18)

HED: heavy episodic drinking; MAST: Michigan Alcohol Screening Test.

Table 2

2 × 2 Cross-tabulation of frequencies of lifetime AUD diagnoses from the Composite International Diagnostic Interview (CIDI) at ages 25–26 and 35–36 years.

Ages 35–36 years			
Ages 25–26 years	No AUD	AUD	Total
No AUD	67	27	94
AUD	24	65	89
Total	91	92	183

AUD: alcohol use disorder.

Table 3

Contrast analyses comparing means of distal and proximal measures of AUD men with AUD-free men and of AUD men who received treatment with untreated AUD men.

Category and measure	AUD versus no AUD		Treatment versus no treatment	
	<i>t</i>	<i>d</i> (CI for <i>d</i>)	<i>t</i>	<i>d</i> (CI for <i>d</i>)
<i>Distal Measures</i>				
Socioeconomic status and cognitive-related				
Education	-2.37*	-.35 (-.67, -.04)	-.94	-.14 (-.51, .23)
Parental education	-2.43*	-.36 (-.68, -.04)	-1.53	-.23 (-.60, .15)
Income				
First assessed	-1.26	-.19 (-.51, .13)	-1.70	-.25 (-.63, .12)
Time averaged	-1.54	-.23 (-.55, .09)	-.60	-.09 (-.46, .28)
Vocabulary				
First assessed	-1.21	-.18 (-.50, .14)	-1.02	-.15 (-.53, .22)
Time averaged	-1.65	-.25 (-.56, .19)	-1.25	-.19 (-.56, .19)
Mean <i>d</i>	-.26		-.18	
Psychopathology				
Elliott Delinquency Scale				
First assessed	2.24*	.33 (.02, .65)	.96	.14 (-.23, .52)
Time averaged	2.51*	.38 (.06, .69)	2.12*	.32 (.01, .63)
Drug-related arrests	-.56	-.08 (-.40, .23)	.22	.03 (-.34, .41)
Total arrests	1.89	.28 (-.04, .60)	1.41	.21 (-.16, .58)
Depressive symptoms				
First assessed	1.22	.18 (-.13, .50)	1.94	.29 (-.08, .66)
Time averaged	2.14*	.32 (.01, .63)	1.14	.17 (-.20, .54)
Mean <i>d</i>	.24		.19	
<i>Drug related</i>				
Parental alcohol use				
First assessed	1.10	.16 (-.15, .48)	.88	.13 (-.24, .50)
Time averaged	1.98*	.30 (.01, .59)	1.06	.16 (-.21, .53)
Marijuana use				
First assessed	2.22*	.33 (.01, .65)	1.33	.20 (-.17, .57)
Time averaged	5.01***	.75 (.42, 1.07)	.92	.14 (-.24, .51)
Other drug use				
First assessed	2.12*	.32 (.01, .63)	.04	.01 (-.36, .38)
Time averaged	5.48***	.82 (.49, 1.15)	1.62	.24 (-.13, .62)
Age at first intoxication	-4.00***	-.60 (-.92, -.28)	-2.75**	-.41 (.03, .79)
Alcohol expectancies				
First assessed	1.32	.20 (-.12, .51)	.22	.03 (-.34, .41)
Time averaged	3.89***	.58 (.26, .90)	.75	.11 (-.26, .49)
Mean <i>d</i>	.45		.16	
Proximal measures				

Category and measure	AUD versus no AUD		Treatment versus no treatment	
	<i>t</i>	<i>d</i> (CI for <i>d</i>)	<i>t</i>	<i>d</i> (CI for <i>d</i>)
Social influence				
Peers' heavy drinking				
First assessed	4.18***	.62 (.30, .95)	-.48	-.07 (-.44, .30)
Time averaged	4.68***	.70 (.37, 1.02)	-.18	-.03 (-.40, .35)
Partners' heavy drinking				
First assessed	3.02**	.45 (.13, .77)	.27	.04 (-.33, .41)
Time averaged	2.87**	.43 (.11, .75)	.20	.03 (-.34, .40)
Peers' influence				
First assessed	1.79	.27 (-.12, .66)	1.06	.16 (-.29, .61)
Time averaged	3.13**	.47 (.13, .81)	1.58	.24 (-.16, .63)
Partners' influence				
First assessed	5.25***	.78 (.45, 1.12)	.77	.12 (-.26, .49)
Time averaged	6.20***	.93 (.59, 1.27)	1.01	.15 (-.23, .53)
Mean <i>d</i>	.58		.13	
Alcohol-related				
Heavy episodic drinking				
First assessed	1.03	.15 (-.16, .47)	.29	.04 (-.33, .42)
Time averaged	7.82***	1.17 (.83, 1.51)	1.93	.29 (-.09, .66)
Alcohol consumption				
First assessed	3.63***	.54 (.22, .86)	1.47	.22 (-.15, .59)
vTime averaged	5.70***	.85 (.52, 1.18)	1.98	.30 (-.08, .67)
Alcohol problems				
First assessed	1.13	.17 (-.15, .49)	.49	.07 (-.30, .45)
Time averaged	7.45***	1.11 (.78, 1.45)	3.26***	.49 (.11, .86)
Alcohol-related arrests	.18***	.62 (.30, .95)	3.77***	.56 (.18, .94)
Mean <i>d</i>	.66		.28	

AUD: alcohol use disorder; CI for *d* = 95% confidence interval for *d*. Positive values of *d* indicate that AUD men had a higher mean than the AUD-free men for the first contrast for each measures, and the AUD men with a treatment had a higher mean than the untreated AUD men in the second contrast for that measure. In the calculation of mean *ds*, the sign for the effect size of age at first intoxication was reversed because youthful drinking is a risk factor for alcohol misuse.