Published in final edited form as:
Drug Alcohol Depend. 2011 January 1; 113(1): 21–28. doi:10.1016/j.drugalcdep.2010.06.017.

Onset and Course of Alcoholism Over 25 Years in Middle Class Men

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Abstract

Background—Patterns of drinking and alcohol problems change with age. However, few studies use multiple data points and detailed history spanning early adulthood to middle age. This study reports such data from 373 men in the San Diego Prospective Study.

Methods—Data were generated at baseline (T1) at ~age 20, and through face-to-face follow-up interviews ~every 5 years in >90% of these eligible Caucasian and relatively higher educated men. Subjects were placed into 4 groups regarding their course: 62.5% with no alcohol use disorder (AUD); 17.2% with AUD onset <age 30 and a chronic course; 6.7% with onset ≥age 30 and no recovery; and 13.7% with AUD onset <age 30 and maintained remission for >5 years before the 25-year followup.

Results—On a univariate level, low level of response (LR) to alcohol, family history of AUDs, and higher Novelty Seeking at ~age 20 predicted AUDs with onset before age 30 (mean age ~25), but among these only LR predicted later onset (mean age 38) as well. Additional predictors of AUDs included demography (lower education), and greater involvement with alcohol, drugs, and nicotine prior to T1. Sustained remission from AUDs among alcoholics was predicted by lower T1 and T10 drinking frequencies, and being separated or divorced at T10, along with a trend for higher Reward Dependence.

Conclusion—These data indicate that information available in the late teens to early 20's can help predict the future onset and course of AUDs, and underscore the importance of longitudinal studies in substance use disorders.

Keywords

alcohol; sensitivity; alcoholism clinical course

1. Introduction

Heavy drinking usually emerges in the late teens, and the alcohol use disorders (AUDs) of abuse or dependence are often apparent by the mid twenties (Johnston et al., 2008; Schuckit et al., 1998). While important variations over time are likely to be seen for a person's

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drinking patterns and problems, and while many alcoholics have onsets later than the mid twenties (Lynskey et al., 2003; Whelan, 1995), at any age an AUD indicates a likelihood of future recurring alcohol problems and an elevated risk for morbidity and early death (Breslow and Graubard, 2008; Schuckit et al., 1998).

One predictable pattern of change over time for both persons with and without AUDs is that the quantities of intake are likely to decrease as individuals enter middle and later life (Bjork et al., 2008; Breslow and Smothers, 2004; Moos et al., 2004a, b; Moos et al., 2009; Zhang et al., 2008). Thus, it is important to follow groups of drinkers over time. This change can reflect the development of medical problems or the use of medications for which heavier drinking is proscribed, as well as physiological alterations that impact on the effects of alcohol with advancing age. The latter include higher proportions of body fat and associated lower levels of body water, with resulting increased blood alcohol concentrations (BACs) per drink, and enhanced brain reactions to most depressant drugs as people grow older (Kalant, 1998; Lucey et al., 1999). Therefore, with advancing age, even lower daily alcohol intake contributes to increasing risks for falls and hip fractures, cancer, coronary disease and early death (Cumming et al., 1997; Hanson and Li, 2003; Leipzig et al., 1999).

The age-related higher BACs per drink and elevated risks for accidents and medical problems have prompted recommendations that the upper limits of acceptable levels of drinking in older individuals (Grønbæk et al., 1998; Han et al., 2009) should be no more than two to three drinks per day or seven or more standard drinks per week, although some place the upper limit per week at 14 drinks (American Geriatric Society, 2003; Moos et al., 2009). However, half of drinkers age 60 and above exceed the guidelines for “safe” drinking (Kirchner et al., 2007; Merrick et al., 2008), ~25% of older drinkers consume ≥ 14 drinks per week (Zhang et al., 2008), and as many as 20% report five or more drinks per occasion five or more times per year (Moos et al., 2009). In the prior year ~12% of drinking men and women age ≥ 60 years reported consumption levels that placed them in modest or high risk drinking categories (Sacco et al., 2009), and the prevalence of substance use disorders, including AUDs, in older individuals has been estimated to be between 2% and 5%, with most of these diagnoses missed by clinicians (Lynskey et al., 2003).

Thus, unhealthful drinking and AUDs are important problems in middle-aged and older individuals, and it is clinically useful to understand the predictors and correlates of these conditions. Predictors of AUDs in any group are likely to include a family history (FH) of these disorders (Bennett et al., 1999; Cotton, 1979; Perreira and Sloan, 2001), a vulnerability that reflects a >40% heritability. Relevant genes contribute through intermediate characteristics such as disinhibition or sensation-seeking and a low level of response (LR) to alcohol (McGue, 1999; Schuckit, 2009; Sher, 1991). Regarding LR, the need for higher doses of alcohol to produce desired effects is associated with the subsequent consumption of higher numbers of drinks per occasion, and this phenotype characterizes children of alcoholics, has a >40% heritability, and predicts a higher future risk for AUDs (e.g., Heath et al., 1999; Schuckit and Smith, 2000; Trim et al., 2009; Volavka et al., 1996). Other factors related to a vulnerability toward heavier drinking and alcohol problems include demographic characteristics (e.g., male gender, lower education and a single or divorced marital status), prior smoking and illicit drug use, and previous higher alcohol quantities, frequencies, and alcohol problems (Dawson, 1995 in Moos, 2004a, b; Jacob et al., 2009; Karlamangla et al., 2006; Moos et al., 2004a, b; Perreira and Sloan, 2001; Schutte et al., 2003).

Once an AUD develops, factors similar to those predicting onset, but operating in the opposite direction, might predict remission. The Diagnostic and Statistical Manuals (DSM) of the American Psychiatric Association base the diagnosis of AUDs on repetitive problems,

and, therefore, full sustained remission is defined as the absence of any abuse or dependence criteria items for a year or more (APA, 2000). Reflecting data that for most people with alcohol dependence continued remission is likely to require abstinence from drinking, many of those in remission do not drink at all, or if so, consume alcohol rarely and in low amounts (Cox et al., 2004; Maisto et al., 2007; Mann et al., 2005). However, in some studies as many as 30% to 40% of those who fit DSM remission criteria have consumed at least some alcohol on occasion (Dawson et al., 2005; Vaillant et al., 2003). Higher probabilities of developing and maintaining remission are seen in the absence of pre-existing disinhibition and related personality disorders, demographic characteristics of higher education and income, evidence of stable relationships (including ongoing marriages and having children in the home), as well as less severe prior patterns of alcohol-related problems and lower levels of alcohol intake (Booth, 2004; Dawson et al., 2005, 2006; Jacob et al., 2009; Moos et al., 2004a, [2005]; Schutte et al., 2001, 2003, 2006; Weisner et al., 2003). Such longer-term remission from problems associated with AUDs is relatively common, with several studies estimating rates of 40% to 60%, especially when alcoholics are higher functioning and followed into their 50's and beyond (Grant, 1996; Ojesjo et al., 1981; Smith et al., 1999; Vaillant, 2003). While only about 25% of alcohol-dependent individuals might ever seek help, professional treatment for alcohol problems, and/or participating in self-help groups such as Alcoholics Anonymous (AA) improves outcome (Humphreys and Moos, 2007; LoCastro et al., 2009; Longabaugh et al., 2005; Moos and Moos, 2005; Moos et al., 2004a).

The variations in alcohol intake and problem patterns throughout the lifespan (Sartor et al., 2003; Schuckit et al., 1997) underscore the need for longitudinal studies that follow populations from early in their drinking careers on to middle age. However, relatively few investigators have used such long-term prospective approaches (Perreira and Sloan, 2001; Schulenberg and Maggs, 2008). While existing studies have added important information to the literature, there is a need for more prospectively gathered detailed data across as many time points and years as possible. Our group recently used Discrete Time Survival Analysis to evaluate a limited number of time invariant (i.e., baseline) predictors of the pattern (or hazard function) of onset of AUDs over 25 years, focusing on that single outcome in a modest sized sample (Trim et al., 2009). The current paper extends the prior results by evaluating 3 types of outcomes using an expanded set of baseline follow-up variables gathered ~every 5 years from face-to-face semi-structured interviews. The 3 goals of these analyses were to: 1) identify the characteristics at ~age 20 that predicted the onset of an AUD; 2) to evaluate baseline variables that predicted earlier versus later onsets of these conditions; and 3) for those men with an AUD onset before age 30, to identify characteristics that predicted remission. Based on a literature review and prior research, we hypothesized that robust predictors of the onset and course of AUDs would include an FH of AUDs, a low LR to alcohol, early life disinhibition (e.g., higher Novelty Seeking), less lifetime stability or achievement (e.g., in education, marital status, etc.), and higher prior intake of alcohol, nicotine, and illicit drugs.

2. Methods

The data reported here were generated from the San Diego Prospective Study (SDPS), a longitudinal investigation using informed consent procedures as approved by the University of California, San Diego (UCSD) Human Subjects Protections Committee. The subjects, or probands, were originally identified between 1978 to 1988 at baseline (Time 1 or T1) when they were 18-to-25-year-old Caucasian (including White Hispanic) men (Schuckit and Gold, 1988). Subjects with early onset of AUDs (e.g., in the teens) in the context of severe antisocial behaviors were excluded because these characteristics had already been identified as contributing to a wide range of substance problems and related disorders (Schuckit et al., 1970; Winokur et al., 1970), and the focus of the SDPS was to search for additional

characteristics linked specifically to AUDs. The probands first responded to a questionnaire mailed to students and non-academic staff at UCSD, and, if of interest to the protocol, were contacted and interviewed using a semi-structured instrument similar to the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA) interview (Bucholz et al., 1994; Hesselbrock et al., 1999; Schuckit and Gold, 1988; Schuckit and Smith, 1996). Subjects all had experience with alcohol, but none met criteria for alcohol abuse or dependence as described in the Third (DSM-III) and Third-Revised (DSM-IIIR) versions of the Diagnostic and Statistical Manual of the American Psychiatric Association (American Psychiatric Association 1980, 1987). Probands were selected as pairs of family history positive (FHP) men with alcohol-dependent biological fathers (alcohol-dependent mothers were excluded to avoid fetal alcohol effects) matched with family-negative (FHN) controls on demography, drinking patterns, and the history of ever having used an illicit drug or nicotine (Schuckit and Gold, 1988).

At baseline all probands participated in an evaluation of their LR to alcohol following consumption of 0.75 ml/kg of laboratory-grade ethanol which was administered over ~10 minutes as a 20% by volume solution mixed with a carbonated sugar-free and caffeine-free beverage. LR scores were generated through observation of changes from baseline for subjective feelings of intoxication, standing steadiness (body sway), and alcohol-related changes in cortisol, using z-scores to combine results across measures, with a focus on the time of usual peak BACs of ~0.06 gm/dl of ethanol at approximately 60 minutes after drinking. For LR, the subjective high score was, by definition, zero before subjects drank the beverage, and at 1 hour after drinking rose to a mean of 16.1 (SD=9.40) units on a 36-point scale, while the increase in body sway from before the drink to 1 hour later was 11.4 (18.6) cm of movement. T1 data were also gathered regarding other factors hypothesized to relate to the future AUD risk including demography (e.g., education, marital status, and frequency of practicing a religion from a score of none [zero] to two [frequent]), the recent six-month quantity and frequency of drinking, the lifetime occurrence of 9 alcohol-related problems (i.e., blackouts, work problems, losing friends, felt a need to cut down on drinking, tried to stop, ever sought advice about alcohol from a doctor, ever visited a hospital or clinic for a consequence of drinking [e.g., an accident], ever stayed overnight in a hospital related to drinking, or alcohol contributing to a breakup of a relationship), as well as the lifetime smoking history and pattern of use of illicit drugs. In addition, data were gathered on three personality characteristics projected to relate to the AUD risk as generated from the 100-item Tri-Dimensional Personality Questionnaire, focusing on the three major scales of Novelty Seeking, Reward Dependence, and Harm Avoidance (Cloninger et al., 1987). The internal consistency of the scales is estimated at >.7, with good structural and construct validities (Chen et al., 2002).

All 453 original probands were located ~10 years after baseline at Time 10 (T10), by which time 4 men had died, and 99.3% of the remainder agreed to participate in SSAGA-like interviews. Data were gathered to update demography and FH status, smoking and drug histories, as well as the pattern during the follow-up interval for the quantity and frequency of alcohol intake, and the occurrence of 11 non-diagnostic alcohol problems (i.e., the 9 questions asked at T1 plus drinking before noon or feelings of lost control of drinking), along with the 11 DSM-IIIR criteria items for alcohol abuse and dependence. At followup, separate interviews were used to gather information on the proband from an additional informant (usually the spouse), with any disagreements between information from spouse and proband coded as the worst-case scenario (e.g., the largest number of problems from either source) (Bird et al., 1992). Subsequent followups gathered similar data at 15 years (T15 with 98% participation), 20 years (T20 where 96% participated), and the current ongoing 25-year (T25) followup with a projected 94% completion rate for probands regarding all follow-up epochs.

For the DSM-IIIR criteria for alcohol abuse and dependence, the age of onset was recorded as the actual age when 3 or more criteria had developed and adversely impacted on functioning. The subjects were placed into mutually exclusive categories based on an AUD diagnosis, and, if they had met criteria, the age at which the AUD developed or remitted. For most analyses the outcome of interest was the combination of alcohol abuse and/or dependence, reflecting data that these two diagnoses represent a unidimensional factor in most analyses, the proposed movement in DSM-V to a single alcohol use disorder encompassing items from both abuse and dependence, and to maximize statistical power (Borges et al., 2010; Bucholz et al., in press). When appropriate, dependence and abuse according to DSM III-R were also evaluated separately. Recovery from an AUD was recorded if the individual evidenced no items from the abuse and dependence criteria over at least the final 5-year follow-up period. Based on the goals of these analyses stated in the Introduction, 4 groups were created, including Group 1 who evidenced no AUD over the 25 years subsequent to baseline; Group 2 with an onset of an AUD by the first followup (at T10 or before age 30) and maintenance of an alcohol diagnosis throughout all subsequent followups; Group 3 who developed an AUD after the 10-year interview; and Group 4 with an AUD onset before age 30 along with persisting remission from diagnostic criteria for at least the T25 follow-up period. None of the later-onset alcoholics in Group 3 had recovered by the T25 interview.

The 4 groups were subsequently evaluated for characteristics projected to relate to the risk for AUDs (e.g., LR, FH, demography, and substance use histories); for Groups 2 through 4, the histories of receiving help with their alcohol problems (e.g., treatment from a clinician or participation in Alcoholics Anonymous [AA]); as well as the pattern of relevant characteristics regarding the prediction of later onset AUDs and the prediction of remission. Chi square (for categorical data) and ANOVA (for continuous variables) were used to determine the overall significance across the 4 groups. If significant, planned comparisons based on the goals stated in the Introduction were carried out. These included an evaluation of all 373 men, comparing the combined Groups 2 through 4 who evidenced an AUD versus Group 1 with no AUD diagnosis during the 25 years; a comparison of Group 3 men who had an onset after T10 versus Group 1 men with no AUD; within the 140 alcoholics, comparisons between those who developed their AUD after the T10 followup versus those with an earlier onset (Group 3 versus Groups 2 and 4); and a comparison within the 115 alcoholics who developed their AUD by T10 of those who evidenced remission (Group 4) versus those with a chronic course (Group 2). Logistic regressions with backward elimination were then used to evaluate all relevant significant items identified in each planned comparison.

3. Results

The 373 subjects included in these analyses were San Diego Prospective Study probands who participated in the ongoing 25-year followup to date and who had complete data for all of the required analyses. They represent an estimated 94% of those who had been scheduled for interview. These men were an average age of 46.5 (3.29) years at their 25-year followup, all were Caucasian (including White Hispanic), and 53.1% had an alcohol-dependent father. Over the 25 years, the rate of AUDs in the FHP probands was 44.9% and in FHNs was 29.1% ($\chi 2 = 9.90$, $p < .002$). At baseline 18.5% were married, 26.5% reported having no religious preference, while the values for Novelty Seeking were a mean of 15.6 (4.78) with a range of 3 to 32 and for Reward Dependence were 17.7 (4.41) with a range of 6 to 28.

Among the 373 probands, 140 men (37.5%) met criteria for alcohol abuse or dependence during the 25-year followup, with an average age of onset of an AUD of 27.2 (6.60) years. The proportion of alcoholics (Groups 2 through 4) who ever met criteria for alcohol

dependence was 71.4% overall, with the remainder only meeting criteria for alcohol abuse. Over the 25 years of the study, 10.7% of the 140 alcoholics had ever received treatment for their AUD from a healthcare provider, and an additional 12.1% had participated in a self-help group such as Alcoholics Anonymous, but had never received formal treatment. Therefore, a total of 22.8% of the 140 had received either or both of these sources of help.

As shown in Table 1, the 4 groups included 233 drinkers (62.5%) in Group 1 who had never developed an AUD during the relevant follow-up interval; 64 men in Group 2 (17.2% of the total and 45.7% of the 140 probands with an AUD) who developed their disorder during the first 10-year followup and maintained an AUD for all subsequent evaluations; 25 individuals in Group 3 (6.7% of the total and 17.9% of the alcoholics) who had an AUD onset after the T10 followup and a subsequent chronic course; and 51 men in Group 4 (13.7% of the total sample and 36.4% of those with an AUD) who were alcoholic by T10 and who subsequently recovered from their AUD by T20. Across Groups 2, 3, and 4, the ages of onset of the AUD were 24.5 (3.33), 37.9 (6.83), and 25.2 (3.80) years, respectively ($F = 94.8$, $p < .001$). While the Tukey HSD post-hoc analysis (the approach most appropriate across the three groups) for ages of onset for Groups 2 versus 4 was not significant ($p = .67$), Groups 2 and 4 were each different from Group 3 ($p < .001$). For Group 3, 8 men (32.0% of Group 3 and 5.7% of the 140 alcoholics) developed their disorder at age 40 or above.

Table 1 presents the distribution across the 4 groups for T1 and T10 potential predictors of the onset and course of AUDs. At the bottom of the table we also report the percent who met criteria for alcohol dependence in each AUD group, along with a summary of treatment received for AUDs and for 12-step program participation for relevant groups through T25. As all variables in Table 1 were significantly different across the 4 groups, we carried out a series of planned comparisons, with results presented in the four last columns in Table 1. First, men in the combined Groups 2 through 4 who had developed an AUD were compared to the non-diagnostic Group 1. Almost all baseline variables were significantly different across the two broad diagnostic categories, including an association for AUDs with lower LR values, an FHP designation, higher Novelty Seeking, and higher T1 alcohol and substance use patterns. The only baseline variable that did not significantly differentiate between Groups 1 versus Groups 2–4 was T1 Reward Dependence. While the comparisons across non-alcoholic and alcoholic groups for T10 variables reflected the fact that all but 25 of the 140 alcoholics had manifested their diagnosis by the 10-year follow-up, the lower T10 figures for alcoholics for ever having been married and for practicing a religion, and the higher percent who smoked and used drugs during the first decade of follow up may be worth noting.

Additional information regarding the prediction of AUDs can be gathered by comparing the later onset subjects in Group 3 (who did not have an AUD at T10) with men in Group 1. Here, compared to those in Group 1 with no diagnosis, Group 3 men with later onset AUDs had significantly lower T1 LR, fewer years of schooling, and higher usual T1 drinking quantities. However, at T1 Groups 1 and 3 men were similar on FH, the percent with non-diagnostic alcohol problems, smoking histories, Novelty Seeking, and Reward Dependence. At T10, a time frame before their AUD onset, Group 3 men had higher drinking frequencies and quantities and demonstrated a trend for a higher proportion with any T10 alcohol problems ($p = .07$).

The third planned evaluation of results from Table 1 involved comparing subjects with an AUD onset after T10 (Group 3) to the alcoholics who evidenced onsets earlier than the T10 followup (combined Groups 2 and 4). As shown in the second to last column in Table 1, at T1 the men who would experience a later onset of their AUD were less likely than other alcoholics to have reported any of the 9 non-diagnostic alcohol problems and to smoke, and

had lower scores than other alcoholics for Novelty Seeking. Once again, while T10 alcohol items reflect the differences between groups in their AUD status at the 10-year followup, the higher proportion of ever married men by T10 in Group 3 may be worth noting, as might their lower proportion of those who used an illicit drug between T1 and T10. Later-onset subjects were also more likely than those with AUDs by T10 to have developed only alcohol abuse, not dependence. Interestingly, the frequencies of drinking were similar at T10 across earlier versus later onset alcoholics.

The final planned comparison evaluates predictors of remission (Group 4) versus a chronic course (Group 2) for men with an AUD onset by T10. At T1, compared to men who would become alcoholics with a chronic course, those with subsequent remission had lower drinking frequencies and a trend for higher scores on Reward Dependence ($p = .09$). At T10, a time when both groups were already demonstrating their AUD, those who subsequently remitted were more likely to be divorced or separated and reported lower usual and maximum drinking frequencies, although drinking quantities were more similar. While not shown in the table, the direction of the relationship to remission was the same for those divorced as for those separated at T10.

For each of the planned comparisons, all items that differentiated across the relevant groups were then entered into regression analyses predicting the relevant outcomes, as shown in Table 2. For the predictors of developing an AUD (Groups 2, 3, and 4) among all 373 men, the regression revealed that 7 of the 10 baseline (T1) items predicting an AUD contributed significantly to the equation. Note only items from Table 1 that were significant in any comparison are listed in Table 2, with “NA” indicating when a variable was not relevant to a specific column of that table, either because it was not significant for that outcome in Table 1 or was not appropriate to test in the regression for that outcome. An example of the latter is that T10 alcohol frequency could not be used to predict an AUD by T10, and is “NA” for data column 1, but was relevant for the evaluation of predictors of remission among those with an AUD at T10 in data column 4. The T1 items that contributed to an estimated 41% of the variance for development of an AUD (the pseudo R^2), included all 3 key variables of lower LR, an FHP status, and higher Novelty Seeking scores. Also significant were higher T1 alcohol use and problem histories, and lower education.

Outcomes for the 140 alcoholics (Groups 2–4) reflected a combination of alcohol dependence (71% of Groups 2 – 4) and abuse (29%). To better understand the implications of this approach, the major analyses for Groups 1 versus 2 through 4 were repeated to evaluate the two diagnoses separately. A regression predicting dependence revealed significant contributions for most T1 items significant in the first data column of Table 2, including a lower LR (OR = 0.64); the FHP status (OR = 2.14); fewer years of education (OR = 0.71); higher T1 alcohol frequency (OR = 1.08), quantity (OR = 1.46), and problems (OR = 2.59); as well as higher Novelty Seeking (OR = 1.07), with $R^2 = .50$. When the regression was repeated for abuse outcomes, the results revealed significant contributions to the prediction of abuse for a lower LR (OR = 0.50); fewer years of education (OR = 0.78); and higher T1 alcohol frequencies (OR = 1.06) and quantities (OR = 1.06), with $R^2 = .65$.

The second regression in Table 2 focused on the prediction of later onset AUDs (Group 3) for men in Groups 1 and 3. Here, while significant on its own in Table 1, LR did not add to the equation that generated an R^2 of .57. Of the significant items in Table 1, only T1 education and T10 drinking frequencies were significant in the regression.

The regression analysis in the third data column of Table 2 describes the performance of the 3 significant T1 items from Table 1 regarding the prediction of later onset AUDs among alcoholics. Two T1 variables contributed significantly to the regression predicting later

onset among alcoholics, including the absence of non-diagnostic alcohol problems at T1 and lower scores for Novelty Seeking, combining for a pseudo R^2 of .55. Adding the presence or absence of a dependence (versus abuse only) diagnosis to the regression did not contribute significantly to the regression.

The data in column 4 of Table 2 evaluates predictors of recovery for these men who demonstrated an AUD by T30. The regression demonstrates that the predictors of remission included lower drinking frequencies at both T1 and T10, higher Reward Dependence, and a T10 marital status of having been divorced or separated (R^2 = .30). To evaluate the impact of abuse versus dependence diagnosis, the regressions were re-evaluated after adding the abuse/dependence designation, but the results did not change from those listed in Table 2. To test the possible impact of having received alcohol-related treatment or participating in AA for this recovery-related outcome, these items were also added to the regression, but did not contribute significantly to the prediction of outcome.

4. Discussion

This paper describes the predictors of the development of AUDs and aspects of their course over 25 years for 373 men who have been followed through personal interviews about every 5 years since age 20. The SDPS is one of the few prospective evaluations of a non-clinical sample enriched for the alcoholism risk through selection of half of the subjects as FHPs and describing individuals from blue- and white-collar more high functioning families. With a follow-up rate of >90% across all time points, as well as data gathered through face-to-face interviews with both the probands and a corroborating resource person, information was generated regarding life elements originally hypothesized to be related to the development and course of alcohol abuse or dependence.

In the SDPS, 37% of the probands developed an AUD, with significantly higher rates among the FHPs and those with low LRs. Despite the high levels of education and income of these probands and the exclusion of men with AUD onset in the teens, the current outcomes were consistent with what one might expect from other FHP/FHN samples from the general population. Also similar to the literature were the relationships of the AUD risk with demographic characteristics (e.g., lower education), histories of higher prior alcohol quantities, frequencies and problems, as well as higher rates of smoking and illicit drug use at baseline (Dawson, 1995; Karlamangla et al., 2006; Shutte et al., 2003). Thus, our decision to exclude very early onset alcoholics in order to focus on risk factors more uniquely related to AUDs and not substance use disorders in general still produced a population that, despite their higher education and income, is likely to mirror the AUD risk factors for the general population in the U.S.

Regarding the first of 3 major AUD risk factors discussed in the Introduction (LR, FH, and Novelty Seeking) those who developed alcoholism had LRs at about age 20 that were twice as low as those for the non-AUD group. On a univariate level, compared to the Group 1 non-alcoholics, LR values were significantly lower both for men who developed AUDs at a mean age of ~25 (Group 2) and those with an average onset of age 38 (Group 3), with the latter AUD onset occurring >15 years after original testing. In addition, a low LR appeared equally robust in predicting alcohol abuse and dependence. The performance of LR as a predictor of later onset AUDs is consistent with an earlier evaluation of SDPS probands that revealed that, while an increasing sensitivity to alcohol did occur in many subjects with advancing age, this increase might apply mostly to lighter drinking men and those more sensitive to alcohol at age 20 (Schuckit and Smith, 2004). Those with an earlier low LR were more likely to maintain their need for more drinks for effects into middle adulthood. While higher Novelty Seeking also predicted an onset of alcohol abuse or dependence by

age 30, this variable did not relate to an AUD onset after age 30. FHP status was also only related to the development of AUDs prior to age 30.

Another goal of these analyses was to evaluate potential predictors of later onset AUDs. In some studies about one-third of alcoholics developed their disorder later in life, with as many as 10% of those with AUDs only meeting criteria for their condition at age 40 or older (Lynskey et al., 2003; Schuckit, 1977; Wehlen, 1995). In the current study where subjects with onsets of AUDs in the teens had been excluded, 6.7% of the 373 men had an AUD onset after the age of 30 (at a mean age of ~38 years), a figure that translated to 17.9% of the 140 alcoholics. Univariate T1 predictors of a later onset (Group 3 versus Group 1) included lower T1 LR and education and higher drinking quantities, along with higher T10 drinking patterns and problems, with T1 education and T10 drinking frequency contributing to a regression with a pseudo R^2 of .57. Later onset among alcoholics (Group 3 versus Groups 2 and 4) on a univariate level was predicted by lower T1 alcohol problems, smoking, and Novelty Seeking, with the T1 problems and Novelty Seeking contributing to the regression where an estimated 55% of the variance of outcome was explained.

Overall, remission among the earlier onset alcoholics was predicted by lower T1 and T10 drinking frequencies, a trend for higher Reward Dependence, along with T10 marital separation or divorce. Almost all of these items significantly contributed to a regression analysis that explained ~30% of the variance. While it is difficult to predict remission a decade or more later (Schuckit et al., 1986; Zucker et al., 2006), the role of lower earlier drinking frequencies is consistent with some prior reports regarding the importance of this variable (e.g., Gruenewald et al., 2009). Higher Reward Dependence, included in these analyses because of the trend for this characteristic to be associated with a better outcome in Table 1, relates to greater levels of sensitivity to the need for offering help, greater understanding of people's problems, and efforts to strive for doing well in endeavors. Measured prior to the onset of alcoholism, the performance of this variable in Table 2 underscores the possibility that long-standing personality attributes might not only predict the development of AUDs but might also help indicate a higher probability of good outcomes.

While being married by T10 (~age 30) was associated with the absence of an AUD diagnosis overall (Group 1) and with a later onset of alcohol abuse or dependence (Group 3) among alcoholics, being separated or divorced at T10 related to entering recovery among men with earlier AUDs (Group 4). The salience of the latter finding awaits replication, as only a relatively small number of men had this event, but if robust, the result might reflect a role of the breakup in highlighting the importance of working toward recovery, or decreases in stressors related to ending a problematic relationship.

The focus on the absence of AUD criterion items in defining recovery and inclusion of individuals as being in recovery despite the continuation of some drinking is consistent with the guidelines for remission outlined in DSM-IV where, just as the diagnostic criteria for AUDs rest with patterns of problems, so do the related remission criteria (American Psychiatric Association, 2000). Among the 51 men in Group 4, 24 (47%) were either totally abstinent during the most recent 5 years of follow-up or were reported by themselves and their spouses as consuming one or fewer drinks per occasion with no evidence of heavy drinking periods. The overall remission rate of 44% for the 115 men with an AUD at T10 and, thus, sufficient time of follow up, is also consistent with some rates of outcome likely to be seen in non-clinical samples of alcoholics in the general population (Ojesjo et al., 1981; Smith et al., 1999; Vaillant, 2003).

Among the 51 recovered men (Group 4), almost 22% had received help for their alcohol problems, a level that was not different from those in Group 2 with a chronic course. This lack of difference for treatment exposure was observed regardless of whether Group 4 men were abstinent or drank but had demonstrated none of the 11 DSM-IIIR and DSM-V alcohol abuse and dependence problems. However, the current study was not structured to evaluate the impact of treatment. The sample size, the low proportion of individuals receiving help from professionals or AA, the absence of detail gathered regarding the intensity of treatment or AA, along with the use of 5-year follow-up periods, make it difficult to establish cause-and-effect relationships between receiving help and recovery. Many alcohol-dependent people achieve remission from drinking or alcohol problems in the absence of treatment and, thus, a large proportion of the variance of outcome may operate independently of such interventions (Cunningham et al., 2000; Dawson et al., 2006). Nonetheless, there is evidence from other studies that formal treatment and self-help group participation are likely to be associated with better outcomes overall (LoCastro et al., 2009; Mueller et al., 2007).

It is also important to note that these analyses combined abuse and dependence into one overall AUD outcome. This was done because of the unidimensional nature of the abuse and dependence items in the DSM, as well as the reported movement toward a single list of criterion items for the substance use disorders in DSM-V (Borges et al., 2010; Bucholz et al., in press). However, the predictors of AUDs, later onset, and future remission operated relatively similarly for those with abuse or dependence. Thus, while the number of subjects with abuse was too small to generate robust separate analyses, the results reported here may be relevant to both the current DSM-IV and DSM-V approaches.

The data presented here must be considered in light of the methods used. The caveats include the relatively small number of alcoholics overall, and the relatively large number of potential predictors considered, although the number of items used in regressions was more modest. Limitations in statistical power open the possibility that additional important predictors of AUD development and clinical course are likely to be found in larger samples, and the number of predictors underscores the possibility of Type 1 errors. Also, the population used here was Caucasian, male, and well educated, making it unclear whether the same predictors would operate in more racially-diverse populations, women, or individuals with earlier onsets of their AUDs associated with more antisocial behaviors earlier in life. In addition, because the mean age of these probands at followup was ~47 years, additional cases of AUDs and changes in the relationships of key variables to outcomes may be observed as these men enter their sixth decade of life (Hanson and Li, 2003; Lucey et al., 1992.

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Table 1

Comparisons of T1 and T10 Variables across 4 Alcohol Diagnosis Groups with Planned Comparisons for 373 Men*

	Variable mean (sd) or percent for each of 4 groups				Planned comparisons for groups with significant overall differences [F-test/ χ^2]			
Variables	**Group: No AUD n = 233**	**Group 2: Chronic n = 64**	**Group 3: Later Onset n = 25**	**Group 4: Recovery n = 51**	**Developed AUD: Group 2,3,4 vs. 1**	**Later Onset vs. No AUD: Group 3 vs. 1**	**Later vs. Earlier Onset: Group 3 vs. 2,4**	**Remission in Early Onset: Group 4 vs. 2**
LR	−.33 (0.90)	−.72 (0.77)	−.77 (0.77)	−80 (0.64)	23.4^c	5.73^a		
FHP √	46.8	68.8	52.0	62.7	9.9^b			
T1 Variables								
Age (yrs)	22.8 (1.75)	22.1 (1.87)	22.5 (2.12)	22.2 (1.72)	9.4^b			
Education (yrs)	16.2 (1.65)	15.5 (1.14)	15.5 (1.58)	15.4 (1.22)	19.6^c	4.0^a		
Usual drink freq	7.6 (6.05)	12.4 (6.23)	9.6 (7.11)	8.8 (5.11)	20.7^c			11.2^c
Usual drink quant	2.4 (1.26)	3.6 (1.57)	3.2 (1.56)	3.5 (1.35)	56.8^c	9.6^b		
Any alc problem √	27.0	67.2	32.0	54.9	32.0^c		7.4^b	
Use illicit drug √	54.5	76.6	72.0	72.5	14.5^c			
Smoke √	15.9	35.9	16.0	37.3	14.6^c		3.9^a	
Novelty Seeking	14.8 (4.47)	17.3 (5.33)	14.9 (4.20)	17.3 (4.87)	16.5^c		4.6^a	
Reward Dependence	17.4 (4.40)	17.9 (4.11)	16.9 (4.29)	19.3 (4.65)				2.9^d
T10 Variables								
Ever married √	73.0	46.9	76.0	45.1	17.8^c		7.4^a	
Sep/divorced √	3.9	1.6	0.0	15.7				7.8^b
Practice religion	0.69 (0.84)	0.41 (0.61)	0.52 (0.71)	0.39 (0.57)	11.0^c			
Usual drink freq	10.2 (7.86)	19.2 (7.37)	18.5 (8.31)	14.2 (6.41)	73.5^c	25.0^c		14.4^c
Usual drink quant	2.4 (1.23)	4.1 (1.68)	3.0 (1.21)	4.1 (1.71)	94.0^c	4.0^a	10.4^a	
Max drink freq	15.3 (8.75)	24.7 (6.62)	22.8 (7.63)	22.2 (6.57)	90.3^c	17.2^c		4.0^a
Max drink quant	7.6 (4.04)	14.1 (5.04)	10.7 (4.66)	13.9 (5.48)	146.4^c	13.2^c	8.7^b	
Any alc problem √	48.5	96.9	68.0	96.1	70.5^c	3.4^d		

	Variable mean (sd) or percent for each of 4 groups				Planned comparisons for groups with significant overall differences [F-test/ χ^2]			
Variables	**Group: No AUD n = 233**	**Group 2: Chronic n = 64**	**Group 3: Later Onset n = 25**	**Group 4: Recovery n = 51**	**Developed AUD: Group 2,3,4 vs. 1**	**Later Onset vs. No AUD: Group 3 vs. 1**	**Later vs. Earlier Onset: Group 3 vs. 2,4**	**Remission in Early Onset: Group 4 vs. 2**
AA or treatment √	0.0	9.4	0.0	15.7	24.2[c]			
Use illicit drug √	56.7	92.2	72.0	88.2	37.4[c]		6.4[a]	
Smoke √	17.2	39.1	24.0	45.1	21.3[c]			
25 Yr Summary Variable								
Any AA/Treatment √	0.0	26.6	16.0	21.6	NA	NA	NS	NS
Alc Dependent AUD √	0.0	82.8	52.0	66.7	NA	NA	5.6[c]	4.3[c]

[*] T1 = baseline and T10 = 10-year follow ups; AUD = alcohol use disorder; Chronic (Group 2) had an onset before T10 and continued to meet alcohol abuse or dependence criteria through the 25 yr follow up (T25); Later Onset (Group 3) = met AUD criteria after T10; Recovery (Group 4) = ≥ 5 years prior to T25 when no AUD criteria were met; LR = level of response to alcohol; FHP = Family history positive for parental alcoholism; freq and quant = frequency and quantity of drinking in the prior 6 mo at T1 or 10 years at T10; max = maximum freq and quant during the 10 yrs of follow up; alc = alcohol; AA or treatment = alcoholics anonymous attended and/or alcohol treatment from a health care professional; sep/divorced = divorced or separated during the intervals; NS = tested in that specific comparison but was nonsignificant; NA = was not tested because was inappropriate to evaluate to that specific comparison.

√ Data are in percent. Remaining items are mean (sd).

[a] $p \leq .05$

[b] $p \leq .01$

[c] $p \leq .01$

[d] $p < .10$

Table 2

Significant Items in Regressions Predicting Each of Four Outcomes: Wald χ^2 (Odds Ratio)*

Variables	Developed AUD: Group 2,3,4 vs. 1 N=373 Wald χ^2 (Odds Ratio)	Later Onset vs. No AUD: Group 3 vs. 1 N=258 Wald χ^2 (Odds Ratio)	Later vs. Earlier Onset: Group 3 vs. 2,4 N=140 Wald χ^2 (Odds Ratio)	Remission in Early Onset: Group 4 vs. 2 N=115 Wald χ^2 (Odds Ratio)
LR	10.37^c (0.60)	NS	NA	NA
FHP √	6.36b (1.85)	NA	NA	NA
T1 Variables				
Age (yrs)	NS	NA	NA	NA
Education (yrs)	58.1^c (0.74)	59.59^c (0.78)	NA	NA
Usual drink freq	10.88^c (1.07)	NA	NA	4.32^a (0.93)
Usual drink quant	17.67^c (1.46)	NS	NA	NA
Any alc problem √	8.83^b (2.30)	NA	6.46^a (0.31)	NA
Use illicit drug √	NS	NA	NA	NA
Smoke √	NS	NA	NS	NA
Novelty Seeking	4.70^a (1.06)	NA	13.97^c (0.94)	NA
Reward Dependence	NA	NA	NA	10.19^c (1.10)
T10 Variables				
Sep/divorced √	NA	NA	NA	5.18^a (15.04)
Usual drink freq	NA	20.55^c (1.13)	NA	6.43^c (0.93)
Usual drink quant	NA	NS	NA	NA
Any alc problem	NA	NS	NA	NA
Pseudo R^2	.41	.57	.55	.30

*See Table 1 for term definitions. A specific reminder regarding definitions is that NS = tested in that specific regression but was nonsignificant; NA = was not tested because was not significant in Table 1 or was inappropriate to evaluate for that specific outcome.

√Data are in percent. Remaining items are mean (sd).

^{a}p ≤ .05

^{b}p ≤ .01

^{c}p ≤ .001