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Similarities and Differences of Longitudinal Phenotypes Across Alternate Indices of Alcohol Involvement: A Methodologic Comparison of Trajectory Approaches

Kristina M. Jackson and **Kenneth J. Sher**
Department of Psychological Sciences, University of Missouri—Columbia

Abstract

Extant developmental research distinguishing young adults who moderate versus persist in alcohol consumption has not consistently evaluated the domain of alcohol involvement being modeled, making it difficult to compare findings across studies. In the present study, the authors characterized the developmental course of 5 indices of alcohol involvement using a prospective (6-wave) sample of 377 young adults (Year 1 age = 18.52 years; 55% female; 51% with family history of alcoholism) over 11 years. Growth mixture models were applied to each measure. Despite similarity in trajectory shape, predicted prevalences varied, and the consistency of trajectory classifications across alternate indices revealed low agreement. Correlates of drinking course, however, were somewhat robust across alcohol index. The finding that trajectories are conditional on the specific indices used suggests that it may be hazardous to generalize across alternate indices of alcohol involvement.

Keywords

alcohol; drinking; trajectory; course; developmental

Early young adulthood (ages 18–25 years) represents the period of peak prevalence for alcohol use (Gallup Organization, 1987) and alcohol use disorders (AUDs) (Grant, 1997), and the transition from high school to college is a time of risk for heavy drinking (Baer, Kivlahan, & Marlatt, 1995; Johnston, O'Malley, & Bachman, 2002a, 2002b). As young adults age beyond college, however, many exhibit a tendency to moderate heavy alcohol involvement (Donovan, Jessor, & Jessor, 1983; Perkins, 1999). In the population, alcohol consumption (particularly heavy drinking), problem drinking, and AUDs tend to peak in the early 20s and then show a decline over the third decade of life (Dawson, Grant, Stinson, & Chou, 2004; Fillmore, 1988; Jessor, Donovan, & Costa, 1991; Johnston et al., 2002a, 2002b; Muthén & Muthén, 2000). Against this normative decrease, many young adults continue to drink heavily (Windle, 1988), continue to have drinking problems (Fillmore, 1988; Tubman, Vicary, von Eye, & Lerner, 1990; Windle, 1988), and manifest AUDs (Zucker, Fitzgerald, & Moses, 1995). Thus, a central question of research into the etiology of alcoholism concerns factors that distinguish young adults who moderate their alcohol consumption from those who persist in heavy drinking. Yet, until recently, relatively little research has addressed variability in typical courses of alcohol involvement during this rapidly changing period of life of early young adulthood (recently termed "emerging adulthood"; Arnett, 2000). In the present study, we addressed three central research questions: (a) whether there exist prototypical courses of

Correspondence concerning this article should be addressed to Kristina M. Jackson, who is now at the Center for Alcohol and Addiction Studies, Brown University, Box G-BH, Providence, RI 02912. kristina_jackson@brown.edu.

alcohol involvement, (b) the extent to which these courses are consistent across operationalization of alcohol involvement, and (c) whether these different courses vary in their etiological correlates.

Recent research has begun to chart the longitudinal course of alcohol involvement during adolescence and young adulthood. Theoretical (Zucker, 1987, 1994; Zucker et al., 1995) and empirical (e.g., Bennett, McCrady, Johnson, & Pandina, 1999; Chassin, Pitts, & Prost, 2002; Colder, Campbell, Ruel, Richardson, & Flay, 2002; Schulenberg, O'Malley, Bachman, Wadsworth, & Johnston, 1996; Schulenberg, Wadsworth, O'Malley, Bachman, & Johnston, 1996; Tucker, Orlando, & Ellickson, 2003) work has suggested that several prototypical courses can be identified. Although results with respect to the specific characterization of course and associated prevalences vary somewhat from study to study, there is consensus in findings revealing the presence of a nonuser/stable low-use course, a chronic or high-use course, a "developmentally limited" course (maturing out of drinking), and some evidence for a later onset or increasing course.

Recent research on the developmental course of alcohol involvement has varied considerably with respect to the specific variables and domains assessed and includes indices of alcohol involvement such as heavy/binge drinking (e.g., Chassin et al., 2002; Schulenberg, O'Malley et al. 1996; Schulenberg, Wadsworth, et al., 1996; Tucker et al., 2003), problem drinking (e.g., Bennett et al., 1999), and quantity–frequency (e.g., Colder et al., 2002). Assuming moderate associations among various indices of alcohol involvement, it is reasonable to hypothesize that trajectories based on one type of measure may be similar to those based on others (e.g., quantity–frequency vs. binge drinking vs. problems). However, the lack of studies comparing the correspondence of trajectories based on alternate definitions of alcohol involvement makes it difficult to compare findings across studies. To the extent that researchers have begun to reify these trajectories as summary measures of drinking course, it is critical to determine the extent to which such correspondence exists. Lack of correspondence can seriously limit the types of generalizations that can be made from one investigation to another if study particulars are not adequately considered.

Correlates of Drinking Course

In the process of investigating the construct validity of drinking trajectories, several investigations have attempted to identify etiological correlates of drinking course, often looking at the possible influence of demographic variables (e.g., sex, race) as well as measures of genetic disposition to alcoholism, personality/temperament, life events, and motivations for substance use. The findings with respect to several of these covariates are summarized below.

Sex

Men generally consume more alcohol and experience more alcohol-related problems than do women (Ager et al., 1996; Baer et al., 1995; Harford & Grant, 1994; Wilsnack & Wilsnack, 1997). In terms of course of alcohol involvement, findings are inconsistent, with some studies finding gender differences (e.g., Baer et al., 1995; Bennett et al., 1999; Chassin et al., 2002; Curran, Muthén, & Harford, 1998; Hill, White, Chung, Hawkins, & Catalano, 2000; Schulenberg, Wadsworth, et al., 1996; Tucker et al., 2003) and others failing to find differences (Bates & Labouvie, 1997; Duncan et al., 1997; Pape & Hammer, 1996; Wills, McNamara, Vaccaro, & Hirky, 1996). Generally, when a difference is observed, being male is associated with increased or persistently high alcohol involvement.

Family History of Alcoholism

Positive family history of alcoholism is associated with increased risk for AUDs (Sher, 1991; West & Prinz, 1987; Windle & Searles, 1990). Although Chassin and colleagues found that paternal alcoholism was associated with positive growth in alcohol use (Chassin, Curran, Hussong, & Colder, 1996) and with likelihood of belonging to an early onset (increasing) drinking group (Chassin et al., 2002), other researchers have failed to detect a family history effect on drinking course (Baer et al., 1995; Bates & Labouvie, 1997; Bennett et al., 1999).

In a similar vein, higher parental substance use in general has been shown to be associated with a more maladaptive (persistently heavy or increasing use) course of alcohol involvement (White, Johnson, & Buyske, 2000; Wills et al., 1996).

Behavioral Undercontrol

Indices of behavioral undercontrol, such as conduct disorder and delinquency, predict alcohol use and alcohol-related problems both cross-sectionally (Moss & Kirisci, 1995; Neighbors, Kempton, & Forehand, 1992) and prospectively (Brown, Gleghorn, Schuckit, Myers, & Mott, 1996; Johnson, Arria, Borges, Ialongo, & Anthony, 1995). In addition, behavioral undercontrol is a robust predictor of course of alcohol involvement among adolescents and young adults (Bates & Labouvie, 1997; Bennett et al., 1999; Chassin et al., 2002; Colder et al., 2002; Hill et al., 2000; Stice, Myers, & Brown, 1998; Tucker et al., 2003; White, Xie, Thompson, Loeber, & Stouthamer-Loeber, 2001; Wills et al., 1996). This has been shown to be true for temperamental constructs (i.e., harm avoidance and disinhibition) as well as antisocial, externalizing, risk-taking, and delinquent behavior. In general, findings reveal that those who began drinking early and had persistently high rates of drinking, in particular, exhibited greater behavioral undercontrol than did other groups. In the present study, we examined a temperamental index of behavioral undercontrol (novelty seeking) as well as a behavioral one (conduct disorder).

Negative Affectivity

Depression and anxiety disorders are associated with alcohol use in adolescents (Galaif, Chou, Sussman, & Dent, 1998; Hussong, Hicks, Levy, & Curran, 2001) and young adults (Kushner & Sher, 1993). Although indices of negative affectivity, such as depression, have shown associations with drinking course, particularly for those courses represented by elevated drinking (Chassin et al., 2002) and rapid escalation (Colder et al., 2002), findings have been inconsistent. Stice, Myers, and Brown (1998) showed that negative affectivity and internalizing predicted abstinence (vs. escalation to moderate alcohol use) but failed to show that either construct predicted escalation to heavy drinking. Bates and Labouvie (1997) found that emotional outbursts and self-derogation predicted continued heavy use versus continued low use. In addition, both societal- and individual-level research indicate that suicidal ideation, attempted suicide, and completed suicide are associated with alcohol use and abuse (Grant & Hasin, 1999; Lester, 2000) as well as alcohol dependence (Knopik et al., 2004). To date, however, no research has examined the influence of suicidal ideation or attempt on drinking course. In the present study, we examined diagnosis with a Diagnostic and Statistical Manual of Mental Disorders (3rd edition, *DSM–III*; American Psychiatric Association, 1980) depression or anxiety disorder as well as lifetime suicidal thoughts.

Reasons for Drinking

Drinking to regulate negative affect (e.g., tension reduction) robustly predicts alcohol use and problems (Cooper, Frone, Russell, & Mudar, 1995; Mann, Chassin, & Sher, 1987). In the only study to examine the association between reasons for drinking and course of alcohol involvement, Bennett et al. (1999) showed that persistent and developmentally limited courses

of drinking tended to have the highest escape and enhance reasons for use, followed by moderate (and low) groups. In the present study, we examined affect-regulation reasons for drinking as a predictor of drinking course.

Alternate Definitions of Alcohol Involvement

Although there is wide variation in how alcohol involvement is defined, alcohol involvement can be categorized into distinct but correlated domains: alcohol consumption and alcohol-related consequences or problems, and syndromal alcohol abuse and dependence. Heavy (or binge) alcohol consumption is related to higher probabilities of alcohol-related consequences (Harford, Wechsler, & Muthén, 2002; O'Hare, 1990; O'Neill, Parra, & Sher, 2001; Wechsler & Nelson, 2001) and higher rates of diagnosable AUDs, both cross-sectionally and prospectively (O'Neill et al., 2001; we note that prospective analyses suggest that this relation may be more robust during late adolescence than during later young adulthood, as individuals become more experienced with drinking alcohol).

However, some research suggests only a moderate association between consumption and problems, with only a small proportion of users actually reporting alcohol problems (Bailey & Rachal, 1993; Sadava, 1985, 1990; Stice, Barrera, & Chassin, 1998). Bailey and Rachal (1993) observed only moderate intercorrelations between indices of consumption frequency, alcohol-related problems, and symptoms of dependency, and Sadava (1990) computed the average correlation between consumption and problems as $r = .40$ across a set of general population studies. Bailey and Rachal (1993), Sadava (1990), and Stice, Barrera, and Chassin (1998) proposed distinct risk factors for moderate drinking and alcohol problems/consequences; Bailey and Rachal's findings (but not Stice, Barrera, & Chassin's findings) support this. No research to date, however, has explored the congruence between different courses of drinking on the basis of indices of alcohol consumption, alcohol consequences/problems, and alcohol use disorder or examined the extent to which these different courses can be differentially predicted by risk factors.

Overview

We evaluated the extent to which trajectories based on different indices of alcohol involvement tend to identify the same people. First, developmental trajectories were identified across five indices of alcohol involvement ranging in degree of severity, including alcohol use disorder, alcohol dependence, alcohol consequences, heavy drinking, and alcohol quantity–frequency. Then, pattern and prevalence of trajectory group membership was compared across indices. In addition, etiological predictors of trajectories based on different indices of alcohol involvement were compared.

Method

Sample and Procedure

Our sample was taken from a prospective study of college students (i.e., Sher, Walitzer, Wood, & Brent, 1991). During the 1987–1988 academic year, 3,156 incoming, first-time college freshmen at a large, midwestern university were recruited to participate in the study over the telephone. Participants were screened for family history of alcoholism with the Short Michigan Alcoholism Screening Test (F-SMAST and M-SMAST for paternal and maternal alcoholism, respectively; Selzer, Vinokur, & van Rooijen, 1975), adapted for assessing parental alcoholism (Crews & Sher, 1992), and sections of the Family History-Research Diagnostic Criteria interview (FH-RDC; Endicott, Andreasen, & Spitzer, 1978). Participants were classified as family history positive (FH+) for alcoholism if they scored a 4 or greater on the F-SMAST (in some cases, 3 was used as a cut score; see Sher et al., 1991) and if their biological father was

diagnosed with alcoholism with the FH-RDC. Participants were classified as family history negative (FH-) for alcoholism if they had no biological first-degree relatives with alcoholism or drug abuse or antisocial personality disorder and no second-degree relatives with alcoholism or drug abuse. Hence, our sample consisted of only those respondents who exhibited positive or negative (but not intermediate or indeterminate) family history of paternal alcoholism; consequently, approximately half of our sample was high risk (reflecting a substantial oversampling of persons with a positive family history of alcoholism). From these participants, a final sample consisting of 489 participants (47% male) was retained, including 113 FH− men, 118 FH+ men, 124 FH− women, and 132 FH+ women.[1] Baseline age was 18.5 years; 94% of the sample was Caucasian. Participants were assessed at baseline and at five subsequent occasions over the next 10 years (Years 2, 3, 4, 7, and 11). Assessment included clinical interview and self-administered questionnaire. Interviews were conducted primarily in person; telephone interviews increased in frequency as participants relocated (42% by Year 11). At Year 11, 410 participants (84% of the original 489) were reinterviewed; 33 refused further participation, 39 were not assessed (because they were never located, never scheduled, or failed to complete the survey), and 7 were deceased. A more complete portrayal of subject retention and loss over all six waves of assessment is presented in Jackson and Sher's (2003) study. For the present study, 377 participants (77% of the original 489) provided complete data at all six waves. At all waves, informed consent was given for participation.

Measures

We used five indices of alcohol involvement, including an interview-based alcohol use disorder assessment and four questionnaire-based measures: alcohol consequences, alcohol dependence, alcohol quantity–frequency, and heavy drinking.

AUD—Past-year alcohol abuse and dependence diagnoses were assessed with the Diagnostic Interview Schedule (DIS; Robins, Helzer, Croughan, Williams, & Spitzer, 1985): DIS Version III-A (Robins et al., 1985) at Years 1 and 2; DIS Version III-R (Robins, Helzer, Cottler, & Goldring, 1989) at Years 3, 4, and 7; and DIS Version-IV (Robins, Cottler, Bucholz, & Compton, 1997) at Year 11. To maintain continuity, diagnoses were made according to criteria from the *DSM–III* (American Psychiatric Association, 1980). A single AUD diagnosis was assigned if a participant met criteria for alcohol abuse and/or dependence.

Alcohol dependence—A count of past-year alcohol dependence symptoms was assessed with a 13-item scale (α from .70 to .85, over six waves) developed for the present study (Sher et al., 1991). Item responses included 0(*no, never*), 1 (*yes, but not in the past year*), 2 (*once in the past year*), 3 (*twice in the past year*), and 4 (*three or more times in the past year*). Sample items included experiencing shakes after quitting or cutting back on alcohol, needing larger amounts of alcohol to feel an effect, and not remembering part of the previous evening after drinking.

Alcohol consequences—A count of past-year alcohol consequences was assessed with a 14-item scale (α from .72 to .75), also developed for the present study (Sher et al., 1991). Item responses were identical to alcohol dependence responses. Sample items included getting into trouble at work or school because of drinking, finding oneself in a sexual situation when drinking that was later regretted, and receiving a lower grade on a test or paper because of drinking.

Alcohol quantity–frequency—Past-year quantity–frequency of alcohol consumption was computed by taking the product of per-week drinking quantity (number of drinks per drinking

[1]Two of the 489 participants learned after study entry that they had been adopted as children.

day, expressed as standard drink equivalents) and past-week drinking frequency (scaled to reflect drinking occasions per week). The variable was log-transformed (after adding 1) to reduce skew.

Heavy drinking—Frequency of heavy drinking in the past month was assessed with a single item: "In the past 30 days, how many times have you had five or more drinks at a single sitting, either of beer, wine, wine coolers, liquor, or some combination of these?" We retained the ordinal scaling of the item, which ranged from 0 (didn't drink five or more drinks at a single sitting in the past 30 days) to 7 (*every day*). Although we originally transformed this item to reflect weekly drinking values, we experienced model convergence problems with this highly skewed variable and consequently retained the original ordinal item.

Predictors of course—Predictors included sex, family history of alcoholism, conduct disorder symptom count, novelty seeking, lifetime diagnosis with a *DSM–III* depression or anxiety disorder, the presence of suicidal thoughts in lifetime, and affect-regulation reasons for drinking. All were taken from Year 1. Sex and family history of alcoholism (as described above) were assessed at screening for all respondents. Novelty seeking was assessed with the 34-item novelty-seeking subscale of the 98-item Tridimensional Personality Questionnaire (TPQ; Cloninger, 1987b). A symptom count for conduct disorder, diagnoses of lifetime generalized anxiety and depression, and the single-item measure assessing the presence of suicidal thoughts in lifetime were assessed with the DIS Version III-A (Robins et al., 1985). Finally, affect-regulation reasons for drinking (e.g., "I drink because it helps me to relax") were assessed with 7 items from a 15-item instrument adapted from Cahalan, Cisin, and Crossley (1969).

Data Analytic Technique

To identify trajectories of drinking, we used a mixture modeling procedure (Jones, Nagin, & Roeder, 2001; Muthén, 2001a, 2001b; Muthén & Muthén, 2000; Nagin, 1999), latent growth mixture models (LGMMs). These models "mix," in a single estimation procedure, the continuous nature of a latent growth curve model with the categorical nature of group membership. Typical latent growth curve models assume that respondents come from the same population, with identical mean levels (intercepts) and growth patterns (slopes) over time (with individual variation represented by the intercept and slope factor variances). LGMMs, however, allow for different populations to have unique intercepts and slopes. In essence, LGMMs estimate a unique latent growth curve (with individual variability) for each underlying population. For applications of this technique in the substance use area, see Chassin et al. (2002), Colder et al. (2001, 2002), and Li, Barrera, Hops, and Fisher (2002).

For our categorical measure (AUD), we used latent class growth analysis (LCGA), which is a procedure much like latent class analysis but which incorporates the time-ordered nature of the data (Muthén, 2001a). The latent classes are particular subtypes or groups of individuals who empirically display similar patterns of diagnosing with AUD, and the observed variables represent the presence (1) or absence (0) of a diagnosis with AUD at Years 1, 2, 3, 4, 7, and 11.

All models to estimate trajectories were estimated with Mplus, Version 2.13 (Muthén & Muthén, 1998–2004). The base model included intercept and linear slope. The categorical AUD model also included a quadratic slope (models with a quadratic slope would not converge

for continuous alcohol measures).[2] Regressions to predict group membership from baseline predictors of drinking course were also estimated with Mplus.

Results

Identification of Trajectories

To maximize meaningful comparisons, we selected a four-class model for each measure. For most cases, this was the best-fitting model in terms of model fit and class interpretability and size (see Table 1 for fit indices). Model fit was evaluated with information criteria fit indices (Bayesian information criterion [Schwartz, 1978]; Akaike's information criterion [Akaike, 1987]). Table 1 also presents entropy values, which represent precision of classification based on posterior probability values (Muthén & Muthén, 1998-2004); an entropy value close to 1.0 indicates clear classification (little overlap among trajectories).

Figures 1 through 5 show the developmental courses for alcohol use disorder, alcohol dependence, alcohol consequences, alcohol quantity–frequency, and heavy drinking, respectively. Group membership was characterized by the following courses: nondrinking or nonproblematic drinking, which ranged from 45% to 74%; developmentally limited, which decreased over time, ranging from 8% to 26%; later onset, which increased over time, ranging from 2% to 19%; and chronic (this course also often exhibited a decrease over time; however, it was the most chronic of all courses), which ranged from 3% to 22%.

Comparison of Trajectories

To compare agreement between class membership for each measure, respondents were first assigned to classes on the basis of their most likely group membership (unweighted by probability of class membership), and then contingency tables were created for each pairwise comparison (10 in all).[3] Agreement between indices, assessed by Cohen's kappa (Cohen, 1960), ranged from $\kappa = .26$ to $\kappa = .54$ (see below the diagonal in Table 2). These represent moderate levels of agreement (Goodman & Kruskal, 1954). Ancillary analyses examined hand-calculated kappa estimates that were weighted by probability of group membership, which were very similar in magnitude ($\kappa = .21$ to $\kappa = .48$; see above the diagonal in Table 2). Note that these "weighted" kappas are not weighted kappas as the term is traditionally used in the literature to describe a kappa variant that takes into account gradation in level of disagreement (Cohen, 1968). Rather, they are unweighted kappas for estimates that were weighted by probability of group membership.

One concern is that the association between any two measures is actually driven by the nonproblem groups. We recomputed the kappas after eliminating those two groups from the comparison (see Table 3). On average, the magnitude of the kappas was reduced by about two thirds (*Mdn* = 66%), suggesting that, overall, different patterns among users account for the majority, but not all, of the concordance between the indices. Inspection of specific pairs indicated substantial variability after eliminating nonusers (39% to 94% of the original value of the kappa remained after removing nonusers); however, there was no specific pattern suggesting that nonusers account for concordance among certain constructs.

[2]We note that the AUD model with a linear slope only produces trajectories and class prevalences that are very similar to the model with the quadratic and linear slopes.

[3]When possible, estimates that are weighted by the probability for group membership are preferred, as it ensures that individuals who are "cleanly" assigned to a group (e.g., 100% likely to be in the chronic group, 0% likely to be in any other group) are not treated identically to individuals whose profiles are less well defined (e.g., 51% likely to be in the chronic group, 49% likely to be in the later-onset group). However, it was not possible to create contingency tables with this weighted approach, and for these analyses, we assigned respondents to their most likely class.

Given that presenting each of the 10 pairwise contingency tables would be cumbersome, we also present a summary figure (see Figure 6) documenting the location of trajectory agreement (i.e., along the diagonal) and disagreement (i.e., off-diagonal cells). To determine the particular combinations of courses that most contribute to agreement between two trajectories, the cell chi-square statistics for each cell were plotted with bar graphs for each of the 10 comparisons. Corresponding courses were observed at a rate greater than what would be expected by chance (i.e., agreement along the diagonal). Associations between AUD and other indices were lower than those between any other comparison. This may be due to the larger negative slope and higher initial value of the developmentally limited course for AUD than for the other indices.

Correspondingly, disagreement along the off diagonal was considerable, although the chronic course was occasionally associated with the developmentally limited and later-onset courses. Although the chronic course tended to decline over time for some of the alcohol indices (AUD, alcohol dependence, alcohol consequences), this would not explain why it was associated with the developmentally limited course for other alcohol indices or why it was at times associated with the later-onset course.

Etiological Predictors

Finally, we examined third-variable prediction of group membership for each of the indices. Predictors (sex, family history of alcoholism, conduct disorder symptom count, novelty seeking, lifetime diagnosis with a *DSM–III* depression or anxiety disorder, presence of suicidal thoughts in lifetime, and affect-regulation reasons for drinking) were treated as exogenous to class membership. Continuous measures (conduct disorder symptom count, novelty seeking, and reasons for drinking) were standardized to provide a more meaningful metric for odds ratios. A multinomial logistic regression procedure was used (Agresti, 1990). Five comparisons of interest were examined: the three drinking groups versus the nondrinking group (consistent with the literature) and developmentally limited versus chronic (begin at the same point but diverge) and later onset versus chronic (begin at different points but converge). All models were run in Mplus, which allows for parameters to be weighted by probability of group membership. For the five psychosocial predictors, sex and family history were controlled.

In Table 4, we present odds ratios for each measure of alcohol involvement and each of the seven predictors. Relative to the nondrinking/nonproblematic drinking group, likelihood of membership in the chronic group was generally predicted by all etiological predictors, although considerably less so for family history of alcoholism, depression/anxiety, and suicidal thoughts. In general, likelihood of belonging to the developmentally limited class was significantly predicted by greater conduct disorder, novelty seeking, and reasons for drinking. Likelihood of belonging to the later onset class was positively predicted by greater reasons for drinking and, for measures of more problematic drinking (AUD, alcohol dependence, alcohol consequences), greater conduct disorder. There were few differences between the chronic group and the developmentally limited and later-onset groups. Those who remitted from the problematic alcohol involvement measures were less likely to exhibit conduct disorder or endorse affect-regulation reasons for drinking than were those who continued their drinking. In addition, those who increased their alcohol involvement over young adulthood were more likely to exhibit conduct disorder (for the consumption measures) or endorse novelty seeking (particularly for the problematic measures). In sum, with a few exceptions, prediction tended to be relatively consistent across measures of alcohol involvement, despite the differences in trajectory structure and membership and the low-to-moderate agreement among trajectories.

Discussion

The present study examined congruence of drinking trajectories over an 11-year interval across five indices of alcohol involvement, including AUD, alcohol dependence, alcohol

consequences, heavy drinking, and alcohol quantity–frequency, and explored the extent to which etiological predictors of drinking trajectory varied across indices.

Agreement Across Alternate Indices of Alcohol Involvement

For the five alcohol indices, we observed four developmental courses tracking alcohol involvement across early young adulthood. Given the epidemiological data showing the decline in heavy and problematic drinking during this developmental period (e.g., Dawson et al., 2004; Johnston et al., 2002a, 2002b), it was not surprising that we observed a developmentally limited course for each measure. In fact, for most indices of alcohol involvement, even the more chronic course tended to evince a decline over time. We examined whether the slopes for the chronic and developmentally groups were in fact identical by examining the 95% confidence intervals for the slope parameter. The two slopes were identical for alcohol consequences (but not for the other measures), suggesting that, for this measure, despite different initial levels, these two groups shared a normative decrease. We do note that the developmentally limited course may be less evident in nonstudents. In fact, there is evidence that although college students surpass their noncollege student peers in heavy drinking after high school, they exhibit lower levels of heavy drinking in their 30s (Jackson, Sher, & Park, 2005), and recent analyses of the National Epidemiologic Survey on Alcohol and Related Conditions (Dawson et al., 2004) revealed that those not in college and living independently tended to have more stable heavy drinking rates than did those in college, who revealed a more developmentally limited pattern.

Despite relatively similar trajectory shapes, predicted prevalences varied considerably across alternate indices of alcohol involvement. The indices of alcohol dependence and alcohol consequences showed the greatest number of participants in the nonproblematic drinking category and the fewest in either the chronic (for alcohol consequences) or later-onset (for alcohol dependence) classes. Whereas most of the individuals who reported having had some alcohol consequences tended to show a developmentally limited pattern, alcohol dependence contained large frequencies of both chronic and developmentally limited groups. Alcohol quantity–frequency showed the fewest in the nondrinking category and the greatest in the developmentally limited category, which is consistent with the notion of abundant drinking during college. Likewise, heavy drinking showed a large group of developmentally limited individuals, although an equally large group of individuals who persistently binge drank was observed. Consistent with the idea of a Type I alcoholic (Cloninger, 1987a), there was a substantial group (19%) with a later-onset trajectory for the AUD measure; none of the other indices showed later-onset groups of such magnitude (all others < 10%).

Of greatest importance for the present study, trajectory classifications across alternate indices of alcohol involvement revealed only small-to-moderate agreement. This has profound implications for researchers who wish to generalize developmental courses among indices of alcohol involvement. Not surprisingly, more similar indices showed greater agreement, with the two consumption indices (quantity–frequency and heavy drinking) showing the greatest agreement, along with the two questionnaire-based indices of more pathological alcohol involvement (alcohol dependence and alcohol consequences). Agreement with the AUD measure tended to be lowest, which is likely due in part to the interview-based assessment and categorical nature of AUD. As would be expected, heavy drinking showed stronger agreement with the problem indices (AUD, alcohol dependence, alcohol consequences) than did alcohol quantity–frequency. Although the concordance between nonusing groups contributed to a good part of the agreement between indices, eliminating these groups did not diminish the relation entirely. Most of the correspondence was due to concordance between corresponding groups, although, at times, there was a tendency for the chronic group to be classified as developmentally limited or later onset.

Etiological Predictors

Both the temperamental and behavioral measures of behavioral undercontrol exhibited strong prediction of alcohol group membership across all indices. This is consistent with much of the literature showing effects for measures of harm avoidance and disinhibition and antisocial, externalizing, risk taking, and delinquent behavior on drinking trajectories (e.g., Bates & Labouvie, 1997; Bennett et al., 1999; Chassin et al., 2002; Colder et al., 2002; Hill et al., 2000; Stice, Myers, & Brown, 1998; Tucker et al., 2003; Wills et al., 1996). Substance use such as drinking may occur in the context of general problem behavior, whereby behavioral undercontrol and substance use are two of a number of behaviors associated with a deviant lifestyle (Farrell, Danish, & Howard, 1992; Jessor & Jessor, 1977). For the problem alcohol indices only, behavioral undercontrol was associated with relatively heavy and persistent drinking during early young adulthood (i.e., the early college years) versus drinking that remitted over time. In addition, and not surprisingly, behavioral undercontrol was greater for those whose drinking was chronic than for those who had a later onset of drinking.

In addition, affect-regulation reasons for drinking were highly predictive of group membership, with a monotonically decreasing level from chronic to developmentally limited to nondrinking trajectories (reasons for drinking in the later-onset group were greater than in the nondrinking group but were not necessarily greater than those of the other two groups). This is consistent with Bennett et al. (1999), who found that escape and enhance reasons for use were greatest for the persistent and developmentally limited drinking groups. The effects for negative affect were less robust, supporting previous work showing this to be a weaker predictor than is behavioral undercontrol (e.g., Colder et al., 2002).

Consistent with cross-sectional analyses of our own data (Sher et al., 1991), we expected to see the strongest effect of family history on more problematic measures of alcohol involvement. We did, in fact, find a difference between chronic and nondiagnosing groups for AUD and alcohol dependence. Our prior work showed that family history relates to the persistence of problematic alcohol involvement in early adulthood rather than to the initiation into heavier drinking (Jackson, Sher, Gotham, & Wood, 2001); prediction of membership in chronic but not developmentally limited drinking trajectories is consistent with this. However, surprisingly, we failed to observe differences among the diagnosing groups (e.g., chronic vs. developmentally limited or later onset).

In addition, there were fewer sex effects than might be expected. Being male did tend to predict heavy drinking course, however, which may be due in part to the convention of defining binge drinking as five drinks at a time for both men and women, despite individual differences—in body mass, body water content, and gastric metabolism of alcohol (Lieber, 1997)—associated with gender. These gender differences may not have been as readily observable with a more gender-specific definition of binge drinking (Wechsler, Dowdall, Davenport, & Rimm, 1995). However, gender effects were noted for other measures where the definitions were not gender specific (e.g., AUD; also for alcohol consequences and heavy drinking using univariate tests; results not shown).

Despite the epidemiological literature on “maturing out,” we found few risk factors that actually distinguished between individuals who moderate versus persist in their alcohol consumption. Some of the more temperamental measures (e.g., conduct disorder, novelty seeking, reasons for drinking) predicted membership in the chronic group, relative to the developmentally limited group, for the problematic alcohol indices. Following the framework suggested by Sher, Gotham, and Watson (2004), these constructs (particularly conduct disorder and novelty seeking) could be considered “stable vulnerability indicators” that distinguish course and may be indexing a severity-graded vulnerability process such that the developmentally limited course is a less severe form of the chronic course. Conversely, those in the chronic group may

be vulnerable to the influence of more time-varying, situational risk factors, which were not assessed (e.g., peer influence, life stressors), that could increase their risk of problematic alcohol involvement.

In sum, despite small-to-moderate agreement in trajectory classification, we observed roughly similar patterns of prediction across trajectories based on different indices of alcohol involvement. Although we could not explicitly test this, alcohol quantity–frequency appeared to show the smallest effects, and the most severe indices of problematic drinking appeared to show the strongest effects, especially for the chronic trajectories.

Conceptualization of Developmental Course

Recent research in substance use, and in developmental psychopathology in general, has attempted to model heterogeneity in developmental course and to determine the risk and protective factors associated with a less maladaptive course. Although course can be represented by modeling trajectories, as in the present article, it is certainly not the only way to represent course. Research has explored correlates of individual growth curves representing initial level (intercept) and growth (using polynomial slope factors) of a construct using a growth curve modeling approach (Muthén & Curran, 1997). For situations where modeling growth in a process is not a study objective, chronicity can be represented as a continuous variable using state-trait modeling (e.g., Jackson & Sher, 2003), which posits that behavior over time can be captured by a single latent variable. One of the strengths of state-trait models is in the multiple assessments of a single construct. Increasing the number of assessments increases our assessment of reliability, akin to Cronbach's alpha increasing with number of scale items (Nunnally & Bernstein, 1994). In fact, behavior averaged over a number of events has been shown to be more predictable than is a single instance of behavior (Epstein, 1979). Current work in our lab is exploring the extent to which modeling behavior as a chronic process is more powerful than exploring behavior at individual time points. Concordance between alternate indices of alcohol involvement can also be represented with the state-trait model as well as by agreement between developmental courses. Preliminary work (Sher & Jackson, 2004) has shown that aggregated (trait) correlations between alternate indices of alcohol involvement are considerably greater than are the median zero-order correlations across six waves. Variable-centered approaches such as state-trait modeling complement the more person-centered approaches such as that taken in the present study.

Final Comments

In sum, we found that longitudinal trajectories of alcohol involvement were conditional on the indices used. Recognition of this general finding should lead researchers to be cautious in generalizing longitudinal patterns across different facets of alcohol involvement, and care must be taken not to overreify empirically derived trajectories. Although person-based approaches to resolving course of alcohol involvement are almost certainly an advance in thinking about variations in alcohol involvement over the life course, we should not lose sight of the fact that trajectories need to be referenced to the indices, timing, and observation periods used.

Thus, from our perspective, characterization of trajectories represents a step forward in the study of developmental aspects of alcohol involvement in the life course. However, we are still at an early stage of research with respect to understanding the implications of these new methodologies for how well we "carve nature at her joints." Our present findings suggest that, although carvings based on different facets of alcohol yield similar structures and show roughly similar patterns of correlates, they are proportioned differently (i.e., yield different prevalences). Also, we do not yet know how best to choose the number and timing of measurement occasions to yield optimally resolved portrayals of life-course trajectories. Progress in our understanding individual differences in alcohol use over the life course will

require a careful consideration of the developmental meanings of different domains of alcohol involvement and longitudinal designs that are optimally sensitive to the underlying phenomena.

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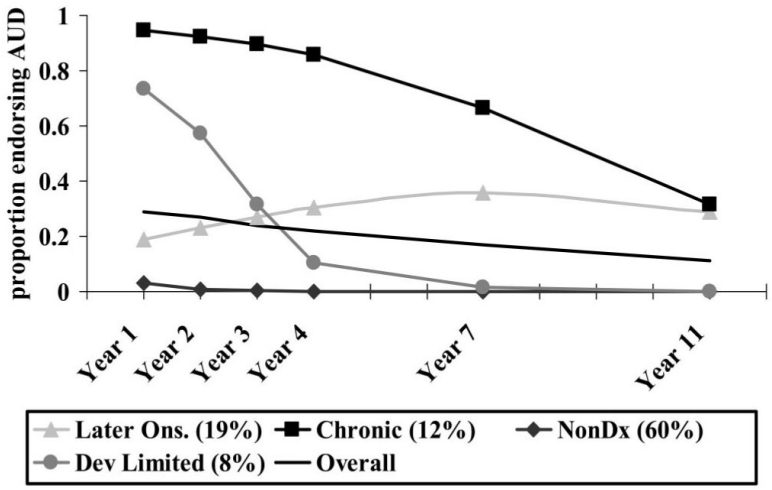

Figure 1.

Latent class growth analysis model for alcohol use disorder (AUD) at Waves 1–6, weighted by estimated class probabilities. *N* = 377. Later Ons. = later onset; NonDx = nondrinking or nonproblematic drinking; Dev Limited = developmentally limited.

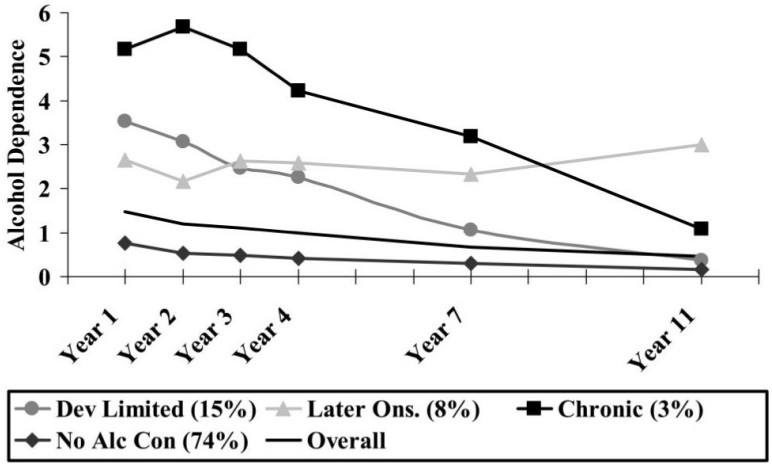

Figure 2.

Mixture model for alcohol dependence at Waves 1–6, weighted by estimated class probabilities. *N* = 377. Alcohol dependence symptoms are scored as follows: 0 = *no, never*, 1 = *yes, but not in the past year*, 2 = *once in the past year*, 3 = *twice in the past year*, and 4 = *three or more times in the past year*. Dev Limited = developmentally limited; Later Ons. = later onset; No Alc Con = no alcohol consequences.

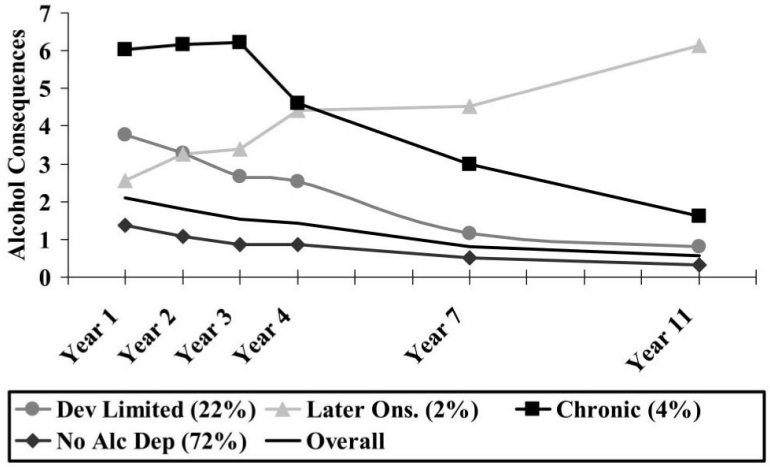

Figure 3.

Mixture model for alcohol consequences at Waves 1–6, weighted by estimated class probabilities. *N* = 377. Alcohol consequence symptoms are scored as follows: 0 = *no, never*, 1 = *yes, but not in the past year*, 2 = *once in the past year*, 3 = *twice in the past year*, and 4 = *three or more times in the past year*. Dev. Limited = developmentally limited; Later Ons. = later onset; No Alc Dep = no alcohol dependence.

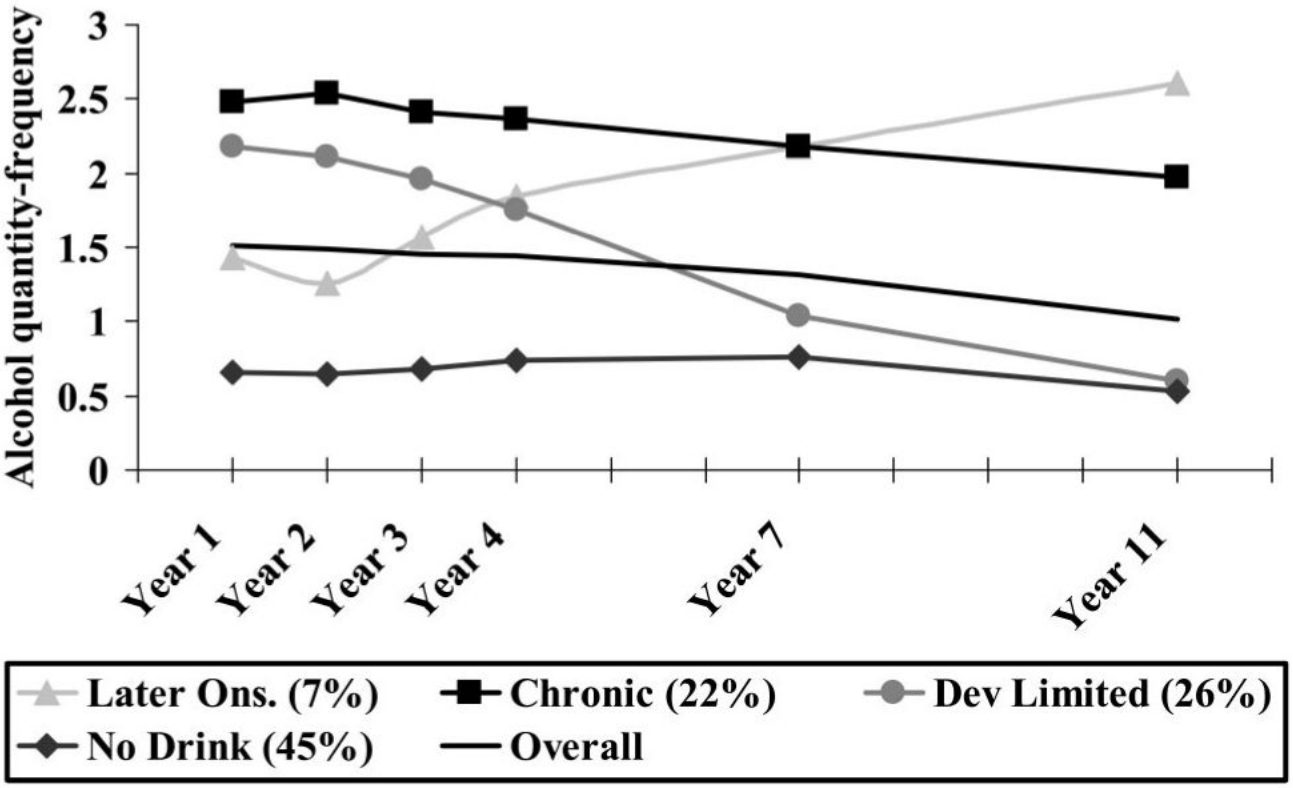

Figure 4.
Mixture model for alcohol quantity–frequency at Waves 1–6, weighted by estimated class probabilities. *N* = 377. Alcohol quantity–frequency is scaled as the log of (the product of per-week drinking quantity and past-week drinking frequency) +1. Later Ons. = later onset; Dev Limited = developmentally limited.

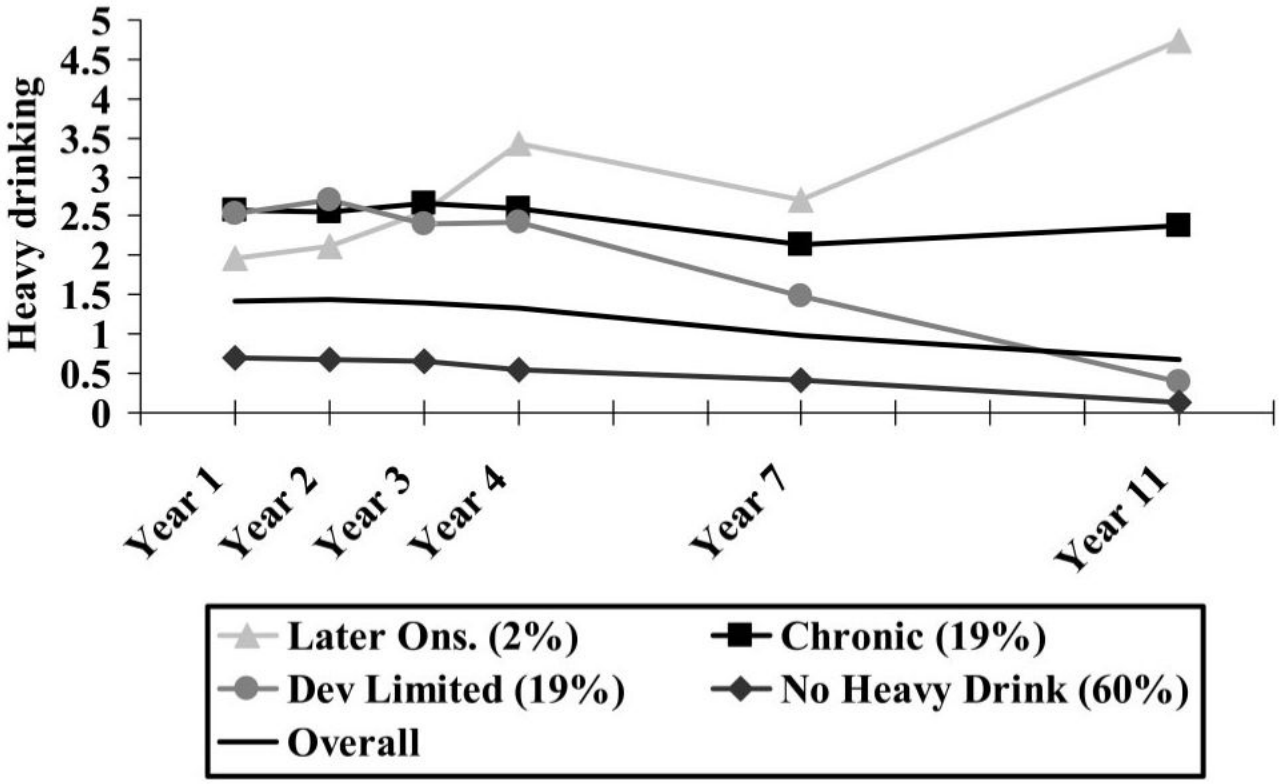

Figure 5.

Mixture model for heavy drinking at Waves 1–6, weighted by estimated class probabilities. *N* = 377. Heavy drinking was scored as 0 = didn't drink 5 or more drinks at a single sitting in the past 30 days, 2 = *once during the past 30 days*, 3 = *two to three times during the past 30 days*, 4 = *once or twice a week*, 5 = *three to four times a week*, 6 = *five to six times a week*, and 7 = *nearly every day*. Later Ons. = later onset; Dev Limited = developmentally limited.

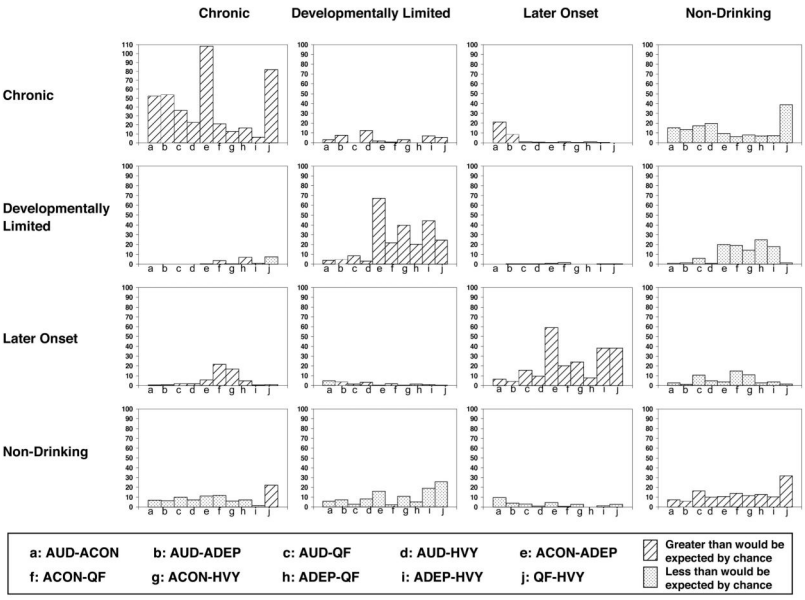

Figure 6.

Summary of trajectory agreement represented by cell chi-square statistics for each of the five alcohol measures. Dark bars reflect values that are greater than would be expected by chance, and light bars indicate values that are lower than would be expected by chance, based on the marginals. Note that a chi-square value with a single degree of freedom of 3.84 is significant at $p < .05$, and a chi-square value with a single degree of freedom of 6.64 is significant at $p < .01$. AUD = alcohol use disorder; ACON = alcohol consequences; ADEP = alcohol dependence; QF = alcohol quantity–frequency; HVY = heavy drinking.

Table 1

Model Fit for Five Indices of Alcohol Involvement

Measure of alcohol involvement and fit index	Number of classes			
	2-class	3-class	4-class	5-class
Alcohol use disorder				
AIC	1,563.74	1,551.65	1,519.46	1,522.66
BIC	1,583.40	1,583.10	1,562.71	1,577.71
Entropy	.88	.85	.76	.76
Alcohol dependence				
AIC	7,456.31	7,354.70	7,313.14	—[a]
BIC	7,511.36	7,421.55	7,391.78	—[a]
Entropy	.94	.94	.90	—[a]
Alcohol consequences				
AIC	6,879.14	6,779.28	6,751.90	6,743.92
BIC	6,934.19	6,846.13	6,830.55	6,834.37
Entropy	.94	.92	.92	.90
Alcohol quantity–frequency[b]				
AIC	4,953.96	4,929.53	4,904.80	4,880.60
BIC	5,009.01	4,996.38	4,983.45	4,971.04
Entropy	.68	.67	.73	.77
Heavy drinking				
AIC	6,622.62	6,592.86	6,497.64	6,513.64
BIC	6,677.86	6,659.93	6,576.55	6,604.34
Entropy	.94	.83	.87	.90

Note. *N* = 377. AIC = Akaike's information criterion; BIC = Bayesian information criterion.

[a] Model would not converge.

[b] Variable is log-transformed (after adding 1).

Table 2

Comparison of Trajectories Using Cohen's Kappa

Measure of alcohol involvement	AUD	Alcohol dependence	Alcohol consequences	Alcohol quantity–frequency	Heavy drinking
AUD	—	.21	.28	.26	.24
Alcohol dependence	.26	—	.48	.28	.32
Alcohol consequences	.30	.54	—	.30	.34
Alcohol quantity–frequency	.32	.31	.32	—	.43
Heavy drinking	.28	.38	.38	.50	—

Note. $N = 377$. Kappas based on unweighted estimates are shown below the diagonal; kappas based on weighted estimates are shown above the diagonal (note that these are all unweighted kappas, but the data are either weighted or not weighted by probability of group membership). AUD = *DSM-III* alcohol use disorder.

Table 3

Comparison of Trajectories Using Cohen's Kappa After Removing Nonusers

Measure of alcohol involvement	AUD	Alcohol dependence	Alcohol consequences	Alcohol quantity–frequency	Heavy drinking
AUD	—	.16 (76%)	.19 (68%)	.19 (73%)	.10 (42%)
Alcohol dependence	.16 (61%)	—	.43 (90%)	.19 (68%)	.14 (44%)
Alcohol consequences	.16 (53%)	.49 (91%)	—	.36 (83%)	.21 (62%)
Alcohol quantity–frequency	.24 (75%)	.21 (68%)	.30 (94%)	—	.39 (91%)
Heavy drinking	.11 (39%)	.18 (47%)	.24 (63%)	.45 (90%)	—

Note. $N = 377$. Kappas based on unweighted estimates are shown below the diagonal; kappas based on weighted estimates are shown above the diagonal (note that these are all unweighted kappas, but the data are either weighted or not weighted by probability of group membership). Numbers in parentheses are percentages of the original kappas that remained after nonusers were removed. AUD = *DSM-III* alcohol use disorder.

Table 4

Odds Ratios and Significance Values for Univariate Prediction of Trajectories for Five Pairwise Comparisons Across Each Measure of Alcohol Involvement, for Seven Predictor Variables

Predictor variable	AUD	Alcohol dependence	Alcohol consequences	Alcohol quantity–frequency	Heavy drinking
Sex (male = 1, female = 0)					
Chrn vs. non	6.66[a]**	1.98	7.94*	5.66**	6.22**
Dev vs. non	1.37[a]	0.97	1.41	1.13	3.17**
Late vs. non	1.56[a]	2.37	2.00	3.07	12.40*
eDv vs. chrn	0.20[a]**	0.49	0.18†	0.20**	0.51†
Late vs. chrn	0.23[a]**	1.20	0.25	0.54	1.99
Family history of alcoholism					
Chrn vs. non	3.44**	4.38*	10.53†	1.33[b]	1.25
Dev vs. non	2.87†	1.36	1.79	1.56[b]†	1.86
Late vs. non	2.77†	5.97	2.25	2.56[b]†	1.60
Dev vs. chrn	0.84	0.31	0.17	0.82[b]	1.49
Late vs. chrn	0.80	1.36	0.21	0.56[b]*	1.28
Conduct disorder					
Chrn vs. non	5.08**	3.73**	3.13**	2.73**	2.12**
Dev vs. non	2.89**	1.91**	1.61*	2.61	2.03**
Late vs. non	2.58**	2.30	1.64*	1.81	0.42
Dev vs. chrn	0.57*	0.51**	0.51*	0.94	0.96
Late vs. chrn	0.51	0.62	0.53*	0.67†	0.20*
Novelty seeking					
Chrn vs. non	2.67**	3.59**	3.88**	3.05**	2.16**
Dev vs. non	1.82**	2.00	1.87**	2.62**	2.56**
Late vs. non	1.34	1.70	1.39	1.68	1.91
Dev vs. chrn	0.68	0.56*	0.48†	0.85	1.16
Late vs. chrn	0.50**	0.47	0.36**	0.56	0.82

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Predictor variable	AUD	Alcohol dependence	Alcohol consequences	Alcohol quantity–frequency	Heavy drinking
Depression/anxiety					
Chrn vs. non	3.67*	3.32*	2.99	1.30	1.01
Dev vs. non	4.13	1.62	1.43	1.48	1.20
Late vs. non	1.64[c]	0.50	0.31	1.41	—[d]
Dev vs. chrn	0.75	0.49	0.48†	0.85	1.32
Late vs. chrn	0.38[c]†	0.15	0.36**	0.56	—[d]
Suicidal thoughts					
Chrn vs. non	4.32**	4.32*	3.18	4.16	3.30**
Dev vs. non	0.47	2.59*	0.95	1.93	1.84
Late vs. non	4.32**	0.42	1.61	6.19	0.56
Dev vs. chrn	0.64	0.85	0.78	0.86	0.81†
Late vs. chrn	1.00	0.63	0.87	1.08	0.69
Affect-regulation reasons for drinking					
Chrn vs. non	6.00**	15.26**	7.08**	4.16[e]**	5.67**
Dev vs. non	2.74**	5.12**	4.13**	3.59[e]**	5.68**
Late vs. non	5.01**	4.99**	4.29**	1.90[e]*	10.40**
Dev vs. chrn	0.46*	0.34**	0.58	0.82[e]	1.01
Late vs. chrn	0.84	0.33*	0.61	0.56[e]*	1.79

Note. = N = 377. All models control for sex and family history, with the exception of the sex and the family history analyses. Chrn = chronic; dev = developmentally limited; Late = later onset; non = nondrinking or nondiagnosing; AUD = *DSM-III* alcohol use disorder.

[a] Owing to an inestimable model that was due to low numbers of drinkers in the female family history negative group, these analyses were conducted as logistic regressions outside of the mixture modeling framework.

[b] Owing to an inestimable model because of the low numbers of drinkers in the family history negative group, these analyses were conducted as logistic regressions outside of the mixture modeling framework, by assigning each participant to his or her most likely class and running a series of logistic regressions.

[c] As model could not be estimated with family history of alcoholism, parameters are from a model without this term.

[d] Parameter was inestimable because 100% of the later onset group had a history of depression.

[e] Owing to large odds ratios in excess of 1,600, analyses were conducted as logistic regressions outside of the mixture modeling framework, by assigning each participant to his or her most likely class and running a series of logistic regressions.

[†] $p < .10$.

[*] $p < .05$.

[**] $p < .01$.

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