

Received Date : 07-Jul-2016

Revised Date : 19-Feb-2017

Accepted Date : 22-Feb-2017

Article type : Original Research Article

Using Factor Mixture Models to Evaluate the Type A/B Classification of Alcohol Use Disorders in a Heterogeneous Treatment Sample

Tom Hildebrandt, Psy.D.^a, Elizabeth E. Epstein, Ph.D.^{b,c}, Robyn Sysko, Ph.D.^a, Donald A. Bux, Jr., Ph.D.^d

^aDepartment of Psychiatry, Mount Sinai School of Medicine, One Gustave L Levy Place, Box 1230, New York, NY 10029

^bCenter of Alcohol Studies, Rutgers – The State University of New Jersey, Smithers Hall, 607 Allison Road, Piscataway, NJ 08854-8001

^cDepartment of Psychiatry, University of Massachusetts Medical School, 365 Plantation Street, Worcester, MA 01605

^dDepartment of Psychiatry, Montefiore Medical Center, 334 East 148th Street, Bronx, NY 10451-5707

Corresponding Author: Tom Hildebrandt, Psy.D., Department of Psychiatry, Mount Sinai School of Medicine, One Gustave L Levy Place, Box 1230, New York, NY 10029, Phone: 212-659-8673, Fax: 212-849-2561, Email: tom.hildebrandt@mssm.edu

This article has been accepted for publication and undergone full peer review but has not been through the copyediting, typesetting, pagination and proofreading process, which may lead to differences between this version and the Version of Record. Please cite this article as doi: 10.1111/acer.13367

This article is protected by copyright. All rights reserved.

Acknowledgements: This work was supported by the National Institutes of Health: NIAA R29-AA 09894, NIAAA P50-AA08747, NIAA T32-AA07569, and NIDA K23 DA-024043

The authors have no conflict of interest to declare.

Abstract

Background: The type A/B classification model for alcohol use disorders (AUDs) has received considerable empirical support. However, few studies examine the underlying latent structure of this subtyping model, which has been challenged as a dichotomization of a single drinking severity dimension. Type B, relative to type A, alcoholics represent those with early age of onset, greater familial risk, and worse outcomes from alcohol use. **Method:** We examined the latent structure of the type A/B model using categorical, dimensional, and factor mixture models in a mixed gender community treatment-seeking sample of adults with an AUD. **Results:** Factor analytic models identified 2-factors (drinking severity/externalizing psychopathology and internalizing psychopathology) underlying the type A/B indicators. A factor mixture model with 2-dimensions and 3-classes emerged as the best overall fitting model. The classes reflected a type A class and two type B classes (B1 and B2) that differed on the respective level of drinking severity/externalizing pathology and internalizing pathology. Type B1 had a greater prevalence of women and more internalizing pathology and B2 had a greater prevalence of men and more drinking severity/externalizing pathology. The 2-factor, 3-class model also exhibited predictive validity by explaining significant variance in 12-month drinking and drug use outcomes. **Conclusions:** The model identified in the current study may provide a basis for examining different sources of heterogeneity in the course and outcome of AUDs.

Keywords: type A/B classification, factor analysis, factor mixture model, typology

Introduction

Classifying individuals with alcohol use disorders (AUDs) into meaningful subtypes has resulted in numerous typologies derived from a range of methodologies and clinical or research objectives. A considerable typology literature reports on subtypes A versus B, with mixed support (Babor et al., 1992; Babor & Caetano, 2006). Briefly, type A drinkers relative to type B have a later age of onset, fewer childhood and familial risk factors, a lower quantity and frequency of drinking, more limited consequences from drinking, and lower rates of drug and psychopathology related comorbidity than type B drinkers. Type A/B classification is noted in research (e.g., Brown et al., 1994), community (e.g., Carpenter et al., 2006), and national samples (e.g., Carpenter & Hasin, 2001), and appears to moderate treatment response (e.g., Kranzler et al., 1996) and disease progression (Carpenter et al., 2006). Recent population data indicate that Type B drinkers experience greater odds of persistent drinking problems and poorer response to treatment over three year follow-up (Tam, Mulia, & Schmidt, 2014). Investigations of alcohol typology derived strictly by diagnostic information yield inconsistent results suggesting as many as five groups varying in severity (e.g., Beseler et al., 2012; Casey, Adamson, & Stringer, 2013; Ko et al., 2012), although prospective studies suggest age of onset distinguishes these typologies with adult onset alcohol dependent drinkers experiencing more internalizing symptoms than early onset individuals (Meier et al., 2013). Questions about these strictly categorical models and their results led Hasin and colleagues (2013) to recommend that alcohol diagnosis constitute one category varying in dimensional severity for *Diagnostic and Statistical Manual (DSM)-5*.

Empirical support for traditional dimensional approaches is also not consistent. Factor analytic studies of *DSM III-R* or *IV* (American Psychiatric Association, 1987; 2000) criteria support the existence of a unidimensional latent construct broadly characterized as “alcohol problem severity” (e.g., Ray et al., 2008). Other studies indicate separate abuse and dependence

dimensions that drive the diagnostic criteria (Harford & Muthén, 2001), particularly when accounting for item biases related to gender, age, and race/ethnicity (Harford et al., 2009).

Extensions of these models have been used to establish reliability of this unidimensional severity construct across substance using populations (Shmulewitz, Greene, & Hasin, 2015).

Dimensional latent trait models, like item response theory (IRT) models, have the advantage of examining specific diagnostic criteria within a modern psychometric framework. Using IRT, distinct *DSM-IV* abuse and dependence criteria were associated with greatest and least problem severity, respectively, contrary to the prevailing model of lower relative severity in alcohol abuse compared to dependence (Saha et al., 2007). McBride et al. (2011) found support for a unidimensional model using *DSM* criteria, although acceptable 3-class or 2-factor models also fit data in this large population sample. Models focused on symptoms other than *DSM* criteria have also been proposed, with dimensional severity underlying AUDs that provides a relevant statistical and diagnostic model with the greatest potential for predictive validity (e.g., Krueger et al., 2004).

Strictly categorical and dimensional models are complicated by important sources of covariation (e.g., psychiatric comorbidity, gender; Del Boca & Hesselbrock, 1996; Epstein et al., 2002; Morgenstern et al., 1997). Typologies may perform less well for women and the type A/B classification has not been replicated in female alcoholics (McGue et al., 1997). Two continuous traits may explain some of the covariance complexity encountered in alcohol subtyping literature. Krueger and colleagues (2005) proposed that AUDs vary as a function of an externalizing continuum with some evidence that AUD subtypes vary by a dimension of severity that positively correlates with externalizing symptoms (Swift et al., 2016).

Theoretically, three dimensions contribute to this continuum (externalizing, internalizing, and problematic substance use) and account for between-subject variation linked to comorbidity and gender. However, the relevant contribution of age remains underdeveloped. Externalizing

and substance use disorders co-occur at higher rates in younger patients, whereas internalizing disorders and substance use disorders co-occur at higher rates in older adults (Chan et al., 2008). Thus, some theoretical and empirical data support dimensional variation in AUDs associated with either externalizing or internalizing psychopathology (Del Boca & Hesselbrock, 1996).

Given these competing conceptual frameworks, hybrid classification models have also been considered; that is, models that simultaneously incorporate dimensional and categorical constructs by positing discrete classes of alcoholics within which members may be differentiated on one or more dimensionals. A two-class (low endorsement vs. high endorsement for abuse and dependence criteria), single factor model (i.e., single dimension within each class) was found in the first application of this type of model to *DSM-IV* abuse and dependence criteria (Muthén, 2006). The two latent classes were differentiated primarily by probability of impairment in social and work activities and did not correspond to the *DSM-IV* alcohol abuse and dependence categories, which is also consistent with Kuo et al. (2008) and McBride et al. (2013) who found similar dimensionality with 3-classes.

The present study is an extension of modeling research (Hildebrandt et al., 2007; Saha et al., 2006), and is the first to compare hybrid models of alcohol dependence using the clinical indicators traditionally used to derive the type A/B classification in a treatment seeking sample. Given the superior fit of hybrid models when relying on *DSM-IV* criteria, we hypothesized a similar advantage of hybrid models to the classical categorical and dimensional approaches for explaining the heterogeneity in AUDs. As both psychopathology and drug use severity contribute to variation in these subtypes, we also hypothesized that the hybrid model would include two factors reflective of these dimensions.

Materials and Methods

Setting and Participants

The sample included 342 subjects from five treatment outcome studies from the Rutgers University Alcohol Research Center (ARC) with lifetime or current (i.e., last 6 months) *DSM-III-R* alcohol abuse (4%) or dependence (96%), with or without a comorbid SUD (see Epstein et al., 2002 for a detailed description of all 342 subjects). In efforts to identify and limit influence of outliers, we reduced the sample to 281 participants based on boxplots and Mahalanobis distance of the 17 type A/B indicators. The remaining sample was 24.2% women, with a mean age of 42 years ($SD = 13.64$; range 18-79 years). The majority identified as Caucasian (67%) or African American (24.2%). A total of 84.3% completed at least a high school degree with 52.2% employed full or part-time.

Measures

Demographics.

A self-report questionnaire assessed basic demographic information, including: age, gender, ethnicity, and marital, educational, and employment status.

Substance Use.

The *Structured Clinical Interview for DSM-III-R* (SCID; Spitzer et al., 1990) assessed current (past 6-months) or lifetime abuse and dependence (inter-rater reliability: alcohol abuse/dependence = 0.75; substance abuse/dependence = 0.84). The Time-Line-Follow-Back (TLFB; Sobell et al., 1980) measured the frequency of alcohol and substance use for the 6 months prior to baseline, with the exception of 107 subjects at two sites, for whom a 3-month period was queried (see Epstein et al., 2002). Frequency data from the TLFB have demonstrated good convergence with other measures (e.g., $r = 0.79$; Maisto et al., 1982), and high rates of test-retest reliability for alcohol and substances (Sacks et al., 2003; Sobell et al., 1979). The Rutgers Consequence of Use questionnaire (RCU; Rhines et al., 1997), a scale with

established internal consistency ($\alpha = 0.94$), quantified alcohol and drug consequences. The Michigan Alcoholism Screening Test- Brief Version (MAST-B; Pokorny et al., 1972) measured lifetime severity of alcoholism with a weighted sum reflecting lifetime consequences. The 10 items of the abbreviated version are highly correlated with the full 25-item scale ($r = 0.95-0.99$). Treatment history was assessed using a structured interview created for the Alcohol Research Center to yield frequency, type, and duration of all inpatient and outpatient episodes of treatment for alcohol and/or drug problems.

Family History.

The Family History Research Diagnostic Criteria (FHRDC; Andreason et al., 1977) interview was administered to the proband to obtain data on family members' alcohol use disorders. Percent of first-degree relatives affected by an alcohol use disorder was calculated for each subject, correcting for relatives about whom the proband had no information.

Psychopathology and Personality.

The SCID-I and SCID-II (Spitzer et al., 1990) assessed current and lifetime Axis I and II disorders (mean kappa for this sample= 0.87; Epstein et al., 2002). The Hyperkinesis/Minimal Brain Dysfunction Scale (HK/MBD; Tarter et al., 1977) is a 50-item, retrospective, self-report checklist of behavioral symptoms prior to age 12 with a total score and four subscales (Alterman & McLellan, 1986) and good test-retest reliability ($r = 0.93-0.95$ for a 7-week interval) and significant correlations with parental or sibling observations ($r = 0.47$; De Obaldia & Parsons, 1984). The NEO Personality Inventory- Revised (NEO-PI-R; Costa & McCrae, 1992) is a 240 item self-report inventory measuring five domains of normal adult personality: Neuroticism, Extroversion, Openness, Agreeableness, and Conscientiousness, and 30 facets of personality such as depression or anxiety. Internal consistency for the domains range from 0.86 to 0.92, and three month test-retest reliability ranges from 0.75-0.83.

Procedures

Assessment.

Each participant was recruited and assessed at ARC after detoxification as close to treatment entry as possible. After providing informed consent, participants completed baseline interviews and self-report measures described above. Interviewers were trained research assistants (see Morgenstern et al., 1998; Morgenstern et al., 1997).

Variables to Derive the A/B model.

As in other studies of A/B typology, measures matched Babor et al.'s (1992) original variables as closely as possible. Table 1 describes operationalization of the 17 variables for the modeling and the sample means and standard deviations.

Statistical Analyses

Dimensional models.

Exploratory factor analysis (EFA) with a robust maximum likelihood estimator first established a common dimensional structure to the data, which were subsequently followed by a series of latent trait (LTA) structure equation models. We chose this confirmatory method to estimate comparative fit of specific models for comparison to competing models and the two-parameter model because of its use in previous AUD studies. Two-parameter models yield an intercept (α) and slope parameters (β) linking the latent trait to the type A/B item scores. These parameters are similar to the (α) difficulty and (β) discrimination parameters used to link the (θ) to the observed responses in recent dimensional evaluations of *DSM* criteria (e.g., Saha et al., 2006).

Categorical models.

Estimation of subtypes was conducted via two statistical procedures. *K*-means cluster analyses (using SPSS 14.0) sorted individuals into groups based on a calculated Euclidean distance defined by means on each indicator variable (Aldenderfer & Blashfield, 1984). As in

more modern categorical modeling approaches (e.g., latent class analysis), this methodology makes no assumptions about local independence, or the statistical assumption that the estimated variable (i.e., alcohol subtype) is responsible for all of the shared variability between indicators (i.e., covariation between 17 type A/B indicators). Along with cluster analyses, latent class analyses (LCAs) were conducted using *Mplus* version 7.1 (Muthén & Muthén, 1998-2015), which uses the specification of a nominal latent variable (i.e., alcohol subtype) to explain the variability in a set of observed variables (type A/B indicators). Local independence is assumed with LCA and a maximum likelihood estimator provided a range of traditional goodness of fit indicators. This methodology accounts for measurement error in the model, significance tests for superiority of nested models using bootstrapped chi square tests, and a range of goodness of fit statistics available to compare competing models (Nylund et al., 2007).

Factor Mixture Models.

Hybrid models were estimated using *Mplus* version 7.1, which allows for the simultaneous estimation of latent dimensions and latent subtypes (Lubke & Muthén, 2005). Models were evaluated using AIC, BIC, Entropy, and bootstrap adjust likelihood ratio tests for comparing nested models. Lower AIC and BIC and higher entropy indicate better model fit. Measurement invariance was assumed between classes (i.e., factor loadings and intercepts are equal among subtypes). This restriction can be relaxed, but results in a larger number of estimated parameters which can greatly reduce power (Lubke & Neale, 2006). The dimensions provide a source of systematic variation within each subgroup, thus relaxing the assumption of local independence.

Missing data.

Missing data were replaced via a missing-at-random (MAR) function using maximum likelihood method with an expectation maximization algorithm. Missing data is allowed to vary as a function of covariates but covariates cannot have any missing data. For 6 and 12 month

follow-up data, 17.1% to 31.3% of data were missing depending on the outcome variable.

Subjected to Little's test of missing completely at random (MCAR) using matrix with all 17 Type A/B predictors, covariates, and outcome variables $\chi^2(270) = 262.38, p = .62$, indicating an appropriate assumption of MAR.

Results

Dimensional Models

Factor Analyses.

Type A/B categorical models were first tested using the original 17 A/B indicators. Box plots and Mahalanobis distance were used to eliminate multivariate outliers, leaving 281 participants for model testing. Only seven respondents endorsed benzodiazepine use and problems with model convergence complicated estimation with all 17 indicators. Thus, benzodiazepine use was added to drug use to simplify the model and aid in convergence. After this merge, the remaining 16 variables were subjected to exploratory factor analysis and all subsequent analyses. A two factor model provided adequate fit to the data, with two factors containing Eigenvalues above 1.0 (4.22 and 2.08 respectively). The root mean square of residual for the two factor model was 0.039. We also examined scree plots to confirm that additional factors offered little new information with minimal variance explained. Thus, factor mixture analysis (FMA) models were conducted using both 1-factor and 2-factor solutions.

Factor 1 was labeled as drinking severity/externalizing psychopathology because a majority of the heavy loading items were traditional alcohol severity indicators such as mean drinks per drinking day, dependence severity, and MAST total score, and for antisocial traits (low conscientiousness and antisocial personality; see Table 2). The second factor was labeled internalizing psychopathology because the heaviest loading items were depression and anxiety measures and there were weak loadings for the drinking severity items. To support our use of

Varimax rotations, we also used Geomin rotations and found consistent results with evidence of only a weak correlation between factors ($r = 0.22$, $SE = 0.13$).

Categorical Models

Latent Class Analyses.

Tables 3 and 4 show the 5-class LCA results and model fit for 2-5 class LCA models, respectively. A 5-class model was the best fitting, although the 2-class model provided the most parsimonious option, it did not survive BLRTs for nested models. Increasing the number of classes across models yielded increased entropy, lowered AIC and BIC and significantly better fit by BLRT suggesting reliable evidence that increasing classes yielded a better fitting model. The 5-class model suggests two larger classes (1 and 4) that roughly correspond to types A and B. Two of the remaining classes (2 and 5) deviate most clearly by greater antisocial symptoms and drug use from larger type A (class 2) and type B (class 5) classes. Class 3 is most closely aligned with type A, but reports more dependence symptoms despite late age of onset.

Hybrid Models

Factor Mixture Models.

A different pattern of results emerged from the 1-factor FMA models when the single factor was constrained to be invariant across latent classes. Adding a latent dimension reduced enough heterogeneity to yield only 2-classes, which largely reflected the type A/B distinction. However, 2-factor FMA models showed a slightly different pattern with the 3-class model proving the best fit overall and fit the data and significantly better than the 2-class model [$\chi^2(4) = 79.91$, $p < .001$]. Comparisons between models suggested that FMA models were generally superior to LCA and LTA models in terms of loglikelihood value, AIC, and BIC (see Table 4). Of the FMA models, the 2-factor model provided the best fit to the data. The superiority of the

2-factor model is not surprising considering the results of the EFA and LTA, which suggested a multidimensional structure to the data (see Table 5).

To determine if fit could be improved by removing the between class parameter constraints, a second model was estimated. Results indicated removing parameter constraints slightly improved model fit ($-2LL = -5127.003$, $AIC = 9387.22$, $BIC = 9639.012$, $Entropy = .890$), but this difference was not significant [$\chi^2(16) = 2.780$, $p = 1.0$]. The same pattern was true when individual items were evaluated for invariance, with evidence largely suggesting factorial invariance for these 16 indicators. Thus, a 2-factor, 3-class FMA model with equality constraints placed on parameter estimates provided the best fit and suggested the 16 indicators are measuring the same continuous latent dimensions (drinking severity/externalizing psychopathology and internalizing psychopathology) across classes.

Table 5 reports the parameter estimates for the best fitting hybrid model, with slopes representing the strength of the relationship between the individual indicator and the latent construct. We conceptualized factor 1 as a drinking severity/externalizing, and factor 2 as an internalizing psychopathology factor to be consistent with the EFA/LTA results. Compared to factor 2, the highest factor loadings for factor 1 were heavy drinking, mean drinks, dependence severity, relief drinking, physical conditions related to alcohol use, and antisocial personality. Conversely, the highest loadings for factor 2 were depression and anxiety. The slopes can be interpreted as the amount of change in latent indicator (e.g., age of first drinking) per one unit increase in the latent factor, controlling for the other latent dimension and latent class. For example, drinkers began drinking about 0.8 years earlier for every one unit increase in latent externalizing/drinking severity, controlling for the effects of latent class and latent psychopathology on age of first drink.

To further clarify differences between classes, model estimated means are reported in Table 6. Class 1, termed “A” since it is similar to Type A, appears to have later age of onset of alcohol problem, and lower indicators of severity for AUD and drug use, and for both internalizing and externalizing psychopathology. Class 2, called “B1” since it appears to be one grouping within the classic Type B subtype, individuals had elevated family history, highest scores on childhood disorders, and moderate levels of externalizing risk factors. Class 2 scored highest on indicators of alcohol severity (years heavy drinking, relief drinking, medical/physical consequences) and internalizing psychopathology (anxiety and depression). Conversely, Class 3 (or “B2”) had more risk factors (family history, low conscientiousness, early age of onset), high recent use of alcohol and drugs, recent and lifetime social consequences, and externalizing psychopathology (antisocial personality disorder). Class 2 (“B1”) was strongly related to the latent dimension of internalizing psychopathology, while Class 3 (“B2”) was most strongly related to the latent dimension of drinking severity/externalizing psychopathology. The relationship between severity/externalizing psychopathology and internalizing psychopathology was ($\beta = 0.001$, $SE = 0.016$), suggesting these latent dimensions, when accounting for subtype, were distinct and likely to be independent sources of between-subject variation.

Factor Mixture Model Validation.

To examine concurrent validity of the three classes, models were estimated with demographic variables as covariates. Tables 7 and 8 summarize the relationships between demographics, latent class, and latent dimension. Females were significantly more likely to be in Class 2 (“B1”), with increased odds of about 10% relative to Class 3 (“B2”) and those who completed some college were significantly less likely to be in Class 1 (“A”), with decreased odds of about 10% relative to Class 3. No other significant differences on demographic variables were observed. Being female was associated with significantly lower latent drinking

severity and significantly more comorbid psychopathology; having some college education and being married was associated with lower drinking severity and psychopathology.

Tables 9 and 10 report the predictive effects of the model on 12-month drinking and drug use outcomes. A separate model, controlling for baseline level of drinking outcome, examined each drinking variable to maximize interpretation using a latent drinking outcome variable defined by the outcomes listed in these tables. The effects of latent class were estimated using posterior probability assignments for individual cases¹. Class 1 (type A) had significantly better outcomes and Class 3 (Type B2) demonstrated the worst outcomes. The unique effects of latent severity and psychopathology were also significant. Both dimensions were associated with worse drinking and drug use outcomes over 12-months. However, a significantly stronger relationship was observed between latent severity/externalizing dimension and poor drinking outcomes (mean drinks per drinking day, days before first drink, and days before first heavy drink) than for the internalizing dimension, whereas the relationship between latent internalizing psychopathology was associated with significantly worse drug use outcomes (number of illegal drug uses). These effects were invariant across groups, suggesting a stable effect of each dimension despite mean differences in each factor across three subgroups.

Discussion

This study examined the latent framework of the type A/B classification system and represents an important progression in the evolution of subtyping research because of the unique framework that incorporates several parallel theories of AUD psychopathology.

Classification of AUDs has been approached from the perspective of *DSM-IV* and *DSM-5* criteria (e.g., Harford & Muthén, 2001; Harford et al., 2009; Shmulewitz D, Greene ER, &

¹To our knowledge, model based prediction using latent nominal variables as the predictor is not possible. Posterior assignment was chosen because it is the easiest to interpret, although results using class probability estimates as the independent variable yielded similar results.

Hasin D, 2015), drinking severity (Helzer et al., 2007;), and internalizing—externalizing psychopathology (Jenkins et al., 2011). Each perspective offers value for understanding the heterogeneity in drinking populations and identifying those with the greatest clinical risk, and our data suggest interesting options for synthesizing these nosological frameworks.

Strict categorical models (i.e., local independence of indicators within groups) suggested significant heterogeneity in this community sample, with upwards of five distinct subgroups. Multi-dimensional models offered more parsimonious quantification of this population heterogeneity in both LTA and FMA approaches. The multidimensional nature was largely supportive of two dimensions approximating drinking severity/externalizing psychopathology and internalizing psychopathology. Within the FMA models, 3-classes appeared to capture the type A and split type B, with two separate groups reflecting a group “B1” with greater internalizing pathology and a group “B2” with greater externalizing pathology, with concurrent and predictive validity. Thus, individual differences can be explained by an integrated model including drinking severity/externalizing pathology and internalizing psychopathology and this model offers the most parsimonious explanation of AUD heterogeneity.

Conceptualizations of AUD pathology based on comorbid personality constructs (e.g., antisocial traits (Morgenstern et al., 1997) or comorbid axis I psychopathology (e.g., internalizing vs. externalizing (Eaton et al., 2011) have also offered some compelling explanations for the individual differences among drinkers. Prospective studies suggest 3-class models differ in these symptoms over time with high externalizers having earlier onset and adult onset AUDs having greater internalizing pathology (Meier et al., 2013). Greater internalizing and externalizing psychopathology increases odds for alcohol dependence, with comorbidity occurring along both of these dimensions carrying the highest risk (Dawson et al., 2011). The results from our study provide evidence for a slightly more differentiated model

whereby the elevations along both dimensions were found among the type B1 (Class 2) and B2 (Class 3) subtypes compared to type A (Class 1), but that B1 and B2 subtypes were characterized by higher values on either a drinking severity/externalizing dimension or internalizing dimension. Specifically, the antisocial traits (i.e. Class 3, or B2 subtype) captured in this study were most strongly associated with the externalizing/drinking severity dimension, whereas Class 2, or B1 subtype was more strongly associated with an internalizing (depression and anxiety) dimension (see Table 8). This finding is not surprising given the strong relationship between antisocial behavioral syndromes and drinking in both men and women (Goldstein et al., 2007).

Although men and women were in all three subtypes, type B1 (Class 2) had a higher percentage of women and higher scores on the latent internalizing pathology dimension.

Emerging evidence suggests that gender differences in latent internalizing and externalizing pathology explains much of the sex differences in axis I psychopathology (Kramer et al., 2008). Women are theorized to have different AUD risks factors and trajectories than men (Epstein et al., 2007) including higher percentages of anxiety and depression in drinking samples (Greenfield et al., 2010). Data from this study are consistent with indications of a key role for anxiety and depression in the severity of AUD for women, and provide a basis for future analyses testing this hybrid model separately by gender.

An increasing number of hybrid models have support (e.g., Eaton et al., 2012), but focus on modeling variability from the existing *DSM* criteria or patterns of comorbidity. To our knowledge, this study is the first to incorporate a diverse set of premorbid risk factors, substance specific indicators, and comorbid psychiatric symptoms into a hybrid model for AUDs and provide a nosological framework with the potential to cut across personality and axis I comorbidity. Consistent with multi-factorial etiological vision of AUD nosology described by Kendler (2011), our findings present an integration of potential explanatory

factors for AUDs. Eaton, Rodriguez-Seij, Carragher, and Krueger (2015) cited the strong evidence for internalizing-externalizing structure to psychopathology to argue that this structure could be used to understand heterogeneity within clinical groups. Most germane to this study, gender differences in latent internalizing-externalizing supports the likelihood that men and women express behaviors (e.g., problem drinking) via different latent psychopathology factors (Eaton et al., 2012). However, the implications for *DSM* or other classifications systems are not entirely clear. Helzer et al. (2007) argue that dimensional systems need to anchor their dimensionality in categorical distinctions. The hybrid model estimated with type A/B indicators satisfies this recommendation; however, it does not rely on existing systems like the newly developed severity scale embedded in *DSM-5* AUD criteria (Fazzino et al., 2014). Comparisons of the type A/B FMA with a *DSM* based classification would provide some interesting insights into the generalizability of this structure across indicator sets.

Data from the current study also provide predictive validity supporting the model. The identified subtypes include one class (Class 1) with low scores across the type A/B indicators, a second class (Class 2, or B1) with the greatest amount of anxiety, depression, and relief drinking, and a third class (Class 3, or B2) with the greatest degree of antisocial traits, drug and alcohol use, and consequences from their drinking and drug use. The latter group engaged in the greatest amount of drinking and drug use in the 12 months following treatment and began drinking on average about a month before those in the low risk group. Variability within these groups was also explained by the latent dimensions of drinking severity and psychopathology. The latter finding suggests that there are individual differences between members of these groups that defined by level of psychopathology or drinking severity that is also uniquely predictive of drinking and drug use outcomes.

There are a number of limitations to the current study. Our sample size is modest for estimating factor mixture models, which limited our ability to examine metric invariance across groups in considerable detail and to detect less prevalent subgroups (Lubke & Neale, 2008), and also contains a relatively small proportion of females. Identifying intercept differences between latent classes will be a necessary step in replication of this finding with a larger sample that has a more balanced gender distribution. Additionally, the prospective validators were specific to drinking and drug use. It will be important to examine these prospective effects on depression, anxiety, and antisocial traits to establish the reliability of the identified factors over time, and evaluate the clinical utility of the scheme given the large number of measures needed for subtyping. Although the internalizing-externalizing dimension appears to be stable (Eaton, Krueger, & Oltmanns, 2011; Hicks et al., 2007), there is also evidence of a general psychopathology factor that contributes to this bifactor structure (Waldman, Poore, van Hulle, Rathouz & Lahey, 2016) and it is possible that our results were influenced by attrition. Finally, the sample used in this study was recruited from community treatment settings and it will be important to examine this model in non-treatment seeking individuals and across different types of treatment settings to determine the limits to generalizability of these results.

In summary, the data from this study revealed distinct dimensional aspects of the type A/B alcoholic model. These dimensions are consistent with broader models of psychopathology, specifically the internalizing—externalizing dimensions (Levy, 2010) that have been used to identify variability within other diagnostic categories and link specific and nonspecific genetic and environmental risks to psychopathology (Tackett et al., 2013; Eaton et al., 2015). Varying along these dimensions, three subpopulations emerged marked by a lower risk type A group, a primarily internalizing type B1, and primarily drinking severity/externalizing type B2 group. The two type B subgroups varied by gender which may

provide a model for examining gender differences in AUD severity and provide a basis for examining other sources of heterogeneity such as genetic risk or moderating effects on treatment.

References

- Aldenderfer MS, Blashfield RK (1984) Cluster Analysis. Sage Publications, Newbury Park, CA.
- Alterman AI, McLellan AT (1986) A factor-analytic study of Tarter's "hyperactivity-MBD" questionnaire. *Addict Behav* 11:287-294.
- Andreasen NC, Endicott J, Spitzer RL, Winokur G (1977) The family history method using diagnostic criteria: Reliability and validity. *Arch Gen Psychiatry* 34:1229-1235.
- American Psychological Association (1987) Diagnostic and Statistical Manual of Mental Disorders, 3rd ed. revised. Author, Washington, D.C.
- American Psychological Association (2000) Diagnostic and Statistical Manual of Mental Disorders, 4th ed. text revision. Author, Washington, D.C.
- Babor TF, Caetano R (2006) Subtypes of substance dependence and abuse: implications for diagnostic classification and empirical research. *Addiction* 101 Suppl 1:104-110.
- Babor TF, Hofmann M, DelBoca FK, Hesselbrock V, Meyer RE, Dolinsky ZS, Rounsaville B (1992) Types of alcoholics, I. Evidence for an empirically derived typology based on indicators of vulnerability and severity. *Arch Gen Psychiatry* 49:599-608.
- Beseler CL, Taylor LA, Kraemer DT, & Leeman RF. (2012). A latent class analysis of DSM-IV alcohol use disorder criteria and binge drinking in undergraduates. *Alcoholism: Clinical and Experimental Research*, 36: 153–161.
- Brown J, Babor TF, Litt MD, Kranzler HR (1994) The Type A/Type B distinction - subtyping alcoholics according to indicators of vulnerability and severity, in Types of

Alcoholics: Evidence from Clinical, Experimental, and Genetic Research (Babor TF, Hesselbrock V, Meyer RE, Shoemaker W eds), pp 23-33. Annals of the New York Academy of Sciences, New York.

Casey M, Adamson G, Stringer M (2013). Empirical derived AUD subtypes in the US general population: a latent class analysis. *Addict Behav* 38(11): 2782-2786.

Carpenter KM, Hasin DS (2001) Reliability and discriminative validity of the Type I/II and Type A/B alcoholic subtype classification in untreated problem drinkers: A test of the Apollonian-Dionysian hypothesis. *Drug Alcohol Depend* 63:51-67.

Carpenter KM, Liu X, Hasin DS (2006) The type A-type B classification in a community sample of problem drinkers: Structural and predictive validity. *Addict Behav* 31:15-30.

Chan YF, Dennis ML, Funk RR (2008) Prevalence and comorbidity of major internalizing and externalizing problems among adolescents and adults presenting to substance abuse treatment. *J Subst Abuse Treat* 34:14-24.

Costa PT, McCrae RR (1992) Revised NEO Personality Inventory and NEO Five-Factor Inventory: Professional Manual. Psychological Assessment Resources, Odessa, FL.

Dawson DA, Goldstein RB, Moss HB, Li TK, Grant BF (2011) Gender differences in the relationship of internalizing and externalizing psychopathology to alcohol dependence: likelihood, expression and course. *Drug Alcohol Depend* 112:9-17.

De Obaldia, R., Parsons, O. A., 1984. Relationship of neuropsychological performance to primary alcoholism and self-reported symptoms of childhood minimal brain dysfunction. *J Stud Alcohol.*, 45, 386-392.

Del Boca FK, Hesselbrock MN (1996) Gender and alcoholic subtypes. *Alcohol Health Res World* 20:56-62.

Eaton NR, Krueger RF, Oltmanns TF (2011) Aging and the structure and long-term stability

of the internalizing spectrum of personality and psychopathology. *Psychol Aging* 26:987–993.

Eaton NR, Keyes KM, Krueger RF, Balsis S, Skodol AE, Markon KE, Grant BF, Hasin DS (2012) An invariant dimensional liability model of gender differences in mental disorder prevalence: Evidence from a national sample. *J Abnorm Psychol* 121:282-288.

Eaton NR, Rodriguez-Seijas C, Carragher N, Krueger RF (2015) Transdiagnostic factors of psychopathology and substance use disorders: a review. *Soc Psychiatry Psychiatr Epidemiol* 50:171-182.

Epstein EE, Fischer-Elber K, Al-Otaiba Z (2007) Women, aging, and alcohol use disorders. *J Women Aging* 19:31-48.

Epstein EE, Labouvie E, McCrady BS, Jensen NK, Hayaki J (2002) A multi-site study of alcohol subtypes: classification and overlap of unidimensional and multi-dimensional typologies. *Addiction* 97:1041-1053.

Fazzino TL, Rose GL, Burt KB, Helzer JE (2014) A test of the DSM-5 severity scale for alcohol use disorder. *Drug Alcohol Depend* 141:29-43.

Goldstein RB, Dawson DA, Saha TD, Ruan WJ, Compton WM, Grant BF (2007) Antisocial behavioral syndromes and DSM-IV alcohol use disorders: results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Alcohol Clin Exp Res* 31:814-828.

Greenfield SF, Back SE, Lawson K, Brady KT (2010) Substance abuse in women. *Psychiatr Clin North Am* 33:339-355.

Harford TC, Muthén BO (2001) The dimensionality of alcohol abuse and dependence: a multivariate analysis of DSM-IV symptom items in the National Longitudinal Survey of Youth. *J Stud Alcohol* 62:150-157.

Harford TC, Yi HY, Faden VB, Chen CM (2009) The dimensionality of DSM-IV alcohol use disorders among adolescent and adult drinkers and symptom patterns by age, gender, and race/ethnicity. *Alcohol Clin Exp Res* 33:868-878.

Helzer JE, Bucholz KK, Gossop M (2007) A dimensional option for the diagnosis of substance dependence in DSM-V. *Int J Methods Psychiatric Research* 16 Suppl 1:S24-33.

Hicks BM, Blonigen DM, Kramer MD, Krueger RF, Patrick CJ, Iacono WG, McGue M (2007) Gender differences and developmental change in externalizing disorders from late adolescence to early adulthood: a longitudinal twin study. *J Abnorm Psychol* 116:433–447.

Hildebrandt T, Langenbucher JW, Carr SJ, Sanjuan P (2007) Modeling population heterogeneity in appearance- and performance-enhancing drug (APED) use: applications of mixture modeling in 400 regular APED users. *J Abnorm Psychol* 116:717-733.

Jenkins MB, Agrawal A, Lynskey MT, Nelson EC, Madden PA, Bucholz KK, Heath AC (2011) Correlates of alcohol abuse/dependence in early-onset alcohol-using women. *Am J Addiction* 20:429-434.

Ko JY, Martins SS, Kuramoto SJ, Chilcoat HD. (2010). Patterns of alcohol-dependence symptoms using a latent empirical approach: Associations with treatment usage and other correlates. *J of Stud on Alcohol Drug* 71: 870–878.

Kendler KS (2011) Levels of explanation in psychiatric and substance use disorders: implications for the development of an etiologically based nosology. *Mol Psychiatry* 17:11-21.

Kramer MD, Krueger RF, Hicks BM (2008) The role of internalizing and externalizing liability factors in accounting for gender differences in the prevalence of common

psychopathological syndromes. *Psychol Med* 38:51-61.

Kranzler HR, Burleson JA, Brown J, Babor TF (1996) Fluoxetine treatment seems to reduce the beneficial effects of cognitive-behavioral therapy in type B alcoholics. *Alcohol Clin Exp Res* 20:1534-41.

Krueger RF, Markon KE, Patrick CJ, Iacono WG (2005) Externalizing psychopathology in adulthood: A dimensional-spectrum conceptualization and its implications for DSM-V. *J Abnorm Psychol* 114:537-550.

Krueger RF, Nichol PE, Hicks BM, Markon KE, Patrick CJ, Iacono WG, McGue M (2004) Using Latent Trait Modeling to Conceptualize an Alcohol Problems Continuum. *Psychol Assess* 16:107-119.

Kuo PH, Aggen SH, Prescott CA, Kendler KS, Neale MC (2008) Using a factor mixture modeling approach in alcohol dependence in a general population sample. *Drug Alcohol Depend* 98:105-114.

Levy F (2010) Internalizing versus externalizing comorbidity: neural circuit hypothesis. *Aust N Z J Psychiatry* 44:399-409.

Lubke G, Neale MC (2006) Distinguishing between latent classes and continuous factors: Resolution by maximum likelihood? *Multivariate Behav Res* 41:499-532.

Lubke G, Neale M (2008) Distinguishing between latent classes and continuous factors with categorical outcomes: Class invariance of parameters of factor mixture models. *Multivariate Behav Res* 43:592-620.

Lubke GH, Muthén B (2005) Investigating population heterogeneity with factor mixture models. *Psychol Methods* 10:21-39.

McGue M, Slutske W, Taylor J, Iacono WG (1997) Personality and substance use disorders: I. Effects of gender and alcoholism subtype. *Alcohol Clin Exp Res* 21:513-520.

Meier MH, Caspi A, Houts R, Slutske WS, Harrington H, Jackson KM, Belksy DW, Poulton

R, Moffitt TE (2013). Prospective developmental subtypes of alcohol dependence from age 18 to 32: implications for nosology, etiology, and intervention. *Dev Psychopathol* 25(3): 785-800.

Morgenstern J, Kahler CW, Epstein E (1998) Do treatment process factors mediate the relationship between Type A-Type B and outcome in 12-Step oriented substance abuse treatment? *Addiction* 93:1765-1776.

Morgenstern J, Langenbucher J, Labouvie E, Miller KJ (1997) The comorbidity of alcoholism and personality disorders in a clinical population: prevalence rates and relation to alcohol typology variables. *J Abnorm Psychol* 106:74-84.

Muthén B (2006) Should substance use disorders be considered as categorical or dimensional? *Addiction* 101 Suppl 1:6-16.

Muthén LK, Muthén BO (1998-2015) *Mplus* (Version 7.1). Los Angeles, CA.

Nylund K, Muthén B, Asparouhov T (2007) Deciding on the Number of Classes in Latent Class Analysis and Growth Mixture Modeling. *Struct Equ Modeling* 14:535-569.

Pokorny AD, Miller BA, Kaplan HB (1972) The brief MAST: A shortened version of the Michigan Alcoholism Screening Test. *Am J Psychiatry* 129:342-345.

Ray LA, Kahler CW, Young D, Chelminski I, Zimmerman M (2008) The factor structure and severity of DSM-IV alcohol abuse and dependence symptoms in psychiatric outpatients. *J Stud Alcohol Drugs* 69:496-499.

Rhines KC, McCrady BS, Morgan TJ, Hirsch LS (1997) Integrated assessment of alcohol and drug use: The Rutgers consequences of use questionnaire.

Sacks JA, Drake RE, Williams VF, Banks SM, Herrell JM (2003) Utility of the Time-Line Follow-Back to assess substance use among homeless adults. *J Nerv Ment Dis* 191:145-153.

Saha TD, Chou SP, Grant BF (2006) Toward an alcohol use disorder continuum using item

response theory: results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Psychol Med* 36:931-941.

Saha TD, Stinson FS, Grant BF (2007) The role of alcohol consumption in future classifications of alcohol use disorders. *Drug Alcohol Depend* 89:82-92.

Sanislow CA, Little TD, Ansell EB, Grilo CM, Daversa M, Markowitz JC, Pinto A, Shea MT, Yen S, Skodol AE, Morey LC, Gunderson JG, Zanarini MC, McGlashan TH (2009) Ten-year stability and latent structure of the DSM-IV schizotypal, borderline, avoidant, and obsessive-compulsive personality disorders. *J Abnorm Psychol* 118:507-519.

Schuckit MA, Tipp JE, Smith TL, Shapiro E, Hesselbrock VM, Bucholz KK, Reich T, Nurnberger JI (1995) An evaluation of type A and B alcoholics. *Addiction* 90:1189-1203.

Shmulewitz D, Greene ER, Hasin D (2015) Commonalities and differences across substance use disorders: phenomenological and epidemiological aspects. *Alcohol Clin Exp Res* 39:1878-1900.

Sobell MB, Maisto SA, Sobell LC, Cooper AM, Cooper T, Saunders B (1980) Developing a prototype for evaluating alcohol treatment effectiveness, in *Evaluating alcohol and drug abuse treatment effectiveness: Recent advances* (Sobell LC, Sobell MB, Ward E eds), pp. 129-150, Pergamon, New York.

Spitzer RL, Williams JBW, Gibbon M, First MB (1990) User's guide for the structured clinical interview for DSM-III-R: SCID. American Psychiatric Association, Washington, DC.

Swift W, Slade T, Carragher N, Coffey C, Degenhardt L (2016) Adolescent predictors of a typology of DSM-5 alcohol use disorder symptoms in young adults derived by latent class analysis using data from an Australian cohort study. *J Stud Alcohol*

Drugs 77:757-65.

Tackett JL, Lahey BB, van Hulle C, Waldman I, Krueger RI, Rathouz PJ. (2013) Common genetic influences on negative emotionality and a general psychopathology factor in childhood and adolescence. *J Abnorm Psychol* 122: 1142-53.

Tam, TW, Mulia, N, Schmidt, LA (2014) Applicability of Type A/B alcohol dependence in the general population. *Drug Alcohol Depend* 138:169-76.

Tarter RE, McBride H, Buonpane N, Schneider DU (1977) Differentiation of alcoholics. Childhood history of minimal brain dysfunction, family history, and drinking pattern. *Arch Gen Psychiatry* 34:761-768.

Waldman ID, Poore HE, van Hulle C, Rathouz PJ, Lahey BB (2016). External validity of a hierarchical dimensional model of child and adolescent psychopathology: tests using confirmatory factor analyses and multivariate behavior genetic analyses. *J Abnorm Psych* 125:1053-1066.

Table 1

Measures used to operationalize Type A/B variables

A/B Criteria	M (SD)	Measure
Premorbid Risk Factors		
Familial Alcoholism ^{A<B}	26.47(25.33)	FHRDC: percent first degree relatives with alcohol abuse/dependence
Childhood disorder ^{A<B}	15.03(8.82)	HK/MBD: total score
Conscientiousness ^{A>B}	40.65 (12.54)	NEO-PI R: conscientiousness domain score
Onset Problem Drinking ^{A>B}	24.93 (12.41)	Alcohol SCID: age of onset of DSM-III-R alcohol abuse or dependence
Alcohol and Other		
Substances		
Drinks per drinking day ^{A<B}	10.28(12.55)	TLFB: mean drinks per drinking day in last 3 months prior to treatment

Relief Drinking ^{A<B}	38.7%	Alcohol SCID: item A11 assessing relief drinking in last 6 months
Dependence Severity ^{A<B}	7.21(2.74)	Alcohol SCID: criterion A symptom count (except relief drinking) in last 6 months
Benzodiazepine Use ^{A<B}	5.3%	TLFB: percent of days using benzodiazepines in last 3 months
Drug use ^{A<B}	17.86(38.95)	TLFB: total number of usages of any drug other than benzo. in last 3 Months
Chronicity and Consequences		
Medical Conditions ^{A<B}	36.7%	one or more alcohol-related medical problems in lifetime
Physical Consequences ^{A<B}	16.21(9.75)	RCU: average frequency of 11 physical consequences in last 6 months
Social Consequences ^{A<B}	13.47(10.18)	RCU: average frequency of 13 social consequences in last 6 months
Lifetime Severity ^{A<B}	17.86(7.97)	MAST-B: total weighted score

Years Heavy Drinking ^{A<B}	17.11(18.88)	Alcohol SCID: current age minus age of onset
Psychiatric Symptoms		
Depression ^{A<B}	5.58(4.85)	SCID I: criterion A symptom count for current or lifetime major depression & dysthymia
Antisocial Personality ^{A<B}	2.11(3.93)	SCID II: symptom count for conduct disorder and adult ASP
Anxiety ^{A<B}	56.68(9.78)	NEO-PI R: anxiety facet score from Neuroticism domain

ASP= Antisocial Personality; DSM= Diagnostic and Statistical Manual of Mental Disorders; FHRDC=Family History Research Diagnostic Criteria; HK/MBD= Hyperkinesia/Minimal Brain Dysfunction Scale; MAST-B=Michigan Alcoholism Screening Test- Brief Version; NEO-PI-R= NEO Personality Inventory- Revised; RCU= Rutgers Consequence of Use Questionnaire; SCID=Structured Clinical Interview for DSM-IV; TLFB= Time Line Follow-Back

Table 2

Factor Loadings for 2-Factor Solution (N = 281)

Type A/B Indicator	Factor 1 (Drinking Severity/Externalizing Psychopathology)	Factor 2 (Internalizing Psychopathology)
1. Familial Alcoholism	0.208	0.156
2. Childhood Disorder	0.580	0.523
3. Conscientiousness	-0.527	-0.213
4. Age of Onset	-0.005	-0.211
5. Mean Drinks	0.527	0.046
6. Relief Drinking ^a	0.750	-0.115
7. Dependence Severity	0.881	-0.023
8. Drug use	-0.133	0.302
9. Medical Consequences ^a	0.643	-0.030
10. Physical Consequences	0.796	0.066
11. Social Consequences	0.650	0.223
12. Lifetime severity	0.588	0.434
13. Years of Heavy Drinking	0.399	0.220
14. Antisocial Personality	0.431	0.317
15. Depression	0.242	0.721
16. Anxiety	0.134	0.688

^a Dichotomous item estimated using logistic scale. Varimax rotations reported. Robust maximum likelihood estimator used.

Accepted Article

Table 3

Model Estimated Means and Standard Deviations of 5-Class Solution

	Class 1 (n=91)	Class 2 (n=30)	Class 3 (n=35)	Class 4 (n=113)	Class 5 (n=12)
1. Familial Alcoholism	22.86(3.09)	27.18(5.27)	12.24(2.99)	33.59(3.42)	26.23(24.54)
2. Childhood Disorder	12.72(0.94)	21.91(2.39)	10.04(1.21)	15.63(2.33)	21.76(13.70)
3. Conscientiousness	45.12(1.38)	32.22(2.92)	47.20(2.38)	38.21(1.39)	35.93(5.60)
4. Age of Onset	22.49(0.97)	17.02(0.72)	51.29 (2.64)	21.03(0.83)	22.49(5.13)
5. Mean Drinks	5.34(0.79)	6.85(1.33)	7.05(0.90)	11.91 (0.98)	50.47(26.25)
6. Relief Drinking	9.6%	0%	32.8%	69.2%	100%
7. Dependence Severity	5.23(0.33)	5.33(0.73)	6.29(0.42)	9.43(0.19)	10.33(0.45)
8. Square Root Drug Use	.989(.34)	8.67(0.79)	0.08(0.08)	1.50(0.73)	7.60(2.16)
9. Medical Consequences	24.8%	27.2%	31.3%	44.7%	35.6%
10. Physical Consequences	8.90(0.90)	15.54(2.32)	9.52(1.17)	22.91(1.05)	28.00(1.88)
11. Social Consequences	6.88(0.82)	16.38(2.33)	5.28(1.07)	19.38(1.33)	22.05(5.21)
12. Lifetime severity	12.27(1.27)	17.03(1.72)	14.71(1.23)	11.74(0.70)	22.86(3.94)
13. Years of Heavy Drinking	19.89(1.56)	11.39(1.52)	8.22(1.27)	19.55(0.99)	15.92(6.50)
14. Antisocial Personality	0.04(0.03)	.29(0.12)	0.06(0.04)	0.08(0.08)	0.37(.48)
15. Depression	3.43(0.47)	7.53(0.95)	4.49(0.82)	6.45(0.73)	10.66(1.64)
16. Anxiety	51.84(1.01)	60.89(2.91)	53.73(1.88)	59.41(1.42)	62.10(3.60)

Note. Drug use is combination of benzodiazepine and other drug use. We used square root transformations of this variable in model estimation of 5-class solution to facilitate convergence speed.

Table 4

Summary of Model Fit Statistics and Classification Quality for Dimensional, Categorical, and Mixed Models of Alcohol Users.

LCA-Model	H0 -2LL Value	# Parameters	BLRT	AIC	aBIC	Entropy
2-Class	-5344.243	48		10784.487	10958.612	0.846
3-Class	-5331.832	65	24.82**	10738.665	10949.460	0.866
4-Class	-5311.562	82	40.54**	10731.123	10935.843	0.916
5-Class	-5294.688	99	33.75**	10433.376	10803.393	0.905
FMA 1-Factor						
2-class	-5303.612	49		10705.223	10882.977	0.382
3-class	-5303.610	51	.002	10709.223	10894.232	0.422
4-class	-5299.832	53	7.56	10715.423	10910.244	0.542

5-class	-5295.834	55	7.80	10722.323	10915.378	0.601
---------	-----------	----	------	-----------	-----------	-------

FMA 2-Factor

2-class	-5201.693	63		10533.386	10769.181	0.872
---------	-----------	----	--	-----------	-----------	-------

3-class	-5129.783	67	143.82***	9393.566	9637.336	0.896
----------------	------------------	-----------	------------------	-----------------	-----------------	--------------

4-class	-5283.234	71	-306.90	10728.321	10899.328	0.808
---------	-----------	----	---------	-----------	-----------	-------

5-class	-5309.200	75	-51.93	10788.432	10904.232	0.799
---------	-----------	----	--------	-----------	-----------	-------

LTA Model

1-Factor	-5473.866	44		11039.733	11062.198	--
----------	-----------	----	--	-----------	-----------	----

2-Factor	-5354.572	58	207.59***	10833.144	10863.423	--
----------	-----------	----	-----------	-----------	-----------	----

Note. LCA = latent class analysis. FMA = factor mixture analysis. LTA = latent trait analysis. AIC = Akiake Information Criterion. BIC = Bayesian Information. Criterion. ** $p < .01$. *** $p < .001$. aBIC = sample size adjusted BIC. BLRT = bootstrap Lo-Mendel-Rubin Test.

Table 5

Slope and Intercepts for 2-Factor 3-Class Factor Mixture Model

	Externalizing/ drinking severity	Internalizing Psychopathology	Intercept
1. Familial alcoholism	1.350 (0.234)	0.882 (0.388)	0.199 (0.086)
2. Childhood disorder	1.820 (0.416)	1.145 (0.419)	-0.352 (0.091)
3. Conscientiousness	-0.930 (0.301)	-0.387 (0.449)	0.287 (0.098)
4. Age of Onset	-0.812 (0.398)	-1.259 (0.533)	0.346 (0.116)
5. Mean Drinks	2.660 (0.578)	0.480 (0.179)	-0.314 (0.095)
6. Relief Drinking ^a	2.880 (0.054)	-1.327 (0.062)	1.322 (0.381)
7. Dependence Severity	4.674 (0.334)	-0.563 (1.247) ^b	-0.361 (0.147)
8. Drug use	-0.174 (0.094)	1.605 (0.463)	-0.482 (0.053)
9. Medical Consequences ^a	1.429 (0.285)	-3.628 (0.485)	0.803 (0.194)
10. Physical Consequences	2.993 (0.673)	0.063 (0.137)	-0.533 (0.164)
11. Social Consequences	2.417 (0.386)	1.379 (0.914) ^b	-0.499 (0.137)
12. Lifetime severity	2.938 (0.311)	1.285 (0.497)	-0.237 (0.096)
13. Years of Heavy Drinking	-3.396 (0.148)	-2.497 (0.374)	0.009 (0.085) ^b
14. Antisocial Personality	1.885 (0.376)	0.975 (0.477)	-0.361 (0.077)
15. Depression	1.095 (0.364)	3.430 (0.565)	-0.303 (0.091)
16. Anxiety	1.265 (0.689)	2.472 (0.122)	-0.262 (0.097)

Note. ^a = logistic threshold parameter. ^b = not significant at $p < .05$. All other parameters are significant at $p < 0.05$.

Table 6

Model Estimated Indicator Means for 2-Factor 3-Class Factor Mixture Model

Type A/B Indicator	Class 1 (“A”) (n = 133 (16 women))	Class 2 (“B1”) (n = 103 (37 women))	Class 3 (“B2”)(n = 45 (10 women))
1. Familial alcoholism	8.57 (19.16)	26.45 (24.82)	39.20 (30.71)
2. Childhood disorder	13.84 (7.94)	22.61 (8.60)	19.14 (9.75)
3. Conscientiousness	53.80 (6.26)	46.83 (12.35)	40.14 (12.38)
4. Age of Onset	26.48 (13.51)	19.60 (7.63)	18.71 (3.14)
5. Mean Drinks	9.49 (9.16)	10.51 (5.18)	13.14 (6.48)
6. Relief Drinking ^a	11.0%	44.4%	29.3%
7. Dependence Severity	6.58 (2.42)	9.80 (2.86)	7.68 (2.63)
8. Drugs	3.61 (8.06)	19.20 (14.33)	23.63 (22.51)
9. Medical Consequences ^a	16.7%	48.8%	34.5%
10. Physical Consequences	15.64 (8.34)	26.40 (4.87)	22.17 (10.33)
11. Social Consequences	11.05 (9.42)	17.79 (9.42)	25.40 (5.68)
12. Lifetime severity	17.08 (6.32)	19.51 (8.34)	24.60 (2.88)
13. Years of Heavy Drinking	10.40 (6.26)	17.46 (9.80)	14.25 (6.85)
14. Antisocial Personality	1.82 (3.59)	2.80 (3.59)	4.71 (5.53)
15. Depression	4.32 (3.01)	8.34 (2.84)	5.80 (2.86)
16. Anxiety	14.32 (9.07)	51.23 (11.12)	41.03 (12.45)
Latent Severity/Externalizing	0.01 (0.02)	0.12 (0.09)	0.46 (0.03)
Psychopathology			

Latent Internalizing	-0.02 (0.01)	0.46 (0.03)	0.20 (0.05)
Psychopathology			

Note. ^a dichotomous variable, frequency of medical. Standard Deviations reported for indicators, and standard error reported for latent variables.

Table 7

Summary of the Relationships between Demographics and Latent Class

Demographics	Class 1 (“A”) (n = 133)		Class 2 (“B1”) (n = 103)				Class 3 (“B2”)(n = 45)			
	n	%	n	%			n	%		
	(Mean)	(SD)	(Mean)	(SD)	Exp(B)	95% CI	(Mean)	(SD)	Exp(B)	95% CI
Age	(42.8)	(11.7)	(45.7)	(15.2)	1.020	.904-1.137	(34.7)	(9.5)	.900	.767-1.133
Female	16	12.0%	37	35.9%	1.105	1.002-1.208	10	22.2%	1.061	.995-1.127
Married	47	35.3%	41	39.8%	.925	.777-1.073	16	35.6%	.882	.741-1.023
Some College	81	60.9%	60	58.1%	1.010	.916-1.104	22	48.9%	.901	.878-.924
Caucasian	86	64.6%	69	66.9%	1.055	.945-1.165	33	73.3%	1.094	.985-1.203

Note. Posterior probability assignments used to generate sample size counts within each cell.

Table 8.

Regression of Latent Severity/Externalizing and Internalizing Psychopathology on Demographic Predictors

Demographics	Latent Factor 1		Latent Factor 2	
	(Drinking Severity/Externalizing)		(Internalizing Psychopathology)	
	β	SE	β	SE
Age	1.191	0.744	0.231	0.141
Female ^a	-0.132***	0.019	0.157***	0.024
Married ^a	-0.033	0.023	-0.551***	0.036
College Degree ^a	-0.122*	0.046	-0.230***	0.022
Caucasian ^a	0.011	0.029	0.020	0.030

Note. Unstandardized regression coefficients reported. ^adichotomous item

* $p < .05$.

** $p < .01$.

*** $p < .001$.

Table 9

The Effects of Latent Class on 12-month Drinking and Drug Use outcomes.

	Latent Class 1 ("A")		Latent Class 2 ("B1")				Latent Class 3 ("B2")			
12 Month Outcomes	(n = 133)		(n = 103)				(n = 45)			
	Mean	SD	Mean	SD	β	SE	Mean	SD	β	SE
Percent Drinking Days	16.5	30.2	20.5	32.5	4.05	2.96	24.4	32.3	8.13*	3.45
Mean Drinks Per Drinking Day	6.25	4.49	8.86	7.9	2.73	3.44	14.6	13.1	8.168*	3.57
Number Days before first drink	114.4	91.2	113.4	89.1	-1.01	4.43	98.3	90.8	-14.78**	3.99
Number Days before first heavy drink	140.1	88.9	120.1	89.3	-20.45*	7.11	109.9	92.1	-30.98**	7.48
Number Illegal Drug Uses	4.63	35.5	9.11	42.0	4.57	3.88	14.5	37.2	9.01*	3.29
Percent Drug use per day	1.63	10.4	3.54	14.9	1.85	2.44	7.03	18.9	5.03**	1.25
Days until first drug use	170.2	49.1	168.2	52.3	1.99	3.57	150.8	66.5	20.45**	3.92

Note. Unstandardized regression coefficients reported, * $p < .05$, ** $p < .01$, *** $p < .001$.

Table 10

Twelve Month Drinking Outcomes Predicted by Latent Severity and Psychopathology

12 Month Outcomes	Latent Factor 1		Latent Factor 2	
	(Drinking Severity/Externalizing)		(Internalizing Psychopathology)	
	β	SE	β	SE
Drinking Days/Valid Days	0.033**	0.010	0.021*	0.014
Mean Drinks Per Drinking Day _a	1.121**	0.023	0.560**	0.021
#Days before first drink _a	-0.023**	0.004	-0.066**	0.013
#Days before first heavy drink _a	-0.981***	0.019	-0.025*	0.011
#Illegal Drug Uses _a	0.455**	0.111	1.341***	0.156
% Drug use per day	0.234**	0.072	0.411***	0.068
Days until first drug use	-0.178**	0.051	-0.311***	0.057

Note. N = 281 using missing at random. Unstandardized regression coefficients reported. *M*plus model test used to compare mean differences between parameter estimates in each factor across outcome variables. _a = mean difference significantly greater than zero ($\alpha = .01$).

* $p < .05$, ** $p < .01$, *** $p < .001$.