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Pathways to Vulnerability for Alcohol Problem Severity in a Treatment-Seeking Sample

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Abstract

Objectives—The present investigation examined the role of gender, family history of alcohol and drug use disorders, temperament, childhood behavior problems, and adult psychopathology, on adult alcohol use disorder (AUD) severity.

Methods—Structural equation modeling was used to examine multiple etiological pathways to adult alcohol use disorder (AUD) severity. Participants included 335 treatment-seeking males and females with current or lifetime DSM-III-R alcohol dependence (96%) or abuse (4%) enrolled in one of five treatment outcome studies. Extensive assessment at treatment entry used a mixture of retrospective and current self-report.

Results—Results identified two significant paths associated with a latent factor of adult alcohol use disorder severity at entry to treatment. In Path 1, male gender and family history of drug use disorder predicted greater childhood behavior problems, which predicted antisocial personality disorder (ASPD), borderline personality disorder (BPD), and anxiety disorders (ADs), with anxiety disorders leading directly to alcohol use disorder severity. In Path 2, family history of alcohol use disorder predicted difficult temperament in childhood, which predicted borderline personality disorder, major depressive disorder (MDD) and anxiety disorders; both major depressive disorder and anxiety disorders in turn predicted alcohol use disorder severity at treatment entry.

Conclusions—The present findings build on the literature on heterogeneity in developmental risk processes leading to the expression of adult alcohol use disorder symptomology among patients presenting for alcohol use disorder treatment.

Keywords

alcohol use disorders; heterogeneity; risk trajectories; psychological disorders; alcohol use disorders treatment

INTRODUCTION

Despite advances in empirically-based therapies to treat alcohol use disorders (AUDs) over the past 20 years (1–3), typical treatment effect sizes have remained modest, and no single treatment approach has been shown to consistently yield superior outcomes over another (4–6). Treatment development efforts in general have not taken into account heterogeneity in the etiology and maintenance of AUDs (7–9), and thus, broad ranging alcohol treatments may not be applicable to all subtypes of problem drinkers (10, 11). The purpose of the current study is to test multiple pathways of vulnerability for adult alcohol problem severity based on risk factors at three stages of development (see Figure 1) among a heterogeneous sample of treatment-seeking individuals who meet criteria for an AUD. A structural model of heterogeneity in the association between inherited risk factors (male gender and family history of alcohol and drug problems), childhood risk factors (childhood behavior problems, and difficult temperament), adult psychopathology risk factors (depression, anxiety and personality disorders), and alcohol problem severity at entry to AUD treatment is proposed and tested.

Etiological studies examining risk factors for diagnosis of alcohol abuse or dependence have highlighted both the salience of psychiatric disorders with AUDs and the importance of a developmental perspective. In particular, current research on etiology of AUDs points to two broad dimensions of comorbidity as major risk domains underlying development of AUDs: externalizing and/or internalizing psychopathology (12). However, much of the research to date has focused on non-treatment-seeking samples, has targeted AUD diagnosis as a dichotomous endpoint without considering AUD severity, and has not fully addressed the possibility of multiple distinct pathways, that is, heterogeneity in development of the same endpoint disorder.

Background

Inherited Risk Factors—Twin, adoption, and candidate-gene studies have found that there is a strong genetic component of AUDs (13–15). Also, several typologies and risk pathways associated with adult AUD diagnoses have been proposed in the literature; common elements in most of these models are that the inherited traits of male gender and a family history of AUDs significantly increase risk for the development of alcohol use problems and AUDs (7, 8, 16–18).

Childhood Risk Factors—Research has also linked a variety of developmental factors to adult AUDs. Externalizing problems such as "difficult temperament" characterized by emotionality, fussiness, irritability, difficulty soothing, and arrhythmicity in biological functions (19), behavioral under-control characterized by aggression/rebelliousness, drive for stimulation, aversion to routine, as well as lack of constraint (20), and childhood

behavior problems such as attention deficit/hyperactivity, and oppositional defiant disorder, have most notably been associated with family history of AUDs, male gender, and the manifestation of adult AUDs (21–28), as have internalizing characteristics such as anxiety and depression (29–31).

In a seminal study, Jackson et al. (32) followed a sample of 449 male and female non-alcohol dependent freshmen from a large Midwestern university over the course of seven years, half of whom had a paternal history of alcoholism. They found that those with a family history of AUDs scored high on measures of behavioral under-control, and that behavioral under-control and male gender predicted the likelihood of belonging to a group of chronic problem drinkers in adulthood. Wennberg and Bohman (33) found that factors similar to behavioral under-control (higher levels of extraversion and aggression), and lower levels of social reactivity in childhood (age 4) were associated with adult alcohol problems at ages 25 and 36. Using a mixed sample of students and staff from the University of California San Diego, Schuckit and Smith (34), however, found that behavioral under-control in young adulthood on its own did *not* mediate the link between positive family history of alcohol dependence, and risk for adult alcohol-related problems in a sample of adult males with an AUD (12.8% meeting criteria for alcohol dependence, and 8.8% for alcohol abuse in the past 15 years). Rather, their results showed that behavioral under-control in young adulthood was associated with alcohol problems through alcohol expectancies, peer drinking, and coping. Thus, while trajectory-based studies support the presence of multiple distinct risk pathways leading to problematic alcohol use, findings remain inconclusive about which individual-difference domains and factors link early family risk for an AUD to childhood developmental processes, and to later adult alcohol-related risk and diagnosis. Further, most research to date in this area has examined individuals with AUDs who are not in treatment, and therefore, may not accurately characterize the full spectrum of individuals with AUDs.

Adult Risk Factors—In addition to examining childhood risk factors, studies have focused on the associations among family history of an AUD, adult psychopathology, and adult AUDs and alcohol related problems. Family history of AUDs is known to confer high risk for mood disorders, and mood disorders may lead to AUDs (9, 35–37). For instance, Young-Wolff and colleagues' (38) finding that the use of alcohol to improve mood mediated the relationship between depression and AUDs provides support for the “self-medication” hypothesis. The development of AUDs among individuals with mood disorders may in some cases be the result of chronic alcohol use to suppress mood disorder symptomology (39–41).

Similarly, stress response dampening (42) has been suggested as a mechanism explaining the strong association of anxiety disorders and AUDs. Individuals with a positive family history of AUDs have been found to experience greater stress dampening as a result of using alcohol, therefore putting them at greater risk of developing alcohol problems (43–45). Similar effects have been seen in individuals with a family history of anxiety disorders (39, 46), providing further evidence of the shared genetic vulnerability of anxiety disorders and AUDs. In addition, a growing body of literature implicates anxiety sensitivity in the development of AUDs (see 47).

Borderline personality disorder (BPD) though considered an adult disorder because it cannot be formally diagnosed until 18 years of age, nevertheless has its portent in childhood and early adolescence externalizing problems. The temperamental and mental state abnormalities that resemble aspects of the BPD phenotype emerge early before fully manifesting in BPD symptomology in adolescence or adulthood (48, 49). Similarly antisocial personality disorder (ASPD) first emerges as conduct disorder (50) and is formally diagnosed after the age of 18. Both of these disorders are thought to play a causal role in the development of AUDs, particularly because of associated problems of disinhibition and emotion regulation (26, 51). As such, BPD and ASPD both frequently co-occur with AUDs (52). Notably, family history of a substance use disorders is thought to be a predictor of the development of BPD (53), and family history of AUDs has been shown to increase risk for ASPD (54, 55).

The Current Study

Although the bivariate association between the aforementioned factors and AUDs has been well elucidated in the literature, a limited body of research has examined multiple developmental pathways to AUD diagnoses in treatment-seeking problem drinkers by examining inherited, childhood, and family risk factors. Additionally, previous studies have typically relied on dichotomous outcome variables (e.g., AUD diagnosis, yes or no), which fail to capture individual problem severity among those seeking treatment for AUDs. The current study, therefore, extends previous work by exploring multiple pathways of risk factors via distinct externalizing and internalizing childhood and adult problems that are comorbid with AUDs, and are hypothesized here to predict alcohol problem severity in a clinical sample of adults entering treatment for AUDs. Using a sample diverse in age, gender, ethnicity, and socioeconomic status, the present trajectory model sought to examine temperament, childhood behavior problems and adult psychopathology (e.g., Axis I and II diagnosis) as hypothesized mediators of the association between early risk factors (e.g., familial risk and gender), and AUD severity at the time of treatment entry in adulthood. Indicators of alcohol use problem severity included AUD symptom count at treatment entry, degree of alcohol related problems, and alcohol consumption pattern. It was hypothesized that multiple pathways leading to more severe alcohol problems in adulthood would emerge, stemming from gender and early familial risk.

MATERIALS & METHODS

Participants

For a detailed description of study design, measures, and participants see Epstein et al. (7). Briefly, this study reports on data from individuals with a current (within last 6 months), or lifetime Diagnostic and Statistical Manual of Mental Disorders III-R (DSM-III-R; 56) diagnosis of alcohol abuse (4%) or dependence (96%) who participated in one of five treatment outcome studies conducted jointly through the Rutgers University Alcohol Research Center. The sample included participants receiving treatment in both in-and out-patient settings, heterogeneous in terms of gender, age and ethnicity. From a total of 462 individuals who consented to participate in the Alcohol Research Center study, 86 participants completed one third or less of the baseline assessment battery. Of the remaining

participants, alcohol diagnosis information was missing for 19, and 22 participants did not meet criteria for either alcohol abuse or dependence. These individuals were excluded from analyses. As such, the final number of participants used in the present analysis was 335.

Measures

Demographic Information—Age, gender, race, and educational status were collected with a brief self-report questionnaire, and are summarized in Table 1.

Substance Related Problems

The Structured Clinical Interview for DSM-III-R (Alcohol & Other Drugs Section):

The alcohol and drug portions of the SCID-I (57) were administered to assess lifetime and current abuse or dependence on alcohol and other substances. Reported SCID-I inter-rater reliability is high for AUD diagnoses ($\kappa = 0.96$) and other substance use disorders ($\kappa = 0.85$; 58).

The Time-Line Follow-Back Interview (TLFB): The TLFB (59), a calendar-based alcohol consumption measurement instrument, was used to assess percent days drinking (PDD) and drinks per drinking day (DDD) over the six-month period preceding the baseline interview. The TLFB has high test-retest reliability in outpatient samples with $r = +0.85$ (59), as well as high convergent validity as assessed by correlations between drinker and collateral reports with r ranging from $+0.80$ to $+0.88$ (60).

The Michigan Alcoholism Screening Test - Brief Version (MAST-B): The MAST-B (61) is a 22-item screening instrument for lifetime alcohol problems. Each item is rated as either “yes” or “no”, with 1 or 0 points scored for each item. A total score is derived by summing each the items. The MAST-B has high specificity (96.4%) in clinical populations (62).

Family History

Family History Research Diagnostic Criteria (FH-RDC) Interview: Information about family history of alcohol and drug disorders in study participants was collected using the structured widely used FH-RDC interview (63). Percent of first-degree relatives with an alcohol or drug use disorder was calculated for each participant.

Psychopathology

The Structured Clinical Interview for DSM-III-R (SCID-I & II): The full SCID-I interview (64) assessed current and lifetime Axis-I mood and anxiety disorders. To reduce participant burden, only the Cluster-B section of the SCID-II interview (65) was used to assess current or lifetime Axis-II pathology; relevant to the present study were the diagnoses of BPD and ASPD. Test-retest reliability is high for both SCID-I ($\kappa = 0.61$; 66) and SCID-II ($\kappa = 0.75$; 67).

Childhood Behavior Problems

The Hyperkinesia/Minimal Brain Dysfunction Scale (Hk/MBD): The Hk/MBD (24), which uses a 3-point Likert-scale, was used to collect self-report data of childhood behavior

problems. The Hk/MBD yields a total score as well as scores for four subscales: hyperactivity/impulsivity, attention socialization, antisocial behavior, and learning problems. The total score was used in these analyses. Test-retest reliability ($r = +0.93$) and convergent validity ($r = +0.86$) for the Hk/MBD are high (68).

Temperament

The Dimensions of Temperament Survey – Revised (DOTS-R): The DOTS-R (69) was used to assess difficult temperament. The DOTS-R is comprised of 10 subscales: General Activity Level, Sleep Activity Level, Approach-Withdrawal, Flexibility-Rigidity, Mood, Rhythmicity-Sleep, Rhythmicity-Eating, Rhythmicity Daily Habits, Distractibility and Persistence. The DOTS-R has high internal consistency with alphas ranging from 0.62 to 0.84 (70). A difficult temperament index was calculated following the method used by Giancola (71), and Windle and Lerner (69). High scores on the difficult temperament index reflected high activity level, withdrawal, rigidity, distractibility, and low approach, flexibility, mood, rhythmicity, and persistence.

Procedure

Assessment—Participants were recruited, provided informed consent, and completed baseline self-report and interviews at their treatment site. All interviewers were bachelors or masters level research assistants (training procedures have been described in more detail in 72). The measures described above were drawn from a larger battery of measures, completion of which took roughly 4–5 hours at baseline. Participants completed subsequent assessments at 6 months and 12 months post-baseline. Only baseline data were used for the current study.

Analysis

Theoretical Model—Before analyses were conducted variables known to predict problem alcohol use were selected and organized according to a theoretically plausible developmental model of vulnerability. As such, we examined variation in pathways leading from gender and family drug and alcohol problems, to difficult temperament and childhood behavior problems, to adult psychopathology, and finally to adult alcohol use severity and problems at treatment entry. It was hypothesized that multiple pathways leading to more severe alcohol problems in adulthood would emerge, stemming from gender and early family risk (see Figure 1). While much of the past research has focused on these factors as they develop in adolescence or young adulthood in non-clinical, non-treatment-seeking samples, we examined genetic, childhood and adult risk factors as predictors of problem severity in an adult sample seeking treatment for AUDs.

Risk factors were assessed retrospectively using self-report measures, either on dimensions of characteristics such as temperament that have been shown to be apparent as early as infancy and stable across the lifespan (69, 73), or on childhood behavior problems before the age of 12. Genetic risk factors were estimated using a family history interview. Using retrospectively collected self-reported data, the model tested the association among sets of indicators that reflected traits and/or problems that originated at various ages in developmental progression. The term 'stage' is used here to describe the temporal order of

these sets of risk indicators, as they are purported to emerge through the lifespan in the literature.

The variables in “Stage 1” include gender, percent of first-degree relatives with an AUD, and percent of first-degree relatives with a drug use disorder diagnosis, other than alcohol or tobacco. “Stage 2” consists of childhood risk factors, including childhood behavior problems (measured by Hk/MBD) and difficult temperament (measured by DOTS-R). “Stage 3” consists of adult psychopathology risk factors, including Axis I major depressive disorder (MDD) and any anxiety disorder (AD), and the Axis II disorders antisocial personality disorder (ASPD) and borderline personality disorder (BPD). “Stage 4” is the outcome variable of alcohol problems/alcohol use severity at entry to treatment (AUD severity).

While Stages 3 and 4 may in some instances be inverted or temporally equivalent (i.e., reciprocally affecting each other during the same temporal stage), psychopathology precedes alcohol use problems in the current tested model for two reasons. First, the primary focus of the study was to examine a theoretical model of the development of AUD severity, so that is our primary endpoint variable. Second, psychopathology has been found to precede the development of alcohol diagnoses at least for many individuals. The average age of onset of an anxiety disorder is usually earlier than that of an AUD in population-based samples (74). Other evidence suggests that mood disorders are often primary to AUDs (9, 37, 75, 76), and depression has been found to increase the risk of developing an AUD (76). Further, AUDs have been found not to increase risk of developing depressive disorders (77). In terms of personality disorders, they are considered to be primary to AUDs, as they are defined by the DSM 5 (78) as long-standing patterns of behavior having their roots in childhood and adolescence, before the onset of alcohol dependence typically develops. Though this model is theoretically sound, the cross-sectional nature of the data call for testing an alternative model with Stages 3 and 4 flipped. Such a model was tested in addition to the a priori theoretical model.

Measurement Model—Confirmatory factor analysis was conducted to create a single latent construct reflecting alcohol problem severity. The latent construct included current or lifetime alcohol dependence diagnosis (yes or no), number of DSM-III-R AUD symptoms endorsed, PDD, DDD, and MAST-B score. This model was constructed using Mplus 3.0 Structural Equation Modeling software using maximum likelihood imputation and maximum likelihood estimation. Model fit was assessed using the comparative fit index (CFI; .90–.95 = good fit, >.95 = very good fit; 79), the root mean square error of approximation (RMSEA; <.05 = very good fit; 79), the standardized root mean squared residual (SRMR; <.08 = very good fit; Hu & Bentler, 1999), and the relative normed chi-square, calculated as chi-square divided by degrees of freedom (χ^2/df), which accounts for inflated chi-square values in large samples (80). Conservatively, relative normed chi-square values < 2 represent very good fit (81). It should be noted that structural equation models assume a degree of disturbance or error loaded onto each endogenous variable.

Structural Equation Model—Structural equation modeling analyses were also conducted using Mplus 3.0 and estimated using maximum likelihood imputation and estimation. Model fit was again assessed using the CFI, RMSEA, SRMR, and relative normed chi-square

values. The initial model was constructed such that variables from Stage 1 were predictors of those in Stage 2, which were predictors of those variables in Stage 3, which were predictors of those in Stage 4 (see Figure 1). Models were modified by progressively removing non-significant ($p > .10$) paths and including theoretically justified modification index changes in an iterative fashion. Paths from male gender as well as family history of drug use disorders to difficult temperament were trimmed because of null associations in iterations of the structural equation model, as was the path from family history of AUD to childhood behavior problems. The path between childhood behavioral problems and difficult temperament was also trimmed because this path was non-significant.

RESULTS

Confirmatory Factor Analysis

The latent variable, alcohol problem severity was constructed using confirmatory factor analysis and then entered into the structural equation model. Current or lifetime diagnosis of alcohol dependence (yes or no), number of DSM-III-R AUD symptoms endorsed, PDD, DDD, and total weighted MAST-B score were entered as indicators of AUD severity. This latent alcohol severity model was found to have excellent fit (CFI=.989; RMSEA= .054; SRMR= .028; relative normed chi-square= 1.96). Alternative models were fitted that included other indicators of AUD severity, but this structure showed the best fit.¹ The construct variables had the following factor loadings: Current or lifetime diagnosis of alcohol dependence= .35, number of DSM-III-R AUD symptoms endorsed= .90, PDD= .28, DDD= .51, and total weighted MAST-B score= .68.

Structural Equation Model

Means and frequencies of model variables are reported in Table 2. Overall, the model's endogenous variables (ASPD, BPD, MDD, and AD) explained 8% of the variance in the latent factor, AUD severity. Two distinct pathways were found from Stage 1 predictors to the Stage 4 outcome (see Figure 2). The model from which these two paths emerged had good fit (CFI= .902; RMSEA= .050; SRMR= .058; relative normed chi-square= 1.84). Covarying the exogenous variables in the final model did not alter the fit.

In one path, male gender and family history of a drug use disorder predicted more childhood behavior problems (Hk/MBD score), while childhood behavior problems predicted a greater likelihood of adult psychopathology, specifically having ASPD, BPD, and AD. Lastly, diagnosis of an AD predicted current AUD severity.

In the second path, family history of an AUD predicted higher scores on the difficult temperament index (DOTS-R). In turn, difficult temperament predicted a greater likelihood of adult psychopathology, specifically having BPD, MDD, and AD where MDD and AD predicted current AUD severity.

¹Other indicators of AUD severity investigated included age of onset of alcohol abuse or dependence, and number of adverse alcohol related consequences.

In sum, the two paths differed in that path 1 did not include the vulnerability factors of difficult temperament or family history of an AUD, showed a risk profile for men, and only AD led to AUD severity; whereas path 2 was not specific to men, did not include the vulnerability factors of family history of drug use disorder, or childhood behavior problems, while AD as well as depression led to AUD severity.

Mediation analyses revealed a significant indirect effect of greater family history of drug use disorders leading to greater likelihood of ASPD through greater childhood behavior problems (Sobel's test, $z = 3.02$, $SE = .00$, $p = .003$). In addition, a positive history of AD mediated the positive relationship between difficult temperament and AUD severity (Sobel's test, $z = 2.45$, $SE = .58$, $p = .014$), while a positive history of MDD mediated the positive relationship between difficult temperament and AUD severity (Sobel's test, $z = 2.62$, $SE = .27$, $p = .009$).

Because of the cross-sectional nature of the data, we tested an alternative model by flipping Stages 3 and 4. This allowed us to test whether AUD severity was potentially a precursor to anxiety, depressive, or personality disorder pathology, as opposed to being a consequence of it. This model failed to converge, indicating very poor fit, even after attempts to improve model fit, such as setting parameters to 1, and resetting starting values. This observation supports the validity of the temporal relationship in the a priori model.

DISCUSSION

This study sought to extend prior findings on developmental risk factors and trajectories to adult alcohol problems by examining how gender, family history of drug or alcohol problems, childhood behavior problems, difficult temperament, and adult psychopathology lead to severity of alcohol problems in a heterogeneous sample of treatment-seeking participants with alcohol use disorders (AUDs). We assessed the influence of these factors using path modeling and latent factor analysis in theoretically plausible temporal pathways to examine whether one or more risk profiles would be associated with pre-treatment AUD severity.

In the present study, a latent factor capturing AUD severity was derived using the composite of variables, including lifetime alcohol dependence diagnosis, number of DSM-III-R AUD symptoms endorsed, percent of days drinking (PDD), mean drinks per drinking day (DDD), and the Brief Michigan Alcoholism Screening Test (MAST-B) score. The model's endogenous variables explained 8% of the variance in AUD severity. While this figure may not seem ostensibly large, it should be remembered that this figure represents pure variance, distinct from model disturbances, which are accounted for in structural equation modeling.

Two distinct pathways to adult AUD severity emerged. The first pathway began with male gender or family history of drug abuse or dependence, not alcohol abuse or dependence, as might be expected. This then led to childhood behavior problems. Childhood behavior problems then led to the adult psychopathology including antisocial personality disorder (ASPD), borderline personality disorder (BPD), and anxiety disorders (AD). In turn, AD emerged as the direct predictor of AUD severity. The second pathway began with family

history of an AUD, which led to difficult temperament, and then adult psychopathology, including BPD, AD, and major depressive disorder (MDD), then finally to AUD severity via the MDD and AD paths.

The pathways revealed in this model suggest being male and/or having a family history of drug use disorders may be linked to AUD severity in instances where childhood behavioral problems transmute to AD, but not ASPD or BPD. In addition, having a family history of AUD predicted AUD severity through AD and MDD pathways. Sartor and colleagues (8) previously showed that male gender and familial alcohol and other drug abuse predicted early alcohol use, as did adolescent behavioral problems characterized by conduct disorder and attention deficit hyperactivity disorder. In turn, they showed generalized anxiety disorder predicted progression to AUD. It is possible that the latent AUD severity factor in the present study is picking up on an additional nuance in the relationship between AD, MDD and AUD.

Both of these pathways are concordant with previous work demonstrating an association between anxiety, depression and AUDs. In addition, the positive findings for the primary model, and the null findings for the alternative model (wherein Stages 3 and 4 were flipped) support the idea that affective disorders such as ADs and MDD often precede the onset of AUD symptomology—an observation substantiating the self-medication hypothesis (30, 39, 40).

These findings uncover two distinct pathways to adult alcohol problem severity, providing support for the notion of heterogeneity in the development and maintenance of problematic alcohol use behavior. Moreover, the endpoint AUD severity factor utilized in the present investigation was unique from prior studies, in that it was comprised of a latent score of AUD severity derived from individual measures of problematic alcohol involvement. Other studies have examined these factors independently as observed variables, or have used binary dependent variables of AUD diagnosis that lack individual problem severity specificity. The use here of a latent variable to measure AUD severity allowed for the measurement of problematic alcohol use that is implied by the measured variables, but not directly observed, allowing us to capture a broader characterization of the construct. In addition, whereas previous studies examining developmental trajectories have typically utilized “at-risk” samples, this study adds to the literature in that the sample was comprised of a diverse and relatively severe group of individuals with alcohol dependence or abuse. Previous research has suggested that problem drinkers who seek treatment often have greater alcohol-related problems, alcohol use severity, and psychiatric distress than those who do not seek treatment (82). Thus, these findings can be generalizable to a more severely affected group of problem drinkers, and may inform early interventions and treatments that target populations at risk for developing AUDs.

Limitations

There are several limitations of this study that bear mentioning. First, while structural equation modeling is a powerful statistical technique for examining causal relationships between two or more variables, inferring strict causality among variables must be cautioned. In addition, while this technique allows a fit of several specified models, alternative

pathways cannot be ruled out. Additionally, conclusions about causality among variables in our model must be made with caution, as none of the variables of interest were directly manipulated, and because data were collected in a cross-sectional, retrospective manner. Concerns about the use of retrospective data should be reduced, however, given that the characteristics examined have been shown to emerge at specific points in the lifespan (83). Gender and family history, for example, are implicit, regardless of measurement point. Additionally, temperament is thought to be determined before or very soon after birth and then to be stable into adulthood (84–86). Although some data were collected retrospectively, this is not the first study to do so; previous clinical studies have used the Hk/MBD and DOTS-R to retrospectively measure childhood behavior problems (7, 24, 87, 88). Despite the cross-sectional design of our study, statistically significant paths in a theoretically plausible developmental model were identified. Future research should further examine and attempt to replicate these pathways using a longitudinal, prospective design.

Conclusions

In sum, the present findings add to previous research showing that family history of substance use disorders, gender, behavioral problems, and emotional disorders are differentially associated with the problematic alcohol use. These results are concordant with previous work using “at-risk” samples and dichotomous outcomes variables in that they show that substantial heterogeneity exists in the developmental risk factor pathways leading to AUD severity in a treatment-seeking sample, and that these pathways are not mutually exclusive. Further, these results indicate that developmental pathways and individual risk factors can be used to predict a broader construct of alcohol problem severity, as well distinct diagnostic factors. Behavioral dyscontrol, and affective disorders appear to be consistent across multiple risk categories, and therefore may interact with family history and subsequent developmental trajectories that lead to alcohol use problems. Although not all individuals with AUDs present with pre-morbid psychopathology, identification of discrete pathways has implications for targeted assessment of the specific factors that maintain current alcohol use behavior for an individual who presents for AUD treatment.

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Stage 1	Stage 2	Stage 3	Stage 4
Inherited/early Vulnerability	Childhood Behavior Problems	Adult Psychopathology	Alcohol Use Disorder Severity at Treatment Entry
Male Gender	Childhood Behavior Problems	Antisocial Personality Disorder	Current or Lifetime Diagnosis of Alcohol Dependence
% First Degree Relatives with a Drug Use Disorder	Difficult Temperament	Borderline Personality Disorder	Number of DSM-III-R Alcohol Use Disorder Symptoms Endorsed
% First Degree Relatives with an Alcohol Use Disorder		Lifetime Depression	Percent Days Drinking
		Lifetime Anxiety	Drinks per Drinking Day
			Michigan Alcoholism Screening – Brief Version Test Score

Figure 1.
Proposed Variables in Developmental Trajectories of Alcohol Use Disorder Severity.

Figure 2.

Structural equation model, CFI= .902; RMSEA= .050; SRMR= .058; relative normed chi-square= 1.84, showing two developmental trajectories to alcohol use disorder problem severity. *Notes:* Standardized beta coefficients reported. Paths represented by solid lines significant at $p < .05$, paths represented by dotted lines not significant.

Table 1

Sample Characteristics (n = 335)

Age (Mean & <i>SD</i>)	41.6	(14.0)
Gender	254 male	(75.8%)
Race		
African American	88	(26.3%)
European American	225	(67.2%)
Hispanic	13	(3.9%)
Not reported	9	(2.7%)
Education		
Some high school	50	(14.9%)
High school or GED	93	(27.8%)
Some college	126	(37.6%)
College graduate	30	(9.0%)
Some graduate school	15	(4.5%)
Graduate degree	21	(6.3%)

Notes. *SD*= Standard deviation

Table 2
Means and Standard Deviations / Frequencies, and Zero-order Correlations Among Model Variables

	<i>M(SD) / Freq.</i>	1	2	3	4	5	6	7	8	9	10	11	12	13
1. % Male Gender	75.8													
2. % 1 st degree relative with drug use disorder	11.1 (17.8)	-.11												
3. % 1 st degree relative with AUD	24.8 (24.2)	-.12*	.50*											
4. Childhood behavior problems (HK/MBD)	15.5 (9.1)	.22*	.25*	.16*										
5. Difficult temperament (DOTS-R)	2.4 (1.4)	-.04	.07	.11	.12									
6. Antisocial personality disorder diagnosis	15.2%	.11	.20*	.15*	.39*	.11								
7. Borderline personality disorder diagnosis	11.5%	-.12	.17*	.11	.19*	.24*	.32*							
8. History depression diagnosis	35.6%	-.10	.10	.15*	.13*	.21*	.16*	.26*						
9. History anxiety diagnosis	11.2%	-.00	.03	.10	.16*	.20*	.11	.04	.10					
10. Alcohol dependence diagnosis	96.0%	.06	-.05	-.02	-.01	.01	.05	.03	.10	.08				
11. Number AUD symptoms	8.0 (2.6)	.16*	.05	.14*	.15*	.15*	.10	.13*	.21*	.18*	.52*			
12. Percent days drinking	59.7 (39.7)	.17*	-.05	-.04	.06	-.01	-.06	-.04	.01	.01	.19*	.28*		
13. Drinks per drinking day	14.9 (13.4)	.18*	.05	.06	.16*	.05	.21*	.11	.11*	.09	.15*	.45*	.22*	
14. MAST-B score	17.4 (8.2)	.13*	-.01	.10	.05	.14*	.17*	.14*	.08	.08	.22*	.62	.14*	.38*

Notes.

M = Mean, *SD* = Standard deviation, *Freq.* = Frequency, *AUD* = Alcohol use disorder, *MAST-B* = Michigan Alcohol Screening Test – Brief Version

* $p < .05$.