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Addiction resistance to alcohol: What about heavy drinkers who avoid alcohol problems?

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ABSTRACT

Background: Some individuals are resistant to alcohol use disorders despite high levels of intake. Addiction Resistance (AR) measures the disparity between alcohol consumption and alcohol use disorder (AUD) symptoms, such that some persons exhibit few (AUD) symptoms despite higher intake. The validity of the concept and the factors contributing to AR are not well understood. The aim of this study was to predict AR based on variables related to risk for addiction that are measured in the Family Health Patterns Project.

Method: Participants were healthy young adults (n = 1122) with and without a family history of alcohol and other substance use disorders who were given measures of mood stability and risk-taking tendencies, and were interviewed to determine alcohol intake, AUD symptoms, and other substance use disorders (SUD). AR was calculated using maximal lifetime alcohol intake and number of AUD symptoms.

Results: A principal components analysis was run with varimax rotation, which yielded three components: Component 1 indexed behavioral and mood regulation, Component 2 encompassed family and environmental factors, and Component 3 included cognitive factors. A multiple regression analysis revealed that Component 1 and Component 2 were predictive of AR whereas Component 3 was not.

Discussion: Individuals who reported greater emotional stability, norm adherence, risk avoidance, and fewer family members with substance use disorders were more resistant to AUD despite higher alcohol intake. These findings suggest that AUD risk and resistance may represent different points of the same continuum.

1. Introduction

Alcohol use disorders (AUD) are a major public health problem, with lifetime prevalence rates as high as 29.1% among adults aged 18 and older (Grant et al., 2015). It is estimated that 1 in 10 deaths among working age adults are from alcohol related causes each year in the United States (Stahre et al., 2014). Additionally, greater AUD severity corresponds to more significant lifetime disability (Grant et al., 2015). In light of such statistics, understanding risk factors for AUD provides an essential step in coping with the health burden of AUD.

However, experience shows us that there are heavy drinkers who appear to avoid problems stemming from alcohol use. Recent work has focused on this notion of addiction resistance to alcohol (AR), capturing the intuitive idea that some persons resist developing an AUD despite heavy levels of intake (Kendler and Myers, 2015). Characterizing persons high in AR may clarify the interplay between risk factors and resilience to alcohol's deleterious effects. Kendler and Myers (2015), recently addressed this issue and identified a set of characteristics of individuals who reported heavy intake and who did not meet criteria for an AUD. The present paper evaluated the relationships between traditional risk factors for AUD and AR using data from the Family Health Patterns (FHP) cohort, a sample of healthy young adults with varying levels of risk for AUD. We compared FHP results with those of Kendler and Myers (2015) and asked whether AR persons simply lacked the standard risk factors or if risk factors for AUD had differential relationships with AR.

The best-known AUD risk factor is having a family history (FH) of alcoholism, with persons with a FH having 4–6 times the lifetime incidence relative to persons without a FH (Merikangas and Avenevoli, 2000). Adolescents and young adults with a FH of alcoholism show

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other characteristics thought to contribute to AUD risk, including a high prevalence of behavioral disinhibition or behavioral undercontrol (Sher et al., 1991; Tarter et al., 2004; Zucker et al., 2011), mood dysregulation (Yoshimasu et al., 2016), mild cognitive deficits (Acheson et al., 2011), and exposure to early life adversity (ELA) (Acheson et al., 2018; Vincent et al., 2017). Although these risk factors seem to be consistently identified, little is known about protective factors for AUD.

Empirical work shows that volume of alcohol consumption is an imperfect indicator of AUD, particularly when heavy episodic drinking is not taken into account (Greenfield et al., 2014). Further, the prevalence of heavy episodic drinkers who do not develop an AUD has been shown to range from 58% to 82%, depending on age (Linden-Carmichael et al., 2017). Reflecting this, the diagnostic criteria for AUD do not specify any threshold level of intake, instead relying on social, behavioral, legal, and health consequences associated with the person's drinking habit (American Psychiatric Association, 2013). However, it is notable that the peak prevalence of heavy drinkers developing an AUD is as high at 42% (Linden-Carmichael et al., 2017). Additionally, heavy drinking may result in negative health consequences independent of an AUD diagnosis (Goel et al., 2018; Topiwala and Ebmeier, 2018). Regardless, considering alcohol intake and AUD symptoms simultaneously may provide better insights into the prevention, treatment, and outcomes of alcohol-related problems.

Using a large twin sample in a prospective study, Kendler and Myers (2015) showed that AR was estimated to be 34.8% heritable and relatively stable over the course of a 12-month period. Factors that predicted greater risk and lower AR were: a denser FH, childhood sexual abuse, diagnosis of generalized anxiety disorder (GAD), major depressive disorder (MDD), or antisocial personality disorder (ASPD; Kendler and Myers, 2015). In addition to identifying low levels of established risk factors, Kendler and Myers (2015) identified personal mastery, or an individual's perception of control over outcomes in their life, as a characteristic that contributed positively to the AR construct (Pearlin and Schooler, 1978). In the study by Kendler and Myers, normative traits of behavioral and mood regulation were not extensively evaluated, with the exception of neuroticism (Kendler and Myers, 2015). Although differences were observed in AR between clinical and non-clinical groups (Kendler and Myers, 2015), it is possible that normative variations in behavioral and mood regulation, as well as other risk factors for AUD, may correspond to AR. FH, ELA, and trait measurements of behavioral and mood regulation have been measured in the FHP cohort (Kendler and Myers, 2015). Thus, the FHP cohort may be effective in evaluating what risk factors for AUD are most predictive of AR, which may better elucidate the construct of AR.

The FHP sample provides a perspective on the AR concept. The FHP cohort was selected to characterize persons varying in risk for AUD with particular emphasis on healthy young adults with and without a FH of substance use disorders studied at a single time point. A recent FHP analysis of AUD risk identified internalizing characteristics and mood instability, externalizing characteristics and history of ELA, and cognitive characteristic involving working memory and decision making (Acheson et al., 2018). The current study sought to evaluate if these characteristics corresponding to AUD risk similarly predicted varying levels of AR. Specifically, we hypothesized that higher levels of typical risk factors for AUD would be predictive of lower AR.

2. Materials and methods

2.1. Participants

Participants were a community sample of healthy young adults (n = 1122), ranging in age from 18 to 30 years. Average age of the sample was 23.68 and average level of education attained was 15.22. Additional demographic information is presented in Table 1. Reported drinking and drug use characteristics are reported in Table 2. Inclusion criteria included: no current use of CNS-acting medication, maintaining contact with at least one biological parent, and having no history of neurological damage or impairment. Participants were excluded if they had never consumed a full drink of alcohol. Unlike some previous studies of this cohort (Lovallo, 2013), individuals positive for alcohol abuse or dependence histories were not excluded. All participants completed informed consent prior to participation and were financially compensated for their participation. The FHP was approved by the Institutional Review Board of the University of Oklahoma Health Sciences Center and the VA Medical Center in Oklahoma City, OK.

Table 1
Sample Demographic Characteristics.

	Frequency (N)	Percent
Age		
18–20	250	22.3
21–25	602	53.7
26–30	270	24.1
Sex		
Male	446	39.8
Female	676	60.2
Race		
White	859	76.6
Black	113	10.1
Native American	37	4.4
Asian	19	1.6
Biracial	51	4.5
Other	49	4.4
Ethnicity		
Hispanic	115	10.2
Non-Hispanic	1007	89.8
Education		
High school or less	240	22.1
Some college	615	54.8
College degree	194	17.3
Graduate degree	65	5.8

Table 2
Sample Drinking and Drug Use Characteristics.

	M (SD)	Percent of Sample
Drugs Used	1.7 (1.78)	100
Age at First Drink	16.3 (2.84)	78.1
Intent to get Drunk	1.2 (2.61)	48.9
AUDIT	4.5 (3.75)	74.4

Note. Drugs Used = lifetime number of psychoactive substances ever tried not for a medical purpose, Age at First Drink = age of first full alcoholic drink, Intent to get Drunk = number of days over the last month that participant drank with the intention of becoming intoxicated, AUDIT = Alcohol Use Identification Test total score, Percent of Sample = the percentage of the FHP sample that reported on each characteristic.

2.2. Procedure

Following prescreening by telephone, participants were interviewed in the laboratory by a trained research assistant under the supervision of a licensed clinical psychologist using the Computerized Diagnostic Interview Schedule, which was based on DSM-IV criteria (C-DIS-IV; Blouin et al., 1988). Additional interviews collected information on FH density. Participants were also given psychological self-report measures. Parents were interviewed to confirm reports of FH in 20.5% of cases.

2.3. Materials and procedure

2.3.1. Addiction resistance

Maximal lifetime alcohol use in drinks per year and AUD symptoms

were obtained using the alcohol abuse and dependence sections of the C-DIS-IV. Maximal lifetime use in drinks per year was based on the product of the following two questions: 1) "During the year when you drank the most, how many of the 52 weeks did you drink at all?" and 2) "During the weeks when you had something to drink, how much would you usually drink from Monday to Sunday?" AUD symptom questions were asked with reference to participants' year of highest alcohol intake. AUD symptom questions were asked in a screening format based on branching logic, thus not every participant was asked every question. Because of this, a proportion was calculated based on the number of positively endorsed criteria divided by the number of questions asked, such that a larger proportion indicated a greater percentage endorsement of AUD criteria. On average, participants were asked 7.24 (SD = 1.10) symptom questions out of the 11 AUD symptom criteria. The Alcohol Use Disorder Identification Test (AUDIT; Saunders et al., 1993) was used to evaluate the contrast validity of this AUD severity measure. Previous studies have implemented the AUDIT as a measure of AUD severity (Fede et al., 2019).

AR was then determined by calculating the residuals of the regression between maximal lifetime use and proportion of endorsed AUD criteria. Specifically, individuals with higher than predicted AUD criteria based on alcohol intake would be characterized as having a lower resistance to AUD, while individuals with fewer than predicted AUD criteria would have higher resistance as implemented by Kendler and Myers (2015). For example, an individual with higher than average AUD symptoms based on alcohol intake would have lower AR than an individual with lower than average AUD symptoms based on alcohol intake. In this way, AR captures those exhibiting lower than expected AUD symptoms based on their amount of alcohol intake.

2.3.2. Family history density

The Family History Research Diagnostic Criteria (FH-RDC) was used to determine participants' biological parents' and grandparents' history of substance use disorders (SUD; Andreasen et al., 1977). SUD was categorized as a family member having at least two criteria endorsed by the participant. As part of the FHP, biological parents are contacted to confirm participant reports. Parental reports were attained for approximately one-fifth of the full FHP cohort (20.5%). Participant and parent agreement of FH status was observed in 90% of these reports. This information was gathered to verify participant reports of FH. Family history density was calculated by adding together all family members (i.e., biological parents and grandparents) who were categorized as having a SUD, with scores ranging from 0-6.

2.3.3. Early life adversity

Questions taken from the C-DIS-IV posttraumatic stress disorder (PTSD) section were used to create ELA composite scores, which closely resemble assessment of adverse life events conducted in studies by Caspi (Caspi et al., 2002, 2003). The PTSD section covers physical and sexual abuse ("Have you ever been raped or sexually assaulted by a relative?" "Have you ever been raped or sexually assaulted by someone not related to you?" "Have you ever been mugged or threatened with a weapon, or experienced a break-in or robbery?"), and emotional adversity ("Before you were 15 was there a time you did not live with your biological mother for at least 6 months?" "Before you were 15, was there a time you did not live with your biological father for at least 6 months?"). This score ranged from 0 (no adverse events) to 5.

2.3.4. Behavioral disinhibition

The California Personality Inventory – Sociability scale (CPI-So; Gough, 1994) assessed conformity to social norms, with higher scores indicating a greater degree of conformity. Persons at higher risk for addiction have been shown to exhibit lower CPI-So scores (Sorocco et al., 2015). The Psychopathic Personality Inventory (PPI) is a self-report instrument that has two factors based on Hare's Psychopathy Checklist (Hare and Neumann, 2005). PPI Factor II score describes poor behavioral regulation and a tendency toward norm violation. PPI Factor II has been found to be positively related to alcohol use and dependence in community samples (Benning et al., 2003).

2.3.5. Neuroticism

Emotional stability was assessed using the neuroticism subscale of the Eysenck Personality Inventory (EPI-N; Eysenck and Eysenck, 1964; Meites et al., 1980). Higher scores indicate greater levels of neuroticism. Neuroticism has been shown to be associated with greater risk for AUD (Larkins and Sher, 2006).

2.3.6. Delay discounting

Delay discounting or devaluing of delayed rewards were assessed with a paper and pencil questionnaire (Acheson et al., 2011; Kirby et al., 1999). The questionnaire consisted of 27 items where participants were asked to choose between a smaller immediate reward or a larger delayed reward for each item. Discount rate estimates (k), based on a hyperbolic function of value over time by Mazur (1987), were calculated for each participant then subjected to a **log** transformation. Delay discounting is associated with cognitive ability, including working memory capacity (Murphy and Garavan, 2011; Szuhany et al., 2018). Higher rates of delay discounting imply more rapid loss of perceived value over time and correspond to more impulsive decision making. Increased discounting of delayed rewards is associated with nearly all types of SUD (Amlung et al., 2017; Bickel et al., 2014; MacKillop et al., 2011).

2.3.7. Mental age

Global cognitive functioning was measured using the Shipley Institute of Living scale (Zachary et al., 1985), which consisted of vocabulary and abstraction subscales and yielded a mental age estimate in years ranging up to 22 years. Higher scores are associated with higher mental age. Observed scores in this sample ranged from 9.2 to 20.6. SUD diagnosis has been associated with poorer verbal reasoning, which is a major component of metal age (Latvala et al., 2009).

3. Data analysis

AR was calculated using the relationship between the maximal lifetime alcohol use score and the proportion of endorsed AUD criteria. Bivariate correlation coefficients of all measured variables are presented in Table 3. Due to collinearity among the dependent variables, a factor analysis was conducted using principal components with a varimax rotation. Component loadings, eigenvalues, and explained variance are reported in Table 4. Component scores were then computed using a regression method. A multiple linear regression analysis was run to determine which components were most predictive of AR. Because age, years of education, and sex were found to be significantly related to either AR or a component score, they were included as covariates in the regression model.

4. Results

Using a subsample of participants who completed the AUDIT, the relationship between the percent of AUD symptoms endorsed on the C-DIS-IV interview and AUDIT scores was evaluated to assess validity of our measure of AUD severity. The AUDIT and percent of AUD symptoms endorsed was significant and positive, $r(1, 833) = .42$, $t = 13.53$, $p < .001$, which supported the use of this measure as a proxy for AUD severity. The relationship between maximal lifetime alcohol use and AUD symptoms was significant and positive, $r\ (1, 1120) = .58$, $t = 24.09$, $p < .001$. As expected, persons who consumed alcohol at a higher rate were more likely to report AUD symptoms. This relationship accounted for 34% of the observed variance in AUD symptoms. The distribution of AR was unimodal and was significantly leptokurtotic ($p < .001$). Additionally, AR was significantly and positively skewed

Table 3
Bivariate Pearson Correlation Coefficients Between All Predictor Variables.

	M (SD)	1	2	3	4	5	6	7
1. AR	−0.31 (20.66)	–						
2. FH density	0.92 (1.14)	−.16**	–					
3. ELA	0.94 (1.05)	−.13**	.38**	–				
4. CPI-So	29.87 (5.80)	.26**	−.33**	−.44**	–			
5. PPI-II	12.57 (2.38)	−.26**	.18**	.25**	−.57**	–		
6. EPI-N	7.00 (4.42)	−.23**	.23**	.20**	−.37**	.46**	–	
7. Delay dis.	−4.45 (1.39)	−.10**	.13**	.16**	−.22**	.16**	.09**	–
8. Mental age	17.47 (1.50)	.06*	−.19**	−.21**	.25**	−.07*	−.11**	−.26**

Note. M = mean, SD = standard deviation, AR = addiction resistance, FH density = family history density of SUDs, ELA = early life adversity, CPI-So = California Personality Inventory – Sociability, PPI-II = Psychopathic Personality Inventory – Factor II, EPI-N = Eysenck Personality Inventory – Neuroticism, Delay dis. = discount estimate rate (k), Mental age = Shipley Institute of Living scale mental age in years. All coefficients were tested against two-tailed significance.

** p < .01.

* p < .05.

Table 4
Rotated Factor Analysis Loadings for Variables Used to Predict AR.

Measure	Component 1	Component 2	Component 3
CPI-So	**−.68**	**−.41**	−.23
PPI-II	**.88**	.07	.06
EPI-N	**.75**	.11	−.01
FH Density	.11	**.81**	.05
ELA	.21	**.76**	.13
Delay Discounting	.16	−.06	**.83**
Mental Age	.03	−.29	**−.72**
Eigenvalue	2.58	1.16	0.94
Variance Explained (%)	36.9	16.5	13.4

Note. CPI-So = California Personality Inventory – Sociability, PPI-II = Psychopathic Personality Inventory – Factor II, EPI-N = Eysenck Personality Inventory – Neuroticism, FH Density = family history density of SUDs, ELA = early life adversity, Delay Discounting = discount estimate k, Mental Age = Shipley Institute of Living Scale. Component loadings above .40 are bolded.

($p < .001$), however the skew statistic was less than 2, indicating that there were not problems with skewness for parametric analysis. An independent samples *t*-test revealed that AR was significantly lower among males than females ($t = 2.14$, $p = .032$).

The factor analysis identified three principal components, which explained 66.7% of the total variance. Component loadings, eigenvalues, and variance explained by each component are reported in Table 4. Component 1, encompassed the CPI-So, PPI-II, and EPI Neuroticism scores. Based on the direction of the component loadings, higher scores on the Component 1 reflected greater behavioral disinhibition and poorer emotional stability. Component 2 comprised FH Density and ELA. Additionally, the CPI-So also had a notable loading on Component 2 (.41). Based on the direction of the component loadings, higher scores on Component 2 indicated greater exposure to ELA and a denser FH. Component 3 was comprised of the Shipley mental age score and delay discounting performance. Based on the direction of the component loadings, higher Component 3 scores indicated a lower mental age and greater discounting of delayed rewards.

Following a multiple linear regression, the components explained a significant amount of variance in AR, adjusted $R^2 = .10$, F (3, 1118) = 41.38, $p < .001$. The respective components were tested for their contributions to unique variance in AR. Standardized beta weights, *t*-tests, and tolerances for the predictors and covariates are presented in Table 5. Component 1 explained 7.9% of unique variance in AR and was negatively related to AR. Based on the direction of the loadings, poorer behavioral and mood regulation was associated with poorer AR. Component 2 explained 1.0% of unique variance in AR and was negatively associated with AR. Based on the direction of the loadings, greater ELA and FH density were associated with poorer AR. Component 3 did not explain a significant portion of unique variance in AR.

Table 5
Standardized Betas and t-tests for Variables in Multiple Regression Analysis.

	Standardized Beta	*t*	*p*	Tolerance
BMR	−.29	−9.84	< .001	.97
FEF	−.12	−3.62	< .001	.86
CF	−.06	−1.87	.056	.90
Sex	.05	1.72	.09	.97
Age	−.03	−.089	.38	.86
Education	−.01	−.010	.77	.71

Note. BMF = Behavioral and Mood Regulation, FEF = Familial and Environmental Factors, CF = Cognitive Factors.

5. Discussion

The current study evaluated correlates of AR to identify characteristics of persons who have few AUD symptoms despite high levels of alcohol intake. This approach is complimentary to a focus on persons who develop AUD by characterizing individuals who are less symptomatic than expected of AUD based on their level of alcohol consumption. The primary goal of this analysis was to determine what ways traditional AUD risk factors were related to AR. Previously, alcohol consumption was found to explain 32% of the variance of endorsed AUD criteria (Kendler and Myers, 2015), while the current study found 34% of explained variance between alcohol use and AUD criteria. The similarity of these values is notable since somewhat different questions were asked to attain maximal alcohol use and count of AUD symptom criteria. Additionally, similar factors explored by Kendler and Myers (2015) were also found to be predictive of AR in the current study. Variables relating to behavioral and mood regulation were found to be the strongest unique predictors of AR. Furthermore, the second most predictive component of AR contained FH density and ELA. Kendler and Myers (2015) found both FH and childhood sexual abuse were found to be predictive of AR. Although there is some concordance between the current study and Kendler and Myers (2015), the present study found that many traditional AUD risk factors were predictive of AR. Notably, however, cognitive factors in general were unrelated to AR.

The current study assessed the potential contribution of cognitive factors, such as delay discounting and mental age, to the development of AR. Both variables had significant bivariate relationships with AR. However, as a conjoined component, they were unrelated to AR. These findings are counterintuitive based on previous studies linking cognitive factors, specifically verbal reasoning and delay discounting, to risk for AUD (Claus et al., 2011; Latvala et al., 2009). However, both delay discounting and mental age were found to be correlated with nearly all other predictors. Notable exceptions include mental age being uncorrelated with PPI-II and delay discounting being uncorrelated with

neuroticism. Cognitive and decision-making factors being related to other risk factors for addiction has previously been documented in the FHP sample (Acheson et al., 2011; Lovallo, 2013; Lovallo et al., 2013). It is possible that the variables used in this study to measure cognitive factors have indirect relationships with AR. Future research should investigate these potential relationships, and other measures of cognitive functioning to explain AR.

Component 1 – comprised of the CPI-So, PPI-II, and EPI-N – was the strongest predictor of AR. This finding indicates that normal behavioral inhibition, norm adherence, and emotional stability are resilience factors that confer greater resistance to AUD. This is consistent with a recent study showing that externalizing and internalizing tendencies are associated with AR among first year college students (Cooke et al., 2017). It is possible that behavioral inhibition and emotional stability and development of AR are both influenced by similar underlying neural systems underlying effortful control and incentive reactivity (Zucker et al., 2011). Normal development of these systems may increase resistance to the development of AUD. Additional research is needed parse the causal relationships between these neural systems, behavioral inhibition and emotional stability, and AR.

Other work has repeatedly identified FH as a risk factor, implicating genetic factors although specific genes have so far eluded detection (Reilly et al., 2017). Component 2 in the present data was primarily comprised of FH density and ELA suggesting a combination of genetic and environmental factors that may contribute to AR. It is worth noting that FH and ELA as measured here are not inherently independent variables and in some sense may not easily be separated. Greater FH density may confer risk through heritable means including vulnerability to aspects of the household environment. ELA captures the family environment and may itself be an expression of a disrupted and abusive family. Accordingly FH and ELA capture possible feedback relationships between inherited risk and family environment that are not fully separable.

It should be noted that the CPI-So had a small but notable factor loading on Component 2, even after rotation. This is likely due to the multidimensional nature of the CPI-So subscale, which contains items dealing with antisocial tendencies and home and family life (Gough, 1994). Items related to poorer home and family warmth have been shown to be most related FH of SUD status (Vincent et al., 2017). Thus, it is not surprising that in the current analysis the CPI-So shared variance in the FEF factor. However, our previous analysis showed that the strongest single predictor of SUD was the person's CPI-So score, indicating that the individual's manifestation of rule violation, impulsive behavior, and lack of empathy as captured in these scores, was the most proximal contributor to SUD outcome. The same analysis showed that ELA and an FH of SUD background were also significant contributors to SUD outcomes, suggesting that FH background may capture genetic vulnerabilities, ELA may result in early stress exposure that acts on the presumed vulnerability, and antisocial characteristics may point to the risk prone phenotype (Vincent et al., 2017).

Since the current study found that traditional risk factors were predictive of AR, questions arise concerning whether risk and resistance factors for AUD are divergent constructs, or rather, reflect different ends of a continuum. A recent analysis of the FHP cohort found that externalizing tendencies, such as behavioral disinhibition and lack of norm adherence, and experience of ELA was more strongly associated with FH status, FH density, and probability of an AUD diagnosis than internalizing tendencies and general cognitive factors (Acheson et al., 2018). This study was in line with previous research that has identified behavioral dysregulation as a key risk factor for AUD (Sher et al., 1991; Tarter et al., 2004; Zucker et al., 2011). In the current study, better behavioral regulation was related to greater AR. Similarly, FH status has been consistently demonstrated to be a potent risk factor for AUD (Merikangas and Avenevoli, 2000), and was found to be related to poorer AR in the current study. Thus, resistance, using the presented measurement method, and risk for AUD may represent different points on the same continuum.

Based on the current analyses, it appears that individuals with fewer commonly identified risk factors for AUD exhibit less alcohol-related impairment despite relatively higher levels of alcohol intake. Overall, these findings support previous research on identified risk factors for SUD. All AUD-related risk factors analyzed in this study had significant bivariate relationships with AR. Additionally, the two primary components extracted from these joined variables uniquely predicted AR. Thus, factors corresponding to greater AR appear to be largely similar to those related to lower risk for AUD. However, the current study was unable to evaluate protective factors such as mastery, previously related to AR (Kendler and Myers, 2015). It is possible that protective factors may be differentially related to risk or resistance to AUD.

The present results should be interpreted in light of limitations. The primary measures used to determine AR were based on autobiographical memory of alcohol use. Specifically, recall bias may have affected participants' reports of their drinking histories. However, there is no reason to suspect that recall bias differentially affected reports of those with low and high AR. Thus, the primary findings of this study were likely not significantly impacted by recall bias. Additionally, a raw count of AUD symptomatology was not available since not all questions were asked of all participants due to the nature of the computerized interview. However, the estimated shared variance between alcohol use and symptoms in present study closely resembles the estimate from the previous study on AR (Kendler and Myers, 2015). The measure of ELA in the current study may be limited in nature, and thus the relationship of ELA to AR may be underestimated. Another limitation is that measurement in the current study was limited to well-known risk factors for AUD. Potential protective traits, such as mastery, were not able to be considered. The age range for subjects enrolled in the FHP was between 18 to 30 years, thus the present results may not generalize to children, adolescents, or middle-aged or older adults individuals. The current study was cross sectional, thus causal relationships were unable to be evaluated. Finally, although efforts were made to sample generally from the community, the sample was ultimately gathered by convenience and not true randomness.

Overall, results from the current study suggest that AR may reflect an absence of risk factors for AUD. Inherent to the AR construct is that there are individuals who consume higher amounts of alcohol, but do not exhibit AUD symptoms. Thus, focusing on clearer indications of AUD symptoms, such as behavioral and mood dysregulation may be more effective for prevention, diagnosis, and treatment of AUD. However, it should be noted that individuals exhibiting heavy drinking but few AUD, representing high AR, are likely at risk for negative health consequences of heavy drinking (Goel et al., 2018; Topiwala and Ebmeier, 2018) and may develop additional AUD symptoms. Future studies are needed to determine if there are protective or risk factors that influence AR and AUD diagnosis differentially. Additionally, future research should consider potential gene by environment interactions that could contribute to the development of AR.

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Contributors

J. Hoffmeister performed statistical analyses and drafted the first version of the manuscript. A. Cohoon performed data management, consulted on statistical analyses, and edited the manuscript. K. H. Sorocco, A. Acheson, and W. R. Lovallo edited all versions of the manuscript. All authors read and approved the final manuscript.

Declaration of Competing Interest

No conflict declared.

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