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Does variance in drinking motives explain the genetic overlap between personality and alcohol use disorder symptoms? A twin study of young women

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Abstract

Background—Genetic risk for alcohol dependence has been shown to overlap with genetic factors contributing to variation in dimensions of personality. Though drinking motives have been posited as important mediators of the alcohol-personality relation, the extent to which the genetic covariance between alcohol use disorder (AUD) symptoms (i.e. abuse and dependence criteria) and personality is explained by genetic factors contributing to variation in drinking motives remains unclear.

Methods—Using data from 2,904 young adult female twins, the phenotypic and genetic associations among personality dimensions (constraint [measured by the Multidimensional Personality Questionnaire; Tellegen, 1982], conscientiousness, neuroticism, and agreeableness [measured by the NEO-PI; Costa & McCrae, 1985]), internal drinking motives (enhancement and coping motives [measured by the Drinking Motive Questionnaire; Cooper, 1994]), and AUD symptoms were tested.

Results—Significant genetic associations were found between all personality measures and AUD symptoms. Coping motives showed significant genetic overlap with AUD symptoms and most personality measures, whereas enhancement motives were not significantly heritable. Adjusting for coping motives, genetic correlations between AUD symptoms and traits of neuroticism and agreeableness were no longer statistically significant.

Conclusions—Findings suggest that genetic variation in drinking to cope might account for a considerable proportion of the genetic covariance between specific personality dimensions and AUD symptoms.

Keywords

behavior genetics; personality; drinking motives; alcohol use disorders

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Numerous studies have linked personality traits to alcohol use, negative consequences from drinking, and alcohol use disorders (AUDs; see Sher, Trull, Barthalow, & Vieth, 1999; Sher, Grekin, & Williams, 2005; Littlefield & Sher, 2010 for reviews). Empirical evidence suggests that traits related to impulsivity/behavioral undercontrol (e.g., lack of constraint) exhibit especially strong relations with AUDs and related outcomes (see Sher et al., 1999). A recent meta-analysis of studies including Five-Factor personality traits (i.e., openness to experience, conscientiousness, extraversion, agreeableness, neuroticism) concluded that alcohol use and related outcomes were associated with low conscientiousness, low agreeableness, and high neuroticism (Malouff, Thorsteinsson, Rooke, & Schutte, 2007). Indeed, this configuration of traits is associated with a wide array of psychopathology (e.g., Trull & Sher, 2004; see De Pauw & Mervielde, 2010, for a recent discussion of this highly replicable finding).

Numerous theoretical explanations have been posited regarding the link between personality and AUDs (Littlefield & Sher, 2010). Genetic diathesis models suggest that the covariance between personality and AUDs is at least partially mediated by common genetic factors (Cloninger, 1987). Indeed, Slutske and colleagues (2002) found that approximately 40% of genetic variation in alcohol dependence was accounted for by genetic variance in behavioral undercontrol in both men and women and that negative emotionality accounted for a modest (though significant) 4% of genetic variation in alcohol dependence in men. These findings suggested that genetic diathesis for AUDs is at least partially related to genetic variation in specific personality traits.

Based on affect regulation models that hypothesize some individuals are predisposed to use alcohol to manage both positive and negative mood states, internal drinking motives (i.e., reasons for drinking) have also been suggested and empirically established as statistical mediators of the personality-alcohol relation (e.g., Cooper, Frone, Russell, & Mudar, 1995; Cox & Klinger, 1988, 1990; see Kuntsche, Knibbe, Gmel, & Engels, 2006 for a review). Specifically, studies have shown that neuroticism is robustly associated with coping motives (i.e., drinking to alleviate negative emotional states; Cooper, Agocha, & Sheldon, 2000; Loukas, Krull, Chassin, & Carle, 2000; Kuntsche, von Fischer, & Gmel, 2008; Stewart & Devine, 2000; Stewart, Loughlin, & Rhyno, 2001), whereas low conscientiousness and agreeableness have been linked to both enhancement (i.e., drinking to enhance positive emotional states) and coping motives (Stewart & Devine, 2000; Theakston et al., 2004). Traits related to impulsivity/behavioral undercontrol have been associated with both enhancement motives (Colder & O'Connor, 2002; Cooper, et al., 2000; Cooper et al., 1995; Comeau, Stewart, & Loba, 2001) and coping motives (e.g., Cooper et al., 2000; Littlefield, Sher, & Wood, 2010; see Littlefield & Sher, 2010, for a discussion on the tenuous relation between enhancement motives and personality).

In a recent review, Kuntsche, Knibbe, Gmel, and Engels (2005) concluded that enhancement motives are directly related to heavy drinking (Carey, 1993; Cooper, Russell, Skinner, & Windle, 1992; Read, Wood, Kahler, Maddock, & Palfai, 2003; Schulenberg, Wadsworth, O'Malley, Bachman, & Johnston, 1996). Coping motives, however, are directly related to alcohol-related problems as well as heavy drinking (Carpenter & Hasin, 1999; Cooper et al., 1995; Cooper, Russell, & George, 1988; Holahan, Moos, Holahan, Cronkite, & Randall, 2001; Holahan, Moos, Holahan, Cronkite, & Randall, 2003; McNally, Palfai, Levine, & Moore, 2003; Simons, Correia, & Carey, 2000; Windle & Windle, 1996; see also Cooper et al., 2008; Littlefield & Sher, 2010).

Associations between drinking motives and alcohol outcomes also appear, to some degree, to be genetically influenced. The heritability of drinking motives, especially those related to coping with negative moods, has been documented, at least in females (Agrawal et. al.,

2008; Prescott et al., 2004). However, the heritability of enhancement motives has been shown to be more inconsistent, in so far as evidence suggests that they are heritable only in certain subgroups or when certain environmental factors are present (see Agrawal et al., 2008; Kristjansson et al., 2011). Further, Prescott et al. (2004) found a substantial portion of genetic variation in AUDs overlapped with genetic variation in drinking to manage mood states. Mood-related drinking motives also have appeared to account for the genetic overlap between alcohol dependence and mood disorders associated with personality. Young-Wolff et al. (2009) recently documented that mood-related drinking motives accounted for nearly all the genetic covariance between depression (a significant phenotypic and genetic covariate of neuroticism; Fanous et al., 2002; Kendler et al., 1993; Kendler et al., 2006) and alcohol dependence.

In sum, personality traits, especially those related to impulsivity/behavioral undercontrol and, to a lesser extent, neuroticism are found to relate to alcohol use and AUDs. Additionally, there is significant genetic covariation between AUDs and personality. Further, internal drinking motives have been shown to significantly account for the personality/alcohol relation. Finally, genetic variation in coping motives has been found to covary with genetic variation in AUDs and additionally, to account for the genetic overlap of AUDs with personality-related mood disorders (i.e, Young-Woff et al., 2009). However, the extent to which genetic variance in drinking motives: a) overlaps with genetic variance in theoretically-related personality constructs, or b) accounts for the genetic covariance between personality and AUDs, has yet to be documented. Significant overlap in the genetic variation in drinking motives with genetic variation in personality would suggest that common biological pathways may contribute to the well documented, albeit modest, associations between personality dimensions and AUDs. By documenting the important role of genetic influences on drinking motives that also influence the extent of genetic covariation between personality and AUDs, we may begin to outline a potential pathway for the link between normative personality and the pathology associated with vulnerability to AUD.

Thus, the current paper sought to address three primary research questions: (a) are there genetic influences on AUD symptoms, measures of personality and measures of internal drinking motives? (b) is the covariation between AUD and personality, personality and motives, and between motives and AUD, partly attributable to shared genetic factors; and (c) if so, to what extent do internal drinking motives account for the genetic co-variation between personality dimensions and AUD symptomatology?

Method

Participants and Procedure

Interview data on 3,787 young adult female twins (mean age 22 years, range 18–29) and questionnaire data on 3,656 of these women were collected during a follow-up phase of the Missouri Adolescent Female Twin Study (MOAFTS) conducted during 2002–2005 (Heath et al., 1999; Heath et al., 2002). The Missouri Adolescent Female Twin Study or ‘MOAFTS’ (Principal Investigator Heath) consists of a cohort of female same-sex twin pairs born between 1 July 1975–30 June 1985 who were identified from birth records for a mid-western US state (Heath et al., 1999; Slutske et al., 2004). Twins were eligible to participate if both members of the twin pair had survived past infancy, were not adopted at birth and if their biological mother was a resident of the state at the time of their birth. Using a cohort-sequential sampling design, twins and their parents were invited to participate in the baseline interviews with at least one biological parent (whenever possible, the biological mother) during 1994–1999, when the twins were 13, 15, 17 or 19 years old. Recruitment of 13-year-olds continued over a 2-year period as twins became age-eligible. After obtaining

permission from parents and assent from twins, a telephone diagnostic interview was administered to the twins and their parents. Of the 2,369 pairs who were identified as live-born, 95.6% were located. The final sample of twins interviewed at baseline for each cohort included 1633 pairs (72.5% of pairs targeted), including 579, 291, 367 and 373 pairs aged 13, 15, 17 and 19 years, respectively (n = 3446). About 13% reported African American ancestry and 40% of the participants came from rural residences. Further details regarding sample recruitment and characteristics are given elsewhere (Heath et al., 1999; Knopik et al., 2005).

Subsequently, 3,059 women from this baseline interview were re-interviewed in 2002–2005, along with an additional 728 women, from the baseline sampling frame, who had not been interviewed previously. To minimize sampling bias all twins were invited to participate, provided that they had not indicated previously an unwillingness to participate in future studies. Data from these follow-up interviews (N=3,787), along with data from the questionnaires (returned by 3,661 women) that accompanied them were used in the present study. Approximately, 14.6% of the sample is identified as being of African-American ancestry.

Importantly, as alcohol involvement was only queried in those individuals who reported a lifetime history of 6 of more drinking instances (N=2,904), only these subjects had informative data for twin analyses. For the seven main study variables (i.e., four personality variables, two alcohol motive variables, one AUD symptom count), the number of twins that were included in analyses (i.e., twins with no missing values on exogenous variables of age and ethnicity) ranged from 2,196 for conscientiousness (corresponding to 669 monozygotic and 519 dizygotic twin groups, including some unpaired observations, used in univariate analyses) to 2,402 for alcohol symptoms (corresponding to 674 monozygotic and 527 dizygotic twin groups).

Measures

Alcohol Use Disorder Symptoms—Comprehensive diagnostic interviews were conducted using an adaptation of the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA; Bucholz et al., 1994; Hesselbrock, Easton, Bucholz, Schuckit, & Hesselbrock, 1999), an instrument designed to assess alcohol use disorders (AUDs) and related psychiatric disorders in adults. A log-transformed count of abuse (four items) and dependence (seven items) symptoms from the Diagnostic and Statistical Manual, version 4 (DSM-IV; American Psychiatric Association, 1994) were used to assess AUD symptoms, which were summed over the 4 abuse and 7 dependence criteria. As mentioned above, only individuals reporting six or more drinking instances (*n*= 2,904) were assessed for alcohol disorder symptoms. We opted not to code light/infrequent drinkers as “0” for the AUD symptom count, given our focus on drinking motives (that are referenced to a subject's own reason for drinking, making it meaningless to assess abstainers). In instances when one twin drank and the other twin did not, only data on the twin who drank was used, thus converting the twin pair into singletons. These individual twins do not contribute to any estimates of covariance but they are included in all estimates of variance. Of the 2,904 individual twins, 301 came from a twin pair where the co-twin had a valid non-missing observation but reported drinking on less than 6 instances.

Personality—Neuroticism (mean [M] = 1.73, standard deviation [SD] = .64), conscientiousness (M=2.73, SD = .50), and agreeableness (M=2.74, SD = .47), were assessed by self-report measures from the NEO-PI (Costa & McCrae, 1985). Control (M=2.44, SD = .40), a subscale of constraint, was assessed with items from the

Multidimensional Personality Questionnaire (Tellegen, 1982). These measures were scored in accordance with recommendations by these authors.

Drinking motives—Drinking motives were collected in the self-report mailed questionnaires using the drinking motive questionnaire (DMQ; Cooper, 1994). The DMQ assess four motive factors with five items per factor (i.e., social, conformity, coping, and enhancement). These scales were scored by summing responses (ranging 1-5) across the five items per scale and then dividing the summed items by five, resulting in scores that ranged from 1-5 for each scale. Given that internal drinking motives have shown the most robust relations with personality variables (see Kuntsche et al., 2006), coping (M=.94, SD = 1.02) and enhancement (M=1.85, SD = 1.24) motives were used in the current study.

Analytic Procedure

Multigroup analyses that compared estimates between monozygotic and dizygotic twins were used to fit univariate and bivariate genetic models (Eaves, Last, Young, & Martin, 1978) using full information maximum likelihood (FIML) with robust standard errors (Satorra & Bentler, 1994) in Mplus version 6 (Muthén & Muthén, 2010). FIML allows for the analysis of data containing missing values[1]. Analyses proceeded as follows:

a. The proportion of the total variance in AUD symptoms and in each personality and motive measure that could be explained by additive genetic factors (A), environmental influences shared by members of a twin pair (C), and nonshared environmental influences (E) were estimated using univariate models.

b. Bivariate models were then fitted to the data to test the extent to which genetic factors contributed to the covariance between (i) personality and AUD symptoms; (ii) measures of personality and of motives; and (iii) measures of motives and AUD symptoms.

c. Next, in order to examine the extent to which motives accounted for the genetic covariance between the respective personality measures (i.e., neuroticism, conscientiousness, agreeableness, control) and AUD symptoms, genetic correlations between these constructs were estimated after partialling out the variance in each construct attributable to, respectively, coping and enhancement motives (as well as linear and quadratic age and African-American ethnicity). This was done by regressing out the effects of motives from both personality and AUD symptoms and analyzing the residuals in a series of bivariate twin models.

Results

Associations among personality, motives, and AUD symptoms

Table 1 presents the phenotypic correlations between personality, drinking motives, and AUD symptoms. Adjusting for age (linear and quadratic) and ethnicity, all variables were significantly correlated. The phenotypic correlations between personality and AUD symptoms were small to small-medium in magnitude (Cohen, 1977) with absolute values ranging from .13 to .20. The correlations between personality and coping motives were roughly medium in effect size, whereas the correlations between enhancement motives and personality were small in magnitude (see Table 1). The correlations between drinking motives and AUD symptoms were roughly medium in magnitude, ranging from .32 to .38.

[1]Ancillary analyses using listwise deletion yielded similar results and identical conclusions as those analyses currently presented and discussed in the manuscript.

Genetic and environmental influences on personality, motives, and AUD symptoms

With the exception of enhancement motives, significant evidence was found for genetic influences on all study constructs. As shown in Table 2, omitting enhancement motives, MZ correlations were higher than DZ correlations, suggesting the role of heritable influences[2]. Nonshared environmental influences were significant for all constructs as well. Shared environmental influences were non-significant for all constructs. Given that enhancement motives were not significantly heritable, in line with our research aims, this construct was not included in subsequent analyses. Thus, remaining analyses focused on the relationship between personality, AUD symptoms and coping motives.

Genetic correlations among personality, coping motives, and AUD symptoms

The extent to which genetic factors contributed to the bivariate relationship (i.e. covariation) between AUD symptoms, personality measures and coping motives are shown in Table 3. All personality measures demonstrated significant genetic correlations with AUD symptoms. All personality constructs, with exception of conscientiousness, also demonstrated significant genetic overlap with coping motives. Coping motives shared significant and substantial genetic overlap with AUD symptoms (see Table 3).

Genetic correlations between personality and AUD symptoms, adjusting for coping motives

Bivariate models that examined the covariance between residualized personality and AUD symptoms, after regressing out the effects of coping motives, were then fit to these data. These estimates are displayed in Table 4. After adjusting for coping motives, the significant genetic correlations (Rg=0.33) between neuroticism and AUD symptoms was reduced in magnitude (Rg=0.06) and was no longer statistically significant. Similarly, adjusting for coping motives, the genetic correlation between agreeableness and AUD symptoms were no longer statistically significant (see Tables 3 and 4). Though the genetic overlap between the other personality constructs (conscientiousness and control) and AUD symptoms remained statistically significant, the estimates were both reduced (see Tables 3 and 4).

Discussion

The link between AUDs and specific personality factors is well established. Moving further, identifying mechanisms in which personality traits, such as neuroticism, relate to negative health outcomes has been identified as a “top priority” for research (Lahey, 2009). Though previous research has suggested that the genetic diathesis for AUDs is partially mediated by genetic variation in personality (Slutske et al., 2002) and that internal drinking motives act as a mediator in the alcohol-personality relation (Cooper et al., 1995), this is the first paper, to our knowledge, to document the overlap in genetic variance between coping motives and theoretically-related personality constructs. Additionally, this paper demonstrates, for the first time, the extent to which genetic variability in coping motives contributes to genetic covariance between personality and AUD symptoms. Summaries and implications of these findings are discussed below.

[2]MZ correlations were more than twice the DZ correlations for some study constructs (e.g., control), suggesting non-additive genetic influences (i.e., dominance or epitasis; see Neale & Maes, 2002). However, the current sample lacks adequate power to detect non-additive genetic influences (see *Limitations* section).

Do genetic factors contributing to variation in personality also contribute to genetic variation in coping motives?

Current findings suggested that genetic variation in the personality traits of neuroticism, conscientiousness, agreeableness, and control significantly covaried with genetic variability in coping motives. Importantly, a substantial portion of the genetic variation in neuroticism overlapped with genetic factors contributing to variation in coping motives. These findings suggest that the robust link between neuroticism and coping motives (see Kuntsche et al., 2005) is likely due, in part, to common genetic influences.

However, consistent with other findings using this sample (Argawal et al., 2008), enhancement motives were not significantly heritable. The heritability of enhancement motives might depend on other factors. For example, in this same dataset, Kristjansson et al. (2011) found enhancement motives were not significantly heritable in non-regular smokers but were significantly heritable in regular smokers. Considering that gene by non-shared environment interactions are included in unique environmental effects on variability in ACE models, it is possible that genetic factors contribute to enhancement motives but primarily in the presence of certain environmental influences. Further, low heritability can arise through poor reliability of a phenotypic measure. The test-retest reliability of the enhancement scale in this sample could not be calculated as data were collected at one time point only, so we cannot comment on this form of reliability in this sample. Though studies examining the test-retest reliabilities of the drinking motive questionnaire are less common in general, at least one other study has documented high test-retest reliability of enhancement motives (i.e., intraclass correlation coefficient = .78 across a roughly 95-day period) using a scale that is nearly identical to the enhancement scale used in the current sample (see Grant, Stewart, O'Connor, Blackwell, & Conrod, 2007). Further, the enhancement motive scale from the DMQ demonstrated high internal consistency in the current sample (alpha = .91); this high reliability is consistent with other studies documenting the high internal reliability of the enhancement motive scale (e.g., Kuntsche, Stewart, & Cooper, 2008). Given, to our knowledge, that estimates of heritability for Cooper's well-validated drinking motive questionnaire have only been conducted in the MOAFTS dataset, it is difficult to conclude whether the heritability of enhancement motives extends to other samples with differing characteristics than those found here.

Does genetic variance in coping motives explain the genetic overlap between personality and AUD symptoms?

Previous work (e.g., Slutske et al., 2002) established a genetic link between AUDs and traits related to behavioral undercontrol and, to a lesser extent, neuroticism. Additional research has linked genetic variability in internal drinking motives, especially those related to negative moods, to the genetic diathesis for AUDs (Prescott et al., 2004; Agrawal et al., 2009). Building on these findings, the current paper suggests that the genetic overlap between neuroticism and AUDs can almost entirely be explained by the role of coping motives. Further, adjusting for coping motives, the genetic covariance between AUD symptoms and agreeableness were no longer statistically significant though the adjusted genetic correlation was similar in magnitude to the unadjusted correlation. Overall, coping motives appear to account, at least partially, for genetic overlap between AUD and personality, especially in regards to neuroticism.

Theoretical Implications

The significant genetic covariation among personality, coping motives, and AUD symptoms has several theoretical implications. Young-Wolff et al. (2009) suggested that the overlap among alcohol dependence, mood-related drinking motives, and major depression may suggest overlapping dysregulations in reward and stress pathways (see Lotrich & Pollock,

2004; Markou, Kosten, & Koob, 1998; Nellissery et al., 2003; Rao, 2006; Samochowiec et al., 2006); similar mechanisms may account for the genetic overlap between personality, coping motives, and AUDs.

Further, given that genetic variance in alcohol-related disorders shares significant genetic overlap with other substance use disorders (such as illicit drug use disorders [DUDs]; see Prescott, Maes, & Kendler, 2005, for a general review and Kendler, Myers, & Prescott, 2007 for a recent empirical paper), and personality traits similar to those assessed here appear to relate primarily to abuse and dependence symptoms of illicit drugs (e.g., cannabis) through genetic factors (Agrawal, Jacobson, Prescott, & Kendler, 2004), it may be that similar mechanisms broadly contribute to overlap between AUDs, DUDs, personality, and substance use motives. Future studies should examine the extent to which that shared genetic overlap between AUDs, DUDs, and personality may be accounted for genetic variability in motives for substance use. Finally, considering that personality and coping motives appear to be important developmental covariates of problematic alcohol involvement (Littlefield, Sher, & Wood, 2009; Littlefield, Sher, & Steinley, 2010; Littlefield et al., 2010; Littlefield, Sher, & Wood, 2010), genetic overlap among these constructs should be considered from a developmental perspective in future studies.

Limitations

Several limitations of the current study should be noted. The current sample was restricted to young adult female twin-pairs from the Midwest so findings need replication in samples that include men and individuals from other settings and age ranges. The relatively modest sample size precluded the examination of other models (such as non-additive genetic models).

Phenotypic relations between personality and AUD symptoms in this sample generally reflected small effect sizes. Thus, though genetic correlations between AUD symptoms and personality ranged in absolute values from 0.33-0.50, phenotypic correlations ranged from 0.13-.20. . Therefore, other unmeasured genetically-influenced variables (e.g., level of response to alcohol; Schuckit & Smith, 1996) besides personality may contribute to both phenotypic and genetic variability in AUDs.

As noted elsewhere (e.g., Littlefield et al., 2010), though drinking motives are often theoretically discussed and empirically examined as mediators of the personality-alcohol involvement relation (see Kuntsche et al., 2006 for a broad review), the data presented in this paper are correlational in nature. Thus, though the current paper documents the genetic correlations among personality, drinking motives, and alcohol-related outcomes, causal relations cannot be determined. For example, though adjusting neuroticism and AUD symptoms for coping motives results in a low-magnitude, non-significant genetic correlation, it does not necessarily confirm this to be the mechanism of its effect. Though beyond the scope of the current paper, future research could investigate alternative models to those presented here.

Though this study may document the genetic overlap between personality, coping motives, and AUD symptoms, genetic variability in complex phenotypes, such as neuroticism, may be comprised of small (i.e., less than 1%) additive effects arising from many loci (e.g., Shifman et al., 2007; Terracciano et al., 2008). Thus, the shared genetic variability among personality, coping motives, and AUDs is most likely comprised of small effects from numerous loci and large samples will most likely be needed to determine which common genetic variants contribute to these phenotypes (see Shifman et al., 2007; Terracciano et al., 2008).

In addition, there are several assumptions made in behavioral genetic studies that, if violated, may lead to biases in estimating parameter estimates and compromise the validity of finding. Heritability estimates may be inflated (and environmental influences diminished) if the equal-environment assumption is violated (i.e., if non-genetic factors cause MZ twins to be more similar than DZ twins; Scarr, 1968), if epitasis is present but ignored (i.e., gene-by-gene interactions; Rao, Morton, & Yee, 1974), or if there is the presence of an AxC interaction (Jinks & Fulker, 1970). However, it should also be noted that the current study allowed for common environment (despite being non-significantly different from zero for all phenotypes), which is commonly fixed to zero but can lead to the inflation of the additive genetic influence. Similarly, the presence of an AxE interaction may inflate estimates of unshared environmental influences. On the other hand, heritability estimates may be diminished (and environmental influences inflated) if there is assortative mating (Gottesman, 1963) or if there is heterogeneity in the influence of environmental factors across groups being studied (e.g., linear changes in the influence of common environment across social classes or personality types; Morton, 1974). Extended family designs (e.g., stealth design, cascade design; see Keller, Medlan, & Duncan, 2009) can be used to overcome some of the aforementioned assumptions, but data from other family members (e.g., parents) were not available for analyses in the current study.

The five-factor traits extraversion (E) and openness to experience (O) were not included for several reasons. First, recent meta-analytic results regarding the relation between five-factors of personality and alcohol outcomes suggests that these two constructs are not significantly related to alcohol outcomes (see Malouff et al., 2007). Second, especially in regards to openness to experience, these personality constructs are not typically considered to be especially relevant to the nomological network of internal drinking motives, personality, and AUD symptoms (see Kuntsche et al., 2006). Third, in consideration of the aforementioned points, we believed that including these constructs would add length and complexity to the paper for very little empirical or theoretical benefit. Consistent with the larger literature, the phenotypic correlation between AUD symptoms and E was small and non- significant ($r = 0.01$, $p = 0.53$) as was the correlation between AUD symptoms and O ($r = 0.01$, $p = 0.57$) in the current sample. Given that a major aim of the paper is to estimate the extent to which internal drinking motives account for the overlap between personality and AUD symptoms, the lack of overlap demonstrated undermined the justification for further analyses.

Though this study was largely consistent with Slutske et al.'s (2002) previous findings regarding the overlap between personality and the genetic risk for alcohol dependence, we found significant genetic overlap between neuroticism and AUD symptoms in this sample of women. Slutske et al. (2002) concluded that genetic variance in negative emotionality was only significantly related in men. There are several possible reasons for the divergent results between studies that are discussed below. The average age, age range, and geographical location of participants in the Slutske et al. study (for men, average age = 42.7, age range 28-89 years; for women, average age = 44.8, age range 27-90; residing in Australia) is divergent from the current sample of women (mean age 22 years, range 18–29, from the United States). Thus, a greater proportion of the sample used by Slutske et al. (2002) had passed through the period of greatest risk for high alcohol use and abuse, rather than being in the midst of this period (i.e., emerging adulthood; Arnett, 2000); these differences may have contributed to somewhat disparate findings. Further, Slutske et al. (2002) used a sum of TPQ Harm Avoidance (TPQ; Cloninger, Przybeck, & Svrakic, 1991) and EPQ-R Neuroticism (EPQ-R; Eysenck, Eysenck, & Barrett, 1985) whereas neuroticism from the NEO-PI (Costa et al., 1985) was used for the current study. Though these measures ostensibly measure a similar constructs (i.e., the tendency to experience negative emotions), correlations in a separate data set (see Sher, Walitzer, Wood, & Brent, 1991, for more

details) demonstrated that the NEO-short form measure of neuroticism correlates with a short-form measure of TPQ harm avoidance at .61 and EPQ neuroticism at .78, suggesting these measures are strongly related though not isomorphic. Further, the NEO-PI measure of neuroticism includes a facet measure of impulsiveness. Given the robust relation between impulsiveness and AUDs (see Littlefield & Sher, 2010 for a thorough discussion), it is possible that a full NEO PI Neuroticism scale would more strongly relate to AUD than the measures used by Slutske et al. (2002) [3]. Thus, the differences in sample characteristics as well as measures may have contributed to divergent findings involving negative emotionality/neuroticism.

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TABLE 1

Phenotypic correlations between AUD symptoms, drinking motives, and personality

	1	2	3	4	5	6
1. AUD Symptoms						
2. Coping Motives	0.38					
3. Enhancement Motives	0.32	0.56				
4. Neuroticism	0.13	0.32	0.07			
5. Conscientiousness	-0.17	-0.20	-0.10	-0.30		
6. Agreeableness	-0.16	-0.20	-0.07	-0.36	0.26	
7. Control	-0.20	-0.20	-0.13	-0.11	0.48	0.26

Note. AUD = Alcohol use disorder. No correction was made for multiple testing to the reported significance levels. All estimates significant at $p<.001$ with the exception enhancement motives with agreeableness ($p = .03$). All correlations adjusted for age (linear and quadratic) and ethnicity.

Table 2

Parameter estimates (95% confidence intervals) for the ACE models.

	MZ correlation	DZ correlation	Additive Genetic	Shared Environmental	Non-shared Environmental
Neuroticism	0.44	0.25	0.39 (0.18, 0.58)	0.04 (-0.16, 0.22)*	0.57 (0.53, 0.60)
Agreeableness	0.47	0.22	0.44 (0.39, 0.49)	0.00	0.56 (0.53, 0.58)
Conscientiousness	0.42	0.21	0.41 (0.35, 0.45)	0.00	0.59 (0.56, 0.62)
Control	0.30	0.05	0.26 (0.18, 0.32)	0.00	0.74 (0.71, 0.78)
Coping	0.27	0.07	0.23 (0.16, 0.29)	0.00	0.77 (0.73, 0.80)
Enhancement	0.37	0.28	0.14 (-0.10, 0.36)*	0.18 (-0.01, 0.36)*	0.68 (0.63, 0.72)
AUD Symptoms	0.39	0.15	0.37 (0.31, 0.42)	0.00	0.63 (0.60, 0.66)

Note. AUD = Alcohol use disorder. All estimates adjusted for age (linear and quadratic) and ethnicity. All monozygotic (MZ) and dizygotic (DZ) correlations were significant at $p<.05$ except DZ coping and control correlation. Instances where MZ correlation is less than the additive genetic estimate is due to rounding error.

*As denoted by a confidence interval including 0.00, this parameter is not significant.

Table 3

Genetic correlations (with 95% confidence intervals) among personality, drinking motives, and AUD symptoms.

	AUD Symptoms	Coping Motives
Coping Motives	0.82 (0.78, 0.88)	-
Neuroticism	0.33 (0.07, 0.50)	0.68 (0.66, 0.68)
Conscientiousness	-0.40 (-0.15, -0.62)	-0.32 (0.02, -0.70)*
Agreeableness	-0.45 (-0.31, -0.59)	-0.59 (-0.40, -0.77)
Control	-0.50 (-0.32, -0.66)	-0.38 (-0.15, -0.61)

Note. AUD = Alcohol use disorder. All correlations adjusted for age (linear and quadratic) and ethnicity.

*As denoted by a confidence interval including 0.00, this parameter is not significant

Table 4

Genetic correlations (with 95% confidence intervals) between AUD symptoms and personality, adjusting for coping motives.

	Adjusting for Coping Motives
	AUD Symptoms
Neuroticism	0.06 (-0.36, 0.32)*
Conscientiousness	-0.29 (-0.02, -0.59)
Agreeableness	-0.31 (0.03, -0.69)*
Control	-0.36 (-0.14, -0.59)

Note. AUD = Alcohol use disorder. All correlations adjusted for age (linear and quadratic) and ethnicity.

*As denoted by a confidence interval including 0.00, this parameter is not significant.