A Multivariate Behavior Genetic Investigation of Dual-Systems Models of Alcohol Involvement

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ABSTRACT. Objective: Dual-systems models hypothesize that individuals who tend to be drawn to risky behavior and are low in self-control are at greatest risk for alcohol use disorder (AUD). Importantly, these models assume that behavioral approach tendencies and self-control are distinct. This study investigated hypotheses and assumptions central to dual-systems models. **Method:** Participants were 3,509 members of a national twin registry (58% female). Structured interviews assessed alcohol use and AUD symptoms. Self-report questionnaires assessed individual differences in approach tendencies, namely for general risky behavior (sensation seeking) and substance use (positive expectancies), and behavioral control. Regression models tested nonadditive, interaction effects on alcohol involvement, as proposed by the dual-systems model. Multivariate behavior genetic models investigated the incremental validity of these interaction effects and whether approach tendencies and behavioral control explain distinct variance in alcohol involvement. **Results:** In regression models, we found interaction effects consistent with the dual-systems model for women but in the opposite direction for men. After accounting for additive main effects in behavior genetic models, however, these interaction effects played a negligible role phenotypically and genetically. Further, sensation seeking and positive expectancies explained phenotypic and genetic variance in alcohol involvement that was distinct from behavioral control. Behavioral control, however, did not explain distinct variance in alcohol involvement. **Conclusions:** Contrary to dual-systems models, this study suggests that all of the variance in alcohol involvement explained by behavioral control is also shared with the tendency to engage in risky behavior (sensation seeking) and substance use (positive expectancies). Further, interaction effects central to dual-systems models failed to explain additional variance beyond basic main effects. Thus, more parsimonious models may better explain AUD. (*J. Stud. Alcohol Drugs, 79,* 617–626, 2018)

WITHIN THE LAST DECADE, increasing attention has been devoted to investigating dual-systems models of broad constructs such as decision making (Kahneman, 2003, 2011; Metcalfe & Mischel, 1999) and risky behavior (Steinberg, 2010), and specific constructs such as alcohol use disorder (AUD) (Houben & Wiers, 2009; Magid et al., 2007; Stacy & Wiers, 2010; Thush et al., 2008). Dual-systems models attribute AUD to an interplay between two complementary systems, a bottom-up (e.g., mesolimbic; Koob & Le Moal, 2008) emotion-based system that is characterized by approach tendencies, and a top-down (e.g., prefrontal; Goldstein & Volkow, 2011) cognitive-based system that is characterized by behavioral control. Specifically, those who tend to be drawn to risky behavior and are low in behavioral control are hypothesized to be at greatest risk for alcohol problems. Thus, dual-systems models assume that interaction effects capturing this interplay cannot be explained by additive, main effects of these constructs. Further, these models posit that each system serves as a distinct risk process for AUD, thus warranting the inclusion and delineation of both constructs. These fundamental assumptions are often overlooked, but it is important to clarify whether interaction models better explain AUD than simpler models and whether these are actually distinct risk processes. The current study used a twin sample to investigate assumptions and hypotheses of the dual-systems model of AUD.

The constructs represented in dual-systems models correspond to personality taxonomies (Harden & Tucker-Drob, 2011; Quinn & Harden, 2013; Shulman et al., 2015). Gray's behavioral approach system has informed many conceptualizations of approach-based tendencies (Gray, 1972, 1990), including sensation seeking, defined as the tendency to seek "varied, novel, complex, and intense sensations and experiences" (Zuckerman, 1994, p. 27). Further, the association between behavioral approach and alcohol use is mediated by domain-specific conceptualizations, namely alcohol expectancies (Wardell et al., 2012). Behavioral control has been measured by personality facets related to conscientiousness and can be defined by Tellegen's MPQ Control Scale, which closely resembles (lack of) planning from the UPPS impul-

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sivity scale and assesses the "tendency to be planful, reflective, and careful rather than impulsive" (Shiner et al., 2002, p. 1172; Tellegen, 1982; Tellegen & Waller, 2008; Whiteside & Lynam, 2001). Of note, factor analyses suggest these constructs load onto separate impulsivity facets (sensation seeking and lack of planning), which are moderately correlated with each other (r = .34) and with alcohol involvement (r = .17–.37) (Coskunpinar et al., 2013; Smith et al., 2007; Whiteside & Lynam, 2001). Conceptually, these constructs are also not mutually exclusive, as the tendency to carefully plan ahead is unlikely to result in the pursuit of risky behavior and subsequent consequences. Given the relatedness of these personality constructs, it is unclear whether they represent distinct risk processes for alcohol involvement.

Additive genetic factors explain a significant proportion of variance in alcohol involvement (50%–61%; Kendler & Prescott, 2006), sensation seeking (48%–63%; Koopmans et al., 1995), and behavioral control (49%; Hur & Bouchard Jr., 1997). Few studies have investigated common genetic factors among approach- and control-based personality measures. Using a small sample of twins reared apart (n = 106), Hur and Bouchard Jr. (1997) attributed 55% of the genetic variance in behavioral control to sensation seeking. Similarly, analyzing the same twin sample used in the current study, we found that sensation seeking and behavioral control share genetic factors in men and environmental factors in women (Ellingson et al., 2013a). We are unaware of any work that has partitioned the genetic variance in AUD into approach- or control-based measures, but prior work has found shared genetic factors for AUD and a measure that assessed both sensation seeking and behavioral control (Slutske et al., 2002).

The present study extends this work to investigate the nature of the relationship of approach tendencies (sensation seeking, positive expectancies) and behavioral control with alcohol involvement. Generalized linear models tested interaction effects between these constructs on alcohol involvement. There has been mixed empirical support for these interaction effects, with considerable variation across study samples (e.g., adolescence to middle adulthood) and methodological characteristics (e.g., self-report and behavioral measures). Behavior genetic models were then conducted to decompose alcohol involvement into variance specific to each personality measure, variance shared by both personality measures, an interaction of these measures, and other factors (i.e., residual variance). The most straightforward prediction from the dual-systems model would be that these constructs explain distinct variance in alcohol involvement via main effects and produce significant, nonadditive interaction effects that are unexplained by main effects. If these constructs explain some of the same variance in alcohol involvement via main effects with the presence of significant interaction effects, this would still be consistent with the dual-systems model but would suggest a more complex relationship than currently proposed. If main effects of these constructs explain the same variance in alcohol involvement and interaction effects are negligible, this would be difficult to reconcile with the dual-systems model.

Method

Participants

Members of the national community-based Australian Twin Registry (ATR) Cohort II participated in a study that primarily focused on gambling addiction (80.4% recruitment rate; for more details, see Slutske et al., 2009). In 2004–2007, 4,764 participants completed a self-report, paper-pencil questionnaire about personality characteristics and a semi-structured psychiatric telephone interview that assessed alcohol use and AUD symptoms. Participants who denied ever having a drink (i.e., lifetime abstainers; n = 140) were excluded from analyses in the current article, as the conceptual model of impulse control moderating effects on alcohol problems does not apply to these individuals. Further, sex-limited expression of genetic risk was beyond the scope of the current article; therefore, opposite-sex twin pairs were excluded from analyses (n = 1,115). Participants in the current study were 3,509 individuals (58.0% female, M_{age} = 37.6, SD = 2.3) from 1,385 complete twin pairs (monozygotic females [MZF] = 485, MZ males [MZM] = 333, dizygotic females [DZF] = 349, DZ males [DZM] = 218) and 739 incomplete pairs. Trained lay interviewers conducted structured assessments and were supervised by a clinical psychologist; interviews were recorded and reviewed at random to ensure acceptable quality. The Institutional Review Boards at the University of Missouri and the Queensland Institute of Medical Research approved all data collection methods.

Measures

Personality. Participants in the ATR were administered the 40-item Zuckerman Sensation Seeking Scale (Zuckerman et al., 1964). Sensation seeking, as conceptualized by Zuckerman and dual-systems researchers, assesses dopaminergic approach-based tendencies underlying risky behavior (Steinberg et al., 2008; Zuckerman & Kuhlman, 2000). Six items from the Zuckerman Sensation Seeking Scale query substance use and were used to assess positive expectancies (example items: "I feel best after taking a couple drinks," "I often like to get high [drinking liquor or smoking marijuana]," and "Keeping the drinks full is the key to a good party"). The remaining 34 items were used to assess sensation seeking (example items: "I like to have new and exciting experiences and sensations even if they are a little frightening, unconventional, or illegal," "I am very restless if

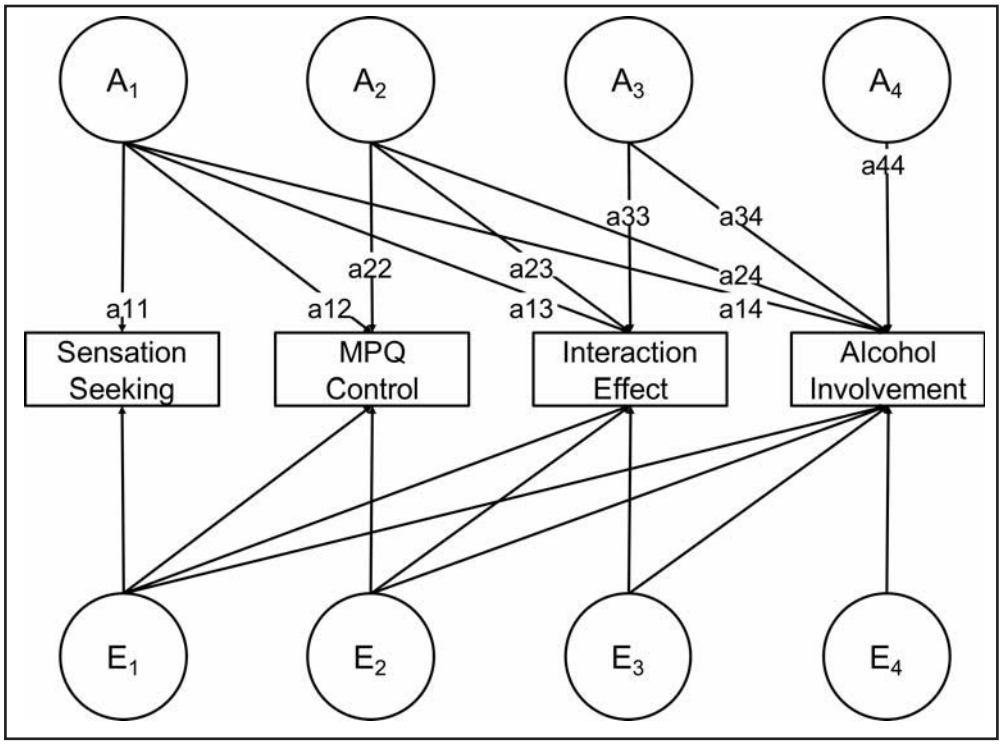

FIGURE 1. A quadrivariate structural equation model quantifying the extent to which variance in alcohol involvement (alcohol quantity × frequency, heavy drinking, max drinks, and alcohol use disorder symptoms). Separate models were run for sensation seeking and positive expectancies. For simplicity, only one individual from a twin pair is illustrated for the sensation-seeking model. Latent variables represent the genetic and environmental variance for the total main effect explained by sensation seeking (A_1, E_1); the remaining main effect variance explained by MPQ Control (A_2, E_2); the remaining variance explained by an interaction effect of sensation seeking and MPQ Control (A_3, E_3); and all variance that is unexplained by these main and interaction effects (A_4, E_4). In this model, sensation seeking was entered first; thus, the genetic variance in alcohol involvement that is explained by MPQ Control (represented by A_2) and cannot be explained by sensation seeking (represented by A_1) is: [(a22 × a24)/(a11 × a14 + a22 × a24 + a33 × a34 + a44 x a44)]. To determine the proportion of variance in alcohol involvement that is specific to sensation seeking, this model was re-run with MPQ Control entered first. The proportion of genetic variance attributable to nonadditive interaction effects, after accounting for main effects, is: [(a33 × a34)/(a11 × a14 + a22 × a24 + a33 × a34 + a44 x a44)]. Similar estimates were derived for the environmental variance in alcohol involvement.

I have to stay at home for any length of time," "I would like to try parachute jumping"). Both measures were included so that findings could be compared to dual-systems models of general, risky behavior (e.g., Steinberg et al., 2008) and AUD (e.g., Houben & Wiers, 2009). The lower-bound internal consistency (ω; McDonald, 1999) was .91 for sensation seeking and .88 for positive expectancies.

Participants were also administered the Multidimensional Personality Questionnaire (MPQ; Tellegen, 1982), from which the 20-item Control scale was used to assess behavioral control (ω = .92). Of note, Tellegen and his colleagues have described the MPQ Control scale as a measure of "behavioral control" (example items: "I tend to value and follow a rational, 'sensible' way of doing things," "I am very level headed and always like to keep my feet on the ground," "Before I get into a new situation I like to find out what to expect from it,") (Shiner et al., 2002, p. 1172).

Alcohol involvement. Participants were queried about the 12-month period in which they drank the most. This period included the past year for 57.6% of participants, and early adulthood for 42.4% of participants (M_{age} = 22.7, SD = 5.7). Thus, the lifetime levels of heaviest drinking were assessed for all participants.

Peak alcohol use was assessed by four items: (a) drinking frequency (days per year consuming at least one drink), (b) typical drinking quantity (number of drinks per drinking occasion), (c) intoxication frequency (days per year had slurred speech, unsteady gait when drinking), and (d) hangover frequency (days per year did not feel well the day after drinking). These items have demonstrated adequate test–retest reliability (Ellingson et al., 2013b) and were combined into two measures of alcohol use. A measure of drinking volume was computed as the drinking frequency multiplied by the typical quantity, labeled Alcohol QF (M = 706.3, SD = 918.6 drinks per year during peak drinking). Heavy drinking was computed as average frequency of intoxication and hangover (M = 30.2, SD = 51.3 heavy drinking days per year). In addition, a measure of the most drinks ever consumed in a 24-

hour period, labeled Max Drinks, was used (M = 15.7, SD = 12.5 drinks).

Alcohol use disorder. The AUD section from the World Health Organization Composite International Diagnostic Interview (CIDI) was used to assess lifetime AUD symptoms (Robins et al., 1988). The current study used a lifetime AUD symptom count according to the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-5; American Psychiatric Association, 2013), excluding craving, which was not assessed in this version of the CIDI. These symptoms demonstrated adequate internal consistency in the current sample (ω = .94). On average, participants endorsed 1.3 lifetime AUD symptoms (SD = 1.8). All alcohol involvement and AUD measures were log-transformed to adjust for nonnormality.

Analytic procedures

Phenotypic analyses. Pearson correlation coefficients among personality and alcohol measures were estimated using Mplus, Version 7.4 (Muthén & Muthén, 1998). Regression models testing interaction effects were conducted using PROC GENMOD in SAS Version 9.3 (SAS Institute Inc., Cary, NC) to investigate interaction effects hypothesized by dual-systems models (Houben & Wiers, 2009; Thush et al., 2008). Analyses accounted for the nonindependence of observations obtained from members of the same family. Personality measures were mean-centered to reduce the influence of collinearity. Regression models included quadratic effects to control for spurious interactions (Lubinski & Humphreys, 1990). For all analyses, outliers were dropped if they had high leverage (greater than twice the sample mean for leverage) and high influence (Cook's $D > \frac{4}{n-2-1}$) on results (Chatterjee & Hadi, 1986), and men and women were analyzed separately. Models were conducted separately for sensation seeking and positive expectancies.

Univariate behavioral genetic analyses. Structural equation models (SEMs) were fitted in Mplus to estimate the proportion of variance in personality and alcohol phenotypes attributable to additive genetic (A), shared environmental (C), and unique environmental (E) factors. These models use genetically informed data and impose variance and covariance constraints, from which latent variables are assumed to represent the biometrical (ACE) factors for each personality measure (e.g., A is correlated 1.0 for MZ and .5 for DZ pairs). Thus, behavioral genetic models estimated the covariances between MZ-twin pairs (calculated as A + C) and DZ-twin pairs (calculated as .5 × A + C) and the proportion of phenotypic variance attributable to the biometrical factors.

Multivariate behavioral genetic analyses. Quadrivariate SEMs were fitted to decompose the phenotypic variance in alcohol involvement into genetic and environmental variance explained by main and interaction effects of the personality measures. Models were conducted separately for sensation seeking and positive expectancies on the four measures of alcohol involvement (alcohol QF, heavy drinking, max drinks, AUD symptoms), resulting in eight sets of models. Figure 1 shows a model run with sensation seeking. Alcohol involvement was decomposed into genetic and environmental latent variables that represented the total variance due to the main effect of sensation seeking or positive expectancies (A_1, E_1); the remaining variance explained by the main effect of MPQ Control (A_2, E_2); the remaining variance explained by nonadditive interaction effects hypothesized by dual-systems models (A_3, E_3); and variance unexplained by these main and interaction effects (A_4, E_4). Thus, A_2–A_4 and E_2–E_4 are residual variables that are unexplained by variables with a lower subscript (e.g., A_2 is variance unexplained by A_1; A_3 is variance unexplained by A_2 and A_1).

To decompose the variance in alcohol involvement, we used the Cholesky approach, from which a 4 × 4 triangular matrix containing parameter estimates was derived for each of the A, C, and E factors (Loehlin, 1996; Neale & Maes, 2005). Each matrix contained 10 elements, 4 path coefficients on the diagonal corresponding to the variance for each phenotype and 6 on the off-diagonal corresponding to the covariance between each pair of phenotypes. Further, path analysis of latent variable loadings was implemented using the tracing rules in a MODEL CONSTRAINT command in Mplus. (Loehlin, 2004). This approach attributed the variance in alcohol involvement (the rightmost variable in Figure 1) to latent variables in a stepwise manner (from left to right in Figure 1). With this consideration, the model shown in Figure 1 was also run with MPQ Control as the leftmost variable. Thus, models estimated the variance distinct to MPQ Control and approach tendencies (sensation seeking, positive expectancies), as well as the common variance explained by both measures (e.g., due to MPQ Control and sensation seeking).

Results

Phenotypic analyses

Descriptive statistics for personality, interaction terms, and alcohol involvement are displayed in Table 1. MPQ Control was moderately correlated with sensation seeking and positive expectancies. Correlations for alcohol involvement measures were strongest for positive expectancies, moderate for sensation seeking, and weak for MPQ Control. Alcohol involvement was also weakly correlated with interaction terms in women, and associations were negligible in men.

Participants with missing data or excessive influence and leverage on regression model coefficients were dropped, resulting in 1,905 women and 1,272 men analyzed in regression models. Regression coefficients are displayed in Table 2. For the sensation-seeking models, all alcohol phenotypes were associated with MPQ Control and sensation seeking.

Table 1. Phenotypic correlations and descriptive statistics for measures of personality and alcohol involvement

Variable	1.	2.	3.	4.	5.	6.	7.	8.	9.
Personality									
1. MPQ Control		-.45 (.02)	-.34 (.02)	-.22 (.02)	-.21 (.02)	-.18 (.02)	-.22 (.02)	-.24 (.02)	-.23 (.02)
2. Sensation seeking	-.38 (.03)		.47 (.02)	-.13 (.02)	N.A.	.27 (.02)	.27 (.02)	.31 (.02)	.24 (.02)
3. Positive expectancies	-.27 (.03)	.44 (.02)		N.A.	-.24 (.02)	.50 (.02)	.49 (.02)	.49 (.02)	.47 (.02)
Interaction terms									
4. MPQ Control × Sensation Seeking	-.16 (.03)	-.06 (.03)	N.A.		N.A.	-.07 (.02)	-.10 (.02)	-.07 (.02)	-.06 (.02)
5. MPQ Control × Positive Expectancies	-.11 (.03)	N.A.	-.14 (.03)	N.A.		-.07 (.02)	-.10 (.02)	-.07 (.02)	-.11 (.02)
Alcohol involvement									
6. Alcohol QF	-.15 (.03)	.17 (.03)	.44 (.02)	-.002 (.03)	-.03 (.03)		.71 (.01)	.59 (.02)	.51 (.02)
7. Heavy drinking	-.17 (.03)	.21 (.03)	.44 (.02)	-.03 (.03)	-.04 (.03)	.73 (.01)		.57 (.02)	.61 (.01)
8. Max drinks	-.22 (.03)	.26 (.03)	.44 (.02)	.02 (.03)	.02 (.03)	.60 (.02)	.53 (.02)		.52 (.02)
9. AUD symptoms	-.18 (.03)	.12 (.03)	.40 (.02)	.01 (.03)	-.03 (.03)	.52 (.02)	.56 (.02)	.49 (.02)	
n	1,303 \ 1,936	1,304 \ 1,934	1,304 \ 1,934	1,303 \ 1,936	1,303 \ 1,936	1,474 \ 2,035	1,474 \ 2,035	1,471 \ 2,033	1,474 \ 2,035
M	0.57 \ 0.65	0.47 \ 0.36	0.41 \ 0.29	0.03 \ 0.02	0.02 \ 0.02	6.17 \ 5.28	2.58 \ 1.79	3.01 \ 2.24	0.79 \ 0.48
SD	0.21 \ 0.20	0.15 \ 0.16	0.28 \ 0.26	0.04 \ 0.03	0.07 \ 0.05	1.48 \ 1.63	1.7 \ 1.66	0.64 \ 0.61	0.68 \ 0.60
Cronbach's α	.81	.84	.69	N.A.	N.A.	N.A.	N.A.	N.A.	.79
McDonald's ω	.92	.93	.88	N.A.	N.A.	N.A.	N.A.	N.A.	.94

Notes: Correlations for males are presented on the lower diagonal, females on the upper diagonal. Standard errors for phenotypic correlations are provided in parentheses. Internal consistency is presented for the full sample. MPQ = Multidimensional Personality Questionnaire; alcohol QF = typical quantity × typical frequency of use; heavy drinking = composite of hangover and intoxication frequency; max drinks = most drinks ever consumed in a 24-hour period in lifetime; AUD symptoms = DSM-5 alcohol use disorder symptom count, without craving symptom; N.A. = not applicable.

For the positive expectancies models, MPQ Control was not associated with alcohol QF or heavy drinking in men, but all main effects of positive expectancies were significant at $p < .001$. There were interaction effects of MPQ Control and sensation seeking in women for heavy drinking and lifetime AUD symptoms. The nature of these interactions was consistent with the dual-systems model, with associations between alcohol involvement and sensation seeking tempered among women highest in behavioral control. For men, there were interaction effects involving sensation seeking on alcohol QF and positive expectancies on max drinks, but in the opposite direction proposed by the model. That is, heavier alcohol involvement occurred among men high in sensation seeking and high in behavioral control. Of note, there were no statistically significant interaction effects when analyses excluded outliers, suggesting that these effects are not robust against influential cases.

Twin correlations

Table 3 displays twin correlations for MZ and DZ pairs. Cross-trait, cross-twin correlations for alcohol involvement were consistently stronger for MZ pairs than DZ pairs for MPQ Control (MZ = .07–.21, DZ = .01–.14), sensation seeking (MZ = .08–.22, DZ = -.01–.22), and positive expectancies (MZ = .18–.41, DZ = .04–.25). Twin correlations appeared to be weaker for MPQ Control, suggesting that familial factors may account for less covariation between

Table 2. Regression coefficients of main and interaction effects for behavioral control, sensation seeking, and positive expectancies on alcohol involvement

	Sensation-seeking models			Positive expectancies models		
Alcohol phenotype	MPQ Control	Sensation seeking	Interaction	MPQ Control	Positive expectancies	Interaction
Women						
Alcohol QF	-0.81 (0.21)***	2.11 (0.25)***	-2.65 (1.38)	-0.16 (0.16)	3.39 (0.14)***	0.53 (0.58)
Heavy drinking	-1.22 (0.22)***	1.98 (0.27)***	-5.36 (1.37)***	-0.42 (0.19)*	3.16 (0.14)***	0.33 (0.65)
Max drinks	-0.41 (0.08)***	0.87 (0.10)***	-1.05 (0.57)	-0.25 (0.06)***	1.14 (0.05)***	-0.03 (0.23)
Lifetime AUD symptoms	-0.55 (0.08)***	0.53 (0.10)***	-1.74 (0.55)**	-0.22 (0.07)**	1.04 (0.05)***	-0.22 (0.25)
Men						
Alcohol QF	-1.08 (0.27)***	1.18 (0.33)***	2.74 (1.29)*	-0.19 (0.24)	2.78 (0.20)***	-0.44 (0.65)
Heavy drinking	-0.83 (0.30)**	2.36 (0.36)***	-1.01 (1.59)	-0.29 (0.25)	3.06 (0.20)***	-0.62 (0.70)
Max drinks	-0.47 (0.11)***	0.99 (0.14)***	0.19 (0.51)	-0.34 (0.10)***	1.18 (0.09)***	0.59 (0.27)*
Lifetime AUD symptoms	-0.60 (0.11)***	0.41 (0.14)**	-0.08 (0.65)	-0.33 (0.10)***	1.01 (0.07)***	0.21 (0.30)

Notes: Regression coefficients are standardized. Standard errors are in parentheses. MPQ = Multidimensional Personality Questionnaire; alcohol QF = typical quantity × typical frequency of use; heavy drinking = composite of hangover and intoxication frequency; max drinks = most drinks ever consumed in a 24-hour period in lifetime; AUD symptoms = DSM-5 alcohol use disorder symptom count, without craving symptom. Models were conducted separately for sensation seeking and positive expectancies.
*$p < .05$; **$p < .01$; ***$p < .001$.

TABLE 3. Within-trait and cross-trait twin correlations between measures of personality and alcohol involvement

Alcohol phenotype	MZF (659 pairs)	MZM (487 pairs)	DZF (542 pairs)	DZM (436 pairs)
MPQ Control				
MPQ control	.36 (.04)***	.44 (.05)***	.21 (.05)***	.17 (.07)*
Sensation seeking	.20 (.03)***	.23 (.04)***	.16 (.04)***	.10 (.05)
Positive expectancies	.16 (.03)***	.21 (.04)***	.14 (.04)***	.02 (.05)
Alcohol QF	.13 (.03)***	.12 (.04)**	.10 (.04)**	.07 (.05)
Heavy drinking	.18 (.03)***	.15 (.04)***	.11 (.04)**	.07 (.05)
Max drinks	.17 (.03)***	.21 (.03)***	.12 (.04)**	.05 (.05)
Lifetime AUD symptoms	.15 (.03)***	.16 (.04)***	.14 (.04)***	.08 (.05)
Sensation seeking				
Sensation seeking	.50 (.03)***	.44 (.04)***	.29 (.05)***	.32 (.07)***
Positive expectancies	.29 (.03)***	.24 (.04)***	.20 (.04)***	.17 (.06)**
Alcohol QF	.22 (.03)***	.11 (.04)**	.19 (.04)***	.08 (.05)
Heavy drinking	.22 (.03)***	.12 (.04)**	.22 (.04)***	.05 (.05)
Max drinks	.24 (.03)***	.21 (.03)***	.19 (.04)***	.09 (.05)
Lifetime AUD symptoms	.17 (.03)***	.09 (.04)*	.15 (.04)***	.01 (.05)
Positive expectancies				
Positive expectancies	.46 (.04)***	.47 (.04)***	.26 (.05)***	.27 (.07)***
Alcohol QF	.39 (.03)***	.36 (.03)***	.25 (.04)***	.16 (.05)**
Heavy drinking	.41 (.03)***	.35 (.03)***	.21 (.04)***	.18 (.05)***
Max drinks	.38 (.03)***	.40 (.03)***	.21 (.04)***	.14 (.05)**
Lifetime AUD symptoms	.34 (.03)***	.30 (.03)***	.13 (.04)**	.17 (.05)**

Notes: Standard errors are provided in parentheses. MZF = monozygotic females; MZM = monozygotic males; DZF = dizygotic females; DZM = dizygotic males; MPQ = Multidimensional Personality Questionnaire; Alcohol QF = typical quantity × typical frequency of use; heavy drinking = composite of hangover and intoxication frequency; max drinks = most drinks ever consumed in a 24-hour period in lifetime; AUD symptoms = DSM-5 alcohol use disorder symptom count, without craving symptom.
*$p < .05$; **$p < .01$; ***$p < .001$.

alcohol involvement and MPQ Control, relative to sensation seeking and positive expectancies.

Univariate models

Table 4 displays estimates of the proportion of variance in personality and alcohol phenotypes associated with genetic and environmental factors. A significant proportion of variance in all personality and alcohol phenotypes was attributed to A, except sensation seeking in men (.25, $p = .12$). Of note, E explained the largest proportion of variance in all phenotypes, except for max drinks in men, and C accounted for a negligible proportion of variance in all phenotypes, except max drinks in women (.20, $p = .045$). Therefore, multivariate models constrained the effect of C to 0.

Multivariate models

Multivariate models decomposed the proportion of phenotypic variance in alcohol involvement into each personality measure, including additive and nonadditive variance, and unexplained variance (see the left panel of Figures 2 and 3). MPQ Control accounted for a negligible proportion of variance for all alcohol phenotypes after accounting for sensation seeking or positive expectancies (.001–.06 [*SE*s = .001–.03], $p = .06$–.89), except for lifetime AUD when modeled with sensation seeking in women (.04 [*SE* = .02], $p = .04$). After accounting for MPQ Control, sensation seeking accounted for a small but statistically significant proportion of variation for all alcohol phenotypes in women (.03–.06 [*SE*s = .01–.02], $p < .05$), but only for heavy drinking in

TABLE 4. Estimates of the proportion of variance in measures of personality and alcohol involvement attributable to genetic and environmental factors

	Women			Men		
Phenotype	Additive genetics	Shared environment	Unique environment	Additive genetics	Shared environment	Unique environment
Personality						
MPQ Control	.32 (.13)*	.05 (.11)	.64 (.04)***	.44 (.04)***	.00 (.00)	.56 (.04)***
General sensation seeking	.42 (.12)***	.08 (.11)	.50 (.03)***	.25 (.16)	.19 (.15)	.56 (.04)***
Positive expectancies	.40 (.12)**	.06 (.11)	.54 (.04)***	.42 (.17)*	.06 (.15)	.52 (.04)***
Alcohol involvement						
Alcohol QF	.33 (.11)**	.18 (.10))	.50 (.03)***	.49 (.04)***	.00 (.00)	.51 (.04)***
Heavy drinking	.40 (.11)***	.07 (.10)	.53 (.03)***	.41 (.04)***	.00 (.00)	.59 (.04)***
Max drinks	.29 (.11)**	.20 (.10)*	.51 (.03)***	.61 (.03)***	.00 (.00)	.39 (.03)***
Lifetime AUD symptoms	.37 (.04)***	.00 (.00)	.63 (.04)***	.46 (.04)***	.00 (.00)	.55 (.04)***

Notes: Standard errors are provided in parentheses. Estimates displayed as .00 are smaller than .01. MPQ = Multidimensional Personality Questionnaire; Alcohol QF = typical quantity × typical frequency of use; heavy drinking = composite of hangover and intoxication frequency; max drinks = most drinks ever consumed in a 24-hour period in lifetime; AUD symptoms = DSM-5 alcohol use disorder symptom count, without craving symptom.
*$p < .05$; **$p < .01$; ***$p < .001$.

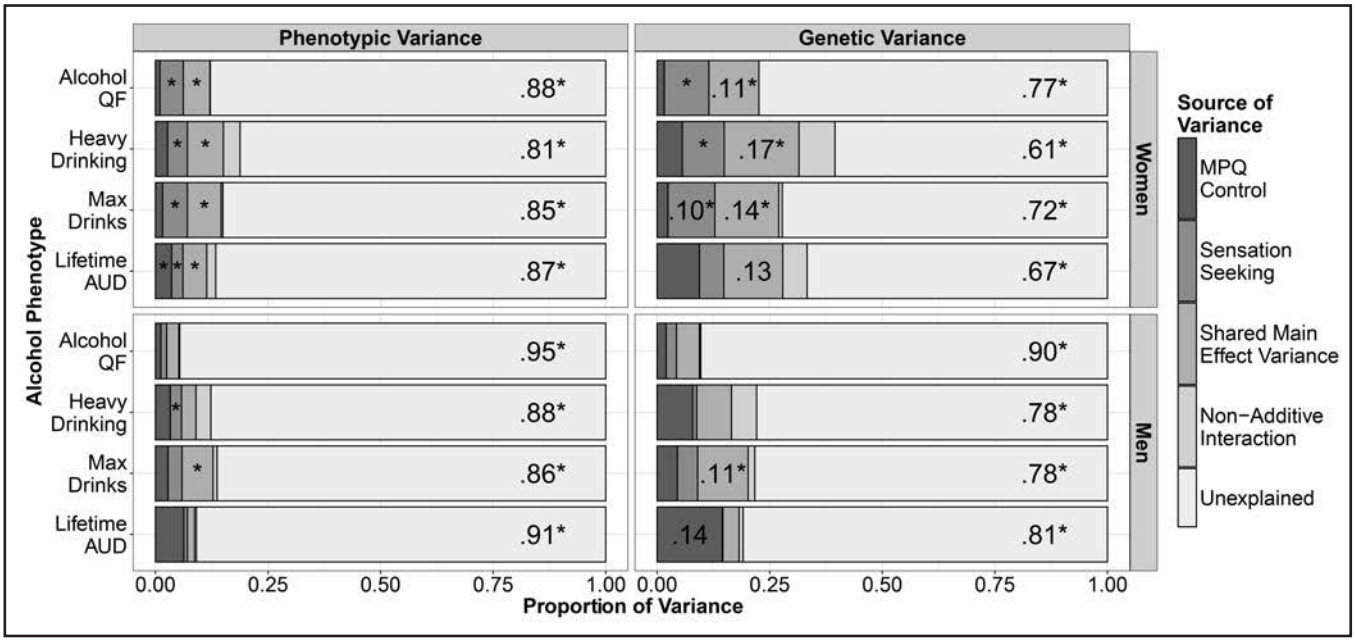

FIGURE 2. Estimates of the proportion of phenotypic (left panel) and genetic (right panel) variance in alcohol phenotypes attributable to main and interaction effects from models of sensation seeking and MPQ Control. Proportion estimates that were less than 0.10 are not displayed. Asterisks indicate statistical significance, even when proportion estimates fell below 0.10. The "Unexplained" variance is due to all sources not included in the model (i.e., neither main nor interaction effects).

men (.03 [*SE* = .01], $p = .01$). In contrast, positive expectancies accounted for a moderate proportion of variation in all alcohol phenotypes, even after accounting for MPQ Control. Of note, main effects of MPQ Control and sensation seeking explained the same variance for all alcohol phenotypes in women (.05–.08 [*SE*s = .02–.03], $p < .05$), but only for max drinks in men (.07 [*SE*s = .03], $p = .03$). MPQ Control and positive expectancies also explained the same variance for all alcohol phenotypes (.05–.10 [*SE*s = .02–.04], $p < .05$). After accounting for additive variance, interaction effects were negligible for all alcohol phenotypes (.001–.04 [*SE*s = .002–.03], $p = .21–.90$). Thus, behavioral control does not appear to be a distinct risk factor for most alcohol phenotypes, and interaction effects explained a negligible proportion of phenotypic variance in all alcohol phenotypes. Together, MPQ Control and sensation seeking explained 5%–19% of the variance in alcohol involvement, and MPQ Control and positive expectancies explained 23%–37%.

After we accounted for sensation seeking or positive expectancies, the effects of A_2 were negligible, suggesting that behavioral control does not explain genetic variance in alcohol involvement that is distinct from these constructs (.001–.14 [*SE*s = .001–.08], $p = .06–.99$; see the right panel of Figures 2 and 3). When modeling sensation seeking and accounting for MPQ Control, A_2 explained alcohol use (alcohol QF, heavy drinking, max drinks; .09–.10 [*SE*s = .04], $p < .05$) but not lifetime AUD in women (.05 [*SE*s = .03], $p = .11$). Among men, however, A_2 was unrelated to alcohol involvement in this model (.001–.05 [*SE*s = .01–.03], $p = .18–.87$). Finally, when modeling positive expectancies and accounting for MPQ Control, the effects of A_2 were large and significant for all phenotypes. Thus, positive expectancies appear to explain genetic variance that is distinct from behavioral control in women and men, and sensation seeking plays a similar role in women.

Of note, main effect variance attributable to both MPQ Control and sensation seeking explained alcohol use (but not AUD) in women and max drinks in men. Similarly, main effect variance attributable to both MPQ Control and positive expectancies accounted for a substantial proportion of variation in all alcohol phenotypes, except alcohol QF in men (.09 [*SE* = .05], $p = .06$). That is, genetic variance in all personality measures was associated with alcohol involvement, but much of the variance explained by behavioral control was also explained by sensation seeking and positive expectancies. After we accounted for main effects of personality, the effects of A_3 were negligible, suggesting that interaction effects do not explain genetic variation in alcohol involvement (.001–.08 [*SE*s = .001–.07], $p = .21–.97$). Thus, at the genetic level, behavioral control does not appear to be a distinct risk factor for alcohol involvement, and interaction effects explained a negligible proportion of genetic variance in all alcohol phenotypes. Together, MPQ Control and sensation seeking explained 10%–40% of the genetic variance in alcohol involvement, and MPQ Control and positive expectancies explained 50%–75%.

Aggregated across all personality measures, environmental factors explained a small proportion of variation in

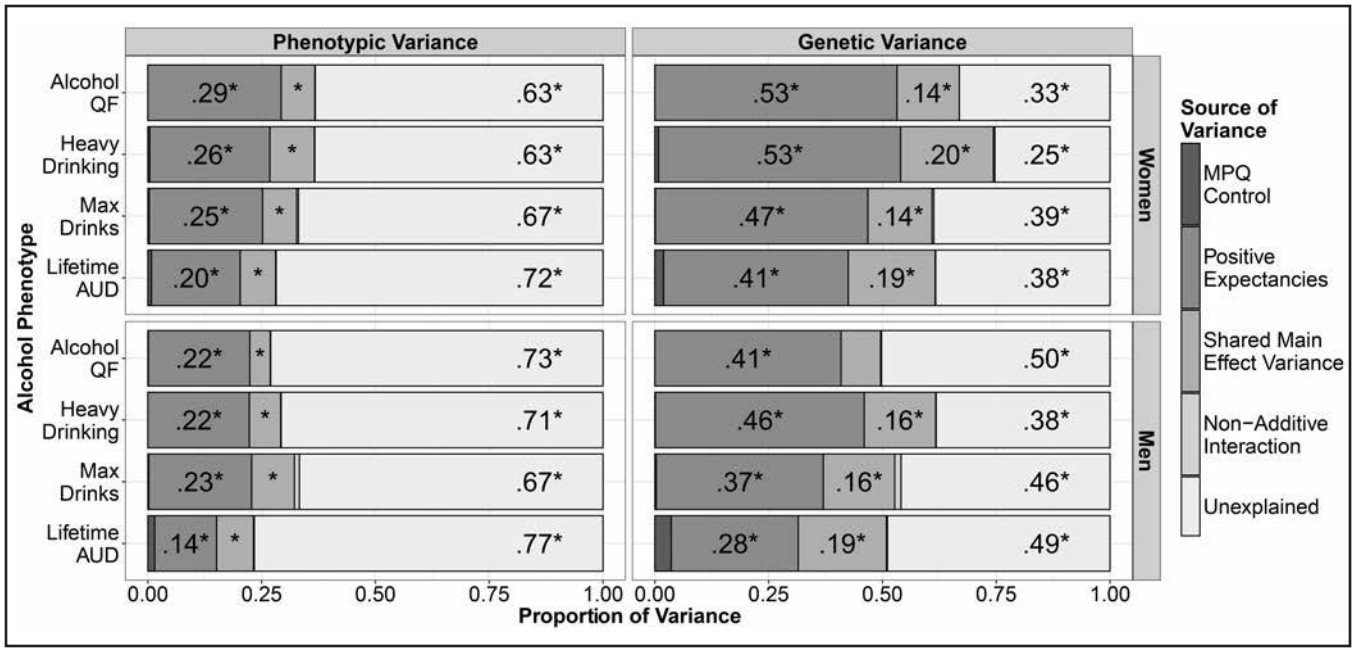

FIGURE 3. Estimates of the proportion of phenotypic (left panel) and genetic (right panel) variance in alcohol phenotypes attributable to main and interaction effects from models of positive expectancies and MPQ Control. Proportion estimates that were less than 0.10 are not displayed. Asterisks indicate statistical significance, even when proportion estimates fell below 0.10. The "Unexplained" variance is due to all sources not included in the model (i.e., neither main nor interaction effects).

alcohol involvement (0%–6%). Only E_2, when representing variance related to positive expectancies in women, was associated with alcohol involvement (.02–.06 [*SE*s = .01–.02], p = .01–.05). All behavior genetic models were also run with outlying observations, but results did not change.

Discussion

These findings are largely counter to the dual-systems model. Phenotypic analyses identified two significant interaction effects in the hypothesized direction in women, but two interaction effects in the opposite direction in men. Further, in behavior genetic models, the proportion of phenotypic, genetic, and environmental variation attributable to nonadditive interaction effects was negligible after accounting for main effects of personality measures. Last, behavioral control was largely found not to be distinct from sensation seeking and positive expectancies as a risk factor for alcohol involvement.

These findings are consistent, however, with the proposal that risky behavior may have more to do with sensation seeking or cravings than behavioral control (Duckworth & Steinberg, 2015). The genetic intersection between these risk factors is also consistent with growing neuroimaging evidence suggesting that, despite being implemented by separate neural regions, projections between mesolimbic reward (Koob & Le Moal, 2008) and prefrontal control substrates (Goldstein & Volkow, 2011) are associated with behavior. For example, white matter projections from the prefrontal cortex to the striatum are associated with trait measures of impulsivity (e.g., novelty seeking and persistence; Lei et al., 2014), as well as behavioral measures (e.g., delay discounting and response inhibition) in healthy and alcohol-dependent samples (Courtney et al.; Peper et al., 2013). Considering the current findings in light of this literature, phenotypic and genetic variance for behavioral control may be associated with alcohol involvement indirectly, via projections to the reward system. To elucidate the genetic relation between alcohol involvement, self-control, and approach tendencies, neuroimaging research and large-scale genome-wide association studies will be needed.

The current findings may point to potential mechanisms of treatment. All variance in alcohol involvement explained by behavioral control could be attributed to sensation seeking or positive expectancies, suggesting that interventions are unlikely to act directly on self-control, independent of these constructs. Interventions targeting craving (e.g., naltrexone; Maisel et al., 2013), however, may act independently of control.

Limitations and future directions

The measurement of approach tendencies and behavioral control should be considered when evaluating empirical findings of dual-systems models. Investigations of dual-systems models of alcohol involvement often use behavioral

measures, which correlate weakly with analogous self-report measures (Cyders & Coskunpinar, 2012; King et al., 2014). Further, incorporating neuroimaging with behavioral measures of approach tendencies and behavioral control may improve the operationalizations of these constructs; however, self-report measures tend to correlate more strongly with alcohol involvement (Donny et al., 2003; Salemink et al., 2015). Finally, the current study found substantial sex differences, which were beyond the scope of this article but should be explored in future studies.

The implications of general and domain-specific measures of approach tendencies should also be considered when interpreting these results. Sensation seeking may not assess approach tendencies for substance use, making it a poor fit for dual-systems models of alcohol involvement. For example, sensation seeking consisted of the Zuckerman Sensation Seeking Scale without substance use items, which yielded negligible associations with alcohol involvement, counter to prior findings with the Zuckerman Sensation Seeking Scale (Hittner & Swickert, 2006). In contrast, positive expectancies may be a proxy for problematic substance use. It is unclear whether general or specific measures best represent these systems, and empirical work will be important for identifying optimal operationalizations of approach tendencies and their relation to alcohol involvement.

Summary

There has been an increasing focus on dual-systems models, which assume that approach tendencies and behavioral control are distinct processes for risky behavior (Casey et al., 2005; Harden & Tucker-Drob, 2011; Quinn & Harden, 2013; Somerville et al., 2010; Steinberg, 2010). The current findings suggest that, although conceptually distinct, behavioral control is not separate from sensation seeking or positive expectancies with regard to alcohol involvement. Further, we found little support to justify the inclusion of nonadditive interaction effects to explain alcohol involvement. Thus, there was little support for the dual-systems model, and a more parsimonious model (e.g., main effects of only sensation seeking and/or behavioral control) may adequately capture the effects central to the dual-systems model.

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The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

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