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Shared genetic contributions to early-onset drinking and drinking to cope motives

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Abstract

Introduction—Recent evidence from empirical studies indicates that individuals who begin drinking at an early age may be more likely to use alcohol to cope with negative mood states and stress; however, the mechanisms underlying this association are unclear. One possibility is that early drinking directly increases risk for drinking to cope (DTC). Alternatively, the association between early drinking and DTC may be indirect, attributable to overlapping genetic or environmental factors. No prior genetically informative study has investigated the sources of covariation underlying the early-onset drinking-DTC association.

Method—Early-onset drinking (before age 15) was assessed using structured clinical interviews in a sample of 7130 male and female participants aged 19–56 years from the Virginia Adult Twin Study of Psychiatric and Substance Use Disorders (VATSPSUD, Kendler & Prescott, 2006). DTC was assessed using the mood management scale of the alcohol use inventory (Horn & Wanberg, 1983). The sources of the covariation between early first drink and DTC were estimated using bivariate twin modeling.

Results—Early drinking onset was reported by 28% of males and 16% of females and was associated with significantly higher DTC scores (phenotypic correlation: males = .19, females = .22). Results from bivariate twin models indicated that the association between early-onset drinking and DTC was completely attributable to shared genetic factors that contribute to both behaviors.

Conclusions—Greater DTC among early-onset drinkers may not reflect a direct causal process, as shared biological pathways may explain vulnerability to stress-related drinking seen among early-onset drinkers.

Keywords

Drinking motives; Alcohol; Early-onset drinking; Twin; Genetic

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Contributors

Dr. Young-Wolff conceptualized this paper, conducted the statistical analysis, and wrote the first draft of the manuscript. All authors have participated in the manuscript preparation and have approved the final manuscript.

Conflict of interest

No conflict of interest exists.

1. Introduction

Early initiation of alcohol use is a well-established predictor of alcohol-related problems and alcohol use disorders (Buchmann et al., 2009; DeWit, Adlaf, Offord, & Ogborne, 2000; Grant & Dawson, 1997; Pedersen & Skrandal, 1998; Pitkänen, Lyyra, & Pulkkinen, 2005; Reboussin, Song, Shrestha, Lohman, & Wolfson, 2006). There is some evidence that this risk may be due to increases in stress-related drinking associated with early alcohol initiation. Experimental studies have found that young rodents exposed to alcohol have greater self-administration of ethanol in response to stressors than those with later exposure (Füllgrabe, Vengeliene, & Spanagel, 2007; Siegmund, Vengeliene, Singer, & Spanagel, 2005). Several human studies also suggest that early-onset drinking may increase risk for stress-related drinking. In a population-based sample, past-year stressful life events were associated with greater alcohol consumption among individuals who began drinking by age 14 but not among those with later drinking onset (Dawson, Grant, & Li, 2007). These findings have been replicated and expanded in our community sample of adult twins (Lee, Young-Wolff, Kendler, & Prescott, in press) and in a longitudinal sample of young adults (Blomeyer et al., 2011).

Drinking motives are robust proximal predictors of drinking behaviors (Carpenter & Hasin, 1999; Cooper, Russell, Skinner, Frone, & Mudar, 1992) and the elevated risk for stress-related drinking among early-onset drinkers may be due in part to a greater vulnerability or stronger motivation for drinking to cope (DTC). Supporting evidence for this hypothesis comes from studies showing that early-onset drinking is associated with deficits in self-regulatory behaviors and coping skills (Cooper et al., 1992; DeWit et al., 2000; Fromme & River, 1994; Laurent, Catanzaro, & Callan, 1997; Lewis et al., 2008; Moos, Brennan, Fondacaro, & Moos, 1990), a greater tendency to drink under conditions of unpleasant emotions (Buchmann et al., 2010) and higher endorsement of DTC (Rothman, Edwards, Heeren, & Hingson, 2008).

It is important to understand the basis for the association between early onset of alcohol use and the later use of alcohol to regulate emotions. One mechanism is via direct effects of alcohol on the developing brain. Adolescence is a period of substantial structural and functional remodeling of the brain, particularly in areas associated with the maturation of emotional, behavioral, and cognitive regulation (Clark, Thatcher, & Tapert, 2008; Spear, 2000; Witt, 2010). Heavy alcohol use in early adolescence is associated with brain damage and neuropsychological deficits (Alfonso-Loeches & Guerri, 2011). The neurotoxic effects of adolescent drinking could hinder the development of healthy alternative emotion regulation strategies (Sher & Grekin, 2007) creating a vicious cycle of drinking to cope with stressors and negative mood. Drinking during adolescence may also impede achievement of age appropriate developmental tasks (e.g., school attendance, making and maintaining close friends) (Masten, Faden, Zucker, & Spear, 2008), potentially limiting opportunities to learn coping skills while increasing risk for DTC.

An indirect mechanism for the relation between early-onset drinking and DTC is through the association of both with familially-transmitted risk for alcohol misuse. Family history of alcoholism is associated with early-onset drinking and strong positive expectancies and motives for drinking (Chandler, Elgar, & Bennett, 2006; Dawson, 2000; Mann, Chassin, & Sher, 1987; Sher, Walitzer, Wood, & Brent, 1991; Worobec et al., 1990). The overlap could be due to family environment if heavy drinking parents create environments that increase risk for offspring early-onset drinking and DTC (e.g., via modeling parental drinking behaviors and attitudes). Alternatively, elevated DTC among early-onset drinkers might be attributable to shared genetic factors. Twin studies have shown that early-onset drinking is genetically influenced (Liu et al., 2004; McGue, Pickens, & Svikis, 1992) and shares

overlapping genetic liability with alcohol use disorders (Grant et al., 2006; Prescott & Kendler, 1999). Genetic influences also contribute to DTC (Agrawal et al., 2008) and this genetic liability overlaps with alcohol use disorders and major depression (Prescott, Cross, Kuhn, Horn, & Kendler, 2004; Young-Wolff, Kendler, Sintov, & Prescott, 2009).

Despite evidence that early-onset drinking and DTC are each genetically influenced, no prior genetically-informative study has investigated the basis of the early-onset drinking–DTC overlap. We address this gap in the literature by using data from a large sample of personally-interviewed twins to: 1) examine the association between early-onset drinking and DTC, and 2) estimate the relative genetic and environmental sources of this association.

2. Method

2.1. Participants and procedures

The sample comprised individuals interviewed for the VATSPSUD, a longitudinal study of psychopathology in two samples of adult twins. The samples were originally identified through state birth certificates compiled by the Virginia Twin Registry, and include Caucasian twins born between 1934 and 1974. Data for the current study come from wave four of data collection in female twin pairs conducted on 1995–1997 and from wave two of data collection in male–male and male–female twin pairs conducted on 1995–1998 (Kendler & Prescott, 2006).

Lifetime abstainers ($n = 425$) were excluded from these analyses. Participants were not administered the drinking motives questionnaire if they had never consumed 12 or more drinks within a year ($n = 230$) and an additional 719 participants did not return the motives questionnaire. Aside from lifetime abstainers, all participants who completed the clinical interview were included in the analyses regardless of whether they completed the drinking motives questionnaire. The analyses for this study are based on data from 7130 individuals who completed the clinical interview, including 5568 individuals from 2784 complete pairs (444 monozygotic (MZ) female, 282 dizygotic (DZ) female, 646 MZ male, 455 DZ male, 957 DZ opposite sex (DZO)) and 1562 individuals whose cotwin did not participate or was excluded for low drinking. The mean age of the sample is 36.7 ($SD = 8.8$, range 20–61).

2.2. Measures

DTC were measured using the mood management scale of the alcohol use inventory (Horn & Wanberg, 1983), which included seven items assessing drinking to unwind, forget, relieve tension, or overcome depressed mood (e.g., do you drink to change your mood?). Scores were calculated by summing the item scores (0 = no, 1 = yes) and dividing by the number of items. The internal consistency estimate (Cronbach alpha) for the DTC scale was 0.82 and the test–retest correlation was 0.85 (Prescott et al., 2004). DTC scores were positively skewed and log-transformed prior to analysis.

Age at drinking onset was assessed using the question, “How old were you the first time you had a drink?” (defined as 12 oz beer, 5 oz wine, or 1.5 oz spirits). Average age at first drink was 16.5 ($SD = 3.6$). We followed the convention in the literature of defining early-onset drinking as younger than age 15 (e.g., Grant & Dawson, 1997) and created a dichotomous variable for analysis (1 = onset age <15.0, 0 = onset age $\geq$ 15.0 and non-drinkers).

2.3. Statistical analyses

Based on the premise that MZ twins share all of their genetic and common environmental factors, and DZ twins share on average half of their genetic variation and all of their common environmental factors, bivariate twin models were utilized to partition the early-

onset drinking–DTC covariance into additive genetic (A) variance shared by twins due to genetic alleles that combine additively; common environmental (C) variance shared by twins reared together; and non-shared environmental (E) variance unique to individuals. Structural equation modeling was used to fit a bivariate twin model that assigns overlapping variance equally to both measures (see Young-Wolff, Kendler, Ericson, & Prescott, 2011). Models were fit to raw data using all available data (i.e., using data on early-onset drinking even if DTC scores were not available, and data from twins in incomplete pairs). In this model (Fig. 1), the first set of factors (A_{overlap} , C_{overlap} , and E_{overlap}) contribute to both early-onset drinking and DTC, the second set are unique to early-onset drinking (A_{onset} , C_{onset} and E_{onset}), and the third set are unique to DTC (A_{DTC} , C_{DTC} , E_{DTC}). Twin models were fit using Mplus™ software version 5.2 (Muthén & Muthén, 2007) using the WLSMV estimation option (for details of twin modeling with Mplus, see Prescott, 2004). We compared a fully saturated model to several reduced models. The goodness of fit of nested alternative models was evaluated using χ^2 difference tests calculated using the Mplus DIFFTEST option.

3. Results

Approximately 28% of males and 16% of females reported drinking onset prior to age 15. The means (and SD) of DTC scores were 1.92 (0.04) among males and 1.61 (0.07) among females. Early-onset drinking was significantly correlated with DTC at the individual level (males: $r = 0.19$ (.02); females: $r = 0.22$ (.03)). The pattern of cross-twin cross-variable correlations (i.e., one twin's early-onset with cotwin's DTC) were consistent with genetic influences on the overlap (i.e., MZ correlations > DZ correlations). These values (and SE) were: MZM = 0.14 (.04), DZM = 0.11 (.05), MZF = 0.21 (.05), DZF = -0.03 (.07), DZO male_{ONS}–female_{DTC} = 0.11 (.05), female_{ONS}–male_{DTC} = 0.15 (.05). As reported previously for a slightly different sample from our study, MZ pair correlations were greater than DZ pair correlations for both for early-onset drinking (Prescott and Kendler, 1999) and DTC (Prescott et al., 2004). In the present study, for early-onset, MZM = 0.48 (.06), DZM = 0.35 (.08), MZF = 0.38 (.05), DZF = 0.23 (.03), DZO = 0.21 (.06), and for DTC, MZM = 0.34 (.04), DZM = 0.17 (.05), MZF = 0.38 (.05), DZF = 0.07 (.06), DZO = 0.16 (.04).

Fit statistics for biometric modeling are reported in Table 1. The least restrictive model (1) estimated all possible unique and overlapping parameters, allowed sex differences in these parameter estimates, and allowed sexes to have different sources of genetic variance.¹ Dropping sex differences in genetic sources (model 2) did not significantly worsen model fit, and genetic sources were modeled as fully overlapping for the sexes in subsequent models. Requiring males and females to have the same values for the genetic and environmental parameters (model 3) decreased model fit, and subsequent models allowed parameter estimates to vary across sex. Forcing all overlapping influences (i.e., A_{overlap} , C_{overlap} , E_{overlap}) to zero (model 4) significantly decreased model fit, indicating there is a significant association between early-onset drinking and DTC. The overlapping common environmental influence (i.e., C_{overlap}) was estimated near zero in model 2, and was fixed at zero (model 5) with minimal change in fit. The two paths of common environmental influences contributing to early-onset drinking and DTC, respectively, could also be fixed at zero without worsening model fit (model 6).

Forcing A_{overlap} to zero (model 7) substantially worsened model fit, indicating that genetic influences contribute significantly to the overlap of early-onset drinking with DTC. Model 8 forced E_{overlap} to zero to test whether the early onset-DTC relation is entirely due to genetic

¹Genetic correlations <0.5 indicate that the genetic factors contributing to early-onset drinking and DTC are not completely overlapping in males and females.

influences. This did not fit worse than model 6, so model 8 was selected as the best-fitting model for representing the basis for the early-drinking onset–DTC association (Fig. 1).

4. Discussion

The current study is the first genetically informative investigation of the sources of covariance between early-onset drinking and DTC. Early-onset drinkers reported higher DTC motives than those with later drinking onset. Genetic influences accounted for approximately 1/3 of the variance in DTC (in males, $(19 + 14) = 33\%$ and females $(22 + 12) = 34\%$), with the remainder of variance attributable to non-shared environmental factors. More than half of the genetic variance contributing to DTC was shared with early-onset drinking (males $(19/(19 + 14) = 58\%)$; females $(22/(22 + 12) = 65\%)$). Genetic influences contributed to a substantial proportion of the variance in early-onset drinking in males $((19 + 29) = 48\%)$ and females $((22 + 46) = 68\%)$, with the remainder of variance attributable to non-shared environmental influences. Roughly 40% $(19/(19 + 29))$ and 32% $(22/(22 + 46))$, respectively, of genetic liability for early-onset drinking was shared with DTC in males and females. Moreover, there was evidence that virtually all of the early-onset drinking–DTC association (males $r = .19$; females $r = .22$) was attributable to overlapping genetic influences.

Early-onset drinking and DTC are complex behaviors that tend to cluster with other risk factors for problematic drinking, and multiple mechanisms likely contribute to their overlapping genetic risk. We cannot rule out the possibility that genetic influences on DTC are directly mediated or potentiated via early-onset drinking. However, the present findings suggest that greater DTC among early-onset drinkers may not reflect a direct causal process. Rather, shared genetic mechanisms (e.g., genetically influenced physiological responses to alcohol) may contribute to both vulnerability for DTC and risk for stress-related drinking seen among early drinkers. Moreover, given evidence that both early-onset drinking (Grant et al., 2006; Prescott & Kendler, 1999) and DTC (Prescott et al., 2004; Young-Wolff et al., 2009) share genetic liability with alcohol use disorders, the current results point to the possibility that the well-known correlation between early-onset drinking and alcohol use disorders may be partially attributable to genetic influences shared with DTC. Prevention efforts that teach early-onset drinkers adaptive coping strategies and stress-reduction techniques may be particularly beneficial, and perhaps of equal clinical significance as interventions focused primarily on delaying age at first drink. Ongoing studies of adolescent twins will help unravel these etiological mechanisms, including how drinking motives unfold as experience with alcohol accumulates.

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Notes: Results are shown for one twin in a pair. Estimates are from Model 8 in Table 1. A=additive genetic factors; C=common environmental factors; E=non-shared environmental factors. A_{overlap} , C_{overlap} and E_{overlap} are factors shared between early drinking onset and drinking to cope, A_{onset} , C_{onset} and E_{onset} are specific to early drinking, and A_{DTC} , C_{DTC} and E_{DTC} are specific to DTC. For model identification, A_{overlap} , C_{overlap} and E_{overlap} are equated. Thresholds, means variances and covariances were estimated and allowed to differ across sex. Parameters fixed at zero are indicated by ---.

Fig. 1.

Path diagram for the association between early-onset drinking and drinking to cope motives (DTC) in males and females. Notes: results are shown for one twin in a pair. Estimates are from model 8 in Table 1. A = additive genetic factors; C = common environmental factors; E = non-shared environmental factors. A_{overlap} , C_{overlap} and E_{overlap} are factors shared between early drinking onset and drinking to cope, A_{onset} , C_{onset} and E_{onset} are specific to early drinking, and A_{DTC} , C_{DTC} and E_{DTC} are specific to DTC. For model identification, A_{overlap} , C_{overlap} and E_{overlap} are equated. Thresholds, means variances and covariances were estimated and allowed to differ across sex. Parameters fixed at zero are indicated by ---.

Table 1

Goodness-of-fit results from twin models for association between early alcohol use and drinking to cope motives (DTC).

Model	Overall fit			Model difference test			
	χ^2_{df}	Free param	RMSEA	Compared to model #	$\Delta\chi^2$	$\Delta Param$	P
1 Full model, sex differences in genetic sources	49.6	23	0.029	–	–	–	–
2 Full model, same genetic sources in M and F	50.5	20	0.027	1	1.98	3	0.58
3 Full model, same ACE parameters in M and F	77.6	11	0.036	2	32.4	9	<0.001
4 Full model, drop overlap (i.e., overlap = $C_{overlap} = E_{overlap} = 0$)	206.5	14	0.075	2	159.8	6	<0.001
5 Full model, drop $C_{overlap}$	49.7	18	0.025	2	0.46	2	0.79
6 No $C_{overlap}$, drop C_{onset} , C_{DTC}	52.4	14	0.024	5	3.70	4	0.45
7 No C ($C_{overlap} = C_{onset} = C_{DTC} = 0$), drop $A_{overlap}$	85.9	12	0.040	6	41.6	2	<0.001
8 No C ($C_{overlap} = C_{onset} = C_{DTC} = 0$), drop $E_{overlap}$	53.7	12	0.024	6	2.77	2	0.25

Note. χ^2_{df} = Mplus WLSMV chi-square values are approximate, but chi-square difference test values ($\Delta\chi^2$) are exact. Param = parameters. RMSEA = root mean square error of approximation. $\Delta\chi^2$ = difference in log-likelihoods between nested models. $\Delta Param$ = difference in number of free parameters relative to comparison model. A = additive genetic influences, C = shared environmental influences, E = non-shared environmental influences. P-value <0.05 indicates significantly worse fit than comparison model. Bold = best-fitting model.