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The Moderating Effect of Religiosity on the Genetic Variance of Problem Alcohol Use

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Abstract

Background—Previous studies have demonstrated that the heritability of alcohol related phenotypes depends upon the social background in which it is measured (e.g., urbanicity, marital status, and religiosity). The aim of the current study was to identify whether religiosity moderated the genetic variance of problem alcohol use in males and females at two time points: adolescence and early adulthood.

Method—Participants were 312 male MZ pairs, 379 female MZ pairs, 231 male DZ pairs, 235 female DZ pairs, and 275 opposite sex DZ pairs participating in the University of Colorado Center on Antisocial Drug Dependence. Religiosity was measured using the Value on Religion Scale (Jessor and Jessor 1977), and problem alcohol use was measured using the Composite International Diagnostic Interview-Substance Abuse Module (Cottler et al. 1989). Data were analyzed using a model-fitting approach to the twin data.

Results—In adolescence, genetic variance of problem alcohol use decreased significantly with increasing levels of religiosity in both males and females whereas in early adulthood, religiosity did not moderate the genetic variance of problem alcohol use in either males or females.

Conclusion—Religiosity appears to moderate the genetic effects on problem alcohol use during adolescence, but not during early adulthood. The reduced genetic variance for problem alcohol use in adolescence may be the consequence of greater social control in adolescence than in young adulthood.

Keywords

Problem alcohol use; heritability; moderation; religiosity; adolescence

Introduction

Many alcohol related phenotypes are heritable in both adolescents and adults (Heath et al. 1991; Heath et al. 1997; Heath and Martin 1994; Hopfer et al. 2005; Kendler et al. 1994; Pagan et al. 2006; Rhee et al. 2003). However, the heritability of these phenotypes is contextually dependent on a number of social factors. For adolescents, a study of adolescent lifetime alcohol *use* reported a heritability of 72%, but only in those who did not have parental permission to drink (Maes et al. 1999). In adolescents who had parental permission to drink, genes were not a salient contributor to the variance of lifetime alcohol use. Similarly, genetic risk for drinking *frequency* was found to be more salient in twins from

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urban (vs. rural) backgrounds (Rose et al. 2001) and twins whose peers use drugs (Dick et al. 2007); the heritability of male drinking *patterns* was higher in those whose parent were frequent drinkers than in those from non/low drinking households (Cleveland and Wiebe 2003). Moreover, heritability of *intoxication* frequency was found to be more heritable in independent twins than in dependent twins in both adolescence and early adulthood (Penninkilampi-Kerola et al. 2005), In studies of adult alcohol phenotypes, Heath et al. (Heath et al. 1989) showed that the heritability of levels of alcohol *consumption* were around 0.50 for married women, but increased to 0.76 for unmarried women. *Frequency* of use was found to be more heritable in twins who displayed greater independence from their co-twin (vs. those who were dependent on their co-twin) (Penninkilampi-Kerola et al. 2005).

Religiosity, a term used to describe multiple related constructs such as religious beliefs, religious values, and religious behavior, is a protective factor for alcohol use (Heath et al. 1999; Winter et al. 2002), and has also been shown to moderate the genetic influence on alcohol use initiation, particularly in females. In a study of 1967 Dutch twin pairs 12–26 years of age, genetic influences on *initiation* of alcohol use were attenuated in participants from religious backgrounds, compared with non-religious backgrounds (Koopmans et al. 1999). For individuals without a religious upbringing, heritability estimates of 40% and 39% were reported for males and females, respectively; for individuals with a religious upbringing heritability was estimated at 25% and 0% for males and females, respectively. Similar findings have also been reported for the influence of religiosity on the heritability of behavioral disinhibition (Boomsma et al. 1999), smoking initiation (Timberlake et al. 2006), and neuroticism (Willemsen and Boomsma 2007). In all of these studies, heritability estimates were lower in populations with a religious upbringing or self described religiousness. These findings are consistent with Heath and colleagues (1999) theory that genetic influences on behavioral outcomes are attenuated in environments where individuals' choices are more limited.

Shanahan and Hofer (2005) described three models by which environmental factors moderate genetic variance for behavioral traits. The first, consistent with that proposed by Heath et al (1999), was a social control model, which suggests that people are prevented from behaving in a certain manner as the result of social factors, regardless of genetic predisposition. Consequently, the genetic variance (and heritability) is attenuated in more controlling environments. The second model, social distinction, proposes that genes become more important in the etiology of a phenotype in environments where the trait is rare. The social push perspective (Raine 2002), in which a benign environment (one that neither encourages nor discourages a particular behavior) is associated with a higher heritability, is an example of social distinction. Finally, the third model was a social expression model, where it is proposed that specific environments trigger genetic variance. For instance, exposure to extremes of alcohol using peers (i.e. where all peers drink) may trigger any pre-existing, but previously un-expressed, genetic risk to drink. These models are described in detail elsewhere (Button et al. 2009; Shanahan and Hofer 2005).

The aim of the current study was to test the hypothesis that religiosity is a protective factor for problem alcohol use in adolescents and young adults, and to examine the extent to which genetic effects are contextually dependent on religiosity. Based on previous findings we expect genetic influences to be attenuated in participants who are more religious, as predicted by the social control model. Furthermore, studies have shown that shared environmental variance is greater for religiosity in adolescence than in adulthood, suggesting that there is more social control in adolescence (Boomsma et al. 1999; Button et al. under review; Koenig et al. 2005). Therefore, we might expect religiosity to influence alcohol problems differently in adolescents compared with adults. Given previous evidence of greater social control of religiosity in adolescence than in adulthood, we hypothesize that

religiosity may attenuate the heritability of alcohol problems more in adolescence than in adulthood. This is the first study, to our knowledge, to examine this potential age difference for alcohol problems.

Materials and Methods

Participants

Participants were from the Longitudinal Twin Study (LTS) and Community Twin Study (CTS), two community-based twin samples, assessed as a part of the Center on Antisocial Drug Dependence (CADD). The LTS twins were recruited through the Colorado Department of Health's Division of Vital Statistics and were included in the CADD sample as they reached their 12th birthday. Twins from the CTS were recruited through the Department of Health and 170 out of 176 school districts in Colorado. Since these samples were community based twin samples and not selected on religiosity or alcohol related phenotypes, combining the two into one large sample was permissible. Data were available for 2754 individual twins at time 1 (male = 1311, female = 1443), and 2486 individual twins at time 2 (male = 1153, female = 1333). These individuals comprised 689 identical, or monozygotic (MZ), twin pairs (male = 311, female = 378), and 740 fraternal, or dizygotic (DZ) twin pairs (male = 230, female = 235, opposite sex = 275), for whom data were available for at least one twin at one or more waves. Twins were 12–18 years of age (mean = 14.51, S.D. = 2.10) at time 1 and 17–29 (mean = 19.63, S.D. = 2.46) at time 2. The mean age difference across waves is 5.3 years (S.D. 0.99). Twin zygosity was determined using a 9-item assessment of physical characteristics (Nichols and Bilbro, Jr. 1966) completed by the interviewer and by genotyping 11 highly informative short tandem repeat polymorphisms. Twin pairs were categorized as MZ if they had similar physical characteristics and were concordant on all markers and DZ if they presented with dissimilar physical characteristics and were discordant on any of the markers.

Measures

Religiosity was assessed using five items from the Adolescent Health and Development Questionnaire (Jessor and Jessor 1977), often referred to as the Value on Religion Scale, that assess how important different aspects of religion are to the individual. Participants were asked, “How important is it to you”:

1. To be able to rely on religious counsel or teaching when you have a problem?
2. To believe in God?
3. To rely on your religious beliefs as a guide for day-to-day living?
4. To be able to turn to prayer when you're facing a personal problem?
5. To attend religious services regularly?

Responses were scored as “not important at all” (1), “somewhat important” (2), “important” (3), and “very important” (4). The five items were highly correlated, and the Cronbach's alpha was 0.92 at wave 1 and 0.95 at wave 2. Religiosity scores were taken as the average score across all 5 items, unless more than 1 item was missing, in which case they were scored as missing. Given that these items assessed importance rather than actual behavior, religiosity in this study is more accurately described as an assessment of religious values, which is similar to the concept of intrinsic religion (Bouchard, Jr. et al. 1999). Religiosity scores were regressed on age separately within sex, and the standardized residuals were used for further analyses.

Problem Alcohol Use was assessed using items pertaining to whole life alcohol dependence and abuse from the Composite International Diagnostic Interview-Substance Abuse Module (CIDI-SAM; Cottler et al. 1989). The CIDI-SAM is a structured diagnostic interview that assesses DSM-IV abuse and dependence symptoms. At wave 1 the pencil and paper interview was used, whereas computerized CIDI-SAM was used at wave 2. Endorsed symptoms were summed and scores were regressed on age separately for males and females, then log transformed to approximate a normal distribution.

Analyses

All descriptive analyses were conducted using SPSS 15 (SPSS Inc. 2006). A Mann-Whitney U test was used to test for sex differences in all scores prior to any transformations of the data, and the correlation between age and each phenotype was estimated using a Pearson's correlation coefficient. Correlations between phenotypes were estimated using the Pearson's correlation coefficient after age-regressions and transformations.

Model fitting analyses were conducted using Mx (Neale 2004), and the fit of all models was assessed using the minus twice the log-likelihood (−2LL) fit statistic. The model we applied to our data was the model described by Purcell (Purcell 2002), often referred to as a gene-environment interaction model (see Figure 1). In this model, the phenotypic variance can be partitioned into the latent, or unmeasured, genetic (a), shared environment (c), and non-shared environment (e) effects, and also the interaction between each of these parameters (β_X, β_Y, and β_Z respectively) with a moderating variable (i.e. religiosity). The model also examines the moderating effect of religiosity on the means (β_M). Any gene-environment correlation between religiosity and problem alcohol use (i.e. any genetic influences shared in common by the two phenotypes) are included in the means model, and are not explicitly tested in the model. To test the significance of each of the interaction parameters, we dropped each one in turn and compared the fit of the nested models (those in which the parameter was dropped) with the fit of the model in which all parameters were freely estimated. Furthermore, differences across sex were examined by comparing the fit of a model in which male and female estimates were free to differ with a model in which they are constrained to be the same. Comparison of the fit of nested models with the full model was tested using a likelihood ratio test. That is, the difference in the −2LL between the full and nested models was treated as a χ^2 statistic, with degrees of freedom equal to the difference in the number of estimated parameters. A significant χ^2 ($p < 0.05$) indicates that the nested model provides an unsatisfactory fit to the data, and thus, it should be rejected in favor of the fuller model. A non-significant χ^2 indicates that the constraints assumed for the nested model do not significantly worsen the fit to the data; the nested model would be preferred as more parsimonious.

Although a number of papers report changes in heritability, these are often misleading. The heritability is the *proportion* of phenotypic variance due to genetic influences, and can change as the result of a moderation of genetic variance, shared environment variance, non-shared environment variance, or any combination of these. Therefore, it is possible for the heritability to change, even when the genetic variance remains constant. Moreover, it is even possible for the genetic variance to increase while the heritability decreases. Consequently, only changes in absolute variances were reported in the current paper.

Results

As presented in Table 1, on average males reported higher levels of problem alcohol use symptoms than females at both waves; however, this difference reached significance only in young adulthood. Moreover, consistent with previous studies, on average females reported significantly higher religiosity than males at both waves. Furthermore, the mean levels of

religion significantly decreased for both males and females from wave 1 to wave 2, as determined using the Mann Whitney U test (males: $z = 5.61$, $p < 0.001$; females: $z = 6.70$, $p < 0.001$), and levels of problem alcohol use significantly increased across wave (males: $z = 16.28$, $p < 0.001$; females: $z = 21.02$, $p < 0.001$). A significant negative correlation was detected between religiosity and problem alcohol use; for males correlations were −0.11 and −0.20 in adolescence and young adulthood respectively, while for females the correlations between religiosity and problem alcohol use were −0.15 (adolescence) and −0.13 (young adulthood). Correlations of the twin-pair differences for religiosity and alcohol problem use provide a within-family replication of the between-family correlations described above. That is, we analyzed the data to determine whether the twins who scored higher on religiosity than their co-twin also scored lower than their co-twin on alcohol problem use. Results were largely comparable to the between-family scores, and importantly, were also negative; for males, the correlations were −0.03 (non-significant) in adolescence and −0.11 (significant at the 0.05 level) in young adulthood. For females, the correlations were −0.13 (significant at the 0.01 level) in adolescence and −0.11 (significant at the 0.05 level) in young adulthood.

Constraining the male and female parameter estimates to be equal resulted in a significant deterioration of fit for all models when compared to the models in which male and female parameters were estimated freely (Table 2). Consequently, male and female parameters were estimated separately in all further models. Consistent with the significant negative correlations between religiosity and problem alcohol use, reported above, it was not possible to drop the main effect of religiosity on the mean scores of the alcohol problem scores for males or females at either wave. Therefore, our results show that higher levels of religiosity are associated with lower mean levels of problem alcohol use.

It was not possible to drop the moderating effect on any parameter for either males or females during adolescence without a significant worsening in fit of the model. In both males and females, the genetic variance and non-shared environment variance are attenuated in participants with higher religiosity scores, whereas the shared environment variance increased (Figure 2). The total phenotypic variance of adolescent problem use was attenuated for higher levels of religiosity for both males and females.

For male problem alcohol use at the second assessment, it was possible to drop the moderating effect of religiosity on all parameters without a significant reduction in the fit of the model. Consequently, the phenotypic variance for male problem alcohol use remained constant irrespective of religiosity. For females, it was possible to drop the moderating effects of religiosity on the genetic variance and the shared environment variance, but not on the non-shared environment variance. Therefore, both the non-shared environment and total phenotypic variance were attenuated at higher levels of religiosity.

Discussion

The aims of the current study were to identify whether religiosity is a protective factor for problem alcohol use in adolescents and young adults, and to examine the extent to which religiosity moderates the magnitude of the genetic influence of problem alcohol use. Consistent with previous findings (e.g. Koopmans et al. 1999) our results suggested that religiosity did significantly protect against the development of problem use in both adolescence and early adulthood. Moreover, we also demonstrated that, consistent with previous work, both the phenotypic variance and genetic variance was attenuated in higher levels of religiosity. However, although the variance was attenuated in both adolescents and young adults, the genetic variance was attenuated only in the adolescents. As discussed below, these findings provide general support for the social control hypothesis.

Our findings of lower mean levels of problem alcohol use at higher levels of religiosity further add to the literature reporting a protective effect of religiosity against drinking alcohol. A number of reasons for this decrease are possible. Certain religions may forbid, or at least restrict the use of alcohol, reducing its use in religious followers. People who subscribe to religious beliefs may also be naturally less inclined to drink alcohol and therefore develop problems with alcohol. Also, people who hold religion as important may be more likely to associate with other religious individuals, and less with individuals who promote alcohol consumption. In contrast to the study by Koopmans et al. (1999), we found an attenuated variance associated with higher levels of religiosity for alcohol problem scores in adolescence and young adulthood. This lower variance is consistent with the social control theory in which religiosity restricts behavior and subsequently the total phenotypic variance of alcohol problems.

Genetic variance is attenuated at higher levels of religiosity in adolescence, consistent with the social control hypothesis. That is, the environment is sufficiently influential (i.e. there is likely to be pressure to *not* drink) that it over-rides any genetic predispositions. Some additional evidence for the social control hypothesis is found in the increase in shared environmental variance in more religious adolescents, suggesting that people who are religious are closer to their families, and are more influenced by them. Previous studies have demonstrated that religiosity in adolescents is almost entirely due to shared environmental effects (Boomsma et al. 1999; Koenig et al. 2008), and it is possible that this is because adolescents tend to follow their parents in religious matters. This increased likelihood to be religious if your parents are, and the control religious parents potentially exert over their adolescent offspring, would influence the siblings being raised in the same family similarly, and manifest itself as a shared environmental effect on alcohol related behavior. This is providing that there is variation among religious households in their attitudes towards drinking. For example, it has been demonstrated that Catholics are more at risk for alcohol related phenotypes than other religious affiliations (Heath et al. 1997), and this might be reflected in the etiology of alcohol phenotypes. This finding was replicated for adult males in the current study.

Our finding of a moderating effect of religiosity on the etiology of adolescent alcohol problem scores is consistent with other studies demonstrating a moderating effect in adolescence (Cleveland and Wiebe 2003; Dick et al. 2007; Koopmans et al. 1999; Penninkilampi-Kerola et al. 2005). The lack of a significant moderating effect on the heritability in young adulthood is in contrast to the previous limited findings in adulthood, which showed a significant moderating effect of both marital status (Heath et al. 1989) and co-twin dependence (Penninkilampi-Kerola et al. 2005). However, both of these studies focused on alcohol use, rather than the more extreme alcohol problems, as was assessed in the current study. Furthermore, our results showed that religiosity significantly decreases longitudinally from adolescence to young adulthood, providing direct evidence of a decline in social control.

The results from the current study should be considered with respect to a number of limitations. We applied a normalizing transformation to our alcohol data prior to model fitting. This has the potential to eliminate some sorts of the interaction effects that might have been detected in the untransformed data. A related issue is that the moderation effects may be scale-specific. That is, the moderation may not be present when religiosity is scored in a different way. We tested this possibility by re-analyzing the data using summed scores of religiosity rather than the mean. We found that, similar to the results described above, there was a significant moderating effect of religiosity on genetic and environmental effects in adolescence but not young adulthood, and that these moderations went in the same direction as those previously reported. Consequently, these results add confidence to the

findings discussed above. Furthermore, as described in the method section, our model does not test for the moderation of those genetic effects on alcohol problems shared in common with religiosity; the interaction effects we report here are on those variance components specific to alcohol use. Finally, it is possible that the five-item scale of religiosity used in this study is only a proxy for a more robust values construct such as “Conformity to Social Norms”. The development of alcohol abuse or dependence symptoms in this largely under-21 population requires a willingness to flout legal restrictions on alcohol use for minors, which could be moderated by a willingness to adhere to community expectations for adolescents.

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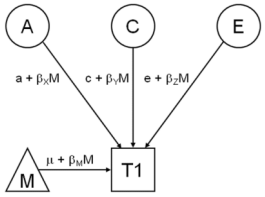

Figure 1.

A gene-environment interaction model

Partial path diagram showing only a single twin: a, c, and e = unmoderated genetic, common environment and non-shared environment components; β_X, β_Y and β_Z = moderated components of a, c and e respectively. β_M = main effect of moderator; M = moderator; μ = grand mean

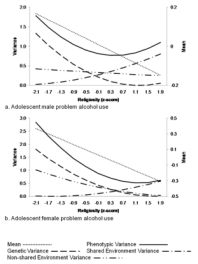

Figure 2.
Change in mean, genetic variance, shared environment variance, and non-shared environment variance for male (a) and female (b) problem alcohol use at time 1.

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Table 1

Male and female descriptive data and tests of sex differences in untransformed data

Phenotype	Female				Male				Mann Whitney U test	
	N	Mean	SD	AgeR	N	Mean	SD	AgeR	Z	p
Time 1										
Religiosity	1434	2.71	.94	−0.19	1301	2.56	0.94	−0.18	4.34	<0.001
Problem alcohol use	1440	0.28	.97	0.31	1307	0.30	1.00	0.34	0.61	0.54
Time 2										
Religiosity	1146	2.48	1.05	$-0.06^{n/s}$	976	2.28	1.04	$0.04^{n/s}$	4.45	<0.001
Problem alcohol use	1326	1.12	2.00	0.22	1144	1.64	2.25	0.29	6.99	<0.001

N = number of participants, S.D. = Standard Deviation, AgeR = correlation of phenotype with age

Table 2

Model fitting results

Model	Adolescence					Early adulthood				
	−2LL	df	χ^2	Δdf	p	−2LL	df	χ^2	Δdf	p
Full	6982.98	2695				5098.37	1953			
Equate Sex	7020.57	2701	37.59	6	<0.001	5115.55	1959	17.18	6	0.01
Drop β_Mf	7014.04	2696	31.06	1	<0.001	5115.79	1954	17.42	1	<0.001
Drop β_Xf	7027.98	2696	45.00	1	<0.001	5098.73	1954	0.36	1	0.55
Drop β_Yf	6996.61	2696	13.63	1	<0.001	5098.81	1954	0.44	1	0.51
Drop β_Zf	7005.98	2696	23.00	1	<0.001	5108.09	1954	9.71	1	0.002
Drop β_Mm	6990.64	2696	7.66	1	0.006	5123.05	1954	24.68	1	<0.001
Drop β_Xm	7005.470	2696	22.49	1	<0.001	5098.84	1954	0.47	1	0.49
Drop β_Ym	7001.942	2696	18.96	1	<0.001	5099.60	1954	1.23	1	0.27
Drop β_Zm	6987.643	2696	4.66	1	0.03	5099.24	1954	0.87	1	0.35

β_M = effect of moderator on the mean, β_X, β_Y and β_Z = moderated components of a, c and e respectively, f = female parameters, m = male parameters; −2LL = minus twice the log likelihood, *df* = degrees of freedom, χ^2 = chi square (estimated as the difference between the nested model and the full model), Δ *df* = difference in degrees of freedom between the full and nested models, *p* = probability estimate for the χ^2.