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Drinking Motives in Female Smokers: Factor Structure, Alcohol Dependence, and Genetic Influences

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Abstract

Background—Alcohol and tobacco use often co-occur. Human and animal studies indicate that nicotine increases alcohol's rewarding effects and the motivation to consume it. The aims of this study were to examine whether the factorial architecture of self-reported motivations to consume alcohol differed between regular and nonregular cigarette smokers while taking into account the lifetime history of alcohol dependence and psychopathology, and to estimate the genetic and environmental influences on the motivations.

Methods—Using data on 2,189 monozygotic and dizygotic female twins, we examined the factorial structure (item thresholds and factor loadings, means, and variances) of the items from the Drinking Motives Questionnaire (DMQ) in regular and nonregular smokers. Post hoc tests examined the association between the latent drinking motives factors and alcohol dependence in both groups. Twin models were fitted to the latent drinking motives factors, testing for variations in the magnitude of additive genetic, shared, and nonshared environmental influences between the groups.

Results—The 4 DMQ factors (social, conformity, coping, and enhancement) were recovered in both groups, and their measurement structure was consistent across the groups. Regular smokers reported higher levels of coping, enhancement, and social motives while nonregular smokers reported higher conformity motives. Alcohol dependence was associated with higher scores on all motives in both groups; however, in a regression analysis that included all of the motives as predictor variables, only coping was significantly related to alcohol dependence. While twin models revealed evidence for substantially greater genetic influences on enhancement ($h^2 = 0.40$), coping ($h^2 = 0.35$) and social ($h^2 = 0.37$) drinking motives in regular compared to nonregular smokers, the power to statistically distinguish the 2 groups was low.

Conclusions—While the measurement structure of the drinking motive factors appears to be similar across regular and nonregular smokers, regular smokers report more motivation to drink for internal affect-related reasons and to obtain social reward. Of all the motives, coping was the most robust predictor of alcohol dependence in both the regular and the nonregular smokers. Further, genetic influences might play a larger role in drinking motives among regular smokers, which provides tentative evidence for latent genetic × smoking status interactions.

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Keywords

Alcohol; Tobacco; Drinking Motives; Genetic Influences; Alcohol Dependence

The prevalence and chronicity of combined cigarette and alcohol use are already evident in early adulthood (Jackson et al., 2008). Cigarette smoking increases the pleasure derived from drinking alcohol and vice versa (McKee et al., 2004). Approximately 90% of individuals with alcohol dependence (AD) have a history of regular smoking, and estimates suggest that <10% of them successfully quit smoking (DiFranza and Guerrera, 1990). This may in part account for why over 50% of alcoholic smokers ultimately die from a tobacco-related disease (Hurt et al., 1996).

Biologic mechanisms contribute to cigarette and alcohol co-use. Smoking and alcohol use phenotypes are influenced by genetic and environmental factors (Kendler and Prescott, 2006; Kendler et al., 2007; Knopik et al., 2004; Lessov et al., 2004; Madden et al., 1999, 2004) and might share some common genetic influences (Kendler et al., 2007; True et al., 1999). Although the molecular and cellular mechanisms of action differ, both drugs affect common neural pathways and neurotransmitter systems (for reviews see Dani and Harris, 2005; Davis and de Fiebre, 2006; Dick and Bierut, 2006; Drews and Zimmer, 2009; Funk et al., 2006; Grucza and Bierut, 2006; Tyndale, 2003), and laboratory research in both humans and rodents suggest nicotine potentiates the rewarding effects of alcohol and/or the motivation to consume it (Barrett et al., 2006; Blomqvist et al., 1996; Clark et al., 2001; Kouri et al., 2004; Larsson and Engel, 2004; Le et al., 2000, 2003; Perkins et al., 1995; Potthoff et al., 1983; Smith et al., 1999).

Few studies have examined whether tobacco use affects the motivation to consume alcohol outside of experimental settings where dosage levels, administration schedules, and environmental factors are tightly controlled. The Motivational Model of Alcohol Use (Cox and Klinger, 1988) can be used to characterize the association between motivations and a range of individual and environmental variables. The model assumes that the decision to drink alcohol is based on its current incentive value, where its incentive value is determined by the change in current affective state that is anticipated to be brought about by consuming alcohol compared to not consuming alcohol (e.g., “How often do you drink because it gives you a pleasant feeling”; Cooper, 1994). In line with this model, if chronic nicotine potentiates alcohol-induced positive affective change, regular smokers should anticipate that alcohol will elicit larger or more rewarding affective changes compared to nonregular smokers, which should modify drinking motives. However, the extent to which the factorial architecture and measurement characteristics of drinking motives differ across types of smokers is largely unknown.

Drinking motives are known to relate to specific drinking behaviors and drinking problems in adolescents (Kuntsche et al., 2005). In adult samples as well, drinking to cope with negative affect has been found to be a robust predictor of alcohol use disorders (AUDs) and AUD symptoms (Beseler et al., 2008; Carpenter and Hasin, 1998a; Cooper et al., 1988, 1992). However, if exposure to cigarettes modifies drinking motives, it may be hypothesized that smoking status may alter the relationship between drinking motives and AD.

Similar to other aspects of alcohol-related behaviors, genetic influences also play a role in individual differences in drinking motives. While a study of adult twins (Prescott et al., 2004) found genetic factors to account for 23 to 48% of the individual differences in the drinking motives in women, genetic influences in men were less prominent. In prior analyses in a young adult female sample, however, the role of heritable influences on

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motives was modest, ranging from 11 to 33% (Agrawal et al., 2008). However, no study to date has explored the extent to which genetic influences on drinking motives are moderated by smoking status.

The architecture of motivations to drink may differ in several aspects in those who smoke. In this study, using a young adult female twin sample, we examine the following:

1. Whether the factor structure of the 4 latent factors representing social, conformity, enhancement and coping motives of the DMQ differed between regular smokers and nonregular smokers, and whether these differences would persist after adjusting for demographic variables and comorbid psychopathology.
2. Whether each drinking motive was associated with DSM-IV AD diagnoses and comorbid psychopathology and whether the strengths of the relationships between the drinking motives and AD differed between the 2 groups.
3. Whether the magnitude of genetic, shared, and nonshared environmental influences on each motive varied across nonregular and regular smokers. This tested for a latent genetic × smoking status interaction.

MATERIALS AND METHODS

Participant Recruitment and Characteristics

Interview data on 3,787 young adult women (mean age 21.99 years, range 18 to 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 to 2005. The "MOAFTS" study (PI Andrew Heath) consists of a cohort of female twin pairs born between July 1, 1975 and June 30, 1985. Twins, who were identified from birth records, were eligible to participate if both members of the twin pair had survived past infancy, were not adopted at birth and if their biologic mother was a resident of the state at the time of their birth. Using a cohort sequential sampling design for initial recruitment, interviews were attempted with at least one biologic parent (wherever possible, the biologic mother) and both twins during 1994 to 1999, when the twins were 13, 15, 17, or 19 years old. Recruitment of the 13-year-olds continued over a 2-year period as twins became age-eligible. After obtaining permission from parents, a telephone diagnostic interview was administered to the twins and their parents. Of the 2,369 twin pairs identified as live-born, 95.6% were located. The final sample of twins interviewed at baseline for each cohort included 1,633 pairs (72.5% of pairs targeted), including 579, 291, 367, and 373 pairs aged 13, 15, 17, and 19 years, respectively (n = 3446).

Subsequently, 3,060 women from this baseline interview were re-interviewed in 2002 to 2005, along with 728 women from the baseline sampling frame, who had not been interviewed previously. To minimize the sampling bias, all twins were invited to participate, provided they had not indicated previously an unwillingness to participate in future studies and provided that parents at baseline had not refused permission for the family to be contacted. The interview was adapted from the Semi-Structured Interview for the Study of the Genetics of Alcoholism (SSAGA; Bucholz et al., 1994) for telephone administration with restriction to DSM-based diagnoses. The interview also included a DSM-based nicotine dependency assessment based on the CIDI (Cottler et al., 1991). The sample in the present study included 2,189 women, aged 18 to 29 (Mn = 21.96), of European American descent who participated in the wave 4 follow-up interviews, provided questionnaire data, who reported at least one full drink lifetime and who also reported smoking at least one cigarette lifetime (i.e., all participants were exposed to smoking). The sample included 857 twin pairs

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and 475 singletons. A total of 508 pairs were monozygotic (MZ) twins and 349 were dizygotic (DZ) twins.

Measures

Drinking Motives—The Drinking Motives Questionnaire, Revised (DMQ-R; Cooper, 1994), was the primary outcome measure in this study. The DMQ-R measures 4 types of drinking motives: (i) enhancement (positive/internal; e.g., drink to improve positive mood state); (ii) coping (negative/internal, e.g., drink to relieve negative mood state); (iii) conformity (negative/external, e.g., drink to avoid social consequences); and (iv) social (positive/external, e.g., drink to obtain social rewards). The DMQ-R uses 5 items to measure each drinking motive (20 items total). In this study, the DMQ items were scored on an ordinal scale with 6 response categories (1 = never, 2 = almost never, 3 = some of the time, 4 = about half of the time, 5 = most of the time, 6 = almost always). Examination of the item response distributions indicated the participants tended not to endorse “most of the time” and “almost always” for the 5 conformity motive items, and the endorsement frequencies would not support multigroup analyses (see Data Analysis section below). Consequently, these responses were collapsed into a single category. Although the response distributions for the other 15 items were skewed, collapsing response categories was not necessary.

Smoking Status—Similar to previous research (Madden et al., 1997), regular smokers (n = 1181) were defined as those who had smoked 100 or more cigarettes lifetime or as those who had smoked only 21 to 99 cigarettes but reported smoking at least weekly for a period of 2 months or longer prior to the interview. Nonregular smokers (n = 1008) were defined as those who had smoked at least one but <100 cigarettes lifetime and did not report weekly smoking during the 2 months prior to the interview. As our interest was primarily on alcohol and tobacco co-users, those who had drank but never smoked at least one cigarette (n = 633) were excluded from these analyses.

Data Analysis

Validity of the 4-Factor Structure of the DMQ-R—Although confirmatory factor analytic studies using older adolescent and young adult samples provide empirical support for coping, enhancement, conformity, and social motives at the latent level (Cooper, 1994; Kuntsche et al., 2006, 2008; MacLean and Lecci, 2000), we first examined the validity of the 4-factor structure of the DMQ-R in our sample. These analyses were conducted with confirmatory factor analyses (CFAs) appropriate for ordered categorical data (Lubke and Muthén, 2004; Muthén, 1984) using mean and variance adjusted weighted least squares estimation (WLSMV) that accounted for missing observations in Mplus 5.2 (Muthén and Muthén, 1998–2007). The CFAs were conducted in the regular and nonregular smoker groups separately to ensure the 4-factor model (with correlated factors) fit the data adequately in both groups. Standard errors (for these and all other models) were adjusted for nonindependent observations because of familial clustering using the TYPE = COMPLEX and the CLUSTER commands. Adequacy of model fit was determined using the Comparative Fit Index (CFI), Tucker-Lewis Index (TLI), and Root Mean Square Error of Approximation (RMSEA).

Measurement Invariance of the DMQ-R Across Regular and Nonregular Smokers—Valid comparisons of latent factor means between 2 or more groups require the measurement structure of the factors to be sufficiently invariant across the groups (Lubke and Muthén, 2004; Meredith, 1993). Once the 4-factor model was determined to fit the DMQ-R data from each group reasonably well, multigroup CFAs were used to test for invariance in the thresholds and factor loadings (but not residual variances) across the groups. According to Lubke and Muthén (2004), this level of measurement invariance (MI)

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is necessary to make meaningful between-group comparisons. MI was determined with a chi-square difference test that used the Mplus DIFFTEST procedure as is necessary for models using the WLSMV estimator (Muthén and Muthén, 1998–2007).

To identify appropriate indicator items (items for which the factor loadings are fixed to "one" to set the scale of the latent factors) for the multigroup CFAs, we first conducted exploratory factor analyses (allowing for correlations among the factors) in each group separately. For each factor, the item with the Geomin-rotated loadings that were most similar across the groups was selected as an indicator.

The CFAs were identified using the DELTA parameterization (Muthén and Muthén, 1998–2007). The baseline (less restrictive) model was identified by: (i) setting the factor means to zero in both groups; (ii) setting the scale factors to one in both groups; (iii) freely estimating the factor variances and covariances in both groups; and (iv) freely estimating the thresholds and factor loadings in both groups. The more restrictive model (i.e., nested measurement invariance model) was identified by (i) setting the factor means in the nonregular smoker group to zero; (ii) freely estimating the factor means in the regular smoker group; (iii) setting the scale factors to one in the nonregular smoker group; (iv) freely estimating the scale factors in the regular smoker group; (v) freely estimating the factor variances and covariances in both groups; and (vi) constraining the thresholds and factor loadings to be equal across the groups.

Differences in Drinking Motives Between Regular and Nonregular Smokers— Next, we tested for between-group differences in the latent drinking motive factor means. Here, the factor mean for each drinking motive was adjusted for DSM-IV psychopathology. Zygosity (0 = MZ, 1 = DZ) and age (grand-mean centered) were also included as covariates. To adjust for DSM-IV psychopathology, we created composite "mood disorder problems" and "externalizing problems" variables. This was done because the base rates of most diagnoses (excluding AD) were too low to be included in the analyses as separate variables. The mood problems variable discriminated participants who reported unusually elevated mood on a nondiagnostic mania screen or who met diagnostic criteria for social phobia, panic attack, or major depression (coded 1) from those who did not (coded 0). The externalizing problems variable discriminated participants who met criteria for drug dependence or who reported 3 or more criteria for conduct disorder (coded 1) from those who did not (coded 0). Alcohol dependence (coded in the same manner) was entered into the model as a separate variable, which allowed us to examine the specific relationships of AD with the latent drinking motive factors.

Interactions of AD and the Covariates With Smoking Status—We tested for interactions of zygosity, age, mood disorder, and externalizing problems, and AD with smoking status within the multigroup CFA. To do so, we regressed the 4 latent drinking motive factors onto these variables, and we allowed the regression coefficients to vary across the regular and nonregular smoker groups. Then, we constrained the coefficients to be equal across the groups and tested the change in model fit using a chi-square difference test.

Genetic Analyses—We used Mplus to compute and output factor scores for each drinking motive for each participant. To do so, we used a multigroup CFA where we controlled for age by regressing each latent drinking motive factor onto the centered age variable. In this model, the regression coefficients associated with age were freely estimated across the 2 groups. Using the raw factor scores from MZ and DZ twin pairs, we used Mx (Neale, 2004) to estimate univariate twin models to partition the variance in the drinking motive scores into 3 sources: additive genetic (A), shared environmental (C), and unique environmental (E). We fit univariate models to the factor scores for the full sample and also

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separately to the regular and nonregular smokers. In the latter analyses, the magnitudes of A, C, and E across regular and nonregular smokers were allowed to vary with statistical tests examining whether they could be equated to each other to test for a latent genetic × smoking status interaction. Here, concordant regular, concordant nonregular, and discordant smoker pairs were modeled. Means for the motives were allowed to vary across the groups, which controlled for the spurious effect of latent genetic–environment correlation (i.e., smoking, the putative environment) mediating mean levels of self-reported motives (Purcell, 2002).

RESULTS

Table 1 shows that the regular smokers were significantly older and had significantly higher rates of all types of DSM-IV psychopathology compared to the nonregular smokers. The group means for each motive (computed using observed scores) also are shown. Regular smokers had significantly higher observed mean values for the coping and enhancement motives and significantly lower values for the conformity motives.

Validity of the 4-Factor DMQ-R Structure

The 4-factor structure (with correlated factors) adequately fit the DMQ-R data in the regular and nonregular smoker groups. In regular smokers, CFI = 0.954, TLI = 0.987, RMSEA = 0.077, and in the nonregular smokers, CFI = 0.950, TLI = 0.985, and RMSEA = 0.075.

Measurement Invariance

Tests for invariance of thresholds and factor loadings indicated that MI was not obtained, χ^2 (adjusted df, 48) = 127.98, $p < 0.001$. However, noninvariance was attributed to threshold differences between just 3 items: (i) How often do you drink to get high? (enhancement); (ii) How often do you drink because you feel more self-confident or sure of yourself? (coping); and (iii) How often do you drink to be sociable? (social). This indicated that the regular and nonregular smokers tended to use the response categories for these 3 items differently (Millsap and Yun-Tein, 2004). The CFA model assumed the response categories were cut-points on an underlying continuous latent response distribution (Lubke and Muthén, 2004; Muthén, 1984). Higher threshold values for the regular smoker group indicated that compared to nonregular smokers, regular smokers had higher values on this underlying distribution before they chose a higher response category (e.g., almost never) over a lower response category (e.g., never).

When the thresholds for these 3 items were freely estimated across the groups, partial MI was obtained, χ^2 (adjusted df, 43) = 56.65, $p = 0.08$. Notably, when we deleted these items from the model and again tested for MI, the thresholds and factor loadings for the remaining 17 items were fully invariant across the groups, χ^2 (adjusted df, 43) = 57.14, $p = 0.07$. Here, we report results from the model with partial MI, because follow-up CFAs (that used only the 17 invariant items) yielded identical results.

Interactions of AD and Covariates With Smoking Status

Compared to the model where regression coefficients associated with zygosity, age, mood disorder, and externalizing problems and AD were freely estimated across the groups, constraining the coefficients did not result in a significant decrease in model fit, χ^2 (adjusted df, 12) = 17.245, $p = 0.1406$. This indicated the size of the coefficients in regular smokers did not significantly differ from the size of the coefficients in the nonregular smokers; we did not find evidence for interactions of smoking status with AD or the covariates (i.e., there was no evidence that the relationships between the drinking motives and alcohol dependence differed between regular and nonregular smokers). The unstandardized coefficients (i.e.,

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partial regression coefficients) for each variable and for each latent drinking motive factor are shown in Table 2.

Relationships of AD With the Drinking Motives

After adjusting for zygosity, age, and mood disorder and externalizing problems, AD diagnosis was significantly associated with higher scores on enhancement, coping, conformity, and social motives. Additionally, mood disorder problems were associated with higher scores on coping and conformity motives and lower scores on enhancement and social motives. Also, a 1-year increase in age was associated with a 0.035 decrease in enhancement scores and 0.019 decrease in coping scores. Finally, externalizing problems were associated with higher coping scores. In a follow-up regression analysis, we examined the specificity of each drinking motive as a predictor of AD. After adjusting for enhancement, social, and conformity motives, only coping was significantly associated with AD ($z = 6.76$, $p < 0.001$) in both groups.

Differences in Drinking Motive Scores Between Regular and Nonregular Smokers—The covariate-adjusted differences between group means are shown in Table 3. Positive values indicate larger mean values for the regular versus the nonregular smoker group. After adjusting for zygosity, age, mood disorder and externalizing problems, and AD, the means for enhancement, coping, and social motives in the regular smokers were larger than those in the nonregular smokers. Conversely, the mean for conformity in the regular smokers was significantly lower than that in the nonregular smokers.

Genetic Analyses

Genetic analyses are based on 837 twin pairs. Of these, 217 MZ and 152 DZ pairs were concordant for regular smoking; 170 MZ and 80 DZ pairs were concordant nonregular smoking; and 107 MZ and 111 DZ pairs were discordant for smoking status. Table 4 shows the standardized estimates (A, C, and E) from the univariate twin models for each drinking motive as estimated for the entire sample (Panel 1), the MZ and DZ correlations for twin pairs concordant and discordant for smoking status (Panel 2) and the standardized estimates for each drinking motive from the univariate twin models stratified by smoking status (Panel 3).

Results of the univariate twin models for the entire sample (which included twin pairs discordant for smoking status) suggest statistically significant additive genetic effects on the scores for coping (30%) and social (25%) motives, but not on the scores for enhancement and conformity. Shared environment effects accounted for a significant portion of the variance in enhancement scores, but not in the scores for any other motives. For all 4 of the motives, the remaining variation in the scores was attributable to nonshared environment.

Examination of the MZ and DZ twin pair correlations for each drinking motive in the twins concordant for smoking status suggests an interesting trend. Specifically, in twins concordant for regular smoking, the size of the DZ twin pair correlation for each drinking motive is about half the size of the MZ twin pair correlation. In contrast, the MZ and DZ correlations are about equal in the twin pairs concordant for nonregular smoking. This suggests the influence of additive genetic effects on the drinking motive scores in regular smokers but not in nonregular smokers.

In the univariate twin models stratified by smoking status, there were no statistically significant differences in the additive genetic influences on the drinking motives between the regular and nonregular smokers. Specifically, the chi-square difference tests indicated that constraining the additive genetic (a), shared environmental (c), and nonshared

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environmental coefficients (e) to be equal across the groups did not result in a significant change in the fit of the model for enhancement χ^2 (3) = 2.69, p = 0.443, for coping, χ^2 (3) = 2.32, p = 0.501, for conformity, χ^2 (3) = 3.12, p = 0.374, or for social motives χ^2 (3) = 3.30, p = 0.348. As shown in Panel 3 in Table 4, the confidence intervals for the standardized estimates of the additive genetic influences on the drinking motives in the regular and in nonregular smokers all overlap. However, it is notable that the point estimates for the additive genetic influences on all of the drinking motives are consistently larger in the regular versus the nonregular smokers. Further, additive genetic influences on enhancement, coping, and social motive scores reached statistical significance in regular smokers, but did not in nonregular smokers.

To investigate this further, we computed another series of models for each drinking motive in the regular and nonregular smoker groups. Here, we dropped A, C, or A and C from the models and tested for significant deterioration in model fit using chi-square difference tests. For the regular smokers, dropping A from the models for enhancement and coping resulted in a significant deterioration in model fit, χ^2 (1) = 5.35, $p < 0.05$ and χ^2 (1) = 3.96, $p < 0.05$, respectively. In contrast, dropping C from these models did not result in a significant deterioration in model fit ($p > 0.05$ for both). For conformity and social motives, dropping either A or C did not result in a significant deterioration in model fit, whereas the models that dropped both A and C yielded a significantly poorer fit to the data, χ^2 (2) = 9.27, $p < 0.05$ and χ^2 (2) = 52.55, $p < 0.05$, respectively.

In the nonregular smoker group, dropping C resulted in significantly poorer fit for enhancement, χ^2 (1) = 7.51, $p < 0.05$, and social motives χ^2 (1) = 3.86, $p < 0.05$. For coping and conformity motives, A or C could be dropped without significantly reducing model fit. However, dropping both A and C yielded a significant deterioration in the fit of the model for coping, χ^2 (2) = 19.50, $p < 0.05$ and for conformity, χ^2 (2) = 6.47, $p < 0.05$.

These results are summarized in Panel 4 in Table 4, where the standardized variance estimates for the best-fitting models (either A–E, C–E, or A–C–E) are shown. Taken together, our results suggest a trend where drinking motives in regular smokers are genetically influenced more so than in nonregular smokers.

DISCUSSION

We sought to examine the architecture of drinking motives in a sample of young adult female smokers. The results from this study indicate the following: (i) while motives have a similar factorial structure across regular and nonregular smokers, those who are regular smokers are more motivated to drink for coping, enhancement, and social reasons, but are less motivated to drink for conformity reasons; (ii) after adjusting for demographic variables and psychopathology, those with AD are more motivated to drink for coping, enhancement, conformity, and social reasons compared to those without AD; and (iii) there is tentative evidence that drinking motives are more heritable in regular smokers than in nonregular smokers.

Differences in Drinking Motives Between Regular and Nonregular Smokers

Consistent with our expectations, regular smokers had significantly higher coping and enhancement motive scores compared to nonregular smokers, which suggests that regular smokers are more motivated to consume alcohol for internal, mood-related reasons. This result is in line with laboratory studies in animals and humans.

Evidence from rodent models suggests that nicotine potentiates the rewarding effects of alcohol and increases the motivation to consume it. For example, treatment with nicotine

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injections or subcutaneous nicotine capsules increases alcohol consumption (Blomqvist et al., 1996; Larsson and Engel, 2004; Potthoff et al., 1983; Smith et al., 1999) and increases and/or prolongs motivation to obtain alcohol (Clark et al., 2001; Le et al., 2000, 2003, 2009). Conversely, treating rats with the nicotinic receptor antagonist, mecamylamine, decreases alcohol consumption (Blomqvist et al., 1996; Larsson and Engel, 2004; Le et al., 2000).

Placebo-controlled co-administration studies with humans have found the same general pattern. For example, administration of transdermal nicotine (21 mg) has been found to increase reports of feeling drunk, feeling ethanol's effects, feeling euphoria, and the desire to drink in male smokers (Kouri et al., 2004). Nonnicotine-dependent male smokers, who received nicotinized cigarettes but not denicotinized cigarettes, reported increased motivation to consume alcohol compared to water using a progressive ratio schedule (Barrett et al., 2006). A study of deprived smokers by Perkins and colleagues (1995) found that compared to either drug alone, intranasal nicotine enhanced alcohol's stimulating effects and decreased its sedative effects. Conversely, treating nonsmokers with mecamylamine decreased subjects' self-reported desire to consume alcohol and reduced alcohol's euphoric and stimulant effects (Chi and de Wit, 2003).

Assuming these laboratory results generalize to other settings, one explanation for our results is that regular smokers generally find alcohol to be more rewarding. As drinking experiences accumulate (and holding demographic variables and psychopathology constant), regular smokers would anticipate that alcohol will elicit more positive affective change compared to nonregular smokers. According to the motivational model, this would result in the regular smokers reporting more motivation to drink for both coping and for enhancement reasons.

We also found that regular smokers were more motivated to drink for social (positive, external) reasons. Given that we adjusted for demographic factors and differences in the rates of psychopathology between the 2 groups, this also could be explained by the additive or interactive effects of nicotine on alcohol. If smoking is associated with stronger or more positive alcohol-induced affective changes, alcohol should serve as a better "social lubricant" for regular compared to nonregular smokers. Again, as drinking experiences accumulate, the positive reinforcement value of consuming alcohol in social settings would be greater for regular smokers leading to reporting of higher social motive scores.

In contrast, regular smokers reported significantly lower conformity motive scores compared to nonregular smokers, suggesting that regular smokers were less motivated to consume alcohol to avoid social consequences such as peer pressure to drink, not being "liked" and feeling "left out." There is evidence that adolescents who smoke are more likely to select friends who smoke (Engels et al., 1997), and there is a robust link between the smoking behavior of one's peers and current and future smoking behavior among adolescents and young adults (Ennett et al., 2008; Hoffman et al., 2006; Kobus, 2003; White et al., 2008). Coupled with evidence for peer influences on smoking in women (Rose et al., 1996), one explanation for our finding of lower conformity scores in regular smokers is that smoking behaviors, not alcohol consumption, might be the primary route for smokers to feel amalgamated with their social networks. Alternatively, because of the stigma associated with smoking in the present sociocultural milieu, regular smokers might also report lower conformity motive scores because they are less sensitive to social consequences generally.

Drinking Motives and Alcohol Dependence

Our results suggest that all drinking motives were more prominent in those with AD, and these relationships persisted even after adjusting for demographic variables and mood

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disorder and externalizing problems. However, in follow-up regression analyses in which all 4 motives were simultaneously regressed onto AD, only coping remained significantly associated with AD. These results are in line with previous studies reporting that coping is a specific and robust predictor of AUDs in adult samples (Carpenter and Hasin, 1998a,b, 1999; Cooper et al., 1988; Prescott et al., 2004; Young-Wolff et al., 2009).

We found no evidence that smoking status interacted with AD (or demographic or psychopathology variables) to influence the drinking motive scores. Interestingly, those with mood disorder problems were more motivated to drink for negative reinforcement reasons (to cope with negative mood and to avoid social consequences) but were less motivated to drink for positive reinforcement reasons (to enhance positive mood and to obtain social reward). This is likely due to the pervasiveness of negative affective states that characterize most mood disorders; individuals dealing with chronic negative affect would have fewer opportunities to experience the positive reinforcement value of consuming alcohol.

Genetic Influences on Drinking Motives in Regular and Nonregular smokers

To our knowledge, our study is the first to investigate the differences in the biometric structure of drinking motives as a function of smoking status. Largely consistent with Agrawal and colleagues (2008), we found modest heritable influences on coping and social motives and not on enhancement motives. While the present study did not find statistical support for heritable influences on conformity motives, the estimate was well within the confidence parameters of the prior study and may be attributable to our sample being restricted to European American subjects only and to those who were exposed to cigarettes. Across the smoking groups, while there was evidence for a greater magnitude of heritable influences on the motives in regular smokers, these differences were not statistically significant, potentially because of the reduced power.

Despite the potential influence of Type II error, our analyses suggest that in young adult women the motivation to consume alcohol for internal, mood-related reasons, and perhaps for positive, social reasons are genetically influenced more so in regular than in nonregular smokers. The slight differences in the heritabilities across the 2 groups indirectly imply that different mechanisms might be operating in individuals who are regular and nonregular smokers. First, the differences might be attributed to latent genetic × smoking interactions where exposure to differences in the social milieu (i.e., exogenous environments) of regular and nonregular smokers, such as situations where drinking and smoking jointly occur, modifies the magnitude to which latent genetic factors influence motives. Also, nicotine itself could be considered a modifying “endogenous environment.” Nicotine is known to produce numerous changes in neurobiologic structure and function (Laviolette and van der Kooy, 2004; Markou, 2008), and these alterations might contribute to differential manifestation of genetic influences on the drinking motives. Second, the motives might share common genetic etiologies with heritable phenotypes that differ between the regular and nonregular smoker groups. Specifically, significantly more regular smokers reported AD, mood disorder, and externalizing problems, and almost 50% of the regular smokers reported nicotine dependence (ND). Thus, motive-influencing genetic factors are embedded in genetic contexts associated with ND, AD, and psychopathology more so in the regular than in the nonregular smokers, and the different genetic contexts might alter the magnitude of heritable influences on the motives (either through correlations or interactions among these latent genetic factors). Third, differences in heritabilities could also be attributed to the nonrandom placement (Evans et al., 2002) of motive-influencing genotypes in the regular versus nonregular smoking environments. However, this constitutes a latent genetic–smoking correlation (rGE; Purcell, 2002; Scarr and McCartney, 1983)—our analyses control for rGE to some extent by allowing for smoking to mediate motive scores. However, the extent to which genetic factors influencing regular smoking overlap with genetic factors

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influencing motives, and further, the effects of smoking on this shared heritable component require significantly greater power and are beyond the scope of these analyses. Taken together, our results tentatively suggest a latent genetic × smoking status interaction, where (i) smoking itself modifies the magnitude of heritable variation in the motives; and/or (ii) smoking indexes a cluster of other heritable phenotypes (e.g., AD, ND, psychopathology) that aggregate and share genetic vulnerability with drinking motives. Further research with larger samples will be needed to disentangle the nature of these effects and to isolate whether similar or differing mechanisms are responsible for these fluctuations in heritability across the groups. Importantly, latent genetic influences refer to variation at the population level—the use of measured genotypes and their impact on individual differences in motives in regular and nonregular smokers may provide insight into the specific mechanisms at play.

Limitations

This study had a few limitations that might influence the interpretation and generalizability of our results. First, participants for these analyses were European American, Missouri-born female twins who might not be representative of individuals from other ethnic backgrounds or geographic regions, and our results might not extend to samples of different ages or to men. Comparable data on men is not available. Although the Drinking Motives Questionnaire (DMQ) factor structure was generally robust across African Americans and Caucasians in development samples (Cooper, 1994; Cooper et al., 1992), preliminary analyses suggested that the factor structure might be distinct in our sample of about 200 African-American subjects. However, the small sample size precludes any formal test of ethnic differences and data from these subjects were excluded from the present study. Second, although we controlled for demographic variables and psychopathology, other potential confounding factors (e.g., personality, stressors) might exist that remain unaccounted for. Third, we do not account for heterogeneity in regular smokers (e.g., regular smokers who are nicotine dependent), particularly the extent to which nicotine dependence influences the structure of drinking motives. However, follow-up analyses indicated that after adjusting for AD, mood disorder, and externalizing problems, the effects of ND on the drinking motive factors did not reach statistical significance. Further, as mentioned above, there was limited power to detect differential heritability of the drinking motives in regular and nonregular smokers, and we were not able to distinguish the mechanisms that contribute to this provisional effect. Finally, there was limited power to distinguish the sources of familial variance influencing the motives within the regular and nonregular smoker groups. In a classical twin study such as ours, this requires larger sample sizes (Neale et al., 1994).

Drinking motives are hypothesized to be the most proximal factors that influence the decision to use alcohol (Cox and Klinger, 1988). In conclusion, our study shows that in young adult women, smoking status can modify the architecture of drinking motives and possibly the extent to which heritable factors influence motivations to drink. An important area for future research will be to disentangle genetic and motivational mechanisms that contribute to alcohol and nicotine co-use in women and in men in both laboratory and naturalistic settings.

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Table 1

Descriptive Statistics for Regular Smoker and Nonregular Smoker Groups and Results of Statistical Tests for Between-Group Differences

	Regular smokers (*n* = 1,181)	Nonregular smokers (*n* = 1,008)	Between-group differences	
	Mn (SD) range	**Mn (SD) range**	***z***	***p***
Enhancement scale	3.03 (1.27)[a] 1–6	2.80 (1.27)[c] 1–6	4.01	$p < 0.001$
Coping scale	2.12 (1.06) 1–6	1.81 (0.96) 1–6	6.99	$p < 0.001$
Conformity scale	1.36 (0.64)[b] 1–5	1.43 (0.69)[c] 1–5	−2.65	$p < 0.01$
Social scale	2.90 (1.19)[b] 1–6	2.81 (1.20)[c] 1–6	1.62	ns
Age	22.21 (2.67) 18–29	21.66 (2.78) 18–28	4.07	$p < 0.001$
DSM-IV psychopathology (lifetime)	Frequency (%)	Frequency (%)	Odds ratio (95% CI)	
Nicotine dependence	560 (47.4%)	–	–	
Alcohol dependence	168 (14.2%)	61 (6.1%)	2.56 (1.89–3.50)	
≥3 Conduct disorder criteria	78 (6.8%)[d]	8 (0.8%)[e]	9.07 (4.44–18.53)	
Drug dependence	92 (7.8%)	13 (1.3%)	6.47 (3.48–12.02)	
Externalizing problems composite (conduct disorder & drug dependence)	144 (12.2%)	19 (1.9%)	7.23 (4.38–11.92)	
Elevated mood (mania screen)	99 (8.4%)	42 (4.2%)	2.10 (1.44–3.08)	
Major depression	334 (28.3%)	153 (15.2%)	2.20 (1.76–2.76)	
Social phobia	211 (17.9%)	101 (10.0%)	1.95 (1.51–2.53)	
Panic attack	202 (17.1%)	94 (9.3%)	2.01 (1.54–2.61)	
Mood disorder problems composite (elevated mood, depression, social phobia, & panic attack)	515 (43.6%)	256 (25.4%)	2.27 (1.88–2.74)	

Based on

[a] 1,180,

[b] 1,179,

[c] 1,007,

[d] 1,143, and

[e] 999 individuals with complete data.

All between-group differences in rates of psychopathology variables are statistically significant, $p < 0.01$, after adjusting standard errors for nonindependence because of familial clustering.

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Table 2

Results From the Regression of the Drinking Motive Factors onto Zygosity, Age, Mood Disorder Problems, Externalizing Problems, and Alcohol Dependence. Unstandardized Partial Regression Coefficients Are Shown

Drinking motive	Zygosity	Age	Mood disorder problems	Externalizing problems	AD
Enhancement	0.003	−0.035***	−0.125*	0.106	0.675***
Coping	0.000	−0.019*	0.156***	0.179*	0.742***
Conformity	0.019	−0.003	0.296***	−0.005	0.352***
Social	−0.043	−0.005	−0.120**	0.038	0.677***

Results are based on standard errors that are adjusted for nonindependence because of familial clustering. Coefficients are equal across the smoking groups.

*** $p < 0.001$;

** $p < 0.01$;

* $p < 0.05$.

Alcohol Clin Exp Res. Author manuscript; available in PMC 2011 August 14.

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Table 3

Covariate-Adjusted Group Mean Differences Between Regular and Nonregular Smokers, Standard Errors (SE), *z*-Test Results

Drinking motive	Group mean difference	SE	*z*	*p*
Enhancement	0.168	0.066	2.56	<0.05
Coping	0.302	0.067	4.50	<0.001
Conformity	−0.268	0.101	−2.65	<0.01
Social	0.178	0.073	2.43	<0.05

Standard errors are adjusted for nonindependence because of familial clustering. Positive group mean differences indicate larger mean values for the regular smoker group.

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Table 4

e Twin Models of Drinking Motives and MZ and DZ Correlations for Twin Pairs Concordant and

PANEL 1: Standardized univariate estimates of additive genetic (A), shared environment (C) and nonshared environment (E)										
		Coping			**Conformity**			**Social**		
		0.30 (0.04–0.38)			0.16 (0.00–0.24)			**0.25** (0.01–0.45)		
		0.01 (0.00–0.22)			0.00 (0.00–0.15)			0.13 (0.00–0.33)		
		0.69 (0.62–0.78)			**0.84** (0.76–0.93)			**0.62** (0.55–0.69)		
PANEL 2: Genetic correlations, stratified by smoking status, in MZ (r_{MZ}) and DZ (r_{DZ}) twin pairs										
		Coping			Conformity			Social		
nokers	Disc.	Concordant Regular Smokers	Concordant Nonreg. Smokers	Disc.	Concordant Regular Smokers	Concordant Nonreg. Smokers	Disc.	Concordant Regular Smokers	Concordant Nonreg. Smokers	Disc.
	0.394	0.371	0.298	0.096	0.189	0.147	0.119	0.413	0.398	0.294
	0.326	0.152	0.216	0.074	0.096	0.194	−0.087	0.282	0.374	0.139
PANEL 3: Standardized estimates of A, C and E from the full univariate models, stratified by smoking status										
		Coping			Conformity			Social		
		0.36 (0.06–0.45)	0.14 (0.00–0.38)[c]		0.17 (0.00–0.30)[c]	0.04 (0.00–0.27)[c]		**0.37** (0.04–0.51)[c]	0.06 (0.00–0.47)[a]	
		0.00 (0.00–0.28)[b]	0.14 (0.00–0.34)[c]		0.02 (0.00–0.19)[c]	0.14 (0.00–0.29)[c]		0.05 (0.00–0.34)[c]	0.33 (0.00–0.47)	
		0.64 (0.55–0.75)	**0.72** (0.61–0.84)		**0.81** (0.70–0.93)	**0.82** (0.69–0.96)		**0.58** (0.49–0.69)	**0.61** (0.51–0.72)	
PANEL 4: Standardized estimates of A, C and E from the best-fitting univariate models, stratified by smoking status										
		Coping			Conformity			Social		
		0.35 (0.24–0.45)	0.14 (0.00–0.38)[c]		0.17 (0.00–0.30)[c]	0.04 (0.00–0.27)[c]		**0.37** (0.04–0.51)[c]	–	
0.53)		–	0.14 (0.00–0.34)[c]		0.02 (0.00–0.19)[c]	0.14 (0.00–0.29)[c]		0.05 (0.00–0.34)[c]	**0.39** (0.29–0.49)	
0.66)		**0.65** (0.55–0.76)	**0.72** (0.61–0.84)		**0.81** (0.70–0.93)	**0.82** (0.69–0.96)		**0.58** (0.49–0.69)	**0.61** (0.51–0.72)	

ups that include 217 MZ and 152 DZ twin pairs concordant for regular smoking, 170 MZ and 80 DZ twin pairs concordant for nonregular
king status. Panel 2 shows twin pair correlations for MZ and DZ twins concordant and discordant for smoking status. Panel 3 shows

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standardized variance estimates for the full univariate twin models stratified by smoking status. Panel 4 shows the standardized variance estimates from the best-fitting models. Standardized variance estimates in bold differ significantly from zero, $p < 0.05$.

Disc. = discordant for smoking status.

[a] A could be dropped.

[b] C could be dropped.

[c] Either A or C, but not both, could be dropped from the model.