

---

# Genetic Influences on Vulnerability to, and Protective Factors for, Adolescent Drinking

---

Elizabeth A. Siewert, Michael C. Stallings, and John K. Hewitt

*Institute for Behavioral Genetics, University of Colorado, Boulder, Colorado, United States of America*

Using behavioral genetic analyses, we investigated and present a possible relationship between adolescent alcohol use and six domains of common problem behaviors in a community-based sample of 633 twin pairs who were under the legal drinking age of 21 (mean age = 15.0 years). The underlying etiology of the six problem behavioral domains, classified as conduct problems, hyperactivity, school problems, low self-esteem, neuroticism, and social withdrawal, was previously described (Siewert et al., 2003) as two heritable and genetically distinct dimensions of problem behavior. We took the two best-fitting models from that study (one that proposed a generalized behavior problem factor along with an internalizing behavior factor, and one that proposed an externalizing behavior factor along with an internalizing behavior factor) and extended the analyses in this study to include an index of alcohol use. Our results suggest that there is a strong genetic relationship between adolescent alcohol use and a broad spectrum of both externalizing and internalizing behavioral problems. The individual who seems to be at risk for either generalized or specifically externalizing behavioral problems is also at risk for adolescent alcohol use. However, the individual who exhibits internalizing problem behaviors appears to be protected from adolescent alcohol use. We propose that adolescent alcohol consumption needs to be understood in the context of these genetically influenced externalizing and internalizing propensities.

---

Alcohol use is pervasive among adolescents in the United States, even though it is illegal for those under 21 years of age. According to the *Monitoring the Future* survey (Johnston et al., 2003), a nationwide survey of drug and alcohol habits of adolescents within the United States, approximately 20%, 35% and 48% of eighth, tenth and twelfth graders respectively reported having used alcohol at least once in the past 30 days, while 7%, 18% and 31% of eighth, tenth and twelfth graders respectively reported having been drunk in the past 30 days. While these numbers have consistently declined every year since 1999, these estimates still indicate widespread teenaged drinking. Young et al. (2002) report that between the ages of 12 and 18 the self-reported lifetime use of

alcohol (defined as having ever used alcohol more than five times) increased from 2% for 12-year-olds to 68% for 18-year-olds. They found rates of alcohol abuse (*DSM-IV* criteria; APA, 1994) to be 0.4% for 12-year-olds and increasing to 18.4% for 18-year-olds. In addition to the problem of illegal alcohol use by adolescents, early alcohol use is associated with other behaviors that have serious negative consequences such as use of other illegal substances, behaviors that result in traumatic injuries, and risky sexual behavior (Armstrong & Costello, 2002; Parker et al., 1994; Tarter & Vanyukov, 1994; Wechsler et al., 1994). Poor socioeconomic and employment outcomes, as well as psychiatric disorders in adulthood, have also been associated with early alcohol use (Brook et al., 2002; Myers et al., 1998; Sher & Gotham, 1999; Turnbull et al., 1990).

However, not all individuals use alcohol, and among those who do, the amounts consumed vary greatly. There is increasing evidence that individual differences in adolescent alcohol consumption cannot be properly understood in isolation from individual differences in a wider range of behavioral problems (Kendler et al., 2003; Krueger et al., 2002; Young et al., 2000). To investigate this relationship between adolescent alcohol use and other behavioral problems, we extended our prior analyses of adolescent behavioral problems to incorporate an index of alcohol use. Previously, through exploratory factor analysis of 75 items from the Drug Use Screening Inventory (DUSI; Tarter, 1990; Tarter & Hegedus, 1991), we derived six problem behavioral domains underlying the DUSI domains. Three domains were characterized by externalizing behaviors: conduct problems, hyperactivity and school problems; and three domains were characterized by internalizing behaviors: low self-esteem, neuroticism, and social withdrawal (Siewert et al., 2004).

---

*Received 24 June 2004; accepted 6 August, 2004.*

*Address for correspondence: John Hewitt, Institute for Behavioral Genetics, University of Colorado, 447 UCB, Boulder, Colorado, 80309-0447, USA. E-mail: John.Hewitt@colorado.edu*

In a subsequent study, behavioral genetic analyses indicated an underlying etiology of these six problem behavior domains which could best be explained by one general genetic factor influencing all six domains, and a second latent genetic factor influencing the three internalizing domains (Siewert et al., 2003). An alternative model postulating two genetic factors with one factor influencing the three externalizing domains and the second influencing the three internalizing domains fits almost as well, but required a correlation of .75 between the two factors. However, one single general genetic factor did not provide an adequate fit. All of these models included a specific genetic factor for each domain and a full Cholesky nonshared environmental decomposition. From these findings, it is apparent that internalizing and externalizing problems share much of their genetic etiology; however, there appear to be distinct genetic influences on internalizing behavioral domains.

In the current study, we extend our previous analyses to investigate how these behavioral domains are related to alcohol use in adolescents. Since the six domains are only moderately intercorrelated, they may facilitate our interpretation and understanding of separable behavioral components of risk for alcohol use in adolescents.

## Methods

### Subjects

The subjects in this study were twins who had participated in the Colorado Adolescent/Adult Twin Study (CATS; Siewert et al., 2003, 2004). We selected those subjects who were younger than the legal drinking age of 21 years ( $M = 15.0$ ,  $SD = 2.36$ ). Informed consent was obtained from the individual twins, or a parent if the twins were under age 18. The Human Research Committee at the University of Colorado, Boulder, approved the research protocols and consent forms and the data were protected by a Confidentiality Certificate issued by the Department of Health and Human Services.

There were 633 twin pairs in this study. The zygosity of each same-sex twin pair was initially determined using responses to questions on the study registration form. The twins and a rater (usually a parent) were asked if they were identical or fraternal and how frequently they were mistaken for each other. Additionally, in some cases, two trained testers rated the similarity of the twins based on a 10-item assessment comparable to the form developed by Nichols and Bilbro (1966). Using this information, the twins were classified as monozygotic (MZ) or dizygotic (DZ). The final twin sample for this study included 119 MZ male, 208 MZ female, 76 DZ male, 100 DZ female and 130 opposite-sex pairs. Subsequently, the zygosity assignment was confirmed for 253 out of 259 twin pairs (97.7%) by genotyping the twins' DNA using nine highly polymorphic Short Tandem Repeat markers.

### Measures

The six measures of problem behavior were obtained through exploratory factor analysis of 75 items from the Drug Use Screening Inventory (Tarter & Hagedus, 1991). Summary scores were computed as unit-weighted sums of the items loading on each of the six factors. These domain scores were then normalized by applying square root transformations, and regressed on age, age-squared, sex, sex-by-age, and sex-by-age-squared as previously reported (Siewert et al., 2003). All analyses were performed on the residuals.

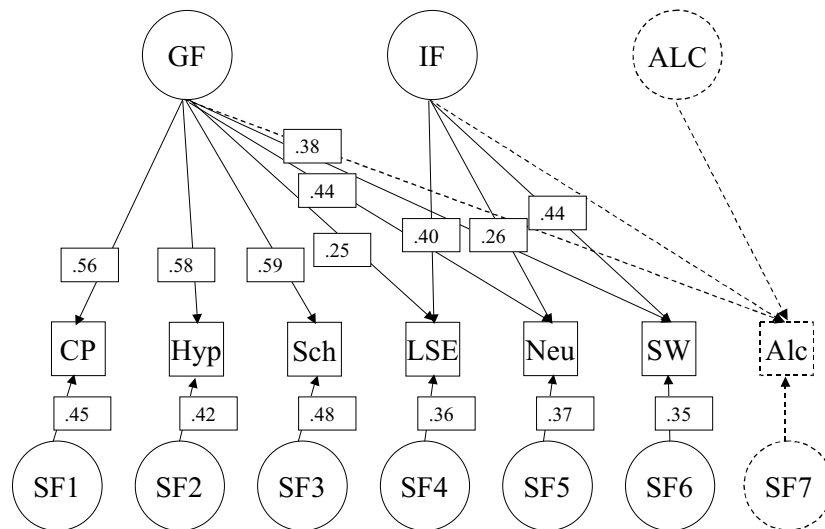
We used two Likert Scale items that asked about alcohol use to assess the drinking habits of these twins. One question addressed the frequency of drinking: 'How many times in the last month have you used alcohol?', and the second addressed the amount of drinking: 'If you drank in the last month, how many drinks do you usually have at one time?'

We chose to treat these two items as independent indices of alcohol use and summed them to obtain an overall alcohol-use index. We used this summary statistic because the sum, compared to the product (which would give an indication of volume of alcohol), had a distribution that was less skewed with a smaller kurtosis, and thus could be transformed to be more normally distributed. The correlation between these two summary statistics was .94. The resulting sum was normalized using a log transformation, and then age- and sex-corrected using standard regression in the same manner as described above. Subsequent analyses were performed on the residuals.

### Models

We used standard multivariate behavioral-genetic analyses (Neale & Cardon, 1992) to decompose the phenotypic covariance structure among our seven behavioral measures into additive genetic, nonadditive genetic, shared environmental and nonshared environmental sources of variance and covariance. Using the Mx software (Neale, 1999), we fit multivariate twin models to the five 14 X 14 variance/covariance matrices (one for each of the five zygosity groups) obtained using SAS (2000). Note that with MZ and DZ twins reared together, nonadditive genetic effects and shared environmental effects are confounded and cannot be estimated simultaneously.

In our previous report (Siewert et al., 2003), we found no significant evidence of nonadditive genetic or shared environmental influences and no significant sex differences in the etiology of the six problem behavior domains. Genetic influences on the six behavior domains could be modeled as two latent genetic factors: the first as a general genetic factor (GF) loading on all six behaviors, and a second genetic factor (IF) loading on only the three internalizing behaviors (low self-esteem, neuroticism and social withdrawal). Additionally, we modeled specific individual latent genetic factors and a Cholesky factorization of the non-shared environment loading on each of the six behavior domains. An alternative model with two correlated



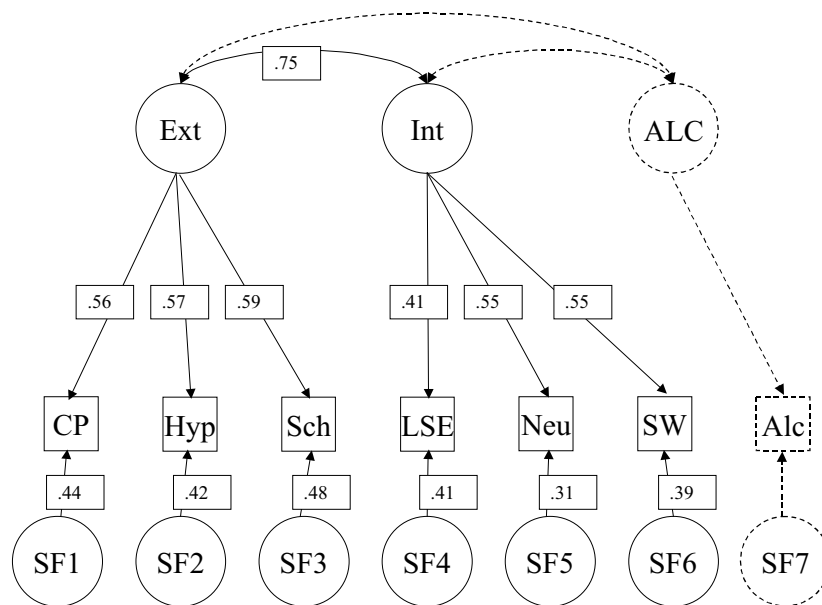
**Figure 1**

General factor/internalizing factor basis model: the loadings indicated for the general factor (GF), the internalizing factor (IF), and the specific factors are from a previous report (Siewert et al., 2003).

latent genetic factors that allowed the first factor to load only on the externalizing behavioral domains, and the second factor to load only on the internalizing behavioral domains, while retaining the specific genetic and nonshared environmental factors, as in the first model, also provided a good, although not the best, fit to the data.

In the current study, we included both these models as bases to further investigate the relationship between these behaviors and alcohol use in adolescents. We extended each of these two models to include an index

of alcohol use. We again considered the possibility of nonadditive genetic influences, shared environmental influences, and sex limitations contributing to the variance in the alcohol-use phenotype, but since these contributions were not significant, we did not include them in further analyses. Figures 1 and 2 picture the latent genetic factor structure for each model (general/internalizing and externalizing/internalizing respectively), with the results of the previous study shown in solid lines (point estimates of the genetic loadings/correlations indicated), and extensions to the



**Figure 2**

Externalizing factor/internalizing factor basis model: the loadings indicated for the externalizing factor (Ext), the internalizing factor (Int), and the specific factors are from a previous report (Siewert et al., 2003).

**Table 1**  
Phenotypic Correlations Among the 6 Behavioral Domains and the Alcohol-Use Index

	CP	Hyper	SchProb	LSE	Neu	SocWith	AlcUse
CP	1.000						
Hyper	.502	1.000					
SchProb	.483	.519	1.000				
LSE	.231	.232	.234	1.000			
Neu	.410	.451	.369	.488	1.000		
SocWith	.302	.261	.285	.456	.473	1.000	
AlcUse	.178	.140	.276	.015	.084	-.019	1.000

Note: conduct problems (CP), hyperactivity (Hyper), school problems (SchProb), low self-esteem (LSE), neuroticism (Neu), social withdrawal (SocWith), alcohol use (AlcUse).

model incorporating the index for alcohol use shown as dashed lines. For simplicity of presentation, only the genetic structure is shown. Alternative models were analyzed by constraining one or more of the dashed lines to be zero. For investigations of the genetic structure, a full Cholesky decomposition of the nonshared environmental covariance structure was used.<sup>1</sup> The model with the best fit was selected based on the Akaike Information Criterion, a fit index which takes into account the parsimony, as well as the fit, of the model (Akaike, 1987).

**Results**

**Correlations**

Table 1 shows the phenotypic correlations among the six behavior domains and the alcohol-use index. Although there were modest correlations between the Neuroticism domain and the three externalizing behavior domains, in general the three externalizing behavior domains tended to cluster, as did the three internalizing behavior domains (Siewert et al., 2003). The correlations between the six behavior domains with the alcohol-use index ranged from -.019 to .276. All three internalizing behavior domains had very low correlations with the alcohol-use index. In fact, the correlations with low self-esteem (.015) and social

withdrawal (-.019) were not significantly different from zero.

The correlations between Twin 1 and Twin 2 for the six behavior domains and the alcohol-use index are summarized in Table 2. The correlations used a listwise deletion for missing values, so therefore the number of twin pairs in Table 2 does not correspond to values previously indicated under the subjects heading. The correlations between MZ twins are generally twice that of the correlations between DZ twins. This is indicative of considerable genetic influence on these behavioral traits. Although there was little difference between the correlations of MZ males and DZ males for the alcohol-use index, and for the low self-esteem measure the DZ male correlation even exceeded the MZ male correlation, there was no significant shared environmental component to the variance in these measures within a multivariate model. Additionally, we investigated whether there were any sex differences for the shared environmental component of the alcohol-use index, and found no significant differences ( $\chi^2 = 3.020, df = 1, p = .082$ ).

**Model Fits**

Table 3 presents the resulting fits of the hypothesized models. The first three models were based on the 2-factor model where the first latent genetic factor influenced a generalized problem behavior, and the

**Table 2**  
Correlations Between Twin 1 and Twin 2 by Zygosity

	Male		Female		M/F
	MZ (n = 116)	DZ (n = 72)	MZ (n = 201)	DZ (n = 97)	DZ (n = 127)
CP	.642	.226	.494	.226	.285
Hyper	.659	.187	.501	.237	.183
SchProb	.619	.300	.563	.301	.286
LSE	.184	.303	.430	.214	.165
Neu	.424	.023	.434	.206	.220
SocWith	.318	.264	.559	.245	.205
AlcUse	.664	.557	.569	.282	.285

Note: conduct problems (CP), hyperactivity (Hyper), school problems (SchProb), low self-esteem (LSE), neuroticism (Neu), social withdrawal (SocWith), alcohol use (AlcUse).

**Table 3**

The Fits of 7 Models Where Models 1–3 are Based on the General Factor/Internalizing Factor Basis Model, and Models 4–7 are Based on the Externalizing Factor/Internalizing Factor Basis Model

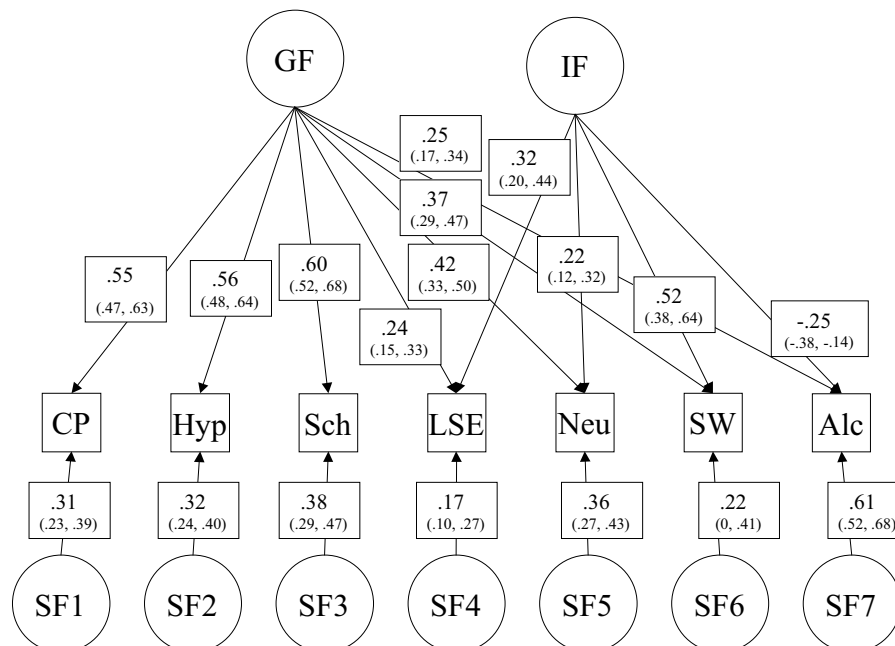
Model fits				
General factor/internalizing factor models	$\chi^2$	<i>df</i>	<i>p</i> -value	AIC
1 3 Latent A (loading 6 & 3 & 1)	530.81	481	.05	–431.19
2 2 Latent A (loading 7 & 3)	507.31	480	.19	–452.69
3 2 Latent A (loading 7 & 4)	488.27	479	.38	–469.73
Externalizing factor/internalizing factor models				
4 3 Latent A (loading (3 & 3 & 1) with $r_{Ext/Int}$ , $r_{Ext/Alc}$ and $r_{Int/Alc}$ )	504.19	481	.22	–457.81
5 3 Latent A (loading 3 & 3 & 1) with $r_{Ext/Int}$ and $r_{Ext/Alc}$ )	504.22	482	.23	–459.78
6 3 Latent A (loading 3 & 3 & 1) with $r_{Ext/Int}$ )	538.12	483	.04	–427.88
7 2 Latent A (loading 4 & 4) with $r_{Exp/Int}$ )	504.19	481	.22	–457.81

second latent genetic factor specifically influenced internalizing behaviors (see Figure 1). Model 1 hypothesizes a third latent genetic factor specifically influencing alcohol use that is independent of the other two latent genetic factors. This model fit the data poorly ( $\chi^2 = 530.81$ ,  $df = 481$ ,  $p = .05$ , AIC = –431.19).

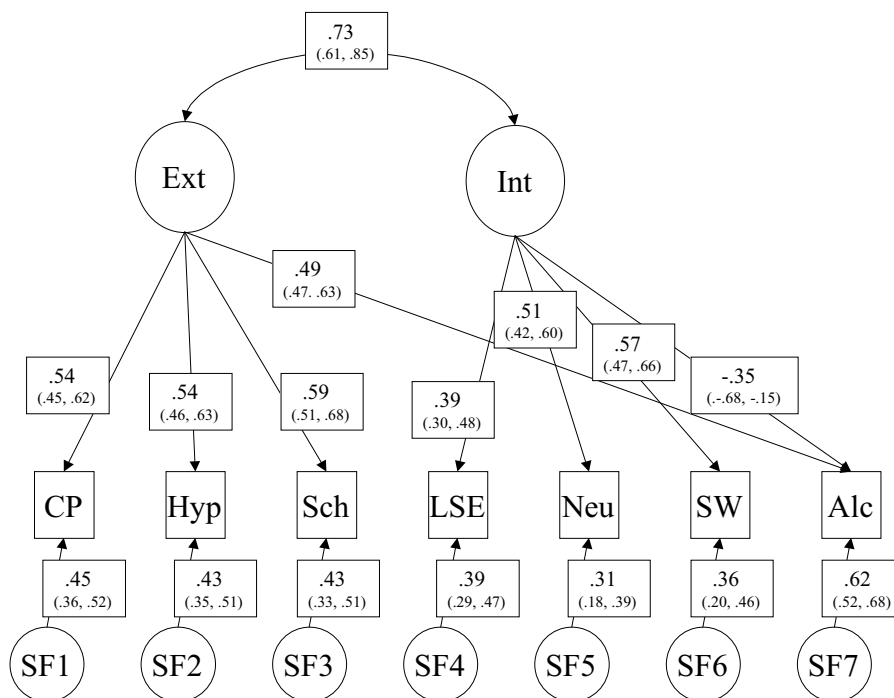
Models 2 and 3 hypothesize either the generalized or both the generalized and the internalizing factors influencing alcohol use. Although both these models fit well, the better fitting model by Akaike Information Criterion was Model 3, where both the generalized and the internalizing latent genetic factors influence alcohol use. Figure 3 depicts the resultant

loadings from Model 3 of the two latent factors on the six behavior domains and the alcohol-use index. Of note is the negative loading (–.25) of the internalizing latent factor on alcohol-use index.

The next four models tested were adaptations of the second basis model in which the two hypothesized latent genetic factors were correlated externalizing and internalizing factors (see Figure 2). While maintaining this correlation, Models 4, 5 and 6 hypothesized a third latent genetic factor loading on the alcohol-use index with the associated correlation pathways. Model 4 allows for correlations between the third latent factor, and both the externalizing and

**Figure 3**

Estimated loadings (95% CI) of latent genetic factors in best fitting model, based on the general factor/internalizing factor basis model. General factor (GF), internalizing factor (IF), specific factor (SF), conduct problems (CP), hyperactivity (Hyp), school problems (Sch), low self-esteem (LSE), neuroticism (Neu), social withdrawal (SW), alcohol use (Alc).



**Figure 4** Estimated loadings (95% CI) of latent genetic factors in best fitting model, based on the Externalizing Factor/Internalizing Factor Basis Model. Externalizing factor (Ext), internalizing factor (Int), specific factor (SF), conduct problems (CP), hyperactivity (Hyp), school problems (Sch), low self-esteem (LSE), neuroticism (Neu), social withdrawal (SW), alcohol use (Alc).

the internalizing latent factors. Model 5 allows for correlations only between the third latent factor and the externalizing latent factor, and Model 6 has no correlations between the third latent factor and either of the other two latent factors. Both Models 4 and 5 fit well, but since Model 5 is a more parsimonious model, it was the preferred model by the Akaike Information Criterion. When the model hypothesized a latent factor influencing alcohol use that is independent of the other two latent factors as in Model 6 (and as in Model 1), the result is a poor fit ( $\chi^2 = 538.12$ ,  $df = 483$ ,  $p = .04$ ,  $AIC = -427.88$ ).

Instead of modeling the covariances between the index of alcohol use and the other six behavior domains by correlating the third latent genetic factor with the other two latent genetic factors as in Models 4 and 5, we modeled the covariances by allowing the externalizing and the internalizing latent genetic factors to both load on the alcohol-use index (Model 7). This is essentially the same model as Model 4, and results in the same chi-squared statistic ( $\chi^2 = 504.19$ ,  $df = 481$ ,  $p = .22$ ,  $AIC = -457.81$ ), but it provides a clearer understanding of the relationship between these two clusters of behavior problems and alcohol use in adolescents. As in the best fitting model from the General/Internalizing basis model, note that, in this model (Figure 4) as well, the loading from the internalizing latent genetic factor onto the alcohol-use index is negative (-.35).

### Discussion

In this study, we sought to understand the genetic contributions to the complex mechanisms that influence alcohol use among adolescents who are younger than the legal drinking age of 21. Others have examined the phenotypic relationship between adolescent alcohol use and other characteristics. Guy et al. (1994) found that adolescent drug use is negatively associated with measures of socialization and obedience, and positively associated with measures of extraversion and later adult drug use. Mezzich et al. (1993) reported two variants of adolescent alcohol abuse/dependence: one characterized by behavioral dyscontrol, and the other characterized by problems of depressive and anxiety disorders. Miller-Johnson et al. (1998) found that conduct problems at sixth grade are a predictor of future adolescent substance use, while depressive problems are not. Tapert et al.'s (2002) findings indicated that limited attentional abilities (not necessarily attention deficit/hyperactivity disorder [ADHD]) predicts adolescent alcohol use, while Loeber et al. (1999) reported that ADHD does not, but persistent delinquency does predict persistent substance use. In this investigation, we extended our previous studies in which we first developed six clearly separable and interpretable behavioral domains (Siewert et al., 2004) and secondly analyzed the genetic and environmental influences on these domains (Siewert et al., 2003) using a community-

based sample of 633 twin pairs. From the results of the second study, we used two models as bases for this study of the genetic relationship of behavioral problems with alcohol use in adolescents. One basis modeled two latent genetic factors where one latent factor could be thought of as a generalized behavioral problem factor that loaded on all six behavioral domains, and the second latent factor could be thought of as an internalizing behavioral problem factor loading only on the cluster of three internalizing behavioral domains. The other basis also modeled two latent genetic factors. However, in this model the first latent factor loaded only on the cluster of the three externalizing behavioral domains, and the other latent factor loaded only on the cluster of the three internalizing behavioral domains. The data required the two latent factors in this model to be correlated.

We can draw several inferences from our analyses. First, we found a strong genetic relationship between alcohol use and a broad spectrum of externalizing behavior problems in adolescence. This corroborates Krueger et al. (2002) who found evidence for a highly heritable (81%) general factor linking measurements of antisocial behavior, conduct disorder, alcohol dependence, drug dependence, and a personality assessment of 'lack of constraint' in 17-year-old twins, and Young et al. (2000) who earlier reported a highly heritable factor of behavioral disinhibition loading on substance use, conduct problems, ADHD, and novelty-seeking. In epidemiological studies of adults, the comorbidity of substance use and common psychiatric disorders (Kendler et al., 2003) or alcohol dependence and conduct disorder (Slutske et al., 1998) were found to be predominantly genetic. This leads one to consider the types of interventions that can be established for adolescents with a genetic predisposition to early alcohol use, such as to teach them coping strategies to successfully compensate for this genetic predisposition.

We also found that regardless of the starting basis model, both latent genetic factors that influenced the problem behaviors also directly influenced alcohol use in adolescents. Of particular interest is the result that, in spite of the internalizing behavior domains being poorly correlated with the alcohol-use index, when the factor loadings from the general/externalizing latent factor were accounted for, the internalizing latent factor appears to have a protective effect on alcohol use.

From these results, we can formulate a relationship between alcohol use and common problem behaviors among adolescents. We propose that among adolescents, the degree of alcohol use is a result of both behavioral inhibition and externalizing behavioral factors. Those adolescents who, for genetic reasons, do not interact well with their peers (are neurotic, socially withdrawn and have a poor self image) are less likely to use alcohol. On the other hand, the adolescents who, for genetic reasons, are behaviorally and socially disinhibited (characterized by conduct

problems, hyperactivity and school problems) are more likely to use alcohol before it is legal.

The question remains: Will these socially withdrawn adolescents become involved with alcohol use and abuse when they reach young adulthood? Cloninger (1987) describes a Type 1 alcoholism which is characterized by late-onset and comorbid with internalizing psychopathology, and a Type 2 alcoholism, characterized by early-onset (before age 25) and comorbid with antisocial behavior. Babor et al. (1992) characterizes this phenomenon as Type A alcoholism characterized by late-onset, with fewer childhood problems, and less severe alcohol-related and psychopathological problems, and Type B alcoholism characterized by early-onset, with childhood risk factors, familial alcoholism, and greater severity of alcohol related and psychopathological problems. These findings underscore the importance of investigating the developmental processes of both early-onset and late-onset alcoholism. Is the socially withdrawn adolescent who is seemingly protected from illegal alcohol use the same individual who is at risk for Type 1/Type A alcoholism when he/she reaches adulthood? If so, what is the developmental pathway to this late-onset alcoholism? What interventions would facilitate the prevention of either type of adult alcoholism developing given the behavioral characteristics of the adolescent? Litt et al. (1992) report that Type A alcoholics responded better to interactional treatment, while Type B alcoholics showed better results with coping skills training. Our study provides support to the growing body of research that finds that adolescent alcohol consumption needs to be understood in the context of a wider range of behavioral characteristics. Directed interventions to avert adolescent or adult onset of alcoholism need to take into consideration these genetically influenced externalizing and internalizing propensities of the individual.

### Endnote

- 1 Analyses of the environmental covariance structure showed that the only significant nonshared environmental path between the six behavior domains and the alcohol-use index was between the school problem measure and the alcohol-use index ( $\chi^2 = 14.17$ ,  $df = 1$ ,  $p < .01$  for the GF/IF model, and  $\chi^2 = 16.72$ ,  $df = 1$ ,  $p < .01$  for the Ext/Int model).

### Acknowledgments

This work was supported by NIH grants EY12562 and DA11015. Ascertainment of the twin samples was supported in part by a grant from the Veteran's Administration, V554P-3828/3829.

### References

- Akaike, H. (1987). Factor analysis and AIC. *Psychometrika*, 52, 317-332.

- American Psychiatric Association (APA). (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- Armstrong, T. D., & Costello, E. J. (2002). Community studies on adolescent substance use, abuse, or dependence and psychiatric comorbidity. *Journal of Consulting and Clinical Psychology, 70*, 1224–1239.
- Babor, T. F., Hofmann, M., DelBoca, F. K., Hesselbrock, V., Meyer, R. E., Dolinsky, Z. S., & Rounsaville, B. (1992). Types of alcoholics. I. Evidence for an empirically derived typology based on indicators of vulnerability and severity. *Archives of General Psychiatry, 49*, 599–608.
- Brook, D. W., Brook, J. S., Zhang, C., Cohen, P., & Whiteman, M. (2002). Drug use and the risk of major depressive disorder, alcohol dependence, and substance use disorders. *Archives of General Psychiatry, 59*, 1039–1044.
- Cloninger, C. R. (1987). Neurogenetic adaptive mechanisms in alcoholism. *Science, 238*, 410–416.
- Guy, S. M., Smith, G. M., & Bentler, P. M. (1994). Consequences of adolescent drug use and personality factors on adult drug use. *Journal of Drug Education, 24*, 109–132.
- Johnston, L. D., O'Malley, P. M., & Bachman, J. G. (2003). *Monitoring the future. National results on adolescent drug use: Overview of key findings, 2002*. Retrieved February 11, 2004, from <http://www.monitoringthefuture.org>
- Kendler, K. S., Prescott, C. A., Myers, J., & Neale, M. C. (2003). The structure of genetic and environmental risk factors for common psychiatric and substance use disorders in men and women. *Archives of General Psychiatry, 60*, 929–937.
- Krueger, R. F., Hicks, B. M., Patrick, C. J., Carlson, S. R., Iacono, W. G., & McGue, M. (2002). Etiologic connections among substance dependence, antisocial behavior, and personality: Modeling the externalizing spectrum. *Journal of Abnormal Psychology, 111*, 411–424.
- Litt, M. D., Babor, T. F., DelBoca, F. K., Kadden, R. M., & Cooney, N. L. (1992). Types of alcoholics, II. Application of an empirically derived typology to treatment matching. *Archives of General Psychiatry, 49*, 609–614.
- Loeber, R., Stouthamer-Loeber, M., & White, H. R. (1999). Developmental aspects of delinquency and internalizing problems and their association with persistent juvenile substance use between ages 7 and 18. *Journal of Clinical Child Psychology, 28*, 322–332.
- Mezzich, A., Tarter, R., Kirisci, L., Clark, D., Buckstein, O., & Martin, C. (1993). Subtypes of early age onset alcoholism. *Alcoholism. Clinical and Experimental Research, 17*, 767–770.
- Miller-Johnson, S., Lochman, J. E., Coie, J. D., Terry, R., & Hyman, C. (1998). Comorbidity of conduct and depressive problems at sixth grade: Substance use outcomes across adolescence. *Journal of Abnormal Child Psychology, 26*, 221–232.
- Myers, M. G., Stewart, D. G., & Brown, S. A. (1998). Progression from conduct disorder to antisocial personality disorder following treatment for adolescent substance abuse. *American Journal of Psychiatry, 155*, 479–485.
- Neale, M. C. (1999). *Mx: Statistical Modeling* (5th ed.). Richmond, VA: Department of Psychiatry, Medical College of Virginia.
- Neale, M. C., & Cardon, L. R. (1992). *Methodology for genetic studies of twins and families*. NATO ASI series. Dordrecht, the Netherlands: Kluwer Academic.
- Nichols, R. C., & Bilbro, W. C., Jr. (1966). The diagnosis of twin zygosity. *Acta Geneticae Statisticae Medicinae, 16*, 265–275.
- Parker, D. A., Harford, T. C., & Rosenstock, I. M. (1994). Alcohol, other drugs, and sexual risk-taking among young adults. *Journal of Substance Abuse, 6*, 87–93.
- SAS. (2000). *SAS/STAT user's guide* (Version 8). Cary, NC: SAS Institute.
- Sher, K. J., & Gotham, H. J. (1999). Pathological alcohol involvement: A developmental disorder of young adulthood. *Developmental Psychopathology, 11*, 933–956.
- Siewert, E. A., Stallings, M. C., & Hewitt, J. K. (2003). Genetic and environmental analysis of behavioral risk factors for adolescent drug use in a community twin sample. *Twin Research, 6*, 490–496.
- Siewert, E. A., Stallings, M. C., & Hewitt, J. K. (2004). Factor structure and concurrent validity of the drug use screening inventory in a community adolescent sample. *Addictive Behaviors, 29*, 627–638.
- Slutske, W. S., Heath, A. C., Dinwiddie, S. H., Madden, P. A., Bucholz, K. K., Dunne, M. P., Statham, D. J., & Martin, N. G. (1998). Common genetic risk factors for conduct disorder and alcohol dependence. *Journal of Abnormal Psychology, 107*, 363–374.
- Tapert, S. F., Baratta, M. V., Abrantes, A. M., & Brown, S. A. (2002). Attention dysfunction predicts substance involvement in community youths. *Journal of American Academy of Child and Adolescent Psychiatry, 41*, 680–686.
- Tarter, R. (1990). Evaluation and treatment of adolescent substance abuse: A decision tree method. *American Journal of Drug and Alcohol Abuse, 16*, 1–46.
- Tarter, R., & Hegedus, A. (1991). The Drug Use Screening Inventory: Its application in the evaluation and treatment of alcohol and drug abuse. *Alcohol Health and Research World, 15*, 65–75.
- Tarter, R. F., & Vanyukov, M. (1994). Alcoholism: A developmental disorder. *Journal of Consulting and Clinical Psychology, 62*, 1096–1107.



- Turnbull, J. E., George, L. K., Landerman, R., Swartz, M. S., & Blazer, D. G. (1990). Social outcomes related to age of onset among psychiatric disorders. *Journal of Consulting and Clinical Psychology, 58*, 832–839.
- Wechsler, H., Davenport, A., Dowdall, G., Moeykens, B., & Castillo, S. (1994). Health and behavioral consequences of binge drinking in college. A national survey of students at 140 campuses. *Journal of American Medical Association, 272*, 1672–1677.
- Young, S. E., Corley, R. P., Stallings, M. C., Rhee, S. H., Crowley, R. J., & Hewitt, J. K. (2002). Substance use, abuse and dependence in adolescence: Prevalence, symptom profiles and correlates. *Drug and Alcohol Dependence, 68*, 309–322.
- Young, S. E., Stallings, M. C., Corley, R. P., Krauter, K. S., & Hewitt, J. K. (2000). Genetic and environmental influences on behavioral disinhibition. *American Journal of Medical Genetics, 96*, 684–695.
-