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# Personality at Ages 16 and 17 and Drinking Problems at Ages 18 and 25: Genetic Analyses of Data from *FinnTwin16-25*

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We enrolled more than 3500 same-sex twins from 5 consecutive Finnish birth cohorts into a longitudinal study as each cohort reached age 16. Twins completed the Psychopathic Deviate (Pd) Scale of the Minnesota Multiphasic Personality Inventory at baseline, Sensation Seeking Scale items as each cohort reached age 17, and later, at average ages 18.5 and 25, the Rutgers Alcohol Problem Index (RAPI). Using raw maximum likelihood estimation, we fit a Cholesky model to the 4 variables assessed at 4 ages across the 4 twin types; we estimated genetic and environmental influences on the stability of alcohol problems across development and the genetic and environmental contributions to predictive correlations between adolescent personality and later alcohol-related behavior problems. With one exception, the phenotypic, genetic, and environmental correlations were very similar for males and females. The exception was that the lagged associations of Pd and RAPI reflect a higher genetic correlation among males than females and a higher environmental correlation among females than males. Our analyses suggest that developmental changes underlying variation in alcohol problems from late adolescence to early adulthood differ for males and females. In males, the main change is decreased variation due to shared environmental effects; the magnitude of genetic effects is stable over time, and the high genetic correlation, .95, suggests that the same genetic influences are important at both ages. Among females, in contrast, genetic influences decline in magnitude from age 18 to 25, and at least part of the genetic effect evident at age 25 differs from the genetic effect evident at age 18.

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A number of recent twin studies have focused on developmental trajectories of alcohol use from early adolescence into early adulthood (Hopfer et al., 2003; Rose & Dick, 2004/2005). And for obvious reasons: Drinking is typically initiated in early adolescence, alcohol dependence often originates by late adolescence, and earlier drinking onset is a risk factor, albeit

one of uncertain meaning, for the later development of alcoholism (Grant & Dawson, 1997). Identifying and distinguishing the interplay of genetic and environmental factors in the initiation of drinking and in childhood predictors of alcohol abuse in early adulthood is, accordingly, a high research priority, one reflected in twin studies conducted in Australia (Heath & Martin, 1988), Finland (Rose, Dick, Viken, Pulkkinen, et al., 2001; Viken et al., 1999), Minnesota (Han et al., 1999; King et al., 2004), Missouri (Bucholz et al., 2000), the Netherlands (Koopmans et al., 1999), and Virginia (Maes et al., 1999), and in analyses of data from a multistate United States (US) sample (Hopfer et al., 2005).

However much such research has enhanced our understanding of developmental patterns of adolescent drinking, its implications for understanding clinical problems associated with drinking are more limited. In part, that is because the studied samples of twins are population-based, and the twins are in their initial stages of alcohol use, so few exhibit symptoms of alcohol dependency. In a Finnish twin sample modestly enriched for familial risk, only 12% exhibited any symptoms of alcohol dependency at age 14, less than 1% met diagnostic criteria, and no genetic effects were found on symptom counts among either boys or girls (Rose et al., 2004). But there are other complexities: trajectories of high-density drinking exhibit different developmental pathways from adolescence into early adulthood (Schulenberg et al., 1996). And there is wide variation in the drinking behaviors of alcohol-dependent males and frequent fluctuations in the course of their individual drinking histories from adolescence into midlife (Sartor et al., 2003). It is likely that multiple developmental pathways to alcoholism exist, and likely, as well, that different pathways have different genetic loadings.

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Received 10 March, 2006; accepted 13 November, 2006.

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Individual differences in frequency/quantity/density of adolescent alcohol use correlate but moderately with individual differences in alcohol-related problems; some heavy adolescent drinkers report many drinking-related problems, but others do not (Thombs & Beck, 1994). Continuity between adolescent drinking and early adult outcomes is strongly associated with family background factors, possibly reflecting those factors rather than direct consequences of adolescent drinking (Wells et al., 2004). There is evidence of gender differences in the development patterning of adolescent alcohol use and in the transitioning in or out of heavy drinking, from late adolescence into early adulthood (Jackson et al., 2001). And there is some evidence that genetic factors have less influence on high-density drinking in young adult women than men (King et al., 2005), and suggestive evidence that gender moderates the associations of drinking patterns, drinking-related problems, and symptoms of psychological distress (Geisner et al., 2004).

Taken together, this research literature invites a longitudinal twin study that is focused directly on negative consequences of alcohol use, to explore developmental modulation of genetic and environmental contributions to alcohol-related behavioral problems from late adolescence to early adulthood. With appropriate data from Finnish twins, we sought to study the genetic and environmental contributions to stability of drinking problems across this developmental period. We ask whether genetic and environmental contributions to age-to-age consistency of drinking-related behavior problems differ in men and women. To data from two occasions of self-reported drinking-related problems, we added two prospectively measured dimensions of personality, given research evidence that personality antecedents assessed in adolescence predict risk for alcoholism (e.g., Finn et al., 2000; Sher et al., 2000). Using data from the four assessments made in *FinnTwin16-25*, we report a longitudinal analysis of drinking problems reported at ages 18 to 19 and 23 to 27 and the association of drinking problems with two risk-relevant dimensions of personality, assessed from earlier self-reports obtained at ages 16 and 17.

## Method

### Setting

A half-century ago, the Finnish Foundation for Alcohol Studies appointed a research team to plan a twin study of genetic influences on alcohol use. Using nationwide records maintained by parish churches, all twin brothers born in Finland during the 1920s were identified, and the resulting research publication, *Inheritance of Drinking Behavior* (Partanen et al., 1966) became a classic contribution to the field. In 1974, 1 year after demographic data on all Finnish citizens had been collated into the Population Register Center (PRC), the Finnish Twin Cohort Studies were established, and a baseline questionnaire was mailed out in 1975 (Kaprio

et al., 1978). During the mid-1980s, birth cohorts of younger twins were identified from the PRC and longitudinal studies of adolescent Finnish twins (including the *FinnTwin16-25* study for this report) began.

As a setting in which to conduct longitudinal twin research, Finland offers advantages shared with other Nordic countries, as well as some unique to it (Rose, 2006). Finnish twins from any given birth cohort can be ascertained readily and followed throughout their lives. The PRC contains data on all Finnish citizens; each newborn Finn is given a unique identifying number that incorporates date of birth and a linkage to the newborn's biological mother. The PRC contains a current residential address for each individual Finn and updated information on the family composition and its births, deaths, marriages and divorces. Irretrievable loss of Finns to follow-up across their lifetimes is minimized, given access to their residential addresses and the individualized linkage of each Finnish citizen to health and institutional outcome measures. In common with their Nordic neighbors, Finns have a long history of civil registration and of voluntary participation in epidemiological research; compliance with research requests by Finnish twins and their families is among the highest anywhere, and neither incomplete ascertainment nor self-selection biases create inescapable concerns.

Other aspects of Finnish society, unusual if not unique, make it a superb setting for longitudinal twin research. These importantly include the uniformly high quality of Finnish public education and the nationwide delivery of healthcare services within the Finnish welfare state. Structured along the Nordic welfare model, the Finnish system applies the principle of universality, granting the right of all citizens to education and healthcare, regardless of their residence, occupation or socioeconomic position. Organized childcare is an integral feature of contemporary Finland, dropouts from comprehensive school are rare, and one in three adult Finns, the highest percentage among all European Union EU countries, has a university or other tertiary qualification.

The extraordinary quality of Finland's comprehensive educational system is evident in ongoing research conducted by the Organization for Economic Cooperation and Development (OECD); reports from the OECD show that Finnish public education achieves the highest international standard in both reading and mathematics, and the high rankings are achieved with remarkably modest between-school variation and little association with familial socioeconomic status (OECD's Programme for International Student Assessment, PISA; [www.pisa.oecd.org](http://www.pisa.oecd.org)). PISA is a 3-yearly study of knowledge and skills of over 250,000 15-year-olds in the 30 OECD member countries and PISA partner countries. Finnish adolescents achieved top ranks in both reading performance and mathematical literacy, and importantly for genetic research, their high achievement was much less influenced by differences in their school or

family backgrounds than was found for most participant countries. Between-school differences accounted for 35% of the variation in reading performance across all participant OECD countries, but only 12% of individual differences in reading in Finland. For Finnish adolescents, variance in mathematics literacy attributable to between-school differences was but one tenth of the OECD average. Socioeconomic status differences were the strongest single factor in the PISA assessments, accounting for 21% of total variance in reading performance in results from the US and averaging 20% across all OECD countries; in contrast, socioeconomic status differences accounted for only 9% of the variance in performance of Finnish school children.

A similar pattern of nationwide homogeneity holds for the delivery of health services. Of uniformly high quality, it is available to all, regardless of social circumstance. Adjusted for its population, the availability of hospital beds and practicing physicians places Finland favorably among world nations, and the nationwide quality of medical services, from prenatal care to extended care of the elderly, is reflected in high life expectancy and a low rate of infant mortality, for which Finland, with its Nordic neighbors, Iceland and Sweden, ranks among the first five world countries.

Finally, like other population isolates, Finland may offer advantages in genetic mapping for complex traits (Peltonen et al., 2000; Varilo & Peltonen, 2004). The history of Finland is a history typical of isolates: a small number of founders, subsequent isolation, rapid expansion and bottlenecks created by war and environmental disruption. A number of mostly recessive diseases are enriched in Finland, while others common elsewhere are almost nonexistent.

Finland's demographic registries, the high quality and relative uniformity of its educational training and healthcare delivery, and its history as a geographic and linguistic isolate, make Finland an unusual living laboratory for genetically informative research.

### Sample

Using the PRC, we ascertained twins in five consecutive nationwide birth cohorts (1975–1979) and enrolled them into a longitudinal study as they reached age 16 in 1991 to 1995. Response rates were high and unrelated to twin type, so the realized sample (called *FinnTwin16-25*) contained equal thirds of brother–brother, sister–sister, and brother–sister twin pairs. Questionnaires were mailed out during 10 months of each year, across the 5-year baseline period, to achieve a narrow age-standardization (Kaprio et al., 2002). The first follow-up, at age 17, used a similar procedure, staggering mail out of questionnaires by birth date across 60 months' time. Subsequently, we telescoped the procedure, sending questionnaires quarterly for Wave 3, when twins were aged 18 to 19 (mean 18.4, hereafter designated as age 18) and semi-annually for Wave 4, when the twins were aged 22 to 27 (mean 24.6, hereafter designated as age 25); individual response rates approached or exceeded 90%

through age 18 for both twin brothers and sisters, and at the age 25 follow-up, response rates remained above 90% for females, but participation declined to 83% among males. Zygosity of same-sex pairs was determined by questionnaire responses obtained separately at baseline from both co-twins and one or both of their parents (Rose et al., 2001).

### Measures

Finland is a bilingual country: 6% to 7% of the population speaks Swedish, rather than Finnish. Information contained in the PRC indicates each family's preferred language, and, accordingly, all *FinnTwin* questionnaires were prepared in both Swedish and Finnish; questionnaire content translated from English was back-translated as needed.

The Rutgers Alcohol Problem Index (RAPI; White & Labouvie, 1989) was used to assess negative consequences of drinking at ages 18 and 25. RAPI is a 23-item checklist of behavior problems consequent to consuming alcohol; item content includes injury to self and others, neglected responsibilities, emotional problems, and personal and interpersonal loss associated with drinking. One RAPI item concerning interference from alcohol use with schoolwork or exam preparation was deleted, because all Finnish twins had completed mandatory education when first assessed at age 18. Our Finnish adaptation of RAPI, therefore, contained 22 items, with four response alternatives for reporting frequency of each consequence. Developed and widely used in the US, RAPI exhibits good internal consistency (White & Labouvie, 1989; coefficient alpha for our 22-item scale was .87); RAPI has been employed effectively in other cultures from New Zealand (Fergusson & Horwood, 2000) to Norway (Pedersen & Skrandal, 1996). As with many measures of problem behavior, RAPI scores show a strong positive skew. We report descriptive data on untransformed RAPI data to facilitate comparisons with other data sets, but we used log-transformed data to compare means for males and females, to compute twin correlations and for biometric analyses.

The Minnesota Multiphasic Personality Inventory (MMPI) Psychopathic Deviate (Pd) Scale (Dahlstrom et al., 1972) was included in the baseline questionnaire administered at age 16. The scale includes 50 true/false items related to family conflict, social isolation, life dissatisfaction, and difficulty dealing with authority figures. Test–retest stability is quite high and given its derivation, internal consistency of the Pd scale is satisfactory; coefficient alpha in our age 16 Finnish data was .65. A peak score on Pd is a common characteristic of MMPI profiles from alcoholic and delinquent samples. MMPI profiles routinely obtained from entering college students showed that elevated Pd Scale scores distinguished men later hospitalized for alcoholism from matched controls (Loper et al., 1973); an early result consistent with evidence, now substantial, that pre-alcoholics tend to be more impulsive, nonconforming and gregarious.

In subsequent research individual differences in personality predictive of alcohol abuse have been broadly conceptualized as ‘disinhibited’ or ‘externalizing’ dimensions of personality: novelty- or sensation-seeking and boredom susceptibility, combined with fearlessness, impulsiveness, and inattention. Whether self-reported or rated by parents, teachers, or peers, these measures predict an earlier onset of drinking and an earlier trajectory to high-density drinking. The age 17 follow-up included 24 items drawn from the Sensation Seeking Scale (SSS; Zuckerman, 1979), a widely used measure of the novelty-seeking and disinhibitory dimensions of personality, and its items have well-established psychometric properties. The 24 items in our short form included representative items from all SSS subscales. The item format of the SSS requires a forced choice of two alternatives, for example, a preference for friends who ‘are excitingly unpredictable’ versus those who ‘are reliable and predictable’.

### Analyses

We performed our analyses with and without inclusion of individual twins who reported abstinence from alcohol prior to the RAPI assessments at ages 18 and 25; the number of consistently abstinent twin individuals was very small (~5% of twins contributing data for these analyses consistently reported abstaining on each assessment in which they participated, with abstinence sharply declining from age 16 to 25), and no obvious difference was found by their exclusion; accordingly, we report results using data from all same-sex twins for whom the necessary data across four occasions were available. Individual twins from incomplete pairs were included if their zygosity was known (e.g., if both co-twins responded at age 16, but only one at age 25), and for that reason, the number of individual twins in our analyses is a bit larger than twice the numbers of twin pairs. To simplify the multivariate analyses and their interpretation, and to focus on genetic associations between personality and later drinking-related outcomes, we restricted our analyses to same-sex twin pairs. Our analyses fit a Cholesky model to the four variables across the four twin types, using raw maximum likelihood estimation in Mx (Neale et al., 1999). The saturated baseline model estimated additive genetic (A), shared environmental (C), and unshared environmental (E) effects for all four variables, freely estimated genetic and environmental covariances among the four variables, and allowed for sex differences on all parameters. We fit a nested submodel that constrained estimates to be equal for males and females to test for sex differences.

### Results

Descriptive results, based on raw scores for each measure, are shown in Table 1. The means and variances of scores from both adolescent personality measures, Pd and SSS, are very similar across gender

**Table 1**  
Descriptive Statistics for the Four Measured Variables

	Pd16	SSS17	RAPI18	RAPI25
<b>Males</b>				
Mean	16.97	12.31	29.52	29.17
(SD)	(5.19)	(4.01)	(7.73)	(7.98)
N	1619	1525	1480	1337
<b>Females</b>				
Mean	17.07	12.41	28.59	26.49
(SD)	(5.36)	(4.15)	(7.03)	(6.18)
N	1902	1867	1826	1691

Note: Pd16 = psychopathic deviate scale of the MMPI assessed at age 16; SSS17 = 24 items from the Sensation Seeking Scale completed at age 17; RAPI18 = Rutgers Alcohol Problem Index at age 18; RAPI25 = Rutgers Alcohol Problem Index at age 25; the N listed for each cell is the number of individual male or female twins with data for each measure.

Pd was assessed at age 16, SSS at 17, and RAPI at ages 18 and 25.

(tests on differences in both means and variances non-significant), but mean RAPI scores at age 18 are higher among males ( $t = 3.67$ ,  $p < .001$ ), and both means ( $t = 10.848$ ,  $p < .001$ ) and variances ( $F = 80.30$ ,  $p < .001$ ) are higher for male RAPI scores at age 25. Note, as well, that by age 25, the RAPI mean and variance decreased among females but not among males. Samples with data at age 25, here shown for individual twins from same-sex pairs, comprise 83% of the baseline age 16 sample among males, and 89% of the age 16 sample among females.

Phenotypic correlations for male and female twin pairs are shown in Table 2. These correlations parallel the consistency of descriptive results across gender; the correlations are strikingly similar for brother–brother and sister–sister twin pairs — none of the corresponding correlations differed significantly when an equality constraint was tested in Mx, and none differ by a magnitude greater than .03. Table 2 shows that, for both men and women, the Pd–SSS correlation is quite modest, and, across gender, that Pd correlates more highly with RAPI than does SSS at both age 18 and at age 25. That deserves emphasis because the Pd–RAPI correlations are lagged an additional year in time.

Table 3 presents the twin correlations for the four variables. For both personality predictors assessed in

**Table 2**  
Phenotypic Correlations

	Pd16	SSS17	RAPI18	RAPI25
Pd16		.17	.36	.29
SSS17	.15		.24	.17
RAPI18	.34	.27		.51
RAPI25	.26	.17	.50	

Note: Variables as defined in Table 1; all correlations significant  $p < .01$ .

Data from males above the diagonal, females below.

**Table 3**  
Twin Correlations and Heritabilities

Twin Correlations ( <i>N</i> Pairs)				
	Pd16	SSS17	RAPI18	RAPI25
MZM	.60 (344)	.65 (330)	.64 (306)	.48 (276)
DZM	.37 (439)	.33 (408)	.47 (397)	.24 (315)
MZF	.60 (500)	.60 (495)	.59 (478)	.48 (428)
DZF	.35 (435)	.35 (422)	.29 (408)	.24 (366)
Heritabilities (95% CIs)				
	Pd16	SSS17	RAPI18	RAPI25
Males	.60 (.54, .65)	.65 (.59, .70)	.47* (.39, .61)	.48 (.39, .55)
Females	.61 (.56, .66)	.61 (.56, .66)	.61 (.55, .66)	.48 (.41, .54)

Note: Variables as defined in Table 1; MZM = monozygotic male twin pairs; DZM = dizygotic male twin pairs; MZF = monozygotic female twin pairs; DZF = dizygotic female twin pairs; 95% confidence intervals for the heritability estimates shown inside parentheses. \*Heritability estimates from AE models except for the estimate for RAPI18 from males, where a significant effect of common environments,  $C = .20 (.09-.26)$ , was found.

adolescence, and for the RAPI at age 25, there is again a striking similarity of monozygotic (MZ)/dizygotic (DZ) twin correlations across gender. For RAPI at age 18, the MZ correlations for males and females are quite similar, but the FDZ correlation for RAPI is much lower than that for MDZ twins. We fit a baseline Cholesky model allowing sex differences on all parameters, and rejected a nested submodel constraining estimates to equality across brother–brother and sister–sister twin pairs,  $\Delta\chi^2(30) = 121.05, p < .001$ . Genetic and unshared environmental effects were significant for all variables, but the only significant shared environmental effect was for RAPI at age 18 in males, with an estimate of .20 (95% confidence intervals [CI] = .09–.26). To avoid problems in estimating genetic and environmental correlations among non-significant C parameters, the final model set the C effects for the other variables to zero,  $\Delta\chi^2(19) = 10.45$ , compared with the baseline model. Genetic estimates from this final model are shown in Table 3, along with 95% CIs. As might be expected based on the twin correlations, the heritability estimates for the personality measures and for the RAPI at age 25 are very similar for males and females. The only substantial difference is for RAPI at age 18 where the substantial familiarity for males is attributed to genetic and shared environmental influences, while all of the familial influences for females are genetic.

Table 4 shows results for the genetic contributions to the associations between variables over time. Gender differences in genetic correlations were tested by the change in  $\chi^2$  when male and female estimates were constrained to be equal. In general, and consistent with the phenotypic correlations, the genetic correlations show similar patterns for males and females, with a larger genetic contribution to the

**Table 4**  
Additive Genetic Correlations

	Pd16	SSS17	RAPI18	RAPI25
Pd16		.22 (.12, .31)	.72 (.59, .82)	.50 (.39, .62)
SSS17	.22 (.13, .30)		.43 (.32, .54)	.36 (.24, .48)
RAPI18	.48 (.40, .55)	.39 (.30, .47)		.95 (.80, .99)
RAPI25	.34 (.24, .44)	.26 (.15, .36)	.74 (.65, .81)	

Note: Variables as defined in Table 1. All correlations are significant at  $p < .05$ .

The 95% confidence intervals for each correlation are shown in parentheses. Magnitude of genetic correlations from males and females differed significantly ( $p < .05$ ) for that of Pd16 with RAPI at both ages 18 and 25, and for the correlation of RAPI18 with RAPI25.

Data from males above the diagonal, females below.

correlations of Pd with RAPI than for SSS with RAPI. But there are two notable differences as well: Pd has a higher genetic correlation with drinking problems among males than among females and a higher environmental correlation with drinking problems among females than among males. The second gender difference is that the stability of genetic influences on drinking-related problems is higher among males.

Parallel results for the unshared environmental contributions to associations between the four measures over time are shown in Table 5. These results dramatize the greater influence unshared environment has among women in mediating predictive associations between adolescent personality and later drinking problems; for women, that influence is significant for all four associations, with the lower bound of each confidence interval exceeding zero, for men, none of the four associations reach significance.

**Table 5**  
Unshared Environmental Correlations

	Pd16	SSS17	RAPI18	RAPI25
Pd16		.08 (-.02, .18)	-.08 (-.17, .02)	.05 (-.05, .15)
SSS17	.08 (.00, .17)		.03 (-.07, .14)	-.05 (-.15, .06)
RAPI18	.17 (.09, .25)	.12 (.04, .20)		.22 (.10, .32)
RAPI25	.17 (.08, .26)	.11 (.02, .19)	.25 (.16, .33)	

Note: Variables as defined in Table 1. For female twins, all four correlations between personality measures and RAPI18 and RAPI 25 are significant at  $p < .05$ ; those estimates and their 95% confidence intervals, inside parentheses, are shown in bold; none of the corresponding correlations are significant for twin brothers. Magnitude of unshared environmental correlations from males and females differed significantly ( $p < .05$ ) for the correlation of Pd16 with RAPI18 and for the correlation of SS17 with RAPI25.

Data from males above the diagonal, females below.

## Discussion

In combination, the estimated heritabilities and genetic correlations obtained from these analyses suggest that influences on developmental changes in alcohol problems from late adolescence to early adulthood differ between men and women. In males, the main change is a decrease in variation from shared environmental effects; the magnitude of genetic effect is stable, and the genetic correlation of .95 suggests that the same genetic influences are important at both ages. For females, there is a decrease in the magnitude of genetic influences from 18 to 25, and at least part of the genetic influences present at age 25 is different from the genetic influences present at age 18. For both men and women, about half of the phenotypic variation in alcohol problems reported at age 25 is due to genetic variance. But contributions of genetic and unshared environmental factors to associations of individual differences in mid-adolescent personality with drinking problems in later adolescence and early adulthood differ between men and women. For women, unshared environment significantly contributes to those associations; among men it does not. Conversely, additive genetic factors make a larger contribution to these personality–problem correlations among men than among women.

We studied twin sibling similarities for drinking-related problems from age 18 to age 25. What changes in sibling similarities are to be expected over this period? An obvious expectation is reduced resemblance, as twin siblings move from their shared childhood parental home to individualized adult lives with nonrelatives; separating from one another and from their parents should attenuate similarity, because family structure, family size, family status, parental modeling of substance use and use by shared peers influence adolescent drinking patterns. When familial and neighborhood characteristics are no longer shared, sibling similarity will decline. Results in Table 3 confirm the obvious expectation: The RAPI correlation from MZ twin brothers is reduced from age 18 to age 25 by a quarter; that for DZ twin brothers is halved. Correlations for twin sisters are attenuated less dramatically. Age-related effects may be more evident among Finnish males, because nearly all experience compulsory military service between age 18 and 25, and that experience may be associated with easy access and high exposure to alcohol and drinking interactions with peers; individual differences in acute and acquired tolerance, with a genetic basis, would serve to attenuate drinking patterns more so among DZ than MZ co-twins.

In an earlier report on these Finnish twins (Mustanski et al., 2003), we related the Pd Scale, as a measure of social deviance, and a smaller subset of SSS items, chosen as a measure of excitement seeking, to drinking and alcohol problems at age 18; our interest then was to test a hypothesis that personality risk factors for drinking differed from those predictive of drinking problems and that both associations were genetic in nature. Here, with follow-up data on RAPI scores reported from the fourth wave of assessment at

ages 22 to 27, we replicate the finding that Pd correlates more highly than does SSS with RAPI outcomes and that the association is mediated in part by genes. We now add the finding that consistency of the alcohol-related outcomes assessed by RAPI is mediated genetically and much more so in males than females.

As always, these findings should be interpreted in the context of strengths and limitations of the research. The generalizability of these findings should be tested to assess whether characteristics specific to these Finnish birth cohorts might moderate the results we obtained. Ours was a population-based sample of Finnish twins, born 1975 to 1979 and coming of age in the early 1990s. That was a turbulent period for Finland as it experienced severe economic challenges following the dissolution of the Soviet Union, which had been its major trading partner. Those challenges accelerated Finland's emergence as a high-tech, electronic leader in the world economy and cemented Finland's membership in the European Union. What were the drinking patterns of Finnish adolescents as our studied twins matured into mid-adolescence? Epidemiological data based on large samples of 15- and 16-year-olds from 26 European countries were obtained in 1995, as the last cohorts of *FinnTwin16* were enrolled into study (ESPAD; Hibell et al., 1997). ESPAD data illustrate that high-density drinking is common among Finnish adolescents; compared to their European peers, the percentage (15%) of Finnish 15-year-olds who reported consuming beer on three or more occasions during the preceding month was but a third of that reported by age-matched Danes, half of that reported in the United Kingdom and lower than was found for 15-year-olds in the US. But 28% of 15-year-old Finns reported they had been drunk 10 times or more during the past year, ranking them second only to Danes and more than three times that reported by American 15-year-olds. In short, Finnish adolescents drink less frequently than adolescents in many other cultures, but they tend to drink in high density. Such binge-drinking patterns may influence age-to-age consistency of drinking-related problems over time, as well as the predictive association of antecedent personality assessments with later drinking problems.

Our suggestive evidence of gender differences in genetic and environmental influences on developmental changes in alcohol problems from late adolescence to early adulthood is provocative. Consistent with other emerging evidence of gender modulation in risk pathways for alcoholism, these results invite further study; data collection now underway with a second *FinnTwin* sample, initially assessed at ages 11 and 12 and now maturing into their early 20s, will permit replication and extension of these findings.

## Acknowledgments

*FinnTwin16-25* has been supported by awards from the National Institute on Alcohol Abuse and Alcoholism (grants AA-08315, AA-00145 and

AA-12502) to RJR, with supplementary support from the Academy of Finland (Grant 44069) awarded to JK.

## References

- Bucholz, K. K., Heath, A. C., & Madden, P. A. F. (2000). Transitions in drinking in adolescent females: Evidence from the Missouri Adolescent Female Twin Study. *Alcoholism: Clinical and Experimental Research*, *24*, 914–923.
- Dahlstrom, W. G., Welch, G. S., & Dahlstrom, L. E. (1972). *An MMPI handbook. Volume I: Clinical interpretation*. Minneapolis, MN: University of Minnesota Press.
- Fergusson, D. M., & Horwood, L. J. (2000). Alcohol abuse and crime: A fixed-effects regression analysis. *Addiction*, *95*, 1525–1537.
- Finn, P. R., Sharkansky, E. J., Brandt, K. M., & Turcotte, N. (2000). The effects of familial risk, personality, and expectancies on alcohol use and abuse. *Journal of Abnormal Psychology*, *109*, 122–133.
- Geisner I. M., Larimer, M. E., & Neighbors, C. (2004). The relationship among alcohol use, related problems, and symptoms of psychological distress: Gender as a moderator in a college sample. *Addictive Behaviors*, *29*, 843–848.
- Grant, B., & Dawson, D. (1997). Age at onset of alcohol use and its association with DSM-IV alcohol abuse and dependence: Results from the National Longitudinal Alcohol Epidemiological Survey. *Journal of Substance Abuse*, *9*, 103–110.
- Han, C., McGue, M. K., & Iacono, W. G. (1999). Lifetime tobacco, alcohol and other substance use in adolescent Minnesota twins: Univariate and multivariate behavior genetic analyses. *Addiction*, *94*, 981–993.
- Heath, A. C., & Martin, N. G. (1988). Teenage alcohol use in the Australian Twin Register: Genetic and social determinants of starting to drink. *Alcoholism: Clinical and Experimental Research*, *12*, 735–741.
- Hibell, B., Andersson, B., Bjarnason, T., Kokkevi, A., Morgan, M., & Narusk, A. (1997). *The 1995 ESPAD report: Alcohol and other drug use among students in 26 European countries*. Stockholm: Modin Tryck AB.
- Hopfer, C. J., Crowley, T. J., & Hewitt, J. K. (2003). Review of twin and adoption studies of adolescent substance use. *Journal of the American Academy of Child and Adolescent Psychiatry*, *42*, 710–719.
- Hopfer, C. J., Timberlake, D., Haberstick, B., Lessem, J. M., Ehringer, M. A., Smolen, A., & Hewitt, J. K. (2005). Genetic influences on quantity of alcohol consumed by adolescents and young adults. *Drug and Alcohol Dependence*, *78*, 187–193.
- Jackson, K. M., Sher, K. J., Gotham, H. J., & Wood, P. K. (2001). Transitioning into and out of large-effect drinking in young adulthood. *Journal of Abnormal Psychology*, *110*, 378–391.
- Kaprio, J., Pulkkinen, L., & Rose, R. J. (2002). Genetic and environmental factors in health-related behavior: Studies on Finnish twins and twin families. *Twin Research*, *5*, 358–365.
- Kaprio, J., Sarna, S., Koskenvuo, M., & Rantasalo, I. (1978). The Finnish Twin Registry: Formation and compilation, questionnaire study, zygosity determination procedures, and research program. In W. E. Nance (Ed.), *Twin research: Part B. Biology and epidemiology* (pp. 179–184). New York: Alan R. Liss.
- King, S. M., Burt, S. A., Malone, S. M., McGue, M., & Iacono, W. G. (2005). Etiological contributions to heavy drinking from late adolescence to young adulthood. *Journal of Abnormal Psychology*, *114*, 587–598.
- King, S. M., Iacono, W. G., & McGue, M. (2004). Childhood externalizing and internalizing psychopathology in the prediction of early substance use. *Addiction*, *99*, 1548–1559.
- Koopmans, J. R., Slutske, W. S., van Baal, G. C., & Boomsma, D. I. (1999). The influence of religion on alcohol use initiation: Evidence for genotype × environment interaction. *Behavior Genetics*, *29*, 445–453.
- Loper, R. G., Kammeier, M. I., & Hoffman, H. (1973). MMPI characteristics of college freshman males who later became alcoholics. *Journal of Abnormal Psychology*, *82*, 159–162.
- Maes, H. H., Woodard, C. E., Murrelle, L., Meyer, J. M., Silberg, J. L., Hewitt, J. K., Rutter, M., Simonoff, E., Pickles, A., Carbonneau, R., Neale, M. C., & Eaves, L. J. (1999). Tobacco, alcohol and drug use in eight- to sixteen-year-old twins: The Virginia Twin Study of Adolescent Behavioral Development. *Journal of Studies on Alcohol*, *60*, 293–305.
- Mustanski, B. S., Viken, R. J., Kaprio, J., & Rose, R. J. (2003). Genetic influences on the association between personality risk factors and alcohol use and abuse. *Journal of Abnormal Psychology*, *111*, 282–289.
- Neale, M. C., Boker, S. M., Xie, G., & Maes, H. H. (1999). *Mx: Statistical modeling* (5th ed.). Richmond, VA: Department of Psychiatry, Virginia Commonwealth University.
- Partanen, J., Bruun, K., & Markkanen, T. (1966). *Inheritance of drinking behavior: A study on intelligence, personality, and use of alcohol of adult twins* (The Finnish Foundation for Alcohol Studies, Vol. 14). Helsinki: Almqvist & Wiksell.
- Pedersen, W., & Skrandal, A. (1996). Alcohol and sexual victimization: A longitudinal study of Norwegian girls. *Addiction*, *91*, 565–582.
- Peltonen, L., Palotie, A., & Lange, K. (2000). Use of population isolates for mapping complex traits. *Nature Review*, *1*, 182–190.
- Rose, R. J. (2006). Introduction. In L. Pulkkinen, J. Kaprio, & R. J. Rose (Eds.), *Socioemotional development and health from adolescence to adulthood* (pp. 1–25). Cambridge, UK: Cambridge University Press.

- Rose, R. J., & Dick, D. M. (2004/2005). Gene–environment interplay in adolescent drinking behavior. *Alcohol Research and Health*, 28, 222–229.
- Rose, R. J., Dick, D. M., Viken, R. J., & Kaprio, J. (2001). Gene–environment interaction in patterns of adolescent drinking: Regional residency moderates longitudinal influences on alcohol use. *Alcoholism: Clinical and Experimental Research*, 25, 637–643.
- Rose, R. J., Dick, D. M., Viken, R. J., Pulkkinen, L., & Kaprio, J. (2001). Drinking or abstaining? A genetic epidemiological study. *Alcoholism: Clinical and Experimental Research*, 25, 1594–1604.
- Rose, R. J., Dick, D. M., Viken, R. J., Pulkkinen, L., & Kaprio, J. (2004). Genetic and environmental effects on conduct disorder and alcohol dependence symptoms and their covariation at age 14. *Alcoholism: Clinical and Experimental Research*, 28, 1541–1548.
- Sartor, C. E., Jacob, T., & Bucholz, K. K. (2003). Drinking course in alcohol-dependent men from adolescence to midlife. *Journal of Studies on Alcohol*, 64, 712–719.
- Schulenberg, J., O'Malley, P. M., Backman, J. G., Wadsworth, K. N., & Johnston, L. D. (1996). Getting drunk and growing up: Trajectories of frequent binge drinking during the transition to young adulthood. *Journal of Studies on Alcohol*, 57, 289–304.
- Sher, K. J., Bartholow, B. D., & Wood, M. D. (2000). Personality and substance use disorders: A prospective study. *Journal of Consulting and Clinical Psychology*, 68, 818–829.
- Thombs, D. L. B., & Beck, K. H. (1994). The social context of four adolescent drinking patterns. *Health Education Research*, 9, 13–22.
- Varilo, T., & Peltonen, L. (2004). Isolates and their potential use in complex gene mapping efforts. Commentary. *Current Opinion in Genetics and Development*, 14, 316–323.
- Viken, R. J., Kaprio, J., Koskenvuo, M., & Rose, R. J. (1999). Longitudinal analyses of the development of drinking and of drinking to intoxication in adolescent twins. *Behavior Genetics*, 29, 455–461.
- Wells, J. E., Horwood, L. J., & Fergusson, D. M. (2004). Drinking patterns in mid-adolescence and psychosocial outcomes in late adolescence and early adulthood. *Addiction*, 99, 1529–1541.
- White H. R., & Labouvie, E. W. (1989). Towards the assessment of adolescent problem drinking. *Journal of Studies on Alcohol*, 50, 30–37.
- Zuckerman, M. (1979). *Sensation seeking: Beyond the optimal level of arousal*. Hillsdale, NJ: Erlbaum.
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