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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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Abstract

We enrolled >3,500 same-sex twins from five consecutive Finnish birth cohorts into longitudinal study as each cohort reached age 16. Twins completed the Psychopathic Deviate (Pd) Scale of the MMPI 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 four variables assessed at four ages across the four 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 and a higher environmental correlation among females. 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, 0.95, 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.

A number of recent twin studies have focused on developmental trajectories of alcohol use from early adolescence into early adulthood (Hopfer, Crowley & Hewitt, 2003; Rose & Dick, 2004/05). 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 & Kaprio, 2001; Viken, Kaprio, Koskenvuo & Rose, 1999), Minnesota (Han, McGue & Iacono, 1999; King, Iacono & McGue, 2004), Missouri (Bucholz, Heath & Madden, 2000), The Netherlands (Koopmans, Slutske, van Baal & Boomsma, 1999), Virginia (Maes, et al, 1999), and in analyses of data from a multi-state USA sample (Hopfer, et al, 2005).

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However much such research has enhanced understanding 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 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, Dick, Viken, Pulkkinen & Kaprio, 2004). But there are other complexities: trajectories of high-density drinking exhibit different developmental pathways from adolescence into early adulthood (Schulenberg, O'Malley, Bachman, Wadsworth & Johnston, 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, Jacob & Bucholz, 2003). It is likely that multiple developmental pathways to alcoholism exist, and likely, as well, that different pathways have different genetic loadings.

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, Horwood & Fergusson, 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, Sher, Gotham & Wood, 2001). And there is some evidence that genetic factors have less influence on high-density drinking in young adult women than men (King, Burt, Malone, McGue & Iacono, 2005), and suggestive evidence that gender moderates the associations of drinking patterns, drinking-related problems, and symptoms of psychological distress (Geisner, Larimer & Neighbors, 2004).

Taken together, this research literature invites 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 mid-adolescence predict risk for alcoholism. Using data from the four assessments made in *FinnTwin16-25*, we report a longitudinal analysis of drinking problems reported at ages 18 - 19 and 23 - 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

As a setting in which to conduct longitudinal twin research, Finland offers some unique advantages to those shared with other Nordic countries (Rose, 2006). Finnish twins can be ascertained readily and followed throughout their lives. The Population Register Centre (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 biological mother; the PRC contains a current residential address for each individual and information on family structure, births, deaths, marriages and divorces. Irretrievable loss of Finns to follow-up is minimized, given access to

residential addresses and the individualized linkage of each Finnish citizen to health, insurance, and institutional outcome measures. Finns have a long history of voluntary participation in epidemiological research, and compliance with research requests among Finnish twins and their families is very high. Finnish public education achieves a very high 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 (*Programme for International Student Assessment*; www.pisa.oecd.org). Delivery of health services, of uniformly high quality, is available to all, regardless of social circumstance. Finland's Population Register Centre, the nation's high quality and relative uniformity of educational training and health care delivery, combined with its history as a geographic and linguistic isolate, make Finland an unusual living laboratory for genetically informative research.

Finland is a bilingual country: 6-7% 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.

Sample

Using the Population Register Centre, we ascertained twins in five consecutive nation-wide birth cohorts (1975 – 79) and enrolled them into longitudinal study as they reached age 16 in 1991 - 1995. Response rates were high and unrelated to twin type, so the realized sample (called *FinnTwin16*) 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, Pulkkinen & Rose, 2002). The first follow-up, at age 17, used a similar procedure, staggering mail out of questionnaires across 60 months time. Subsequently, we telescoped the procedure, sending questionnaires quarterly for wave 3, when twins' were ages 18 – 19 (mean 18½, here designated as age 18) and semi-annually for wave 4, when the twins were ages 22-27 (mean 24.6, hereafter designated as age 25); individual response rates were $\geq 90\%$ through age 18, but declined at the age 25 follow-up, with lower compliance among the young adult male twins.

Measures

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 school work or exam preparation was deleted, because all Finnish twins had completed mandatory education when assessed at age 18. Our Finnish adaptation of RAPI, therefore, contained 22 items, with 4 response alternatives for reporting frequency of each consequence, yielding a scale scored from 22-88. Developed and widely used in the USA, RAPI exhibits good internal consistency (White & Labouvie, 1989) and has been employed effectively in other cultures from New Zealand (Fergusson & Horwood, 2000) to Norway (Pedersen & Skrondal, 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 compute twin correlations and for biometric analyses.

The Minnesota Multiphasic Personality Inventory (MMPI) Psychopathic Deviate (Pd) Scale (Dahlstrom, Welsh & Dahlstrom, 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. Internal consistency and retest reliability of the Pd scale is satisfactory. A peak score on Pd is a common

characteristic of MMP 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, Kammemier, & Hoffman, 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 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 dimension of personality. The 24 items came from all SSS subscales. The item format of the SSS is a forced choice of two alternatives, e.g., 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 so small that no obvious difference was found by their exclusion, and we report results using data from all same-sex twins for whom the necessary data across four occasions were available. Our analyses fit a Cholesky model to the four variables across the four twin types, using raw maximum likelihood estimation in Mx (Neale, Boker, Xie & Maes, 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, but both means and variances for RAPI are higher among males at both age 18 and 25; note, as well, that the RAPI mean and variance decrease by age 25 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 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 adolescence, and for the RAPI at age 25, there is again a striking similarity of MZ/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% CI=.09, .26). To avoid problems in estimating genetic and environmental correlations among nonsignificant 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% confidence intervals. 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 correlations to the associations between variables over time. 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 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 and a higher environmental correlation with drinking problems among females. 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 the confidence intervals exceeding zero; for men, none of the four reach significance.

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 25 are different from the genetic influences present at 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 non-relatives; separating from one another and from their parents should attenuate similarity, because family structure, family size, and 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, albeit 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, Viken, Kaprio & Rose, 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 18½; 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 4th wave of assessment at ages 22-27, 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- 79 and coming of age in the early 1990's. That was a turbulent period for Finland as it experienced severe economic challenges following 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 15-16 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 ≥ 3 occasions during the preceding month was but a third of that reported by age-matched Danes, half of that reported in the U.K. and lower than was found for 15 year-olds in the USA. 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 3X 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 11-12 and now maturing into their early 20's, will permit replication and extension of these findings.

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Table 1

Descriptive Statistics for the four measured variables; Pd was assessed at age 16, SSS at 17, and RAPI at ages 18 and 25.

	Pd16	SSS17	RAPI18	RAPI25
Males				
Mean	16.97	12.31	29.52	29.17
(SD)	(5.19)	(4.01)	(7.73)	(7.98)
N	1619	1525	1480	1337
Females				
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

Table 2

Phenotypic Correlations: Data from Males above the Diagonal, Females below

	Pd16	SSS17	RAPI18	RAPI25
Pd16				
SSS17	.15			
RAPI18	.34	.27		
RAPI25	.26	.17	.50	

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

Table 3**Twin Correlations and Heritabilities**

Twin Correlations (N 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% CI's)				
	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.

Table 4

Additive Genetic Correlations: Data from Males above the Diagonal, Females below

	Pd16	SSS17	RAPI18	RAPI25
Pd16				
SSS17	.22 (.13, .30)			
RAPI18	.48 (.40, .55)	.39 (.30, .47)		
RAPI25	.34 (.24, .44)	.26 (.15, .36)	.74 (.65, .81)	

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

Table 5

Unshared Environmental Correlations: Data from Males above the Diagonal, Females below

	Pd16	SSS17	RAPI18	RAPI25
Pd16				
SSS17	.08 (.00, .17)			
RAPI18	.17 (.09, .25)	.12 (.04, .20)		
RAPI25	.17 (.08, .26)	.11 (.02, .19)	.25 (.16, .33)	

Note: Variables as defined in Table 1. For female twins, all correlations between personality measures and RAPI18 and RAPI 25 are significant at $p < .05$; the 95% Confidence Intervals shown in bold; none of the corresponding correlations are significant for twin brothers.