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GENETIC AND ENVIRONMENTAL INFLUENCES ON ALCOHOL USE: DF ANALYSIS OF NLSY KINSHIP DATA

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Summary. Research designs to study alcohol use and abuse have included twin, adoption and family history/high risk studies. Results have consistently implied a genetic factor in the aetiology of alcohol abuse. However, less research has been conducted in search of environmental factors. This study uses kinship structure in a large national dataset (the National Longitudinal Survey of Youth) to estimate (using DeFries–Fulker analysis) the extent of the shared genetic, non-shared genetic, shared environmental and non-shared environmental influences on alcohol use. The NLSY kinship sample contained 3890 pairs of cousins, half-siblings, full-siblings and twins between the ages of 14 and 21 in the initial year of the survey (1979). Estimates of heritability (h^2) and shared environment (c^2) were small to moderate for the entire dataset for both light drinking and heavy drinking behaviour, with h^2 estimates slightly higher in each case. Non-shared genetic measures of self-esteem and locus of control accounted for a significant portion of the remaining variance in heavy drinking behaviour. Race and gender patterns showed c^2 and h^2 estimates that were also small to moderate for both light and heavy drinking behaviour. Significant non-shared effects were found for the White group for heavy drinking behaviour, and for male pairs for both heavy and light drinking behaviour. Additionally, implications and future directions are discussed.

Introduction

The focus of much recent research on alcohol use and abuse has been on the determination of biological and environmental indicators for alcohol abuse. A number of studies have supported the familial nature of alcoholism (Cotton, 1979; Goodwin *et al*., 1972; Goodwin, 1994; Schuckit, 1985). Specifically, Schuckit (1985) reported, '. . . the risk for developing severe alcohol-related problems appears to increase with the number of alcoholic relatives and closeness to their genetic relationship to the subject (p. 6).' However, the design of many of the studies and the quality of the data have typically led to results that only weakly imply a genetic component. Schuckit (1985)

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points out, 'The complex interactions between genotype and environment make it difficult to determine cause and effect' (p. 33). Designs do exist, however, that can explicitly model and separate genetic and environmental influences.

Three primary designs have been used previously to identify family influences on alcohol use and abuse. Adoption studies use children who were adopted soon after birth. If biological parents and the adopted offspring share some measurable symptom – e.g. alcoholism – while the adoptive parents do not, this implies that the symptom is at least partially hereditary. Adoption studies have been discussed and reported by Goodwin (1976, 1981), Bohman (1978) and Cadoret & Gath (1978). Alternatively, other researchers have employed twin studies, which many feel to be the most effective method of implying a genetic component in polygenic traits. Identical (monozygotic, MZ) twins share an identical genetic makeup, with a coefficient of genetic relatedness of $R = 1{\cdot}0$. Fraternal (dizygotic, DZ) twins share half of their genes on average, with $R = 0{\cdot}50$. If the concordance rates are higher for the MZ twin pairs than for the DZ pairs then heredity is implicated as a potential cause. Discussion and presentation of twin studies of alcohol use can be found in Shuckit (1981), Goodwin (1994), Kaij (1960), Partanen, Bruun & Markannen (1966) and McGue, Pickens & Svikis (1992). Finally, some researchers have used family history (or high risk) studies, ones in which 'there has been a substantial effort to differentiate individuals who are at putative risk for becoming alcoholics by virtue of their family history' (Searles, 1990, p. 89). In the family history studies, the researcher typically examines the concordance of family members who exhibit alcoholism or alcohol abuse. Family history studies of alcohol use can be found in Cotton (1979) and Harford, Parker & Grant (1992).

However, genetic influences exist in the context of environmental influences, as well. Recent publications have documented the importance of environmental influences in accounting for differences among siblings (as opposed to similarities) in behavioural traits (Rowe & Plomin, 1981; Scarr & Grajek, 1982; McCall, 1983; Plomin & Daniels, 1987; Rodgers & Rowe, 1985; Searles, 1988). Apparently, non-shared environmental influences often make children residing in the same family as different from one another as pairs of children selected randomly from the population (Plomin & Daniels, 1987). This perspective recognizes four theoretical sources of influence on individual differences in behavioural outcomes: shared and non-shared genetic influences, and shared and non-shared environmental influences.

The goal of the current research was to take advantage of new data and methods to partition variance in alcohol use and abuse between genetic, shared environmental and non-shared environmental influences. It improved on previous research in a number of ways. First, it was based on a national probability sample. Second, it used a wide array of kinship levels, including twins, siblings, half-siblings and cousins, in approximately representative proportions. Third, it combined genetic and environmental components within the same analytic procedures.

Methods

DF analysis and the adaptation to non-shared influences

DF analysis, originally proposed by DeFries & Fulker (1985), is a regression model that estimates parameters reflecting genetic and environmental influences. The model requires measures of a trait and at least two levels of genetic relatedness to estimate h^2

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and c^2 on the given trait. The DF model will not be extensively defined, because a large methodological literature can be consulted for that purpose (DeFries & Fulker, 1985; Cherny *et al*., 1992; Cherny, DeFries & Fulker, 1992; LaBuda, DeFries & Fulker, 1986; Rodgers, Rowe & Li, 1993). However, a brief discussion of the DF model in general and the Rodgers *et al*. (1993) extension will be presented.

The DF model, as proposed by DeFries and Fulker, is as follows:

$$K_1 = b_0 + b_1 K_2 + b_2 R + b_3 (K_2 * R) + e, \quad (1)$$

where K_1 is the score on a relevant trait or behaviour (e.g. a measure of alcohol abuse) for the first member of the kinship pair, K_2 is the score for the second member of the kinship pair, R is the measure of genetic relatedness ($R = 1{\cdot}0$ for identical twins, $R = 0{\cdot}5$ for fraternal twins and siblings, $R = 0{\cdot}25$ for half-siblings, and $R = 0{\cdot}125$ for cousins), the b values are least squares regression coefficients, and e is the residual. In settings where there is not a proband (i.e. it is arbitrary which member of the pair is K_1 and K_2), the scores are double-entered so that both members of the kinship pair appear in the K_1 and K_2 positions. After double-entry the parameter estimates will be unbiased (up to the assumptions of the model), but the significance tests should be adjusted by re-running the tests with sample sizes adjusted back to the number of unique pairs (a conservative adjustment).

The assumptions of the model include genetic additivity, trivial assortative mating and equal shared environmental influences across the levels of genetic relatedness. Additionally, it is assumed that all of the critical variables are included in the model; specifically, that there are no non-shared environmental influences impacting the trait. If there are important non-shared environmental influences, they will be absorbed in the residuals of equation (1), which can bias the results.

Rodgers *et al*. (1993) extended the DF model by proposing a method of accounting for non-shared environmental and genetic influences. The model used kinship differences as specific measures of non-shared environmental influences. The interaction of the measures of kinship difference and the genetic coefficient was used to assess the influence of non-shared genetic influences. The DF model with the Rodgers *et al*. extension is as follows:

$$K_1 = b_4 + b_5 K_2 + b_6 R + b_7 (K_2 * R) + b_8 \mathrm{ENVDIF} + e \text{ and} \quad (2)$$

$$K_1 = b_9 + b_{10} K_2 + b_{11} R + b_{12} (K_2 * R) + b_{13} \mathrm{ENVDIF} + b_{14} (\mathrm{ENVDIF} * R) + e, \quad (3)$$

where ENVDIF is a difference score for the two kin on measuring a specific non-shared influence, and the variables, parameters, and residual are defined as before.

This method may also be used to study the effects of personality differences. Personality differences are one indicator of the non-shared environment (though they are not direct measures of the non-shared environment itself). A number of personality difference measures may be entered sequentially in search of non-shared personality influences, while simultaneously estimating c^2 and h^2. In this paper, various measures of personality differences will be entered, including locus of control and self-esteem, into equations (2) and (3) above. Significant estimates on either of these personality measures will indicate a trait that underlies differences in alcohol use, after having accounted for genetic and environmental similarity.

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The NLSY data

The NLSY began in 1979 as a household probability sample in which all 14- to 21-year-old youth in selected households were surveyed, with $N = 12{,}686$. In the 1992 survey, 90% of the original participants remained. Each survey consisted of a number of questions repeated from previous years, in addition to a number of unique questions. In 1984 to 1988, the survey included a set of 37 questions relating to alcohol use and resultant symptoms from extensive use. These questions were factor analysed; then the measurement scales developed from two of the factors were used as input measures into equations (2) and (3) above. The questions are listed in Table 1.

There are a number of variables that could potentially account for non-shared environmental differences between kinship pairs in alcohol use, including peer networks, socioeconomic status, parenting and family environment (Hesselbrock & Hesselbrock, 1990). However, the NLSY data files offered few such measures. The NLSY did collect measures of two personality characteristics: Rosenberg's Self Esteem shortened scale in 1980, and a shortened version of Rotter's Locus of Control measure in 1979, two well-studied scales with established reliability and validity. Although these are not direct measures of environmental effects, they may well correlate with non-shared influences on alcohol use, and also exhibit a direct link to alcohol use. Further, both these personality characteristics have face validity for being related to alcohol use. At least one study has reported that an ability to relate well to others – social competence – is important in reducing the risk for future pathology (Rae-Grant, Thomas, Offord & Boyle, 1988). Hesselbrock & Hesselbrock (1990) stated: 'Social competence is thought to provide an effective response to a variety of environmental hazards that predispose the individual to maladaptive outcomes, such as peer pressure to use alcohol and the general availability of alcohol' (p. 81).

The linking algorithm

Because the NLSY sample was a household sample, the dataset included many kinship links, including monozygotic and dizygotic twins, full-siblings, half-siblings, cousins and step/adoptive siblings. However, there was no single variable included in the dataset capable of defining each of the various levels. Rodgers (1996) presented a linking algorithm to estimate the level of genetic relatedness for each of the 3890 sibling pairs. The algorithm was similar to work by Rodgers *et al*. (1993) in which they developed a linking algorithm for the NLSY–Children dataset. That algorithm was validated using height measures from each member of the kinship pairs, and shown to be effective. These kinship links have been used successfully in a number of other behaviour genetic studies treating delinquency (Rodgers, Buster & Rowe, 1999), sexual behaviour (Rodgers, Rowe & Buster, 1999), and intellectual development (Bjornsdottir, 1996). Only a brief description of the linking algorithm will be presented here.

Cousins were identified by self reports, and were assigned an $R = 0{\cdot}125$. This assignment was made if both members of the kinship pair identified the other as such. There was no information to distinguish between first cousins and other cousins.

Although it was easy to identify same-sex twins by virtue of a shared birth date and reports of gender, there was no direct measure in the NLSY dataset to define genetic relatedness of same-sex twins. The MZ twins would normally be assigned an $R = 1{\cdot}0$,

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Table 1. NLSY alcohol questions

NLSY alcohol questions
Ever had a drink?
Had any alcoholic beverages in the last month?
Drinking ever interfered with school work?
Drinking ever interfered with your job?
Frequency of 6/more drinks at 1 time last month.
Number of days drank alcohol in the last month.
Number of days had 1 drink in the last month.
Number of days had 2 drinks in the last month.
Number of days had 3 drinks in the last month.
Number of days had 4 drinks in the last month.
Number of days had 5 drinks in the last month.
Number of days had 6/more drinks in the last month.
Total number of days had a drink last month.
Frequency going to bars last month.
Number of days had a hangover in the last month.
Number of days drank alcohol in the last week.
Number of cans/bottles of beer consumed last week.
Number of glasses of wine consumed last week.
Number of drinks w/liquor consumed last week.
Drinking ever interfere w/school work?
Drinking ever interfere w/job?
Ever felt aggressive/cross while drinking?
Ever got into a heated argument while drinking?
Ever gotten into a fight while drinking?
Ever try cut down/quit drink but failed?
Are you afraid you might be/become an alcoholic?
Do you have difficulty stopping drinking until you are completely intoxicated?
Are you unable to remember things done while drinking?
Do you often take a drink 1st thing in the morning?
Do your hands shake a lot the morning after drinking?
Have you gotten high/tight when drinking alone?
Have you kept drinking after you promised not to?
Have you stayed away from work because of a hangover?
Have you gotten high/tight on the job?
Have you nearly/lost job because of drinking?
Has drinking led to quitting a job?
Has drinking hurt you chances for promotion/raises?

while the DZ twins would be assigned an $R = 0{\cdot}50$. Approximately half of same-sex twins are MZ twins, while the remaining half are DZ twins. Thus, same-sex twin pairs were assigned an $R = 0{\cdot}75$ and opposite-sex twin pairs were assigned an $R = 0{\cdot}50$.

The NLSY did not include an explicit indicator of genetic relatedness for the sibling pairs. Although two members of a pair may identify each other as being brothers, sisters, etc., it is possible that the pairs are actually half-siblings or step/adoptive

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siblings. The linking algorithm used a retrospective time line ranging from birth to 18 years of age (created in the 1988 survey when respondents were from 23 to 30 years old) which consisted of questions indicating whether the respondent lived with their biological mother and father at each age. The critical target year was 1979, the only year in which it was certain the pair lived in the same household. A pair that shared both a biological mother and a biological father in 1979 were assigned an $R = 0{\cdot}50$. A pair that shared one biological parent, father or mother, but not both was assigned an $R = 0{\cdot}25$. A pattern indicating a pair that shared neither a biological father nor mother was assigned an $R = 0{\cdot}0$.

The remaining patterns were ambiguous, in that although both members answered 'no' to at least one of the parents, it was not certain whether the members actually shared this biological parent. Pairs that were either full- or half-siblings were assigned an $R = 0{\cdot}375$. Another variable was used when the father status was uncertain. The variable indicated the distance of the biological fathers' residence from each of the members. If the estimates of both members of the kinship pair were close, the members were classified as sharing the same biological father. The algorithm was also stepped back 2 and 4 years from 1979 to fill in the remaining pairs' R values. The risk of doing so was that siblings might not have been living in the same household, and this risk increased with the number of years the algorithm looked back.

The algorithm resulted in the classification of 2338 kinship pairs out of the original 3890, a 60% classification rate. The identified pairs broke down into 32 twin pairs, 1877 full-sibling pairs, 43 half-sibling pairs, 76 cousin/adoptive sibling pairs and 310 pairs assigned an $R = 0{\cdot}375$ as either full- or half-siblings.

Analyses

The DF analysis models were fitted to two measurement scales defined from a factor analysis of the 37 alcohol variables, using the kinship links defined in the linking algorithm. The kinship correlations and heritability estimates were calculated for the total dataset, by race, and by gender-pair. As described earlier, the DF models were run on the double-entered dataset. The significance tests were calculated with the corrected standard errors and degrees of freedom: i.e. the tests are adjusted for the number of unique pairs rather than the number of double-entered pairs.

Additional analyses were run introducing the self-esteem and locus of control variables into the model suggested by Rodgers *et al.* (1993). The variables were introduced as signed difference scores between the members of each pair evaluating the non-shared environmental effect and entered into the models in equations (2) and (3) as ENVDIF values. The difference scores were also crossed with the R coefficient as an ENVDIF*R interaction term to estimate the non-shared genetic component. These analyses were also run by race and gender-pair.

Results

Factor analysis results

A principal component analysis (PCA) with a varimax rotation was conducted on the 37 drinking variables, which identified distinct factors associated with heavy and light drinking. Table 2 includes a list of the variables within each factor and the

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Table 2. Resulting factors

Factor 1 (in order of loading): heavy drinking factor	Factor 2 (in order of loading): light drinking factor
Frequency of 6/more drinks at 1 time last month	Number of days drank alcohol in the last month
Number of days had 6/more drinks in the last month	Total number of days had a drink last month
Number of cans/bottles of beer consumed last week	Number of days had 1 drink in the last month
Number of days drank alcohol in the last week	Number of days had 2 drinks in the last month
Number of days drank alcohol in the last month	Number of days drank alcohol in the last week
Total number of days had a drink last month	Had any alcoholic beverages in the last month
Number of days had 5 drinks in the last month	Number of day had 3 drinks in the last month
Number of drinks w/liquor consumed last week	Frequency going to bars last month
Frequency going to bars last month	
Number of days had a hangover in the last month	
Number of days had 4 drinks in the last month	
% of variance: 22	% of variance: 7

percentage of variance accounted for by each factor. The eleven variables loading above 0·40 on the heavy drinking factor were scored as binary indicators and summed to define the dependent variable for heavy drinking. The eight variables loading above 0·40 on the light drinking factor were treated identically. (Note that a third factor was also identified by the PCA, interpreted as 'symptoms of alcohol use'. Since the goal of this study was to identify measures of alcohol use, however, this third factor was not used.)

Analysis of the overall dataset for factors 1 and 2

Kinship correlations by genetic category for the complete dataset are presented in Table 3. Table 3 also presents the descriptive statistics for each of the kinship pairs, including sample sizes, means and standard deviations for both Factors 1 and 2: heavy drinking and light drinking respectively.

As previously discussed in this paper, the DF model provides estimates of variance accounted for by shared environment (c^2) and heritability (h^2). It is difficult to define an appropriate statistical test for the point estimates, because the adjustment for double-entry is a conservative adjustment to an unknown degree. Therefore, the standard errors and the statistical tests have been adjusted according to the number of unique pairs, and results for $\alpha = 0{\cdot}10$ and $\alpha = 0{\cdot}05$ will be presented for testing each of the estimates. The former will be referred to as 'approaching significance', while the latter will be referred to with the usual terminology as 'significant'.

When the basic DF model was fitted to the overall dataset, the estimates for c^2 and h^2 were 0·17 and 0·25, and 0·13 and 0·34 respectively for the heavy and light drinking factors. Although not an appreciable difference in the first case, the genetic component appeared slightly more important in the determination of light drinking (Factor 2).

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Table 3. Descriptive statistics

Verbal description	*R*	*N*	Mean	Standard deviation	Kinship corr.
Factor 1 (heavy drinking)					
Cousins	0·125	146	3·61	3·53	0·23
Half-siblings	0·250	82	4·76	3·40	0·23
Ambiguous siblings	0·375	590	4·24	3·20	0·24
Full-siblings	0·500	3646	4·23	3·31	0·30
Twins	0·750	54	4·11	3·31	0·46
Factor 2 (light drinking)					
Cousins	0·125	146	3·43	3·07	0·22
Half-siblings	0·250	82	4·32	2·74	0·21
Ambiguous siblings	0·375	598	4·24	2·69	0·21
Full-siblings	0·500	3660	4·18	2·90	0·30
Twins	0·750	54	4·22	2·93	0·48

Because approximately 70% for heavy drinking and 40% for light drinking of the variance remained (attributable to non-shared influences and measurement error) it appeared the extended DF model would have some potential contribution to identify non-shared variance.

The next step involved fitting the model described in equation (3) and using the self-esteem and locus of control variables to account for specific measures of non-shared influence. When introduced into the model with Factor 1, the non-shared genetic measures for both the self-esteem and locus of control variables approached significance ($p<0{\cdot}10$). Both regression coefficients were positive, suggesting that for high relatedness, high personality differences matched high alcohol use and low personality differences matched low use, while for low relatedness, the opposite patterns were obtained. None of the non-shared measures was significant for Factor 2.

Analysis by race

The basic DF analysis by race resulted in: $c^2=0{\cdot}18$ and $h^2=0{\cdot}24$ (Factor 1) and $c^2=0{\cdot}21$ and $h^2=0{\cdot}21$ (Factor 2) for Whites; $c^2=0{\cdot}19$ and $h^2=0{\cdot}02$ (Factor 1) and $c^2=0{\cdot}05$ and $h^2=0{\cdot}38$ (Factor 2) for Blacks; and $c^2=0{\cdot}00$ and $h^2=0{\cdot}33$ (Factor 1) and $c^2=0{\cdot}01$ and $h^2=0{\cdot}32$ (Factor 2) for Hispanics. Each of the three groups differ from one another in some form. The estimates for Blacks were reversed from one factor to the other, while the estimates for Whites and Hispanics were consistent across the two factors. Additionally, the estimates of c^2 and h^2 for the Whites were approximately the same (and equally balanced) for both factors.

The extended model including non-shared influences (equation (2)) produced no significant findings for either the Hispanics or Blacks. However, the non-shared genetic measure of self-esteem approached significance ($p<0{\cdot}10$) for the White group, as did the non-shared genetic measure of locus of control ($p<0{\cdot}10$). Both of these findings were for Factor 1.

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Table 4. DF analysis results

	Factor 1				Factor 2			
Subgroup	*N*	h^2	c^2	R^2	*N*	h^2	c^2	R^2
Total dataset	4518	0·25	0·17	0·08	4540	0·34	0·13	0·09
Whites	2666	0·24	0·18	0·09	2682	0·21	0·21	0·10
Blacks	1096	0·02	0·19	0·04	1096	0·38	0·05	0·05
Hispanics	756	0·33	0·00	0·03	762	0·32	0·01	0·03
Male pairs	1284	0·10	0·27	0·10	1288	0·28	0·16	0·08
Female pairs	1158	0·45	0·13	0·12	1166	0·49	0·12	0·13
Opposite-sex pairs	2076	0·55	−0·07	0·04	2086	0·57	−0·05	0·05

Note: *N* values are total individuals. These are adjusted back to total pairs to run significance tests.

Analyses by gender-pair

The basic DF model analysis by gender-pair resulted in $c^2 = 0{\cdot}27$ and $h^2 = 0{\cdot}10$ (Factor 1) and $c^2 = 0{\cdot}16$ and $h^2 = 0{\cdot}28$ (Factor 2) for the male pairs, $c^2 = 0{\cdot}13$ and $h^2 = 0{\cdot}45$ (Factor 1) and $c^2 = 0{\cdot}12$ and $h^2 = 0{\cdot}49$ (Factor 2) for the female pairs, and $c^2 = -0{\cdot}07$ and $h^2 = 0{\cdot}55$ (Factor 1) and $c^2 = -0{\cdot}05$ and $h^2 = 0{\cdot}57$ (Factor 2) for the opposite-sex pairs. Similar to the analysis by race, each of the three types of pairs differ from each other in some form. The estimates for the male pairs were reversed from the first factor to the second, while the estimates for the female and opposite-sex pairs remained consistent with higher h^2 in each case. However, while the female pairs also exhibited (small) shared environmental influences, the opposite-sex pairs did not.

The extended model accounting for non-shared influences resulted in no significant effects for either the female pairs or the opposite-sex pairs. However, both the non-shared personality and genetic measures of self-esteem were significant ($p < 0{\cdot}05$) for Factor 1 for the male pairs, and approached significance for the non-shared personality measure of self-esteem for Factor 2 ($p < 0{\cdot}10$). The sign of the *t* statistics for the self-esteem non-shared personality measures was negative indicating that the members of the kinship pairs with higher self-esteem scores had lower levels of alcohol intake. The signs of the regression coefficient for the non-shared genetic influences were both negative, suggesting that for high relatedness, high personality differences match low alcohol use and low personality differences match high use, with the opposite pattern for low relatedness. The estimates for the basic DF analysis of the total group, by race group and by gender-pair, are found in Table 4.

Discussion

As discussed earlier, there is considerable evidence found in past research indicating that alcohol use and abuse have genetic or at least familial ties. The major work described in each of the three types of studies – adoption, twin and family history/high risk – concurred on this point. However, a number of issues have been neglected in the alcohol literature, including direct estimates of the extent of the genetic component,

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extensive review of the contribution of environmental factors, and review of the contribution of non-shared environmental influences. The purpose of this paper was to provide estimates of both genetic and environmental influences and evaluation of the relative importance of at least two specific non-shared measures of environmental influence. The goals have been completed through the use of the DF procedure applied to a national dataset with multiple kinship levels, resulting in clear and interpretable findings. These findings are consistent with earlier ones in the alcohol literature. While there is relatively little research in the area of environmental influences of alcohol use, authors such as Cloninger, Bohman & Sigvardsson (1981) have recognized the importance of both genetics and environment in drinking behaviour, or more specifically, alcoholism. Finally, the non-shared environmental sources proposed by numerous authors to be important in accounting for the variability in personality measures have been shown to be effective in accounting for variance in alcohol use.

Two types of drinking variables were identified in this study: the first (Factor 1) a measure of heavy drinking behaviour, and the second (Factor 2) consisting of a measure of light drinking behaviour. It is to be expected that the genetic, environmental and non-shared influences on these two variables would differ somewhat, and possibly substantially. In fact, this was the case in many of the analyses. The total group exhibited shared environmental (c^2) estimates of 0·13 and 0·17 for the two factors and heritability (h^2) estimates of 0·25 for Factor 1 and 0·34 for Factor 2. These differences are not large, though the importance of the genetic component appeared to increase somewhat for light drinking behaviour.

While the specific measures of non-shared environmental influence were not significant for Factor 2 for the total group, this is not to say that non-shared influences are unimportant for light drinking behaviour. The success of the model depends upon the selection of non-shared environmental variables. There are a number of non-shared environmental variables – for example, parental discipline, parental involvement, peer influence – that could conceivably contribute to this behaviour. Perhaps the most intriguing of the list is the peer influence. Although two youths may be of similar age and reside in the same household, their peer networks may be vastly different. For example, two brothers may be separated in age by a single year, yet the older youth attends a high school while the younger of the two attends a middle school. The extracurricular activities of the two schools would certainly be different, as would the peer associations. It is also possible – and in fact likely – that two youths could attend the same school but associate with very different 'types' of peers participating in very different types of activities. Future research should focus on the identification of these non-shared variables. The non-shared measures of self-esteem and locus of control were found to be important here in accounting for variation in heavy drinking behaviour for the total group.

The analyses by gender-pair produced some interesting results, consistent with the findings of Cotton (1979). In that study, Cotton noted that although there is (1) a high rate of alcoholic fathers of alcoholics, and (2) a higher incidence of alcoholism in fathers and brothers of alcoholics than mothers and sisters, there are higher rates of familial alcoholism for female alcoholics than for men. Correspondingly, results from the current study suggest that there is a stronger genetic component for female pairs than for male pairs. In fact, the total variance accounted for by the genetic component

alone was 0·45 and 0·49 for the female pairs on the two factors. The estimates were less consistent for male pairs. The environmental component was higher than the genetic for male pairs on Factor 1, but reversed for Factor 2, suggesting that men may drink for genetic reasons, but that heavy drinking is more environmental. And while self-esteem is an important measure in accounting for non-shared personality influences for male pairs, neither self-esteem nor locus of control were important for female pairs.

The analyses by race were equally interesting. The Hispanic group produced environmental estimates of zero for both factors, and heritability estimates in the region of 0·32–0·33 for the two factors. The estimates for the Black group were somewhat different from the Hispanics. The environmental estimate was much larger for the heavy drinking factor, but heritability was dominant for the light drinking factor. This implies that heavy drinking behaviour is more the result of an environmental effect for Blacks, but light drinking is more genetically related (which, interestingly, matched the basic pattern for the male pairs). There were no significant non-shared influences found in this study for either the Hispanics or the Black groups.

The White group was more balanced in terms of the environmental and genetic effects. While the Hispanic and Black groups displayed larger estimates on one effect or the other and near-zero on the remaining, the White group produced approximately equivalent estimates for each effect, and for both factors. Additionally, the non-shared genetic effect for both the self-esteem and locus of control variables were significant for the White group in the determination of heavy drinking. This implies that the personality variables self-esteem and locus of control are more important in the determination of heavy drinking for Whites than for the Hispanic or Black groups.

This study has contributed to the work in the alcohol-use field in several ways. First, previous research was supported showing that alcohol use has an important hereditary component, and estimates were provided, by subgroup, of the extent of the genetic component. Second, the importance of the environmental component had been elaborated, particularly among males and Blacks for heavy drinking, and estimates were provided of the extent of the environmental contribution. Third, the importance of non-shared factors in the determination of alcohol use was highlighted, and further research on this topic is certainly supported. Non-shared influences that carefully measure environmental differences were not available in the current data, although the analysis of personality differences was suggestive that such measures would be useful. Using representative data with kinship structure to apportion variance between these theoretical sources is a useful step in understanding the aetiology of alcohol use and abuse.

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References

BJORNSDOTTIR, A. (1996) *Gender Differences in Mathematics: Genetic and Environmental Influences with Special Emphasis on High and Low Ability*. Doctoral Dissertation, University of Oklahoma.

Bohman, M. (1978) Genetic aspects of alcoholism and criminality – through a material of adoptions. *Archs gen. Psychiat.* **35**, 269–276.

Cadoret, R. & Gaith, A. (1978) Inheritance of alcoholism in adoptees. *Br. J. Psychiat.* **132**, 252–258.

Cherny, S. S., Cardon, L. R., Fulker, D. W. & DeFries, J. C. (1992) Differential heritability across levels of cognitive ability. *Behav. Genet.* **22**, 153–162.

Cherny, S. S., DeFries, J. C. & Fulker, D. W. (1992) Multiple regression of twin data: A model-fitting approach. *Behav. Genet.* **22**, 489–497.

Cloninger, R. C., Bohman, M. & Sigvardsson, S. (1981) Inheritance of alcohol abuse: Cross-fostering analysis of adopted men. *Archs gen. Psychiat.* **38**, 861–868.

Cotton, N. (1979) The familial incidence of alcoholism. *J. Stud. Alcoholism* **40**, 89–116.

DeFries, J. C. & Fulker, D. W. (1985) Multiple regression analysis of twin data. *Behav. Genet.* **15**, 467–473.

Goodwin, D. (1976) *Is Alcoholism Hereditary?* Oxford University Press, New York.

Goodwin, D. (1981) Adoption studies of alcoholism. In: *Twin Research 3: Epidemiological and Clinical Studies*, pp. 71–76. Edited by L. Gedda, P. Paris & W. Webb. Alan R. Liss, New York.

Goodwin, D. (1994) *Alcoholism: The Facts* (2nd edn). Oxford University Press, New York.

Goodwin, D., Schulsinger, F., Hermansen, L., Guze, S. B. & Winokur, G. (1972) Alcohol problems in adoptees raised apart from biological parents. *Archs gen. Psychiat.* **28**, 238–243.

Harford, T. C., Parker, D. A. & Grant, B. F. (1992) Family history, alcohol use and dependence symptoms among young adults in the United States. *Alcoholism: Clin. Exp. Res.* **16**, 1042–1046.

Hesselbrock, V. & Hesselbrock, M. (1990) Behavioral/social factors that may enhance or attenuate genetic factors. In: *Genetics and Biology of Alcoholism*. Edited by C. R. Cloninger & H. Begleiter. Cold Spring Harbor Laboratory Press, New York.

Kaij, L. (1960) *Alcoholism in Twins*. Almquist and Wiksell, Stockholm.

LaBuda, M. C., DeFries, J. C. & Fulker, D. W. (1986) Multiple regression analysis of twin data obtained from selected samples. *Genet. Epidemiol.* **3**, 425–433.

McCall, R. B. (1983) Environmental effects on intelligence: The forgotten realm of discontinuous nonshared within family factors. *Child Dev.* **17**, 408–415.

McGue, M., Pickens, R. W. & Svikis, D. S. (1992) Sex and age effects on the inheritance of alcohol problems: A twin study. *J. abnorm. Psychol.* **101**, 3–17.

Partanen, J., Bruun, K. & Markannen, T. (1966) *Inheritance of Drinking Behavior: A Study of Intelligence, Personality, and Use of Alcohol of Adult Twins.* Finnish Foundation for Alcohol Studies, Helsinki, Finland.

Plomin, R. & Daniels, D. (1987) Why are children in the same family so different from one another? *Behav. Brain Sci.* **10**, 1–60.

Rae-Grant, N. J., Thomas, D., Offord, X. & Boyle, M. (1988) Risk, protective factors, and the prevalence of behavioral and emotional disorders in children and adolescents. *J. Am. Acad. Child Adoles. Psychiat.* **28**, 262.

Rodgers, J. L. (1996) *NLSY Youth Linking Algorithm* (unpublished).

Rodgers, J. L., Buster, M. & Rowe, D. C. (1999) Genetic and environmental influences on delinquency: DF analysis of NLSY kinship data (unpublished).

Rodgers, J. L. & Rowe, D. C. (1985) Does contiguity breed similarity? A within-family analysis of nonshared sources of IQ differences between siblings. *Devl Psychol.* **21**, 743–746.

Rodgers, J. L., Rowe, D. C. & Buster, M. (1999) Nature, nurture, and first sexual intercourse in the USA: fitting behavioural genetic models to NLSY kinship data. *J. biosoc. Sci.* **31**, 29–41.

Rodgers, J. L., Rowe, D. C. & Li, C. (1993) Beyond nature vs. nurture: DF analysis of nonshared influences on problem behaviors. *Devl Psychol.* **30**, 374–384.

ROWE, D. C. & PLOMIN, R. (1981) The importance of nonshared (E1) environmental influences in behavioral development. *Devl Psychol.* **17**, 517–531.

SCARR, S. & GRAJEK, S. (1982) Similarities and differences among siblings. In: *Sibling Relationships: Their Nature and Significance Across the Lifespan.* Edited by M. E. Lamb & B. Sutton-Smith. Erlbaum and Associates, Hillsdale.

SCHUCKIT, M. (1981) Twins studies on substance abuse: An overview. In: *Twin Research 3: Epidemiological and Clinical Studies*, pp. 61–70. Edited by L. Gedda, P. Paris & W. Webb. Alan R. Liss, New York.

SCHUCKIT, M. (1985) Studies of populations at high risk for alcoholism. *Psychiat. Dev.* **3**, 31–63.

SEARLES, J. S. (1988) The role of genetics in the pathogenesis of alcoholism. *J. abnorm. Psychol.* **97**, 153–167.

SEARLES, J. S. (1990) Methodological limitations of research on the genetics of alcoholism. In: *Genetics and Biology of Alcoholism*. Edited by C. R. Cloninger & H. Begleiter. Cold Spring Harbor Laboratory Press, New York.