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GRADUATE COLLEGE

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

A Dissertation

SUBMITTED TO THE GRADUATE FACULTY

in partial fulfillment of the requirements for the

degree of

Doctor of Philosophy

By

Maury A. Buster
Norman, Oklahoma
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GENETIC AND ENVIRONMENTAL INFLUENCES ON ALCOHOL USE:
DF ANALYSIS OF NLSY KINSHIP DATA

A Dissertation APPROVED FOR THE
DEPARTMENT OF PSYCHOLOGY

BY

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Abstract

Alcohol use and abuse are topics that have been studied for many years. The research, including twin studies, adoption studies, and family history / high risk studies have focused primarily on the genetic or familial ties as related to these topics. Accordingly, results have consistently implied a genetic factor in the determination of alcohol abuse. However, little research has been conducted in search of environmental factors in the determination of alcohol use and abuse. Additionally, recent publications from other areas have documented the importance of “nonshared” genetic and environmental influences in accounting for the variability in personality measures. This study uses the NLSY dataset and a biosocial modeling approach called DeFries-Fulker (DF) Analysis to estimate the extent of the shared genetic and environmental influences on alcohol use. Additional analyses using an extended version of the DF model are conducted to identify nonshared genetic and environmental effects on alcohol use.

DF analyses were conducted for the entire set of kinship pairs in the NLSY dataset, with additional analyses by race and by gender pair. The 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 behavior. The h^2 estimate was slightly higher in each case. Nonshared genetic measures of self-esteem and locus of control accounted for a significant portion of the remaining variance in heavy drinking behavior.

DF analyses by race produced interesting findings. Each of the groups -- Whites, Blacks, and Hispanics -- differed from each other in some form. In each case, the c^2 and h^2 estimates were small to moderate for both light and heavy drinking behavior.

Significant nonshared effects were found for the White group for heavy drinking behavior. The gender pair analyses were similar to those by race. Each of the gender pairs -- female-female, male-male, and opposite-sex -- differed from each other in some form, and the c^2 and h^2 estimates were again small to moderate for light and heavy drinking. Significant nonshared effects were found for male pairs for both heavy and light drinking behavior.

The results are presented in relation to earlier research findings. Additionally, implications and future directions are discussed.

Introduction

Alcohol use and abuse are topics that have drawn attention and research for a number of years. Although most topics enjoy abbreviated periods of review in the social sciences, alcohol-related studies have endured. Much of the focus has surrounded the determination of biological and environmental indicators for alcohol abuse. Most studies have supported the familial nature of alcoholism (Cotton, 1979; Goodwin, 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 of 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 imply a genetic component. Schuckit (1985) points out, “The complex interactions between genotype and environment make it difficult to determine cause and effect” (p. 33). The degree to which genetics and the environment are responsible for alcohol use and abuse deserves more research.

More recently, there has been a surge of publications documenting the importance of the environmental differences among siblings as opposed to among families in accounting for the variability in personality measures (Rowe and Plomin, 1981; Scarr and Grajek, 1982; McCall, 1983; Plomin and Daniels, 1987; Rodgers and Rowe, 1985, Plomin and Daniels, 1987; Searles, 1988). These differences, more commonly known as nonshared environmental influences, have been shown to make children residing in the same family as different from one another as pairs of children selected randomly from the population (Plomin and Daniels, 1987).

In this study, I will use a biosocial modeling approach originally proposed by DeFries and Fulker (1985), and an extension proposed by Rodgers, Rowe, and Li (1993) to estimate the genetic and environmental influences on alcohol use. The procedure, known as DF analysis, uses kinship pairs with multiple levels of relatedness to estimate genetic and shared environmental influences. Additionally, the Rodgers et. al extension partitions the environmental component into shared and nonshared environmental influences. The model requires, as input, measures of relatedness between pairs of siblings. The data are taken from the National Longitudinal Survey of Youth (NLSY). Using an algorithm developed and described by Rodgers (1996), various levels of genetic relatedness are identified within the 3,890 sibling pairs of the NLSY dataset.

I will begin with a review of the literature on alcohol use. Secondly, I will discuss the DF procedure. Following, I will describe the dataset and the linking algorithm. Finally, I will present the Method, Results, and Discussion sections.

Review of the Literature on Alcohol Use

Cotton (1979) noted,

“Alcoholism, whether treated as a symptom or a disease, has long been considered by both clinicians and researchers to be a familial disorder. The literature on alcoholism abounds with statistics on its incidence in families, included as general background in descriptive studies of samples of alcoholics and as the focus of investigations of the familial incidence of alcoholism” (p. 89).

As Cotton noted, the literature abounds with familial evidence -- regardless of the design of the study. Researchers have put to use a number of designs in the study of familial markers, but three predominate in the alcohol literature, including adoption, twin, and high risk studies. Schuckit (1985) states, “The 1970s and early 1980s have seen an explosion

of family, twin, and adoption studies in alcoholism, with almost every investigation concluding that genetic factors are important” (p.32). I will briefly discuss the three dominant designs and include a review of the major studies applying them.

Adoption Studies

Goodwin (1976) writes,

“Not all children are raised by the people who provide their genes, their ‘real’ parents. Some are adopted by foster parents. When the adoption occurs in early infancy; when the foster parents are unrelated to the genetic parents; when the adopted child has no contact with the genetic parents or other blood relatives - then the conditions are suitable for a nature-nurture study” (p. 59).

The essence of the adoption study is to identify those children who were adopted soon after birth, and raised thereafter by adoptive parents. If both the biological parents and their adopted offspring systematically develop some measurable symptom, while the adoptive parents do not, this would imply that the symptom is hereditary. Thus, if the biological children of alcoholic parents tend to become alcoholics, this would imply a genetic component in the determination of alcoholism - because the biological members share only their genetic disposition. Of course this only holds if the adoptive parents are not also alcoholic. While the implications of the adoptive design appear obvious, problems indeed exist.

Goodwin (1976) pointed out several drawbacks in the use of the adoption design. First, we must assume that the parents identified as biological are in fact biological. The mother is easily identified as the biological parent, but the father, especially in adopted cases, may be difficult to specify. Second, although the child is typically adopted at an early age, the child has shared a common environment with at least the mother for nine

months before birth. Additionally, the mother may have nursed the child prior to the adoption. Finally, adoption studies are difficult because of the cooperation, or lack thereof, provided by various authorities. It is extremely difficult to track biological parents, often compounded by the passage of 20 years or more. An additional problem not addressed by Goodwin is the assumption that the environments of the biological and the adoptive parents are uncorrelated. Because placement agents often match adoptive children to parents with similar backgrounds, this assumption may not be viable. Regardless of the problems associated with the design, adoption studies are abundant in the alcohol literature.

In a longitudinal study of Danish adoptive children, Goodwin (1981) reported that adopted sons of alcoholic biological parents (typically fathers) displayed a high incidence of alcoholism. Specifically, ten of the 55 probands were alcoholics, while only four of the 78 controls were alcoholics. Of those who were alcoholics, there were no differences between the sons of alcoholics and the control group alcoholics in terms of resulting problems, or the tendency for heavy drinking. Additionally, the non-adopted brothers who lived with the biological parents were also highly likely to become alcoholics, but no more so than were the adoptive children. Sample size precluded any conclusions for females. Goodwin did however suggest that heredity is at least a partial determinant for alcoholism for females.

Bohman (1978) studied the Swedish registers of 2,000 adopted children born between 1930 and 1949. The study, known as the Swedish Adoption Study, reported similar results to those found by Goodwin (1981). Specifically, as the incidence of

registration for alcoholism increased among parents, the incidence also increased for the registration of the children of those alcoholic parents.

One of the strengths of the Swedish study is the accuracy of the records of biological parents maintained by the Swedish authorities. As pointed out earlier, the paucity of records for biological parents (specifically fathers) is one of the weaknesses of the adoption design in the United States. Whereas the U.S. records are often incomplete, the Swedish records of alcohol use were kept by a Temperance Board, and records of treatment were kept by the National Health Insurance.

Cadore and Gath (1978) studied a sample of 84 adults raised by adoptive parents. Their results again reflected those found in both the Danish and Swedish studies. Of those adoptees with alcoholic relatives, they found a higher incidence of alcoholism as compared to the control group. Additionally, they found a higher incidence of childhood conduct disorder among children who were later diagnosed as alcoholic.

Twin Studies

Twin studies, at least on the surface, appear to be the most effective method of implying a genetic component in polygenic traits. Twins occur in two forms, identical (monozygotic) and fraternal (dizygotic). Identical twins share the same genetic makeup, and their coefficient of genetic relatedness is therefore 1.0. However, fraternal twins share only half of their genes on the average, with a genetic relatedness coefficient of .50. Therefore, Schuckit (1981) states, “. . . the concordance within identical or monozygotic (MZ) twin pairs for genetically influenced factors should be higher than the level of resemblance or similarity for fraternal or dizygotic (DZ) twins sharing only 50% of their

genes” (p.63). If the concordance rates are higher for the MZ twin pairs than for the DZ pairs then heredity may be a factor.

While the twin study design appears strong, there are a number of problems associated with it. Schuckit (1981) describes a number of biases in the twin approach. First, twins are not representative of the population. Specifically, he writes, “. . . factors observed in twins cannot necessarily generalize to the population at large, as twins might differ on their increased rates of infant mortality, lower birth rate, slightly lower IQ, and their tendency to be the product of women of higher mean age” (p.64). Second, there are inherent sampling problems in twin studies which may render a group of twins more similar to one another on a given trait than would be expected in the general population of twins. Third, there are difficulties in establishing the zygosity of the twin pairs. At least one study has shown visual classification error rates at 60% (when, of course, no other form of classification --e.g., questionnaire, Karoyotyping is used). Goodwin (1976) notes a fourth bias in the twin design, the assumption that MZ and DZ twins share the same environments. In reality, parents and significant individuals may treat the DZ twins very differently, whereas the MZ twins are not only treated the same, but also dressed the same.

According to Goodwin (1994), there have been 10 studies where MZ twins were compared with DZ twins in relation to alcoholism. The general findings have been consistent, concluding that MZ twins are more similar in terms of drinking habits than are DZ twins. There are at least two large-scale studies that have been conducted using the twin approach. The first is the Swedish study conducted by Kaij in 1960, and the second

is the Finnish study by Partanen, Bruun, and Markannen (1966). Although the results were not identical between the two studies, the general findings seem to coincide.

The Swedish study was conducted with 174 male twin pairs as subjects. The pairs were chosen if at least one partner was registered at a temperance board in response to some indication of alcohol abuse. The results appear to indicate at least a familial influence upon the decision to drink, and both the frequency and the quantity of alcohol intake. Kaij (1960) also reports MZ pair concordance for alcoholism at 51%, compared to 28% for the DZ pairs.

The Finnish study included 902 male twins, between the ages of 28 and 37. Whereas the Swedish study found higher concordance for alcoholism in the MZ twins than DZ twins, the Finnish study did not find differences between the MZ and DZ groups with regard to the consequences of drinking. As Goodwin (1976) points out, the consequences of drinking are “. . . perhaps the most widely accepted criterion today for diagnosing alcoholism” (p.81). Partanen et. al. did however conclude that the frequency and the amount of drinking were more concordant for the MZ pairs than for the DZ pairs. This finding mirrors that reported in the Swedish study.

Family History / High Risk Studies

Searles (1990) wrote,

“Recently, there has been a substantial effort to differentiate individuals who are at putative risk for becoming alcoholics by virtue of their family history. This quasi-genetic approach seeks to identify factors (e.g., personality traits, physiological, neuropsychological, and biochemical processes) that distinguish those individuals who will and will not eventually exhibit behavioral or social problems with alcohol” (p. 89).

These studies are more commonly known as high risk studies. They seem to be the logical extension of the family history studies. In the family history studies, the researcher typically examines the concordance of family members who exhibit alcoholism or alcohol abuse. In the high risk studies, the researcher typically classifies subjects into various categories which indicate the number and closeness of alcoholic relatives. Once this process is complete, the researcher can compare the groups in search of presymptomatic trait markers of alcohol abuse (Searles, 1988). The high risk studies are fairly recent in the literature, but the family history studies are quite common in older research.

Cotton (1979) provides an extensive review of the family history literature. She notes several trends across the 39 studies that were examined, including; consistently higher findings of sibling alcoholism in samples of alcoholics than in samples of nonalcoholics, higher incidence of alcoholism in near relatives of alcoholics than in distant relatives, a high rate of alcoholic fathers (25-50%) of alcoholics, and higher incidence of alcoholism in fathers and brothers of alcoholics than mothers and sisters. In light of the latter two findings, it is surprising to find that higher rates of familial alcoholism were found for female alcoholics than for men. All but two of the studies that Cotton examined reported higher rates of alcoholism in the families of female alcoholics than in the families of male alcoholics.

Harford et. al. (1992) used the high risk design to study the 12,686 respondents in the NLSY. The respondents were classified into four mutually exclusive groups, according to the existence and closeness of alcoholic relatives. They found that a positive family history (having at least one alcoholic relative), and greater alcohol consumption

place both males and females at greater risk for symptoms of dependence. Harford et. al. appropriately pointed out, "In our analysis, we cannot disentangle genetic from environmental influences, but we can determine whether support can be found for the hypothesis that having both positive family history and greater alcohol use places adult men and women at elevated risk for symptoms of dependence" (p.1043).

Schuckit (1985) provides an in-depth discussion of the high risk design, as well as a review of the high risk literature. He reports that there has been little evidence emanating from the high risk studies to indicate that high risk and low risk groups differ on measurable personality traits and breakdown of ethanol. However, evidence suggests that the high risk and low risk groups differ significantly in terms of their reaction to ethanol. The high risk groups have exhibited less intense reactions to ethanol, and have also exhibited unique EEG patterns.

The problems with the high risk design are abundant. Schuckit (1985) provides an extensive critique of the design. He defines four problem areas, including; inherent shortcomings, uncontrolled factors, generalizability, and financial drawbacks. The primary difficulty appears to be the generalizability of the studies. The high risk studies have not always reported similar results. In defense of the high risk design, all of the studies have not been similar in terms of the age, sex, or socioeconomic status of the subjects. Therefore, the findings of the high risk studies have, to date, not provided information we can generalize to the population in general. Additionally, the design of the studies has not allowed the partitioning of the genetic and environmental components. As was pointed out earlier, researchers such as Harford (1992) have acceded this point.

The goal of the current research is to take advantage of new data and methods to partition variance in alcohol use and abuse between several conceptual sources of family influence. Genetic, shared environmental, and nonshared environmental influences will be investigated. This investigation will use the same data as Hartford et. al. (1992), along with kinship structure not available to Hartford et. al., and a new analytic approach based on DF analysis

DF Analysis and the Adaptation to Nonshared Influences

DF Analysis was originally proposed by DeFries and Fulker (1985). It is a regression model that requires, as input, measures of genetic relatedness between pairs of siblings. The model requires measures of a trait and at least two levels of relatedness to estimate genetic and shared environmental influences (h^2 and c^2) on the given trait. I will not provide an exhaustive discussion of the DF model, but rather explain it in the context of a review of the published papers where it has been applied (DeFries and Fulker, 1985; Cherny, Cardon, Fulker, & DeFries, 1992; Cherny, DeFries, & Fulker, 1992; LaBuda, DeFries, & Fulker, 1986; Rodgers, Rowe, and Li, 1993). However, I will provide a brief discussion of the DF model in general and the Rodgers et. al (1993) extension.

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

$$K_1 = b_0 + b_1K_2 + b_2R + b_3(K_2 * R) + e \quad (1)$$

where K_1 is the score 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 ($R=1.0$ for identical twins, $R=.5$ for fraternal twins and siblings, $R=.25$ for half siblings, and $R=.125$ for cousins), the b 's 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. In this process 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 rerunning the tests with sample sizes adjusted back to the number of unique pairs (a conservative adjustment).

The assumptions of the model include additivity of the genetic and environmental influences, no 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, there are no nonshared environmental influences impacting the trait. If there are important nonshared environmental influences, they will be absorbed in the residuals of equation (1), which can bias the results. Although the first three assumptions often seem plausible, this last assumption appears likely to be violated.

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

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

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

where ENVDIF is a difference score for the two kin on a specific measured environmental source, and the variables, parameters, and residual are defined as before.

This method may also be used to study the effect of personality differences. Personality differences are one indicator of the nonshared environment (though they are not direct measures of the nonshared environment itself). A number of personality difference measures may be entered sequentially in search of nonshared personality influences, while simultaneously estimating c^2 and h^2 . In this paper, I will enter various measures of personality differences, 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.

The NLSY Data and the Kinship Linking Algorithm

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 $N=12,686$ (Rodgers, 1996). In the 1992 survey, 90% of the original participants remained. Each survey consists of a number of questions repeated from previous years, in addition to a number of unique questions. In 1984 through 1988, the survey included a set of 37 questions relating to alcohol use and resultant symptoms from extensive use. These questions will be factor analyzed; then the measurement scales developed from two of the factors will be used as input measures into equations (2) and (3) above. The content of these questions are listed in Table 1.

Insert Table 1 Here

There are a number of variables that could potentially account for nonshared 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 offer 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 nonshared influences on alcohol use, and as well exhibit a direct link 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, and Boyle, 1988). Hesselbrock et. al (1990) state, "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

The NLSY files contain a number of kinship links, including monozygotic and dizygotic twins, full siblings, half siblings, cousins, and step/adoptive siblings. However, there is no single variable included in the dataset capable of defining each of the various

levels. Rodgers (1996) describes a linking algorithm (written by Buster & Rodgers in 1995-96) to estimate the level of genetic relatedness for each of the 3,890 sibling pairs. The algorithm was similar to work by Rodgers, Rowe, and Li (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 very effective. The remainder of this section deals with the logic behind the linking algorithm for the NLSY-Youth.

Two variables were constructed in the linking process, including R , defined as the genetic coefficient, and $CONF$, describing the level of confidence in the assigned R value. If a pair was unambiguously assigned to a particular R value, the $CONF$ value was set to 1.0. If there were two possible kinship values for a given pair, but one or the other was logically certain, an average of the two R values was assigned, and $CONF$ set to .50. Several other values of $CONF$ were assigned and discussed in Rodgers (1996).

The cousins were unambiguously identified, and assigned an $R=.125$ and $CONF=1.0$. This assignment was made if both members of the kinship pair identified the other as such. There was no effort made to distinguish between first cousins and all others, but the effects should be minimal given the relatively small genetic coefficient assigned.

Although it was easy to identify same-sex twins by virtue of a shared birth date and reports of gender, there is no direct measure in the NLSY dataset to define genetic relatedness of same-sex twins. Approximately half of the same-sex twins will be MZ twins, while the remaining half will be DZ twins. The MZ twins would normally be

assigned an $R=1.0$, while the DZ twins would be assigned an $R=.50$. However, because there is no direct evidence of zygosity in the NLSY files, all same-sex twin pairs were assigned an $R=.75$ and $CONF=.50$. The opposite-sex twin pairs were assigned an $R=.50$ and $CONF=1.0$.

The remaining sibling pairs were critical because they comprised such a large percentage of the sample. The NLSY does not include a valid measure 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 even step/adoptive siblings, but consider each other to be brothers or sisters. Therefore, 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). The time line consisted of a number of questions regarding 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. The respondent's ages were linked to 1979 and an ordered quadruple developed indicating whether each sibling lived with their biological mother and father. A pattern of (1, 1, 1, 1) indicated a pair that share both a biological mother and a biological father, and thus were assigned an $R=.50$ and $CONF=1.0$. Any pattern with three 1s and one 0 indicates a pair that shared one biological parent, father or mother, but not both. Therefore, the pairs must be half siblings, and were therefore assigned an $R=.25$ and $CONF=1.0$. Any pattern with two consecutive 0s or a pattern of (0, 1, 1, 0) indicates a pair that shares neither a biological

father or mother, and were therefore step/adoptive siblings with an assigned $R=0.0$ and $CONF=1.0$.

The remaining patterns (0, 1, 0, 1), (1, 0, 1, 0), and (0, 0, 0, 0) were ambiguous, in that although both members answered “no” to at least one of the parents, it is not certain whether the members actually shared this biological parent. Another variable was used in the second case (1, 0, 1, 0), where the fathers were 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 considered to have shared the same biological father. The algorithm was also stepped back two and four years before 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. Thus, four years was as far back as it went.

The final group of unassigned pairs contained a number of individuals who were greater than 18 years of age in 1979. Efforts to resolve the genetic coefficients for these pairs were unsuccessful.

The algorithm resulted in the classification of 2,338 kinship links/pairs out of the original 3,890. The final file consisted of four variables - ID of kin1, ID of kin2, R , and $CONF$. The identified pairs broke down into 32 twin pairs of unknown zygosity, 1,877 full sibling pairs, 43 half sibling pairs, 76 cousin/adoptive sibling pairs, and 310 pairs assigned an $R=.375$ as either full or half siblings (Rodgers, Buster, and Rowe, 1996).

The validation of the linking algorithm was conducted using height as a validation variable. “A large body of literature suggests that height has heritability of approximately

$h^2=.90$, and a trivial level of shared environmental variances around $c^2=0$ " (Rodgers, Buster, & Rowe, 1996, p.15). The height correlations were examined for each of the kinship categories defined in the linking algorithm. The correlation for the $R=0.0$ group was conspicuously high, undoubtedly due to the misclassification of some pairs in this group. The correlation approximated that of the cousins, therefore the $R=0.0$ group was reclassified as $R=.125$. The remaining height correlations followed the expected pattern of increasing values. The DF model was run using the height measures, resulting in h^2 and c^2 estimates within reason -- $h^2=.71$ and $c^2=.12$. However, the analyses by race were less consistent. Estimates for Whites -- $h^2=.89$ and $c^2=.05$ -- were the closest to the widely accepted values discussed earlier. Estimates for Blacks and Other Races were $h^2=.69$ and $c^2=.04$, and $h^2=.46$ and $c^2=.19$ respectively. The $R=.375$ group was recoded as $R=.50$ and the analyses rerun, resulting in improved h^2 estimates for both the total group and the Black group -- $h^2=.88$ and $h^2=.83$ respectively.

Although the estimates support the quality of the linking algorithm, finer links appeared possible. Using measures of height, a number of the $R=.75$, $R=.375$, and nonassigned pairs were resolved. The twin category, classified as $R=.75$ contained both MZ twins and DZ pairs -- $R=1.0$ and $R=.50$. The 50 percent of twin pairs closest in height were reclassified as $R=1.0$, while the other 50 percent were classified as $R=.50$. The $R=.375$ category was redefined by comparing the absolute height difference of pairs to the expected value for full siblings. Those pairs whose absolute difference value was at least as close to that of the expected value for full siblings were reclassified as $R=.50$, and

the remaining pairs were reclassified as $R=.25$. Finally, this same procedure was applied to those pairs previously unclassified.

While the first algorithm resulted in the classification of 2,338 pairs, the additional analyses resulted in the classification of 3,419 pairs -- 88 percent of the original 3,890 pairs. This second file included the 3,419 pairs with the same four variables defined in the first file. The new classifications are 20 MZ pairs ($R=1.0$), 3,090 full siblings ($R=.50$), 233 half siblings ($R=.25$), and 76 cousin/adoptive pairs ($R=.125$).

The validity analyses were run as before, producing total group estimates of $h^2=.86$ and $c^2=.15$. The analyses by race produced heritability estimates of $h^2=1.05$ for Whites, $h^2=.68$ for Blacks, and $h^2=.67$ for Other Races, and c^2 estimates approximately equal to zero for each.

Both datasets will be used in further analyses. Although there certainly remain misclassifications in both datasets, this will show up in the error terms of each of the models.

Method

A common criticism of the twin, adoption, and high risk designs is the lack of generalizability to the population. Each of the three designs consist of a limited subject pool. This study is unique because it 1) is based on a probability sample from the entire U.S. population, and 2) estimates directly the level of the genetic and environmental components in the determination of alcohol use.

Hypotheses

Earlier studies have consistently resulted in an implied genetic component in the determination of alcohol use. Additionally, there have been consistent differences between males and females, as well as among the various races. I expect the general findings of this study will be similar in this respect. However, I will also provide estimates of the extent of the genetic component. Additionally, I expect to find significant nonshared environmental influences similar to those found by Rae-Grant et. al. (1988). Specifically, I expect to find that the self-esteem and locus of control variables will be significant nonshared environmental indicators of alcohol use

Analyses

The DF analysis models are fit to two measurement scales defined in the factor analysis of the 37 alcohol variables, and including the kinship links defined in the linking algorithm. The kinship correlations and heritability estimates are calculated for the total dataset, by race, and by gender-pair, for both datasets. As described earlier, the DF models are run on the double-entered dataset. The significance tests are 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 are run introducing the self-esteem and locus of control variables into the model suggested by Rodgers, Rowe, and Li (1993). The variables are introduced as signed difference scores between the members of each pair, and then crossed with the R estimate to evaluate both the nonshared environmental and nonshared genetic components. These analyses are also run by race and gender-pair, and on both datasets.

For the final set of analyses, pairs from both the upper and lower 25 percent of the distributions for each dependent variable are examined separately. The pairs are trimmed from the total group according to the value of the dependent variable for kin1. The subgroups are then subjected to the same analyses described above for the entire group.

Results

Factor Analysis Results

A principal components analysis with a varimax rotation was conducted on the 37 drinking variables, resulting in three distinct factors. These were interpreted as, 1) a measure of heavy drinking behavior, 2) a measure of light drinking behavior, and, 3) a measure indicating resulting symptoms from alcohol use. Table 2 includes a list of the variables within each factor and the percent of variance accounted for by each factor. The eleven variables loading above .40 on factor one were set to dichotomous items and an additive score across the factor used as the dependent variable. Therefore, any score greater than or equal to one for each question was reassigned a “1”, while all other responses were set to “0”. The total possible score on the final dependent variable (Factor 1) is an “11”. It should be noted that since the questions are independent of each other, a respondent who had 5 drinks on a given day in the previous month did not necessarily also have four drinks, three drinks, etc. The eight variables loading above .40 on factor two were treated identically, while factor three was not examined, because the indicators of heavy and light drinking were more interpretable from a measurement standpoint. In the original analysis, factors two and three were in reverse order. However, because the focus is on heavy and light drinking, they have been reversed.

Insert Table 2 Here

At first glance it appears that heavy and light drinking behaviors may be independent of each other. It seems plausible that someone consistently drinks in small amounts, or, changes their drinking patterns from week days to week ends, while others consistently drink in large amounts. In either case, this study attempts to identify differential effects between the two behaviors.

Analysis of the Overall Dataset #1 for Factors 1&2

Kinship correlations by genetic category for the complete Dataset #1 (the smaller dataset based on 2,338 kinship pairs) are presented in Table 3. Analyses on Dataset #2 were inconsistent and are therefore not presented here. 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.

Insert Table 3 Here

As previously discussed in this paper, the DF model provides estimates of both shared environment (c^2) and heritability (h^2). As explained by Rodgers, Rowe, and Buster (1996), it is difficult to define an appropriate statistical test for the point estimates. Therefore, the standard errors and the statistical tests have been adjusted according to the

number of unique pairs (a conservative adjustment), and results for $\alpha=.10$ and $\alpha=.05$ will be presented for testing each of the estimates.

When the basic DF model was fit to the overall Dataset #1, the estimates for c^2 and h^2 were .17 and .25, and .13 and .34 respectively for the two factors. Although not an appreciable difference in the first case, the genetic component appears slightly more important in the determination of light drinking (Factor 2). Because approximately 50 to 60 percent of the variance remained (attributable to nonshared influences and measurement error) it appeared the extended DF model would have some potential contribution.

The next step involved the models described in Equations 2 and 3 and the use of the self-esteem and locus of control variables to account for specific measures of nonshared influence. When introduced into the model with Factor 1, the nonshared genetic measures for both the self-esteem and locus of control variables were significant ($p<.10$). None of the nonshared measures were significant for Factor 2.

Analysis by Race

The basic DF analysis by race resulted in c^2 and h^2 estimates of .18 and .24 (Factor 1) and .21 and .21 (Factor 2) for Whites, .19 and .02 (Factor 1) and .05 and .38 (Factor 2) for Blacks, and .002 and .33 (Factor 1) and .01 and .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. 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 nonshared influences produced no significant findings for either the Hispanics or Blacks. However, the nonshared genetic measure of self-esteem was significant ($p < .10$) for the White group, as well as the nonshared genetic measure of locus of control ($p < .10$). Both of the significant findings were for Factor 1.

Analyses by Gender-pair

The basic DF model analysis by gender-pair resulted in c^2 and h^2 estimates of .27 and .10 (Factor 1) and .16 and .28 (Factor 2) for the male pairs, .13 and .45 (Factor 1) and .12 and .49 (Factor 2) for the female pairs, and -.07 and .55 (Factor 1) and -.05 and .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 are reversed from the first factor to the second, while the estimates for the female and opposite-sex pairs remain consistent with higher h^2 in each case. However, while the female pairs also exhibit (small) shared environmental influences, the opposite-sex pairs do not.

The extended model accounting for nonshared influences resulted in no significant effects for either the female pairs or the opposite-sex pairs. However, both the nonshared personality and genetic measures of self-esteem were significant ($p < .05$) for Factor 1 for the male pairs, as well as the nonshared personality measure of self-esteem for Factor 2 ($p < .10$). The sign of the t statistics for the self-esteem nonshared personality measures were negative indicating that the members of the kinship pairs with higher self-esteem scores had lower levels of alcohol intake.

The estimates for the basic DF analysis of the total group, by race group, and by gender-pair are found in Table 4.

Insert Table 4 Here

Analysis of the Extreme Groups

The final set of analyses involved the extreme scores -- both high and low -- for Factors 1 and 2. "Extreme" scores are defined as those scores at or below the first quartile and at or above the third quartile of the distribution. For this analysis, scores were not double-entered, and extreme individuals were considered probands.

The bottom 31 percent of scores for both Factor 1 and Factor 2 were zero and therefore could not be analyzed. However, the upper 29 percent of the distribution for Factor 1 ranged from a score of seven to 11, while the upper 21 percent of the distribution for Factor 2 ranged from a score of six to eight (See Table 5).

Insert Table 5 Here

DF analyses on Factor 1 produced estimates for shared environment and heritability of $c^2=.11$ and $h^2=.14$ respectively. Further review of the correlations by genetic relatedness revealed high correlations for both the cousin and half-sibling groups -- .30 and .41 respectively -- while the correlations for the full-sibling and twin groups were .12 and .06. DF analysis on Factor 2 produced essentially zero estimates -- $c^2=.02$ and

$h^2=.03$. These findings were not very interpretable. While other studies have found useful results with this type of analysis of extreme groups, the skewness of the alcohol measures prevented a useful analysis of these variables.

Summary

Generally, the findings described herein were clear and interpretable (with the exception of the extreme group analysis). Although the findings were not consistent from Factor 1 to Factor 2 for each group, this is interpretable and to be expected. The nonshared influences -- self-esteem and locus of control -- were shown in some cases to be important factors in the determination of both Factor 1 and Factor 2. These patterns will be summarized and discussed in the next section.

Discussion

As discussed earlier, there is considerable evidence found in past research indicating that alcohol use and abuse have genetic or at the very least familial ties. The major works described in each of the three types of studies -- adoption, twin, and family history/high risk -- have 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, extensive review of the contribution of environmental factors, and review of the contribution of nonshared 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 nonshared measures of environmental influence. The goals have been completed through the use of the DF procedure, resulting in clear and interpretable findings. These findings are consistent with earlier ones in the

alcohol literature. While there is little research in the area of environmental influences of alcohol use, authors such as Cloninger, Bohman, and Sigvardsson (1981) have recognized the importance of both genetics and environment in drinking behavior, or more specifically, alcoholism. Finally, the importance of nonshared environmental sources proposed by numerous authors (Rowe and Plomin, 1981; Scarr and Grajek, 1982; McCall, 1983; Plomin and Daniels, 1987; Rodgers and Rowe, 1985; Plomin and Daniels, 1987; Searles, 1988), in accounting for the variability in personality measures has been shown effective in accounting for variance in alcohol use. While this study does not provide directly relevant assistance for parents in child rearing, the findings herein should be used to redirect future research in the alcohol use field.

I have described two types of drinking variables in this study, the first (Factor 1) a measure of heavy drinking behavior, and the second (Factor 2) consisting of a measure of light drinking behavior. It is to be expected that the genetic, environmental, and nonshared 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 .13 to .17 for the two factors and heritability (h^2) estimates of .25 for Factor 1 to .34 for Factor 2. These differences are not large, though the importance of the genetic component appeared to increase somewhat for light drinking behavior.

While the specific measures of nonshared environmental influence studied here were not significant for Factor 2 for the total group, this is not to say that nonshared influences are unimportant for light drinking behavior. The success of the model depends

upon the selection of nonshared environmental variables. There are a number of nonshared environmental variables -- for example, parental discipline, parental involvement, peer influence -- that could conceivably contribute to this behavior. 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 nonshared variables. The nonshared measures of self-esteem and locus of control were found to be important here in accounting for variation in heavy drinking behavior for the total group.

The analyses by gender-pair produced some interesting results, consistent with the suspicious findings noted by Cotton (1979). In that study, Cotton noted that although there is, 1) a high rate of alcoholic fathers of alcoholics, and, 2) 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. I found that there is indeed a stronger genetic component for female pairs than for male pairs. In fact, the total variance accounted for by the genetic component alone was .45 and .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 some for genetic reasons, but that heavy drinking is more environmental. And while self-esteem is an important measure in accounting for nonshared personality influences for male pairs, neither self-esteem or 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 neighborhood of .32 to .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 behavior is more the result of an environmental affect for Blacks, but light drinking is more genetically related (which, interestingly, matched the basic pattern for the male pairs). There were no significant nonshared influences found in this study for either the Hispanic 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 nonshared 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 furthered the work in the alcohol use field in several ways. First, I have supported the belief that alcohol use has an important hereditary component and provided estimates, by subgroup, of the extent of the genetic component. Second, I have pointed out the importance of the environmental component, particularly among males and blacks for heavy drinking, and provided estimates of the extent of the environmental contribution. Third, I have issued a call to examine the importance of nonshared factors in the determination of alcohol use. This call reflects perhaps the most critical oversight in a field of study with such a long history. Nonshared influences that carefully measure environmental differences were not available in the data used here, 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 etiology of alcohol use and abuse.

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Table 1
Alcohol (NLSY) Questions

1. Ever had a drink
2. Had any alcoholic beverages in the last month
3. Drinking ever interfered with school work
4. Drinking ever interfered with your job
5. Frequency of 6/more drinks at 1 time last month
6. Number of days drank alcohol in the last month
7. Number of days had 1 drink in the last month
8. Number of days had 2 drinks in the last month
9. Number of days had 3 drinks in the last month
10. Number of days had 4 drinks in the last month
11. Number of days had 5 drinks in the last month
12. Number of days had 6/more drinks in the last month
13. Total number of days had a drink last month
14. Frequency going to bars last month
15. Number of days had a hangover in the last month
16. Number of days drank alcohol in the last week
17. Number of cans/bottles of beer consumed last week
18. Number of glasses of wine consumed last week
19. Number of drinks w/liquor consumed last week
20. Drinking ever interfere w/school work
21. Drinking ever interfere w/job
22. Ever felt aggressive/cross while drinking
23. Ever got into a heated argument while drinking
24. Ever gotten into a fight while drinking
25. Ever try cut down/quit drink but failed
26. Are you afraid you might be/become an alcoholic
27. Do you have difficulty stopping drinking until you are completely intoxicated
28. Are you unable to remember things done while drinking
29. Do you often take a drink 1st thing in the morning
30. Do your hands shake a lot the morning after drinking
31. Have you gotten high/tight when drinking alone
32. Have you kept drinking after you promised not to
33. Have you stayed away from work because of a hangover
34. Have you gotten high/tight on the job
35. Have you nearly/lost job because of drinking
36. Has drinking led to quitting a job
37. Has drinking hurt your chances for promo/raises

Table 2
Resulting Factors

Factor 1 (In order of loading)

1. Frequency of 6/more drinks at 1 time last month
2. Number of days had 6/more drinks in the last month
3. Number of cans/bottles of beer consumed last week
4. Number of days drank alcohol in the last week
5. Number of days drank alcohol in the last month
6. Total number of days had a drink last month
7. Number of days had 5 drinks in the last month
8. Number of drinks w/liquor consumed last week
9. Frequency going to bars last month
10. Number of days had a hangover in the last month
11. Number of days had 4 drinks in the last month

% of Variance: 22

Factor 2 (In order of loading)

1. Number of days drank alcohol in the last month
2. Total number of days had a drink last month
3. Number of days had 1 drink in the last month
4. Number of days had 2 drinks in the last month
5. Number of days drank alcohol in the last week
6. Had any alcoholic beverages in the last month
7. Number of days had 3 drinks in the last month
8. Frequency going to bars last month

% of Variance: 7

Factor 3 (In order of loading)

1. Have you kept drinking after you promised not to
2. Ever got into a heated argument while drinking
3. Are you afraid you might be/become an alcoholic
4. Ever felt aggressive/cross while drinking
5. Are you unable to remember things done while drinking
6. Do you have difficulty stopping drinking until you are completely intoxicated
7. Ever try cut down/quit drink but failed
8. Ever gotten into a fight while drinking
9. Have you gotten high/tight when drinking alone
10. Do your hands shake a lot the morning after drinking
11. Drinking ever interfered w/job

% of Variance: 9

Table 3
Descriptive Statistics

Factor 1 (Heavy Drinking)

<u>Verbal Description</u>	<u>R</u>	<u>N</u>	<u>Mean</u>	<u>Standard Deviation</u>	<u>Corr</u>
Cousins	.125	146	3.61	3.53	.23
Half Siblings	.250	82	4.76	3.40	.23
Ambiguous Siblings	.375	590	4.24	3.20	.24
Full Siblings	.500	3,646	4.23	3.31	.30
Twins	.750	54	4.11	3.31	.46

Factor 2 (Light Drinking)

<u>Verbal Description</u>	<u>R</u>	<u>N</u>	<u>Mean</u>	<u>Standard Deviation</u>	<u>Corr</u>
Cousins	.125	146	3.43	3.07	.22
Half Siblings	.250	82	4.32	2.74	.21
Ambiguous Siblings	.375	598	4.24	2.69	.21
Full Siblings	.500	3,660	4.18	2.90	.30
Twins	.750	54	4.22	2.93	.48

Table 4
DF Analysis Results

Subgroup	N	Factor 1			N	Factor 2		
		h²	c²	R²		h²	c²	R²
Total Dataset	4,518	.25	.17	.08	4,540	.34	.13	.09
Whites	2,666	.24	.18	.09	2,682	.21	.21	.10
Blacks	1,096	.02	.19	.04	1,096	.38	.05	.05
Hispanics	756	.33	.002	.03	762	.32	.01	.03
Male Pairs	1,284	.10	.27	.10	1,288	.28	.16	.08
Female Pairs	1,158	.45	.13	.12	1,166	.49	.12	.13
Opposite-sex Pairs	2,076	.55	-.07	.04	2,086	.57	-.05	.05

NOTE: Ns are total individuals. These are adjusted back to total pairs to run significance tests.

Table 5
Distributions of Factors 1 & 2

Factor 1	Frequency	Cumulative Percent
0	2313	31.3
2	355	36.1
3	494	42.8
4	629	51.3
5	756	61.5
6	709	71.1
7	783	81.7
8	673	90.8
9	456	97.0
10	170	99.3
11	55	100.0

Factor 2	Frequency	Cumulative Percent
0	2310	31.2
1	3	31.2
3	86	32.4
4	660	41.3
5	1373	59.8
6	1454	79.4
7	994	92.8
8	532	100.0