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GENETIC LOADING FOR ALCOHOLISM: NEW EVIDENCE FOR SUBTYPES

By

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ABSTRACT

GENETIC LOADING FOR ALCOHOLISM: NEW EVIDENCE FOR SUBTYPES

By

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Current research on the etiology of alcoholism suggests that genetic vulnerability plays a role in the development of alcoholism. This study, using a population-based sample and a broad set of independent variables, examined how genetic loading for alcoholism affects developmental course of alcoholism and what moderating effects various environmental variables might have on genetic load. Results confirmed that high genetic loading for alcoholism was related to earlier onset and greater severity of drinking difficulties and more frequent alcohol use. More importantly, alcoholic subtype was a significant factor in predicting etiologic pathways into alcoholism. Among Type 2 alcoholics, genetic loading for alcoholism contributed strongly to later alcohol problems, as did history of socialization to aggression. For Type 1s, rearing in an alcoholic environment was the only predictor of adult alcohol problems, suggesting that socialization to using alcohol as a coping mechanism is most important for this subtype.

To Tom

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Introduction and Literature Review

Research into the etiology of alcoholism has been hampered by the lack of a widely shared definition of alcoholism. Because of this lack of consistency, it is often difficult to compare research across studies; one cannot be sure that the same populations are under study.

The classical definition of alcoholism was formulated by E.M. Jellinek in the 1950's. He decried the broader medical profession's use of the word 'alcoholism' for any type of heavy drinking. Instead, Jellinek (1952) distinguished two types of alcoholics (addictive and non-addictive) in whom excessive drinking occured due to underlying pathology. Using the medical model, he applied the disease conception of alcohol addiction not to excessive drinking per se, but to the 'loss of control' over drinking that accompanied it. He considered this loss of control to be a disease condition that only occured in addictive alcoholics.

More modern formulations of the medical model of alcoholism include the diagnostic criteria of DSM-III-R (A.P.A.,1987). As did Jellinek, DSM-III-R considers alcoholism to be a disease with some unknown etiology. It defines people with alcohol dependence syndrome as those who have any three of the following symptoms: a loss of control over alcohol intake, a desire to cut down on intake, a great deal of time spent obtaining alcohol, frequent intoxication or withdrawal symptoms which interfere with role obligations, participation in important social or occupational activities reduced due to alcohol use, continued use despite recognition of a problem with alcohol, tolerence of alcohol, withdrawal symptoms, or intake of alcohol to avoid withdrawal. Because alcoholism is thought to exist in differing degrees of severity, not all of these indicators need be present to define a person as an alcoholic.

Another medical model of alcoholism which has recently been proposed is Edward and Gross's (1976) Alcohol Dependence Syndrome. The seven facets of the syndrome are narrowing of the drinking repetoire, salience of drink-seeking behavior, increased

tolerance to alcohol, repeated withdrawal symptoms, repeated relief of withdrawal symptoms by further drinking, subjective awareness of a compulsion to drink and reinstatement of the syndrome after abstinence. Edward and Gross' definition of alcoholism differs from DSM-III-R in that social problems resulting from drinking are not included as indicators of alcohol dependence (Caetano, 1985).

The term 'alcoholism' has been used differently in various studies; not all use the DSM-III-R criteria (or those of its predecessor DSM-III) to obtain samples of alcoholics. Bohman (1978), for example, defined his sample of alcoholics based on law-breaking: alcoholics were those who had been registered with local Temperence Boards set up to regulate misuse of alcohol. Defining alcoholism in such a manner is problematic, since criteria for being registered with the Temperence Board have changed over time. Cadoret, Troughton and O'Gorman (1987) considered alcoholics to be those who had one or more recorded social or medical problems because of drinking (i.e. lost a job due to drinking, got a divorce due to drinking). In similar fashion, Schuckit (1984) defined alcoholism as the occurence of one of several alcohol- related major life problems, such as dropping out of school. One problem with such definitions is that they do not clearly differentiate alcoholics from 'problem' drinkers, who may also have had difficulties functioning in society due to excessive drinking. One way to distinguish between these two groups may relate to Jellinek's (1952) original definition of alcoholism; 'problem' drinkers might well be conceptualized as Jellinek's 'non-addictive' alcoholics, who do not lose control over their drinking but may still drink to excess.

Mulford and Miller (1960) described alcoholism as a function of a set of social norms which gave the individual a certain way of thinking about or defining himself, alcohol, and the relationship between the two. Alcoholic drinking, in Mulford and Miller's conceptualization, allowed an alcoholic to redefine himself and his relationship to others in a way that was more self-satisfying. Unlike those discussed previously, this definition conceptualized alcoholism within a social learning theory framework.

One important question needs to be answered in order to produce a useful definition of alcoholism: is alcoholism one end of a continuum that includes 'heavy' or 'problem' drinkers? Goodwin (1979) has argued, based on his studies of Danish adoptees, that alcoholism results from a different developmental path than does 'heavy drinking'. However, research by Clark and Cahalan (1976) has indicated that more severe forms of alcoholism merge gradually with problem and heavy drinking. A distinct, pathological type of alcoholism does not appear to stand out on a population curve of those with drinking problems (Peele, 1986).

Finally, the issue of whether there are several different kinds of alcoholism, each with its own etiology, remains. Zucker (1987a) has proposed that four different alcoholisms, each with its own cause and course, exist: antisocial alcoholism, developmentally limited alcoholism, developmentally cumulative alcoholism and negative affect alcoholism. Cloninger and Bohman's (1981) research also points to the need to develop definitions of alcoholism which are specific to different alcoholic subpopulations.

Alcoholism: A Genetic Etiology?

What kinds of evidence led to the idea that inherited factors might play a role in the development of alcoholism? One of these was family studies. Such studies indicated that rates of alcoholism were much higher among the families of alcoholics than among the general population. Cotton's (1979) literature review encompases 39 studies of alcoholics. She found that regardless of the nature of the non-alcoholic population used as a comparison group, an alcoholic was more likely than a non-alcoholic to have a mother, father or other close relative who was alcoholic. The fact that alcoholics were twice as likely to report parental alcoholism as other psychiatric patients implied that a high rate of familial alcoholism was specific to alcoholics.

Two-thirds of the studies that Cotton reviewed found at least 25% of all alcoholics to have alcoholic fathers. One third of alcoholics were found to have at least one alcoholic

parent. Cotton noted that if anything, her figures erred on the side of conservatism due to demonstrated underreporting of mental illness and alcoholism in the 39 studies.

Family studies also indicate that the rate of alcoholism is increased in the offspring of alcoholics. Goodwin (1979) reported that sons of alcoholics were four times as likely as were sons of non-alcoholics to become alcoholics themselves. Winokur, Reich, Rimmer and Pitts (1970) also investigated the occurence of alcoholism in children of alcoholics. They diagnosed 31% of the sons of male alcoholics to be alcoholic upon reaching adulthood; an astonishing 51% of sons of female alcoholics were found to be alcoholic as adults.

The fact that the rate of alcoholism is higher within families of alcoholics than in the general population does not by itself prove that a genetic vulnerability to alcoholism exists. An alternative explanation would be that similar environments are recreated within alcoholic families, such that offspring in each generation are exposed to an alcohologenic environment. But when coupled with results from animal research, the concept of a genetic role in the development of alcoholism begins to be a plausible hypothesis.

Animal studies have demonstrated strain and line differences in the acquisition of alcohol preference, sensitivity, tolerance and dependence. Building animal models for alcohol preference, for example, provides information about genetic mechanisms related to voluntary excessive consumption in humans. Lumeng, Hawkins and Li (1977) were able to use selective breeding to produce a true-breeding strain of rats that voluntarily consumed large amounts of alcohol. However, it could be argued that the rats were drinking the alcohol because they liked the taste or smell, rather than, like humans, for its mood altering effects. To demonstrate that this was not the case, Waller, McBride, Gatto, Lumeng and Li (1984) inserted devices directly into the stomachs of rats which provided the rats with their choice of water or alcohol. Rats bred from the alcohol-preferring strain self-infused with up to 9.4 g of alcohol per kilogram body weight each day as compared to the non-alcohol preferring strain of rats which self-infused a maximum of .7 g per kilogram body

weight per day. Apparently, the rats bred for alcohol preference were seeking the pharmacological effect of the alcohol. The fact that animals usually refuse to drink alcohol (Petrakis, 1985) makes this research even more intriguing.

Genetics have also been shown to play a role in animals' sensitivity to alcohol. McClearn and Kakihana (1981) developed two strains of mice which they called short sleep (SS) or long-sleep (LS) based on the duration of unconciousness after receiving a dose of ethanol. Selective breeding over several generations produced large differences in sensitivity to alcohol. Quantities of ethanol that merely anesthetized SS mice were close to lethal for LS mice. Mice selected for their genetically lower sensitivity to alcohol have also been shown to develop alcohol tolerance more quickly during prolonged exposure to alcohol (Gallaher and Gionet, 1988).

Lastly, two groups of researchers have developed animal models which demonstrate a genetic contribution to alcohol dependence in mice. Crabbe, Kosobud and Young (1983) and Allen, Petersen, Wilson and McClearn (1983) have selectively bred different strains of mice which can be distinguished on the basis of the severity of their withdrawal symptoms after alcohol is removed from their diet.

Therefore, indirect research evidence points to a genetic contribution to the development of alcoholism in humans. More direct evidence from human genetic studies will now be reviewed.

Genetics and Human Alcohol Consumption

Twin Studies

There are several kinds of studies which allow conclusions to be drawn about the role of inheritence in the development of alcoholism. One type is the twin study, where drinking behavior in monozygous (MZ) twins (who are assumed to be genetically identical) is compared to drinking behavior in dizygous (DZ) twins (who are related as normal

siblings). Higher rates of concordant drinking behavior in the monozygous twins can then be attributed to their more similar genotype.

In one early twin study, Partanen, Brunn and Markannen (1966) investigated drinking behavior in twins with 'normal' drinking patterns. The only inclusion criteria for the study were that subjects be a member of a twin pair born in Finland between 1920 and 1929 and that they currently be living in Finland. After recruiting 902 male twin pairs, the group found a significantly higher concordance rate in amount and density (i.e. frequency) of drinking among MZ twins than among DZ twins. Heritability was calculated to be .36 for amount of drinking and .39 for density of drinking; Partanen et al. concluded that genetic factors influence drinking patterns among normal individuals.

Similar research was conducted by Kaprio, Koshenvuo, Langinvanio, Romanov, Sarna and Rose (1987) who also assessed 'normal' drinking behavior. They compared 879 male pairs of monozygous twins and 1940 pairs of dizygous twins from the Finnish Twin Cohort. The Finnish Twin Cohort consists of all like-sex twin pairs born in Finland in 1958 among whom both twins were living in 1967. Subjects used in this study comprised nearly all surviving male twins in the 25-49 age range. Information was requested on the frequency (number of days of alcohol use), quantity (amount of alcohol used) and density (number of days of excessive use) of alcohol consumption per month and the frequency of passouts in the previous year. Results confirmed genetic effects for frequency, quantity and density of drinking, but not for passouts. Kaprio's group found heritability rates that were similar to those of Partanen: .39 for frequency of beer consumption, .38 for frequency of spirit consumption, .40 for density of consumption and .36 for quantity of consumption.

Partanen's work was also confirmed by Gabrielli and Plomin (1985) whose sample included 46 MZ and 44 DZ twin pairs as well as 46 unrelated pairs of subjects who were raised together. 203 subjects were females and 143 were males; median level of schooling was 14.5 years. Unrelated pairs of adoptees were included to assess the importance of

shared family environment in alcohol consumption, independent of genetic influence. The Colorado Alcohol Behavior Questionnaire was used to assess amount, frequency and rate of alcohol consumption. Gabrielli and Plomin found significantly higher correlations of both amount and rate of alcohol consumption among MZ twins. Based on this data, they calculated a 66% heritability for rate and 25% heritability for amount of alcohol consumption. Gabrielli and Plomin concluded that genetic influences on drinking behavior were more important than shared family environment.

The classic twin study which investigated alcohol abuse among twin pairs was Kaij's (1960). Kaij's subjects were 292 Swedish twins. At least one member of each twin pair had been registered with Temperence Boards set up to control excessive drinking. After categorizing each twin in terms of alcohol consumption, Kaij found that the co-twin of an index MZ twin was much more likely to fall into the same category of alcohol consumption as his twin than the co-twin of an index DZ twin. For example, he found that among chronic alcoholics, 71.4% of co-twins in MZ pairs fell in the same category as their index twin, while this was only true of 32.3% of co-twins in DZ pairs. Based on these results, Kaij concluded that alcohol abuse was largely an inherited trait.

Murray, Clifford and Gurling (1983), however, criticized Kaij's findings on several grounds. One criticism was the low proportion of MZ twins in the study. This raised the possibility that some MZ twin pairs were mistakenly labelled as DZ. How this would lower the drinking concordance rate in the DZ group relative to the MZ group was not explained, however. A more important criticism presented by Murray's group was that Kaij's sample was not representative of alcoholics in general. For example, alcoholics registered with the Temperence Board were much more likely to have been convicted of alcohol-related criminal acts than most alcoholics.

A more recent study of twins with drinking problems was conducted by Hrubec and Omenn (1981). Hrubec and Omenn accessed records of the VA to collect data on alcohol-

dependent twin pairs. They found MZ twins to be more concordant than DZ twins on rates of alcoholism, cirrhosis of the liver, and alcohol psychosis.

Murray et al. (1983) also criticized Hrubec and Omenn's methodology. They pointed out that overall detection rate of alcoholics among veterans in the study was lower than would be expected. They argued that the higher concordance rate among MZ twins than among DZ twins could be explained away if alcoholic co-twins of MZ index alcoholics were simply identified more often than alcoholic co-twins of DZ index alcoholics. Murray and his coworkers felt that alcoholic co-twins of MZ probands were indeed more likely to be identified by the armed forces because their relatedness was more noticeable than that of DZ twins.

It has been argued that twin studies which find higher concordance rates for MZ than DZ twins do so simply because MZ twins share a more similar environment than DZ twins (Scarr, 1968). For example, parents may be more likely to treat their children alike if the children look identical then they may be to treat their children alike if the children do not resemble one another. Such factors could lead to more concordant drinking behavior among MZ than among DZ twins in the absence of genetic loading for alcoholism. However, several studies have disputed this argument by examining twins whose zygosity was incorrectly classified (Scarr and Carter-Saltzmann, 1970; Matheny, 1979). Results showed that true zygosity, not self-perceived zygosity or zygosity perceived by parents, determined similarity of the twins' behavior. Loehlin and Nichols' (1976) research also helped to resolve this issue. They correlated the similarity of twin pairs' social environment in childhood with the similarity of their scores on the California Personality Inventory and National Merit Test in adolescence. Results showed little or no correlation between similarity of social environment as children and similarity of intellectual and personality styles in high school.

Therefore, it appears that the higher concordance rates of drinking behavior for MZ twins are not an artifact. They cannot be explained away by assuming that MZ twins share

a more similar environment than DZ twins. However, the concordance rates for MZ twins are not even close to the 100% one would expect if alcoholism was caused entirely by genetic factors. In order to differentiate the effects of nature and nurture more clearly, it is necessary to turn to adoption studies.

Adoption Studies

Adoption studies allow researchers to more clearly separate the effects of heredity from the effects of environment. Assuming that the child of an alcoholic biological parent is separated from that parent shortly after birth, it is possible to see if the child's proposed genetic predisposition for alcoholism affects him even in a family environment where alcohol abuse does not occur.

One of the earliest adoption studies on children of alcohol abusers was conducted by Roe and Burks (1945). Roe and her associate found that none of the adopted-away sample of children of alcoholics and none of their matched controls showed any symptoms of alcohol dependence. They concluded that heredity had no influence on the development of alcoholism. However, these results may have been biased by the fact that children of alcoholic biological parents were more often placed in rural homes (where the opportunity to drink was infrequent) than children of non-alcoholic parentage (Cloninger et al., 1981). Also, because Roe and Burks never personally interviewed the natural parents, questions have arisen as to whether fathers of the index cases were really alcoholic (Goodwin et al., 1973). Finally, Schuckit (1980) has questioned Roe and Burks' results on the basis of small sample size.

The adoption study which began the modern debate over the contribution of genetics to alcoholism was that of Goodwin and his colleagues in 1974 (Murray et al., 1983). Goodwin et al.'s (1974) sample consisted of male Danish adoptees. The index group was made up of sons of alcoholics raised by non-alcoholic foster parents while the control group was made up of sons of non-alcoholics paired on age and circumstances of adoption.

Data on sons of alcoholics who were raised by their biological parents was also included. Results showed that sons of alcoholic biological parents were four times more likely to become alcohol abusers than sons of non-alcoholic biological parents, regardless of whether or not an alcoholic parent raised them. However, sons of alcoholics did not differ from controls when the dependent measure was rate of heavy drinking. These results strongly indicated that no matter what family environment sons of alcoholics were raised in, some type of genetic factor made them more vulnerable to alcoholism (but not to heavy drinking).

Murray et al. (1983) thought it curious that widening criteria for drinking pathology to include heavy drinking totally eliminated Goodwin's argument for a genetic diathesis in alcoholism. Pointing out that this contradicts the evidence that heavy drinking and alcoholism are closely related, they asked:

> " Could it be that Goodwin's findings are simply an artifact produced by the threshold for alcoholism accidentally dividing heavy drinkers in the index and control group unevenly?"

In another criticism of Goodwin's work, Fillmore (in press) suggested that Goodwin and colleagues' results were biased because of socioeconomic (SES) differences between the control and index group. She proposed that children of alcoholic biological parents would be more likely to be adopted out to lower class parents than children of normal biological parents. There is substantial evidence for the existence of social class differences in rates of alcoholism (Zucker, 1987a); (Cahalan and Cisin, 1976); more environmental press for deviance (i.e. alcoholism and antisocial behavior) seems to exist in lower socioeconomic groups. Given this fact, if adopted by lower SES parents, the index group in Goodwin's study would have been at higher risk for alcoholism simply because rates of

alcoholism are higher in lower class families. There would be no need to include heredity in an explanation of group differences.

A different adoption study by Cadoret and Gath (1978) involved 84 adoptive families. Families were recruited through the 1939-1965 adoption records of the Iowa Childrens' and Family Services Agency. In six cases, the biological parents of the adoptive child were judged to have been alcoholic. A significant difference was found in alcoholism rates in the two groups such that adoptive children with an alcoholic biological parent were much more likely to become alcohol dependent. Questions about the validity of this study have been raised because of the use of parental interview to diagnose alcoholism in adoptees, the differential sample loss among families with alcoholic versus non-alcoholic adoptees, the possibility of impressionistic diagnosis of alcoholism in biological parents by adoption agency staff, and small sample size (Murray et al., 1983); (Fillmore, in press). However, a follow-up study by Cadoret, Troughton and O'Gorman (1985) was much more tightly controlled. The sample was drawn from Lutheran Social Services records. Alcoholism in the adoptees was diagnosed using DSM-III criteria during personal interviews. To assess sample loss, adoptive families who refused to participate were evaluated for alcohol use and found to be equivalent to participating families. The same type of results were found as before, indicating a genetic influence on the development of alcohol abuse for both males and females.

Some of the best data from adoption studies comes from the research of C. Robert Cloninger and Martin Bohman. One of their studies (Cloninger, Bohman and Sigvardsson, 1981) involved 862 male Swedish adoptees, of whom 151 had some record of alcohol abuse. Subjects were part of the Stockholm Adoption Study. The sample included all persons born out of wedlock between 1930 and 1949 who were placed for adoption. Each of the adoptees, based on his drinking behavior, was classified as belonging to one of four groups. Data about both adoptive and natural parents was collected as well. Analysis of Cloninger et al.'s data showed a significant correlation

between alcohol dependence in the biological parents and alcohol dependence in the adoptees.

The researchers also wished to know if the biological parents of severely alcohol dependent adoptees had differed from biological parents of mildly alcohol dependent adoptees. They were able to demonstrate the existence of two types of alcoholism in the alcohol abusing adoptees which were associated with psychological standing of the natural parents. One type of alcoholism (male-limited or Type II) was proposed to be highly heritable from father to son and to result in a moderate degree of alcoholism in the son. Supporting this hypothesis, male-limited alcoholics were found to have no excess of alcoholic mothers. In addition, Type II alcoholism was found to be associated with criminality and severe alcoholism in the adoptees' natural father. Therefore, in the male-limited alcoholic, antisocial behavior and alcoholism were found to be closely linked. Cloninger, Sigvardsson and Bohman (1988) also found that personality traits associated with antisocial behavior such as novelty-seeking behavior and harm avoidance which were measured in a large sample of eleven year olds predicted early-onset alcoholism (i.e. Type 2) in adulthood. Such information is important in light of studies by Vaillant (1983) and McCord and McCord (1962) showing that childhood antisocial behavior is a good predictor of later alcohol problems.

The other type of alcoholism described by Cloninger et al. (milieu-limited or Type I) was proposed to be somewhat heritable from either biological parent and was associated with milder alcohol dependence and no record of criminality in the natural parents. In addition, Type I alcoholism was thought to be influenced by post-natal environmental factors, and to result in either a mild or severe degree of alcohol abuse depending on the degree of post-natal environmental stress. In order to reconcile these findings with Goodwin's (1974), which implied that family environment was not important in the development of alcoholism, Cloninger et al. (1985) suggested that Goodwin's sample consisted of male-limited, not milieu-limited, alcoholics.

A replication of the group's results among 913 female Swedish adoptees from the Stockholm Adoption Study (Bohman et al., 1981) confirmed the initial findings. First, there was a three-fold excess of alcohol abusers among adopted daughters of alcoholic biological mothers as compared to daughters of non-alcoholics. Biological fathers with a record of criminality and severe alcoholism had very few alcoholic daughters. This is consistent with the prediction that male-limited alcohlism is mostly passed on to sons. However, there was a high degree of alcohol abuse among daughters of biological parents who were not involved in criminal activity and whose alcohol abuse was mild, supporting the idea that milieu-limited alcoholism is heritable by either sex. Bohman et al. concluded that alcoholism in women generally fit the Type 1 pattern.

The work of Bohman and Cloninger is important for several reasons. It not only replicated earlier findings of a genetic contribution to alcoholism, but it was one of the first pieces of literature to suggest different degrees of heritability for different types of alcoholism. Considered from this perspective, it is possible that adoption studies such as Roe and Burks' (1945) found no evidence for a genetic contribution to alcoholism simply because they studied children of 'milieu limited' alcoholics in adaptive family environments (Cloninger et al., 1985).

As stated earlier, the validity of this research can be questioned because of its reliance on registration with the Temperence Board to define alcoholism. The fact that legal criteria for registration with the Temperence Board changed over time calls into question the similarity of parental alcoholics to their alcoholic offspring (Fillmore, in press). At the very least, it may limit how generalizable Bohman and Cloninger's results are to other alcoholic populations.

Searles (1988) has also pointed out several flaws in the Cloninger studies. He noted that in contrast to data from epidemiological studies, Cloninger and his colleagues found no increased risk for alcohol abuse as a function of age in their sample. This could be another indication that their sample is atypical of alcoholics in general. In addition, Searles pointed

out that the group's system for classifying subjects as mild, moderate or severe was inadequate; he suggested that their findings might simply be an artifact of their criteria for abuse.

The evidence from both adoption and twin studies leads to similar conclusions. Confirming the findings from twin studies which indicated that genetic factors influenced the development of alcoholism, most of the adoption studies found higher rates of alcoholism among adoptees whose natural parents had been alcoholics. Although some of the studies were methodologically flawed, the fact that replications kept producing the same pattern of results indicates that there is indeed a genetic component to the development of alcohol abuse. However, as a caveat, it is important to remember that it is not at all clear that parents who give their children up for adoption are representative of the general alcoholic population; they may show more signs of antisocial behavior (Murray et al., 1983). Lastly, there was little consensus among adoption studies about the degree to which post-natal family environment affects genetic loading for alcoholism.

There is obviously a need for well-controlled studies which define alcoholism in a useful way and which also have large samples such as Cloninger et al.'s (1981). Also, there is a need for research which makes even greater distinctions between alcoholic subpopulations and the degree of heritability of each.

If one assumes that alcoholism is indeed influenced by genetic factors, then what exactly is being inherited? Factors from biochemical abnormalities to temperament differences have been implicated. The evidence for such factors will now be reviewed.

What Do Alcoholics Inherit?

Schuckit (1980), in a broad overview of possible biological mediators of alcoholism, suggested five possible mechanisms through which a genetic predisposition for alcoholism could express itself. The first of these was that individuals at risk for alcoholism could inherit different acute responses to doses of alcohol. For example, alcohol might produce

more intense pleasure for alcoholics. The second of these was that high risk individuals might inherit different subacute reactions to alcohol. As a third mechanism through which a genetic predisposition could express itself, Schuckit proposed that high-risk individuals might be more vulnerable to chronic alcohol exposure than other individuals. A fourth proposed mechanism was differences in the way individuals at risk metabolize alcohol as compared to individuals not at risk. Finally, Schuckit suggested that high-risk individuals might inherit factors which affected psychological parameters such as temperament.

Biochemical Abnormalities

It is often hypothesized that alcoholics inherit a biochemical abnormality which somehow affects their interactions with alcohol. Schuckit and Rayses (1979) proposed that alcoholics produce higher amounts of acetaldehyde than non-alcoholics; because alcetaldehyde is a breakdown product of alcohol metabolism in the liver, genetic variations in the efficiency of alcohol-metabolizing enzymes would affect acetaldehyde concentrations in the body. To prove that this was an inherited vulnerability, Schuckit and Rayses compared non-alcoholic subjects with a positive family history of alcoholism to matched controls with no family history of alcoholism. Results confirmed that after drinking alcohol, the subjects with a positive family history had significantly higher breath concentrations of acetaldehyde. Unfortunately, attempts to replicate this important finding have been unsuccessful (Knop, Angelo and Christensen,1981).

Another biochemical abnormality which may be inherited by alcoholics is low levels of monoamine oxidase (MAO), an mitochondrial enzyme that catalyzes the oxidative deamination of biogenic amines. (Faraj, Lenton, Kutner, Camp, Stammers, Lee, Lolies and Chandora, 1987). Monoamine oxidase is involved in brain neurotransmitter metabolism, but is also found in blood platelets. (Alcohol and Health, 1987). Initial research suggested that chronic alcoholics had MAO levels which were lower than normal (Oreland et al., 1983); (Faraj et al., 1987). Puchall, Coursey, Buchsbaum and Murphy

(1983) were than able to demonstrate that MAO level was genetically determined. 75 subjects with either high or low MAO levels were chosen and MAO level was correlated with that of their parents. Results showed significant and positive correlations.

Low MAO levels have been shown to be correlated with a tendency to increase or 'augment' stimulus intensity (Buchsbaum, Landau, Murphy and Goodwin, 1973); alcoholics as a group are likely to be stimulus augmenters (Petrie, 1967). In addition, low MAO levels are associated with the type of fast tempo and vigorous behavioral response style which is typical of alcoholics (Tarter, Alterman and Edwards 1985).

Von Knorring, Bohman, Von Knorring and Oreland (1985), building upon the work of Cloninger and Bohman, first showed that the male- limited (highly heritable)/ milieu-limited (somewhat heritable) typology could validly differentiate alcoholics in a clinical setting. The typology was used to classify 31 male and five female alcoholics treated through a university outpatient psychiatric clinic. They then demonstrated that the MAO levels of milieu-limited alcoholics did not differ significantly from those of healthy controls, whereas male-limited alcoholics had significantly lower MAO levels than controls. Findings of lower MAO levels among Type 2 alcoholics were confirmed by Pandey, Fawcett, Gibbons, Clark and Davis (1988).

Brain Abnormalities

Some researchers feel that alcoholics inherit an anomalous brain structure which leads to some type of neurological dysfunction. Schuckit (1984) proposed that alcoholics were less able than non-alcoholics to use internal cues to estimate their blood alcohol level (BAL) after drinking. His sample consisted of 23 non-alcoholic male college students with either a positive or negative family history of alcoholism. After consuming alcohol, subjects with a family history of alcoholism had significantly lower self-ratings of intoxication than controls. These results indicate that alcoholics may inherit a deficit in the ability to learn to

process cues about internal state, especially when the internal state experienced is alcohol-induced.

Several studies have been conducted on the electroencephalograms (EEGs) of persons at high risk for alcoholism. Propping (1977), in a study of 52 healthy twin pairs, showed that the extent of alcohol action on the resting EEG was under genetic control. After giving subjects a dose of ethanol, he recorded their EEG's; EEG's of MZ twins reacted identically to alcohol loading whereas EEG's of DZ twins became more dissimilar. Propping and his colleagues then conducted a follow-up study on relatives of alcoholics and matched controls (Propping, Kruger and Nark, 1981). They found that non-drinking females with a positive family history of alcoholism had a significantly poorer EEG synchronization than female controls. No such effect was found for males, however.

Pollock, Volavka, Mednick, Goodwin et al. (1984) found that after consuming alcohol, 44 subjects at high risk for alcoholism could be differentiated from 28 matched controls by their EEG alpha frequencies. Subjects in the high-risk group showed significantly greater increases in slow alpha frequencies and decreases in fast alpha frequencies. The researchers interpreted the results to mean that EEGs could function as a biological marker for an inherited central nervous system (CNS) sensitivity to the effects of alcohol among alcoholics. Gabrielli, Mednick, Volavka, Pollock et al. (1982) found that 27 young high- risk children of alcoholics showed more beta wave activity in their EEG's than 27 matched controls.

Begleiter, Porjesz, Bihari and Kissin (1984) studied visually produced event-related brain potentials (ERPs) among 25 non-drinking sons of alcoholic fathers and matched controls with no family history of alcoholism. They found significant group differences in the P3 component of the ERP. Begleiter's group proposed that because P3 potentials reflect processes involved in revising representations stored in memory, alcoholics might inherit deficits in memory processing.

While this area of research is promising, a major problem exists. Although each study on inherited brain wave abnormalities has found subjects at high risk for alcoholism to differ from healthy controls, two studies have rarely found the same anomalous brain wave patterns (Peele,1986). Therefore, findings may well be sample specific. Also, none of these studies have discussed in depth how an inherited brain abnormality would lead to alcoholism.

Temperament

It is also possible that alcoholics inherit a temperament that makes them more vulnerable to alcohol abuse. Tarter, Alterman and Edwards (1985), in an elegant discussion, identified six temperament dimensions which might play a part in vulnerability to alcoholism. These six factors were activity level, attention span /persistence, soothability, emotionality, reaction to food and sociability. Tarter's group presented evidence that at least some of these temperament dimensions had a genetic component and then discussed possible underlying biological mechanisms. This piece of work is important because it is one of the few papers which describes how inherited biological dysfunctions could express themselves as factors which would hinder an alcoholic's functioning in his environment.

Statement of the Problem

After reviewing the literature, it seems clear that some genetically-based vulnerability to alcoholism exists. Animal models have been developed which show that true-breeding, alcohol-preferring strains of rats can be produced with relative ease. The evidence from twin and adoption studies also indicates that the children of alcoholic parents inherit some factor which places them at risk for alcoholism. However, much of the research suffers

from poor methodology and problems with the way in which alcoholism is defined. Therefore, generalizability becomes questionable.

Moreover, as previously discussed, although the best evidence which supports the theory of genetic loading for alcoholism comes from adoption studies, it is not at all clear that parents who give their children up for adoption are representative of the general alcoholic population. If adoption studies have indeed used samples which tend to be more heavily involved in antisocial behavior, it is possible that alcoholism is highly heritable only among such groups. The present study addresses this issue by studying genetic loading for alcoholism within a sample who were not adopted out by their natural parents.

Furthermore, few studies have investigated how genetic loading for alcoholism might influence various different drinking-related variables; those which have done so have only considered a limited range of factors, such as frequency and amount of drinking. This study, using male subjects, examines the relationship between genetic loading for alcoholism (as measured by family expression of alcoholism) and age of first drunkenness, lifetime number of areas of drinking problems, percent of the lifespan characterized by alcohol problems and quantity-variability and frequency of drinking. In addition, alcoholic male subjects are characterized as Type I or Type II alcoholics in order to test whether these different drinking-related variables are more heritable among male-limited alcoholics. Such data are unique in that they provide information on the degree to which familial (and ostensibly genetic) density of alcoholism affects the onset, duration and severity of alcohol problems. They also help differentiate which aspects of drinking problems are under genetic control.

Lastly, the study examines the role of other posited factors that moderate heritability of alcohol abuse. While the effects of factors such as environmental press for deviance (as defined by SES), exposure to an alcoholic caretaker and antisocial behavior have been touched upon in various studies of genetic loading for alcoholism, they have been addressed neither simultaneously nor systematically. Due to the nature of the sample,

findings from this study are only generalizable to a more impulsive population in which drinking problems are already substantial.

Hypotheses

Hypothesis 1a: There will be a positive correlation between genetic loading for alcoholism and problems with alcohol in adulthood (i.e. early onset of drinking-related difficulties, longer percentage of the lifespan characterized by alcohol problems, high number of lifetime problems with alcohol and heavy intake of alcohol as defined by quantity, frequency and variability of alcohol use).

Hypothesis 1b: Genetic loading for alcoholism will only be positively correlated with the variables listed in Hypothesis 1a among Type 2 alcoholics.

Hypothesis 2: Current environmental press for deviance (as defined by adult SES) will moderate the effects of genetic loading for alcoholism. Higher SES will be related to fewer alcohol problems in respondents.

Hypothesis 3: Environmental press for deviance during childhood (as defined by childhood SES) will moderate the effects of genetic loading for alcoholism. Respondents with higher SES during childhood will have fewer alcohol problems in adulthood than will respondents with lower SES during childhood.

Hypothesis 4: Exposure to an alcoholic environment as a child (e.g. being reared by an alcoholic) will moderate the effects of genetic loading for alcoholism. Respondents who were reared by alcoholic parents will have more alcohol-related difficulties than those who were not.

Hypothesis 5: Childhood antisocial behavior will moderate the effect of genetic loading for alcoholism. Respondents who displayed childhood antisocial behavior will have more alcohol problems than those who did not.

Hypothesis 6a: Adult antisocial behavior will be highly correlated with alcoholism.

Hypothesis 6b: Adult antisocial behavior will be highly correlated with alcoholism among Type 2 but not among Type 1 alcoholics.

Methods

Subjects

This study utilized data from 125 men participating in the Michigan State University Longitudinal Study (Zucker, Noll and Fitzgerald, 1986). The larger research project tracks the development of a group of families with children who are at elevated risk for conduct disorders and alcoholism because of their male gender and because their fathers are alcoholic. These alcoholic men are identified from the population of all males convicted of drunk driving in the mid-Michigan Tri-County area. In order to meet selection criteria, potential respondents must have had a blood alcohol concentration (BAL) of. 15% (150 mg/ 100ml) or higher when arrested (indicating the development of substantial alcohol tolerance) or must have had a BAL of .12% but also have had a history of an additional alcohol-related driving offense. In addition, potential respondents must have a male offspring between three and six years of age and must be residing with the child's mother at initial contact. After being approached by court probation officers about the study, those men who agree to be contacted (currently 77%) are recruited into the M.S.U. Longitudinal Study by project staff (the acceptance rate at this stage is 90%). Subjects are screened using items from the SMAST (Short Michigan Alcohol Screening Test; Selzer, 1975) shortly after recruitment and again later with items from the Diagnostic Interview Schedule (DIS Version III) (Robins, Helzer, Croughan and Ratcliffe, 1980) to verify that they do indeed meet the Feighner research diagnostic criteria (Feighner, Robins, Winokur, Guze et. al., 1972) for either probable or definite alcoholism. Currently, 88% of fathers in the study meet a definite diagnosis. Participating families are compensated at the completion of each wave of data collection; families currently receive $250 for their involvement. For the purposes of this study, it is desirable to have a high degree of variance in the distribution of subjects' current level of alcohol abuse. Therefore, a subset of men has been included who come from families which function as matched controls for the alcoholic

Table 1

Demographic Characteristics of Subjects

	X (s.d.)	Range
Age	30.6 (4.6)	22-47
Years of Education	12.7 (4.0)	7 -20
Number of Marriages	1.2 (0.5)	0 -3
Annual Family Income	$26,983 (16,294)	$2,000-$62,500
Number of Children in Household	2.4 (0.9)	1-5

These families reside in the same census tract as high risk families and are homogeneous with them for age of target male offspring, family economic status and neighborhood; however, neither parent may meet Feighner criteria for alcoholism or for other drug abuse/ dependence (see Table 1 for sample demographics).

Data Collection

Data are collected by trained project staff who are blind to family risk status. Because of the large volume of data collected, a number of contacts with the family are necessary, involving 18 hours of time for each parent and seven hours of time for the target child. These contacts include questionnaire sessions, semi-structured interviews and interactive tasks. Six of the data collection sessions take place in the family home, while two take place on the M.S.U. campus.

Genograms are collected from the family during the last contact of the first assessment phase. Subjects are first asked to produce a family tree which extends back to the grandparental generation and which includes such second degree relatives as aunts, uncles and first cousins. First names, sex, ages and/or birthdates of each relative are recorded if known by the subject. Subjects are then given a list of various physical and psychological disorders (Table 2) and, for each disorder listed, asked if any of the persons recorded on the family tree suffered from it. Any additional information provided by subjects about their family, such as disorders not included on the standardized list, is also recorded.

Measures

A. Alcoholic subtype

In order to determine whether alcoholic subjects fit a Type I or Type II classification, an algorithm (Figure 1) was used which utilizes the criteria laid out by Cloninger's group (Von Knorring et al., 1985; Cloninger, 1987). The measure uses questions from the

Table 2
Possible Family Illnesses [a]

Alcoholism
Allergies or Asthma
Anemia
Arthritis (Rheumatoid Arthritis)
Cancer/ Leukemia
Color Blindness
Depression
Diabetes
Drug Abuse
Emphysema
Epilepsy/ Convulsion Disorder
Gout
Heart Problems/ Heart Disease
High Blood Pressure (Hypertension)
Hyperactivity
Kidney/ Bladder Problems
Learning Disabilities
Liver Disease
Manic Depressive Illness (Bipolar Disorder)
Nervous Breakdown
Schizophrenia
Stroke
Suicide
Tuberculosis
Tumor
Other Illness

[a] List given to respondents during the genogram interview

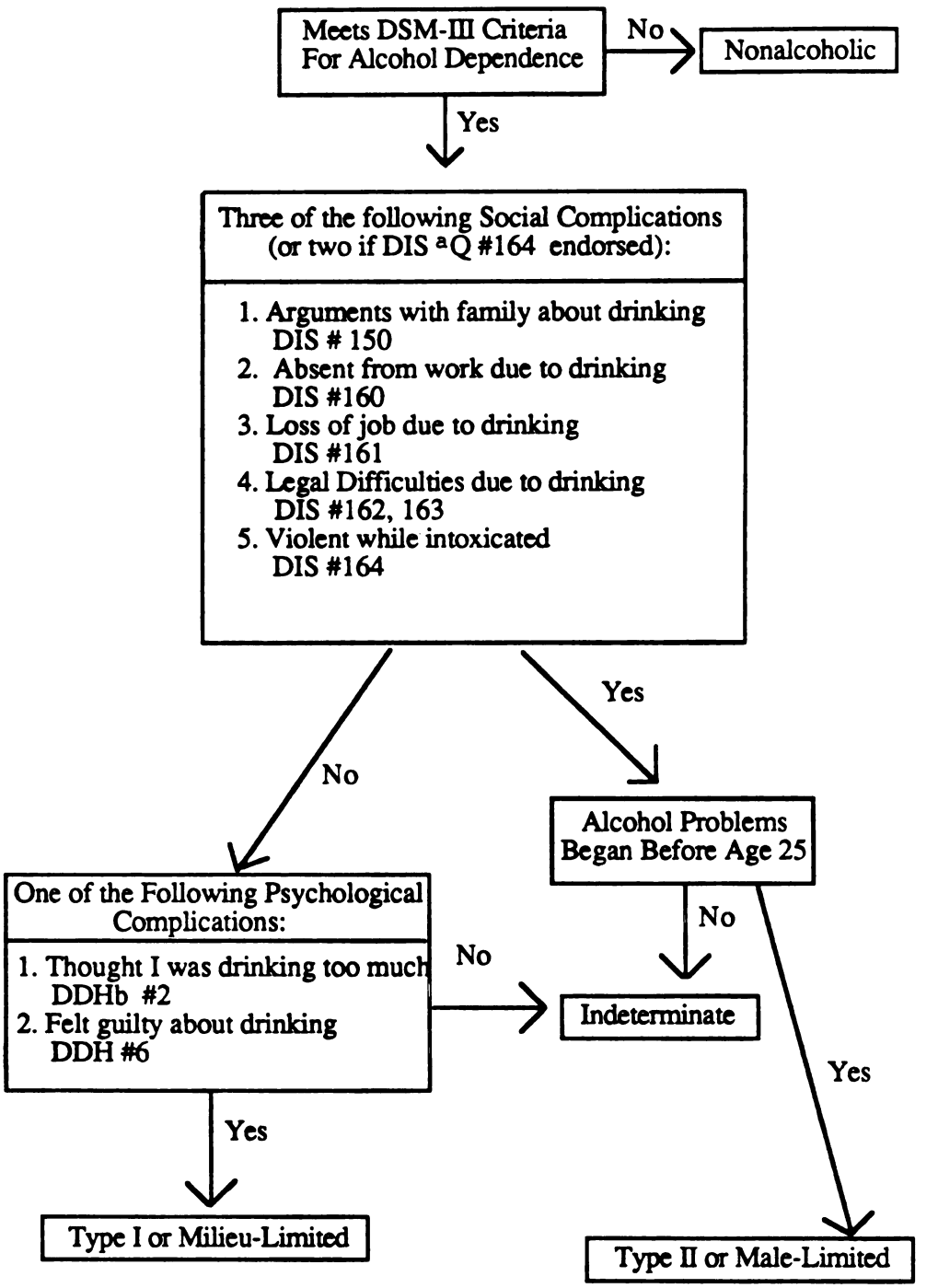


Figure 1

Diagram for Classification of Alcoholic Subtype

[a]DIS #s refer to questions from the Diagnostic Interview Schedule-III
[b]DDH #s refer to items from Drinking and Drug History

DIS- Version III (Robins et al., 1980) and the Drinking and Drug History (Zucker and Noll, 1980) to determine alcoholic subtype. Both these instruments provide information about problems associated with alcohol use. Subjects who met Feighner criteria for probable or definite alcoholism, whose drinking had not incurred social consequences (such as loss of a job) and who had suffered psychological distress over their drinking were coded as Type I alcoholics. Subjects who met Feighner criteria for probable or definite alcoholism, whose drinking began before age 25 and whose drinking had incurred social complications were coded as Type II alcoholics.

B. Drinking measures

1. Lifetime Alcohol Problem Score (LAPS)

In order to determine degree of alcohol related difficulty over the lifecourse, the LAPS (Zucker, 1989) was used. Information from which LAPS is determined is provided by the DIS, Drinking and Drug History and SMAST. LAPS is a composite score derived from three different components: age of first drunkeness, variety of alcohol problems and life percent involving alcohol problems These three subscores are standardized and are calculated as 1) the squared reciprocal of age at which respondent first reported being drunk 2) the number of areas in which drinking problems are reported and 3) the number of years between respondents' first drinking problem and most recent drinking problem multiplied by the squared reciprocal of his age. The measure effectively distinguishes between alcoholics and non-alcoholics and is moderately to strongly correlated with a range of external measures of alcohol-related difficulty.

2. Current Alcohol Consumption

To determine quantity, frequency and variability of drinking, data from the Drinking and Drug History (Zucker and Noll, 1980) was utilized. This self-report

questionnaire assesses amount of alcohol intake in the past six months, using an extended version of the standardized survey questions developed by Cahalan, Cisin and Crossley (1969). Data are coded for quantity-variability and frequency of alcohol consumption, where frequency is a yearly figure based upon number of drinking episodes in the past six months (doubled) and quantity-variability taps both the modal amount of alcohol consumed on any given drinking occasion and the maximum amount consumed when there is variation in consumption. The measure used is a revised version of Cahalan, Cisin and Crossley's Alcohol Consumption Index: the QFV-R (Zucker and Davies, 1989), which provides a more extended range for the measure. Alcoholics who are not currently drinking are not included in computations that involve either of the current consumption measures.

C. Genograms

The measure used to obtain information on alcoholism in the families of subjects is the genogram. The genogram utilizes an interview method known as the family history method, where subjects provide data on psychiatric and physical disorders in other family members. (Thompson, Orvaschel, Prusoff and Kidd, 1982). Several studies have investigated the reliability and validity of the family history method. O'Malley, Carey and Maisto (1986) compared young adults' reports of alcohol use and alcohol-related problems in their parents to the parents' self-report; the two were found to be highly correlated (e.g. the Pearson correlation between students' and fathers' estimates of average monthly consumption was .72). Thompson et al. (1982) compared subjects' reports of various psychiatric illnesses in their relatives to diagnoses made by psychiatrists during personal interviews. They found that the family history method generated few false positives (specificity = .96) for alcoholism, but that subjects often classified alcoholic relatives as unaffected, producing many false negatives (sensitivity = .57). Offspring were found to produce the most accurate reports of illness, as compared to spouses and parents.

Thompson et al. concluded that positive diagnoses generated by the family history method are highly likely to be accurate, but that the true incidence of alcoholism in subjects' relatives will be underestimated.

1. Familial expression of alcoholism (FEA): an index of genetic loading

The FEA score is not a true measure of genetic loading for alcoholism, as it includes data from respondents' siblings (who contribute no genetic material to the respondent). Rather, FEA indexes respondents' genetic loading for alcoholism by reflecting the density of alcoholism in respondents' families as well as the degree of relatedness of these alcoholic family members to respondents. In order to assign each subject a FEA score, alcoholic family members were identified by using the subjects' genograms. Although genograms provided data on cousins, only data about parents, siblings, grandparents, aunts and uncles were used in the analyses, since it became clear that in this data set, subjects' familiarity with more distant relatives was insufficient to allow them to accurately label these relatives as alcoholic or not.

Next, the degree of relatedness between the subject and each alcoholic relative was determined. The degree of relatedness between two family members can be expressed by a value known in human genetics as the coefficient of relationship (Figure 2). Once coefficients of relationship were determined for the subject and each alcoholic relative, genetic loading scores were calculated by 1) within each generation, summing the coefficients of relationship for all alcoholic relatives 2) multiplying this sum by the ratio of alcoholics in each generation to the total number of family members in that generation and 3) summing the subscores across generations. A sample calculation of FEA is shown in Figure 3.

D. Antisocial behavior

The NIMH Diagnostic Interview Schedule (DIS-Version III) (Robins et al., 1980) was used to measure childhood and adult antisocial behavior. The DIS is a semi-

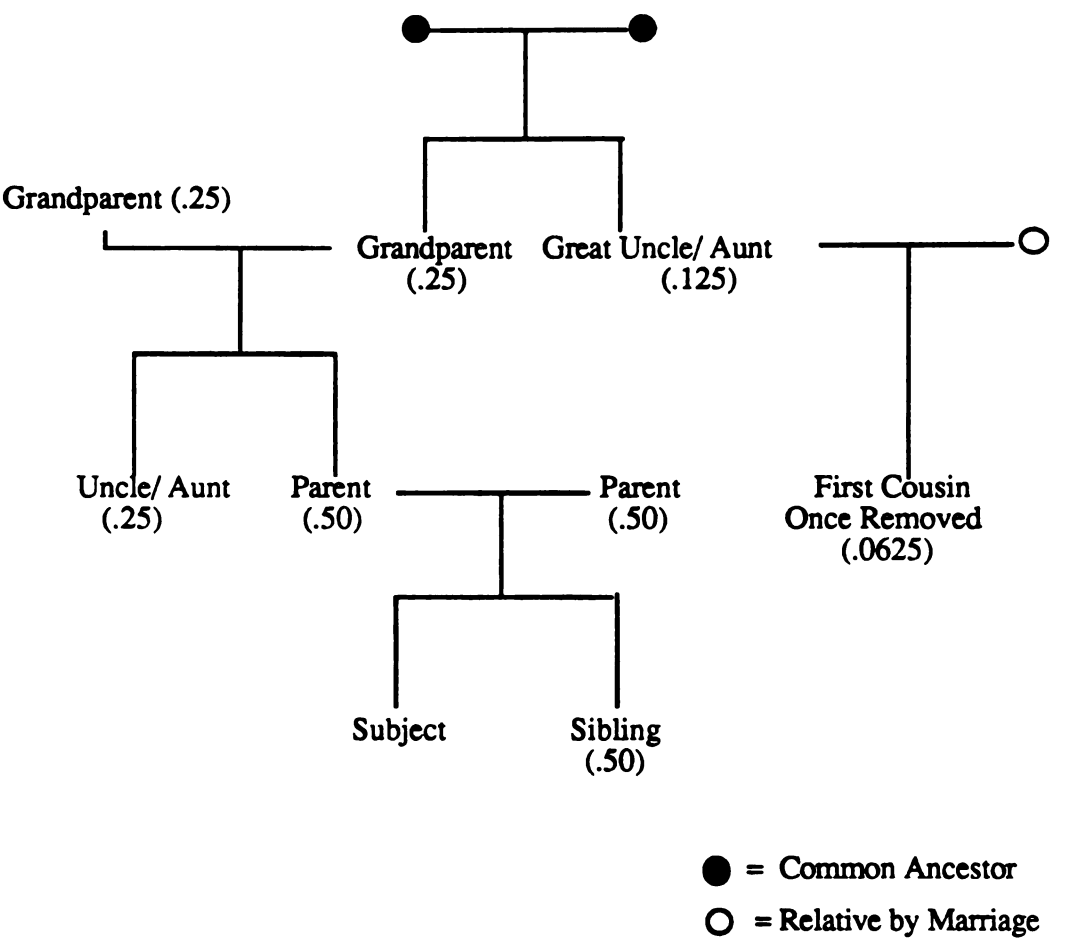
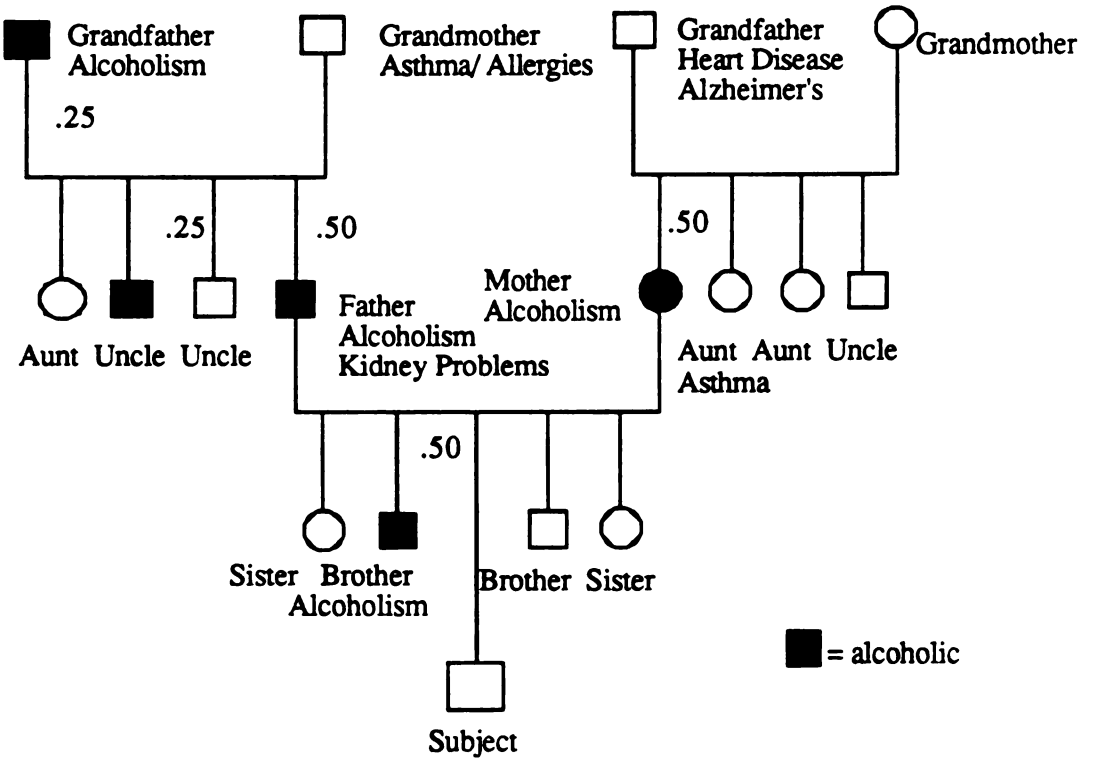


Figure 2
Coefficient of Relationship For Hypothetical Subject and His Extended Family



Step 1: within each generation, sum the coefficients of relationship for all alcoholic relatives

G1: .25 + 0 = .25
G2: .25 + .50 + .50 = 1.25
G3: .50 + 0 = .50

Step 2: multiply this sum by the ratio of alcoholics in each generation to the total number of family members in that generation

G1: (.25).25 = .06
G2: (1.25).38 = .48
G3: (.50) .20 = .10

Step 3: sum subscores across generations

FEA = .06 + .48 + .10 =.64

Figure 3
Sample Calculation: Family Expression of Alcoholism

structured interview developed for use in a multisite study of rates of mental disorder in the general population (Helzer, Robins, McEvoy, Spitnagel, Stolzmann, Farmer and Brockington, 1985). It was designed to make diagnoses by three systems: DSM-III, the Feighner criteria and Research Diagnostic Criteria (Robins, Helzer, Croughan and Ratcliff, 1980). Inter-rater reliability of the DIS has been reported to be high (k=.94) (Hesselbrook, Stabenau, Hesselbrook, Mirkin and Meyer, 1982), as has its test-retest reliability (Wittchen, Burke, Semler, Pfister, Cranach and Zandig, 1989). Validity of the DIS has been tested by examining its concordance with the Schedule for Affective Disorders and Schizophrenia-Lifetime (Endicott and Spitzer, 1978); agreement across diagnoses was found to be quite high (k=.80) (Hesselbrook et al., 1982). The DIS was used to obtain a count of the number of DSM-III symptoms of child and adult antisocial behavior experienced by each subject.

E. Socioeconomic Status (SES)

Information used to rate socioeconomic status comes from the Demographic Questionnaire (Zucker and Noll,1980). Childhood and adulthood SES of subjects was calculated using the Duncan TSE12 Socioeconomic Index (Stevens and Featherman, 1981), an occupationally-based measure of social prestige. Significant evidence exists in the sociological literature to suggest that occupation, not income or education, is the optimal indicator of SES and that the perceived prestige of an occupation best captures its underlying socioeconomic dimension (Mueller and Parcel, 1981).

F. Environmental Exposure to Alcoholism

To assess degree of exposure to an alcoholic environment (e.g. being reared by an alcoholic parent or stepparent) as a child, the Demographic Questionnaire and genograms were used. The Demographic Questionnaire identified people involved in

raising respondents, while genogram data provided a means for assessing drinking status of subjects' parents and/or other primary caretakers. Degree of exposure to an alcoholic environment was rated on a scale which assigned one point for each alcoholic involved in raising the respondent (e.g. parents, stepparents, grandparents). An additional point was added for any *pairing* of alcoholic caretakers (e.g. two alcoholic parents). Thus, a respondent raised by an alcoholic mother would receive a score of one, whereas a respondent raised by an alcoholic father and an alcoholic stepmother would receive a score of three. Additional points were added for a pairing because of the potentiating effect of having two caretakers who both agree about, rather than have conflict over, heavy consumption (Reider, Zucker, Maguin, Noll and Fitzgerald, 1989).

Results

Relationship Between Family Expression of Alcoholism and Drinking Variables

Overall Relationships

Pearson product-moment correlations between family expression of alcoholism and alcohol involvement variables for the entire sample of men (including non-alcoholics) are presented in Table 3. Correlations were significant between FEA and LAPS (r=.32, p<.001), age of first drunkeness (r=-.28, p<.01), lifetime variety of alcohol problems (r=.33, p<.001) and frequency of alcohol consumption in the last six months (r=.26, p<.01).

Type1 vs. Type 2 Alcoholics

Alcoholic males in the sample were further classified as either Type 1 or Type 2. Some of these men (14%) were not codable as either type; they were excluded from these analyses. Inspection of scatterplots of FEA and LAPS revealed distributions for Type 1s and 2s which were similar to that of the overall sample of men; no significant skewness was found, justifying continued analyses of these two subtypes.

The two types of alcoholics were found to differ on a number of variables which assess life difficulty. As shown in Table 4, t-tests demonstrated that Type 2 alcoholics had significantly lower socioeconomic status (t= 2.58, p<.01). They also reported significantly higher levels of child antisocial behavior (t=-5.86, p<.0001) and adult antisocial behavior (t=-6.43, p<.0001) than did Type 1 alcoholics. Moreover, they experienced significantly more separations/ divorces from their partners (t=-4.23, p<.0001). Type 2 alcoholics also scored significantly higher on Lifetime Alcohol Problems Score (t=-3.28, p < .002). However, the two groups did not differ significantly on consumption variables such as frequency of drinking in the past six months or quantity-variability of drinking in the

Table 3
Correlation Between Family Expression of Alcoholism and Alcohol Involvement for Men.

FEA and Alcohol Problem Indices	(n=124)
LAPS [a]	.32 * *
Age First Drunk [b]	-.28 *
Variety [c]	.33 * *
Life Percent [d]	.07
FEA and Alcohol Consumption Indices	**(n=100)**
Quantity-Variability	.16
Frequency	.26 *

Note. Only current drinkers are included for the correlations involving consumption indices.

[a] Lifetime Alcohol Problems Score
[b] Age of First Drunkeness
[c] Lifetime Variety of Alcohol Problems
[d] Life Percent Involving Alcohol Problems
[e] Quantity-Variability of Alcohol Consumption
[f] Frequency of Alcohol Consumption

* $p < .01$ * * $p < .001$

Table 4
Differences Between Type 1 (n=25) and Type 2 (n=60) Alcoholics on Demographic/ Life Difficulty Variables.

	X (s.d.)		t	r_{pb}
	Type 1	Type 2		
Socioeconomic Status (Duncan TSE12)	35.5 (16.4)	26.2 (12.0)	2.58 *	.50
Number of Child Antisocial Behavior Symptoms from DIS	1.7 (1.5)	4.4 (2.8)	-5.86 * * *	.69
Number of Adult Antisocial Behavior Symptoms from DIS	2.5 (1.0)	4.4 (1.7)	-6.43 * * *	.71
Number of Separations/ Divorces from Partner(s)	.9 (1.0)	2.2 (1.8)	-4.23 * * *	.61
Lifetime Alcohol Problems Score	9.8 (1.4)	11.2 (2.0)	-3.28 * *	.55
QFV-R Frequency Classification	6.2 (2.6)	5.9 (2.8)	.54	.26
QFV-R Quantity-Variability Classification	13.4 (6.7)	15.0 (6.9)	-.84	.32
Beck Depression Inventory Score	2.6 (2.9)	3.8 (3.7)	-1.45	.38

Note. Figures in parentheses are standard deviations.

* $p < .05$ * * $p < .01$ * * * $p < .001$

past six months. Current level of depression, as indexed by scores on the short form of the Beck Depression Inventory (Beck and Beck, 1972) also did not differ between the two groups.

The relationship between alcohol involvement variables and family expression of alcoholism for Type 1 and Type 2 alcoholics is presented in Table 5. For Type 2s, family expression of alcoholism was found to be significantly positively correlated to overall LAPS (r=.27, p<.05) and to the LAPS subscore assessing lifetime variety of alcohol problems (r=.30, p< .05). Frequency of alcohol consumption was also significantly related to FEA (r=.38, $p \leq .01$). For Type I alcoholics, all correlations between FEA and drinking variables were found to be non-significant. However, sample size in the Type 1 group is small; moreover, the magnitude of the relationships between FEA and drinking variables was not significantly different in the Type 2 group than in the Type 1 group. Therefore, initial analyses do not support the hypothesis that drinking difficulties are more heritable among Type 2 alcoholics.

Because the FEA measure of genetic loading includes the contribution of alcoholic parents, it is confounded with the environmental effects of being reared by an alcoholic caretaker. In order to estimate the relationship between FEA and alcohol involvement variables while adjusting for the effects of being reared by an alcoholic caretaker, partial correlations were also calculated; alcoholic rearing environment was used as the control variable.

Table 6 shows the correlations between FEA and the alcohol involvement variables after the effects of being reared by an alcoholic were statistically controlled. For Type 2 alcoholics, the correlation between family expression of alcoholism and LAPS remained significant (r=.28, p<.05), and virtually identical, as did the correlations between family expression of alcoholism and lifetime variety of alcohol problems (r= .28, p< .05) and between FEA and frequency of alcohol consumption (r= .36, p< .05). FEA and age of first drunkeness were also found to be significantly negatively correlated (r = -.27,

Table 5
Correlation Between Family Expression of Alcoholism and Alcohol Involvement for Type 1 Versus Type 2 Alcoholics

	Type 1	Type 2
FEA and Alcohol Problem Indices	(n=25)	(n=60)
LAPS [a]	.18	.27 *
Age First Drunk [b]	-.33	-.23
Variety [c]	.27	.30 *
Life Percent [d]	-.18	-.01
FEA and Alcohol Consumption Indices	(n=20)	(n=42)
Quantity-Variability	-.14	.26
Frequency	.03	.38 * *

Note. Only current drinkers are included for the correlations involving consumption indices.

[a] Lifetime Alcohol Problems Score
[b] Age of First Drunkeness
[c] Lifetime Variety of Alcohol Problems
[d] Life Percent Involving Alcohol Problems
[e] Quantity-Variability of Alcohol Consumption
[f] Frequency of Alcohol Consumption

* $p < .05$ * * $p \leq .01$

Table 6
Correlation Between Family Expression of Alcoholism and Alcohol Involvement for Type 1 Versus Type 2 Alcoholics After Controlling for Parental Alcoholism in the Rearing Environment.

	Type 1	Type 2
FEA and Alcohol Problem Indices	(n=25)	(n=60)
LAPS [a]	-.21[+]	.28 * [+]
Age First Drunk [b]	.13	-.27 *
Variety [c]	-.01	.28 *
Life Percent [d]	-.25	.02
FEA and Alcohol Consumption Indices	(n=20)	(n=42)
Quantity-Variability	-.35	.17
Frequency	-.15	.36 *

Note. Only current drinkers are included for the correlations involving consumption indices.

[a] Lifetime Alcohol Problems Score
[b] Age of First Drunkeness
[c] Lifetime Variety of Alcohol Problems
[d] Life Percent Involving Alcohol Problems
[e] Quantity-Variability of Alcohol Consumption
[f] Frequency of Alcohol Consumption

* $p < .05$

[+] coefficients with this superscript differ significantly from each other ($p < .05$)

$p<.05$). Therefore, these relationships are unaffected by rearing environment. In contrast, for Type 1 alcoholics correlations between FEA and drinking variables remained non-significant, but the direction of the relationship was now found to be opposite from that originally observed. In addition, a significant difference was now found in the magnitude of the correlation between FEA and LAPS for Type 1 and Type 2 alcoholics ($z= 2.00$, $p< .05$). The fact that partialling out the shared variance between FEA and alcoholic rearing environment differentially affected the relationship between FEA and LAPS for Type 1s versus Type 2s is consistent with findings that a significant difference ($z= 2.14$, $p < .05$) existed in the magnitude of the correlation between FEA and rearing environment for Type 1s ($r=.39$, $p < .01$) and Type 2s ($r=.74$, $p < .01$).

Relationship between Lifetime Alcohol Problems Score and Other Life Difficulties

Correlations between life difficulty variables for Type 2 alcoholics are presented in Table 7 (figures below the diagonal). For these men, LAPS was found to be significantly positively correlated with childhood antisocial behavior ($r=.39$, $p< .01$) and adult antisocial behavior ($r= .41$, $p<.01$) as well as with FEA ($r=.27$, $p<.05$). Childhood antisocial behavior was also positively correlated with adult antisocial behavior ($r=.31$, $p<.05$). No significant relationship between LAPS and socioeconomic status was found. However, childhood antisocial behavior was found to be negatively correlated with adult SES ($r= -.32$, $p<.05$). Adult antisocial behavior was also negatively correlated with adult SES ($r= -.36$, $p<.01$).

Table 7 also shows the relationships between life difficulty variables for Type 1 alcoholics (figures above the diagonal). Probably as a result of low power, few correlations were significant. Patterns were also different from those found in the Type 2 group. For Type 1 men, LAPS was found to be significantly correlated only with being reared in an alcoholic environment ($r=.42$, $p< .05$); neither childhood nor adulthood antisocial

Table 7

Correlations among Life Difficulty Variables for Type 1 (n=25) and Type 2 (n=60) Alcoholic Men.

	CASB[a]	AASB[b]	FEA[c]	CSES[d]	ASES[e]	LAPS[f]	REARENV[g]
CASB		.33	.04	.20	-.15	.03	.17
AASB	.31 *		.23	.04	-.26	.21	.23
FEA	.16	.07		-.17	.14	.19	.74 * *
CSES	.03	-.14	.01		.59 * *	.04	.10
ASES	-.32 *	-.36 * *	-.12	.14		-.28	.03
LAPS	.39 * *	.41 * *	.27 *	.10	-.19		.42 * *
REARENV	-.01	-.19	.39 * *	-.09	-.09	.03	

Note. Correlations above the diagonal are for Type 1 alcoholic men; those below the diagonal are for Type 2 alcoholic men

[a]Number of Child Antisocial Behavior Symptoms from DIS
[b]Number of Adult Antisocial Behavior Symptoms from DIS
[c]Family Expression of Alcoholism
[d]Child Socioeconomic Status (Duncan Socioeconomic Index)
[e]Adult Socioeconomic Status (Duncan Socioeconomic Index)
[f]Lifetime Alcohol Problem Score
[g]Alcoholic Rearing Environment

* $p < .05$ * * $p < .01$

behavior were significantly correlated with LAPS. Childhood socioeconomic status was positively correlated with adult socioeconomic status (r= .59, p<.01).

Path Analysis

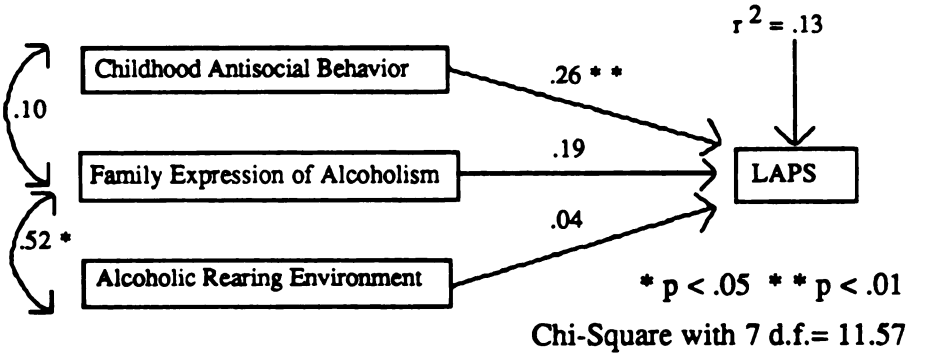
The analyses presented so far clearly point to the appropriateness of a developmental conceptualization of more than one type of alcoholism, each with its separate causal chain. In order to test this statistically, a stacked LISREL (Joreskog and Sorbom,1978) analysis was performed. A stacked group analysis allows a statistical test of the appropriateness of using different path models for two or more mutually exclusive groups. A stacked LISREL model is first estimated with effect coefficients constrained to be equal between groups (hereafter called the combined model); the model is then reestimated with effect coefficients allowed to vary for groups (hereafter called separate models). The difference chi-square for combined versus separate models then provides a test of the goodness of fit when models for the two groups are allowed to differ.

The stacked groups analysis involved three of the most distal predictors of lifetime alcohol problems: family expression of alcoholism, alcoholic rearing environment and childhood antisocial behavior. In the combined model, childhood antisocial behavior, FEA and alcoholic rearing environment were all used as predictors of LAPS. For the separate models, alcoholic rearing environment was used to predict LAPS among Type 1 alcoholics and FEA and childhood antisocial behavior were used to predict LAPS among Type 2 alcoholics; these submodels were chosen based upon preliminary LISREL analyses (see Figure 6, Appendix A). LAPS was selected as the dependent variable for these analyses rather than a dummy variable coded as alcoholic /nonalcoholic because it more adequately characterizes the variation in extent of alcohol problems than does a binary characterization schema; in addition, the score is highly correlated with alcohol dependence /non-dependence.

Figure 4 presents the results of this LISREL analysis. The difference chi-square for combined versus separate models for Type 1 and Type 2 alcoholics is significant at the .05 level, indicating that the data fits significantly better when different models are allowed. Moreover, the combined model explains only 13 percent of the variance in LAPS, while the separate models respectively account for 18 percent and 20 percent of the variance in LAPS. Type 1 alcoholism is best predicted by alcoholic rearing environment while Type 2 alcoholism is predicted by childhood antisocial behavior and FEA.

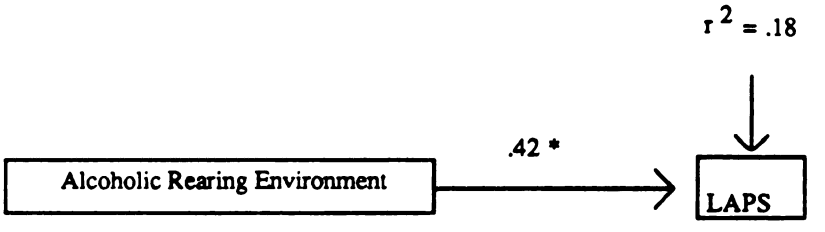
Because of small sample size in the Type 1 group, few life difficulty variables are significantly related. Thus, the unitary path presented in Figure 4 remains the best model of Type 1 alcoholism; alcoholic rearing environment remains the only significant predictor of later alcohol problems for this data set. An expanded path model for Type 2 alcoholics which best fits the data is shown in Figure 5. The model shows that childhood antisocial behavior has both a direct effect on alcoholism and an indirect effect through its relationship to adult antisocial behavior. FEA misses significance at the .05 level in the path model, but is retained because of its significance in the univariate correlations and because it remains a trend ($p < .10$).

Combined model for Type 1 and Type 2 Alcoholics

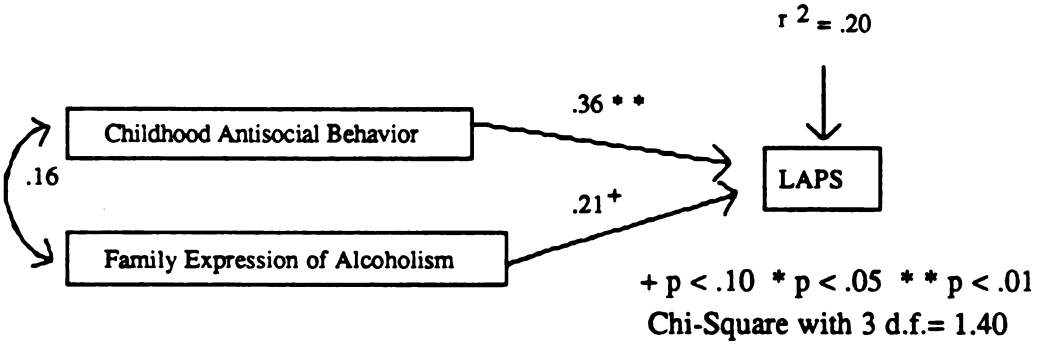


Separate models for Type 1 and Type 2 Alcoholics

Type 1



Type 2



Difference Chi-Square for combined versus separate models: X = 10.17, p < .05

Figure 4
Comparison of Path Models for Type 1 and Type 2 Alcoholics

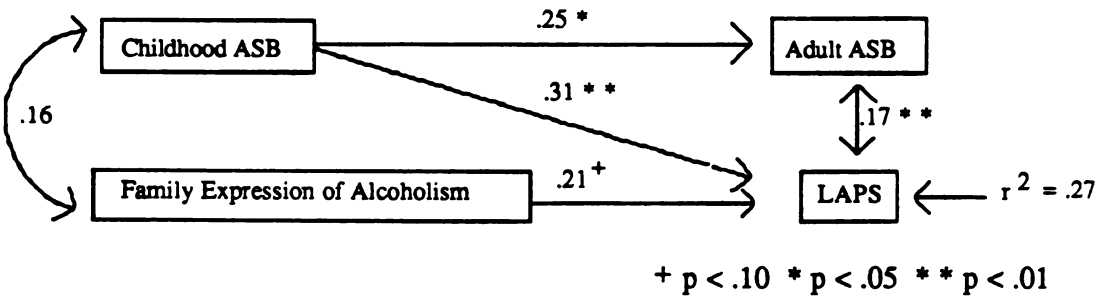


Figure 5

Extended Path Model for Type 2 Alcoholics

Discussion

The research literature clearly points to a genetically-based diathesis in the etiology of some forms of alcoholism. Although the Washington University group has begun to addressthe question of how inherited factors might affect the developmental course of alcoholism (Cloninger, 1987), the role of other variables which might systematically affect the relationship of genetic loading for alcoholism to alcohol related problems (e.g. onset, density, severity) is still in its infancy. In addition, the role of other variables which might systematically affect the relationship of genetic loading for alcoholism to alcohol-related problems has not yet been systematically addressed. The present study replicates those earlier studies which implicate genetic factors in the etiology of alcoholism. In addition, it specifies more clearly the domains of possible genetic influence during the lifespan and the effects of variables such as socioeconomic status and antisocial behavior as they interact with genetic loading for alcoholism.

Hypothesis 1a predicted that a positive correlation would be found between genetic loading for alcoholism and problems with alcohol in adulthood. Supporting this hypothesis, significant positive correlations were found for the overall sample between family expression of alcoholism and LAPS. This finding reaffirms the role of genetic vulnerability in later alcohol-related difficulties. The result also parallels findings from adoption and twin studies. In addition, the present findings more clearly link respondents' alcohol problems to those in the respondents' extended families in addition to those of their parents.

Two of the LAPS subscores, age of first drunkeness and lifetime variety of alcohol problems, were also found to be positively correlated with family expression of alcoholism. These findings imply that genetic factors play a role in determining both the onset of alcoholism, that is, the age at which alcohol use first becomes problematic, and the severity, or pervasiveness, of alcohol problems during the lifespan. Inherited factors

appear to affect the course of drinking problems over the lifespan by providing an additional push toward earlier alcohol abuse; earlier use likely provides more opportunity for a wide range of alcohol problems to develop. This finding is in contrast to those of Vaillant (1989), who has suggested that age of onset of alcoholism is uncorrelated with genetic load and is driven purely by environmental factors.

The correlation between the third LAPS subscore, life percent, and FEA was low order and non-significant, lending no support to Hypothesis 1a. This subscore quantifies the extent of invasiveness of alcohol problems; one possible explanation for the non-significant correlation is that the length of time that alcohol-related difficulties persist is impacted by numerous environmental factors which act to control drinking, such as family pressure to stop drinking and court-ordered involvement in rehabilitation programs after drunk driving arrests. These factors would be anticipated to affect duration of drinking problems much more directly than would genetic loading for alcoholism.

FEA was also found to be positively correlated with one of the alcohol consumption indices: frequency. Earlier studies, such as those of Kaij (1960), Partanen (1966) and Kaprio et al. (1987) have indicated that there are genetic effects upon such drinking variables as frequency and density of alcohol consumption. However, family expression of alcoholism is a lifetime measure whereas the frequency variable indexes current alcohol consumption; consumption might be expected to fluctuate over time and therefore to be uncorrelated with FEA. Findings from the present study demonstrate that family expression of alcoholism can in fact predict both current and lifetime drinking problems.

Hypothesis 1b predicted that once alcoholic males in the sample were divided by subtype, genetic loading for alcoholism would only be positively correlated with alcohol problem and consumption variables among Type 2 alcoholics. Before completing the genetic analyses, comparisons were made between the two groups on other life difficulty variables in order to confirm that Type 1s and Type 2s were distinctive enough and that further analyses were warranted.

Comparisons between the two groups demonstrated that both types currently experienced similar rates of current alcohol consumption. However, Type 2 alcoholics reported significantly higher scores on LAPS. As expected, Type 2 alcoholics also reported significantly higher levels of childhood and adult antisocial behavior, such as difficulty maintaining stable relationships with partners. In addition, they had significantly lower adult (i.e. achieved) socioeconomic status, although no differences were noted in childhood SES (an index of social class origins).

These data indicate that the alcoholic sample may seem homogeneous when characterized by way of alcohol consumption, but that Type 1 and Type 2 alcoholics are actually quite different in their life experiences. The data suggest that the course of Type 1 alcoholism is more benign, since the consequences of drinking do not invade as much of the lifespace. The distinctiveness of Type 1s and 2s supports the concept of different etiological pathways into alcoholism, which is addressed in more detail by the LISREL analyses.

Significant positive correlations were found for Type 2 alcoholics between LAPS, the LAPS subscore lifetime variety of alcohol problems, current frequency of alcohol consumption and family expression of alcoholism. Although all correlations between FEA and drinking variables were found to be non-significant for Type 1 alcoholics, power was low in this group due to small sample size. Thus, the possibility that family expression of alcoholism did in fact predict later alcohol problems among Type 1s cannot be ruled out. In addition, the magnitude of the relationship between FEA and drinking variables was not significantly different between the Type 1 and Type 2 groups. Therefore, initial findings provided no support for Hypothesis 1b, because they do not confirm that alcohol-related problems are more heritable among Type 2 alcoholics. However, the FEA measure of genetic loading is confounded by the effects of being reared by an alcoholic caretaker; because the Cloninger et al. (1981) data indicate that post-natal environment differentially

affects Type 1 and Type 2 alcoholics, analyses were rerun while adjusting for the effect of being reared in an alcoholic environment.

Hypothesis 1b was now supported by the data; significant differences were now found in the magnitude of the correlation between family expression of alcoholism and LAPS for Type 1 and Type 2s. These findings suggest that the magnitude of the initial correlation between LAPS and family expression of alcoholism for Type 1 alcoholics is accounted for by the inclusion of alcoholic biological parents, who for the most part reared the respondent, as well as contributing to his genetic makeup. Once the confounding effects of rearing environment are controlled, the data clearly indicate that genetic loading for alcoholism plays a much more important role in the development of alcoholism among Type 2 than among Type 1 alcoholics. On the other hand, removing environmental effects from the correlation between LAPS and FEA clearly shows the importance of alcoholic rearing environment in the etiology of Type 1 alcoholism.

The differences which appear between Type 1 and Type 2 alcoholics on the genetic-environmental continuum are similar to distinctions made by Zucker (1987a) in his discussion of alcoholic typologies. He suggests that antisocial alcoholism (i.e. Type 2) has a heavy genetic diathesis; moreover, he proposes that a history of socialization to aggression, not necessarily to excessive alcohol consumption, is a necessary etiologic factor. On the other hand, in developmentally cumulative alcoholism (i.e. Type 1) any potential genetic diathesis is proposed to be environmentally mediated; it is the role of socialization involving exposure to an alcoholic parent and leading the future alcoholic to regard alcohol use as a coping mechanism which is seen as vital.

Influence of Other Variables on the Developmental Course of Alcoholism

Hypotheses 2 through 6 predicted that adult socioeconomic status, childhood socioeconomic status, alcoholic rearing environment, childhood antisocial behavior and

adult antisocial behavior would also affect the extent of lifetime alcohol-related difficulties. These hypotheses were tested through univariate correlations and, in a more complex manner, through path analysis.

Univariate correlations lend support to both Hypothesis 5 and Hypothesis 6b. Child and adult antisocial behavior were found to be significantly related to LAPS among Type 2 alcoholics but not among Type 1s. At one level, this is yet another confirmation of widespread research findings which suggest that early onset antisocial behavior which continues over the lifespan is systematically correlated with alcohol-related difficulty in adulthood (McCord and McCord, 1962; Zucker and Gomberg, 1986). However, the fact that the correlation between childhood antisocial behavior and LAPS is close to zero for Type 1 alcoholics suggests that early delinquency is only one route into alcohol problems. Other routes may a) not be as dependent upon childhood antisociality as they are upon other childhood influences b) be susceptible to developmentally later influences or c) involve some combination of these two sets of contributory effects. Further research should investigate these other potential sources of effect.

Data from the univariate correlations also supported Hypothesis 4. Rearing by an alcoholic caretaker was found to be significantly correlated with LAPS among Type 1 alcoholics, indicating, as previously discussed, the importance for this group of early exposure to alcohol abuse.

No support was found in the univariate analyses for a direct effect of either adult socioeconomic status or childhood socioeconomic status on lifetime alcohol problems (Hypotheses 2 and 3). It should be noted that the general lack of significant relationships between variables in the Type 1 group may well be due to small sample size, resulting in low power. An alternative explanation, that variables which are etiologic in Type 1 alcoholism were not adequately addressed in this study, will be discussed later.

Both analyses of the relationship between family expression of alcoholism and drinking variables, and those involving LAPS and the non-drinking variables which might

have a potentiating effect on genetic loading for alcoholism, suggest that different factors play a role in the development of alcoholism for Type 1s and 2s. Supporting the hypothesis that Type 1s and 2s are developmentally distinct, a stacked groups LISREL analysis confirmed that in order to provide the best fit for the data, different models should be used for the two groups. Out of four potential distal predictors of later alcohol problems (i.e. family expression of alcoholism, childhood antisocial behavior, childhood SES and alcoholic rearing environment), family expression of alcoholism and childhood antisocial behavior are precursive to alcohol difficulties for Type 2 alcoholics. Exposure to an alcoholic rearing environment is predictive of later alcohol problems for Type 1s.

For Type 2 alcoholics, the preliminary path model can be expanded to included more proximal predictors of alcohol difficulties. In this expanded path model, both adult antisocial behavior and LAPS were used as dependent variables. This approach was taken because the direction of causality between antisocial behavior and LAPS was not indicated by the LISREL analyses and also because other research in this area has failed to specify which of these two variables is precursive (Cadoret et al., 1985). It is likely, in fact, that antisocial behavior and alcoholism act synergistically, each driving the other. The expanded path model also shows that childhood antisocial behavior continues to have a direct effect on lifetime alcohol problems, but also influences these problems indirectly through adult ASB. Thus, delinquent behavior as a child is predictive of antisociality in adulthood, which as discussed above, drives alcohol problems.

The expanded path model for Type 2s indicates that family expression of alcoholism makes a seperate contribution to LAPS which is not mediated by other variables. It also shows that FEA makes a genetic contribution which is specific to alcoholism, as FEA does not predict antisocial behavior. Therefore, although this data set does not elaborate the mechanism through which genetic predisposition for alcoholism is expressed, it indicates that the mechanism may well be alcoholism-specific for this subtype.

Therefore, path analysis provides further confimation of Hypotheses 5 and 6. Type 2 alcoholism, then, can be explained as a function of antisocial behavior over the life course and genetic loading for alcoholism. These findings extend recent work by Cloninger, Sigvardsson, and Bohman (1988) which suggests that in very young adolescents, personality traits associated with antisocial behavior are predictive of Type 2, but not Type 1 alcoholism in adulthood.

For Type 1 alcoholics, exposure to an alcoholic rearing environment remains the only variable which predicts later problems with alcohol use. One possible reason (besides low power) why the data may not be capturing the developmental course of Type 1 alcoholism is that variables etiologic to this form of alcoholism may not have been included in these analyses. Zucker (1987a) suggests that adolescent problem drinking becomes developmentally cumulative alcoholism (i.e. Type 1) when poor career and marital adaptations occur, providing the alcoholic with fewer external resources upon which to draw. Therefore, in order to properly capture the path to alcohol difficulties for Type 1 alcoholics, the nature of Type 1s' intimate relationships and career satisfaction probably need to be assessed.

Future Directions

This study was able to replicate findings by a number of other researchers that genetic loading contributes to the development of alcoholism. However, the nature of the data (i.e. the genograms) used to measure familial alcoholism was somewhat imprecise and possibly of lower reliability. Other familial-genetic researchers, such as Thompson et. al. (1982) have gained access to populations where personal interviews with extended family members can be conducted. Replication of results presented in this study with data

obtained from collateral informants would be another step which should be pursued by investigators who are interested in the developmental role of genetics in alcoholism.

Although many variables which lead to the development of Type 2 alcoholism were identified here, the available data set failed to adequately describe Type 1 alcoholism. Further investigation is necessary to identify those developmental factors which push the Type 1 alcoholic to experience drinking difficulties; intimate relationships in childhood and adulthood as well as career satisfaction may well be fruitful areas for study. Differences in the experience of depression may be another.

It is important to note that results from this study are limited to men. They do not provided any information about the relationship between genetic loading for alcoholism and drinking problems among women, nor about the developmental course of alcoholism in women. Although Cloninger et al. (1987) suggest an etiologic process for alcoholic women which is similar to that of Type 1 men, use of a broader variable domain may well reveal a different path into alcohol problems for females. Future research should also attempt to apply the antisocial alcoholic/ non-antisocial alcoholic dichotomy to women in order to evaluate its possible relationship to an inherited predisposition to alcoholism across both sexes.

Summary

Current etiologic research strongly suggests that genetic vulnerability plays a role in the development of alcoholism. Studies using animal models have demonstrated that strain and line differences exist in the acquistion of alcohol preference, sensitivity, tolerance and dependence. Evidence which impinges more directly on alcoholism in humans comes from family studies. Several sources have shown that alcoholics are significantly more likely than non-alcoholics to have alcoholic parents, siblings and children. Both twin and adoption studies, traditional behavioral genetic tools for analyzing heritability, have also been used to investigate the etiology of alcoholism. Findings from these studies also

indicate that children of alcoholics inherit some factor which puts them at risk for alcoholism.

However, few researchers have related genetic loading for alcoholism to a broad range of drinking-related variables; thus, the issue of precisely what might be inherited remains unclear. In addition, prior studies have only crudely assessed the effect of other factors such as environmental press for deviance, antisocial behavior and exposure to an alcoholic parent as these might moderate genetic vulnerability. Therefore, the present study, which used a population-based sample and a broadly based set of independent variables, was able to more clearly examine these potential contributory effects.

An addition question of significance was the extent to which earlier work on two types of alcoholism (Cloninger's male-limited and milieu-limited types) could be replicated. Should this be possible, the study also planned to model the contributory role of other psychosocial factors than those originally posited by Cloninger's group.

Results of the study confirmed both the effects of genetic loading for alcoholism and the impact of other variables upon alcohol-related difficulties in adulthood. Positive correlations were found between family expression of alcoholism and lifetime alcohol problem score, as well as between FEA and two LAPS subscores: age of first drunkenness and lifetime variety of alcohol problems. Frequency of alcohol use was also found to be positively correlated with family expression of alcoholism.

The role of genetic loading for alcoholism in later alcohol problems was also found to be different among Type 1 and Type 2 alcoholics. For Type 2s, genetic loading for alcoholism contributed strongly to the later development of alcoholism, whereas for Type 1s, being reared in an alcoholic environment, rather than inheritance of a predisposition to alcoholism, was key.

Finally, childhood and adulthood antisocial behavior were found to contribute heavily to the etiology of Type 2 alcoholism; therefore, a history of socialization to aggression, rather than to excessive alcohol consumption, is suggested as an etiologic

factor for this type of alcoholism. For Type 1 alcoholics, no variables other than alcoholic rearing environment were found to be related to adult alcoholism. Thus, the role of socialization involving exposure to an alcoholic caretaker, which leads to heavy use of alcohol as a coping mechanism, was the only etiologic factor identified for Type 1s.

This study replicates findings by a number of researchers which demonstrate that genetic loading contributes to the development of alcoholism; at the same time, it specifies a particular alcoholic subset for whom genetic predisposition to alcoholism is a necessary etiologic factor. In addition, results more clearly link respondents' alcohol problems to those in their extended families. Finally, the study demonstrates the importance of other factors, such as being reared in an alcoholic environment and antisocial behavior, in the etiology of alcoholism. It remains for future research to more fully chart these etiologic pathways.

List of Appendices

Appendix A
Full Models of Distal Predictors of LAPS for Type1s and Type 2s

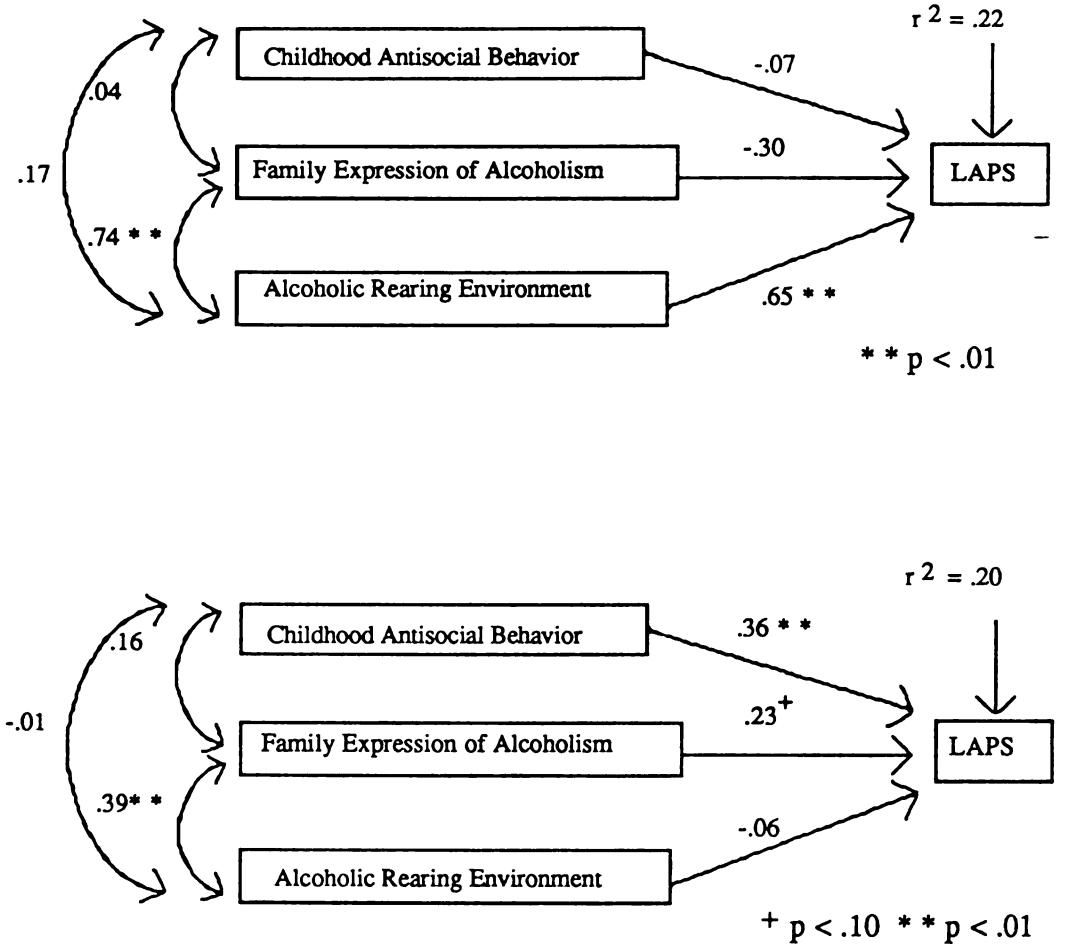


Figure 6
Full Models of Distal Predictors of LAPS for Type 1 and Type 2 Alcoholics

Appendix B
Demographic Questionnaire

DEMO 11/89

Respondent Number: _____
Given By: _____
Date: _____
T1.0
Ans. Chk: _____

MICHIGAN STATE UNIVERSITY
FAMILY PROJECT
East Lansing, Michigan 48824

Background Information

We would like to ask you a few questions about yourself. The questions ask about your life during the time you were growing up as well as now. Please answer all of them as completely as possible. (PLEASE PRINT).

1. What is your date of birth?

_____ _____ _____
MONTH DAY YEAR

2. Where were you born?

_____ _____ _____
CITY/TOWN (COUNTY IF RURAL) STATE COUNTRY (IF NOT U.S.)

3. Where did you live most of the time until you were 18?

_____ _____ _____
CITY/TOWN (COUNTY IF RURAL) STATE COUNTRY (IF NOT U.S.)

4. Until you were 18, about how many times did your family move.

CIRCLE ONE 0 1 2 3 4 5 6 7 or more.

5a. Did you live together with both of your natural parents for most of the time from birth to 18? CIRCLE ONE.

YES (If Yes, go to question 6) NO (If No, go to question 5b)

5b. What was the main reason your parents did not live together with you during that time? CIRCLE ONE

1. Mother died
2. Father died
3. Both parents died
4. Parents divorced or separated
5. Parents never lived together
6. Other (Please explain) _____

_____

5c. Which adult(s) did you live with most of the time from birth to 18? CIRCLE ONE.

2. Mother, but no adult male
3. Father, but no adult female
4. Mother and step-father
5. Father and step-mother
6. Other (Please explain) _____

_____

6. Who was the main wage earner in your home while you were growing up? CHECK ONE

(a) your father ____
(b) your mother ____
(c) someone else ____
What was their relationship to you _____

ABOUT YOUR NATURAL (BIOLOGICAL) FATHER

7a. Where was he born?

_____ _____
STATE COUNTRY (IF NOT U.S.)

ABOUT THE ADULT MALE WHO LIVED WITH YOU MOST OF THE TIME UNTIL YOU WERE 18.
(This could be your natural father, or stepfather, or someone else).

7b. What kind of work did this adult male do (the adult male who lived with you most of the time until you were 18?) That is, what was his occupation? _____

_____

(For example: electrical engineer, machinist, stock clerk, assembly line worker, farmer)

7c. What were his most important activities or duties?

_____

(For example: keep account books, filing, selling cars, operate printing press, finish concrete)

7d. What kind of business or industry was this?

_____

(For example: TV and radio mfg., Retail shoe store, Automobile manufacturing [Oldsmobile], State Labor Dept., Farm work)

7e. What was the highest grade of school he completed?

CIRCLE THE HIGHEST GRADE COMPLETED.

None	0								
Elementary	1	2	3	4	5	6	7	8	
High School	9	10	11	12					
College	1	2	3	4				Degree?	_____
Graduate School	5	6	7	8+				Degree?	_____

AGAIN, A QUESTION ABOUT YOUR NATURAL (BIOLOGICAL) FATHER

7f. How would you describe his primary cultural/ethnic heritage? CIRCLE ONE

(a) White
(b) Black
(c) Hispanic/Hispanic-American
(d) Native American (American Indian)
(e) Asian/Asian American
(f) Other (describe) _____

ABOUT YOUR NATURAL (BIOLOGICAL) MOTHER

8a. Where was she born? _____ _____
STATE COUNTRY--IF NOT U.S.

ABOUT THE ADULT FEMALE WHO LIVED WITH YOU MOST OF THE TIME UNTIL YOU WERE 18.
(This could be your natural mother, or stepmother, or someone else).

8b. What kind of work did this adult female do (the adult female who lived with you most of the time until you were 18?) That is, what was her occupation?

_____

_____
(For example: electrical engineer, file clerk, assembly line worker bookkeeper, sales clerk)

8c. What were her most important activities or duties?

_____
(For example: keep account books, filing, selling clothing, teach fifth graders)

8d. What kind of business or industry was this?

_____
(For example: TV and radio mfg., Retail shoe store, Automobile manufacturing [Oldsmobile], State Labor Dept.)

8e. What was the highest grade of school she completed?

CIRCLE THE HIGHEST GRADE COMPLETED.

None	0								
Elementary	1	2	3	4	5	6	7	8	
High School	9	10	11	12					
College	1	2	3	4			Degree?		_____
Graduate School	5	6	7	8+			Degree?		_____

AGAIN, A QUESTION ABOUT YOUR NATURAL (BIOLOGICAL) MOTHER:

8f. How would you describe her primary cultural/ethnic heritage?

CIRCLE ONE

(a) White
(b) Black
(c) Hispanic/Hispanic-American
(d) Native American (American Indian)
(e) Asian/Asian American
(f) Other (describe) _____

9a. Until you were 18, what religion was practiced in your home most of the time? CIRCLE ONE

1. Protestant
2. Roman Catholic
3. Jewish
4. None, no religion
5. Other religion (please explain) _____

_____

9b. What denomination? (please try to specify fully)_____

_____

9c. Until you were 18, how often did you attend religious services? CIRCLE ONE

1. several times a week
2. about once a week
3. 2-3 times a month
4. once a month or less
5. never

10a. What is your religious preference now? CIRCLE ONE

1. Protestant
2. Roman Catholic
3. Jewish
4. None, no religion
5. Other religion (please explain _____

_____

10b. What denomination? (please try to specify fully) _____

_____

10c. About how often did you attend religious services in the last year? CIRCLE ONE

1. several times a week
2. about once a week
3. 2-3 times a month
4. once a month or less
5. never

10d. Regardless of your attendance at religious services, how religious do you consider yourself to be?

1. not religious at all
2. not very religious
3. fairly religious
4. very religious

11. What was the highest grade you completed? CIRCLE THE HIGHEST GRADE COMPLETED.

None	0							
Elementary	1	2	3	4	5	6	7	8
High School	9	10	11	12				
Post High School								
Voc-Tech School	1	2	3					
College	1	2	3	4			Degree?	_____
Graduate School	5	6	7	8+			Degree?	_____

12a. What kind of work are you doing (what is your occupation)?

_____

_____

(For example: electrical engineer, machinist, stock clerk, assembly line worker, teacher, farmer)

12b. What are your most important activities or duties?

_____

(For example: keep account books, filing, selling cars, operate printing press, finish concrete, teach fifth graders, answer phone).

12c. What kind of business or industry is this?

_____

(For example: TV and radio mfg., Retail shoe store, Automobile manufacturing [Oldsmobile], State Labor Dept., Farm work)

12d. Are you: CHECK ONE

an employee of a PRIVATE company, business or individual for wages, salary, or commission? _____

a GOVERNMENT employee (federal, state, county, or local government? _____

self-employed in OWN business, professional practice, or farm?

own business not incorporated _____

own business incorporated _____

working WITHOUT pay in a family business or farm _____

12e. Approximately what is your present annual <u>family</u> income? CIRCLE ONE

1.	under $4,000	6.	$16,001--$20,000
2.	$ 4,001--$ 7,000	7.	$20,001--$30,000
3.	$ 7,001--$10,000	8.	$30,001--$50,000
4.	$10,001--$13,000	9.	$50,001--$75,000
5.	$13,001--$16,000	10.	over $75,000

13. How many times have you been married? CIRCLE ONE

0 1 2 3 4+

14a. What was the date of your marriage to your (present) spouse?

_____

14b. If married more than once, what was the date of your first marriage? _____

15a. List the children you have had from your present marriage or any previous marriages. Please list all children, starting with the oldest, and include birthdate, sex, and check (✓) if the child lives with you now.

	FIRST NAME ONLY	BIRTHDATE (mo/day/year)	SEX	LIVING WITH YOU NOW	NOT LIVING WITH YOU NOW
1.					
2.					
3.					
4.					
5.					
6.					
7.					
8.					

15b. Now please circle the names of the children you listed in Question 15a above who are from your present marriage. If all are from your present marriage just check a mark here_____.

THANK YOU FOR FILLING OUT THIS QUESTIONNAIRE

Appendix C
Diagnostic Interview Schedule

DIAGNOSTIC INTERVIEW SCHEDULE

DECK 01

Version III (5/81)

Family Number: _____

Given By: _____

Date: _____

Test: PRE PT1 PT2 PT3 Post

Ans. Chk: _____

CODE

1 = no
2 = below cnt
3 = drugs or alc
4 = mod exp
5 = yes

ID CODE

ENTER TIME BEGAN _____ AM PM

AM
PM

08/

1. RECORD SEX AS OBSERVED.

MALE 1
FEMALE 2

13/

2. How old were you on your last birthday?

ENTER AGE: ☐☐

14/

3. What is your birthdate?

MONTH ☐☐
DAY ☐☐
YEAR ☐☐

16/

4. Are you presently married or are you widowed, separated, divorced, or have you never been married?

Married 1
Widowed .....(SKIP TO Q. 6)..... 2
Separated .....(SKIP TO Q. 6)..... 3
Divorced .....(SKIP TO Q. 6)..... 4
Never married ....(SKIP TO Q. 6)..... 5

22/

5. Are you currently living with your (husband/wife)?

No .....(SKIP TO Q. 7)..... 1
Yes .....(SKIP TO Q. 7)..... 5

23/

6. Are you currently living with someone as though you were married?

No 1
Yes 5

24/

IF NEVER MARRIED, GO TO Q. 11

DECK 09

196. Now I'd like to ask about your life as a child. Let's begin with some questions about school. Did you ever repeat a grade? 17/

No .....(SKIP TO Q. 197)....	①
Yes .....(ASK A).....	④

A. Did you get held back more than once? 18/

No, only once	②
Yes, more than once	⑤

197. How were your grades in school—better than average, average, or not so good? 19/

Better than average ..(SKIP TO Q. 198)....	①
Average .....(SKIP TO Q. 198)....	②
Not so good .....(ASK A).....	⑤

A. Did your teachers think you did about as well as you could or did they think you had the ability to do much better? 20/

Did as well as could .....(SKIP TO Q. 198)	②
Could have done much better .(ASK B).....	⑤

B. How old were you when your teachers first felt that way? 21/22/

ENTER AGE & GO TO Q. 198 ☐☐ ⓪ ① ② ③ ④ ⑤ ⑥ ⑦ ⑧ ⑨ / ⓪ ① ② ③ ④ ⑤ ⑥ ⑦ ⑧ ⑨

INTERVIEWER: IF R SAYS "DK": ASK C

C. Do you think it was before you were 15 or later than that? 23/

Under 15 .....(RECORD 01 ABOVE)	
15 or more .....(RECORD 95 ABOVE)	
Still DK .....(RECORD 98 ABOVE)	

198. Did you frequently get into trouble with the teacher or principal for misbehaving in school? (ELEMENTARY, JUNIOR HIGH, OR HIGH SCHOOL) 24/

No .....(SKIP TO Q. 199)....	①
Yes .....(ASK A).....	④

A. How old were you when you first got into trouble for misbehaving in school? 25/26/

ENTER AGE & GO TO Q. 199 ☐☐ ⓪ ① ② ③ ④ ⑤ ⑥ ⑦ ⑧ ⑨ / ⓪ ① ② ③ ④ ⑤ ⑥ ⑦ ⑧ ⑨

INTERVIEWER: IF R SAYS "DK": ASK B

B. Do you think it was before you were 15 or later than that? 27/

Under 15 .....(RECORD 01 ABOVE)	
15 or more .....(RECORD 95 ABOVE)	
Still DK .....(RECORD 98 ABOVE)	

DECK 09

199. Were you ever expelled or suspended from school? (ELEMENTARY, JUNIOR HIGH OR HIGH SCHOOL)

No ..... (SKIP TO Q. 200) .....① 28/
Yes ..... (ASK A) .....④

A. How old were you when you were first expelled or suspended?

ENTER AGE & GO TO Q. 200 ☐☐ ⑥①②③④⑤⑥⑦⑧⑨ ⑩①②③④⑤⑥⑦⑧⑨ 29/

INTERVIEWER: IF R SAYS "DK": ASK B.

B. Do you think it was before you were 15 or later than that?

Under 15 .....(RECORD 01 ABOVE) 31/
15 or more ....(RECORD 95 ABOVE)
Still DK .....(RECORD 98 ABOVE)

200. Did you ever play hooky from school at least twice in one year?

No .....(SKIP TO Q. 201) .....① 32/
Yes ..... (ASK A) .....④

A. Was that only in your last year in school or before that?

Last year only .... (SKIP TO Q. 201).....② 33/
Before last year ...(ASK B AND C).....④

B. Did you play hooky as much as 5 days a year in at least two school years, not counting your last year in school?

No .....① 34/
Yes .....⑤

C. How old were you when you first played hooky?

ENTER AGE & GO TO Q. 201 ☐☐ ⑥①②③④⑤⑥⑦⑧⑨ ⑩①②③④⑤⑥⑦⑧⑨ 35/

INTERVIEWER: IF R SAYS "DK": ASK D

D. Do you think it was before you were 15 or later than that?

Under 15 .....(RECORD 01 ABOVE) 37/
15 or more ....(RECORD 95 ABOVE)
Still DK .....(RECORD 98 ABOVE)

201. Did you ever get into trouble at school for fighting?

No ..... (SKIP TO Q. 202).....① 38/
Yes .....(ASK A) .....④

A. Did that happen more than once?

No .....(SKIP TO Q. 202).....① 39/
Yes .....(ASK B AND C).....④

DECK 09

B. Were you sometimes the one who started the fight?

No ..... ① 40/
Yes ..... ⑤

C. How old were you when you first got into trouble for fighting at school?

ENTER AGE & GO TO Q. 202 ☐☐ ⓪①②③④⑤⑥⑦⑧⑨ 41/

INTERVIEWER: IF R SAYS "DK": ASK D

D. Do you think it was before you were 15 or later than that?

Under 15 .... (RECORD 01 ABOVE) 43/
15 or more ... (RECORD 95 ABOVE)
Still DK .... (RECORD 98 ABOVE)

202. Before age 18, did you ever get into trouble with the police, your parents or neighbors because of
O fighting (other than for fighting at school)?

No ...(SKIP TO INSTRUCTIONS BEFORE Q. 202E) ① 44/
Yes .....(ASK A) ..... ⑤

A. Did that happen more than once?

No .....(SKIP TO C)..... ② 45/
Yes .....(ASK B)..... ⑤

B. Were you sometimes the one who started the fight?

No ..... ① 46/
Yes ..... ⑤

C. At what age did you first get into trouble because of fighting (away from school)?

ENTER AGE AND SKIP TO Q. 203: ☐☐ ⓪①②③④⑤⑥⑦⑧⑨ 47/

INTERVIEWER: IF R SAYS "DK": ASK D

D. Do you think it was before you were 15 or later than that?

Under 15 ...(RECORD 01 ABOVE & SKIP TO Q. 203) 48/
15 or more ..(RECORD 95 ABOVE & SKIP TO Q. 203)
Still DK ..(RECORD 98 ABOVE & SKIP TO Q. 203)

INTERVIEWER: ARE BOTH Q. 201 AND 202 CODED 1?

HH NO .....(SKIP TO Q. 203)..... ① 50/
YES .....(ASK E)..... ⑤

E. Even though you didn't get into trouble for fighting, did you start fights more than once before you were 15?

No ..... ① 51/
Yes ..... ⑤

DECK 09

203. When you were a kid, did you ever run away from home overnight?

No ..... (SKIP TO Q. 204) .....① 52/
Yes ..... (ASK A) .....④

A. Did you run away more than once?

No, just once .....② 53/
Yes, more than once .....④

B. How old were you when you first ran away from home overnight?

ENTER AGE & GO TO Q. 204 ☐☐ 54/

INTERVIEWER: IF R SAYS "DK": ASK C

C. Do you think it was before you were 15 or later than that?

Under 15 ....(RECORD 01 ABOVE) 55/
15 or more ...(RECORD 95 ABOVE)
Still DK ....(RECORD 98 ABOVE)

204. Of course, no one tells the truth all the time, but did you tell a lot of lies when you were a child or teenager?

No ..... (SKIP TO Q. 205) .....① 57/
Yes ..... (ASK A) .....④

A. How old were you when you first told a lot of lies?

ENTER AGE & GO TO Q. 205 ☐☐ 58/

INTERVIEWER: IF R SAYS "DK": ASK B

B. Do you think it was before you were 15 or later than that?

Under 15 ....(RECORD 01 ABOVE) 60/
15 or more ...(RECORD 95 ABOVE)
Still DK ....(RECORD 98 ABOVE)

205. When you were a child, did you more than once swipe things from stores or from other children or steal from your parents or from anyone else?

No .....(SKIP TO Q. 206).....① 61/
Yes .....(ASK A).....④

A. How old were you when you first stole things?

ENTER AGE & GO TO Q. 206 ☐☐ 62/

INTERVIEWER: IF R SAYS "DK": ASK B

B Do you think it was before you were 15 or later than that? DECK 09

Under 15 .....(RECORD 01 ABOVE)
15 or more ....(RECORD 95 ABOVE)
Still DK .....(RECORD 98 ABOVE) 64/

BEGIN DECK 10

206. When you were a kid, did you ever intentionally damage someone's car or do anything else to destroy or severely damage someone else's property?

No .....(SKIP TO Q. 207).....① 65/
Yes .....(ASK A).....②

A. How old were you when you first did that?

ENTER AGE & GO TO Q. 207 ☐☐ ⓪①②③④⑤⑥⑦⑧⑨ 66/

INTERVIEWER: IF R SAYS "DK": ASK B

B. Do you think it was before you were 15 or later than that?

Under 15 .....(RECORD 01 ABOVE)
15 or more ....(RECORD 95 ABOVE)
Still DK .....(RECORD 98 ABOVE) 11/

207. Were you ever arrested as a juvenile or sent to juvenile court?

No .....(SKIP TO Q. 208).....① 12/
Yes .....(ASK A).....②

A. How old were you the first time?

ENTER AGE & GO TO Q. 208 ☐☐ ⓪①②③④⑤⑥⑦⑧⑨ 13/

INTERVIEWER: IF R SAYS "DK": ASK B

B. Do you think it was before you were 15 or later than that?

Under 15 .....(RECORD 01 ABOVE)
15 or more ....(RECORD 95 ABOVE)
Still DK .....(RECORD 98 ABOVE) 15/

208. Have you ever been arrested since 18 for anything other than traffic violations?

No .....(SKIP TO Q. 209).....① 16/
Yes .....(ASK A AND B).....②

A. Have you been arrested more than once?

No, just once .....② 17/
Yes, more than once ⟶ ①*

B. Have you ever been convicted of a felony?

No .....① 18/
Yes ⟶ ①*

CODE	
1 = no	4 = med exp
2 = below cnt	5 = yes
3 = drugs or alc	

DECK 10

209. Have you had at least four traffic tickets in your life for speeding or running a light or causing an accident?

No ..... ① 19/
Yes → ①*

210. Now I'm going to ask you about your sexual experience. In general, has your sex life been important to you, or could you have gotten along as well without it?

Somewhat important ..... ①
Gotten along as well without it ..... ②
No sexual experience ..... ③ 20/

SUPPLEMENT USED:	IF NO SEX EXPERIENCE OR REFUSES SEX QUESTIONS, SKIP TO Q. 523.
NO SUPPLEMENT:	IF NO SEX EXPERIENCE, SKIP TO Q. 216.
	IF REFUSES SEX QUESTIONS, SKIP TO INSTRUCTIONS ABOVE Q. 214.

211. Has having sexual relations ever been physically painful for you?

MD: _____ SELF: _____ ① ② ④ ⑤ 21/

212. Has there been a period of several months in your life when having sex was not pleasurable for you (even when it wasn't painful)?

MD: _____ SELF: _____ ① ② ③ ④ ⑤ 22/

213. Have you had any (other) kind of sexual difficulties (FOR MEN, such as a period of two months or more when you had trouble having an erection)?

MD: _____ SELF: _____ ① ② ③ ④ ⑤ 23/

INSERT SUPPLEMENT HERE

217. Since age 18, have you been in more than one fight that came to swapping blows (other than fights with your [husband/wife/partner])?

INTERVIEWER: IF R VOLUNTEERS "ONLY AS REQUIRED BY JOB," CODE 1.

No ..... ① 14/
Yes → ①*

218. Have you ever used a weapon, like a stick, knife, or gun, in a fight since you were 18?

INTERVIEWER: IF R SAYS "ONLY AS REQUIRED BY OCCUPATION," CODE 1
IF R SAYS "WIELDED BUT MISSED," CODE 5

No ..... ① 15/
Yes → ①*

CODE	
1 = no	4 = med exp
2 = below cnl	5 = yes
3 = drugs or alc	

DECK 11

219. Since you were 18, did you ever hold three or more different jobs within a five-year period?

INTERVIEWER: OMIT CHANGES VOLUNTEERED AS DUE TO JOB ENDING, RETURN TO SCHOOL, ILLNESS OR MATERNITY, TRANSFER OF SPOUSE, BECOMING HOUSEWIFE. COUNT CHANGES IN MAIN JOB ONLY.

No ..... ① 16/
Yes → ①*

220. Have you been fired from more than one job?

No ..... ① 17/
Yes → ①*

221. Since you were 18, have you quit a job three times or more before you already had another job lined up?

INTERVIEWER: OMIT QUITTING VOLUNTEERED AS DUE TO RETURN TO SCHOOL, ILLNESS OR MATERNITY, TRANSFER OF SPOUSE, BECOMING HOUSEWIFE.

No ..... ① 18/
Yes → ①*

222. On any job you have had since you were 18, were you late or absent an average of 3 days a month or more?

INTERVIEWER: IF R SAYS "NO SET SCHEDULE": CODE 1

RECORD ALC/MED: ① ② → ①* 19/

223. How many months out of the last five years have you been without a job?

ENTER # MONTHS: ☐☐ ⓪ ① ② ③ ④ ⑤ ⑥ ⑦ ⑧ ⑨ 20/

INTERVIEWER: IF LESS THAN 6 MOS. SKIP TO Q. 224.
IF 6 MOS. OR MORE, ASK B
IF R SAYS "DK," ASK A.

A. Do you think it was less than 6 months or more than that?

Less than 6 mos. ..... (RECORD 01 ABOVE & SKIP TO Q. 224) 22/
6 mos. or more ..... (RECORD 95 ABOVE & ASK B)
Still DK ..... (RECORD 98 ABOVE & ASK B)

B. For how much of that time did you want to work but were not able to find a job?

ENTER # MONTHS: ☐☐ ⓪ ① ② ③ ④ ⑤ ⑥ ⑦ ⑧ ⑨ 23/

INTERVIEWER: IF B = 6 MOS. OR MORE, SKIP TO E

DECK 11

223. (Continued)

C. For how much of that time were you not looking for work because of emotional or mental problems or because of problems with drugs or alcohol?

ENTER # MONTHS: ☐☐ 00 01 02 03 04 05 06 07 08 09 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25/

INTERVIEWER: IF B + C = 6 MOS. OR MORE, SKIP TO E

D. How much time (besides that) were you just not interested in working but not in school, or physically ill (or retired or a housewife)?

ENTER # MONTHS: ☐☐ 00 01 02 03 04 05 06 07 08 09 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 27/

E. **INTERVIEWER: DO B + C + D = 6 MONTHS OR MORE WITHOUT WORK?**

NO 0 29/
YES → 1*

224. Have you ever used an alias or an assumed name?

INTERVIEWER: IF R VOLUNTEERS ONLY PEN OR STAGE NAME: CODE 1.

No 1 30/
Yes → 5*

225. Have you thought that you lied pretty often since you have been an adult?

No 1 31/
Yes 5

226. Have you ever traveled around for a month or more without having any arrangements ahead of time and not knowing how long you were going to stay or where you were going to work?

INTERVIEWER: IF R VOLUNTEERS "ONLY ON VACATION FROM JOB": CODE 1.

No 1 32/
Yes → 5*

227. Has there ever been a period when you had no regular place to live, for at least a month or so?

No 1 33/
Yes → 5*

DECK 11

INTERVIEWER: DOES R HAVE CHILD (Q. 14 = 01 OR MORE) OR
KK HAS R ACTED AS PARENT (Q. 14A = 5)?

NO ..(SKIP TO INSTRUCTIONS BEFORE Q. 232) ..... ①
YES .....(ASK Q. 228) ..... ② 34/

228. Have you sometimes left young children under 6 years old home alone while you were shopping or out doing anything else?

INTERVIEWER: IF R VOLUNTEERS "ONLY IN EMERGENCY," "FOR LESS THAN 30 MINUTES," OR "CHILD COULD BE HEARD OR COULD COME THERE": CODE 1.

No ..... ① 35/
Yes ..... ② *
Never lived with child ..... (SKIP TO INSTRUCTIONS BEFORE Q. 232) ..... ③

229. Have there been times when a neighbor fed a child (of yours/you were caring for) because you didn't get around to shopping for food or cooking, or kept your child overnight because no one was taking care of him at home?

INTERVIEWER: IF R VOLUNTEERS "ONLY IN EMERGENCY": CODE 1.

No ..... ① 36/
Yes ..... ② *

230. Has a nurse, or social worker or teacher ever said that any child (of yours/you were caring for) wasn't being given enough to eat or wasn't being kept clean enough or wasn't getting medical care when it was needed?

No ..... ① 37/
Yes ..... ② *

231. Have you more than once run out of money for food for your family because you had spent the food money on yourself or on going out?

No ..... ① 38/
Yes ..... ② *

INTERVIEWER: HOW MANY "5"'s" HAVE BEEN CODED IN Qs. 208-231 (BEGINNING ON PAGE 55)?

LL

NONE, ONE OR TWO ..(SKIP TO Q. 235) ①
THREE OR MORE .....(ASK Q. 232)..... ②

39/

232. Did you ever talk to a doctor about any of these things you did like (SPECIFY "5"'s" WHICH HAVE BEEN CODED, BEGINNING WITH Q. 208, p. 55)

No ..... ①
Yes ..... ⑤

40/

INTERVIEWER: IS R OLDER OR YOUNGER THAN 26 YEARS OLD?

MM

26 OR OLDER .....(ASK Q. 233)..... ①
YOUNGER THAN 26 ..(SKIP TO Q. 234). ②

41/

233. Did you do any of these things between the ages of 18 and 25?

No .....(ASK A)..... ①
Yes ....(SKIP TO Q. 234)..... ⑤

42/

A. Was there some reason you couldn't have done these things between 18 and 25, for instance, because you were ill in bed that whole time (or in jail/not married/had no children)?

No ....(HAD OPPORTUNITY)..... ①
Yes ...(NO OPPORTUNITY)..... ⑤

43/

234. When is the last time you did any one of these things like (MENTION CODED "5"'s" BEGINNING WITH Q. 208)?

CODE MOST RECENT TIME POSSIBLE

Within last 2 weeks ..... ①
Within last month ..... ②
Within last 6 months ..... ③
Within last year ..... ④
Within last 3 years ..... ⑤
More than 3 years ago .....(ASK A).... ⑥

44/

IF MORE THAN 3 YEARS AGO:

A. How old were you the last time you did any of those things?

ENTER AGE: ⓪ ① ② ③ ④ ⑤ ⑥ ⑦ ⑧ ⑨
⓪ ① ② ③ ④ ⑤ ⑥ ⑦ ⑧ ⑨

45/

Appendix D
Drinking and Drug History Form

Information on Drinking and Other Drug Use
(6/1/89) (13 pages)

R Number: _____
Given By: _____
Date: _____
T1.0
Ans. Chk: _____
P6

This questionnaire takes about 15 minutes to complete. All information will be used for research only and will be kept strictly confidential. If you are not sure of the answer to a question please answer the best you can. Please try to answer each item.

A. THE FOLLOWING QUESTIONS ARE ABOUT YOUR DRINKING OF ALCOHOLIC BEVERAGES:

1. HOW OLD WERE YOU THE FIRST TIME YOU EVER TOOK A DRINK? DO NOT COUNT THE TIMES WHEN YOU WERE GIVEN A "SIP" BY AN ADULT.

_____ years old.

2. OVER THE LAST 6 MONTHS, ON THE AVERAGE, HOW MANY DAYS A MONTH HAVE YOU HAD A DRINK?

_____ days a month.

3. OVER THE LAST 6 MONTHS, ON A DAY WHEN YOU ARE DRINKING, HOW MANY DRINKS DO YOU USUALLY HAVE IN 24 HOURS? (A DRINK IS A 12 OZ. CAN, GLASS OR BOTTLE OF BEER; A 4 OZ. GLASS OF WINE; A SINGLE SHOT; OR A SINGLE "MIXED DRINK.")

_____ drinks per 24 hours.

4. OVER THE PAST 6 MONTHS, WHEN YOU GOT DRUNK, HOW BAD WAS YOUR HANGOVER?

_____ Never bad
_____ Not bad
_____ A little less than average
_____ Average
_____ A little more than average
_____ Pretty Bad
_____ Terrible
_____ Worst possible
_____ Never drank enough to get hangover

CONTINUE ON THE NEXT PAGE.

8. THE FOLLOWING QUESTIONS ARE ABOUT YOUR DRINKING PATTERNS. IN ANSWERING THE QUESTIONS, PLEASE THINK ABOUT WHAT YOU HAVE DONE ON THE AVERAGE OVER THE LAST SIX MONTHS.

1. WHEN DRINKING WINE:

a. HOW OFTEN DO YOU USUALLY HAVE WINE OR A PUNCH CONTAINING WINE?

_____ 3 or more times a day
_____ 2 times a day
_____ Once a day
_____ Nearly every day
_____ 3 or 4 times a week
_____ once or twice a week
_____ 2 or 3 times a month
_____ About once a month
_____ Less than once a month, but at least once a year
_____ Less than once a year
_____ NEVER [If checked, go to question #2a]

b. THINK OF ALL THE TIMES YOU HAD WINE OR A PUNCH CONTAINING WINE RECENTLY. WHEN YOU DRINK WINE, HOW OFTEN DO YOU HAVE 10 OR MORE GLASSES?

_____ Nearly every time: SKIP TO QUESTION #2 BELOW
_____ More than half the time: SKIP TO QUESTION #2 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

c. WHEN YOU DRINK WINE, HOW OFTEN DO YOU HAVE AS MANY AS 7 TO 9 GLASSES?

_____ Nearly every time: SKIP TO QUESTION #2 BELOW
_____ More than half the time: SKIP TO QUESTION #2 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

d. WHEN YOU DRINK WINE, HOW OFTEN DO YOU HAVE AS MANY AS 5 to 6 GLASSES?

_____ Nearly every time: SKIP TO QUESTION #2 BELOW
_____ More than half the time: SKIP TO QUESTION #2 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

e. WHEN YOU DRINK WINE, HOW OFTEN DO YOU HAVE AS MANY AS 3 to 4 GLASSES?

_____ Nearly every time: SKIP TO QUESTION #2 BELOW
_____ More than half the time: SKIP TO QUESTION #2 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

f. WHEN YOU DRINK WINE, HOW OFTEN DO YOU HAVE 1 TO 2 GLASSES?

_____ Nearly every time
_____ More than half the time
_____ Less than half the time
_____ Once in a while
_____ NEVER

2. <u>WHEN DRINKING BEER</u>

a. HOW OFTEN DO YOU USUALLY HAVE BEER?

_____ 3 or more times a day
_____ 2 times a day
_____ Once a day
_____ Nearly every day
_____ 3 or 4 times a week
_____ Once or twice a week
_____ 2 or 3 times a month
_____ About once a month
_____ Less than once a month, but at least once a year
_____ Less than once a year
_____ NEVER [If checked, go to question #3a]

b. THINK OF ALL THE TIMES YOU HAD BEER RECENTLY. WHEN YOU DRINK BEER, HOW OFTEN DO YOU HAVE 10 OR MORE CANS, GLASSES OR BOTTLES?

_____ Nearly every time: SKIP TO QUESTION #3 BELOW
_____ More than half the time: SKIP TO QUESTION #3 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

c. WHEN YOU DRINK BEER, HOW OFTEN DO YOU HAVE AS MANY AS 7 TO 9 CANS, GLASSES OR BOTTLES?

_____ Nearly every time: SKIP TO QUESTION #3 BELOW
_____ More than half the time: SKIP TO QUESTION #3 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

d. WHEN YOU DRINK BEER, HOW OFTEN DO YOU HAVE AS MANY AS 5 TO 6 CANS, GLASSES OR BOTTLES?

_____ Nearly every time: SKIP TO QUESTION #3 BELOW
_____ More than half the time: SKIP TO QUESTION #3 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

e. WHEN YOU DRINK BEER, HOW OFTEN DO YOU HAVE AS MANY AS 3 to 4 CANS, GLASSES OR BOTTLES?

_____ Nearly every time: SKIP TO QUESTION #3 BELOW
_____ More than half the time: SKIP TO QUESTION #3 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

f. WHEN YOU DRINK BEER, HOW OFTEN DO YOU HAVE 1 TO 2 CANS, GLASSES OR BOTTLES?

_____ Nearly every time
_____ More than half the time
_____ Less than half the time
_____ Once in a while
_____ NEVER

3. <u>WHEN DRINKING WHISKEY OR LIQUOR</u>

a. HOW OFTEN DO YOU USUALLY HAVE WHISKEY OR LIQUOR (SUCH AS MARTINIS, MANHATTANS, HIGHBALLS, OR STRAIGHT DRINKS INCLUDING SCOTCH, BOURBON, GIN, VODKA, RUM, ETC.)?

_____ 3 or more times a day
_____ 2 times a day
_____ Once a day
_____ Nearly every day
_____ 3 or 4 times a week
_____ Once or twice a week
_____ 2 or 3 times a month
_____ About once a month
_____ Less than once a month, but at least once a year
_____ Less than once a year
_____ NEVER [If checked, go to question #4]

b. THINK OF ALL THE TIMES YOU HAD DRINKS CONTAINING WHISKEY OR OTHER LIQUOR RECENTLY. WHEN YOU HAVE HAD THEM, HOW OFTEN DO YOU HAVE 10 OR MORE DRINKS?

_____ Nearly every time: SKIP TO QUESTION #4 BELOW
_____ More than half the time: SKIP TO QUESTION #4 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

c. WHEN YOU HAVE HAD DRINKS CONTAINING WHISKEY OR OTHER LIQUOR, HOW OFTEN DO YOU HAVE AS MANY AS 7 TO 9 DRINKS?

_____ Nearly every time: SKIP TO QUESTION #4 BELOW
_____ More than half the time: SKIP TO QUESTION #4 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

d. WHEN YOU HAVE HAD DRINKS CONTAINING WHISKEY OR OTHER LIQUOR, HOW OFTEN DO YOU HAVE AS MANY AS 5 TO 6 DRINKS?

_____ Nearly every time: SKIP TO QUESTION #4 BELOW
_____ More than half the time: SKIP TO QUESTION #4 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

e. WHEN YOU HAVE HAD DRINKS CONTAINING WHISKEY OR LIQUOR, HOW OFTEN DO YOU HAVE 3 TO 4 DRINKS?

_____ Nearly every time: SKIP TO QUESTION #4 BELOW
_____ More than half the time: SKIP TO QUESTION #4 BELOW
_____ Less than half the time
_____ Once in a while
_____ NEVER

f. WHEN YOU HAVE HAD DRINKS CONTAINING WHISKEY OR LIQUOR, HOW OFTEN DO YOU HAVE 1 TO 2 DRINKS?

_____ Nearly every time
_____ More than half the time
_____ Less than half the time
_____ Once in a while
_____ NEVER

4. <u>WHEN DRINKING ANYTHING</u>, CHECK HOW OFTEN YOU HAVE ANY DRINK CONTAINING ALCOHOL, WHETHER IT IS WINE, BEER, WHISKEY OR ANY OTHER DRINK. MAKE SURE THAT YOUR ANSWER IS NOT LESS FREQUENT THAN THE FREQUENCY REPORTED ON ANY OF THE PRECEDING QUESTIONS.

_____ 3 or more times a day
_____ 2 times a day
_____ Once a day
_____ Nearly every day
_____ 3 or 4 times a week
_____ Once or twice a week
_____ 2 or 3 times a month
_____ About once a month
_____ Less than once a month, but at least once a year
_____ Less than once a year

---

5. Now <u>a question about earlier in your life</u>: HOW OLD WERE YOU THE FIRST TIME YOU EVER DRANK ENOUGH TO GET DRUNK?

_____ years old.

6a. WE ARE ALSO INTERESTED IN THE OCCASIONS THAT MAY BE RARE (OR NOT), WHEN PEOPLE DRINK A LOT MORE THAN THEY USUALLY DO. IN THE LAST SIX MONTHS, THINK OF THE 24 HOUR PERIOD WHEN YOU DID THE MOST DRINKING; THIS WOULD BE A DAY SOMEWHERE IN THE PERIOD BETWEEN _____ (month), _____ (year) AND NOW.

On that day, how many drinks did you have? (A drink is a 12 oz. can, bottle, or glass of beer, a 4 oz. glass of wine, a single shot, or a single mixed drink).

_____ 30 or more drinks
_____ 25 - 29 drinks
_____ 20 - 24 drinks
_____ 15 - 19 drinks
_____ 10 - 14 drinks
_____ 7 - 9 drinks
_____ 5 - 6 drinks
_____ 3 - 4 drinks
_____ 1 - 2 drinks
_____ none

6b. APPROXIMATELY WHEN DID THIS HAPPEN? _____ (month), _____ (year).

6c. NOW ANSWER THIS QUESTION FOR ANY TIME IN YOUR LIFE BEFORE THESE LAST SIX MONTHS. IN THE 24 HOUR PERIOD WHEN YOU DID THE MOST DRINKING, HOW MANY DRINKS DID YOU HAVE?

_____ 30 or more drinks
_____ 25 - 29 drinks
_____ 20 - 24 drinks
_____ 15 - 19 drinks
_____ 10 - 14 drinks
_____ 7 - 9 drinks
_____ 5 - 6 drinks
_____ 3 - 4 drinks
_____ 1 - 2 drinks
_____ none

6d. APPROXIMATELY WHEN DID THIS HAPPEN? _____ (month), _____ (year)

ANSWER KEY FOR QUESTIONS BELOW:

1	2	3-5	6-10	11-20	21-50	51-100
101-250	251-500	501-1000	1000+ (more than 1000)			

C. NOW SOME QUESTIONS ABOUT OUTCOMES PEOPLE SOMETIMES HAVE BECAUSE OF DRINKING. HAVE YOU EVER HAD ANY OF THE FOLLOWING HAPPEN BECAUSE OF YOUR DRINKING?

		YES NO (check one)	HOW MANY TIMES (approx.- see key)*	AGE first time	AGE most recent time
1.	Missed school or time on job	___ ___	_____	_____	_____
2.	Thought I was drinking too much	___ ___	_____	_____	_____
3.	Gone on a binge of constant drinking for 2 or more days	___ ___	_____	_____	_____
4.	Lost friends	___ ___	_____	_____	_____
5.	My spouse or others in my family (my parents or children) objected to my drinking	___ ___	_____	_____	_____
6.	Felt guilty about my drinking	___ ___	_____	_____	_____
7.	Divorce or separation	___ ___	_____	_____	_____
8.	Took a drink or two first thing in morning	___ ___	_____	_____	_____
9.	Restricted my drinking to certain times of day or week in order to control it or cut down, (like after 5PM, or only on weekends, or only with other people)	___ ___	_____	_____	_____
10.	Been fired or laid off	___ ___	_____	_____	_____
11.	Once started drinking, kept on going till completely intoxicated	___ ___	_____	_____	_____
12.	Had a car accident when I was driving	___ ___	_____	_____	_____

* SELECT YOUR ANSWER FROM KEY AT THE TOP OF THE PAGE

Questions continue on the next page.

ANSWER KEY FOR QUESTIONS BELOW:

1	2	3-5	6-10	11-20	21-50	51-100
101-250	251-500	501-1000	1000+ (more than 1000)			

		YES	NO (check one)	HOW MANY TIMES approx- see key)*	AGE first time	AGE most recent time
13.	Kept on drinking after I promised myself not to	___	___	_____	_____	_____
14.	Had to go to a hospital (other than accidents)	___	___	_____	_____	_____
15.	Had to stay in a hospital overnight	___	___	_____	_____	_____
16.	Had the shakes "the morning after"	___	___	_____	_____	_____
17.	Heard or saw or felt things that weren't there, hallucinations) several days after stopping drinking	___	___	_____	_____	_____
18.	Had blackouts (couldn't remember later what you'd done while drinking)	___	___	_____	_____	_____
19.	Been given a ticket for drunk driving (DWI or DUIL)	___	___	_____	_____	_____
20.	Had a jerking or fits (convulsions) several days after stopping drinking	___	___	_____	_____	_____
21.	Been given a ticket for public intoxication, drunk and disorderly, or other nondriving alcohol arrest	___	___	_____	_____	_____
22.	Had the D.T.'s (delirium tremens, shakes, sweating, rapid heart, etc.) within 2 - 3 days after stopping drinking	___	___	_____	_____	_____

* SELECT ANSWERS FROM THE KEY AT THE TOP OF THE PAGE

D. THE LAST SECTIONS OF THIS QUESTIONNAIRE DEAL WITH VARIOUS DRUGS OTHER THAN ALCOHOL. THERE IS STILL A LOT OF TALK THESE DAYS ABOUT THIS SUBJECT, BUT VERY LITTLE ACCURATE INFORMATION, PARTICULARLY ABOUT PATTERNS OF USE OF THESE SUBSTANCES IN ADULTHOOD. THEREFORE, WE STILL HAVE A LOT TO LEARN ABOUT THE ACTUAL EXPERIENCES OF PEOPLE YOUR AGE.

WE HOPE THAT YOU CAN ANSWER ALL QUESTIONS; BUT IF YOU FIND ONE WHICH YOU FEEL YOU CANNOT ANSWER HONESTLY, WE WOULD PREFER THAT YOU LEAVE IT BLANK.

REMEMBER THAT YOUR ANSWERS WILL BE KEPT STRICTLY CONFIDENTIAL AND THEY ARE NEVER CONNECTED WITH YOUR NAME. THAT IS WHY THIS QUESTIONNAIRE IS IDENTIFIED ONLY WITH A CODE NUMBER.

THE FOLLOWING QUESTIONS ARE ABOUT CIGARETTES (CHECK THE BEST ANSWER):

1a. HAVE YOU EVER SMOKED CIGARETTES?

_____ Never (GO TO QUESTION 3)
_____ Once or twice
_____ Occasionally but not regularly
_____ Regularly in the past
_____ Regularly now

1b. HAVE YOU SMOKED CIGARETTES DURING THE PAST 12 MONTHS?

_____ Never (GO TO QUESTION 3)
_____ Once or twice
_____ Occasionally but not regularly
_____ Regularly for a while during this year, but not now
_____ Regularly now

2. HOW FREQUENTLY HAVE YOU SMOKED CIGARETTES DURING THE PAST 30 DAYS?

_____ Not at all
_____ Less than one cigarette per day
_____ One to five cigarettes per day
_____ About one-half pack per day
_____ About one pack per day
_____ About one and one-half packs per day
_____ Two packs or more per day

E. THE FOLLOWING QUESTIONS ARE ALL ABOUT NON-PRESCRIPTION USE OF DRUGS, EITHER FOR RECREATION OR FOR SELF-MEDICATION.
(MARK ONE SPACE FOR EACH LINE).

3. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU USED MARIJUANA (GRASS, POT) OR HASHISH (HASH, HASH OIL)	0 Occasions	1-2 Occasions	3-5 Occasions	6-9 Occasions	10-19 Occasions	20-39 Occasions	40-99 Occasions	100-1000 Occasions	More than 1000
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )

(MARK ONE SPACE FOR EACH LINE).

	0 Occasions	1-2 Occasions	3-5 Occasions	6-9 Occasions	10-19 Occasions	20-39 Occasions	40-99 Occasions	100-1000 Occasions	More than 1000
4. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU USED LSD (ACID)									
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )
5. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU USED PSYCHEDELICS OTHER THAN LSD (LIKE MESCALINE, PEYOTE, PSILOCYBIN, PCP)									
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )
6. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU USED COCAINE (COKE OR CRACK)									
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the past 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )
7. AMPHETAMINES ARE SOMETIMES PRESCRIBED BY DOCTORS TO HELP PEOPLE LOSE WEIGHT OR TO GIVE PEOPLE MORE ENERGY. THEY ARE SOMETIMES CALLED UPPERS, UPS, SPEED, CRYSTAL, CRANK, BENNIES, DEXIES, PEP PILLS, AND DIET PILLS. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU TAKEN AMPHETAMINES ON YOUR OWN--THAT IS, WITHOUT A DOCTOR TELLING YOU TO TAKE THEM										
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )

(MARK ONE SPACE FOR EACH LINE).

8. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU USED QUAALUDES (QUADS, SOAPERS, METHAQUALONE) ON YOUR OWN-- THAT IS, WITHOUT A DOCTOR TELLING YOU TO TAKE THEM	0 Occasions	1-2 Occasions	3-5 Occasions	6-9 Occasions	10-19 Occasions	20-39 Occasions	40-99 Occasions	100-1000 Occasions	More than 1000
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )

9. BARBITURATES ARE SOMETIMES PRESCRIBED BY DOCTORS TO HELP PEOPLE RELAX OR GET TO SLEEP. THEY ARE SOMETIMES CALLED DOWNS, DOWNERS, GOOFBALLS, YELLOWS, REDS, BLUES, RAINBOWS. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU TAKEN BARBITURATES ON YOUR OWN -- THAT IS, WITHOUT A DOCTOR TELLING YOU TO TAKE THEM	0 Occasions	1-2 Occasions	3-5 Occasions	6-9 Occasions	10-19 Occasions	20-39 Occasions	40-99 Occasions	100-1000 Occasions	More than 1000
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )

10. TRANQUILIZERS ARE SOMETIMES PRESCRIBED BY DOCTORS TO CALM PEOPLE DOWN, QUIET THEIR NERVES, OR RELAX THEIR MUSCLES. LIBRIUM VALIUM, AND MILTOWN ARE ALL TRANQUILIZERS. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU TAKEN TRANQUILIZERS ON YOUR OWN -- THAT IS, WITHOUT A DOCTOR TELLING YOU TO TAKE THEM	0 Occasions	1-2 Occasions	3-5 Occasions	6-9 Occasions	10-19 Occasions	20-39 Occasions	40-99 Occasions	100-1000 Occasions	More than 1000
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )

(MARK ONE SPACE FOR EACH LINE).

11. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU USED HEROIN (SMACK, HORSE, SKAG)	0 Occasions	1-2 Occasions	3-5 Occasions	6-9 Occasions	10-19 Occasions	20-39 Occasions	40-99 Occasions	100-1000 Occasi	More than 1000
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )

12. THERE ARE A NUMBER OF NARCOTICS OTHER THAN HEROIN, SUCH AS METHADONE, OPIUM, MORPHINE, CODEINE, DEMEROL, PAREGORIC, TALWIN, AND LAUDANUM. THESE ARE SOMETIMES PRESCRIBED BY DOCTORS. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU TAKEN NARCOTICS OTHER THAN HEROIN ON YOUR OWN-- THAT IS, WITHOUT A DOCTOR TELLING YOU TO TAKE THEM	0 Occasions	1-2 Occasions	3-5 Occasions	6-9 Occasions	10-19 Occasions	20-39 Occasions	40-99 Occasions	100-1000 Occasions	More than 1000
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )

13. ON HOW MANY OCCASIONS (IF ANY) HAVE YOU SNIFFED GLUE, OR BREATHED THE CONTENTS OF AEROSOL SPRAY CANS, OR INHALED ANY OTHER GASES OR SPRAYS IN ORDER TO GET HIGH	0 Occasions	1-2 Occasions	3-5 Occasions	6-9 Occasions	10-19 Occasions	20-39 Occasions	40-99 Occasions	100-1000 Occas.	More than 1000
In your lifetime?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 12 months?	( )	( )	( )	( )	( )	( )	( )	( )	( )
During the last 30 days?	( )	( )	( )	( )	( )	( )	( )	( )	( )

F. NOW SOME OTHER QUESTIONS ABOUT NONPRESCRIPTION USE OF DRUGS. HAVE YOU EVER HAD ANY OF THE FOLLOWING OUTCOMES BECAUSE OF YOUR USE OF THE NONPRESCRIPTION DRUGS ASKED ABOUT IN SECTION E (THE LAST SECTION)?

ANSWER KEY FOR QUESTIONS BELOW:

1	2	3-5	6-10	11-20	21-50	51-100
	101-250		251-500	500+ (more than 500)		

		YES	NO	HOW MANY TIMES (approx) (see key)*	AGE first TIME	AGE most recent TIME
1.	Missed school or time on job	___	___	_____	_____	_____
2.	Lost friends	___	___	_____	_____	_____
3.	Been divorced or separated	___	___	_____	_____	_____
4.	Been fired or laid off	___	___	_____	_____	_____
5.	Had a car accident when you were driving	___	___	_____	_____	_____
6.	Had to go to a hospital (other than accidents)	___	___	_____	_____	_____
7.	Had to stay in hospital overnight	___	___	_____	_____	_____
8.	Had to see a doctor because of drug use (unintentional overdose) or had a doctor say drugs had harmed your health	___	___	_____	_____	_____
9.	Gone through physical withdrawal from drugs	___	___	_____	_____	_____
10.	Been arrested for possession or sale of drugs other than marijuana	___	___	_____	_____	_____

* SELECT YOUR ANSWER FROM KEY AT THE TOP OF THE PAGE

IIa. Have you ever taken drugs intravenously (using a needle)? Don't count shots you were given by a doctor or nurse or shots you may have taken for treatment of diabetes.

_____NO _____YES

IIb. IF YES, WHAT DRUGS HAVE YOU TAKEN INTRAVENOUSLY (IV)? _____

IIc. AT WHAT AGE DID YOU FIRST TAKE AN IV DRUG? _____ years old.

IId. AT WHAT AGE WAS THE MOST RECENT TIME? _____ years old.

List of References

List of References

Allen, D. L., Petersen, D. R., Wilson, J. R., McClearn, G.E. and Nishimoto, T.K. (1983). Selective breeding for a multivariate index of ethanol dependence in mice: results from the first five generations. Alcoholism: Clinical and Experimental Research, 7, 443-337.

American Psychiatric Association (1987). Diagnostic and Statitical Manual of Mental Disorders (Third Edition-Revised). Washington, D.C., A.P.A.

Beck, A.T. and Beck, R. W. (1972) Screening depressed patients in family practice: A rapid technique. Postgraduate Medicine, 52, 81-85.

Begleiter, H., Porjesz, J., Bihari, B. and Kissin, B. (1984). Event-related brain potentials in boys at risk for alcoholism. Science, 225, 1493-1496.

Bohman, M. (1978). Some genetic aspects of alcoholism and criminality. Archives of General Psychiatry, 269-276.

Bohman, M., Sigvardsson, S., and Cloninger, C. R. (1981). Maternal inheritance of alcohol abuse: Cross-fostering analysis of adopted women. Archives of General Psychiatry, 38, 265-269.

Buchsbaum, M. S., Landau, S., Murphy, D. and Goodwin, F. (1973) Average evoked response in bipolar and unipolar affective disorders: Relationship to sex, age of onset and monoamine oxidase. Biological Psychiatry, 7, 199-212.

Cadoret, R. J. and Gath, A. (1978). Inheritance of alcoholism in adoptees. British Journal of Psychiatry, 132, 252-258.

Cadoret, R. J., O'Gorman, T. W., Troughton, E. and Haywood, E. (1985). Alcoholism and antisocial personality: Interrelationships, genetic and environmental factors. Archives of General Psychiatry, 42, 161-167.

Cadoret, R. J., Troughton, E. and O'Gorman, T. W. (1987). Genetic and environmental factors in alcohol abuse and antisocial personality. Journal of Studies on Alcohol, 48, 1-8

Caetano, R. (1976). Two versions of dependence: DSM-III and the Alcohol Dependence Syndrome. Drug and Alcohol Dependence, 15, 81-103.

Cahalan, D., Cisin, I. and Crossley, H. (1969). American Drinking Practices: A National Study of Drinking Behavior and Attitudes. New Brunswick, New Jersey: Rutgers Center of Alcohol Studies.

Cahalan, D. and Cisin, I. (1976) Drinking behavior and drinking problems in the United States. In B. Kissin and H. Begleiter (Eds.) The Biology of Alcholism (Vol. 4) Social Aspects of Alcoholism. New York: Plenum Press.

Clark, W. B. and Cahalan, D. Changes in problem drinking over a four-year span. Addictive Behaviors, 1, 251-259.

Cloninger, C. R. (1987). Neurogenic adaptive mechanisms in alcoholism. Science, 236, 410-416.

Cloninger, C. R., Bohman, M. and Sigvardsson, S. (1981). Inheritance of alcohol abuse: Cross-fostering analysis of adopted men. Archives of General Psychiatry, 38, 861-868.

Cloninger, C. R., Bohman, M., Sigvardsson, S. and VonKnorring, A-L. (1985). Psychopathology in adopted-out children of alcoholics: The Stockholm Adoption Study. In M. Galanter (Ed.) Recent Developments in Alcoholism, Vol. 3. New York: Plenum Press.

Cloninger, C. R., Sigvardsson, S. and Bohman, M. (1988). Childhood personality predicts alcohol abuse in young adults. Alcoholism: Clinical and Experimental Research, 12, 494-505.

Cotton, N. S. (1979). The familial incidence of alcoholism: A review. Journal of Studies on Alcohol, 40, 89-116.

Crabbe, J. C., Kosobud, A. and Young, E. R. (1983). Genetic selection for ethanol withdrawal severity: Differences in replicate mouse lines. Life Sciences, 33, 995-962.

Edwards, G. and Gross, M. M. (1976). British Medical Journal, 1, 1058.

Endicott, J. and Spitzer, R. L. (1978). A diagnostic interview. Archives of General Psychiatry, 35, 837-844.

Faraj, B., Lenton, J., Kutner, M., Camp, V., Stammers, T., Lee, S. R., Lolies, P. and Chandora, D. (1987). Prevalence of low monoamine oxidase function in alcoholism. Alcoholism: Clinical and Experimental Research, 11, 464-468.

Feighner, J., Robins, E., Guze, S., Woodruff, R., Winokur, G. and Munoz, R. (1972). Diagnostic criteria for use in psychiatric research. Archives of General Psychiatry, 26, 57-63.

Fillmore, K. M. (1987). The 1980's dominant theory of alcohol problems- Where is it leading us? Unpublished manuscript.

Gabrielli, W., Mednick, S., Volavka, J., Pollock, V., Schulsinger, F. and Itile, T. (1982). Electroencephalographs in children of alcoholics fathers. Psychophysiology, 19, 404-407.

Gabrielli, W. and Plomin, R. (1985). Drinking behavior in the Colorado Adoptee and Twin Sample. Journal of Studies on Alcohol, 46, 24-31.

Gallaher, E. J. and Gionet, S.E. (1988) Initial sensitivity and tolerance to ethanol in mice genetically selected for diazepam sensitivity. Alcoholism: Clinical and Experimental Research, 12, 77-80.

Goodwin, D. W. (1979). Alcoholism and Heredity: A review and a hypothesis. Archives of General Psychiatry, 36, 57-62.

Goodwin, D. W., Schulsinger, F., Moller, N., Hermansen, L. , Guze, S. and Winokur, G. (1973). Alcohol problems in adoptees raised apart from alcoholic biological parents. Archives of General Psychiatry, 28, 238-243.

Goodwin, D. W., Schulsinger, F., Moller, N., Hermansen, L. Winokur, G. and Guze, S. (1974). Drinking problems in adopted and non-adopted sons of alcoholics. Archives of General Psychiatry, 31, 164-169.

Helzer, J., Robins, L., McEvoy, L., Spitnagel, E., Stolzmann, R., Farmer, A. and Brockington, I. (1985). A comparison of clinical and Diagnostic Interview Schedule diagnoses. Archives of General Psychiatry, 42, 657-666.

Hrubec, Z. and Omenn, G.S. (1981). Evidence of genetic predisposition to alcoholic cirrhosis and psychosis. Alcoholism: Clinical and Experimental Research, 5, 207-215.

Jellinek, E. M. (1952). Current notes: Phases of alcohol addiction. Quarterly Journal of Studies on Alcoholism, 13, 673-684.

Joreskog, K. and Sorbom, D. (1978). LISREL-V- a general computer program for estimating a linear structural equation system involving multiple indicators of unmeasured variables. Chicago: National Educational Resources.

Kaij, L. and McNeil, T. F. (1960). Genetic aspects of alcoholism. Advanced Biological Psychiatry, 3, 54-65.

Kaprio, J., Koskenvuo, M., Langinvainio, H., Romanov, K., Sarna, S. and Rose, R. (1987). Genetic influences on use and abuse of alcohol: A study of 5638 adult Finnish twin brothers. Alcoholism: Clinical and Experimental Research, 11, 349-355.

Knop, J., Angelo, H., and Christensen, J. (1980). Is the role of acetaldehyde in alcoholism based on an analytical artifact? Lancet, 2, 102.

Loehlin, J. C. and Nichols, R. C. (1976). Heredity, Environment and Personality. Austin: University of Texas Press.

Lumeng, L., Hawkins, T. D., and Li, T. K. (1977). In R. G. Thurman (Ed.) Alcoholism and Aldehyde Metabolizing Systems, Vol. 3. New York: Academic Press.

Matheny, A. P. (1979). Appraisal of parental bias in twin studies: ascribed zygosity and IQ differences in twins. Acta Geneticae Medicae et Gemellologiae (Roma), 28, 155-160.

McClearn, G. E. and Kakihana, R. (1981). Selective breeding for alcohol sensitivity: Short sleep and long sleep mice. In G.E. McClearn and V.G. Erwin (Eds) Developmental of Animal Models as Pharmacogenic Tools, Research Monograph #6. Washington, D.C.: National Institute on Alcohol Abuse and Alcoholism.

McCord, W. and McCord, J. (1962). A longitudinal study of the personality of alcoholics. In D. J. Pittman and C. R. Snyder (Eds.) Society, Culture and Drinking Patterns. New York: J. Wiley and Sons.

Mueller, C. and Parcel, T. (1981). Measures of socioeconomic status: alternatives and recommendations. Child Development, 52, 13-30.

Mulford, H. A. and Miller, D. E. (1960). Drinking in Iowa V: Drinking and alcoholic drinking. Quarterly Journal of Studies on Alcohol, 21, 483-499.

Murray, R. M., Clifford, C. A and Gurling, H. M. (1983). Twin and adoption studies: How good is the evidence for a genetic role? In M. Galanter (Ed.) Recent Developments in Alcoholism, Vol 1. New York: Plenum Press.

O'Malley, S. S., Carey, K. B. and Maisto, S. A. (1986). Validity of young adult's reports of parental drinking practices. Journal of Studies on Alcoholism, 47, 433-435.

Oreland, L., Witberg, A., Winblad, B., Fowler, C., Gottsfries, C. and Kiianmaa, K. (1983). The activity of monoamine oxidase-A and -B in brains from chronic alcoholics. Journal of Neural Transmission, 56, 73-83.

Pandey, G. N., Fawcett, J., Gibbons, R., Clark, D. C. and Davis, J. M. (1988). Platelet monoamine oxidase in alcoholism. Biological Psychiatry, 24, 15-24.

Partanen, J., Brunn, K. and Markannen, T. (1966). Inheritance of Drinking Behavior. Helsinski: Finnish Foundation for Alcohol Studies.

Petrakis, P. L. (1985). Alcoholism: An Inherited Disease. Washington, D. C.: U.S. Government Printing Office.

Petrie, A. (1967). Individuality in Pain and Suffering. Chicago: University of Chicago Press.

Peele, S. (1986). The implications and limitations of genetic models of alcoholism and other addictions. Journal of Studies on Alcohol, 47, 63-73.

Pollock, V. E., Volavka, J., Mednick, S., Goodwin, D., Knop, J. and Schulsinger, F. (1984). Electroencephalographic findings. In D. W. Goodwin, K. T. Van Dusen and S. A. Mednick (Eds.) Longitudinal Research in Alcoholism. Boston: Kluwer-Nijhoff.

Propping, P. (1977). Genetic control of ethanol action on the central nervous system: An EEG study in twins. Human Genetics, 35, 309-334.

Propping, P. L., Kruger, J. and Nark, M. (1981). Genetic predisposition to alcoholism: an EEG study in alcoholics and their relatives. Human Genetics, 59, 51-58.

Puchall, L. B. Coursey, R. D. Buchsbaum, M. and Murphy, D. (1980). Parents of high risk subjects defined levels of monoamine oxidase activity. Schizophrenia Bulletin, 6, 338-345.

Reider, E., Zucker, R., Maguin, E., Noll, R., and Fitzgerald, H. (1989, August). Alcohol involvement and violence toward children among high-risk families. Paper presented at the 97th annual meeting of the American Psychological Association, New Orleans, LA.

Roe, A. and Burks, B. (1945). Adult adjustment of foster children of alcoholic and psychotic parentage and the influence of the foster home. Memoir of the Section on Alcohol Studies. New Haven: Yale University Press.

Robins, L. Helzer, J., Croughan, J. and Ratcliffe, K. S. (1980). The NIMH Diagnostic Interview Schedule: Its history, chacteristics and validity. St Louis, MO:, Washington University School of Medicine.

Scarr, S. Environmental bias in twin studies. (1986). In S. G. Vandenberg (Ed.) Progress in Human Behavior Genetics. Baltimore: Johns Hopkins University Press.

Scarr, S. and Carter-Saltzmann, L. (1979). Twin method: Defense of a critical assumption. Behavioral Genetics, 9, 527-542.

Schuckit, M. A. (1980). Alcoholism and genetics: Possible biological mediators. Biological Psychiatry, 15, 437-447.

Schuckit, M. A. (1984). Subjective responses to alcohol in sons of alcoholics and control subjects. Archives of General Psychiatry, 41, 879-884.

Schuckit, M. A. and Rayses, V. (1978). Ethanol ingestion: Differences in blood acetaldehyde concentrations in relatives of alcoholics and controls. Science, 203, 54-55.

Searles, J. S. (1988). The role of genetics in the pathogenesis of alcoholism. Journal of Abnormal Psychology, 97, 153-167.

Selzer, R. (1975). A self-administered short Michigan Alcohol Screening Test. Journal of Studies on Alcohol, 36, 117-126.

Stevens, G. and Featherman, D. L. (1981). A revised socioeconomic index of occupational status. Social Science Research, 10, 364-395.

Tarter, R. E., Alterman, A. I. and Edwards, K. L. (1985). Vulnerability to alcoholism in men: A behavioral-genetic persepctive. Journal of Studies on Alcohol, 46, 339-356.

Thompson, W. D., Orvaschel, H., Prusoff, B., and Kidd, K. (1982). An evaluation of the family history method for ascertaining psychiatric disorders. Archives of General Psychiatry, 39, 53-58.

Vaillant, G. E. (1983). The Natural History of Alcoholism. Cambridge: Harvard University Press.

Vaillant, G. E. (1989). The pendulum swings the other way: The role of environment obscured by genes. Archives of General Psychiatry, 46, 1151.

Von Knorring, A-L., Bohman, M., Von-Knorring, L. and Oreland, L. (1985). Platelet monoamine activity as a biological marker in subgroups of alcoholics. Acta Psychiatrica Scandinavica, 72, 51-58.

Waller, M. B., Mc Bride, W. J., Gatto, G., Lumeng, L., and Li., T. K. (1984). Intragastric self-infusion of ethanol by ethanol preferring and non-preferring rats. Science, 225, 78-80.

Winokur, G., Reich T., Rimmer, J. and Pitts, F. N. (1970). Alcoholism III. Diagnosis and familial psychiatric illness in 259 alcoholic probands. Archives of General Psychiatry, 23, 104-111.

Wittchen, H., Burke, J., Semler, G., Pfister, H., VonCranach, M. and Zaudig, M. (1989). Recall and datings of psychiatric symptoms: Test-retest reliability of time-related symptom questions in a standardized psychiatric interview. Archives of General Psychiatry, 46, 437-443.

Zucker, R. A. (1987a). The four alcoholisms: A developmental account of the etiologic process. In P. C. Rivers (Ed.) Alcohol and Addictive Behavior: Nebraska Symposium on Motivation, 1986. Lincoln, NE: University of Nebraska Press.

Zucker, R. A. (1989). The Lifetime Alcohol Problems Score. Unpublished manuscript, Michigan State University, Department of Psychology, East Lansing, MI.

Zucker, R. A. and Davies, W. H. (1989). The revised Quantity- Frequency- Variability Index: Rationale and formulae. Unpublished manuscript, Michigan State University, Department of Psychology, East Lansing, MI.

Zucker, R. A. and Gomberg, E. A. (1986). Etiology of alcoholism reconsidered: The case for a biopsychosocial process. American Psychologist, 41, 783-793.

Zucker, R. A. and Noll, R. B.. (1980b). Drinking and Drug History. Unpublished manuscript, Michigan State University, Department of Psychology, East Lansing, MI.

Zucker, R. A. , Noll, R. B. and Fitzgerald, H. (1986). Risk and coping in children of alcoholics. Unpublished manuscript, Michigan State University, Department of Psychology, East Lansing, MI.