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Alcoholism as a discrete personality variable| Implications for its heritability and treatment

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ALCOHOLISM AS A DISCRETE PERSONALITY VARIABLE:

IMPLICATIONS FOR ITS HERITABILITY AND TREATMENT
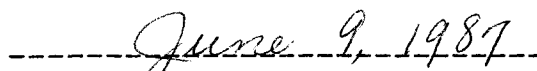

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Alcoholism as a Discrete Personality Variable:
Implications for its Heritability and Treatment (84pp)

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   There are at least two different kinds of personality variables, continuous variables and class variables. Continuous variables are dimensions or characteristics possessed to some degree by all individuals. Class variables are not distributed on a continuum, but rather are distributed into discrete classes.
   In this study the application of taxometric methods, based on a maximum covariance model, shows that alcoholism is a class variable rather than a continuous variable. As a class variable, alcoholism is possessed by only certain individuals and not by others. It is not on a continuum existing in some degree in all persons. Individuals belong either to the discrete class of alcoholics or to the discrete class of nonalcoholics. The implication of this result is that, as a class variable, alcoholism is much more likely to be inherited than if it were a continuous variable. Further evidence has thus been gathered to show that alcoholism has a genetic component. Implications of this finding for the treatment of alcoholism are also discussed.

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CHAPTER I

According to Gangestad and Snyder (1985), there are at least two types of personality variables: continuous variables and class variables. Continuous variables are dimensions or characteristics possessed to some degree by all individuals. Psychologists commonly refer to them as traits. For example, impulsivity--as measured by the Personality Research Form (Jackson, 1974)--is often thought to be on a continuum. Some people are more impulsive while others are less impulsive; however, all carry the trait or variable of impulsivity.

The other type of personality variable referred to by Gangestad and Snyder is the class variable. Class variables, as opposed to continuous variables, are not distributed on a continuum but rather are distributed into discrete classes. Class variable theory suggests that individuals differ not in degree but in kind. For example, if we now assume that impulsivity is a class variable with only two categories, we imply that only people belonging to one category have the trait of impulsivity; those belonging to the other category do not possess the trait.

Gangestad and Snyder (1985) argue that if comparative individual differences can be distributed

either as continuous dimensions or discrete classes then we may ask whether any specific difference between individuals is properly conceptualized as a continuous or as a class variable. For purposes of this thesis, the question then becomes: "Is alcoholism a class variable, possessed by only certain individuals and not by others, or is it on a continuous dimension possessed in some degree by all individuals?" If it is a discrete variable with two classes, then individuals belong to either the discrete class of alcoholics or to the discrete class of nonalcoholics, and this has very specific implications for the treatment of alcoholism.

If alcoholism is considered to be a class variable, we are assuming that all individuals belonging to the class--alcoholics--share some underlying entity, structure or event that affects their outward or phenotypic characteristics. The phenotypic characteristics of alcoholics include loss of control over drinking, problems with employment, legal and interpersonal difficulties due to alcohol use, blackouts, preoccupation with the chemical and personality changes such as overly aggressive behavior. This pattern of similar outward characteristics or phenotypic covariation can then be explained as the manifestation of the latent class variable: alcoholism.

By using measurement techniques explicated by Meehl

and Golden (1982), and utilized by Gangestad and Snyder (1985), we expect to be able to determine whether the latent variable underlying alcoholism is a class variable. Detection of the presence of a class variable in alcoholism could be of special interest in the debate surrounding the questions: (a) Is alcoholism heredity? (b) Can alcoholics be taught to control their drinking? A class variable may be more strongly argued to be hereditary and one who carries a gene (complex) for alcoholism may never find it possible to engage in controlled drinking.

The argument for a class model of personality versus a continuous model proceeds along the lines of the argument of a genetic versus an environmental approach to human behavior. Persons who argue for a genetic explanation of alcoholism development, such as Goodwin (1979), claim that there are certain individuals who are predisposed to the disorder because of a genetic influence. Proponents of an environmental explanation for alcoholism development, such as Roe (1945), claim that individuals become alcoholic because of environmental pressures. Class variables have a rather specific etiology that suggests a genetic influence, while continuous variables have a rather diffuse etiology, suggesting little or no genetic influence (Gangestad and Snyder, 1985). Thus, if alcoholism can

be determined to be a class variable, using Meehl's
(1977) taxometric techniques, it is much more likely to
be genetic in origin than if it is a continuous
variable.

There are at least two different classes of
individuals who consume alcohol. One of these classes
is alcoholic, while the other class is nonalcoholic
(controlled drinkers or social drinkers). Surrounding
the class of alcoholics, there exists an argument. Can
alcoholics be taught to control their drinking? One
position, the disease concept position, argues that
alcoholism is an either/or situation: that one is either
alcoholic or one is not, and if one is alcoholic, it is
highly unlikely that he or she can be taught to control
alcohol use. The disease concept proponents propose the
existence of a specific dichotomous etiological factor,
probably a threshold effect, operating in the
development of alcoholism. It seems likely that this
particular factor has its roots in genetics. The other
side of the argument maintains that there is no disease
process and that people are not necessarily, by class,
alcoholic or nonalcoholic and that they can be taught to
control their drinking. If it is possible to
conceptualize and measure the variable of alcoholism
along a continuous dimension, it would seem that
individuals who fall on the less alcoholic side of the

continuum of alcoholism may be able to control their drinking. However, if alcoholism is a class variable then alcoholism would seem to be an either/or situation and it would appear to be highly unlikely that alcoholics could be taught to successfully control their drinking. In that event, the treatment of choice would appear to be abstinence.

We should be able to use the taxometric methods of Meehl and Golden (1982) to detect the existence of latent class structures. These taxometric methods can be applied at any time one is able to conjecture the presence of a class variable. In alcoholism, a class structure can be conjectured on the basis of evidence that alcoholism is hereditary (Goodwin, 1979). Once the presence of a class structure is conjectured in this matter, a set of indicators--items from a drinking history scale and/or from measures such as the MacAndrew Scale (MacAndrew, 1965)--can be used to discriminate between the two classes. If alcoholism can be shown to be a class variable, and thus in all likelihood hereditary, arguments in favor of teaching controlled drinking would not seem to be cogent. The purpose of this study is to provide evidence that alcoholism is a class variable and therefore in all likelihood hereditary.

Before discussing the proposed methods used to

tease out whether alcoholism is a class or a continuous variable, we will examine the research that exists on the heritability of alcoholism and the related problem of controlled drinking.

THE GENETICS OF ALCOHOLISM

Because professionals find it almost impossible to agree upon a definition for the construct of alcoholism, it should come as no surprise that the etiology is uncertain. While most people are able to limit their alcohol use to quantities that do not interfere with occupational, familial, emotional, social and/or physical functioning, there are a few drinkers (10% by most estimates) who drink to the point of causing dysfunction in one or more of those areas. There is a difference of opinion as to whether the inability to control alcohol use is a function of social, psychological or genetic factors, with the best guess being that it is a combination of the three. Animal studies, genetic marker studies, family studies, twin studies and adoption studies provide evidence for a genetic influence on the development of alcoholism; that material and a brief discussion of what it is that is inherited will be presented below.

Family Studies

As Goodwin (1971) noted, the world-wide lifetime expectancy rate for alcoholism among males is between 3% and 5%; for females, it is between .1% and 1%. It has long been known that elevated alcoholism rates occur among family members of alcoholics and thus alcoholism is said to run in families. Goodwin (1971) cited several studies which found high alcoholism rates among family members. For example, Boss (1929) examined the siblings and parents of 909 male and 166 female alcoholics and found that alcoholism occurred in 53% of the fathers, 6% of the mothers, 30% of the brothers and 3% of the sisters. Winokur et al. (1968) found a high rate of alcoholism among the full siblings of identified alcoholics. Among the full siblings of male alcoholics, the lifetime expectancy for excessive drinking was 46% for the brothers and 5% for the sisters. The lifetime expectancy for alcoholism among the full siblings of female alcoholics was 50% for the brothers and 8% for the sisters.

These two studies are typical of the findings of other researchers studying the incidence of alcoholism within families. As Goodwin (1971, p. 54) noted, "Without known exception, every family study of alcoholism, irrespective of country of origin, has shown much higher rates of the disorder among the relatives of

alcoholics than apparently occurs in the general population."

There seems to be no doubt, based on the family studies, that alcoholism does run in families. The problem is to tease out whether alcoholism runs in families because of genetics, because of environment or because of an interaction between the two. In addition to the family method, four other methods have been used for studying the heritability of alcoholism. The four will be presented in increasing order of the confidence and generalizability that can be placed in their results.

Animal Studies

Self-selection experiments have been done in an effort to breed animals that will preferentially drink alcohol solutions over water. If it can be shown that some strains of mice can be bred to prefer alcohol over water solutions, evidence is provided for the genetic transmission of at least alcohol preference, if not alcoholism. Some studies reported by Goodwin (1976) found just such results. However, extrapolating from animal studies to humans is no small task. For an animal to resemble a human alcoholic, the animal would have to: "a) spontaneously drink enough alcohol to become intoxicated while foods and fluids of equal

caloric value were also available; b) drink enough to have withdrawal symptoms such as shakes and seizures when the alcohol is withdrawn, and c) drink to relieve these withdrawal symptoms when alcohol is again available" (Goodwin, 1976, p. 62).

Because of the generalizability problem, the evidence for human genetic transmission is extremely fragile and will not be reviewed in detail here. The interested reader is referred to Kissin (1976) for an in-depth discussion of the animal literature.

Genetic Marker Studies

If a positive association can be found between alcoholism and other characteristics that are known to be inherited, much support is given to the genetic argument. For example, if every color-blind individual in a family was alcoholic while none of the noncolor-blind individuals were, it would follow that alcoholism, just like color-blindness, is hereditary. Studies have been done (Goodwin, 1971) in an effort to link alcoholism with such inherited traits as color-blindness, ability to taste certain substances and blood types. The results of such studies are highly contradictory and unconvincing. For example, Cruz-Coke and Varela (1966) found that color-blindness, cirrhosis and alcoholism were associated and claimed that

alcoholism was transmitted by an X-borne recessive gene.
Fialkow, Thuline, and Fenster (1966) also found an
association between alcoholism and color-blindness but
discovered that the color-blindness disappeared when the
alcoholism subsided. Because of the contradictory
findings and the lack of clarity this brings to the
topic of heritability of alcoholism, the genetic marker
studies will not be addressed further in this paper.
For a more detailed review, the reader can consult
Goodwin (1971).

Twin Studies

An important method for examining the presence of a
genetic factor in the development of alcoholism is to
compare identical (monozygotic) twins to fraternal
(dizygotic) twins. This approach assumes that
monozygotic and dizygotic twins differ with respect to
genetic makeup but not with respect to environmental
influences. The prediction is that genetic disorders
will more often be concordant among identical twins than
among fraternal twins.

The first large-scale study to examine alcoholism
using the twin method was performed by Kaij (1966) and
was reported by Goodwin (1971). Kaij located 174 male
twin pairs in Sweden. At least one partner was
registered with a temperance board because of a

conviction for drunkenness or other alcohol abuse incidents. He conducted personal interviews and established zygosity by anthropometric and blood type measures. The concordance rate for alcohol abuse in the monozygotic twins was found to be 54%, while in the dizygotic twins it was 28%; the difference was statistically significant at the .05 level. Kaij also found that the more severe cases of alcoholism had higher concordance rates, indicating that the more severe forms may be more rigidly genetically determined.

The Kaij study discovered that social and intellectual deterioration were related to zygosity as well. A heavy-drinking monozygotic twin was more likely to have a light-drinking partner showing signs of deterioration than was a dizygotic twin with one partner who was deteriorated. Thus, alcohol-related deterioration seems to be linked to something other than alcohol consumption. From a Finnish study (Partanen, Bruun, & Markkanen, 1966) reported by Goodwin (1971), the evidence for a genetic predisposition to alcoholism is not so clear. Partanen et al. studied a large proportion of the twins born in Finland between 1920 and 1929. The subjects included 902 male twins between the ages of 28 and 37. Zygosity determination was based on a combination of anthropological measures and serological analysis. In an attempt to increase the

generalizability of their findings, the authors also examined a sample of brothers who were the same age as the twins. In contrast to Kaij's findings, Partanen et al. discovered no differences between identical and fraternal twins with respect to consequences from drinking. ¡As Goodwin (1971) noted, drinking consequences are probably the most widely accepted criterion for the diagnosis of alcoholism.¿ Frequency and amount of drinking were significantly more concordant among identical twins than among fraternal twins. Abstinence as well was more concordant among identical than fraternal twins. They found no evidence for heritability of arrests for drunkenness, nor for various social complications related to drinking. Partanen et al.'s findings seem to suggest that the severe forms of alcoholism are not as highly heritable as Goodwin (1976), Kaij (1966), Bohman (1978) and Cloninger, Bohman, and Sigvardsson (1981) indicate they are.

Other twin studies are commented on by Madden (1984). He reported that Hrubec and Omenn (1981) found a significantly higher concordance for alcoholism among identical twins than among fraternal twins. However, Gurling et al. (1981) found similar rates of alcohol dependence for both types of male twins and discovered an even higher concordance rate among fraternal female

twins. Loehlin (1972), Pedersen (1981) and Cederlof et al. (1977) found the concordance rates for heavy drinking to be higher among identical than fraternal twins. However, Cederlof et al. found no substantial differences for amount of consumption. Jonsson and Nijlsson (1968) examined questionnaire data from 7,500 Swedish twin pairs. They found no differences between the two types with respect to adverse consequences from drunking, nor did they find any differences between identical and fraternal twins with respect to amount of alcohol consumption. They did find a greater concordance between identical twins for the choice between abstinence and non-abstinence.

Weaknesses of the twin method, which may explain the contradictory findings, were examined by Goodwin (1971, 1976). First of all, there is the ubiquitous problem of defining alcoholism that continues to plague alcoholism research in general. Different studies may use varying criteria for diagnosing alcoholism and this may result in contradictory findings. Believers in a genetic basis for alcoholism may overdiagnose the disorder in identical twins and underdiagnose it in fraternal twins. The opposite, of course, may be true for those who lean toward an environmental explanation for the development of alcoholism.

Although it is assumed that identical twins and

fraternal twins are treated the same, this may not be the case. It is known that a person's appearance plays a large role in how he or she is treated by other people (Goodwin, 1971). Based on appearance, identical twins should be treated equally but this would not necessarily be so for fraternal twins.

Goodwin (1971) went on to make the point that identical twins, as opposed to fraternal twins, tend to develop deeper relationships with their partners and to have similar life experiences. These similarities could result in different environmental pressures for alcoholism development. Identical twins also tend to live longer and more often have similar vocational, educational and marital status than do fraternal twins.

Because of the methodological problems associated with the twin studies, adoption studies are believed to provide the most credible data for teasing out genetic from environmental effects upon the etiology of alcoholism. These studies will be looked at next.

Adoption Studies

In adoption studies, the adopted-away children of alcoholic biological parents are compared with the adopted-away children of nonalcoholic biological parents. An attempt is made to determine if the two groups of adoptees have different rates of adult

alcoholism. If the rate of adult alcoholism is found to be different for both sets of adoptees, evidence is provided for a genetic influence, since the environmental factors should be negated by the adoption. Because of the importance and confidence that is placed in their results, adoption studies will be presented in some detail here.

Roe's 1945 Study. The first adoption study to examine the issue of alcoholism was conducted by Roe (1945). She obtained information about 49 foster children of both sexes. Their ages ranged from 20 to 40. Twenty-two of them were from "normal" parentage, and 27 of them had a biological parent described as a heavy drinker. It was found that 70% of the children of heavy-drinking parentage used alcohol while 64% of the children from "normal" parentage used alcohol. Roe discovered that the adopted-away children of heavy drinkers had more adjustment problems; however, these differences were small. Since no individuals in either group developed drinking problems as adults, it was concluded that there was no evidence for a genetic predisposition to alcoholism.

The Roe study has been criticized by Goodwin (1976) and Bohman (1981). The major objections include the lack of a firm diagnosis of alcoholism in the "heavy-drinking" parents and small sample size. In addition,

children of heavy drinkers were older at time of
adoption placement and were more frequently placed in
rural areas or small towns where the risk of alcoholism
was less than in urban areas. None of the heavy-
drinking parents had ever been treated for alcoholism
and it is unclear that they really were alcoholic.

Goodwin's 1973 Study. Goodwin, Schulsinger,
Hermansen, Guze, and Winokur (1973) looked at 55 male
adoptees chosen from a pool of 5,483 adoption cases in
Denmark from 1924 to 1947. The sample consisted of
children who had a biological parent with a record of
hospitalization for alcoholism. The adoptees had been
separated from their biological parents before the first
six weeks of life and were adopted by nonrelatives.
They had no known contact with their biological
relatives subsequent to adoption. Two control groups
were chosen using the above criteria, with one
exception: none of the members of the control groups had
a biological parent with a record of hospitalization for
alcoholism or alcohol abuse. The two control groups
differed in that one of the groups had a biological
parent who had been hospitalized for a psychiatric
disturbance other than alcoholism. No members of the
other control group had a parent with a record of
hospitalization for psychopathology. Since analysis
showed no significant differences between the two

control groups, they were pooled to form one control group of 78 subjects.

The only demographic variable that distinguished between the controls and the probands was the divorce rate. There were three times as many divorces among the probands than among the controls. The adoptive parents of the probands and the controls were found to be similar in terms of depression, alcoholism and other psychopathology.

Analysis of variance on the two groups indicated that only severe alcoholism distinguished between the two. As compared with the controls, the probands had significantly more (p <.05) hallucinations, treatment for drinking (p <.05), morning drinking (p <.02), loss of control (p <.02) and alcohol-related problems including marital trouble, employment difficulties, police trouble and drunken-driving arrests (p <.02). Goodwin classified persons as moderate, heavy, problem and alcoholic drinkers. The controls had about as many moderate drinkers as the probands (45 as opposed to 51). The controls included more (although not statistically significantly more) heavy drinkers (36 as opposed to 22). There were also more problem drinkers among the controls as well (14 as opposed to 9) but again statistical significance was not reached. It has been suggested by Goodwin (1976), Bohman (1978) and Cloninger

et al. (1981) that severe forms of alcoholism appear to be especially susceptible to genetic influence. Based on Goodwin's (1973) findings, it may very well be that severe alcoholism is not on a continuum with social and problem drinking but is discretely distributed as a separate entity.

Remarkably, there was no difference between the groups with respect to various other problems including drug abuse, depression, other psychopathology and heavy smoking. It is particularly striking that genetics seemed to play a larger role in the development of alcoholism than it did in the development of disorders such as depression and drug abuse.

The 1974 Goodwin Study. Goodwin et al. (1974) compared drinking problems and other psychopathology in sons of alcoholics raised by their alcoholic biological parents with drinking problems and other psychopathology in their brothers who had been adopted away. Thirty-five siblings of 20 of the original Goodwin subjects were located and examined. The environment of the adoptees presumably was of a quality that would lessen the risk of alcoholism development and as a result the adopted-away children should have a lower rate of alcoholism as adults.

Several environmental variables were examined and reported on in the study. The biological parents were

of relatively low social class as compared to the adoptive parents. The sons who remained in the biological parents' homes were of lower socioeconomic status as adults than were their adopted-away brothers. The non-adopted brothers seemed to have had a more disruptive childhood and more school problems. There was more psychopathology among the biological parents than among the adoptive parents.

It was discovered that, while the adopted and non-adopted sons differed significantly with respect to personality disturbances (the incidence of personality disturbance was higher in the adoptees), they did not differ significantly with respect to alcoholism. The authors concluded that foster care did not lessen the risk for development of alcoholism.

Bohman's 1978 Study. In an effort to investigate the presence of a genetic predisposition to criminality as well as to alcoholism, Bohman (1978) looked at adoptees born in Stockholm, Sweden, between 1930 and 1939. The study was confined to children adopted away prior to age 3 (most of them had been separated from their biological parents in the first few months of life). The Swedish Criminal Register and Excise Board (alcohol abuse registration) were perused to determine the presence or apparent absence of criminality and/or alcohol abuse. Criminality was defined as the

imposition of a sentence of more than 60 "day fines" (a fine assessed on the basis of a defendant's daily income). Adoptees whose biological parents appeared in the register for alcohol abuse and/or criminality were compared with adoptees whose biological parents had no such record. In an effort to keep the two factors as separate as possible, subjects with a parent appearing in both registers were excluded.

Male adoptees with a biological father registered for alcohol abuse had a significantly greater representation in the official register than did adoptees whose biological father was not registered (p <.01). Male adoptees whose biological mother was registered for alcohol abuse likewise had a higher registration rate than did those whose biological mother was not registered (p <.01).

However, male adoptees whose father had a criminal record alone were not overly represented in the criminal register. Twelve and one-half percent of them were registered as compared to 12.0% of those whose fathers had no such record. Similar findings were presented for female adoptees as well (12.6% as compared to 12.4%).

The risk of alcoholism or criminality could not be adequately determined for female adoptees, because so few of them were registered.

The Bohman data suggested that, while there appears

to be a genetic component in the development of alcoholism, no such conclusion can be drawn for criminality. Bohman (1978) claimed that different results for two different types of social problems adds strength to the argument that there is a genetic predisposition to alcoholism. If bias were operating in the study, it should apply equally to both alcoholism and criminality.

In a follow-up control study, Bohman (1978) found nearly identical results. Adoptees were matched with controls on the variables of age, sex, age at time of placement, occupational category of the adoptive parents, and ages of the biological and adoptive parents at the time of the child's birth. A correlation was found between the biological parents' alcohol abuse and their sons' alcohol abuse but there was no firm relationship between criminality in the biological parents and their sons.

The 1981 Cloninger Study. Operating under the assumption that susceptibility to alcoholism is a function of genetic and environmental interaction, Cloninger et al. (1981) examined the inheritance of alcoholism in 862 Swedish men adopted by non-relatives. The average age at time of placement was 8 months. At the time of the study, their ages ranges from 23 to 43. The Cloninger group used cross-fostering analysis, a

technique used to examine each possible combination of genotype and environment, to determine how adoptees with particular types of congenital backgrounds reacted to different types of adoptive placement.

Cloninger et al. identified four different patterns of adoptee alcohol abuse: a) non-abusers; b) mild abusers--had one registration for abuse by the Temperance Board and had never been treated for alcoholism; c) moderate abusers--2 to 3 registrations for alcohol abuse without treatment; d) severe abusers-- 4 or more registrations and compulsory treatment or psychiatric hospitalization with a diagnosis of alcoholism.

Based on the cross-fostering analysis, two types of alcohol abuse were identified. Biological fathers of type 1, milieu-limited, alcoholics were characterized by mild alcohol abuse, minimal criminality and no alcoholism treatment. The mothers of the milieu-limited alcoholics were characterized by mild abuse and minimal criminality. The post-natal environment was shown to determine both the frequency and the severity of the alcoholism in the susceptible sons. The alcoholism was marked by usually isolated and mild problems, although at times the problems were severe. With post-natal (environmental) provocation, the calculated risk of alcoholism in congenitally-predisposed sons was twice as

high as the risk for the geneal population. Without
post-natal provocation, the relative risk for
development of alcoholism was the same as for the
general population.

Biological fathers of the type 2, male-limited,
alcoholics were characterized by severe alcohol abuse,
severe criminality and extensive treatment for
alcoholism. The biological mothers resembled the
general population. The post-natal environment did not
affect the frequency of the sons' alcoholism. It could,
however, affect the severity. The alcoholism was
characterized by recurrent and moderate problems which
could be severe at times. The calculated risk in
congenitally-predisposed sons in this group was found to
be nine times that of the general population, regardless
of the post-natal environment.

Thus, like Goodwin, Cloninger found that there
seemed to be a type of alcohol abuse that was passed
from father to son, was highly heritable and was
associated with the biological father's extensive
treatment for alcohol abuse.

The 1978 Cadoret and Gath Study. Cadoret and Gath
(1978) looked at 84 adoptees chosen from among adopted
infants born in Des Moines from 1939 to 1965. At the
time of the study, all were age 18 or older. They had
been separated from their biological parents at birth

and had no further known contact with the biological parents.

Age of adoptee, time spent in foster care, age of the biological mother at time of birth, socioeconomic status of adoptive home, psychopathology other than alcoholism in the biological parents, and behavioral problems in the adoptive family were all unrelated to adoptee alcoholism. Adoptee childhood conduct disorder was positively, although not significantly ($p <.06$), correlated with alcoholism in the adoptees. Alcoholism in the biological parents (as defined by two or more social or medical complications associated with alcoholism, or hospitalization for detoxification) was highly correlated with the development of alcoholism in their children ($p <.001$).

The 1980 Cadoret Study. Cadoret, Cain, and Grove (1980) examined 92 male subjects aged 18 and over. Adoptees raised apart from their alcoholic biological parents were compared with adoptees raised apart from their nonalcoholic biological parents. Environmental factors including psychiatric or alcohol problems in the adoptive family, exposure to discontinuous mothering, and socioeconomic status of the adoptive family did not significantly distinguish between the two groups. Presence of a first-degree biological relative with alcoholism ($p <.03$) and presence of alcoholism in a

second-degree biological relative ($p < .02$) did distinguish between the two groups. In addition, adoptee childhood conduct disorder approached significance in predicting the development of alcoholism in the adoptees as adults. ($p < .06$).

Patrilineal Transmission. Because of the findings that indicate patrilineal (father to son) transmission of alcoholism (Goodwin et al., 1973; Bohman, 1978), Bohman, Sigvardsson, and Cloninger (1981) decided to study a population of female adoptees to see if a similar pattern existed for them.

The authors examined a population of 913 adopted women. Among them were 307 alcoholic biological fathers and 51 alcoholic biological mothers. The adoptees' ages ranged from 23 to 43. Like the Cloninger (1981) study, this study employed the technique of cross-fostering analysis in an attempt to tease out the relative importance of genetic and environmental influences. However, unlike Cloninger et al., they could not find any significant environmental effects operating in the development of alcoholism.

They found that if the biological mother was the alcohol-abusing parent, the risk of the daughter's alcohol abuse was increased four times (10.3% as compared to 2.8%, $p < .05$). However, if the biological father was the alcohol abuser, then the risk for alcohol

abuse in the daughters was not significantly greater than the control group's risk (3.5% as compared to 2.8%, $p > .50$). If both biological parents were alcohol abusers, the daughters had a greater risk for alcohol abuse than the controls but the difference was not significant (9.8% as compared to 2.8%, $p < .10$).

Summary of the Adoption Studies. While the adoption studies nearly unanimously implicate a genetic component in the development of alcoholism, they are not without methodological problems of their own (Goodwin et al., 1973; Goodwin, 1976). Although these problems do not seem so severe as the methodological problems of twin studies, they do need to be addressed; a brief summary of these difficulties follows.

The adoptees spent at least the first few weeks of life in the care of their biological mother. These mothers may have differed in unsuspected ways from the mothers of nonalcoholics. For example, they may have been alcoholic themselves or had other forms of unreported psychopathology.

It is possible that infants with a known alcoholic parent were matched with less desirable foster parents. However, since the adoptive parents of the two groups did not differ with respect to educational or economic status, this selective bias does not seem to be a major difficulty.

Cloninger et al. (1981) raised the objection that the information about the adoptive parents was gathered from the adoptees themselves and as such might not be accurate.

Bohman (1981) claimed that transmission of alcoholism may be mediated, not by genetic factors, but rather by the intrauterine or lactational enviornment. He (1981) described a model for this in which mice exposed to alcohol-selecting mothers during pregnancy or lactation drank more alcohol than mice not so exposed. However, the Goodwin et al. (1973) study of male adoptees indicated otherwise. In that study, 85% of the biological parents who were alcoholic were the fathers and thus hardly could have contributed to the intrauterine or lactational enviroments.

Madden (1984) claimed that interstudy differences might be the result of changing definitions or measurements of alcoholism between studies. Madden also asserted that the selective choice of subjects through their volunteer status or antisocial activity that brought them the attention of the studies in the first place might have biased the results.

Despite the methodological flaws discussed above, there does appear to be a good deal of evidence supporting a genetic predisposition to at least certain types of alcoholism. Assuming this to be the case, the

next question that needs to be answered is: "What is it that is inherited?"

WHAT IS INHERITED?

There may be many factors that underlie a genetic influence in alcoholic vs. nonalcoholic individuals (Schuckit, 1979). These include the possibility of a unique reaction to a single dose of alcohol. For example, high-risk individuals may receive greater pleasure from the ingestion of alcohol, while low-risk individuals may receive greater discomfort from it.

Goodwin (1979) reported on a number of cases which indicate that genetic control is an important factor regulating drug metabolism. These studies report that identical twins metabolize a wide variety of drugs, including alcohol, at nearly identical rates, while fraternal twins show varying rates of metabolism. With alcohol, there seems to be an implication of the metabolic step in the liver where ethyl alcohol is broken down by the enzyme alcohol dehydrogenase into acetaldehyde which, in turn, is broken down into acetic acid by the enzyme aldehyde dehydrogenase. This enzymal activity appears to be different in alcoholics than it is in nonalcoholics. The actions of acetaldehyde and its role in the development of alcoholism will be

discussed next.

Differences in Acetaldehyde Levels

Milam and Ketcham (1981) reported that Lieber (1976) discovered the same amount of alcohol produced much higher blood acetaldeyde levels in alcoholics than in nonalcoholics. Lieber hypothesized that this was due to malfunctioning of the liver enzyme system. However, Lieber's study had a circularity problem because it could not answer the question, "Does the metabolic abnormality result from alcoholism or is it present prior to its development?

Schuckit's (1979) research seemed to solve the nagging circularity question and suggested that there is a difference prior to the onset of the disorder. After screening out alcoholics, Schuckit selected 20 males who had an alcoholic parent or sibling and matched them with controls with no familial alcoholism. After drinking ethanol-7up conbinations over a 5-minute period, blood acetaldehyde concentrations were gathered at 15 and 30 minutes and subsequent half-hour levels during the following three hours. Acetaldehyde levels differed significantly between the two groups at each interval (p <.004). Schuckit (1979, p. 54) speculated that "the increased acetaldehyde levels could mediate the short-term effects of alcohol, resulting in an altered

(perhaps heightened) state of intoxication. It is equally possible that the individuals predisposed to alcoholism are more vulnerable to organ damage from acetaldehyde. This higher acetaldehyde plateau might facilitate the formation of condensation products with monoamine metabolites resulting in the production of addicting morphine-like alkaloids." ¡It should be noted, nowever, that the ability of modern techniques to accurately measure acetaldehyde levels in human tissue may be questionable. A discussion of those measurement problems is beyond the scope of this paper; the interested reader is referred to Wartburg (1980)¿.

The Tetrahydroisoquinolines

The role of the acetaldehyde metabolites, the isoquinolines, in the development of alcoholism has stimulated a good deal of interest and research. Some of these findings will be briefly summarized below.

The tetrahydroisoquinolines (THIQs) are formed through a condensation reaction between aldehydes such as acetaldehyde and catecholamines such as dopamine. One of the THIQs (tetrahydropapaveroline or THP) is found naturally in the poppy plant where it acts as an intermediary in the biosynthesis of morphine. In addition to being precursors of morphine, a known addicting and euphoriant drug, they are thought to be

addictive themselves (Blum, Hamilton, Hirst, & Wallace, 1978).

Collins and Bigdeli (1975) produced evidence that one of the THIQs, salsolinol, can be produced in the brain of live rats pretreated with pyrogallol and then given ethanol intraperitoneally. Pyrogallol was used to increase the blood acetaldehyde levels because without it no traces of salsolinol could be found. The authors suggested that the pyrogallol increased acetaldehyde concentrations to the point where they resembled the concentrations that result from the chronic ingestion of alcohol by human alcoholics. The suggestion here is that, for some reason--possibly genetic, acetaldehyde levels need to reach a certain point before the production of the THIQ is possible.

Myers and Melchoir (1977) produced abnormal alcohol intake in laboratory rats by exposing them to tetrahydroisoquinolines. Rats who preferred water to alcohol solutions were given alcohol solutions that were increased in step-wise fashion from 3 to 30% over a period of 12 successive days. THP was delivered directly into the cerebral ventricles of the rats automatically every 15 minutes for 12 days. The animals were given a choice between water and the gradually-increasing alcohol solutions. Within three to six days from the start of the THP infusion, the rats (who

normally wouldn't drink alcohol solutions at all) began to drink them in ever-increasing amounts. They drank to the point of intoxication and had withdrawal symptoms upon removal of the alcohol.

In a follow-up study, Myers (1978) found that the pattern of excessive alcohol drinking continued up to 6 months later, indicating that the action of the alkaloids might be irreversible. Myers (1978) suggested that alcoholics and nonalcoholics differ in the manner in which their bodies handle the THIQs, as follows:

1.   The isoquinolines may be formed peripherally (as opposed to within the brain itself) and do not cross the blood-brain barrier until repeated bouts of heavy drinking actually damage the barrier, allowing them access to the brain. Nonalcoholics probably don't drink enough to cause this damage.

2.   The nonalcoholic may not form the metabolites peripherally in the appropriate concentrations or in the correct chemical structure to exert an influence on behavior.

3.   Genetically, the biochemical makeup of the alcoholic may allow the production of the chemicals within the brain itself rather than peripherally; increased alcohol intake would cause the alkaloids to be synthesized in increased amounts.

4.   Perhaps the alcoholic does not enzymatically

degrade the metabolites fast enough to prevent them from being stores in the brain. When eought is stored over a long period of time, abnormal intake of alcohol is triggered.

5. There may be a specialized interaction within the alcoholic individual involving monoamine, amino acid or calcium ions.

At least four of the five postulates offered by Myers could be explained by differential genetic influences in the alcoholic as oppoed to the nonalcoholic individual.

The Biphasic Problem

It has been noted by Agarwal, Harada, and Goedda (1981) that some North American Indians, Japanese, Chinese and other Orientals exhibit frequent signs and symptoms of high blood acetaldehyde concentrations. These signs and symptoms include increased facial flushing, increased skin temperature, peripheral vasodilation, higher heart rates, nausea, abdominal discomfort and chest distress.

Agarwal et al. (1981) produced experimental evidence of low levels of aldehyde dehydrogenase in Japanese liver tissue. Low levels of this enzyme could explain abnormally high acetaldehyde concentrations in the bloodstream. High blood acetaldehyde levels would

explain the hypersensitivity to alcohol that is seen in some Japanese. This hypersensitivity is unique to certain races and lends credibility to the genetic argument.

Additionally, the rate of alcoholism in Oriental people in general is much lower than the rate throughout the rest of the world (Milam & Ketcham, 1981). The inability to tolerate alcohol because of elevated blood acetaldehyde levels could explain this situation. However, a dilemma is posed here. How is it that increased acetaldehyde concentrations create an aversion to alcohol in some people and yet seem to lead to an affinity for alcohol in others? Perhaps further research will provide answers to this most intriguing question.

Alcoholism and the Electroencephalogram

As Pollock et al. (1983) noted, it has been known for years that the electroencephalograms (EEGs) of chronic alcoholics are poorly synchronized. In addition, it has been found that alcohol improves the synchronization of the EEG (Propping, Kruger, & Mark, 1981).

Some of the first evidence that these brain wave differences may be genetically determined was produced in a twin study performed by Propping (1977). He found

that the EEGs of identical twins reacted similarly to alcohol while the EEGs of fraternal twins varied. This discovery was compatible with the findings of Vogel, Schalt, and Kruger (1979) as reported by Pollock et al. (1983).

Propping et al. (1981) presented evidence suggesting that persons with a genetic predisposition to alcoholism might be characterized, in part, by deficient alpha activity. Pollock et al. (1983) hypothesized that, without alcohol, these people might never reach the pleasant states associated with alpha brain wave activity and hence might be more vulnerable to the effects of alcohol.

In an effort to determine whether alpha brainwave activity differences were present prior to the development of chronic alcoholism, Pollock et al. administered ethyl alcohol to the biological sons of alcoholics and to a group of men whose biological fathers were not alcoholic. After alcohol administration, the sons of the alcoholics exhibited greater increases in slow alpha energy and greater decreases in fast alpha energy than did the controls. The results suggest that sons of alcoholics may be physiologically more sensitive to the effects of alcohol and that this sensitivity is manifested, in part at least, in the EEG.

Other recent research has focused on evoked brain potential (EBP) (Elmasian, Neville, Woods, Schuckit, & Bloom, 1982). These studies measure a single brain wave in response to a stimulus. The authors discovered that EBPs from normal drinkers with a family history of alcoholism and EBPs from normal drinkers without a family history of alcoholism were significantly different. EBPs elicited in conjunction with subjects' decisions about task-relevant stimuli were of lower amplitude in those persons with a family history of alcoholism. In addition, both the latency of the positive component and reaction times to correctly detected targets were significantly later in individuals with a family history of alcoholism. These differences were found both before and after the ingestion of alcohol.

Begleiter, Porjesz, Bihari, and Kissin (1984) provided the first evidence that inferred neurological deficits might be present prior to alcohol abuse. They measured the voltage of the P3 wave, a brain wave related to attention and learning, in 7- to 13-year-old sons of alcoholic fathers. The boys were asked to make decisions about a picture of a head displayed at various angles, and during this process the voltage of the P3 wave was measured. Begleiter et al. found that, when compared to a control group, the sons of alcoholics had

a significant deficiency in P3 wave voltage.

The findings that individuals with a possible genetic predisposition to alcoholism seem to have a neurological deficit is interesting but it doesn't explain why they are vulnerable to alcoholism if they drink. The deficits could be linked to chemical abnormalities or they could be associated with behavioral problems. Further research is needed to help clarify the role of the nervous system in the development of alcoholism.

CONTROLLED DRINKING VS. ABSTINENCE

The term "controlled drinking" was first introduced into the literature by Reinert and Bowen (1968) to describe an observed outcome of alcohol treatment in which the patient resumed moderate drinking by observing strict rules of self-control. For the purposes of this thesis, the terms "normal drinking" and "controlled drinking" will be used interchangeably to indicate this type of alcohol use.

Davies' 1962 Study

The controlled drinking and abstinence controversy began when Davies (1962) presented the first evidence that some alcoholics apparently had been able to attain

normal or controlled drinking status.

No follow-up of the Davies' research was reported until Edwards (1985) published an article presenting evidence that questioned Davies' findings. Edwards attempted to reconstruct the history of the seven Davies subjects from the time of their discharge from Maudsley Hospital until 1983. Of the seven, Edwards found that five had not been able to maintain the controlled drinking over either the length of Davies' original follow-up or subsequently. The other two remaining subjects were able to engage in trouble-free drinking over the period. However, Edwards raised the point that they may never have been truly dependent in the first place. In addition to addressing the problem of defining dependency, Edwards suggested that future studies should utilize corroboration of the subjects' self-reports with those of concerned others and official records.

While the Edwards research was the first scientific questioning of Davies' results, it should not be concluded that the Davies research went unnoticed. For example countless letters referring to the Davies research were sent to the editors of <u>Quarterly Journal on Alcohol Studies</u> over the next decade.

A particularly comprehensive and widely quoted review of the literature was published by Pattison

(1966). In the review, Pattison attacked the notion that abstinence is the only reasonable goal in alcoholism treatment. In particular, Pattison addressed the issue of treatment evaluation and other problems centering around the difficulty of defining alcoholism. Because of the wide range of alcoholism syndromes, the various stages of the illness and the may types of personalities who become alcoholic, Pattison claimed that variable methods of treatment are needed.

Armed with the Davies study and the Pattison review, behaviorally-oriented psychologists began to question whether or not alcoholism could be treated successfully by teaching alcoholics to control alcohol consumption. After Mark and Linda Sobell introduced the concept of Individualized Behavior Therapy for alcoholism, they became the focus of the abstinence and controlled-drinking controversy. The Sobells collaborated with Pattison (1977) in a review of the literature, citing 74 studies which appeared to produce evidence that some alcoholics could successfully return to normal drinking. The original Sobell (1973) study has become the focal point for the current controversy between abstinence and controlled drinking. Because the widely-quoted and controversial Sobell (1973) study is representative of other controlled-drinking studies, that study and its follow-up will be discussed. The

famous Rand Report (1976) will also be looked at briefly.

The 1973 Sobell Study

Subjects of the Sobell (1973) study were 70 male patients, all diagnosed as gamma alcoholics (Jellinek, 1960) because they had withdrawal symptoms and deteriorated health, financial and social status due to drinking. The patients were voluntarily admitted to Patton State Hospital in California from April 1970 to February 1971. All of them volunteered to participate in the research study. Based on an interview, those who could socially identify with Alcoholics Anonymous (AA), requested abstinence and/or lacked social support for controlled drinking were always assigned to the non-drinking condition. Persons who requested controlled drinking and had significant outside support for it were considered for the controlled drinking condition. After the treatment goal was established, the subjects were randomly assigned to a control group receiving the conventional hospital treatment (group therapy, AA, drug, physio- and industrial therapy) or an experimental group receiving 17 behavioral treatment sessions in addition to the conventional treatment. Thus, each of the treatment groups differed only in the treatment goal. Twenty of the subjects were assigned to a group

with a controlled-drinking goal and designated as CD-E.
Their matched controls were designated as CD-C. Fifteen
of the subjects were assigned to a group with a
treatment goal of abstinence and were designated as ND-
E. The remaining 15 subjects were assigned to be their
controls and were designated as ND-C.

At six-week and six-month follow-up, information
was obtained from the subjects and their collateral
information sources. In terms of functioning well or
not functioning well, the difference between CD-E and
CD-C subjects was significant (p <0.05) at the six-week
follow-up. The authors saw evidence for the
continuation of the trend at six months, although a
statistical analysis was not done because at the time of
the report only 48 of the 70 subjects had been due for
six-month follow-ups. Differences between the ND-E and
ND-C were not significant at the six-week interval but
at six months the differences were found to be
significant (p <.0.05).

Indices of behavioral change--including vocational
status, use of therapeutic supports and evaluation by
collateral sources of the subjects' general functioning-
- indicated that both the CD-E and ND-E subjects were
doing significantly better than their controls. It
appeared, therefore, that the treatment paradigms of
abstinence and controlled drinking were both equally

effective.

Caddy's Third-Year Follow-Up to the Sobell Study

Caddy, Addington, and Perkins (1978) conducted a third-year independent follow-up study of 53 of the 70 subjects of the original Sobell study. In addition to directly contacting the subjects, the authors interviewed collateral information sources. In terms of percentage of days abstinent and vocational status (job satisfaction), both the controlled drinking and the abstinent subjects appeared to be functioning better than their respective controls. In terms of percentage of days drunk, occupational status (actual state of employment), general health and index of general adjustment, the controlled-drinking subjects appeared to be functioning better than their controls. On these same measures, there were no apparent differences between the abstinent subjects and their controls. In terms of percentage of days controlled drinking, percentage of days incarcerated and drinking control index, there was no difference in functioning between the two groups and their respective controls. Thus, the Caddy et al. study affirmed the Sobell conclusion that controlled drinking and abstinence were equally effective treatment goals for alcoholism.

A Re-evaluation of the Sobell and Caddy Studies

The Sobells concluded in their book (1978) that many of the CD-E subjects engaged in limited, nonproblem drinking throughout the follow-up period. Therefore, it seemed to them that controlled drinking was an effective therapy for alcoholism. However, both the Sobell (1973) and Caddy et al. (1978) studies must be questioned in light of evidence presented by Pendery, Maltzman, and West (1982).

Pendery et al. addressed only the issue of whether or not controlled drinking is a desirable treatment goal for alcoholism. The authors were not concerned with whether or not the CD-E subjects fared better than their controls. Their findings were significantly different from those of Sobell and Caddy et al. and will be summarized below.

In addition to interviews with the patients, Pendery et al. examined the records of hospitals and other facilities. One of their major concerns was that, although all 20 of the CD-E subjects were reported to have withdrawal symptoms and therefore gamma or late-stage alcoholism, only 16 actually met the criteria completely. The other four did not seem to have the necessary withdrawal symptoms. Of those 16, thirteen were rehospitalized for alcoholism treatment within approximately one year of discharge. The remaining

three of the 17 reported unfavorable outcomes centered
around alcohol-related hospital admissions. These
reports were substantiated by hospital records. It is
also noteworthy that two of these were among the six
listed by Caddy (1978) as enjoying the most satisfactory
outcomes. They were reported by Caddy to be functioning
well 100% of the year.

Findings were similar for the four with respect to
questionable dependence. One stated that, upon
discharge from the research project, his drinking
worsened and he lost his job. After surgery, he
moderated his drinking but still got very intoxicated on
weekends. Multiple alcohol-related arrests did not
occur until later in the Pendery et al. follow-up. Two
of the other Sobell successes reported intermittent
excessive drinking but no arrests until after the third
year follow-up.

On long-term follow-up, Pendery et al. found that
eight controlled-drinking subjects continued to drink
excessively, either repeatedly or intermittently,
throughout the follow-up and had one or more of the
following verified alcohol-related consequences from
1979 to 1981: job loss, arrests, marital breakup and
hospitalization for alcoholism and related serious
physical illness. Six of the controlled-drinking
subjects were abstaining completely by the end of the

follow-up. Four had suffered alcohol-related deaths
included heart attack, ethanol-induced respiratory
failure, suicide and drowning. The drowning victim had
a blood alcohol concentration of .30, which is three
times the legal limit for intoxication in most states.
One could not be located and one was an apparent success
although, as mentioned earlier, it is doubtful that he
was a gamma alcoholic in the first place.

The Rand Report

The third significant publication that lent
credence to the position that alcoholics could be taught
to control their drinking and that, indeed, controlled
drinking was as attainable a treatment goal as
abstinence was the famous Rand Report (Armor, Polich, &
Stambal, 1976).

This research team looked at data from an original
pool of 14,000 non-DWI (driving while intoxicated)
clients admitted into hospitalized treatment at 44
National Institute on Alcohol Abuse and Alcoholism
(NIAAA) treatment centers (ATC) throughout the country
from September 1972 until April 1974.

Results indicated substantial improvement on a
number of measures for clients of NIAAA treatment
centers. The rate of improvement approached 70% for
consumption and behavioral impairment. Social

adjustment yielded a mixed outcome, with gains made in employment and income but no change in marital status. Both the six-month and the 18-month follow-ups yielded remission rates of nearly 70%. The authors concluded that remission was independent of controlled drinking or abstinence. When relapse rates were examined, they were found to be just as low for the normal drinkers as for the long-term abstainers and independent of signs of physical addition.

SUMMARY

Evidence has been presented that strongly suggests vulnerability to alcoholism is at least partially genetic in origin. Whether that genetic predisposition is transmitted as a biochemical, neurological or some other abnormality remains uncertain at this time. We will try a new approach to this area using the techniques developed by Meehl and Golden (1982), and explicated by Gangestad and Snyder (1985), to see if we can provide further evidence that genetics plays a role in the development of alcoholism.

CURRENT STUDY

Much of the literature on alcoholism supports the
assumption that underneath its development lies an
attribute or structure that either alone or by
interaction with the environment causes some persons to
develop alcoholism when they drink while others do not.
To understand why it is more likely that a class
variable--rather than a continuous variable--is genetic
in origin, we need first to consider the etiology of the
two variables. Of prime importance to this
understanding is the concept of normality. The
continuous variable is likely to be normally distributed
because numerous independent antecedent events have all
contributed to its development (Gangestad & Snyder,
1985). This diffuse pattern of etiology is compatible
with learning theory. Strict learning theory would
subscribe to the notion that it is not genetics but
environment that plays the major role in the development
of personality. Personality does not result from an
underlying genetic predisposition but rather unfolds
because of our interaction with the environment.

Class variables, on the other hand, are not
normally distributed; they are discretely distributed.
The etiology is not diffuse, it is specific. As
Gangestad and Snyder (1985, p. 321) note: "Specific

etiology refers to the operation of a necessary and sufficient factor, or a necessary but not sufficient factor, which is itself a discrete entity." In other words, if alcohol dependence were entirely genetic in origin, the latent class variable would be the only factor necessary for its development. It has been shown that, while genetics probably plays a role in the development of alcohol dependence, the environment is important as well. Thus, it seems more likely that the latent variable is a necessary but insufficient factor in the development of dependence. More simply put, class variables are discretely distributed and therefore diffuse causation is not the proper model for this conceptualization. The observable behaviors descriptive of alcohol dependence are probably not the result of the interaction of independent antecedent events but rather are the result of an interaction between the underlying genetically-based factors and the environment.

Testing the Class Model

As explained by Gangestad and Snyder, before we test for the presence of a class model, we should have at least some minimal theoretical or empirical reasons for postulating the existence of a class variable. At least two kinds of reasons are sufficient for this: a) one should have reason to believe that a particular

etiology produces consistent behavioral mainfestations; this is called the etiological springboard to a class model, and b) if one is aware of "contemporaneous causal relationships that specify differences between individuals in kind rather than in degree, or that proposes that individuals possess discretely different internal structures that influence behavior" (Gangestad & Snyder, p. 322), then one has a contemporaneous-theoretical springboard.

Based on the evidence gathered in the introduction to this paper, there exists sufficient reason to conjecture that the etiology of the behavior seen in alcohol dependence is at least partly genetic in nature. Therefore, we have sufficient reason to test for the presence of a class variable in the etiology of alcoholism.

Utilizing the Technology

Because of the newness of the techology, we will reproduce the arguments that originated with Meehl and were later delineated by Gangestad and Snyder. The basis for this technology is provided by the following assumption (Gangestad & Snyder, 1985): if a class variable exerts strong influence on some domain of observable events, then these events are discontinuously, rather than continuously, distributed.

Specifically, a class variable will exhibit a particular pattern among the covarience of its indicators. Thus, we will examine the covariances among a set of indicators between alcoholics and nonalcoholics and see if they exhibit this pattern. If we look at covariances lover the levels of the underlying variable, the plot of the covariances should be peaked toward the middle.

The methods of Meehl and Golden can be used to detect the presence of a latent class variable. These methods can be used when the state of the knowledge allows one to conjecture the presence of a dichotamous class variable and to supply a set of indicators believed to discriminate between the two classes. Since we are able to conjecture both the presence of a class variable and to provide a set of indicators to distinguish between the two classes, we should be able to proceed with this method.

CHAPTER II

METHOD

The Indicators

The Drinking History Questionnaire, the MacAndrew
Scale, the Comprehensive Drinker Profile, the Mortimer
Filkens Test and the Western Personality Inventory were
examined for 60 items (Appendix A) from which we could
choose a set of ten indicators. A pilot study was
conducted on the 60 items to determine which items were
related between groups but not within groups. The items
were given to 50 alcoholics, identified as such by their
responses to the Drinking History Questionnaire
(Appendix B) and to 50 nonalcoholics, identified as such
by responses to the Drinking History Questionnaire.
Criteria for classification are included in Appendix C.
Those ten items (Appendix D) with the highest
correlation between groups but lowest correlation within
groups were chosen for the actual study. Ideally, the
items should only intercorrelate in a sample because
they discriminate between the two classes. They should
be relatively independent of one another in order to map
the construct more thoroughly. For example, we would
not want to choose two obviously highly correlated items
such as "I have a hard time stopping drinking after one

or two drinks" and "I sometimes find it difficult to stop drinking once I have started."

Subjects

The indicators were given to 125 male alcoholics, identified as such by responses to the Drinking History Questionnaire. The indicators were also given to 200 male nonalcoholics, identified as such by responses to the Drinking History Questionnaire. Females were excluded due to apparently differing base rates for alcoholism.

The alcoholic subjects were males involved in alcoholism treatment at Galen State Hospital, Warm Springs, Montana, Montana State Prison, Deer Lodge, Montana, Rocky Mountain Treatment Center, Great Falls, Montana, Providence Treatment Center, Great Falls, Montana, and Recovery Foundation, Missoula, Montana. They also included members of Alcoholics Anonymous in Missoula, Montana and Psychology 110 students at the University of Montana. The Psychology 110 students participated in the study as a course requirement. The rest of the alcoholic population volunteered to complete the questionnaires. The average age of the alcoholics was 31.0

The nonalcoholic subjects included members of Bethel Baptist, First Evangelical, and Christian

Missionary Alliance Churches in Missoula, Montana. All
volunteered to complete the questionnaires. Other
nonalcoholic subjects were Psychology 110 students at
the University of Montana who completed the
questionnaires to fulfill course requirements. The
average age of the nonalcoholics was 37.8

Data Analysis

Covariances among the items were plotted by level
of response to the indicators (Figure 1). The
covariance between each of 45 possible item pairs was
plotted for eight levels of responses to the indicators.
The levels ranged from 0 alcoholic responses to the
remaining 8 items to 8 alcoholic responses to the
remaining 8 items. For a detailed explanation of this
method see Appendix E.

COVARIANCE X 10

CORRESPONDING 9-POINT SCALE

CHAPTER III

<u>RESULTS</u>

Figure 1 clearly shows that the plot of the
covariances among items by level of response to the
indicators peaks toward the middle (at number 3). As
mentioned previously, this particular pattern is
indicative of the existence of a class variable. In
addition, an independent base rate estimation yielded a
base rate of .381 (meaning that the original population
was 38.1% alcoholic and 61.9% nonalcoholic). To obtain
that figure, for each individual item the total number
of alcoholic responses was divided by the total
population. That figure was then averaged for the ten
items. The resulting average was .381. This compares
with an actual sample of 38.5% alcoholic and 61.5%
nonalcoholic. Since this calculation is independent of
the computation of the covariance plot, it provides
independent support for a genetic basis for alcoholism.
It should be noted that attempts to use Meehl and
Golden's (1982) methods of base rate estimation, based
on sketchy descriptions, failed to produce reasonable
values and Meehl and Golden's counsel is being sought
concerning the computations involved. This matter not
withstanding, the excellent agreement between the

simplest base rate estimation and the proportion of

alcoholics in the sample provides support for the results.

CHAPTER IV

DISCUSSION

The results clearly indicate that alcoholism has a latent class variable underlying its development. The plot of the covariances by level of responses to the indicators yielded a curve that was unmistakably peaked in the central part, indicative of the existence of a class variable. In addition, working backwards, the base rate for alcoholism in our population was reproducible from the data. Since this is independent of the computation used to obtain the covariance curve, it provides a control measure and strengthens the argument that alcoholism is a class variable. It either exists or it does not; there is not a continuum.

These results are analogous to the findings of Gangestad and Snyder (1985), who discovered that self monitoring, too, is a class variable. The results of the present study are made all the more remarkable by the fact that we were able to obtain results consistent with those of Gangestad and Snyder without the benefit of an extremely large sample size. In their study, Gangestad and Snyder used a population of 1918 individuals. The present study was able to obtain results using a population of 325. This should be

encouraging to others who may be considering utilizing the taxometric methodology, but who are concerned about the sample size required for adequate results.

Before moving on to the implications of these results, a control measure devised by Gangestad and Snyder merits some discussion, as it, too, strengthens the argument for the ability of the maximum covariance method to detect class personality variables. Gangestad and Snyder wanted to see if the maximum covariance method would fail to detect a class variable when it should. In other words, would one obtain the peaked maximum covariance curve applying the methods to a continuous variable? To study this Gangestad and Snyder examined impulsivity. They matched measures of impulsivity with measures of self monitoring in terms of (a) average intercorrelations between items, (b) range of intercorrelations between items, and (c) range of item difficulties. They then performed the same taxometric analysis and found no peakedness in the covariance curve. In addition, the base rate estimation of latent classes was not consistent with the presence of a latent class variable. Thus, it is clear that the methods will fail to detect a class variable when there is none present.

As Gangestad and Snyder (1985) suggest, there are cases, such as self-monitoring, and now alcoholism,

where the data support the contention that people differ in kind rather than in degree. Future research with this methodology could prove fruitful in understanding the origins of other personality characteristics as well.

Implications for Alcoholism Development and Treatment

The results of this study may be most important for the understanding they provide concerning the origins of alcoholism and the implications for its treatment. There is evidence from family, twin, animal, adoption studies and now, from the unique perspective of taxometric analysis, that alcoholism has a major, if not overriding, genetic component. Of all these, taxometric analysis, alone, avoids the pervasive problem of alcoholism definition. As such, it provides the most conclusive evidence yet for the heritability of alcoholism. As mentioned earlier, the existence of a class variable suggests specific as opposed to diffuse etiology. A class variable, with its specific etiology, is much more likely to be genetic in origin than is a continuous variable, with its diffuse etiology.

In looking at alcoholism treatment, there are nearly always two arguments that are encountered. One is whether alcoholism results from personality problems and life difficulites or whether alcoholism causes

personality problems and life difficulties. In using the same set of data, Vaillant and Milofsky (1983), and Zucker and Lisansky Gomberg (1986) reach different conclusions. Vaillant suggests that life problems stem from alcoholism and Zucker and Lisansky Gomberg suggest that alcoholism results from life problems. Given the present results, we cannot ignore the importance that genetics plays in the biology of alcoholism. There is a major genetic component to alcoholism and it only makes sense that the predisposed individual be extremely careful with his or her alcohol use.

This conclusion leads to the second argument one encounters: the controlled drinking versus abstinence argument. This controversy is a continuing one (Taylor, Helzer and Robins, 1986; Cook, 1985). This study adds to the growing body of evidence that alcoholism is not merely learned; there are real differences between the alcoholic and the nonalcoholic. An analogy can be drawn to something as simple as diabetes and sugar. Just as the diabetic can best avoid symptoms of diabetes by avoidng sugar, so can the alcoholic best avoid symptoms of alcoholism by avoiding alcohol. With intake under control, proper treatment can help the alcoholic deal with the psychosocial aspects of the problem and learn how to prevent relapse.

The implications for prevention of alcoholism are

clear. Those with a family background of alcoholism must be made aware of the large risks inherent to them should they make the personal choice to use the chemical. And, given the high probability of relapse (Brownell, Marlatt, Lichtenstein, and Wilson, 1986) the most sensible approach to the problem is probably prevention. The key to prevention is education. With adequate education concerning the risks genetics poses for the development of alcoholism, people will be able to make informed decisons concerning their chemical use before alcoholism has had a chance to develop.

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APPENDIX A

60 ALCOHOLISM INDICATORS

1. Drinking helps me feel more confident.
2. Drinking seems to ease personal problems.
3. I sometimes feel bad about my drinking.
4. I am always able to stop drinking when I want to.
5. I have neglected my obligations, my family, or my work for two or more days in a row because of drinking.
6. I have had trouble remembering what I did the night before while I was drinking.
7. I sometimes need a drink or two in the morning to get going.
8. I have gone to someone for help with my drinking.
9. I am able to drink more now than I used to without feeling the same effect.
10. Friends and relatives think I am a normal drinker.
11. My relatives are upset with the way I live.
12. I am sometimes bothered by nervousness (irritable, fidgety or tense).
13. My judgement is better than it ever was.
14. I have recently undergone a great stress.
15. I have never been in trouble with the law
16. I sweat very easily even on cold days.
17. I am moderate in all my habits.
18. I do not feel that I have abnormal problems.
19. I have lived the right kind of life.
20. I would like to wear expensive clothes.
21. I like to read newspaper articles on crime.
22. I can not keep my mind on one thing.
23. I wish that I could be as happy as others seem to be.
24. My home life is as happy as it should be.
25. Drinking helps me make friends.
26. There is a history of problem drinking in my family.
27. Much of the time I feel that I have done something wrong or sinful.
28. I enjoy a race or game more when I bet on it.
29. I like (or liked) school.
30. I readily become one hundred per cent sold on a good idea.
31. I am certainly lacking in self-confidence.
32. I wish people would stop telling me how to live my life.
33. A drink or two gives me energy to get started.
34. 4 or 5 drinks affect my driving.
35. I have never been in trouble with the law.
36. I know who is responsible for most of my troubles.
37. My drinking has never caused problems between my

spouse (boyfriend/girlfriend, other family members) and me.

38. I often become quarrelsome and abusive when I drink.
39. I like to cook.
40. My parents often objected to the kind of people I went around with.
41. I am a good mixer.
42. I frequently notice that my hand shakes when I try to do something.
43. I was fond of excitement when I was a child.
44. Evil spirits possess me at times.
45. Many of my dreams are about sex matters.
46. I seem to make friends about as quickly as others do.
47. I drink when I get angry.
48. If I were a reporter, I would very much like to report sporting news.
49. I have few or no pains.
50. I drink because I need it when I am tense or nervous.
51. I have a cough most of the time.
52. I pray several times every week.
53. I drink because I like the taste.
54. I do many things which I regret afterward (I regret things more or more often than others seem to).
55. I drink when I want to forget everything.
56. My table manners are not quite as good at home as when I am out in company.
57. I drink because it helps me to forget my worries.
58. In school, I was sometimes sent to the principal for cutting up.
59. My soul sometimes leaves my body.
60. I have been quite independent and free from family rule.

Appendix B

DRINKING HISTORY QUESTIONNAIRE

1. What are your present drinking habits?
   (Check one or more of the following, as they apply)
   _____ Daily drinking
   _____ Evening drinking
   _____ Weekend Drinking
   _____ Social drinking, drinking with friends at parties, bars
   _____ Occasional very heavy drinking due to emotional stress, celebrations,
           other reasons (specify) _____
   _____ Other (specify) _____

2. Think of the times you have been drinking recently. On an average, how many drinks
   did you have?

_____ 1-2	_____ 7-8	_____ 13-14
_____ 3-4	_____ 9-10	_____ 15-16
_____ 5-6	_____ 10-12	_____ 17-18
		_____ 19 or more

3. Have you consumed any alcohol in the past two months?
   _____ Yes   _____ No

4. How many days ago was your last drink? _____days

5. Have you drunk daily in the past two months?
   _____ Yes   _____ No

6. Do you find it almost impossible to live without alcohol?
   _____ Yes   _____ No

7. Have your periods of not drinking alcohol been longer in the past two months than
   in any other previous two month period?
   _____ Yes   _____ No

8. What do you usually drink?
   _____ Beer   _____ mixed drinks
   _____ Wine   _____ straight drinks

9. Are you always able to stop drinking when you want to?
   _____ Yes   _____ No

10. Where do you do most of your drinking?
    _____ At home
    _____ Away from home (bars, lounges, restaurants, parties, etc.) Specify _____
    _____ Other (specify) _____

11. Do you drink during your work day?
    _____ Yes   _____ No

12. With whom do you do your drinking?
    _____ alone   _____ with friends   _____ people from
    _____ family   _____ neighbors   work

13. Were your drinking habits ever different from what they are now?
    _____ Yes   _____ No

14. If you answered yes to Question 13, what were your habits previously?
    (Check one or more of the following as they apply)
    _____ Daily drinking including before noon and/or on the job
    _____ Evening drinking
    _____ Weekend drinking
    _____ Social drinking, drinking with friends at parties, bars
    _____ Occasional very heavy drinking due to emotional stress, celebrations,
          other reasons (specify) _____
    _____ Other (specify) _____

15. If you answered yes to Question 13, when and why did your drinking habits change?
    _____ began drinking because of marital problems
    _____ began drinking because of job problems
    _____ began drinking because of group of friends
    _____ stopped drinking for some reason: (specify) _____

16. Is it difficult for you to stop drinking after one or two drinks?
    _____ Yes   _____ No

17. Do you consider yourself to be:
    _____ very light drinker   _____ fairly heavy drinker
    _____ fairly light drinker   _____ heavy drinker

18. What were the drinking habits in your parents' home?
    (Check one or more of the following as they apply)
    _____ drinking not allowed in the home
    _____ drinking on social occasions only
    _____ regular moderate drinking
    _____ regular heavy drinking (By whom?) _____
    _____ one or more family members with drinking problem (Who?) _____

19. Do most of your friends drink?
    _____ Yes   _____ No

20. Do friends or relatives think you drink more or less than other people who drink?
    _____ less   _____ more   _____ same

21. Do you feel that you drink more or less than other people who drink?
    _____ less   _____ more   _____ same

22. What is your attitude about driving after drinking?
    (Check one or more of the following as they apply)
    _____ I have no rule about this.
    _____ I don't take any special care in my driving after drinking.
    _____ I drive after drinking (often, sometimes, seldom, never). (Underline what fits)
    _____ I make special efforts to avoid driving after drinking (i.e. by taking a taxi,
          leaving car home, having a friend drive me).

23. How long have you been employed at your present job?
    _____ not employed   _____ 4 months
    _____ 1 month   _____ 5 months
    _____ 3 months   _____ 6 months or more

24. What was your family income last month? (include all sources)
    _____ 0-50   _____ 100-200   _____ 400-600
    _____ 50-100   _____ 200-400   _____ more than 600

25. What was your personal income last month?
    _____ 0-50   _____ 100-200   _____ 400-600
    _____ 50-100   _____ 200-400   _____ more than 600

26. How many hours do you spend on the job a week?
_____ Not Employed   _____ 30-45 hours
_____ Less than 15 hours   _____ Over 45 hours
_____ 15-30 hours

27. In the last two months has your salary:
_____ decreased?
_____ remained the same ?
_____ increased ?

28. Has your drinking caused you to lose a job?
_____ Yes   _____ No

29. Have you gotten into trouble at work because of drinking?
_____ Yes   _____ No

30. Have you ever neglected your obligations, your family, or your work for two or more days in a row because you were drinking?
_____ Yes   _____ No

31. How many days in the last year did you miss from work (or take sick leave) because of drinking?
_____ 1-3 days   _____ a week or more
_____ 4-5 days   _____ none

32. Do you presently have any hobbies or special interests outside your job?
_____ Yes   _____ No

33. Do you feel you could still do better for yourself as far as your vocation or work is concerned?
_____ Yes, I could do much better for myself.
_____ Yes, some improvement is possible.
_____ No, I am satisfied with my present vocational status.

34. Have you had any severe medical problems in the past two months?
_____ Yes   _____ No
Specify: _____

35. In the past two months has your drinking gotten
_____ worse?
_____ about the same?
_____ better?

36. Have you awakened the morning after some drinking the night before and found that you could not remember a part of the evening before?
_____ Yes   _____ No

37. Do you need a drink the "morning after" to get rid of a hangover?
_____ Yes   _____ No

38. Would you like assistance with drinking problems at this time?
_____ Yes   _____ No

39. How would you describe your overall health?
_____ Below average
_____ Average
_____ Above average

40. Have you attended any therapy sessions over the past month?
_____ Yes   _____ No

41. Have you ever attended a meeting of Alcoholics Anonymous (AA) other than as a guest?   _____ Yes   _____ No

42. Have you ever passed out in the past year due to excessive drinking?
_____ Once or twice   _____ A few times   _____ Never

43. Have you ever been told that you have liver trouble or cirrohsis?
_____ Yes   _____ No

44. Have you had delirium tremen (D.T.'s), severe shaking, heard voices, or seen things that weren't there after heavy drinking?
_____ Yes   _____ No

45. Have you ever gone to anyone for help about your drinking?
_____ Yes   _____ No

46. Have you ever been in a hospital because of drinking?
_____ Yes   _____ No

47. Have you ever been a patient in a psychiatric hospital or on a psychiatric ward of a general hospital where drinking was a part of the problem?
_____ Yes   _____ No

48. Have you ever been seen at a psychiatric or mental health clinic or gone to a doctor, social worker, or clergyman for help with an emotional problem in which drinking had played a part?   _____ Yes   _____ No

49. People drink for different reasons. How important would you say that each of the following is to you as a reason for drinking? (Put a check mark in proper column for each item.)

	very important	fairly important	not at all important
a. I drink because it helps me to relax.	_____	_____	_____
b. I drink to be sociable.	_____	_____	_____
c. I like the taste.	_____	_____	_____
d. I drink because the people I know drink.	_____	_____	_____
e. I drink when I get angry.	_____	_____	_____
f. I drink when I want to forget everything.	_____	_____	_____
g. I drink to celebrate special occasions.	_____	_____	_____
h. A drink helps me to forget my worries.	_____	_____	_____
i. A small drink improves my appetite.	_____	_____	_____
j. I accept a drink because it's the polite thing to do in certain situations.	_____	_____	_____
k. A drink helps cheer me up when I'm in a bad mood.	_____	_____	_____
l. I drink because I need it when I'm tense and nervous.	_____	_____	_____

50. Put check mark in proper column for each item.

Have you:	Often	Sometimes	Seldom	Never
felt tense or nervous?	_____	_____	_____	_____
felt suspicious?	_____	_____	_____	_____
felt worried about things?	_____	_____	_____	_____
felt jealous?	_____	_____	_____	_____
felt depressed, lonely?	_____	_____	_____	_____
felt angry?	_____	_____	_____	_____
had difficulty sleeping?	_____	_____	_____	_____
had thoughts of suicide?	_____	_____	_____	_____
attempted suicide?	_____	_____	_____	_____

51. Have you gotten into fights, verbal or physical, when drinking?
_____ Yes   _____ No

52. Drinking sometimes has an adverse effect on people. Check those of the following if they apply to you.
    _____ Quarrelsome and abusive language
    _____ Physical abuse
    _____ Failure to support family (missing work, etc.)
    _____ Undependable when drinking, irresponsibility, absent from home
    _____ Fear and worry about you by family
    _____ If none of the above, how are you affected? _____

53. Have you ever been arrested, even for a few hours, because of drunk behavior (other than this DUI)? _____ Yes _____ No

54. Have you been charged with any drunken driving offenses, besides the one for which you were referred to us in the past six months?
    _____ Yes _____ No

55. Have you had any personal crises in the past six months such as death in the family, severe illness? _____ Yes _____ No

56. In the past two months, has your relationship with people
    _____ become worse? _____ remained the same? _____ become better?

57. Do you ever feel bad about your drinking?
    _____ Yes _____ No

58. Does your spouse (boyfriend/girlfriend, other family members, or friend) ever worry or complain about your drinking?
    _____ Yes _____ No

59. Has your drinking ever created problems between you and your spouse (boyfriend/girlfriend, other family members, or friend)?
    _____ Yes _____ No

60. Has your spouse (boyfriend/girlfriend, other family members, or friend) ever gone to anyone for help about your drinking?
    _____ Yes _____ No

61. Have you ever lost friends or girlfriends/boyfriends because of drinking?
    _____ Yes _____ No

62. Over the past six months, do you feel your living conditions have
    _____ become worse? _____ remained the same? _____ become better?

63. Has your circle of friends changed in the past six months? _____ Yes _____ No

64. Do you think you have a problem with (or because of) drinking?
    _____ Yes _____ No _____ Unsure

65. Do you believe that you have alcoholism?
    _____ Yes _____ No _____ Unsure

APPENDIX C

CRITERIA FOR IDENTIFYING ALCOHOLICS USING THE

DRINKING HISTORY QUESTIONNAIRE

A. Anyone who exhibits any one of the following:

 1. Two or more previous alcohol related arrests.

 2. Loss of control of drinking.

 3. Prior diagnosis of alcoholism by a competent authority.

B. Anyone who exhibits two or more of the following
    indicators.

 1. One prior alcohol related arrest.

 2. Employment problems due to drinking.

 3.   Previous contact with social or medical facilities for
    problems where drinking was involved.

 4. Blackouts.

 5. D.T.s

 6. Passed out due to drinking.

 7. Cirrhosis or fatty liver.

 8. Shaking--especially in the morning after drinking.

 9. Family and/or social problems as a result of drinking.

APPENDIX D

THE TEN INDICATORS

This questionnaire is for males only. Be sure to put your age in the upper right hand corner. It is not necessary to give your name.

For these questions a true or false answer is needed. Please respond to every statement. Do not spend too much time on any one question. Answer each question in the order in which it appears. In the space to the left of the number of the question, place an F for statements that don't fit for you and a T for statements that do fit for you. There are no right or wrong answers.

1. I am always able to stop drinking when I want to.
2. I often become quarrelsome and abusive when I drink.
3. I have lived the right kind of life.
4. I drink when I get angry.
5. My drinking has never caused problems between my spouse (boyfriend/girlfriend, other family members) and me.
6. I am moderate in all my habits.
7. I sometimes need a drink or two in the morning to get going.
8. I have neglected my obligations, my family, or work for two or more days in a row because of drinking.
9. My home life is as happy as it should be.
10. I drink because it helps me to forget my worries.

APPENDIX E

<u>PREDICTING A PEAKED COVARIANCE CURVE</u>
(Adapted from Gangestad and Snyder, 1985)

Consider the two items, i and j, selected from the set of the eight conjectured items. If two classes exist within any sample (for convenience, let us call them the class of highs and the class of lows), it is an algebraic truth that the sample covariance between the two indicators is equal to the sum of three terms:

$$cov(ij) = p\ covh(ij) + q\ covl(ij) + pq\ \Delta i \Delta j,$$

where

p = the proportion of highs in the sample;
q = the proportion of lows in the sample;
covh(ij) = the covariance between the indicators within the subsample of highs;
covl(ij) = the covariance between the indicators within the subsample of lows;
Δi = the difference between the mean i scores within the subsample of highs and within the subsample of lows; and
Δj = the difference between the mean j scores within the subsample of highs and within the subsample of lows.

We have ideally assumed that the two indicators are independent within the classes and thus that the within-class covariances are equal to zero. If this assumption holds, then the only source of covariance within the total sample will be the third term in the expression above. Thus,

$$cov(ij) = pq\ \Delta i \Delta j$$

Of course, before we have started we do not know what p and q are for any given sample, nor do we have estimates of i or j for any given population nor, in fact, do we know whether two classes do actually exist. As the above formula reveals, however, if two classes do exist (and when Δi and Δj are held relatively constant), we expect the covariance between i and j in a sample to be some function of the relative proportions of the two classes p and q. Thus, for instance, if we could somehow select a pure sample of alcoholic individuals, we would expect cov(ij) to be near zero because cov(ij) = (1.00)(.00)   i j = 0. Similarly, if we could select a pure sample of nonalcoholic individuals, we would also expect cov(ij) to be near zero. Suppose now that we select a sample of 1/4 one class and 3/4 of the other. Then we would expect cov(ij) to be other than zero because (.25)(.75) $\Delta i \Delta j$ = (.1875)   i j. Moreover, if i and j are keyed in the conjectured direction, as we assume here, then we would expect this value to be positive. And, if we select a sample of 40% of one

class and 60% of the other class, we would expect some larger value still because $(.40)(.60)$ i j .1875 i j. Finally, it is a simple mathematical truth that because the product pq is maximal when there exist equal numbers from each class in the sample (i.e., $p = q = 1/2$), as long as i and j are held constant, cov(ij) is also expected to be maximal when $p = q = 1/2$.

Given this fact, we can create a powerful bootstraps effect (Cronbach & Meehl, 1955). For our item pair i and j, we take the remaining six items of our conjectured eight item pool and construct a 7-point scale (with values ranging from 0-6). If, as we have already assumed, these six items discriminate between the classes, then this small scale also discriminates between the classes. And, if our items i and j do not highly correlate with any of the six items within the classes, as we have also already assumed, then i and j will not correlate very highly with the small scale within the classes. Let us now use this 7-point scale to select different subsamples, each corresponding to the set of individuals who obtained a given score on the scale. If the above conditions hold (once again, testable for fit afterwards) and if two classes really do exist, then the seven different subsamples we have created should have a different p and q. The seven subsamples, however, should have similar Δi and Δj. (These latter values, in fact, should be similar to i and j for the entire sample.)

If two classes exist and if the smaller of the two classes is large enough so that the latent frequency distributions on the 7-point scale cross, then there will exist a scale value within which $p=q= 1/2$. Moreover, if the latent frequency distributions are monomodal and are not too unequal in size (so that the smaller of p and q equals at least .2), this value will be located somewhere toward the middle of the scale. Samples associated with values toward the extremes are expected to be composed of more disparate p and q. Given our previous results, this expectation yields the following prediction: If a class variable underlies responses to the items as conjectured, the seven sample covariances between i and j plotted as a function of the values on the 7-point scale should be peaked--maximal toward the middle and nearer to zero toward the extremes.