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Cognitive Functioning of Male Adult Children of Alcoholics and Control Subjects While Under the Influence of Acute Alcohol Intoxication

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COGNITIVE FUNCTIONING OF MALE ADULT CHILDREN OF ALCOHOLICS AND CONTROL SUBJECTS WHILE UNDER THE INFLUENCE OF ACUTE ALCOHOL INTOXICATION

by
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This thesis, submitted by Bette Bakke in partial fulfillment of the requirements for the Degree of Master of Arts from the University of North Dakota, has been read and is hereby approved by the Faculty Advisory Committee under whom the work has been done.

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This thesis meets the standards for appearance, conforms to the style and format requirements of the Graduate School of the University of North Dakota, and is hereby approved.

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ABSTRACT

Previous research has suggested that adult children of alcoholics (ACAs) are at increased risk for the development of alcoholism. Differences between ACA and control subjects have been reported for a range of cognitive, affective and behavioral measures in addition to certain components of the auditory evoked potential, supporting speculation that biological or psychological markers exist as predictors of future alcoholism. The present study examined 20 males ACA and 20 male control subjects under either placebo or alcohol experimental conditions using cognitive measures (Digit Span, Trail Making, Digit Symbol) at baseline, peak and descent phase of the session that have been associated with proposed evoked potential and neuropsychological deficits among subjects with a positive history for alcoholism. The results failed to demonstrate predicted baseline deficits among ACA subjects in any of the dependent measures or placebo expectancy effects from either group. These previous results demonstrating cognitive deficits in ACA functioning were discussed in terms of research designs that possibly were confounded by subject drinking histories. The ACA subjects were found to demonstrate superior recovery of function on the Digit Span backward test at the descent phase of testing. These results appeared to support a hypothesis that ACA's are less influenced and recover faster from the effects of acute

alcohol intoxication. This conclusion would appear consistent with previous studies describing diminished mood state changes, decreased sensitivity to bodily sensations and underestimates of blood alcohol levels by ACA subjects. Recommendations for future research are provided.

CHAPTER I

INTRODUCTION AND LITERATURE REVIEW

Research and clinical observations have shown that alcoholism is a complex problem facing the western world that cannot be explained by a simple psychological or biological model. In the last forty years a prolific amount of research has been done attempting to find its pathogenesis. Many theories have been advanced to account for its etiology. For example, there are biological models (Goodwin, 1979, 1985; Cloninger, Reich, Sigvardsson, von Knorring & Bohman, 1988; Porjesz & Begleiter, 1983; Hrubec & Omenn, 1981) neuropsychological models (Begleiter & Porjesz, 1984, 1987; Elmasian, Neville, Woods, Shuckit, Bloom, 1982, Pfefferbaum, 1980), social-learning models (Collins & Marlatt,1981), tension-reduction (Cappel & Herman, 1972) and expectancy theories (Goldman, Brown, & Christiansen, 1987) , the self-awareness model (Hull, 1981), the self-handicapping model, (Berglas & Jones, 1978) and the opponent-process theory (Shipley, 1987) all contributing to the understanding of alcoholism. Although it has been shown that biological, psychological and social factors are relevant to the mediation of drinking behavior, an unequivocal etiological model has yet to emerge.

The present study examined the effects of family history for alcoholism and acute intoxication on cognitive processes that have been shown to be associated with evoked potential components found most sensitive to blood alcohol levels and family history for alcoholism. The cognitive abilities examined in this study also have been shown to be impaired in chronic alcoholics. Elevated blood alcohol levels have been found to strongly influence particular evoked potential components among non-alcoholic adult subjects, and some of these same brain wave response patterns have been observed among chronic alcoholics not under the influence of alcohol. Most interestingly, studies are emerging to indicate that male biological offspring of alcoholics also show some of the same evoked potential deviations as those observed among chronic alcoholics and non-alcoholic subjects while under the influence of alcohol. Moreover, alcoholic patients appear to show deficits in verbal-nonverbal learning and memory task performance, abstract reasoning abilities and perceptual-spatial motor skills (Porjesz & Begleiter, 1988; Kleinknecht & Goldstein, 1972; Leckliter & Matarazzo, 1989.)

Adult Children of Alcoholics (ACA) and Risk for Alcoholism

Reports of increased risk for alcohol abuse among ACA's have been common. Claydon (1987) estimated that ACA's were four times more likely to report a possible drinking problem. Goodwin's (1979) adoption studies reported similar findings among adoptee offspring in the general Denmark population. The national average for alcohol consumption has been estimated at around 0.96 ounces or

approximately two drinks per day. (Khavari & Farber, 1978; Nobel, 1978). Male subjects reporting alcoholism in both parents estimated their average daily consumption at 4.6 ounces of ethanol (approximately 9 drinks) in a recent study of college students (Schumacher, 1990). The female subjects in the Schumacher study reported an average consumption of 1.8 ounces, and both groups were significantly higher than male and female control subjects reporting 1.2 and 0.94 ounces respectively. Wallace (1989) estimated that 80 to 85% of the patients who enter treatment centers report alcoholism in their immediate families. The increased risk for male adult alcohol abuse among ACA's appears fairly well established.

Research comparing psychological variables associated with acute alcohol intoxication between male ACA and nonACA subjects revealed subjective response differences between the two groups (Savoie, Emory & Moody-Thomas, 1988; Vogel-Sprott & Chipperfield, 1986; O'Malley & Maisto, 1985; Schuckit, 1980, 1984). Using self-report measures such as the Subjective High Assessment Scale (SHAS) (Schuckit, 1982), the Sensation Scale(SS) (Maisto, Connors, Tucker, McCollam & Adesso, 1980) and the Multiple Affect Adjective Checklist (MAACL) (Zuckerman & Lubin, 1965) it has been consistently shown that male ACA subjects are generally less sensitive to the subjectively perceived effects of alcohol than male nonACA subjects despite comparable blood alcohol levels. These finding are particularly more pronounced under moderate doses of alcohol (Schuckit, 1984). Because ACA males show a comparative insensitivity to the subjective effects of alcohol leading to a less

efficient monitoring of their alcohol consumption, it has been speculated that this may be a factor which contributes to the development of alcoholism.

Convincing evidence for genetic influences on adult alcoholism will be followed by a review of the literature attempting to isolate predictors or markers that identify these high risk individuals prior to the experience of drinking problems. Literature discussing neuropsychological and cognitive impairment found in alcoholics appear to offer the most promise of achieving this important task. The present literature review addresses genetic linkages , P300 evoked potentials, neuropsychological and cognitive findings, and cognitive and EP correlates in succession.

Genetic Linkages

Benefiting from the genetic clinical studies with schizophrenia and affective disorders, researchers have been able to follow the same methodological approaches in studying alcoholism. One approach has been to establish that the disease runs in the family or that there is a familial vulnerability to the disease. Cloninger et al. (1988) looked at the changes in alcohol use with respect to cohort effect. The temporal trends that are occurring in the United States reflect an increase in alcohol consumption per capita. This increase necessitated a different approach in analyzing the inheritance of alcoholism, specifying the age of onset, cumulative lifetime risk of men and women in each cohort, in addition to the parameters of the models of inheritance. It was found that the lifetime risk for alcoholism in the general population

has increased, but that the risk for women appears lower than that for men. One finding that is frightening is the observed trend of higher risk to younger subjects. For example, the risks of developing alcoholism by age 25 was shown to increase with the year of birth. Their study showed that men born before 1924 have a 34% risk, those men born between 1925 and 1934 show a risk of 44% compared to 52% risk for men born from 1935 to 1944. Those born between 1945 and 1954 evince a 63% risk for development of alcoholism compared to the 67% risk for those born after 1954.

As a result of their Swedish adoptee studies, Cloninger, Sigvardsson & Bohman (1988) also found evidence for two types of alcoholism. According to Cloninger and colleagues, the Type 2 alcoholism appears to be entirely genetic in nature and is limited to males. It was found that male offspring whose parents show this type of alcoholism are at a nine times greater risk for developing alcoholism. Type 2 alcoholism is characterized by an early onset, usually in the early teens, petty criminality and an inability to abstain from alcohol on a day-to-day basis. Type 1 alcoholism develops more slowly and appears later in life. This type of alcoholism, which occurs in both males and females appears to develop as a result of environmental and genetic influences. It was noted that drunk driving is typically the only alcohol-related problem Type 1 alcoholics will encounter with law enforcement officials. Type 1 alcoholics appear to be able to abstain from alcohol consumption on a daily basis, but encounter loss of control over their drinking behavior when they do drink. Thus, it would

appear that research is beginning to discover the types of alcoholics who may be at a greater biological risk in the development of alcoholism.

Beginning in 1970, Goodwin and his colleagues (1979) began a series of adoption studies in Denmark. The study looked at four different groups of subjects, all children of alcoholics. The first groups consisted of sons of alcoholics that had been raised by nonalcoholic foster-parents. The second groups consisted of sons of alcoholics raised by their alcoholic parents. The third group was made up of daughters whose biological parents were alcoholic, but were raised by nonalcoholic foster-parents; and the fourth group contained daughters raised by biological alcoholic parents. Results of this study led Goodwin to conclude that individuals with alcoholic relatives are four times more likely to develop alcoholism than are adults in the general population.

Another methodological approach that is used to tease apart environmental and genetic factors is twin studies. In an effort to seek evidence for genetic predisposition for alcohol-related, organ-specific complications, Hrubec & Omenn (1981) examined male twins pairs in the National Academy of Science-National Research Council Twin Registry. Eleven thousand, eight-hundred and sixty-four monozygotic twins, 15,108 dizygotic twins, and 4,876 twins of unknown zygocity were sampled. While the number of affected individual dizygotic twins (94%) slightly exceed the monozygotic twins (86%), looking at the number of twin pairs both affected by the disease, they found a higher casewise concordance rate among

monozygotic twins than among the dizygotic twins, 23.6% and 11.86% respectively. These results provide evidence in favor of a genetic predisposition for alcoholism and alcohol-related complications.

Results such as these have been an impetus for many researchers to find biologic and genetic markers associated with alcoholism. The National Institute of Alcohol Abuse and Alcoholism has supported ongoing research in the genetic linkages, specifically with respect to the neurophysiological, neuropsychological and cognitive development associated with alcoholism (Vejnoska, 1984).

P300 Evoked Potential

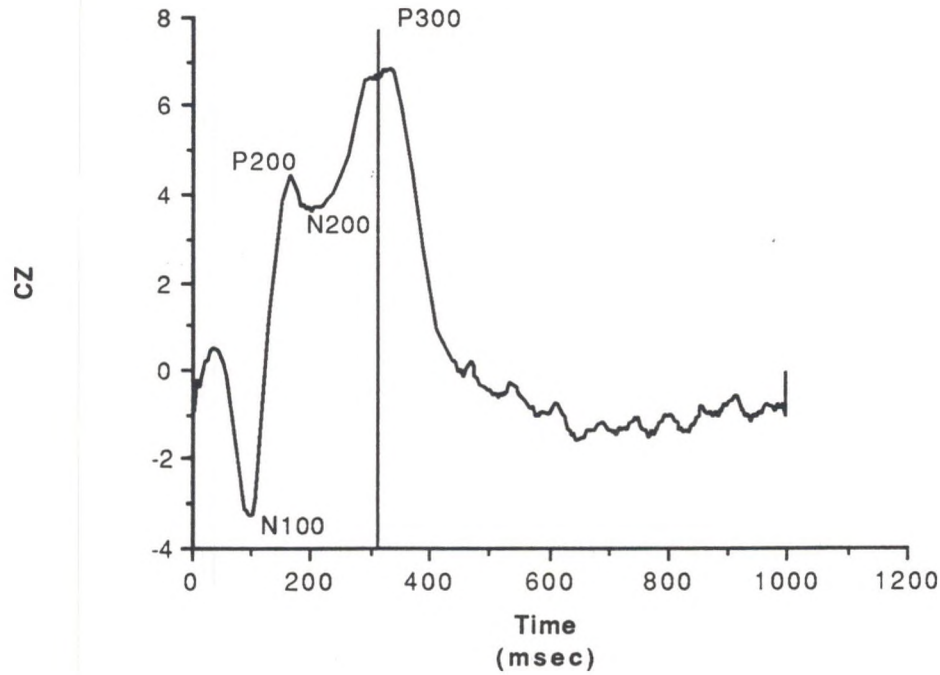
With the advent of modern computer technology, scientists are now able to measure the human brain's reaction to stimuli by looking at the evoked potential (EP). EP methodology appears to provide a non-invasive approach in measuring the brain processes of auditory and visual stimuli. For example if a flashing light is presented, EP recordings can track the signal as it proceeds from the retina, the optic nerve, the brain stem, up to the visual cortex. It is done by placing electrodes on the scalp of the individual and the electrical response is recorded using signal averaging techniques to pull out the time-locked evoked activity.

Figure 1 is a pictorial representation of a typical evoked potential of a normal healthy subject. The N100 component is a large negative deflection that occurs at a latency of about 80-110

msec. following presentation of a stimulus with healthy subjects. It is thought to be sensitive to the selection

FIGURE 1.

Evoke Potential of Healthy Subject



of both relevant and irrelevant stimuli. In a relevant (to be attended) stimulus modality the amplitude of N100 is enhanced and alternately reduced to irrelevant (to be ignored) modalities. (Hillyard, Hink, Schwent, & Price, 1973). Another negative deflection which occurs at a latency of about 200 msec. and also appears to be modality specific is the N200 component of the evoked potential. It is considered to be an early index of stimulus evaluation time; the more difficult the discrimination the longer the N200 latency. (Renault & Lesevre, 1979). Finally, the P300 component is a large positive deflection that occurs approximately

300-500 msec after the stimulus. It has been established that the amplitude of this component indexes the significance of a stimulus and plays a role in memory (Begleiter, Porjesz, Bihari & Kissin, 1984). A significantly reduced P300 amplitude suggests a reduced capacity to assess significance or allot the neural resources needed for encoding the specific event. It is thought that the N100 and P300 components functionally reflect different selective processes. N100 appears to index the preferential admittance of all stimuli having a common simple sensory attribute, i.e., pitch or position in space. An analogy is the ability to listen and attend to one conversation at a noisy cocktail party, suppressing irrelevant stimuli. The P300 component, on the other hand, appears to be reflecting selective processing and analysis of sensory information. This would be analogous to recognizing the specific contents of the cocktail party conversation.

A typical EP paradigm used to elicit P300 is what is referred to as an "oddball" task. For example, using the auditory modality, tone bursts of different pitches or frequencies are presented biaurally. The infrequent tones, designated as the target or oddball stimuli, are generally presented randomly 20% of the time, while the frequent or non-target tone bursts are presented 80% of the time. The subject is asked to count the number of target tones presented or to press a button each time a target tone is heard. The non-target tones have been shown to elicit enhanced amplitudes in the N100 and N200 components of the EP, leaving the P300

component unaffected. However, the presentation of the oddball stimuli will elicit an increased amplitude in the P300 component.

Begleiter and Porjesz (1981, 1984, 1987, 1988) have done extensive research looking at EP's of alcoholics and their offspring. Many of the studies have shown several EP deficits in abstinent alcoholics, especially with the P300 component. These finding led them to investigate the possibility that offspring of alcoholics may also show the same deficits. The results of the studies have shown that young sons of alcoholics who have never ingested alcohol have significantly lower P300 amplitudes as compared to matched groups of control children. The fact that P300 deficits are present in both the abstinent alcoholic patients and offspring of alcoholics suggests that this neurophysiological deviation may be present before the development of alcoholism.

Whipple and Nobel (1987) also investigated the effects of familial alcoholism on the P300 component of the visual ERP and looking at the possibility of transgenerational commonalities existing on the P300 measure. Thirty-nine father-son pairs were divided into three groups. One group consisted of recovering alcoholics with a positive history of alcoholism (RA-FH+), another group consisted of nonalcholics with a positive family history of alcoholism (NA-FH+), and a final group consisting of nonalcoholic subjects with a negative family history of alcoholism (NA-FH-). The sons, aged 8 to 12 years, were categorized the same as their fathers. They did not find significant differences in amplitudes of the P300 between the group fathers, however, they found prolonged

P300 latencies with the RA-FH+ and NA-FH+ fathers compared to NA-FH- . Additionally, the latency for the RA-FH+ sons were also significantly longer than the other two groups. A significant relationship between the father and son P300 latencies was found for the thirty-nine pairs examined $r = .39$ ($p < .02$).

Pfefferbaum, Horvath, Roth Clifford & Kopell (1980) investigated whether acute alcohol intoxication produces observable impairment in EP responses among 18 healthy, male social drinkers ranging in age from 19 to 26 years. It has been shown that acute ingestion of alcohol will reduce EP components within the 30-400 msec range regardless of the stimulus modality. A frequent stimulus produced prominent N120 and P200 components during the baseline condition and a marked reduction in amplitude and an effect on latencies while subjects were under the influence of alcohol. The oddball or target stimuli produced prominent P300 components with longer latencies while subjects were under the influence.

In a second experiment, Pfefferbaum and his colleagues examined EP's among 10 chronic male alcoholics, abstinent from alcohol for at least two weeks, and 10 age matched controls. It was observed that the alcoholics and controls did not differ with respect to their N120 or P200 amplitude or latency in response to the frequent stimuli, but the former group did show markedly prolonged P300 latencies in response to both target and non-target stimuli.

The prolonged P300 latency in response to target stimuli was produced by acute administration of alcohol in the first experiment and also observed in the chronic alcoholics who were not

intoxicated. Pfefferbaum postulated that alcohol may effect earlier sensory sensitive processes (N120 and P200) but not the later P300 component which is more sensitive to cognitive processes. Chronic use appears to leave the earlier component unaffected, but produces a longer latency in the later P300 component. It should be noted that family history for alcoholism for the adult non-alcoholic subjects was not mentioned. These neurophysiological findings are consistent with the research to be reviewed shortly showing impaired cognitive functioning observed in chronic alcoholics.

Elmasian, Neville, Woods, Schuckit and Bloom (1982) examined 15 pairs of male subjects using an evoke related potential (ERP) auditory vigilance task in baseline, peak and placebo alcohol conditions to examine the effects of family drinking history on CSN functioning while under the influence of alcohol. The 15 pairs were divided into three dosage groups: placebo, low dose (0.56g/kg) and high dose (0.94 g/kg). Each pair consisted of one subject with a positive family history for alcoholism (FH+) matched for sex, age and drinking habits with a subject with a negative family history for alcoholism (FH-). Three ERP recording blocks approximately 21 minutes in duration were investigated; the first occurring before ingestion of alcohol or placebo, the second immediately following a half hour drinking period, with the third block one half hour after the second block. Results indicated that the P300 amplitude was markedly suppressed in blocks 2 and 3 for FH+ subjects for both high, low and placebo conditions. The data revealed a significant block X family history interaction for peak altitude and average

latency measures. Significantly delayed P300 latencies for FH+ subjects also was evident from block 1 to block 2 to block 3, with FH+ subjects found to be behaviorally less accurate that FH- subjects in responding to target stimuli. These results have lead Elmasian to believe that family history for alcoholism and P300 have a strong relationship. The Elmasian and Begleiter team results collectively argue strongly that alcohol is not required for P300 differences in brain functioning between FH+ and FH- individuals. Reduction of P300 amplitude and latency among FH+ subjects may suggest lower levels of cognitive stimulus evaluation while under the influence of alcohol. Acute ingestion of alcohol by nonalcoholics appears to produce P300 characteristics that look very similar to those found in abstinent alcoholics. Most striking of all findings were ACA P300 amplitudes and latencies in response to the alcohol placebo that mimicked the brain functioning of nonACA control subjects in response to actual alcohol doses. ACA males appear to show strong idiosyncratic neurophysiological responses to placebo doses.

Neuropsychological and Cognitive Findings

With the exception of the Wernicke-Korsakoff syndrome, it has been observed that alcoholics do not show across-the-board cognitive deficiencies. Consequently, identifying neuropsychological impairment in alcoholics who do not show behavioral evidence of cognitive impairment has been more problematic. Because of the myriad demographic factors such as educational and occupational background, age, gender, duration and pattern of alcohol abuse, and

alcohol-related factors,for instance, nutritional deficiencies and liver dysfunction, it has been important to look for sensitive measures that will assess the subtle changes in information-processing abilities due to chronic alcohol abuse.

Detoxified, neurologically intact alcoholics generally earn IQ scores in the average to bright average range when intellectual functioning is assessed using the Wechsler-Bellvue or the Wechsler Adult Intelligence Scale. But further analysis indicates that they perform more poorly than nonalcoholics on one or more subtests, usually Block Design, Object Assembly, Digit Symbol and Digit Span (Kleinknecht & Goldstein, 1972). These consistently replicated results appear to reflect impairment related to visual-spatial functions, problem-solving ability and memory. The potential utility of the P300 component as a genetic marker has led researchers to investigate whether some of the cognitive impairments seen in alcoholics can be explained on the basis of family history and if this premorbid neuropsychological influence has any real life significance.

To assess the possibility of premorbid neurological deficits in alcoholics, Schaeffer, Parsons, & Yohman (1984) compared FH+ and FH- individuals on a battery of neuropsychological tests assessing abstraction/problem-solving, verbal, learning/memory and perceptual-motor ability. They looked at four groups: FH+ alcoholic males, FH- alcoholic males, FH+ nonalcoholic males and FH- nonalcoholic males. It was observed that FH+ alcoholics performed significantly poorer than FH- nonalcoholic controls on the

Learning/Memory, Abstract/Problem Solving and the Perceptual-Motor clusters on several neuropsychological tests. While the differences between the two alcoholic groups did not reach statistical significance, there was an observed trend for FH+ alcoholics to perform more poorly than FH- alcoholics. This may suggest that there is a subset of alcoholics, specifically those with a positive family history for alcoholism which may predispose those individuals who begin drinking to a neuropsychological disadvantage or vulnerability.

Reporting unpublished data from the familial alcoholism high risk studies in Denmark, Goodwin (1983) found that nonalcoholic sons of alcoholic fathers had poorer performances on the Halstead Category Test than nonalcoholic sons of nonalcoholic fathers after ingesting alcohol. It was suggested that low scores on the categories test found in previous studies with alcoholics, which were attributed to the deleterious effects of alcohol, may necessitate revised interpretation in light of this finding.

Part of the the first phase of the Danish longitudinal study on alcoholism, Drejer, Theilgaard, Teasdale, Schulsinger & Goodwin (1985) looked at young males at high risk for alcoholism using a battery of neuropsychological measures. The high risk males (N=134) were sons of alcoholics found through a national demographic register which listed all admission and discharge dates of psychiatric departments as far back as 1918. The alcoholic fathers had at least one main diagnosis of alcoholism or a secondary discharge diagnosis of alcoholism with the main diagnosis as

alcohol-related, eg, psychopathy. Control subjects (N=70) were carefully matched according to age and birth order, mother's age and marital status at the time of the subject's birth and parental social class.

The neuropsychological battery consisted of twelve tests examining handedness, general intelligence, memory, attention, field dependence, categorizing, organizing and planning ability. Results of this study revealed the high risk group to perform significantly poorer on the WAIS vocabulary subtest, Halstead Category Test and the Porteus Maze Test. These findings reveal that FH+ males are significantly different than FH- males on general intelligence and have poorer categorizing and planning ability.

Tarter, Hegedus, Goldstein, Shelly and Alterman (1984) have found that FH+ male delinquents compared to FH- delinquent males performed more poorly on Part B of the Trail Making Test as well as on the Semantic Memory and Figural Memory subtests of the Wechsler Memory Scale and the Peabody Picture Vocabulary Test.

Reed, Grant & Adams (1987) sought to examine the relationship of family history of alcoholism in first degree relatives to neuropsychological performances in abstinent alcoholics looking at the strength of the family history or the genetic loading. One group consisted of individuals with a strong family history, a parent plus one other first-degree relative. The second group consisted of individuals having only one alcoholic parent. A weak family history consisted of individuals having a non-parent first-degree relative positive and the fourth group contained males with no first-degree

relative positive. Administering the extended Halstead-Reiten Battery, they found no significant difference in neuropsychological functioning related to family history for alcoholism which led them to conclude that the presence of first-degree alcoholic relatives does not predict later neurological status in adult males.

Alterman, Gerstley, Goldstein and Tarter (1987) also examined the strength of familial alcoholism on cognitive performance. Eighty-one alcoholic men participating in a Veterans Administration inpatient program were divided into three groups: the first group had no history for alcoholism, the second group had individuals with at least one alcoholic parent and the third group consisted of individuals with an alcoholic sibling, grandparent or uncle. Ten neuropsychological tests were used that had been shown to discriminate between alcoholics and nonalcoholics. The results of this study did not confirm the hypothesis that FH+ subjects would perform worse than FH- subjects.

The presence of hyperkinesis and minimal brain dysfunction (Hk-MBD) observed in young males has been implicated as a possible etiological factor in the development of alcoholism (Tarter, McBride, Buopane & Schneider, 1977). To examine whether the cognitive deficits found in alcoholics are a result of alcohol abuse or a premorbid vulnerability marked by Hk-MBD, Workman-Daniels & Hesselbrock (1987) examined three groups of subjects. One sample consisted of subjects with a positive family history (FH+) for alcoholism, one group consisted of offspring of nonalcoholic

parents. The third group was a comparison sample of young detoxified alcoholics.

Each subject was administered Trail Making A & B, the Category Test, the Rhythm and Tactual Performance Test, and the Wechsler Intelligence Scale from the Halstead-Reitan battery to assess attention, memory and concentration. These neuropsychological measures have been related to childhood Hk-MBD; therefore, they hypothesized that FH+ subjects who reported a higher number of Hk-MBD behavior in childhood would show poorer neuropsychological performance than FH- subjects. The results of this study did not support this hypothesis nor the idea that Hk-MBD is a premorbid factor responsible for cognitive deficits found in alcoholics.

The findings of Reed et al. (1987), Workman-Daniels & Hesselbrock (1987) and Alterman et al. (1987), contradict those of Goodwin & Hill (1975); Tarter & Ryan (1983); Tarter et al. (1977) and Drejer et al. (1985). It was suggested that the studies that found cognitive differences based upon family history for alcoholism examined subjects who were atypical for high risk research. Tarter looked at delinquents, Schaeffer studied middle-aged subjects and Drejer's findings may have been confounded by a higher incidence of antisocial alcoholism in their families.

These studies have assessed the possibility of neuropsychological deficits in alcoholics and individuals with a positive history for alcoholism. In reviewing the literature, there is divided evidence for a genetically transmitted predisposition. None

of the studies, however, looked at cognitive functioning of FH+ subjects and FH- subjects while under the influence of alcohol. This study was interested in examining individuals with a positive family history for alcoholism using an alcohol paradigm and neuropsychological measures that have been shown to be sensitive to brain dysfunctions and the P300 component of the evoked potential.

Cognitive- EP Correlates

Individuals with severe forms of cognitive impairment produced by congenital problems or brain injury typically show substantially longer P300 latencies in simple auditory and visual paradigms (Brown, Marsh & LaRue, 1982). Polich, Howard & Starr (1983) speculated that the broad cognitive impairment observed among these individuals may be related to more fundamental memory deficits that could also be reflected in the longer P300 latency. They investigated relationships between P300 latency and memory capability within a group of 96 neurologically normal subjects ranging in age from 5 to 87 years. The evoked potentials recording were obtained using a standard auditory P300 paradigm. The Digit Span subtest of the Wechsler Adult Intelligence Scale (WAIS) was selected as an important index of attention and immediate memory recall.

Insignificant relationships were found between Digit Span performance and the latency of any auditory evoked potential components prior to the P300. The P300 component obtained to the rare tones were observed to consist of two distinct subcomponents

which they labelled P3a (range: 220-320 msec) and P3b (range: 300-450 msec). Significant negative correlations were observed between mean P3a and P3b range latencies and memory scores (r= -.47, t(83)=4.79, p<001, and r= -.36, t(94)=3.69 p<.001, respectively). Shorter P300 latencies were associated with better memory scores. Removing the variability of the P300 latency due to age still showed the correlation existed irrespective of age (P3a r=-.52, P3b r= -.40).

Polich et al. (1983) speculated that these results reflect the importance of "context" updating of the stimulus environment. They suggest that the P300 latency reflects brain functions which may mediate retention of recently encoded material for comparison with new incoming information. An individual's capacity to maintain a mental representation may rely heavily on brain functions reflected in the P300 component. Certain forms of neurological impairment or chemically induced altered brain states may impair P300 functions that result in slower internal context processing.

Howard and Polich (1985) generated similar findings in their examination of Digit Span and auditory evoked potentials among 24 children (ages 5 to 14 years) and 24 adults (ages 20 to 40 years). They found a negative relationship between P300 and Digit Span scores which was most apparent for the younger subjects.

The Present Study

The present study sought to investigate differences in cognitive functioning between male high risk FH+ and FH- subjects while under the influence of alcohol with a placebo condition. It appears that the P300 latency reflects the cognitive processes of

attention, discrimination of significant stimuli and context updating. Digit Span performance appears to be a sensitive behavioral manifestation of P300 latency. Digit Symbol and the Trailmaking tests appear unusually sensitive to alcohol-related brain impairment.

CHAPTER II
METHODOLOGY

Subjects

A total of forty male subjects enrolled in undergraduate psychology courses participated as subjects in the present study earning extra credit points for their respective classes. Twenty subjects were Adult Children of Alcoholics (ACAs) who were identified as such by using the criteria set forth by the Children of Alcoholics Screening Test (CAST) (Pilat & Jones, 1985). Additionally, the ACA's were biological offspring of an alcoholic father whose mother was not identified as alcoholic. Twenty subjects were nonACA's, who were identified as such by scoring a zero on the CAST.

All subjects were between the ages of 21 and 38 years and were white Americans since it had been suggested that racial differences may occur in alcohol metabolism (Reed, Kalant, Gibbins, Kapur & Rankin, 1976).

All subjects denied using prescription or nonprescriptive drugs which may influence alcohol metabolism . All subjects were screened for drinking problems or alcoholism using the Michigan Alcoholism Screening Test (MAST) (Selzer, 1971). Additionally, all subjects indicated a tolerance for moderate amounts of alcohol

which was determined by their responses to the Khavari Alcohol Test (KAT) (Khavari, 1978).

Independent Measures

The independent measures used were family history for alcoholism status, alcohol dosage, and phase of intoxication. The design consisted of two between factors: the subject factor being ACA and nonACA status and the treatment factor consisted of Alcohol and Placebo dosage. Baseline, peak and descending level of blood alcohol concentration (Block 1, Block 2, Block 3) was the within subjects factor.

Dependent Measures

The Digit Span subtest is used in the Wechsler Adult Intelligence Scale (WAIS) and the Wechsler Memory Scale. It is comprised of two different tests, Digit Forward and Digits Backwards. It is assumed that these two tests measure highly correlated behavior in normal subjects up through middle age (Lezak, 1983). Differences between the two tests have been shown to appear with age and in some populations with brain impairment. Digits forward is considered to reflect efficiency in attention with Digits Backwards requiring a more effortful activity of holding pieces of information in short term memory while mentally juggling them around. A Digits Forward score of 6 falls within the normal range and raw scores of 4 or 5 for Digits Backwards is considered within the normal limits (Spitz, 1972) . The raws scores for each test were considered separately therefore, the Wechsler scoring system was not relevant to the present study.

Both tests required auditory attention during which an examiner read aloud seven pairs of random number sequences at a rate of one digit per second. Digits Forward was presented first. The subject was asked to repeat a sequence of digits in the same order in which they were presented. There were seven levels of sequences containing two trials at each level. The levels increased in number from three to nine digits. The subject continued until failure of two trials at the same level or all nine digits were successfully repeated. Digits Backwards contained number sequences two to eight digits long. After hearing a number sequence the subject's task was to repeat the digits back in reverse order. Testing continued following the same guidelines as Digits Forward.

Digit Symbol is thought to measure visual-motor dexterity, attentiveness, persistence and quickness. This is the only subtest of the WAIS that requires on-the-spot learning. This test has been shown to be consistently sensitive to cognitive deficits in chronic alcoholics (Goldman, Klisz & Williams, 1985). Digit Symbol is a symbol substitution task which consists of four rows of 25 blank squares with numbers above each square. Above the rows is a printed key that pairs each number with a nonsense symbol. The subject's task was to fill in the blank square as quickly as possible with the symbol that corresponds to the number. The subject was given 90 seconds and the score reflected the number of correct and completed squares. Scores from 52 to 57 are considered within the normal range for subjects between the ages of 16 and 34.

The Trail Making Test is another test among those that have been found to be sensitive in detecting cognitive deficits as a result of chronic alcohol abuse (Leckliter & Matarazzo, 1989). Trail Making is a timed test of visual information-processing efficiency which requires attention and speed. The test was given in two parts: Trails A and Trails B. In Trails A the subject was asked to draw lines to connect consecutively numbered circles that appeared on a worksheet. The subject was told to work quickly without lifting the pencil from the paper. Trails B contained circles with numbers and letters and the subject was asked to draw a line to connect the circles alternating between the number and letter sequence, i.e., 1 A 2 B 3 C 4 D and so on. Scores were considered according to time for completion and number of errors.

Each of the three tests had three alternate forms and presentation was counterbalanced across all subjects to avoid practice effects with the repeated measures.

The WAIS-R vocabulary subtest, known to be a valid measure of general intellectual functioning, was also administered.

Screening Measures

The Michigan Alcoholism Screening Test (MAST) (Selzer, 1971) is a 25-item instrument that was devised as an attempt to detect early drinking problems and alcoholism (see Appendix A). It was developed with the understanding that individuals with a drinking problem may have a tendency to be defensive and not answer with complete honesty. It was validated in a way that attempted to reduce the likelihood of false negatives. Originally, the MAST was

designed to be administered orally, however, it may be self-administered as well. Because of the potential problem concerning the lack of candor on the part of the alcoholic respondent the validity of this screening method has been questioned. However, in an experiment Selzer (1967) carefully instructed 99 hospitalized alcoholics to lie about their drinking problems using the MAST. Despite these instructions more than 92 percent disclosed enough information to be classified as alcoholics. This lead Selzer to believe that alcoholics have a problem with lying about their problem in a consistent way, therefore the MAST was able to detect problem drinking and alcoholism despite the false negatives. Moreover, the self-administered MAST was studied by Selzer, Vinokur and Van Rooijen (1975) who gave it to four different groups and it was concluded that "a self-administered MAST questionnaire has substantial reliability and validity with the scores relatively unaffected by age and the denial of socially undesirable characteristics." Silber, Capon and Kuperschmit (1985) evaluated the contribution of the MAST with respect to the detection of alcoholism among college and/or university students. They found that the MAST is an appropriate and reliable assessment device in detecting alcoholism and alcohol related problems among the college population. It was determined that a score of 10 or more is considered diagnostic of alcoholism, therefore the current study excluded those individuals scoring 10 or above on the MAST.

The Children of Alcoholics Screening Test (CAST) (Pilat & Jones, 1985) is a 30-item screening instrument developed to

identify children five years old through adult who are currently living with or have lived with an alcoholic parent or parents (see Appendix B). This screening test measures the child of an alcoholic's emotions, attitudes, perceptions and experiences related to their parents' drinking behavior. Normative data were derived from clinically diagnosed children of alcoholics (ACAs), self-identified ACAs and a control group. The CAST has a validity coefficient of .78. All thirty items significantly discriminated ACAs from control subjects. A cut-off score of six or more reliably identified 100 percent of the ACA group. A reliability coefficient of .98 was reported. The present study utilized the same exclusion criteria. Six additional questions were added to determine biological status of parent but were not used in determining ACA status.

The Khavari Alcohol Test (KAT) consists of four questions relating to three of the types of alcoholic products: beer, wine and liquor. Respondents are asked to indicate how much and how often they usually drink each of the three products, in addition to how much they have drank the maximum amount (See Appendix C). An index of each beverage along with an index of annual absolute alcohol intake consumption can be computed from the responses to the items. These indices reflect an annual quantity of alcohol consumption ranging from total abstinence to extreme daily consumption. In order to determine the validity of the KAT, data were collected from two samples of diagnosed alcoholics from a metropolitan area and from a university-based psychiatric hospital,

and three samples of nonalcoholic men and women union workers, army reservists, and male and female university students. Results showed that many of the KAT scales were able to reliably discriminate between the two alcoholic and three nonalcoholic groups. Using test-retest reliability, the reliability coefficients ranged from r=.78 to r=.98 for the 12 separate correlation coefficients, with a mean correlation of r=.92. The purpose for this screening device was to determine those subjects that were able to tolerate moderate amounts of alcohol. Subjects scoring .25 to 2.0, which reflects an an average daily alcohol consumption to be one to four drinks, were considered eligible to participate in the current study.

Procedure

All subjects were initially contacted by phone. Upon agreement to participate, a letter was sent stating the date, time and place of the experiment that they had been invited to attend. The letter contained instructions not to consume anything (with the exception of water) after 2 p.m. on the day of their scheduled session. Additionally, they were requested to abstain from tobacco and alcohol use for 24 hours prior to the session. All subjects were tested between 3:30 p.m. and 10 p.m. Upon arrival for the experimental session, photo identification was checked to ascertain that the subject was 21 years or older. All subjects read and signed a consent form (See Appendix D) and were asked about their compliance with the pre-experimental instructions they received in the letter. Subjects were weighed using a standard weight scale to

determine how much alcohol may be administered to the subjects in the alcohol treatment condition before testing began. Subjects were tested by an examiner blind to the dosage and family history treatment condition. Subjects were first given the WAIS-R vocabulary subtest followed by the administration of the three dependent measures. Upon completion they began ingestion of two equal size drinks. The alcohol was 80 proof Phillip's vodka. The alcohol was in a solution consisting of 1 part vodka and 2 parts masking solution consisting of a double concentration of lemonade flavored with peppermint extract. Subjects in the alcohol condition received 1.0 mL of absolute alcohol per kilogram of body weight. Subjects in the non-alcohol treatment group received water in place of the vodka with the rim of the glass swabbed with one mL of vodka. They were instructed to drink slowly and evenly, making each drink last 20 minutes. The forty minute period was followed by a 15 minute absorption period, allowing the blood alcohol to reach its peak. At this point the subjects were asked to rinse their mouths for five minutes and blood alcohol estimates were taken. A breathalyzer was used to estimate blood levels of alcohol from breath samples. Subjects were again tested with alternate forms of the three tests. Following a 30 minute period, blood alcohol readings were taken again, after which, the experimenter administered the final block of testing using a third alternate form of the dependent measures. Subjects in the alcohol condition were required to remain in the laboratory until it was determined that

sobriety had been achieved, (a breathalyzer reading of .02 or less) at which time the subject was allowed to leave.

Hypotheses

The present literature review strongly suggests that neurophysiological differences exist between male ACA's and nonACA's, but equivocal evidence has been advanced for differences in cognitive functioning between these two groups. None of the studies reviewed investigated the effects of acute alcohol intoxication on cognitive functioning in these high risk males.

The present study attempted to examine the effects of acute alcohol intoxication in ACA's using two neuropsychological measures that have been shown to reveal cognitive impairment in chronic alcoholics eg., Digit Symbol and Trailmaking B. Digit Span, a measure that has been correlated with the cognitive processes associated with the P300 component of the evoked potential was also investigated.

It was hypothesized that ACA subject performance will be negatively influenced compared to the nonACA controls on all three dependent measures at baseline, peak blood alcohol levels (BAL) and at descent. Moreover, it was expected that ACA's may also perform more poorly in the placebo condition at peak BAL and descent which may reflect behavioral evidence for the Elmasian et al study which revealed a placebo effect in ACA P300 latencies.

Research Design

A 2 x 2 x 3 completely randomized design was used. The two between factors which served as the major independent variables

were ACA subject status and alcohol treatment condition. The one within subjects factors were baseline, peak and descending level of blood alcohol concentration (Block 1, Block 2 and Block 3 respectively).

Statistical Analyses

Analysis of variance (ANOVA) was conducted on all three measures to determine whether there were statistically significant main effects or interactions for the three independent variables.

Analysis of covariance was performed on all three measures for age, blood alcohol levels at peak and descent phases of session, the KAT, MAST and WAIS-R vocabulary scores.

CHAPTER 3

RESULTS

Descriptive Statistics

Twenty ACA subjects and 20 nonACA subjects participated in the present study. A 2 (ACA status) x 2 (alcohol condition) analysis of variance for age, KAT, MAST, CAST and vocabulary test was conducted. No significant differences between groups were found for age, the KAT screening measure and the vocabulary test. A significant main effect for ACA status was found for both the MAST F (1,36) = 5.597 p= .024 and the CAST F (1,36) = 104.636 p< .001, with ACA subject mean scores on these two screening measures significantly higher than nonACA subject mean scores.

A 2 (ACA status) x 2 (alcohol condition) x 2 (phase of session at peak and descent) analysis of variance was conducted on blood alcohol level estimates. A significant main effect for phase of session was found F (1,36) =4.916 p= .034 indicating the mean BAL estimates to be significantly lower at block 3 (M= .050) than at block 2 (M= 0.057). No significant main effects were found for ACA status F (1,36)= 1.487 p=.231. No significant interaction main effects were found for ACA x alcohol condition F (1,36)=1.487 p= .231, ACA status x phase of session F (1,36)= 2.306 p= .138, or ACA status x alcohol condition x phase of session F (1.36) = 2.306 p= .138. These results indicate that no significant differences in blood

alcohol level estimates were found between ACA and nonACA subjects at peak and descent phases of session. Table 1 presents the means and standard deviations for age, screening measure and WAIS-R vocabulary scores, and blood alcohol levels at peak (block 2) and descent (block 3) for the ACA and control subjects in the alcohol and placebo treatment conditions.

TABLE 1

MEANS AND STANDARD DEVIATIONS FOR ADULT CHILDREN OF ALCOHOLICS AND CONTROL SUBJECTS FOR AGE, KHAVARI ALCOHOL TEST(KAT), MICHIGAN ALCOHOL SCREENING TEST (MAST), CHILDREN OF ALCOHOLICS SCREENING TEST (CAST), WAIS-R VOCABULARY SUBTEST(VOCAB) RAW SCORES AND BLOOD ALCOHOL LEVEL ESTIMATES (BAL)

	ACA				CONTROL			
	Alcohol		Placebo		Alcohol		Placebo	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Age	23.00	2.05	25.60	4.80	3.30	2.90	22.70	3.10
KAT	.93	.49	.97	.70	.93 .	60	1.11	1.17
MAST	4.00	2.79	5.30	2.68	2.20	2.82	2.80	2.60
CAST	13.70	6.23	12.30	3.98	0	0	0	0
Vocab	43.30	10.76	44.70	6.29	45.00	10.73	42.30	9.48
BAL								
Block 2	0.061	0.012	0	0	0.059	0.008	0	0
Block 3	0.055	0.013	0	0	0.045	0.009	0	0

Dependent Measures

A simple 2 (ACA status) x 2 (alcohol condition) x 3 (phase of session) analysis of variance was conducted for each of the Digit Span, Trail Making B, and Digit Symbol dependent measures. In these

alcohol level estimates were found between ACA and nonACA subjects at peak and descent phases of session. Table 1 presents the means and standard deviations for age, screening measure and WAIS-R vocabulary scores, and blood alcohol levels at peak (block 2) and descent (block 3) for the ACA and control subjects in the alcohol and placebo treatment conditions.

TABLE 1

MEANS AND STANDARD DEVIATIONS FOR ADULT CHILDREN OF ALCOHOLICS AND CONTROL SUBJECTS FOR AGE, KHAVARI ALCOHOL TEST(KAT), MICHIGAN ALCOHOL SCREENING TEST (MAST), CHILDREN OF ALCOHOLICS SCREENING TEST (CAST), WAIS-R VOCABULARY SUBTEST(VOCAB) RAW SCORES AND BLOOD ALCOHOL LEVEL ESTIMATES (BAL)

	ACA				CONTROL			
	Alcohol		Placebo		Alcohol		Placebo	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Age	23.00	2.05	25.60	4.80	3.30	2.90	22.70	3.10
KAT	.93	.49	.97	.70	.93 .	60	1.11	1.17
MAST	4.00	2.79	5.30	2.68	2.20	2.82	2.80	2.60
CAST	13.70	6.23	12.30	3.98	0	0	0	0
Vocab	43.30	10.76	44.70	6.29	45.00	10.73	42.30	9.48
BAL								
Block 2	0.061	0.012	0	0	0.059	0.008	0	0
Block 3	0.055	0.013	0	0	0.045	0.009	0	0

Dependent Measures

A simple 2 (ACA status) x 2 (alcohol condition) x 3 (phase of session) analysis of variance was conducted for each of the Digit Span, Trail Making B, and Digit Symbol dependent measures. In these

analyses all significant effects were defined by a $p < .05$. Newman-Keuls (Myers, 1979) post hoc procedures were utilized when required with alpha set to .05.

Digit Span Results

No significant main effect for ACA status or phase of session was found in the analyses for Digit Span Forward. A significant alcohol main effect was found $F(1,36) = 8.75$ $p < .006$. Analysis of this effect revealed mean Digit Span Forward scores for subjects in the alcohol condition (M= 8.07) were significantly lower than the mean of subjects in the placebo condition (M= 9.57).

The analysis of Digit Span backward scores revealed significant main effects for the experimental subjects $F(21,36)= 7.22$ $p<.01$, with subjects in the alcohol condition scoring lower (M=6.22) than placebo subjects (M=7.53). A significant main effect for phase of session was also found $F(2,72)=4.584$ $p<.01$. Subsequent Newman-Keuls analysis revealed that performance from block 1 (M= 7.2) to block 2 (M= 6.4) significantly decreased, followed by a significant improvement at block 3 (M=7.02). Figure 2 illustrates this main effect for phase of session. No significant main effect was found for ACA status $F(1,36)=1.26$ $p=.27$.

Analyses also generated a significant ACA x alcohol X block interaction effect $F(2,72)=4.97$ $p<.01$, which revealed that ACA subjects under the influence of alcohol significantly decreased their Digit Span backward

performance from block 1 (M=7.4) to block 2 (M=5.1) with improved performance at the descent point of phase of session (M=7.2). No significant differences were found between block 1 and block 3, and Newman-Keuls analyses revealed an absence of significant differences in the four ACA and nonACA , alcohol and placebo cells at baseline. Significant differences between ACA and nonACA subjects in the alcohol and placebo condition were not found at block 2, however, significant differences in Digit Span backward scores for ACA and nonACA subjects in the alcohol condition were revealed at block 3. It should be noted that ACA subjects in the alcohol condition revealed the only significant phase of session differences. This effect is entirely due to the improvement in performance of the ACA subjects from block 2 to block 3. This three-way interaction is illustrated in Figure 3.

Trail Making B Results

No significant main effects for ACA status or alcohol condition were found, however a main effect for phase of session was observed F (2,72) = 3.51 $p < .036$. Subsequent analysis indicated an increase in performance on the Trail Making B task for subjects in both treatment groups from block 1 (M= 52.37 sec.) to block 2 (M=49.02) to block 3 (M= 44.75 sec.). An illustration of this phase of session main effect is found in Figure 4.

A significant two-way interaction effect was found for alcohol condition and phase of session F (2,72) = 3.35 $p < .041$. Newman-Keuls analysis showed increased performance from block 1 (M= 52.6 sec.) to block 2 (M= 42.6 sec.) for subjects in the placebo condition, while subjects in the alcohol treatment condition

revealed a significant increase in performance from block 2 (M= 55.4 sec.) to block 3 (M=44.8 sec.) $p<.05$. Further analyses revealed a significant difference in performance between the alcohol and placebo conditions at block 2 with mean scores of 55.4 sec. and 42.6 sec. respectively. This two-way interaction effect is illustrated in Figure 5.

Digit Symbol Results

No significant main effect for ACA status was found F (1,36) = 1.27 p= .268, for the Digit Symbol task. Mean Digit Symbol scores produced a significant main effect for alcohol F (1,36) = 4.225 $p <$ .048, in that subjects in the alcohol treatment condition performed significantly poorer (M= 64.98) than subjects in the placebo treatment conditions (M= 70.51).

A significant main effect also was found for phase of session with the Digit Symbol task F (2,72) = 9.067 $p < .001$. Analysis indicated that performance decreased from block 1 (M= 68.27) to block 2 (M= 65.82) and then showed improved performance from block 2 (M=65.82) to block 3 (M= 69.15) for all subjects in both treatment conditions. Figure 6 illustrates this phase of session

main effect. Further analyses revealed a significant treatment x phase of session effect F (2,72) = 9.139 $p < .001$. Newman-Keuls analysis revealed that subjects in the placebo treatment condition performed significantly better from block 1 (M=69.05) to block 3 (M=73.05) and from block 2 (M=69.45) to block 3(M=73.05) with no improvement from block 1 to block 2. Subject performance in the alcohol treatment condition declined from block 1 (M= 67.5) to block 2 (M= 62.2), but showed significant improvement from block 2 to block 3 (M= 65.25), and no significant improvement from block 1 to block 3. This interaction effect is illustrated in Figure 7.

Table 2 presents the means and standard deviations for the three dependent measures examined in this study at baseline (block 1), peak (block 2) and descent (block 3). The statistical significance and directionality of the results for the three dependent measures examined in this study were not altered by the extraction of variance attributable to a range of covariates including age, peak and descent blood alcohol levels, KAT and MAST scores.

Figure 2.

Mean Digit Span Backward Scores at Phase of Session

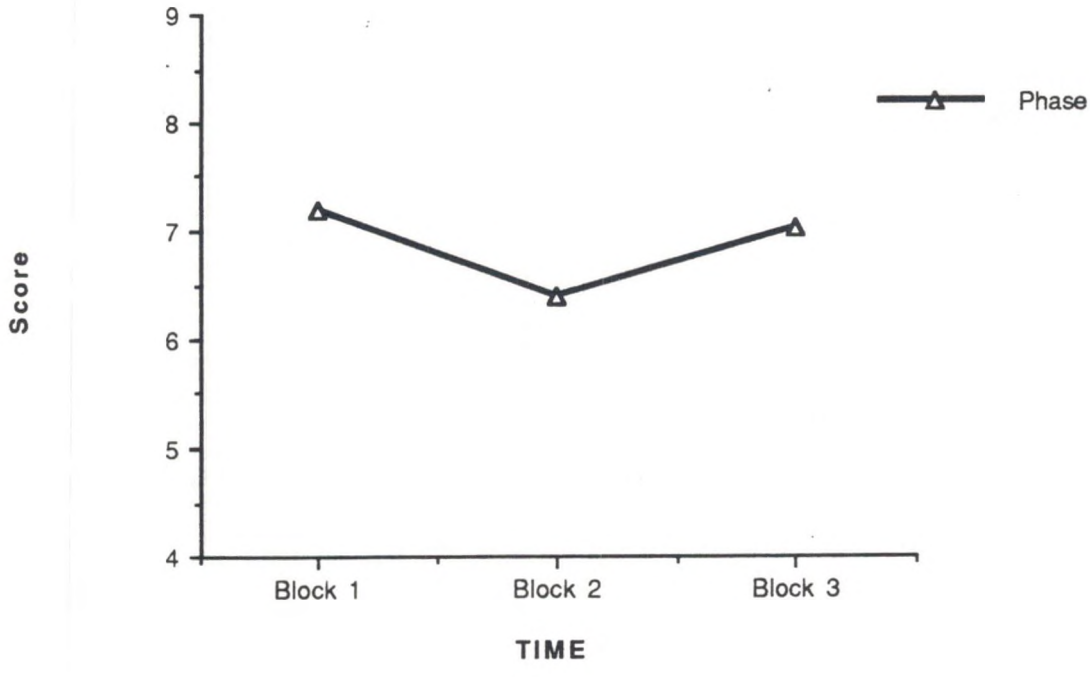


Figure 3.

Mean Digit Span Backward Scores for ACA and Non-ACA Subjects at Phase of Session for Alcohol and Placebo Conditions

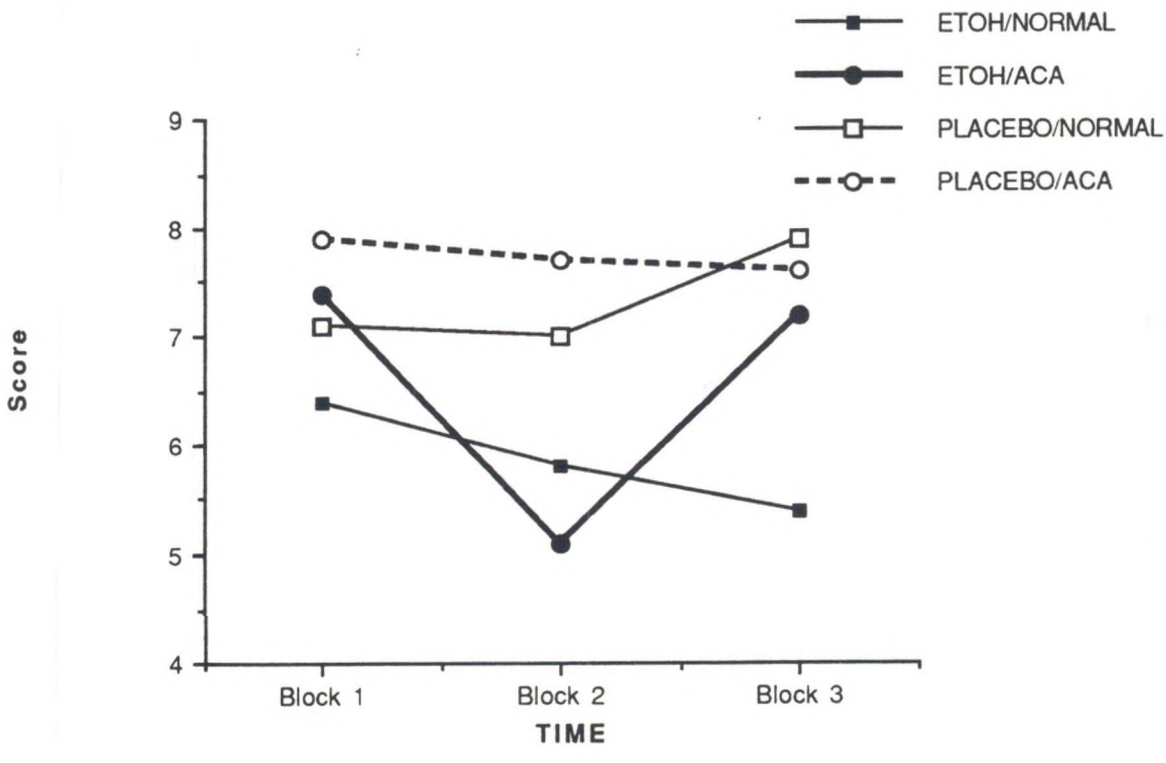


Figure 4.

Mean Trail Making B Scores at Phase of Session

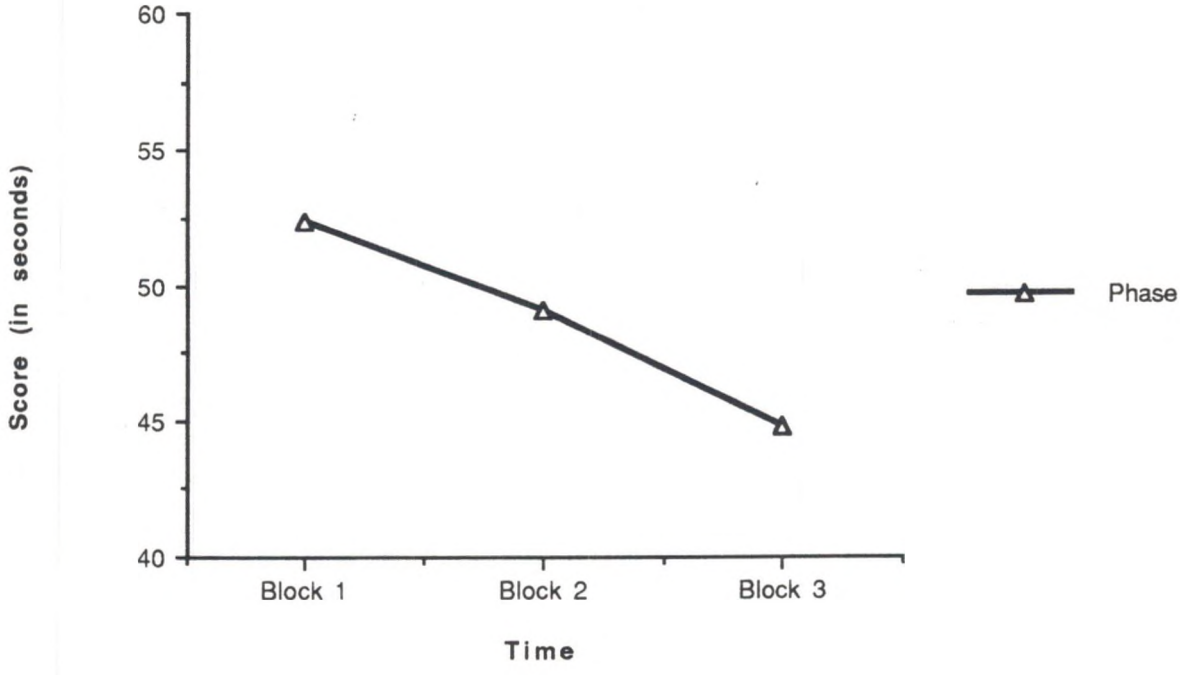


Figure 5.

Mean Trail Making B Scores at Phase of Session for Alcohol and Placebo Conditions

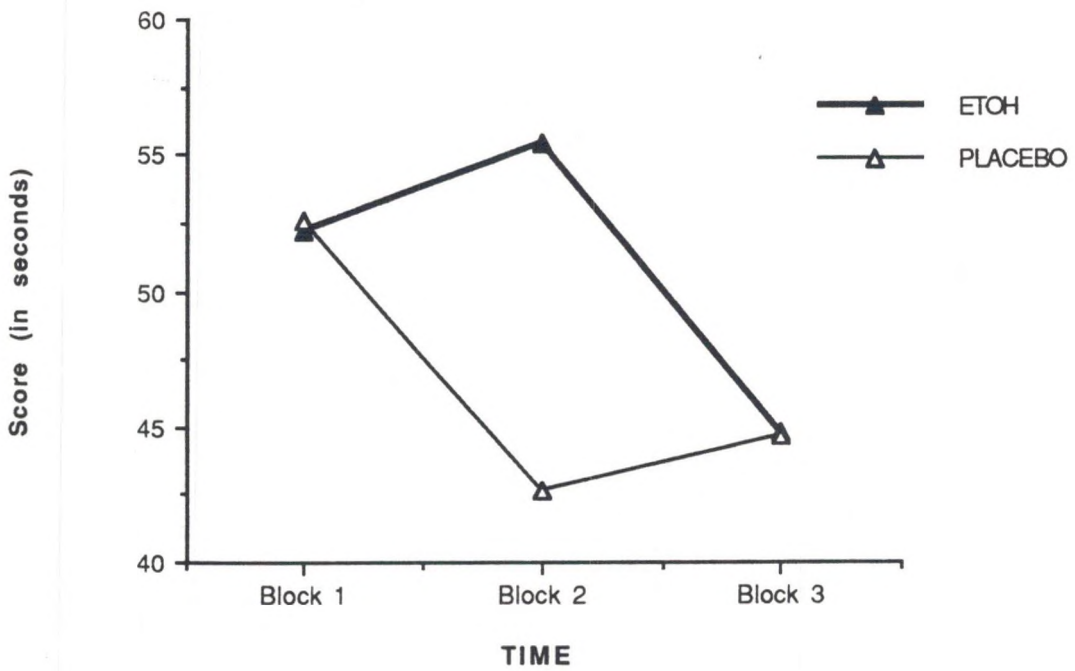


Figure 6.

Mean Digit Symbol Scores at Phase of Session

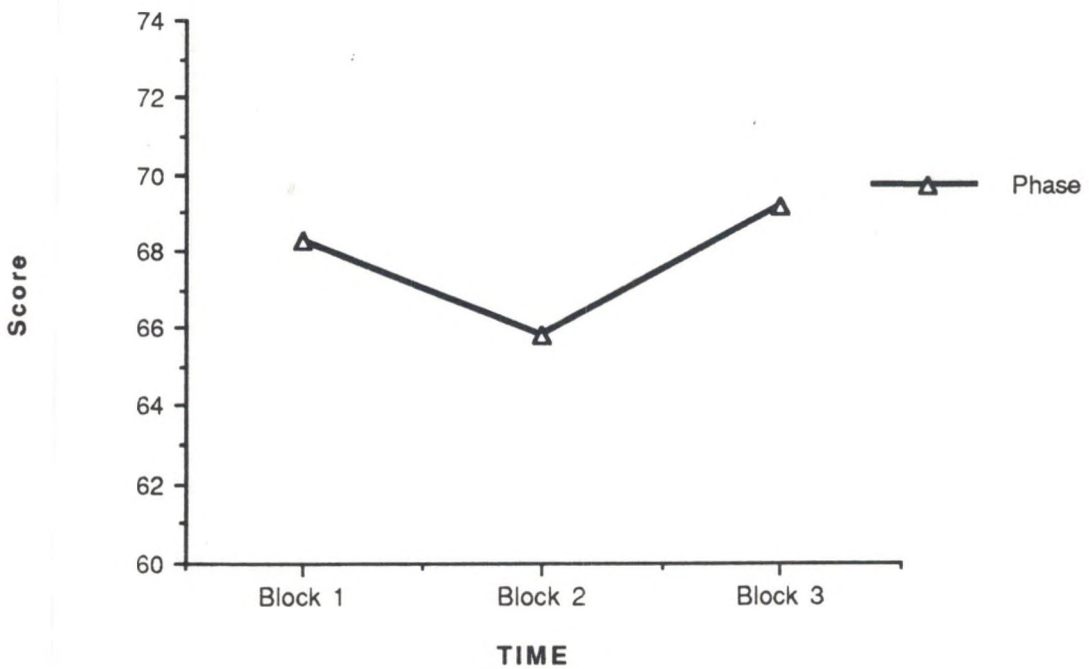


Figure 7.

Mean Digit Symbol Scores at Phase of Session for Alcohol and Placebo Conditions

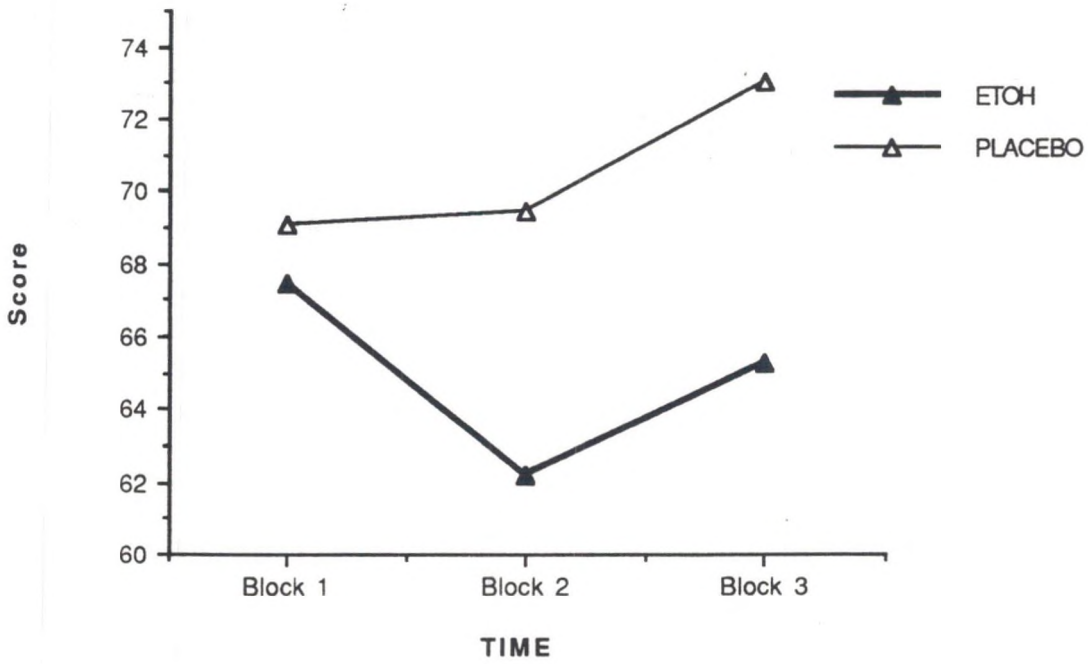


TABLE 2

MEANS AND STANDARD DEVIATIONS FOR ADULT CHILDREN OF ALCOHOLICS AND CONTROL SUBJECTS FOR DIGIT SPAN FORWARD, DIGIT SPAN BACKWARD, TRAIL MAKING B, AND DIGIT SYMBOL SCORES AT BLOCK 1, BLOCK 2 AND BLOCK 3

	ACA				CONTROL			
	Alcohol		Placebo		Alcohol		Placebo	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Digit Span Forward								
Block 1	8.80	1.99	9.60	1.20	7.40	0.80	9.50	1.36
Block 2	8.30	2.45	9.70	2.00	7.10	1.04	9.40	2.15
Block 3	8.50	2.50	9.60	2.10	8.30	1.61	9.60	1.62
Digit Span Backward								
Block 1	7.40	1.28	7.90	2.54	6.40	1.35	7.10	1.97
Block 2	5.10	1.44	7.70	1.18	5.80	1.60	7.00	1.41
Block 3	7.20	1.60	7.60	2.72	5.40	1.28	7.90	1.86
Trail Making B								
Block 1	58.70	12.14	52.90	39.75	45.69	9.78	52.30	9.12
Block 2	60.20	23.65	41.70	14.20	50.60	6.77	43.60	11.53
Block 3	2.70	8.49	42.50	11.46	46.90	11.97	46.90	8.99
Digit Symbol								
Block 1	68.30	10.32	71.30	8.69	66.70	4.75	66.80	7.74
Block 2	64.10	8.59	71.50	9.28	60.30	7.36	67.40	8.27
Block 3	64.60	10.58	75.80	9.92	65.90	7.77	70.30	8.79

CHAPTER IV

DISCUSSION

No significant main effects were found for ACA status on the three dependent measures examined in this study. These results could be interpreted to support Reed, et al. (1982), whose findings led them to conclude that cognitive performance in non-intoxicated subjects cannot be predicted by family history for alcoholism. The present results do appear to contradict those of Schaeffer, et al. (1984) and Tarter et al. (1984), who found ACA male subjects to perform more poorly than nonACA's on neuropsychological measures, with neither study examining subjects while under the influence of alcohol. The present study was unique in providing an examination of cognitive functioning of ACA subjects under the influence of alcohol at baseline, peak and descent phases of acute intoxication. The significant three-way interaction between ACA status, alcohol treatment condition and phase of session for the Digit Span backward measure is most interesting in possibly isolating a cognitive ability that differentiates ACA from control subjects in their response to alcohol. It was hypothesized that ACA subjects would perform more poorly than controls under the influence of alcohol and would continue to show relative deficits when measures were reported at the descent phase of session. The results of this study supported an opposite conclusion that ACA subjects were able to recover from alcohol effects quicker than controls as indicated by

superior Digit Span backward performance at descent. Figure 3 illustrates this interesting effect.

Significant differences were not detected in Digit Span forward or backward performance between ACA and control subjects in neither the alcohol or placebo treatment conditions at baseline which appears to demonstrate equivalency in ability to mentally attend, concentrate and juggle information in short-term memory. It should be recalled that Digit Span scores have been associated with the P300 component of an evoked potential. Previous research has claimed delayed P300 latencies in young ACA males prior to the development of a drinking history (Begleiter & Porjesz, 1980; 1983; 1984; Elmasian, et al., 1982), leading to an unsupported hypothesis that Digit Span baseline deficits would be found among ACA subjects. Apparently, the Digit Span task represents a rough correlate of evoked potential parameters which was not sensitive enough in the present study to reveal difference between ACA's and controls. Digit Span backward performance was effected deleteriously by alcohol for both ACA and control subjects to a similar degree at the peak of intoxication, failing to support the anticipated short-term memory differentials between subjects differing in family history for alcoholism. Interestingly, significant differences were found to appear at the descent phase of the session, demonstrating more rapid recovery of performance by ACA males subjects under the influence of alcohol.

Evidence exists that ACA and control subjects differ in their expectancies about the effects of alcohol (O'Malley et al., 1985; Savoie, et al., 1988; Vogel-Sprott et al., 1986; Schuckit, 1980,1984). Several studies have consistently found ACA males to differ from nonACA males in their perceptions of and reactions to the effects of alcohol in spite of the fact that blood alcohol levels were the same for both groups. These studies found that ACA males reported themselves as less intoxicated than nonACA males. It was also found that ACA males showed more stable mood-state ratings than nonACA males in response to the course of alcohol absorption and elimination from the bloodstream. Schuckit (1984) hypothesized that the subjective responses to the effects of alcohol may predispose ACA males to a greater risk for the development of alcoholism. The rationale is that ACA males are insensitive to the internal cues associated with acute alcohol intoxication and therefore are unable to recognize the drug effect and modulate their drinking. This finding is particularly marked when ACA subjects are given moderate doses of alcohol, similar to the amount administered in the present study. Schuckit found that differences between male ACA and nonACA subjective responses differ more at low blood alcohol concentrations than at higher doses. ACA subjects in the present study seemed to be able to recuperate faster from the effects of alcohol as supported by significant improvement in their Digit Span backward performance, while neither placebo group showed significant changes across phase of session. The Digit

Span backward task appears to be a more sensitive measure than the Digit Span forward task since it requires more effortful mental activity.

It has been suggested that ACA and nonACA subjects differ in their expectancies about the effects of alcohol because individuals who have lived with an alcoholic family member are likely to have had different exposure to its effects (O'Malley & Maisto, 1985). The present comparisons between ACA and control subjects in the placebo conditions would not appear to support hypotheses about differences in expectancy effects adversely influencing the measures examined in this study. However, the differences in performance observed at the descent phase of session between ACA and control subjects in the alcohol condition appear to support the findings of subjective response differences between the two groups at low blood alcohol concentrations. In view of the fact that the present study did not find significant expectancy effects in the placebo conditions, it could be speculated that the observed differences may be due to innate neuropsychological sensitivity differences in ACA and control subjects. Perhaps the ACA subjects were experiencing less overall subjective alcohol effect due to acute sensitivity. This could explain the rapid recovery in Digit Span backward performance for this group. Both groups reported similar drinking histories, therefore it is unlikely that the differential brain sensitivity was acquired through years of drinking. Rather, it could be argued that male ACA's may be

predisposed to develop an acute tolerance to the effects of alcohol. Digit Span backward appears to be a sensitive measure for detecting this brain sensitivity to low blood alcohol concentrations in nonalcoholic ACA subjects.

Significant main effects for alcohol were found for Digit Span Forward and Backward and the Digit Symbol measures, but not on Trail Making B. This would suggest that Digit Span and Digit Symbol are sensitive to the acute effects of alcohol for young nonalcoholic males, but the Trail Making B measure is not.

This result seems to support the findings of Leckliter and Matarazzo (1989). After reviewing the influence of age, gender, education, IQ and alcohol abuse on the Halstead-Reitan neuropsychological test battery (HRB), they concluded that at least five of the HRB tests appear to be sensitive to the effects of alcohol, one of which was Trail Making B. However, they cautioned against attributing poorer scores solely to the effects of alcohol. Age, gender, education and IQ may also influence performance on these measures and should be considered when assessing the influence of alcohol on performance. Moreover, Eckardt, Ryback and Paulter (1980) reported that drinking history is the best predictor of performance on the HRB, accounting for seventy percent of the variance. The subjects in the present study were not alcoholics nor did they report problem drinking histories. The studies presented in the literature review, citing decreased performance on Trail Making B with FH+ males may be reflecting cumulative effects of alcohol

for those subjects that do not appear to exist for the subjects in the present study.

Analysis of Trail Making B did, however, reveal a main effect for phase of session. Both treatment groups showed an increase in performance from block 1 to block 2 to block 3. This indicates that subjects in both conditions were able to significantly improve their performance by the third trial. It may be suggested that the observed increase in performance can be attributed to practice effects experienced by both groups. The two-way interaction effect of alcohol x block provides additional support for this hypothesis. The placebo group showed a significant increase in performance from block 1 to block 2, whereas the alcohol treatment group evinced a significant improvement from block 2 (peak BAL) to block 3. It appears that although, alcohol did not significantly decrease performance at block 2 it did hamper any practice effect that was observed in the placebo group. Interestingly, the alcohol subjects were able to match their performance to that of the placebo group at block 3 (M= 44.8 and 44.7 respectively). This again supports the notion that Trail Making B may not be a sensitive measure for effects of acute alcohol intoxication in nonalcoholic males who do not have histories of high levels of alcohol consumption.

Analyses of the Digit Symbol measure also revealed a significant main effect for alcohol. Individuals who perform well on this task appear to be learning the nonsense symbols associated with the number in the key and are thereby able to perform the task

at a faster speed. Conversely, individuals who demonstrate poorer performance may not be learning the nonsense symbols and are required to look up at the key more often, slowing down their performance. The results of this study appear to indicate that alcohol hinders this on-the-spot learning, in addition to attentiveness, visual-motor dexterity and speed in the performance of this task for nonalcoholic males. The puzzling finding with this measure was the absence of significant improvement for the placebo groups from block 1 to block 2, who subsequently demonstrated significantly improved performance from block 2 to block 3. These results are difficult to interpret. It is possible that subjects in the placebo conditions were experiencing a negative expectancy effect at block 2 which may have compromised their performance. This interpretation appears to be contradicted by the results that were found on the Trail Making B measure from block 1 to block 2, where subjects in the placebo conditions show significant improvement. However, in an exploratory effort to determine whether the observed recovery of cognitive functioning in detoxified alcoholics was a function of time or experience, Goldman, et al. (1985) found that recovery of performance on Trail Making B was not time-dependent but experience-dependent. On the other hand, improved performance on the Digit Symbol task was determined to be a function of time. Performance of Trail Making B and the Digit Symbol tasks requires visual-information processing, attention, hand-eye coordination and speed, however, Digit Symbol requires the additional task of

learning. Therefore, it could be speculated that this measure is more robust to practice effects with repeated measures. The lack of comparison ACA and nonACA control groups in determining practice effects following repeated measures does not allow for clear interpretation of these results.

The present study investigated cognitive functioning of nonalcoholic ACA and nonACA males while under the influence of alcohol. Results of this study did not indicate significant differences between ACA and control subjects in baseline functioning on any of the three dependent measures. These findings have been supported by previous research in this area. Studies that have found cognitive deficits in ACA males were examining subjects that were drinking heavily and determined to already be at high risk for alcoholism (Goodwin et al., 1975; Tarter et al., 1984; Drejer, et al., 1985). The ACA subjects who participated in this study were carefully screened for alcoholism and alcohol abuse problems, intended to exempt them from a "Type 2" alcoholic classification which Cloninger (1988) linked to genetic heritage. Previous ACA research with such "high risk" drinking subjects appears to confound the research design by confusing ACA and drinking history effects. The present study focused on ACA effects by examining only male social drinkers. Acute intoxication studies with subjects who already drink excessively also has been questioned on ethical grounds.

General Conclusions

The present literature review examined studies which suggested both P300 evoked potential delayed latencies and deficits in neuropsychological performance among adult children of alcoholics. These finding have been interpreted by many researchers as the first step in identifying a neurological deficiency or marker for risk of alcohol dependency. Other studies have revealed diminished responsiveness of ACA subjects on measures of mood state, bodily sensations, perceived level of intoxication and other more subjective indicators which describe a more robust psychological response to the substance, which would appear less consistent with neurological impairment. The present results appear to support the latter model by demonstrating an absence of baseline differences on three neuropsychological measures and apparent increased ACA resiliency in recovering from the effects of acute alcohol intoxication on the Digit Span backward measure.

Perhaps the apparent inconsistencies in "impaired" and "robust" interpretations of ACA neuropsychological functioning can be accounted for by closer future examination of selection criteria in ACA studies. Neuropsychological differences in adulthood may indeed be largely a function of the drinking history of the subject. Further, P300 idiosyncrasies of children of alcoholics may reveal a fascinating neuropsychological correlate to the subjective differences observed in many ACA subjects by their response to acute alcohol intoxication. Further evoked potential research may

combine cognitive, mood state and behavioral measures to test hypotheses about associations between brain wave functioning and subjective response to alcohol. Sensory modalities involved in testing must be given far greater attention. For example, the Digit Span backward task is entirely auditory in nature, much like the P300 studies which use predominantly auditory evoked potential. Visual evoked potential studies were much less consistent in revealing ACA differences. These future research considerations may help to explain the apparent divergent findings of previous studies.

Finally, results of the present study did provide a measure which seems to be sensitive in differentiating between ACA and nonACA subjects while under the influence of alcohol. The Digit Span backward test provides a most interesting measure because it appears to be able to detect differences in acute sensitivity to alcohol between male ACA and non ACA subjects who do not report problem drinking histories. Although the present study did not measure subjective responses, it appears to support previous research citing subjective response differences between ACA and nonACA subjects in their reactions to alcohol intoxication.. This interpretation would indicate the importance of subjective ratings to the effects of alcohol intoxication in future research with high risk for alcoholism males. Continued research in this area may

allow for the detection of individuals at high risk for developing alcoholism before it becomes a serious problem to them, their families and society.

APPENDIX A

MICHIGAN ALCOHOL SCREENING TEST (MAST)

Please circle either Yes or No for each item as it applies to you.

Yes No (2)	1. Do you feel you are a normal drinker?
Yes No (2)	2. Have you ever awakened the morning after some drinking the night before and found that you could not remember a part of the evening
Yes No (1)	3. Does your wife/husband (or do your parents) ever worry or complain about your drinking?
Yes No (2)	4. Can you stop drinking without a struggle after one or two drinks?
Yes No (1)	5. Do you ever feel bad about your drinking?
Yes No (2)	6. Do friends or relatives think you are a normal drinker?
Yes No (0)	7. Do you ever try to limit your drinking to certain times of the day or to certain places?
Yes No (2)	8. Are you always able to stop drinking when you want to?
Yes No (5)	9. Have you ever attended a meeting of Alcoholics Anonymous?
Yes No (1)	10. Have you ever gotten into fights when drinking?
Yes No (2)	11. Has drinking ever created problems with you and your wife/husband?
Yes No (2)	12. Has your wife/husband (or other family member) ever gone to anyone for help aboutyour drinking?

Yes	No	(2)	13. Have you ever lost friends or girlfriends/boyfriends because of your drinking?
Yes	No	(2)	14. Have you ever gotten into trouble at work because of drinking?
Yes	No	(2)	15. Have you ever lost a job because of drinking?
Yes	No	(2)	16. Have you ever neglected your obligation, your family, or your work for two or more days in a row because you were drinking?
Yes	No	(1)	17. Do you ever drink before noon?
Yes	No	(2)	18. Have you ever been told you have liver trouble? Cirrhosis?
Yes	No	(5)	19. Have you ever had delirium tremens (DT's), severe shaking, heard voices, or seen things that weren't there after heavy drinking?
Yes	No	(5)	20. Have you ever gone to anyone for help about your drinking?
Yes	No	(5)	21. Have you ever been in a hospital because of drinking?
Yes	No	(2)	22. Have you ever been a patient in a psychiatric hospital or on a psychiatric ward of a general hospital where drinking is part of the problem?
Yes	No	(2)	23. 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 played a part?
Yes	No	(2)	24. Have you ever been arrested, even for a few hours, because of drunken behavior?
Yes	No	(2)	25. Have you ever been arrested for drunk driving after drinking?

APPENDIX B

CHILDREN OF ALCOHOLICS SCREENING TEST

(CAST)

Please check the answer below that best describes your feelings, behavior, and experiences related to a parent's alcohol use. Take your time and be as accurate as possible. Answer all 36 questions by checking either "Yes" or "No."

Sex: Male_____ Female_____ Age_____

Father		Mother			QUESTIONS
Yes	No	Yes	No		
___	___	___	___	1.	Have you ever thought that one of your parents had a drinking problem?
___	___	___	___	2.	Have you ever lost sleep because of a parent's drinking?
___	___	___	___	3.	Did you ever encourage one of your parents to quit drinking?
___	___	___	___	4.	Did you ever feel alone, scared, nervous, angry, or frustrated because a parent was not able to quit drinking?
___	___	___	___	5.	Did you ever argue or fight with a parent when he or she was drinking?

___ ___ ___ ___ 6. Did you ever threaten to run away from home because of a parent's drinking?

___ ___ ___ ___ 7. Has a parent ever yelled at or hit you or one other family member when drinking?

___ ___ ___ ___ 8. Have you ever heard your parents fight when one of them was drunk?

___ ___ ___ ___ 9. Did you ever protect another family member from a parent who was drinking?

___ ___ ___ ___ 10. Did you ever feel like hiding or emptying a parent's bottle of liquor?

___ ___ ___ ___ 11. Do many of your thoughts revolve around a problem drinking parent or difficulties that arise because of his or her drinking?

___ ___ ___ ___ 12. Did you ever wish that a parent would stop drinking?

___ ___ ___ ___ 13. Did you ever feel responsible for and guilty about a parent's drinking?

___ ___ ___ ___ 14. Did you ever fear that your parents would get divorced due to alcohol?

___ ___ ___ ___ 15. Have you ever withdrawn from and avoided outside activities and friends because of embarrassment and shame over a parent's drinking problem?

___ ___ ___ ___ 16. Did you ever feel caught in the middle of an argument or fight between a problem drinking parent and your other parent?

___ ___ ___ ___ 17. Did you ever feel that you made a parent drink alcohol?

___ ___ ___ ___ 18. Have you ever felt that a problem drinking parent did not really love you?

___ ___ ___ ___ 19. Did you ever resent a parent's drinking?

___ ___ ___ ___ 20. Have you ever worried about a parent's health because of his or her alcohol use?

___ ___ ___ ___ 21. Have you ever been blamed for a parent's drinking?

___ ___ ___ ___ 22. Did you ever think your father was an alcoholic?

___ ___ ___ ___ 23. Did you ever wish your home could be more like the homes of your friends who did not have a parent with a drinking problem?

___ ___ ___ ___ 24. Did a parent ever make promises to you that he or she did not keep because of drinking?

___ ___ ___ ___ 25. Did you ever think your mother was an alcoholic?

___ ___ ___ ___ 26. Did you ever wish that you could talk to someone who could understand and help the alcohol-related problems in your family?

___ ___ ___ ___ 27. Did you ever fight with your brothers and sisters about a parent's drinking?

___ ___ ___ ___ 28. Did you ever stay away from home to avoid the drinking parent or your other parent's reaction to the drinking?

___ ___ ___ ___ 29. Have you ever felt sick, cried, or had a "knot" in your stomach after worrying about a parent's drinking?

___ ___ ___ ___ 30. Did you ever take over any chores and duties at home that were usually done by a parent before he or she developed a drinking problem?

___ ___ ___ ___ 31. Is this your biological parent?

___ ___ ___ ___ 32. Does this parent <u>presently</u> drink excessively in your opinion?

___ ___ ___ ___ 33. Has this parent ever been physically abusive to your mom/dad while under the influence of alcohol?

___ ___ ___ ___ 34. Has this parent ever been abusive to you while under the influence of alcohol?

___ ___ ___ ___ 35. Do you believe that this parent's father (your grandfather) had a drinking problem?

___ ___ ___ ___ 36. Do you believe that this parent's mother (your grandmother) had a drinking problem?

____ ____ **Total Number of "YES" Answers**

APPENDIX C

Khavari Alcohol Test (KAT)

Name_____ Date_____

This is a series of questions about the use of alcoholic beverages. What beverages people drink, how much, and how often. Please check the statement <u>that best applies to you</u>.

1. How often do you usually drink beer? _____
2. How often do you usually drink wine? _____
3. How often do you usually drink whisky or liquor?_____

A. daily
B. 3 or 4 times a week
C. twice a week
D. once a week
E. 3 or 4 times a month
F. twice a month
G. once a month
H. 3 or 4 times a year
I. twice a year
J. once a year
K. I have tried, but don't drink it now
L. I have never tried

---

4. Think of all the times you have had <u>beer</u> recently. When you drink beer, how much beer do <u>YOU USUALLY DRINK</u> each time in cans or glasses?

_____ cans or glasses _____ I don't drink beer.

Think of all the times you have had <u>wine</u> recently. When you drink wine, how much wine do <u>YOU USUALLY DRINK</u> each time in glasses (4 oz.)?

_____ glasses _____ I don't drink wine.

Think of all the times you have had whiskey or liquor recently. When you drink whiskey or liquor, how much do YOU USUALLY DRINK each time (in mixed drinks, approximately 1 oz. shots)?

_____ drinks _____ I don't drink liquor.

---

5. Each time you drink beer, what is the MOST YOU DRINK at one time?

_____ cans or glasses _____ I don't drink beer.

Each time you drink wine, what is the MOST YOU DRINK at one time?

_____ glasses _____ I don't drink wine.

Each time you drink liquor, what is the MOST YOU DRINK at one time?

_____ drinks _____ I don't drink liquor.

---

6. [USE THE RESPONSE POSSIBILITIES FROM QUESTION #1]

How often do you drink this MOST amount of beer? ____

How often do you drink this MOST amount of wine? ____

How often do you drink this MOST amount of liquor? ____

APPENDIX D

Consent Form

You are invited to participate in a study about the effects of acute doses of alcohol on cognitive functioning. You are being asked to participate in the study because your responses to a previous screening questionnaire suggested that you would be able to tolerate moderate doses of alcohol. All information collected during the screening and data collection facets of this study will be kept strictly confidential. All questionnaire and data protocols will be coded with the key maintained by the project director. All data will be kept in locked quarters.

You will be asked to present your driver's license or equivalent identification to determine that your age is 21 years or older prior to further participation.

Prior to consumption of alcohol you will be given a vocabulary test and a series of tests to assess your cognitive functioning, after which you will be asked to consume a drink that will contain either alcohol or a non-alcohol beverage. The amount of beverage that you will receive will be 1.0 mL per kilogram of your body weight. The dose will be divided into two drinks, and you will be given 40 minutes to consume both drinks.

We understand that you consumed no food or beverages other than water within the previous four hours. We also understand that you have not ingested any drugs including alcohol, caffeine, nicotine or any medications within the past 24 hours. We understand that you agree to remain in the lab until you are deemed sober by a breath estimate of your blood alcohol level (BAL < .02) which will require approximately 3 to 4 hours. You agree to allow us to drive you home if transportation is necessary.

All information gathered during this study will be kept strictly confidential and no identifiable individual results will be released. You will be assigned a code number which will be used on all forms. You may discontinue participation in the study at any time that the procedure makes you feel personally uncomfortable.

There is a slight possibility you may experience some nausea if you are administered alcohol. This possibility is very unlikely since the dosage has been administered safely many times before, and your drinking history suggests tolerance within the acceptable range for the moderate amount used in this study.

The benefits from participation in this study are improved understanding of how alcohol influences cognitive functioning. You may consider your participation of educational benefit, learning from your performance as a subject in a scientific investigation. You will be assigned randomly to the treatment condition in order to insure unbiased results.

You will receive class credit in return for participation in accordance with the amount of time that you spend in this experiment. Your decision whether or not to participate will not prejudice your future relations with UND or the Psychology Department. If you decide not to participate, you are free to discontinue at any time without prejudice.

The investigators involved will make themselves available to answer any questions that occur to you in the future. You may direct any questions to either Bette Bakke at 777-3017 or Dr. Alan King at 777-3644. You will be given a copy of this form if you wish to have one. Medical treatment will be available as it is to any member of the general public in similar circumstances. Payment for any such treatment must be provided by you or your third party payor.

I have read all of the above information and willingly agree to participate in this study as explained to me by:

_____	_____	_____
Research Assistant	Date	Subject

_____	_____
Witness	Date

APPENDIX E
SUMMARY ANOVA TABLES

TABLE 3

Summary ANOVA for Digit Span Forward

Source	SS	df	MS	F-Test	Sig.
ACA	8.533	1	8.533	1.106	0.301
Alcohol	67.500	1	67.500	8.747	0.006
ACA x Alcohol	4.800	1	4.800	0.622	0.436
Error	277.806	36	7.717		
Phase of Session	2.817	2	1.408	0.859	0.428
ACA x Phase of Session	2.817	2	1.408	0.859	0.428
Alcohol x Phase of Session	2.150	2	1.075	0.656	>0.500
ACA x Alc. x Phase of Session	1.550	2	0.775	0.473	>0.500
Phase of Session x Error	118.000	72	1.639		
Total	485.972	119	4.084		

TABLE 4

Summary ANOVA for Digit Span Backward

Source	SS	df	MS	F-Test	Sig.
ACA	9.075	1	9.075	1.260	0.270
Alcohol	52.008	1	52.008	7.219	0.011
ACA x Alcohol	0.675	1	0.675	0.094	> 0.500
Error	259.367	36	7.205		
Phase of Session	14.150	2	7.075	4.584	0.014
ACA x Phase of Session	4.650	2	2.325	1.506	0.229
Alcohol x Phase of Session	8.717	2	4.358	2.824	0.066
ACA x Alc. x Phase of Session	15.350	2	7.675	4.972	0.010
Phase of Session x Error	111.133	72	1.544		
Total	475.124	119	3.993		

TABLE 5

Summary ANOVA for Trail Making B

Source	SS	df	MS	F-Test	Sig.
ACA	136.535	1	136.533	0.237	0.500
Alcohol	67.500	1	512.529	0.891	0.352
ACA x Alcohol	488.033	1	488.033	0.848	0.364
Error	20711.806	36	575.331		
Phase of Session	1168.517	2	584.259	3.510	0.036
ACA x Phase of Session	665.817	2	332.909	2.000	0.143
Alcohol x Phase of Session	1115.217	2	557.608	3.349	0.041
ACA x Alc. x Phase of Session	233.317	2	116.6580	.701	>0.500
Phase of Session x Error	11986.438	72	166.478		
Total	37018.438	119	311.078		

TABLE 6

Summary ANOVA for Digit Symbol

Source	SS	df	MS	F-Test	Sig.
ACA	276.032	1	276.032	1.270	0.268
Alcohol	918.531	1	918.531	4.225	0.048
ACA x Alcohol	83.333	1	83.333	0.383	>0.500
Error	7827.305	36	217.425		
Phase of Session	237.649	2	118.825	9.067	<0.001
ACA x Phase of Session	17.117	2	8.558	0.653	>0.500
Alcohol x Phase of Session	239.517	2	119.75	9.139	<0.001
ACA x Alc. x Phase of Session	53.517	2	26.758	2.042	0.138
Phase of Session x Error	43.529	72	13.105		
Total	10596.516	119	89.046		

TABLE 7

Summary ANOVA for Blood Alcohol Levels at Peak and Descent Phase of Session

Source	SS	df	MS	F-Test	Sig.
ACA	0.000	1	0.000	1.487	0.231
Alcohol	0.058	1	0.058	591.542	< 0.001
ACA x Alcohol	0.000	1	0.000	1.487	0.231
Error	0.004	36	0.000		
Phase of Session	0.000	1	0.000	4.916	0.034
ACA x Phase of Session	0.000	1	0.000	2.306	0.138
Alcohol x Phase of Session	0.000	1	0.000	4.916	0.034
ACA x Alc. x Phase of Session	0.000	1	0.000	2.306	0.138
Phase of Session x Error	0.002	36	0.000		
Total	0.065	79	0.001		

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