12-2000

Cognitive Inhibition and Impulsivity in Adult Children of Alcoholics and Controls

Louise A. Weller

COGNITIVE INHIBITION AND IMPULSIVITY IN

ADULT CHILDREN OF ALCOHOLICS AND CONTROLS

by

Louise A. Weller
Master of Arts, University of North Dakota, 1997

A Dissertation

Submitted to the Graduate Faculty

of the

University of North Dakota

In partial fulfillment of the requirements

for the degree of

Doctor of Philosophy

Grand Forks, North Dakota
December
2000

This dissertation, submitted by Louise A. Weller in partial fulfillment of the requirements for the Degree of Doctor of Philosophy from the University of North Dakota, has been read by the Faculty Advisory Committee under whom the work has been done and is hereby approved.

_____
(Chairperson)

_____

_____

_____

_____

This dissertation 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.

_____
Dean of the Graduate School

_____
8-13-00
Date

PERMISSION

Title   Inhibitory processes and Impulsivity as measured with a group
of Adult Children cf Alcoholics and a Control Group

Department   Psychology

Degree   Doctor of Philosophy

In presenting this dissertation fulfillment of the requirements for a graduate degree from the University of North Dakota, I agree that the library of this University shall make it available for inspection. I further agree that permission for extensive copying for scholarly purposes may be granted by the professor who supervised my dissertation work or, in his absence, by the chairperson of the department or the Dean of the Graduate School. It is understood that any copying or publication or other use of this dissertation or part thereof for financial gain shall not be allowed without my written permission. It is also understood that due recognition shall be given to me and to the University of North Dakota in any scholarly use which may be made of any material in my dissertation.

Signature   _Louise A. Weller_

Date   _August 4, 2000_

TABLE OF CONTENTS

LIST OF TABLES

ACKNOWLEDGEMENTS

I would like to express my appreciation to Ric Ferraro, Alan King, Richard Wilsnack, Sharon Wilsnack, and Andrea Zevenbergen for their support and participation as members of my dissertation advisory committee. I would like to especially thank my advisor, Tom Petros, for his support, encouragement, and guidance during all phases of this dissertation.

ABSTRACT

The genetic transmission of a pattern of impairments associated with alcoholism

has been supported by research literature (Dawson, Harford, & Grant, 1992; Schuckit,

1986). No single factor appears to cause the development of a substance abuse problem,

but a family history of alcoholism may be one predictive factor (Goodwin, 1985). The

offspring of alcoholics are more likely to display disinhibited behavior and impulsivity

(Pihl, Peterson, & Finn, 1990) and are more likely to develop drinking problems than the

general population (Goodwin, 1971). Researchers have found patterns of cognitive

deficits (Tartar, Jacob, Bremer, 1989) and neuropsychological differences (Gabriella &

Mednick, 1983) associated with adult children of alcoholics (ACA) status. Several

researchers have questioned if those deficits may be associated with a set of inherited traits

which precede alcoholism, rather than be a consequence of alcohol abuse (Knop,

Teasdale, Schulsinger, & Goodwin, 1985). ACAs have also been found to display deficits

in learning new material presented in a visual paradigm (Schandler, Cohen, & Antick,

1992). This study addressed the relationship between ACA status, cognitive inhibition,

impulsivity, and visuospatial learning in ACAs. It was proposed that groups of male and

female ACAs, as compared to control groups of male and female nonACAs, would exhibit

heightened impulsivity and specific deficits in cognitive inhibition, as measured by tests

purported to find differences between groups in these domains.

CHAPTER I

INTRODUCTION

It is only in the last three decades that the term "adult children of alcoholics" (ACA) has been used. Research has identified this group of individuals as a distinct population, requiring intervention and treatment specifically for the problems resulting from being the offspring of an alcoholic parent (Brown, 1988). Researchers in this field have found that alcohol dependence and problem drinking, generally referred to as alcoholism, affects more than just the individual who imbibes. It also has a deep and lasting effect on the family of the problem drinker. It is estimated that more than 28 million Americans share the experience of growing up in an alcoholic home (Brown, 1983). There is evidence supporting the theory that these individuals are at elevated risk for alcoholism themselves (e.g., Goodwin, 1971; Cotton, 1979; Pollock, Schneider, Gabrielli, & Goodwin, 1987; Sher, Walitzer, Wood, & Brent, 1991) though it has yet to be determined what proportion of that risk can be attributed to hereditary versus environmental factors (Woodside, 1983). Epidemiological data suggest that approximately 25% of the sons of alcoholics will themselves develop serious drinking problems while the figure for the population as a whole is only 4% (Goodwin, 1971).

The first published study which at all addressed the issue of children of alcoholics was included as part of memoir writings by Roe and Burks (1945, as cited in Brown,

1983), in which they noted that the children of alcoholics were often themselves alcoholic. Over the next decade, literature on alcoholism and its effects on families centered primarily on the alcoholic and the alcoholic's behavior. This singular focus on the alcoholic began to change with the work of Jackson (1954) who outlined stages in a developmental disease process of alcoholism for the spouse and family of the alcoholic. Fox (1962) built on this work by suggesting that every member of the family of the alcoholic is uniquely affected by the drinking parent. These early studies drew attention to the problem of alcoholism as a family disease and the focus of research began to shift from the alcoholic alone to include the interactions, adjustments, and development of the family with the alcoholic member.

It is Cork (1969) who is credited with raising public and professional awareness about ACAs. Her book, The Forgotten Children, is viewed as the starting point not only of research into ACAs but a national social movement. It became a popular topic in news magazines in the 1970s, spawning support groups such as Adult Children of Alcoholics (ACOA), modeled after Al-Anon (Brown, 1983). Research literature into the unique problems of ACAs began to proliferate in the late 1970s. The research found serious and enduring negative psychological effects for children raised in the home of an active alcoholic (Schuckit, 1986; West & Prinz, 1987), where uncertainty and instability are often prevalent. As previously mentioned, research has indicated that about one-quarter of ACA male children grow up to become alcoholics themselves. In a prospective, longitudinal study of all children born on the island of Kauai in 1955, Werner (1986) found that 40% of the children of alcoholics experienced serious problems coping with life,

whether as alcoholics themselves or with another psychiatric diagnosis, by the age of 18.

In a study of female ACAs and a comparison group of female nonACAs, Jones and

Zalewski (1994) tested the hypothesis that ACAs are more prone to depression than

nonACAs. They found that ACAs were significantly more likely to experience death or

divorce in their families, both parental and familial, more likely to have a relative with a

psychiatric illness, more likely themselves to drink heavily, and more prone to depression.

The chaotic nature of home life with an alcoholic has been suggested as one of the

principle sources of stress for the children in the home (Shinn, 1978). Parental anxiety and

depression and parental absence or neglect, problems often present in alcoholic homes,

may cause developmental, emotional, and cognitive problems in children. Ervin, Little,

Streissguth, and Beck (1984) assessed 50 children raised by an alcoholic father and 50

children raised by a nonalcoholic father. Only 19 of the alcoholic fathers were the

biological parent. The children of the alcoholic fathers scored significantly lower than the

children of the nonalcoholic fathers on the Wechsler Intelligence Scale for Children-

Revised (WISC-R) Full Scale IQ, Performance IQ, and Verbal IQ. The authors concluded

that intellectual functioning was related to the presence of an alcoholic father in the home,

the impact on the child not necessarily being the result of heredity. They suggested that

the chaotic and unpredictable nature of home life with an active alcoholic, rather than

genetic inheritance, is the source of stress for children that may lead to future antisocial

and alcoholic behavior and poorer cognitive performance (Shinn, 1978).

Many clinical reports indicate that ACAs have emotional problems and adjustment

difficulties (Ashby, Mangine, & Slaney, 1995; Black, 1979; Chafetz, Blane, & Hill, 1971;

Hectman, Weiss, & Perlman, 1984; Jones, 1968; Sher, Walitzer, Wood, & Brent, 1991)

and are more likely than nonACAs to become substance abusers (Hawkins, Catalano, &

Miller, 1992; Pandina & Johnson, 1990; Pihl, Peterson, & Finn, 1990; Turner, Cutter,

Worobec, O'Farrell, Bayog, & Tsuang, 1993). Hardwick, Hansen, and Bairnsfather

(1995) found that the presence of parental substance abuse in the home was predictive of

difficulties in reality testing in children. Chassin, Curran, Hussong, and Colder (1996)

looked at parent alcoholism effects on their children's substance abuse and found that

having an alcoholic parent was predictive of adolescent substance abuse. They also found

that those adolescents who had an alcoholic parent also had a steeper rate of substance use

than their nonACA peers.

Some researchers assert that because ACAs are also likely to be the offspring of

parents with other drug abuse and affective, anxiety, or antisocial personality disorders, it

is difficult to attribute specific problems in the children to the parental alcoholism (Helzer

& Pryzbeck, 1988). Roosa, Sandler, Gehring, Beals, and Cappo (1988) conceptualized a

model which considers parental alcoholism as a chronic condition that leads to an increase

in stressful events experienced by the child. In an alcoholic parental home the occurrence

of a range of stressful experiences, such as interparental arguments, expressions of

parental hostility to the child, neglect of the child, economic hardships, parental illnesses

and accidents, and legal repercussions would create environmental stressors that are rare

or nonexistent in the homes of other children. In addition, children of alcoholics are

known to experience more familial disruptions such as divorce and numerous residence

changes (Schulsinger, Knop, Goodwin, Teasdale, & Mikkelson, 1986). Roosa et al.

(1988) administered an instrument, the Children of Alcoholics Life-Events Schedule (COALES), to 228 adolescent children, 56 of whom identified themselves as the children of alcoholics. This questionnaire was designed to measure the stressful experiences of children in an alcoholic home. They found that the children raised in alcoholic homses experienced significantly fewer good events and significantly more bad events in their homes. The researchers theorized that at least some of the negative influence on the lives of ACAs was due to the disturbances present in the alcoholic home.

It is difficult to disentangle the genetic and environmental sources of variation which bear on the development of a child raised in an alcoholic home. There is considerable research that indicates that the disruptive alcoholic home sets the stage for later social, behavioral and psychological problems in the ACA individual (Brown & Finkelhor, 1986; Helzer & Pryzbeck, 1988; Steinglass, Bennett, Wolin, & Reiss, 1987; Wolin, Bennett, Noonan, & Teitelbaum, 1980; Wilsnack, Vogeltanz, Klassen, & Harris, 1994; Windle, Windle, Scheidt, & Miller, 1995).

There have also been numerous studies conducted with ACAs over the last three decades which present evidence to support the hypothesis that alcoholism may be an inheritable trait and family alcoholism may contribute significantly to the development of alcoholism in the offspring of alcoholic drinkers (Bohman, 1978; Chassin, Rogosch, & Barrera, 1991; Goodwin, Schulsinger, Hermansen, Guze, & Winokur, 1973; Goodwin, Schulsinger, Moller, Hermansen, Winokur, & Guze, 1974; Schuckit, Goodwin, & Winokur, 1972; Sher, Gershuny, Peterson, & Raskin, 1997). However, exactly what is inherited has yet to be agreed upon. It may be that there is a factor directly affecting

drinking behavior. Alternatively, the genetic influence may be on neuropsychological processes, personality variables, emotional lability, hyperactivity, executive functioning in the brain, or some combination of those variables.

Goodwin et al. (1973) found that sons of alcoholics who were adopted by the age of six weeks, never knowing their biological fathers, were nearly four times as likely to become alcoholic as adopted sons of nonalcoholics. Goodwin et al. (1974) found no significant differences between adopted and nonadopted sons of alcoholics in the later development of alcohol dependency, and concluded that being reared by an alcoholic parent did not affect the development of alcoholism (in males) as much as simply being the biological offspring of an alcoholic father. Schuckit et al. (1972) found that children with at least one alcoholic biological parent who were raised in nonalcoholic families with half-siblings had a higher alcoholism rate than the half-siblings who did not have an alcoholic biological parent. Bohman (1978) confirmed the findings of Goodwin's studies in research on 2,324 Swedish adoptees. Male adoptees whose natural fathers were alcoholic developed drinking problems at a 20% rate while the rate was only 6% in a comparison control group. Although the results were not statistically significant because the sample was too small, Bohman identified a similar trend for the sons of female alcoholics as well. These early studies provided evidence that genetic factors might be implicated in the vulnerability of alcoholic offspring to develop alcoholism and related psychopathology, and inspired additional studies of genetic transmission and family concordance rates.

Cotton (1979) reviewed 39 studies of the familial incidence of alcoholism and concluded that rates of alcoholism were higher among relatives of alcoholics than in the

general population: alcoholics were approximately four to five times more likely than nonpsychiatric patients to have alcoholic parents. A longitudinal study recruited several hundred boys in the Boston area between 1940 and 1963 as a study on juvenile delinquency and followed them as men at ages 25, 31, and 47. Several reports were written over the years with a complete analysis written by Drake and Vaillant in 1988. They found no significant difference between men with alcoholic fathers ($\underline{n}$ = 149) and men without alcoholic fathers ($\underline{n}$ = 250) in the prevalence of alcohol abuse (as defined by the DSM-III (American Psychiatric Association, 1980) with prevalence rates of 14% and 16%, respectively. However, they did find that alcohol dependence was more than twice as prevalent among men with alcoholic fathers, with a rate of 28% versus 12%, a ratio of 2.3 to 1. Alcohol abuse was classified as a residual category of dependence (DSM-III) marked by the occasional use of alcohol despite knowledge of persistent or recurrent problems and the risk of physical hazard. Alcohol dependence was noted as being marked by increased and marked tolerance to the substance, withdrawal symptoms, inability to control the use of the substance, and frequent intoxication.

In 1981, Cloninger, Bohman, and Sigvardsson suggested that there were two forms of alcoholism which they labeled Type 1 and Type 2. Type 1 alcoholics were those who began to drink later in life, usually due to stressors in the environment, and only incidentally did some report a family history of alcoholism. Type 1 alcoholism is associated with feeling a loss of control about the drinking behavior and guilt and fear about dependence on alcohol. Type 2 alcoholics typically had an early onset of drinking problems, a conduct-disordered youth and antisocial adulthood, and could report an

alcoholic father. This drinking behavior occurred regardless of external circumstances and was often associated with impulsive-aggressive behavior. Cloninger et al. theorized that the Type 2 alcoholic behavior was heavily dependent on genetic factors and was associated with a spectrum of behaviors and characteristics including conduct disorder, hyperactivity, sensation or novelty seeking, and antisocial personality features.

Twin studies have shown the concordance rate for alcoholism to be higher in monozygotic twins than dizygotic twins. Kaij (as cited in Silvia & Liepman, 1991) found a 71% concordance rate of alcoholism for monozygotic twins versus a 32% rate for dizygotic twins, and the more severe the alcoholism, the greater was the discrepancy between twin concordance rates. Winokur, Reich, Rimmer, and Pitts (1970) found monozygotic twins to be more concordant for alcoholism than other siblings, but only for male alcoholics and not female alcoholics. Another study, conducted by Partanen (as cited in Silvia & Liepman, 1991) in Finland found that identical twins were more concordant than fraternal twins for quantity and frequency of drinking though not for adverse consequences of drinking.

McGue, Pickens, and Svikis (1992) conducted a seven-year study of siblings for the purpose of investigating the genetic transmission of alcoholism. They analyzed the responses of 356 twin pairs. Analysis of the male same-sex twin data revealed a moderate and significant heritability. Male monozygotic twins of probands with alcohol dependence were more likely than male dizygotic twins (both siblings being male) to report alcohol and drug abuse. For women, rates of problem drinking behavior did not differ between monozygotic and same-sex dizygotic cotwins. Opposite-sex dizygotic twins showed a

cross-sex transmission: alcohol problems were greatest among male cotwins of female probands while female cotwins of male probands did not display the same rate of alcohol problems.

Some of the research evidence does seem to indicate gender differences as found in studies focusing on familial transmission of alcoholism, though the findings are mixed. Goodwin, Schulsinger, Knop, Mednick, and Guze (1977) found an increased risk for alcoholism among the adopted-away sons of alcoholics, but not the daughters. In the Cloninger et al. (1981) study, an increased risk in alcohol dependence was found in both the adopted-away sons and daughters of alcoholics, though the heritability was lower in the daughters than the sons. Bohman, Sigvardsson, and Cloninger (1981) found that although the risk for alcoholism in the sons of alcoholics is seven times greater than for nonalcoholics, the risk for alcoholism in the daughters is only four times greater (10.3%) than for the daughters of nonalcoholics (2.8%). Pickens, Svikis, McGue, Lykken, Heston, and Clayton (1991) found alcohol dependence to be inheritable by both men and women, though alcohol abuse was somewhat less heritable in women. Another twin study conducted by Gurling, Murray, and Clifford (as cited in McGue, Pickens, & Svikis, 1992) found no statistically significant differences in alcohol use between monozygotic and dizygotic twins for either males or females. In a meta-analysis of family studies, Pollock, Schneider, Gabrielli and Goodwin (1987) found that the rate of alcohol dependence was lower among the children of alcoholic mothers than the children of alcoholic fathers.

McGue, Pickens, and Svikis (1992) did not suggest that women are less likely to suffer problems with alcohol or that they have a less severe form of alcoholism. They

suggested that the sex difference in transmission of alcohol problems may not lie in differences in drinking behavior but in differences in other clinical pathology. Female twins were much more likely than male twins to report treatment for depression and other forms of substance abuse. The increased use of drugs among female twins was most often abuse of prescription drugs rather than street drugs, again suggesting a different societal pattern.

The finding of higher rates of depression in ACA women corroborates findings by Goodwin, Schulsinger, Knop, Mednick, and Guze (1977) as well as other researchers (Winokur, 1971; Winokur & Coryell, 1991). Berkowitz and Perkins (1988) assessed 860 college students and found that ACAs were more likely to report greater self-depreciation than nonACAs, with the difference between ACAs and nonACAs being greater for women than men. Bush, Ballard, and Fremouw (1995) assessed 57 ACA college students and 100 nonACA college students for depression. They found that ACAs had higher levels of depressive features, lower self-esteem, and a depressive attributional style; females had higher overall scores on the rating of depression than males.

Dawson and Grant (1996) examined data collected from 42,862 U.S. adults to explore the relationship between familial alcohol history and alcohol dependence and depression. The data was drawn from the 1992 National Longitudinal Alcohol Epidemiologic Survey, sponsored by the National Institute on Alcohol Abuse and Alcoholism, and was collected by personal interviews. Slightly more than 50% of the adults surveyed had positive family histories of alcoholism with 9.4% reporting 25% or more of their first- and second-degree relatives as alcoholic. The survey found the

predicted evidence for the transmission of familial alcoholism, with the odds of having experienced lifetime alcohol dependence, alone or comorbid with major depression, increasing in direct proportion to the percentage of first- and second-degree relatives identified as alcoholics. Men with a positive family history (FH+) of alcoholism were more likely to report alcohol dependence (20.3%) than men with a negative (FH-) family history (8.7%). FH+ men were more likely to report comorbid alcohol dependence and depression (6.3%) than FH- men (1.4%); FH+ men were more likely to report depression alone (6.3%) than FH- men (3.1%). FH+ women were more likely to report alcohol dependence (8.4%) than FH- women (2.8%); FH+ women were more likely to report comorbid alcohol dependence and depression (4.0%) than FH- women (0.8%). FH+ women were more likely to report depression alone (11.6%) than FH- women (4.6%). More men were likely to report alcohol dependence (14.6%) than women (5.9%) while more women (8.4%) were likely to report depression than men (4.7%). The highest rates of alcohol dependence and depression were found in those individuals who reported the greatest number of alcoholic relatives.

Exactly what is being inherited has been the subject of many studies. Subsequent to the Cloninger et al (1981) report, other researchers have documented correlations between early conduct disordered behavior and ACA status (Belliveau & Stoppard, 1995; Knop, Teasdale, Schulsinger, & Goodwin, 1985; Sher & McCrady, 1984; Vaillant, 1983). ACA youth have consistently been described as conduct disordered (Cadoret & Gath, 1978; Chafetz, Blane, & Hill, 1971; Schulsinger, Knop, Goodwin, Teasdale, & Mikkelson, 1986; Tartar, Hegedus, Goldstein, & Alterman, 1984). Sher and McCrady (1984)

reported higher scores on the MacAndrew Alcoholism Scale (MAC; MacAndrew, 1965) and Minnesota Multiphasic Personality Inventory (MMPI) subscale scores for school maladjustment for ACA adolescents as compared to a group of nonACA adolescents. In a Danish longitudinal study, teachers who were blind to children's ACA status rated 134 students who were ACA as significantly more impulsive with more behavioral undercontrol than 70 children who were nonACA (Knop, Teasdale, Schulsinger, & Goodwin, 1985).

Belliveau and Stoppard (1995) compared 118 ACA college students (88 female, 30 male) and 307 nonACA students on their responses to the Clinical Analysis Questionnaire (CAQ; Cattell & Sells, 1974), which yields scores on personality factors and dimensions of psychopathology. They found higher scores for the ACA students on measures of depression, psychoticism and neuroticism, as well as general maladjustment. Sher, Walitzer, Wood, and Brent (1991) compared 253 ACA college freshman and 237 nonACA college freshmen on alcohol and drug use, psychopathology, cognitive ability, and personality. The ACAs reported more alcohol and drug problems, higher rates of psychopathology such as major depressive episodes and various anxiety disorders, and scored higher on indices of behavioral undercontrol, psychoticism, neuroticism, and impulsivity.

Pihl, Peterson, and Finn (1990) described male ACAs as being characterized by conduct disorder or antisocial personality, and as children or adolescents who often presented with a comorbid hyperactivity/attention deficit disorder. Sons of male alcoholics, as a group, appear to be impaired in their ability to concentrate, pay attention,

and control their motor behavior sufficiently. As compared to groups of males whose pedigrees do not include a significant family history of alcoholism, they tend to be quicker to resort to aggression in social situations. They often seem to delight in breaking the rules and, though they are sometimes gregarious and extroverted, they often get into trouble with others.

Opposing evidence has also been reported. A comparison study of college-age men conducted by Alterman, Bridges, and Tarter (1986) found no differences in drinking behavior or conduct behavior or consequences from drinking between a group with a family history of alcoholism and a group with no family history. Alterman, Searles, and Hall (1989) tested 27 ACA subjects with a first-degree relative who was alcoholic (mother or father), 26 ACA subjects with a second-degree relative who was alcoholic and 30 nonACA subjects. They found no differences between the three groups in their drinking behavior, their scores on the MacAndrew Alcoholism Scale, measures of adolescent antisocial behavior, or measures of sensation seeking or hyperactivity.

There have also been studies with mixed results. Knop, Teasdale, Schulsinger, and Goodwin (1985) found no differences in the drinking behavior of 18- and 19-year-old sons of alcoholic and nonalcoholic fathers, though they did find the groups differed on some aspects of impulsivity and early conduct problems. Schuckit and Sweeney (1987), in a similar study, found no difference in alcohol intake between young men of legal drinking age based on family history. They did, however, find that the individuals with more of a family pedigree of problem drinking displayed more alcohol-related consequences from their drinking.

Other researchers have found significant differences between ACA individuals and nonACA individuals in comparisons of physiological, neurological, neuropsychological, and cognitive measures. Finn, Earleywine, and Pihl (1991) found some interesting physiological differences between their groups of subjects (see description of groups earlier), exploring the concept that men with a multigenerational family history of alcoholism would have a stimulus-response regulatory deficit. This deficit had been manifested as a cardiovascular hyperreactivity to unavoidable shock, or overactivated response. Most published research studies in which unavoidable electric shock was delivered, found response inhibition and cardiac deceleration to be the norm (Finn, Zeitouni, & Pihl, 1990). In this study, the multigenerational family history group experienced an increased heart rate change as a result of exposure to unavoidable shock as compared to the groups with no such pedigree. The men with a multigenerational family history of alcoholism were also more sensitive to alcohol's reactivity dampening effect (they experienced a lowered cardiovascular response as a result of alcohol ingestion). Previous studies had indicated that ACA individuals were more responsive to alcohol's dampening effect (Finn & Pihl, 1988). As predicted, they found that their multigenerational group reacted significantly differently than the father-only group and the nonACA group with a significantly dampened reactivity after ingestion of alcohol. The authors also administered the Neuroticism and Extroversion subscales and the four subscales of the Sensation Seeking Scale (Eysenck, 1975). A canonical discriminant analysis found that sensation seeking and disinhibitory personality traits loaded onto the same canonical variable with the significant physiological responses of cardiovascular

hyperreactivity to unavoidable shock and increased sensitivity to the reactivity-dampening effects of alcohol for the multigenerational group.

Other physiological differences between ACA groups and nonACA groups have been found in response to alcohol challenge tests. In an alcohol challenge test, participants drank a beverage (which may or may not have been alcohol) and then subjectively rated themselves on a scale from 0 to 36 on several feelings associated with intoxication, such as feeling "high", "intoxicated","sleepiness","floating sensation", and "nausea" (Schuckit, 1987). The participants were also rated on their eyes-open steadiness while standing, or body sway, in an apparatus that evaluated levels of sway in both anterior-posterior and lateral planes. This alcohol challenge test was administered to 454 men between the ages of 18 and 25 who were evenly divided between ACA and nonACA status. Approximately 40% of the participants with positive family histories had significantly lower levels of change on body sway (static ataxia), or change in movement from steadiness, and reported significantly lower scores on their subjective feelings of intoxication. Schuckit (1994b) followed up 222 men ten years after the initial assessment to find if there was any difference in alcohol dependency between the ACA and nonACA groups. By the time of follow-up, 42 (34%) of the ACA men had developed alcohol abuse or dependence while only 13 (13%) of the nonACA group had developed drinking problems. When analyzing the physiological data, or body sway, from the original assessment to the later rate of alcohol abuse of the participants, the subjects among the 20% with the least response to alcohol had an alcoholism rate of 43% while those among the 20% with the greatest response had a rate of only 11%. These findings were

replicated and further confirmed in a second and larger follow-up study conducted by Schuckit and Smith (1997).

With respect to the central nervous system, disturbances in the regulation of motor processes appear to be associated with an increased risk for developing alcoholism. Hegedus, Tartar, Hill, Jacob, and Winsten (1984) replicated the findings of Lipscomb, Carpenter and Nathan (1979) in a study measuring static ataxia, or upper body sway. In the Hegedus et al. study, 20 young ACA men, 22 young men who were the sons of depressed fathers, and 15 nonACAs whose fathers also had no other psychiatric diagnosis, were compared. The ACA sons were significantly more ataxic than sons of depressed fathers or normal controls, with the two latter groups not significantly different from each other. All groups were sober when tested. It should be noted, however, that the findings of static ataxia in ACAs were not replicated in a 1988 study conducted by Wilson and Nagoshi.

Other researchers have explored biological and neuropsychological differences in ACAs by looking at variables which are known to be genetically transmitted and are also distinctive for alcoholics, such as certain electroencephalographic (EEG) patterns. Jones and Holmes (1976) found that awake EEG patterns of detoxified alcoholics, taken when they were sitting at rest, contained excess fast EEG activity and deficient alpha, theta and delta activity. As this EEG pattern of fast activity is genetically transmitted (Propping, 1977), the researchers next investigated whether this EEG pattern was antecedent to alcoholic drinking behavior and, if so, if it would be found in the children of alcoholic fathers before drinking behavior began. Gabrielli, Mednick, Volavka, Pollock,

Schulsinger, and Itil (1982) administered several psychological, neurological, and medical tests, including an EEG, to 265 Danish children, aged 11 to 13, of whom 27 were the male children of alcoholic fathers. Gabrielli et al. hypothesized that the fathers' alcoholism would be associated with high frequency EEG activity (above 18 Hz) in the children, similar to the pattern found in the alcoholic fathers. This was confirmed in the male offspring: fast EEG activity, which is frequently found in alcoholics and which is heritable, was also found to be a characteristic of their male children.

Begleiter, Porjesz, and Kissin (1982) found neuroanatomical differences between ACA and nonACA male alcoholics using cortical event-related potential (ERP) techniques and computerized tomography (CT). The ERP waveform, with a duration of approximately one-half second, is an electrical response in the brain to a brief sensory stimulus and is derived from the EEG by signal-averaging techniques. The early components (<100 ms) of the wave appear to vary with change in "objective" stimulus characteristics; the later components (100-500 ms) appear to vary with change in "subjective" evaluation (Pihl, Peterson, & Finn, 1990). Begleiter et al. found neurophysiological deficits in the ACA group, implicating brain stem, limbic and cortical structures as well as widened sulci and enlarged ventricles. The ACAs were characterized as having a decrease or delay of various components of the ERP response to stimulus presentations where one must voluntarily allocate attentional resources (Pihl, Peterson, & Finn, 1990). It was suggested that this means ACAs may have difficulty with voluntary modulation or control of the orienting response, which involves the inhibition of ongoing behavior, and redirection of attention.

A series of studies followed these initial finding, searching for additional possible EEG markers associated with the genetic transmission of a predisposition to alcohol dependence. One part of the ERP is a positive wave observed between 300 and 600 ms after an anticipated but rare event (target stimulus), the P300. The latency of the P300 wave to reach peak altitude correlates with an individual's ability to respond selectively to the anticipated stimulus. The amplitude to target stimuli are decreased or absent in alcoholics (Porjesz & Begleiter, 1981) and has been found to be attenuated in the preadolescent sons of alcoholics (Begleiter, Porjesz, Bihari, & Kissin, 1984) and adult ACAs (Begleiter & Porjesz, 1988). These findings suggest that P300 deficits may predate the onset of alcoholism and may serve as a marker for the inherited risk of alcoholism.

Steinhauer and Hill (1993) tested two groups of children between the ages of 8 and 18 using an EEG to determine if the P300 could serve as a marker for the risk of developing alcoholism. The high-risk (ACA) group of 51 children had an average 4.1 first- and second- degree alcoholic relatives; the low-risk group of 42 children had no known relatives with a history of alcoholism or any other psychiatric diagnosis. Each child performed two tasks during which auditory ERPs were recorded. The experiments consisted of a simple counting task followed by a choice reaction time task of identifying high-pitched or low-pitched tones. The results indicated significantly lower P300 amplitude for male high-risk children, as compared to male low-risk children. No significant differences were observed among females. This lower P300 wave amplitude for male high-risk children showed greater reduction with older subjects, indicating it may be related to developmental processes in high-risk children.

Ramsey and Finn (1997) explored the influence of incentives on the amplitude of P300 in high- and low-risk men in their early 20s. The men with positive family histories of alcoholism displayed the expected attenuation of P300 amplitude, as had been found in earlier research studies. There was no significant change in the amplitude due to the incentive condition for the high-risk group. The men with no family history, as expected, displayed a significantly increased P300 amplitude in response to an incentive. The lack of change in P300 amplitude of ACAs in response to an incentive suggests a deficit in the motivational system of ACAs, which correlates with research done with men who have a history of antisocial behavior (Forth & Hare, 1989). Most recently, Van Der Stelt, Geesken, Gunning, Snel, and Kok (1998) have confirmed smaller amplitude P300 waves in ACAs, this time using a visual paradigm for the target stimulus.

A 1998 study from Holguin, Corral, and Cadaveira provided results which offered a more complicated picture. They studied boys and girls from families with alcoholic fathers with no second-degree alcoholic relatives, families with alcoholics fathers with additional alcoholic relatives, and a control group with no alcoholic relatives. They presented both visual and auditory discrimination tasks with three different stimuli. The expected low P300 amplitude with boys from families with greater alcoholism did not consistently reach significance when compared to boys from the other two groups. There was overall no main effect for gender, though when females from high-density alcoholic families were compared to the other two groups of females, the alcoholic family group of females displayed a lower P300 amplitude.

There have been two longitudinal studies of children and P300 testing. Berman, Whipple, Fitch, and Noble (1993) followed a group of boys four years after the initial testing and found that those children with the lowest P300 amplitude at baseline were more likely to be involved with substances. Hill, Steinhauer, Lowers, and Locke (1995) followed 20 children, testing them eight years after the initial testing. They found that those with the lowest P300 amplitudes at the initial testing were those most likely to be involved in substance abuse at the time of the second testing.

Whipple, Parker, and Noble (1988) combined ERP evaluation and a battery of cognitive tests. They tested 15 detoxified and recovering alcoholic fathers (A+) who had a strong family history of alcoholism and their sons (mean age 10.1 years); 15 nonalcoholic fathers (NA+) with a family history of alcoholism and their sons (mean age 10.5 years); and 15 nonalcoholic fathers (NA-) with no family history of alcoholism and their sons (mean age 10.2 years). On comparison of mean amplitude on the EEG, the A+ fathers had the lowest mean amplitudes, significantly below the NA- fathers, with the NA+ fathers falling between the two groups. The A+ sons had amplitudes significantly below the NA+ and NA- groups, which were virtually identical. The A+ and NA+ fathers had significantly lower Full Scale IQ scores reflecting reduced performance on the visuoperceptual and memory subtests of the WAIS. This result was replicated in their sons. Other researchers have examined the cognitive abilities of ACAs, to explore further whether some deficits in cognitive functioning routinely found in alcoholics might be antecedent to the development of alcoholic behavior, rather than be solely a consequence of alcohol abuse. Gabrielli and Mednick (1983) compared the scores of Danish children

who were at high risk for developing alcoholism, due to a family history of alcoholism, to a control group, using the Wechsler Intelligence Scale for Children. They found that the high-risk group had significantly lower scores on Similarities and Vocabulary subtests, and they produced lower Verbal IQ and Full Scale IQ scores.

Schaeffer, Parsons, and Yohman (1984) tested four different groups of adult males: 41 detoxified alcoholic ACAs, 27 detoxified alcoholic nonACAs, 19 nonalcoholic ACAs, and 43 men who were nonalcoholic and nonACA. The groups were administered several tests for vocabulary and verbal ability, learning and memory, problem solving and conceptualization. As might be expected, the alcoholics differed significantly from the nonalcoholic nonACA group in the abstracting/problem solving and learning/memory dimensions. They also found that the nonalcoholic ACA males performed significantly worse than the nonalcoholic nonACA males on the abstracting/problem solving and perceptual-motor clusters. The primary differences between the two nonalcoholic groups were on the tests that required higher or more complex cognitive functioning. For example, the groups varied little on vocabulary, sentence writing, pegboard or digit span, but the ACA group performed more poorly on such tests as the Wechsler Memory Scale Semantic Memory test (Wechsler, 1945), the Booklet Category Test (McCampbell & Defilippis, 1979), the Trail Making Test B, and the Conceptual Level Analogy Test (Willner, 1970).

Tartar, Hegedus, Goldstein, Shelly, and Alterman (1984) tested 41 delinquent adolescents, 16 of whom had an alcoholic biological father and 25 of whom had fathers free of alcoholism. Adolescents with paternal alcoholism performed more poorly than

those with no such paternal alcoholism on tests measuring attention, memory, perceptual-motor coordination, motor speed, spatial sequencing, reading comprehension, and language capacity.

Tartar, Hegedus, Winsten and Alterman (1984) tested 16 ACA male delinquents and 25 nonACA male delinquents and found the ACA group scored higher on the MMPI on the subscales of hysteria, hypochondriasis, and depression, though not in the pathological ranges, while the nonACA group proved to be less impulsive on the Matching Familiar Figures Test (Kagan, Rosman, Day, Albert, & Phillips, 1964). They also tested cognitive capacities and found that, although general intellectual capacity between the groups (Verbal IQ, Performance IQ, and Full Scale IQ on the Wechsler Intelligence Scale for Children-Revi.. ~d; Wechsler, 1974) was not significantly different, the ACAs performed more poorly on tests measuring attention, memory, perceptual-motor coordination, motor speed, spatial sequencing, language capacity, and a test of reading comprehension.

Drejer, Theilgaard, Teasdale, Schulsinger, and Goodwin (1985) examined 134 Danish male ACAs and 70 male control subjects with an average age of 19 years old, on tests of handedness, general intelligence, memory, attention, field dependence, categorizing ability, and organizing and planning. They found the ACA group performed more poorly on the Vocabulary subtest of the Wechsler Adult Intelligence Scale, the Halstead Category test, and the Porteus Maze test, but not on tests of memory, attention, or field dependence. The authors asserted that their findings confirm other research which shows ACAs to be deficient in verbal tests (Gabrielli & Mednick, 1983) and tests which

measure impulsivity (Knop, Teasdale, Schulsinger & Goodwin, 1985). Wilson and

Nagoshi (1988) tested 53 subjects reporting an alcoholic parent and 191 control subjects

as part of the Colorado Alcohol Research on Twins and Adoptees program, using

numerous physiological, neuropsychological and cognitive tests. They found that ACAs

who displayed cognitive deficits impairing their problem-solving abilities generally also

had significantly fewer years of education and generally scored lower on vocabulary tests.

A 1989 study conducted by Tartar, Jacob and Bremer tested 16 sons of alcoholics

who began to drink before the age of 24 (early onset), 17 sons of alcoholics who began

drinking after the age of 24 (late onset), 30 boys with no family history of alcoholism, and

29 sons of depressed men with no drinking history. All boys were between the ages of 8

and 17. The sons of early-onset alcoholics obtained lower performance IQ scores and Full

Scale IQ scores than the subjects in the other three groups. They also performed more

poorly on tests of auditory and verbal attention and cognitive inhibtion. Sher, Walitzer,

Wood, and Brent (1991) compared 253 ACA children and 237 nonACA children who

were incoming college freshmen on alcohol and drug use, psychopathology, cognitive

ability, and personality. While the ACAs reported more alcohol and drug problems and

greater behavioral undercontrol and neuroticism, they also displayed lower academic

achievement and less verbal ability. On the Wechsler Adult Intelligence Scale-Revised

(WAIS-R; Wechsler, 1981), ACAs scored significantly lower on the Block Design,

Similarities, and Vocabulary subtests.

Harden and Pihl (1995) pursued the neuropsychological hypothesis that high-risk

ACA status is associated with deficits in the executive functions associated with the

frontal lobe. They proposed a study of the hypothesis that the frontal lobes are implicated in the highest levels of hierarchical cognitive functioning, strategic planning and problem solving. They tested 14 ACA boys and 14 nonACA boys whose mean age was 12.1 years. The boys in the ACA group were of multigenerational alcoholic families, with at least a father, paternal grandfather, and one other male relative alcoholic. The boys were matched for age, grade level and IQ scores. Results from the analysis of the cognitive test battery indicated that the high-risk boys performed significantly more poorly on tests of frontal lobe functioning, such as the Wisconsin Card Sorting Test, a test of word fluency, and the Paired Associates Recall test (Wechsler, 1981). The ACA group also made more errors and scored as more impulsive in responding on the Matching Familiar Figures Test (Kagan et al, 1964).

There have been studies with ACAs which did not indicate the neuropsychological and cognitive deficits previously delineated. Hesselbrock, Stabenau, and Hesselbrock (1985) divided young alcoholics into three groups based on parental alcoholism: two alcoholic parents, one alcoholic parent, and no alcoholic parents. They found no systematic differences on the Halstead Reitan Trail Making Test, Category Test, or Tactual Performance Test (TPT) between the groups. Reed, Grant and Adams (1987) divided 84 male adult alcoholics into four groups: parent plus additional relatives who were alcoholic, parent only alcoholic, relative only alcoholic, and no relatives alcoholic. Subjects were administered the WAIS Vocabulary and Digit Symbol subtests, the Halstead Reitan Trail Making Test, Category Test, TPT, and two of the Wechsler Memory Scale (WMS) subtests. Statistical analysis revealed no systematic between-group

differences in neuropsychological performance. This replicated findings on these particular instruments obtained by Schuckit (1985) and Hesselbrock et al. (1985).

Schuckit, Butters, Lyn, and Irwin (1987) compared a group of male ACAs to a control group using the Category Test of the Halstead-Reitan Battery and found no differences between the groups. The authors did speculate, however, if a failure to find group differences might be due to subject selection, in that they used upper-level college students who, by virtue of their having succeeded in college for a minimum of two years, might not be representative of all male ACAs. Workman-Daniels and Hesselbrock (1987) administered a similar battery to one group of six men and 15 women (mean age 24.6) who had one alcoholic parent, another group of 13 men and eight women with no alcoholic relatives (mean age 25.3), and one group of detoxified alcoholics, 10 men and 11 women (mean age 27.5), parent status unknown. In addition to the WAIS subtests, WMS Visual Reproduction and Paired Associate Learning subtests, and Halsted-Reitan tests, they also administered the Benton Visual Retention Test. The only significant differences between the groups were lower WAIS Full Scale IQ, Verbal IQ, and Performance IQ scores in the group of detoxified alcoholics as compared to both of the other groups.

Studies conducted by Alterman, Searles, and Hall (1989) and Alterman and Hall (1989) also failed to find substantive differences between groups of college men divided into groups according to whether they had an alcoholic father, an alcoholic second-degree relative, or no alcoholic relatives. These studies did not find differences in drinking behavior or consequences of drinking behavior between the groups.

Bates and Pandina (1992) reported on a very large and ambitious project which spanned several years. A pool of 659 young men and women, aged 18 to 24, were separated into five groups: no family history of alcoholism, alcoholic father and at least one alcoholic grandparent, alcoholic father only, alcoholic mother only, and one or more alcoholic grandparent(s) only. They were administered the WAIS Digit Span, Block Design, and Digit Symbol subtests, the Halstead Reitan Category and Trail Making tests, a spatial relations test, a vocabulary test, and an abstraction test. When groups with any family history of alcohol use were combined, no significant differences were found between the groups in vocabulary, verbal and nonverbal abstraction, or the majority of visuospatial skills. Overall, they found no evidence to link cognitive vulnerability with a positive family history of alcoholism, even in the area of visuospatial skills.

Other researchers have found deficits in visuospatial skills in ACAs to be a consistent finding. Schandler, Brannock, Cohen, Antick, and Caine (1988) examined visuospatial processing in young children with a family history (FH+) of alcoholism (n = 18) comparing them to a group with no family history (FH-) of alcoholism (n = 18). The children, ranging in age from six to eleven years old, were administered a visuospatial paired-associate learning task requiring the learning of five nonsense shapes in one of five distinct positions on a grid consisting of five lines radiating from center at equal 72° angles. A paired associate paradigm was used, in which each shape served as the stimulus and its grid position served as the response associate. During each trial the nonsense shape was first presented in the center of the grid for a 3-second period. The shape was then removed and there was a 5-second presentation of the grid only. This was followed

by a 3- second presentation of the shape in its associated grid position. The child was then required to indicate the grid position associated with each shape during the 5-second presentation of the grid only. The response was a button press in one of five corresponding positions on the identical grid display panel by the child's dominant hand. The stimulus presentations were organized by trial blocks, with each block containing all five shapes presented in random order. Learning was defined as one correct series of responses during one block trial. The FH+ children displayed significantly poorer performance than the FH- children. The FH+ children required significantly more trials to achieve learning, emitted fewer correct responses and committed more errors.

The authors hypothesized that their findings were suggestive of visuospatial difficulties being an etiological factor in developing alcoholism because they are related to the deficits of attention and information processing found in other studies of ACA groups. To further study this hypothesis, they applied the same learning paradigm to a group of adults (mean age 31) of whom 17 were FH+ (nine men and eight women) and 17 were FH- (ten men and seven women) (Schandler, Cohen, McArthur, Antick, & Brannock, 1991). Although all those participating achieved learning criterion, the visuospatial learning performance of the FH+ group was inferior to the FH- group. The FH+ group required significantly more trials to achieve learning criterion, required more time to produce a correct response, and emitted relatively fewer correct responses across learning trials.

Garland, Parsons, and Nixon (1993) also found that FH+ individuals displayed impaired visuospatial learning when compared to a group of FH- subjects. The

participants were 16 men and 16 women with FH+ and a control group of 16 men and 16

women who were FH-. Using the Schandler et al. (1988) visuospatial learning paradigm,

they found the visuospatial learning performance of the FH+ males to be inferior to the

FH- males; no significant learning deficit was observed for the FH+ female participants

although the trends were similar to the male groups. The authors suggested these results

might be due to heritable factors of neuropsychological dysfunction or other variables,

such as attention problems or affective disturbance, any or all of which might be an

etiological factor in alcoholism. These findings have been replicated and expanded upon

in other studies of visuospatial deficits in ACAs (Schandler, Brannock, Cohen, & Mendez,

1993; Schandler, Cohen & Antick, 1992).

As has been reviewed, numerous studies have demonstrated that ACAs are at

heightened genetic risk for the development of alcoholism and an accompanying spectrum

of psychosocial and neuropsychological problems. While some research has not supported

these claims, the preponderance of the literature suggests that ACAs exhibit specific

neuroanatomical differences, demonstrate certain cognitive deficits, face a four- to seven-

fold greater chance of being alcoholic themselves, and are at heightened risk for the

development of problematic psychological and personality traits. The precise nature of

this increased risk is still unknown, but a number of markers implicate specific functions of

the frontal cortex. Peterson, Finn, and Pihl (1992) noted that ACAs perform more poorly

than controls on cognitive tests of classification and planning generally associated with

prefrontal function. They noted from Granit (1977) that the prefrontal cortex provides the

physiological substrate for the cognitive functions associated with abstract classification

and planning, which have been shown in several studies of ACAs to be deficit in that group. The aforementioned P300 wave, which is generated in response to attention being given to a novel stimuli, is generated in the frontal cortex (Begleiter & Porjesz, 1988). Also, more recent research regarding inhibitory processes, which will be reviewed, has also implicated the prefrontal cortex in the inhibition or modulation of the function of various subcortical structures, including those governing threat or novelty response.

Numerous studies of ACA children, particularly male, have strongly associated ACA status, hyperactivity, attention deficits, conduct disorder, and a heightened risk for substance abuse (Pihl, Peterson, & Finn, 1990). These are linked to the processes of behavioral inhibition, which is regulated by the executive functioning system of the frontal cortex (Barkley, Grodzinsky, & DuPaul, 1992). As children develop, fine motor coordination, the allocation of attentional resources, and the planning and execution of goal-directed behavior is shaped and organized in the substrates of the frontal cortex. In evaluating psychopathology in children, adolescents, and adults, there is often an overlap or comorbidity of the symptoms associated with attention deficits, hyperactivity, conduct disorder, substance abuse and a host of related cognitive and neuropsychological deficits. The frontal lobes are involved in central executive functions, such as planning and monitoring of behavior, with the pattern of connections between sensory and motor areas meeting in the frontal lobes (Harnishfeger & Bjorklund, 1994). Dempster hypothesized that there is extensive neuropsychological evidence that dysfunction of the frontal lobes leads to inhibitory deficits in behavior, cognition, emotion, and personality (as cited in Harnishfeger & Bjorklund, 1994).

Longitudinal studies of childhood and adolescent precursors of adult alcohol abuse consistently identify a cluster of behavioral traits (e.g., disinhibited, undercontrolled, or impulsive) that can significantly predict high levels of adult alcohol abuse (Cloninger, Sigvardsson, & Bohman, 1988; Hechtman, Weissman, & Perlman, 1984). Rydelius (1983) suggested a relationship between ACA status, impulsive behavior patterns, and early onset of alcohol problems. Cloninger et al. (1981, 1988) proposed a "disinhibited novelty-seeking" temperament that is heritable and predictive of future substance abuse. Cloninger developed a scale for the measurement of personality dimensions labeled "novelty-seeking," "harm avoidant," and "reward dependent." The Tridimensional Personality Questionnaire (TPQ; Cloninger, 1987) delineates traits of impulsivity, reflection, excitability, and stoic rigidity. Gorenstein and Newman (1980) proposed that impulsive behavior patterns, antisocial personality, and early-onset alcoholism could be viewed as variable expressions of a general disinhibitory psychopathology. Inhibition is central to the control of social behavior. Behavioral inhibition allows individuals to control strong emotional responses, i.e., aggressive, sexual, and appetitive, and to delay gratification (Bjorklund & Harnishfeger, 1995).

Gray (as cited in Finn, Kessler, & Hussong, 1994) proposed a theory that deficits in behavioral inhibition are associated with decrements in the ability to form conditioned responses to stimuli that signal punishment, rather than insensitivity to the punishment itself. Past research has suggested that disinhibited personality traits are associated with a relative insensitivity to cues for punishment, such as a failure to inhibit behavior even in the presence of cues for punishment (Newman, 1987) or to learn from the consequences

of antisocial behavior (Virkkunen & Linnoila, 1993). Finn, Kessler, and Hussong (1994) tested 16 ACA males and 16 nonACA males using an aversive stimuli model that investigated the correlation between ACA status and behavioral inhibition. The basic hypothesis of the study was that the ACA group would condition more poorly than the nonACA group to signals for punishment (electric shock) as this group would theoretically be more likely to engage in behavior that has a higher probability for negative consequences. The results indicated a relationship between ACA status and electrodermal underresponsivity to stimuli signaling an aversive event. The ACA participants showed a consistent pattern of smaller responses to the conditioned stimuli signaling electric shock and poor differential responsivity to signals for shock or no shock conditions. The authors suggested that a weak behavior inhibition system is the mechanism mediating the interrelationship between a family history of alcoholism, impulsive behavior, and later alcohol problems.

Whether at the basic neuronal level or as expressed in complex cognitive and behavioral systems, human activity basically relies on processes that are excitatory or inhibitory. Excitatory processes increase the likelihood that messages will be sent and acted upon. Inhibitory processes decrease the likelihood that initial stimuli will result in a response. Neuropsychological research has identified the associative cortex of the frontal lobes, the prefrontal cortex, as the primary locus of behavioral inhibition (Bjorklund & Harnishfeger, 1995). Research with brain-damaged adults and brain-lesioned animals have supported theories that the ability to inhibit and control behavior has its locus in this area of the brain. Animals with frontal lesions show emotional and behavioral deficits, a

general hyperactivity of movement, particularly to novel stimuli, an inability to inhibit attention to irrelevant stimuli, and a general disinhibition of behavior (Bjorklund & Harnishfeger, 1995).

Luria (as cited in Harnishfeger, 1995) identified various forms of impairment in inhibition resulting from frontal lobe dysfunction, including the inability to plan, direct, and monitor cognitive processing, the inability to stop an ongoing repetitive behavior, and the inability to commence a new pattern of behavior that is different from overlearned stereotypic responses. Luria also identified an attentional inhibition, with patients with frontal lobe damage being unable to control orientation to irrelevant stimuli while being unable to orient correctly to relevant stimuli. Deficits in inhibition result in the activation and processing of irrelevant information during cognitive processing (Hasher & Zacks, 1988). Inhibition is a developmental challenge which results in an active suppression process, removing task-irrelevant information from working memory. Selective attention to relevant stimuli requires efficient inhibition of wandering attention (Bjorklund & Harnishfeger, 1995). The cognitive ability to control attention processes is an early step in the development of self-control with later developments in symbolic thought and flexible planning dependent on successfully meeting the earlier challenge.

Ecological psychologists view attention as a naturally evolved and essential survival mechanism because each organism is surrounded and bombarded with sensory information, some of which may be necessary for survival. For action to occur which is useful and essential, selective attention must choose what is relevant from the available information, while disregarding the rest (Enns & Burack, 1997). Some mechanism, or

system of mechanisms, is required to ensure that the brain's attention system selects what is important in the environment to which to attend. Several theories have tried to explain what that system of attention is and how it operates. Cherry (1953) was an early researcher into the phenomenon of attention, noting that people have to ability to attend to only one voice or conversation in the middle of a room filled with talking people. He further explored this using a dichotic listening technique in which subjects used headphones with one voice talking in one ear and a different message being spoken into the other ear. He found that subjects could effectively attend to (shadow) the message in one ear but were unable to remember what was said into the other ear. Listeners would remember certain details, such as a change in gender or speed of speaking in the unattended ear, but none of the semantic content could be remembered.

One of the earliest and best known theories of attention was Broadbent's (1958) filter or early selection theory. This theory supposes a three-stage process of selective attention. All stimuli is impinged on the sensory register, a large capacity but very short-term staging area. A selective filter identifies that which will be further processed, depending on features such as pitch or intensity, with other sensory data being discarded. The stimuli is shunted along a limited- capacity channel to the detection device where decisions are made to process further the stimuli into short term memory. There are several incoming channels and the selective filter switches rapidly from channel to channel, one channel at a time. Because this is a serial process and must, by its nature, be a laborious process, this early selection theory is also nicknamed the bottleneck theory. Broadbent theorized that meaning is only attached after the information passes the

detection device, so we only know or become aware of information which has been passed through the selective filter.

Moray (1959) found that subjects in a dichotic listening task would recognize their names being spoken in the unshadowed ear. According to Broadbent's early selection theory, this should not have happened. If analysis of meaning is carried out in the detection device, their names should have been filtered out at the selective filter. Deutsch and Deutsch (1963) proposed a late-selection theory in which all incoming sensory data is sent on for further processing. Selection for attention takes place at the level of the working memory. Subjects then should recognize information under almost any circumstances, even when presented in the unshadowed ear. Lewis (1970) was one of the first researchers to test this theory by presenting two lists of words, one in the shadowed ear and one in the unshadowed ear. He found that when the synonym of a word in the shadowed ear was presented in the unshadowed ear, there was a latency in the subject's speaking the shadowed word. He suggested that the subjects had recognized a semantic relationship between the words, causing the latency, supporting a late-selection theory.

Triesman (1960) found that when she switched the meaningful message from the attended ear to the unattended ear, the subjects would change their attention to the previously unattended ear to shadow the meaningful message. She developed a much more complex attenuation model of attention involving three different levels of processing. At the first level, the physical properties of the stimuli (i.e., loudness, intensity, brightness) are analyzed. The second level determines whether the stimuli are linguistic and, if so, groups the stimuli into understandable blocks, i.e., phonemes, syllables, words. At the

third level of processing, meaning is assigned. Attenuation theory also holds that incoming information not immediately processed is not discarded, but rather is attenuated, awaiting additional information before a decision is made to discard it or combine or associate it with new incoming information. Rejected stimuli are only filtered out partially, rather than completely. Recognition of attenuated material takes place through accumulation of information or activation in detector units (Pashler, 1998). Unattended attenuated material would not produce enough activity to cause the corresponding detector to reach a threshold for processing. However, when the detector represents a concept that is somehow related to concepts already activated, a process known as semantic priming (Meyer & Schvaneveldt, 1971), partial activation will trigger recognition.

Posner (1980) suggested three different attentional systems: arousal, limited-capacity attention, and selectivity. Arousal refers to the excitation experienced by the organism, including alertness and cognitive readiness, in response to sensory stimulation. Limited-capacity attention is based on the premise that each organism has a limit to its cognitive capacity. For example, if an individual is given two different tasks to accomplish which used the same resources, such as dichotic listening or accessing long term memory, these are done serially even if rapidly. One task will be given precedence and the other will be accomplished secondarily. Selective attention refers to the specificity with which attentional resources are allocated to task demands.

Inhibition was not involved in these early theories of selective attention nor in most theories of cognitive development. The developmental psychology of attention processes

was dominated by the aforementioned information-processing models, as well as other theories, in which inhibitory processes were not treated as particularly useful (Harnishfeger, 1995). In the past decade, several researchers have begun exploring models that employ inhibitory mechanisms as an essential ingredient in several domains of social, motoric and cognitive development. Luria (1961) and Saltz, Campbell, and Skotko (1983) demonstrated that inhibitory control, the ability to stop, slow, or pause responding, is a separate developmental challenge from excitatory mechanisms, or the ability to initiate responding, by observing the behavior of very young children. Infants are unable to guide their own behavior through external or internal speech. They are unable to use verbal commands to stop (inhibit) their ongoing behaviors. Later, toddlers become capable of using the external verbal commands of others to direct their behavior, but are not yet able to use personal, internalized, verbal instructions to regulate their own behavior. Development of the verbal control of behavior happens slowly over time and occurs from the outside in, with external verbal control being achieved before internal verbal control (Harnishfeger, 1995) and is distinct from excitatory mechanisms, or the ability to initiate responding.

Cognitive inhibition is an active process, involving the suppression of previously activated cognitive processes, the clearing or removal of task-irrelevant actions or attention from consciousness, and resistance to interference from potentially attention-capturing processes (Bjorklund & Harnishfeger, 1995). It differs from behavioral inhibition in that the latter involves the control of overt behavior, such as resisting temptation, motor inhibition, delay of gratification, and impulse control (Harnishfeger,

1995). Cognitive inhibition involves the control of cognitive contents or processes, i.e., thought suppression or the intentional control of the contents of consciousness, the clearing of incorrect inferences from memory, the suppression of context-inappropriate meanings of words with multiple meanings, and the gating of irrelevant information from working memory during memory processing (Harnisfeger, 1995).

Both behavioral and cognitive inhibition become more efficient as children develop. Infants will try to reach a toy placed behind a box by reaching through the box; by two years of age the children are able to inhibit that response and display the more flexible response of reaching around the box (Diamond, 1988). In another set of experiments with very young children, Diamond repeatedly placed toys out of sight in a covered well (well A) and the infant was allowed to retrieve the toy several times (as cited in Harnishfeger, 1995). Next the toy was hidden in full view of the infant in a different well (well B). Infants younger than one year usually continued to reach to well A. Diamond argued that the perseverative error is due, in part, to inhibitory inefficiency. Healthy infants were able to develop that efficiency by the end of the first year. This developmental challenge is important because later success in school is dependent on the acquisition of increasingly better skills to inhibit irrelevant stimuli, focus attention and control the processes of working memory.

Although they are manifested differently and it is useful to distinguish between behavioral and cognitive inhibition, they are clearly related (Harnishfeger, 1995). One aspect of this relationship is the use of cognitive inhibition to facilitate behavioral inhibition. Mischel, Shoda, and Rodriguez (1989) tested children's ability to delay

gratification by setting each of them in front of a plate of treats, explaining that if they could sit there without touching the treats, they would receive a greater reward later. This test of ability to inhibit impulsive responding was facilitated by the cognitive process of thought suppression. This cognitive process refers to the attempt to keep unwanted thoughts out of conscious awareness. The children reported that they accomplished this by thinking distracting and fun thoughts that were unrelated to the reward or by talking or singing to themselves. Olson (1989) reviewed research literature of cognitive and behavioral inhibition using factor analysis and found that various measures of inhibitory control cohere into three higher order factors: ability to delay gratification, motor inhibition, and cognitive inhibition. Olson found a significant correlation between motor inhibition and delay of gratification, while the correlations were not significant between behavioral inhibition factors and measures of cognitive inhibition. Nevertheless, the overlap of behavioral and cognitive factors is demonstrated in several areas, including attention deficit/hyperactivity disorder, conduct disorder, obsessive-compulsive disorder, and schizophrenia (Harnishfeger & Bjorklund, 1994).

Inhibition has been presented as an active cognitive process. It is the stop signal for individual functioning. It allows the individual to stop a thought or a behavior. It allows the spotlight of attention to be refocused, clearing away what was previously attended to and allowing the process of attention to continue. While often the result or consequences are overt and observable, inhibition begins with a cognitive process. Research has provided evidence that alcoholics have an impaired ability to inhibit their behavior. Other research has clearly suggested that many of the cognitive deficits

experienced by alcoholics may be antecedent to the drinking behavior and have been identified in their young or nondrinking offspring. As inhibition is an active cognitive process, this study proposed to investigate inhibitory processes in ACAs.

Impulsivity has been defined in a variety of ways and encompasses several cognitive and behavioral aspects which may or may not correlate with each other. Dictionary definitions of impulsivity include the idea of an act moving onward with sudden force, a wave of excitation transmitted through bodily tissues and nerve fibers that results in physiological activity or inhibition, a sudden spontaneous inclination incitement to some unpremeditated action, and/or a propensity or natural tendency usually other than rational (Gove, 1965). Dickman (1990) identified two distinct subtypes of impulsivity: functional and dysfunctional. Functional impulsivity describes the use of action in a very quick fashion, but just at the right time, which results in a positive outcome. Functional impulsivity represents the tendency to engage in rapid, error-prone information processing (i.e., to act with little forethought) when such a strategy is optimal. Stock brokers who work on the exchange floor are likely successful because of the exercise of functional impulsivity. Dysfunctional impulsivity consists of similar tendencies toward thoughtless, spontaneous action; however, in this case, the consequences are negative. Dysfunctional impulsivity represents the tendency to engage in rapid, error-prone information processing because of an inability to use slower, more methodical approaches. Dickman suggests that these are separate processes and that dysfunctional impulsivity reflects a breakdown in the control of information processing due to stress. Individuals who demonstrate higher levels

of impulsivity may experience difficulties in inhibiting the more impulsive, error-prone reactions in favor of more methodical and careful strategies.

The focus of attention by both psychologists and sociologists has been upon the dysfunctional aspect of impulsivity because of its deleterious impact on human behavior. In recent years, the domains of arousal, attention, and impulsivity have become areas of intense research scrutiny. Schachar, Tannock, and Logan (1993) include in their definition of impulsivity: (1) the tendency to execute actions too quickly or in an unreasoned or unreflective manner; (2) difficulties in withholding actions or difficulties in inhibiting actions once they have been commenced; and (3) the tendency to seek out immediate gratification at the expense of longer-term goals. In their research on children with Attention-Deficit/Hyperactivity Disorder (ADHD), the authors suggested that there is a link between the cognitive construct of inhibition and impulsivity as a behavioral construct. Deficient inhibitory control results in a greater likelihood that a response will escape executive control and a behavior will be executed. How much deficits or impairments in inhibitory control are related to overt impulsive behavior is still a research question which needs to be further addressed. Certainly there is a face valid and intuitive assumption that can be drawn about the relationship between deficits in cognitive inhibitory control mechanisms and impulsive responding. For that reason, this study investigated not only the potential for differences in inhibition between ACA individuals and control individuals, but also investigated factors of impulsivity.

Further investigation of the cognitive ability to inhibit or suppress responding and impulsivity was conducted using a stop-signal paradigm (Logan, 1994). In the stop-signal

paradigm, participants are engaged in a computer-administered forced-choice reaction time task. Occasionally, and unpredictably, participants are presented with a stop signal (a tone generated by the computer) that instructs them to withhold their motor response to the primary task. Stopping, or activating inhibition in thought or action, is an extreme form of control. Stopping is a clear case of executive function and example of cognitive control over thinking and behaving. Schachar and Logan (1990a) found that hyperactive children had trouble stopping on stop-signal trials. Not only were the groups of hyperactive children slower to inhibit their behavior than the control group of children, they were less likely to inhibit their behavior altogether, responding more often than normals on stop-signal trials. To discover if it was due to not noticing the stop signal, Schachar and Logan (1990b), in a second research project, ran a dual-task experiment presenting the same stimuli they used in their stop-signal experiment but requiring the children to make an overt response to the stop signal, as well as an overt response to the primary task. Hyperactive children detected the signal as often as normal controls, and showed the same refractory effect in their reaction time. The deficiency in stopping was then due to an inability to inhibit their behavior, not a deficiency in detection.

Logan (1994) suggested that behavioral inhibition is a separate process from excitation, and that the observed process likely reflects what occurs at the neuronal level. Neurologically, a tendency to decrease firing tempers a tendency to increase firing. A single, global mechanism may be responsible for ability to stop or inhibit performance. De Jong, Coles, Logan, and Gratton (1990) examined ERPs in the EEG while subjects processed go and stop signals. Their analysis suggested two mechanisms of inhibition: a

central one that operated selectively, inhibiting central preparation of the required response, and a peripheral one that operated nonselectively, inhibiting any and all responses (Logan, 1994). Stop-signal inhibition involves a whole process, the stopping process, working against the excitatory or arousal processes.

Impulsivity is thought to play a role in both attention/hyperactivity disorders in childhood and alcohol problems later in life (Pelham & Lang, 1993). The construct of high impulsivity is central to defining ADHD and is one of the core symptoms (American Psychiatric Association, 1994). Similarly, personality characteristics related to impulsivity and deficits in inhibitory control have long been studied by researchers in alcoholism, with many arguing that behavioral undercontrol or disinhibition plays a role in the development of the disorder (Cloninger, 1987; Sher, Walitzer, Wood, & Brent, 1991). Problems in impulse control are also thought to play a role in other highly comorbid disorders, such as conduct disorder in children (West & Prinz, 1987) and antisocial personality in adults (Tartar, 1988). Several studies have shown that children with externalizing problems, including impulse control, behavioral undercontrol, and deficits in inhibitory control are at increased risk for developing substance abuse problems as adolescents and adults (Hechtman, Weiss, & Perlman, 1984; Zucker & Gomberg, 1986). While this risk is likely heightened and exacerbated by the presence of the child in a family system which is rife with tension, stress and negative influences, it is also well-substantiated in the research literature that generational effects are embedded in a biopsychosocial paradigm which includes a genetic component. For that reason, this study proposed to investigate components of impulsivity and inhibitory control in ACAs.

Studies have used the Matching Familiar Figures Test (MFFT; Kagan, Rosman, Day, Albert, & Phillips, 1964) as an instrument to investigate cognitive dimensions of reflectivity and impulse control (Parker & Bagby, 1997). The task requires subjects to search several similar pictures for one that matches a criterion picture exactly. The premise is that impulsive subjects will be unable to delay responding in the course of analyzing the stimuli and will make an impulsive initial selection. Subjects are rated according to the speed and accuracy of their responses. Several researchers have found that children who display impulsivity and hyperactivity have done more poorly on the MFFT (e.g., Biederman, Munir, & Knee, 1987; Campbell, Douglas, & Morgenstern, 1971; Rapoport, Quinn, Bradbard, Riddle, & Brooks, 1974) than control groups. Messer (1976) reviewed the research literature on the MFFT and found the measure to have adequate psychometric properties. Validity ratings have been mixed, however. The MFFT has been found to be associated with performance tests, such as the Porteus Maze Test (Gow & Ward, 1982) and the Draw-a-Line-Slowly test (Bentler & McClain, 1976), that assess impulsivity-related constructs but correlations with teacher and observer ratings have been low (Parker & Bagby, 1997). Other researchers have also noted that performance on the MFFT improves with the age of the test groups (Salkind & Wright, 1977); older subjects make fewer errors than younger subjects.

Another widely used test of attention and impulsivity in both children and adults is the Stroop test, first demonstrated by Stroop in 1935. In this test subjects are asked to name the ink color of a word and ignore the word semantically. Ink color naming is slower when the ink color and the word meaning are incongruent (e.g., the word "green"

printed in red ink) than when they are congruent (e.g., the word "green" printed in green ink), or when they are neutral (e.g., a string of letters or a noncolor word printed in green). In the Stroop effect, the irrelevant color word interferes with the cognitive processing of the ink color and responding is inhibited. Occasionally the word reading cannot be inhibited and the word is read rather than the ink color, in spite of the individual's attempt to suppress the word reading. Studies of individuals with disorders of attention, impulsivity and other psychopathology of areas of the frontal cortex have found that impaired groups have performed more poorly (e.g., Seidman, Biederman, Faraone, Milberger, Norman, Seiverd, Benedict, Guite, Mick, & Kiely, 1995). The Stroop is a useful overall measure of several processes that appear to be related to impulsivity, attention and concentration, ability to maintain a set, and inhibition of inappropriate responses (Zaparniuk & Taylor, 1997).

Seidman et al. (1995) tested 65 ADHD males, aged 9 to 20, and 45 normal controls using several tests of neuropsychological functioning, including the Stroop test. On the results of the Stroop test, they found ADHD subjects were significantly impaired on scores of color-word interference.

Priming refers to the triggering of specific memories by a particular cue, e.g., the recall of a fire engine or an apple can be primed by the word "red." Triesman (1960) had theorized in her attention attenuation theory that incoming sensory data can be attenuated, or temporarily set aside, until primed for attention by an accumulation of data or incorporation with previously acquired knowledge (Rafal & Henik, 1994). A large body of research has documented greater speed and accuracy of performance in responding to a

target word (e.g., doctor) when it follows a semantically-related prime word (e.g., nurse) than when it follows an unrelated prime word (e.g., dog). As with the Stroop effect, priming is demonstrated by the automatic accessing of the word meaning. Accessing the meaning of the written symbol "red" is automatic; it requires no intention, it happens whether one wants it to or not. A word automatically activates or primes its meaning in memory and, as a consequence, primes or activates meanings closely associated with it (Ashcraft, 1994). This priming makes related meanings easier to access: because of priming, associations are easier and quicker. The Stroop effect is one example of negative priming: its effect is to slow responses to a stimulus rather than facilitate it. Negative priming refers to an increase in reaction time to a target if the target was the distractor in the trial immediately preceding. For the Stroop paradigm, color naming is slower if the color corresponds to the preceding distractor word. If the internal depiction of an object which is to be ignored is associated with inhibition when a target object is being selected, the processing of a subsequent stimulus which uses the inhibited depiction should then be impaired. In a priming procedure, when the inhibited stimulus is presented as a probe for identification, reaction time to name the probe should then be increased. For example, participants might be presented with two overlapping drawings in which one object is a vase drawn in red ink and the other is a sled in blue ink (control display) and they are told to attend to the vase and ignore the sled. Then in an "ignored repetition" trial, they are to attend to the vase again, this time superimposed over a flower rather than the previous background distractor. When the flower is presented in the probe display, superimposed

over a neutral, meaningless distractor, reaction time is longer to identify the previously

ignored flower. Such a result is consistently observed in negative priming tasks.

Negative priming has been a test paradigm for cognitive inhibition using various

tasks and challenges, including picture naming (Tipper, 1985), letter naming (Tipper &

Cranston, 1985), letter matching (Neill, Lissner & Beck, 1990) lexical decision (Yee,

1991), and letter capitalization identification (Ferraro & Okerlund, 1996) as well as

variations on the Stroop effect. Diverse populations in which the inhibitory process which

allows priming is deficient or impaired have been administered negative priming tests.

These populations include schizophrenics (Laplante, Everett & Thomas, 1992), obsessive-

compulsives (Enright & Beech, 1990, 1993), and schizotypals (Ferraro & Okerlund,

1996) as well as impulsive children (Visser et al., 1996).

A popular measure of attention, vigilance, and impulsivity is the continuous

performance test (CPT). CPTs present a series of stimuli, e.g., letters, numbers, or

objects, that the examinee must monitor for the presence of predetermined targets. The

stimuli may be presented as visual or auditory. The examinee is instructed to press a key

on the computer keyboard when a specific target appears, e.g., the letter "X," and not to

press when other letters appear. In some conditions, the target appears with no warning.

Other conditions include cued conditions when a specific letter cues the examinee that the

target letter is to appear next, e.g., the letter "A" before the letter "X." Most CPTs yield

measures of missed targets (i.e., errors of omission), which are generally considered to

reflect inattention, and false alarms (i.e., errors of commission), which are generally

considered to reflect impulsivity. Reaction time and reaction time variability are also recorded (Matier-Sharma, Perachio, Newcorn, Sharma, & Halperin, 1995).

A variation on the CPT is a rapid visual performance test (Wesnes & Revell, 1984) during which numbers are presented at the rate of 100 per minute and subjects were instructed to press a response button as quikcly as possible when they detected sequences of three consecutive odd or three consecutive even digits. Three measures of performance were made during each ten minute presentation of the task: the probability of correctly detecting an experimental target (probability of a hit = total number of correctly detected targets/number of targets presented), the average time taken to respond to an experimental target, and the number of responses made in error. Wesnes and Revell (1984) used this test to determine the effects of administration of scopolamine and nicotine on efficiency in the performance of a rapid information processing task.

This study proposed to investigate inhibitory processes and impulsivity in male and female ACAs as compared to groups of males and female nonACAs. As previously reviewed, there is a compelling body of research indicating that ACAs, as a group, demonstrate a spectrum of specific cognitive and neuropsychological differences from comparative groups of nonACAs. Some of these differences displayed in alcoholics have been suggested to be antecedent to drinking behavior and to be genetically transmitted. As impairment in behavioral inhibition and problems with impulsivity are demonstrated by alcoholics and are also noted in children with disorders of inattention, hyperactivity, and impulsivity, disorders which have been shown to be comorbid with a heightened risk for alcoholism, might there also be a pattern of impairment in cognitive inhibition and

impulsivity in ACAs? This question was explored using measures of inhibition and impulsivity, including the MFFT, the Stroop test, a negative priming test, and a CPT. Personality measures of impulsivity, harm avoidance, and novelty seeking were also administered. It was hypothesized that the ACA groups would demonstrate poorer inhibitory control, greater impulsivity, longer reaction times to negative priming, and more errors of commission on a CPT. It was also expected that personality scales would find them to be more impulsive and novelty-seeking than comparative control groups.

CHAPTER II

METHOD

Participants

Participants were recruited from college students attending a Midwestern university who were taking introductory and developmental psychology courses and who received class credit or $25 for participation. Adult community members, aged 18 to 42, were also solicited with an offer of $25 for participation. Initial screening for placement in the ACA or nonACA groups was accomplished using an 11-question instrument developed by Petros and Weller (1998; see Appendix A), asking participants to indicate if either their mother or father has or has had a drinking problem, the extent of the problem, and whether any second-degree relatives have had a drinking problem. Participants who qualified for the ACA group had to have had one parent and at least one, and preferably two, second-degree relatives who were reported by the respondent to have (or have had) a drinking problem. If the drinking parent was the mother, the participant had to know that the mother was not drinking during her pregnancy, or the potential participant was unable to proceed with the study. This was required due to the potential confounding effects of fetal alcohol syndrome or effects. Participants who qualified for inclusion in the nonACA group must have had no reported drinking problems with either parent or with any second-degree relatives.

As to the importance of a multigenerational positive family history, several studies have hightlighted differences between groups of ACAs with either single or multigenerational pedigrees. For example, Finn, Earleywine, and Pihl (1992) examined the potential differences between three groups of men (mean age 23) using discriminant analysis: one group had a multigenerational family history of alcoholism ($\underline{n}$ = 40), one group had only an alcoholic father ($\underline{n}$ = 19), and one group had a negative family history ($\underline{n}$ = 36). The participants were measured on several neurobiological and personality factors. A discriminant function analyses generated a linear combination of psychophysiological and personality variables that significantly discriminated the extent of the participant's family history of alcoholism and correctly classified 62% of all the subjects into their family grouping. The analysis provided an indication of heterogeneity between the groups, clearly separating the multigenerational individuals from the other two groups.

Based on the screening responses, four pools of research participants were developed and contacted to participate further: one group of males with a family history positive for alcoholism (ACA) and a second group of males with the family history negative for alcoholism (nonACA); one group of females with a family history positive for alcoholism (ACA) and a second group of females with a family history negative for alcoholism (nonACA). Individuals were called at random from these pools to offer them the opportunity to participate in the research study. The study included 120 participants: 29 male ACAs, 30 male nonACAs, 31 female ACAs, and 30 female nonACAs. This

participant pool was selected based on research by Schuckit (1994), who recommended group size at 30 subjects in each group.

Materials

Each participant was administered the Vocabulary and Block Design subtests of the Wechsler Adult Intelligence Scale-Revised (WAIS-R; Wechsler, 1981). This two-subtest combination is popular as a short-form screening instrument for intellectual functioning, correlating well with the Full Scale IQ of the WAIS (Sattler, 1992). The Vocabulary subtest consists of 35 words arranged in order of increasing difficulty. Each word is presented orally and in writing, and the subject is required to give a definition orally to the examiner. Responses are scored 0, 1, or 2 depending on the quality of the response, with more accurate responses receiving higher scores. The test is discontinued after the participant commits three consecutive failures. The Block Design subtest utilizes four to nine two-dimensional, red-and-white blocks which are either all white on one side, all red on one side, or half red and white. The examinee is shown drawings of abstract designs and asked to replicate the pictured design, using the blocks. Scores are assigned based on the length of time to replicate the design and accuracy of the design reproduction. The subtest is discontinued after three consecutive failures.

Participants were administered two personality scales: the Extroversion/Introversion and Impulsivity/Sociability portions of the Eysenck Personality Questionnaire (EPQ; Eysenck, 1975), and the Tridimensional Personality Questionnaire (TPQ; Cloninger, 1987). The Eysenck is a 54-item self-report scale that assesses four dimensions: neurotic introversion, stable introversion, neurotic extroversion, and stable

extroversion. Extraversion has been found to be correlated with greater impulsivity and weaker inhibitory control (Dickman, 1990; Newman, Wallace, Schmitt, & Arnett, 1997). The TPQ is a 100-item self-report scale (true-false format) developed to assess three broad personality dimensions which Cloninger suggested could differentiate those at risk for alcoholism from those with less risk: novelty seeking, harm avoidance, and reward dependence (Cloninger, Sigvardsson, & Bohman, 1988). The Novelty Seeking scale purports to measure the tendency toward frequent exploratory activity and exhilaration in response to novel or appetitive stimuli. Individuals high in novelty seeking are said to be impulsive, exploratory, excitable, distractible, and easily provoked to prepare for fight or flight. The Harm Avoidance scale purports to measure the tendency to be uncertain about one's personal safety, thereby responding to aversive stimuli by learning the appropriate behavior to avoid punishment. Individuals high in harm avoidance are characterized as cautious, fearful, inhibited, and shy. In contrast, individuals low in harm avoidance are uninhibited, confident, carefree and energetic. The Reward Dependence scale was designed to measure the facility to acquire conditioned signals of reward or relief from punishment, which serves to increase resistance to extinction of previously rewarded behavior. Individuals high in reward dependence are sentimental, sensitive to social cues, and eager to help and please others. Those who are lower than average in reward dependence are socially detached, emotionally cool, independently self-willed, and tough-minded.

Participants were asked to complete the following psychosocial measures: Spielberger State-Trait Anxiety Inventory, (Spielberger, Gorsuch, & Lushene, 1967), the

Beck Depression Inventory (Beck, 1978), the Wahler Symptoms Inventory (Wahler, 1983), and the Khaveri Alcohol Test (KAT; Khaveri & Farber, 1978). Measures of anxiety, depression, and somatization have been used in prior studies of ACAs. Results have consistently indicated that ACAs, as a group, score higher than nonACAs on instruments that measure these domains (Dawson & Grant, 1994; Samson, 1994; Weller, 1997). The Khaveri Alcohol Test has also been used in previous research and has often indicated that ACAs, as a group, actually tend to drink less than the nonACA group (Samson, 1994; Weller, 1997).

The Spielberger State-Trait Inventory (Spielberger et al, 1967) is a 20-item, self-report instrument which asks individuals to endorse items which express how they are feeling at the present moment and then how they generally feel. It is a multiple choice answer format. The Beck Depression Inventory (Beck, 1978) consists of 21 groups of four statements which explore feelings and actions such as sleeping habits, feelings of depression, suicidal ideation, and life satisfaction. The responder checks one of the four statements which is closest to current feelings or behaviors. The Wahler Physical Symptoms Inventory (Wahler, 1983) is a 42-item, self-report questionnaire listing a variety of physical problems an individual may experience. Questions are scored on a 5-point Likert scale. Zero indicates that the respondent almost never experiences the symptom and five indicates that the respondent experiences the symptom nearly every day. The questionnaire queries physical well-being and includes such physical symptoms are losing weight, heart trouble, dizzy spells, and shakiness. The subject's score on the test is a sum of their responses to all the test items. The Khaveri Alcohol Test (KAT)

consists of four questions relating to three beverage types: beer, wine, and liquor. Participants are asked how much and how often of each type of product they usually drink, the maximum they have ever drank, and how often they drink the maximum amount. The number derived from calculating these amounts represents each individual participant's total annual consumption of absolute alcohol

Participants were administered the color word version of the Stroop effect test, presented as a computerized task via the Micro-Experimental Laboratory (MEL; St. James, Schneider, & Rodgers, 1994). Each individual performed one complete trial of the Stroop task, divided into three blocks of 36 stimulus presentations each. Within each block there were 36 control stimuli ("xxxx" in color), 36 congruent stimuli (the word and color matching), and 36 incongruent stimuli (the word and color mismatching). The words were presented on the color monitor of a computer. The participant was asked to read the color of each stimulus presentation as quickly as possible. The task of the participant was to ignore the color word (or row of x's) and to name the color in which the stimulus was displayed, responding as quickly as possible while avoiding errors. The examiner recorded the recitation in order to score for accuracy and kept time on a stopwatch. The time was recorded on a record sheet. The responses were scored for accuracy and the percentage correct was recorded on the record sheet.

In the negative priming task, a computerized task, participants were presented with two-letter displays (e.g., A-b) and were required to indicate as quickly and accurately as possible which letter was the uppercase letter. If the uppercase letter was on the left side of the display, the participants were instructed to press the "1" key with the index finger of

the left hand. If the uppercase letter was on the right side of the display, participants were instructed to press the "0" key with the index finger of their right hand. Following typical negative priming convention, in the A-b example the A is relevant (uppercase) while the b is irrelevant (lowercase). On control trials, the next display might be (f-J). On critical trials, the next display might be B-e. In the case of the critical trial, the lowercase b, which was previously irrelevant, now becomes relevant (uppercase B). Individuals are typically slower on critical trials than control trials because they must inhibit the irrelevant information across trials (Ferraro & Okerlund, 1996). There were 136 priming trials of which 68 were critical and 68 were control.

A visuospatial paired-associates learning task was adapted from previous research (Schandler, Cohen, & Antick, 1992) for use on the computer. During learning, participants received presentations of eight different Vanderplas and Garvin (1959) "nonsense" shapes with matched median association and heterogeneity values. Each shape was presented in one of eight positions on a grid comprised of eight lines radiating from center at 45-degree angles. A paired-associate learning paradigm was incorporated, with each shape serving as a stimulus and its grid position serving as the response associate. During each learning trial a 2-second duration stimulus image was first presented depicting one of the eight shapes in the center of the grid. This was immediately followed by a 2-second duration response image consisting of the grid with a question mark presented in the center. Finally, a 3-second information feedback image was presented, displaying the shape in its associated grid position.

The participant was required to indicate the grid position for each shape only during the 2-second presentation of the question mark image. The response was a key press in one of eight corresponding positions on the numeric keypad at the right of the computer keyboard. Each stimulus shape was associated with the same grid position throughout learning. The stimulus presentations were organized by trial blocks, with each block containing all eight shapes presented in random order. Learning was defined as a correct series of response during one trial block.

The computerized Matching Familiar Figures Test (MFFT; Hummel-Schluger & Baer, 1996) is a recently modified edition of the original MFF (Kagan, Rosman, Day, Albert, & Phillips, 1964) task, which was hand-administered as a set of cards. The new computerized version presents a picture at the top of the computer screen with eight pictures in two rows of four each on the lower portion of the screen. Seven of the pictures are very similar to the exemplar at the top while only one of the pictures is an exact match. It is the task of the participant to select the exact match. The task is scored in number of seconds for latency, as averaged over the 12 trials of the task, and the number of errors committed, averaged over the 12 trials.

A computerized continuous performance task was developed for use in this study, based on a rapid serial visual performance task (RSVP; Wesnes & Revell, 1984) developed to be used in nicotine studies. The RSVP presented single-digit numbers serially at the rate of 100 per minute. There were 5 blocks of 250 numbers each. The task of the participant was to notice when three numbers in a row were even or three numbers in a row were odd. When this occurred, the participant was instructed to hit the "enter"

key on the computer keyboard. There were 20 sets of three numbers in a row and 20 sets of two numbers in a row. Hits would be scored when the participant correctly hit the key when three numbers in a row were odd or three in a row were even. Errors would be noted when the participant did not respond with a key-press to an odd or even three-number presentation. The test developed by Wesnes and Revell was modified for the current study to include a false alarm condition. False alarms (errors of commission) would be noted when participants hit the key after two in a row as a measure of impulsivity.

A computerized matching program was also used in this study. The first presented set were block designs. An exemplar was displayed at the top of the computer screen. Five block designs were presented in a row across the bottom of the computer screen. It was the task of the participant to select which of the five designs at the bottom was an exact match to the block design exemplified at the top. These block designs were further categorized as easy and difficult, rotated and unrotated. The second presented set were cube designs which appeared to be three-dimensional. Again an exemplar was displayed at the top of the computer screen. Five cube designs were presented in a row across the bottom of the computer screen. It was the task of the participant to select which of the five cubes at the bottom was an exact match to the cube design at the top. These cube designs were further categorized as easy and difficult, rotated and unrotated. The computer scored for accuracy and reaction time.

Procedure

Initial screenings were conducted in psychology classes, where potential

participants were asked to voluntarily complete the screening form. Community

screenings were accomplished at a local community college, an Air Force base, and

through public advertisement. The potential participants were given the Family History

Alcohol Screening form (Appendix A) and a consent form (Appendix B) to complete,

noting that participation was voluntary. They were asked to indicate whether they would

be interested in further participation for additional research credit or monetary

remuneration by giving their full name and telephone number. Those selected were

contacted by telephone and an appointment time was set to administer the full battery of

research instruments.

Upon arrival in the research lab, each participant completed an additional consent

form (Appendix C) and was asked to complete a more comprehensive questionnaire about

his/her family history (Appendix D). Next each participant was administered the WAIS

Vocabulary and Block Design subtests. After this was completed, the participant was

seated in front of a computer. The examiner stayed in the testing area to guide the

participant through each procedure and to answer any questions. Participants were

administered, in random order, the cognitive instruments: the negative priming test, the

Stroop effect test, the RSVP, the Blocks and Cubes matching test, and the Matching

Familiar Figures Test. Participants were next administered the EPQ, the TPQ, the

Spielberger State-Trait Anxiety Inventory, the Beck Depression Inventory, the Wahler

Symptoms Inventory, and the Khaveri Alcohol Test. The complete administration took

between two and three hours for most participants. When the administrations were complete, the participant was paid $25 or three hours of class credit, as the participant requested.

CHAPTER III

RESULTS

The means for Age, and the scaled scores from the WAIS-III Vocabulary and Block Design subtests are presented Table 1, as a function of family history of alcoholism and gender. A 2 (Family History) X 2 (Gender) analysis of variance (ANOVA) on these measures revealed no significant differences.

Table 1. <u>Means and Standard Deviations for Screening Variables</u>

Variable		Female FH+	Female FH-	Male FH+	Male FH-
Age	Mean	23.35	21.30	24.07	23.43
	(SD)	(6.47)	(4.81)	(5.39)	(5.19)
WAIS Block Design					
	Mean	11.84	11.30	11.72	12.67
	(SD)	(2.73)	(2.37)	(2.30)	(2.60)
WAIS Vocab	Mean	10.55	10.67	11.38	10.87
	(SD)	(2.22)	(1.56)	(2.70)	(1.72)

A 2 (Family History) X 2 (Gender) ANOVA was conducted on the indices of depression, anxiety, physical health, drinking behavior, and individual and family drug use. The means and standard deviations for these measures are presented in Table 2. The analysis of the BDI scores revealed a significant main effect of family history,

F (1,119) = 10.387, p < .002, with the FH+ group (M = 8.88) scoring significantly higher than the FH- group (M = 4.72). A significant interaction of gender with FH was also observed, F (1,119) = 5.033, p < .027. A subsequent analysis of this interaction using a Tukey procedure (Myers & Well, 1991) indicated that male ACAs scored significantly higher than male nonACAs while there was no significant difference between female ACAs and nonACAs. The analysis of the measure of state anxiety (STAI-1) indicated a significant main effect of gender, F (1,119) = 4.109, p < .045, with higher scores for female participants (M = 37.69) than males (M = 34.14). The analysis of the measure of trait anxiety (STAI-2) revealed a significant main effect of family history, F (1,119) = 5.212, p < .024, with the FH+ group scoring significantly higher (M = 39.75) than the control group (M = 35.37). The results of the Wahler Physical Symptoms Inventory indicated a significant main effect of gender, F (1,119) = 6.19, p < .02, with females scoring significantly higher (M = 40.03) than males (M = 30.44) and a significant main effect of family history, F (1,119) = 4.894, p < .029, with the FH+ group scoring significantly higher (M = 39.57) than the control group (M = 31.07). The responses to the Khaveri Alcohol Test indicated a significant main effect of gender, F (1,119) = 14.356, p < .001, with a greater number of ounces of alcohol consumed annually by male participants (M = 485.72) than females (M = 122.16).

Table 2. <u>Means and Standard Deviations of Various Measures</u>

Variable		Female FH+	Female FH-	Male FH+	Male FH-
BDI	Mean	7.61	6.33	10.24	3.10
	(<u>SD</u>)	(7.58)	(8.10)	(8.20)	(3.83)
STAI-1	Mean	38.81	36.53	35.97	32.37
	(<u>SD</u>)	(10.15)	(10.75)	(8.42)	(8.23)
STAI-2	Mean	40.23	38.20	39.24	32.53
	(<u>SD</u>)	(12.18)	(11.22)	(10.87)	(6.72)
Wahler	Mean	42.45	37.53	36.48	24.60
	(<u>SD</u>)	(22.19)	(22.13)	(23.68)	(13.75)
Kahvari	Mean	114.099	130.486	660.87	316.40
	(<u>SD</u>)	(150.12)	(189.86)	(915.09)	(503.28)

A 2 (Family History) X 2 (Gender) ANOVA was conducted on the Eysenck Personality Inventory (EPI). The scoring of the EPI produced three scores, one for the dimension of Extroversion, one for Impulsivity, and one for Sociability. The means and standard deviations are indicated in Table 3. A significant main effect of gender was found for the EPI extroversion scale, $\underline{F}$ (1,119) = 13.388, $\underline{p}$ < .001, with males ($\underline{M}$ = 14.47) scoring higher than females ($\underline{M}$ = 11.59). A significant main effect of gender was also found for the EPI impulsivity scale, $\underline{F}$ (1,119) = 22.268, $\underline{p}$ < .001, with males ($\underline{M}$ = 4.92) scoring higher than females ($\underline{M}$ = 3.39). There were no significant differences observed in the analyses of the Sociability measure. Finally, there were no significant differences between the ACA and nonACA groups on any indices of this personality scale.

Table 3. Means and Standard Deviations for the EPI

Variable		Female FH+	Female FH-	Male fH+	Male FH-
EPI-E/I	Mean	12.06	11.10	14.69	14.27
	(SD)	(4.24)	(4.43)	(3.67)	(4.89)
EPI-Imp	Mean	3.55	3.23	5.24	4.60
	(SD)	(1.59)	(1.28)	(1.75)	(2.33)
EPI-Soc	Mean	7.35	6.70	7.72	8.30
	(SD)	(3.12)	(3.80)	(2.59)	(3.27)

A 2 (Family History) X 2 (Gender) ANOVA was conducted on the Tridimensional Personality Questionnaire (TPQ). The scoring of the TPQ produced three scores, one for Novelty Seeking, one for Harm Avoidance, and one for Reward Dependence. The means and standard deviations are indicated in Table 4. There was a significant main effect of gender for the TPQ Novelty Seeking scale, $F(1,119) = 16.404$, $p < .001$, with males ($M = 18.88$) producing higher scores than females ($M = 14.67$). There was also a significant main effect of family history for the TPQ Novelty Seeking scale, $F(1,119) = 6.025$, $p < .001$, with the FH+ group ($M = 17.98$) scoring higher than the control group ($M = 15.50$). There was a significant main effect of gender for the TPQ Harm Avoidance scale, $F(1,119) = 24.729$, $p < .001$, with females ($M = 16.05$) scoring higher than males ($M = 9.86$). There was a significant main effect of gender for the TPQ Reward Dependence scale, $F(1,119) = 11.847$, $p < .001$, with females ($M = 21.10$) scoring higher than males ($M = 17.93$).

Table 4. <u>Means and Standard Deviations of the TPQ</u>

Variable		Female FH+	Female FH-	Male FH+	Male FH-
TPQ Novelty Seeking					
	Mean	15.26	14.07	20.90	16.93
	(<u>SD</u>)	(6.67)	(4.62)	(5.77)	(5.72)
TPQ Harm Avoidance					
	Mean	16.55	15.53	10.45	9.30
	(<u>SD</u>)	(6.91)	(7.49)	(6.22)	(6.45)
TPQ Reward					
Dependence	Mean	20.87	21.33	17.83	18.03
	(<u>SD</u>)	(4.80)	(3.41)	(5.78)	(5.85)

The number of trials and errors to reach criteria on the Paired Associates task, the number correct, and the average latency to respond were subjected to separate 2 (Family History) X 2 (Gender) ANOVA. The means and standard deviations are found in Table 5. A significant main effect for number of trials to criteria was found, $\underline{F}$ (1,119) = 9.232, $\underline{p}$ < .003, with the FH+ group ($\underline{M}$ = 6.63) needing significantly more trials than the FH- group ($\underline{M}$ = 5.37) to complete the task. A significant main effect of family history was also found for the number of errors to criterion, $\underline{F}$ (1,119) = 4.452, $\underline{p}$ < .037, with FH+ participants making significantly more errors ($\underline{M}$ = 14.85) than FH- participants ($\underline{M}$ = 11.79) Finally, the analysis of the latency data revealed a significant main effect of family

Table 5. Means and Standard Deviations for the Paired Associates Task

Variable		Female FH+	Female FH-	Male FH+	Male FH-
Pair Assc	Mean	6.45	5.93	6.83	4.80
# Trials	(SD)	(2.32)	(2.39)	(2.51)	(1.92)
Pair Assc	Mean	32.03	27.40	33.00	25.70
# correct	(SD)	(10.44)	(9.88)	(12.66)	(8.91)
Pair Assc	Mean	965.47	862.88	977.28	940.55
latency	(SD)	(179.58)	(179.12)	(247.53)	(202.25)
Pair Assc	Mean	14.74	13.90	14.96	9.67
# errors	(SD)	(8.39)	(9.03)	(7.71)	(6.29)

history, $F (1,119) = 4.023$, $p < .047$, indicating longer lateness to respond for the FH+ group ($M = 971.38$ seconds) as compared to the FH- group ($M = 901.72$ seconds).

In the Stroop task, the participants were presented with three blocks of 36 stimulus presentations each. The congruent block was comprised of 12 presentations each of the words "red," "green," and "yellow" with the color and semantic word presentation matching. The control block was comprised of 36 presentations of "xxxx" in each of the three colors; twelve of the presentation were printed in red, twelve were green, and twelve were yellow.. In the incongruent condition, the names of the color words were presented with the semantic presentation of the word and the color in which it was presented mismatched. For example, the word "red" might be printed in green while the word

"green" might be printed in yellow. Each participant was instructed to read the color of each stimulus presentation as quickly as possible while ignoring the semantic presentation.

The Stroop task produced two measures for each condition, the latency to completion and the percent correct. The means and standard deviations for each condition for both measures are presented in Table 6. The measures examined were reaction time and percent correct for each of the congruent, the control, and the incongruent conditions. A 2 (Family History) X 2 (Gender) ANOVA was conducted for each dependent variable. There were no effects of FH status or gender on the response latency or percent correct for the congruent or control conditions. A significant main effect of gender was found in the incongruent condition for percent correct, $F (1,119) = 4.771$, $p < .032$, with females ($M = 96.30$) producing a lower percentage of correct responses than males ($M = 97.52$). There was a significant main effect of family history in the incongruent condition for percentage of correct responses, $F (1,119) = 7.816$, $p < .006$, with participants in the FH+ group ($M = 96.12$) producing a lower percentage of correct responses than those in the FH- group ($M = 97.68$).

Each participant's performance on the Negative Priming task was represented by a reaction time score for each trial in which the participant responded to which letter was the uppercase letter. The mean latency for control trials and critical trials was computed for each participant for both positions. Trials associated with errors and trials with latencies less than 200 milliseconds or greater than 1,000 milliseconds were deleted from these calculations. The average latency was computed over position for each participant. The measure of priming for each participant was the difference between control and

Table 6. Means and Standard Deviations for the Stroop Task

Variable		Female FH+	Female FH-	Male FH+	Male FH-
Stroop	Mean	15.164	15.57	15.75	16.27
RT/Cong	(SD)	(2.60)	(2.62)	(2.38)	(2.84)
Stroop	Mean	99.82	99.81	99.87	99.91
Pct/Cong	(SD)	(.373)	(.38)	(.33)	(.38)
Stroop	Mean	17.59	17.50	18.31	18.54
RT/Ctrl	(SD)	(2.32)	(2.725)	(2.34)	(2.77)
Stroop	Mean	99.34	99.72	99.52	99.57
Pct/Ctrl	(SD)	(.88)	(.43)	(.59)	(.84)
Stroop	Mean	25.98	25.30	26.67	26.38
RT/Incgrt	(SD)	(4.51)	(4.86)	(4.60)	(4.795)
Stroop	Mean	95.26	97.38	97.05	97.98
Pct/Incgrt	(SD)	(3.49)	(2.59)	(2.51)	(3.24)

critical trials. A 2 (Family History) X 2 (Gender) ANOVA of this measure produced no significant results.

Performance on the MFFT task was measured by the amount of time needed to respond to each of the twelve tasks and the number of errors committed when matching the correct picture with the exemplar. Latency was measured as the mean amount of time in seconds and was found by taking the total amount of time for the task and dividing it by twelve. If the initial response was incorrect, the subject had to make selections until the

correct response was made. The error rate was derived by taking the total number of errors in the task and dividing by the twelve presentations (e.g., if there was a total of 36 errors, the error rate would be 3.0). A significant main effect of family history was found for latency on the MFFT, F (1,119) = 5.226, p < .024, with the FH+ group (M = 21.14 seconds) faster than the FH- group (M = 28.61 seconds) in completing the entire task.

Table 7. Means and Standard Deviations for Negative Priming and the MFFT

Variable		Female FH+	Female FH-	Male FH+	Male FH-
NP	Mean	- 12.21	2.76	- 8.71	- 5.76
	(SD)	(39.51)	(32.03)	(53.88)	(31.75)
MFFT Latency	Mean	20.46	23.75	21.87	33.47
	(SD)	(9.76)	(16.76)	(14.04)	(26.47)
MFFT Errors	Mean	1.47	1.58	1.65	1.32
	(SD)	(.72)	(.67)	(.60)	(.75)

Performance on the RSVP task was examined with a 2 (gender) x 2 (ACA status) x 5 (number of trials) mixed ANOVA. Performance was represented as median reaction time and mean reaction time when responding to a false alarm (two numbers in a row but not three) and the percentage of responses to the false alarm, as well as median reaction time and mean reaction time when responding correctly (to three odd or even numbers consecutively) and the percentage of correct hits. The results are also displayed for overall percentage of misses to the presentation of three consecutive odd or even numbers. There was a significant main effect of gender for percentage of hits, F (1,119) = 6.28,

$\underline{p} < .014$), with males making more hits than females. There was a significant main effect of gender for percentage of misses, $\underline{F}$ (1,119) = 4.73, $\underline{p} < .032$, with females making more misses.

A 2 (Family History) X 2 (gender) ANOVA was conducted on the results of the Matching Blocks and Cubes task. The means and standard deviations are listed in Table 8. A significant main effect of gender for block identification was found, $\underline{F}$ (1,119) = 4.24,

Table 8. <u>Means and Standard Deviations for RSVP</u>

Variable		Female FH+	Female FH-	Male FH+	Male FH-
False Alarm	Mean	55.99	58.04	60.02	58.94
Median RT	(<u>SD</u>)	(8.41)	(8.5)	(8.4)	(8.7)
False Alarm	Mean	57.42	56.71	59.01	57.92
Mean RT	(<u>SD</u>)	(7.22)	(7.5)	(6.8)	(7.8)
False Alarm	Mean	17.93	14.56	17.01	13.31
Percentage	(<u>SD</u>)	(10.73)	(11.67)	(12.02)	(10.03)
Hits	Mean	90.88	88.82	85.99	87.55
Median RT	(<u>SD</u>)	(8.8)	(8.8)	(9.25)	(8.62)
Hits	Mean	89.50	87.53	85.68	86.85
Mean RT	(<u>SD</u>)	(6.6)	(6.5)	(7.3)	(5.9)
Hits	Mean	27.64	29.54	33.27	36.41
Percentage	(<u>SD</u>)	(12.44)	(14.7)	(10.85)	(16.39)
Misses	Mean	54.62	56.05	49.83	50.43
Percentage	(<u>SD</u>)	(11.39)	(14.48)	(12.02)	(14.39)

$p < .042$, with males ($\underline{M} = 86.52$) performing better than females ($\underline{M} = 76.88$).

A significant main effect of gender for cube identification was found, $\underline{F}$ (1,119) = 10.39,

$p < .002$, with males ($\underline{M} = 82.71$) identifying the correct cube to exemplar more often than

females ($\underline{M} = 66.80$).

Table 9. <u>Means and Standard Deviations for the Matching Block and Cube Task</u>

Variable		Female FH+	Female FH-	Male FH+	Male FH-
Blocks	Mean	762.27	756.34	742.91	825.05
Median RT	(<u>SD</u>)	(222.2)	(216.9)	(199.8)	(235.3)
Blocks	Mean	703.89	670.33	631.55	737.66
Mean RT	(<u>SD</u>)	(169.8)	(231.8)	(165.6)	(231.1)
Blocks	Mean	74.63	79.13	83.93	89.13
%Correct	(<u>SD</u>)	(29.6)	(20.42)	(27.44)	(24.26)
Cubes	Mean	701.31	798.10	695.71	693.73
Median RT	(<u>SD</u>)	(269.5)	(209.8)	(241.1)	(240.4)
Cubes	Mean	740.03	862.53	789.83	817.95
Mean RT	(<u>SD</u>)	(317.0)	(212.4)	(192.2)	(228.1)
Cubes	Mean	63.35	70.26	79.07	86.34
% Correct	(<u>SD</u>)	(31.65)	(21.2)	(30.87)	(24.7)

CHAPTER IV

DISCUSSION

This study proposed to investigate cognitive inhibition processes and impulsivity between groups of female and male individuals who had a family history of alcohol problems and those with no such family history. It was hypothesized that a pattern of impairment in cognitive inhibition and a tendency to respond more impulsively to stimuli would be found with the group of individuals with a family history of alcoholism.

The participants were administered cognitive tests designed to measure their ability to inhibit responding or tendency to respond impulsively. On the Stroop task, which measures the ability to inhibit responding, the ACA groups produced fewer correct responses to the task. When presented with the word of a color printed in a different color from the semantic presentation and told to name the color rather than read the word, the ACA participants were more likely than the nonACA participants to make an error and read the semantic presentation rather than name the color. They were more likely to have difficulty in inhibiting the semantic response than the participants who had no family history of alcoholism. Although the responses of both groups were very accurate, the FH+ group was significantly less accurate. The groups were only differentiated on their ability to inhibit responding to the incongruent stimuli. Therefore, the FH+ group was less able to inhibit a dominant response when the task demands required such inhibition.

In previous research (Biederman, Munir, & Knee, 1987), the MFFT has differentiated groups with higher impulsivity from those who respond less impulsively. In the current study, performance on the MFFT indicated that the FH+ groups completed the task in significantly less time than the FH- groups. Although the FH+ group made slightly more errors than the FH- group (1.56 versus 1.45), the difference did not approach conventional levels of significance. One way in which the current findings differed from previous research results was that earlier research was conducted using the original hand-administered instrument. The computerized task used in this study was developed in 1996 by Hummel-Schluger and Baer. When compared with the earlier hand-administered instrument, it was found to correlate moderately, with a .61 for latency and a .40 for error scores. The change from the hand administration to the computer administration may be one reason for a difference in findings. The current study found that the ACA group performed as hypothesized as far as speed of performance, completing the task more quickly than the nonACA group. The failure to accomplish the error performance hypothesis may have been due to the matching of participants by intellectual level, which may have been more powerful in this study than any differences due to family history. Another difficulty in this study was the age of the participants. Salkind and Wright (1977) found that performance on the MFFT improves with the age of the test groups. This may be another explanation to account for the lack of differentiation between the groups in the error rate. Perhaps if the task were more difficult, larger differences in error rates between FH+ and FH- subjects would have been observed.

The Paired Associates task provided quite robust results. The ACA groups performed in a similar fashion as groups with a positive family history of alcoholism, presented in earlier research (Garland, Parsons, & Nixon, 1993; Schandler, Brannock, Cohen, & Mendez, 1993). The ACA groups required significantly more trials to complete the task than did the nonACA groups. The ACA participants also took longer in each trial to successfully complete the task and made significantly more errors. The Paired Associates task was successful at differentiating the groups and clearly supported the effort to assure that group membership was valid.

The learning performance displayed by the results of the Paired Associates task supported the hypothesis that persons with a family history of alcohol problems have a visuospatial learning performance inferior to persons with no family history of alcoholism (Schandler, Cohen, & Antick, 1992). This inferiority was reflected both in reduced speed of learning and in a significantly larger number of error responses.

While the negative priming task did not produce scores which significantly differentiated the groups, it should be noted that the scores for the FH+ group were lower than for the FH- group, indicating that there may have been some slower latency for the FH+ group. If the task had been longer, results might have reached significance.

The absence of any significant group differences for the RSVP task was disappointing given that the task has been used extensively in previous studies and was effective in indicating performance declines over time (Wesnes & Revell, 1984; Parrot & Winder, 1989). The test was used in studies of administration of scopolamine and nicotine and was sensitive enough to indicate when drug adminstration changed the ability to pay

attention, the amount of time to react to the stimuli, and the error rate. A modification to the test was made for the current study to include a false alarm condition. No change over the five blocks of administration was found for the number of hits, the error rate, or the newly developed false alarm condition. One difference between the current findings and previous research may be related to practice time. Previous work utilizing the RSVP task used extensive practice (Parrot & Winder, 1989) while the present study used minimal practice, and thus the task may have been too difficult given the amount of practice time. The RSVP task may also be insensitive to group differences. This was the first published administration of the RSVP task in a test comparing performance between groups by family history of alcoholism. A recent comparison of 15 adults with a history of impulsive behavior with 15 normal control (Dougherty, Bjork, Marsh, & Moeller, 2000) were tested on a more conventional continuous performance task and the researchers found the impulsive group to have elevated errors of commission, lower stimulus discrimination between target and nontarget stimuli, and shorter response latencies.

The results of the Block and Cube task failed to find any significant differences between the family history groups. The findings with the matching task actually agreed with the MFFT results, in that there were no differences between the family history groups on the error rate for matching to an exemplar. This may again have been due to the higher intellectual functioning of the groups, being individuals who were, for the most part, college or technical school enrollees, which may have compensated for any differences in the groups in problems attending to a stimulus and taking the time to make good choices.

The results from the personality measures were mixed. No significant differences between FH+ and FH- groups were observed on the Extroversion/Introversion, Impulsivity, or Sociability scales of the EPI. Beaudoin, Murrray, Bond and Barnes (1997) tested 982 male and female participants with the hypothesis that the ACAs would be lower than nonACAs in self-esteem, and higher in neuroticism and psychoticism. They found the ACA groups to differ from nonACAs on factors such as lower sociability and higher impulsivity, as well as overall higher neuroticism and psychoticism. Finn, Earlywine, and Pihl (1992) tested 95 college males who either did or did not have a family history of alcoholism. They found their FH+ group differed significantly from an FH- group on the scales measuring neuroticism and experience-seeking, but not on measures of extroversion, disinhibition, or thrill-seeking. The findings of this current study were more similar to those obtained by Sher, Walitzer, Wood, and Brent (1991) who found no significant differences on the EPI subscales of extroversion or impulsivity between family history groups.

On the TPQ, the Novelty Seeking scale differentiated between the family history groups with the ACA groups scoring significantly higher than the nonACA groups. Cloninger (1988) described novelty-seeking as a heritable tendency toward frequent exploratory activity and intense exhilaration in response to a novel stimuli and found that males with a family history of alcoholism tended to score higher on indices of novelty-seeking. In the current study, this scale did differentiate the family history groups, perhaps indicating that, even with participants with the age and intellectual level to be attending college, this scale is particularly sensitive to differentiating between the family history

groups. Sher, Walitzer, Wood, and Brent (1991) found their groups to be differentiated by novelty-seeking with both male and female FH+ groups scoring significantly higher on the novelty-seeking scale than FH- groups. Galen, Hendersen, and Douglas (1997) found that high novelty-seeking and low harm-avoidance were positively correlated with a higher frequency of early-onset drinking among 140 adolescent ACAs.

The Cloninger model of family transmission of alcoholism also predicts that adults with a family history of alcoholism would also be lower in harm avoidance and reward dependence than individual with no such family hisotry. Individuals who are harm-avoidant have a facilitated capacity to learn from their experience in order to avoid punishment and sometimes feel uncertain about their safety. Individuals who are reward-dependent repeat behavior in order to receive benefits, or be rewarded, and base future actions on their desire to be relieved from punishment. In this study, there was no significant difference found between the family history groups on either the Reward Dependence or Harm Avoidance scales. This is not, however, inconsistent with othr studies of the TPQ. Meszaros, Lenzinger, Hornik, Fureder, Willinger, Fischer, Schonbeck, and Aschauer (1999) found the Novelty Seeking scale was effective in predicting early onset alcohol abuse and discriminated alcoholics with antisocial behavior from their non-antisocial counterparts. In their analysis, however, they found the Harm Avoidant and Reward Dependence scales to be less consistent at discriminating between ACA and nonACA groups.

The family history groups also differed on other characteristics. The ACA groups scored significantly higher on the BDI than the nonACA groups. This difference was

primarily attributable to the higher BDI scores of the male members of the ACA group. This was consistent with other findings that men with a positive family history of alcohol problems also experience more problems than men who do not have a family history of alcohol problems with depression (Belliveau & Stoppard, 1995; Bush, Ballard, & Fremouw, 1995; Dawson & Grant, 1998). The ACA groups also reported more family members who had been diagnosed with depression, which is again consistent with previous research (Dawson & Grant, 1998).

The ACA groups also provided higher scores on the Trait scale of the STAI. Both males and females were experiencing higher levels of enduring, trait-like anxiety characteristics. This finding was in agreement with the results of a study conducted with 253 ACAs and 237 nonACAs (Sher, Walitzer, Wood, & Brent, 1991) in which they found that ACAs were at significantly greater risk of meeting the criteria for a diagnosis of Agoraphobia, Social Phobia, Simple Phobia, or Generalized Anxiety Disorder.

To summarize the personality findings of the family history groups, this study found that the ACAs, as a group, were more likely than nonACAs to be novelty-seeking, and to endorse more symptoms of depression and trait anxiety. They were also more likely to report having family members who had been diagnosed with depression.

While not the focus of this study, there were a few findings of differences as a function of gender which were of interest. On the Stroop task, there were no gender differences on the congruent or control tasks. On the incongruent task, the female participants had more difficulty inhibiting the semantic naming response than males. This was in variance to other research which has found no gender differences for the number of

correct responses in the incongruent condition of the Stroop task (Ben-Tovim, Walker, & Douros, 1993; Boone, 1999). Women have been found to complete the task faster than men (Mekarski, Cutomore, & Suboski, 1996; Strickland, D'Elia, James, & Stein, 1997).

On the RSVP task, the female participants missed more of the three number combinations than their male counterparts. A search of previous research to discover what gender differences have been noted on performance on continuous performance tasks reveals that most research has been done on same-sex groups. A study conducted in 1997 on 435 first- and second-grade children (mean age 7.9 years) indicted that girls made fewer errors than boys on a CPT (Pascualvaca, Anthony, Arnold, Rebok, Ahearn, Kellam, & Mirsky, 1997). A study of 22 ADHD children and 19 normal controls, aged 6 to 21 years, found no differences on CPT performance by gender (Seidel & Joschko, 1990).

On the Matching Blocks and Cubes task, males were better than their female counterparts at identifying the correct block exemplar. This was also true for cube identification. This is consistent with previous research showing that males tend to perform superior to females on tests of spatial perception and mental rotation (e.g., Linn & Petersen, 1985).

Regarding the personality characteristics of the EPI, it was found that males scored higher than females on the Extroversion scale as well as the Impulsivity scale. These findings are consistent with other research which identified gender differences on the EPI (Clift & Wilkins, 1993; La Grange, Jones, Erb, & Reyes, 1995). Males also scored higher on the TPQ Novelty Seeking scale. On the other hand, female participants scored

significantly higher on the Reward Dependence and Harm Avoidance scales, a consistent finding with earlier research by Cloninger et al. (1991).

This study found significant differences on specific areas of processing between the groups of interest, those with and without a family history of alcoholism. Care was taken to delineate the groups by asking that the individuals in the FH+ group had one natural parent and one additional second-degree relative who could be defined as alcoholic. This careful scrutiny of the family pedigree was more likely to identify individuals with a family transmission of alcoholism factors and has been recommended by ACA researchers (Finn, Earleywine, & Pihl, 1982; Schuckit, 1994a). The success of this effort still depends, however, on self-report and an individual's personal definition of alcoholism, which is inherently a flawed procedural definition. Each individual was asked to answer specific questions pertaining to medical problems, work-related problems, marital problems and legal problems of family members. In order to qualify for inclusion in the FH+ group, the individual needed to identify at least three alcohol-related problems in the life of the alcohol-abusing parent. Nevertheless, the decision that the troubles at home or at work for the parent were alcohol-related, and that the parent was alcoholic, was still a qualified opinion. The questionnaire developed for this study attempted to minimize this problem by asking very specific questions to limit the vagueness of each participant's self-report.

This was also a self-selected group in that some portion of the university students who could participate in the study by completing the screening form were unwilling to do so. It is not known if they did not participate because they knew of the subject matter of the screening or they simply were not interested in research participation. Also, almost

half of those who appeared to qualify for inclusion in the FH+ group were unwilling to participate. How the individuals who agreed to participate differed from those who refused is not known. Neither is it known how that might have influenced the outcome of the study. FH+ participants were randomly asked at the conclusion of the testing if they still considered that their participation was legitimate, based on their family history, and in no case did anyone respond differently.

Another limitation on this study may have been effect size. A calculation of effect size on the findings in this study indicate that effects sizes were very modest to quite small. It may very well be that a sample size of 30 was too small to find effects, particularly for a moderator variable such as family history. A brief review of the literature in which effect sizes were calculated indicates that family history is not a major variable by itself, but rather is a moderator variable.

Having found some modest differences between our groups cannot, nevertheless, lead to definitive statements about the genetic influences of alcoholic behavior on neuropsychological responding. Although there does seem to be a great deal of evidence that indicates that alcoholism may be genetically transmitted in families and although there is quite a bit of evidence that there is a pattern of personality characteristics and cognitive anomalies that coincides with that pedigree, it is not possible to state definitively that those secondary characteristics are due to the primary criteria of alcoholism. The confound of environmental influences is enormous (Searles, 1988). The literature about the effects of the alcoholic home and chaotic environments both inside the home and due to outside peer pressure is extensive and will not be reviewed here. Nevertheless, the contributions of

these external influences are very difficult to tease apart from what might be genetic. Future work should include measures to assess the participants' memories of how stable or chaotic were their home environments.

While the preponderance of research seems to indicate that genetic influences are paramount, there is still a large number of studies which have not found significant personality or cognitive differences between the groups. Alterman, Searles, and Hall (1989) found few significant differences between groups of male college students based on a family history of alcoholism. They did not, however, ascertain whether their "high-risk" group, those with an alcoholic parent, were part of a multigenerational family pattern of alcoholism.

Bates and Pandina (1992) also found no significant differences between ACA and nonACA groups when they tested 1,270 subjects at three different times over three years. They concluded that their findings did not support any hypothesis that premorbid cognitive deficits were to be found in the offspring of individuals with a family history of alcoholism. This large and complex study has, nevertheless, some procedural problems. Of the 1,270 subjects solicited from the community, 677 were used in the analysis. Of that number, 384 were in the FH- group. The FH+ group was subdivided into four groups: mother only alcoholic, father only alcoholic, grandparents only alcoholic, and parent and a second-degree relative. The multigenerational group consisted of 33 subjects. When the scores of the cognitive testing were initially compared, the FH+ groups were comprised of all 293 subjects, most of whom had only a single parent alcoholic or only a second-degree relative alcoholic. The groups were split apart for further analysis, but the disparity in

group sizes may have resulted in inconsistent findings. The researchers did not indicate that their statistical analyses included computation for groups of unequal size. They also mentioned that their groups may have been somewhat unrepresentative of the general population because they were above the average in family income.

The current study provided mixed results, some of which may be due to the age and intellectual development of the particular participants. By the time young people reach college, there has been a winnowing out process that significantly impacts the potential pool of participants. Many children from the troubled homes of alcoholics do not do well academically (Ervin, Little, Streissguth, & Beck, 1984) and might not go on to college, for a variety of reasons. By testing college or vocational school students, as was done in this study, many potential participants are not included. To assess the greatest number of individuals who might conceivably be at risk for a genetic transmission of a spectrum of difficulties, including heightened potential for alcohol abuse themselves, personality impairments, and cognitive difficulties, it would be preferable to use a younger population. Because of the higher incidence of the offspring of alcoholics being more likely to use alcohol themselves, and at a younger age (Cloninger, 1988), testing should be done with young people who have not yet begun to use substances themselves. Even with the younger group, there will still be a problem with self-selection of group membership. Young sensation-seeking, conduct disordered individuals, assuming such from Cloninger's hypothesis about some of the personality characteristics of Type II alcoholics, might decline to participate. Also, participants of that age group would require parental permission, and alcoholic families might choose to keep their children from participation in

research that might reveal problems in the family. In light of the many factors involved in the winnowing-out process to arrive at the population who participated in this study, the findings of group differences as a function of family history would seem to be particularly robust.

Because individual differences will always exist, future research needs to be done with groups that maximize the opportunity to account for individual variances. Schuckit (1994a) suggested that study groups number at least 30 participants each, which the current study set as the goal for number of individuals per group. It might be suggested to use even larger numbers because of the number of variables for which to account when doing similar studies. Because of the complex genetic influences likely to be involved, only a minority of the offspring of alcoholics are likely to carry the genetic factors associated with the increased risk (Schuckit, 1994a). The investigator also needs to closely question participants so that behavioral definitions of alcoholic behavior of family members, such as legal, professional, or familial problems caused by drinking, are in accord with the reported opinion of alcoholism made by the participants. This is likely the most critical feature of participant selection. This study developed an in-depth questionnaire to identify a family history of alcoholism. It would be recommended that future research be very stringent in identifying the familial transmission of an alcohol problem. The questionniare needs to be followed up with a personal interview to verify the details. The ACA group must include only those participants for whom a family history of alcoholism has been clearly identified.

The preponderance of literature published on the differences between adult children of alcoholics, as a group, and those without that pedigree indicate that ACAs, as a group, may indeed start life with certain cognitive, psychosocial, and personality deficits. It is quite unlikely that a specific gene for alcoholism exists, but there may be several genetic variables, each with a cognitive or behavioral impairment, that add up to a unique combination that predispose the individual for future problems regulating their behavior, or place them at a higher risk for alcoholism than the general population. Those difficulties with behavioral inhibition and impulsivity may be part of a spectrum which includes problems with abstaining from the use of intoxicating substances. Research which links behaviors we can assess or measure by psychological testing with specific brain chemistry may ultimately be identified on DNA sequences. It will likely be some combination of cognitive deficits, dysregulation in behavioral regulation or executive functioning, high tolerance for alcoholic beverages, and a pattern of personality responding that will one day be identified as the antecedent factors that come together to cause alcoholism. This one small study replicated previous research that indicated that ACAs, as a group, have more difficulty learning a visuospatial task than nonACAs. It also found that these individuals, as a group, also experience more difficulty inhibiting an erroneous cognitive response. We did not find that ACAs were more impulsive, but that may have been more a function of the measures selected for this study than the actual performance of the participants. We also found that, as a group, ACAs report more depression and trait anxiety, which has been linked by other researchers to a familial pattern associated with substance use problems.

In summary, this study proposed to compare groups of men and women based on their status as ACA or nonACA on tests of cognitive inhibition, impulsivity, and various personality characteristics. The current research found that, in this study, ACAs were more likely to have difficulty inhibiting their responding and learning in a visuospatial paradigm. They were not, however, found to react more impulsively nor did they have more difficult with sustained attention. They tended to be more sensation- or novelty-seeking, but not more likely to be socially-impulsive or harm-avoidant. It is suggested that future research use groups who are younger than this current study's college-age participants.

APPENDICES

APPENDIX A

Subject Number_____   FS_____   MS_____

Please check the answer below that descirbes the drinking behavior of each of your parents. Take your time and answer as accurately as possible. Check "yes," "no," or "dk" (don't know), for each question. Please answer only for your biological parents.

Father				Mother			
	Yes	No	DK		Yes	No	DK

1. Is this your biological father? ___ ___ ___   1. Is this your biological mother? ___ ___ ___

2. Did your father regularly   ___ ___ ___   2. Did your mother regularly   ___ ___ ___
   drink alcoholic beverages?   drink alcoholic beverages?

3. Did your father's alcohol use ___ ___ ___   3. Did your mother's alcohol use ___ ___ ___
   cause any health problems   cause any health problems
   for him?   for him?

4. Did your father's alcohol use ___ ___ ___   4. Did your mother's alcohol use ___ ___ ___
   cause any problems for him at   cause any problems for him at
   work or interference with his   work or interfere with his
   performance at work?   performance at work?

5. Did you father's alcohol use ___ ___ ___   5. Did your mother's alcohol use ___ ___ ___
   cause marital or relationship   cause marital or relationship
   problems?   problems?

6. Did your father's alcohol use ___ ___ ___   6. Did your mother's alcohol use ___ ___ ___
   ever result in a drunk driving   ever result in a drunk driving
   arrest or arrest for public   arrest or arrest for public
   intoxication?   intoxication?

7. Have you ever thought that   ___ ___ ___   7. Have you ever thought that   ___ ___ ___
   your father had a drinking   your mother had a drinking
   problem?   problem?

8. Has your father ever received ___ ___ ___   8. Has your mother ever received ___ ___ ___
   treatment for alcoholism?   treatment for alcoholism?

9. Did you ever think your   ___ ___ ___   9. Did you ever think your mother ___ ___ ___
   father was an alcoholic?   was an alcoholic?

10. Have any of your father's   ___ ___ ___   10. Have any of your mother's   ___ ___ ___
    relatives (his parents, brothers,   relatives (her parents, brothers,
    sisters) ever received treatment   sisters) ever received treatment
    for alcoholism or had a drinking   for alcoholism or had a drinking
    problem?   problem?

APPENDIX B

Consent Form

You are invited to participate a study being conducted by Louise Weller, a doctoral candidate in the Psychology Department of the University of North Dakota, as part of her dissertation research. This study will examine the relationship between parental alcohol consumption and their adult children's performance on several aspects of cognitive functioning.

Today you will be asked to complete a questionnaire about the alcohol use of your parents and extended family. The entire procedure will consume about ten minutes. Please answer each question as honestly as possible. Your responses to the items will be held in strict confidence. If you are willing to be included in further research, please write your name and telephone number on the top of the form. If you do not want to be included in further research, you may decline to participate.

If you are uncomfortable when filling out this questionnaires you may terminate your participation without consequence at any time. In addition, if after completing the questionnaire, you become upset, you should contact the researcher, Louise Weller at 777-3326 or Dr. Tom Petros at 777-3260. Should you experience any psychological discomfort due to completing this questionnaire, the campus counseling center, located at O'Kelly Hall, phone 777-2127, provides counseling services to university students at no charge. The Psychological Services Center, located at 210 Montgomery Hall, in the 3100 block of University Avenue, phone 777-3691, provides psychological services to students and community members on a sliding scale fee basis, and any charges incurred will be the responsibility of the individual.

The benefits from this study stem from an improved understanding of how parental drinking and various psychological measures of their adult children may be related. Immediate benefits to you would be the opportunity to experience what scientific research is about.

In return for your participation today, your instructor will provide class credit. Your decision whether or not to participate will not prejudice your future relations with UND or the Psychology Department. If you decide to participate, you are free to discontinue participation at any time without prejudice. Should you decide to provide your name and phone number as an indication that you are interested in participating in the full research project, you may be telephoned. Further participation may earn additional class credit or a financial remuneration.

There are no physical risks involved with participation in this project. However, some people may become anxious or angry because they are taking tests and being asked about personal and sensitive information. However, your name will not appear on any of the questionnaires that you complete today. You will only be identified by a subject number. All test data will be kept strictly confidential in the researcher's office for three years, after which it will be shredded.

The investigators involved will make themselves available to answer any questions that you have regarding this study. In addition you are encouraged to ask any questions that occur to you in the future. You are not required to enter into this research if you wish not to. Any questions you have will be answered by calling Louise Weller at 777-3536 or Tom Petros at 777-3260. You may have a copy of this form if you want one.

_____ Telephone #_____ Date_____
Please print name

_____
Signature

I was 18 years or older on my last birthday. _____Yes   _____No

APPENDIX C

Consent Form

You are invited to participate a study being conducted by Louise Weller, a doctoral candidate in the Psychology Department of the University of North Dakota, as part of her dissertation research. This study will examine the relationship between parental alcohol consumption and their adult children's performance on several aspects of cognitive functioning.

Today you will be asked to complete seven paper-and-pencil questionnaires and four computerized tasks. The entire procedure will consume about two hours. Please put forth your best effort on these tasks. Some of the questionnaires will ask you about your feelings, attitudes, and some of your activities. Please answer each question as honestly as possible. Your responses to the items will be held in strict confidence and your name will not be associated with your questionnaires, only your subject number.

If you are uncomfortable when filling out these questionnaires you may terminate your participation without consequence at any time. In addition, if after filling out the questionnaires, you become upset, you should contact the researcher, Louise Weller at 777-3326 or Dr. Tom Petros at 777-3260. Should you experience any psychological discomfort due to completing these tests, the campus counseling center, located at O'Kelly Hall, phone 777-2127, provides counseling services to university students at no charge. The Psychological Services Center, located at 210 Montgomery Hall, in the 3100 block of University Avenue, phone 777-3691, provides psychological services to students and community members on a sliding scale fee basis, and any charges incurred will be the responsibility of the individual.

The benefits from this study stem from an improved understanding of how parental drinking and various psychological measures of their adult children may be related. Immediate benefits to you would be the opportunity to experience what scientific research is about.

In return for your participation you will receive class credit or a financial remuneration in accordance with the amount of time 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 to participate, you are free to discontinue participation at any time without prejudice. Should you discontinue at some point less than full completion, a prorated amount of credit or payment, consistent with the amount of time you spend in the experiment, will be given.

There are no physical risks involved with participation in this project. However, some people may become anxious or angry because they are taking tests and being asked about

personal and sensitive information. However, your name will not appear on any of the questionnaires that you complete today. You will only be identified by a subject number. All test data will be kept strictly confidential in the researcher's office for three years, after which it will be shredded.

The investigators involved will make themselves available to answer any questions that you have regarding this study. In addition you are encouraged to ask any questions that occur to you in the future. You are not required to enter into this research if you wish not to. Any questions you have will be answered by calling Louise Weller at 777-3536 or Tom Petros at 777-3260. You may have a copy of this form if you want one.

_____ Telephone #_____ Date_____
Please print name

_____
Signature

I was 18 years or older on my last birthday. _____Yes   _____No

APPENDIX D

Code No._____

Please answer each of the following questions. Please check only one answer unless otherwise indicated by the question.

1. Your age: _____.

2. Your sex: Female_____   Male_____

3. Which of the following best describes your racial/ethnic background?

_____   A. African-American
_____   B. Asian
_____   C. Caucasian
_____   D. Hispanic
_____   E. Native-American
_____   F. Other (please specify):_____

4. If you are in college or tech school, what is your current class ranking?

_____   A. First year student
_____   B. Second year student
_____   C. Third year student
_____   D. Fourth year student
_____   E. Other (please specify):_____

5. If you are in college or tech school, what is your current major?_____

6. What is your current marital status?

_____   A. Never married
_____   B. Married
_____   C. Separated
_____   D. Divorced
_____   E. Widowed

7. Are you currently in an intimate relationship?

Yes _____   No _____   If yes, for how long?
                                   _____ Less than 3 months
                                   _____ 3 - 12 months
                                   _____ 1 - 5 years
                                   _____ More than 5 years

8. How many intimate relationships have you had that lasted more than 3 months? _____

9. How much do you smoke?
        _____   A. Never smoked
        _____   B. Have quit for more than a year
        _____   C. Have quit for less than one year
        _____   D. Currently smoke less than one pack per day (PPD)
        _____   E. Currently smoke one PPD

10. How much caffeine do you drink (include coffee, soft drinks, and tea)?

      _____ A. None
      _____ B. 1 - 2 cups per day (CPD)
      _____ C. 3 - 4 CPD
      _____ D. 5 -6 CPD
      _____ E. 7 - 10 CPD
      _____ F. 11 or more CPD

11. Do you take any prescribed medications?

    Yes _____ No_____ If yes, what medication(s) do you take, what amount, and why?

_____

_____

12. Do you regularly take any over-the-counter medications?

    Yes _____ No_____ If yes, what medication(s) do you take, what amount, and why?

_____

_____

13. Have you used any drugs recreationally?

    Yes _____ No_____

If yes, please check the specific drug used, amount used, and how often:

Drug   Amount and frequency

    _____ a. Pot, marijuana, hash
    _____ b. Amphetamines, uppers, speed, stimulants
    _____ c. Barbiturates, sedatives, downers, sleeping pills, qualudes
    _____ d. Tranquilizers, valium, librium
    _____ e. Cocaine, coke, crack
    _____ f. Heroin, methadone
    _____ g. Other opiates - demerol, morphine, percocet
    _____ h. Psychedelics - LSD, peyote, mescaline, PCP
    _____ I. Other (specify):_____

14. Do you use drugs recreationally now:

    Yes_____ No_____ If yes, what are you using, in what amount, and how often?

_____

15. Have you ever misused any prescription drugs?

    Yes_____ No_____ If yes, what have you used, in what amount, and how often?

_____

_____

16. Do you have any medical problems currently?

    Yes_____   No_____   If yes, what medical problem do you have?

_____

_____

17. Have you ever been hospitalized medically?

    Yes_____   No_____   If yes, what medical problem did you have?

_____

_____

18. Have you ever seen a counselor or psychiatrist ?

    Yes_____   No_____   If yes, for what were you seen, when, and for how long?

_____

_____

19. Have you ever had problems with depression and/or anxiety?

    Yes_____   No_____   If yes, what medical problem do you have?

_____

_____

20. As far as you know, were there any problems with your mother's pregnancy or delivery of you?

    Yes_____   No_____   I don't know_____
    If yes, please describe.

_____

_____

21. As far as you know, did you walk, talk, and sit up on time?

    Yes_____   No_____   I don't know_____
    If no, please describe.

_____

_____

22. Have you experienced any legal problems, such as disorderly conduct or public intoxication, either as a juvenile or as an adult?

    Yes_____   No_____
    If yes, please describe.

_____

_____

23. Have you ever been diagnosed and/or treated for attention-deficit/hyperactivity disorder or a learning disability?

Yes_____   No_____

_____

_____

24. Were you ever in any special classes in school?

Yes_____   No_____   If yes, what kinds of special classes were you in?

_____

_____

25. Did you ever have to repeat a grade?

Yes_____   No_____   If yes, what medical problem do you have?

_____

_____

26. How would you best describe your grades in school: in high school and in college?

_____   a. Average
_____   b. Better than average
_____   c. Worse than average

27. Is there anyone in your family who has been diagnosed with attentional problems?

Yes_____   No_____   I don't know_____
If yes, who had the attention problem and how did it cause difficulty?

_____

28. Is there anyone in your family who has been diagnosed with a learning disability?

Yes_____   No_____   I don't know_____
If yes, who had the problem and how did it cause difficulty?

_____

_____

29. Has your mother ever been diagnosed and/or treated for depression?

Yes_____   No_____   I don't know_____
If yes, when and what treatment did she receive?

_____

_____

_____

30. Has your mother ever been diagnosed and/or treated for anxiety (including generalized anxiety, phobia, post traumatic stress disorder, acute stress disorder, or obsessive-compulsive disorder)?

Yes_____   No_____   I don't know_____
If yes, when and what treatment did she receive?

_____

_____

31. Has your mother ever been diagnosed and/or treated for any other psychiatric illness?

Yes_____   No_____   I don't know_____
If yes, what illness and what treatment did she receive?

_____

_____

32. To your knowledge, has your mother ever had a drinking problem or abused alcoholic beverages?

Yes_____   No_____   I don't know_____
If yes, when and for how long?

_____

_____

If the answer to the above question is yes, do you know *for sure* if your mother was drinking during her pregnancy with you?

Yes, she drank during her pregnancy_____   No, she did not_____   I don't know_____

33. If the answer to 32 was yes, your mother had a drinking problem, please answer the following:

How old was she when she started drinking? _____ (approximate as close as you can)
Is she currently experiencing a drinking problem? _____
Has she received treatment for this problem? _____
Has it caused medical problems?_____
Has it caused work-related problems?_____
Did it cause marital or family problems?_____
Has she been arrested for DUI?_____

34. Has your mother ever abused drugs (medically or recreationally)?

Yes_____   No_____   I don't know_____
If yes, when and for how long?

_____

_____

35. Has your father ever been diagnosed and/or treated for depression?

    Yes_____   No_____   I don't know_____
    If yes, when and what treatment did he receive?

    _____

    _____

36. Has your father ever been diagnosed and/or treated for anxiety (including generalized anxiety, phobia, post traumatic stress disorder, acute stress disorder, or obsessive-compulsive disorder)?

    Yes_____   No_____   I don't know_____
    If yes, when and what treatment did he receive?

    _____

    _____

37. Has your father ever been diagnosed and/or treated for any other psychiatric illness?

    Yes_____   No_____   I don't know_____
    If yes, what illness and what treatment did he receive?

    _____

    _____

38. To your knowledge, has your father ever had a drinking problem or abused alcoholic beverages?

    Yes_____   No_____   I don't know_____
    If yes, when and for how long?

    _____

    _____

39. If the answer to 38 was yes, your father had a drinking problem, please answer the following:

    How old was he when she started drinking? _____ (approximate as close as you can)

    Is he currently experiencing a drinking problem? _____

    Has he received treatment for this problem? _____

    Has it caused medical problems?_____

    Has it caused work-related problems?_____ _____

    Did it cause marital or family problems?

    Has he been arrested for DUI?

40. Has your father ever abused drugs (medically or recreationally)?

    Yes_____   No_____   I don't know_____

    If yes, when and for how long?

    _____

    _____

98

41. Please circle the highest educational level or grade your mother completed:

    1   2   3   4   5   6   7   8   9   10   11   12   13   14   15   16   Grad+

42. Please circle the highest educational level or grade your father completed:

    1   2   3   4   5   6   7   8   9   10   11   12   13   14   15   16   Grad+

43. To your knowledge, did your mother ever get into trouble with the law?

Yes_____   No_____   I don't know_____
If yes, when (how old was she) and what happened?

_____

_____

44. To your knowledge, did your father ever get into trouble with the law?

Yes_____   No_____   I don't know_____
If yes, when (how old was he) and what happened?

_____

_____

45. Has anyone else in your family ever been diagnosed and/or treated for depression?

Yes_____   No_____   I don't know_____
If yes, when and what treatment did s/he receive?

_____

_____

46. Has any other family member been diagnosed and/or treated for anxiety (including generalized anxiety,
    phobia, post traumatic stress disorder, acute stress disorder, or obsessive-compulsive disorder)?

Yes_____   No_____   I don't know_____
If yes, when and what treatment did s/he receive?

_____

_____

47. Has anyone else in your family ever been diagnosed and/or treated for any other psychiatric illness?

Yes_____   No_____   I don't know_____
If yes, what illness and what treatment did s/he receive?

_____

_____

99

48. To your knowledge, has any other family member had a drinking problem or abused alcoholic beverages?

Yes_____   No_____   I don't know_____
If yes, what relation was (is) it, and when and for how long?

_____

_____

49. If the answer to 48 was yes, this family member had a drinking problem, please answer the following:

How old was s/he when she started drinking? _____ (approximate as close as you can)
Is s/he currently experiencing a drinking problem? _____
Has s/he received treatment for this problem? _____
Has it caused medical problems?_____
Has it caused work-related problems?_____
Has s/he been arrested for DUI?_____

50. Has any other family member ever abused drugs (medically or recreationally)?

Yes_____   No_____   I don't know_____
If yes, when and for how long?

_____

_____

51. Does any family member experience seizures or other neurological problem?

Yes_____   No_____   I don't know_____
If yes, please describe.

_____

_____

REFERENCES

Alterman, A. I., & Hall, J. G. (1989). Effects of social drinking and familial alcoholism risk on cognitive functioning: Null findings. Alcoholism: Clinical and Experimental Research, 13, 799-803.

Alterman, A. I., Searles, J. S., & Hall, J. G. (1989). Failure to find differences in drinking behavior as a function of familial risk for alcoholism: A replication. Journal of Abnormal Psychology, 98, 1-4.

American Psychiatric Association. (1994). Diagnostic and statistical manual of mental disorders (4th ed.). Washington, DC: Author.

Ashby, J. S., Mangine, J. D., & Slaney, R. B. (1995). An investigation of perfectionism in a university sample of adult children of alcoholics. Journal of College Student Development, 36, 452-455.

Ashcroft, M. H. (1994). Human memory and cognition. New York: Harper Collins College Publishers.

Barkley, R. A., Grodzinsky, G., & DuPaul, G. (1992). Frontal lobe functions in attention deficit disorder with and without hyperactivity: A review and research report. Journal of Abnormal Child Psychology, 20, 163-186.

Bates, M. E., & Pandina, R. J. (1992). Familial alcoholism and premorbid cognitive deficit: A failure to replicate subtype differences. Journal of Studies on Alcohol, 53, 320-327.

Beaudoin, C. M., Murray, R. P., Bond, J. J., & Barnes, G. E. (1997). Personality characteristics of depressed or alcoholic adult children of alcoholics. Personality & Individual Differences, 23, 559-567.

Beck, A. T. (1978). Depression Inventory. Philadelphia, PA: Center for Cognitive Therapy.

Begleiter, H., & Porjes, B. (1988). Potential biological markers in individuals at high risk for developing alcoholism. Alcoholism: Clinical and Experimental Research, 12, 488-493.

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

Begleiter, H., Porjesz, B., & Kissin, B. (1982). Brain dysfunction in alcoholics with and without a family history of alcoholism. Alcoholism: Clinical and Experimental Research, 6, 136 (abstract).

Belliveau, J. M., & Stoppard, J. M. (1995). Parental alcohol abuse and gender as predictors of psychopathology in adult children of alcoholics. Addictive Behaviors, 20 (5), 619-625.

Ben-Tovim, D. I., Walker, M. K., & Douras, G. (1993). Failure to demonstrate gender differences in interference to information-processing of body-shape stimuli. Perceptual and Motor Skills, 76, 456-458.

Berkowitz, A., & Perkins, H. W. (1988). Personality characteristics of children of alcoholics. Journal of Consulting and Clinical Psychology, 56, 206-209.

Biederman, J., Munir, K., & Knee, D. (1987). Conduct and oppositional disorder in clinically referred children with attention deficit disorder: A controlled family study. Journal of the American Academy of Child and Adolescent Psychiatry, 26, 724-727.

Bjorklund, D. F., & Harnishfeger, K. K. (1995). The evolution of inhibition mechanisms and their role in human cognition and behavior. In F. N. Dempster & C. J. Brainerd (Eds.), Interference and inhibition in cognition (pp. 142-173). San Diego, CA: Academic Press, Inc.

Bohman, M. (1978). Some genetic aspects of alcoholism and criminality. Archives of General Psychiatry, 35, 269-276.

Boone, K. B. (1999). Neuropsychological assessment of executive functions: Impact of age, education, gender, intellectual level, and vascular status on executive test scores. The human frontal lobes: Functions and disorders (pp. 247-260). New York: Guilford Press.

Broadbent, D. E. (1958). Perception and communication. London: Pergamon Press.

Brown, S. (1988). Treating adult children of alcoholics: A developmental perspective. New York: John Wiley & Sons, Inc.

Bush, S. I., Ballard, M. E., & Fremouw, W. (1995). Attributional style, depressive features, and self-esteem: Adult children of alcoholic and nonalcoholic parents. Journal of Youth and Adolescence, 24, 177-185.

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

Campbell, S. B., Douglas, V. I., & Morgenstern, G. (1971). Cognitive styles in hyperactive children and the effect of methylphenidate. Journal of Child Psychology and Psychiatry, 12, 55-67.

Chafetz, M., Blane, H., & Hill, M. (1971). Children of alcoholics. Journal of Studies on Alcohol, 32, 687-698.

Chassin, L., Curran, P. J., Hussong, A. M., & Colder, C. R. (1996). The relation of parent alcoholism to adolescent substance use: A longitudinal follow-up study. Journal of Abnormal Psychology, 105, 70-80.

Chassin, L., Rogosch, F., & Barrera, M. (1991). Substance use and symptomology among adolescent children of alcoholics. Journal of Abnormal Psychology, 100, 449-463.

Cherry, E. C. (1953). Some experiments on the recognition of speech, with one and with two ears. Journal of the Acoustical Society of America, 25, 975-979.

Clift, S. M., Wilkins, J. C., & Davidson, E. A. (1993). Impulsiveness, venturesomeness and sexual risk-taking among heterosexual GUM clinic attenders. Personality & Individual Differences, 15, 403-410.

Cloninger, C. R. (1987). A systematic method for clinical description and classification of personality variants. Archives of General Psychiatry, 44, 473-588.

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

Cloninger, C. R., Sigvardsson, S., & Bohman, M. (1988) Childhood personality predicts alcohol abuse in young adults. Alcoholism: Clinical and Experimental Research, 12, 494-504.

Cork, M. (1969). The forgotten children. Toronto: Addiction Research Foundation.

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

Dawson, D. A., & Grant, B. F. (1996). Family history of alcoholism and gender: Their combined effects on DSM-IV alcohol dependence and major depression. Journal of Studies on Alcohol, 59, 97-106.

Dawson, D. A., Harford, T. C., & Grant, B. F. (1992). Family history as a predictor of alcohol dependence. Alcoholism: Clinical and Experimental Research, 16, 572-575.

Deutsch, F. A., & Deutsch, D. (1963). Attention: Some theoretical considerations. Psychological Review, 70, 80-90.

Diamond, A. (1988). Abilities and neural mechanisms underlying A not B performance. Child Development, 59, 523-527.

Dickman, S. J. (1990). Functional and dysfunctional impulsivity: Personality and cognitive correlates. Journal of Personality and Social Psychology, 58, 95-102.

Dougherty, D. M., Bjork, J. M., Marsh, D. M., & Moeller, F. G. (2000). A comparison between adults with conduct disorder and normal controls on a Continuous Performance Test: Differences in impulsive response characteristics. Psychological Record, 50, 203-219.

Drejer, K., Theilgaard, A., Teasdale, T. W., Schulsinger, F., & Goodwin, D. W. (1985). A prospective study of young men at high risk for alcoholism:

Neuropsychological assessment. <u>Alcoholism: Clinical and Experimental Research, 9</u>, 498-502.

El-Guelbaly, N., & Orford, D. (1977). The offspring of alcoholics: A critical review. <u>American Journal of Psychiatry, 134</u>, 357-365.

Enns, J. T., & Burack, J. A. (1997). Attention, development, and psychopathology: Bridging disciplines. In J. T. Enns & J. A. Burack (Eds.), <u>Attention, development, and psychopathology</u> (pp. 3-30). New York: The Guilford Press.

Ervin, C. S., Little, R. E., Streissguth, A. P., & Beck, D. E. (1984). Alcoholic fathering and its relation to child's intellectual development: A pilot investigation. <u>Alcoholism: Clinical and Experimental Research, 8</u> (4), 362-365.

Eysenck, H. J., & Eysenck, S. B. G. (1975). <u>Manual for the Eysenck Personality Questionnaire</u>. San Diego, CA: Educational and Industrial Testing Service.

Ferraro, F. R., & Okerlund, M. (1996). Failure to inhibit irrelevant information in non-clinical schizotypal individuals. <u>Journal of Clinical Psychology, 52</u>, 389-394.

Finn, P. R., Earleywine, M., & Pihl, R. O. (1992). Sensation seeking, stress reactivity, and alcohol dampening discriminate the density of a family history of alcoholism. <u>Alcoholism: Clinical and Experimental Research, 16</u>, 585-590.

Finn, P. R., & Pihl, R. O. (1987). Men at high risk for alcoholism: The effect of alcohol on cardiovascular response to unavoidable shock. <u>Journal of Abnormal Psychology, 96</u>, 230-236.

Finn, P. R., & Pihl, R. O. (1988). Risk for alcoholism: A comparison between two different groups of sons of alcoholics on cardiovascular reactivity and sensitivity to alcohol. Alcoholism: Clinical and Experimental Research, 14, 704-712.

Finn, P. R., Zeitouni, N. C., & Pihl, R. O. (1990). Effects of alcohol on psychophysiological reactivity to nonaversive and aversive stimuli in men at high risk for alcoholism. Journal of Abnormal Psychology, 99, 79-85.

Forth, A. E., & Hare, R. D. (1989). The contingent negative variation in psychopaths. Psychophysiology, 26, 676-682.

Fowles, D. C. (1987). Application of a behavioral theory of motivation to the concepts of anxiety and impulsivity. Journal of Research in Personality, 21, 417-435.

Fox, R. (1962). Children in an alcoholic family. In W. C. Bier (Ed.), Problems in addiction: Alcoholism and narcotics. New York: Fordham University Press.

Gabrielli, W. F., & Mednick, S. A. (1983). Intellectual performance in children of alcoholics. The Journal of Nervous and Mental Disease, 171 (7), 444-447.

Gabrielli, W. F., Mednick, S. A., Volavka, J., Pollock, V., Schulsinger, F., & Itil, T. (1982). Electroencephalograms in children of alcoholic fathers. Psychophysiology, 19, 404-407.

Galen, L. W., Hendersen, M. J., & Whitman, R. D. (1997). The utility of novelty seeking, harm avoidance, and expectancy in the prediction of drinking. Addictive Behaviors, 22, 93-106.

Garland, M. A., Parsons, O. A., & Nixon, S. J. (1993). Visual-spatial learning in nonalcoholic young adults with and those without a family history of alcoholism. Journal of Studies of Alcohol, 54, 219-224.

Goodwin, D. W. (1971). Is alcoholism hereditary? A review and critique. Archives of General Psychiatry, 25, 545-549.

Goodwin, D. (1985). Alcoholism and genetics. Archives of General Psychiatry, 42, 171-174.

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

Goodwin, D. W., Schulsinger, F., Knop, J., Mednick, S., & Guze, S. B. (1977). Psychopathology in adopted and nonadopted daughters of alcoholics. Archives of General Psychiatry, 34, 1005-1009.

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

Gorenstein, E. E., & Newman, J. P. (1980). Disinhibitory psychopathology: A new perspective and a model for research. Psychological Review, 87, 801-315.

Gove, P. B., et al (Eds.). (1965). Webster's seventh new collegiate dictionary. Springfield, MA: G. & C. Merriam Company.

Gow, L., & Ward, J. (1982). The Porteus Maze Test in the measurement of reflection-impulsivity. Perceptual and Motor Skills, 54, 1043-1052.

Harden, P. W., & Pihl, R. O. (1995). Cognitive function, cardiovascular reactivity, and behavior in boys at high risk for alcoholism. Journal of Abnormal Psychology, 104, 94-103.

Hardwick, C. J., Hansen, N. D., & Bairnsfather, L. (1995). Are adult children of alcoholics unique? A study of object relations and reality testing. The International Journal of the Addictions, 30 (5), 525-539.

Harnishfeger, K. K. (1995). The development of cognitive inhibition: Theories, definitions, and research evidence. In F. N. Dempster & C. J. Brainerd (Eds.), Interference and inhibition in cognition (pp. 175-204). San Diego, CA: Academic Press, Inc.

Harnishfeger, K. K., & Bjorklund, D. F. (1994). A developmental perspective on individual differences In inhibition. Learning and Individual Differences, 6, 331-355.

Hawkins, J. D., Catalano, R. F., & Miller, J. Y. (1992). Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: Implications for substance use prevention. Psychological Bulletin, 112, 64-105.

Hechtman, L., Weiss, G., & Perlman, T. (1984). Hyperactives as young adults: Past and current substance abuse and antisocial behavior. American Journal of Orthopsychiatry, 54, 415-425.

Hegedus, A. M., Tartar, R. E., Hill, S. Y., Jacob, T., & Winsten, N. E. (1984). Static ataxia: A possible marker for alcoholism. Alcoholism: Clinical and Experimental Research, 8 (6), 580-528.

Helzer, J. E., & Pryzbeck, T. R. (1988). The co-occurrence of alcoholism with other psychiatric disorders in the general population and its impact on treatment. Journal of Studies on Alcohol, 49, 219-224.

Hesselbrock, V. M., Stabenau, J. R., & Hesselbrock, M. N. (1985). Minimal brain dysfunction and neuropsychological test performance in offspring of alcoholics. In M. Galanter (Ed.), Recent Developments In Alcoholism, Vol. 3 (pp. 65-82). New York: Plenum Press.

Hill, S. Y., Steinhauer, S., Lowers, L., & Locke, J. (1995). Eight-year longitudinal follow-up of P300 and clinical outcome in children from high-risk for alcoholism families. Biological Psychiatry, 37, 823-827.

Holguin, S. R., Corral, M., & Cadaveira, F. (1998). Visual and auditory event-related potentials in young children of alcoholics from high- and low-density families. Alcoholism: Clinical and Experimental Research, 22, 87-96.

Hummel-Schluger, A. O., & Baer, J. S. (1996). A computer-controlled administration of the Matching Familiar Figures Test. Behavior Research Methods, Instruments, & Computers, 28, 93-95.

Jackson, J. K. (1954). The adjustment of the family to the crisis of alcoholism. Quarterly Journal of Studies on Alcohol, 15, 562-586.

Jackson, C. P., & Matthews, G. (1988). The prediction of habitual alcohol use from alcohol related expectancies and personality. Alcohol & Alcoholism, 23, 305-314.

Jacob, T., & Leonard, K. (1986). Psychosocial functioning in children of alcoholic fathers, depressed fathers and control fathers. Journal of Studies on Alcohol, 47, 373-380.

Johnson, J. L. (1990). Forgotten no longer: An overview of research on children of chemically dependent parents. In T. M. Rivinus (Ed.), Children of chemically dependent parents (pp. 29-54). New York: Brunner/Mazel Publishers.

Jones, D. J., & Zalewski, C. (1994). Shame and depression proneness among female adult children of alcoholics. International Journal of the Addictions, 29, 1601-1609.

Jones, J. W. (1983). The Children of Alcoholics Screening Test. Chicago, IL: Camelot Unlimited.

Jones, F. W., & Holmes, D. S. (1976). Alcoholism, alpha production and biofeedback. Journal of Consulting and Clinical Psychology, 44, 224-228.

Jones, M. C. (1968). Personality correlates and antecedents of drinking patterns in adult males. Journal of Consulting and clinical Psychology, 32, 2-12.

Kagan, J., Rosman, B. L., Day, D., Albert, J., & Phillips, W. (1964). Information processing In the child: Significance of analytic and reflective attitudes. Psychological Monographs, 78 (1, Whole No. 578).

Khavari, K. A., & Farber, P. D. (1978). A profile instrument for the quantification and assessment of alcohol consumption: The Khavari Alcohol Test. Journal of Studies on Alcohol, 9, 1525-1538.

112

Knop, J., Teasdale, T. W., Schulsinger, F., & Goodwin, D. W. (1985). A prospective study of young men at high risk for alcoholism: School behavior and achievement. Journal of Studies on Alcohol, 46, 273-278.

La Grange, L., Jones, T. D., Erb, L, & Reyes, E. (1995). Alcohol consumption: Biochemical and personality correlates in a college student population. Addictive Behaviors, 20, 93-103.

Linn, M. C., & Petersen, A. C. (1985). Emergence and characterization of sex difference in spatial ability: A meta-analysis. Child Development, 56, 1479-1498

Lipscomb, T., Carpenter, J., & Nathan, P. (1979). Static ataxia: A predictor of alcoholism. British Journal of Addiction, 74, 289-294.

Logan, G. D. (1994). On the ability to inhibit thought and action: A users' guide to the stop signal paradigm. In D. Dagenbach & T. H. Carr (Eds.), Inhibitory processes in attention, memory, and language (pp. 189-239). San Diego, CA: Academic Press.

Matier-Sharma, K., Perachio, N., Newcorn, J. H., Sharma, V., & Halperin, J. M. (1995). Differential diagnosis of ADHD: Are objective measures of attention, impulsivity, and activity level helpful? Child Neuropsychology, 1, 118-127.

McCampbell, E., & Defilippis, N. A. (1979). The development of a booklet form of the Category Test: A preliminary report. International Journal of Clinical Neuropsychology, 1, 33-35.

McGue, M., Pickens, R. W., & Svikis, D. S. (1992). Sex and age effects on the inheritance of alcohol problems: A twin study. Journal of Abnormal Psychology, 101, 3-17.

Mekarski, J. E., Cutmore, T. R. H., & Suboski, W. (1996). Gender differences during processing of the Stroop task. Perceptual and Motor Skills, 83, 563-568.

Messer, S. B. (1976). Reflection-impulsivity: A review. Psychological Bulletin, 83, 1026-1052.

Meszaros, E. L., Hornik, K., Fureder, T., Willinger, U., Fischer, G., Schonbeck, G., & Aschauer, H. N. (1999). The Tridimensional Personality Questionnaire as a predictor of relapse in detoxified alcohol dependents. Alcoholism: Clinical and Experimental Research, 23, 483-486.

Meyer, D. E., & Schvaneveldt, R. W. (1971). Facilitation in recognizing pairs of words: Evidence of a dependence between retrieval operations. Journal of Experimental Psychology, 90, 227-234.

Mohs, R. C., Tinklenberg, J. R., Roth, W. T., & Kopell, B. S. (1978). Slowing of short-term memory in alcoholics. Journal of Studies on Alcohol, 39, 1908-1915.

Moray, N. (1959). Attention in dichotic listening: Affective cues and the influence of instructions. Quarterly Journal of Experimental Psychology, 11, 56-60.

Newman, J. P. (1987). Reaction to punishment in extraverts and psychopaths: Implications for the impulsive behavior of disinhibited individuals. Journal of Research on Personality, 21, 464-480.

Newman, J. P., Wallace, J. F., Schmitt, W. A., & Arnett, P. A. (1997). Behavioral inhibition system functioning in anxious, impulsive and psychopathic individuals. Personality and Individual Differences, 17, 583-592.

Pandina, R. J., & Johnson, V. (1990). Serious alcohol and drug problems among adolescents with a family history of alcoholism. Journal of Studies on Alcohol, 51, 278-282.

Parker, J. D., & Bagby, R. M. (1997). Impulsivity in adults: A critical review of measurement approaches. In C. D. Webster & M. A. Jackson (Eds.), Impulsivity: Theory, assessment, and treatment (pp. 142-157). New York: The Guilford Press.

Parrott, A. C.; Winder, G. (1989). Nicotine chewing gum (2 mg, 4 mg) and cigarette smoking: Comparative effects upon vigilance and heart rate. Psychopharmacology, 97, 257-261.

Pascualvaca, D. M., Anthony, B. J., Arnold, L. E., Rebok, G. W., Ahearn, M. B., Kellam, S. G., & Mirsky, A. F. (1997). Attention performance in an epidemiological sample of urban children: The role of gender and verbal intelligence. Child Neuropsychology, 3, 13-27.

Pashler, H. E. (1998) The psychology of attention. Cambridge, MA: The Massachusetts Institute of Technology Press.

Pelham, W. E., & Lang, A. R. (1993). Parental alcohol consumption and deviant child behavior: Laboratory studies of reciprocal effects. Clinical Psychology Review, 13, 763-784.

Peterson, J. B., Finn, P. R., & Pihl, R. O. (1992). Cognitive dysfunction and the inherited predisposition to alcoholism. Journal of Studies on Alcohol, 53, 154-160.

115

Pickens, R. W., Svikis, D. S., McGue, M., Lykken, D. T., Heston, L. L., & Clayton, P. J. (1991). Heterogeneity in the inheritance of alcoholism: A study of male and female twins. Archives of General Psychiatry, 48, 19-28.

Pihl, R. O., Peterson, J., & Finn, P. R. (1990). Inherited predisposition to alcoholism: Characteristics of sons of male alcoholics. Journal of Abnormal Psychology, 99, 291-301.

Pollock, V. E., Schneider, L. S., Gabrielli, W. F., & Goodwin, D. W. (1987). Sex of parent and offspring in the transmission of alcoholism: A meta-analysis. Journal of Nervous and Mental Disease, 173, 668-673.

Porjesz, B., & Begleiter, H. (1981). Human evoked brain potentials and alcohol. Alcoholism: Clinical and Experimental Research, 5, 304-317.

Posner, M. I. (1980). Orienting of attention. Quarterly Journal of Experimental Psychology, 32, 3-25.

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

Ramsey, S. E., & Finn, P. R. (1997). P300 men with a family history of alcoholism under different incentive conditions. Journal of Studies on Alcohol, 58, 606-616.

Rapoport, J., Quinn, P. O., Bradbard, G., Riddle, D., & Brooks, E. (1974). Imipramine and methylphenidate treatments of hyperactive boys. Archives of General Psychiatry, 30, 789-793.

Reed, R., Grant, I., & Adams, K. M. (1987). Family history of alcoholism does not predict neuropsychological performance in alcoholics. Alcoholism: Clinical and Experimental Research, 11, 340-344.

Rice, G. E., & Meyer, B. J. F. (1986). Prose recall: Effects of aging, verbal ability and reading behavior. Journal of Gerontology, 41, 469-480.

Roe, A., & Burks, B. (1945). Adult adjustment of foster children of alcoholics and psychotic parentage and the influence of the foster home. In Memoirs of the section on alcoholism studies, No. 3. New Haven University Press.

Roosa, M. W., Sandler, I. N., Gehring, M., Beals, J., & Cappo, L. (1988). The Children of Alcoholics Life-Events Schedule: A stress scale for children of alcohol-abusing parents. Journal of Studies on Alcohol, 49, 422-429.

Rydelius, P. A. (1983). Alcohol abusing teenage boys: Testing a hypothesis on the relationship between alcohol abuse and social background factors, criminality and personality in teenage boys. Acta Psychiatrica Scandinavica, 68, 368-380.

Salkind, N. J., & Wright, J. C. (1977). The development of reflection-impulsivity and cognitive efficiency. Human Development, 20, 377-387.

Samson, P. M. (1994). Differences in cognitive functioning among adult children of alcoholics and non-alcoholics. Unpublished, Masters Thesis, University of North Dakota, Grand Forks, ND.

Sattler, J. M. (1992). Assessment of children - Revised and Updated Third Edition. San Diego: Jerome M. Sattler, Publisher, Inc.

Schachar, R. J., & Logan, G. D. (1990a). Impulsivity and inhibitory control in normal development and childhood psychopathology. Developmental Psychology, 26, 710-720.

Schachar, R. J., & Logan, G. D. (1990b). Are hyperactive children deficient in attentional capacity? Journal of Abnormal Child Psychology, 18, 493-513.

Schachar, R. J., Tannock, R., & Logan, G. D. (1993). Inhibitory control, impulsiveness, and attention deficit hyperactivity disorder. Clinical Psychology Review, 13, 721-739.

Schaeffer, K. W., Parsons, O. A., & Yohman, J. R. (1984). Neuropsychological differences between male familial and nonfamilial alcholics and nonalcoholics. Alcoholism: Clinical and Experimental Research, 8, 347-351.

Schandler, S. L., Brannock, J. C., Cohen, M. J., Antick, J., & Caine, K. (1988). Visuospatial learning in elementary school children with and without a family history of alcoholism. Journal of Studies on Alcohol, 49, 538-545.

Schandler, S. L., Brannock, J. C., Cohen, M. J., & Mendez, J. (1993). Spatial learning deficits in adolescent children of alcoholics. Experimental and Clinical Psychopharmacology, 1, 207-214.

Schandler, S. L., Cohen, M. J., & Antick, J. R. (1992). Activation, attention, and visuospatial learning in adults with and without a family history of alcoholism. Alcoholism: Clinical and Experimental Research, 16, 566-571.

118

Schandler, S. L., Cohen, M. J., McArthur, D. L., Antick, J. R., & Brannock, J. C. (1991). Spatial learning deficits in adult children of alcoholic parents. Journal of Consulting and Clinical Psychology, 59, 312-317.

Schuckit, M. A. (1985). Ethanol induced changes in body sway in men at high alcoholism risk. Archives of General Psychiatry, 42, 375-379.

Schuckit, M. A. (1986). Alcoholism and affective disorders: Genetic and clinical implications. American Journal of Psychiatry, 143, 140-147.

Schuckit, M. A. (1987). Biological vulnerability to alcoholism. Journal of Clinical and Consulting Psychology, 55, 301-309.

Schuckit, M. A. (1994a). A clinical model of genetic influences in alcohol dependence. Journal of Studies on Alcohol, 55, 5-17.

Schuckit, M. A. (1994b). Low level of response to alcohol as a predictor of future alcoholism. American Journal of Psychiatry, 151, 184-189.

Schuckit, M. A., Butters, N., Lyn, L., & Irwin, M. (1987). Neuropsychologic deficits and the risk for alcoholism. Neuropsychopharmacology, 1, 45-53.

Schuckit, M. A., Goodwin, D. W., & Winokur, G. (1972). A study of alcoholism in half siblings. American Journal of Psychiatry, 128, 1132-1136.

Schuckit, M. A., & Smith, T. L. (1996). An 8-year follow-up of 450 sons of alcoholic and control subjects. Archives of General Psychiatry, 53, 202-210.

Schuckit, M. A., & Smith, T. L. (1997). Assessing the risk for alcoholism among sons of alcoholics. Journal of Studies on Alcohol, 58, 141-145.

Schuckit, M. A., & Sweeney, S. (1987). Substance use and mental health problems among sons of alcoholics and controls. Journal of Studies of Alcohol, 48, 528-534.

Schulsinger, F., Knop, J., Goodwin, D. W., Teasdale, T. W., & Middelson, U. (1986). A prospective study of young men at high risk for alcoholism. Archives of General Psychiatry, 43, 755-760.

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

Seidel, W. T., & Joschko, M. (1990). Evidence of difficulties in sustained attention in children with ADDH. Journal of Abnormal Child Psychology, 18, 217-229.

Seidman, L. J., Biederman, J., Faraone, S. V., Milberger, S., Norman, D., Seiverd, K., Benedict, K., Guite, J., Mick, E., & Kiely, K. (1995). Effects of family history and comorbidity on the neuropsychological performance of children with ADHD: Preliminary findings. Journal of the American Academy of Child and Adolescent Psychiatry, 34, 1015-1024.

Selzer, M. L. (1971). The Michigan Alcoholism Screening Test: A quest for a new diagnostic instrument. American Journal of Psychiatry, 127 (12), 1653-1658.

Sher, K. J., Walitzer, K. S., Wood, P. K., & Brent, E. E. (1991). Characteristics of children of alcoholics: Putative risk factors, substance use and abuse, and psychopathology. Journal of Abnormal Psychology, 100 (4), 427-448.

Shinn, M. (1978). Father absences and children's cognitive development. Psychological Bulletin, 85, 295-324.

120

Silvia, L. Y., & Liepman, M. R. (1991). Biological aspects. In T. M. Rivinus (Ed.), Children of chemically dependent parents (pp. 1-28). New York: Brunner/Mazel Publishers.

Spielberger, C. D., Gorsuch, R. L., & Lushene, R. (1967). The State-Trait Anxiety Inventory. Palo Alto, CA: Consulting Psychologists Press.

St. James, J., Schneider, W., & Rodgers, K. A. (1994). Mel Lab: Experiments in perception, cognition, social psychology & human factors. Pittsburgh, PA: Psychology Software Tools Inc., Publisher.

Steinglass, P., Bennett, L. A., Wolin, S. J., & Reiss, D. (1987). The Alcoholic Family. New York: Basic Books, Inc.

Steinhauer, S. R., & Hill, S. Y. (1993). Auditory event-related potentials in children at high risk for alcoholism. Journal of Studies on Alcohol, 54, 408-421.

Strickland, T. L., D'Elia, L. F., James, R., & Stein, R. (1997). Stroop color-word performance of African-Americans. Clinical Neuropsychologist, 11, 87-90.

Stroop, J. R. (1935). Studies of interference In serial verbal reactions. Journal of Experimental Psychology, 18, 643-662.

Tartar, R. E. (1988). Are there inherited behavioral traits that predispose to substance abuse? Journal of Consulting and Clinical Psychology, 56, 189-196.

Tartar, R. E., Hegedus, A. M., Goldstein, G., Shelly, C., & Alterman, A. I. (1984). Adolescent sons of alcoholics: Neuropsychological and personality characteristics. Alcoholism: Clinical and Experimental Research, 8, 216-222.

Tartar, R. E., Jacob, T., & Bremer, D. A. (1989). Cognitive status of sons of alcoholic men. Alcoholism: Clinical and Experimental Research, 13 (2), 232-235.

Triesman, A. M. (1960). Contextual cues in selective listening. Quarterly Journal of Experimental Psychology, 12, 242-248.

Turner, W. M., Cutter, H. S. G., Worobec, T. G., O'Farrell, T. J., Bayog, R. D., & Tsuang, M. T. (1993). Family history models of alcoholism: Age of onset, consequences and dependency. Journal of Studies on Alcohol, 54, 164-171.

Vaillant, G. E. (1983). Natural history of male alcoholism. V: Is alcoholism the cart or the horse to sociopathy? British Journal of Addiction, 78, 317-326.

Vanderplas, J. M., & Garvin, E. A. (1959). The association value of random shapes. Journal of Experimental Psychology, 57, 147-153.

Van Der Stelt, O., Geesken, R., Gunning, W. B., Snel, J., & Kok, A. (1998). P3 scalp topography to target and novel visual stimuli in children of alcoholics. Alcohol, 15, 119-136.

Virkkunen, M., & Linnoila, M. (1993). Brain serotonin, Type II alcoholism, and impulsive violence. Journal of Studies on Alcohol, Suppl. 11, 163-169.

Visser, M., Das-Smaal, E., & Kwakman, H. (1996). Impulsivity and negative priming: Evidence for diminished cognitive inhibition in impulsive children. British Journal of Psychology, 87, 131-140.

Wahler, H. J. (1983). Wahler Physical Symptoms Inventory. Los Angeles, CA: Western Psychological Services.

Wechsler, D. (1945). A standardized memory scale for clinical use. Journal of Psychology, 19, 8795.

Wechsler, D. (1981). Wechsler Adult Intelligence Scale-Revised. New York: The Psychological Corporation.

Weller, L. A. (1997). Prose recall differences between a group of adult children of alcoholics and a control group. Unpublished, Masters Thesis, University of North Dakota, Grand Forks, ND.

Werner, E. E. (1986). Resilient offspring of alcoholics: A longitudinal study from birth to age 18. Journal of Studies on Alcohol, 47, 34-40.

Wesnes, K., & Revell, A. (1984). The separate and combined effects of scopolamine and nicotine on human information processing. Psychopharmacology, 84, 5-11.

West, M. O., & Prinz, R. J. (1987). Parental alcoholism and childhood psychopathology. Psychological Bulletin, 102, 204-218.

Weyers, P., Krebs, H., & Janke, W. (1995). Reliability and construct validity of the German version of Cloninger's Tridimensional Personality Questionnaire. Personality & Individual Differences, 19, 853-861.

Whipple, S. C., Parker, E. S., & Noble, E. P. (1988). An atypical neurocognitive profile in alcoholic fathers and their sons. Journal of Studies on Alcohol, 49, 240-244.

Willner, A. E. (1970). Towards development of more sensitive clinical tests of abstraction: The Analogy Test. Proceedings of the 78th Annual convention of the American Psychological Association, 5, 553-554.

Wilsnack, S. C., Vogeltanz, N. D., Klassen, A. D., & Harris, T. R. (1994). Childhood sexual abuse and women's substance abuse: National survey findings. Paper presented at the American Psychological Association Conference on Psychosocial and Behavioral Factors in Women's Health, Washington, D.C.

Wilson, J. R., & Nagoshi, C. T. (1988). Adult children of alcoholics: Cognitive and psychomotor characteristics. British Journal of Addiction, 83, 809-820.

Windle, M., Windle, R. C., Scheidt, D. M., & Miller, G. B. (1995). Physical and sexual abuse and associated mental disorders among alcoholic inpatients. American Journal of Psychiatry, 152, 1322-1328.

Winokur, G., & Coryell, W. (1991). Familial alcoholism in primary unipolar major depressive disorder. American Journal of Psychiatry, 148, 184-188.

Winokur, G., Reich, T., Rimmer, J., & Pitts, F. N. (1970). Alcoholism III: Diagnosis and family psychiatric illness. Archives of General Psychiatry, 23, 104-111.

Winokur, G., Rimmer, J., & Reich, T. (1971). Alcoholism IV: Is there more than one type of alcoholism? British Journal of Psychiatry, 118, 525-531.

Wolin, S. J., Bennett, L. A., Noonan, D. L., & Teitelbaum, M. A. (1980). Disrupted family rituals: A factor in the intergenerational transmission of alcoholism. Journal of Studies on Alcohol, 41, 199-214.

Woodside, M. (1983). Children of alcoholic parents: Inherited and psycho-social influences. Journal of Psychiatric Treatment and Evaluation, 5, 531-537.

Workman-Daniels, K. L., & Hesselbrock, V. M. (1987). Childhood problem behavior and neuropsychological functioning in persons at risk for alcoholism. Journal of Studies on Alcohol, 48, 187-193.

Zaparniuk, J., & Taylor, S. (1997). Impulsivity in children and adolescents. In C. D. Webster & M. A. Jackson (Eds.), Impulsivity: Theory, assessment, and treatment, (pp. 158-179). New York: The Guilford Press.

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

Zuckerman, M. (1993). P-impulsive sensation seeking and its behavioral, psychophysiological biochemical correlates. Neuropsychobiology, 28, 30-36.