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COGNITIVE FUNCTIONING OF SONS OF ALCOHOLICS DURING EARLY ELEMENTARY SCHOOL YEARS AS RELATED TO SUBTYPES OF FAMILIAL ALCOHOLISM

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Edwin Poon

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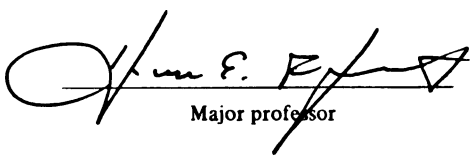

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By

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ABSTRACT

COGNITIVE FUNCTIONING OF SONS OF ALCOHOLICS DURING EARLY ELEMENTARY SCHOOL YEARS AS RELATED TO SUBTYPES OF FAMILIAL ALCOHOLISM

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This study examined the relationship between cognitive functioning and paternal alcoholism subtype in early school-aged children of alcoholics. Results showed that children of antisocial alcoholics (AALs) displayed poorer general cognitive functioning and academic achievement than children of non-antisocial alcoholics (NAALs) and controls. In addition, children of AALs exhibited relative deficits in verbal processing abilities; no such difference was found between children of NAALs and controls. The fact that children of AALs had the poorest abstract planning abilities and highest level of impulsivity suggests that these children may have specific deficits in frontal lobe functioning, which may also put them at greater risk for developing later peer and behavioral problems. It appears that elementary school aged children of AALs may already be launched into a trajectory which places them at heightened risk for deviant behaviors in later life, including problematic alcohol use.

To mom, dad, and Uncle K.K.

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INTRODUCTION

In the 1992 National Household Survey on Drug Abuse, over 80 percent of men between the ages of 18 and 25 reported some consumption of alcohol in the past year (Substance Abuse and Mental Health Services Administration, 1993). Moreover, one in four men in the United States will meet the criteria for alcohol abuse/dependence sometime in the course of their lifetime (Zucker, Fitzgerald, & Moses, 1995). These statistics indicate that alcoholism is one of the major health problems in this country. In order to understand the development of alcoholism, much of the research has focused on identifying factors that increase the risk for problem drinking.

Alcoholism tends to run in families (Dawson, Harford, & Grant, 1992). For example, Beardslee, Son, and Vaillant (1986) found that parental alcoholism was highly correlated with alcohol use and alcoholism among children of alcoholics (COAs). However, prevalence rates suggest that only a portion of these children will actually become alcoholics. What are some of the factors that increase the likelihood for an alcoholism outcome for these children? Research has shown that environmental, genetic, and behavioral factors contribute to the risk for developing alcoholism (Sher, 1991; Zucker & Fitzgerald, 1991; Fitzgerald, Sullivan, Ham, Zucker, Bruckel, & Schneider, 1993). Moreover, recent literature on COAs suggests that cognitive dysfunction may play an important role in the development of alcoholism (West & Prinz, 1987; Sher, Walitzer, Wood, & Brent, 1991; Noll,

Zucker, Fitzgerald, & Curtis, 1992). For instance, several investigators have already found that COAs exhibit poorer cognitive performance when compared with non-COAs (Noll & Zucker, 1983; Goodwin, 1983; Tarter, Hegedus, Goldstein, Shelly, & Alterman, 1984).

However, some studies do not support the notion that COAs have poorer cognitive abilities than non-COAs (Reed, Grant, & Adams, 1987; Schuckit, Butters, Lyn, & Irwin, 1987; Johnson & Rolf, 1988; Alterman, Searles, & Hall, 1989). Such inconsistent findings may stem from the substantial heterogeneity of alcoholic families. Research has shown that variation among alcoholic families such as co-occurrence of parental psychopathology and high density of family alcoholism (multigenerational alcoholism) affect the vulnerability of COAs (Chassin, Rogosch, & Barrera, 1991; Ellis, Bingham, Zucker, & Fitzgerald, 1997; Finn, Sharkansky, Viken, West, Sandy, & Bufferd, 1997). For example, Ellis, Bingham, Zucker, and Fitzgerald (1997) reported that preschool age children with antisocial alcoholic fathers differ from children of non-antisocial alcoholic and non-alcoholic fathers on a number of indicators of child risk, including measures of risky temperament, externalizing behavior problems, and hyperactivity. Moreover, only a few studies have investigated the relationship between alcoholic family subtype and the cognitive functioning of COAs (Tarter, Jacob, Bremer, 1989a; Pihl, Peterson, Finn, 1990; Harden & Pihl, 1995).

This study examined the relationship between cognitive functioning and paternal alcoholism in a sample of 198 male COAs and non-COAs. Two subtypes

of paternal alcoholism were investigated: antisocial alcoholism and non-antisocial alcoholism. Each was compared with a non-alcoholic control group. In particular, the study investigated whether or not the cognitive abilities of male COAs differed as a function of paternal alcoholism subtype, with children of antisocial alcoholics performing worst. In addition, it examined the relationship between cognitive functioning performance and impulsivity.

REVIEW OF THE LITERATURE

Alcoholism and the Brain

For many years, chronic alcoholics were known to be at risk for one specific kind of neurological impairment; namely, the Wernicke-Korsakoff Syndrome (WKS). This syndrome is believed to be caused by thiamin deficiency, a result of malnutrition due to chronic alcohol use. Neurological damage from WKS occurs in the diencephalic region of the brain including the dorsomedial thalamus and the mamillary bodies (Victor, Adams, & Collins, 1971; Charness and DeLaPaz, 1987; Jacobson, & Lishman, 1990). In addition to brain lesions, WKS is often accompanied by cognitive impairment. In general, chronic alcoholics with WKS exhibit anterograde and retrograde memory deficits, while their intellectual skills, as measured by intelligence tests, are mostly preserved (Charness, 1993; Bolden, 1994). Furthermore, studies have found that alcoholics with WKS display perceptual (Oscar-Berman, 1980), conceptual (Kovner, Mattis, Goldmeier, & Davis, 1981) and psychomotor difficulties (Parsons & Nixon, 1993).

In a landmark paper, Courville (1955) reported that chronic alcoholics exhibited widespread cortical atrophy and he argued that the damage results from alcohol neurotoxicity rather than dietary deficiency. Since then, researchers have reported that alcoholics are susceptible to various other neurological disorders including cerebellar degeneration, heptocerebral degeneration, Marchiafava-

Bignami syndrome, and central pontine myelinolysis (Tuck & Jackson, 1991; Charness, 1993). Neuropathological and brain imaging studies have shown that alcoholics without WKS suffer diffuse cortical atrophy as well as ventricular enlargement (Courville, 1955; Von-Cramon, Hebel, & Schuri, 1985; Lishman, 1990; Pfefferbaum, Sullivan, Mathalon, & Lim, 1997).

Consistent with neuroimaging studies, neuropsychological studies provide evidence of cognitive impairment in alcoholics without WKS. Using the Wechsler Adult Intelligence Scale (WAIS), researchers have found that alcoholics with no signs of WKS showed deficits on Performance subtests which assess visuo-motor and visuo-spatial skills (Parsons & Leber, 1981; Goldman, 1986). For instance, Loberg (1980) evaluated WAIS subtest scores attained by male alcoholics and normal drinkers and found that alcoholics obtained significantly lower scores than non-alcoholics on several performance subtests including Block Design and Digit Symbol. Conversely, their verbal abilities remained mostly intact (Parsons & Farr, 1981).

The relationship between cognitive impairment and alcoholism was further substantiated by studies that found alcoholic inpatients performed poorly on the Halstead-Reitan Battery, a neuropsychological test that is sensitive to brain damage (Fitzhugh, Fitzhugh, & Reitan, 1965; Jones & Parsons, 1971; Smith, Burt, & Chapman, 1973). Recently, Tamkin and Dolenz (1990) studied the performance of 104 alcoholic inpatients in a VA hospital on several neuropsychological tests including the Weigl Color-Form Sorting Test,

Vocabulary, Similarities, and Digit Symbol subtests of the WAIS, and Trails A and B of the Halstead-Reitan Battery. As predicted, the results showed that alcoholics performed worse on most cognitive tests (Trials A and B, and all WAIS subtests) as compared to non-alcoholics.

Not all alcoholics who exhibit cognitive dysfunction show signs of neurological disorder. In a recent study, Tuck and Jackson (1991) examined the relationship between neurological disorders and cognitive dysfunction in alcoholics. Alcoholics with no neurological disorder were found to be significantly younger than those with neurological dysfunction. Moreover, neuropsychological testing revealed that the former group performed significantly worse on tests of frontal lobe function than matched controls. Results suggested that alcoholics may suffer cognitive impairment before the onset of any clinical signs of neurological disorder.

In summary, the literature indicates that chronic alcoholics suffer a wide range of neurological disorders resulting in brain lesions. Moreover, alcoholics with no clinical signs of WKS or other neurological disease exhibit various cognitive deficits that may precede brain damage measurable via imaging studies.

Cognitive Deficits as a Consequence of Alcohol Abuse

Prenatal exposure to high levels of alcohol is known to produce cognitive impairment in children. For instance, children whose mothers drank alcohol excessively during their pregnancies often were mentally retarded (Streissguth, Herman, & Smith; 1978, Smith and Eckhardt, 1991), had slower information

processing ability (Jacobson, Jacobson, Sokol, & Martier, 1993), and impaired visual-motor processing ability (Janzen, Nanson, & Block, 1995). Moreover, some investigators have suggested that alcohol can also induce cognitive impairment in adults.

On an acute level, alcohol intoxication has been shown to have a negative effect on cognitive performance (Golby, 1989; Miller, 1992). In one study, Peterson, Rothfleisch, Zelazo, and Pihl (1990) examined the hypothesis that acute alcohol intoxication will produce cognitive change that is similar to the neuropsychological impairment suffered by individuals with prefrontal damage. Seventy-two moderate social drinkers were tested on tasks associated with frontal cortex (e.g. Porteus Maze Test), temporal cortex (e.g. Logical Memory of the WMS-R), and parietal-occipital cortex (e.g. Albert's Simple Test of Visual Neglect) after they received one of three different doses of alcohol: high (1.32 ml/kg), medium (0.66 ml/kg), and low (0.132 ml/kg). The results indicated that a high dose of alcohol significantly impaired such cognitive functions as planning, verbal fluency, memory, and complex motor control.

In cases of chronic alcohol abuse/dependence, three different models have been proposed to describe the specific action of alcohol on the workings of the brain. First, the generalized/diffuse model suggests that alcohol abuse/dependence may cause non-specific neurological damage (Parsons & Leber, 1981). Early evidence for this model has come from the results of neuropathological studies that showed alcoholics suffer diffuse brain damage (Courville, 1955; Mancall,

1961). For instance, Lynch (1960) examined the brains of eleven chronic alcoholics at *post mortem* and found that 20 to 40% of the cortical cells were lost. However, since the majority of the subjects in these studies were elderly, aging may have contributed to the neuronal damage observed in some of these studies. Subsequently, Goldstein and Shelly (1982) argued that neuropsychological impairments exhibited in alcoholics resemble the deficits found in patients with nonalcoholic diffuse brain damage rather than the deficits found in patients with frontal lobe damage. Despite the significant findings, Goldstein (1987) warned that the generalized/diffuse model is not sufficient to explain the pattern of neuropsychological deficits found among all alcoholics. He further suggested that genetic and antecedent cognitive functioning might play a role in the cognitive functioning of different alcoholic subtypes.

The second model proposes that right-brain functions are more susceptible to impairment as a result of alcohol abuse/dependence than are left-brain functions (Leber, Jenkins, and Parsons, 1981; Berglund, Hagstadius, Risberg, Johanson, Bliding, & Mubrin, 1987). Early evidence for this model has come from neuropsychological studies that showed alcoholics performed much worse on tasks that are mediated by the right hemisphere. For example, Chandler and Parsons (1977) found that acute alcohol intoxication impaired recognition and memory performance when material was presented to the left visual field, while performance was equal to that of controls when material was presented to the right visual field. However, more recent studies have failed to validate this model (Ellis

& Oscar-Berman, 1985; Oscar-Berman & Weinstein, 1985). In one study, Akshoomoff, Delis, and Kiefner (1989) administered the Block Design subtest of the WAIS-R to four groups of male subjects; detoxified chronic alcoholics, right hemisphere damaged patients, left hemisphere damaged patients, and normal controls. Analyses of block construction strategies and errors revealed that alcoholics did not suffer the type of visuo-spatial impairment that was seen in right hemisphere damaged subjects. Moreover, their strategies and errors fell between the left and right hemisphere damaged patients suggesting that both hemispheres might be damaged as a result of chronic alcohol abuse.

Finally, the executive function deficit model suggests that chronic alcoholism results in cognitive impairment that is associated with brain functions of the anterior-basal region including the frontal and diencephalic structures (Tarter, 1975). According to Ron (1977), autopsy reports of chronic alcoholics have shown that the frontal brain region is more susceptible to damage from alcohol and the damage is often more severe as compared to the rest of the brain. Subsequent neuropathological studies have also found that chronic alcoholic patients suffer neuronal loss in the frontal cortex (Harper & Kril, 1985; Harper & Kril, 1989; Jacobson & Lishman, 1990).

Results of neuropsychological investigations on alcoholics further support the executive function deficit model (Bergman, 1987; Gebhardt, Naeser, & Butters, 1984; Ron, 1987). For example, Steingass, Sartory, and Canavan (1994) assessed the performance of chronic alcoholics on cognitive tests that measure

orientation, premorbid and current intelligence, organicity, attention, learning, and ability to form categories. The results showed that alcoholics exhibited perseveration and impaired ability to identify semantic categories, both of which are associated with frontal lobe dysfunction. Similarly, Gilman et al. (1997) examined the neuropsychological functioning of chronic alcoholic patients and found that they performed poorly on the Halstead Impairment Index, Halstead Category Test, and Wisconsin Card Sort Test, all of which are known to be sensitive to frontal lobe pathology. In addition, these researchers showed that the neuropsychological performance of alcoholics was correlated with the metabolic abnormality found in the frontal region of the cerebral cortex.

Many studies have used the Mazes test to measure frontal lobe functioning. This test provides a visual-spatial assessment of motor planning, organization and goal directed behavior. Performance on Mazes is considered to depend on planning ability and foresight, which are cognitive abilities thought to be mediated by the frontal brain systems. Alcoholics have been shown to perform poorly on this task (MacDonell, Skinner, Glen, 1987; Bowden, 1988). For instance, Bowden (1988) examined the performance of twenty male alcoholics using a test of complex maze learning. The results indicated that alcoholics performed worse than matched controls, providing additional evidence for deficits in frontal lobe functioning.

In summary, the literature on cognitive functioning of alcoholics suggests that alcohol can induce various cognitive impairments. Moreover, the weight of

the evidence appears to reside with the executive function deficit model rather than the generalized/diffuse or right hemisphere model. Nonetheless, there are shortcomings in some of the studies on the alcohol related cognitive impairment. For instance, the cognitive deficits seen during acute alcohol intoxication are usually transient rather than long lasting. In cases of chronic alcohol abuse, subclinical thiamin-dependent brain damage has been observed in alcoholics who did not show any signs of WKS (Harper, 1983). This raises the question of whether the cognitive damage exhibited in chronic alcoholics is due to alcohol neurotoxicity or thiamin deficiency.

Moreover, other personality or behavioral factors might have an influence on the cognitive performance of alcoholics. For example, Glenn, Errico, Parsons, King, and Nixon (1993) examined the role of antisocial, affective, and childhood behavioral characteristics in the neuropsychological performance of alcoholics. Although none of the subjects met a clinical diagnosis of anxiety, depression, or antisocial personality, all three factors were found to be negatively related to cognitive performance in alcoholics. In another study, Waldstein, Malloy, Stout, and Longabaugh (1996) found that the paths to neuropsychological impairment differed between antisocial and non-antisocial alcoholics. In antisocial alcoholics, cognitive deficits were predicted by less education, childhood symptoms of conduct disorder, drinks per day, and history of head injury. Meanwhile, the cognitive performance of non-antisocial alcoholics was predicted by self-reported history of diagnosed attention deficit disorder, verbal learning disability, and

symptoms of nonverbal learning disability. These findings raise the possibility that the cognitive functioning of alcoholics may be moderated by co-occurring psychopathology such as antisocial personality.

Cognitive Deficits as a Risk Factor for Alcohol Abuse

Although it appears that chronic alcohol abuse can cause cognitive impairment, some researchers have posited that cognitive deficits may be present before the onset of alcoholism. If cognitive impairment precedes the abuse, then cognitive deficits may contribute to the risk of developing alcoholism. To determine whether cognitive deficits increase the vulnerability of alcohol abuse, some researchers have compared the cognitive performance of alcoholics with a positive family history of alcoholism to those with a negative family history of alcoholism. Findings from these studies generally indicate that some cognitive deficits exhibited in alcoholics are associated with positive family history of alcoholism (Malloy, Noel, Rogers, Longbaugh, & Beattlie, 1989; Glenn & Parsons; 1990). However, results are difficult to interpret because chronic alcohol use also impairs cognitive functioning. Alternatively, researchers have begun to examine the neuropsychological functioning of children of alcoholics (COAs) who are non-alcoholics themselves. For example, Deckel, Bauer and Hesselbrock (1995) recently investigated the relationship between anterior brain dysfunction and alcoholic behaviors in 91 young adult men with no history of alcohol dependence. Subjects whose mother had a history of alcoholism were excluded from this study. The results showed that neuropsychological tests that assess

anterior brain functioning predicted the age at which subjects took their first drink and their score on the Michigan Alcohol Screening Test. Other studies have also found that cognitive deficits are present in children who are at risk for developing alcohol abuse/dependence (Noll & Zucker, 1983; Tarter, Hegedus, Goldstein, Shelly, & Alterman, 1984; Noll, Zucker, Fitzgerald, & Curtis, 1992).

The following section reviews a number of studies that investigated the cognitive abilities of COAs. Three areas of cognitive functioning have been studied extensively among COAs: Verbal ability, abstract planning/reasoning, and academic achievement.

a) Verbal Ability

Research has consistently shown that COAs exhibit poorer verbal processing skills as compared to non-COAs. For example, Gabrielli and Mednick (1983) studied the WISC scores of 265 Danish children to examine whether there were differences in verbal abilities between COAs and non-COAs. The results indicated that COAs obtained a lower Verbal IQ score, suggesting that verbal deficits may be antecedent to alcoholism. Ervin, Little, Streissguth, and Beck (1984) also tried to dissociate the effect of alcohol dependence and positive family history of alcoholism on cognitive functioning by studying young COAs who were not yet using high amounts of alcohol. Children who were raised by alcoholic fathers were found to have lower Verbal, Performance, and Full Scale IQ than those who were raised by non-alcoholic fathers although the means for both groups fell within the average range. In a prospective longitudinal study of

alcoholism, Knop, Teasdale, Schulsinger, and Goodwin (1985) also examined the cognitive ability of sons of alcoholic and non-alcoholic fathers. Based on teachers' reports, sons of alcoholics were found to have poorer verbal abilities as compared to sons of non-alcoholics. Several other recent studies have confirmed that verbal ability among COAs is significantly lower than non-COAs (Sher, Walitzer, Wood, & Brent, 1991; Ozkaragoz and Noble, 1995).

b) Abstract Reasoning/Planning

COAs also appear to exhibit problems in abstract reasoning and planning abilities, which are thought to be mediated by the frontal brain region. Three of the WISC subtests, Block Design, Similarities, and Mazes, have been widely used by investigators to measure abstraction and conceptual reasoning abilities. First, several studies have reported that COAs performed worse on Block Design than non-COAs (Schaeffer, Parsons, & Yohman, 1984; Whipple, Parker, & Noble, 1988). For instance, Schaeffer, Parsons, and Yohman (1984) examined the cognitive ability of alcoholics with either a positive or negative family history of alcoholism and found that only those with positive family history of alcoholism showed impaired performance on Block Design. The authors concluded that deficits on abstracting/problem solving and perceptual-motor skills are related to family history of alcoholism independent of the effect of alcoholism itself. Even in studies where the difference did not reach significance, mean scores on Block Design were still consistently lower among COAs than non-COAs (Gabrielli & Mednick, 1983; Ervin et al., 1984; Drejer, Theilgaard, Teasdale, Schulsinger, &

Goodwin, 1985).

The Similarities subtest of the WISC-R was designed to measure abstract verbal reasoning. Three studies have found that COAs performed worse on Similarities than non-COAs (Ervin et al., 1984; Gabrielli & Mednick, 1983; Sher, Walitzer, Wood, & Brent, 1991). In one of the studies, Sher, Walitzer, Wood and Brent (1991) examined the cognitive functioning of 490 COAs and non-COAs and found that performance on WISC-R Similarities was significantly lower among COAs.

Finally, the Mazes subtest of the WISC-R was designed to measure planning ability and foresight. In one study, Tarter, Jacob, and Bremer (1989b) examined the cognitive status of sons of community dwelling alcoholic men. Although the cognitive deficits were not severe, sons of alcoholics were impaired on planning ability (e.g., Porteus Mazes), psychomotor efficiency (e.g., Symbol Digit), and inhibitory control (e.g., Stroop). Jacob et al. concluded that planning deficits found in COAs may be due to anterior cerebral dysfunction. Other researchers have also found that poor performance on the Mazes test is associated with positive family history of alcoholism (Drejer, et al., 1985; Gillen & Hesselbrock, 1992).

Recently, Pihl and Bruce (1995) proposed an information-processing model that explains the relationship between the deficit of abstract planning and behavioral problems among male COAs. According to Pihl et al., dysfunction in the prefrontal cortex may lead to deficits in abstract planning and reasoning, which

in turn limit the response options of the child and cause the child to act impulsively. Problems with impulse control may result in frustration, boredom, and also in higher rates of punishment from adults, all of which may ultimately be linked to problems such as conduct disorder and early problematic alcohol use. Thus, impulsivity may mediate the relationship between cognitive impairment and risk of alcoholism.

c) Academic Achievement

Tarter and his colleagues (Hegedus, Alterman, and Tarter, 1984) posited that cognitive deficits may lead to poor academic performance among COAs. These researchers found that delinquent sons of alcoholics performed significantly worse on a standardized achievement test as compared to matched controls. In addition, educational achievement was found to be correlated with neuropsychological performance, supporting the hypothesis that cognitive deficits are associated with poor academic performance and risk for alcoholism. More recently, Murphy, O'Farrell, Floyd, and Connors, (1991) found that children of long-term alcoholic fathers had lower grade point average (GPA) than children of control families. Similar results have been reported in several other studies (Ervin et al., 1984; Bennett, Wolin, & Reiss, 1988; Sher, Walitzer, Wood, & Brent, 1991; Chandy, Harris, Blum, & Resnick, 1993).

Methodological Issues

In summary, it appears that COAs can be characterized by poorer verbal and abstract planning abilities and lower academic achievement. However, some

studies have not been able to replicate the findings of cognitive deficits in the COA population. For instance, Reed, Grant, and Adams (1987) found that positive family history of alcoholism did not predict poorer performance on WAIS Vocabulary, Digit Symbol, and the Halstead-Reitan Battery in alcoholics. However, the authors noted that alcohol neurotoxicity may have masked premorbid cognitive differences between alcoholic COAs and non-COAs. Alternatively, the negative finding may stem from the method used in collecting parental drinking history. Reed et al. only used third party information to determine parental alcoholism and the fathers were not interviewed.

Johnson and Rolf (1988) also did not find significant differences between children of alcoholic and non-alcoholic families on Verbal and Performance IQ or measures of academic achievement. As a result, they concluded that impaired cognitive functioning should not be considered as a risk factor for alcoholism. However, the non-significant finding may due to the authors' selection of subjects. Specifically, Johnson and Rolf used socially and economically non-disadvantaged children whose parents were recovered alcoholics whereas other studies that have found cognitive deficits in COAs used subjects of active alcoholic parents. In a recent study of cognitive functioning of early school-aged COAs, Puttler (1996) found that female offspring of recovering alcoholics have IQs and academic achievement similar to those of girls from control families, and significantly higher than girls of active alcoholics. Thus, it is possible that active parental alcoholism is a marker of alcoholism severity, which may influence the

relationship between cognitive deficits and positive family history of alcoholism.

Schuckit, Butters, Lyn, and Irwin (1987) also questioned whether or not cognitive deficits increase vulnerability to alcoholism. In their study, they found that collegiate COAs and non-COAs performed equally well on several neuropsychological tests including a category test, a trail making test, body sway, word recall, and a missing digit test. Similarly, Alterman, Searles, and Hall (1989) examined the cognitive performance of college men at high (paternal alcoholism), medium (only second-degree relatives who were alcoholics) and low risk (no alcoholic relatives) for alcoholism and found no relationship between the cognitive measures and family history of alcoholism. However, these college subjects had already achieved relatively high academic success despite their COA status. Therefore, it is arguable that whatever factors helped them to overcome a positive family history of alcoholism may also have protected them from cognitive impairment.

The inconsistent findings regarding the cognitive ability of COAs may also stem from the substantial heterogeneity found in the alcoholic population and among families of alcoholics. Indeed, research has indicated that there are at least two different types of male alcoholics: antisocial and non-antisocial alcoholics (Cloninger, 1987; Zucker, Ellis, & Fitzgerald, 1993; Zucker, 1987; 1994). Antisocial alcoholics are likely to drink more alcohol, have an earlier onset of alcoholism, display more alcohol-related problems, and have more co-morbid psychopathology such as depression and anxiety as compared to alcoholics

without antisocial personality (Hesselbrock, Meyer, & Keener, 1985; Zucker, 1987). Moreover, it has been hypothesized that among alcoholics with antisocial personality (ASP) the brain system that modulates behavioral responses to the effects of alcohol and other environmental stimuli may differ from that of other alcoholics (Cloninger, 1987). Consistent with this hypothesis is the finding that alcoholics with ASP exhibit a variety of neuropsychological impairments. In one study, Malloy, Noel, Rogers, Longabaugh, and Beattie (1989) examined how age, gender, years of drinking, and ASP affected neuropsychological functioning of alcoholics. Alcoholics with co-morbid ASP were found to be more impaired on a variety of neuropsychological tests (WAIS, the Wechsler Memory Scale, and the Halstead-Reitan Neuropsychological Battery) than were alcoholics without co-morbid ASP. When the effect of age, gender, and years of drinking were controlled, ASP still contributed significantly to the cognitive impairment.

Although research has suggested that alcoholism is a heterogeneous disorder, very few studies have looked at the effect of different alcoholic family subtypes upon offspring. Studies that did account for the heterogeneity of alcoholic families have found that different parental characteristics are associated with specific behavioral and psychological problems (Chassin, Rogosch, & Barrera, 1991; Ellis, Bingham, Zucker, & Fitzgerald, 1997; Finn, Sharkansky, Viken, West, Sandy, & Bufferd, 1997). For instance, Chassin, Rogosch, and Barrera (1991) evaluated parental characteristics as risk factors for externalizing and internalizing symptoms and alcohol use in a community sample of COAs.

They found that externalizing problems are associated with parental alcoholism, and the effect is mediated by the co-occurrence of parental antisocial personality disorder. In a prospective study on COAs, Ellis, Bingham, Zucker, and Fitzgerald (1997) reported that children from family with different alcoholic subtypes (antisocial and non-antisocial alcoholism) differed on a number of child risk factors. Particularly, children of antisocial alcoholics exhibited increased level of risky temperament, externalizing problems, and hyperactivity. Further analyses using structural equation modeling techniques revealed that the pathway of influencing structure differed between the children from families with alcoholic subtypes, suggesting that the development of vulnerability for alcoholism may be unique for each subtype. Finn, Sharkansky, Viken, West, Sandy, and Bufferd (1997) also found that antisocial traits and negative affect were significantly higher in COAs with a high level of familial antisocial personality and alcoholism as compared to children of non-alcoholics. Furthermore, children from alcoholic families with high levels of depression and anxiety disorders showed elevated levels of antisocial traits, hypomania, and sensation-seeking behavior.

Even fewer studies have investigated whether alcoholic family subtype is related to cognitive functioning of COAs. In one study, Tarter, Jacob, and Bremer (1989a) assessed the performance of young sons of early onset alcoholics, late onset alcoholics, normal social drinking fathers, and depressed fathers on several cognitive tests including the WISC, 5 subtests from the Detroit Tests of Learning Aptitude, and Standard Progressive Matrices. Sons of early onset alcoholics

showed poorer attention capacity and verbal ability than sons of the normal social drinking fathers. However, there were no differences between the cognitive performance of sons of early onset alcoholics and late onset alcoholics. Harden and Pihl (1995) examined the cognitive functioning of sons of multigenerational alcoholic families. Multigenerational alcoholic families are characterized by dense family histories of alcoholism and also by higher rates of other types of psychopathology such as antisociality. Harden and Pihl found that sons of multigenerational alcoholic families performed the worst on Spatial Conditional Associate-Learning, Self-ordered Pointing (concrete), Wisconsin Card Sort Test (perseveration responses), and Word Fluency, all of which are tests of frontal lobe functions (i.e. planning and goal directed behavior).

STATEMENT OF THE PROBLEM

Based on the previous literature review, it appears that COAs exhibit various cognitive deficits that may put them at risk directly or may act synergistically with other child behavior problems such as aggression and oppositionality to increase risk for alcoholism. However, methodological and procedural inconsistencies among studies have hampered research in this area. In addition, little work has been done to determine whether or not parental alcoholic subtype moderates the relationship between cognitive functioning and positive family history of alcoholism. The present study examines cognitive functioning of male COAs during the early school age years as related to differences in familial alcoholism subtype.

First, focusing on a younger age group eliminates the possibility that alcohol neurotoxicity will confound the results of the study since children in the current sample have not been exposed to alcohol use. Moreover, there is evidence that some cognitive differences may be present in high risk children as early as the preschool years. In a study of preschool COAs, Noll and Zucker (1983) found that COAs exhibited poor fine motor coordination, poor completion of adaptive behavioral task and low levels of language development on the Yale Developmental Inventory. Noll, Zucker, Fitzgerald and Curtis (1992) found that preschool COAs were significantly different from matched controls in global developmental quotient (DQ) as well as in fine-motor and personal/social skills on

the Revised Yale Developmental Schedules (RYDS). The adaptive and language scores were also lower among COAs as compared to matched controls. Regression analyses revealed that the differences in cognitive functioning between COAs and non-COAs were related to the lower quality of cognitive, social, and emotional stimulation available to the child in the home environment.

Second, subtypes of paternal alcoholism have been found to provide a useful way of understanding risk variations among COAs, and they may provide a way of identifying a subset of COAs with greater cognitive impairment as well. Further, it is possible that children of antisocial alcoholics may be at highest risk for cognitive impairment as children of antisocial alcoholics have previously been identified as being at highest risk for a variety of behavioral impairments. The current study attempts to investigate how the performance of COAs on cognitive tests is moderated by paternal alcoholism subtype, in particular, antisocial vs. non-antisocial alcoholism. Such information is unique in demonstrating that subtyping alcoholic families based upon degree of antisociality is a useful method for explaining the heterogeneity of cognitive functioning found among COAs.

HYPOTHESES

Specific hypotheses are as follows:

1. Children of antisocial alcoholics (AALs) will show poorer general intellectual functioning as compared to children of non-antisocial alcoholics (NAALs). Children of NAALs will form an intermediate group that will show lower levels of intellectual functioning than children of controls.
2. Children of AALs will show lower verbal skills as compared to children of NAALs and controls. Children of NAALs will form an intermediate group that will show lower verbal skills than children of controls.
3. Children of AALs will show the lowest academic achievement among children of all groups. Children of NAALs will form an intermediate group that will show lower academic achievement than children of controls.
4. Children of AALs will show poorer abstract planning and reasoning abilities than children of NAALs and controls. Children of NAALs will form an intermediate group that will show poorer abstract planning and reasoning abilities than children of controls.
5. Children of AALs will show higher level of impulsivity as compared to children of NAALs. Children of NAALs will form an intermediate group that will show higher level of impulsivity than children of controls. In addition, level of impulsivity will be negatively correlated with verbal abilities, abstract planning and reasoning skills and academic achievement among COAs.

6. A specific set of cognitive characteristics can be developed to distinguish children of AALs from children in the other groups.
7. Intellectual functioning and impulsivity among COAs will be predicted by socioeconomic status, familial alcoholism subtype, parental intellectual abilities and density of alcoholism in the family.

METHOD

Participants

Subjects for the present study were 198 families participating in the etiologic portion of the Michigan State University - University of Michigan Longitudinal Study (Zucker & Fitzgerald, 1991). This ongoing longitudinal project utilizes population-based recruitment strategies to access alcoholic men and their families and a contrast group of families with non-substance abusing parents. All families were Caucasian. The limited ethnic/racial composition was dictated by the fact that census data in the area where initial data collection took place indicated that other ethnic and racial groups would represent less than 10% of the sample. Given the extensive literature demonstrating a substantial relationship between patterns of alcohol involvement and ethnic/racial status and the fact that effective analyses for such differences could not be undertaken with the proposed study sample size, it was decided to exclude such variation rather than have it contribute to error. During the initial contact, all families were invited to participate in a long term study of family and health and child development. Families are assessed at three year intervals beginning at Wave 1 when the male target child (MTC) were ages 3 to 6. All families received some payment for participation in each data collection interval. The data in the present study were from the MTC second data collection wave. The boys ranged in age from 6 to 9.

At time of initial recruitment, all alcoholic fathers were required to have a

3-0 to 5-11 year old son with whom they were living and also to be residing with the child's mother. Mother's drinking status was assessed for alcoholic families, but maternal alcoholism was neither a requirement nor a basis for exclusion criteria, no child manifested characteristics sufficient for a diagnosis of fetal alcohol syndrome.

Alcoholic families were recruited by way of father's drinking status. Alcoholic fathers were identified in one of two ways. The first group was recruited from the population of all convicted drunk drivers in a four county area of mid-Michigan. Thereafter, all males meeting from the family recruitment criteria involving child age and coupling status who had a blood alcohol concentration (BAC) of 0.15% (150 mg/100 ml) or higher when arrested, or a BAC of 0.12% if a history of prior alcohol-related driving offenses existed, were asked for permission to have their names released for contact by study staff. 79% agreed to have their name released, and of those, 92% agree to participate. At initial contact, a positive alcoholism diagnosis was established using the Short Michigan Alcoholism Screening Test (SMAST; Selzer, 1975); this diagnosis was subsequently verified by way of the NIMH Diagnostic Interview Schedule-Version III (DIS; Robins, Helzer, Croughan & Ratcliffe, 1980). All of these men met a 'definite' or 'probable' criterion for alcoholism using the Feighner Diagnostic Criteria (Feighner, Robins, Guze, Woodruff, Winokur, & Munoz, 1972), with 92% making a 'definite' diagnosis. Later, DSM-III-R diagnoses were also established although this was not a basis for study inclusion; 73% of the

alcoholic men met either moderate or severe alcohol dependence criteria.

The second strategy involved recruiting alcoholic father out of the same neighborhoods where drunk driver alcoholic father resided. These families were accessed during neighborhood canvasses for nonalcoholic (control) families. Thus, they provided an ecologically comparable subset of high risk families drawn out of the same social stratum as the drunk drivers, but where the alcoholism was identified by way of community survey rather than by way of legal difficulty. These alcoholic fathers also met Feighner criteria for probable or definite alcoholism (85% made a definite diagnosis), had children and partners who met the same inclusion criteria as the drunk driving group, but had no drunk driving or drug involved arrest record occurring during the lifetime of the 3 to 5 year old target child.

In addition to alcoholic families, a group of community control families were recruited via door-to-door community survey techniques. These families were recruited out of the same neighborhoods as neither parent met Feighner criteria for alcoholism or for other drug abuse/dependence. In addition, efforts were made to match control families with alcoholic families on the basis of family socioeconomic status by recruiting controls from the same neighborhood in which the risk family lived. Canvassers initiated a door-to-door search a block away from the alcoholic family, staying within the same census tract, and screened for nonalcoholic families with a child of appropriate age. However, in some cases locating a neighborhood control proved impossible due to high levels of drug

and/or alcohol abuse among potential control families living in neighborhoods where the alcoholic families resided. In such cases, the recruitment moved to an adjacent neighborhood and in some instances it was necessary to go even more broadly afield in order to locate another socio-demographically comparable community in which to continue the search. Ninety-three percent of families who met eligibility criteria as controls agreed to participate.

Data Collection

Data were collected by trained project staff who were blind to family risk status. Because of the large volume of data collected, a number of contacts with the family were necessary. Wave Two data collection took place across nine data collection sessions, seven of which took place in the family home and two of which took place on a university campus. The visits involved approximately fifteen hours of contact time for each parent and seven hours of time for the target child. Contacts included questionnaires sessions, semi-structured interviews and interactive tasks.

Measures

Family Demographic. Information on family demographics came from a questionnaire assessing parental education, occupation and family income. Socioeconomic status (SES) of each parent was calculated using the Duncan TSE12 Socioeconomic Index (Stevens & Featherman, 1981), an occupationally-based measure of social prestige. In order to obtain a measure of SES which would best capture the environment of the MTC, family SES was calculated using

an average of the mother's and father's SES when both parents work and the employed parent's score where only one parent worked. Special scores reflecting the lowest possible Duncan ratings were utilized for families where neither parent was employed.

Parental Alcoholism. At the first Wave of data collection, information on current and lifetime prevalence of alcohol problems was gathered using the Short Michigan Alcohol Screening Test (SMAST) and the Diagnostic Interview Schedule - Version III (DIS). The SMAST (Selzer, 1975) is a well validated inventory used extensively to assess alcohol problem. The DIS is a structured interview that allows trained lay interviewers to gather extensive physical, alcohol and drug related, and mental health (symptomatic) information that can then be computer processed to yield diagnoses by way of the three major nosological systems in use today (DSM-III; Feighner, RDC). In addition, when given by a trained clinician it also offers the opportunity to obtain an extensive psychiatric clinical history. At Wave 2, all parents completed the SMAST and DIS again to obtain information on their current alcoholic problems based on the past three year interval. The diagnosis of current or lifetime alcohol abuse/dependence was made by a trained clinician for each wave of data collection using the DSM-IV criteria based on the information provided on the SMAST and DIS. A summary of paternal alcohol abuse diagnosis is presented in Table 1.

Antisocial Personality. The DIS also gathers extensive information about psychiatric symptomatology including antisocial behavior. A diagnosis of

antisocial personality disorder (ASP) was made by a trained clinician using the DSM-IV based on the information provided on the DIS. Unlike alcoholism, which may remit, ASP was assumed to be a lifelong disorder. Therefore, a diagnosis of ASP was made based upon Wave 1 data and men who met criteria at Wave 1 were assumed to have ASP at Wave 2 as well. A summary of ASP diagnosis is presented in Table 1.

Alcoholic Subtype. In order for children from alcoholic families to be identified as offspring of antisocial alcoholics (AALs) or non-antisocial alcoholics (NAALs), their fathers were classified as one or the other alcoholic subtype. Alcoholic fathers who meet the DSM-IV diagnosis for ASP via the DIS were classified as AALs and those who did not have an ASP diagnosis were classified as NAALs. As previously mentioned, alcoholism diagnoses were available both for the Wave 1 and Wave 2 data collection periods. Most AALs showed continuity in alcoholic symptomatology and met diagnostic criteria for abuse or dependence at both data collection periods (n = 30) although a small number no longer met abuse/dependence criteria and were dropped from the study (n = 2). Among NAAL men, some showed continuity of alcohol abuse/dependence across the two data collection periods while some did not. Therefore, children of NAALs were further subdivided into two groups, children of NAALs-current (positive paternal diagnosis of both periods) (n = 64) and NAALs-remission (positive paternal diagnosis only at wave 1) (n = 38). In addition, any control families where parents had developed an alcohol abuse/dependence diagnosis during wave

Table 1

Descriptive Statistics Pertaining to Paternal Alcohol and Antisocial Personality Diagnoses (Dx) in the Study Sample (N=198)

	n	% of Sample	% of subjects receiving Dx
Alcohol Abuse/Dependence Dx (lifetime)			
None (no Dx)	66	33	--
Abuse	13	7	10
Dependence without physical dependence	36	18	27
Dependence with physical dependence	83	42	63
Alcohol Abuse/Dependence Dx (last 3 years)			
None (no Dx)	104	53	--
Abuse	25	9	26
Dependence without physical dependence	29	15	31
Dependence with physical dependence	40	20	43
Antisocial Personality Dx (lifetime)			
No	168	85	--
Yes	30	15	--

Note: Diagnoses based on DSM-IV criteria.

2 were dropped from the study (n = 6) leaving an n of 66.

General Cognitive Functioning. Cognitive functioning of COAs was measured by the Wechsler Intelligence Scale for Children-Revised (WISC-R; Wechsler, 1974). The WISC-R is designed for use with children from ages 6 to 16 years and consists of six Verbal Scales (Information, Similarities, Arithmetic, Vocabulary, Comprehension, and Digit Span) and six Performance Scales (Picture Completion, Picture Arrangement, Block Design, Object Assembly, and Coding or Mazes). The Full Scale IQ was based on the summed Verbal and Performance scores while scores on the Digit Span and Mazes were not used in establishing IQ scores. Verbal, Performance and Full Scale IQs have been shown to have high reliabilities across ages 6 to 16, the average coefficients being .94, .90, and .96, respectively. In addition, correlations between the WISC-R, the Wechsler Preschool and Primary Scale of Intelligence (WPPSI), the Wechsler Adult Intelligence Scale (WAIS), and the Stanford-Binet Intelligence Scale are high and positive, with average correlations being .82, .95, and .73, respectively.

Executive Functioning. Using subtest scaled scores from the WISC-R, two indexes were composited as measures of executive functioning. An Abstract Planning Index (API) was calculated by adding the Mazes and the Picture Arrangement scaled scores. Similarly, an abstract reasoning index (ARI) was calculated by adding the Block Design and the Similarities scaled scores.

Academic Achievement. The WRAT-R (Jastak & Wilkinson, 1984) was used to measure the academic skills of reading, spelling, and arithmetic. This test

is divided into two levels: Level 1 is administered to children from ages 5 to 11 years; level 2 is intended for individuals from 12 years of age through adulthood. The Reading subtest examined the recognition, naming, and pronunciation of words given out of context. The Spelling subtest consisted of copying marks, resembling letters, writing the name, and writing single words to dictation. The Arithmetic subtest consisted of counting, reading number symbols, solving oral problems, and performing written computations. This edition of the WRAT was standardized using a stratified national sample. The test-retest reliability coefficients for Level 1 and 2 range from .79 to .97 and the correlations between the WRAT-R and other achievement and ability tests (e.g., California Achievement Test; the Stanford Achievement Test) range from the .60's to the .80's.

Impulsivity. The Delay of Gratification (Funder, Block, and Block, 1983) was used as an approximate measure of verbal impulsivity of the child. The Delay of Gratification was developed to evaluate the child's ability to delay gratification (i.e. impulse control). Subsequent to the child's intellectual assessment session, the child is thanked for their participation and told they can have a present. As the present is being shown, the examiner apologized to the child and said there is one more task they must first complete. The child is shown a complex block design task (Design #11 - WAIS-R) and is told they must first complete this task before getting their present. Observations were then made regarding the child's behavior (e.g. verbal remarks about the present) and timed until the present is taken.

Scores on this brief laboratory task, using a large sample of four year-year-old children (N=116), have been compared to ratings of child personality by examiners and teachers using the California Child Q-sort (Funder et al., 1983). Personality data (Q-sorts) were available for the children when they were 3, 4, 7, and 11 years of age. Findings demonstrated that boys at age four who delayed gratification on this task were described as being more attentive, more able to concentrate, more reasonable, and more cooperative. These results were interpreted as demonstrating the relationship between task performance at age four and measures of personality related to ego control or undercontrol that remained very stable during the lengthy time period of this project (Funder et al., 1983). Analysis of data from Time 1 of the MSU-UM Longitudinal Study involving two observers independently scoring the child's behaviors demonstrated excellent inter-rater reliability (N=12; r=.97). For the purpose of this study, two variables, Total Comments (total number of comments made) and Time Delay (amount of time before child takes present, up to a maximum of 90 seconds) were chosen as measures of impulse control.

Family Expression of Alcoholism. Information on alcoholism in the target children's families was obtained via a family history interview, or genogram, where the child's parents provided data on psychiatric and physical disorders for themselves and for other family members. Parents were first asked to produce a family tree extending back to their aunts, uncles, and first cousins. They were then given a standardized list of various physical and psychological disorders,

including alcoholism, and asked to identify any relatives who were affected by any of the listed disorders; this process created a genogram. Any additional information provided by parents about their family, such as disorders not included on the list, was also recorded.

Several studies have investigated the reliability and validity of the family history method. O'Malley, Carey and Maisto (1986)compared young adults' reports of alcohol use and alcohol related problems in their parents to the parents' self-report; the two were found to be highly correlated (e.g. the Pearson correlation between students' and fathers' estimates of average monthly consumption was .72). Thompson, Orvascel, Prusoff and Kidd (1982) compared subjects' reports of various psychiatric illnesses in their relatives to diagnoses made by psychiatrists during personal interviews. They found that the family history method generated few false positives (specificity = .96) but more false negative (sensitivity = .57). Offspring were found to produce the most accurate reports of illness as compared to spouses and parents. Therefore, positive diagnoses generated by the family history method are highly likely to be accurate, although true incidence of alcoholism in relatives appears to be underestimated (Thompson et. al., 1982).

The family expression of alcoholism (FEA) score used here was derived from genogram data. In order to determine FEA scores for each child, alcoholic family members were primarily identified by using the genograms of each of the child's parents. However, if diagnostic information from other measures collected by the larger research project identified parents as alcoholic, even though not self-

identified as such on the genogram, they were also coded as alcoholics for the purpose of calculating FEA scores. Thereafter, the individual genograms completed by each of the child's parents were combined to produce a genogram for the child. Although the genograms provided data on cousins and other more distant relatives, only data on the target child's parents, aunts/uncles, grandparents and great aunts/uncles, were used in the analyses, since it became clear during genogram coding that in this data set, lack of familiarity with more distant relatives did not allow respondents to accurately label them as alcoholic or not.

After identification of alcoholic relatives, the child's first degree relatives, such as parents, were allotted a weighting of .50. Second degree relatives, such as grandparents, aunts and uncles, were allotted a weighting of .25. More distant relatives such as great aunts and uncles were allotted a weighting of .125. FEA scores were then calculated by 1) within each generation, summing the weightings for all alcoholics relatives 2) multiplying this sum by the ratio of alcoholics to total number of family members in that generation and 3) summing the subscores across generations.

Since the child's FEA score includes points assigned for alcoholic parents, who both raised him and contributed to his genetic makeup, FEA cannot be considered to be a pure measure of genetic loading for alcoholism as separate from the effects of being raised by an alcoholic. However, the FEA score reflects the density of alcoholism in the child's extended family as well as the degree of relatedness of these alcoholic family members to the child. Additionally, most of

the alcoholic relatives contributing to the child's FEA score would not have participated in his day to day rearing. Thus, FEA does give some index of inherited risk for alcoholism.

Power Analyses

Power analyses were performed to assess the chance of detecting statistical significance if it in fact existed. Using a more conservative approach, effect size was set at a medium level (f = 0.25; R-squared = 0.13) and criterion for significance (alpha) was set at 0.05, two-tailed. According to Cohen (1977), a power of .80 or higher (i.e. a beta risk of .20 or lower) is considered adequate in most social science research. With approximately 200 cases distributed evenly among 4 groups, the estimated power for a one-way analysis of variance (ANOVA) was 0.85. The estimated power remained at 0.85 when a covariate (R-squared = 0.02) was added to the design (ANCOVA). For multiple regression design, a set of four predictors with a medium level of R-squared (i.e. 0.13) yielded an overall power of .99. Further analysis indicated that the power for the fourth variable to detect an R-squared increment of 0.03 (i.e. from 0.10 to 0.13) fell to 0.74.

RESULTS

Missing Data

Before beginning analyses, all variables were screened for missing data. A group mean substitution procedure was used to estimate missing data for each variable. This data estimation method was considered appropriate based on the assumptions that all subgroups differed significantly for variables with missing data and that variance was relatively homogeneous within each subgroup. Under such conditions, estimating missing data with groups mean is unlikely to attenuate variance of dependent variables. No more than five percent of values were estimated in this manner for any of the variables.

Background Characteristics

Table 2 presents a summary of sociodemographic information (parent age, child age, parent years of education, parental IQ, and family socioeconomic status [SES]) for the sample. One-way analyses of variance (ANOVAs) were conducted on each of the dependent variables listed above. The results showed significant effects of familial alcoholism subtype on parent age, parent years of education, parental IQ, and SES (see Table 2).

Post-hoc comparisons between AAL, NAAL-cur, NAAL-rem, and controls families using Tukey's B Test revealed that mothers from AAL families were significantly younger than mothers from NAAL-cur, NAAL-rem, and control families. In addition, mothers from AAL families had significantly lower IQ and

Table 2

Analysis of Variance for Background Characteristics (N = 198)

	Risk Group				
	AALs (n=30) M (SD)	NAALs-cur (n=64) M (SD)	NAALs-rem (n=38) M (SD)	Controls (n=66) M (SD)	F
Mothers					
Age	30.8 (3.5)	34.7 (3.6)	34.8 (4.3)	34.1 (3.9)	7.89** [abc]
Education in Years	12.1 (1.6)	13.5 (2.1)	13.3 (1.9)	13.7 (1.8)	5.35** [abc]
Full Scale IQ	91.4 (12.7)	101.2 (15.7)	101.0 (12.1)	101.3 (12.7)	4.38** [abc]
Fathers					
Age	33.7 (4.6)	37.1 (4.2)	37.1 (6.0)	35.5 (4.8)	3.91* [ab]
Education in Years	12.2 (1.5)	13.8 (2.5)	13.7 (2.1)	14.7 (2.2)	9.03** [abcd]
Full Scale IQ	87.5 (10.6)	100.4 (16.3)	98.7 (15.4)	102.9 (15.1)	7.56** [abc]
Children Age	7.5 (1.0)	7.6 (1.0)	7.4 (1.0)	7.4 (0.9)	0.88
Family SES [1]	253.6 (64.6)	334.3 (132.2)	346.0 (119.9)	378.5 (138.3)	7.01** [abc]

[1] Duncan TSEI2 (Stevens & Featherman, 1981)
*p < .05; **p <.01.
[a] AALs < NAALs-cur, Tukey's B
[b] AALs < NAALs-rem, Tukey's B
[c] AALs < Controls, Tukey's B
[d] NAALs-cur < Controls, Tukey's B

fewer years of education than mothers from NAAL-cur, NAAL-rem, and control families. Fathers from the AAL families were significantly younger than fathers from the NAAL-cur and NAAL-rem families. They also had significantly lower IQ and fewer years of education than fathers from NAAL-cur, NAAL-rem, and controls families. Moreover, fathers from the control families reported significantly more years of education than fathers from the other groups. Additionally, SES was significantly lower among AAL families as compared to NAAL-cur, NAAL-rem, and control families. Age of the target child did not differ significantly across the four groups.

Analyses of Variance

Means and standard deviations for all dependent variables are presented in Table 4 - 8. Significance for all multivariate tests was determined using Wilks' Lamda. In addition, Tukey's B Test was used to conduct post-hoc comparisons in cases where significant main effects were found.

Global Cognitive Abilities. A multivariate analysis of variance (MANOVA) design was used to examine the overall main effect of familial alcoholism subtype (RISK) on measures of child's global cognitive ability. MANOVA was used to control for Type 1 error which can arise when multiple comparisons are performed. The results showed a significant main effect of RISK [$F(9,468)=2.93$, $p<.01$] (see Table 3). Univariate ANOVAs revealed that the four groups differed significantly on Verbal [$F(3,194)=3.64$, $p<.05$], Performance [$F(3,194)=5.66$, $p<.01$], and Full Scale IQ [$F(3,194)=6.30$, $p<.01$]. Post-hoc

Table 3

Multivariate Analysis of Variance for Global Cognitive Measures (N=198)

	Risk Group				
	AALs (n=30) M (SD)	NAALs-cur (n=64) M (SD)	NAALs-rem (n=38) M (SD)	Controls (n=66) M (SD)	F
Verbal IQ	100.1 (11.6)	106.4 (13.8)	105.1 (12.3)	109.5 (13.8)	3.64* [a]
Performance IQ	99.5 (12.1)	108.1 (16.0)	104.1 (12.4)	111.9 (15.6)	5.66** [abc]
Full Scale IQ	99.5 (10.5)	107.9 (14.3)	104.8 (12.1)	111.8 (14.6)	6.30** [abc]

*p < .05; **p <.01.

[a] AALs < Controls, Tukey's B
[b] AALs < NAALS-cur, Tukey's B
[c] NAALs-rem < Controls, Tukey's B

comparisons using Tukey's B Test showed children from AAL families had significantly lower Performance and Full Scale IQ than children from NAAL-cur and control families. In addition, children of AALs were found to have lower Verbal IQ than children of NAALs-cur and controls. Children of NAALs-rem had significantly lower Performance and Full Scale IQ than children from control families. No differences were found between children from NAAL-cur and control families.

To determine whether group differences existed after variability in SES had been accounted for, a multivariate analysis of Covariance (MANCOVA) was conducted on IQ scores with SES as the covariate. The results indicated a continued significant main effect of RISK [$F(9,460)=2.76$, $P<.01$], with univariate analysis of covariance (ANCOVAs) showing significant group differences for Verbal [$F(3,191)=2.97$, $P<.05$], Performance [$F(3,191)=5.06$, $P<.01$], and Full Scale IQ [$F(3,191)=5.66$, $P<.01$].

As shown in Table 3, consistent differences were found between children of AALs, NAALs-rem, and children in the other two groups. Nevertheless, such differences at the mean level do not provide information regarding rates of children in each group who fell in the range where intellectual deficits are considered clinically meaningful. In order to evaluate the relationship between group status and clinical-range intellectual deficits, percentages of children from each group falling below an IQ score of 80 were calculated (i.e. IQs falling outside the average range). Results indicate that 3.3% of AAL children and 4.7% of

NAAL-cur children met the criteria for clinically significant intellectual deficit on the WISC-R, whereas none of the children from NAAL-rem and control families met the same criteria. Odds ratios were calculated as an estimate of relative risk for clinically significant intellectual deficit; relative risk indices greater than or equal to three indicate strong effects (Chassin, Rogosch, & Barrera, 1991). Chi-square statistics were calculated to assess the degree of association in each 2 x 2 contingency table from which the odds ratio was calculated. The odds ratio for children from NAAL-cur families was calculated to be 3.2 (X^2=3.17, n.s.), suggesting that rates of intellectual impairment did not differ significantly from controls. Similarly, the chi-square for children of AALs indicated that they did not vary significantly from controls (Odds ratio = 2.3; X^2=2.22, n.s.).

Academic Achievement. MANOVA results did not show significant main effects of RISK upon academic achievement [F(9,467)=1.45, p<.17]. However, univariate ANOVAs revealed that the four groups differed significantly on Spelling [F(3,194)=3.25, p<.05] and Arithmetic [F(3,194)=3.20,p<0.05] skills (see Table 4). Post-hoc comparisons using Tukey's B Test indicated children from AAL families had significantly lower Spelling and Arithmetic scores than children from control families. No significant differences were found between NAAL-cur, NAAL-rem, and control families.

Results of a MANCOVA on academic achievement with SES as covariate also failed to demonstrate a main effect of RISK [F(99,460)=1.37, p<.20]. However, univariate ANCOVAs continued to show significant group differences

Table 4

Multivariate Analysis of Variance for Academic Achievement Measures (N=198)

	Risk Group				
	AALs (n=30) M (SD)	NAALs-cur (n=64) M (SD)	NAALs-rem (n=38) M (SD)	Controls (n=66) M (SD)	F
Reading	92.1 (15.6)	97.5 (14.3)	93.6 (13.9)	99.9 (15.2)	2.64
Spelling	89.1 (15.2)	94.5 (14.3)	91.0 (14.4)	98.0 (15.3)	3.25* [a]
Arithmetic	92.4 (12.9)	96.0 (14.4)	94.6 (14.1)	100.8 (13.9)	3.52* [a]

*$p < .05$

[a] AALs < Controls, Tukey's B

for Spelling [F(3,191)=3.06, p<.05] and Arithmetic scores [F(3,191)=2.98, p<.05] after SES had been accounted for. An additional MANCOVA was conducted with child's Full Scale IQ added as a second covariate to determine whether the group demonstrated differences in academic achievement beyond that which could be accounted for by general intelligence. No significant group differences on either Spelling or Arithmetic scores were found once variance related to Full Scale IQ had been accounted for.

Executive Functioning. Results of a MANOVA using executive functioning measures revealed a significant main effect of RISK [F(6,386)=2.34, p<.05] (see Table 5). Univariate ANOVAs indicated that the four groups differed significantly on both the Abstract Planning Index (API) [F(3,194)=3.54, p<.05] and Abstract Reasoning Index (ARI) [F(3,194)=2.82, p<.05]. Post-hoc comparisons using the Tukey's B Test showed children from AAL families had a significantly lower score on the API and ARI than children from control families. No differences were found between children from NAAL-cur, NAAL-rem, and control families.

Results of a MANCOVA with SES as the covariate revealed a trend toward an overall main effect of RISK [F(6,380)=2.00, P<.07], with univariate ANCOVAs showing continued significant group differences for the API [F(3,191)=2.88, p<.05] and a trend for the ARI [F(3,191)=2.46, p<.07]. An additional MANCOVA was conducted with child's Full Scale IQ (residualized without the variance from API and ARI) added as a second covariate to determine

Table 5

Multivariate Analysis of Variance for Executive Functioning Measures (N=198)

	Risk Group				
	AALs (n=30) M (SD)	NAALs-cur (n=64) M (SD)	NAALs-rem (n=38) M (SD)	Controls (n=66) M (SD)	F
Abstract Planning Index – API [1]	21.4 (4.9)	23.9 (5.2)	22.7 (4.1)	24.6 (4.7)	3.54* [a]
Abstract Reasoning Index – ARI [2]	21.4 (4.8)	22.9 (5.8)	22.5 (5.3)	24.6 (5.6)	2.82* [a]

[1] API = Mazes Scaled Score + Picture Arrangement Scaled Score
[2] ARI = Block Design Scaled Score + Similarity Scaled Score

*$p < .05$

[a] AALs < Controls, Tukey's B

whether group differences existed once variability in general intelligence had been accounted for. The results revealed a continued significant main effect of RISK [F(6,378)=29.7, p<.01]. Univariate ANCOVAs showed the four groups differed significantly on the ARI [F(3,190)=9.43, p<.01].

Impulsivity. A MANOVA was conducted to examine main effects of RISK on impulse control. Table 6 shows the results of the MANOVA, which revealed a significant main effect of RISK [F(6,386)=2.89, p<.01]. Univariate ANOVAs showed that the four groups differed significantly on Total Comments score [F(3,194)=3.98, p<.01]. Post-hoc comparisons using Tukey's B Test indicated children from AAL families made significantly more comments than children from NAAL-cur and control families.

Results of a MANCOVA with SES as the covariate also showed a main effect of RISK for impulsivity measures [F(6,380)=3.00, P<.01]. Univariate ANCOVAs revealed significant group differences for Total Comments score [F(3,191)=4.21, p<.01]. An additional MACOVA was conducted with child's Full Scale IQ added as a second covariate to determine whether group differences existed once variability in general intelligence had been accounted for. The results showed a continued significant main effect of RISK [F(6,378)=2.26, p<.05], with univariate ANCOVAs indicating significant group differences on Total Comments score [F(3,190)=2.75, p<.05].

Table 6

Multivariate Analysis of Variance for Impulsivity Measures (N=198)

	Risk Group				
	AALs (n=30) M (SD)	NAALs-cur (n=64) M (SD)	NAALs-rem (n=38) M (SD)	Controls (n=66) M (SD)	F
Total Comment [1]	4.7 (7.7)	1.8 (4.3)	2.2 (5.1)	1.2 (2.4)	4.22* [ab]
Time Delay in sec [2]	80.3 (23.6)	89.3 (6.8)	83.5 (20.9)	83.5 (22.3)	2.06

[1] Total Comments = number of comments the child made during the task.
[2] Time Delay = Child's ability to delay gratification when presented with a gift-wrapped toy.

*$p < .10$

[a] AALs > NAALs-cur, Tukey's B
[b] AALs > Controls, Tukey's B

Correlational Analyses

Table 7 presents the results of correlational analyses that explored the relationship between impulse control and cognitive functioning. For children of AALs, the Total Comments score was found to be significantly negatively correlated with Verbal IQ ($r=-.36$, $p<.10$), Performance IQ ($r=-.45$, $p<.05$) and Full Scale IQ ($r=-.49$, $p<.01$). In addition to being related to IQ scores, Total Comments was also more strongly related to ARI score for children of AALs ($r=-0.47$, $p<.01$). Correlations between Total Comments and academic achievements were non-significant.

For children of NAALs-cur, Total Comments was found to be significantly negatively correlated with Performance IQ ($r=-.28$, $p<.05$) and Full Scale IQ ($r=-.22$, $p<.10$), as well as the Abstract Reasoning Index ($r=-.33$, $p<.01$). In addition, Total Comments was significantly correlated with Spelling score ($r=-.22$, $p<.10$) for children of NAALs-cur. For children of NAALs-rem, Total Comments was found to be significantly negatively correlated with Reading ($r=-.37$, $p<.05$) and Spelling score ($r=-.45$, $p<.01$). For children from controls families, none of the correlations between Total Comments and cognitive measures were significant.

Discriminant Function Analysis

A multivariate discriminant functions analysis was conducted to test the hypothesis that a specified set of cognitive characteristics could help distinguish children in the four groups. Seven cognitive measures were chosen as predictor variables: WRAT-R Reading and Arithmetic scores, Performance and Verbal IQs,

Table 7

Correlations Between Impulsivity and Cognitive Functioning

	AALs (n=30)	NAALs-cur (n=64)	NAALs-rem (n=38)	Controls (n=66)
Total Comments and IQ				
Verbal IQ	-.36*	-.08	.25	-.09
Performance IQ	-.45**	-.28**	.11	-.16
Full Scale IQ	-.49***	-.22*	.21	-.14
Total Comments and Executive Functioning				
Abstract Planning Index	-.29	-.18	-.04	-.13
Abstract Reasoning Index	-.47***	-.33***	-.04	-.12
Total Comments and Academic Achievement				
Reading	.04	-.20	.37**	.07
Spelling	-.04	-.22*	.45***	.17
Arithmetic	-.30	-.07	.26	.08

*$p < .10$; **$p < .05$; and ***$p < .01$

Table 8

Results of Discriminant Function Analysis for Familial Alcoholism Subtypes

	Standardized Coefficient		
Variables	Function 1	Function 2	Function 3
WRAT-R Reading	0.26	-0.01	0.77
WRAT-R Arithmetic	0.07	0.79	-0.36
Performance IQ	0.69	-0.28	0.54
Verbal IQ	0.52	-0.87	-0.57
Total Comments	-0.58	0.36	0.45
API	0.02	-0.05	0.30
ARI	-0.66	1.20	-0.31

	Group Centroids		
Category	Function 1	Function 2	Function 3
AALs	-0.83	0.13	0.07
NAALs-cur	0.13	-0.16	0.09
NAALs-rem	-0.19	-0.11	-0.19
Controls	0.36	0.16	-0.02

Table 9

Discriminant Function Analysis Predictions of Familial Alcoholism Subtype from Factors

	Predicted Group Membership			
	AALs	NAALs-cur	NAALs-rem	Controls
Actual Groups				
AALs	**14**	6	7	3
(n = 30)	**46.7%**	20.0%	23.3%	10.0%
NAALs-cur	12	**22**	8	22
(n = 65)	18.8%	**34.4%**	17.2%	34.4%
NAALs-rem	10	4	**16**	8
(n = 39)	26.3%	10.5%	**42.1%**	21.1%
Controls	11	12	9	**34**
(n = 64)	16.7%	18.2%	13.6%	**51.5%**

Percent of "grouped" cases correctly classified: 43.43%

Abstract Planning and Reasoning Indexes, and Total Comments score on the Delay of Gratification. All variables were entered independently and were used simultaneously to determine the group membership (AAL, NAAL-cur, NAAL-rem, and control) of the children. Wilks' Lambda and univariate F-ratio statistics showed that Performance IQ was the most important variable to the analyses [Wilks' Lamda=.92; $F(3,194)=5.66$, $p<.01$] followed by Total Comments [Wilks' Lambda=.094; $F(3,194)=3.97$, $p<.01$].

Three functions were generated using the seven variables (see Table 8). The first function accounted for eighty-three percent of the variance and had a canonical correlation of 0.37. The second function accounted for eleven percent of the variance and had a canonical correlation of 0.15. The last function accounted for five percent of the variance and had a canonical correlation of 0.10. None of the three canonical discriminant functions generated from the analysis were significant. The relative contribution of each factor to the three functions are indicated by the absolute values of the weights shown in Table 9. Group centroids after the final step of the analyses are also shown in the same table. Overall, only 43% of the children were correctly classified in comparison to 25% at chance level. Children from AAL and control families were the most distinguishable among the four groups with 47% and 52%, respectively, correctly classified (see Table 9).

Hierarchical Multiple Regression Analysis

In order to determine which variables might be predictive of cognitive deficits and impulsivity in COAs, a series of hierarchical multiple regression analyses were performed. Full Scale IQ and Total Comments were selected as the dependent variables. Two regression analyses were conducted using a three-step design for entering the predictors. Family sociodemographic (SES) was entered on the first step, parental IQ (mother and father IQ) on the second step and risk group status on the third step for the regression utilizing Full Scale IQ as the dependent variable. Child age was also entered on the first step for the regression utilizing Total Comments as the dependent variable, as Total Comments was not standardized within age groups. This procedure was intended to test whether familial alcoholism subtype (RISK) significantly predicted COAs' cognitive functioning and impulsivity after variance attributable to family SES and parental IQ was accounted for.

Table 10 presents the results of the regression analysis for variables predicting child's Full Scale IQ. The overall regression equation was significant, $F(4,191)=12.14$, $p<.01$ and accounted for 20% of the variance in child intellectual functioning. SES was found to account for 11% of the variance. Parental IQ also had a significant relationship with child's Full Scale IQ accounting for an additional 7% of the variance. However, only mother IQ was significantly related to child's Full Scale IQ. Finally, family alcoholism subtype (RISK) significantly predicted children's Full Scale IQ accounting for 2% percent of the variance after

Table 10

Summary of Hierarchical Regression Analysis for Variables Predicting Child's Full Scale IQ (N = 196)

Variable	B	SE B	β
Step 1			
SES	0.02	0.01	.18*
Step 2			
Mothers' Full Scale IQ	0.18	0.08	.18*
Fathers' Full Scale IQ	0.13	0.07	.14
Step 3			
Risk Group	2.00	0.86	.16*

*p < .05.

	F change	R^2 change	Cumulative R^2
Step 1	22.86**	0.10	0.10
Step 2	14.07**	0.07	0.18
Step 3	12.14**	0.02	0.20

**p < .01

Table 11

Summary of Hierarchical Regression Analysis for Variables Predicting Total Comments score (N = 196)

Variable	B	SE B	β
Step 1			
SES	$4x10^{-3}$	$3x10^{-2}$	.10
Age	-1.04	0.33	-.22**
Step 2			
Mothers' Full Scale IQ	-0.05	0.03	-.16*
Fathers' Full Scale IQ	-0.01	0.03	-.05
Step 3			
Risk Group	-0.81	0.32	-.18*

*p < .05; **p < .01.

	F change	R^2 change	Cumulative R^2
Step 1	3.87*	0.04	0.04
Step 2	4.16**	0.04	0.08
Step 3	4.75**	0.03	0.11

*p < .05; **p < .01

all other variables were accounted for.

Table 11 presents the results of the regression analysis for variables predicting Total Comments score (impulsivity). Child Age was included as a demographic variable in this regression equation because Total Comments scores were not standardized by age. The overall regression equation was significant, $F(5,190)=4.75$, $p<.01$. In step one, SES and Child Age together accounted for 4% of the variance but only Child Age was significantly related to impulsivity. Paternal IQ also had a significant relationship with Total Comments accounting for an additional 4% of the variance. However, t-test results showed that only mother IQ was significantly related to Total Comments. Finally, RISK was found to be significantly related to Total Comments accounting for 3% of the variance after all other variables were accounted for.

To determine whether the density of alcoholism in the child's family also predicted cognitive deficits and impulsivity, two additional regression analyses were conducted in which family expression of alcoholism (FEA) was entered on the third step along with the RISK variable. The results showed that RISK and FEA together accounted for an additional 2% of the variance in Child Full Scale IQ and 3% in Total Comments, the same amount of variance that RISK alone had accounted for. In addition, t-tests did not show that FEA was significantly related to either Child Full Scale IQ or Total Comments.

DISCUSSION

Recent literature has suggested that cognitive deficits may precede the onset of alcoholism and are part of the risk matrix for the development of alcohol use disorders (West & Prinz, 1987; Sher, Walitzer, Wood, & Brent, 1991; Noll, Zucker, Fitzgerald, & Curtis, 1992). However, studies which have examined the cognitive functioning of children of alcoholics (COAs) are marked by inconsistency, with some studies unable to document cognitive compromise among COAs (Alterman, Searles, & Hall, 1989; Johnson & Rolf, 1988; Schuckit, Butters, Lyn, & Irwin, 1987). There is increasing evidence that indicates alcoholism is a heterogeneous disorder with various subtypes each having a different onset, course, prognosis and etiology (Cloninger, 1987; Zucker, Ellis, & Fitzgerald, 1993; Zucker, Fitzgerald, & Moses, 1995). However, few studies have conceptually integrated these two areas and examined the relationship between familial alcoholism subtype and cognitive performance of COAs, particularly within a developmental framework using young COAs.

The present study extended earlier research by showing that poorer general intellectual functioning previously documented among COAs as compared to non-COAs is present in the early elementary school years. Results of this study are consistent with an earlier set of analyses from the MSU-UM Longitudinal Study (Noll, Zucker, Fitzgerald & Curtis, 1992) which found lower global developmental quotient as well as lower adaptive and language scores among

preschool aged COAs.

In addition, findings confirmed that certain groups of COAs are at highest risk for such negative cognitive outcomes. Specifically, children of AALs obtained significantly lower Performance and Full Scale IQ scores than children of NAALs-cur and controls. Overall, children of AALs displayed the poorest global cognitive functioning, supporting the hypothesis that children from AAL families are most susceptible to relative cognitive deficits. Moreover, children of NAALs-rem also exhibited lower Performance and Full Scale IQ than children of controls, indicating that children of NAALs are also susceptive to relative cognitive deficits although the deficits may be less extensive.

Results of the present study also showed that Verbal IQ was worst among children of AALs, while children of NAALs and controls did not differ in verbal abilities. Such relative deficits in verbal processing skills could place children of AALs at higher risk for a variety of problems, including poor peer relations (Skarpness & Carson, 1986; Gallagher, 1993) and poor academic achievement (Tarter, Jacob, & Laird, 1993; Kaplan, 1993; Dash, Mohanty, & Kar, 1989). These may in turn lead to other deviant behaviors in adolescence including earlier, more problematic alcohol use (Spoth, Redmond, Hockaday, & Yoo, 1996; Ohannessian & Hesselbrock; 1993; Schulenberg, Bachman, O'Malley, & Johnston, 1994; Ellickson & Hays, 1991).

Although children from AAL families had the poorest general cognitive functioning, mean scores on IQ tests were well within normal limits. Other

studies have reported that COAs generally fall in the normal range on intelligence tests even though their scores are significantly lower than non-COAs (Gabrielli & Mednick, 1983; Ervin et al., 1984; Bennett, Wolin, & Reiss, 1988). Further, the hypothesis that increased base rates of cognitive dysfunction (i.e. borderline or lower IQ) would be found among children of AALs as compared to controls was not supported. Again, this may suggest that although children of AALs have relatively poorer cognitive abilities, they do not have increased risk for problems such as borderline intellectual functioning or mental retardation. However, it is important to note that there is strong evidence that intelligence has been rising among children (and the population in general) in recent decades (Lynn, 1994; Fuggle, Tokar, Grant, & Smith, 1992; Emanuelsson & Svensson, 1990; Flynn, 1984). For instance, Lynn (1994) reported that IQ scores among American children have increased by 3.5 points between 1978 and 1989. Therefore, an alternative explanation for the present findings is that since the intelligence test use in the current study (the WISC-R) was normed and standardized in 1974, IQ scores were somewhat inflated, causing difficulties in identifying children whose intellectual abilities fell outside the average range.

As predicted, children of AALs showed the poorest scores on all measures of academic achievement. No such differences were found once variance from IQ was accounted for, suggesting that academic difficulties during early school years may be closely linked to general intelligence. Contrary to prediction, children of NAALs did not perform worse than children of controls. Previous studies have

documented poorer academic achievement among COAs (Bennett, Wolin, & Reiss, 1988; Ervin et al., 1984; Hegedus et al., 1984); however, the present study clearly showed the presence of such difficulties as early as first and second grade. It appears that children of AALs are at the highest risk for academic difficulties, which may lead to repeating grades or ultimately to dropping out of school. In an earlier study, Schuckit and Chiles (1978) found that children of parents who were both alcoholic and antisocial had the lowest grade point average and were most likely to repeat a grade in school. Furthermore, Rhodes and Jasinski (1990) found that alcoholic men with a positive family history of alcoholism were more likely to meet the academic discrepancy criteria for learning disabilities. In the future, it will be important to document problems such as rates of formal learning disabilities in this sample as children progress further in school.

The present study also predicted differences in executive functioning among children of AALs, NAALs, and controls; this hypothesis was partially confirmed. Results showed that children from AAL families exhibited significantly poorer abstract planning and reasoning skills than children from control families. The effect of risk status upon abstract reasoning ability remained after variance due to SES and general intelligence was accounted for. Children of AALs were also found to have the highest level of impulsivity as compared to children of NAALs-cur and controls. These findings suggest that early school-aged children of AALs have specific impairments in executive function believed to be mediated by prefrontal brain cortex. These impairments place them at

particular risk for the development of a variety of behavioral problems including attention deficit/hyperactivity disorder, conduct disorder, and later alcoholism (Werner, 1986; Tarter, 1988). Sher (1991) suggested that impulsivity may also directly mediate the relationship between COA status and problem drinking, as individuals with an impulsive cognitive style are particularly likely to use drinking as a coping mechanism because of its immediate reward value.

Although there were no significant differences between children of NAALs-cur and NAALs-rem on cognitive measures, it should be noted that children of NAALs-cur performed better on measures of intelligence, academic achievement and executive functioning than children of NAALs-rem. This finding is somewhat surprising; the initial separation of children of NAALs into two subgroups was made due to the assumption that children of NAAL fathers who were currently alcoholic would be at higher risk for cognitive difficulties than children of NAAL fathers who had stopped drinking at a problematic level. Several studies have reported reduction of negative outcomes in COA after the recovering of alcohol problems (Moos & Billings, 1982, Chassin et al., 1991; Puttler, 1996). In one study, Puttler (1996) found that daughters of recovering alcoholics had higher IQs, spelling ability, and lower levels of externalizing and total behavioral problems than those of active alcoholics. One possible explanation for the current finding is that lower parental IQ may have served as a more proximal risk factor for poorer cognitive outcome among children of NAALs-rem than current paternal drinking status. Demographic information

revealed that both mothers and fathers in the 'remission' group had higher IQs than those in the 'current' group. As the two groups of children of NAALs never differed significantly from one another, it remains for future research to determine the relative importance of current paternal alcoholism to offspring's cognitive outcome as compared to a lifetime history of paternal alcoholism.

Differences were also documented in the degree of relatedness between impulsivity and various cognitive variables in the current sample. For all children of AALs and NAALs-cur, high levels of impulsivity were significantly associated with poor visuo-motor abilities as well as impaired executive functioning as measured by abstract reasoning skills. In addition, impulsivity was significantly negatively correlated with verbal abilities among children of AALs. These finding can be explained in a number of ways. First, children with poor impulse control may have a more difficult time staying on task, especially tasks that are more challenging (i.e. IQ tests; Baer, Novick, & Hummel-Schluger, 1995), resulting in poor learning and performance. Alternatively, low cognitive abilities may lead to poor problem solving ability and behavioral undercontrol. For instance, Peterson, Finn, and Pihl (1992) reported that sons of male alcoholics displayed a pattern of automatic hyper-reactivity to a variety of stimuli and concluded that cognitive deficits may underlie such hyper-reactivity. However, it is also possible that the same underlying brain substrates cause both behavioral dysregulation and poor cognitive functioning among children of AALs, which then may act synergistically to worsen later psychosocial outcomes. Surprisingly, impulsivity

was not significantly related to abstract planning abilities among COAs. Perhaps Total Comments reflects a different aspect of impulse control ("verbal" impulsivity) as compared to the API (motoric disinhibition). The lack of relatedness between impulse control and cognitive functioning among control children found in the present study is also intriguing and suggests the possibility that behavioral undercontrol and poor intellectual abilities are less clearly linked for non-COAs, although more restricted range on the impulse control variable among controls may account for these findings as well.

Impulsivity was not significantly associated with academic achievement for children AALs and controls. For children of NAALs-cur, impulsivity was significantly negatively correlated with spelling scores. Perhaps the most surprising finding is the observation that impulsivity was positively correlated with reading and spelling scores for children of NAALs-rem. This finding is somewhat unexpected since many studies have reported a strong relationship between academic problems and poor impulse control (Dawes, Tarter, & Kirisci, 1997; Weithorn, Kagen, & Marcus, 1984; Karmos, 1981). Possibly, lower academic achievement in the early elementary school years may be more purely related to poor intellectual functioning or other factors; thus, the impact of impulsivity may not be not as salient during this age period. Future data collection with the present sample will clarify whether the relationship between poor impulse control and low academic achievement will become more apparent at later ages.

Despite clear evidence for differences in outcome among the children in the

current study, results fail to identify a discrete set of cognitive characteristics that would characterize children of AALs, NAALs or controls. A limited set of cognitive variables, several of which were significantly correlated with each other, were available for use in the present discriminant function analyses; this may have reduced the likelihood of capturing a unique cognitive profile for children from each family subtype. Nonetheless, a higher percentage of children from AAL families were correctly classified, suggesting that it may be easier to identify a specific cognitive profile among children of AALs that puts them at risk for alcoholism. At future waves of data collection, more comprehensive neuropsychological testing will be completed, which may increase the likelihood of capturing such a profile for children of AALs.

Finally, regression analyses showed that family SES and parental intellectual functioning were important in predicting children's general intelligence and impulsivity. In particular, maternal, but not paternal IQ, was predictive of child outcome. This is a robust finding from other studies (Plomin & DeFries, 1985; Yeates, MacPhee, Campbell, & Ramey, 1983) and likely reflects the higher level of involvement of mothers in early child rearing. It is important to note that when entered on the last step of the regression analyses, risk group status continued to account for variance in child intellectual ability and impulsivity, suggesting that the family subtype marker was useful in predicting child outcome over and above family SES and parental IQ. However, only a small percent of variance in child outcome was accounted for in each of the two regression

equations (20% for Child's Full Scale IQ and 11% for Total Comments score). It is likely that other variables not included in this study (e.g. amount of stimulation in the home during early development, exposure to prenatal toxins such as alcohol) may also be relevant to the cognitive performance of COAs.

Limitation of the Study

The current study provides strong support for the idea that risk for cognitive dysfunction found in COAs varies as a function of familial alcoholism subtype and that the presence of paternal antisocial alcoholism is a useful marker for predicting early cognitive problems as well as the types of early behavioral and emotional problems documented in previous studies (Ellis, Zucker and Fitzgerald, 1997). However, it is important to note that there are several limitations to the present work. First, the cognitive measures used in this study were somewhat limited in scope and focused mainly on global functioning. It is possible that other cognitive deficits that are more subtle exist among children of alcoholics but were not detected in this study. Obtaining a complete profile of the neuropsychological functioning of COAs may help clarify the exact nature of such deficits as well as providing additional information regarding brain-behavioral linkages.

Moreover, the current study only examined the cognitive functioning of COAs at a particular point in time. Cognitive abilities of children at this age are still developing rapidly. It is also conceivable that the differences in cognitive functioning between children of AALs, NAALs and controls may increase over time. As with the majority of the literature on cognitive functioning in COAs, the

present study included only male children of alcoholics. However, the larger study from which the present sample was drawn is currently tracking a group of female COAs, which will allow future research regarding cognitive functioning in this understudied population.

Finally, the subtyping approach used in the present study was useful in showing that heterogeneity in alcoholic families needs to be accounted for when studying the cognitive outcome of COAs. However, there are some disadvantages to this method. First, such a categorical approach does not account well for the fluid nature of risk and the fact that risk factors may change over time. For example, it is likely at future waves of data collection that some fathers will no longer fit a given subtype due to changes in their drinking status (from alcoholic to non-alcoholics and vice versa). It is also important to note that a subtyping approach does not allow conclusions to be drawn regarding the relative contribution of paternal antisociality and paternal alcoholism to the poorer cognitive functioning observed among children of AALs. Cognitive deficits have also been documented among adolescents whose parents exhibited antisocial personality disorder in the absence of substance abuse. Future work may therefore use alternative approaches to when attempting to determine the etiology of cognitive deficits among young COAs.

Finally, as children in the current study were only six to nine years old, linkages between cognitive functioning and later alcohol problems could not be directly assessed. Again, it is important to continue tracking these children in

order to clarify various pathways into later alcoholism.

Summary

The current study showed that differences in cognitive functioning and academic achievement between COAs and non-COAs are present in the early elementary school years. It also demonstrated that the inconsistent findings from the literature on cognitive functioning in COAs may in part be explained by the fact that heterogeneity of risk among alcoholic families is seldom accounted for. Specifically, children of AALs displayed the worst general cognitive functioning and academic achievement as compared to children of NAALs and controls. In addition, children from AAL families exhibited relative deficits in verbal processing abilities; no such difference was found between children of NAALs and controls. The fact that children of AALs had the poorest abstract planning abilities and highest level of impulsivity suggests that these children may have specific deficits in frontal lobe functioning, which may also put them at greater risk for developing later peer and behavioral problems. It appears that elementary school aged children of AALs may already be launched into a trajectory which places them at heightened risk for deviant behaviors in later life, including problematic alcohol use.

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