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THE NEUROPSYCHOLOGICAL OUTCOME OF COMMUNITY ALCOHOLICS: PSYCHIATRIC DISORDERS, NEUROMEDICAL PROBLEMS, AND DRINKING HISTORY

By

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ABSTRACT

THE NEUROPSYCHOLOGICAL OUTCOME OF COMMUNITY ALCOHOLICS: PSYCHIATRIC DISORDERS, NEUROMEDICAL PROBLEMS, AND DRINKING HISTORY

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This study examined the validity of three different neuropsychological models of alcohol abuse: the diffuse dysfunction model, the right hemisphere deficits model, and the frontal dysfunction model. In addition, three potential sources of variance that might influence the neuropsychological outcome of alcoholics were explored: psychiatric disorders, neuromedical risk, and drinking history. Participants were 327 adults drawn from the University of Michigan – Michigan State University Longitudinal Study. Neuropsychological functioning was assessed using the Wechsler Adult Intelligence Test – Revised, the MicroCog, and the Symbol Digit Modality Test. Results showed that alcoholics performed poorly on a wide range of neuropsychological measures. Moreover, perceptual motor functioning appears to be most sensitive to alcohol abuse. Among all the drinking variables, chronic history of alcoholism was the most reliable predictor for neuropsychological outcome. Path analysis revealed that depression mediated the relationship between history of alcoholism and perceptual motor functioning. In addition, higher level of current alcohol problems predicted poorer performance in visuospatial functioning. The current study extended earlier research by showing that poorer neuropsychological

performance previously documented among alcoholics in clinical populations is present in a community-based population of alcoholics. Although the pattern of deficits seems to be most consistent with the diffuse brain dysfunction model, none of the three theoretical provided a definitive framework to describe the effect of alcohol on brain functioning. Further, the results indicated that the direct neurotoxic effect of alcohol is partially moderated by other alcohol-related factors including depression and drinking pattern. In light of the current findings, it appears that past approaches to studying the neuropsychological functioning of alcoholics namely, comparing the three different theoretical models, may not capture the full range of possible neuropsychological effects of sustained and/or intensive alcohol consumption. Future research should focus on developing a more comprehensive theory that incorporates the both the direct and indirect effect of alcoholism on neuropsychological functioning.

To my mom and sister, your love and support are the foundation of my success.

To my dad, I will always miss you.

To Eric, my life is blessed with love and happiness because of you.

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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). In Michigan, 51 percent of adolescent (9-12th grade) reported that they are current drinkers (Michigan Department of Community Health, 1997). 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, 1997). These statistics indicate that alcoholism is one of the major health problems in the United States.

One of the adverse consequences of chronic alcoholism is brain impairment`. Neuroimaging and neuroradiological studies have shown that cortical atrophy, ventricular dilation, and reduced brain weight are commonly observed among chronic alcoholics (Rourke & Loberg, 1996). In addition, alcoholics have been found to perform poorly on various neuropsychological tests that are sensitive to brain damage (Charness, 1993). While the neurotoxicity of alcohol is well established, the exact mechanism of such effects is still relatively unknown. Three different theoretical models have been proposed to describe the specific action of alcohol on the brain. First, the diffuse dysfunction model suggests that alcohol abuse/dependence causes non-specific neurological damage (Goldstein & Shelly, 1982). The second model proposes that the right hemisphere of the brain is more prone to damage from alcohol abuse/dependence than the left hemisphere (Jones & Parsons, 1972). Finally,

the frontal lobe dysfunction model suggests that alcohol-induced brain damage is specifically concentrated in the anterior-basal region including the frontal, limbic and diencephalic structures (Tarter, 1975).

Other factors that are associated with alcoholism might also mediate the relationship between alcohol abuse and brain dysfunction. These factors include head injuries (HillBom & Holm, 1986), antisocial personality disorder (Waldstein, Malloy, Stout, & Longabaugh, 1996), depression (Shafer et al., 1991), and drinking patterns (O'Donnell, De Soto, & De Soto, 1994). It is likely that the etiology of neuropsychological deficits in alcoholics is multifactorial. Therefore, it is important that these alcohol-related factors are taken into consideration when evaluating the neuropsychological functioning of alcoholics.

The proposed study sought to examine the effects of chronic alcohol use on neuropsychological outcome among community alcoholic and non-alcoholic men. Specifically, the validity of three different theoretical models of alcohol effects on brain functioning was evaluated: diffuse dysfunction model, right hemisphere deficit model, and frontal lobe dysfunction model. It was hypothesized that community alcoholic men would show greater neuropsychological deficits than controls. Moreover, deficits in executive functioning would be most severe, supporting the frontal lobe dysfunction model. The current study also investigated variables that might contribute to the neuropsychological outcome in alcoholics. Three potential sources of variance were explored: depression, neuromedical risk (i.e. problems with nervous

system), and drinking history (i.e. history of alcoholism, current alcohol problems, and recent alcohol use).

REVIEW OF THE LITERATURE

Neuropsychological Functioning of Alcoholics

For many years, severe 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. The early clinical presentation of WKS includes global confusion, abnormal eye movements, and gait ataxia. Behavioral symptoms such as disorientation of time and place, apathy, and emotional blandness are also prominent among alcoholics with Korsakoff's psychosis. Alcoholics with WKS are also expected to suffer from severe memory deficits (Lezak, 1995). Specifically, Korsakoff patients have great difficulties learning new verbal and nonverbal information (anteriograde amnesia). In addition, they have trouble recalling historical events that occurred close to the time of onset of the disorder (retrograde amnesia). However, semantic memory (e.g. rules, general principle etc.) remains mostly intact, which may account for the preservation of general intelligence (Bolden, 1994; Butters & Cermak, 1980). Other neuropsychological deficits, including visual-spatial (Oscar-Berman, 1980), conceptual (Kovner, Mattis, Goldmeier, & Davis, 1981), executive functioning (Joyce & Robins, 1991), and psychomotor skills (Parsons & Nixon, 1993), have also been noted among alcoholics with WKS.

In a landmark paper, Courville (1955) reported that chronic alcoholics suffer widespread cortical atrophy and he argued that the damage is the result of

alcohol neurotoxicity rather than dietary deficiency. Since then, evidence from neuroradiological and neuropathological studies have indicated that there is a second kind of alcoholism-related brain impairment that is independent of the Korsakoff's Amnesia (Bergman, Borg, Hindmarsh, Idestrom, & Mutzell, 1980; Harper & Kril, 1990). Similarly, Butters and Salmon (1986) reported that cognitive deficits associated with chronic alcohol use (i.e. visual-spatial processing, abstraction, and problem solving) are independent of those related to WKS (i.e. Amnesia).

The relationship between cognitive dysfunction and alcoholism was further substantiated by studies that found alcoholics performed poorly on the Halstead-Reitan Neuropsychological Battery (HRNB), a set of measures that is sensitive to brain damage (Fitzhugh, Fitzhugh, & Reitan, 1965; Jones & Parsons, 1971; Smith, Burt, & Chapman, 1973). More recently, Tuck and Jackson (1991) reported that alcoholics with no neurological disorder performed significantly worse on various neuropsychological tests than matched controls. These results suggest that chronic alcoholism could cause cognitive impairment even before the onset of any clinical signs of neurological disorder.

The following section reviews studies that examined the neuropsychological functioning of alcoholics with no clinical signs of WKS.

Intellectual Performance

Typically, alcoholics with no signs of WKS are reported to have global intellectual abilities within the normal range (Gordon, Kennedy, & McPeake, 1988; Page & Schaub, 1977). However, when being examined with a cognitive

test like the Wechsler Adult Intelligence Scale (WAIS), alcoholics often show deficits in the performance subtests, which assess perceptual motor and visuospatial skills while their verbal abilities remain mostly intact (Parsons & Farr, 1981; Parsons & Leber, 1981; Goldman, 1986). For example, Loberg (1980) examined the neuropsychological functioning of male alcoholics and normal drinkers and found that alcoholics obtained average general intelligence but they had significantly lower scores than control subjects on several performance subtests including Block Design and Digit Symbol.

More recently, Hambidge (1990) examined the intellectual functioning of adult men ages 18 to 65 who were admitted to an inpatient psychiatric hospital for alcohol related problems. Results showed that more than half of the participants (58%) displayed visuospatial impairment on the WAIS. In contrast, only 3% of the participants exhibited verbal deficits. Similarly, Mahony and Doherty (1996) reported that detoxified alcoholics displayed impaired performance on Block Design and Digit Symbol of the WAIS and WAIS-R, whereas Vocabulary and Digit Span scores were within the normal range. These findings further support the notion that chronic alcoholics are more likely to display deficits in performance intelligence while their verbal intelligence is relatively unimpaired.

Learning and Memory

Early studies on memory functioning of alcoholics without WKS have yielded negative findings (Parsons & Prigatano, 1977; Ryan & Butters, 1980). For instance, Loberg (1980) reported that alcoholic men performed within normal

limits on the Wechsler Memory Scale (WMS-R) and did not exhibit any signs of gross memory deficits. More recent studies, however, have shown that alcoholics might suffer mild deficits in learning and memory. In one study, Ryan and Lewis (1988) investigated the validity of the WMS-R by comparing scores of recently detoxified chronic alcoholics with matched controls. Results indicated that alcoholics performed significantly worse on all five WMS-R index scores as compared to controls. Moreover, the pattern of performance was comparable between the two groups.

Other studies that looked at the different components of memory function (learning, recall, retention etc.) have also found deficits among alcoholics. Using a revised scoring method of the WMS, Nixon, Kujawski, Parsons, and Yohman (1987) compared the semantic and figural memory abilities of detoxified male alcoholics with control subjects. Findings indicated that both immediate and delay recall of verbal and figural materials were significantly worse among alcoholics than controls. Surprisingly, alcoholics did not show greater deficits on the recall of figural material than verbal material, suggesting that the levels of impairment might be comparable across modalities. Alcoholics and controls also did not differ significantly in rate of forgetting (retention ability), indicating that the ability to retain learned materials was relatively intact among alcoholics. The authors concluded that the memory deficit found in alcoholics might lie in the initial acquisition process.

More recently, Sherer, Nixon, Parsons, and Adams (1992) reported that alcoholics performed better on verbal memory functioning than brain damaged

patients. Moreover, alcoholics were significantly slower in acquiring verbal information, suggesting that alcoholic memory deficits might be the result of inferior acquisition processes rather than retrieval difficulties. Beatty, Hames, Blanco, Nixon, and Tivis (1995) also found that alcoholics displayed deficits on measures of anteriograde spatial memory (Figural Memory Test, Rey-Osterrieth Figure, and New Map Test). Overall, alcoholics had greater difficulties learning and remembering unfamiliar spatial objects than controls. The findings indicated that these deficits were related to both the failure to acquire spatial information and poor retention ability.

Some researchers have posited that alcoholism results in premature aging of memory functioning. Kramer, Blusewicz, and Preston (1989) tested this hypothesis by comparing the memory performance of younger alcoholics and older non-alcoholics. Using the California Verbal Learning Test (CVLT), the authors showed that the effects of alcoholism and aging on memory functioning were quite different. Although alcoholism and aging were found to be associated with measures of immediate and delay recall, younger alcoholics performed more poorly on recognition and produced more frequent intrusion and false positive errors than older non-alcoholics.

Visuospatial Ability

There is consistent evidence to support the finding that chronic alcoholics have visuospatial deficits. For example, using a paired-associate learning paradigm, Shelton, Parsons, and Leber (1984) examined the verbal and visuospatial abilities of chronic alcoholic patients and matched controls. Results

showed that alcoholics performed significantly worse than non-alcoholics on visuospatial learning. In contrast, no differences in verbal learning were found. In another study, Kramer, Blusewicz, Robertson, and Preston (1989) assessed the effect of chronic alcoholism on visuospatial processing ability using the Block Design test from the WAIS-R. Male chronic alcoholics were found to have greater difficulties completing the designs than controls. Analyses of the configuration patterns showed that alcoholics were more likely to make errors on the outer configuration of the design. These findings suggested that chronic alcohol use might have a negative effect on visuospatial information processing.

Beatty, Hames, Blanco, Nixon, and Tivis (1995) also reported that inpatient alcoholics performed poorly on several visuospatial measures including Block Design, Rey-Osterrieth Complex Figure, and Benton Line Orientation Test. Specifically, alcoholics made less accurate copies on the Rey-Osterrieth, indicating difficulties in judging position of objects in space in relation to one another. They also displayed poorer visuospatial scanning and construction abilities as compared to controls. Further analyses revealed that alcoholics were more likely to break configuration design on the Block Design test and made more searching errors on the Letter/Symbol Cancellation Task.

More recently, Sher, Martin, Wood, and Rutledge (1997) studied the relationship between alcohol use disorders and neuropsychological functioning in young adults. Five neuropsychological factors were examined: language/verbal memory, visuospatial ability, motor speed, Booklet Category performance, and attention. Results indicated that subjects who met criteria for alcohol use

disorders performed more poorly on measures of visuospatial ability than those with no alcohol diagnosis. Moreover, subjects with alcohol dependence showed greater deficits in visuospatial ability and motor speed as compared to those with alcohol abuse. Sher and his colleagues concluded that alcohol use disorders are associated with deficits in visuospatial ability and the severity of the deficits may depend on the type of diagnosis.

Executive Functioning

Detoxified alcoholics have been shown to exhibit problems in abstract planning and reasoning abilities, which are thought to be mediated by the frontal brain region (Grant, 1987, Rourke & Loberg, 1996). Using the California Card Sorting Test, Beatty, Katzung, Nixon, and Moreland (1993) studied the abstraction and concept formation abilities of a group of inpatient alcoholics and matched controls. Subjects were presented with multiple sets of six cards each and were asked to sort them into two groups with three cards in each group. Each card had several different features (e.g. size, shape, color, nature of words) and could be sorted into different groups based on the sorting principles. When sorting was completed, subjects were asked to explain the principle they used to sort the cards. Results showed that alcoholics identified fewer correct concepts as compared to controls, suggesting deficits in abstraction abilities. Moreover, alcoholics were unable to provide explanations of the concepts that they correctly identified. These findings indicated that alcoholics have difficulties isolating relevant information and eliminating non-relevant information. Beatty and his colleagues also reported that alcoholics made more perseverative sorts and

perseverative verbalization. It appeared that these errors are independent of the abstraction deficits and contributed to their overall difficulties in performing problem-solving tasks. Other studies using the Wisconsin Card Sort Test and Category Test have also shown that alcoholics exhibit deficits in abstract reasoning and perseveration errors (Adams et al., 1993; Grant & Reed, 1985; Ron, Acker, & Lishman, 1980; Steingass, Sartory, & Canavan, 1994; Sullivan et al., 1993).

Another neuropsychological test many studies have used to measure abstract planning is the Mazes test. This test provides a visuospatial 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 system. Bowden (1988) examined the performance of twenty male alcoholics using a test of complex maze learning and found that alcoholics performed worse than matched controls. Using the Porteus Maze Test, MacDonell, Skinner and Glen (1987) also found that chronic alcoholics had greater difficulties with planning ability than controls.

In summary, chronic alcoholics have been found to exhibit deficits in various neuropsychological functions including learning and memory, visuospatial ability, and executive functioning. While the poor cognitive outcome of alcoholics is relatively well established, the exact mechanism of such effects is still largely unknown.

Neuropsychological Models of Alcoholism

Acute alcohol intoxication has been shown to have a negative effect on cognitive performance (Golby, 1989). 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 theoretical models have been proposed to describe the specific action of alcohol on the brain.

Diffuse Dysfunction Model

First, the diffuse dysfunction model suggests that alcohol abuse/dependence might 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 diffused brain damage (Courville, 1955; Mancall, 1961). For instance, Lynch (1960) examined the brain 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.

Goldstein and Shelly (1982) compared the neuropsychological profile of patients with various types of brain damage (frontal, right hemisphere, or diffuse) with chronic alcoholic inpatients. The results indicated that neuropsychological impairments exhibited in alcoholics resemble the deficits found in patients with non-alcoholic diffuse brain damage rather than the deficits found in patients with frontal lobe damage. Moreover, the authors noted that the measures used in this study may not be selective enough to rule out specific damages to the brain (e.g. frontal lobe). Despite the significant findings, Goldstein (1987) warned that the diffuse dysfunction model is not sufficient to explain the pattern of neuropsychological deficits found among all alcoholics. He further suggests that genetic and antecedent cognitive functioning may play a role in the cognitive functioning of different alcoholic subtypes.

More recently, Tivis and Parson (1995) argued that the reason that chronic alcoholics do not show verbal deficits is because the tasks are often well learned and well rehearsed. The authors studied the verbal-spatial and visual-spatial functioning of alcoholics to determine if damages are present in both hemispheres. Results indicated that alcoholics performed worse on both verbal-spatial and visual-spatial tasks, suggesting that chronic alcohol use might affect brain functioning in a non-specific manner.

Right Hemisphere Deficit Model

The second model proposes that the right hemisphere of the brain is more prone to damage from alcohol abuse/dependence than the left hemisphere (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 task that are innervated by the right hemisphere. For example, Chandler and Parsons (1977) reported that acute alcohol intoxication impaired recognition and memory performance when material was presented to the left visual field, while the performance was equal to controls when material was presented to the right visual field. Moreover, several studies have indicated that chronic alcoholics displayed impaired performance on visual spatial tasks, functions that are thought to be mediated by the right hemisphere (Kramer, Blusewicz, Robertsons, & Preston, 1989; Parsons & Leber, 1982; Wilkinson, 1987).

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 subjects: detoxified chronic alcoholic men, right hemisphere damaged men, left hemisphere damaged men, and normal male controls. Analyses of block construction strategies and errors revealed that alcoholics did not suffer visuospatial impairment which 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.

Frontal Lobe Deficit Model

The frontal lobe deficit model suggests that alcohol-induced brain damage is specifically concentrated in the anterior-basal region including the frontal, limbic 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 and the damage is often more severe as compared to the rest of the brain.

Results of neuropsychological investigations on alcoholics also concur with the frontal lobe deficit model (Bergman, 1987; Gebhardt, Naeser, & Butters, 1984; Ron, 1987). For example, Steingass, Sartory, and Canavan (1994) examined the cognitive functioning of 105 chronic alcoholics between the age of 28 and 69. Results showed that chronic alcoholics suffer a decline in IQ and learning ability. Further analyses of the data revealed that alcoholics exhibit perseveration and impaired ability to find semantic categories, both of which are associated with frontal lobe dysfunction. Similarly, Gilman et al. (1998) 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.

Most recently, Ratti et al. (1999) reported that heavy drinkers (daily alcohol intake was more than 100 grams for at least the past 15 years) exhibited deficits in attentional abilities. In contrast, no differences between alcoholics and controls were noted on visuospatial measures. Neuroradiological data (localization of morphological cerebral changes) showed that alcoholics suffered widespread cortical and subcortical atrophy although the atrophy is more marked in frontal lobes than in the other structures. Ratti and her colleagues concluded that these findings were consistent with the frontal lobe deficit hypothesis.

Mediating Factors

Although there is evidence to support the claim that alcohol causes neuropsychological deficits, the exact mechanism is still relatively unknown. In addition, other factors might also have an influence on the neuropsychological outcome of chronic alcoholics. The following section reviewed studies that have examined the influence of comorbid psychiatric disorders, neuromedical problems, and drinking history on neuropsychological differences in alcoholics.

Psychiatric Disorders

The comorbidity of psychiatric disorders (e.g. personality disorders, depression, anxiety) among chronic alcoholics is quite common. For example, DeJong, Van den Brink, Harteveld, and Van der Wielen (1993) reported that 78% of hospitalized alcoholics had received at least one personality disorder diagnosis. More recently, Penick et al. (1994) investigated the lifetime comorbidity of major psychiatric disorders in male alcoholics drawn from six Veterans Administration Medical Centers. Results indicate that 62% of the

subjects met the Psychiatric Diagnostic Interview (PDI) criteria for alcoholism and at least one additional psychiatric disorder. Moreover, depression and antisocial personality were the most common co-occurring disorders reported by alcoholics (36% and 24% respectively).

A number of researchers have studied the relationship between depression and alcohol use disorders (Brown & Schuckit, 1988; Schuckit, Irwin, & Smith, 1994). For instance, Brown, Inaba, Gillin, Schuckit, Stewart, and Irwin (1995) examined the level of depressive symptoms among hospitalized patients who met diagnostic criteria for alcohol dependence and/or affective disorder. Upon admission, 42% of alcoholics reported experiencing depressive symptoms that reached clinical significant levels (based on the Hamilton Rating Scale for Depression). After 3 weeks of abstinence, only 6% of alcoholics continued to present elevated levels of depressive symptoms.

Depression has also been shown to affect the neuropsychological performance of alcoholics. For example, Loberg (1980) reported that depression was significantly related to impaired scores on Performance IQ in alcoholics. He later suggested that the poor performance might be linked to the lack of motivation and psychomotor retardation associated with depression (Loberg, 1986). Sinha, Parsons, and Glenn (1989) also studied the relationship between depression and neuropsychological performance in alcoholics. Results showed that depression, as measured by the Beck Depression Inventory, significantly correlated with the overall impairment index regardless of the family history of alcoholism. The authors concluded that depression could potentially be a

confounding factor in the relationship between alcohol use and cognitive functioning.

More recently, Schafer et al. (1991) conducted a longitudinal study that examined the role of depression on cognitive performance in detoxified male alcoholics. A brief neuropsychological battery (Trail Making Test, Digit Symbol and Vocabulary of WAIS, and Visual Search Test) was administered to all subjects upon admission to the hospital, before discharge, and 3 months afterward. Results showed that depression was a significant factor in predicting neuropsychological performance at admission. However, upon discharge (several weeks later), only premorbid intelligence significantly predicted neuropsychological scores. The authors concluded that levels of depression have a negative impact on the cognitive functioning of alcoholics.

The relationship between antisocial personality and alcoholism has also been studied extensively. In indeed, research has indicated that there are at least two different types of male alcoholics: antisocial and non-antisocial alcoholic (Cloninger, 1987; Zucker, Ellis, & Fitzgerald, 1993; Zucker, 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), brain systems that modulate behavioral responses to the effects of alcohol and other environmental stimuli may differ from those 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 measures (WAIS, WMS, 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.

More recently, Glenn, Errico, Parsons, King, and Nixon (1993) examined the role of antisocial, affective, and childhood behavioral characteristics in the neuropsychological performance of alcoholics. Although all subjects did not meet 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 path to neuropsychological impairment differed for antisocial and non-antisocial alcoholics. For antisocial alcoholics, cognitive deficits were predicted by less education, childhood symptoms of conduct disorder, drinks per day, and history of head injury. Conversely, 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 suggest that neuropsychological functioning of alcoholics may be mediated by co-occurring antisocial personality disorder.

Neuromedical Risk

Studies have indicated that poor physical health is associated with chronic alcohol use. For example, Glenn, Parsons and Stevens (1989) reported that alcoholics had significantly more health problems than community controls in four physical health domains: medical problems, alcohol-related disorders, trauma history, and drug use. Moreover, various medical problems including head injury and liver disease might also contribute to the neuropsychological deficits found in chronic alcoholics (Rourke and Loberg, 1996). In one study, Adams and Grant (1986) investigated the influence of neuromedical risk factors on neuropsychological functioning in recently detoxified alcoholics. Seven domains of neuromedical risk were evaluated: early developmental, learning disability, head injury, toxicity, neurological, anoxic, and sick risk. Subjects who endorsed one or more risk items were classified as "at risk". Results indicated that recently detoxified alcoholics with positive neuromedical risk performed worse than those without on all neuropsychological measures. Moreover, the impairment exhibited by recently detoxified alcoholics was beyond the additive effects of aging, alcohol status and risk. The authors concluded that neuromedical risk negatively affected the neuropsychological performance in the recently detoxified alcoholics and suggested that history of neuromedical risk might predispose alcoholics to neuropsychological deficits as a result of drinking.

Head injury is one of the neuromedical problems that might influence the neuropsychological performance of alcoholics. For example, HillBom and Holm (1986) reported that alcoholics who sustained traumatic brain injury performed

worse than those who did not admit to having suffered any brain injury on the Halstead-Reitan Neuropsychological Battery (e.g. Finger Tapping, Tactual Performance Test, Trails Making Test). No such differences were found in the control group. The authors concluded that head injury might cause more extensive damage to the brain in alcoholics than non-alcoholics.

Drinking History

Several studies have explored the relationship between drinking patterns and neuropsychological functioning. For example, Svanum and Schladenhauffen (1986) reported that increasing life time alcohol consumption was related to level of impairment on the Category Test and Trails Making B, suggesting deficits in higher cognitive functioning (i.e. set-shifting, concept formation). Further regression analysis revealed that lifetime drinking total predicted the level of impairment. In addition, increasing years of heavy drinking was found to be related to an increasing frequency of impairment on Halstead-Reitan tests. Eckardt, Stapleton, Rawlings, Davis, and Grodin (1995) also found that greater lifetime alcohol consumption predicted poorer performance on the several neuropsychological measures including the Boston Naming Test, Speech Sound Perception Test, and Rey-Osterrieth Complex Figure. In addition, alcoholics with longer period of abstinence (10 or more weeks) showed better neuropsychological performance than those with shorter period of abstinence (less than 2 weeks).

More recently, Horner, Waid, Johnson, Latham, and Anton (1999) examined the relationship between neuropsychological functioning and alcohol

consumption in alcoholics. Findings indicated that recent amount of alcohol consumption (last 3 months) was related to mild cognitive deficits in verbal memory and reaction time. Specifically, recent alcohol consumption was negatively correlated with verbal and visuospatial memory, executive functions, and cognitive speed. In another study, Beatty, Tivis, Stott, Nixon, and Parsons (2000) examined the relationship between neuropsychological functioning of alcoholics and consumption variables. Results showed that long-term (10 or more years) and short-term (4 to 9 years) alcoholics did not differ in the pattern of neuropsychological deficits (SILS Vocabulary and Abstraction Scales and Digit Symbol of WAIS-R) as both group performed more poorly than controls. Moreover, measure of recent drinking history accounted for almost 5 percent of the variance in neuropsychological performance of alcoholics. In contrast, no relationship between length of drinking and neuropsychological impairment was noted. Beatty and his colleagues cautioned that the neuropsychological measures they used were not complete and other measures such as Block Design and Object Assembly of the WAIS, which assess visuospatial information processing, might be more sensitive to detecting deficits in chronic alcoholics.

Length of abstinence might also have an influence on the neuropsychological functioning in alcoholics. Rourke and Grant (1999) reported that recently detoxified alcoholics exhibited various neuropsychological deficits including, abstract reasoning, learning, and complex perceptual-motor integration. Moreover, those who resumed drinking in the two-year follow-up period continued to show neuropsychological deficits in abstraction and cognitive

flexibility, complex perceptual-motor functioning, and simple motor skills. On the other hand, those who continued to stay abstinent showed significant improvement in abstraction and cognitive flexibility regardless of their age. Most importantly, the study showed that the neuropsychological performance of long-term abstinence alcoholics (average of 4.3 years of abstinence) was comparable to matched controls, suggesting that long-term abstinence might lead to normal neuropsychological status. The authors noted that the extent of the neuropsychological recovery might depend on the age at which the alcoholic stops drinking and the type of neuropsychological ability.

Based on the literature review, chronic alcohol use appears to have a negative impact on cognitive functioning. However, the underlying mechanisms mediating the relationship between alcoholism and neuropsychological deficits offer many possibilities. Several theoretical models have been proposed to explain the neuropsychological deficits observed in alcoholics, although none of them are able to definitively capture the relationship. Furthermore, factors other than the presumed direct neurotoxic effect of alcohol might influence the neuropsychological consequences of alcoholism (see Figure 1). Alcoholism is a multidimensional disorder with strong associations to other psychiatric disorders and health problems. These alcohol-related factors could play an important role in determining the neuropsychological functioning of alcoholics.

PROPOSED STUDY

The proposed study sought to examine the effects of alcohol on neuropsychological outcome among community alcoholics and non-alcoholics. The use of non-clinically accessed alcoholics made the sample more representative of alcoholics and their families than is generally true of treatment populations. Four domains of neuropsychological functioning (i.e. memory, visuospatial, perceptual motor, and executive functioning) were assessed using the MicroCog, the Wechsler Adult Intelligence Scale-Revised (WAIS-R), and the Symbol Digit Modality Test (SMDT). In addition, Full Scale IQ score from the WAIS-R and the General Cognitive Functioning Index from the MicroCog were used to assess general cognitive ability (see Figure 1). These measures were chosen specifically to examine the validity of the three different neuropsychological models of alcohol abuse as described in the literature review section. The diffuse dysfunction model suggests that alcoholics would show poorer general cognitive ability as well as relative deficits across all neuropsychological measures. The right hemisphere model predicts that alcoholics would perform worse on visuospatial tasks alone. In contrast, the frontal lobe deficit model predicts that alcoholics would show selective deficits in tasks that assess executive functioning.

The second goal of this study was to examine variables that might influence the neuropsychological outcome of alcoholics. Three major sources of variance were explored: psychiatric disorders, neuromedical risk, and drinking

history. The disorder that is of greatest interest is mood disorder, which was estimated by the Hamilton Depression Scale. Neuromedical risk was determined based on the number of nervous system related neuromedical problems subjects endorsed on the Health History Questionnaire. Finally, drinking history was assessed using the Drinking and Drug Use Questionnaire. Specifically, history of alcoholism, current alcohol problems, and recent alcohol consumption were used to assess subjects' drinking pattern.

HYPOTHESES

1. Neuropsychological deficits would be most severe among alcoholics who also met the DSM-IV diagnostic criteria for antisocial personality disorder (i.e. antisocial alcoholics). Moreover, executive functioning tasks would be most impaired, supporting the frontal lobe deficit model. Non-antisocial alcoholics would form an intermediate group that would show poorer neuropsychological functioning across all measures than controls.
2. Antisocial alcoholics would most likely be classified as suffering from "brain impairment" as compared to non-antisocial alcoholics and controls. Non-antisocial alcoholics would also more likely be classified as suffering from "brain impairment" than controls.
3. Drinking variables would mediate a poorer neuropsychological outcome. Moreover, history of alcoholism would be the strongest predictor among all drinking variables.
4. A greater history of alcoholism would directly predict increased neuromedical risks, higher current levels of depression and poorer memory functioning. In addition, increased current neuromedical risks and higher current level of depression would simultaneously predict poorer memory functioning.
5. A greater history of alcoholism would directly predict increased neuromedical risks, higher current levels of depression and poorer perceptual motor functioning. In addition, increased neuromedical risks and higher current

level of depression would simultaneously predict poorer perceptual motor functioning.

6. A greater history of alcoholism would directly predict increased neuromedical risks and higher current alcohol consumption. In addition, increased neuromedical risks and higher current alcohol consumption would simultaneously predict poorer visuospatial functioning.
7. A greater history of alcoholism would directly predict increased neuromedical risk, higher levels of current alcohol consumption and poorer executive functioning. In addition, increased neuromedical risk and current alcohol consumption would simultaneously predict poorer executive functioning.

METHOD

Participants

Subjects for the present study were drawn from the University of Michigan - Michigan State University Longitudinal Study (Zucker et al., 2000). 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. During the initial contact, all families were invited to participate in a long-term study of family and health and child development. Families were assessed at three-year intervals beginning at Wave 1 when the male target child (MTC) was age 3 to 5. All families received some payment for participation in each data collection interval.

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 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 fathers out of the same neighborhoods where drunk driver alcoholic fathers 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.

At the time this project was carried out, 332 adult from the UM-MSU Longitudinal Study had completed the neuropsychological battery at Wave 4. Five participants were omitted from the study due to their large percentages of missing data (over 20 percent). Table 1 presents a summary of the demographic information (gender, age, and years of education) for the sample.

Data Collection

Data were collected by trained project staffs who were blind to family risk status. In most cases, the data were collected during a single campus visit. The visit involved approximately four hours of contact time for each parent. Contacts included questionnaires sessions, semi-structured interviews and interactive tasks. The data used in this study came from Wave 1 to Wave 4.

Table 1.

Background Characteristics of Participants

	M	SD
Alcoholics		
Male (n=102)		
Age	42.99	5.47
Years of Education	13.78	2.64
Female (n=66)		
Age	39.77	4.59
Years of Education	13.85	2.38
Non-Alcoholics		
Male (n=51)		
Age	41.70	4.78
Years of Education	15.02	2.50
Female (n=108)		
Age	40.81	3.86
Years of Education	13.57	1.86

Measures

Alcoholism Diagnosis

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; Robin, Helzer, Croughan, Ratcliffe, 1980). 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). At subsequent waves, all subjects 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. For the present study, an alcoholic was defined as someone who met the DSM-IV criteria for alcohol abuse and/or dependence during the his/her lifetime.

Alcoholic Subtype

Alcoholic subtype was determined based upon alcoholism diagnosis and lifetime antisocial personality disorder (ASP) diagnosis. 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 at Wave 1. Unlike alcoholism,

which may remit, ASP, as an Axis II disorder, was presumed to be lifelong. Alcoholics with co-morbid ASP were classified as antisocial alcoholics (AALs) while those without ASP diagnosis were classified as non-antisocial alcoholics (NAALs). Finally, those who did not meet criteria for alcoholism and ASP diagnoses were classified as non-alcoholics. (controls).

Drinking Variables

Current Drinking. Two measures were used to assess subjects' current drinking problem: alcohol problems, and alcohol consumption. Both variables were gathered using the Drinking and Other Drug Use Questionnaire (Zucker, Fitzgerald, & Noll, 1990). This questionnaire incorporates already much tested items from the 1978 NIDA Survey (Johnston et al., 1979), from the American Drinking Practices Survey (Cahalan, Cisin, & Crossley, 1969) and from the V.A. Medical Center (University of California) San Diego, Research Questionnaire for Alcoholics (Schuckit, 1978). All of the items have been extensively used in a variety of survey and clinical settings. They provide data on drinking patterns including age of first drunkenness, quantity, frequency and variability of alcohol consumption, frequency of drug use, and multiple questions on consequences and troubles related to the use of these substances. Items have been carefully reviewed to yield information sufficient to provide diagnoses according to DSM-IV diagnostic criteria. Alcohol problems were determined based on the total number of drinking problems subjects endorsed on the instrument (31 items). Recent alcohol consumption (last month) was calculated based on the average number

of days per month subjects had a drink and the number of drinks on a day when they drank.

History of Alcoholism. Alcoholism diagnosis was coded based on the severity of the alcoholism (0 = no diagnosis, 1 = alcohol abuse, 2 = alcohol dependence without physical dependence, and 4 = alcohol dependence with physical dependence). History of alcoholism was created by adding each subject lifetime alcoholism diagnosis at Wave 1 and the three-year diagnosis at each subsequent Wave. This variable was designed to capture both the chronicity and the severity of the subjects' alcoholism.

Neuropsychological Domains

Table 2 presents a summary of the measures used in assessing neuropsychological functioning at Wave 4.

General Cognitive Functioning. Two measures were chosen to assess general cognitive ability: Full scale IQ from Wechsler Adult Intelligence Scale-Revised (WAIS-R; Wechsler, 1981) and the General Cognitive Functioning Index from the MicroCog (Powell, Kaplan, Whitla, Weintraub, Catlin, & Funkenstein, 1994). The short form of the WAIS-R was administered to each adult in the study. This test has a composite reliability of .88 and has been demonstrated to be a valid predictor of Full Scale IQ in normal adults (Reynolds et al., 1983) and in neurologically impaired individuals (Ryan, 1985). Evidence generally supports the use of the short form for research when characterizing group performance (Silverstein, 1990; Schretlen, Benedict, & Bobholz, 1994). The short form of the MicroCog is a computer administered and scored test that assesses global

Table 2. Neuropsychological Tests for Memory, Perceptual Motor, Visuospatial, Abstract Reasoning, and General Cognitive Functioning.

Memory	Perceptual Motor	Visuospatial	Abstract Reasoning	General Cognitive Functioning
1. Story Immediate*	1. SDMT (hand)	1. Clock*	1. Analogies*	1. Full Scale IQ**
2. Story Delayed*	2. SDMT (Oral)	2. Block Design**	2. Wordlist*	2. General Cognitive Functioning Index*
		3. Picture Completion**		

Note: * Tests drawn from the MicroCog; ** Tests drawn from the WAIS-R; SDMT - Symbol Digit Modality Test

integrity of neurocognitive functions (Powell et al., 1994). The protocol utilizes 12 subtests covering the following five domains: a) mental control, b) memory, c) calculation/reasoning, d) spatial processing, and e) processing speed. The subtests administered in the Short Form version include Number Forward and Reversed, Wordlist, Story 1 and 2 and Address, Analogies, Math Calculation, Clocks, and Timers. An index score representing global functioning is formed based on the subject's overall performance. The average reliability coefficient for this score is .94.

Memory Functioning. Two subtests from the MicroCog (Story Immediate Recall and Story Delayed Recall) were used to assess memory functioning. The two tests are analogous to those on the Wechsler Memory Scales. Green, Green, Harrison, and Kutner (1994) reported that immediate and delayed stories correlated moderately (.63) with immediate and delayed Logical Memory on the WMS-R. The reliability coefficients for Story Immediate Recall and Story Delayed Recall are .64 and .78 respectively.

Visuospatial Functioning. Three measures were selected to assess visuospatial functioning: Clocks from the MicroCog, Picture Completion from the WAIS-R, and Block Design from the WAIS-R. The Clocks subtest is similar to the traditional clock drawing task. Seven clock faces were presented in turn with hands but no numbers. The subject had to choose the correct time from among five choices. Picture completion is a task that required the subject to identify a missing portion of a picture. Block design is a task that required the subject to replicate geometric designs using colored blocks. The reliability coefficients for

these measures are .69 (Clocks), .81 (Picture Completion), and .87 (Block Design).

Perceptual Motor Functioning. Perceptual motor functioning was assessed using the Symbol Digit Modality Test (SDMT, Smith, 1991). This test was developed to evaluate cerebral dysfunction in children and adult. Numerous studies with over 1000 subjects have demonstrated the sensitivity of this neuropsychological screening test for detection of cerebral dysfunction. According to Rees (1979), scores 1.5 standard deviation below age norm means are suggestive of cerebral dysfunction. Nevertheless, such scores must be interpreted with caution since scores 1.0 standard deviation below age norm means are associated with rather high rates of false positives (9-15%). Significant impairment on both written and oral forms may indicate visual perceptual, visual scanning, or oculomotor deficits and/or general information processing impairment. Both the written and oral forms were given to each subject independently.

Executive Functioning. Three measures were used to assess executive functioning: Wordlist 1, Wordlist 2, and Analogies. All of the measures were drawn from the MicroCog. Wordlist 1 is task that required the subjects to press the Enter key whenever a word appears that belongs to a category specified in each of the four trials. Two categories are phonemic, and the other two are semantic. Wordlist 2 required the subjects to press the Enter key whenever one of the sixteen words from the first list appears on the screen. On the Analogies subtest, the subject was presented a series of 11 relationships and had to

choose among the choices displayed. The reliability coefficients for these measures are .91 (Wordlist 1), .92 (Wordlist 2), and .62 (Analogies).

Neuromedical Risk

The Health History Questionnaire (Carpenter & Lester, 1980) was used to assess personal health and illness status in fifteen areas: hospitalization history; current medication use; allergies; prior illnesses; skin and hair problems; eye, ear, nose, and throat symptoms; heart and lung; G.I. tracts; skeleton and joints; nervous system; alcohol and drug use; general health care patterns; diet and weight control; physical fitness activities; pregnancy and delivery. Neuromedical risk was determined based on the total number of endorsements for the following items drawn from the nervous system section: frequent or severe headaches, loss of balance or dizziness, loss of consciousness, head injury, and persistent numbness or tingling of hands or feet.

Depression

The Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960) was used to assess level of depression. This rating covered a variety of behavioral, affective, somatic, and psychological dimensions associated with depression, and the score is based on the subject's responses as well as the clinician's judgments. The clinician made both a current depression rating and a rating of the level of the subject's depression within the last three years when they were most depressed. The current studied used the worst ever rating for the past three year. Interrater reliabilities are ranged from .80 to .90 (Hamilton, 1960).

RESULTS

Missing Data

Three hundred and thirty-two adults from the UM-MSU Longitudinal Study had completed the neuropsychological assessment at Wave 4. Before beginning analyses, all variables were screened for missing data. Five participants were omitted from the analyses due to their large percentages of missing data (over 20 percent). In addition, two participants were excluded from the analysis due to missing antisocial personality disorder diagnosis. Data imputation was completed for all missing scores of the remaining 327 participants. A total of 293 (2.1%) data points out of the possible 14,147 were missing. All missing data were imputed using the Expectation-Maximization (EM) method of maximum likelihood (ML) estimation. The ML method was chosen because it is considered to be less biased and more efficient than traditional methods such as pairwise deletion and mean substitution (Arbuckle, 1996; Enders & Bandalos, 2001).

Descriptive Statistics

Thirty-eight percent of the female participants and sixty-six percent of the male participants met DSM-IV diagnostic criteria for alcohol abuse and/or dependence. A series of Multivariate Analysis of Variance (MANOVA) Tests were conducted to determine whether there were differences in age and education between groups based on alcoholism diagnosis (DIAGNOSIS). No significant differences were found on these variables [$F(2, 324) = 1.33$, $p = .27$]. Conversely, when the sample was categorized based on alcoholism subtypes (SUBTYPE), significant differences were noted [$F(4, 646) = 3.68$, $p < .01$].

Univariate Analyses of Variance (ANOVAs) revealed that the antisocial alcoholics (AALs), non-antisocial alcoholics (NAALs), and non-alcoholics (controls) differed significantly on both age [$F(2,324) = 3.76$, $p < .05$] and educational level [$F(2,324) = 4.48$, $p < .05$]. Post-hoc comparisons between the three groups using Tukey Test revealed that NAALs were significantly older than AALs. The results also showed that AALs had significantly fewer years of education than NAALs and controls (see Table 3). These results parallel earlier findings from the longitudinal study (e.g. Zucker, Ellis, Fitzgerald, Bingham, & Sanford, 1996).

Multivariate Analysis of Variance

Hypothesis 1

Hypothesis 1 stated that neuropsychological deficits would be most severe among AALs and that abstract reasoning tasks would be most impaired, supporting the frontal lobe deficit model. NAALs would form an intermediate group that would show poorer neuropsychological functioning across all measures than controls. To test this hypothesis, a series of MANOVAs were conducted for general cognitive functioning as well as measures for each of four neuropsychological domains. Scaled scores were used in the analyses to ensure that group differences were not attributable to age and educational level. Means and standard deviations for all dependent variables are presented in Table 3 – 7. Significance for all multivariate tests was determined using Wilks' Lambda. In addition, Tukey Tests were used to conduct post-hoc comparisons in cases where significant main effects were found.

Table 3.

Multivariate Analysis of Variance for Background Characteristics based on Alcoholism Subtypes – Antisocial Alcoholics (AALs), Non-Antisocial Alcoholics (NAALs), and Non-Alcoholics (Controls) (N=327)

	Alcoholism Subtypes			
	AALs (n=24) M (SD)	NAALs (n=144) M (SD)	Controls (n=159) M (SD)	F
Age	39.49 (5.04)	42.10 (5.34)	41.10 (4.18)	3.76*[a]
Years of Education	12.54 (1.53)	14.02 (2.61)	14.04 (2.18)	4.48*[a b]

* $p < .05$
[a] AALs < NAALs, Tukey Test
[b] AALs < Controls, Tukey Test

General Cognitive Functioning. A MANOVA design was used to examine the overall main effect of DIAGNOSIS on measures of general cognitive functioning including Full Scale IQ score and the MicroCog's General Cognitive Functioning Index. The results did not yield a significant main effect [F (2,324) = 1.79, p = .17]. A second MANOVA was conducted to determine if general cognitive functioning differed among alcoholism subtypes (SUBTYPE). The results were also found to be non-significant [F (4,646) = 1.92, p = .11] (see Table 4).

Memory Functioning. Two subtests from the MicroCog, Story Immediate Recall and Story Delay Recall, were used to assess memory functioning. MANOVA results showed a significant main effect of DIAGNOSIS [F (2,324) = 4.39, p < .05]. Univariate ANOVAs revealed that alcoholics performed significantly worse than non-alcoholics on Story Immediate Recall and Story Delay Recall. A second MANOVA was conducted to examine the effect of SUBTYPE on memory functioning. The overall main effect was marginally non-significant [F (4,646) = 2.20, p = .07]. Because of a trend effect, univariate ANOVAs were conducted and revealed a main effect of SUBTYPE on Story Immediate Recall. Post-hoc comparisons indicated that NAALs performed significantly worse than controls on Story Immediate Recall (see Table 5).

Visuospatial Functioning. Three measures were used to assess visuospatial functioning: Clocks, Picture Completion, and Block Design. MANOVA results revealed a significant main effect of DIAGNOSIS [F (3,323) = 2.85, p < .05]. Univariate ANOVAs indicated that alcoholics performed

Table 4.

Multivariate Analysis of Variance for General Cognitive Functioning Measures (N=327)

	Alcoholism Diagnosis		
	Alcoholics (n=168) M (SD)	Non-Alcoholics (n=159) M (SD)	F
MicroCog General Cognitive Functioning	94.79 (13.9)	97.01 (13.6)	2.14
WAIS Full Scale IQ	104.94 (10.6)	107.10 (11.6)	3.10

	Alcoholism Subtypes			
	AALs (n=24) M (SD)	NAALs (n=144) M (SD)	Controls (n=159) M (SD)	F
MicroCog General Cognitive Functioning	90.04 (15.5)	95.58 (13.57)	97.01 (13.6)	2.76
WAIS Full Scale IQ	101.51 (9.7)	105.51 (10.71)	107.10 (11.6)	2.90

Table 5.

Multivariate Analysis of Variance for Memory Functioning Measures (N=327)

	Alcoholism Diagnosis		
	Alcoholics (n=168) M (SD)	Non-Alcoholics (n=159) M (SD)	F
Story Immediate Recall	8.64 (3.77)	9.86 (3.78)	8.51**
Story Delay Recall	8.56 (3.12)	9.37 (3.11)	5.54*

	Alcoholism Subtypes			
	AALs (n=24) M (SD)	NAALs (n=144) M (SD)	Controls (n=159) M (SD)	F
Story Immediate Recall	8.83 (4.09)	8.60 (3.73)	9.86 (3.78)	4.28*[a]
Story Delay Recall	8.58 (3.02)	8.56 (3.15)	9.37 (3.11)	2.76

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

[a] NAALs < Controls

significantly worse than non-alcoholics on Picture Completion and Block Design. A second MANOVA was conducted to examine the effect of SUBTYPE on visuospatial functioning. The overall main effect was non-significant [F (6,644) = 1.63, p = .14] (see Table 6).

Perceptual Motor Functioning. The SMDT was used to assess perceptual motor functioning. Only raw scores were available for this measure. Thus, to determine whether group differences existed after variability in age and educational level had been accounted for, a multivariate analysis of covariance (MANOVA) was conducted on the scores with age and years of education as the covariates. The results showed a significant main effect of DIAGNOSIS [F (2,322) = 4.47, p < .01]. Univariate ANOVAs indicated that alcoholics performed significantly worse than non-alcoholics on both the written and oral version of the SDMT. A second MANCOVA was conducted to examine the main effect of SUBTYPE on perceptual motor functioning. The results yield a significant main effect [F (4,642) = 4.60, p < .01]. Univariate ANOVAs revealed that the three groups differed significantly on both the written and oral scores. Post-hoc comparisons using Tukey Tests showed AALs performed significantly worse than NAALs and controls on both the written and oral SDMT. In addition, NAALs performed significantly worse than controls on oral SDMT (see Table 7).

Executive Functioning. Three measures from the MicroCog were used to assess executive functioning: Wordlist 1, Wordlist 2, and Analogies. Results of a MANOVA did not show a significant main effect of DIAGNOSIS [F (3,323) = 1.77, p = .15]. However, univariate ANOVAs indicated that alcoholics performed

Table 6.

Multivariate Analysis of Variance for Visuospatial Functioning Measures (N=327)

	Alcoholism Diagnosis		
	Alcoholics (n=168) M (SD)	Non-Alcoholics (n=159) M (SD)	F
Clocks	11.64 (0.91)	11.52 (1.16)	1.23
Picture Completion	11.84 (2.65)	12.50 (2.48)	5.31*
Block Design	11.34 (2.42)	12.03 (3.03)	5.18*

	Alcoholism Subtypes			
	AALs (n=24) M (SD)	NAALs (n=144) M (SD)	Controls (n=159) M (SD)	F
Clocks	11.58 (1.02)	11.65 (0.90)	11.52 (1.16)	1.18
Picture Completion	11.31 (2.71)	11.93 (2.64)	12.50 (2.48)	3.26
Block Design	10.99 (2.30)	11.40 (2.44)	12.03 (3.03)	2.82

*$p < .05$

Table 7.

Multivariate Analysis of Covariance for Perceptual Motor Functioning Measures with Age and Education as Covariance (N=327)

	Alcoholism Diagnosis		
	Alcoholics (n=168) M (SD)	Non-Alcoholics (n=159) M (SD)	F
SDMT – Written	55.33 (10.36)	58.54 (10.25)	8.48**
SDMT – Oral	63.67 (10.64)	67.27 (12.54)	7.85**

	Alcoholism Subtypes			
	AALs (n=24) M (SD)	NAALs (n=144) M (SD)	Controls (n=159) M (SD)	F
SDMT – Written	49.75 (9.74)	56.26 (10.11)	58.53 (10.25)	8.71** [a b]
SDMT – Oral	59.38 (14.56)	64.38 (10.88)	67.27 (12.54)	5.83** [a b c]

**$p < .01$

[a]AALs < NAALs
[b]AALs < controls
[c]NAALs < controls

significantly worse than non-alcoholics on Wordlist 2. A second MANOVA was conducted to examine the effect of SUBTYPE on executive functioning. The main effect was significant [$F(6,644) = 2.15$, $p < .05$]. Univariate ANOVAs indicated a significant effect of SUBTYPE on Wordlist 2. Post-hoc comparisons using Tukey Test revealed that NAALs performed significantly worse than controls on Wordlist 2. Univariate also revealed a marginal effect of SUBTYPE on Analogies. Post-hoc comparisons indicated that AALs performed marginally worse than controls on Analogies (see Table 8).

Hypothesis 2

Hypothesis 2 stated that AALs would most likely be classified as suffering from brain impairment as compared to NAALs and controls. NAALs would also more likely be classified as suffering from brain impairment than controls. It was originally proposed that clinical rating of each individual's neuropsychological profile would be obtained to determine the likelihood of brain dysfunction. However, due to difficulties in developing a guideline and finding suitable clinicians to complete the rating, an alternative approach was developed to test the hypothesis. Any neuropsychological score that fell below the 8th percentile, i.e. in the borderline to impaired range, was coded as a positive indicator for brain impairment. All 12 measures were coded based on the above criteria. Subjects with three or more positive indicators were classified as having "brain impairment."

In order to evaluate the relationship between alcoholism subtypes and brain impairment, percentages of subjects from each group having three or more

Table 8.

Multivariate Analysis of Variance for Executive Functioning Measures (N=327)

	Alcoholism Diagnosis		
	Alcoholics (n=168) M (SD)	Non-Alcoholics (n=159) M (SD)	F
Wordlist I	9.95 (2.54)	10.18 (2.65)	0.68
Wordlist II	10.17 (2.63)	10.73 (2.41)	4.05*
Analogy	9.96 (3.16)	10.04 (2.75)	0.07

	Alcoholism Subtypes			
	AALs (n=24) M (SD)	NAALs (n=144) M (SD)	Controls (n=159) M (SD)	F
Wordlist I	9.33 (3.82)	9.94 (3.22)	10.45 (3.13)	1.76
Wordlist II	10.67 (2.82)	9.79 (2.75)	10.51 (2.49)	3.21* [a]
Analogy	7.75 (3.27)	8.99 (3.09)	9.24 (3.12)	2.39

*$p < .05$.

[a] NAALs < Controls

positive indictors of neuropsychological impairment were calculated. Results showed that 20.8% of AALs, 5.6% of NAALs, and 6.3% of controls were categorized with having significant brain impairment. Odds ratios were calculated to estimate the relative risk for significant brain impairment. Ratios that are greater than or equal to three are typically considered as strong effects (Chassin, Rogosch, Barrera, 1991). Chi-square statistics were used to assess the degree of association in each 2 x 2 contingency table from which the odds ratio was calculated. The odds ratio for AALs was calculated to be 3.10 (χ^2 = 5.86, $p < .05$), indicating that AALs were more than three times more likely than controls to have significant brain impairment. In contrast, the chi-square for NAALs showed that they did not vary significantly from controls (Odds ratio = 0.89; χ^2 = 0.07, n.s.) (see Table 9).

Path Analysis

For hypotheses 3 to 7, path analyses were conducted using LISREL 8.3 (Jöreskog & Sörbom, 1999). The maximum likelihood procedure was used to estimate model coefficients and a covariance matrix was analyzed. Standardized parameters were presented to facilitate interpretation. One tailed tests ($p = .05$) was used to determine whether or not each parameter was significant.

Hypothesis 3

Hypothesis 3 stated that drinking variables would mediate a poorer neuropsychological outcome. Moreover, history of alcoholism would be the strongest predictor among all drinking variables. A MIMIC (Multiple Indicators

Table 9.

Relative Risk for Significant Brain Impairment (N=327)

	Alcoholism Subtypes	
	Antisocial Alcoholics (n=24)	Controls (n=159)
Number of Positive Impairment Indictors		
0 – 2	19 (79.2%)	149 (93.7%)
3 and above	5 (20.8%)	10 (6.3%)

Odds Ratio = 3.10; Pearson Chi-Square = 5.86, $p < .05$

	Alcoholism Subtypes	
	Non-Antisocial Alcoholics (n=144)	Controls (n=159)
Number of Positive Impairment Indictors		
0 – 2	136 (94.4%)	149 (93.7%)
3 and above	8 (5.6%)	10 (6.3%)

Odds ratio = 0.89; Pearson Chi-Square = 0.07, n.s.

and Multiple Causes) model design was used to examine the relationship between drinking variables and neuropsychological outcome. The three drinking variables used in the analyses were current alcohol problems, current alcohol consumption, and history of alcoholism diagnosis. One measure from each neuropsychological domain (Wordlist 2, Story Immediate Recall, Block Design, and Hand SDMT) was selected as indictors for the latent variable "neuropsychological outcome."

The overall MIMIC model showing the significant standardized coefficients is presented in Figure 2. The overall goodness of fit for the model was good. Although the resulting Chi-Square showed significant model-data discrepancies [χ^2 (11, $\underline{N}$ = 329) = 21.12, $\underline{p}$ < .05], the GFI and CFI were acceptably large (0.98 and 0.98 respectively) and root mean square error of approximation (RMSEA) was at an acceptable level (.05). Examination of the factor estimates showed that indicator loadings were statistically significant for the latent construct, i.e. neuropsychological outcome. Errors of the terms were not allowed to be freely correlated. Regarding the mediating effect of drinking variables, only history of alcoholism diagnosis significantly predicted neuropsychological outcome. See Appendix A for the Lisrel program and the covariance matrix analysis.

Hypothesis 4

Hypothesis 4 stated that a greater history of alcoholism would directly predict increased neuromedical risks, higher current levels of depression and poorer memory functioning. In addition, increased neuromedical risks and higher current level of depression would simultaneously predict poorer memory

functioning. Story Immediate Recall was chosen as measure of memory functioning. Path analysis was conducted to test the validity of the model. The overall goodness of fit for the model was poor. The Chi-Square indicated significant model-data discrepancies [χ^2 (2, $\underline{N}$ = 329) = 36.35, $\underline{p}$ < .01]. In addition, the CFI was smaller than .90 (.19) and RMSEA was above the .05 level (.23).

To increase model fit, a fifth variable, current alcohol problems was added to the model. The results indicated improvement in goodness of fit: χ^2 (4, $\underline{N}$ = 329) = 40.01, $\underline{p}$ = .001, GFI = 0.95, CFI = 0.85, and RMSEA = 0.17. To further refine the model, neuromedical risk was allowed to directly predict current depression. The modification produced a model that fit the data well: χ^2 (3, $\underline{N}$ = 329) = 2.89, $\underline{p}$ = .41, GFI = 1.00, CFI = 1.00, and RMSEA = 0.00. The final model showing the significant standardized coefficients is presented in Figure 3. The structural paths showed that greater history of alcoholism predicted lower performance on memory functioning, higher current level of depression, and higher level of current alcohol problems. In contrast, none of the immediate measures, i.e. current depression, current alcohol problems, and neuromedical risks significantly predicted memory functioning. See Appendix B for the Lisrel program and the covariance matrix analyzed.

Hypothesis 5

Hypothesis 5 stated that a greater history of alcoholism increased level of lifetime alcohol use would directly predict increased lifetime neuromedical risks, higher current levels of depression and poorer perceptual motor functioning. In

addition, increased lifetime neuromedical risks and higher current level of depression would simultaneously predict poorer perceptual motor functioning. Written score of the SDMT was chosen as measure of perceptual motor functioning. Path analysis was conducted to test the validity of the model. The results indicated a poor fit of data. The Chi-Square showed significant model-data discrepancies [χ^2 (2, $\underline{N}$ = 329) = 36.62, $\underline{p}$ < .001]. In addition, the CFI (0.29) was unacceptably small and RMSEA (.23) was above the .05 level.

To increase model fit, a fifth variable, current alcohol problems was added to the model. The revised model showed improvement in goodness of fit: χ^2 (4, $\underline{N}$ = 329) = 40.01, $\underline{p}$ = .001, GFI = 0.95, CFI = 0.86, and RMSEA = 0.17. To further refine the model, neuromedical risk was allowed to directly predict current depression. The modification produced a model that fit the data well: χ^2 (3, $\underline{N}$ = 329) = 2.89, $\underline{p}$ = .41, GFI = 1.00, CFI = 1.00, and root mean square error of approximation (RMSEA) = 0.01. The final model showing the significant standardized coefficients is presented in Figure 4. The structural paths showed that greater history of alcoholism predicted lower performance on perceptual motor function, higher current level of depression, and higher level of current alcohol problems. In addition, higher current level of depression predicted poorer perceptual motor functioning. Neither current alcohol problems nor neuromedical risks significantly predicted perceptual motor performance. See Appendix C for the Lisrel program and the covariance matrix analyzed.

Hypothesis 6

Hypothesis 6 stated that a greater history of alcoholism would directly predict increased neuromedical risks and higher current alcohol problems. In addition, increased neuromedical risks and higher current alcohol problems would simultaneously predict poorer visuospatial functioning. Block Design was selected as a measure of visuospatial functioning. Path analysis was conducted to test the validity of the model. The model resulted in a good fit of data: χ^2 (3, N = 329) = 1.19, p = .76, GFI = 1.00, CFI = 1.00, and RMSEA = .001. The model showing the significant standardized coefficients is presented in Figure 5. The structural paths showed that greater history of alcoholism predicted higher level of current alcohol problems, which in turn predicted lower level of performance on visuospatial processing. Conversely, greater history of alcoholism did not predict higher neuromedical risk. Further, neuromedical risk did not predict poorer performance on visuospatial processing. See Appendix D for the Lisrel program and the covariance matrix analyzed.

Hypothesis 7

Hypothesis 7 stated that greater history of alcoholism would predict higher levels of neuromedical risk and current alcohol problems. In addition, increased neuromedical risk and current alcohol problems would simultaneously predict poorer executive functioning. Path analysis was conducted to test the validity of the model. The model resulted in a good fit of data: χ^2 (2, N = 329) = 1.01, p = .60 and RMSEA = .000. In addition, the GFT (1.00) and CFI (1.00) were both exceptionally high. The model showing the significant standardized coefficients

is presented in Figure 6. The structural paths showed that greater history of alcoholism predicted higher level of current alcohol problems and poorer executive functioning. Conversely, greater history of alcoholism did not predict higher neuromedical risk. Further, neuromedical risk and current alcohol problems did not predict poorer performance on executive functioning. See Appendix E for the Lisrel program and the covariance matrix analyzed.

DISCUSSION

Research has shown that alcoholism can lead to neuropsychological impairment. However, the underlying mechanisms mediating the relationship between alcoholism and neuropsychological deficits are many in terms of possibilities. By focusing on the neuropsychological functioning of community alcoholics, the current study sought to investigate the validity of the three theoretical models: the diffuse dysfunction model, the right hemisphere deficit model, and the frontal lobe dysfunction model.

The diffuse dysfunction model holds that alcoholics are more likely to show poorer general cognitive ability as well as relative deficits across all neuropsychological domains. Results of the present study showed that alcoholics and non-alcoholics did not differ on measures of general cognitive ability. Similarly, no difference in general cognitive ability was observed among alcoholism subtypes. These findings are inconsistent with the diffuse dysfunction model. Other studies have also shown that alcoholics do not have lower level of intelligence. It appears that standard intelligence tests have limited value when it comes to detecting cognitive deficits among alcoholics.

Although scores on general cognitive functioning did not yield significant finding, alcoholics were found to have a wide range of relative neuropsychological deficits. This was evident in three of the four neuropsychological domains: visuospatial, memory, and perceptual motor functioning. In addition, AALs were found to perform significantly worse than

NAALs and controls on perceptual motor functioning. These results showed that alcoholics suffer generalized neuropsychological deficits, which are indicative of diffuse brain dysfunction. More importantly, perceptual motor functioning was found to be the most significant deficit among AALs and NAALs. SDMT, which was used to assess perceptual motor ability in the present study, is known to be a sensitive measure to brain dysfunction (Lezak, 1995). Thus, the poor performance on SDMT among alcoholics could be interpreted as evidence for the diffuse dysfunction model.

The right hemisphere model suggests that chronic alcoholism affects predominantly functions of the right hemisphere. One of the major functions of the right hemisphere is processing visuospatial information. Therefore, poor performances in visuospatial tasks could be indications of right hemisphere damage. Indeed, the current study revealed that alcoholics performed significantly worse than non-alcoholics on two of the visuospatial measures (i.e. Picture Completion and Block Design). Conversely, no differences were found among AALs , NAALs, and controls on visuospatial ability. One of the visuospatial tasks, Clocks, was proven to be a weak measure due to a marked ceiling effect. This was likely attributed to the fact that the measure has only a few items. Overall, these results suggest that alcoholics suffer visuospatial deficits that might be linked to right hemisphere dysfunction; however, the deficits are unrelated to alcoholism subtypes.

Although visuospatial deficits were evident among alcoholics, other deficits including memory and perceptual motor functioning that are not dominant

functions of the right hemisphere were also noted in the present study. These findings are inconsistent with the idea that the right hemisphere is most vulnerable to damage due to alcoholism. Moreover, some researchers have argued that visuospatial processing is not an exclusive function of the right hemisphere. In a recent study, Beatty, Hames, Blanco, Nixon, and Tivis (1995) examined alcoholics' performances on two different types of visuospatial tasks: featural and configural. The authors noted that visuospatial tasks that require featural analysis (e.g. Picture Completion) are primarily measures of left hemisphere function, whereas those that require configural analysis (e.g. Block Design) are mostly measures of right hemisphere function. Results showed that alcoholics were impaired on both types of visuospatial tasks. A similar conclusion could be drawn from the results of the current study. It is likely that the right hemisphere is not specifically more vulnerable to the effect of alcoholism than the left hemisphere.

The frontal lobe deficit model suggests that frontal lobe functioning is most susceptible to the effect of alcoholism. Many cognitive functions are associated with the frontal lobe including selective attention, abstract reasoning, and planning. In the current study, alcoholics were found to perform significantly worse than non-alcoholics on only one of the three frontal lobe measures, i.e. Wordlist 2, suggesting difficulty in selective attention. Similar results were also observed among alcoholism subtypes. These findings are somewhat surprising given that research has consistently reported frontal lobe deficits in alcoholics. One possible explanation is that the measures chosen to assess executive

functions in this study might not be sensitive to alcohol abuse. In fact, there are a number of abilities involved in executive functioning: selective attention, planning, reasoning, purposive action, and effective performance. Of the all the various abilities of executive functions, only selective attention (Wordlist) and abstract reasoning (Analogies) were assessed in the current study. It is possible that selective attention and abstract reasoning are less affected by alcoholism than other components of executive functions. Future studies should compare the performances of alcoholics on these different components.

There is yet another alternative explanation to the current finding. Some researchers have posited that deficits in Block Design might be related to problem-solving difficulty, a function that is associated with the frontal lobe. For instance, alcoholics have been found to perform adequately on visuospatial tasks that require little or no synthesizing, organizing, or orienting activity (Oscar-Berman & Weinstein, 1985; Tarter, 1975). Moreover, research has demonstrated a frontal constructional difficulty that involves the disruption of one or more of the steps in problem-solving, i.e. intention, programming, regulation or verification (Lezak, 1995). Further, neuroimaging studies have shown that performance on Block Design is correlated with frontal regional blood flow (Rourke and Loberg, 1996). These findings suggest that the poor performance by alcoholics on Block Design alcoholics in the current study might be associated with frontal lobe dysfunction.

The current study showed that alcoholics had poorer memory functioning than non-alcoholics. This finding was extended to alcoholism subtypes by

showing that poor memory functioning is also present in NAALs. Although memory deficits are generally considered to be associated with temporal lobe dysfunction, some researchers have postulated that the frontal lobe is involved in modulating memory functioning. Specifically, the prefrontal cortex is thought be part of the limbic structures that mediate explicit memories. Memory difficulties can occur due to poor execution of the mental strategies that bring recall and memorization into play during memory tasks. Moreover, distraction could also affect memory performance. A person who has poor ability to withstand interference is likely going to have difficulty performing memory tasks. This type of memory difficulty might be relevant to the current findings. As mentioned earlier, alcoholics were found to have poor selective attention. Unlike the Logical Memory in WMS, the Stories subtest in MicroCog required subjects to read the stories on their own, which demand focused attention and concentration. Thus, the current finding of memory difficulty in alcoholics is supportive of frontal lobe dysfunction.

Overall, results of the present study lend support to the diffuse brain dysfunction with the frontal lobe being more susceptible to damage. Among the four neuropsychological domains, perceptual motor functioning appears to be most sensitive to alcohol abuse. As mentioned before, SDMT is sensitive to damage in many areas of the brain. If alcohol does cause mild to moderate damage throughout the brain, it would be expected that alcoholics would perform worse on this task. Notably, perceptual motor functioning was the only neuropsychological domain that AALs performed significantly worse than NAALs

and controls. One of the main features of antisocial alcoholics is that their alcoholism is likely to be more chronic and severe. Perhaps the deficit in perceptual motor functioning is a reflection of such differences.

Although the mean scores on all neuropsychological measures were within normal limits, the proportion of subjects that were classified as having significant brain impairment were much higher among AALs than NAALs and controls. This finding suggests that AALs might be most susceptible to neuropsychological impairment as a result of drinking. One possible explanation is that AALs might have a premorbid cognitive dysfunction before the development of alcoholism. Studies have shown that antisocial personality disorder is related to frontal lobe dysfunction (Deckel, Hesselbrock, & Bauer, 1996; Dinn & Harris, 2000). In addition, AALs are more likely to have a positive family history of alcoholism, which is also linked to cognitive deficits. For instance, studies have shown that certain cognitive deficits are present in children of alcoholics who are at risk for developing alcohol abuse/dependence (Noll, Zucker, Fitzgerald, & Curtis, 1992; Poon, Ellis, Fitzgerald, & Zucker, 2000; Tarter, Hegedus, Goldstein, Shelly, & Alterman, 1984).

A second major goal of the present study was to examine variables that might influence the neuropsychological outcome of alcoholics. Three potential sources of variance were studied: depression, neuromedical risk, and drinking history. Path analyses were conducted on each of the four neuropsychological domains to determine factors that mediate neuropsychological outcome.

Results of the present study showed that history of alcoholism, in terms of length and severity, was the most reliable predictor for neuropsychological outcome among all the drinking variables. Interestingly, none of the current drinking variables were significant predictors of neuropsychological outcome. Further, path analyses showed that current alcohol problem was a significant mediating factor for visuospatial processing only. This finding is somewhat surprising given that current alcohol consumption had been found to predict neuropsychological performance (Beatty et al., 2000; Horner et al., 1999). One could possibly argue that current alcohol problem and current alcohol consumption are two variables that measure different constructs. Specifically, current alcohol problem is more related to symptomatology of alcohol use while recent alcohol consumption measures quantity of alcohol use. However, many alcohol symptoms assessed in this study were closely related to frequency and quantity of use. Thus, it is difficult to conceive that this is the reason for the negative findings. An alternative explanation is that the effect of current alcohol problem on neuropsychological functioning is domain specific whereas the effect of chronic and severe use of alcohol is more global in nature. In fact, the present study supports this argument by showing that increased current alcohol problem is related to poor performance in a specific area, i.e. visuospatial processing. On the other hand, history of alcoholism has a direct effect on various cognitive functions.

Path analysis revealed that length and severity of alcoholism significantly predicted poor performances on three of the four neuropsychological domains:

memory functioning, perceptual motor functioning, and executive functioning (i.e. selective attention). Moreover, for memory functioning and selective attention, no other significant predictors emerged from the path models. These results suggest that length and severity of alcoholism is the main factor that mediates these functions. This finding is consistent with previous research that has shown a strong relationship between chronic alcohol consumption and neuropsychological deficits. In one of the studies, Eckardt, Stapleton, Rawlings, Davis, and Grodin (1995) reported that greater lifetime estimate of alcohol consumption predicted worse performance on Halstead Impairment Index, Speech Sounds Perception, Benton Naming Test, Tactual Performance Test for memory, and the delayed recall portion of the Rey-Osterrieth Complex figure. In contrast, recent alcohol consumption did not have any predictive importance on these measures.

The fact that only history of alcoholism predicted memory functioning is particularly interesting. Studies have shown that alcoholics tend to underestimate and minimize their memory problems (Ryan & Lewis, 1988). Further, the memory deficits observed among alcoholics are often subtle and only apparent when the memory tasks become more difficult. Undiagnosed memory impairment could potentially have a negative impact on treatment efficacy. Thus, it is important to assess the extent of memory deficits in treating chronic alcoholics.

As predicted, path analysis revealed that history of alcoholism strongly predicted current alcohol problem in all four models. In contrast, history of

alcoholism was found to be unrelated to neuromedical risk. This finding is rather disappointing but not entirely inconsistent with the literature. Several studies have failed to document a relationship between head injury, alcoholism, and neuropsychological dysfunction. In one particular study, Alterman, Goldstein, Shelly, Bober, and Tarter (1985) reported that the neuropsychological performance of alcoholics with histories of mild head injury is comparable to those without histories of mild head injury. More recently, Dikmen, Donovan, Loberg, and Machamer (1993) examined the relationship between neuropsychological outcome and alcohol use problem among patients with various level of head injury. Specifically, the study sought to determine whether alcohol abuse prior to any head injury would negatively affect the neuropsychological outcome. Results showed that poor neuropsychological outcome was related to several factors including limited education, neuropsychological impairments, and a lifestyle concurrent with heavy drinking, including an increased risk for head injuries. Contrary to expectation, there was no evidence for a greater head-injury effect in those with more severe alcohol problems. Alternatively, the negative finding could be attributed to the limited scope of the measure. Items that were included in the measures were mostly related to the nervous system. If other alcohol related diseases (e.g. liver cirrhosis) were to be included in the measure, the results might show a stronger predictive power on cognitive functioning.

Depression is another mediating factor that was of interest to this study. Results of the current study showed that chronic alcoholism predicted higher

level of depression, which in turn predicted poorer performance on perceptual motor functioning. This finding is consistent with research and clinical data that indicate depressive individuals are more likely to exhibit psychomotor retardation (Brebion, Amador, Smith, Malaspina, Sharif, & Gorman, 2000; Berndt & Berndt, 1980; Hart & Kwentus, 1987). In one study, Austin (1992) examined the neuropsychological functioning of individuals with a major depressive episode. Results showed that depression is associated with impaired performance on Auditory Verbal Learning Test, Digit Symbol Substitution and the Trail-Making Test. These findings might have significant implications on treatment of alcoholics. In particular, successful treatment of depression in alcoholics could result in significant alleviation of cognitive impairments, which may improve their chance of succeeding in alcohol rehabilitation.

Limitation of the Study

The current study showed that alcoholics exhibit generalized neuropsychological impairment. Moreover, executive deficits appear to be most prominent, suggesting that the frontal lobe might be more susceptible to damage. Results of the present study also showed that AALs are more likely to be classified as neuropsychologically impaired as compared to NAALs and controls. Path analyses revealed that history of alcoholism is the strongest predictor for neuropsychological impairment. Although these are significant findings, it is important to note that there are several limitations to the present work. First, the neuropsychological measures used in this study to assess executive functioning

were somewhat limited in scope and focused mainly on selective attention and abstract reasoning. It is possible that other types of executive functions deficit might exist among alcoholics but were not detected in this study. Using other executive function measures may help clarify the exact nature of such deficits as well as provide additional information regarding brain-behavior linkage.

The current study only examined three potential mediating factors that might contribute to the neuropsychological outcome of alcoholics. Future studies should explore other potential sources of variance such as family history of alcoholism, nutrition, length of abstinence, and age. As mentioned earlier, the measure used in this study to assess neuromedical risk only focused on problems with the nervous system. Other diseases such as liver cirrhosis, HIV, or hypertension might play a significant role in mediating the relationship between alcoholism and neuropsychological functioning.

Summary

The current study showed that community alcoholics performed poorly on a wide range of neuropsychological measures. Moreover, perceptual motor functioning appears to be most sensitive to alcohol abuse. Among all the drinking measures, history of alcoholism was the most reliable predictor for neuropsychological outcome. Path analysis revealed that depression mediated the relationship between history of alcoholism and perceptual motor functioning. In addition, higher current level of alcohol problems predicted poorer performance in visuospatial functioning.

The current study extended earlier research by showing that poorer neuropsychological performance previously documented among alcoholics in clinical populations is present in a community-based population of alcoholics. Although the pattern of deficits seems to be most consistent with the diffuse brain dysfunction model, none of the three theoretical models provided a definitive framework to describe the effect of alcohol on brain functioning. Further, the results indicate that the direct neurotoxic effect of alcohol was partially moderated by other alcohol-related factors including depression and drinking pattern.

In light of the current findings, it appears that the past approaches to studying the neuropsychological functioning of alcoholics namely, comparing the three different theoretical models, may not capture the full range of possible neuropsychological effects of sustained and/or intensive alcohol consumption. Future research should focus on developing a more comprehensive theory that incorporates the both the direct and indirect effect of alcoholism on neuropsychological functioning.

Figure 1.

Multifactorial Etiology of Neuropsychological Functioning of Alcoholics

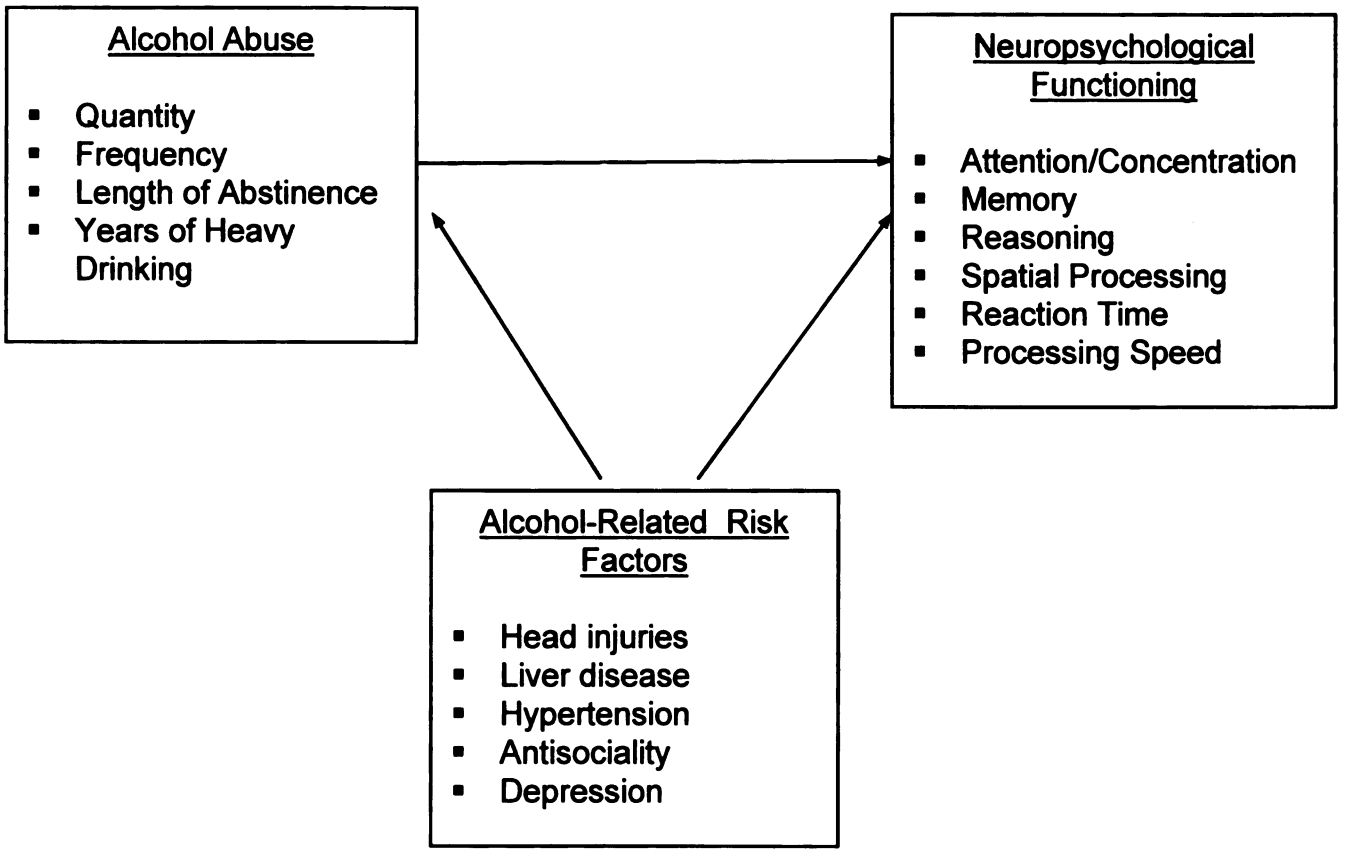


Figure 2.

Drinking Variables and Neuropsychological Outcome

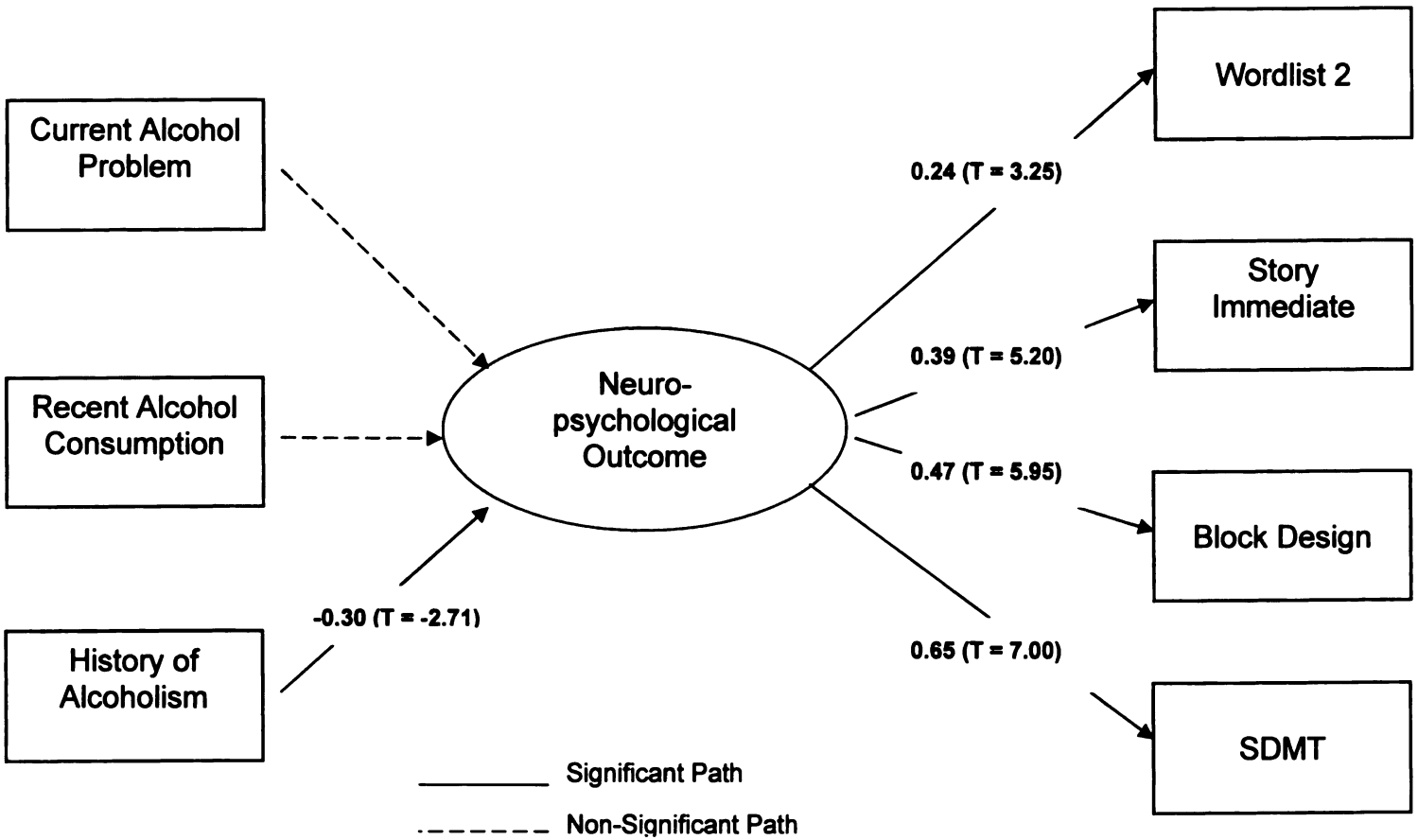


Figure 3.

Structural Equation Model for Memory Functioning

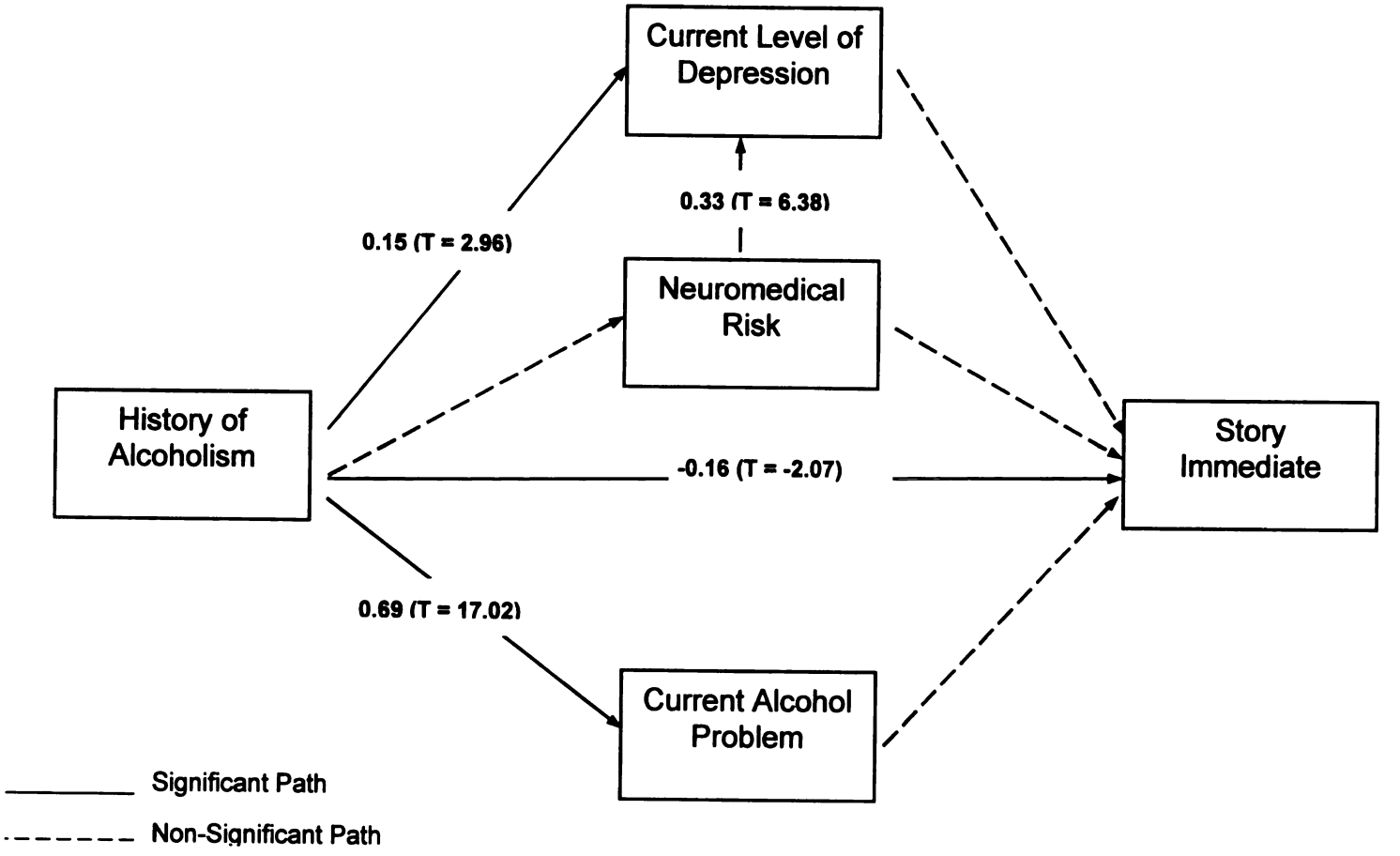
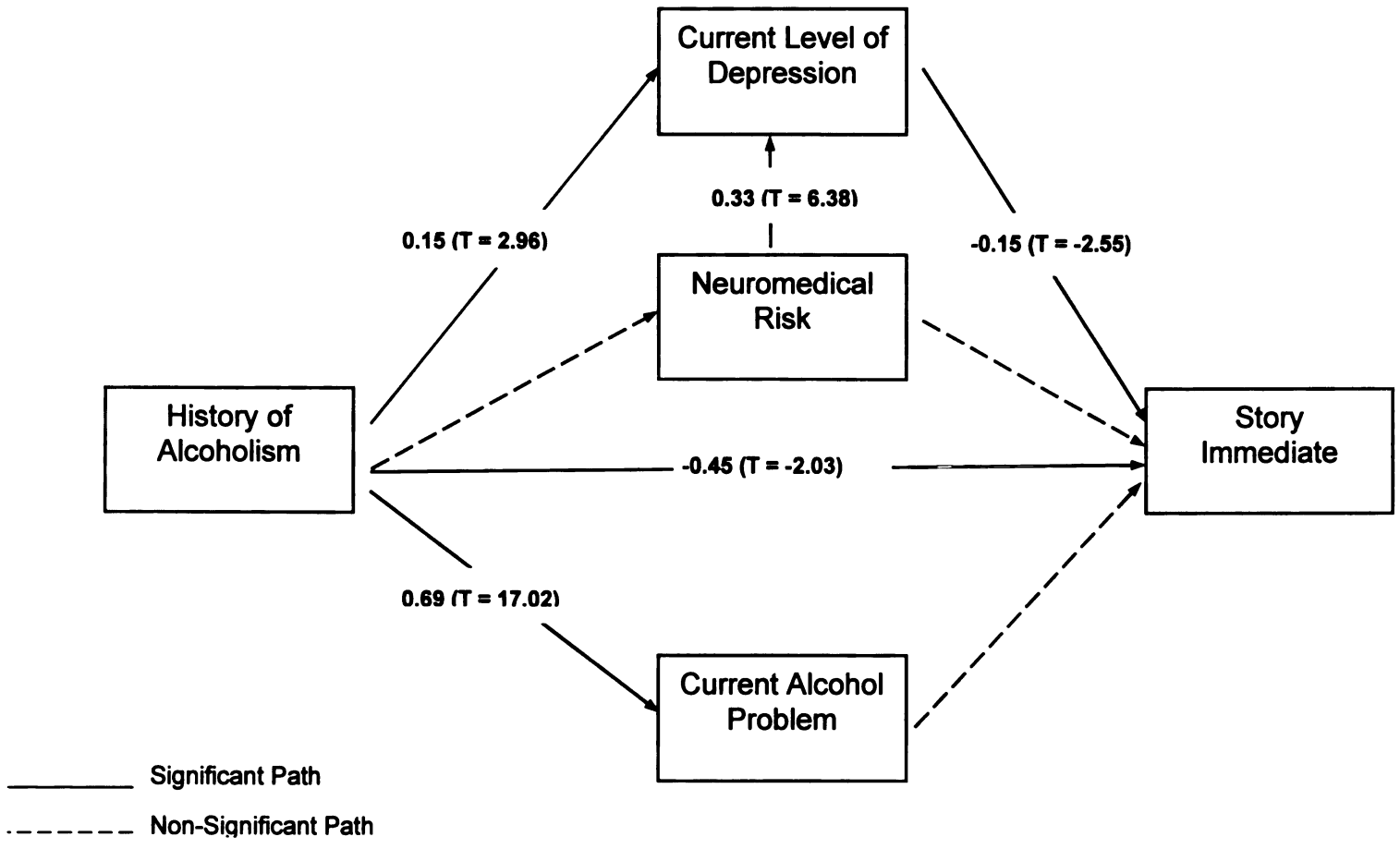


Figure 4.

Structural Equation Model for Perceptual Motor Functioning



Current Level of Depression
Neuromedical Risk
Story immediate
History of Alcoholism
Current Alcohol Problem
Significant Path
Non-Significant Path

Figure 5.

Structural Equation Model for Visuospatial Functioning

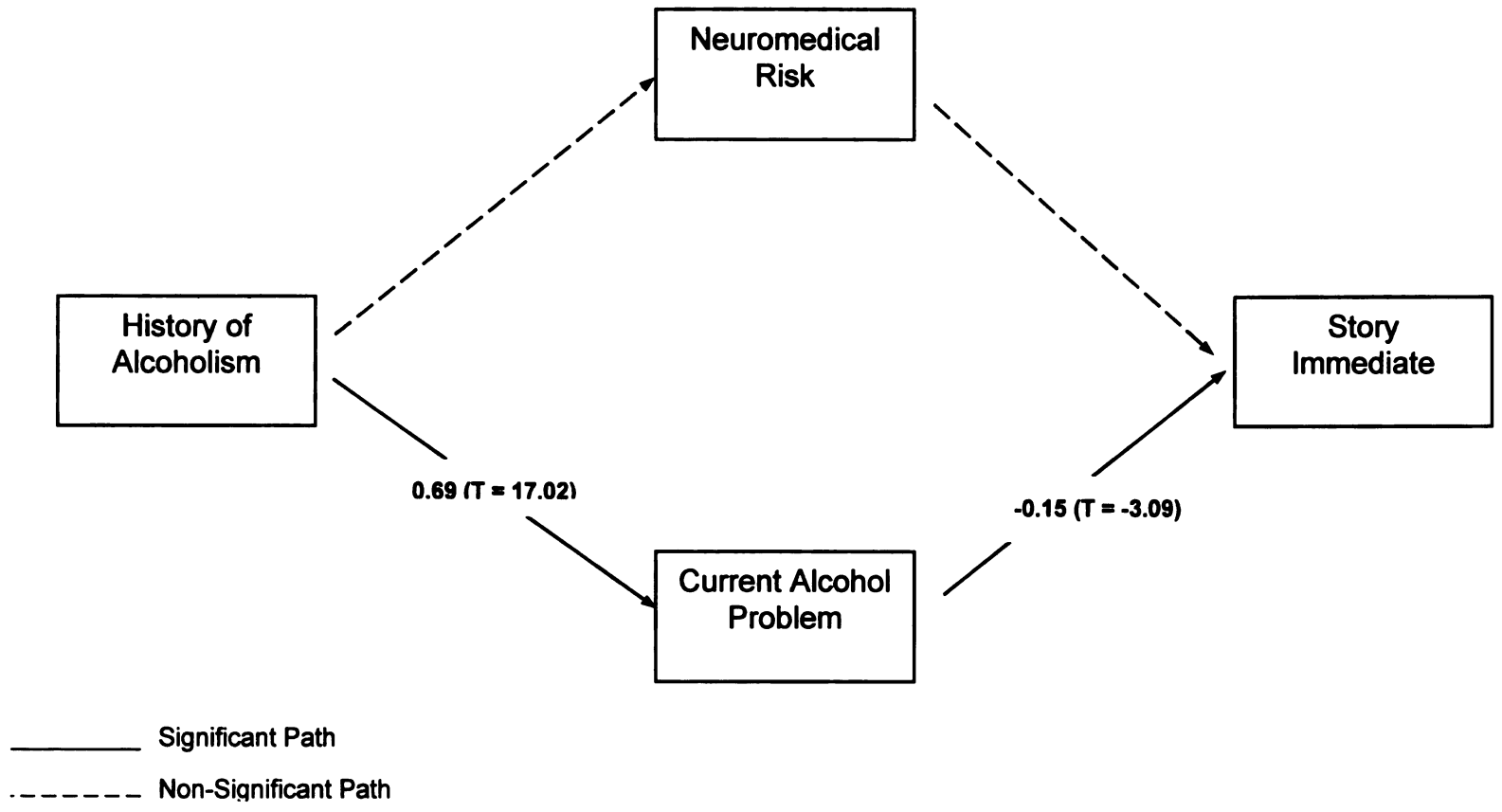
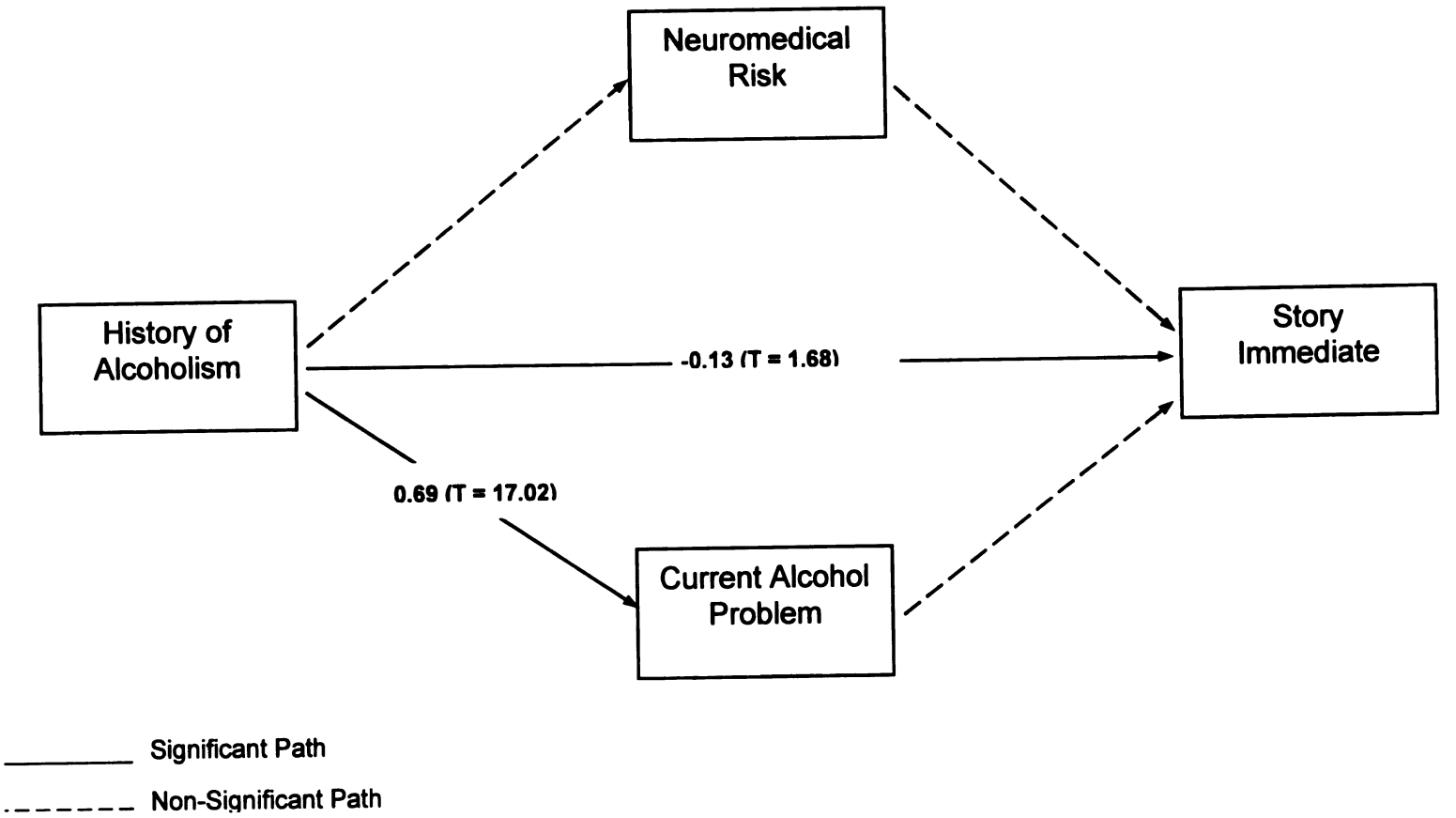


Figure 6.

Structural Equation Model for Executive Functioning



Stay
Immediate
Neuromedical
Risk
Chronic Alcohol
Problem

APPENDICES

APPENDIX A

Lisrel Program – MIMIC Model for Drinking Variables and Neuropsychological Outcome

```
DA NI=7 NO=327
RA FI=C:\LISREL83\DISSERT\RAWDATA\MIMIC.DAT
LA
WORD2 STORY1 WAISBLCK SDMTHAND ALPROB NUMDRINK ALCHRON
SE
1 2 3 4 5 6 7
MO NY=4 NE=1 NX=3 LY=FR
LE
NEUROPSY
PD
OU SE TV EF RS MI
```

APPENDIX A

Covariance Matrix Analyzed – MIMIC Model for Drinking Variable and Neuropsychological Outcome

	ALCHRON	NUMDRINK	ALPROB	SDMTHAND	WAISBLCK	STORY1	WORD2
ALCHRON	7.24						
NUMDRINK	1.61	14.56					
ALPROB	0.33	2.12	7.55				
SDMTHAND	4.74	9.08	9.02	108.62			
WAISBLCK	0.01	-0.41	-1.69	-2.92	12.85		
STORY1	-7.41	-4.97	-13.21	10.05	71.14	1431.58	
WORD2	-0.65	-1.44	-1.27	-5.14	8.64	77.75	12.36

APPENDIX B

Lisrel Program – Path Analysis for Memory Functioning

```
DA NI=5 NO=327
RA FI=C:\LISREL83\DISSERT\RAWDATA\MEM2.DAT
LA
STORY1 HAMILTON NRISK ALPROB ALCHRON
SE
1 2 3 4 5
MO NY=4 NX=1 BE=FI GA=FI PH=FI
FR GA 1 1 GA 2 1 GA 3 1 GA 4 1
FR BE 1 2 BE 1 3 BE 1 4 BE 2 3
PD
OU ME=ML RS MI SE TV EF
```

APPENDIX B

Covariance Matrix Analyzed – Path Analysis for Memory Functioning

	STORY1	HAMILTON	NRISK	ALPROB	ALCHRON
STORY1	14.57				
HAMILTON	-1.83	87.38			
NRISK	0.08	2.38	0.60		
ALPROB	-0.41	5.67	0.11	12.85	
ALCHRON	-1.44	5.01	0.00	8.64	12.36

APPENDIX B

Covariance Matrix Analyzed – Path Analysis for Discovery Functioning

	STORY1	HAMILTON	NRISK	ALPROB	ALCHRON
STORY1	14.57				
HAMILTON	-1.83	47.56			
NRISK	0.09	2.50	1.64		
ALPROB	-0.41	3.40	0.11	12.85	
ALCHRON	-1.44	2.31	0.05	6.64	12.36

APPENDIX C

Lisrel Program – Path Analysis for Perceptual Motor Functioning

```
DA NI=5 NO=327
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LA
SDMTHAND HAMILTON NRISK ALPROB ALCHRON
SE
1 2 3 4 5
MO NY=4 NX=1 BE=FI GA=FI PH=FI
FR GA 1 1 GA 2 1 GA 3 1 GA 4 1
FR BE 1 2 BE 1 3 BE 1 4 BE 2 3
PD
OU ME=ML RS MI SE TV EF
```

APPENDIX C

Lisrel Program – Path Analysis for Perceptual Motor Functioning

```
DA NI=5 NO=327
RA FI=C:/LISREL83/DISSERT/RAWDATA/SPEED.DAT
LA
SDMTHAND HAMILTON NNISK ALPROBAL CHRON
SE
1 2 3 4 5
MO NY=4 NX=1 BE=FI GA=FI PH=FI
FR GA 1 1 GA 2 1 GA 3 1 GA 4 1
FR BE 1 2 BE 1 3 BE 1 4 BE 2 3
PD
OU ME=ML RS MI SE TV EF
```

APPENDIX C

Covariance Matrix Analyzed – Path Analysis for Perceptual Motor Functioning

	SDMTHAND	HAMILTON	NRISK	ALPROB	ALCHRON
SDMTHAND	108.62				
HAMILTON	-15.14	87.38			
NRISK	-0.26	2.38	0.60		
ALPROB	2.92	5.67	0.11	12.85	
ALCHRON	-5.14	5.01	0.00	8.64	12.36

APPENDIX C

Covariance Matrix Analysis — Path Analysis for Structural Motor Functioning

	SOMTHAND	HAMILTON	NRISK	ALPROB	ALCHRON
SOMTHAND	108.82				
HAMILTON	−15.14	37.46			
NRISK	−0.28	1.24	0.46		
ALPROB	2.82	4.11	0.71	12.88	
ALCHRON	−8.14	2.01	0.09	3.84	12.38

APPENDIX D

Lisrel Program – Path Analysis for Visuospatial Functioning

```
DA NI=5 NO=327
RA FI=C:\LISREL83\DISSERT\RAWDATA\SPATIAL.DAT
LA
WAISBLCK NRISK ALPROB ALCHRON
SE
1 2 3 4
MO NY=3 NX=1 BE=FI GA=FI PH=FI
FR GA 2 1 GA 3 1
FR BE 1 2 BE 1 3
PD
OU ME=ML RS MI SE TV EF
```

APPENDIX D

Covariance Matrix Analyzed – Path Analysis for Visuospatial Functioning

	WAISBLCK	NRISK	ALPROB	ALCHRON
WAISBLCK	7.55			
NRISK	-0.19	0.60		
ALPROB	-1.69	0.11	12.85	
ALCHRON	-1.27	0.00	8.64	12.36

APPENDIX E

Lisrel Program – Path Analysis for Executive Functioning

```
DA NI=5 NO=327
RA FI=C:\LISREL83\DISSERT\RAWDATA\EXEC.DAT
LA
WORD2 NRISK ALPROB ALCHRON
SE
1 2 3 4
MO NY=3 NX=1 BE=FI GA=FI PH=FI
FR GA 1 1 GA 2 1 GA 3 1
FR BE 1 2 BE 1 3
PD
OU ME=ML RS MI SE TV EF
```

APPENDIX E

APPENDIX E

Covariance Matrix Analyzed – Path Analysis for Executive Functioning

	WORD2	NRISK	ALPROB	ALCHRON
WORD2	7.24			
NRISK	0.12	0.60		
ALPROB	0.01	0.11	12.85	
ALCHRON	-0.65	0.00	8.64	12.36

APPENDIX E

Covariance Matrix Analysis – Firm Data for Executive Functioning

	WORD2	NRISK	ALPROB	ALCHRON
WORD2	7.24			
NRISK	0.12	0.00		
ALPROB	0.01	0.11	0.12	
ALCHRON	-0.85	0.00	0.44	0.36

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