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NEUROPSYCHOLOGICAL FUNCTIONING AND ATTRITION RATES IN OUTPATIENT SUBSTANCE DEPENDENCE TREATMENT

by

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A Dissertation submitted to the Faculty of the Graduate School,
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ABSTRACT

NEUROPSYCHOLOGICAL FUNCTIONING AND ATTRITION RATES IN OUTPATIENT SUBSTANCE DEPENDENCE TREATMENT

Sandra M. Adams, LCSW, CSAC, ICS

Marquette University, 2010

Numerous neuropsychological factors have been associated with substance dependence, however, very few studies have evaluated the relationship of the neuropsychological functioning and attrition rates in substance dependence treatment. This study examined the relationship of neuropsychological functioning and attrition rates in 68 homeless, substance dependent men participating in outpatient treatment at the 7C's Community Counseling Clinic located in the Guesthouse of Milwaukee, Wisconsin. A neuropsychological battery including the Delis Kaplan Executive Functioning System, the Conners' Continuous Performance Test II, the Wechsler Abbreviated Scale of Intelligence (WASI) and the Wechsler Test of Adult Reading was given to all participants to evaluate neuropsychological function. The neuropsychological functioning was used to predict attrition rates using Survival Analysis and Logistic Regression. The results indicate that the neuropsychological functioning of this group of adult males showed statistically significant impaired functioning on all measures. Of the neuropsychological variables, only the WASI IQ predicted attrition and length of stay which showed a curvilinear relationship to drop out and attrition. Participants with a moderately low WASI IQ score (77-95) were significantly more likely to drop out (p = .012) and more likely to have shorter lengths of stay in treatment (p= .028). In addition, the neuropsychological variables did show a relationship with drop out and length of stay when looking at cases with a median IQ below 94 and those with no prior AODA treatment. Finally, results of calculations on effect size and power analysis show that with a larger sample size (98-170) we could increase the possibility that the neuropsychological variables would predict drop out and attrition and could attain power between .80 and .95.

TABLE OF CONTENT

LIST OF TABLES

Chapter I: Introduction

Substance Abuse and Dependence in the United States

Prevalence of Substance Use Disorders

Recent statistics from the National Survey on Drug Use and Health (NSDUH, 2008) indicated that an estimated 22.3 million Americans aged 12 or older in 2007 met diagnostic criteria for substance dependence or abuse over the past year. Of these, 3.2 million had dependence or abuse issues with both alcohol and illicit drugs, 3.7 million were dependent on or abused drugs but not alcohol, and 15.5 million were dependent on or abused alcohol but not drugs (NSDUH, 2008). NSDUH (2008) also stated that between 2002 and 2007 there was no change in the number of people with substance dependence or abuse (22.0 million in 2002, 22.3 million in 2007; NSDUH, 2008)

Effects of Substance Use Disorders on Individuals and Society

Substance use disorders have an impact on society, families and individuals (American Psychiatric Association {APA}, 2000). Substance use can be associated with violent behavior manifested by fights or criminal activity resulting in injury to the person using the substance or to others (APA, 2000). Likewise, automobile, home and industrial accidents can be a major complication of substance use (APA, 2000). Furthermore, according to the Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition, Text Revision (DSM-IV-TR) approximately half of all highway

fatalities involve either a driver or pedestrian who is intoxicated (APA, 2000). The DSM-IV-TR (APA, 2000) also reported that one in five intensive care admissions are related to alcohol. In addition, most, if not all, psychoactive substances cross from a pregnant woman's blood through the placenta, potentially causing adverse effects on the developing fetus (APA, 2000). When taken repeatedly in high doses by the mother, a number of substances (e.g., cocaine, opioids, alcohol, sedatives, hypnotics and anxiolytics) are capable of causing physiological dependence and withdrawal in the newborn (APA, 2000). Finally, the DSM-IV-TR (2000) reports that possibly 10 percent of individuals with a substance dependence diagnosis commit suicide.

Defining Substance Abuse and Dependence

Substance Abuse is defined based on the criteria listed in the DSM-IV-TR, 2000 that include:

A. A maladaptive pattern of substance use leading to clinically significant impairment or distress, as manifested by one (or more) of the following, occurring within a 12 month period:

(1) recurrent substance use resulting in a failure to fulfill major role obligations at work, school or home (e.g., repeated absences or poor work performance related to substance use; substance-related absences, suspensions, or expulsions from school; neglect of children or household)

(2) recurrent substance use in situations in which it is physically hazardous (e.g., driving an automobile or operating a machine when impaired by substance use),

(3) recurrent substance-related legal problems (e.g., arrests for substance-related disorderly conduct),

(4) continued substance use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance (e.g., arguments with spouse about consequences of intoxication, physical fights).

B. The symptoms have never met the criteria for Substance Dependence for this class of substance (APA, 2000, p. 199).

Substance Dependence is also defined based on the criteria listed in the DSM-IV-TR which states:

A maladaptive pattern of substance use, leading to clinically significant impairment or distress, as manifested by three (or more) of the following, occurring at any time in the same 12-month period:

(1) tolerance, as defined by either of the following:

(a) a need for markedly increased amounts of the substance to achieve intoxication or desired effect,

(b) markedly diminished effect with continued use of the same amount of the substance,

(2) withdrawal, as manifested by either of the following:

(a) the characteristic withdrawal syndrome for the substance [For example, with alcohol withdrawal, two or more of the following symptoms are necessary: autonomic hyperactivity, increased hand tremor, insomnia,

psychomotor agitation, anxiety, nausea or vomiting; and rarely, grand mal seizures or transient visual, tactile, or auditory hallucinations or illusions.]

(b) the same or closely related substance is taken to relieve or avoid withdrawal symptoms,

(3) substance is often taken in larger amounts or over a longer period than intended,

(4) there is persistent desire or unsuccessful efforts to cut down or control the substance use,

(5) a great deal of time is spent in activities necessary to obtain the substance, use the substance, or recover from its effects,

(6) important social, occupational, or recreational activities are given up or reduced because of substance use,

(7) the substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance (e.g., current cocaine use despite recognition of cocaine-induced depression, or continued drinking despite recognition that an ulcer was made worse by alcohol consumption; APA, 2000, p. 197).

This study is a pilot study and one of the first to investigate the effects of neurocognitive functioning on treatment retention. Therefore, in order to increase the likelihood of detecting smaller effect sizes this study aims to maximize the heterogeneity of the sample for this study by only including participants who meet the criteria for the diagnosis of Substance Dependence.

Health Related Effects on Individuals with Substance Use Disorders

Another important area to consider is the impact of substance abuse and dependence on the physical health of the individual. Individuals with substance related disorders often experience deterioration in their general health related to method of induction of substance (i.e., snorting), malnutrition and inadequate personal hygiene (APA, 2000). For example, using a substance intranasally can cause erosion of the nasal septum. The use of contaminated needles can result in human immunodeficiency virus (HIV), hepatitis, tetanus, endocarditis, malaria or other infectious or contagious diseases (APA, 2000). The use of stimulants can result in sudden death from cardiac arrhythmias, myocardial infarction, cerebral vascular accident or respiratory arrest (APA, 2000). Likewise, many medical conditions are commonly associated with alcohol use as alcohol affects nearly every organ in the body. For example, there is an increased rate of cancer of the esophagus and stomach, elevated triglycerides, and peripheral neuropathy in individuals with high alcohol use rates (APA, 2000). Furthermore, liver cirrhosis and pancreatitis are seen in approximately 15% of those who use alcohol heavily (APA, 2000).

Neurological, Neuropsychological and Cognitive Effects of Substance Use Disorders

With regards to central nervous system impact, neurological effects on individuals with substance use disorders include cognitive deficits, memory impairment and degenerative changes in the cerebellum (APA, 2000; Oscar-Berman, Shagrin, Evert & Epstein, 1997). Likewise, multiple negative effects on the frontal lobe (which is associated with executive functions) of the brain, such as reduced

volume and blood flow, have been reported in people with substance use disorders (Bates, Bowden & Barry, 2002; Moselhy, Georgiou & Kahn, 2001; Sullivan, Harding, Pentney, Dlugos, Martin, & Parks, 2003). In fact, many neuropsychological and cognitive deficits have been associated with impairment in functioning for people with a substance use disorder. For example, in people with substance use disorders, reductions in problem solving abilities, abstracting abilities (Oscar-Berman et al., 1997; Ratti, Giardini & Soragna, 2002), verbal fluency and response flexibility (all considered parts of executive functioning) have been found (Dao-Castellana et al., 1998), as have impaired memory and overall executive functioning (Cunha & Novaes, 2004; Rosselli, Ardila, Lubomski, Murray & King, 2001).

Executive functions as an area of interest for this study.

Of the many possible types of neuropsychological impairment, the impairment of the executive functions is what we are primarily interested in for this study. As mentioned above, multiple negative effects on the frontal lobe, which is the lobe associated with executive functioning, have been found in people with substance use disorders (Bates et al., 2002; Cunha & Novaes, 2004; Lezak, 1995; Moselhy et al., 2001; Sullivan et al., 2003). The executive functions are all "necessary for appropriate, socially responsible, and effectively self-serving adult conduct" (Lezak, 1995, p. 650). There are multiple components and possible behavioral disorders associated with impairment in the executive functions which could result in misinterpretation by clinicians and observers (Lezak, 1995) possibly leading to further stigma of individuals with a substance use disorder. The executive functions and impairments will be explicitly defined and discussed in Chapter II.

Homelessness and Substance Dependence

Though homelessness is not the primary area of interest for this study, it is important as the individuals assessed in this study are homeless male residents of the Guesthouse of Milwaukee, Inc. In addition, just as the prevalence of individuals struggling with substance use issues has been well documented in the literature, the prevalence of individuals struggling with homelessness is also well documented (Institute for the Study of Homelessness and Poverty, retrieved January 12, 2006 & March 21, 2009; National Coalition for the Homeless, retrieved January 12, 2006 & March 21, 2009; U.S. Department of Health and Human Services, retrieved January 12, 2006 & March 21, 2009). Finally, the relationship between homelessness and substance use disorders is also established in the literature (National Coalition for the Homeless, retrieved January 12, 2006 & March 21, 2009; SAMHSA, 2003; Solliday-McRoy, Campbell, Melchert, Young & Cisler, 2004; U.S. Department of Health and Human Services, retrieved January 12, 2006 & March 21, 2009) as is the relationship of homelessness and neuropsychological deficits (Gonzalez, Dieter, Natale & Tanner, 2001; Seidman et al., 1997; Solliday-McRoy et al., 2004). Therefore, assessing the relationship of executive functioning and attrition rates in homeless individuals involved in substance dependence treatment seems quite appropriate.

Treatment for Substance Dependence

The National Survey on Drug Use and Health (2008) classifies one as needing treatment if the person has a substance use disorder or one who received treatment at a specialty facility (i.e., hospital inpatient, drug or alcohol rehabilitation, or mental health centers). In 2007, the estimated number of people aged 12 or older needing

treatment for an alcohol or drug problem was 23.2 million (9.4 percent of the total population; NSDUH, 2008). Of these 23.2 million, 2.4 million received treatment at a specialty facility in the past year (NSDUH, 2008). Thus, 20.8 million people needed but did not receive treatment at a specialty treatment facility in 2004 (NSDUH, 2008).

Barriers to Treatment for Substance Dependence

Unfortunately, not everyone who needs substance use treatment actually receives substance use treatment. Of the 20.8 million people who needed but did not receive treatment in 2004, an estimated 1.3 million (6.4%) reported that they felt they needed treatment for their substance use problem (NSDUH, 2008). Of these 1.3 million, 380,000 (28.5%) reported that they made an effort but were unable to get treatment and 955,000 (71.5%) reported making no effort to get to treatment (NSDUH, 20048).

Based on combined data from 2003 and 2004, the NSDUH (2004) reports that of the people who felt they needed but did not receive treatment 40% stated they did not seek treatment because they were not ready to stop using and 34.5% reported cost or insurance barriers, 21.6% reported stigma and 13.9% reported they felt they did not need treatment (at the time) or could handle the problem without treatment. However, among the people who made an effort but were unable to get treatment, 42.5% reported cost or insurance barrier, 25.3% reported they were not ready to stop using, 21.5% report other access barriers and 17.8% report stigma (NSDUH, 2004). Combined data from 2004 and 2007, the NSDUH (2008) reports that of people who felt they needed treatment but did not receive treatment 38.7% said they were not ready to stop, 31.1 % had no insurance and could not afford treatment, 11.6 %

reported concern regarding possible negative effect on job, 11.65% reported not knowing where to go for treatment and 11.1% reported concern of negative opinion of others (NSDUH, 2008). A differentiation could be made between internal and external barriers as reasons people did not receive treatment. For example, lack of insurance and negative stigma could be considered external barriers whereas being not ready to quit or feeling one can handle the problem on their own could be considered internal barriers. Finally, along with lack of financial resources and health insurance, research has also indicated that lack of transportation may be a barrier to engaging in treatment (Knight & Longmore, 1994). For example, if the person has no reliable mode of transportation to the treatment facility, or if the treatment facility is located too far from the person's geographical location, the person may not enter treatment, or may not stay engaged once starting treatment due to the difficulty in getting to treatment.

Neuropsychological impairment as a barrier to treatment

One possible barrier to substance dependence treatment currently under investigation is that of neuropsychological impairment. Bates, Bowden and Barry (2002) have suggested that neuropsychological impairment may limit an individual's treatment engagement and/or may impede treatment completion in traditional outpatient substance abuse treatment. In fact, as discussed above, neuropsychological impairment resulting from substance use disorders is known to often be severe, but it is also true that neurological impairment frequently goes unassessed, unrecognized and untreated in individuals seeking treatment (Bates et al., 2002; Cunha & Novaes, 2004). Although research has begun to evaluate the neuropsychological functioning

and impairment of patients with substance abuse and mental health diagnoses (e.g. Bates et al., 2002, Lezak, 1995; Ratti et al., 2002, Sullivan, Fama, Rosenbloom & Pfefferbaum, 2002; Uekermann, Daum, Schlebusch, Wiebel & Trenckmann, 2002), which will be discussed later, neuropsychology as it relates to substance use disorders is a relatively new arena of study (Fals-Stewart & Bates, 2003).

Stigma and Substance Use Disorders

As mentioned in the NSDUH (2008) study above, concern about stigma has also been identified as a treatment barrier. Stigma, which involves the harm that may come from the label of mental illness or substance dependence, may impede treatment participation (Corrigan, 2004). People who are labeled mentally ill or substance dependent can be harmed publicly with stereotypes (e.g., "All people with mental illness and/or substance dependence are dangerous"), prejudice (e.g., "I agree, people with mental illness and/or substance dependence are dangerous and I am afraid of them"), and discrimination (e.g., "I do not want to be near them; don't hire them at my job"; Corrigan, 2004, p.617). Stigma may lead to people avoiding seeking treatment or staying in treatment in order to avoid the label and escape the public stigma (Corrigan, 2004). However, these barriers to treatment could also offer vital information for development of interventions specifically designed to break down treatment barriers.

"They must not have been ready." "Maybe they didn't want it bad enough." "I guess he just doesn't love us enough." "The bottle is more important than his kid." How often do we as counselors, as well as other people in the substance abuser's life, make these statements about someone who has failed to follow through with a

treatment plan for a substance abuse or dependency problem? In fact, negative stigma and judgments are not only made about substance abusers who do not complete treatment, but individuals with substance abuse problems are often judged and stigmatized harshly for even having the problem in the first place (Knight & Longmore, 1994). Many people, inside and outside of the substance abuse treatment field, seem to assume that the path to solving the problem is obvious - if you have a substance abuse or dependence problem, you go to one of many treatment facilities and get it fixed. On the surface, failure to follow through with prescribed treatment may appear to be a compliance issue. I have heard the stigma substance abusers face reflected in many clinicians' descriptions of people who do not follow through with treatment as "non-compliant," "lazy," "deviant," or having "complete disregard for themselves or their families." Overall, many researchers identify stigma as a barrier to substance use treatment (Corrigan, 2004; NSDUH, 2002 & 2004 & 2008; World Health Organization, 2004). Specifically, the World Health Organization (2004) identifies stigma as one of the main barriers to treatment and care of people with substance dependence and related problems. In addition, the National Mental Health Resource Center reported that no group encounters more stigma than homeless persons with co-occurring (mental health and substance use) disorders (National Mental Health Association retrieved January 12, 2006). Regardless of the level of substance use or which substance a person takes, they have the same rights to health care, education and work opportunities as any other individual (World Health Organization, 2004).

In short, stigma has been identified as one of the relevant factors in identification and treatment of substance use disorders (Corrigan, 2004; NSDUH, 2002 & 2004 & 2008; World Health Organization, 2004). Likewise, multiple neuropsychological deficits have been associated with substance use disorders (Bates et al., 2002; Lezak, 1995; Moselhy et al., 2001; Oscar-Berman et al., 1997; Sullivan et al., 2003) which may be affecting the level of stigma of individuals with a substance use disorder.

Neuropsychology of Substance Use Disorders

Research has suggested that alcoholism affects cognitive functioning such as recall, recognition (Knight & Longmore, 1994, Sullivan et al, 2002) abstract thinking, cognitive flexibility, and persistence and inhibition of competing responses (Zinn, Stein & Swartzwelder, 2004). Over the last decade, researchers have begun to specifically evaluate areas of the brain involved (Ratti et al., 2002). The same is true for drug abusing and dependent patients. Alcohol and drug abuse and dependence are associated with neuroanatomical changes that affect cognitive abilities such as reasoning, learning, memory, decision making and inhibition (Beatty, Tivis, Stott, Nixon & Parsons, 2000; Knight & Longmore, 1994, Pfefferbaum, Sullivan, Rosenbloom, Mathalon & Lim, 1998). Neuroimaging techniques reveal cortical shrinkage (Pfefferbaum et al., 1998), enlarged ventricles and increased space between the gyri of the cerebral cortex (Lilliquist & Bigler, 1992). This has been related to changes in neurobehavioral performance on specific neuropsychological tests of verbal problem solving, conceptual shifting, perceptual-spatial abilities, abstracting, motor speed, information processing and memory (Fals-Stewart & Bates, 2003).

Given these findings, when developing appropriate treatment programs, it would seem necessary to evaluate the patient's abilities and deficits and develop a program that fits their needs. Though research suggests that the neuropsychological deficits may affect treatment efficacy and attrition rates (Sullivan et al., 2002; Zinn et al., 2004), there is a paucity of research connecting neuropsychological deficits and treatment attrition despite use of numerous search engines (including Medline, Ovid, PubMed, PsychInfo, PsychArticles, ScienceDirect, Google) and multiple library systems (including Marquette University and the Medical College of Wisconsin) using multiple search terms (including, but not limited to, "neuropsychology, neuropsychological impairment/deficit, frontal lobe, attrition, drop out, treatment length, executive functioning, cognitive deficits" and various combinations of all of these).

Identifying neuropsychological deficits may be helpful in the development of treatment programs aimed at those substance dependent clients who have neuropsychological impairments (Kass & Silver, 1990). For example, executive function deficits such as deficits in planning and strategizing could affect treatment compliance. If one struggles with planning future events, attendance at treatment could be affected (Zinn et al., 2004). All of these neuropsychological deficits could affect attrition rates which will both be discussed in further detail in Chapter II. Knight and Longmore (1994) have suggested that clients' neuropsychological deficits related to substance use or of other origin may affect clients' attrition rates in substance use disorder treatment programs. However, the connection between neuropsychological impairment and attrition has not been empirically well

established. To date, we do not know the extent to which neuropsychological impairment has an impact on client attrition from substance use disorder treatment. Without such knowledge, we cannot effectively plan and implement substance use disorder treatment programs that might aid individuals suffering from substance use disorders and neuropsychological impairment.

Attrition and Relapse Rates Among Clients in Treatment for Substance Dependence

The purpose of this study is to explore the relationship of neuropsychological functioning and attrition rates in individuals who have engaged in outpatient substance use treatment. Attrition, referring to patients who enter but then drop out of treatment, has been studied through the National Outcomes Measures (2002) and reported on by many sources (Broome, Flynn & Simpson, 1999; DATOS, 2001; Franey & Ashton, 2002; Office of Applied Studies, 2004; Simpson et al., 1997; Simpson, Joe & Rowan-Szal, 1997; Stark, 1992).

The specific statistics reported for drop out rates for people in substance dependence treatment help to confirm the problem of attrition. For example, the medium length of stay specific to outpatient treatment is 76 days, based on 34 states submitting discharge information in 2005 (SAMHSA, 2008). The National Outcomes Measures collected data from 23 states during the year 2002 and found that the median length of stay for completion of outpatient treatment was 78 days (Office of Applied Studies, 2004). However, the median length of stay prior to dropping out was only 32 days In 2002 (Office of Applied Studies, 2004) and 45 days in 2005 (SAMHSA, 2008). Median length of stay for homeless individuals in intensive outpatient treatment was reported to be 45 days in 2005 (SAMHSA, 2008). Likewise,

drop out rates for homeless people from substance abuse treatment have been reported at 66% (SAMHSA, 1998), drop out rates for people with cocaine addiction in outpatient have been reported at 55% (Agosti, Nunes, Ocepek-Welikson, 1996), people with drug abuse in general have been reported to have between 40% to 60% drop out rate according to Marlowe and Dematteo (2003) and 55% according to Sayre, Schmitz, Stotts, Averill, Rhoades and Grabowski (2002). The literature on predicting attrition has been inconclusive. Variables such as sociodemographics, gender, psychiatric comorbidity and substance use severity have all been evaluated in relation to treatment drop out (Sayre et al, 2002).

Another important variable related to clients dropping out of treatment is client relapse. Relapse and attrition have an interactive relationship as each may be a cause or influence of the other. Many variables, including dropping out of treatment, have been related to relapse in substance abuse treatment (Franey & Ashton, 2002). Some of the variables identified include type of drug use (United Nations, 2002), gender (Stocker, retrieved September 20, 2005), support systems (United Nations, 2002), certain medications (United Nations, 2002; World Health Organization, 2004), length and intensity of treatment (Franey & Ashton, 2002; United Nations, 2002) and neuropsychological impairment (Miller, 1991). All of these will be discussed in detail in Chapter II.

The discrepancy between amount of time to treatment completion and actual time spent in treatment for patients who drop out is very important as duration of treatment has been identified as one of the best predictors of outcome for substance abuse treatment (Corrigan, Bogner, Lamb-Hart, Heinemann & Moore, 2005).

Therefore, evaluating variables that may be related to treatment attrition and relapse, such as neuropsychological functioning, would provide valuable, useful information.

Statement of the Problem

Substance abuse and dependence has tremendous effects on the individual and his or her family, as well as on society (APA, 2000). In addition, there are multiple barriers that have been identified as reasons people do not get the treatment they need (NSDUH, 2008). Likewise, many of the people who enter treatment do not complete treatment (NSDUH, 2008). Although we know that individuals suffering from substance dependence also may suffer from neuropsychological deficits, as well as that treatment attrition rates are high for those entering substance dependence treatment, we do not know if or how neuropsychological deficits may affect treatment engagement and attrition. It may be that an understanding of the potential relationship between neuropsychological functioning and attrition could aid researchers and clinicians who are endeavoring to develop helpful treatment programs to do so.

Purpose of the Study

The purpose of this study was to assess the neuropsychological functioning of clients who meet diagnostic criteria for substance dependence according to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition - Text Revision (DSM IV-TR; APA, 2000) and to examine the relationship between neuropsychological functioning and treatment attrition rates. Specifically, the executive functioning of individuals was evaluated. Furthermore, the relationships between substance use diagnosis, treatment attrition, and neuropsychological functioning was investigated. It may be that understanding how deficits in

neuropsychological functioning affect an individual's behaviors (e.g., relapse, missing treatment sessions, and dropping out of treatment) may help to change attitudes of clinicians and others who may currently negatively stigmatize those with substance use disorders (e.g., believing that the individual is lazy, unmotivated, etc.). Understanding the relationships between substance use diagnosis, attrition, and neuropsychological functioning could prove extremely useful in substance use disorder program development, treatment planning, clinician training and stigma reduction.

Research Questions

This study intended to address the following research questions:

(1) What is the level of neuropsychological functioning/impairment of this sample of substance dependent men?

(2) Do deficits in client's neuropsychological abilities including concept identification, cognitive flexibility, divided attention, perseveration, and impulse control predict rates of attrition from substance abuse treatment?

(3) How does neuropsychological functioning relate to relapse rates in those clients seeking treatment for substance dependence?

Definition of Terms

Attrition – "A decline in a population over time" (Reber, 1985, p. 69). For the purpose of this study, attrition refers to the number of participants who begin, but do not complete, treatment due to dropping out.

Executive Functioning - Those capacities that enable a person to engage successfully in independent, purposive, self-serving behavior (Lezak, 1995). Executive

functioning has to do with how a behavior is expressed. Questions about executive functions include *how* or *whether* a person goes about doing something, whereas questions about cognitive functions are phrased in terms of *what* or *how much* one knows (Lezak, 1995). Executive functioning has been identified as a function of the frontal lobe (Lezak, 1995; Moselhy, 2001; Snyder & Nussbaum, 1998).

Homeless – The term "homeless" will be limited to those individuals seeking refuge at the Guesthouse of Milwaukee, Inc. (a local homeless shelter).

Neuropsychological Functioning

Clinical Neuropsychology - The behavioral expression of brain dysfunction (Lezak, 1995); "a sub-discipline within physiological psychology that focuses on the interrelationships between neurological processes and behavior" (Reber, 1985, p.491).

Cognitive Functions - The information handling aspects of behavior, analogous to computer operations of input, storage, processing and output. In more detail, (a) *receptive functions* involve the ability to select, acquire, classify and integrate information; (b) *memory and learning* involve information storage and retrieval, (c) *thinking* concerns the mental organization and reorganization of information; and (d) *expressive functions* are the means though which information is communicated or acted upon (Lezak, 1995). Though these categories can be described as separate concepts, they are interdependent (Lezak, 1995).

Relapse- Any episode of alcohol or drug use by the participant after the date of admission to the substance use treatment program will be considered a relapse.

Substance Dependence - Substance Dependence is defined based on the criteria listed in the DSM-IV-TR which states:

A maladaptive pattern of substance use, leading to clinically significant impairment or distress, as manifested by three (or more) of the following, occurring at any time in the same 12-month period:

(1) tolerance, as defined by either of the following:

(a) a need for markedly increased amounts of the substance to achieve intoxication or desired effect,

(b) markedly diminished effect with continued use of the same amount of the substance,

(2) withdrawal, as manifested by either of the following:

(a) the characteristic withdrawal syndrome for the substance [For example, with alcohol withdrawal, two or more of the following symptoms are necessary: autonomic hyperactivity, increased hand tremor, insomnia, psychomotor agitation, anxiety, nausea or vomiting; and rarely, grand mal seizures or transient visual, tactile, or auditory hallucinations or illusions.]

(b) the same or closely related substance is taken to relieve or avoid withdrawal symptoms,

(3) substance is often taken in larger amounts or over a longer period than intended, (4) there is persistent desire or unsuccessful efforts to cut down or control the substance use,

(5) a great deal of time is spent in activities necessary to obtain the substance, use the substance, or recover from its effects,

(6) important social, occupational, or recreational activities are given up or reduced because of substance use,

(7) the substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance (e.g., current cocaine use despite recognition of cocaine-induced depression, or continued drinking despite recognition that an ulcer was made worse by alcohol consumption; APA, 2000, p. 197).

Importance of the Study

A mere 1.9 million of the 22.2 million people identified as needing treatment for a substance abuse or dependence disorder received treatment in 2003, 2.33 million received treatment of the 23.48 million identified in need in 2004 and 2.4 million of 23.2 million identified as in need in 2008 (SAMHSA, 2005; NSDUH, 2008). For those who wanted but did not enter treatment, factors such as lack of resources including money, medical insurance and transportation, as well as the stigma associated with having a substance dependence problem have been identified as barriers to treatment (Knight & Longmore, 1994; SAMHSA, 2005; NSDUH, 2008). However, the specific reasons individuals enter treatment and then drop out appear to be less clearly understood. The Office of Applied Studies (2004) discussed the median day of dropout (i.e., day 32), but did not discuss why the participant dropped out. Often times when a client does not continue in treatment it is viewed as non-compliance (Glyngdal, Sorenson & Kistrup, 2002). There is strong evidence that the degree to which clients engage and participate in treatment activities is related to the success of substance abuse treatment (Northwest Frontier Addiction Technology

Transfer Center, 2003). Success is not specifically defined in this article but seems to be suggested as following through with recommended treatment. Attrition from substance abuse treatment has been identified by some as one of the greatest problems interfering with the effectiveness of treatment programs (Jacobsen, 2004). To quote Shavelson (2001), "If there is a single consistent finding that has come out of rehab research it is that the longer clients can be maintained in the programs the more likely they are to emerge clean and sober, and stay that way." (Shavelson, 2001, p. 300). Quite obviously, one cannot be expected to benefit from treatment they are not present for. Furthermore, although much research has been conducted regarding neuropsychological impairment resulting from substance abuse and dependence, the research is limited in regards to neuropsychological impairment and treatment attrition. Though over 150 resources were used to research this study, only one was found that researched neuropsychological functioning and attrition rates (Fals-Stewart & Lucente, 1994). However, Fals-Stewart and Lucente's study (1994) included personality disorders with the neuropsychological impairment as it evaluated attrition rates. Likewise, as previously discussed, support exists for the problem of attrition including rates between 40% and 66% (Marlowe & Dematteo, 2003; SAMHSA, 1998). The literature on predicting attrition has been inconclusive. Variables such as sociodemographics, gender, psychiatric comorbidity and substance use severity have all been evaluated in relation to treatment drop out (Sayre et al, 2002).

The purpose of this study is to explore the relationship of neuropsychological functioning and attrition rates in individuals who have engaged in outpatient treatment. These results may provide important implications for treatment planning,

program development and training of clinicians to more effectively meet the needs of patients with a substance dependence disorder.

Brief Introduction to Proposed Methodology

Participants were recruited from the population of clients entering outpatient treatment for substance use disorders at the 7C's clinic within the Guesthouse. All participants agreed to informed, voluntary participation in the study. Participants all had a substance use diagnosis as defined by the DSM-IV-TR and confirmed by an assessment including the Form 90, Addiction Severity Index (ASI) and the Mini International Neuropsychiatric Interview (MINI). Participants had their current level of neuropsychological functioning assessed through the neuropsychological test battery established for this study which included subtests of the Delis-Kaplan, the Conners' Continuous Performance Test II and the WASI. We intended to have data from our established sample size of 100 collected over the course of six months by four clinicians (Addiction Counseling students in training) trained in the use of all assessment tools by licensed psychologists (Dr. Campbell & Dr. Young). As will be discussed later, our final sample size was less than 100. Participants were observed for three months or until the date they dropped out or were discontinued from treatment. Therefore, we intended that over the course of six months, approximately four to five participants per week would need to be evaluated for the study. We intended to complete data collection within nine months which allowed for three months past the six month mark allowing for the three months of observation for the final participants included. This data collection was also supervised by a licensed psychologist (Dr. Todd Campbell) available on a regular basis for questions and

consultation and a neuropsychologist who was also available for questions and consultation (Dr. Terry Young).

The statistical methodologies proposed and used in this study are Survival Analysis (SA) and Logistic Regression (LR). Survival Analysis traditionally has been used for medical research as it is useful for longitudinal studies for things such as survival rates with cancer or organ transplants (Parmar & Machin, 1995). Survival Analysis can be utilized for longitudinal data such as the length in outpatient treatment that we will be studying (Corning & Malofeeva, 2004; Gerstman, 2003, Pamar & Machin, 1995). One difference between survival data and other types of numeric continuous data is that the time to the event occurring (e.g., day of drop out or end of successful treatment) might not be observed in all participants in particular studies due to variables such as death of participants or an end of treatment date that is beyond the length of the study. This non-observed event is accounted for in survival analysis (Corning & Malofeeva, 2004; Parmar & Machin, 1995) making it a very appropriate method for this study. Logistic Regression will allow us to evaluate the relationship between neuropsychological function and the dichotomous variables of drop out vs. no drop out. The different statistical methods allow us to evaluate all important variables in this study.

The dependent variables for this study include the total length of time spent in outpatient treatment prior to drop out and whether or not the participant drops out. The independent variable is the level of neuropsychological functioning or impairment. The null hypothesis of this study is that there will be no relationship between the level of neuropsychological functioning and a participant's length of

attendance in outpatient substance dependence treatment or drop out status. We had also intended to evaluate relapse rates, but as will be discussed below, we were unable to address this issue.

Chapter II: Review of the Literature

This chapter will assist the reader in gaining an understanding of the history of neuropsychological assessment, as well as, the anatomy of neuropsychological functioning. Finally, research specific to executive functioning, substance dependence and dual diagnosis will be presented, as well as, research specific to attrition rates.

Neuropsychology as a Clinical Discipline

Defining Neuropsychology

Neuropsychology is an applied science concerned with the behavioral expression of brain dysfunction (Lezak, 1995; Lezak, Howieson, Loring, Hannay & Fischer, 2004). Clinical neuropsychologists deal with a variety of questions regarding human behavior and brain functioning, a wide range of normal and abnormal behaviors and diverse people with regards to demographics, culture and pathology (Lezak, 1995). Therefore, the practice of neuropsychology requires of its practitioners flexibility of mind, curiosity about the myriad of factors of human behavior and inventiveness with regard to clinical interventions in even the most routine work undertaken (Lezak, 1995; Snyder & Nussbaum, 1998; Stirling, 2002)

Neuropsychologists interact with professionals from many other psychological and medical clinical disciplines, including other psychologists,

psychiatirists, counselors, family practice physicians, gerontologists and emergency room personnel (Lezak, 1995; Lezak et al, 2004; Meier, 1992; Snyder & Nussbaum, 1998). The interaction of neuropsychology with other disciplines allows for a more thorough diagnosis and treatment plan with attention to details regarding brain functioning, strengths and weaknesses for multiple presenting concerns including(but not limited to) behavioral disorders, mood disorders, head injuries, learning disabilities and dementias (Lezak, 1995). At one time, clinical psycholgogists determined brain damage mainly with Wechsler's intelligence tests. Traditional tests are still very useul, but not specific enough to identify specific signs of brain damage such as language deficits and attention problems (Lezak, 1995). With specialized training, neuropsychologists evaluate aspects of intelligence, reasoning, abstraction, attention, executive functions, learning, memory, language, auditory, visual, motor functions and constructional tasks (Lezak, 1995). The neuropsychologist uses specialized assessments to examine the relationship between the brain and behavior helping to identify brain damage, cognitive dysfunction and patient strengths and weaknesses, all of which can be extremely helpful in patient treatment planning and rehabilitation (Seidman, 1998).

Development of Clinical Neuropsychology

Clinical Neuropsychology evolved from its parent disciplines of neurology and psychology, developing an identity of its own in the 1940s (Lezak, 1995). In the 1940s, prior to performing a craniotomy, neurosurgeons relied on electroencephalograms (EEG's), X-rays and neuropsychological reports for localization giving the neuropsychologists a well-defined niche (Lezak, 1995; Ruff,

2003). However, in the 1970s when computerized tomography became available, the neuropsychologist's role in localization became less important. Therefore, the neuropsychologist's role shifted focus to obtaining quantitative descriptions of a patient's cognitive status (Ruff, 2003). Likewise, Lezak (1995) reports, that in the 1940s "psychology's looser constructs were undergoing reexamination in the cold light of operationalism" (Lezak, 1995, p. 3). More specifically, the prominence of "intuitive modus operandi of the earlier armchair and couch theoreticians was giving way to more rigorous-appearing actuarial (statistical probability) techniques" (Lezak, 1995, p. 3). In strict actuarial approaches, the neuropsychologist need not even see the patient, but rather draw conclusions from scores obtained by a technician (Lezak, 1995). However, through the development of testing batteries developed by some of the leaders in the field, neuropsychology developed into more of a mix of the intuitive and actuarial (Lezak, 1995). Some of the leaders and batteries are discussed in the Neuropsychological Assessment section below.

To further distinguish the neuropsychologists in the field of psychology, the formation of the International Neuropsychological Society (INS) was organized in 1966 at the Albert Einstein College of Medicine. This was a landmark for the formal organizational structure of clinical neuropsychology (Meier, 1992). The organization of the INS provided the necessary organizing of a group of neuropsychologists that had been gathering at APA (Meier, 1992). In the 1970s, the organization grew to become international, as well as interdisciplinary, with psychologists, psychiatrists, neurolinguists, neurosurgeons and more becoming members (Meier, 1992). The INS also began publication of *The Journal of Clinical Neuropsychology*, later renamed

Journal of Clinical and Experimental Neuropsychology (Meier**,** 1992). As the INS continued to assume a position of leadership, the stage was set for establishing a division within the American Psychological Association (APA). Finally, in 1980, the Division of Clinical Neuropsychology (Division 40) was formed within APA (Meier, 1992). Though relatively still young as a field, neuropsychology's growth can be noted in an increasing number of clinical practicum sites, journals, clinical internship sites and curriculum planning focused specifically on neuropsychology (Snyder & Nussbaum, 1998).

Neuropsychological Assessment

Early Neuropsychological Test Batteries

As discussed by Lewis and Sinnett (1987), the first neuropsychological test battery was developed by Goldstein and Scheerer. Goldstein, a neuropsychiatrist, and Scheerer were both trained in the Gestalt psychology tradition drawing on experience with brain injured German soldiers in World War I (Lewis & Sinnett, 1987). However, the lack of standardization, lack of objective scoring, lack of reliability and validity data made clinicians hesitant to adopt the battery (Lewis & Sinnett, 1987).

Halstead-Reitan Neuropsychological Test Battery.

A few years later, in 1947 W.C. Halstead, a Northwestern University PhD, initiated the development of the first standardized neuropsychological test battery, as well as the formation of the first neuropsychology laboratory in the United States (Lewis & Sinnett, 1987). Halstead and his graduate student, Ralph Reitan, together developed the Halstead-Reitan Neuropsychological Test Battery (Lewis & Sinnett, 1987), currently one of three commonly used batteries in the United States (Seidman, 1998).

The version of Halstead Reitan Battery (HRB) that is most commonly used at present consists of five subtests including the (a) Category Test, (b) Tactual Performance Test, (c) Rhythm Test, (d) Speech Sounds Perception Test, and (e) Finger Oscillation Test or Finger Tapping Test (Lezak, 1995). The two tests that were part of the original seven that are not commonly used any longer include the Critical Flicker Fusion Test and the Time Sense Test (Lezak, 1995). A distinctive feature of Reitan's handling of examination data of the HRB was his reliance on test scores for predicting nature and site of a lesion (Lezak, 1995). Although the HRB has practical limitations in that it takes a long time to administer and is not considered suitable for thorough examination of patients with sensory or motor handicaps, it offers one of the more reliable psychological means for identifying patients with brain damage (Lezak, 1995).

Luria Nebraska Battery.

Russian neuropsychologist A. R. Luria was the primary developer of the Luria Nebraska Battery (Lezak, 1995). Luria's contributions to neuropsychological testing consist of obtaining sensitive, qualitative, behavioral descriptions, emphasizing the uniqueness of each individual patient (Lezak, 1995). Luria's approach to neuropsychological assessment was clinically focused versus empirically focused (Lezak, 1995). He often administered his assessment battery at a patient's bedside paying particular attention to the *means* the patient used to solve a problem rather than the *outcome* of the test (Lezak, 1995). Luria was more concerned with what he observed clinically versus what the results of the test indicated. As a result of the manner in which Luria approached and administered testing, the data collected on

each individual was rich, but due to the lack of standardized administration procedures (i.e., the testing procedures changed dependent upon the patient's responses, Luria's approach to testing did not allow for duplication (Lewis & Sinnett, 1987). As Luria's approach was very individualized and was difficult to duplicate, it had many qualitative characteristics. Luria's approach to neuropsychological assessment led to many present day neuropsychologists approaching assessment from an integrated qualitative-quantitative approach (Lezak, 1995). Specifically, the assessor can take advantage of the standardized assessments for quantitative analysis while also using clinical training to assist in the attention to more qualitative features for a more eclectic evaluation (Lezak, 1995).

Boston Process.

According to Seidman (1998), the third neuropsychological test battery widely used at present is the Boston Process Neuropsychological Approach, which has many variants. The examiner begins with few measures and focuses the assessment more precisely as more information is learned about the patient (Seidman, 1998). This approach lends itself to a more flexible model of assessment again incorporating the qualitative and quantitative pieces (Seidman, 1998).

Although the Boston approach has many variants, most versions include tests of intelligence, memory, abstraction, naming ability, visuo-constructional, organizational and tests of executive function (Seidman, 1998). Sometimes other tests are added to evaluate dementia, aphasia and personality issues. The emphasis with the Boston battery is more on how patients perform rather than merely whether they

succeed or fail (Seidman, 1998). Therefore, examiners can use the Boston approach to identify possible damage even when the final performance score falls within the identified normal range (Seidman, 1998). Furthermore, Seidman (1998) supports the use of the Boston Process battery when the possibility of malingering is high (such as legal cases with the possibility for monetary gain) because processes are more difficult to fake than are results (Seidman, 1998)

Neuropsychological assessment battery for this study.

The instruments used for this study included specific subtests of the Delis-Kaplan Executive Function System (D-KEFS) and the Conners' Continuous Performance Test II (CPT II) for evaluation of neuropsychological strengths and weaknesses. Furthermore, the Wechsler Abbreviated Scale of Intelligence (WASI) was used for an estimate of general intellectual ability and the Form 90 and Addiction Severity Index (ASI) for substance dependence assessment. In addition, the Addiction Severity Index (ASI) was used to evaluate the severity of problem areas associated with alcohol and drug dependence and the Mini International Neuropsychiatric Interview (MINI) was used to establish any other DSM-IV diagnoses. All of these assessments will be discussed in detail in Chapter III.

Overview of Frontal Lobe Anatomy and Functions

Due to the complex nature of neuroanatomy, this review is meant to be basic, certainly not all-inclusive and exhaustive. The next section provides an overview of the anatomy and specific functions of the frontal lobe.

Frontal Lobe Anatomy

Very simply stated, the brain is divided into two hemispheres, left and right. Furthermore, the cerebral cortex is divided into four lobes: (a) frontal, (b) parietal, (c) temporal, and (d) occipital (Society for Neuroscience, 2002). For the purpose of this study, our focus will be on the frontal lobes.

In humans, the frontal lobes account for approximately one-third of the cerebral cortex (Goldman-Rakic, 1987). The frontal lobe can be subdivided into a number of functional subsystems including: (a) primary motor cortex – responsible for critical to fine motor movement, receives projections from posterior cortical areas involved in somatosensory perceptions as well as subcortical input from the ventral lateral thalamic nucleus, (b) premotor area - involved in sensorimotor integration and complex volitional movement having connections to the parietal lobe, (c) frontal eye fields - permit volitional eye movement in the contralateral visual fields necessary for voluntary gaze and visual search, (d) orbital and basal areas - affecting anosmia (deficiency in smell) and disinhibited personality changes, though few measures are available of orbitofrontal functions in humans, (e) supplemental motor and anterior cingulate gyrus - areas possibly forming a reciprocal system responsible for environmental search and inhibition of exploratory behavior, and (f) the dorsolateral prefrontal subsystem - responsible for executive functions (Kandel, Schwartz & Jessell, 1991; Malloy & Richardson, 1994; Snyder & Nussbaum,1998). Due to the impact alcohol and drugs have been found to have on the dorsolateral prefrontal subsystem (which will be discussed in further detail in the next section), the executive functions of the dorsolateral prefrontal subsystem are the neuropsychological

components we are most interested in for the purpose of this study (Knight & Longmore, 1995; Lezak, 1995; Moselhy, 2001).

Dorsolateral Prefrontal Subsystem and the Executive Functions

As previously mentioned, the dorsolateral prefrontal subsystem is considered mostly responsible for executive functions (Kandel, Schwartz & Jessell, 1991; Malloy & Richardson, 1994; Snyder & Nussbaum, 1998). The executive functions are necessary for appropriate, socially responsible and effective self-serving conduct (Lezak, 1995). The executive functions can be conceptualized as having four components, each involving a set of activity-related behaviors and all having a fair amount of overlap (Lezak, 1995). The four components are (a) volition, (b) planning (c) purposive action, and (d) effective performance (Lezak, 1995; Lezak et al, 2004).

Volition.

Volition, in short, is the capacity for intentional behavior (Lezak, 1995). Volition requires the capacity to formulate a goal or intention. It may be easiest to describe volition by examining deficits in volition. People who lack volitional abilities simply cannot think of anything to do or may be unable to initiate activities except in response to external stimuli such as someone giving them continuous prompting. There are various levels of volitional impairment ranging from mild to much more severe (Lezak, 1995). A mild case of volitional impairment could involve someone successfully engaging in games, chores or familiar routines without prompting, but being unable to assume longer term responsibilities (such as

employment) without outside guidance. Someone with more severe impairment may know the proper use of eating utensils, but may not take the initiative to eat the food placed in front of them without continuous prompting (Lezak, 1995; Shallice & Burgess, 1991).

Planning.

Planning abilities involve the identification and organization of the necessary skills and resources needed to carry out a plan or goal (Klein, 2000). For example, a patient with planning deficits may not be able to plan a future activity such as the steps needed to assemble a swing set (Klein, 2000). One must be able to look ahead, conceive alternatives and weigh out choices. Planning abilities involve reasonably intact memory, good impulse control and capacity for sustained attention (Lezak, 1995). One might find it necessary to repeat questions or instructions several times to patients with planning deficits in order for the patient to be able to direct his or her effort and concentration on completing the task (Stuss & Benson, 1984).

Planning deficits would certainly have implications for treatment. For example, a clinician working with clients who have deficits in planning would need to use repetition when giving instructions or facilitating participation in therapy. Likewise, the clinician may need to use redirection and attention gathering tactics repeatedly and frequently. For example, the clinician may need to verbally redirect clients to remind them to stay on task and maintain their attention to a particular task. Finally, clients may need verbal reminders of appointments repeated in the form of letters, phone calls or any other available sources.

Purposive action.

The translation of a plan into an activity is purposive action (Lezak, 1995). This involves the ability to initiate, maintain, switch, and stop sequences of behavior in an ordered manner (Lezak, 1995). For example, a patient might have a plan to attend an Alcoholics Anonymous meeting, but actually turning that plan into the action of going does not happen. A deficit in purposive action is most important when the actions are not routine. Overlearned, familiar, or automatic tasks are much less vulnerable to frontal lobe damage than more novel tasks (Lezak, 1995). For example, the overlearned task of putting one's shoes on is less likely to be impaired than the attendance at a new meeting in the community.

There are multiple aspects of purposive action that can be impaired. One's ability to self regulate can affect their success at productivity (Lezak, 1995). This gap between planning and activity becomes apparent in patients who are "all talk, no action". This is different from the occasional tendency of someone to not follow through on their word. The gap between planning and action is frequent and persistent giving it a pathological flavor. Flexibility and the capacity to shift one's behavior or thoughts can also be impaired (Zinn et al., 2004), resulting in difficulty conforming to social norms or expectations, rigidity in thinking or behaviors or a tendency to perseverate with behaviors or thought streams (Lezak, 1995; Ratti et al., 2002). For example, a patient who perseverates with behaviors or thought streams might have the exact same routine daily or tell the clinician the same story over and over. The client with rigidity or perseveration issues may present to clinicians as someone who is unmotivated, difficult to redirect or disruptive. Using multiple methods of

redirection and reinforcement could prove helpful versus becoming frustrated and assuming the client is not motivated to participate in treatment.

Effective performance.

A patient's performance on any activity can only be as effective as his/her ability to monitor, self-correct and regulate the qualitative aspects of the delivery of the action (Lezak, 1995). Some patients who suffer deficiencies in executive functioning, including problems in effective performance, may not perceive errors they have made on any task or may perceive errors but do nothing to correct them (Lezak, 1995). They may perform any task erratically or just simply unsuccessfully (Lezak, 1995). Patients with frontal lobe impairment may also have insensitivity to possible consequences (i.e., punishment or reward) which affects their ability to make sound decisions (Bechara, Tranel & Damasio, 2000). A patient's lack of effective performance on any given task may be the result of not perceiving errors or perceiving but not correcting the errors (Lezak, 1995). The lack of self correction may result from an abnormal sense of self awareness or possibly just inertia (lack of purposive action; Lezak, 1995). If one does not perceive what they are doing, it is difficult to correct. Likewise, if one has no inertia, they also would not correct any possible errors.

Another syndrome associated with abnormalities of self-awareness is that of confabulation (Lezak, 1995). Confabulation is defined as the presentation of incorrect, sometimes bizarre information to standard questions (Lezak, 1995). For example, when asked about their recent substance use, the client might report a long story about wild events at work. They might talk while the clinician is speaking and

not directly respond to specific questions without repeated redirection and clarification. Once the response is made, the answer might contain a series of formations of false memory, perceptions or beliefs mixed with some truth, pouring out of irrelevant associations or the response may contain no reality at all. Confabulation is not necessarily related to a memory disturbance, but instead is due to the ability to self correct (Stuss & Benson, 1984**).** Therefore, the patient's lack of effectiveness with any task may be due to multiple issues of self correction, self awareness and self regulation (Stuss & Benson, 1984).

Frontal Lobe Functions

Historically, frontal lobe functions have been poorly understood (Lezak, 1995; Snyder & Nussbaum, 1998). For example, for many years clinicians referred to the prefrontal lobes as the *silent areas* because sensorimotor signs were often absent after prefrontal damage (Snyder & Nussbaum, 1998). However, due to continued research on humans and animals, as well as developments in structural and functional neuroimaging, we now have a much greater understanding of this area of the brain (Snyder & Nussbaum, 1998).

As described with deficits in volition, planning, purposive action and effective performance, frontal lobe disorders affect *how* a person responds to others, which can affect the content of any response (Lezak, 1995). Disorders affecting the frontal lobes tend not to disrupt cognitive functions (such as reporting on specific knowledge) as obviously as does damage in other areas of the brain such as occipital (Lezak, 1995). Therefore, frontal lobe dysfunction may be harder to detect, go undetected or be attributed to other causes such as the client's noncompliance or lack of desire to

participate or address problems (Lezak, 1995). This then impacts the judgments others make about the client and possibly resulting in increased stigma.

Deficits in functioning stemming from damage to the frontal cortex can also affect one's ability to pay attention, as well as one's prospective memory (i.e., one's ability to remember to remember; Lezak, 1995). Therefore, if a patient has attention or memory deficits, they will have a difficult time retaining what is said in treatment, remembering appointments, locations, bus schedules and much more. The deficits in memory and attention could result in frustration and increased drop out rates. Likewise, the deficits in memory and attention may also be misperceived by clinicians and others resulting in continued stigma.

Frontal lobe damage and cognitive functions.

In regards to cognitive functions, frontal lobe disorders usually do not result in the loss of a specific skill or specific information (Bechara et al., 2000; Salloway, 1994; Shallice & Burgess, 1991). In fact, patients with frontal lobe disorders often perform within normal ranges on formal ability tests such as tests of intelligence and tests where they have direction through a series of problems (Bechara et al., 2000; Shallice & Burgess, 1991). Tests of intelligence often ask for factual information which is not affected normally with frontal lobe disorders (Bechara et al., 2000; Shallice & Burgess, 1991). Likewise, in tests where they are directed through a series of tasks, they have the benefit of the examiner's direction**.** Instead, the difficulties in functioning for people with frontal lobe deficits are related to initiating, planning, and organizing abilities, and therefore assessment must include tests designed to examine

the patient's functioning in initiating action, planning tasks, and organizing abilities (Bechara et al., 2000; Shallice & Burgess, 1991).

(Often Misinterpreted) Behavioral Problems Associated with Damage to the Frontal Lobe

Misinterpretation and Stigma

Common complaints from the people around patients with frontal lobe disorders include that the patient seems apathetic, careless, has poor or unreliable judgment, poorly adapts to new situations and has a blunted sense of sensibility (Daffner et al., 2000; Lezak, 1995). In treatment, this might present as someone who does not want help or is uninterested in what the clinician or others have to say. Substance abusing patients with deficits such as information processing, distractibility, difficulty with attention and problem solving can result in the patient missing parts of what they are told leading to issues such as emotional lability, hypersensitivity, low frustration tolerance or paranoia in the patient (Fals-Stewart & Lucente, 1994). Substance abusing patients with such deficits are often described as irritable, impulsive, perseverative and socially disinhibited (Fals-Stewart & Lucente, 1994). Similarly, Bates, Voelbel, Buckman, Labouvie and Barry (2005) reported that cognitive deficits in patients in substance abuse treatment "may lead to lack of motivation and treatment engagement, which are often interpreted as negative client attributes by treatment providers" (Bates, Voelbel, Buckman, Labouvie & Barry, 2005, p. 373). Furthermore, when therapists were informed of the deficits in functioning, the therapists rated participation and therapeutic alliance higher and patients subsequently stayed in treatment longer (Bates et al., 2005).

As previously discussed, the stigma associated with substance use disorders is a relevant factor in why individuals do not engage in treatment (Corrigan, 2004). Understanding and reducing this stigma, which results at least partially from the neuropsychological deficits associated with substance use disorders, is an important reason for this study.

Introduction to Behavior Problems

There are five general behavioral problems that are often misinterpreted in patients with frontal lobe damage adding to the prevalence of stigma associated with substance use disorders (Lezak, 1995). The five behavioral problems, with much overlap amongst them, associated with damage to the frontal lobe include (a) problems in behavior starting, (b) difficulty making mental and behavioral shifts, (c) problems in stopping, (d) deficient self awareness, and (e) a concrete attitude (Lezak, 1995). These five general behavioral problems are often misinterpreted by clinicians, family, friends, coworkers and society in general, leading to the development and perseveration of the stigma that accompanies individuals with frontal lobe damage (Lezak, 1995). The five behavioral problems and their possible misinterpretations are discussed below.

Problems in behavior starting.

The problem of behavior starting relates to the previously discussed volition and purposive action. When compared to their behaviors previous to frontal lobe damage, individuals who suffer with problems in behavior starting exhibit decreased spontaneity, decreased productivity, decreased rate at which behavior is emitted and decreased or lost initiative as compared to the patient's normal level of functioning

(Lezak, 1995). As a result of these particular deficits, patients who exhibit problems in behavior starting may appear lazy or apathetic to the casual observer (Daffner et al., 2000). Many can "talk a good game" but are unable to transform words into action. An extreme dissociation between words and actions has been termed *pathological inertia* (Lezak, 1995). The frontal lobe patient has no problem in describing a viable course of action (e.g., verbally describes when, where and how they will attend a community support meeting), but is unable to carry out the plan (i.e., never actually go to the meeting; Lezak, 1995).

Difficulty making mental and behavioral shifts.

A second behavioral problem associated with purposive action that may manifest as a result of damage to the frontal lobe is that of difficulty in making mental or behavioral shifts (Lezak, 1995). Referred to as *perseveration* or *rigidity* (Lezak, 1995), difficulties are seen in the individual's ability to shift attention from one thing to another, to make changes in physical movement or to maintain flexibility in attitude. Specifically, perseveration refers to repetition or continuation of an act or response to a question or situation (Lezak, 1995). In patients who have damage to the frontal lobe, perseveration tends to be supramodal, meaning that perseveration is exhibited in a variety of situations and a variety of tasks (Lezak, 1995). For example, the client might like to tell the same story of the day they met the president every time they attend a session anywhere with anyone present. Similarly, activities such as stopping at the same bar every night or calling the same drinking friends for support may be due to the individual's inability to change the behavior as the result of frontal lobe damage (i.e., perseveration) rather than being due to the individual's choosing

the behavior, which is what the casual observer might assume without information to the contrary. Perseveration can manifest itself as repetitive prolongation or continuation of an act or sequence of activities, or similar responses to a variety of questions, tasks or situations (Lezak, 1995). I have heard clinicians become frustrated with clients who do the same act expecting different results-often the result of perseveration.

Problems in stopping behaviors or responses.

The third behavioral problem for individuals with frontal lobe damage involves the effective performance component of frontal lobe damage, or more specifically, difficulty stopping behaviors or responses (Bechara et al., 2000; Lezak, 1995). The inability to stop behaviors results in the patient's impulsivity, over reactivity, disinhibition and difficulty holding back a wrong or unwanted response (Lezak, 1995). Because of their behaviors, patients exhibiting difficulties with stopping behaviors are often classified as having a loss of control or control problems (Lezak, 1995). The difficulty in stopping behaviors could result in verbal outbursts that some might find offensive, as well as, an inability to maintain abstinence in any situation where the patient may be exposed to a substance of abuse. These types of behaviors might be misinterpreted as simple noncompliance by the unaware observer. Education regarding the inability to stop behaviors and specific behavioral plans for the client and the clinician could be helpful to the individuals struggling with disinhibition.

Deficient self awareness.

Deficient self awareness, resulting in an inability to perceive performance errors on any given task (for example, forgetting to butter the bread prior to placing it in the frying pan to make grilled cheese, and then wondering why the pan is smoking), inability to appreciate the impact one makes on others, and/or to evaluate social situations appropriately (for example, not noticing that others are upset with you or giving you social cues meant to get you to leave) is the fourth behavioral problem (Lezak, 1995; Stirling, 2002). For example, people with deficient self awareness may be euphoric and self-satisfied at times when such feelings are unwarranted (for e.g., feeling satisfied with one's parenting though only having contact with the child a couple times per year) causing a client to have multiple problems in relationships with friends, family or therapy connections (Lezak, 1995). Deficient self awareness may be misinterpreted as rude, lazy, insensitive or again as non-compliant. Again, education focused on increasing the individual's awareness of the problem and its effects on others, as well as, a plan for alternative actions versus ineffective actions would be imperative.

Loss of abstract attitude.

The fifth problem associated with frontal lobe disorders is due to the loss of the abstract attitude (Lezak, 1995). As a result of impairments in abstracting and conceptual thinking, the patient holds an extremely literal understanding of life where all objects, behaviors or experiences are evaluated only with regards to face value (Stirling, 2002). The patient becomes incapable of planning or sustaining goal directed behavior because they are responding in such a literal manner (Lezak, 1995).

Frustration can again arise with misinterpretations by clinicians and others. This client may be misinterpreted as being “difficult” or possibly immature.

Summary of Executive Functioning Deficits Found in Individuals with Frontal Lobe Damage

Overall, frontal lobe functions, and more specifically, executive functions are large and complex influences in human functioning and behavior. When examining the possible deficits caused by frontal lobe damage, several categories of behavioral problems have been defined (Lezak, 1995; Parsons & Nixon, 1993; Stirling, 2002). However, the behavioral problems exhibited are not exclusive to the defined categories; there is a fair amount of overlap, not to mention that much about the deficits remains unknown (Lezak, 1995; Stirling, 2002). For example, all of the behavioral problems discussed are described as if existing as separate concepts, but in real life the behaviors present as a mixture of some or all categories of possible deficits. Furthermore, the extent to what the exact presentation of behaviors in each individual will be, as related to any neuropsychological damage, is unknown (Lezak, 1995; Snyder & Nussbaum, 1998). The overlap and complicated nature of frontal lobe functions makes assessment and identification of problems difficult (Lezak, 1995). Likewise, the complex nature and presentation of the impairment in the individual, as well as, the misinterpretation by observers provides a breeding ground for frustration, labeling and stigmatization by those who interact with a person with frontal lobe damage. Finally, the five specific behavioral problems discussed and their misinterpretations provide specific areas where stigma is born and magnified

due to the overlapping and often unrecognized problems associated with the frontal lobe (Lezak, 1995).

Neuropsychological Functioning and Substance Dependence

Lewis and Sinnett (1987) discussed the fact that there are many "silent victims" of neuropsychological impairment, referring to the fact that many people's impairments go undetected or misdiagnosed. Some of the silent victims include people with brain injuries, rare metabolic disorders, and more common disorders such as substance abuse

(Lewis & Sinnett, 1987). Bates, Bowden and Barry (2002) estimated that between 50% and 80% of individuals with alcohol use disorders experience mild to severe neurocognitive impairment (Bates et al., 2002). Likewise, Parsons and Nixon (1993) estimated that as many as 50% to 85% of individuals with alcohol use disorders will manifest mild to moderate impairment in some aspect of neuropsychological functioning (Parsons & Nixon, 1993).

Substance abuse and dependence can affect many complex areas and functions of the brain (Lezak, 1995; Parsons & Nixon, 1993). For example, alcohol dependence has been shown to affect the cerebellum, Pukinje cells and many other specific areas of the brain resulting in disruption of motor functioning as well as other specific frontal lobe functions including verbal learning, cognitive planning and attentional set shifting (Sullivan et al., 2003). However, because this study seeks to evaluate the relationship of executive functioning and attrition rates in substance use treatment, I will present a brief overview of the possible effects of alcohol and drug use on the frontal lobe and executive functioning.

Effects of Alcohol Use on Frontal Lobe and Executive Functioning

Reduced brain weight, particularly affecting the frontal lobe has been shown among alcoholic patients upon autopsy and neuroimaging studies (Bates et al., 2002; Sullivan et al., 2003). With such information about the impact of chronic alcohol use on the frontal lobe, researchers have begun to look more seriously at executive functioning and substance use disorders. For instance, Ratti, Giardini and Soragna (2002) evaluated the neuropsychological functioning of 22 male alcoholics (met DSM IV criteria for alcohol dependence, no history of significant drug abuse, ages 30-65, no head injury or medical condition affecting cognitive functions) and 22 non alcoholic controls (no DSM IV diagnosis, no neurological disorders, healthy, right hand dominant, habitually drink less than 40 grams of alcohol per day) using results on several neuropsychological tests. The tests used were Digit Symbol (assessing psychomotor performance), Stroop (selective and focused attention, ability to suppress irrelevant information), Digit Cancellation (selective attention), Trail Making (visual conceptual and visual motor tracking skills, mental flexibility) and the Wisconsin Card Sort Test (WCST; problem solving, abstraction, cognitive flexibility, concept identification, hypothesis generation, ability to use feedback; Ratti, Giardini & Soragna, 2002). This test battery would be appropriate for the author's purpose of evaluating executive functions. For all tests, mean and standard deviation were also calculated. The authors found the alcoholic participants to be impaired in almost every executive function assessed (Ratti et al., 2002). Specifically, results indicated statistically significant differences in the functioning levels between alcoholics and

non-alcoholics (Ratti et al., 2002). The alcoholic participants performed statistically significantly worse than did the non-alcoholic control group in the digit cancellation (M = 44.6 +/- 11.2, p = .0001), the digit symbol (M = 28.2 +/- 14.8, p = .005), the trail making test (M = 167.9 +/- 100.2, p = .01) and reaction test (M = 433 +/- 105, p = .001; Ratti et al., 2002). On the WCST, which evaluates problem solving and abstraction abilities, the alcoholics were also found to be impaired (M = 54.5 +/- 20.0, p = 0.00001). However, they were not impaired on the WCST in the area of perseveration (Ratti et al., 2002).

The strengths of this study include the author's use of a control group (i.e., the non-alcoholic group), and the evaluation of the participant's physical health, so as to not interfere with results. Though the test participants and controls were matched for age, education and IQ, no mention was made of evaluation of socioeconomic status or ethnicity. If the authors were using assessments that were not normed for their participant's socioeconomic status or ethnicity, this could adversely affect the results. Overall, the authors conclude that executive functions are impaired by alcohol dependence (Ratti et al., 2002).

Likewise, researchers have found metabolic abnormalities in the left dorsolateral prefrontal cortex on a study performed on 17 chronic alcoholics including 11 men and 6 women (Dao-Castellana et al., 1998). The subjects were ages 25 to 65 and had been hospitalized for detox from one week to one month. All subjects had been abstinent from alcohol and illicit drugs since hospitalization. Nine normal, non-alcoholic participants were recruited as controls for the imaging studies and neuropsychological evaluations were performed on eight controls (Dao-

Castellana et al., 1998). It is unclear within this study why nine were used for the imaging and only eight for the testing. Likewise, it is unclear why 17 alcoholic participants were studied and only a total of nine, non-alcoholic controls were studied. The controls had normal clinical, neurological and psychiatric examinations and normal MRI images (Dao-Castellana et al., 1998). All subjects had a PET scan and an MRI on the day they were administered the neuropsychological tests. The PET scans found a statistically significant decreased cortical metabolism in the left frontal lobe ($p = 0.048$), mediofrontal region ($p = 0.002$) and close to statistical significance ($p = 0.084$) in the left prefrontal region in the alcoholic participants (Dao-Castellana et al., 1998). Similarly, the MRI showed significant cortical atrophy in the mediofrontal ($p < 0.001$), right dorsolateral prefrontal ($p = 0.005$) and left dorsolateral prefrontal regions ($p < 0.001$) in the alcoholic participants (Dao-Castellana et al., 1998). Likewise, statistically significant reduced verbal fluency ($p = 0.014$) and impaired performance on the Stroop test ($p = 0.003$) were noted on these alcoholic participants (Dao-Castellana et al., 1998). To clarify, the Stroop test evaluates the participant's ability to suppress irrelevant information and enhance relevant information (Lezak, 1995). The Stroop is regarded as a measure of executive functions related to mental control and response flexibility associated with the frontal lobe (Lezak, 1995; Lezak et al, 2004). Verbal fluency, another measure of executive function, was evaluated by having subjects name as many animals as they could within a minute and list as many words as they could that started with the letters *m*, *p*, and *d* in a minute (Dao-Castellana et al., 1998) all of which are measures of speed and ease of verbal production (Lezak, 1995) which was found to be impaired in the alcoholic

participants. Dao-Castellana et al. (1998), concluded that these neuropsychological impairments including verbal fluency and ability to suppress irrelevant information and enhance relevant information may occur prior to other more obvious neurological impairments (such as severe behavioral abnormalities characterized by aggressiveness and breakdowns in family life) accounting for some of the behavioral changes.

There are definite strengths and weaknesses of the Dao-Castellana et al. (1998) study. For example, a follow up study might define the prognosis for the participants or the reversibility of the impairment. One strength of this study includes the author's use of the imaging studies in comparison with the neuropsychological tests, allowing the reader to understand physical changes in the brain as well as differences in an individual's performance when tested. However, the participants in the Dao-Castellan et al. (1998) study range from one week to one month abstinent which could have an impact on their functioning or level of impairment from the substance use. Follow up studies might also want to look at a longitudinal study comparing the impairments in early and later recovery. In addition, Dao-Castellana et al. (1998), make no mention of baseline neuropsychological functioning in the participants so it is unclear how much damage in the participants has occurred due to the alcohol use. This piece would also be interesting to include in future research. There is no mention of previous academic or medical records being evaluated for the possibility of assessing for premorbid functioning or history of trauma and/or injury. Finally, Dao-Castellana et al. (1998), does not mention if the control group was matched to the alcoholic group in the areas of education, socioeconomic status or ethnicity which could all affect how comparable the groups actually are. Overall, this

study does continue to support the relationship between alcohol use disorders and impairment in the frontal lobe.

Sullivan, Fama, Rosenbloom and Pfefferbaum (2002) examined differences in executive functioning between 43 alcoholic women (ages 28-63 years) recruited from inpatient and outpatient programs at the Veterans Affairs Palo Alto Health Care System, outpatient programs at Stanford Medical Center and from community treatment programs and 47 non-alcoholic women (ages 20-85 years) recruited from the community. It is unclear how many of the controls were over age 63 (the top of the age range for the alcoholic group) which could affect the comparability of the two groups. Tests administered to examine executive function included the WCST, Trails B, digit ordering task, and the picture arrangement subtest of the WAIS-R (Sullivan et al., 2002). An ANOVA was performed using six composite scores from each of the two groups including measures of executive function, short term memory, upper limb motor ability, declarative memory, visuospatial ability and balance (Sullivan et al., 2002). A statistically significant group effect was found between the alcoholic women and the control group, $F(1,50) = 5.54$, $p = .02$ (Sullivan et al., 2002). Follow up *t* tests revealed statistically significant performance deficits in the alcoholic group in five of the six areas (p </= .04), including all but upper limb motor ability composite (Sullivan et al., 2002).

One strength of this study was the author's assessment of premorbid intellectual functioning in the alcoholic group using the National Adult Reading Test (NART). The alcoholic group had a statistically significant lower score on the NART than the controls ($p < 0.01$; Sullivan et al., 2002). When the authors attempted to

control for this difference using an ANCOVA, the group differences remained significant for all domains except executive functioning (p = .41; Sullivan et al., 2002). Due to the difference in premorbid functioning in alcoholics and controls, it is unclear if we can attribute the deficits to the substance use of the alcoholic. In addition, although the authors attempted to control for the difference, complete confidence can never be attained when simply using an ANCOVA to control for the difference and to compensate for study design weaknesses (Loftin & Madison, 1991; Thompson, 1992). Sullivan et al. (2002), noted another weakness of this study was the fact that most of the alcoholic group reported being depressed and the control group did not. Previous research has suggested that one factor that may contribute to cognitive impairment in alcoholics is depression (Penick et al., 1994). Therefore, while the use of a control group is definitely a strength of the study by Sullivan et al. (2002), the depression reported by the alcoholic participants is an important difference between the study groups that may have affected the study's results. In addition, the alcoholic group had only 11 of the 43 participants that were free of any comorbid DSM IV diagnosis. Nine of the alcoholic women met criteria for one other DSM IV diagnosis whereas the rest met criteria for two of more other Axis I diagnoses in the DSM IV. On the contrary, the control group was screened with the Structured Clinical Interview for the DSM IV (SCID) and potential controls were excluded if they met DSM IV diagnostic criteria. Therefore, it seems a rather large weakness of the Sullivan et al. (2002), study is the fact that we can not be sure that the results are due to the alcohol use in the test participants rather than their comorbid DSM IV diagnosis.

However, results of other studies examining mood and alcoholism have contradicted the Penick et al. (1994) study mentioned above. For example, Uekermann, Daum, Schlebusch, Wiebel and Trenckmann (2002) compared depressed, non alcoholic (n = 28) nondepressed alcoholic (n = 30) and healthy controls (n = 28). The assessments used included Wechsler Adult Intelligence Scales-Revised (short term memory), Benton Visual Retention Test (visual memory), immediate and delayed recall of three word lists (verbal memory), Fragmented Picture Test (perceptual priming) Mood Rating Scale (mood), three verbal fluency tests, Hayling Test (response suppression) and Cognitive Estimates Test (reasoning; Uekermann et al., 2002). For statistical analysis, ANOVA's with subsequent t-tests using the Bonferroni correction were performed (Uekermann et al., 2002). ANOVA for present state mood revealed significantly (statistically) higher scores for the depressed group when compared to the alcoholic or control group (both $p < 0.0001$; Uekermann et al., 2002). Likewise, the patients with primary depression and the alcoholic group scored significantly higher than the healthy controls ($p < 0.013$; Uekermann et al., 2002). To assess the cumulative effect of depression and alcoholism, the cognitive profiles of depressed and nondepressed alcoholics were compared with those of the control group (Uekermann et al., 2002). It appears, that the authors developed a fourth group from the alcoholic group. They compare those within the alcoholic group that had a significantly higher ($p = 0.0001$) Beck's Depression Inventory score (which is not listed as an instrument used in their section on the instruments used) to the others in the alcoholic group (Uekermann et al., 2002). This results in the comparison of patients with alcoholism that are depressed to

patients with alcoholism that are not depressed. Unfortunately, this process is vaguely described in the article. In addition, the numbers in each group are not reported so it is unclear how many depressed alcoholics are being compared to nondepressed alcoholics. There were no statistically significant differences found with respect to age, general intelligence or history of alcoholism between the depressed alcoholic group and non depressed alcoholic group ($p > 0.34$; Uekermann et al., 2002). The comparisons of these two groups show no statistically significant differences on cognitive measures ($p > 0.10$). In conclusion, the results of the study showed that patients with primary depression and the alcoholic group were impaired with respect to executive functions and memory when compared to healthy controls ($p < 0.05$; Uekermann et al., 2002). In contradiction to Penick et al. (1994) the authors conclude that the lack of a significant difference between depressed and nondepressed alcoholics suggest that the deficits of alcoholics are not necessarily distorted by the depressive symptoms (Uekermann et al., 2002). Given the contradictory reports of the role depression plays with respect to executive functioning in alcoholic patients, further research would be useful in this area. Likewise, current researchers would need to use caution with regard to these variables when evaluating executive function in patients with a substance use issue and possible comorbid depression.

Moselhy, Georgiou and Kahn (2001) have also reviewed the results of many studies that researched the effects of alcohol on the frontal lobe. The studies they reviewed included detailed testing (such as Halstead Category Test, Wechsler Adult Intelligence Scale, WCST and Trail Making test) across both genders, various age groups and multiple countries which indicated that individuals who are diagnosed

with alcohol dependence exhibit deficits in cognitive flexibility, problem solving, verbal and non-verbal abstraction, visuo-motor coordination, learning, conditioning and memory (Moselhy et al., 2001). For example, within Moselhy, Georgiou & Kahn's (2001) review of the literature, multiple studies revealed physical changes in the brain such as wider sulci and fissures. Likewise, younger (mean age 37.5) and older participants (mean age 52.7) had gray matter volume deficits with the older group showing more severe deficits in the prefrontal area through use of CT and MRI scans (Moselhy et al., 2001). Moselhy et al., (2001) referenced eighteen studies with regard to detailed testing across cultures revealing deficits in cognitive flexibility, problem solving, verbal and nonverbal abstraction, visuo-motor coordination, learning, conditioning and memory. Though the specifics of each study are not described, one study of 35 alcoholics compared to 35 nonalcoholic controls revealed significant differences on the Trail Making test and the Halstead Battery (Moselhy et al., 2001). Within this study, the alcoholics were found to be indistinguishable from the non-alcoholics in terms of I Q (Moselhy et al., 2001). Moselhy et al's., literature review (2001) also summarized multiple studies which reported that when several third variables such as anxiety, depression, head injury and family history of alcoholism are controlled for, deficits in neuropsychological measures can still be found. For example, in one of the studies reviewed, 27 alcoholic participants that had a first degree relative with alcoholism were compared to 21 alcoholic participants without a first degree relative with alcoholism (Moselhy et al., 2001). No differences were found between the two groups suggesting that a family history of alcoholism does not appear to impact the effects of alcoholism on the frontal lobe (Moselhy et

al., 2001). Overall, Moselhy et al's extensive literature review provides support for the relationship between alcohol use disorders and frontal lobe impairment (Moselhy et al., 2001).

In an attempt to control for confounding variables in the performance of participants with an alcohol use disorder, Sher, Martin, Wood and Rutledge (1997) used a MANCOVA to control for family history, anxiety, depression, conduct disorder, and loss of consciousness. Factor analysis of 17 neuropsychological tests was performed on 489 undergraduates, half of whom had a history of alcoholism in their biological fathers (Sher et al., 1997). Of the 489 participants, 88 were diagnosed with alcohol abuse and 31 with alcohol dependence (Sher et al., 1997). Confounding variables were diagnosed using the Diagnostic Interview Schedule III to diagnose conduct disorder and the Brief Symptom Inventory for diagnosis of depression and anxiety (Sher et al., 1997). Family history was assessed through the Short Michigan Alcoholism Screening Test (SMAST) and loss of consciousness was assessed with simple questioning (Sher et al., 1997). This is an interesting study as it was performed on undergraduate students instead of a clinical sample like many other studies. One might presume that this non-clinical study might not have the same pattern of deficits as a clinical population. In addition, the individuals with the alcohol use disorders were drawn from the same population as the controls (first year undergraduates at the same institution). The findings from this study indicate that alcohol use disorders are associated with poorer visual spatial ability and reduced motor speed (Sher et al., 1997). These results essentially mirror results derived from studies of clinical samples

(Sher et al., 1997). When all confounds were controlled for through the use of a MANCOVA, statistically significant differences were still found (Sher et al., 1997).

Effects of Drug Use on Frontal Lobe and Executive Functioning

The effects on the frontal lobe are not only applicable to patients with an alcohol abuse or dependence diagnosis. Similar results have been found with drug abusing and dependent patients (Roselli et al., 2001). Forty-two adult (28 male, 14 female) cocaine abusers from a state rehabilitation facility were administered a neuropsychological test battery including the arithmetic and digit subtests from the WAIS-R, California Verbal Learning Test (CVLT), Trail making Test (TMT), Rey Osterrieth Complex Figure (ROCF), WCST, Benton Visual Retention Test (BVRT), Stroop Neurological Screening Test (SNST) and Hooper Visual Organization (Roselli et al., 2001). A control group of 11 females and 6 males with no history of alcohol or drug abuse and no psychiatric or neurological disorders was recruited from student advertisements (Roselli et al., 2001). The control group was given all of the same assessments as the drug dependent group. A MANOVA was performed and statistically significant differences between the drug abusing patient and the controls for the tests used were found on several of the executive function tests (Roselli et al., 2001). The most abnormal scores were observed in attention, memory and the executive functions which could impair participation and retention in treatment (Rosselli et al., 2001). Specifically, statistically significant differences were observed in the WAIS-R arithmetic ($F = 16.92$, $p = .001$) and digit subtest ($F = 13.52$, $p = .001$, CVLT ($F = 3.73$, $p = .05$), TMT Form B ($F = 10.28$, $p = .002$), ROCF ($F = 5.75$, $p = .02$), WCST errors ($F = 7.82$, $p = .007$), WCST number of categories ($F = 5.20$, $p =$

.026), BVRT (F = 16.49, p = .001) and SNST (F = 4.09, p = .04; Roselli et al., 2001). Rosenberg, Grigsby, Dreisbach, Busenbark & Grisby (2002) have found similar results to Roselli et al., using similar assessment tools with solvent abusers. Fifty five solvent abusers (43 males, 12 females) and 61 users (49 males, 12 females) of other drugs, especially cocaine and alcohol, were given a battery of neuropsychological tests including the Wechsler Memory Scale-Revised, Trail Making Test, Digit Cancellation Test, Stroop test, Boston Naming Test, WCST and Behavioral Dyscontrol Scale (Rosenberg et al., 2002). All participants performed poorly, scoring below the mean on most neuropsychological measures (Rosenberg et al., 2002). Solvent abusers performed even more poorly on executive functions than the others (Pillai's Trace = 0.239, p < .001; Rosenberg et al., 2002).

Several neuroimaging techniques have also been used to evaluate the effect of substance abuse and dependence disorders on the brain. For example, computerized tomography has been used to confirm cortical shrinkage and ventricular dilatation among alcoholic samples (Ron, Acker, Shaw & Lishman, 1982). In addition, magnetic resonance imaging (MRI) studies have shown abnormalities among inhalant abusers, cocaine abusers and alcoholics (London, Ernst, Grant, Sonson, & Weistein, 2000; Moselhy et al., 2001; Rosenberg et al., 2002). Functional MRI's (fMRI) have also been used to support the impact on the frontocerebellar regions in chronic alcoholics (Sullivan et al., 2003). Likewise, positron emission tomography (PET) studies have also found abnormalities (Moselhy et al., 2001). Decreased frontal lobe glucose utilization and reduced cerebral blood flow suggest frontal lobe dysfunction (Moselhy et al., 2001). Others also found decreased metabolism of glucose and

reduced cerebral blood flow in the frontal lobes of individuals with alcohol use disorders which may be part of the cause of impaired executive functions (Bates et al., 2002). PET and fluorodeoxyglucose studies have also been used to confirm that specific neuropsychological tests do activate the dorsolateral prefrontal cortex including the Tower of London test (Moselhy et al., 2001). Likewise, the previously mentioned executive functions of volition, planning, purposive action and effective performance can be traced to the frontal lobe (Adams et al., 1995; Dagher, Owen, Boecker & Brooks, 1999). As these functions are related to the frontal lobe and the frontal lobe is found to be affected by substance use, the relationship between impaired executive function and substance dependence is strengthened.

Summary

Overall, research has indicated that we can conclude that alcohol and drug use are associated with physical changes in the brains of users, including reduced size and blood flow in the frontal lobe (Bates et al., 2002; Ron et al., 1982; Sullivan, et al., 2003). Likewise, studies have indicated that individuals who use alcohol and drugs have lowered functioning on a variety of tasks than do their non-abusing counterparts including deficits in digit cancellation, digit symbol, trail making, reaction test, WCST (Roselli et al., 2001), reduced verbal fluency, impaired Stroop performance (Dao-Castellana et al., 1998) visuo-spatial ability and balance (Sullivan et al., 2002), cognitive flexibility, problem solving, verbal and non-verbal abstraction, visuo-motor coordination, learning (Moselhy et al., 2001) and overall executive functions, attention and memory (Roselli et al., 2001; Rosenberg et al., 2002; Sullivan et al., 2002). Additionally, there is some discrepancy in the research regarding the influence

of other factors, such as depression and anxiety on the cognitive functioning in alcoholic patients, and so we are, at present, uncertain if and how these other factors may affect functioning (Penick et al., 1994; Moselhy et al, 2001; Uekermann et al., 2002). However, we do know that the frontal lobe is the area of the brain responsible for executive functions as we have assessments that can measure cognitive functioning and we also have proven imaging techniques that validate that specific neuropsychological tests do indeed examine frontal lobe functioning (Adams et al., 1995; Dagher et al., 1999; Moselhy et al., 2001; Ratti et al., 2002; Roselli et al., 2001; Sullivan, et al., 2003). Therefore, within this study, I was able to evaluate the executive functioning of participants which may be impaired due to their substance dependence and may impact their ability to remain in treatment.

Neuropsychological Functioning and Dual Diagnosis

It is quite possible that any relationship between substance use and neuropsychological deficit can be attributed to third variable confounds related to co-occurring mental health disorders (Sher et al., 1997). Therefore, the relationship of substance use, cognitive/neuropsychological impairment and disorders such as depression, anxiety, personality disorders and thought disorders including schizophrenia will be reviewed.

Mood Disorders

Carpenter and Hittner (1997) evaluated the effects of substance use history and depressive symptoms on the cognitive functioning of 149 male and 72 female dually diagnosed patients with alcohol abuse or dependence, cocaine abuse or dependence and comorbid DSM-III-R affective disorder (Carpenter & Hittner, 1997).

All participants were administered the Beck Depression Inventory (BDI), the Shipley Institute of Living Scale (SILS) and the Screening Test for the Luria Nebraska Neuropsychological Battery (ST-LNNB; Carpenter & Hittner, 1997). Results of a logistic regression analyses using the traditional Shipley abstract indicated a significant effect for previous months alcohol use on probability of impaired classification based on the SILS reasoning performance (Z = 2.01; Carpenter & Hittner, 1997). A marginally significant (statistically) effect for life time alcohol use also emerged indicating that individuals with 5-10 years of regular drinking experience were 2.3 times more likely to be classified as impaired that those with less than 5 years regular consumption (Z = 1.93, p = .05; Carpenter & Hittner, 1997). No statistically significant parameter estimates were demonstrated for cocaine use, depressive symptoms, intravenous (IV) drug use or life time substance use (Carpenter & Hittner, 1997). Results of the logistic regression analyses using the modified Shipley (possible borderline cases omitted) indicated statistically significant effects for life time alcohol use (Z = 2.64) and total number of months of life time substance use other than alcohol and cocaine (Z = 2.23; Carpenter & Hittner, 1997). No statistically significant effects were found for 10 plus years of alcohol use, previous month's alcohol use, cocaine use or depressive symptoms (Carpenter & Hittner, 1997). Logistic regression analyses for the ST-LNNB demonstrated statistically significant effects for education (Z = -2.14) and life time cocaine use (Z = 2.41; Carpenter & Hittner, 1997). The effect for life time cocaine use remained even after controlling for age, education, depressive symptoms, other substance use, IV drug history and previous months use (Carpenter & Hittner, 1997). Overall, the authors

found “no statistically significant effects for age, previous month’s cocaine use, depressive symptoms or any of the interaction terms” (Carpenter & Hittner, 1997, p. 752). Carpenter and Hittner (1997) do acknowledge the weaknesses of their study such as the fact that they are evaluating an inpatient population from a private psychiatric facility which may have resulted in higher functioning sample relative to other studies with other facilities. Likewise, Carpenter and Hittner (1997) did not assess for previous neurocognitive injuries, premorbid conditions (such as learning disabilities) or concurrent health conditions which could affect cognitive functioning such as HIV status. Furthermore, they acknowledged that they did not evaluate Axis II diagnoses which may have provided additional information (Carpenter & Hittner, 1997).

Likewise, Uekermann, Daum, Schlebusch, Wiebel and Trenckmann (2003) studied 30 patients suffering from alcoholism, 28 patients with depression but without alcoholism and 28 healthy controls. After performing an ANOVA, no statistically significant difference between depressed and alcoholic groups was found for short term memory ($p > 0.27$), visual memory ($p > 0.13$), verbal memory ($p > 0.15$) and verbal fluency ($p > 0.39$; Uekermann et al., 2003). However, the results did show that patients with primary depression and alcoholism are impaired with respect to executive functions and memory (Uekermann et al., 2003). The lack of difference found between depressed and nondepressed alcoholics suggests that the results are not distorted by the depressive symptoms (Uekermann et al., 2003). The use of the control group in this study provides interesting information in that we notice that both alcoholism and depression do indeed have an impact on executive functions vs.

healthy controls. However, Uekermann et al., (2003) acknowledge that 27% to 69% of all alcoholics have elevated depression scores and 15% to 28% suffer from major depression. Therefore, the mood disorders are still important variables to consider.

Others have also reported that mood disorders do appear to have a negative impact on neuropsychological functioning in clients with a substance use disorder (Bates et al., 2002; Blume, Davis & Schmaling, 1999; Fals-Stewart & Bates, 2003). Blume, Davis and Schmaling (1999) studied a sample of 22 psychiatric inpatients all with a substance abuse or dependence diagnosis and 14 diagnosed with depression, 3 with schizoaffective disorder, 1 with paranoid schizophrenia and 4 with bipolar disorder (Blume et al., 1999). These authors did find that the full scale IQ scores of participants were at the low end of normal and that the general memory index of the Wechsler Memory Scale-Revised was one standard deviation below the mean (Blume et al., 1999). With these results, the authors stated that dually diagnosed patients would benefit from a thorough neuropsychological assessment (Blume et al., 1999). However, there was no distinction made between dually diagnosed and non-dually diagnosed patients. Therefore, it is unclear if the results of the Blume et al. (1999) study are attributable to the substance use diagnosis or the co-occurring mental health disorder. Likewise, the type of the co-occurring disorder is not distinguished. Therefore, we can not evaluate any possible differences between depression, schizoaffective, schizophrenia and/or bipolar disorder from the Blume et al. (1999) study.

Anxiety

Anxiety, often times along with depression, has been shown to be related to neuropsychological test performance (Bates et al., 2002; Fals-Stewart & Bates, 2003; Glenn, Errico, Parsons, King & Nixon, 1993). For example, Glenn, Errico, Parsons, King & Nixon (1993) reported that neuropsychological test performance was moderately related to anxiety and depression in alcoholic samples but that these affective states did not fully explain their performance deficits. In a sample of 83 male and 48 female alcoholics and 47 male and 36 female non alcoholic controls, a factor analysis revealed three factors including Antisocial Behaviors, Affective symptoms and Childhood Behavioral Disorders (Glenn et al., 1993). In regards to these factors, alcoholics and controls were clearly differentiated with no major difference between genders (multivariate main effect for group, alcoholic > controls, $F(3, 184) = 44.01$, $p < 0.0001$; Glenn et al., 1993). A multivariate analysis was then conducted using five neuropsychological factors as dependent variables and *group*, *group times gender*, and *gender* as independent variables. The main effect for group was again significant with alcoholics scoring lower than controls, $F(5,193) = 8.40$, $p < 0.0001$, with no significant group times gender interaction (Glenn et al., 1993). A significant main effect for gender was also found with females receiving higher scores than males on three factors, $F(5, 193) = 6.42$, $p < 0.001$ (Glenn et al., 1993). In the Glenn et al (1993) study, the best predictor of neuropsychological performance was Childhood Behavioral Disorders. . Secondly, the Affective symptoms were significant predictors of set shifting flexibility and verbal memory (Glenn et al., 1993). The Antisocial Behaviors were not a significant predictor of neuropsychological function in alcoholics or controls (Glenn et al., 1993). The sample

size and use of a control group are definite strengths of the Glenn et al. (1993) study. Likewise, Glenn et al. (1993) perform multiple statistical comparisons to allow for evaluation of gender, alcoholic vs. nonalcoholic, multiple personality variables, mood variables and others. This resulted in a comprehensive evaluation of variables related to neuropsychological function, substance abuse and dual diagnosis.

Personality Disorders

Personality disorders in general and antisocial personality disorder specifically, are acknowledged by many to have an impact on neuropsychological functioning as well as treatment attrition (Fals-Stewart & Lucente, 1994; Kokkevi, Stefanis, Anastasopoulou & Kostogianni, 1998; Roselli et al., 2001). For example, Roselli, Ardila, Lubomski, Murray and King (2001) studied a sample of 42 crack and/or cocaine dependent men and women. A control group of 17 subjects with no history of substance abuse or any psychiatric or neurological disorder was also used in the study (Roselli et al., 2001) The Personality Assessment Inventory (PAI) was given to all to establish an Axis II diagnosis and a neuropsychological test battery was given to all to establish any neuropsychological deficits (Roselli et al., 2001). A MANOVA was used to compare the PAI and neuropsychological test scores of the drug dependent subjects to the controls (Roselli et al., 2001). Statistically significant differences were found on the PAI between the drug dependent and controls ($p >$ 0.0001) indicating that there is a positive relationship between personality and drug dependence (Roselli et al., 2001). This is not an indication that personality is predicting NP function, but rather an indication of the relationship between personality and drug dependence. Specifically, 88% of the drug dependent subjects

obtained an abnormal PAI score (Roselli et al., 2001). Multiple regression analyses were then conducted, with all participants using the neuropsychological test scores as the dependent variable and PAI scores as the independent variable (Roselli et al., 2001). The PAI score associated with drug use (DRG) did predict the score on the WAIS-R arithmetic subtest ($p = 0.01$), California Verbal Learning Test ($p = 0.007$), Stroop color word test ($p = 0.004$) and Benton Visual Retention Test ($p = 0.017$; Roselli et al., 2001). In addition, the PAI score associated with antisocial personality (ANT) predicted the Stroop color word test ($p = 0.03$) but no others (Roselli et al., 2001). The authors therefore concluded that personality does not predict neuropsychological performance, but that there is a relationship between personality and drug dependence (Roselli et al., 2001).

Fals-Stewart and Lucente (1994) administered a battery of neuropsychological tests including the Category Test, the Tactual Performance test, Trails B, Block Design and Digit Symbol tests from the Wechsler Adult Intelligence Scale (WAIS) to 246 residents of a long term residential substance abuse treatment facility (Fals-Stewart & Lucente, 1994). In addition, personality was evaluated with the Millon Clinical Multiaxial Inventory (MCMI-II; Fals-Stewart & Lucente, 1994). Using a cutoff score of $T < 40$ on the neuropsychological test battery to indicate dysfunction, 55 (22.4%) of the residents were classified as cognitively impaired. Fals-Stewart and Lucente (1994) found that patients with cognitive deficits generally scored higher on the MCMI-II than those without impairment ($p < 0.05$). Likewise, Fals-Stewart and Lucente (1994) found that the cognitively intact group stayed in treatment longer ($M = 206.2$ days) versus cognitively impaired ($M = 132.4$ days). The

authors did conduct further statistical analysis to evaluate the effect of the MCMI-II scores on the length of stay as well and concluded that personality and neurocognitive impairment do affect length of stay in substance abusing patients (Fals-Stewart & Lucente, 1994). However, Fals-Stewart and Lucente (1994) did not use a control group that had only cognitive impairment and only personality disorders co-occurring with the substance use disorder. Therefore, we can see that the cognitive impairment is related to the personality disorder, but we can not be sure if the cognitive impairment or the personality disorder affected length of treatment.

Schizophrenia

There are relatively few studies published that evaluate the cognitive status of dually diagnosed people, and even fewer that compare the neurocognitive characteristics of dually diagnosed to non-substance-abusing patients with schizophrenia (Herman, 2004). Part of the difficulty is due to the impaired reality orientation and therefore invalid or unreliable self report data with regards to substance use and current functioning (National Institute on Drug Abuse, 1997). However difficult, Herman (2004) studied 46 dually diagnosed (schizophrenia and substance abuse) and 43 non-substance abusing patients with schizophrenia. The subjects were given subtests of the WAIS III to assess intellectual and memory function, and the Stroop, Controlled Oral Word Association (COWAT) and Trails A & B to assess executive function (Herman, 2004). In addition, quality of life was evaluated using the World Health Organization Quality of Life measure (Herman, 2004). A stepwise multiple regression was used and showed no statistically significant difference between the dually diagnosed group and the non substance

abusing schizophrenic group on the tests of intelligence and memory (Herman, 2004). However, the dually diagnosed group did perform significantly better on tests of executive function including the COWAT ($p < 0.01$), Trails A ($p < 0.00025$), Trails B ($p < 0.01$) and Stroop ($p < 0.05$; Herman, 2004). Therefore, Herman's study (2004) failed to show that dually diagnosed patients will have greater neurocognitive deficit than non-abusing patients with schizophrenia.

Herman's study (2004) could have been even more interesting had he used a third group of only substance abusing patients to give the reader another comparison. Similar results to Herman's (2004) have been found by other researchers as well (see Cleghorn, Kaplan, Szechtman , Szechtman , Brown**,** Franco, 1991; Nixon, Hallford, Tivis, 1996) However, opposite results have also been found. For example, Sevy, Kay, Opler and van Praag (1990) divided 51 schizophrenic inpatients into two groups including those with a cocaine use history and those without. Sevy et al. (1990) found that the dually diagnosed patients were found to be more depressed, less socialized, and performed worse on conceptual encoding and verbal memory (Sevy, Kay, Opler & van Praag, 1990).

In general, schizophrenic patients are found to have deficits in cognitive functions such as information processing and abstract planning (U.S. Department of Health and Human Services, 1999). Likewise, they are reported to have executive function deficits in areas including planning and regulating, goal directed behavior, cognitive flexibility and attention (U.S. Department of Health and Human Services, 1999). More specifically, some believe that schizophrenia is a disorder of the prefrontal lobe which would therefore have an impact upon executive function

(Rains, Sauer & Kant, retrieved September 18, 2005; US Department of Health and Human Services, 1999). Given this information, we will need to be cautious in evaluating any participant who has been diagnosed with schizophrenia due to the intervening impact the schizophrenia could have on the neuropsychological test results.

Summary

Overall, research has suggested the use of caution when evaluating neuropsychological functioning in substance dependent patients. Evidence suggests that disorders such as depression, anxiety, personality disorders and schizophrenia can co-occur with a substance dependence diagnosis and are also possibly related to neuropsychological functioning (Blume et al., 1999; Kokkevi et al., 1998; Roselli et al., 2001; Uekermann et al., 1997; U.S. Department of Health and Human Services, 1999). Finally, these co-occurring disorders may also have an impact on treatment attrition and retention (Fals-Stewart & Lucente, 1994).

Neuropsychological Functioning and Attrition

Of all areas of literature that were researched for this study, the reports on the relationship of neuropsychological functioning and attrition was the sparsest. As the field of neuropsychology is still somewhat new and growing, it seems timely for increased research related to neuropsychology and the impact on attrition rates. More specifically, there has been advancing technology in the field of neuropsychology and a lack of information available on the impact neuropsychological functioning has on attrition.

Search terms used included “neuropsychology, neuropsychological functioning and dysfunction, neurocognitive function, executive function, executive dysfunction, cognitive functioning, cognitive deficits, frontal lobe, frontal lobe impairment, brain, brain impairment, dysexecutive syndrome, dropout rates, attrition, mortality, treatment retention, treatment completion, treatment attendance, treatment outcome, outpatient, substance abuse services, recovery, rehabilitation” and multiple combinations of all of these. I utilized Marquette University’s search engines, assistance from Marquette Library staff, a paid literature search by the Medical College of Wisconsin and all internet offerings such as Google. In addition, I obtained suggestions and direction from practicing neuropsychologists and researchers including Dr. Gina Rehkemper of Waukesha Memorial Hospital’s Neuropsychology Center, Dr. Terry Young of New Life Resources, Dr. Todd Campbell of Marquette University, Dr. Swartzwelder of Duke University Medical Center, Dr. Steve Holliday-Chief of Psychological Services at South Texas Veterans Hospital, Dr. Joe Bleiberg of the National Rehabilitation Hospital in Washington, DC, and Dr. Lisa Drozdick, a neuropsychology researcher at Psych Corp. Through all of these efforts, the findings that actually related to neuropsychological functioning and attrition in outpatient treatment were limited. Over 150 publications were evaluated and only one (thus far) has specifically reviewed neuropsychological functioning and attrition rates in substance use treatment. Fals-Stewart and Lucente (1994) studied neuropsychological impairment along with personality variables and attrition as described in the previous section on neuropsychological function and dual diagnosis (Fals-Stewart & Lucente, 1994). However, even this one publication by

Fals-Stewart and Lucente was not purely addressing neuropsychological impairment with attrition. Therefore, I will present a brief overview of my current findings and will continue to research these issues as I pursue this project.

Researchers studying executive functions and attrition have suggested that executive function deficits *might* contribute to rates of attrition (Ihara, Berrios & London, 2000). Seventeen adults with chronic alcoholism, without amnesia, were given a battery of neuropsychological tests to assess executive function (Ihara et al., 2000). The results of the Ihara et al. (2000) study showed that the participants displayed mild but significant dysexecutive syndrome (DES; meaning a syndrome of impaired executive functioning) even in the presence of unimpaired intelligence and memory. The results of the Ihara et al (2000) study suggest that alcoholic patients have difficulty when demonstrating abstract analysis, critical judgment and flexibility of thought processes. Therefore, the alcoholic patient's ability to respond and participate effectively in conventional substance abuse programs may be limited (Ihara et al., 2000). However, Ihara et al. (2000) did not formally evaluate attrition, it is only suggested that the impairment would likely affect engagement and success in treatment. The suggestion of possible impact on attrition versus the actual evaluation of impact is common in many studies but not empirically investigated (Fals-Stewart & Bates, 2003; Moselhy et al., 2001; Sullivan et al., 2002). Bates, Bowden and Barry (2002) stated that "neurocognitive impairment interferes with the process of alcoholism treatment and is hypothesized to affect outcome as well" (Bates et al., 2002, p. 193). The authors go on to discuss that clients with executive function deficits may have trouble getting to appointments, fail to complete assignments,

behave impulsively and have trouble regulating affect (Bates et al., 2002). Likewise, others have shown that patients with neurocognitive deficits (including spatial ability, mental flexibility, concept formation and nonverbal problem solving) in conjunction with personality disorders stayed in a substance abuse program a shorter amount of time (Fals-Stewart & Lucente, 1994).

Fals-Stewart and Lucente (1994) administered a battery of neuropsychological tests including the Category Test, the Tactual Performance test, Trails B, Block Design and Digit Symbol tests from the Wechsler Adult Intelligence Scale (WAIS) to 246 patients admitted to a long term residential substance abuse treatment facility. In addition, personality was evaluated with the Millon Clinical Multiaxial Inventory (MCMI-II). The neuropsychological tests and the MCMI-II were not administered until 30 days after admission to ensure that residual effects from the substance abuse would not adversely affect performance (Fals-Stewart & Lucente, 1994). Patients who were admitted and stayed less than a month were not included in the study (Fals-Stewart & Lucente, 1994). There is no way of knowing if neuropsychological impairment affected the early drop out of those who did not stay past 30 days. Fals-Stewart and Lucente (1994) found that patients with cognitive deficits have different personality characteristics, as measured by the MCMI-II, and both of these factors influenced the length of stay. Specifically, the cognitively intact group stayed in residential treatment longer (M = 206.2 days and SD = 30.5) than impaired residents (M = 132.4 days and SD = 39.6) which was found to be a significant difference in length of stay, $F(1,242) = 6.83$, $p < .01$ (Fals-Stewart & Lucente, 1994). However, the cognitively impaired group was significantly older (M

= 31 years and *SD* = 6.6) than the cognitively intact group (*M* = 26.3 years and *SD* = 5.1), *t*(246) = 2.39, *p* < .01 (Fals-Stewart & Lucente, 1994). The patients in the impaired group also had longer substance abuse histories (*M* = 134.8 months and *SD* = 67.3) than the intact patients (*M* = 99.6 months and *SD* = 55.6), t(246) = 2.41, *p* < .01. Finally, using Wilk's criterion, the impaired and intact group had profiles on the MCMI-II that were statistically significantly different, *F*(19,226) = 2.10, *p* < .01 (Fals-Stewart & Lucente, 1994). The impaired residents generally scored higher on the MCMI-II subscales than those without impairment, *F*(1,242) = 5.01, p < .05 (Fals-Stewart & Lucente, 1994). Fals-Stewart and Lucente (1994) controlled for age, length of drug use and MCMI-II scale scores through statistical means to conclude that the cognitive deficits indeed affected the length of treatment. However, one cannot be completely confident that variables are actually 'controlled for' without affecting other variables (Loftin & Madison, 1991; Thompson, 1992). Therefore, it is not clear if the neurocognitive deficits, the personality disorders, length of substance use or the combination are responsible for the impact on attrition in this study. In addition, Fals-Stewart and Lucente (1994) also acknowledge weaknesses in their study as the neuropsychological battery they chose was used only for screening and not to identify client strengths and weaknesses.

Multiple studies were reviewed evaluating neuropsychological functioning and substance abuse treatment and outcomes by Knight and Longmore (1994). The authors reviewed findings related to length of abstinence, compliance and relapse rates (Knight & Longmore, 1994). Similarly, cognitive function was compared to amount and length of alcohol usage. Many important factors to consider when

treating a patient with neuropsychological impairment are considered including methods of education, staff response and specific testing techniques (Knight and Longmore, 1994). For example, with specific neuropsychological tests, a patient's strengths and weaknesses can be assessed. Clinicians can be trained on identification of deficits and more effective treatment options including repetition, concrete examples and assisting the client with planning and organizing (Knight & Longmore, 1994). However, the specific relationship between level of neuropsychological impairment and attrition are not formally defined in this publication.

Other authors evaluated executive dysfunction and compared this to functional outcomes including resumption of drinking and occupational status (Moriyama et al., 2002). Moriyama et al. (2002) administered twelve neuropsychological tests to 22 chronic, male alcoholics. These authors found that six of the subtests of the Behavioral Assessment of Dysexecutive Syndrome (BADS) did predict an alcohol-nonspecific outcome (occupation) but not an alcohol-specific outcome (drinking; Moriyama et al., 2002). Other neuropsychological tests used did not predict either of the two outcomes (Moriyama et al., 2002). One reason given for the different performance of the neuropsychological tests was that the discriminative power of the BADS versus the other tests was due to its multicomponent impairment indices (Moriyama et al., 2002). However, once again, the relationship between neuropsychological impairment and length of treatment involvement was not evaluated.

In a more recent study, Zinn, Stein and Swartzwelder (2004) examined neuropsychological functioning in older, male alcoholics. The participants were 27

male, alcohol dependent veterans receiving treatment at the Durham Veterans Affairs Medical Center. All participants met diagnostic criteria for alcohol dependence according to the DSM-IV and were abstinent six weeks or less (Zinn et al., 2004). Control participants (n = 18) were recruited from the Primary Care Clinic of the Durham Veterans Affairs Medical Center (Zinn et al., 2004). The alcohol dependent and control group were not significantly different in age or education. However, the alcohol dependent group had a higher proportion of African Americans (Zinn et al., 2004). All participants were administered a fixed-order neuropsychological battery of seven tests including the COWAT (verbal fluency), Ruff Figural Fluency Test (non-verbal fluency), ASI, Rey Osterrieth Complex Figure Test (nonverbal memory), Hopkins Verbal Learning Test (verbal memory), two subtests of the WAIS-II for abstract reasoning (similarities and matrix reasoning) and Trails Making Test (Trails A for psychomotor speed and Trails B for cognitive flexibility; Zinn et al., 2004). Zinn et al. (2004) found several deficits in executive functioning in treatment seeking, recently abstinent patients. These deficits included reasoning (similarities, p = .05 and matrix reasoning, p = .04), nonverbal fluency (p = .002), performance of timed complex tasks (Trails A, p = .01 and Trails B, p = .003) and discriminative memory (Rey Osterrieth delayed recall, p = .03; Zinn et al., 2004).

Strengths of this study by Zinn et al. (2004) include the use of a control group of similar age to control for the relationship between age and executive function decline and the fact that the authors tested premorbid functioning with an estimated performance IQ. However, the difference in racial characteristics of the alcohol dependent versus control group is mentioned but not addressed which may have

provided interesting information or differences in results. In addition, Zinn et al. (2004) chose to include individuals with a history of non-severe head injuries (33% of controls and 52% of substance abuse patients) to reflect this reality in the greater population of individuals with a substance abuse issue. However, it is unclear what Zinn et al. (2004) considered 'non-severe' or how the results of the study can be linked to substance use versus the head injury. Finally, the authors acknowledged that neuropsychological impairment *may* affect attrition and treatment success but they do not formally evaluate retention and/or attrition (Zinn et al., 2004). I also had the pleasure of personal communication with Dr. Swartzwelder (of the Zinn, Stein & Swartzwelder, 2004) who acknowledged that he did not specifically know of any research that reviewed neuropsychological functioning and attrition rates (Personal communication, 10-5-2004). However, Dr. Swartzwelder did suggest other publications that were reviewed within this paper. Likewise, studies were found which evaluated length and/or amount of alcohol consumed and severity of neuropsychological deficits, but again the relationship between severity of the deficit and attrition was not evaluated, only speculated (Beatty et al., 2000; Munro, Saxton & Butters, 2000).

Part of the difficulty in evaluating or addressing neuropsychological function and attrition rates in outpatient might be the treatment provider's lack of insight or knowledge. Fals-Stewart (1997) evaluated counselor's ability to detect neuropsychological impairment among patients. The author found the counselor's ability to detect impairment was poor (Fals Stewart, 1997). Fals-Stewart (1997) offers several suggestions for improvement including use of specific neuropsychological

evaluation tools upon entry into treatment, emphasizing the importance of incorporating the patient's neuropsychological status into treatment planning, development of referral guidelines for neuropsychological testing, supervision by a trained neuropsychologist and development of more valid and reliable methods of identifying patients at risk for deficits. Likewise, the author mentioned that this is an important variable with treatment planning and success, but again does not formally evaluate the link between neuropsychological deficit and attrition (Fals-Stewart, 1997).

Others also agree that the neurocognitive deficits often go unrecognized in people with a substance use disorder when they seek treatment (Bates et al., 2002). What the clinician identifies as lack of motivation, apathy or noncompliance may all be a result of a neurocognitive deficit. Similarly, few treatment programs for substance use disorders consider the role of neurocognitive impairment (Bates et al., 2002) again supporting the stigma often associated with a person with a substance use disorder. The counselor's lack of success or lack of attempt at identifying neuropsychological deficits, as well as the resulting misperceptions by the clinician again provides reasoning for this current study.

Overall, there is support for the negative impact substance abuse has on neuropsychological functioning (Bates et al., 2002; Fals-Stewart & Lucente, 1997; Zinn et al., 2004). Likewise, there is support for the negative impact neuropsychological deficits have on treatment outcome variables such as relapse rates or occupational stability (Knight & Longmore, 1994; Moriyama et al., 2002). However, the issue of the specific relationship between level of neuropsychological

impairment and attrition rates is still left mostly unanswered. This missing link is what I will address with this study.

Attrition and Retention

Duration of treatment is one of the best predictors of outcome for substance abuse treatment populations (Corrigan et al., 2005). In the United States during the 1970s, the Drug Abuse Reporting Program (DARP) began collecting substance abuse data, followed by Treatment Outcome Prospective Studies (TOPS) that began collecting data from 1979-1981. To follow, from 1991-1993 the Drug Abuse Treatment Outcome Studies (DATOS) collected data in 11 cities, from 96 programs, over 10,000 patients from all treatment types. Together, DARP, TOPS and DATOS collected information from over 65,000 admissions and 272 programs. One of the major themes that resulted from these studies is that longer stays are consistently associated with better outcomes (Franey & Ashton, 2002; DATOS, 2001). For example, one year after treatment, 80% to 90% of long-stay (at least three months) clients who had been using heroin or cocaine weekly prior to treatment were no longer doing so (Franey & Ashton, 2002; DATOS, 2001). However, for clients who left earlier, the figure was 50% to 60%. Franey and Ashton (2002) reported that there is “nothing magical” about the retention periods they chose-longer stays were associated with better outcomes. However, very short stays can also be followed by great improvements (Franey & Ashton, 2002).

Though the relationship between treatment retention and outcome seems clear, there were differences between individual programs that DATOS (2001) studied. Although median lengths of stay were three months in long-term residential (LTR)

and outpatient drug-free treatments (ODF) and one year for clients in outpatient methadone treatment (OMT), there was variation between individual treatment providers. Though specific neuropsychological deficits are not reported, programs treating individuals with more psychological dysfunction usually had shorter retention rates (Simpson et al., 1997). Broome, Flynn and Simpson (1999) examined the psychiatric comorbidity as a predictor of treatment retention using the DATOS data. The psychiatric indicators included lifetime DSM-III-R diagnoses of depression/anxiety and antisocial personality (Broome et al., 1999). Dimensional measures of current symptoms of depression and hostility were also collected. The data collection included structured interviews with clients, a survey of treatment program administrators and program discharge records (Broome et al., 1999). Broome et al. (1999) found that the dimensional measure of current psychiatric symptoms emerged as better predictors of retention than the DSM-III-R diagnoses. In addition, on site mental health services in LTR were associated with better retention for clients with symptoms of hostility (Broome et al., 1999).

So what did DATOS identify as influencing retention? Interestingly enough, whether the client was black or white, male or female, age and drug use profile all made little difference according to Franey and Ashton (2002). What they did find was that more qualitative dimensions related to commitment and motivation were important for retention (Franey & Ashton, 2002). However, motivation is not a given in this sense. It arises from the therapeutic relationship between counselor and client (Franey & Ashton, 2002). In contrast, using logistic regression analysis, Simpson, Joe and Rowan-Szal (1997) found that 35 different patient attributes were associated with

increases in the likelihood of having favorable improved retention. Simpson, Joe and Rowan-Szal (1997) suggested that more comprehensive models of patient attributes, therapeutic processes and environmental influences are needed. Possibly, the patient attributes that need more comprehensive evaluation are neuropsychological strengths and weaknesses. With the DATOS studies, we do not know of the specific training and techniques of individual therapists. Could it be that the clinicians who were more successful at promoting the therapeutic relationship were also more sensitive to the strengths and weaknesses of the client, possibly related to neuropsychological deficit?

Though DATOS, TOPS and DARP did not discuss the neuropsychological functioning of individuals and treatment retention, the studies did emphasize that the "key thing is *remaining* in treatment" (Franey & Ashton, 2002, p. 6). In this study, we evaluate the impact neuropsychological functioning has on attrition to hopefully discover additional variables that may assist individuals in staying in treatment.

Relapse

Substance abuse treatment is plagued by high relapse rates following substance abuse treatment (Walton, 2001). In addition, multiple researchers have evaluated the relationship between neuropsychological functioning and relapse in substance use treatment (Miller, 1991; Society for Neuroscience, 2005; Tapert, Ozyurt, Meyers & Brown, 2004). In the following section, multiple variables related to relapse, including neuropsychological functioning will be reviewed.

Method and Intensity of Treatment, Drug Type and Relapse

In the literature reviewed, relapse rates tended to be reported in conjunction with treatment type or type of drug use. For example, DATOS reported that in the year after completing at least three months of treatment (residential or non-residential), 80-90% of weekly heroin or cocaine addicts were abstinent (Franey & Ashton, 2002). For the clients who left treatment earlier than three months, relapse rates increase with the number abstinent dropping to 50-60% (Franey & Ashton, 2002). With primary cocaine users, only 15% relapsed to weekly cocaine use after at least three months of residential treatment (Franey & Ashton, 2002). Similar findings have been found with other substances. For example, the greatest levels of abstinence from opioids at one year post treatment were associated with 28 day inpatient versus shorter treatments (United Nations, 2002). Likewise, patients who stay for at least one year in outpatient methadone programs have better abstinence rates (specific numbers not reported) than those that leave earlier (United Nations, 2002). In general, it appears that more treatment and more intense levels of treatment lead to greater lengths of abstinence (Franey & Ashton, 2002; United Nations, 2002).

Gender, Support, Pharmacotherapy and Relapse

Just as relapse is related to length and intensity of treatment, relapse is also related to other variables including gender, support and pharmacotherapy (Stocker, retrieved September 20, 2005; United Nations, 2002; World Health Organization, 2004). For example, women have been reported to relapse less frequently than men (Stocker, retrieved September 20, 2005). In a study of 182 women and 148 men in 26 public outpatient drug abuse treatment programs, only 22% of the women versus 32 % of the men relapsed in six months (Stocker, retrieved September 20, 2005). Some

of the possible reasons explored included the intensity of the women's drug use prior to treatment and social support differences (Stocker, retrieved September 20, 2005). However, when evaluated, the variable that was found to explain the difference in relapse rates between women and men was women's willingness to engage in treatment, particularly group treatment (Stocker, retrieved September 20, 2005). Though not found to be a determinant of relapse in Stocker's report (retrieved September 20, 2005), others have identified social support as an important variable related to relapse in substance use treatment (United Nations, 2002). According to a report by the United Nations on a review of evidenced based treatment, social support such as Alcoholics Anonymous meetings and the level of stressful life events such as loss of a job might be more powerful in determining relapse than the type of treatment (United Nations, 2002). Likewise, the United Nations study identifies treatment retention as an important variable in relapse prevention (United Nations, 2002). Specifically, the United Nations study reported that the longer patients are retained in treatment, the more likely lifestyle improvements such as abstinence will be achieved (United Nations, 2002). The United Nations study also reported on the relationship of pharmacotherapy, retention and relapse prevention (United Nations, 2002). Specifically, the use of methadone, buprenorphine, levoalphacetylmethadol, naltrexone and acamprosate were all found to improve retention and rates of relapse (United Nations, 2002; World Health Organization, 2004). However, compliance with patients using the medications as prescribed is paramount to the success of the medication (United Nations, 2002).

Neuropsychological Functioning and Relapse

Another area researchers review with relation to relapse in outpatient substance abuse treatment is the impact of neuropsychological functioning on rates of relapse (Miller, 1991; Society for Neuroscience, 2005; Tapert et al., 2004; World Health Organization, 2004). For example, specific areas of the forebrain have been shown through imaging techniques to be activated by stimuli that induce cravings in substance dependent people which could induce a relapse (World Health organization, 2004). Specifically, the nucleus accumbens area of the forebrain has been shown to be related to intense cravings in substance dependent individuals (Leshner, retrieved September 20, 2005; Society for Neuroscience, 2005). Many neurotransmitters, which are chemical messengers in the brain, have also been studied with relevance to cravings and relapse (Leshner, retrieved September 20, 2005). Specifically, dopamine, serotonin, norepinephrine, glutamate, endogenous opioids and GABA are all neurotransmitters that can affect cravings and relapse (Leshner, retrieved September 20, 2005; World Health Organization, 2004). Chronic substance use can affect the way the neurotransmitters function and the individual's response to the neurotransmitters which then can impact cravings, mood, sleep, sensitivity to pain, aggression and memory (Leshner, retrieved September 20, 2005) which all can impact decision making in recovery.

In a review of the literature, Miller (1991) evaluates neuropsychological and cognitive variables with regard to predicting relapse in substance abusers (Miller, 1991). Miller finds that neuropsychological studies of substance abuse treatment outcomes have generally found intact functioning on most measures for successful recoverers whereas relapsers did more poorly on tests of language, abstract reasoning,

planning and cognitive flexibility which are related to the left hemisphere and frontal lobe (Miller, 1991). In addition, personality characteristics of successful recoverers (with or without treatment) include future goal orientation, frustration tolerance and self efficacy, whereas the relapsers were characterized by impulsivity, antisocial personality and affective traits (Miller, 1991). Through Miller's (1991) review of multiple studies, he concluded that neuropsychodynamic trait of ego autonomy, which includes a reflective, nonimpulsive, goal directed cognitive style, is what is described in successful recoverers. Miller (1991) reported that many researchers focus on programs and treatment techniques which neglect the individual variables which he reports are related to predicting relapse. Miller (1991) admitted his review was not a meta-analysis, but instead a review of what he believed to be substantive issues related to substance abuse outcome. As his review is a selected group of studies, the results could be biased based on the studies he chose to review and include. Miller (1991) concluded with a discussion of individualized treatment options which may be helpful for this study if we find neuropsychological functioning to be related to attrition and relapse.

In addition to Miller's (1991) identified neuropsychodynamic trait of ego autonomy, another individual characteristic reported to be related to neurocognitive ability and relapse in substance abuse treatment is coping style (Tapert et al., 2004). In a study of 43 alcohol dependent male adults, individuals with low levels of coping in role plays of drinking situations consumed more alcohol in six months following treatment than did individuals with high levels of coping (Tapert et al., 2004). The participants were given a neuropsychological battery (including the Halstead-Reitan

Trails A and B, WAIS-R vocabulary and digit symbol subtest, and the Visual Search Test) and the Ways of Coping Questionnaire (WOC; Tapert et al., 2004). The results of this study support a model in which neurocognitive abilities moderated the relationship between coping and substance use treatment outcome (Tapert et al., 2004). Specifically, ten coping factors and five neurocognitive scores were evaluated in hierarchical linear regression with age, years of education and preadmission drinks. With alpha set at .001, the results indicated that maladaptive coping in potential relapse risk situations predict subsequent drinking, particularly for patients with *better* scores on neuropsychological tests (Tapert et al., 2004). Therefore, poor coping was particularly detrimental for those with good cognitive skills. It is unclear from this study if Tapert et al. (2004) is stating that coping skills do not matter if neurocognitive deficits exist, or that they do not matter as much. In addition, another limitation of Tapert et al's (2004) study is that the sample size is small and the authors seem to be drawing many conclusions off the information gathered. For example, the coping style evaluated seems to be a self report measure which could be affected due to changes in responses from cognitively impaired versus not impaired individuals. Likewise, Tapert et al. (2004) did not discuss any comparison between coping styles of the impaired and unimpaired which would have been valuable in assessing this study. However, the issue of coping style as a variable in potential relapse of people with and without neurocognitive deficits is still an important issue. Tapert et al. (2004) concluded with suggestions for treatment matching that could prove valuable for recommendations following this study of the relationship between neuropsychological function and attrition.

Summary

In summary, many variables have been related to relapse in substance abuse treatment. Some of the variables identified include type of drug use (United Nations, 2002), gender (Stocker, retrieved September 20, 2005), support systems (United Nations, 2002), certain medications (United Nations, 2002; World Health Organization, 2004), length and intensity of treatment (Franey & Ashton, 2002; United Nations, 2002) and neuropsychological impairment (Miller, 1991). Addressing an individual's neuropsychological impairment might improve treatment retention resulting in reduced incidence of relapse. This study hopes to identify the relationship between neuropsychological impairment and relapse and attrition rates resulting in suggestions for improving retention in outpatient substance use treatment.

Homelessness

As previously mentioned, the majority of data for this study was collected on homeless male residents of the Guesthouse of Milwaukee, Inc. The assessments were performed on individuals receiving treatment at the 7C's Community Counseling Clinic which is located within the Guesthouse and therefore serves primarily homeless men. The occurrence of individuals becoming homeless at some point in their life has been related to substance use issues, neuropsychological deficits, mental illness, socioeconomic status and other variables (Booth, Sullivan, Koegel & Burnam, 2002; National Coalition for the Homeless, retrieved January 12, 2006). Therefore, for the purposes of this study, the relationship of homelessness with substance use issues, neuropsychological functioning and attrition in substance dependence treatment will be reviewed.

Defining and Identifying Homeless Individuals

In order to study homelessness, one must be able to define what actually constitutes homelessness. This is no easy task as the status of homelessness is oftentimes a temporary condition instead of a permanent condition (National Coalition for the Homeless, retrieved January 12, 2006). Likewise, many homeless individuals are invisible to the researchers due to staying in a car, park or other places researchers cannot effectively search (National Coalition for the Homeless, retrieved January 12, 2006). Furthermore, the definition itself leads to controversy for individuals and organizations due to legal issues, funding and allocation of available resources (Clark & Rich, 2003; National Coalition for the Homeless, retrieved January 12, 2006; U.S. Department of Health and Human Services, retrieved January 12, 2006). Overall, many individuals have an interest in defining and redefining homelessness from a variety of perspectives to meet individual and organizational needs. Therefore, when reviewing the literature on homelessness, it is important to remain cognizant of the difficulties in defining homelessness.

Though controversy has existed on the definition of what exactly constitutes homelessness, researchers have still collected information on the occurrence of homelessness. For example, in a report by the Institute for the Study of Homelessness and Poverty at the Weingart Center (2005), the numbers of homeless people for 56 cities across the United States were published (Institute for the Study of Homelessness and Poverty, retrieved January 12, 2006). Included in the results of the Weingart study (2005) were statistics for Milwaukee, Wisconsin which is where the data was collected for this study. In 2005, the regional population in Milwaukee was

583,624 according to the Weingart study (Institute for the Study of Homelessness and Poverty, retrieved January 12, 2006). The homeless population in Milwaukee was reported as 2,818 which was 0.5% of the population (Institute for the Study of Homelessness and Poverty, retrieved January 12, 2006). The results from all of the cities listed included a homeless population across the cities ranging from less than 0.1% to 1.2% (many cities had <0.1%, Orange County, Arizona had 1.2%; Institute for the Study of Homelessness and Poverty, retrieved January 12, 2006). Likewise, in 2007, the Milwaukee Continuum of Care estimated that there are 1,470 homeless adults and children on a given day (Milwaukee Continuum of Care, 2007). The total reported homeless for the state of Wisconsin in 2007 was 5,648 and for the United States as a whole was 671,859 (National Alliance to End Homelessness, retrieved May 25, 2009).

Many reasons are reported for why so many people have episodic or chronic homelessness including poverty, availability of affordable housing, availability of health care, domestic violence, weak social support, mental illness and addiction disorders (National Coalition for the Homeless, 2005; SAMHSA, 2005). Though all of these issues are important and influential, for the purpose of this study, we are interested in those related to addiction and mental health.

Substance Use and the Homeless

The rates of alcohol and drug abuse are disproportionately high among the homeless population (National Coalition for the Homeless, 2005; SAMHSA, 2005). According to Glasser and Bridgman (1999) alcohol abuse has been found to be as high as 68% among homeless men and 30% among homeless women (Glasser &

Bridgman, 1999). Likewise, in a study by Solliday-McRoy et al., (2004) 93% of the 90 homeless men studied reported having a substance abuse or dependence problem (Solliday-McRoy et al., 2004). Likewise, in a study by Clark and Rich (2003) of 172 adults (79 male, 73 female) that either were homeless or at immediate risk of becoming homeless, approximately half had a diagnosis of a substance use disorder. Even higher numbers are reported by Jainchill, Hawke and Yagelka (2000) who reported that an estimated two-thirds of the homeless are alcohol abusers and half abuse other drugs. In addition, Jainchill, Hawke and Yagelka (2000) reported that among those in shelters, almost 90% are estimated to have alcohol problems and over 60% have problems with other drugs. The extent of the relationship between homelessness and substance use disorders has also been recorded by treatment facilities. In a report from the Drug and Alcohol Services Information System (2003), more than 120,000 admissions to substance abuse treatment facilities were homeless at the time of admission (SAMHSA, retrieved January 12, 2006). It should be clarified that this number (120,000 admissions) represents admissions and not necessarily separate homeless individuals as an individual may have been admitted on more than one occasion.

Mental Illness and the Homeless

Although some have identified substance abuse as the primary individual factor related to homelessness (Jainchill et al., 2000), mental illness is also frequently reported among homeless individuals (Booth et al., 2002; National Coalition for the

Homeless, retrieved January 12, 2006; SAMHSA, 2005). According to the National Coalition for the Homeless in a report dated July of 2005, 20-25% of the single adult homeless population suffers from some form of severe and persistent mental illness (National Coalition for the Homeless, 2005). Likewise, in a study of 438 individuals referred to receive acute psychiatric care in a hospital between 1990 and 1992, 24% were found to be homeless (Kuno, Rothbard, Averyt & Culhane, 2000). However, Kuno et al. (2000) defined homelessness as anyone who had an admission to a shelter between 1990 and 1993, they were not necessarily homeless at the time of admission to the hospital. In Solliday-McRoy et al. (2004) study of 90 homeless men, 50% reported previous mental health diagnoses with the majority (28%) for mood disorders. It is important to note that these reports of mental health issues were self reported by the participants and not specifically investigated by Solliday-McRoy et al (2004).

Neuropsychological Functioning and the Homeless

Though the mental health diagnosis was not specifically investigated by Solliday-McRoy et al. (2004), the neuropsychological functioning of homeless men was assessed. A neuropsychological battery consisting of the Neurobehavioral Cognitive Status Examination, Wechsler Abbreviated Scale of Intelligence, Digit Span Subtest of the WAIS, Rey Auditory Verbal Learning Test (RAVLT), Rey Osterrieth Complex Figure Test (RCFT) and the Letter-Word Identification and Passage Comprehension subtests for the Woodcock Johnson Psychoeducational Battery Revised was given to 90 homeless men living in the Guesthouse shelter, which is the same shelter where I collected the data for this study (Solliday-McRoy et

al., 2004). Impaired cognitive functioning as demonstrated by performance on the Cognistat was found in 80% of the participants with the subtest assessing memory showing the most frequent (64%) impairment (Solliday-McRoy et al., 2004). In addition, the participants scored below average on general intellectual abilities on the WASI with mean Verbal IQ score of 83.73 (*SD* 16.03), mean Performance IQ of 87.07 (SD 14.87) and mean Full Scale IQ of 83.92 (*SD* 15.24; Solliday-McRoy et al., 2004). Nearly half of the sample received scores that fell below 85 on the WASI indicating impaired capacities in a broad range of cognitive abilities (Solliday-McRoy et al., 2004). Likewise, 28% received scores of less than 85 on the digit span subtest suggesting attentional deficits and results from the RAVLT suggested memory and verbal learning deficits in more than half the sample (Solliday-McRoy et al., 2004). Deficits in processing speeds, visuomotor, visual-perceptual integration skills and visuospatial memory were indicated for nearly three quarters of the sample from RCFT results (Solliday-McRoy et al., 2004).

There are many strengths to the Solliday-McRoy et al. (2004) study including the voluntary participation of individuals, the comprehensive instruments used, sample size and the use of statistical procedures to attempt to evaluate the impact and relationship of TBI, substance use and mental illness. In addition, the authors reported that all participants were asked to abstain from any alcohol or drug use for 8 hours prior to testing so as to not interfere with the performance during assessment (Solliday-McRoy et al., 2004). However, it is unclear whether participants were tested for any alcohol or drugs in their system at time of assessment. In addition, it is

unclear why Solliday-McRoy et al. (2004) used an 8 hour gap versus a longer period to allow participants to recover somewhat from any and all substance use.

Results similar to Solliday-McRoy et al. (2004) were found by Gonzalez, Dieter, Natale and Tanner (2001). Gonzalez et al. (2001) also concluded that large numbers of homeless individuals are neuropsychologically impaired. Sixty homeless individuals were given the Abbreviated Halstead-Reitan Battery and the Mini Mental Status Exam (MMSE; Gonzalez et al., 2001). A high incidence of neuropsychological dysfunction was concluded with 80% of participants showing impairment on the Abbreviated Halstead-Reitan and 35% showing impairment on the MMSE (Gonzalez et al., 2001). One strength of this study was the authors' use of a regression analysis to suggest that 29% of the variance in the two instruments used was accounted for by patient education (Gonzalez et al., 2001). Overall, Gonzalez et al. (2001) concluded that as large numbers of the homeless are neuropsychologically impaired, this should be considered for treatment planning.

Other authors have found very similar results to Gonzalez et al. (2001). In a study of 155 homeless men and 49 homeless women, Buhrich, Hodder and Teesson (2000) also found cognitive impairment using the MMSE. Buhrich et al. (2000) used the Composite International Diagnostic Interview, which included an alcohol use disorder section and the MMSE to assess the 204 participants. Of the 204 participants, 20 (10%) were found to be cognitively impaired as compared to a reported 1.7% that are impaired in the general adult population (Buhrich et al., 2000). It is unclear if Buhrich et al. (2000) assessed for current health concerns, head injuries

or current alcohol or drug consumption which could have affected participant's performance on the assessment instruments.

Attrition Rates for Homeless Individuals Receiving Substance Use Services

As previously discussed, many issues influence an individual's homeless status and can be influenced by the homeless status of an individual such as poverty, mental illness, substance use and abuse, health status, access to health care, transportation, support and neuropsychological deficits (National Coalition for the Homeless, 2005; National Mental Health Association, retrieved January 12, 2006). Another variable with an interactive relationship with homelessness, which has been previously reviewed in this paper, is stigma (NSDUH, 2004). Individuals in need of treatment for a substance use disorder that are also homeless face the added stigma of their homeless status (National Mental Health Association, retrieved January 12, 2006). It can be due to any or all of these issues that a homeless individual's ability to complete treatment for a substance use disorder can be compromised (National Mental Health Association, retrieved January 12, 2006).

As mentioned, many variables can affect the rates of attrition for homeless individuals receiving substance abuse services. The substance abuse treatment services available for homeless individuals range from outreach offers of engagement in a human relationship (e.g., the Park Homeless Outreach Project in New York City) to formalized treatment programs inside of shelters (e.g., the 7C'clinic within the Guesthouse in Milwaukee; National Institute on Alcohol Abuse and Alcoholism, retrieved January 27, 2006). Likewise, there are multiple treatment programs of varying intensity that are open to many populations, including homeless individuals,

if they meet the admission criteria (e.g., any hospital program with outpatient treatment, day treatment, inpatient, etc.). Attrition rates for homeless individuals participating in substance abuse treatment can vary on the level of intensity of the program they attend (National Institute on Alcohol Abuse and Alcoholism, retrieved January 27, 2006). As previously discussed, attrition rates in general vary due to length, type and intensity of drug use as well as length, type and intensity of treatment (Franey & Ashton, 2002; Simpson, Joe & Rowan-Szal, 1997). However, the variable of treatment intensity may affect the attrition rates more so in homeless individuals as they simply have other survival demands to attend to if they are attempting to participate in a traditional outpatient program or an inpatient program that they are not immediately admitted into. Indeed, the delay in starting treatment after initial assessment has been reported by some as one of the main reasons for premature exit from a substance abuse treatment program (Zerger, 2002). In addition, there are many variables that often times are depleted or nonexistent in an individual who is homeless that can also affect client attrition rates including employment status, social support networks, positive self efficacy and feeling as if one is close to their ideal self (Northwest Frontier Addiction Technology Transfer Center, 2003). Specifically, attrition rates as high as 58% to 66% have been reported for homeless individuals in substance use treatment (National Institute on Alcohol Abuse and Alcoholism, retrieved January 27, 2006; SAMHSA, 1998) with some reporting rates as high as 80% (Zerger, 2002). The severity of these numbers supports the need for further research on attrition rates from substance abuse treatment for homeless individuals.

Summary

The extent of homelessness in the United States and more specifically in Milwaukee, Wisconsin is a severe and chronic concern (Institute for the Study of Homelessness and Poverty, retrieved January 12, 2006; U.S. Department of Health and Human Services, retrieved January 12, 2006). Homelessness has been found to be related to substance use disorders (Clark & Rich, 2003; Glasser & Bridgman, 1999; Jainchill et al., 2000), mental illness (Booth et al., 2002; Jainchill et al., 2000; National Coalition for the Homeless, 2005) and neuropsychological functioning deficits (Buhrich et al., 2000; Gonzalez et al., 2001; Solliday-McRoy et al., 2004). Furthermore, due to confounding variables, the attrition rates for homeless individuals receiving substance use treatment are reported at rates well over fifty percent (National Institute on Alcohol Abuse and Alcoholism, retrieved January 27, 2006; SAMHSA, 1998; Zerger, 2002). Though much research has been done on substance use treatment and the homeless, the research is lacking in regards to attrition from substance abuse treatment related to neuropsychological deficits. Indeed, not even one study was found that addressed the relationship between neuropsychological deficits and attrition rates from substance abuse treatment in homeless men. Therefore, the importance of this study evaluating neuropsychological functioning and the relationship to attrition rates in substance use treatment is again validated.

Multicultural Considerations

When evaluating neuropsychological functioning one must remain cognizant of possible demographic and multicultural issues related to assessment and diagnosis.

Participant's age, gender and race can all affect performance on particular test instruments (Groth-Marnat, 1999; Posner & DiGirolamo, 2000; Sullivan et al., 2002).

For example, aging can affect performance on many neuropsychological tests (Posner & DiGirolamo, 2000). Older adults are disproportionately disadvantaged on tests of executive function. Specifically, large age related differences are found on tasks that involve shifting sets, which is considered a primary example of executive functioning (Posner & DiGirolamo, 2000).

In relation to gender, Sullivan, Fama, Rosenbloom and Pfefferbaum (2002) reported that alcohol dependent women exhibit a similar pattern of impairment in cognitive tests as men. The areas most severely affected in the women, showing at least a -0.75 standard deviation difference from the healthy controls, involved short term memory and fluency (fluency involves the ability to write/speak as many words starting with a certain letter that one can come up with and creating a variety of designs within prearranged arrays of dots; Sullivan et al., 2002). Scientists have shown how alcoholism affects the nervous system and the brain for decades, but primarily in men (Sullivan et al., 2002). With regards to gender, the American Academy of Neurology (AAN) finds gender to have "consistent but minor effects on neuropsychological assessment" (American Academy of Neurology, 1996, p. 3). The AAN goes on to explain that women perform better on tests of verbal memory than men and suggest that men decline more than women on neuropsychological tests during the normal course of aging (American Academy of Neurology, 1996).

Not only has most of the research been performed with men, but also more specifically, it has been done with European American men (Groth-Marnat, 1999).

Critics believe that most psychological tests are heavily biased and reflect the values of European American middle class society (Groth-Marnat, 1999). Likewise, a study in the Journal of the International Neuropsychological Society (2004) reviewed past studies that investigated cultural bias and neuropsychological testing (Kennepohl, Shore, Nabors & Hanks, 2004). In summarizing several studies, Kennepohl et al. (2004) reported that many medically healthy minorities in the United States are considered cognitively impaired at a much higher rate than European Americans-even when they controlled for other variables such as years of education and socioeconomic status (SES). The Kennepohl et al. (2004) study assessed 71 participants using the African American Acculturation Scale with 40 being tested by a Black examiner and 31 by a White examiner. The results suggested a significant association between level of acculturation and neuropsychological performance even after controlling for other confounding variables such as age, sex, years of education and SES (Kennepohl et al., 2004). Similarly, researchers at Columbia Health Sciences studied whether quality, rather than quantity of education could help explain lower neuropsychological test scores (Dougherty, 2002). By administering a reading test to 384 elderly African Americans and Whites, the researchers found they could eliminate the racial differences in the neuropsychological assessment scores (Dougherty, 2002). The results of the Columbia Health Science study suggest that including an assessment of reading skills will help the neuropsychologists know what scores to expect from people with diverse backgrounds (Dougherty, 2002).

With the issue of bias in mind, one must be cautious when interpreting scores of ethnic, racial, age or gender categories that a specific assessment tool was not

normed on (Groth-Marnat, 1999). For the purpose of this study, I needed to be cautious that the results of the neuropsychological tests are from neuropsychological deficits, not test bias. I wanted to be sure I was measuring executive function deficit in all participants, not just the White males. When evaluating the relationship of executive functioning and attrition, I wanted to be sure to capture those precise variables and not variables related to gender, ethnicity or test bias. Therefore, all of these variables were considered in selection and use of assessment materials.

Chapter III**:** Methodology

The primary purpose of this chapter is to describe the methodology employed in this study of the executive function of patients with a substance dependence diagnosis and effects on attrition. Descriptions of the proposed participants, research design, instruments and procedures are provided.

Participants

Participants were recruited from the adult males seeking services at the 7C's Community Counseling Clinic (7Cs) located within the Guesthouse of Milwaukee. Upon initial contact with 7C's, individuals were assessed for substance related diagnoses. If an individual was identified as having a substance use diagnosis, the researcher presented the purpose and goals of the project, explained the nature of the test instruments, described time requirements and reviewed confidentiality procedures. In addition, it was explained that participation in the study was completely voluntary and had no effect on shelter admission, length of shelter stay or any other service offered at the shelter. An assessment was completed for all of those who agreed to participate.

Funding

In researching various funding opportunities, I contacted major breweries including Miller Brewery and local substance abuse organizations including Aurora and Cornerstone. In addition, many options for grants were researched through National Institute of Health, Division 40 of APA and multiple other online sources.

Unfortunately, no funding was retrieved through any of these sources. As the participants were people staying at the guesthouse, the financial incentive was somewhat less important as we were not be asking them to go anywhere outside of their home. In addition, as all clients who receive services from the 7C's clinic in the Guesthouse are required to complete an initial intake assessment prior to receiving services, the participants would partake in an assessment whether or not they are part of the study. If the study participants are then paid for their time, this could cause discontent for all other and future 7C's clients who are not paid for their time. Therefore, no payment was given to study participants. However, offering some form of payment to participants might have improved participant motivation throughout the assessment process.

Design

The major domains of this study included executive functioning of people with a substance use diagnosis and attrition rates. The data collected was intended to be exploratory in nature and not to be assumed representative as a thorough investigation of these domains.

Independent and Dependent Variables

The dependent variables for this study include the length of time the participant remains in treatment and number of sessions. I had also intended to evaluate time of relapse and frequency of relapse but was unable to gain access to relapse information. The independent variable is level of executive functioning as defined by the scores obtained by our chosen neuropsychological battery which

includes subtests of the Delis Kaplan and the Continuous Performance Test II (CPT II).

Other Variables

A variety of other variables including demographic characteristics, health history (including head injury), and subjective level of fatigue and mood at time of assessment were considered as supplemental variables. All of these variables could be confounding variables when evaluating the impact of neuropsychological functioning and attrition. We want to be sure that any relationship found between neuropsychological functioning and attrition is a true relationship and not one modified by one of these other variables. In addition, if we found that one of the other variables was an important predictor, we would highlight this for treatment suggestions and future research directions as is discussed in detail below.

Sample Size

Sample size is an important variable to consider as the sample size needs to be large enough that an effect of such magnitude to be of scientific significance will also be statistically significant. However, it is just as important that the sample size not be too big that an effect of little scientific importance is still detected as statistically significant (Lenth, 2001). I intended to exert energy and resources in ways that would be clinically and statistically significant. Factors that affect sample size include the Type I error rate, power of the test, and the effect size (Friedman, Furberg & DeMets, 1998). The calculation of sample size, with provisions for adequate levels of significance, power and effect size is discussed below.

The level of significance or alpha (α) level is the probability of making of Type I error or rejecting the null hypotheses when it is true (Grimm & Yarnold, 1995). The determination of where to set alpha is a function of balancing Type I and Type II errors (Grimm & Yarnold, 1995). Type II error occurs when the researcher does not reject a false null hypothesis, a probability called beta (β). For example, raising the level of significance (raising the probability of making a Type I error) from .05 to .10 decreases the probability of making a Type II error. Therefore, in order to find an acceptable balance between Type I and Type II errors, alpha was set at .05 ($\alpha = .05$) which is the preferred standard alpha of many researchers (Friedman et al., 1998).

It is also important to consider Power in the design of a study. Power is defined as the probability of rejecting the null when it is false and is calculated as $1 - \beta$ (Hinkle, Wiersma & Jurs, 1998; Huck, 2000). Due to the inverse relationship of alpha and beta, as alpha (α) increases, beta (β) decreases and power ($1 - \beta$) then increases. Therefore, the larger the power, the more likely one is to reject the null when it is false (Norussis, 2002). Hinkle, Wiersma and Jurs (1998) state that several authors have indicated that Type I errors are typically more serious than Type II errors and therefore suggest a 4:1 ratio of β to α. Therefore, as we have established alpha at .05, the corresponding power is $1 - 4(.05) = .80$ (Hinkle, Wiersma & Jurs, 1998). This power is large enough to help control for Type II errors, but not too large to put unrealistic demands on the researcher for huge sample sizes (Huck, 2000).

Effect size is also considered when determining sample size. Cohen defines effect size as the "degree to which a phenomenon exists" (Cohen, 1977, p. 9). The

effect size score represents the magnitude of the intervening treatment's effect (Grimm & Yarnold, 1995). Huck (2000) describes how Jacob Cohen has suggested that researchers can set the effect size to .20, .50 and .80 depending on whether they are interested in detecting a small, medium or large deviation from the null. However, many researchers warn that identifying the effect size is not so simple (Hinkle, Wiersma & Jurs, 1998). In fact, Cohen himself warns that it is always better for the researcher to specify effect size by thinking about the particular study being conducted rather than just deciding to use one of the accepted values for small, medium and large effect (Huck, 2000). Therefore, to calculate effect size for this study, we should not have arbitrarily picked an effect size, but rather considered what distinguished trivial from meaningful deviations from the null. Although this is not typically a difficult task, determining this requires knowledge about variability in the population being studied (Huck, 2000). For this study, I needed to find similar research on the relationship between neuropsychological function and attrition rates with an effect size reported. As previously discussed, this specific research is extremely rare in current publications. In addition, I reviewed similar types of research, such as attrition rates in outpatient substance dependence treatment, and was unsuccessful at finding any reported effect sizes. The majority of social research produces a small to medium effect size (Grimm & Yarnold, 1995). Therefore, I chose a small effect size as defined by Cohen as an effect size between .20 and .30 realizing this is an inadequate method of determining effect size.

With alpha set at .05, power at .80 and an effect size of .20 to .30, sample size can be chosen using a chart on page 651 of Hinkle, Wiersma and Jurs (1998).

According to this chart, the sample size needed would be between 71 and 155 for a small effect size between .20 and .30 (Hinkle, Wiersma & Jurs, 1998). Again, this determination of sample size is not adequate, but instead a result of the limited data I was working with. Part of the value of this study will be the reported information on effect size for future researchers interested in similar areas of study.

With the limited information available for calculating sample size, sample size was determined mostly as a result of practicality related to resources of this study including time, available participants and manpower to conduct the necessary assessments. With these variables in mind, as well as the variables of power, alpha and effect size, the sample size was arbitrarily set at 100. This allowed for a sample size which is reasonable with the resources available, as well as a sample size that fits with the partial calculations I was able to perform. The 100 needed assessments was feasible to obtain but yet we were unable to complete the 100 assessment due to a change in management of the 7C's clinic.

Instruments

The instruments used for this study included specific subtests of the Delis-Kaplan Executive Function System (D-KEFS) and the Conners' Continuous Performance Test II (CPT II) for evaluation of neuropsychological strengths and weaknesses, the Wechsler Abbreviated Scale of Intelligence (WASI) for an estimate of general intellectual ability. In addition, the Addiction Severity Index (ASI) was used to evaluate the severity of problem areas associated with alcohol and drug dependence and the Mini International Neuropsychiatric Interview (MINI) was used

to establish any other DSM-IV diagnoses. All of these instruments are reviewed in detail below.

Delis-Kaplan Executive Function System (D-KEFS)

The D-KEFS was published by Dean Delis, Edith Kaplan and Joel Kramer to assess key components of executive function (Psychological Corporation, n.d.). The D-KEFS is a neuropsychological test battery consisting of nine subtests that assess higher level cognitive abilities described as executive functions (Dugbartey & Ramsden, in press). The D-KEFS is individually administered in a game like format designed to be engaging for participants to encourage optimal performance in children and adults (Psychological Corporation, retrieved December 13, 2004). Each of the nine subtests comprising the D-KEFS were developed as stand alone measures and include the Trail Making Test, Verbal Fluency Test, Color Word Interference Test, Sorting Test, Twenty Questions Test, Word Context Test, Design Fluency Test, Tower Test and Proverb Test (Dugbartey & Ramsden, in press). The tests of interest for this study included Tower Test, Color Word Interference Test, Trail Making Test and Verbal Fluency. The decision was made to use four rather than all nine of the subtests to focus specifically on executive functions and reduce the time commitment associated with facilitating all nine subtests. These four subtests included in the battery for this study will be discussed in more detail below.

D-KEFS subtests

The nine subtests of the D-KEFS were designed to be autonomous instruments that could be used individually or in combination with other D-KEFS subtests. The selection of which tests to use is determined by the needs and time

constraints of the examiner (Dugbartey & Ramsden, in press). Each subtest takes approximately 20 minutes to administer and score (Dugbartey & Ramsden, in press). In addition, computer scoring software is available which can further reduce the time involved in manual scoring of the subtests (Psychological Corporation, retrieved December 13, 2004). Of the nine subtests of the D-KEFS, the decision was made to use four particular tests to focus specifically on executive functions and reduce the time commitment associated with facilitating all nine subtests.

The four D-KEFS subtests I used for this project included the Tower Test, Color Word Interference, Trail Making Test and Verbal Fluency. We had also intended to use the Sorting Test, but under the guidance of Dr. Terry Young we decided to eliminate it due to time constraints and repetition with other measures. The Tower Test is similar to the Towers of Hanoi test and the Tower of London test. The Tower Test measures spatial planning, rule learning, inhibition of impulsive responding, inhibition of perseverative responding and establishing and maintaining the instructional set (Dugbartey & Ramsden, in press). The Tower Test is a task in which the participant attempts to move five rings across three pegs to build a tower in the fewest number of moves possible (Dugbartey & Ramsden, in press). The Color Word Interference Test is a modified version of the Stroop (1935) test. It measures inhibition of a more autonomic verbal response (reading) in order to generate a conflicting response naming the dissonant ink colors (Dugbartey & Ramsden, in press). The third subtest used was the Trail Making Test. The Trail Making Test is a modified version of the Trail Making test and consists of five conditions instead of two (Dugbartey & Ramsden, in press). The D-KEFS Trail Making Test measures

flexibility of thinking on a visual motor task and assesses whether a deficient score on the switching condition is related to a higher level deficit in cognitive flexibility (Dugbartey & Ramsden, in press). The last subtest of the D-KEFS I used is the Verbal Fluency test. The Verbal Fluency test is a modified version of the Controlled Oral Word Association Test (COWAT, 1969). The Verbal Fluency test is sensitive to frontal lobe involvement in general and left-frontal lobe damage in particular (Dugbartey & Ramsden, in press). The Verbal Fluency test measure fluent productivity in the verbal domain by requiring the participant to generate words in phonemic format from overlearned concepts (Dugbartey & Ramsden, in press).

D-KEFS psychometrics.

The D-KEFS was standardized on a stratified sample of 1,750 individuals including 700 people aged 8-15 years old, 700 people between 16 and 59 years old and 350 between 60 and 89 years old (Dugbartey & Ramsden, in press). The D-KEFS is considered to have "adequate psychometric properties and a strong norming base" (Dugbartey & Ramsden, in press, p. 286). Internal consistency reliability was adequate for composite scores on the Trail Making Test (from .57 to .81), Verbal Fluency Test (from .32 to .90) and Color Word Interference Test (.62 to .86). The test-retest reliability estimate of the D-KEFS was found to be "generally impressive" though "quite variable across age groupings" (Dugbartey & Ramsden, in press, p.283).

In regards to validity, the Mental Measurement Yearbook (15th edition) reported that adequate data was presented on the intercorrelations of various intratest measures of the D-KEFS (Dugbartey & Ramsden, in press). Likewise, some of the D-

KEFS subtests (such as the Sorting Test) showed some impressive correlation results (Dugbartey & Ramsden, in press). However, very limited concurrent validity evidence was available comparing the D-KEFS and other neurocognitive assessments. For example, the correlations between the D-KEFS and the California Verbal Learning Test-Second Edition was rather low (Dugbartey & Ramsden, in press). Overall, the reliability and validity of the D-KEFS are considered to be adequate for researchers (Dugbartey & Ramsden, in press). Furthermore, as the D-KEFS is able to evaluate multiple aspects of executive functions, which are a primary focus for this study, the D-KEFS is a necessity for this study.

Conners' Continuous Performance Test II (CPT II)

The CPT II was developed by C. Keith Conners as a visual performance task which evaluates attentional variables in individuals age six or older (IPS, 2005). The response patterns identified by the CPT II provide information on attention, impulsivity, activation/arousal and difficulties in maintaining vigilance (IPS, 2005). The CPT II is computer administered and scored in approximately 14 minutes (IPS, 2005). After a practice exercise on the computer, the administration begins requiring the participant to press the space bar or click the mouse when any letter except 'X' appears on the computer screen. The computerized program of the CPT II captures response times and records them to the nearest millisecond (Klecker & Sime, in press). Scores are automatically computed, graphed and converted to a text which explains the results to the administrator (Klecker & Sime, in press). The instructions and administration are simple for the participant and the administrator.

CPT II psychometrics.

The CPT II was normed on a sample of 2,682 subjects including clinical and nonclinical samples (IPS, 2005). Normative data include a clinical sample of 378 cases diagnosed with ADHD, 223 adult individuals with neurological impairment and a nonclinical sample of 1920 individuals from the general population (Klecker & Sime, in press). Reliability and validity information is provided in the CPT II Technical Guide and Software Manual (IPS, 2005). Two types of reliability including split half and standard error provide support for the psychometric soundness of the CPT II. The CPT II shows adequate consistency with regards to split half reliability and the standard error measurement values show that scores from the instrument are a reasonable match to the true performance of individuals (IPS, 2005). However, according to a review in the Mental Measurement Yearbook (15^{th} edition), the split half procedure was found to be difficult to follow and the correlations nearly impossible to interpret (Klecker & Sime, in press). Test-retest reliability resulted in a range of .05 to .92 (Klecker & Sime, in press). Statistical validation is discussed in the CPT II manual with regards to demonstrating the tests ability to discriminate between general populations and clinical groups (IPS, retrieved April 27, 2006). The Mental Measurement Yearbook (15^{th} edition) review considers the CPT II a reliable instrument with moderate validity which can be easily used and interpreted (Klecker & Sime, in press). However, the authors of the CPT II do caution that it is not to be used alone as a diagnostic tool (Klecker & Sime, in press). Given the CPT II's positive psychometrics, brief administration and the necessity of attentional

information for evaluating executive function, the CPT II is a valuable tool for this study in conjunction with the other identified assessment tools.

Wechsler Abbreviated Scale of Intelligence (WASI)

The two subtest format of the WASI was developed in 1999 to provide clinicians a reliable method to obtain a brief measure of intelligence on individuals aged 6-89 years old (Harcourt Assessment, retrieved April 27, 2006). Though many short forms of the Wechsler Scales exist, the WASI was developed to provide a consistent, well normed, brief measure of intelligence (Keith, Lindskog & Smith, 2004). The WASI is available in a four or two subtest format giving the administrator control over the time and depth of the assessment (Keith, Lindskog & Smith, 2004). The four subtest format results in a Full Scale IQ (FSIQ, Verbal IQ (VIQ) and Performance IQ (PIQ) with a 30 - minute administration time (Harcourt Assessment, retrieved April 27, 2006). The two subtest form of the WASI includes the Vocabulary subtest and the Matrix Reasoning subtest resulting in a Full Scale IQ (FSIQ) score with a 15 - minute administration time (Harcourt Assessment, retrieved April 27, 2006). For all subtests, raw scores are converted to T scores with all IQ scores having a mean of 100 and a standard deviation of 15 (Keith et al., 2004). For the purposes of this study, we utilized the two subtest form of the WASI to reduce the time the participant is involved in testing.

WASI psychometrics.

The Wechsler Abbreviated Scale of Intelligence was standardized on a national sample of 2,245 children and adults with ages ranging from 6-89 years old (Keith et al, 2004) is considered to have strong psychometric properties (Harcourt

Assessment, retrieved April 27, 2006). The average reliability coefficient for adults on the two subtest format is reported at .96 (Harcourt Assessment, retrieved April 27, 2006). Likewise, the test-retest reliability for the two subtest format is .88 (Harcourt Assessment, retrieved April 27, 2006). In addition, the validity information on the WASI included correlations with other tests, and exploratory and confirmatory factor analysis (Keith et al., 2004). Correlations between the WASI and WAIS III ranged from .66 to .88 for subtests and .76 to .92 for IQ's (Keith et al., 2004). The Mental Measurement Yearbook (2004) review of the WASI considers the correlation with the WAIS III to be the WASI's greatest strength and its greatest weakness as it is not connected to anything but the Wechsler scales (Keith et al., 2004). Overall, the Mental Measurement Yearbook (2004) review considers the WASI to be well standardized and have adequate reliability and validity (Keith et al., 2004). As a brief estimate of intelligence versus a detailed description of intelligence is what we need for this study, the WASI is an appropriate addition to our battery.

Addiction Severity Index (ASI)

The Addiction Severity Index (ASI) was developed in 1980 by A. Thomas McLellan and collaborators from the University of Pennsylvania's Center for the Studies of Addiction (Treatment Research Institute, retrieved February 3, 2006). The ASI is a standardized, multidimensional instrument widely used in the field of substance abuse treatment (Treatment Research Institute, retrieved February 3, 2006). This semi-structured interview was designed to address seven potential problem areas in individuals with a substance use disorder. The seven areas include: (a) medical, (b) employment, (c) alcohol, (d) drug, (e) legal, (f) family/social, and (g) psychiatric

status (McGahan, Griffith, Parente & McLellan, 1986; McLellan, Luborsky, O'Brien & Woody, 1980). The interviewer can gather information on recent substance use (past thirty days) and lifetime problems in all seven of the problem areas (McGahan et al., 1986; McLellan et al., 1980). Therefore, the ASI provides an overview of the problems rather than focusing on any one single area.

ASI psychometrics.

The ASI has been normed on treatment groups including users of alcohol, opiates and cocaine (McLellan et al., 1980). In addition, it has been normed on public and private inpatient and outpatient treatment (McLellan et al., 1980). Finally, it has been normed on males, females, psychiatrically ill substance users, gamblers, homeless, probationers and employee assistance clients (McLellan et al., 1980). Therefore, it is appropriate for the homeless, male, substance abusing population included in this research. The ASI has shown test-retest reliability, split half reliability and internal consistency (McLellan et al., 1980). Likewise, the ASI has shown content, criterion and construct validity (McLellan et al., 1980). Finally, the ASI can be administered in approximately 50 to 60 minutes and can be used free of charge (McLellan et al., 1980) making it a positive part of our chosen assessment battery.

Mini International Neuropsychiatric Interview (MINI)

The Mini International Neuropsychiatric Interview (MINI) is an abbreviated psychiatric structured interview first developed in 1992 by David Sheehan and Yves Lecrubier to meet the need for a short but accurate psychiatric interview (Sheehan et al., 1998). The MINI is designed to assess the major adult Axis I diagnostic

categories, one Axis II diagnosis (antisocial personality disorder) and suicidality (Sheehan et al., 1998). The MINI is available in an electronic version allowing for simple computer administration and scoring which takes approximately 15 minutes.

MINI psychometrics.

The reliability of the MINI was tested with interrater reliability and test-retest reliability. The interrater reliability showed kappa values all above .75 (Sheehan et al., 1998). In addition, 70% of the kappa values were .90 or greater indicating excellent interrater reliability (Sheehan et al., 1998). The test-retest reliability scores included 61% with values over .75 and only one value was below .45. With regards to validity, the MINI was compared to the Structured Clinical Interview for the DSM-III-R (SCID) and the Composite International Diagnostic Interview (CIDI). When compared to the SCID, the MINI showed good or very good kappa values with only one score (current drug dependence) falling below .50 (Sheehan et al., 1998). When comparing the MINI and the CIDI, kappa values were also good or very good for most diagnoses with only two values (simple phobia and generalized anxiety disorder) falling below .50 (Sheehan et al., 1998). The MINI has shown high validation and reliability scores and can be administered in less time (15-30 minutes) than other comparable instruments such as the Structured Clinical Interview for the DSM-III (Sheehan et al., 1998) making it a useful instrument for this study.

Procedures

Informed Consent

When participants arrived for their assessment session, they were read an informed consent document in compliance with current Health Insurance Portability

& Accountability Act 2004 (HIPAA) regulations. This provided them with information regarding voluntary participation, confidentiality, the purpose of the study, and potential risks and/or benefits of participating in this study. Any questions regarding these issues were answered, they signed the consent and were offered a copy of it.

Assessment

All clients of the 7C's clinic are administered the ASI and MINI upon general admission. These instruments are part of the 7C's clinic intake process and therefore all clients entering the clinic are given these assessments, not just the participants for this study. All administrators were Registered Alcohol and Drug Counselors or Certified Alcohol and Drug counselors by the state of Wisconsin and were supervised by Certified Clinical Supervisors. Subsequently, if a substance use diagnosis was established, the neuropsychological test battery including the D-KEFS, CPT II and the WASI were administered after completion of the informed consent procedures. Standard administration was followed for each of the instruments as well as a standard order of administration. The average length of time to complete the neuropsychological test battery was approximately 90 minutes. Multiple clinicians were trained and supervised in the administration of this battery. A licensed psychologist qualified in administration and scoring of the neuropsychological assessment instruments (Dr. Terry Young) provided training prior to the time of assessment and provided supervision throughout the course of this study. All batteries were hand scored first by the assessor for the WASI. The D-KEFS and CPT II are computer scored. The hand scored subtests were then rescored by the lead

investigator to insure accurate scoring. A sample of the assessments were then reviewed by the supervising neuropsychologist and another psychologist (14 batteries or approximately 20%).

Follow Up

Participants were asked to provide contact information at intake to allow for follow up information to be gathered if and when the participant left treatment. Participants were informed of when this person will be contacted and what information would be asked of them. The appropriate releases were signed and attempts were made to contact the listed person when a participant dropped out of treatment to get information on reaching the participant for information on why they left treatment. We were hoping this information would provide a qualitative report of the participant's perception of why they left treatment allowing the study to evaluate the relationship between neuropsychological function and attrition to the participant's report of reasons for attrition. However, we had limited success actually reaching people due to wrong names,

wrong numbers, disconnected numbers and the cooperation of the listed contact person.

Therefore, we were unable to report the intended qualitative piece of this research.

Data Analysis

As we are comparing the relationship between the neuropsychological functioning, attrition rates and relapse rates in outpatient treatment for substance dependent males, there are several variables to evaluate requiring multiple data

analytic techniques. Survival Analysis (SA) and Logistic Regression (LR) are the two primary statistical applications we will used for this study.

Survival Analysis (SA)

Survival Analysis (SA) refers to a group of techniques designed for studying the occurrence of events in longitudinal data (Corning & Malofeeva, 2004). Given that I am evaluating attrition rates from outpatient treatment, which have a well-defined starting point and a 'failure' point, this is an appropriate model for my data (Dobson, 2002; Parmar &Machin, 1995). In SA, the dependent variable is the length of time to an event (Parmar & Machin, 1995). Classically, the event of interest was death-hence the term survival analysis (Luke & Homan, 1998). However, for the purpose of this study, the dependent variables assessed by SA were the number of outpatient sessions attended and total length of time in treatment prior to dropping out. In short, SA assists us in predicting the risk of occurrence of an event given the event has not yet occurred (Corning & Malofeeva, 2004; Luke & Homan, 1998). The advantages of SA over more traditional means of analysis, such as regression and analysis of variance (ANOVA), relate to the longitudinal nature of outpatient treatment that may not be well addressed by other methods (Corning & Malofeeva, 2004). Survival Analysis is important when analyzing data in which risks vary over time (Gerstman, 2003). Survival Analysis allows us to estimate the survival time of participants who complete the study as well as those who do not (Gerstman, 2003). This is considered a distinguishing feature of survival analysis and is referred to as *censoring* (Parmar & Machin, 1995; Venables & Ripley, 2002). Data is considered *right censored* in the event that we do not observe the outcome (end of treatment) for

all participants since data collection ends but the people are still in treatment (Gerstman, 2003; Luke & Homan, 1998). In SA studies, this is the most common type of censoring. However, if participants become unavailable due to physically moving out of the area, death or illness, this is considered *mid censoring* (Gerstman, 2003). We are no longer able to observe them, but they were not necessarily lost due to dropping out in the manner we are concerned with in this study. Therefore, if we had participants in our study that moved, died or otherwise do not represent the dichotomy of someone participating in treatment or dropping out, we could still account for the data and not allow it to have as big of a negative impact on our results. Furthermore, with SA, I could evaluate the time to the *event* (days or sessions to drop out) and what is known as *censor-status* (observation or non-observation of the event-drop out; Luke & Homan, 1998). Or, I could also investigate other variables which influenced survival times such as the neuropsychological functioning of the individual. Survival Analysis can incorporate categorical or continuous variables (Luke & Homan, 1998). With the multiple types of data that my chosen assessment battery produces, I had the opportunity to evaluate the data from both perspectives using impaired vs. non-impaired neuropsychological scores and the continuous type of data that the CPT II reaction time scores offer. Therefore, I was able to evaluate the impact of the client's neuropsychological functioning on survival or time in treatment prior to relapse or dropping out.

Within Survival Analysis there are parametric and nonparametric approaches available for our use. Some researchers report that the parametric approaches have 'fallen out of fashion' with the advent of the more nonparametric approaches

(Venables & Ripley, 2002). Some of the analytic techniques offered by SA include the Cox Proportional Hazards Model, Kaplan-Meier survival function estimation and Life Table analysis (Parmar & Machin, 1995; StatSoft, retrieved March 16, 2006). For this study, we used the Cox Model which is discussed later.

Logistic Regression (LR)

In addition to Survival Analysis, Logistic Regression was also used in analysis of the data. Logistic Regression can be used when the predictor variables are qualitative or quantitative, continuous or categorical, and the criterion variable is dichotomous (Grimm and Yarnold, 2000; Huck, 2000). Our predictor variable of interest is neuropsychological function which is quantitative and continuous and the dichotomous criterion variable was whether the participant dropped out or not. With SA we are able to address the participant's survival in treatment (continuous time oriented variable) as related to neuropsychological functioning, whereas with LR we address whether a relationship exists between neuropsychological function and whether the participant drops out or not (dichotomous variable). Logistic Regression can be used to determine the increase in probability of dropping out of treatment that is associated with neuropsychological functioning while controlling for other variables such age, IQ and types of substances used (Grimm & Yarnold, 2000). Like Linear Regression, Logistic Regression gives each regressor variable a coefficient value that measures the regressor's independent contribution to variations in the dependent variable (University of Exeter, retrieved May 10, 2006). With LR, we can assess whether the independent variables, as a whole, significantly affect the

dependent variable and identify the best variables to use in prediction of treatment drop out (University of Exeter, retrieved May 10, 2006).

Summary of Data Analysis

Survival Analysis and Logistic Regression were used to answer all study questions including identifying the level of neuropsychological impairment, if the impairment predicts rates of attrition from treatment and if a relationship exists between neuropsychological function and attrition. Using the two forms of statistical analysis (LR and SA) allowed us to more precisely and accurately evaluate all variables involved in this study.

In regards to the level of neuropsychological impairment, descriptive statistics and a verbal summary of findings is reported. As it is possible that neuropsychological functioning would improve over the course of the study, the neuropsychological assessment (Delis-Kaplan subtests and CPT II) was to be administered a second time half way through the 90 days we will be monitoring for drop out (45 days into treatment) and again at the 90 day mark. This would provide a quantitative description of any improvement in neuropsychological functioning over time. However, due to limited participation in this aspect of the research, we were unable to report any substantial data related to follow up assessments.

Attrition from substance abuse treatment was evaluated using Logistic Regression with attrition being treated as a dichotomous variable (drop out or no drop out). We intended to predict drop out from the indicators of neuropsychological functioning and control for other variables including age, IQ and any other treatment participation. In addition, Survival Analysis was also used in which the days in

treatment prior to drop out were the dependent variable and neuropsychological functioning was the independent variable. The relationship between neuropsychological function and drop out was evaluated with LR and the relationship between neuropsychological function and survival in treatment was evaluated with SA. Finally, the Cox Proportional Hazards model was used with the Survival Analysis.

In addition to the quantitative data collected, we intended to have a qualitative component to be reported on from the information gathered through the follow up procedures. This information would provide us with the participant's description of why they left treatment which could be compared to the quantitative data. However, as discussed above, this information was not successfully gathered.

Finally, as previously mentioned, no relative effect sizes could be found to be used to determine sample size for this study. Therefore, effect sizes were calculated and reported on as another valuable outcome of this study.

Overall, the use of the multiple statistical techniques allowed for assessment of all independent and dependent variables of interest. In addition, the statistical techniques allowed for evaluation of any confounding variables. Finally, these methods of analysis will provide an enormous amount of information on any relationships between neuropsychological function, attrition, relapse and multiple other potential variables.

Chapter IV: Results

Demographic Description

The present study utilized data from 68 participants. We had planned an n of 100 but due to a change in management of the clinic where we were collecting data, our n was reduced. We collected 75 total neuropsychological assessments on adult males in the 7C's Clinic of the Guesthouse of Milwaukee. Of the 75 completed assessments, seven (initial assessments completed) were eliminated due to facilitator error (initial assessments, facilitator skipped parts of subtest). The average age of our sample was 45 years (*SD* = 9 years). Education levels varied widely: 28% did not finish high school, 37% were high school graduates, and 12% had some college education or an associates degree, 18% graduated from college, and 5% had further education beyond college. None of the participants reported being married: 63% of the participants were single, 23% were divorced, 9% were separated, and 3% were widowed. Two in every three participants were African-American (66.2%), while 25% were Caucasian, and 9% belonged to other racial or ethnic groups (2.9 % Hispanic, 2.9% multiple races, 1.5 % Asian, 1.5% Native American), Three in every four (74.6 %) participants had received prior AODA treatment.

Neuropsychological Functioning

To determine the neuropsychological functioning of the present sample and answer our first research question, we utilized four subtests of the Delis Kaplan including the Tower Test, Color Word Interference, Trail Making Test and Verbal Fluency. We had originally planned to utilize the Sorting Test as well, but for the

sake of time and due to some duplicate information obtained by the sorting test, we limited the Delis Kaplan evaluation to these four subtests. We also used the CPTII, WTAR and WASI to evaluate neuropsychological functioning. All of these results will be reviewed.

D-KEFS.

The Delis Kaplan Executive Function subtests give us multiple scores for each subtest administered. A few key variables that are considered more global achievement measures will be discussed and the rest presented in a table. For most of the measures provided by the D-KEFS, the raw scores are converted to scaled scores with a mean of 10 and a standard deviation of 3. In order to determine whether the sample means in the present study were significantly different than the population norms for each test, a one-sample z-test was employed. Specifically, a two-tailed test was employed with an alpha level of .05. With a sample of 68, and the population parameters specified above, sample means that are 0.71 units higher or lower than the stated population mean are significantly different than the mean. By this criterion, a sample mean of 9.29 is significantly lower than the population mean of 10.

In addition to the standardized scores, the D-KEFS also provides contrast scores that quantify performance on a baseline task and a higher level task or two higher level tasks. These will be discussed more extensively below.

Tower Test.

The Tower Test measures spatial planning, rule learning, inhibition of impulsive responding, inhibition of perseverative responding and establishing and maintaining the instructional set (Dugbartey & Ramsden, in press). To assess how

the sample performed relative to the norm, the distribution of cases according to performance standards was examined. Of the 67 valid scores from the participants on the Tower Test total achievement score, 22 scored below average, 12 scored above average and 33 scored within average range (M = 9.25, SD = 2.97). In the present sample, the mean standardized scale scores for the Tower Test was lower than the criterion level of 9.29 set for rejecting the null hypothesis based on the one-sample z test. This finding suggests that the sample was drawn from a population with a lower than average level of performance on this measure.

Table 4.1

Tower Test total achievement score scaled

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	2	1	1.4	1.5	1.5
	3	1	1.4	1.5	3.0
	4	1	1.4	1.5	4.5
	5	5	7.2	7.5	11.9
	6	6	8.7	9.0	20.9
	7	8	11.6	11.9	32.8
	8	4	5.8	6.0	38.8
	9	8	11.6	11.9	50.7
	10	5	7.2	7.5	58.2
	11	10	14.5	14.9	73.1
	12	6	8.7	9.0	82.1
	13	10	14.5	14.9	97.0
	14	2	2.9	3.0	100.0
	Total	67	97.1	100.0	
Missing	999	2	2.9		
Total		69	100.0		

Trails.

The Trail Making Test is a modified version of the Trail Making test and consists of five conditions instead of two (Dugbartey & Ramsden, in press). The D-KEFS Trail Making Test measures flexibility of thinking on a visual motor task and assesses whether a deficient score on the switching condition is related to a higher level deficit in cognitive flexibility (Dugbartey & Ramsden, in press). The primary scoring measure for each of the five conditions of the D-KEFS Trail Making Test is the number of seconds that the examinee takes to complete each condition again with a mean of 10 and a standard deviation of 3. The first score is the Trail Making test visual scanning score, which is a timed score where the examinee seeks out the number 3 on a page of scattered numbers. Of the 68 valid responses, 18 were below average, 5 were above average and 45 were average (M = 8.8, SD = 3.45). The second score is the number sequencing score where the participant has to seek out and sequence numbers in chronological order. Of the 68 valid responses, 30 were below average, 5 were above average and 33 were average (M = 7.48, SD = 3.95). The third score is the letter sequencing score where the participant has to seek out and sequence letters in alphabetical order. Of the 68 valid responses, 28 were below average, 7 were above average and 33 were average (M = 7.48, SD = 3.93). The fourth score is the number-letter sequencing score where the participant has to sequence numbers and letters in order alternating between the two. Of the 66 valid responses, 29 were below average, 4 were above average and 33 were average (M = 7.6, SD = 3.89). The fifth score is a motor speed score that is calculated by having the participant trace a line while being timed. Of the 67 valid responses, 19 scored below average, 3 scored

above average and 45 scored within the average range (M = 8.8, SD = 2.95). In the present sample, the mean standardized scale scores for the Trail Making Test was lower than the criterion level of 9.29 set for rejecting the null hypothesis based on the one-sample z test. This finding suggests that the sample was drawn from a population with a lower than average level of performance on this measure.

Table 4.2

Trail making test visual scanning scaled

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	1	6	8.7	8.8	8.8
	2	1	1.4	1.5	10.3
	3	1	1.4	1.5	11.8
	4	1	1.4	1.5	13.2
	5	2	2.9	2.9	16.2
	6	1	1.4	1.5	17.6
	7	6	8.7	8.8	26.5
	8	5	7.2	7.4	33.8
	9	10	14.5	14.7	48.5
	10	12	17.4	17.6	66.2
	11	7	10.1	10.3	76.5
	12	11	15.9	16.2	92.6
	13	3	4.3	4.4	97.1
	14	2	2.9	2.9	100.0
	Total	68	98.6	100.0	
Missing	999	1	1.4		
Total		69	100.0		

Table 4.3

Trail Making test number sequence scaled

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	1	10	14.5	14.7	14.7
	2	2	2.9	2.9	17.6
	3	1	1.4	1.5	19.1
	4	4	5.8	5.9	25.0
	5	3	4.3	4.4	29.4
	6	6	8.7	8.8	38.2
	7	4	5.8	5.9	44.1
	8	6	8.7	8.8	52.9
	9	5	7.2	7.4	60.3
	10	13	18.8	19.1	79.4
	11	4	5.8	5.9	85.3
	12	5	7.2	7.4	92.6
	13	3	4.3	4.4	97.1
	15	1	1.4	1.5	98.5
	16	1	1.4	1.5	100.0
	Total	68	98.6	100.0	
Missing	999	1	1.4		
Total		69	100.0		

Table 4.4

Trail Making test letter sequence scaled

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	1	10	14.5	14.7	14.7
	3	5	7.2	7.4	22.1
	4	2	2.9	2.9	25.0
	5	4	5.8	5.9	30.9
	6	4	5.8	5.9	36.8
	7	3	4.3	4.4	41.2
	8	10	14.5	14.7	55.9
	9	6	8.7	8.8	64.7
	10	7	10.1	10.3	75.0
	11	6	8.7	8.8	83.8
	12	4	5.8	5.9	89.7
	13	6	8.7	8.8	98.5
	15	1	1.4	1.5	100.0
	Total	68	98.6	100.0	
Missing	999	1	1.4		
Total		69	100.0		

Table 4.5

Trail Making number letter sequencing scaled score

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	1	10	14.5	15.2	15.2
	2	1	1.4	1.5	16.7
	3	3	4.3	4.5	21.2
	4	1	1.4	1.5	22.7
	5	3	4.3	4.5	27.3
	6	4	5.8	6.1	33.3
	7	7	10.1	10.6	43.9
	8	5	7.2	7.6	51.5
	9	8	11.6	12.1	63.6
	10	3	4.3	4.5	68.2
	11	9	13.0	13.6	81.8
	12	8	11.6	12.1	93.9
	13	4	5.8	6.1	100.0
	Total	66	95.7	100.0	
Missing	999	3	4.3		
Total		69	100.0		

Table 4.6

Trail Making motor speed scaled

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	1	1	1.4	1.5	1.5
	2	1	1.4	1.5	3.0
	3	2	2.9	3.0	6.0
	4	3	4.3	4.5	10.4
	5	6	8.7	9.0	19.4
	6	1	1.4	1.5	20.9
	7	5	7.2	7.5	28.4
	8	5	7.2	7.5	35.8
	9	7	10.1	10.4	46.3
	10	11	15.9	16.4	62.7
	11	16	23.2	23.9	86.6
	12	6	8.7	9.0	95.5
	13	2	2.9	3.0	98.5
	14	1	1.4	1.5	100.0
	Total	67	97.1	100.0	
Missing	999	2	2.9		
Total		69	100.0		

Color Word Interference Test.

The Color Word Interference Test is a modified version of the Stroop (1935) test. It measures inhibition of a more autonomic verbal response (reading) in order to generate a conflicting response naming the dissonant ink colors (Dugbartey & Ramsden, in press). The completion time for each of four measures provides a global measure of performance, again with a mean of 10 and standard deviation of 3. The first condition is color naming which tests the speed at which an examinee can name repeating stimuli of color patches. Of the 68 valid responses, 31 were below average, 4 were above average and 33 were average (M = 7.52, SD = 3.8). The second condition is word reading, which evaluates the examinee's ability to read repeating words as quickly as possible. Of the 68 valid responses, 30 were below average, 5 were above average and 33 were average *(*M = 7.64, SD = 3.85). The third condition is inhibition, which reflects the examinee's ability to inhibit the more automatic task of reading words in order to name the dissonant ink color. Of the 68 valid responses, 25 were below average, 9 were above average and 34 were average (M = 8.22, SD = 3.78). The fourth condition is inhibition/switching, which requires adequate naming speed, reading speed, verbal inhibition and cognitive flexibility. Of the 68 valid responses, 26 were below average, 6 were above average and 36 were average (M = 8.33, SD = 3.64). The mean standardized scale scores for each of the Color Word measures was lower than the criterion level of 9.29 set for rejecting the null hypothesis based on the one-sample z test. This finding suggests that the sample was drawn from a population with a lower than average level of performance on this measure.

Table 4.7

Color Word color naming scaled

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	1	8	11.6	11.8	11.8
	2	1	1.4	1.5	13.2
	3	4	5.8	5.9	19.1
	4	5	7.2	7.4	26.5
	5	1	1.4	1.5	27.9
	6	7	10.1	10.3	38.2
	7	5	7.2	7.4	45.6
	8	3	4.3	4.4	50.0
	9	7	10.1	10.3	60.3
	10	11	15.9	16.2	76.5
	11	9	13.0	13.2	89.7
	12	3	4.3	4.4	94.1
	13	3	4.3	4.4	98.5
	17	1	1.4	1.5	100.0
	Total	68	98.6	100.0	
Missing	999	1	1.4		
Total		69	100.0		

Table 4.8

Color Word word reading scaled

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	1	8	11.6	11.8	11.8
	2	2	2.9	2.9	14.7
	3	1	1.4	1.5	16.2
	4	3	4.3	4.4	20.6
	5	6	8.7	8.8	29.4
	6	6	8.7	8.8	38.2
	7	4	5.8	5.9	44.1
	8	10	14.5	14.7	58.8
	9	3	4.3	4.4	63.2
	10	4	5.8	5.9	69.1
	11	10	14.5	14.7	83.8
	12	6	8.7	8.8	92.6
	14	5	7.2	7.4	100.0
	Total	68	98.6	100.0	
Missing	999	1	1.4		
Total		69	100.0		

Table 4.9

Color Word inhibition scaled

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	1	5	7.2	7.4	7.4
	2	2	2.9	2.9	10.3
	3	3	4.3	4.4	14.7
	4	4	5.8	5.9	20.6
	5	5	7.2	7.4	27.9
	6	2	2.9	2.9	30.9
	7	4	5.8	5.9	36.8
	8	4	5.8	5.9	42.6
	9	11	15.9	16.2	58.8
	10	6	8.7	8.8	67.6
	11	7	10.1	10.3	77.9
	12	6	8.7	8.8	86.8
	13	6	8.7	8.8	95.6
	14	3	4.3	4.4	100.0
	Total	68	98.6	100.0	
Missing	999	1	1.4		
Total		69	100.0		

Table 4.10

Color Word inhibition/switching scaled

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	1	5	7.2	7.4	7.4
	2	1	1.4	1.5	8.8
	3	5	7.2	7.4	16.2
	5	3	4.3	4.4	20.6
	6	4	5.8	5.9	26.5
	7	8	11.6	11.8	38.2
	8	4	5.8	5.9	44.1
	9	8	11.6	11.8	55.9
	10	8	11.6	11.8	67.6
	11	9	13.0	13.2	80.9
	12	7	10.1	10.3	91.2
	13	3	4.3	4.4	95.6
	14	1	1.4	1.5	97.1
	15	2	2.9	2.9	100.0
	Total	68	98.6	100.0	
Missing	999	1	1.4		
Total		69	100.0		

Verbal Fluency.

The Verbal Fluency test is a modified version of the Controlled Oral Word Association Test (COWAT, 1969). The Verbal Fluency test is sensitive to frontal lobe involvement in general and left-frontal lobe damage in particular (Dugbartey & Ramsden, in press). The Verbal Fluency test measures fluent productivity in the verbal domain by requiring the participant to generate words in phonemic format from over learned concepts (Dugbartey & Ramsden, in press). The total correct score for each of three conditions provides a global measure of performance on this task, again with a mean of 10 and standard deviation of 3. The first condition is letter fluency where the examinee generates lexical items while simultaneously observing several rules or restrictions. Of the 68 valid responses, 29 were below average, 6 were above average and 33 were average (M = 8.39, SD = 3.56). The second condition is category fluency, which requires the examinee to retrieve multiple words from high frequency semantic categories. Of the 68 valid responses, 24 were below average, 6 were above average and 36 were average (M = 9.2, SD = 6.8). The third condition is category switching which requires the examinee to retrieve semantic knowledge shifting between two categories. Of the 68 valid responses, 23 were below average, 6 were above average and 39 were average (M = 8.36, SD =3.98). The mean standardized scale scores for each of the Verbal Fluency measures was lower than the criterion level of 9.29 set for rejecting the null hypothesis based on the one-sample z test. This finding suggests that the sample was drawn from a population with a lower than average level of performance on this measure.

As this subtest, as well as all of the D-KEFS subtests were chosen due to their specific relationship with frontal lobe functioning, it makes sense that we would see impairment in each of these measures. This is consistent with our expectations of frontal lobe impairment in this population. More specifically, it is consistent with impairment in the dorsal lateral frontal lobe that is related to executive functioning.

Table 4.11

Verbal Fluency letter fluency total scaled

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	1	2	2.9	2.9	2.9
	2	1	1.4	1.5	4.4
	3	4	5.8	5.9	10.3
	4	4	5.8	5.9	16.2
	5	1	1.4	1.5	17.6
	6	7	10.1	10.3	27.9
	7	10	14.5	14.7	42.6
	8	4	5.8	5.9	48.5
	9	11	15.9	16.2	64.7
	10	5	7.2	7.4	72.1
	11	4	5.8	5.9	77.9

	12	9	13.0	13.2	91.2
	13	2	2.9	2.9	94.1
	14	1	1.4	1.5	95.6
	15	2	2.9	2.9	98.5
	19	1	1.4	1.5	100.0
	Total	68	98.6	100.0	
Missing	999	1	1.4		
Total		69	100.0		

Table 4.12

Verbal Fluency category fluency total scaled

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	1	3	4.3	4.4	4.4
	2	2	2.9	2.9	7.4
	3	2	2.9	2.9	10.3
	4	1	1.4	1.5	11.8
	5	6	8.7	8.8	20.6
	6	4	5.8	5.9	26.5
	7	6	8.7	8.8	35.3
	8	11	15.9	16.2	51.5
	9	3	4.3	4.4	55.9
	10	13	18.8	19.1	75.0
	11	2	2.9	2.9	77.9

Verbal Fluency category fluency total scaled					
	12	7	10.1	10.3	88.2
	13	2	2.9	2.9	91.2
	14	2	2.9	2.9	94.1
	16	1	1.4	1.5	95.6
	18	2	2.9	2.9	98.5
	56	1	1.4	1.5	100.0
	Total	68	98.6	100.0	
Missing	999	1	1.4		
Total		69	100.0		

Table 4.13

Verbal Fluency category switching total correct scaled					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	1	2	2.9	2.9	2.9
	2	3	4.3	4.4	7.4
	3	8	11.6	11.8	19.1
	4	3	4.3	4.4	23.5
	5	2	2.9	2.9	26.5
	6	4	5.8	5.9	32.4
	7	1	1.4	1.5	33.8
	8	6	8.7	8.8	42.6
	9	11	15.9	16.2	58.8
	10	5	7.2	7.4	66.2
	11	11	15.9	16.2	82.4
	12	6	8.7	8.8	91.2

	14	1	1.4	1.5	92.6
	15	2	2.9	2.9	95.6
	16	1	1.4	1.5	97.1
	17	2	2.9	2.9	100.0
	Total	68	98.6	100.0	
Missing	999	1	1.4		
Total		69	100.0		

CPT II.

The CPT II was developed by C. Keith Conners as a visual performance task which evaluates attentional variables in individuals age six or older (IPS, 2005). The response patterns identified by the CPT II provide information on attention, impulsivity, activation/arousal and difficulties in maintaining vigilance (IPS, 2005). The CPT II provides a confidence index value that helps evaluate whether the examinee matches a clinical or non- clinical respondent. In general, values above 50% indicate a closer match to a clinical population, values below 50% indicate a match closer to a nonclinical profile and values at 50% are inconclusive (Conners, 2004). Of the 66 valid profiles, 46 scored above 50%, 7 scored below 5 % and 13 scored at 50% (M = 68.6, SD = 19.28). Using a one-sample z-test, the chance that this sample was drawn from a normal population are less than one in one thousand. This finding confirms that the sample was drawn from a population with lower than average performance on this measure as well. These findings are comparable to the results of other reports in the literature related to attention, impulsivity, activation and arousal being affected in samples of adults with substance use disorders. For example,

as discussed in the literature review above, others have observed impaired attention in adults in substance use treatment (Roselli et e al., 2001). Similar to the D-KEFS results, these results are consistent with defined damage to the frontal lobe and related skill of attention, impulsivity and perseveration.

Table 4.14

CPT II confidence index

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	23.06	1	1.4	1.5	1.5
	28.53	1	1.4	1.5	3.0
	41.2	1	1.4	1.5	4.5
	47.29	1	1.4	1.5	6.1
	49.9	3	4.3	4.5	10.6
	50	13	18.8	19.7	30.3
	53.27	1	1.4	1.5	31.8
	53.43	1	1.4	1.5	33.3

53.77	1	1.4	1.5	34.8
56.52	2	2.9	3.0	37.9
57.47	1	1.4	1.5	39.4
59.29	1	1.4	1.5	40.9
60.57	1	1.4	1.5	42.4
62.22	1	1.4	1.5	43.9
62.85	1	1.4	1.5	45.5
66.2	1	1.4	1.5	47.0
67.82	1	1.4	1.5	48.5
68.1	1	1.4	1.5	50.0
69.47	1	1.4	1.5	51.5
69.62	1	1.4	1.5	53.0
70.76	1	1.4	1.5	54.5
72.04	1	1.4	1.5	56.1
73.31	1	1.4	1.5	57.6
75.56	1	1.4	1.5	59.1
76.68	1	1.4	1.5	60.6
77.46	1	1.4	1.5	62.1
78.19	1	1.4	1.5	63.6
78.71	1	1.4	1.5	65.2
79.48	1	1.4	1.5	66.7
79.66	1	1.4	1.5	68.2
80.48	1	1.4	1.5	69.7
80.61	1	1.4	1.5	71.2
80.67	1	1.4	1.5	72.7
82.02	1	1.4	1.5	74.2
82.67	1	1.4	1.5	75.8
82.7	1	1.4	1.5	77.3
84.28	1	1.4	1.5	78.8
86.3	1	1.4	1.5	80.3
86.64	1	1.4	1.5	81.8
87.08	1	1.4	1.5	83.3
91.1	1	1.4	1.5	84.8

	91.51	1	1.4	1.5	86.4
	93.77	1	1.4	1.5	87.9
	99.9	8	11.6	12.1	100.0
	Total	66	95.7	100.0	
Missing	999	3	4.3		
Total		69	100.0		

Wechsler Test of Adult Reading (WTAR).

We collected data on the WTAR on all participants with the intention of using the information for an estimate of premorbid intelligence. However, since we were only giving the WASI instead of a full WAIS, we were unable to calculate the premorbid functioning. Personal communication with Amy Gabel, PhD, the Director of Client Consultation and Training at Pearson confirmed that there is not data on using the WASI with the WTAR and that the WASI was not intended to replace the WAIS in this situation. (Personal communication, 11-4-2008). Therefore, the WTAR results will not be included.

WASI.

Though many short forms of the Wechsler Scales exist, the WASI was developed to provide a consistent, well normed, brief measure of intelligence (Keith, Lindskog & Smith, 2004). The WASI is available in a four or two subtest format giving the administrator control over the time and depth of the assessment (Keith, Lindskog & Smith, 2004). For this study, we used the two subtest form of the WASI which includes the Vocabulary subtest and the Matrix Reasoning subtest resulting in a Full Scale IQ (FSIQ) score with a 15 minute administration time (Harcourt Assessment, retrieved April 27, 2006). For all subtests, raw scores are converted to T scores with all IQ scores having a mean of 100 and a standard deviation of 15 (Keith

et al., 2004). Of the 62 valid profiles for the WASI, 5 scored in the extremely low IQ range (≤ 69), 9 scored in the borderline range (70-79), 11 scored in the low average range (80-89), 28 in the average range (90-109), 8 in the high average range (110-119) and 1 in the superior range (120-129). The mean for the WASI IQ score was 92.64 with a standard deviation of 15.2. According to the one-sample z-test, samples of 68 with mean less than 97.66 have a less than .05 chance of occurring if the sample was drawn from a population with a mean of 100. The observed sample mean falls below this threshold level, suggesting that the present sample has a significantly lower IQ level. Though the WASI is not an assessment that is specifically associated with the frontal lobe, it does offer additional useful information to incorporate into our overall assessment of the participants executive functioning.

Table 4.15

WASI IQ score

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	60	1	1.4	1.6	1.6
	62	2	2.9	3.2	4.8
	66	1	1.4	1.6	6.5
	69	1	1.4	1.6	8.1
	72	1	1.4	1.6	9.7

WASI IQ score					
	73	3	4.3	4.8	14.5
	75	2	2.9	3.2	17.7
	78	1	1.4	1.6	19.4
	79	2	2.9	3.2	22.6
	80	2	2.9	3.2	25.8
	82	1	1.4	1.6	27.4
	86	2	2.9	3.2	30.6
	88	3	4.3	4.8	35.5
	89	3	4.3	4.8	40.3
	90	2	2.9	3.2	43.5
	91	3	4.3	4.8	48.4
	93	1	1.4	1.6	50.0
	94	1	1.4	1.6	51.6
	96	3	4.3	4.8	56.5
	98	3	4.3	4.8	61.3
	99	3	4.3	4.8	66.1
	101	4	5.8	6.5	72.6
	103	1	1.4	1.6	74.2
	104	2	2.9	3.2	77.4
	105	1	1.4	1.6	79.0
	108	2	2.9	3.2	82.3
	109	2	2.9	3.2	85.5
	110	2	2.9	3.2	88.7
	112	1	1.4	1.6	90.3
	113	2	2.9	3.2	93.5
	115	1	1.4	1.6	95.2
	118	2	2.9	3.2	98.4
	121	1	1.4	1.6	100.0
	Total	62	89.9	100.0	
Missing	999	7	10.1		
Total		69	100.0		

Drop Out and Survival in Treatment

Of the 68 participants followed in this study, 33 stayed in treatment past 90 days (48.5%) and 35 dropped out prior to 90 days (51.5%). Likewise the participant's length of time in treatment from first day of treatment to last day of treatment was a minimum of 13 days and a maximum of 426 with a mean of 120.2 and a standard deviation of 97.03.

Summary of Descriptives

This study utilized data from 68 non-married males with an average age of 45. In our sample 72% had a high school education or beyond, and the majority were African American (66.2%) or Caucasian (25%). Data was collected and analyzed on neuropsychological functioning using subtest of the D-KEFS, WASI IQ and the CPT II. The finding suggests that the sample was drawn from a population with a lower than average level of performance on all measures. There was no correlation between age, education and the reported neuropsychological deficits.

Neuropsychological Deficits Predicting Attrition

To answer our second research question both demographic and psychological variables were considered as potential predictors of treatment attrition (defined in terms of receiving fewer than 90 days of treatment, versus 90 days or more of treatment). The 90 day mark is used by many researchers as the cutoff for treatment retention and is identified as many by an average length of stay (Katz, King, Schwartz, Weintraub, Barksdale, Robinson and Brown, 2005).

In order to identify demographic predictors of treatment attrition, independent groups t-tests were conducted to compare levels of continuous variables, such as age,

between patients who dropped out and those who continued. Chi-squared tests of independence were employed to determine whether dropping out was significantly associated with education, marital status, race, and prior experience of having ADOA treatment. Of these demographic variables, only prior treatment experience was a significant predictor of dropout. As shown in Table 4.16, patients who had no prior treatment experience were more likely to drop out (Chi-squared = 4.664; $df = 1$; $p < .05$).

Table 4.16

Dropout Rates By Previous Treatment History

	Dropout	
	Yes	No
Prior Treatment History	22 (44%)	28 (56%)
No Prior Treatment History	12 (75%)	4 (25%)

Initial efforts to identify such predictors employed Pearson's correlations to examine the linear relationship between neuropsychological measures and length of treatment, as well as independent groups t-tests to test the significance of mean

differences in neuropsychological test scores between participants who dropped out before completing 90 days of treatment, and those who received 90 or more days of treatment. No statistically significant linear relationships were found between neuropsychological measures and these indices of length of treatment.

The absence of significant linear effects might be understandable if the relationships between neuropsychological variables and length of treatment was actually curvilinear. Linear correlations may be quite weak when the underlying relationship between two variables is U-shaped. To detect the presence of curvilinear relationships, scatterplots of data were inspected. The Classification and Regression Trees (CART) program was used to identify cut off points for IQ computationally instead of just visually inspecting the scatterplots and identifying categories (Breiman, Friedman, Stone & Olshen, 1984). The CART software automatically tries out different ways of categorizing the WAIS scores. The cutting point that best predicts the dependent variable is selected. The CART program is regarded as an exploratory data analysis technique (Breiman, Friedman, Stone & Olshen, 1984). The distribution of WASI IQ scores with dropping out, as well as with total days of treatment, appeared to show a curvilinear relationship. As shown in Table 4.17, patients with IQ scores that were low to average were more likely to drop out. Patients with average and above IQ scores, as well as those with borderline to low IQ scores, were less likely to drop out of treatment (Chi-squared = 10.75; df =2 ; $p <$ .01).

Table 4.17

Dropout Rates By WASI IQ

	Dropout	
	Yes	No
WASI Below 77	2 (18.2%)	9 (81.8%)
WASI 77-95	16 (76.2%)	5 (23.8%)
WASI Above 95	13 (43.3%)	17 (56.7%)

Prediction of Attrition and Continuation in Treatment

The main analyses of the present investigation utilized logistic regression and survival analysis to predict attrition and continuation in treatment. Logistic regression was utilized to assess the unique contributions of predictors (i.e., prior treatment, WASI IQ level) on the binary outcome of dropping out before 90 days of treatment or continuing. For the survival analysis, Cox regression was utilized to examine the association between predictors and length of treatment. WASI IQ level was coded in the manner described above in order to examine curvilinear effects of IQ.

Logistic Regression

Both Prior Treatment and WASI IQ level were entered into a logistic regression equation to predict the probability of dropping out of treatment before completing 90 days. The regression analysis was set up so that the medium level of the WASI IQ variable served as a reference category against which the lowest and highest levels were contrasted. As shown in Table 4.18, only WASI IQ, not prior treatment, made a significant unique contribution to the prediction of dropping out. Specifically, patients with low to average IQ scores (77-95) were more likely to drop out than those with borderline to low IQ scores (below 77).

Table 4.18

Logistic Regression Predicting Dropout Rates From Prior Treatment and WASI IQ Level

	Beta	SE	Wald	df	Significance
Prior Treatment	-.971	.752	1.660	1	.196
WASI Level			6.409	2	.041
Low vs.Medium	-2.398	.956	6.295	1	.012
High vs. Medium	-0.979	.682	2.061	1	.376
Constant	.518	.660	0.615	1	.433

The beta weights in logistic regression are conceptually like those in ordinary least squares regression, only now they are predicting the likelihood of dropping out of

treatment. A positive beta means that higher scores on the predictor are associated with a greater likelihood of dropping out. A negative beta means that higher scores on the predictor are associated with a lower likelihood of dropping out. The Wald statistic indicates whether the beta weight is statistically significant. Thus, the results of the logistic regression indicate that subjects with borderline to low IQ scores are significantly less likely to drop out than those with average or above scores. There is a non-significant trend toward lower dropout rates for subjects who have had prior treatment, as well as those who have relatively high IQ scores (i.e.., IQ > 95). While there is no precise formula for computation of R-squared in logistic regression, there are methods for the computation of an analogous measure of effect size, or "pseudo R-squared". The Cox and Snell R-Square for the logistic regression model presented above is .182, while the Nagelkerke R-Square is .243. The overall model accounted for a substantial portion of the variance in the dependent variables.

Survival Analysis/Cox Regression

Both Prior Treatment and WASI IQ Level were entered into a Cox regression equation to predict the survival in treatment (i.e. number of days between the start and end of treatment). As in the preceding analyses, the medium level of the WASI IQ variable (low to average IQ) served as a reference category against which the lowest (borderline to low) and highest (average and above) levels were contrasted. As shown in Table 4.19, one of the WASI IQ contrasts, and not prior treatment, made a significant unique contribution to the prediction of survival. Patients with low to average IQ scores (77-95) had shorter survival times than those with borderline to low IQ scores (below 77).

Table 4.19

Cox Regression Predicting Survival Rates From Prior Treatment and WASI IQ Level

	Beta	SE	Wald	df	Significance
Prior Treatment	-0.324	.315	1.054	1	.305
WASI Level			4.911	2	.086
Low vs. Medium	-0.851	.386	4.853	1	.028
High vs. Medium	-0.383	.311	1.518	1	.218

In the Cox Regression, a negative Beta weight indicates a lower chance of dropping out. The Wald statistic again provides a test of the null hypothesis that the Beta population parameter equals zero. Thus, subjects with borderline to low IQ (below 77) have a significantly lower chance of dropping out across time (i.e., they will remain in treatment for a longer time). There is a non-significant trends toward longer survival among subjects with prior treatment histories and average to high IQ (over 95).

Summary of Logistic Regression and Survival Analysis

The findings of the present study suggest that general intelligence, as assessed by the WASI, predicts dropout and survival in treatment. The effects of IQ are significant even after controlling for, or partialling out, the effects of prior treatment history on dropout rates. Conversely, the effects of prior treatment on dropping out are no longer significant when WASI IQ is entered into the equation. This suggests

that the relationship between prior treatment history and dropping out might be explained in part by differences in WASI IQ. Throughout these analyses, the effects of WASI IQ are curvilinear: patients with low to average IQ are more likely to drop out than those with borderline to low IQ, while those with average or above IQ have an intermediate level of dropping out or persisting in treatment.

Correlations Affecting Attrition and Length of Treatment

In addition to the above statistics, we decided to run further correlations to evaluate whether there were relationships between the NP variables, IQ and prior treatment and whether these relationships affected attrition or length of stay in treatment.

Some correlations were found to exist between the NP variables and IQ which intuitively makes sense and has been found by other researchers (Zinn, Stein & Swartzwelder, 2004). All four of the D-KEFS subtests used (Trail Making Test, Color Word, Verbal Fluency and Tower) had sections that were significantly correlated with IQ. These results can be viewed in table 4.20 below.

Table 4.20

NP and WASI Correlations

NP Measure		Correlation with WASI IQ
TMTvisscRW Trail making test visual scanning raw	Pearson Correlation	-.299
	Sig. (2-tailed)	.018
	N	62
TMTvisscSC Trail making test visual scanning scaled	Pearson Correlation	.297
	Sig. (2-tailed)	.019
	N	62
TMTnumRW trail making test number	Pearson Correlation	-.492

sequence raw		
	Sig. (2-tailed)	.000
	N	62
TMTnumSC trail making test number sequence scaled	Pearson Correlation	.508
	Sig. (2-tailed)	.000
	N	62
TMTletRW trail making test letter sequence raw	Pearson Correlation	-.489
	Sig. (2-tailed)	.000
	N	62
TMTletSC trail making test letter sequence scaled	Pearson Correlation	.542
	Sig. (2-tailed)	.000
	N	62
TMTNLSRAW Trail Making Num/Letter Raw Score	Pearson Correlation	-.667
	Sig. (2-tailed)	.000
	N	60
TMTNLSSC Trail Making Number letter sequencing Scaled score	Pearson Correlation	.672
	Sig. (2-tailed)	.000
	N	60
TMTmsRAW Trails motor speed raw	Pearson Correlation	-.359
	Sig. (2-tailed)	.005
	N	61
TMTmsSC trails motor speed scaled	Pearson Correlation	.344
	Sig. (2-tailed)	.007
	N	61
TMTcmbSS trails combined number + letter sum of scaled scores	Pearson Correlation	.572
	Sig. (2-tailed)	.000
	N	62
TMTcmbCS trails combined number+ letter sequencing composite scaled score	Pearson Correlation	.575
	Sig. (2-tailed)	.000
	N	62
TMTSvVSSSD trails switching vs. visual scanning scaled score dif	Pearson Correlation	.394
	Sig. (2-tailed)	.002
	N	60
TMTSvVSCS trailsswitching vs. visual sanning contrast scaled score	Pearson Correlation	.416
	Sig. (2-tailed)	.001

	N	60
TMTSvNSSSD trails switching vs number sequencing scaled score dif	Pearson Correlation	.192
	Sig. (2-tailed)	.141
	N	60
TMTSvNSCS trails switiching vs. number sequencing contrast sclaed score	Pearson Correlation	.178
	Sig. (2-tailed)	.173
	N	60
TMTSvLSSD Trails switching vs letter sequencing scaled score dif	Pearson Correlation	.150
	Sig. (2-tailed)	.251
	N	60
TMTSvLSCS trailsswitching vs. letter sequencing scaled score contrast scaled score	Pearson Correlation	.146
	Sig. (2-tailed)	.266
	N	60
TMTSvCmbSSD trails switching vs.combined scaled score dif	Pearson Correlation	.103
	Sig. (2-tailed)	.432
	N	60
TMTSvCmbCS trails switching vs. combined contrasst scaled score	Pearson Correlation	.098
	Sig. (2-tailed)	.456
	N	60
TMTSvMSSSD trails switching vs. motor speed scaled score dif	Pearson Correlation	.423
	Sig. (2-tailed)	.001
	N	60
TMTSvMSCS trails switching vs. motor speed contrast scaled score	Pearson Correlation	.416
	Sig. (2-tailed)	.001
	N	60
VFLFtotalRW verbal fluency letter fluency total raw	Pearson Correlation	.579
	Sig. (2-tailed)	.000
	N	62
VFLFtotalSC verbal fluency letter fluency total scaled	Pearson Correlation	.573
	Sig. (2-tailed)	.000
	N	62
VFCFtotalRW verbal fluency category fluency total raw	Pearson Correlation	.601
	Sig. (2-tailed)	.000

	N	62
VFCFtotalSC verbal fluency category fluency total scaled	Pearson Correlation	.171
	Sig. (2-tailed)	.185
	N	62
VFCatSwRW verbal fluency category switching total correct raw	Pearson Correlation	.434
	Sig. (2-tailed)	.000
	N	62
VFCatSwSC verbal fluency category switching total correct scaled	Pearson Correlation	.418
	Sig. (2-tailed)	.001
	N	62
VFtotalSwRW verbal fluency category switching total switching Raw	Pearson Correlation	.494
	Sig. (2-tailed)	.000
	N	62
VFtotalSwSC verbal fluency category switching total switching scaled	Pearson Correlation	.482
	Sig. (2-tailed)	.000
	N	62
VFLFvsCFSSD verbal fluency letter fluency vs category fluency Scaled Score Difference	Pearson Correlation	-.023
	Sig. (2-tailed)	.862
	N	62
VFLFvsCFCSS verbal fluency letter fluency vs category fluency Contrast Scaled Score	Pearson Correlation	-.023
	Sig. (2-tailed)	.862
	N	62
VFCSvsCFSSD verbal fluency category switching vs category fluency Scaled Score Difference	Pearson Correlation	-.139
	Sig. (2-tailed)	.281
	N	62
VFCSvsCFCSS verbal fluency category switching vs category fluency Contrast Scaled Score	Pearson Correlation	-.125
	Sig. (2-tailed)	.333
	N	62
VF1stRW verbal fluency 1st interval total correct raw	Pearson Correlation	.526
	Sig. (2-tailed)	.000
	N	62
VF1stSC verbal fluency 1st interval	Pearson Correlation	.517

total correct scale		
	Sig. (2-tailed)	.000
	N	62
VF2ndRW verbal fluency 2nd interval total correct raw	Pearson Correlation	.611
	Sig. (2-tailed)	.000
	N	62
VF2ndSc verbal fluency 2nd interval total correct scale	Pearson Correlation	.599
	Sig. (2-tailed)	.000
	N	62
VF3rdRW verbal fluency 3rd interval total correct raw	Pearson Correlation	.562
	Sig. (2-tailed)	.000
	N	62
VF3rdSC verbal fluency 3rd interval total correct scale	Pearson Correlation	.549
	Sig. (2-tailed)	.000
	N	62
VF4thRW verbal fluency 4th interval total correct raw	Pearson Correlation	.555
	Sig. (2-tailed)	.000
	N	62
VF4thSC verbal fluency 4th interval total correct scale	Pearson Correlation	.553
	Sig. (2-tailed)	.000
	N	62
VFSLerrRW verbal fluency set loss errors raw	Pearson Correlation	-.186
	Sig. (2-tailed)	.147
	N	62
VFSLerrSC verbal fluency set loss errors scaled	Pearson Correlation	.177
	Sig. (2-tailed)	.168
	N	62
VFREPerrRW verbal fluency repetition errors raw	Pearson Correlation	-.011
	Sig. (2-tailed)	.931
	N	62
VFREPerrSC verbal fluency repetition errors scaled	Pearson Correlation	-.015
	Sig. (2-tailed)	.910
	N	62
VFtotrespRW verbal fluency total responses-correct and incorrect-raw	Pearson Correlation	.545
	Sig. (2-tailed)	.000

	N	62
CWclrnmRW color word color naming raw	Pearson Correlation	-.487
	Sig. (2-tailed)	.000
	N	62
CWclrnmSC color word color naming scaled	Pearson Correlation	.435
	Sig. (2-tailed)	.000
	N	62
CWreadRW color word word reading raw	Pearson Correlation	-.454
	Sig. (2-tailed)	.000
	N	62
CWreadSC color word word reading scaled	Pearson Correlation	.425
	Sig. (2-tailed)	.001
	N	62
CWinhibRW color word inhibition raw	Pearson Correlation	-.555
	Sig. (2-tailed)	.000
	N	62
CWinhibSC color word inhibition scaled	Pearson Correlation	.541
	Sig. (2-tailed)	.000
	N	62
CWinswitRW color word inhibition/switching raw	Pearson Correlation	-.534
	Sig. (2-tailed)	.000
	N	62
CWinswitSC color word inhibition/switching scaled	Pearson Correlation	.505
	Sig. (2-tailed)	.000
	N	62
CWcmbSSS color word combined naming and reading Sum of Scaled Scores	Pearson Correlation	.459
	Sig. (2-tailed)	.000
	N	62
CWcmbCSS color word combined naming and reading Composite Scaled Score	Pearson Correlation	.462
	Sig. (2-tailed)	.000
	N	62
CWErCNRW color word error Color naming raw	Pearson Correlation	-.458
	Sig. (2-tailed)	.000
	N	62

CWErCNSC color word error color naming scaled	Pearson Correlation	.357
	Sig. (2-tailed)	.004
	N	62
CWErWRRW Color word error Word reading Raw	Pearson Correlation	-.338
	Sig. (2-tailed)	.007
	N	62
CWErWRSC Color word error Word reading scaled	Pearson Correlation	.394
	Sig. (2-tailed)	.002
	N	62
CWErInhRW Color word error inhibition raw	Pearson Correlation	-.543
	Sig. (2-tailed)	.000
	N	62
CWErInhSc Color word error inhibition scaled	Pearson Correlation	.553
	Sig. (2-tailed)	.000
	N	62
CWErInSwRw Color Word Error inhibition/Switching raw	Pearson Correlation	-.560
	Sig. (2-tailed)	.000
	N	62
CWErInSwSc Color Word Error Inhibition/Switching Scaled	Pearson Correlation	.592
	Sig. (2-tailed)	.000
	N	62
TTtotalRW Tower Test total achievement score raw	Pearson Correlation	.463
	Sig. (2-tailed)	.000
	N	61
TTtotalSC Tower Test total achievement score scaled	Pearson Correlation	.448
	Sig. (2-tailed)	.000
	N	61
TTrulesRW Tower Test total rule violations raw	Pearson Correlation	-.616
	Sig. (2-tailed)	.000
	N	61
TTrulesPR Tower Test total rule violations cumulative percentile rank	Pearson Correlation	.577
	Sig. (2-tailed)	.000
	N	61
TT1stmvtmR Tower test mean 1st move time ratio score	Pearson Correlation	.026

	Sig. (2-tailed)	.845
	N	61
TT1stmvtmS Tower test mean 1st move time Scaled score	Pearson Correlation	-.011
	Sig. (2-tailed)	.935
	N	61
TTtmprmvR Tower test time per move ratio score	Pearson Correlation	-.219
	Sig. (2-tailed)	.090
	N	61
TTtmprmvS Tower test time per move scaled score	Pearson Correlation	.180
	Sig. (2-tailed)	.165
	N	61
TTmvaccR Tower test move accuracy ratio score	Pearson Correlation	-.239
	Sig. (2-tailed)	.064
	N	61
TTmvaccSc Tower test move accuracy scaled score	Pearson Correlation	.226
	Sig. (2-tailed)	.080
	N	61
TTruleitemR Tower test rule violations per item ratio score	Pearson Correlation	-.651
	Sig. (2-tailed)	.000
	N	61
TTruleitemS Tower test rule violations per item scaled score	Pearson Correlation	.645
	Sig. (2-tailed)	.000
	N	61
CPTconfind CPT II confidence index	Pearson Correlation	-.178
	Sig. (2-tailed)	.170
	N	61

Even more interesting, is that the NP variables are significantly correlated with attrition and length of stay in treatment for cases that have below median IQ for this sample (IQ < 94). Three of the four D-KEFS subtests used correlated significantly on cases with IQ below 94 (Trail Making, Verbal Fluency and Tower Test). The results of this correlation can be viewed in table 4.21 below.

Table 4.21

Correlations (IQ less than 94)

(IQ less than 94)		Drop out	daysintx number of days from 1st day of treatment to last day
TMTvisscRW Trail making test visual scanning raw	Pearson Correlation	-.224	.055
	Sig. (2-tailed)	.226	.769
	N	31	31
TMTvisscSC Trail making test visual scanning scaled	Pearson Correlation	.215	-.022
	Sig. (2-tailed)	.245	.906
	N	31	31
TMTnumRW trail making test number sequence raw	Pearson Correlation	-.494	.240
	Sig. (2-tailed)	.005	.193
	N	31	31
TMTnumSC trail making test number sequence scaled	Pearson Correlation	.446	-.259
	Sig. (2-tailed)	.012	.159
	N	31	31
TMTletRW trail making test letter sequence raw	Pearson Correlation	-.259	.119
	Sig. (2-tailed)	.159	.524
	N	31	31
TMTletSC trail making test letter sequence scaled	Pearson Correlation	.147	-.051
	Sig. (2-tailed)	.429	.786

	N	31	31
TMTNLSRAW Trail Making Num/Letter Raw Score	Pearson Correlation	-.257	.256
	Sig. (2-tailed)	.178	.180
	N	29	29
TMTNLSSC Trail Making Number letter sequencing Scaled score	Pearson Correlation	.269	-.231
	Sig. (2-tailed)	.159	.228
	N	29	29
TMTmsRAW Trails motor speed raw	Pearson Correlation	.005	.106
	Sig. (2-tailed)	.979	.578
	N	30	30
TMTmsSC trails motor speed scaled	Pearson Correlation	-.035	-.079
	Sig. (2-tailed)	.854	.678
	N	30	30
TMTcmbSS trails combined number + letter sum of scaled scores	Pearson Correlation	.338	-.177
	Sig. (2-tailed)	.063	.340
	N	31	31
TMTcmbCS trails combined number+ letter sequencing composite scaled score	Pearson Correlation	.325	-.146
	Sig. (2-tailed)	.074	.432
	N	31	31
TMTSvVSSSD trails switching vs. visual scanning scaled score dif	Pearson Correlation	.093	-.276
	Sig. (2-tailed)	.631	.148
	N	29	29
TMTSvVSCS trailsswitching vs. visual sanning contrast scaled score	Pearson Correlation	.140	-.320
	Sig. (2-tailed)	.468	.090
	N	29	29
TMTSvNSSSD trails switching vs number sequencing scaled score dif	Pearson Correlation	-.246	.114
	Sig. (2-tailed)	.198	.556
	N	29	29
TMTSvNSCS trails switiching vs. number sequencing contrast sclaed score	Pearson Correlation	-.253	.124

	Sig. (2-tailed)	.185	.522
	N	29	29
TMTSvLSSD Trails switching vs letter sequencing scaled score dif	Pearson Correlation	.173	-.178
	Sig. (2-tailed)	.368	.355
	N	29	29
TMTSvLSCS trailsswitching vs. letter sequencing scaled score contrast scaled score	Pearson Correlation	.167	-.163
	Sig. (2-tailed)	.387	.397
	N	29	29
TMTSvCmbSSD trails switching vs.combined scaled score dif	Pearson Correlation	-.046	-.062
	Sig. (2-tailed)	.813	.749
	N	29	29
TMTSvCmbCS trails switching vs. combined contrasst scaled score	Pearson Correlation	-.059	-.042
	Sig. (2-tailed)	.761	.827
	N	29	29
TMTSvMSSSD trails switching vs. motor speed scaled score dif	Pearson Correlation	.278	-.162
	Sig. (2-tailed)	.144	.401
	N	29	29
TMTSvMSCS trails switching vs. motor speed contrast scaled score	Pearson Correlation	.305	-.184
	Sig. (2-tailed)	.108	.338
	N	29	29
VFLFtotalRW verbal fluency letter fluency total raw	Pearson Correlation	.354	-.309
	Sig. (2-tailed)	.051	.090
	N	31	31
VFLFtotalSC verbal fluency letter fluency total scaled	Pearson Correlation	.340	-.317
	Sig. (2-tailed)	.062	.082
	N	31	31
VFCFtotalRW verbal fluency category fluency total raw	Pearson Correlation	.305	-.186
	Sig. (2-tailed)	.095	.315
	N	31	31
VFCFtotalSC verbal fluency category fluency total scaled	Pearson Correlation	-.096	-.041
	Sig. (2-tailed)	.609	.829
	N	31	31
VFCatSwRW verbal fluency	Pearson Correlation	.134	-.002

category switching total correct raw			
	Sig. (2-tailed)	.473	.990
	N	31	31
VFCatSwSC verbal fluency category switching total correct scaled	Pearson Correlation	.099	.010
	Sig. (2-tailed)	.595	.956
	N	31	31
VFtotalSwRW verbal fluency category switching total switching Raw	Pearson Correlation	-.001	.067
	Sig. (2-tailed)	.998	.719
	N	31	31
VFtotalSwSC verbal fluency category switching total switching scaled	Pearson Correlation	-.013	.068
	Sig. (2-tailed)	.947	.717
	N	31	31
VFLFvsCFSSD verbal fluency letter fluency vs category fluency Scaled Score Difference	Pearson Correlation	.047	-.088
	Sig. (2-tailed)	.803	.639
	N	31	31
VFLFvsCFCSS verbal fluency letter fluency vs category fluency Contrast Scaled Score	Pearson Correlation	.047	-.088
	Sig. (2-tailed)	.803	.639
	N	31	31
VFCSvsCFSSD verbal fluency category switching vs category fluency Scaled Score Difference	Pearson Correlation	-.181	.251
	Sig. (2-tailed)	.331	.173
	N	31	31
VFCSvsCFCSS verbal fluency category switching vs category fluency Contrast Scaled Score	Pearson Correlation	-.181	.251
	Sig. (2-tailed)	.331	.173
	N	31	31
VF1stRW verbal fluency 1st interval total correct raw	Pearson Correlation	.308	-.207
	Sig. (2-tailed)	.092	.263
	N	31	31
VF1stSC verbal fluency 1st interval total correct scale	Pearson Correlation	.278	-.209
	Sig. (2-tailed)	.130	.260

	N	31	31
VF2ndRW verbal fluency 2nd interval total correct raw	Pearson Correlation	.401	-.294
	Sig. (2-tailed)	.025	.108
	N	31	31
VF2ndSc verbal fluency 2nd interval total correct scale	Pearson Correlation	.373	-.313
	Sig. (2-tailed)	.039	.087
	N	31	31
VF3rdRW verbal fluency 3rd interval total correct raw	Pearson Correlation	.223	-.180
	Sig. (2-tailed)	.229	.332
	N	31	31
VF3rdSC verbal fluency 3rd interval total correct scale	Pearson Correlation	.155	-.158
	Sig. (2-tailed)	.404	.395
	N	31	31
VF4thRW verbal fluency 4th interval total correct raw	Pearson Correlation	.232	-.146
	Sig. (2-tailed)	.210	.433
	N	31	31
VF4thSC verbal fluency 4th interval total correct scale	Pearson Correlation	.231	-.155
	Sig. (2-tailed)	.212	.405
	N	31	31
VFSLerrRW verbal fluency set loss errors raw	Pearson Correlation	.216	-.120
	Sig. (2-tailed)	.244	.519
	N	31	31
VFSLerrSC verbal fluency set loss errors scaled	Pearson Correlation	-.214	.135
	Sig. (2-tailed)	.248	.468
	N	31	31
VFREPerrRW verbal fluency repetition errors raw	Pearson Correlation	-.062	.278
	Sig. (2-tailed)	.741	.130
	N	31	31
VFREPerrSC verbal fluency repetition errors scaled	Pearson Correlation	-.061	-.162
	Sig. (2-tailed)	.743	.384
	N	31	31
VFtotrespRW verbal fluency total responses-correct and incorrect-raw	Pearson Correlation	.339	-.205
	Sig. (2-tailed)	.063	.268
	N	31	31

CWclrnmRW color word color naming raw	Pearson Correlation	-.325	.183
	Sig. (2-tailed)	.075	.324
	N	31	31
CWclrnmSC color word color naming scaled	Pearson Correlation	.225	-.164
	Sig. (2-tailed)	.224	.378
	N	31	31
CWreadRW color word word reading raw	Pearson Correlation	-.052	.030
	Sig. (2-tailed)	.783	.874
	N	31	31
CWreadSC color word word reading scaled	Pearson Correlation	.124	-.045
	Sig. (2-tailed)	.505	.811
	N	31	31
CWinhibRW color word inhibition raw	Pearson Correlation	-.180	.220
	Sig. (2-tailed)	.333	.233
	N	31	31
CWinhibSC color word inhibition scaled	Pearson Correlation	.126	-.209
	Sig. (2-tailed)	.500	.260
	N	31	31
CWinswitRW color word inhibition/switching raw	Pearson Correlation	.091	-.020
	Sig. (2-tailed)	.626	.916
	N	31	31
CWinswitSC color word inhibition/switching scaled	Pearson Correlation	-.068	.005
	Sig. (2-tailed)	.715	.977
	N	31	31
CWcmbSSS color word combined naming and reading Sum of Scaled Scores	Pearson Correlation	.186	-.112
	Sig. (2-tailed)	.315	.549
	N	31	31
CWcmbCSS color word combined naming and reading Composite Scaled Score	Pearson Correlation	.166	-.096
	Sig. (2-tailed)	.372	.607
	N	31	31
CWErCNRW color word error Color naming raw	Pearson Correlation	-.009	-.073
	Sig. (2-tailed)	.961	.697
	N	31	31

CWErCNSC color word error color naming scaled	Pearson Correlation	.182	-.076
	Sig. (2-tailed)	.326	.684
	N	31	31
CWErWRRW Color word error Word reading Raw	Pearson Correlation	-.191	.004
	Sig. (2-tailed)	.304	.984
	N	31	31
CWErWRSC Color word error Word reading scaled	Pearson Correlation	.275	-.095
	Sig. (2-tailed)	.134	.612
	N	31	31
CWErInhRW Color word error inhibition raw	Pearson Correlation	-.167	.073
	Sig. (2-tailed)	.369	.697
	N	31	31
CWErInhSc Color word error inhibition scaled	Pearson Correlation	.129	-.164
	Sig. (2-tailed)	.491	.379
	N	31	31
CWErInSwRw Color Word Error inhibition/Switching raw	Pearson Correlation	-.130	-.016
	Sig. (2-tailed)	.487	.934
	N	31	31
CWErInSwSc Color Word Error Inhibition/Switching Scaled	Pearson Correlation	.144	-.040
	Sig. (2-tailed)	.439	.831
	N	31	31
TTtotalRW Tower Test total achievement score raw	Pearson Correlation	.300	-.120
	Sig. (2-tailed)	.108	.529
	N	30	30
TTtotalSC Tower Test total achievement score scaled	Pearson Correlation	.293	-.112
	Sig. (2-tailed)	.116	.554
	N	30	30
TTrulesRW Tower Test total rule violations raw	Pearson Correlation	-.495	.453
	Sig. (2-tailed)	.005	.012
	N	30	30
TTrulesPR Tower Test total rule violations cumulative percentile rank	Pearson Correlation	.433	-.353
	Sig. (2-tailed)	.017	.055

	N	30	30
TT1stmvtmR Tower test mean 1st move time ratio score	Pearson Correlation	.041	.015
	Sig. (2-tailed)	.828	.939
	N	30	30
TT1stmvtmS Tower test mean 1st move time Scaled score	Pearson Correlation	-.039	-.040
	Sig. (2-tailed)	.837	.835
	N	30	30
TTtmprmvR Tower test time per move ratio score	Pearson Correlation	.091	.006
	Sig. (2-tailed)	.634	.975
	N	30	30
TTtmprmvS Tower test time per move scaled score	Pearson Correlation	-.117	.020
	Sig. (2-tailed)	.537	.917
	N	30	30
TTmvaccR Tower test move accuracy ratio score	Pearson Correlation	-.137	.135
	Sig. (2-tailed)	.471	.476
	N	30	30
TTmvaccSc Tower test move accuracy scaled score	Pearson Correlation	.145	-.132
	Sig. (2-tailed)	.444	.487
	N	30	30
TTruleitemR Tower test rule violations per item ratio score	Pearson Correlation	-.533	.446
	Sig. (2-tailed)	.002	.014
	N	30	30
TTruleitemS Tower test rule violations per item scaled score	Pearson Correlation	.486	-.419
	Sig. (2-tailed)	.006	.021
	N	30	30
CPTconfind CPT II confidence index	Pearson Correlation	-.335	.259
	Sig. (2-tailed)	.070	.166
	N	30	30

With IQ equal to or greater than 94, none of the NP variables were statistically significantly correlated with attrition and survival in treatment.

In addition to the correlation with IQ below 94, some of the NP variables are correlated significantly with drop out and length of stay in treatment for cases that had no prior AODA treatment. It appears that the NP variables are more important in cases that have had no prior treatment than in cases that had prior treatment. Again, caution must be taked due to the small sample sizes. Two of the four D-KEFS subtests used correlated significantly in cases with no prior treatment (Verbal Fluency and Tower Test). The results can be reviewed in table 4.22.

Table 4.22

Correlations (No Prior AODA Treatment)

(No Prior AODA Treatment)		Drop out	daysintx number of days from 1st day of treatment to last day
TMTvisscRW Trail making test visual scanning raw	Pearson Correlation	.235	-.225
	Sig. (2-tailed)	.381	.402
	N	16	16
TMTvisscSC Trail making test visual scanning scaled	Pearson Correlation	-.225	.202
	Sig. (2-tailed)	.402	.453
	N	16	16
TMTnumRW trail making test number sequence raw	Pearson Correlation	-.288	.097
	Sig. (2-tailed)	.279	.722
	N	16	16
TMTnumSC trail making test number sequence scaled	Pearson Correlation	.346	-.139
	Sig. (2-tailed)	.189	.609
	N	16	16

TMTletRW trail making test letter sequence raw	Pearson Correlation	.199	-.223
	Sig. (2-tailed)	.459	.405
	N	16	16
TMTletSC trail making test letter sequence scaled	Pearson Correlation	-.179	.205
	Sig. (2-tailed)	.507	.446
	N	16	16
TMTNLSRAW Trail Making Num/Letter Raw Score	Pearson Correlation	-.106	.136
	Sig. (2-tailed)	.696	.617
	N	16	16
TMTNLSSC Trail Making Number letter sequencing Scaled score	Pearson Correlation	.206	-.252
	Sig. (2-tailed)	.444	.346
	N	16	16
TMTmsRAW Trails motor speed raw	Pearson Correlation	-.079	.163
	Sig. (2-tailed)	.771	.546
	N	16	16
TMTmsSC trails motor speed scaled	Pearson Correlation	.073	-.166
	Sig. (2-tailed)	.789	.539
	N	16	16
TMTcmbSS trails combined number + letter sum of scaled scores	Pearson Correlation	.090	.040
	Sig. (2-tailed)	.740	.883
	N	16	16
TMTcmbCS trails combined number+ letter sequencing composite scaled score	Pearson Correlation	.064	.081
	Sig. (2-tailed)	.814	.765
	N	16	16
TMTSvVSSSD trails switching vs. visual scanning scaled score dif	Pearson Correlation	.413	-.431
	Sig. (2-tailed)	.112	.096
	N	16	16
TMTSvVSCS trailsswitching vs. visual sanning contrast scaled score	Pearson Correlation	.409	-.431
	Sig. (2-tailed)	.115	.096
	N	16	16
TMTSvNSSSD trails switching	Pearson Correlation	-.146	-.117

vs number sequencing scaled score dif			
	Sig. (2-tailed)	.590	.666
	N	16	16
TMTSvNSCS trails switiching vs. number sequencing contrast sclaed score	Pearson Correlation	-.163	-.106
	Sig. (2-tailed)	.546	.696
	N	16	16
TMTSvLSSD Trails switching vs letter sequencing scaled score dif	Pearson Correlation	.342	-.405
	Sig. (2-tailed)	.195	.119
	N	16	16
TMTSvLSCS trailsswitching vs. letter sequencing scaled score contrast scaled score	Pearson Correlation	.328	-.384
	Sig. (2-tailed)	.216	.142
	N	16	16
TMTSvCmbSSD trails switching vs.combined scaled score dif	Pearson Correlation	.135	-.320
	Sig. (2-tailed)	.619	.228
	N	16	16
TMTSvCmbCS trails switching vs. combined contrasst scaled score	Pearson Correlation	.109	-.293
	Sig. (2-tailed)	.688	.272
	N	16	16
TMTSvMSSSD trails switching vs. motor speed scaled score dif	Pearson Correlation	.146	-.108
	Sig. (2-tailed)	.589	.692
	N	16	16
TMTSvMSCS trails switching vs. motor speed contrast scaled score	Pearson Correlation	.146	-.108
	Sig. (2-tailed)	.589	.692
	N	16	16
VFLFtotalRW verbal fluency letter fluency total raw	Pearson Correlation	-.172	.040
	Sig. (2-tailed)	.525	.882
	N	16	16
VFLFtotalSC verbal fluency letter fluency total scaled	Pearson Correlation	-.187	.064
	Sig. (2-tailed)	.487	.813
	N	16	16
VFCFtotalRW verbal fluency category fluency total raw	Pearson Correlation	.112	-.110

	Sig. (2-tailed)	.679	.686
	N	16	16
VFCFtotalSC verbal fluency category fluency total scaled	Pearson Correlation	.163	-.174
	Sig. (2-tailed)	.547	.518
	N	16	16
VFCatSwRW verbal fluency category switching total correct raw	Pearson Correlation	-.065	-.017
	Sig. (2-tailed)	.810	.949
	N	16	16
VFCatSwSC verbal fluency category switching total correct scaled	Pearson Correlation	-.047	-.046
	Sig. (2-tailed)	.861	.867
	N	16	16
VFtotalSwRW verbal fluency category switching total switching Raw	Pearson Correlation	-.115	-.026
	Sig. (2-tailed)	.671	.925
	N	16	16
VFtotalSwSC verbal fluency category switching total switching scaled	Pearson Correlation	-.105	-.038
	Sig. (2-tailed)	.698	.889
	N	16	16
VFLFvsCFSSD verbal fluency letter fluency vs category fluency Scaled Score Difference	Pearson Correlation	-.323	.215
	Sig. (2-tailed)	.223	.424
	N	16	16
VFLFvsCFCSS verbal fluency letter fluency vs category fluency Contrast Scaled Score	Pearson Correlation	-.323	.215
	Sig. (2-tailed)	.223	.424
	N	16	16
VFCSvsCFSSD verbal fluency category switching vs category fluency Scaled Score Difference	Pearson Correlation	-.185	.088
	Sig. (2-tailed)	.493	.747
	N	16	16
VFCSvsCFCSS verbal fluency category switching vs category fluency Contrast Scaled Score	Pearson Correlation	-.185	.088
	Sig. (2-tailed)	.493	.747
	N	16	16

VF1stRW verbal fluency 1st interval total correct raw	Pearson Correlation	.238	-.267
	Sig. (2-tailed)	.375	.318
	N	16	16
VF1stSC verbal fluency 1st interval total correct scale	Pearson Correlation	.259	-.298
	Sig. (2-tailed)	.333	.262
	N	16	16
VF2ndRW verbal fluency 2nd interval total correct raw	Pearson Correlation	.156	-.236
	Sig. (2-tailed)	.565	.378
	N	16	16
VF2ndSc verbal fluency 2nd interval total correct scale	Pearson Correlation	.168	-.274
	Sig. (2-tailed)	.533	.305
	N	16	16
VF3rdRW verbal fluency 3rd interval total correct raw	Pearson Correlation	-.462	.357
	Sig. (2-tailed)	.071	.175
	N	16	16
VF3rdSC verbal fluency 3rd interval total correct scale	Pearson Correlation	-.593	.464
	Sig. (2-tailed)	.015	.070
	N	16	16
VF4thRW verbal fluency 4th interval total correct raw	Pearson Correlation	-.403	.287
	Sig. (2-tailed)	.121	.281
	N	16	16
VF4thSC verbal fluency 4th interval total correct scale	Pearson Correlation	-.410	.286
	Sig. (2-tailed)	.115	.283
	N	16	16
VFSLerrRW verbal fluency set loss errors raw	Pearson Correlation	-.133	.256
	Sig. (2-tailed)	.623	.338
	N	16	16
VFSLerrSC verbal fluency set loss errors scaled	Pearson Correlation	.182	-.289
	Sig. (2-tailed)	.501	.277
	N	16	16
VFREPerrRW verbal fluency repetition errors raw	Pearson Correlation	.217	-.257
	Sig. (2-tailed)	.419	.336
	N	16	16
VFREPerrSC verbal fluency repetition errors scaled	Pearson Correlation	-.238	.263

	Sig. (2-tailed)	.374	.325
	N	16	16
VFtotrespRW verbal fluency total responses-correct and incorrect-raw	Pearson Correlation	-.041	-.051
	Sig. (2-tailed)	.881	.851
	N	16	16
CWclrnmRW color word color naming raw	Pearson Correlation	-.374	.441
	Sig. (2-tailed)	.153	.087
	N	16	16
CWclrnmSC color word color naming scaled	Pearson Correlation	.406	-.472
	Sig. (2-tailed)	.119	.065
	N	16	16
CWreadRW color word word reading raw	Pearson Correlation	-.011	.088
	Sig. (2-tailed)	.969	.746
	N	16	16
CWreadSC color word word reading scaled	Pearson Correlation	.123	-.152
	Sig. (2-tailed)	.649	.574
	N	16	16
CWinhibRW color word inhibition raw	Pearson Correlation	-.433	.512
	Sig. (2-tailed)	.094	.043
	N	16	16
CWinhibSC color word inhibition scaled	Pearson Correlation	.372	-.466
	Sig. (2-tailed)	.156	.069
	N	16	16
CWinswitRW color word inhibition/switching raw	Pearson Correlation	-.276	.440
	Sig. (2-tailed)	.302	.088
	N	16	16
CWinswitSC color word inhibition/switching scaled	Pearson Correlation	.272	-.461
	Sig. (2-tailed)	.308	.072
	N	16	16
CWcmbSSS color word combined naming and reading Sum of Scaled Scores	Pearson Correlation	.278	-.329
	Sig. (2-tailed)	.296	.213
	N	16	16
CWcmbCSS color word combined naming and reading	Pearson Correlation	.242	-.298

Composite Scaled Score			
	Sig. (2-tailed)	.367	.261
	N	16	16
CWErCNRW color word error Color naming raw	Pearson Correlation	.148	-.147
	Sig. (2-tailed)	.584	.588
	N	16	16
CWErCNSC color word error color naming scaled	Pearson Correlation	-.137	.144
	Sig. (2-tailed)	.614	.594
	N	16	16
CWErWRRW Color word error Word reading Raw	Pearson Correlation	.000	-.144
	Sig. (2-tailed)	1.000	.594
	N	16	16
CWErWRSC Color word error Word reading scaled	Pearson Correlation	.197	.083
	Sig. (2-tailed)	.464	.760
	N	16	16
CWErInhRW Color word error inhibition raw	Pearson Correlation	-.238	.296
	Sig. (2-tailed)	.374	.266
	N	16	16
CWErInhSc Color word error inhibition scaled	Pearson Correlation	.257	-.344
	Sig. (2-tailed)	.336	.193
	N	16	16
CWErInSwRw Color Word Error inhibition/Switching raw	Pearson Correlation	.044	.096
	Sig. (2-tailed)	.872	.724
	N	16	16
CWErInSwSc Color Word Error Inhibition/Switching Scaled	Pearson Correlation	-.041	-.100
	Sig. (2-tailed)	.881	.713
	N	16	16
TTtotalRW Tower Test total achievement score raw	Pearson Correlation	.644	-.506
	Sig. (2-tailed)	.007	.046
	N	16	16
TTtotalSC Tower Test total achievement score scaled	Pearson Correlation	.625	-.478
	Sig. (2-tailed)	.010	.061
	N	16	16
TTrulesRW Tower Test total rule violations raw	Pearson Correlation	-.756	.669
	Sig. (2-tailed)	.001	.005

	N	16	16
TTrulesPR Tower Test total rule violations cumulative percentile rank	Pearson Correlation	.572	-.553
	Sig. (2-tailed)	.020	.026
	N	16	16
TT1stmvtmR Tower test mean 1st move time ratio score	Pearson Correlation	-.197	.220
	Sig. (2-tailed)	.465	.414
	N	16	16
TT1stmvtmS Tower test mean 1st move time Scaled score	Pearson Correlation	.277	-.327
	Sig. (2-tailed)	.298	.217
	N	16	16
TTtmprmvR Tower test time per move ratio score	Pearson Correlation	-.468	.506
	Sig. (2-tailed)	.068	.045
	N	16	16
TTtmprmvS Tower test time per move scaled score	Pearson Correlation	.454	-.523
	Sig. (2-tailed)	.077	.038
	N	16	16
TTmvaccR Tower test move accuracy ratio score	Pearson Correlation	-.113	.132
	Sig. (2-tailed)	.677	.625
	N	16	16
TTmvaccSc Tower test move accuracy scaled score	Pearson Correlation	.084	-.091
	Sig. (2-tailed)	.756	.737
	N	16	16
TTruleitemR Tower test rule violations per item ratio score	Pearson Correlation	-.747	.671
	Sig. (2-tailed)	.001	.004
	N	16	16
TTruleitemS Tower test rule violations per item scaled score	Pearson Correlation	.701	-.661
	Sig. (2-tailed)	.003	.005
	N	16	16
CPTconfind CPT II confidence index	Pearson Correlation	-.168	.254
	Sig. (2-tailed)	.534	.343
	N	16	16

Summary of Correlations

Most importantly, the correlations show that some of the NP variables are correlated significantly with attrition and survival in treatment for cases that have below median (94) IQ. Furthermore, some of the NP variables are correlated significantly with drop out and survival in treatment in cases that had no prior treatment. The NP variables that show statistically significant correlations include Trail Making, Verbal Fluency and Tower test for cases with below median IQ and Verbal Fluency and Tower test for no prior AODA treatment. The Color Word Subtest did correlate with IQ but not when looking at the relationship with drop out or length of stay in treatment. The CPT II test showed no statistically significant correlation. These results will be discussed in further detail in the discussion section.

Effect Size

Finally, as previously mentioned, no relative effect sizes could be found to be used to determine sample size for this study. Therefore, effect sizes were calculated and reported on as another valuable outcome of this study. Information on effect sizes is valuable as it can help determine which variables look most promising as possible predictors of attrition and to provide information on how many more cases one would have to add to the sample size to obtain sufficient statistical power. Effect sizes were computed as the square of the correlation between each NP measure and each outcome. As can be seen in Table 4.23, the effect sizes overall are fairly small. The largest effect size is for the variable Tower Test Rule Violations Per Item Ration Score which has an effect size close to 6% (.059). Again, caution must be taken with interpreting these effect sizes due to small sample size. Using the G-Power software

package (Erdfelder, Faul & Buchner, 1996) power and sample size were calculated. In order to attain statistical power of .80 (meaning there is an 80% chance of rejecting the null hypothesis) we would need a sample size of 98 (Erdfelder, Faul & Buchner, 1996). To obtain a power of .95, we would need a sample size of 170 (Erdfelder, Faul & Buchner, 19696).

Table 4.23

Effect Sizes for NP Measures on Length of Treatment and Dropout.

	Correlation Length of treatment-days	Effect Size Length of treatment -days	Correlation Dropout	Effect Size Dropout
Trail making test visual scanning raw	-0.028	0.000784	0.064	0.004096
Trail making test visual scanning scaled	0.025	0.000625	-0.046	0.002116
trail making test number sequence raw	0.133	0.017689	-0.23	0.0529
trail making test number sequence scaled	-0.128	0.016384	0.172	0.029584
trail making test letter sequence raw	0.043	0.001849	-0.082	0.006724
trail making test letter sequence scaled	-0.01	0.0001	-0.007	0.000049
Trail Making Num/Letter Raw Score	0.032	0.001024	-0.017	0.000289
Trail Making Number letter sequencing Scaled score	-0.014	0.000196	0.011	0.000121
Trails motor speed raw	0.155	0.024025	-0.065	0.004225
trails motor speed scaled	-0.147	0.021609	0.042	0.001764
trails combined number + letter sum of scaled scores	-0.075	0.005625	0.09	0.0081
trails combined number+ letter sequencing composite scaled score	-0.058	0.003364	0.083	0.006889
trails switching vs. visual scanning scaled score dif	-0.037	0.001369	0.057	0.003249
trailsswitching vs. visual sanning contrast scaled score	-0.039	0.001521	0.062	0.003844
trails switching vs number sequencing scaled score dif	0.159	0.025281	-0.183	0.033489
trails switiching vs. number sequencing contrast sclaed score	0.163	0.026569	-0.184	0.033856
Trails switching vs letter sequencing scaled score dif	0.002	0.000004	0.048	0.002304
trailsswitching vs. letter sequencing scaled score contrast scaled score	0.01	0.0001	0.044	0.001936
trails switching vs.combined scaled score dif	0.075	0.005625	-0.075	0.005625
trails switching vs. combined contrasst scaled score	0.084	0.007056	-0.081	0.006561
trails switching vs. motor speed scaled score dif	0.113	0.012769	-0.036	0.001296
trails switching vs. motor speed contrast scaled score	0.103	0.010609	-0.024	0.000576
verbal fluency letter fluency total raw	-0.065	0.004225	0.095	0.009025
verbal fluency letter fluency total scaled	-0.043	0.001849	0.067	0.004489
verbal fluency category fluency total raw	-0.109	0.011881	0.149	0.022201

verbal fluency category fluency total scaled	-0.055	0.003025	-0.036	0.001296
verbal fluency category switching total correct raw	-0.038	0.001444	0.024	0.000576
verbal fluency category switching total correct scaled	-0.045	0.002025	0.023	0.000529
verbal fluency category switching total switching Raw	-0.03	0.0009	-0.06	0.0036
verbal fluency category switching total switching scaled	-0.032	0.001024	-0.066	0.004356
verbal fluency letter fluency vs category fluency Scaled Score Difference	0.1	0.01	-0.089	0.007921
verbal fluency letter fluency vs category fluency Contrast Scaled Score	0.1	0.01	-0.089	0.007921
verbal fluency category switching vs category fluency Scaled Score Difference	0.083	0.006889	-0.123	0.015129
verbal fluency category switching vs category fluency Contrast Scaled Score	0.084	0.007056	-0.136	0.018496
verbal fluency 1st interval total correct raw	-0.04	0.0016	0.101	0.010201
verbal fluency 1st interval total correct scale	-0.035	0.001225	0.091	0.008281
verbal fluency 2nd interval total correct raw	-0.154	0.023716	0.142	0.020164
verbal fluency 2nd interval total correct scale	-0.153	0.023409	0.124	0.015376
verbal fluency 3rd interval total correct raw	-0.061	0.003721	0.082	0.006724
verbal fluency 3rd interval total correct scale	-0.049	0.002401	0.054	0.002916
verbal fluency 4th interval total correct raw	-0.067	0.004489	0.087	0.007569
verbal fluency 4th interval total correct scale	-0.069	0.004761	0.092	0.008464
verbal fluency set loss errors raw	0.014	0.000196	0.051	0.002601
verbal fluency set loss errors scaled	-0.03	0.0009	-0.021	0.000441
verbal fluency repetition errors raw	0.207	0.042849	-0.077	0.005929
verbal fluency repetition errors scaled	-0.138	0.019044	0.02	0.0004
verbal fluency total responses-correct and incorrect-raw	-0.049	0.002401	0.141	0.019881
color word color naming raw	0.161	0.025921	-0.193	0.037249
color word color naming scaled	-0.175	0.030625	0.144	0.020736
color word word reading raw	0.081	0.006561	-0.056	0.003136
color word word reading scaled	-0.121	0.014641	0.11	0.0121
color word inhibition raw	0.157	0.024649	-0.016	0.000256
color word inhibition scaled	-0.171	0.029241	0.01	0.0001
color word inhibition/switching raw	0.067	0.004489	0.075	0.005625
color word inhibition/switching scaled	-0.101	0.010201	-0.047	0.002209
color word combined naming and reading Sum of Scaled Scores	-0.158	0.024964	0.136	0.018496
color word combined naming and reading Composite Scaled Score	-0.152	0.023104	0.129	0.016641

color word error Color naming raw	-0.086	0.007396	0.055	0.003025
color word error color naming scaled	0.034	0.001156	-0.027	0.000729
Color word error Word reading Raw	-0.02	0.0004	-0.109	0.011881
Color word error Word reading scaled	-0.001	0.000001	0.126	0.015876
Color word error inhibition raw	0.076	0.005776	-0.103	0.010609
Color word error inhibition scaled	-0.127	0.016129	0.065	0.004225
Color Word Error inhibition/Switching raw	-0.053	0.002809	-0.008	0.000064
Color Word Error Inhibition/Switching Scaled	0.001	0.000001	0.027	0.000729
Tower Test total achievement score raw	-0.058	0.003364	0.187	0.034969
Tower Test total achievement score scaled	-0.052	0.002704	0.183	0.033489
Tower Test total rule violations raw	0.232	0.053824	-0.212	0.044944
Tower Test total rule violations cumulative percentile rank	-0.135	0.018225	0.095	0.009025
Tower test mean 1st move time ratio score	0.103	0.010609	-0.07	0.0049
Tower test mean 1st move time Scaled score	-0.074	0.005476	0.045	0.002025
Tower test time per move ratio score	0.175	0.030625	-0.115	0.013225
Tower test time per move scaled score	-0.178	0.031684	0.121	0.014641
Tower test move accuracy ratio score	-0.118	0.013924	0.097	0.009409
Tower test move accuracy scaled score	0.116	0.013456	-0.089	0.007921
Tower test rule violations per item ratio score	0.243	0.059049	-0.244	0.059536
Tower test rule violations per item scaled score	-0.214	0.045796	0.206	0.042436
WASI vocab raw score	-0.033	0.001089	0.035	0.001225
WASI vocab T-score	-0.047	0.002209	0.036	0.001296
WASI matrix reasoning raw score	-0.146	0.021316	0.14	0.0196
WASI matrix reasoning T-score	-0.155	0.024025	0.135	0.018225
WASI sum of T-scores	-0.135	0.018225	0.128	0.016384
WASI IQ score	-0.13	0.0169	0.109	0.011881
CPT II confidence index	-0.025	0.000625	-0.021	0.000441
WTAR Standard Score	0.003	0.000009	-0.053	0.002809

Chapter V: Discussion

The purpose of this study was to assess the neuropsychological functioning of clients who meet diagnostic criteria for substance dependence according to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition - Text Revision (DSM IV-TR; APA, 2000) and to examine the relationship between neuropsychological functioning and treatment attrition rates. Specifically, the executive functioning of individuals was evaluated. Furthermore, the relationships between substance use diagnosis, treatment attrition, and neuropsychological functioning was investigated. It may be that understanding how deficits in neuropsychological functioning affect an individual's behaviors (e.g., relapse, missing treatment sessions, and dropping out of treatment) may help to change attitudes of clinicians and others who may currently negatively stigmatize those with substance use disorders (e.g., believing that the individual is lazy, unmotivated, etc.). Understanding the relationships between substance use diagnosis, attrition, and neuropsychological functioning could prove extremely useful in substance use disorder program development, treatment planning, clinician training and stigma reduction.

Neuropsychological and cognitive impairment in substance abusing participants as well as homeless participants has also been reported in existing research as discussed previously. Therefore, the results of this study add to the existing research that confirms that people with a substance abuse issue as well as homeless individuals have a much greater occurrence of NP deficit than the general

population. Though much has been written about impairment in homeless substance abusing people, very little has been written about the relationship of neuropsychological impairment and attrition in treatment. As there is still a paucity of research in this area, the results of this study with regards to neuropsychological deficit, drop out and attrition add to the existing research.

Interpretation of Findings

Summary

This study examined the neuropsychological functioning of homeless, substance dependent men and how this affected attrition, survival in treatment and relapse. In summary, the neuropsychological (NP) functioning of this group of 68 adult males located in the Guesthouse Shelter of Milwaukee as a whole, showed statistically significant impaired functioning on all measures; though not every individual was impaired on every measure, some were impaired on each measure. For example, using a threshold of two standard deviations below the mean, (the threshold of two SD was used as this is often used as a threshold of abnormality, for example, T scores of 70 or higher are noted on psychodiagnostic measures such as the MMPI) 24.6% were impaired on the Trail Making Test Number Sequencing and Letter Sequencing Scaled score, 23.2% were impaired on the Verbal Fluency Category Switching Scaled score and 26.1% were impaired on the Color Word Naming Scaled score. Likewise, with a score of greater than 50% indicating impairment, 66.7% were impaired on the CPT II. Finally, the WASI IQ score showed 7.2 % impaired. Of the neuropsychological variables, only the WASI IQ predicted attrition and length of stay

except not in the expected linear relationship. The WASI IQ showed a curvilinear relationship to drop out and attrition. Prior to examining the results of this study, I had expected to see a linear relationship exist between low IQ and high attrition. The results of this study showed that participants with a low to average WASI IQ score (77-95) were statistically significantly more likely (than those with a borderline to low IQ - <77 or average to high IQ - >95) to drop out (p = .012) and more likely to have shorter lengths of stay in treatment (p = .028). In addition, some NP variables did show a relationship with drop out and length of stay when looking at cases with a median IQ below 94 and those with no prior AODA treatment.

Neuropsychological Impairment as Indicated by the D-KEFS, CPT II, and WASI

The results of this study indicated that this sample was drawn from a population with a lower than average level of performance on all measures. Specifically, the mean standardized scores for all of the D-KEFS subtests including the Tower Test, Verbal Fluency, Color Word and Trail Making test was lower than the criterion level of 9.29 set for rejecting the null hypothesis based on the one sample z test. These assessments were all chosen based on their ability to assess executive functions which are specifically associated with the frontal lobe- the brain area of primary interest in this study. Likewise, using a one sample z-test, the chance that this sample was drawn from a normal population on the CPT II test are less than one in one thousand. Finally, according to the one-sample z-test, the results of the WASI IQ subtest show that samples of 68 with mean less than 97.66 have a less than .05 chance of occurring if the sample was drawn from a population with a mean of 100. The observed sample mean falls below this threshold level, suggesting that the present sample has a

significantly lower IQ level. In practical terms, this indicates that our sample of substance abusing homeless men have a much lower level of cognitive functioning than the general population of non-homeless, non-substance abusing men. We cannot assume causation – that substance use or homelessness causes the impairment- or that the impairment causes homelessness or substance use. However, we can state that this sample in general is impaired and this information needs to be utilized when treating homeless, substance using men.

There are multiple implications of the severity of the NP deficit found in this sample. The NP impairment found by this study, as well as other studies (Burra, Stergiopoulos, & Rourke, 2009; Spence, Stevens & Parks, 2004), suggests that more thorough evaluation of NP strengths and weaknesses should be afforded to all homeless and/or substance abusing clients to develop better-tailored treatment programs and resources. Although we cannot make definitive causal connections from this study this information will be helpful for treatment providers to recognize the deficits as a limitation in the population they are serving. Though not presuming causation from these results, it is the opinion of this writer that a circular relationship exists between NP function, substance use, homelessness and attrition with each of these variables having a cause and effect relationship with each other. For example, substance use can cause NP impairment; NP impairment can cause individuals to make poor choices about substance use. Likewise, NP function can affect attrition; attrition/dropping out of treatment can cause one to be more likely to abuse substances causing more NP damage. Clinicians need to be cognizant that the

discussed deficits likely impair a homeless person's ability to maintain stable housing and follow clinician/staff recommendations.

The NP deficits examined in this study primarily address executive function skills which are specifically associated with the frontal lobe and include four components of (a) volition, (b) planning (c) purposive action, and (d) effective performance (Lezak, 1995; Lezak et al, 2004). These primary skill areas need to be taken into consideration with all goal and treatment planning. For example, clients would benefit from assistance in developing concrete goals, reduced to small, short term objectives. Likewise, they would need encouragement and instruction on how to begin addressing each goal. All instruction and education should be given in multiple media (written, verbal, auditory, etc) with frequent reminders. In addition to the NP components examined by the D-KEFS, the CPT II provides information on attention, impulsivity, activation/arousal and difficulties in maintaining vigilance (IPS, 2005). These specific skills are associated with the dorsal lateral prefrontal cortex. The CPT II is a simple, fast, computer generated instrument that would be easy for clinician to utilize upon primary assessment of clients that are homeless or struggling with substance abuse. This information could be used to increase client and clinician insight and develop goals specific to maintaining attention. For example, clients might be given a binder with all important information to bring with them to each appointment. Likewise, they could be given a wallet size card with reminders of important dates or aspects of treatment. Furthermore, calendars could be given to all clients and clinicians could be trained to remind the client to write all goals on the calendar- including self-care goals, formal appointments and social activities.

Though overall, all four D-KEFS subtests showed impairment in skills associated with the frontal lobe, it is the opinion of this writer that much detail related to frontal lobe function was still missed. For example, I do not believe any of these instruments adequately evaluate the frontal lobe function of behavior starting. With a guided assessment, someone facilitating the moves of the participant, behavior starting is difficult to assess. Likewise, purposive action, the gap between a client stating their intentions, and actually following through on the necessary behaviors is an important component of the frontal lobe (Lezak, 2004). This actual skill is difficult to assess during formal assessment as the facilitator again constantly guides the participant. In real life, people do not have someone with them prompting all the necessary, appropriate behaviors. This appears to be the paradoxical nature of all of the assessments used in the battery for this research. As these are formal, structured assessments, it is difficult to assess some of the more discretionary frontal lobe functions. However, all of the assessments were chosen due to their proclaimed ability to capture these skills. It is the opinion of this writer that these assessments did not adequately capture those components of the dorsal lateral frontal lobe.

Cognitive Functioning Predictive of Attrition and Survival in Treatment

For this study, general intelligence, as assessed by the WASI, predicts drop out and survival in treatment. Specifically, subjects with average to above IQ (>95), as well as those with borderline to low IQ (<77) were less likely to drop out of treatment, whereas subjects with an IQ between 77-95 were more likely to drop out. Likewise, subjects with low to average IQ (77-95) had shorter survival times (i.e., "time in treatment") than those with borderline to low IQ (<77). More precisely,

participants with a low to average WASI IQ score (77-95) were statistically significantly more likely to drop out (p = .012) and more likely to have shorter lengths of stay in treatment (p= .028). These findings indicate that one can predict drop out and length of time in treatment by evaluating the IQ of the participant. However, as previously mentioned, caution must be taken given the small sample size. Confidence in these results could be increased with a study that used a larger sample. Specifically, a Beta of -2.398 (p = .012) was found for predicting drop out and a Beta of -851 (p = .028) was found for predicting survival in treatment. Evaluating the strength of this Beta can be complicated with a binary logistic regression as there is no precise equivalent to an effect size measure like r-squared when predicting a binary outcome (Garson, 2009, retrieved June 9, 2009). We have no exact equivalent to r-squared because our dependent variable is not a continuous variable. Rather, the dependent variable is dichotomous- the odds of dropping out or not. Table 4.17 above shows the percentage of drop out by IQ. The group with IQ between 77 and 95 were over four times more likely to drop out than those with an IQ below 77, and almost twice as likely to drop out than those with an IQ over 95.

To compare these results to existing research, cognitive impairment has been found by multiple researchers in substance abusing adults (Grohman & Fals Stewart, 2004) as well as homeless males (Buhrich, Hodder & Teesson, 2000; Burra, Stergiopoulos & Rourke, 2009; Spence, Stevens & Parks, 2004). In addition, cognitive deficits predicting low treatment retention have also been found by other researchers (Aharonovich, Hasin, Brooks, Liu, Bisage & Nunes, 2005: Fals Stewart & Lucente, 1994). On the contrary, other researchers have found no difference in

treatment retention between high and low cognitive ability (Katz, King, Schwartz, Weintraub, Barksdale, Robinson and Brown, 2005).

The finding that low to average cognitive functioning (IQ between 77 and 95) (as opposed to borderline to low functioning, IQ below 77) is predictive of drop out and survival in treatment has implications for barriers to treatment, treatment planning, clinician training, and continued substance use in clients. These factors, in turn, can have an effect on the community, family and funding sources. To speculate, it appears that patients need to have a minimal level of cognitive functioning in order to drop out of treatment. For example, the results of this study indicate that those with an IQ between 77 and 95 were statistically significantly more likely to drop out (p = .012). To be precise, the group with IQ between 77 and 95 were over four times more likely to drop out than those with an IQ below 77, and almost twice as likely to drop out than those with an IQ over 95. Again, in speculation, for the borderline to low IQ patients, treatment might serve as a kind of sheltered environment. Patients with low to average IQ may have just enough cognitive functioning to follow through on a bad decision about dropping out. Patients with average and above IQ are perhaps more likely to make a good decision regarding persistence in treatment. Those with average and above IQ might be better able to recognize the benefits of treatment such as abstinence, improved mood, improved family functioning and resolution of legal conflicts. Likewise, those with average and above IQ might also be better able to recognize the negative consequences of dropping out such as continued substance use, health issues and family disappointment. However, there are many words of caution when using and interpreting the results of IQ tests. For example, the Wechsler

Adult Intelligence Scales were not designed to assess brain damaged patients and are considered “insensitive to their neurobehavioral problems and cognitive deficits” (Sbordone, Saul & Purisch, 2007, p.357). Likewise, there are many additional concerns reported about the use of the Wechsler IQ including over-interpretation of subtest scores, belief that norms may not be applicable for ethnic minorities, complexity of scoring lending itself to clerical errors by examiners, and subjectivity in scoring (Groth-Marnat, 2003). Furthermore, there is question related to the actual meaning of IQ scores. Many misconceptions are made regarding IQ scores. IQ scores are not fixed and they are “not exact, precise measurements” (Groth-Marnat, 2003, p. 140). When interpreting intelligence scores, one needs to remember that IQ scores are estimates that can be related to a variety of environmental factors (Groth-Marnat, 2003).

Clinicians need to be cognizant of the prevalence of cognitive impairment in substance using clients and the potential negative impact this can have on drop out and survival in treatment. In addition, clinicians might be more likely to give additional attention to the clients with borderline to low IQ. However the results of this study indicate that it is actually the clients in the low to average, not borderline to low range, who may need extra effort. Clinicians should partake in additional training to increase assessment skills to identify clients with low to average IQ. Utilizing a simple, brief, assessment tool, such as the WASI, could provide valuable information to clinicians if included in the standard intake assessment. All clients should be screened for cognitive impairment during the initial assessment with those with low to average IQ being offered additional support services through the treatment process.

Clients that appear to be struggling may need to be referred to a more structured or intense program during the initial months of recovery.

Specific techniques can be employed toward improving outcomes for substance users with cognitive deficits. For example, drug counseling that makes use of mapping techniques has been shown to improve treatment outcome by addressing planning and problem solving issues (Czuchry & Dansereau, 2003b). Mapping techniques would not only be helpful for the client with cognitive deficit, but also the clients with NP deficits addressed above. Using mapping techniques that address planning and problem solving would offer support to clients with NP impairment, especially executive function impairment. Any and all repetition and guidance in planning and problem solving skills will assist these individuals in compensating for such deficits.

Strategies used to address cognitive dysfunction in patients with traumatic head injuries can also be helpful with cognitively impaired substance abusers (Ersche & Sahakian, 2007). For example, information should be presented to clients in a variety of modalities such as written, oral, auditory and visual. Education should be done slowly with repetition and paraphrasing encouraged by the client (William & Evans, 2003). In addition, communication between all clinical staff will help to ensure that cognitively impaired clients are not perceived as deceitful or manipulative. Likewise, treatment providers must keep in mind that cognitively impaired clients' nonadherence to treatment may be a result of the impairment and not caused by denial, resistance, or unwillingness to accept care (SAMHSA, 1998).

Clients demonstrating a low to average IQ warrant special attention in regard to disengaging from services. The client should be made aware of their risk of dropping out or not staying in treatment. This should be discussed thoroughly and goals implemented to address this risk. For example, clients could be given a list of people to call when having thoughts of not returning to treatment. Likewise, the topic of dropping out of treatment should be added to the agenda of regularly addressed issues in the treatment process. Finally, therapist awareness could be elevated by providing a visual cue on the charts of at risk clients. For example, clients at high risk could have a different color label on their chart.

Neuropsychological Function and Attrition/Survival in Treatment

Although some research has indicated a possible relationship (with the assumption that poor NP function would increase attrition) between neuropsychological functioning and attrition rates (Zinn et al., 2004) there is still a paucity of research on this topic. Some authors acknowledged that neuropsychological impairment may negatively affect attrition and treatment success but they have not formally evaluated retention and/or attrition (Zinn et al., 2004). As discussed above, researchers suggest that a relationship between neuropsychological functioning and treatment outcome exists, but empirical attempts to document this relationship have been met with limited success.

The results of this study show that there is a relationship between the neuropsychological variables (D-KEFS) and attrition rates with below median IQ

(below 94) and no prior AODA treatment. However, caution must be taken regarding the confidence in this data due to the small sample size. Specifically, the NP variables that show statistically significant correlations include Trail Making, Verbal Fluency and Tower test for cases with below median IQ and Verbal Fluency and Tower test for no prior AODA treatment. The results indicate that for most of the subtest relationships, there was a negative correlation indicating that the better the subject performed on the measure, the more likely he was to drop out of treatment and/or have a shorter length of stay in treatment. Although perhaps counterintuitive the results are indeed interesting.

The three subtests shown to be predictive of drop out and/or length of stay include the Trail Making test, the Verbal Fluency and the Tower Test. (All three were predictive for subjects with below median IQ and Verbal Fluency and Tower test were predictive in subjects with no prior treatment) The Trail making test measures flexibility of thinking on a visual motor task. There is a negative correlation between the trail making test number sequencing raw score and participant drop out for subjects with below median IQ (r = -.494, p = .005) indicating that the better they scored, the more likely they were to drop out. The r squared for this correlation is .24 indicating that 24% of the variance of the drop out can be accounted for by trail making test predictor. This is a relatively strong prediction. (As a general rule, correlations that account for 10% or less of the variance are weak, those that account for more than 25% are quite strong; Cohen, 1977)

This subtest requires the individual to visually scan and sequence numbers. This subtest might be more of an indication of cognitive functioning and therefore may be

an indication of participants with moderate functioning being more likely to drop out as indicated by the WASI scores being predictive of drop out. Likewise, two of the Tower Test scores, which measure spatial planning, rule violation, inhibition of impulsive responding, inhibition of perseverative responding and establishing and maintaining an instructional set, also showed a negative correlation. Specifically the tower test total rule violations raw score was r = -.495, p = .005 (with an r squared of .24, again relatively strong prediction) and tower test rule violations per item ratio score was r = -.533, p = .002 (with an r squared of .28). This score indicates that the more rule violations a participant had, the less likely they were to drop out or the better they did, the more likely they were to drop out. Similarly, two of the Tower test subtests for subjects that had no prior treatment also showed a correlation that indicates the better the participant performed, the more likely they were to drop out or have a decreased length of stay. The tower test total achievement raw score has a correlation of r = .644, p = .007 (with an r squared of .40, predictive strength increasing) and tower test total rule violations raw score has a correlation of r = -.756, p = .001 (a very strong prediction with an r squared of .56). The only NP subtests that indicated that the better a subject performed, the less likely they were to drop out was the Verbal Fluency third interval for subjects with no prior treatment (r = -.593, p = .015, r squared of .34) and the Verbal Fluency second interval total correct score showed a correlation of r = .401, p = .025 (a somewhat weaker predictor with an r squared of .16) However, these scores are simply an indication of a subject's ability to sustain a verbal response over time and are therefore not one of the more global or primary scores.

We initially had expected to see those with lower NP performance be more likely to drop out and have a lower length of treatment. Though initially the present study results appear counterintuitive, the fact that the groups either had below median IQ or no prior treatment might assist in interpretation. It may be that those with lower IQ that scored better on the NP measures had greater confidence to feel they do not need treatment. The higher NP function and lower cognitive ability might lend itself to poor decisions and an inability to see the potential benefits of treatment. Likewise, as discussed above, any impairment in cognitive functioning can affect a person's ability to make a good decision. It may be that the lower cognitive functioning encouraged a bad decision to drop out of treatment. However, it may also be that those with lower IQ do not benefit as quickly as others. The difference may not be related to decision-making abilities, but simply that they are slower to benefit from treatment. Furthermore, it could be that the WASI IQ score is more an indication of personality functioning than previously believed and that is what is actually affecting the decision to drop out. Personality variables are considered important when evaluating intelligence (Groth-Marnat, 2003). Indeed, Wechsler himself believed that intelligence is influenced by personality as well as other component such as anxiety (Groth-Marnat, 2003). Caution must be taken when interpreting the results of those with no prior treatment as only 16 individuals had no prior treatment. Re-evaluating this issue with a larger sample size could help clarify the results. Other factors that we were not able to formally evaluate in this study also need to be considered in future research. For example, personality was not directly assessed by this study and would be an interesting piece to incorporate in future research.

Also interesting is that the NP subtests that showed no correlation with drop out or attrition - Color Word and CPT II - are both tests of inhibition and impulsivity. Though participants were statistically significantly impaired on both of these measures, neither showed a correlation for participants with below median IQ or those with no prior treatment. This suggests that these aspects of NP impairment are less important when evaluating treatment retention (though still important for treatment planning). It may be that once individuals are engaged in treatment, the impulsivity and inhibition are affecting their performance in treatment, but not whether or not they stay in treatment.

The extent of neuropsychological and cognitive deficit found in individuals with a substance use disorder and/or homeless individuals suggests that increased assessment upon intake should be standard to accurately evaluate the specific needs of individuals, effective treatment planning and efficient disbursement of resources. These results indicate that clinicians need to develop highly individualized treatment plans utilizing specific strengths and identifying weaknesses for each individual for those with and without NP deficits to decrease attrition and increase length of stay. In addition, based on the results of this study, special attention may need to be paid to those with below median IQ and those with no prior treatment. As discussed above, special measures can be taken to help flag the clients that may be more at risk. Likewise, the clients that are identified to be at greater risk of dropping out should be educated about this and specific treatment goals implemented regarding treatment attendance and completion. These clients would also benefit from education regarding the specific expectations and timeline of treatment. One of the subtests

negatively correlated with drop out is the Tower Test. This test addresses the ability to follow rules. In this study, the better the client did with this, the more likely they were to drop out. Clinicians could make use of this strength and actually implement rules that the clients are asked to follow about staying in treatment. Clients could sign an agreement at the beginning of treatment specifying the required attendance and length of treatment and their willingness to comply. The ability to follow rules was shown to be one of the predictors to drop out. This is not to say that following rules caused drop out, however making use of this strength might actually help improve retention. Likewise, behavior modification and contingency management approaches could also be implemented. Participants could be rewarded at intervals in treatment with products such as tokens indicating length of sobriety (as used in AA), products with clinic insignia (t-shirts, pens, bags) or gift certificates to healthy social functions such as movies or restaurants.

Though the results of this study do not specifically support the need for increased NP and cognitive assessment at time of intake, they do seem to suggest that clients would benefit from increased assessment. Increased NP assessment at time of intake can provide specific strengths and weaknesses of each individual to allow the clinician to develop treatment approaches that may best suit a clients needs. The importance of individualized treatment planning cannot be overstated and is well supported in the literature (Adams, 2004). In addition, this would aid in educating the clinician and reducing stigma. The “lazy” “unmotivated” client might actually have serious NP and/or cognitive deficits. This could impact provider perception and expectations. If a provider understands the issues of the client and is given tools to

address the issues, the provider will be more effective. This alone could lead to improved retention. Conversely, if the provider is frustrated and misidentifies the deficits as a lazy client, the provider might direct their energy and resources to other clients they perceive as more receptive. Likewise, funding sources would be more likely to direct resources to clients to assist in situations where a deficit is identified vs. directing resources to someone they think is not wanting or ready for treatment. In order for programs to implement a more thorough assessment at time of intake, clinicians would need further training and programs would need funding for the assessment. Therefore, funding sources, such as private insurance and government resources, all need to be informed of the benefits to increased assessment to allow for allocation and reallocation of resources. Providers and funders alike, all need to rethink priorities when it comes to direction of energy to the assessment process. As discussed above, the current statistics on attrition and survival in treatment show limited success of current treatment programs. This has an overall negative financial impact on individuals, families, employers and funding sources (SAMHSA 2008; TEDS, 2005). Therefore, additional funding up front, could save money overall for the funding sources. By improving assessment, we could provide better treatment, and therefore have a positive impact on the rates of substance abuse. Multiple researchers and agencies have commented on the high cost of substance use on a personal and societal level as discussed thoroughly above (SAMHSA, 2008: TEDS, 2005).

Additional research is warranted to continue to evaluate the relationship between NP functioning, attrition and survival in treatment. Additional research, with

a larger sample size, as well as a sample that included women and non-homeless individuals could prove quite useful. With a larger sample size, more variables that are useful for predicting drop out and attrition might be found. Furthermore, the ability to evaluate all people with a substance use problem, not just homeless men, would provide much useful information that can be generalized to more of the population.

Effect Size

As noted previously, no relative effect sizes could be found to be used to determine sample size for this study. Therefore, in addition to the information presented on neuropsychological functioning, cognitive functioning, drop out and attrition rates, effect sizes were calculated and reported on as another valuable outcome of this study. Information on effect sizes is valuable as it can help determine which variables look most promising as possible predictors of attrition and to provide information on how many more cases one would have to add to the sample size to obtain sufficient statistical power. Specifically, results of calculations on effect size and post-hoc power analysis, with alpha set at .05, showed that with a larger sample size (98-170) we could increase the possibility that the neuropsychological variables would predict drop out and attrition and could attain statistical power between .80 and .95. This information will be valuable to future researchers when planning similar studies.

Limitations/Suggestions for Improvements and Future Research

These results were limited initially by the location we chose to collect the data. As we were collecting data on men living in a homeless shelter, we were bound by rules and regulations of the shelter. We were unable to access urine screen information, which resulted in no information on relapse. The impact on relapse rates could not be calculated as we were not able to access urine analysis information. As urine screens were given by case managers in the Guesthouse, and not by counselors in the 7Cs clinic, we did not have consent to access that information. Future researchers operating in a homeless shelter environment will be well-served to establish a written agreement with the shelter and the participants to be able to access any objective screening measures used. In addition, our study was cut short by a transition of management at the clinic which stopped our data collection at 68 completed batteries instead of the intended 100 batteries.

Another important limitation of this study is that the men living in the shelter were required to attend substance abuse treatment. Therefore, the participation in treatment was not completely voluntary which could affect the participants desire to perform on the given assessments.

In addition, as it is a population of homeless men, the physical state of the participant at the time of testing such as fatigue or hunger could also impact performance affecting the validity of the results we obtained. Future researchers should consider utilizing a brief screening instrument to evaluate a participants general level of hunger or fatigue at various intervals to attempt to control for these

issues. The fact that all participants were homeless men also limits the generalizability of this information to other populations such as women and non-homeless individuals. Likewise, the fact that this treatment took place at the homeless shelter is different from other treatment providers. The men lived in the shelter where they were given treatment. If discharged from the shelter, the men were unlikely to return to the shelter for treatment. This might have been a result of feeling unwelcome at the shelter in general, or maybe a desire to leave that part of their lives behind once they had moved on with independent housing. Most outpatient treatment facilities are not tied physically or emotionally to a person's residence.

Limitations might also arise from the use of multiple assessors to facilitate the neuropsychological assessments. All assessors were masters level students that were trained by a licensed neuropsychologist. However, all of the assessors were new to the neuropsychological battery, which could affect the facilitation of the battery. Moreover, only subjective assessments were used to verify abstinence at time of testing. Future researchers may want to invest in saliva tests or quick urine screens to verify abstinence at time of testing. No patron is allowed to enter the Guesthouse if they are believed to be impaired by any substance so all are subjectively screened at the door. Likewise, no neuropsychological batteries were given to anyone thought to be impaired at the time of the assessment, but again, this was a subjective screening. Similarly, no objective measures were used to evaluate abstinence during treatment. Urine screens, blood tests, saliva samples and breathalyzers would have all provided objective evidence of a participant's use. We did not have access to any of these results during this study.

As suggested by the effect size and power calculations discussed above, another limitation of this study is the sample size. As stated previously, caution must be taken when interpreting this data due to the small sample size. Specifically, much caution needs to be taken when interpreting any relationship between IQ and attrition/length of stay due to the very small sample sizes of those calculations. Our calculations suggest that if we had a larger sample size (98-170) we could increase the possibility that the neuropsychological variables would predict drop out and attrition and could attain power between .80 and .95. This would mean that we are more likely to reject a null hypothesis and therefore increase the likelihood that what our results state is in fact true. Therefore, future researchers would be wise to include a larger sample size.

Finally, the mental health diagnoses were not included in this research and could offer another important variable when evaluating level of neuropsychological impairment and treatment attrition. We are not able to evaluate the possibility of depression, anxiety or any other mental health diagnosis that could have been a variable in these results. It is quite likely that many of these participants did indeed have a dual diagnosis. A diagnosis of a mood disorder, anxiety disorder or personality disorder all could impact results of cognitive and NP functioning. Likewise, these mental health issues could impact one's desire and ability to remain in treatment. We are not able to identify if these issues existed or how they may have impacted the results of this study. Future researchers would be wise to evaluate these variables and attempt to control for them when looking at NP function and attrition.

Likewise, we did not distinguish between types of substances used or length of substance use, which could also offer interesting information. Though we are interested in treating all substance abuse, the types and length of use could have provided more detailed information regarding drop out and survival in treatment. Alcohol, marijuana, cocaine and opiates all can have a negative impact on one's ability to perform on cognitive and NP assessments. Furthermore, different substances can produce different frequency and intensity of cravings for individuals. The cravings alone could have a significant impact on ability to stay in treatment – especially in a program that will remove you from your housing if you are actively using. Likewise, support systems, cognitive function, neuropsychological function and physical health can all be impacted by type of substance used and length of use. This would also be another useful variable for future research for all the reasons stated.

Limitations also arise from the difference between actuarial and qualitative components of assessment. As previously discussed in this paper, in strict actuarial approaches, the neuropsychologist need not even see the patient, but rather draw conclusions from scores obtained by a technician (Lezak, 1995). This approach can be helpful for gathering statistics, but important information is lost from the missing qualitative component. Through the development of testing batteries developed by some of the leaders in the field, neuropsychology has developed into more of a mix of the intuitive and actuarial (Lezak, 1995). However, given that this study utilized multiple facilitators and the primary researcher did not meet many of the participants, the benefits of a more process-oriented approach were lost. Qualitative comments

were gathered on each participant, but the depth of the qualitative component that can be gathered through each assessment was lost.

Reliability and Validity

There are multiple factors that can affect reliability and validity of data obtained in any research. First and foremost are the reliability and validity of the data yielded from the measures used. The reliability and validity of the data used in this study is discussed in detail in Chapter III under the psychometrics of each instrument. In addition to the specifics of each instrument, a number of authors have elaborated on the difficulties of using standardized assessments with ethnic minorities. Given that 75% of the participants in this study are non-White, this is an important factor to consider. Specifically, Suzuki and Kugler, 1995 (as cited in Pope-Davis & Coleman, 1996) summarize areas of concern including inappropriate test content, inappropriate standardization samples, examiner and language bias, inequitable social consequences, measurement of different constructs, differential predictive validity and differences in test taking skills. Therefore, the development of additional norms for these tests and others used on non -White individuals as well as homeless individuals would be of benefit to future researchers.

Furthermore, limitations in the setting could also affect the results of this study. The homeless shelter setting can be loud and distracting at times, which could affect the participant's performance. Likewise, issues related to being homeless, such as hunger and fatigue can also affect one's performance. The use of multiple assessors with

limited experience in facilitating NP assessments is also an important factor to consider when reviewing reliability and validity of the given results. Finally, though the D-KEFS and CPT II both have computerized scoring, the WASI was all hand scored. To ensure accurate scoring, licensed psychologists reviewed 20% of the batteries.

Conclusion

In conclusion, the purpose of this study was to assess the neuropsychological functioning of clients with a substance dependence issue and to examine the relationship between neuropsychological functioning, length of stay in treatment and whether one dropped out of treatment or not. Specifically, the executive functioning of individuals was evaluated, as well as IQ and compared to attrition rates. In this study, a battery of tests including four subtests of the D-KEFS, the WASI IQ and the CPT II were administered to a group of 68 homeless adult males residing at the Guesthouse of Milwaukee. The results indicate that the neuropsychological functioning of this group of adult males showed statistically significant impaired functioning on all measures. Of the neuropsychological variables, only the WASI IQ predicted attrition and length of stay which showed a curvilinear relationship to drop out and attrition. Participants with a low to average WASI IQ score (77-95) were significantly more likely to drop out ($p = .012$) and more likely to have shorter lengths of stay in treatment ($p = .028$). In addition, the NP variables did show a relationship with drop out and length of stay when looking at cases with a median IQ below 94 and those with no prior AODA treatment. However, caution regarding

interpretation was indicated due to the small sample sizes. Finally, results of calculations on effect size and power analysis show that with a larger sample size (98-170) we could increase the possibility that the neuropsychological variables would predict drop out and attrition and could attain power between .80 and .95.

As there is still a paucity of research related to NP functioning, homeless individuals and attrition rates, this information is a valuable addition to existing research. In addition, this study identified implications for professionals that work with substance abusing individuals, specifically Individuals that are homeless with a substance use issue and individuals struggling with a NP deficit. As this study is limited to homeless males, the generalizability of this research is limited. Therefore, suggestions were also made for future research.

Overall, the importance of this research is multifaceted. The information regarding specific variables that are predictive of client success in treatment cannot be underestimated. As discussed, the severity of addiction rates and attrition rates in the United States dictates the need for more information regarding variables that affect client success in treatment. The suggestions from this research regarding therapist approaches, funding allocation and treatment interventions will help reduce drop out/attrition and ultimately help reduce the rates of relapse and continued substance use. Likewise, the more educated providers and the public are on neuropsychological function and substance use disorders/treatment, the greater reduction we will see in stigma. Furthermore, the information regarding effect size will help future researchers plan effective research designs to continue to gather information regarding NP function and attrition/drop out. Individuals with a substance use disorder deserve the

very best care we can offer. With this addition to the literature, and others, we will develop efficient, effective and successful treatment plans for individuals and families affected by substance use disorders.

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