12-2017

Age of Drinking Initiation's Association with Cognitive Functioning

Joshua Seth Goldberg

LOMA LINDA UNIVERSITY
School of Behavioral Health
in conjunction with the
Faculty of Graduate Studies

Age of Drinking Initiation's Association with Cognitive Functioning

by

Joshua Seth Goldberg

A Thesis submitted in partial satisfaction of
the requirements for the degree
Doctor of Philosophy in Clinical Psychology

December 2017

Each person whose signature appears below certifies that this thesis in his/her opinion is adequate, in scope and quality, as a thesis for the degree Doctor of Philosophy.

_____, Chairperson

Grace J. Lee, Assistant Professor of Psychology

_____

Kelly R. Morton, Professor of Psychology and Family Medicine

_____

Ricardo Whyte, Assistant Professor of Psychiatry, School of Medicine

ACKNOWLEDGEMENTS

I would like to express my deepest gratitude to Dr. Grace Lee for continuously providing me the unconditional support to pursue excellence in my graduate studies.

I would also like to my committee members for their advice and direction. Without Dr. Kelly Morton and Dr. Ricardo Whyte, this project would not have been possible. Thank you for your diligence and patience with me throughout my thesis drafting.

To my family and friends, your love and support through this long endeavor has been unmatched. I extend a special thank you to my parents who have been a such a source of such inspiration to me. Thank you for all the sacrifices you have made to allow me to pursue my passion of a career in clinical psychology.

CONTENT

FIGURES

Figures Page

TABLES

Tables Page

ABBREVIATIONS

AUD	Alcohol use disorder
DSM-5	Diagnostic and Statistical Manual of Mental Disorders 5th Edition
CDC	Centers for Disease Control and Prevention
fMRI	Functional magnetic resonance imaging
GABA	Gamma-aminobutyric acid
MMSE	Mini-Mental State Exam
MoCA	Montreal Cognitive Assessment
WAIS-IV	Wechsler Adult Intelligence Scale 4th Edition
MCI	Mild cognitive impairment
RBANS	Repeatable Battery for the Assessment of Neuropsychological Status
CDPHP	Chemical Dependency Partial Hospitalization Program
LLUBMC	Loma Linda University Behavioral Medicine Center
AI	Age of alcohol initiation
ANCOVA	Analysis of covariance
APD	Age of problem drinking
DPD	Drinks per day
NIH	National Institute of Health

ABSTRACT

Age of Drinking Initiation's Association with Cognitive Functioning

by

Joshua Seth Goldberg

Doctor of Philosophy, Graduate Program in Psychology
Loma Linda University, December 2017
Dr. Grace J. Lee, Chairperson

Research has indicated that alcohol abuse is associated with deleterious effects on cognitive functioning later in life, specifically in the neuropsychological domains of immediate memory, delayed memory, and attention. However, research has been mixed regarding how age of initiation into problem drinking affects cognitive health after abstinence. This study aimed to identify if earlier age of alcohol abuse was associated with significant deficits in neuropsychological functioning in comparison to individuals who commenced alcohol abuse later in life. Participants were recruited from an alcohol rehabilitation program within Loma Linda University's Behavioral Medicine Center and were administered the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS) after eight days of substance sobriety. Results indicated that individuals who drank more and began drinking in childhood demonstrated significantly better performance in immediate memory in comparison to individuals who drank less and who initiated into drinking later in life. However, these findings are reflective of pervasive limitations within this study, including low power, low sample size, and operational complexities within the studies primary independent variables.

CHAPTER ONE

INTRODUCTION

Alcoholism is one of the most devastating addictions both in America and around the globe. In 2012, it was estimated that over 17 million adults ages 18 and older suffered from some form of an alcohol addiction in the United States (National Institute on Alcohol Abuse and Alcoholism, 2014). Alcohol misuse costs the United States around $223.5 billion annually due to losses in workplace productivity, health care expenses, criminal justice expenses, and motor vehicle accidents. Globally, alcohol misuse accounts for 5.9% of global deaths and is the fifth most preventable cause of premature death.

Defining Problem Drinking/Alcohol Abuse

In the fifth edition of the Diagnostics and Statistical Manual of Mental Disorders (DSM), the clinical diagnosis of problematic alcohol drinking is described as Alcohol Use Disorder (American Psychiatric Association, 2013). Alcohol Use Disorder (AUD) is characterized by behavioral and physical symptoms, which include withdrawal, tolerance, and cravings that cause significant distress within a timeframe of 12 months (American Psychiatric Association, 2013). With the recent revision of the DSM-5, health professionals now recognize alcohol abuse and alcohol dependence as the same spectrum of AUD's. Alcohol abuse refers to the harmful use of alcohol that affects professional and social life, while also contributing to risky drinking behaviors (Centers for Disease Control and Prevention, 2013). Alcohol Abuse can eventually contribute to Alcohol Dependence (also known as alcoholism), which is encompassed by increased tolerance and withdrawal symptoms associated with alcohol use. AUD's are routinely associated

with elevated alcohol usage. The Centers for Disease Control and Prevention (CDC; 2013) indicate that heavy drinking for men is defined as 15 drinks per week; while heavy drinking for women is defined as 8 drinks per week. Specifically, individuals who abuse alcohol typically drink in large quantities. However, individuals can drink heavily without abusing alcohol as well.

General Health Risks/Consequences

Chronic alcohol abuse has been linked to several health risk factors throughout the human body. Cardiovascular alcohol-related health deficits include heightened risk of stroke, and chronic hypertension (American Heart Association, 2015). More than two million alcoholics currently suffer from some form of liver disease related to chronic alcohol abuse; and, that chronic alcohol abuse increases the risk for cancers of the mouth, esophagus, larynx, pharynx, liver, and breasts (National Institute on Alcohol Abuse and Alcoholism, 2010). Alcohol abuse and excessive drinking have also been associated with a wide breadth of health problems including: digestive problems, diabetes, sexual dysfunction, eye problems, bone loss, weakened immune system, as well as an increase in motor-related accidents, accidental death, suicide, risky sexual practices, and accidental injury (Mayo Clinic, 2014).

Longitudinal research has demonstrated that individuals who abuse and/or become dependent on alcohol are more likely to experience depression and suicidal ideation (Fergusson, Boden, & Horwood, 2013). Individuals meeting criteria for alcohol misuse had twice the likelihood of suffering from depression and were six times more likely to engage in suicidal ideation than individuals who did not exhibit alcohol-related

issues. In fact, individuals with a history of alcohol abuse are likely to experience both anxiety and depression even after a significant duration of abstinence (Desfosses, Meadows, Jackson, & Crowe, 2014). Excessive alcohol use has also been linked to heightened risk of mortality. Additional longitudinal data suggested that middle-aged men who were considered heavy drinkers (five or more drinks per day) had four times the risk of developing functional impairment and twice the risk of developing some form of mental illness compared to controls (Perreira & Sloan, 2002).

CHAPTER TWO

NEUROPSYCHOLOGICAL EFFECTS

With the recent emergence of progress within neuropsychological testing and brain imaging, researchers have investigated the effects of years of alcohol use and abuse on the brain and cognitive function. Some research has shown that light to moderate drinking might provide protection against cognitive decline (Anstey, Mack, & Cherbuin, 2009; Panza, Solfrizzi, Seripa, Imbimbo, & Pilotto, 2010; Pinder & Sandler, 2004; Ruitenberg et al., 2002; Stampfer, Kang, Chen, Cherry, & Grodstein, 2005; Zanjani, Downer, Kruger, Willis, & Schaie, 2013; Zuccalà et al., 2001). Commonly held biological theories suggest that regular to modest alcohol intake might moderate vascular risk factors that can impact the brain. For instance, modest alcohol consumption can increase high-density lipoprotein cholesterol, inhibit platelet aggregation, and help release the neurotransmitter acetylcholine that fosters memory formation (Mukamal, Kuller, Fitzpatrick, Mittleman, & Siscovick, 2014; Stott et al., 2008). However, long-term benefits of modest alcohol consumption do not generalize to heavy drinking.

Rather, chronic alcohol abuse has been associated with significant deficits in neuropsychological functioning. In general, recently detoxified individuals suffering from alcohol abuse perform similarly to non-alcohol abusing populations on measures of verbal ability and full scale IQ tests, but score lower on measures of achievement (Waldron-Perrine & Adams, 2014). Therefore, though overall intelligence is unaffected by chronic alcohol abuse, there may be diminished abilities in certain cognitive domains.

Alcoholic populations routinely demonstrate attentional dysfunction (Stavro, Pelletier, & Potvin, 2013). However, tasks involving visual attention are less impacted

(Bijl, de Bruin, Kenemans, Verbaten, & Böcker, 2005). Researchers maintain that specific detriments in attention reflect diminished working memory functioning within alcoholic-dependent individuals (Ambrose, Bowden, & Whelan, 2001). Moreover, short-term abstinence (approximately 40 days) was associated with significant increases on attention related tasks for chronic alcohol abusers. Within a twin-study, individuals who initiated their alcohol abuse within adolescence have demonstrated subtle yet significant detriments in attention and orienting in an auditory event-related potential task compared to their twin counterparts who did not initiate alcohol use within adolescence (Koskinen et al., 2011). Thus, attentional dysfunction within chronic alcoholic patients is ostensibly reflected in poor working memory functioning.

There are limited studies on chronic alcohol abuse and organizational verbal memory impairments. Verbal memory deficits within alcoholic populations could be the indirect result of liver dysfunction (Fox, Coltheart, Solowij, Michie, & Fox, 2000). Such neurotoxic effects could have profound effects on the hippocampal regions of alcoholics, with adolescent populations being the most susceptible to such damage (De Bellis et al., 2000). However, research has demonstrated that there are much more pronounced deficits in other areas of cognitive ability, specifically memory capacities, within chronic alcohol abusing populations.

Specific domains of memory impairment within alcoholic populations typically include visuospatial memory. Past literature has highlighted that visuospatial memory is resistant to recovery following durations of abstinence. This is in contrast to visuospatial ability, which seems to be impacted through chronic alcohol abuse but not nearly as much as memory-related components of cognitive functioning (Stavro et al., 2013;

Beatty, Hames, Blanco, Nixon, & Tivis, 1995). Specific detriments in visuospatial learning and memory have been associated with significant demyelination within the corona radiata within the cerebellum in an alcoholic sample (Yeh, Simpson, Durazzo, Gazdzinski, & Meyerhoff, 2009).

There is some evidence that alcoholic patients experience specific language difficulties. In an auditory language task aimed at analyzing language comprehension, alcoholic subjects more heavily utilized frontal and temporal regions than healthy controls (Chanraud et al., 2011). Thus, researchers believe that although alcoholics and controls task performance was indistinguishable, alcoholic patients were more heavily taxed when participating in the task. Additionally, alcoholics have been shown to have significant deficits in verbal fluency in comparison to non-alcoholic controls (Hewett, Nixon, Wagner-Glenn, & Parsons, 1991; Cutting, 1978). Individuals categorized as alcoholics exhibited greater fMRI activation of the pars triangularis, the right superior frontal gyrus, and the cerebellar vermis than controls, possibly reflecting the need for compensatory strategies to perform at equal levels of controls. It should be noted that verbal fluency is thought to be highly associated with damage in the frontal regions of the brain. Research involving language does not appear to be as prominent and as well-defined as other areas of neuropsychological functioning within alcoholic populations.

Patients suffering from chronic alcohol abuse commonly present with frontal-executive deficits. Alcohol abusers performed worse than non-abusing controls in an alternate response task of responding flexibly to a task with constantly changing rules to assess frontal-executive function of inhibition (Brokate et al., 2003). This example of difficulties of set shifting is indicative of frontal-executive dysfunction in alcoholic

populations, which is hypothesized to be associated with related neurotoxic effects within frontal regions of the brain (Noel et al., 2001). When presented with a perceptual learning task, detoxified alcoholics did rely more heavily on frontal-executive functions than non-alcoholic controls (Fama, Pfefferbaum, & Sullivan, 2004). Such reliance on executive functions was considered less efficient than utilizing less taxing cognitive resources such as visuoperceptual ability within the same task. Research has also reported that alcoholics exhibit impairments in decision-making ability, also a major component of frontal-executive functioning (Fernández-Serrano, Pérez-García, Schmidt Río-Valle, & Verdejo-García, 2010). Alcoholics suffering from alcohol withdrawal have also demonstrated inhibition deficits while memory and visuospatial ability remain intact (Desfosses et al., 2014; Wollenweber et al., 2014). Thus, difficulties in shifting between tasks, decision-making, and inhibition reflect pronounced frontal-executive deficits in alcoholic populations.

Working memory deficits within alcoholic populations have been associated with a diminished neural efficiency between the cerebellum and frontal regions and are associated with duration of drinking (Brokate et al., 2003; Chanraud, Pitel, Pfefferbaum, & Sullivan, 2011; Sullivan & Pfefferbaum, 2005). Research is limited regarding how the duration of alcohol abuse affects working memory functioning. However, existing literature delineates that chronic alcoholism is associated with a significant slowing of working memory functioning, specifically, cognitive planning (Ritz et al, 2014). Studies indicate that continued alcohol abuse for over ten years is linked to widespread deficiencies in working memory, implicit memory, psychomotor speed, and associate learning (Cairney, Clough, & Jaragba, 2006). Thus, processing speed is likely negatively

affected by chronic alcohol abuse, primarily through the mechanism of psychomotor speed deficits. Hence, participants who had abused alcohol for over ten years exhibited diminished speed of acquiring and analyzing new information, which in turn delayed participants' reaction time, the operationalized modality of processing speed. Chronic alcoholism is also associated with pronounced psychomotor speed and visuoperceptual processing deficits, particularly in women (Flannery et al., 2007). For instance, research examining same-aged individuals with differing alcohol abuse histories illustrated that nonverbal learning may be hindered by alcohol abuse regardless of age (Fein, Bachman, Fisher, & Davenport, 1990).

However, current researchers speculate that slowed psychomotor speed may be confounded by older age, regardless of alcohol consumption and duration. Research analyzing specific patterns and trajectories of deficits of cognitive functioning has been limited and concrete conclusions regarding alcohol abuse on cognitive functioning have yet to be made due to the plethora of confounding variables within alcohol abuse including but not limited to age, years of education, and comorbid medical and psychological conditions.

In summary, it is evident that individuals who abuse alcohol commonly exhibit dysfunction in a wide variety of neuropsychological domains. The most prominent areas of dysfunction include: attentional capacity, memory (primarily working memory), and frontal/executive functioning. Despite the amount of research within the realm of alcohol abuse and diminished cognitive functioning, there seems to be a lack of research that considers how the specific duration of drinking affects such cognitive dysfunction, while controlling for age.

CHAPTER THREE

NEUROBIOLOGICAL MECHANISMS

Scientists believe that chronic and problematic alcohol consumption might influence brain activity through a few different mechanisms. Heavy alcohol consumption, even during one sitting, can produce neurotransmitter imbalances throughout the brain. Four prominent neurotransmitters in the brain that are commonly affected by alcohol use are serotonin (responsible for emotion regulation), acetylcholine (responsible for memory formation and deficient in individuals suffering from dementia), gamma-aminobutyric acid (GABA: inhibitory neuronal response) and dopamine (responsible for reward and pleasure). Persistent problem drinking across the lifespan can cause chemical imbalances in the brain that can prove highly detrimental.

Literature suggests that individuals who are dependent on alcohol experience a depletion of serotonin during withdrawal (Wong et al., 2003). Specifically, research supports the notion that long durations of alcohol abuse reduces central serotoninergic activity and is associated with elevated depressive and anxiety symptoms during periods of withdrawal (Berggren, Eriksson, Fahlke, & Balldin, 2002). Compared to serotonin, dopaminergic neurons are similarly reduced after chronic alcohol abuse. Alcoholics' prefrontal regions display decreased dopamine transmission (Narendran et al., 2014). Lack of prefrontal dopamine could impair executive functions such as attention, working memory, behavioral flexibility, and risk/reward behaviors leading to less inhibition and ability to focus. For example, mice with chronic ethanol exposure have difficulties completing tasks involving significant utilization of orbitofrontal cortex, such as reversal learning tasks (Badanich, Becker, & Woodward, 2011).

Alcohol abuse has profound effects on acetylcholine levels; this neurotransmitter is specifically involved with memory formation and cognition. Specifically, Korsakoff's syndrome patients have presented with a reduction in the number of neurons containing acetylcholine in the nucleus basalis of Meynert in the basal forebrain while chronic alcohol abusers have exhibited similar cholinergic dysfunction within neurons located in the CA1 region of the hippocampus; both regions are associated with memory impairment (Nevo & Hamon, 1995).

Alcohol abuse also has a significant negative effect on GABA. Research suggests that chronic alcohol use can lead to a reduced number of GABA receptors in the brain with associated dysfunction in the efficient binding of GABA to its receptors, eventually resulting in alcohol tolerance (Wong et al., 2003). These neurochemical changes within the brain possibly contribute to tolerance and dependence within alcohol abusing individuals (Nevo & Hamon, 1995). GABA dysfunction forces the body to compensate for GABA inefficiency. When alcohol is withheld from a chronic alcohol abuser, the brain has too few GABA receptors to balance out excitatory neurotransmitters resulting in hyperexcitability, a characteristic of alcohol withdrawal and dependence. GABA's depletion within the brain is associated with an imbalance of excitatory and inhibitory neurotransmission, resulting in possible hyperexcitability as well.

Structural and neuroanatomical evidence of chronic alcohol abuse provides us with a better understanding of where and how alcoholism affects brain functioning. Postmortem studies on brains of problem drinkers show lower overall volume of white matter and weight. More specifically, alcoholics were more likely to suffer from neuronal loss in the cerebral cortex, cerebellum, and the hypothalamus (Trivedi et al., 2013). In an

older study, researchers found sulci widening in 40-60 year alcoholics was comparable to 70-90-year-old controls (Fein et al., 1990), suggesting advanced progression of neurodegeneration. Long-term alcohol abuse also yielded differential white matter results by gender. In addition to generalized white and gray matter reductions in brain volume among alcoholics against controls, female alcoholics had significantly diminished white matter integrity in frontal and temporal regions specifically, whereas males exhibited diminished white matter integrity in the corpus callosum (Ruiz et al., 2013). Thus, research has demonstrated that long-term alcohol abuse can negatively affect white matter volume.

The prefrontal regions of the brain also seem to be largely affected by long-term problematic alcohol consumption. One explanation asserts that the disruption of cerebellar neuronal activity that is typically associated with chronic alcohol abuse can cause brain dysfunction in regions such as the prefrontal cortex (Sullivan, Rosenbloom, & Pfefferbaum, 2000). Not surprisingly, alcoholics have been shown to demonstrate frontal-executive deficits when undergoing neuropsychological testing (Wollenweber et al., 2014). Such research has also been confirmed within mouse models such that ethanol dependent mice had significantly more reversal learning errors than their non-ethanol dependent counterparts (Badanich, Becker, & Woodward, 2011). Mice-models provide us with a clearer understanding of how early-life exposure to alcohol can disrupt normative hippocampal functioning. Pyramidal cells are neurons found within the amygdala, cerebral cortex, and hippocampus that relay excitatory responses to the prefrontal cortex. Exposure to alcohol at early ages is predictive of pyramidal cell death not found in healthy controls (Miki, Harris, Wilce, Takeuchi, & Bedi, 2004). Rats

exposed to chronic high levels of ethanol had more mitochondrial edema within hippocampal neurons than their healthy counterparts leading to corresponding learning and memory deficits (Du et al., 2014).

Another primary focus of neuropsychological alcoholism studies has been the medial temporal lobe, or more specifically the hippocampus. It is believed that memory formation first begins within the hippocampus and then is relayed throughout the cortical regions of the brain. Individuals with alcohol dependence are likely to exhibit impairments related to hippocampal size and function. Recent research demonstrated that patients with alcohol dependence had smaller right hippocampal regions than those of individuals without alcohol dependence (Ozsoy, Candan, & Esel, 2013). Ozsoy et al. also found that age of problem drinking onset was also a significant factor in predicting hippocampal volume. Individuals with adolescent-onset of alcohol abuse had smaller hippocampal volumes than individuals who had begun problem drinking later in adulthood. Further, adolescent-onset alcoholism was associated with smaller right and left hippocampal volume than healthy controls (De Bellis et al., 2000). However, another study found only the alcoholism effect and not the early age of onset effect on hippocampal volume (Laakso et al., 2000). Thus, age of onset may be a significant moderator when examining alcoholism's effect on memory formation but this needs further confirmation.

Functional imaging studies on cognitive deficits after prolonged alcohol abuse have discovered significant cerebellar dysfunction. Alcoholic patients have also been shown to utilize their cerebellar regions more inefficiently compared to non-alcoholic controls (Parks et al., 2003). Such dysfunction was associated with slower self-paced

finger tapping compared to normal controls, reflecting possible motor dysfunction effects. Past literature has highlighted how increased chronic ethanol exposure is associated with significant changes in Purkinje neurons within the cerebellum (Sullivan et al., 2003). Specifically, chronic ethanol exposure predicted a decrease in median path lengths in dendritic arbors and a reduction of the number of synapses among Purkinje cells. Research has demonstrated that alcohol-dependent individuals who have recently become abstinent experience a reduction in neural connectivity between the prefrontal cortex and the inferior cerebellum compared to non-alcohol abusing controls (Rogers, Parks, Nickel, Katwal, & Martin, 2012). A similar lack of synchrony between neural pathways has also been present in alcoholic populations between the cerebellum and posterior cingulate regions (Chanraud et al., 2011). These findings continue to illustrate how prolonged alcohol abuse can negatively affect processing speed capabilities.

Researchers, who are convinced that long-term alcohol abuse can harm the brain, believe that a variety of medical conditions resulting from alcohol abuse can also secondarily lead to cerebral dysfunction. Individuals can contract liver cirrhosis and vitamin deficiencies that negatively affect brain activity and cognitive functioning. Specifically, hepatic encephalopathy occurs when liver damage becomes so great that the liver itself is no longer able to remove toxic substances from the blood stream (Waldron-Perrine & Adams, 2014). Chronic build-up of toxic substances within an individual's blood such as ammonia have been associated with increased levels of lethargy, personality changes, and cognitive deficits. Notable cognitive detriments linked to hepatic encephalopathy include a reduction in psychomotor, visuomotor, and executive performance. Conversely, alcohol abusers routinely present with thiamine deficiency.

This vitamin deficiency is commonly associated with Wernicke-Korsakoff syndrome, which is commonly marked by disorientation, confusion, indifference, inattentiveness, and ataxic gait disturbances. Alternatively, some alcoholics never develop either of these common side effects of alcoholism and still suffer non-age related cognitive dysfunction (Harper, 2009).

CHAPTER FOUR

ASSESSMENT OF NEUROPSYCHOLOGICAL FUNCTION

Neurocognitive impairments are most directly tested and measured using neuropsychological testing. Strides within brain imaging technology have increased the need for more validated and reliable measures for neurocognitive testing. The two most prevalent cognitive impairments found within alcoholic participants are visuospatial ability and executive functioning (Fox, Coltheart, Solowij, Michie, & Fox, 2000; Sullivan et al., 2000). Researchers commonly use a host of neuropsychological measures to adequately measure the most prominent abilities present within an individual's cognitive skills. Neuropsychological assessments test participants across a host of domains usually including: verbal memory, immediate memory, executive functioning, and visuospatial ability (Bates, Labodizzuvie, & Voelbel, 2002). Research studies analyzing the effect of alcohol on overall cognitive ability primarily utilize the Mini Mental State Exam (MMSE), the Montreal Cognitive Assessment (MOCA), or the Wechsler Adult Intelligence Scale (WAIS-IV) to screen for general cognitive functioning.

The MMSE is a very brief screener for cognitive impairment, which takes around five to ten minutes to fully administer. Items comprised within the MMSE assess orientation to time and place, attention, mathematical calculation, language, immediate memory, and delayed memory (Strauss, Sherman, & Spreen, 2006). Despite well-documented clinical utility of the MMSE, research has demonstrated that low educational levels or low intelligence of participants may increase the risk of misclassifying cognitively normal individuals as impaired. Furthermore, the MMSE is considered less than ideal in assessing for mild impairment in psychiatric populations. The MMSE may

be overly sensitive towards verbal items and therefore does not properly measure tasks related to attention, problem-solving, visual-spatial ability and mood. Other criticisms of the MMSE emphasize that the assessment lacks diagnostic specificity, as low MMSE only hint at the possibility of changes in cognition and health. Finally, significant level of measurement error and variation in change in annual scores reflect the MMSE's limited utility in analyzing the progression of a cognitive disease within a patient or a specific patient population.

The Montreal Cognitive Assessment (MOCA) is another popular cognitive assessment utilized to screen for dementia and cognitive impairment. The MOCA typically takes around ten minutes to complete as it was initially designed to screen for Mild Cognitive Impairment. Domains on the MOCA include: short term memory, visuospatial ability, executive functioning, attention, working memory, language, and orientation to time and place (Ismail, Rajji, & Shulman, 2010). The MOCA has been praised for its increased sensitivity in comparison to the MMSE in terms of identifying cognitive complaints within individuals who do not yet experience functional impairment (Smith, Gildeh, & Holmes, 2007). Research has demonstrated that 73% of Mild Cognitive Impairment (MCI) patients score in the abnormal range of the MOCA but in the normal range for the MMSE (Nasreddine et al., 2005). However, the MOCA does not allow for diagnostic specificity, as researchers cannot differentially characterize functioning within each specific cognitive domain. Despite the clinical utility of the MOCA, there is limited evidence that associates the MOCA with neuroimaging indices (Paul et al., 2011).

Another prominent neuropsychological assessment used to assess cognitive ability is the Wechsler Adult Intelligence Scale Fourth Edition (WAIS-IV). The test itself is based on a four-factor structure to analyze: Verbal Comprehension, Working Memory, Perceptual Organization, and Processing Speed (Strauss, Sherman, & Spreen, 2006). Because the WAIS-IV contains ten core subtests, researchers typically select specific subtests such as Digit Span in concordance with other neuropsychological assessments to screen for overall cognitive ability. The Wechsler scales have been widely praised for their reliability and their validity in assessing the four core domains. However, researchers suggest utilizing more abbreviated neuropsychological measures when participant stamina and focus is concerned. Thus, it would seem more reasonable to employ a neuropsychological assessment that could analyze similar domains comprehensively within a shorter time period when dealing with clinical populations involved in outpatient treatment. One such neuropsychological measure is the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS: Randolph, 1998).

The RBANS, originally adapted to analyze dementia, consists of five domains: immediate memory, visuospatial/constructional, language, attention, and delayed memory. One of the key utilities of the RBANS is that it is highly correlated with longer neuropsychological assessments, such as the Wechsler Adult Intelligence Scale (r = .75), but it only requires thirty minutes to complete (Hartman, 2009). Some evidence suggests that the RBANS might be more sensitive toward impairment than Wechsler tests (Strauss, Sherman, & Spreen, 2006). After extensive clinical testing, the RBANS was deemed a validated measure of cognitive differences between Alzheimer's and Huntington's disease patients (Randolph, Tierney, Mohr, & Chase, 2010), and other

forms of cognitive dysfunction such as schizophrenia and bipolar disorder (Dickerson et al., 2004). Research within the last decade has also revealed that total RBANS performance is one of the better neuropsychological measures in predicting total brain volume (Paul et al., 2011). More specifically, the RBANS was a significant indicator of reduced medial temporal lobe volume within individuals suffering from cognitive impairment (England, Gillis, & Hampstead, 2014). The RBANS also contains measurement domains of attention (Digit Span and Coding) that involve the exercise of primarily prefrontal brain regions. Thus, the RBANS could be considered a possibly useful assessment for analyzing cognitive deficits within alcoholic populations. Surprisingly, no alcohol studies have utilized the RBANS as a measure for neuropsychological analysis of long-term alcoholic patients.

One factor that may moderate the effect of alcohol abuse on cognitive function is the duration of alcohol abuse, i.e., how long an individual has been suffering from alcohol abuse. Ralph Ryback's continuum theory (1971) hypothesizes that cognitive faculties diminish as a function of the duration of alcohol abuse. One study of note did not substantiate Ryback's continuum theory although participants within the study only included mild to moderate abusers of alcohol (Horner, Waid, Johnson, Latham, & Anton, 1999). Research has explicitly measured how the duration of abstinence (Stavro et al., 2013) and duration of abuse (Cairney, Clough, & Jaragba, 2007) affects cognitive functioning within chronic alcohol populations, but there is a dearth of literature directly analyzing the effect of years of alcohol abuse on specific aspects of cognitive functioning.

Additionally, another potentially significant factor in the effect of alcohol abuse on cognition is the age of alcohol dependence onset. Previous but limited research has illustrated that age of onset did not predict any significant differences on neuropsychological performance (Kist, Sandjojo, Kok, & van den Berg, 2014) when comparing early (<25 years), middle (25-44 years) to late (>45 years) ages of onset. Nor did a comparison of early to middle age of onset of alcohol dependence find neuropsychological differences (Kok, 2014). However, there may be difficulties in classifying age of onset precisely for these comparisons due to self-report biases. Additionally, research has indicated that profound alcohol abuse may significantly harm brain maturation up to twenty-two years of age, therefore categorical variables measuring age of onset may need to be modified in order to accommodate the pertinent neurocognitive critical period that may be affected by alcohol abuse (Silveri, 2012; Bennett & Baird, 2006). However, existing research has also demonstrated that full brain maturation may be individually reflective of genetics and environmental factors and therefore a definitive cut-off at age of brain maturation may not be fully generalizable (Arain, Haque, Johal, Mathur, Nel, Rais, Sandhu, & Sharma, 2013).

There seems to be a lack of consistency within the literature regarding age of onset and alcohol abuse. Scientists highlighted how such findings might be confounded by possible problems involving classification, namely how later onset alcohol populations may have been misrepresented within previous studies. Namely, older individuals who initiate alcohol abuse later in life are rarely considered in research. Thus, although recent findings have demonstrated that age of alcohol abuse onset is not

significantly associated with detriments cognitive faculties, follow-up studies are needed to more concretely understand this particular finding.

It should be noted that this study served as a continuation of a previous research study (Abeyesinhe, 2014). Briefly, Abeyesinhe conducted neurocognitive assessment on a recovering alcoholic population using the RBANS and discovered that individuals recovering from alcohol abuse scored significantly lower on RBANS subtest scores of list learning and figure copy, indicating worse performance on immediate verbal memory and visuospatial ability. This current study analyzed if the duration of drinking within a similar alcoholic population is associated with pronounced cognitive deficits, as literature is relatively scarce in identifying if the duration of drinking has a significant effect on cognitive functioning.

This current study identified individual factors of chronic alcoholism that may predict neuropsychological performance on the RBANS. Specifically, we analyzed how the age of drinking initiation and amount of alcohol consumed per day affected cognitive functioning.

CHAPTER FIVE

AIMS AND HYPOTHESES

The primary aim of this study was to determine if age at which individuals began to drink and the amount of alcohol consumed per day had a significant impact on indices of attention, immediate memory, and delayed memory. These specific indices were chosen due to the abundance of evidence suggesting that chronic alcoholism affects memory capacity and attentional capabilities, whereas domains of language, and visuospatial abilities do not seem to be as affected. Chronic alcoholism targets frontal regions of the brain, which are essential for initiating and maintaining attention, and medial temporal regions which are vital for memory capacity. Thus, we hypothesized that individuals who initiated alcohol usage during childhood and who were heavier drinkers would score significantly worse than individuals who initiated drinking behaviors during adolescence or adulthood and who reported drinking less per day. We hypothesized that the age of their first drink and amount of alcohol consumed would not have a significant effect on other cognitive indices (language and visuospatial function).

An exploratory aim was to determine if "problem drinking" age (subjective determination of when drinking became problematic) had an effect on overall cognitive functioning in conjunction with drinks consumed per day. Within the structured interview at the end of the RBANS assessment, participants were asked when they perceived their alcohol abuse started causing dysfunction within daily living. It was hypothesized that participants who initiated problem-drinking behaviors during childhood and who consumed heavier amounts of alcohol per day would score significantly worse than participants who began to engage in problem drinking in adolescence or adulthood and

who consumed less drinks per day. Additionally, it was hypothesized that problem drinking in conjunction with drinks per day would be predictive of worse overall functioning on indices of attention, immediate memory, and delayed memory.

CHAPTER SIX

METHODS

Participants and Procedures

Fifty-four participants were recruited from the outpatient Chemical Dependency Partial Hospitalization Program (CDPHP) at the Loma Linda University Behavioral Medical Center (LLUBMC). During the program, participants completed an inpatient detoxification program at the LLUBMC. Participants falling within the age range of 20-89 were included. Additionally, all patients included within this study were able to speak and understand English fluently. Of the 54 patients enrolled, two were excluded due to missing data leaving 52 for the study analyses. Four additional participants were excluded utilizing the outlier labelling rule to help correct for the positive skewness of the data. All patients who were enrolled had an accompanying primary diagnosis of alcohol use disorder (AUD).

Participants who were admitted to the LLUBMC for chemical dependency strictly for alcohol usage were eligible to participate in the study within the first two days of outpatient treatment. Before a clinical researcher administered the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS), patients completed a series of informed consent forms detailing the core purpose of the study while also explaining that their participation in the study was completely voluntary and anonymous. Shortly after the administration of the RBANS, patients completed a structured interview to compile demographic information as well as occupational status, alcohol/drug use history, and medical history. The RBANS assessment and interview were completed at the same appointment as when informed consent was obtained.

Patient Demographic Information

The demographic characteristics of participants are shown in Table 1. Information on age, gender, race, marital status, education, and income were acquired upon CDPHP admission. Twenty-four males (M = 48.13 years, SD=11.84) and 24 females (45.63 years, SD=8.76) were recruited from a local alcohol outpatient program. The majority of participants were Caucasian (63%), married (58%), and relatively well educated with approximately 42% earning college or graduate degrees. Participants also had relatively high to middle class incomes as approximately 47% of participants reported earning around $80,000 annually.

Table 1. Patient Demographics

Total N = 48	N ~ (%)
Gender	
Male	24 (50)
Female	24 (50)
Race	
Caucasian	31 (63)
Hispanic	17 (35)
African American	1 (2)
Other	0 (0)
Marital Status	
Married	28 (58)
Separated	2 (4)
Divorced	3 (6)
Single	3 (6)
Remarried	10 (21)
Widowed	2 (4)
Education	
High School	10 (21)
Some College	18 (38)
Bachelor's or Associate's	15 (29)
Master's/Doctorate	6 (13)
Income	
<10k	3 (6)
10-35k	6 (13)
35-60k	9 (17)
60-80k	8 (17)
80k+	22 (47)
	Mean
Age	46.88 years (SD = 10.46)

Instruments

Neuropsychological Assessment of Cognitive Functioning

Randolph's RBANS (1998) is a neuropsychological assessment used to test the cognitive status of individuals suffering from neurological diseases or head trauma. One of the core advantages to using the RBANS is its brevity. The RBANS takes approximately 30 minutes to administer, as opposed to other cognitive assessments that require a much longer duration to fully administer.

The RBANS is comprised of five indices (immediate memory, delayed memory, visuospatial ability, language, and attention) and twelve subtests (list learning, story memory, figure copy, line orientation, digit span, symbol digit coding, picture naming, semantic fluency, list recall, list recognition, story recall, and figure recall). All index scores are comprised of two subtests except for the delayed memory domain, which consists of four subtests. The RBANS total score provides an overall outcome statistic for an individual's overall neuropsychological functioning. In addition to the total score, individual subscale scores for immediate memory, visuospatial ability, language, attention, and delayed memory were calculated. RBANS raw scores are scaled according to age-weighted norms and percentiles. All subtests are given a subtest raw score. Raw scores of subtests within each domain are added and converted to an age-corrected index score. Index scores were also converted to percentile scores, according to the age-based normative conversions from the RBANS manual.

Immediate Memory

The immediate memory domain assesses an individual's ability to remember and

recall a small amount of information directly after it has been presented. The immediate memory domain was assessed using two subtests: list learning and story memory. List learning consists of a list of 10 unrelated words, read for immediate recall over four trials, with a maximum score of 40. Words compiled within the list learning section are considered moderate-high imagery words with relatively low age of acquisition. High levels of imagery and low age of acquisition of these words is considered helpful in reducing education effects on neuropsychological performance and allows for easing language translation difficulties. The story memory subtest is comprised of a 12-item story, read for immediate recall over two trials, for a total maximum score of 24. Scoring is hinged upon verbatim recall. The stories within the different forms of the RBANS all follow similar structure.

Visuospatial Ability

The visuospatial domain prompts participants to examine, comprehend, and recreate spatial relations. Notably, this domain assesses participants' ability to mentally rotate objects, estimate distance and depth, and navigate the surrounding environment. The subtests used to analyze visuospatial/constructional ability were as follows: figure copy and line orientation. The figure copy subtest prompts participants to draw an exact copy of a complex figure comprised of geometric shapes. The figure is comprised of 10 components, and a structured simplified scoring guide, which provides for a maximum score of 20. It should be noted that an additional detailed scoring guide was published in 2008 to allow for more objectivity and increase inter-rater reliability in scoring this component of the RBANS assessment. Within the line orientation subtest, participants

are presented with an arrangement of 13 lines, beginning at a common point of origin and fanning out across 180 degrees. Each item consists of two target lines that are shown beneath the overall arrangement. Subjects must correctly identify which two lines match with the overall arrangement. Line orientation consists of 10 items, each comprised of two matching lines, for a total maximum score of 20.

Language

The language domain prompts participants to execute communication skills to verbally name and retrieve previously learned material. Two subtests are comprised within this domain are picture naming and semantic fluency. Picture naming is considered a confrontation-naming task, with 10 line drawing objects that the participant must name. In the semantic fluency subtest, participants are allotted one minute to provide as many examples from a semantic category as possible (e.g., animals).

Attention

The RBANS attention domain assesses an individual's ability to select a component of information to focus on in subsequent processing and integration tasks. The attention domain prompts the participant to manipulate previously presented material (visual and oral) that has been stored within the individual's short term memory. This domain includes the following subtests: digit span and coding. In the digit span subtest, subjects are asked to repeat a series of numbers, with stimulus items increasing in length from 2 digits to 9 digits. The items are presented in order of length (shortest to longest), and the test itself is discontinued when the participant fails consecutively at a given string

length. In comparison, coding is an assessment of an examinee's processing speed that is very similar to the Digit Symbol subtest of the Wechsler scales. Subjects are asked to fill in digits matching with corresponding shapes on a coding key as fast as they can. After practice items are completed, participants have 90 seconds to complete as many items as possible.

Delayed Memory

The delayed memory domain of the RBANS requires participants to recall information for an extended length of time. These subtests are presented to the participants approximately 20 minutes after initial presentation. The subtests included within the delayed memory domain are: list learning free recall, list learning recognition, story memory free recall, and figure free recall. The list learning free recall subtest is a free recall of the words from the initial list learning subtest, whereas the list learning recognition subtest is a yes/no recognition task of the original list learning subtest with 10 foils. The story memory free recall subtest is a free recall task from the original story memory subtest. The figure free recall subtest is a free recall task from the original figure copy.

Total Scale

The total scale is the overall outcome statistic for an individual's overall neuropsychological functioning as comprised by all indices of the RBANS (Attention, Immediate Memory, Delayed Memory, Visuospatial/Constructional, and Language).

Additionally, it should be noted that all RBANS index scores were categorized according to typical classification of borderline aptitude: Impaired (standard score < 80) and not impaired (standard score ≥ 80). These categorical outcome variables were utilized for logistic regression outcome variables, whereas RBANS index scores were utilized as continuous outcome variables within multiple Analyses of Covariance (ANCOVAs).

Demographic Data

A brief patient interview was conducted following administration of the RBANS to gather patient information including age, gender, years of education, and income. Years of education was coded as a continuous variable while income was coded categorically with the following values: <10k, 10-35k, 35-60k, 60-80k, and 80+k.

History of Alcohol Use

Substance abuse history was collected from the clinical interview as well. Variables of interest include: age of alcohol initiation, general number of drinks consumed per day during the climax of alcohol abuse, and age of problem drinking initiation.

Age of Alcohol Initiation

Age of alcohol initiation (AI) was obtained from the initial clinical interview. This variable described at what age participants consumed their first alcoholic beverage. This predictor variable was categorized into three distinct ages: childhood (13 years of

age and younger), adolescence (14 to 17 years of age) and early adulthood (18 years of age and older).

Age of Problem Drinking

Additionally, participants were asked at what age drinking became a "problem" for them (age of problem drinking initiation; APD) during the structured clinical interview after completing the RBANS. This variable was categorized into the same distinct categories as alcohol initiation: childhood (13 years of age and younger), adolescence (14 to 17 years of age) and early adulthood (18 years of age and older).

Drinks per Day

Subjects were asked to indicate how many drinks they typically consumed per day (DPD) prior to entering outpatient treatment. Despite the expected variability in subject drinking behavior, alcoholic beverages were quantified utilizing drinking conversions provided by the NIH normative drinking conversions. For ANCOVA analyses, DPD was categorized utilizing a median split: "light" drinking (11 drinks per day or fewer) and heavy drinking (12 drinks per day or greater). A median split was utilized for these analyses because most participants in this study far surpassed the CDC's heavy drinking criteria (15 drinks/week for men, 8 drinks/week for woman) per day (CDC, 2013).

CHAPTER SEVEN

RESULTS

Normality Distribution of Independent Variables

We examined the normality distributions of our independent variables and outcome variables. We found that problem drinking age was normally distributed, whereas age of alcohol initiation and drinks per day were skewed. To correct for skewness, outliers were extracted from age of alcohol initiation and drinks per day through the utilization of the outlier-labeling rule. Specifically, three outliers were extracted from age of alcohol initiation and one outlier was extracted from drinks per day. After extracting these outliers, both age of alcohol initiation and drinks per day were normally distributed according to common cutoff criterion of current research but violated the Shapiro-Wilks test of normality (Kim, 2013). Hence, the data presented in this study displayed a positive skew for both Age of Initiation (AI) and Drinks per Day (DPD) which was included as a limitation in the current study.

Independent Variables of Interest

Descriptive statistics calculated for the three variables of interest (age at first drink, drinks per day, and problem drinking age) are shown in Table 2. Descriptive statistics were also calculated for the RBANS indices, as shown in Table 3.

Table 2. Independent Variable Descriptive Statistics

Variable	N	Mean	SD	Range
Age at first drink	48	15.47 years	3.98	[7,25]
Drinks per day	48	12.17 drinks	5.51	[2,22]
Problem age	22	34.95 years	12.70	[15,64]

Table 3. RBANS Descriptive Statistics

Cognitive Domain	N	M	SD	Range
Attention	48	95.25	15.93	[60,118]
Visuospatial/Constructional	48	86.67	16.72	[50,126]
Language	48	95.60	14.92	[60,132]
Immediate Memory	48	91.56	14.35	[61,117]
Delayed Memory	48	92.19	14.73	[56,124]
Total Scale	48	89.60	13.50	[63,119]

Categorization of Age at First Drink

For our Analyses of Covariance (ANCOVAs), AI was categorized into three categories whereas DPD was categorized into two categories. AI was categorized as follows: childhood (13 years of age and younger), adolescence (14 to 17 years of age) and early adulthood (18 years of age and older), See Table 4 for specific frequencies for each AI category. In terms of DPD, a median split was performed on drinks per day, which yielded two groups (11 drinks per day or less, and 12 drinks per day or more). There are no current CDC criteria for heavy alcohol usage above and beyond the current classification for heavy drinking and the great majority of participants in this current study far surpassed such initial heavy alcohol usage classification. Thus, it was necessary to utilize a median split to classify drinking amount with this study through a median split. See Table 5 for specific frequencies.

Table 4. Frequency Statistics for Age at First Drink

Category	N	Percent of Sample
Childhood	13	27.08
Adolescence	25	52.08
Early Adulthood	10	20.83

Table 5. Frequencies for Drinks per Day

Category	N	Percent of Sample
Low	25	52.10
High	23	47.90

RBANS Performance Group Differences

ANCOVAs were utilized to examine if differences existed within age of alcohol initiation and its association with drinks per day on RBANS index scores while controlling for both years of education and gender. One of the key limitations of this study was that our predictor variables do present with a positive skew and would be considered non-normally distributed according to the Shapiro Wilk's Test of Normality. As explained previously, three outliers were extracted according to the outlier labelling rule to help alleviate the positive skew of the data.

The first ANCOVA model examining the RBANS attention index did not reflect any significant main effects of AI or DPD, or interaction effects between AI and DPD, $p > .05$. The ANCOVA on the RBANS immediate memory index was significant, [F (7,40) = 3.36, $p < .01$, $r^2 = .37$], revealing a significant interaction effect of AI and DPD [F (2, 40) = 3.46, $p<.05$]. Pairwise comparison post-hoc tests revealed that individuals who began drinking in childhood and were heavier drinkers (M = 96.85, SD = 10.43) reported significantly higher scores on the immediate memory index than individuals who also initiated drinking during childhood but were considered lighter drinkers (M = 89.43, SD = 9.43), $p < .05$, see Figure 1. Additionally, participants who initiated drinking during childhood and who were considered heavier drinkers (M = 96.85, SD = 10.43) reported significantly higher scores on the immediate memory index than individuals who initiated drinking in adolescence and who also were classified as heavier drinkers (M = 88.18, SD = 19.85), $p < .05$, see Figure 1. Finally, individuals who initiated drinking during childhood and were classified as heavy drinkers (M = 96.85, SD = 10.43) reported significantly higher scores on the immediate memory index than individuals who initiated

drinking during adulthood and were considered heavier drinkers (M =84.75, SD = 11.32), $p < .05$, see Figure 1.

There were no significant main or interactions effects of AI or DPD, $p > .05$, on the RBANS delayed memory index. Additionally, there were no significant effects of AI or DPD on the remaining RBANS indices of language and visuospatial ability, $p > .05$. As a follow-up analysis, RBANS index scores were dichotomized into two groups and categorized according to standard neuropsychological benchmarks, with standard scores of 80 and above considered "normal" and scores from 79 and below considered "impaired." Logistic regression analyses were conducted to investigate whether AI and/or DPD were significant predictors of cognitive impairment. Within each regression model, education and gender were controlled within the first block of each model. However, our regression analyses revealed no significant effects of AI, DPD or the interaction between the two in predicting cognitive impairment on any RBANS index, $p > .05$.

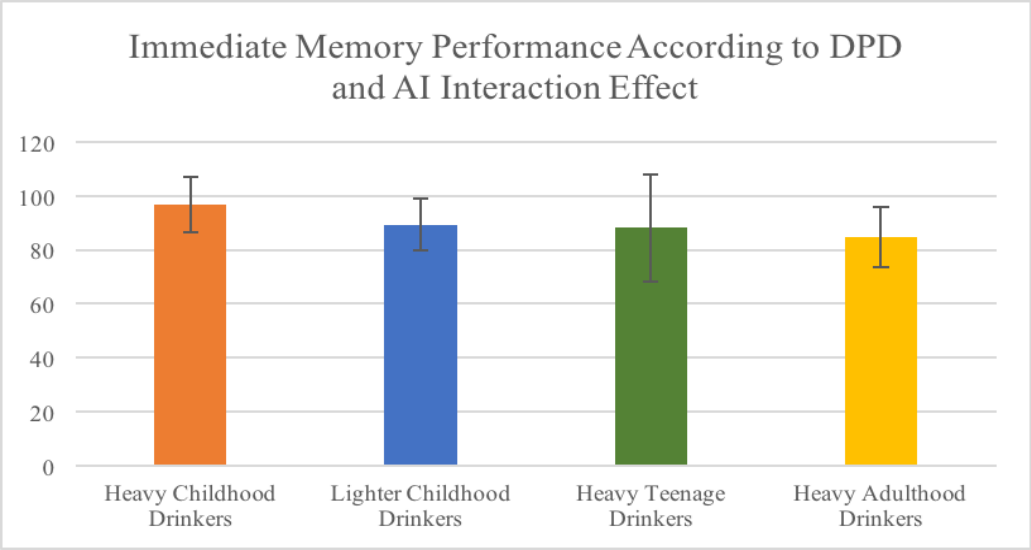

Figure 1. Immediate Memory Performance Interaction Effect

Exploratory Analyses

As an exploratory analysis, we replicated the same analyses above, replacing AI with the APD predictor variable. Because APD was added as a variable in the middle of the study, fewer participants provided this information. Thus, the data for this variable could not be considered generalizable and not enough individuals completed this portion of the current study to conduct appropriate analysis and subsequently make responsible conclusions of the data. However, it should be noted that the correlation between AI and APD was not significant ($r = .12$, $p > .05$). This suggests that there may be discrepancies between the age at which participants consumed their first alcoholic beverage (AI) and when their alcohol abuse subjectively began.

CHAPTER EIGHT

DISCUSSION

This study analyzed cognitive functioning of 48 alcohol-dependent individuals enrolled with the outpatient Chemical Dependency Partial Hospitalization Program at an academic medical center in southern California. Data was collected for this study from May 2013 until May 2016.

The primary purpose of this study was to analyze if prolonged alcohol usage within an alcohol-dependent population would be associated with marked cognitive deficits not accounted for by the general aging process. Research has suggested that chronic alcoholism may affect several domains of cognitive functioning such as memory, attention, and executive functioning.

Our primary hypothesis, which predicted that individuals suffering from alcoholism who had initiated drinking earlier in life would exhibit deficits in cognitive functioning compared to those who initiated drinking behaviors in adolescence or early adulthood was not confirmed. Conversely, individuals who drank more and began drinking in childhood demonstrated significantly better performance in immediate memory in comparison to individuals who drank less and who initiated into drinking later in life. These findings are profoundly counterintuitive and reflect, in our view, pervasive limitations in this study.

Limitations

One of the main limitations of the current study was that the independent variables utilized lacked the necessary variance that is essential to conducting a

scientifically sound experiment. The lack of a control group was a major flaw; a sample of participants who were not alcohol abusers at any stage of their life could have provided a baseline reference in terms of how chronic alcohol abusers perform on a neuropsychological measure in comparison to controls. Furthermore, throughout the study, there was a significant difficulty in finding individuals who had initiated drinking behaviors throughout their lifespan. The overwhelming majority of participants began engaging in alcoholism within their late childhood and early teenage years. Thus, finding an adequate sample of participants reporting a wide enough range in age of alcohol initiation was difficult.

The independent variables incorporated into this study could also have been better defined and operationalized. Age of alcohol initiation may not be a good predictor of cognitive sequelae, as it only provides information as to when an individual drank their first alcoholic beverage and not when their alcohol abuse commenced. Thus, problem drinking (APD) may be more relevant for alcoholism research as it prompts participants to state when their alcoholism truly began. It should also be noted that AI and APD were not significantly correlated, possibly indicating that the age at which a participant's drink was first consumed was not associated with, and therefore not a good proxy for the age at which their alcoholism began.

Defining relative amounts of alcohol consumed was also a significant challenge. To our knowledge, there is no current classification system for specifically heavy drinking over and above what the NIH has defined as "heavy drinking". More specifically, individuals who have surpassed the NIH criteria for heavy drinking are not provided with further classification as to how their drinking levels relate to other

consequences of alcoholism. Thus, criteria for our classification of DPD into high and low drinkers could not be standardized to literature-based classifications because none to our knowledge existed beyond the initial NIH criteria for heavy drinking itself. The median split of 11 drinks per day is somewhat arbitrary; the overwhelming majority of participants included in this study could be considered uncommonly heavy drinkers regardless of their classification in this study alone. As such, the lack of moderate drinkers or non-drinkers in our study resulted in a heavily skewed distribution. Our research indicates that although AI and DPD may be important aspects of an individual's neurocognitive development, an individual's cognitive ability is shaped and molded by a variety of biological and environmental factors.

Aside from the primary independent variables of interest, there were additional limitations within the study. The sample size gathered within this current study is rather small (n = 48), and thus, statistical power to examine the hypotheses may have been inadequate. Specifically, data regarding the specifics of alcohol abuse history could have been more comprehensive. Only 22 participants were asked about the age in which they initiated "problem drinking". Furthermore, participants varied in terms of days of sobriety. Although the discrepancy between days of sobriety was rather small, perhaps an increased length of time within a treatment setting may have yielded different results than those that were compiled within this current study. Data within this current study was cross sectional; perhaps participants may improve upon their RBANS scores after their two week stay in outpatient treatment has been completed. Finally, the RBANS does not provided what is considered a "true" measure of frontal-executive functioning. A frontal

measure (i.e. Wisconsin Card Sorting Task) could have provided important data for such a population that typically presents with difficulties within frontal-related tasks.

Finally, participants included within this experiment often presented with a history of chronic generalized substance abuse as well, despite only being admitted for alcoholism. A more complicated substance abuse history may very well contribute to more profound and lasting cognitive deficits, whereas this study examined alcohol use history alone.

Research Implications and Future Directions

This study suggests that more research is needed regarding how chronic alcoholism contributes to cognitive functioning. Future research targeting duration of problematic alcohol abuse needs to incorporate a more clearly defined independent variable regarding the individual's true age of alcoholism initiation, similar to the age of problem drinking variable (APD). After this age is identified, it is essential to gather as much information regarding the individual's abuse history. Such information could include: attempts at sobriety, length of sobriety, drinks consumed per day, type of alcohol consumed, family history of alcohol abuse, and timeline of other substances used/abused. Each individual's substance history is unique and the more information gathered regarding such history would provide a more comprehensive attempt at analyzing how true age of alcohol initiation affects cognitive functioning later in life. Additionally, it is imperative that future research incorporate a control group for which to compare and contrast cognitive performance between alcoholics and the normative population. It would also be suggested that a more comprehensive battery of neuropsychological

assessments that includes a true frontal-executive measure be utilized. Measures such as the Wisconsin Card Sorting Task (Berg, 1948), Trails B (Army Individual Test Battery, 1944), and The Delis-Kaplan Executive Function System (Delis, Kaplan, & Kramer, 2001) would be appropriate for accurate assessment of frontal-related neuropsychological measures pertinent for future directions.

CHAPTER NINE

CONCLUSION

In summary, this study analyzed the effect of chronic alcohol consumption on cognitive functioning in individuals participating in an outpatient alcohol addiction treatment program at Loma Linda University's Behavioral Medicine Center. This study found that alcohol abusers who initiated alcohol use during childhood and who were classified as heavy drinkers demonstrated better performance on a task of immediate memory functioning in comparison to outpatient alcohol abusers who initiated alcohol use during adolescence and adulthood and who were lighter drinkers. These findings are reflective of limitations in this study which included independent variables which were poorly defined and lacked variance, an absence of a normative control group, and a small sample size. Given the experimental issues within this study, more research is needed to identify the true relationship between age of alcohol initiation and cognitive functioning later in life. Future research directions should target compiling a more detailed history of participant's substance abuse in addition to including more frontal-executive related measures to accurately assess tasks that incorporate frontal lobe function, an area in which alcoholics routinely espouse neuropsychological deficiencies. Future research is required to fully understand the mechanisms behind how alcoholism may affect cognitive functioning with respect to normative cognitive aging.

REFERENCES

Abeyesinhe, S. (2014). Cognitive Function in an alcohol treatment population. (Unpublished doctoral dissertation). Loma Linda University, Loma Linda, CA.

American Heart Association. (2015). *Alcohol and heart health.* Retrieved from: http:// http://www.heart.org/HEARTORG/GettingHealthy/NutritionCenter/HealthyEating/Alcohol-and-Heart-Health_UCM_305173_Article.jsp

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, D.C.

Anstey, K. J., Mack, H. A., & Cherbuin, N. (2009). Alcohol consumption as a risk factor for dementia and cognitive decline: meta-analysis of prospective studies. *The American Journal of Geriatric Psychiatry : Official Journal of the American Association for Geriatric Psychiatry*, *17*(7), 542–55. doi:10.1097/JGP.0b013e3181a2fd07

Army Individual Test Battery. (1944). *Manual of Directions and Scoring*. Washington, DC: War Department, Adjutant General's Office.

Arain, M., Haque, M., Johal, L., Mahtur, P., Nel, W., Rais, A., Sandhu, R., & Sharma (2013). Maturation of the adolescent brain. *Neuropsychiatric Disease and Treatment, 20*(3), 449-461.

Badanich, K. A., Becker, H. C., & Woodward, J. J. (2011). Effects of chronic intermittent ethanol exposure on orbitofrontal and medial prefrontal cortex-dependent behaviors in mice. *Behavioral Neuroscience*, *125*(6), 879–891. doi:10.1037/a0025922

Bartsch, A. J., Homola, G., Biller, A., Smith, S. M., Weijers, H. G., Wiesbeck, G. a., … Bendszus, M. (2007). Manifestations of early brain recovery associated with abstinence from alcoholism. *Brain*, *130*(1), 36–47. doi:10.1093/brain/awl303

Bates, M. E., Labouvie, E. W., & Voelbel, G. T. (2002). Individual differences in latent neuropsychological abilities at addictions treatment entry. *Psychology of Addictive Behaviors : Journal of the Society of Psychologists in Addictive Behaviors*, *16*(1), 35–46. doi:10.1037/0893-164X.16.1.35

Beatty W.W., Hames K.A., Blanco C.R., Nixon S.J., Tivis L.J. (1996) Visuospatial perception, construction and memory in alcoholism. *J Stud Alcohol 57*, 136–143.

Bennett, C.M, & Bair, A. A. (2006). Anatomical changes in the emerging adult brain: a voxel-based morphometry study. *Human Brain Mapping, 27*, 766-777.

Berg, E. A. . (1948). A simple objective technique for measuring flexibility in thinking . *J. Gen. Psychol. 3*9, 15-22.

Bijl, S., de Bruin, E. A., Kenemans, J. L., Verbaten, M. N., & Böcker, K. B. (2005). Effects of chronic alcohol consumption in a visual attention task and an auditory oddball task: an event-related potential study. *Alcoholism, Clinical and Experimental Research*, *29*(11), 2029–2038. doi:10.1097/01.alc.0000187163.52577.0d

Brokate, B., Hildebrandt, H., Eling, P., Fichtner, H., Runge, K., & Timm, C. (2003). Frontal lobe dysfunctions in Korsakoff's syndrome and chronic alcoholism: continuity or discontinuity? *Neuropsychology*, *17*(3), 420–428. doi:10.1037/0894-4105.17.3.420

Cairney, S., Clough, A., Jaragba, M., & Maruff, P. (2007). Cognitive impairment in Aboriginal people with heavy episodic patterns of alcohol use. *Addiction (Abingdon, England)*, *102*(6), 909–15. doi:10.1111/j.1360-0443.2007.01840.x

Chanraud, S., Pitel, A. L., Pfefferbaum, A., & Sullivan, E. V. (2011). Disruption of functional connectivity of the default-mode network in alcoholism. *Cerebral Cortex*, *21*(10), 2272–2281. doi:10.1093/cercor/bhq297

Cutting, J. (1978). Specific psychological deficits in alcoholism. *British Journal of Psychiatry*, *32*(8), 119–122. doi:10.1192/bjp.133.2.119

Centers for Disease Control and Prevention. (2013). *Alcohol and Public Health frequently asked questions.*

Cohen, J., Cohen, P., West, S. G., & Aiken, L. S. (2003). *Applied multiple regression/correlation analysis for the behavioral sciences* (3rd ed.). New York, NY: Taylor and Francis Group.

De Bellis, M. D., Clark, D. B., Beers, S. R., Soloff, P. H., Boring, A. M., Hall, J., … Keshavan, M. S. (2000). Hippocampal volume in adolescent-onset alcohol use disorders. *The American Journal of Psychiatry*, *157*(May), 737–744. doi:10.1176/appi.ajp.157.5.737

Delis, D.C., Kaplan, E., Kramer, J.H. (2001). *The Delis-Kaplan Executive Function System*. San Antonio, TX, Psychological Corporation.

Desfosses, M., Meadows, H., Jackson, M., & Crowe, S. F. (2014). The Relationship Between Neuropsychological Functioning and Mental Health Outcomes of Chronic Alcohol Users Involved in Counselling: Prediction of Treatment Outcome. *Australian Psychologist*, *49*, 287–296. doi:10.1111/ap.12071

Dickerson, F., Boronow, J. J., Stallings, C., Origoni, A. E., Cole, S. K., & Yolken, R. H. (2004). Cognitive functioning in schizophrenia and bipolar disorder: Comparison of performance on the Repeatable Battery for the Assessment of Neuropsychological Status. *Psychiatry Research*, *129*, 45–53. doi:10.1016/j.psychres.2004.07.002

Du, A., Jiang, H., Xu, L., An, N., Liu, H., Li, Y., & Zhang, R. (2014). Damage of hippocampal neurons in rats with chronic alcoholism. *Neural Regeneration Research*, *9*(17), 1611–1615. doi:10.4103/1673-5374.141787

England, H. B., Gillis, M. M., & Hampstead, B. M. (2014). RBANS memory indices are related to medial temporal lobe volumetrics in healthy older adults and those with mild cognitive impairment. *Archives of Clinical Neuropsychology : The Official Journal of the National Academy of Neuropsychologists*, *29*(4), 322–8. doi:10.1093/arclin/acu012

Fama, R., Pfefferbaum, A., & Sullivan, E. V. (2004). Perceptual learning in detoxified alcoholic men: contributions from explicit memory, executive function, and age. *Alcoholism, Clinical and Experimental Research*, *28*(11), 1657–1665. doi:00000374-200411000-00010 [pii]

Fein, G., Bachman, L. E. A., Fisher, S., Davenport, L., & Francisco, S. (1990). Cognitive Impairments in Abstinent Alcoholics.

Fergusson, D. M., Boden, J. M., & Horwood, L. J. (2013). Alcohol misuse and psychosocial outcomes in young adulthood: Results from a longitudinal birth cohort studied to age 30. *Drug and Alcohol Dependence*, *133*(2), 513–519. doi:10.1016/j.drugalcdep.2013.07.015

Fernández-Serrano, M. J., Pérez-García, M., Schmidt Río-Valle, J., & Verdejo-García, A. (2010). Neuropsychological consequences of alcohol and drug abuse on different components of executive functions. *Journal of Psychopharmacology (Oxford, England)*, *24*, 1317–1332. doi:10.1177/0269881109349841

Flannery, B., Fishbein, D., Krupitsky, E., Langevin, D., Verbitskaya, E., Bland, C., … Zvartau, E. (2007). Gender differences in neurocognitive functioning among alcohol-dependent Russian patients. *Alcoholism: Clinical and Experimental Research*, *31*(5), 745–754. doi:10.1111/j.1530-0277.2007.00372.x

Fox, A. M., Coltheart, M., Solowij, N., Michie, P. T., & Fox, G. A. (2000). Dissociable cognitive impairments in problem drinkers. *Alcohol and Alcoholism (Oxford, Oxfordshire)*, *35*(1), 52–54. doi:10.1093/alcalc/35.1.52

Harper, C. (2009). The neuropathology of alcohol-related brain damage. *Alcohol and Alcoholism*, *44*(2), 136–140. doi:10.1093/alcalc/agn102

Hartman, D. E. (2009). Wechsler Adult Intelligence Scale IV (WAIS IV): return of the gold standard. *Applied Neuropsychology*, *16*(Wais Iv), 85–87. doi:10.1080/09084280802644466

Hewett, L. J., Nixon, S.J., Wagner-Glenn, S., & Parsons, O.A. (1991). Verbal fluency deficits in female alcoholics. *Journal of Clinical Psychology*, *47*(5), 716-721.

Horner, M. D., Waid, L. R., Johnson, D. E., Latham, P. K., & Anton, R. F. (1999). The relationship of cognitive functioning to amount of recent and lifetime alcohol consumption in outpatient alcoholics. Addictive Behaviors, 24, 449–453.

Ismail, Z., Rajji, T. K., & Shulman, K. I. (2010). Brief cognitive screening instruments: An update. *International Journal of Geriatric Psychiatry*, *25*(2), 111–120. doi:10.1002/gps.2306

Kim, H.Y. (2013). Statistical notes for clinical researchers: assessing normal distribution (2) using skewness and kurtosis. *Restorative Dentistry*, 52-54.

Kist, N., Sandjojo, J., Kok, R.M., & van den Berg, J.F. (2014). Cognitive functioning in older adults with early, late, and very late onset alcohol dependence. *International Psychogeriatrics*, *26*(11), 1863-1869.

Koskinen, S. M., Ahveninen, J., Kujala, T., Kaprio, J., O'donnell, B. F., Osipova, D., … Rose, R. J. (2011). A Longitudinal Twin Study of Effects of Adolescent Alcohol Abuse on the Neurophysiology of Attention and Orienting. *Alcoholism: Clinical and Experimental Research*, *35*(7), 1339–1350. doi:10.1111/j.1530-0277.2011.01470.x

Kok, R.M. (2014). Treatment of alcohol use disorders in the elderly: an overview of RCTs. *International Psychogeriatrics*, *26*(11), 1767-1770.

Laakso, M., Vaurio, O., Savolainen, L., Repo, E., Soininen, H., Aronen, H. ., & Tiihonen, J. (2000). A volumetric MRI study of the hippocampus in type 1 and 2 alcoholism. *Behavioural Brain Research*, *109*(2), 177–186. doi:10.1016/S0166-4328(99)00172-2

Mayo Clinic. (2014). *Diseases and conditions: Alcoholism*. Retrived from: http:// http://www.mayoclinic.org/diseases-conditions/alcoholism/basics/complications/con-20020866?p=1.

Miki, T., Harris, S. J., Wilce, P. a, Takeuchi, Y., & Bedi, K. S. (2004). Effects of age and alcohol exposure during early life on pyramidal cell numbers in the CA1-CA3 region of the rat hippocampus. *Hippocampus*, *14*(1), 124–34. doi:10.1002/hipo.10155

Mukamal, K. J., Kuller, L. H., Fitzpatrick, A. L., Mittleman, M. A., & Siscovick, D. S. (2014). Prospective Study of Alcohol Consumption and Risk of Dementia in Older Adults, *289*(11), 1405–1413.

Narendran, R., Mason, N. S., Paris, J., Himes, M. L., Douaihy, A. B., & Frankle, W. G. (2014). Decreased Prefrontal Cortical Dopmaine Transmission in Alcoholism. *American Journal of Psychiatry*, *171*(8), 881–888.

Nasreddine, Z. S., Phillips, N. a., Bédirian, V., Charbonneau, S., Whitehead, V., Collin, I., … Chertkow, H. (2005). The Montreal Cognitive Assessment, MoCA: A brief screening tool for mild cognitive impairment. *Journal of the American Geriatrics Society*, *53*(4), 695–699. doi:10.1111/j.1532-5415.2005.53221.x

National Institute on Alcohol Abuse and Alcoholism. (2010). *Beyond Hangovers*.

National Institute on Alcohol Abuse and Alcoholism. (2014). *Alcohol Facts and Statistics*.

Nevo I., Hamon M. (1995) Neurotransmitter and neuromodulatory mechanisms involved in alcohol abuse and alcoholism. *Neurochem Int*, 26, 305–336.

Noel, X., Van der Linden, M., Schmidt, N., Sferrazza, R., Hanak, C., Le Bon, O., De Mol, J., et al. (2001). Supervisory attentional system in nonamnesic alcoholic men. Archives of General Psychiatry, 58, 1152–1158.

Oscar-Berman, M., & Marinković, K. (2007). Alcohol: effects on neurobehavioral functions and the brain. *Neuropsychology Review*, *17*(3), 239–57. doi:10.1007/s11065-007-9038-6

Ozsoy, S., Candan, A., & Esel, E. (2013). Hippocampal volumes and cognitive functions in adult alcoholic patients with adolescent-onset, *47*, 9–14.

Panza, F., Solfrizzi, V., Seripa, D., Imbimbo, B. P., & Pilotto, A. (2010). Peripheral Antioxidant Markers in Mild Cognitive Impairment and its Progression to Dementia, *21*, 1179–1183. doi:10.3233/JAD-2010-101224

Parks, M. H., Morgan, V. L., Pickens, D. R., Price, R. R., Dietrich, M. S., Nickel, M. K., & Martin, P. R. (2003). Brain fMRI activation associated with self-paced finger tapping in chronic alcohol-dependent patients. *Alcoholism, Clinical and Experimental Research*, *27*(4), 704–711. doi:10.1097/01.ALC.0000062759.14944.CF

Paul, R., Lane, E. M., Tate, D. F., Heaps, J., Romo, D. M., Akbudak, E., … Conturo, T. E. (2011). Neuroimaging signatures and cognitive correlates of the montreal cognitive assessment screen in a nonclinical elderly sample. *Archives of Clinical*

Neuropsychology : The Official Journal of the National Academy of Neuropsychologists, *26*(5), 454–60. doi:10.1093/arclin/acr017

Perreira, K. M., & Sloan, F. a. (2002). Excess alcohol consumption and health outcomes: A 6-year follow-up of men over age 50 from the health and retirement study. *Addiction*, *97*, 301–310. doi:10.1046/j.1360-0443.2002.00067.x

Pinder, R. M., & Sandler, M. (2004). Alcohol, wine and mental health: focus on dementia and stroke. *Journal of Psychopharmacology (Oxford, England)*, *18*(4), 449–56. doi:10.1177/0269881104047272

Randolph, C. (1998). *Repeatable Battery for the Assessment of Neuropsychological Status*. San Antonio, TX: The Psychological Corporation.

Randolph, C., Tierney, M. C., Mohr, E., & Chase, T. N. (2010). Journal of Clinical and Experimental Neuropsychology The Repeatable Battery for the Assessment of Neuropsychological Status (RBANS): Preliminary Clinical Validity The Repeatable Battery for the Assessment of Neuro- psychological Status (RBANS): Prelim, (August 2014), 37–41.

Ritz, L., Segobin, S., Le Berre, A. P., Lannuzel, C., Boudehent, C., Vabret, F., … Beaunieux, H. (2014). Brain structural substrates of cognitive procedural learning in alcoholic patients early in abstinence. *Alcoholism, Clinical and Experimental Research*, *38*(8), 2208–16. doi:10.1111/acer.12486

Rogers, B. P., Parks, M. H., Nickel, M. K., Katwal, S. B., & Martin, P. R. (2012). Reduced Fronto-Cerebellar Functional Connectivity in Chronic Alcoholic Patients. *Alcoholism: Clinical and Experimental Research*, *36*(2), 294–301. doi:10.1111/j.1530-0277.2011.01614.x

Ruitenberg, A., van Swieten, J. C., Witteman, J. C. M., Mehta, K. M., van Duijn, C. M., Hofman, A., & Breteler, M. M. B. (2002). Alcohol consumption and risk of dementia: the Rotterdam Study. *Lancet*, *359*(9303), 281–6. doi:10.1016/S0140-6736(02)07493-7

Ruiz, S. M., Oscar-Berman, M., Sawyer, K. S., Valmas, M. M., Urban, T., & Harris, G. J. (2013). Drinking History Associations with Regional White Matter Volumes in Alcoholic Men and Women. *Alcoholism: Clinical and Experimental Research*, *37*(1), 110–122. doi:10.1111/j.1530-0277.2012.01862.x

Ryback, R. (1971). The continuum and specificity of the effects of alcohol on memory: A review. Quarterly Journal of Studies on Alcohol, *32*, 995–1016.

Silveri, M. M. (2012). Adolescent brain development and underage drinking in the United States: Identifying risks of alcohol use in college populations. *The Harvard Review of Pschiatry*, *20* (4), 189-200.

Smith, T., Gildeh, N., & Holmes, C. (2007). The Montreal cognitive assessment: Validity and utility in a memory clinic setting. *Canadian Journal of Psychiatry, 52*(5), 329–332.

Stampfer, M. J., Kang, J. H., Chen, J., Cherry, R., & Grodstein, F. (2005). Effects of moderate alcohol consumption on cognitive function in women. *The New England Journal of Medicine, 352*(3), 245–53. doi:10.1056/NEJMoa041152

Stavro, K., Pelletier, J., & Potvin, S. (2013). Widespread and sustained cognitive deficits in alcoholism: A meta-analysis. *Addiction Biology*, *18*(2), 203–213. doi:10.1111/j.1369-1600.2011.00418.x

Stott, D. J., Falconer, A., Kerr, G. D., Murray, H. M., Trompet, S., Westendorp, R. G. J., … Ford, I. (2008). Does low to moderate alcohol intake protect against cognitive decline in older people? *Journal of the American Geriatrics Society*, *56*(May 2002), 2217–2224. doi:10.1111/j.1532-5415.2008.02007.x

Strauss, E., Sherman, E.S., & Spreen, O. (2006). *A compendium of neuropsychological tests:Administration, Norms, and Commentary* (3rd ed.) . New York: Oxford University Press.

Sullivan, E. V., Rosenbloom, M. J., Pfefferbaum, A., Park, M., & Sciences, B. (2000). Pattern of Motor and Cognitive Deficits in Detoxified Alcoholic Men. *Alcoholism, Clinical and Experimental Research, 24*(5), 611–621.

Sullivan, E. V., Harding, A. J., Pentney, R., Dlugos, C., Martin, P. R., Parks, M. H., … Pfefferbaum, A. (2003). Disruption of frontocerebellar circuitry and function in alcoholism. *Alcoholism, Clinical and Experimental Research*, *27*(2), 301–309. doi:10.1097/01.ALC.0000052584.05305.98

Sullivan, E. V., & Pfefferbaum, A. (2005). Neurocircuitry in alcoholism: a substrate of disruption and repair. *Psychopharmacology*, *180*(4), 583–94. doi:10.1007/s00213-005-2267-6

Trivedi, R., Bagga, D., Bhattacharya, D., Kaur, P., Kumar, P., Khushu, S., … Singh, N. (2013). White matter damage is associated with memory decline in chronic alcoholics: A quantitative diffusion tensor tractography study. *Behavioural Brain Research, 250,* 192–198. doi:10.1016/j.bbr.2013.05.001

Waldron-Perrine, B., & Adams, K. M. (2014). Alcohol-related neuropathology. In M. W. Parsons & T. A. Hammeke (3rd Ed.), *Clinical neuropsychology*: *A pocket handbook for assessment* (363-382). Washington D.C.: American Psychological Assocaition.

Wollenweber, F. a, Halfter, S., Brügmann, E., Weinberg, C., Cieslik, E. C., Müller, V. I., … Eickhoff, S. B. (2014). Subtle cognitive deficits in severe alcohol addicts--do

they show a specific profile? *Journal of Neuropsychology*, *8*(1), 147–53. doi:10.1111/jnp.12001

Wong, D. F., Maini, A., Rousset, O. G., & Brasić, J. R. (2003). Positron emission tomography--a tool for identifying the effects of alcohol dependence on the brain. *Alcohol Research & Health : The Journal of the National Institute on Alcohol Abuse and Alcoholism*, *27*, 161–173.

Yeh, P. H., Simpson, K., Durazzo, T. C., Gasdrinski, S., & Meyerhoff, D. J. (2009). Tract-based spatial statistics (TBSS) of diffusion tensor imaging data in alcohol dependence: Abnormalities of the motivation neurocircuitry. *Psychiatry Research-Neuroimaging 173*,(1), 22-30. doi:10.1016/j.psychchresns.2008.07.012

Zanjani, F., Downer, B. G., Kruger, T. M., Willis, S. L., & Schaie, K. W. (2013). Alcohol effects on cognitive change in middle-aged and older adults. *Aging & Mental Health, 17*(1), 12–23. doi:10.1080/13607863.2012.717254

Zuccalà, G., Onder, G., Pedone, C., Cesari, M., Landi, F., Bernabei, R., & Cocchi, a. (2001). Dose-related impact of alcohol consumption on cognitive function in advanced age: results of a multicenter survey. *Alcoholism, Clinical and Experimental Research*, *25*(12), 1743–8. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11781507

APPENDIX A

RESEARCH STRUCTURED INTERVIEW QUESTIONNAIRE

NAME (first and last name):

_____

Today's Date_____

Demographic Questions

1. Date of Birth: (mm/dd/yyyy) _____/_____/_____
2. Gender: Male/Female (circle one)
3. What city do you live in?

   City: _____
4. Approximately how long have you lived at this address? (Years/Months) _____
5. Race/Ethnicity: (please check one)

   _____ Caucasian
   _____ Hispanic
   _____ African-American/Black
   _____ Asian
   _____ Other (specify) _____
6. What is your marital status:
   [ ]1 Married
   [ ]2 Remarried
   [ ]3 Widowed
   [ ]4 Separated
   [ ]5 Divorced
   [ ]6 Single, never married
7. What is your highest level of education?
   [ ]1 Grade School or Less Education
   [ ]2 High school diploma or equivalent (trade school certificate)
   [ ]3 Some college or Vocational, Business or Trade School
   [ ]4 Associate or Bachelors college degree
   [ ]5 Masters or Doctoral degree

8. Do you have a profession, trade, or skill? _____

9. What is your employment status?
    a. Employed full time
    b. Employed part time
    c. Student
    d. Unemployed

10. What type of health insurance do you currently have?
    [ ]1 I don't have any health insurance
    [ ]2 Private Insurance, Blue Cross, HMO
    [ ]3 Medicare/Medicaid/Medical
    [ ]4 Champus/ Champus VA/other military
    [ ]5 Other type of insurance: _____

11. What is your average household income?
    a. <10,000
    b. 10,000-<35,000
    c. 35,000-<60,000
    d. 60,000-<80,000
    e. 80,000+

Drug and Alcohol history questions

12. Have you previously been in treatment prior for alcohol addiction or drug rehab?
    No, skip to question 16
    Yes

13. How many times previously have you been in treatment for alcohol addiction or drug rehab?_____

14. Did you terminate any of the previous treatments early?
    No, skip to question 16
    Yes

15. Why did you choose to terminate the previous treatments early?
    _____
    _____

16. At what age did you begin drinking alcohol? _____

17. On average, how many drinks do you have per day? _____

18. On average, how many drinks do you consume in one sitting? _____

19. At what age did alcohol become a problem for you?
    _____

20. Are there any other drugs you take either regularly or even on occasion?

    a. Heroin: (#times)_____
       (#years)_____
    b. Methadone: (#times)_____
       (#years)_____
    c. Benzodiazepines (Xanax, Valium, etc.):
       (#times)_____
       (#years)_____

    d. Cocaine: (#times)_____
       (#years)_____
    e. Amphetamines (meth, speed, etc.):
       (#times)_____
       (#years)_____
    f. Cannabis: (#times)_____
       (#years)_____
    g. Hallucinogens (LSD, PCP, mushrooms, etc.):
       (#times)_____
       (#years)_____
    h. Inhalants: (#times)_____
       (#years)_____

21. When was the last time that you had any alcohol or took drugs, other than the medications given to you in treatment?_____

22. How important is it for you to complete treatment for your alcohol/drug problems? (0-5; 0:not at all, 5:extremely important) _____

General Health Questions

23. Have you ever had an injury to your brain? (like concussion, trauma..etc.)
    No
    Yes, please
specify_____

24. Are you being treated for any medical illness at this time?
    No
    Yes, please
    specify_____

25. Have you ever been diagnosed with a chronic medical illness? (like cancer, diabetes, etc.)

    No
    Yes, please
    specify_____

26. Have you ever been diagnosed with a mental health condition (like depression, bipolar…etc.)

    No
    Yes, please
    specify_____

27. Have you ever been diagnosed with a learning disability? (like ADHD, reading disability, writing disability, etc.)

    No
    Yes, please
    specify_____

28. Are you currently taking any medication?
    No
    Yes, please
    specify_____

Stress

29. What do you feel is your current stress level on a scale of 0-10 with 10 the worst and 0 no stress at all?

Legal History

30. Was this admission prompted by the criminal justice system?
    No
    Yes, please
    specify_____

31. Are you on probation or parole?
    No
    Yes

Family History

32. Do you have any relatives that have/had a significant drinking or drug use problem?

    a. Mother
    b. Grandmother
    c. Grandfather
    d. Uncle
    e. Aunt

    f. Father
    g. Grandmother
    h. Grandfather
    i. Uncle
    j. Aunt

    k. Siblings