MILD TRAUMATIC BRAIN INJURY REHABILITATION: A MODEL FOR ADHD TREATMENT

APPROVED BY SUPERVISORY COMMITTEE

Cheryl Silver, Ph.D.

Richard Fulbright, Ph.D.

Karen Brewer-Mixon, Ph.D.

DEDICATION

I would like to thank the members of my Graduate Committee and my parents.

MILD TRAUMATIC BRAIN INJURY REHABILITATION: A MODEL FOR ADHD TREATMENT

by

ASHLEY NICOLE YATES

THESIS

Presented to the Faculty of the School of Health Professions

The University of Texas Southwestern Medical Center

Dallas, Texas

In Partial Fulfillment of the Requirements

For the Degree of

MASTER OF CLINICAL REHABILITATION COUNSELING

Abstract

Attention Deficit Hyperactivity Disorder (ADHD) is a condition that affects approximately 4.4% of adults in the U.S. (Kessler et al., 2006) and is most commonly treated with psychopharmacological interventions. More recently, non-pharmacological interventions have been developed for ADHD. Cognitive Behavioral Therapy (CBT) has emerged as an efficacious treatment for ADHD and typically consists of training compensatory strategies and the use of external aids. The aims of this thesis were to examine the similarities between ADHD and mild Traumatic Brain Injury (mTBI) as well as review treatment options for mTBI and discuss their possible usefulness in treating ADHD. Based on the literature reviewed, there were striking similarities of deficits between ADHD and mTBI, specifically in the executive functioning of both. It is hypothesized that treatment for mTBI could also be beneficial for ADHD. Currently, some of the techniques used to treat ADHD and mTBI overlap. However in, cognitive rehabilitation (CR) for TBI, there is more emphasis on remediation of deficits compared to treatment of ADHD. Also, cognitive tasks for mTBI are more often completed in a real-life setting or as close to a real-life setting as possible. At this time, the literature regarding cognitive rehabilitation specific to mTBI is somewhat limited because it continues to be a growing field of literature. However, CR in general may be a beneficial treatment for the executive functioning deficits that also commonly affect ADHD.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF ABBREVIATIONS

ADHD– Attention Deficit/Hyperactivity Disorder

mTBI– Mild Traumatic Brain Injury

EF – Executive Functioning

CR-Cognitive Rehabilitation

CHAPTER ONE

Statement of the Problem

Executive function (EF) is a complex set of cognitive processes that coordinate to allow an individual to problem solve and effectively plan. EF impairments are well documented in individuals with Attention Deficit-Hyperactivity Disorder (ADHD). Executive dysfunction is also thought to be an important deficit associated with mild traumatic brain injury (mTBI). This paper focuses on the similarities in the executive function of these two disabilities and postulates that due to similarities in the two conditions, behavioral treatments effective for mTBI may also be useful for the treatment of ADHD.

Medication interventions for adults with ADHD are empirically supported for the improvement in the total number of ADHD symptoms. However, compared to healthy controls, individuals with ADHD remain significantly impaired, despite the decrease of some of their symptoms. Currently, medication treatments for ADHD do not fully address the EF deficits that are persistent and disruptive in these individuals' lives. Non-pharmacological treatments have been developed to address those symptoms of ADHD which are not improved by medications, but conflicting data exist regarding individual symptom outcome. On the other hand, a preliminary review of cognitive rehabilitation treatments for mTBI yielded promising results for an improvement of EF deficits. Given the similarities in EF deficits that adults with both of these disorders demonstrate, both groups may also benefit from similar behavioral intervention (e.g., cognitive rehabilitation) strategies.

This paper aims to (1) review the similarities between mTBI and ADHD; and (2) discuss the potential use of mTBI interventions for ADHD. It is hypothesized that cognitive interventions used for mTBI would be beneficial for individuals with ADHD.

CHAPTER TWO

Review of the Literature

Attention Deficit/Hyperactivity Disorder

Attention-Deficit/Hyperactivity Disorder (ADHD) is a behavioral disorder that includes symptoms of inattention, impulsivity, and hyperactivity (American Psychiatric Association, 2013; Biederman & Faraone, 2005). Within ADHD, there exist three subtypes, including Inattentive type, Hyperactive-Impulsive type, and Combined type (American Psychiatric Association, 2013). The subtypes of ADHD are a reflection of the multidimensional nature of attention. Each subtype reflects specific types of attention and behavior as well.

As its name implies, the Inattentive type of ADHD is characterized primarily by symptoms related to inattention (American Psychiatric Association, 2013). These symptoms may present as daydreaming, moving on to another task without completing the previous one, and poor listening skills (Murphy & Barkley, 1996). An individual diagnosed with ADHD, Hyperactive-Impulsive type, can display symptoms of both hyperactivity and impulsivity. Similar to the construct of attention, impulsivity also manifests in a variety of patterns. Symptoms of impulsivity displayed by individuals with ADHD will often include blurting out, risk-taking, and an inability to delay gratification (Barkley, 2006). The hyperactivity symptoms for individuals with ADHD typically include fidgeting and restlessness (Murphy & Barkley, 1996). An individual may be diagnosed with ADHD, Combined type, if his/her symptoms meet criteria from both the Inattentive and Hyperactive/Impulsive categories.

In addition to attentional deficits, many individuals with ADHD also experience symptoms of executive dysfunction (EF; Antshel et al., 2010; Barkley & Murphy, 2010; Brown, Reichel, & Quinlan, 2009; Martel, Nikolas, & Nigg, 2007). The EF deficits in adults with ADHD

often manifest as difficulty planning ahead, difficulty completing tasks on time, disorganization, and inefficient work due to careless errors (Solanto, 2014). The literature suggests that those with ADHD and significant EF deficits experience lower educational and occupational attainment compared to other individuals with ADHD who have less severe executive function deficits and to healthy controls. (Antshel et al., 2010; Barkley & Murphy, 2010; Biederman et al., 2006).

ADHD in the Workplace

EF deficits are thought to be at the core of difficulties in the work environment that many adults with ADHD experience. These difficulties in the workplace place a considerable burden on U.S. society. Doshi et al. (2012) found a cost of between $87 billion and $138 billion annually due to decreased productivity and income losses of adults with ADHD. These results may be related to those obtained by de Graaf et al. (2008) in which they found that adults with ADHD average 8.4 more days per year out of role, 21.7 days per year of decreased work quantity, and 13.6 days per year of decreased work quality relative to those of healthy controls. These data are measured by the World Health Organization (WHO) Disability Assessment Schedule (WHO-DAS 2.0; Chwastiak & Von Korf, 2003). Kessler et al. (2005) found similar results to de Graaf et al. (2008), reporting an average of 35 lost work days due to low work performance among adults with ADHD compared to healthy controls using the WHO Health and Work Performance Questionnaire (HPQ; Kessler et al., 2003). A limitation to these studies is that participants surveyed were not asked to specify what the specific obstacles were that resulted in decreased work productivity. It appears that not only are individuals with ADHD suffering as individuals due to their condition, but this condition also is a costly for others in the work environment and society at large.

Psychological Comorbidity

ADHD has a high comorbidity with other behavioral and psychiatric disorders. Children with ADHD commonly have comorbidities of Oppositional Defiant Disorder, Conduct Disorder, anxiety, and tic disorders (Pliszka, 2003). Additionally, there is much literature on the presence of comorbid learning disorders with ADHD. In a comprehensive literature review, Barkley (2006) culled studies with the most stringent criteria for learning disorders. In this review, he only included studies that used a regression equation suggested by Frick et al. (1991) to identify children with learning disorders. The Frick et al. (1991) equation determines the discrepancy between a child's predicted academic achievement and actual achievement while controlling for other factors such as age. Based on his literature review, Barkley concluded that among children with ADHD, 8-39% will likely have a reading disorder, 12-30% will likely have a math disorder, and 12-37% will likely have a spelling disorder. The lower end of Barkley's ranges is on par with the prevalence of learning disorders in the general population. These large ranges could be attributable to the varying methods among studies used in Barkley's review to determine the prevalence ranges of learning disorders among ADHD. On the other hand, the difficulties in attention children with ADHD experience could also lead to difficulties in learning.

ADHD persists into adulthood, and adults with ADHD have been shown to have higher incidence of comorbid psychiatric disorders. For example, in a 10-year prospective study, Biederman et al. (2006) found that when compared to controls, adults with ADHD were significantly more likely to have comorbid antisocial behaviors, substance dependence, a mood disorder, Tourette's Disorder, or an anxiety disorder. These results were similar to those of Kessler et al. (2006), with the exception that Kessler et al. (2006) also found a higher incidence of comorbid Intermittent Explosive Disorder.

Prevalence

Attention-Deficit/Hyperactivity Disorder has an overall prevalence rate of 5.2% in children worldwide (Polanczyk, Silva de Lima, Horta, Biederman, & Rohde, 2007). However, the Center for Disease Control estimates an even higher prevalence of nine percent in U.S. children (Center for Disease Control and Prevention, 2012). The difference in these two prevalence rates is likely due to the fact that the first study looked at worldwide prevalence and there is considerable variability in estimates of ADHD across various regions of the world. Polanczyk et al. (2007) found that there were significantly lower estimates of ADHD in the geographical regions of Africa and the Middle East compared to those in North America. These researchers speculated that the striking differences in prevalence among geographical regions are likely due to methodological differences in studies in each region. There could also be cultural factors that influence prevalence rates, such as willingness to seek treatment.

Kessler et al. (2006) estimated that the prevalence of adults with ADHD in the U.S. is 4.4%. Typically, longitudinal studies have found that approximately 30% of children diagnosed with ADHD will meet full diagnostic criteria at follow-up in adulthood. Although not all individuals diagnosed in childhood still meet diagnostic criteria at adult follow-up, most of them continue to have functional difficulties (Barbaresi et al., 2013; Biederman, Petty, Clarke, Lomedico, & Faraone, 2011). For example, Biederman et al. (2011) found that although 35% of their sample met full criteria for ADHD at adult follow-up, 43% remained symptomatic and just below the threshold for a full ADHD diagnosis.

Faraone, Biederman, and Mick (2006) suggest that the discrepancy in studies regarding the persistence of ADHD into adulthood is likely due to differing definitions of “persistence” among studies. They suggest that when persistence is determined as either meeting full DSM-IV

(American Psychiatric Association, 1994) criteria or exhibiting a sub-threshold number of symptoms at follow-up, the percentage rises to around 65% of adults who were diagnosed with ADHD as children. Some individuals may be misdiagnosed with ADHD in childhood; instead, they may have another disorder with overlapping symptoms such as anxiety. There could also be psychosocial factors involved that could impact a child's behavior. Another theory posed by Faraone (2000) is that ADHD does indeed persist into adulthood, but the symptoms as described in the DSM-IV do not necessarily reflect ADHD symptoms as they manifest in adults. Faraone (2000) gives the example that many of the symptoms are related to behaviors in the school environment. Furthermore, the studies regarding prevalence described above used criteria from DSM-IV. Criteria in the DSM-5 (American Psychiatric Association, 2013) have been modified and may better reflect ADHD symptoms in adulthood. To this writer's knowledge, there has not been a longitudinal follow-up study that investigates whether the rate of persistence of ADHD from childhood to adulthood has risen when DSM-5 criteria are utilized.

The literature suggests that there are substantial gender differences in ADHD prevalence. Ramtekkar, Riersen, Todorov and Todd (2010) found an overall male:female ratio of 2.28:1 in children and adults ages 7-29. They also found that the gender difference was greatest in adolescence with a ratio of 2.56:1. They suggested that it is possible that ADHD is under-diagnosed in the female population. On the other hand, the discrepancy could also be that ADHD is over-diagnosed in boys because their hyperactive symptoms are more difficult to manage by the parents or in a classroom setting. They also suggest that it could be because females with ADHD typically have more inattentive than hyperactive-impulsive symptoms. Therefore, girls with ADHD may be less disruptive, making it less likely that they are referred by schools or parents for treatment. This pattern may skew samples, resulting in identifying more males than

females because of this higher likelihood for parents to seek treatment for males with more disruptive symptoms (Ramtekkar, Reiersen, Tdorov, & Todd, 2010).

Etiology of ADHD

Neuroimaging and genetic research have provided compelling evidence for a biological etiology of ADHD. Castellanos et al. (2002) conducted a neuroimaging study using magnetic resonance imaging (MRI) with participants who consisted of medicated and non-medicated children with ADHD and controls. When compared to healthy controls, participants with ADHD were found to have significant reductions in total cerebral volume, all measures of cortical gray matter (total gray matter, as well as frontal, parietal, temporal, and occipital gray matter), and all measures of white matter (total white matter, as well as white matter in the frontal, parietal, temporal, and occipital regions). Specifically, the biggest differences between groups were noted in the caudate and the cerebellum. However, participants were scanned longitudinally over ten years and the caudates of participants with ADHD did not differ significantly from those of healthy controls by mid-adolescence. All other measures that had been significantly different at initial scans remained significantly different at the 10-year follow-up.

Some of the findings of reduced brain volume in children with ADHD are similar to findings with adults with ADHD. For example, Seidman et al. (2006) found that adults with ADHD had significantly less total gray matter, as well as lower prefrontal and anterior cingulate cortex volumes. Makris et al. (2007) similarly identified significantly less cortical thickness in adults with ADHD. The brain regions found to be thinner included the prefrontal cortex, lateral inferior parietal cortex, and cingulate regions. An MRI study comparing the scans of non-medicated adults with ADHD and controls found a significant difference in the volume of the orbitofrontal cortex, with reduced volumes in the adults with ADHD (Hesslinger et al., 2002).

Consistent with the findings of Castellanos et al. (2002), Hoogman et al. (2012) found a significant difference in total brain volume between adults who met ADHD criteria and those who did not. Hoogman et al. (2012) found that there was also an association between the total number of self-reported ADHD symptoms and the total brain volume of adults. When broken down into types of symptoms, inattentive symptoms were associated more with total brain volume than were hyperactive symptoms. These findings suggest that there is a negative correlation between ADHD symptoms and an individual's total brain volume. The consistent finding of reduced brain volume and this association of symptom severity and total brain volume of individuals with ADHD supports a neurological basis that underlies ADHD.

Diffusion tensor imaging is a relatively new MRI technique that is used to study white matter circuitry throughout the brain. Individuals with ADHD have been found to have reduced fractional anisotropy in the premotor cortex, right striatum, and right cerebral peduncle compared to healthy controls (Ashtari et al., 2005). Hamilton et al. (2008) also found reduced fractional anisotropy for individuals with ADHD in the corticospinal tract and the superior longitudinal fasciculus when compared to healthy controls. Although brain regions affected in individuals with ADHD vary among studies, it appears that there is a disruption of white matter integrity in individuals with ADHD.

Genetic studies also support a biological etiology for ADHD. Smalley et al. (2000) found that 55% of affected sibling pairs with ADHD also had at least one parent who had ADHD. Adoption studies have reported that the biological parents of individuals with ADHD scored significantly worse on measures of attention than adoptive parents of children with ADHD (Alberts-Corush, Firestone, & Goodman, 1986). Sprich et al. (2000) found that six percent of parents who adopted children with ADHD also had ADHD, whereas 18% of biological parents

of children with ADHD had also been diagnosed with ADHD. Twin studies further support the biological etiology of ADHD. Faraone et al. (2005) found a mean heritability rate of .76 when they averaged the results from 20 twin studies.

Outcomes

The literature suggests that educational and vocational outcomes in the lives of adults with ADHD (treated or untreated) are less favorable than for those in the population at large. Research has shown that lower educational levels and vocational under-attainment outcomes of adults with ADHD cannot be accounted for by intelligence level (Biederman et al., 2008). Biederman et al. (2008) compared adults with and without ADHD on measures of IQ as well as on educational and occupational outcomes. Using an ordered logistic regression, the researchers made predictions of participants' education levels from control participants' Full Scale Intelligence Quotients (FSIQs) from the Wechsler Adult Intelligence Scale-Third Edition (WAIS-III, 1997). The educational levels of only the controls were then used in an ordered logistic regression to predict what the occupational attainment should be. Occupational attainment was measured with the Hollingshead Four Factor Index of Social Status (Hollingshead, 1975). These predictors were then compared to the actual educational and occupational outcomes of both adults with ADHD and healthy controls. Based on participants' FSIQ scores, 84% of the adults with ADHD were expected to be college graduates, but only 50% actually attained that level. Based on predicted educational levels, 96% of participants with ADHD would have attained an occupational level of six or above on the Hollingshead Index (semi-professionals and technicians), but only 58% achieved this level (Biederman et al., 2008). The estimated and observed levels of education and occupation in individuals with ADHD

differed significantly from each other. On the other hand, in healthy controls, FSIQ significantly predicted educational level, and occupational level was significantly predicted by education.

A 16-year follow-up study of the long-term outcomes of men who were diagnosed with ADHD as boys had similar findings to Biederman et al. (2008). Compared to controls, adult males with ADHD had significantly lower socio-economic status, were significantly less likely to be college graduates, and reported significantly more family conflict (Biederman et al., 2012). These findings remained significant after controlling for comorbid psychiatric disorders. Galera et al. (2012) investigated the relationship between symptom severity in children with ADHD and educational, occupational, and socioeconomic outcomes at an 18-year follow-up. This study found greater symptom severity in childhood significantly positively associated with negative educational, socioeconomic, and occupational outcomes as adults.

Research has also found an association between having ADHD and negative life events (Garcia et al, 2012). In this study, negative life events were based on the Life Experience Survey (LES; Sarason, Johnson, & Siegel, 1978). This survey is based on frequently experienced stressful life changes and consists of events such as mortgage foreclosure, sleep disturbances, and history of arrests. Negative life events that were out of the individual's control, such as the death of a loved one, were not included in the analysis. In this study, they used a sample of 211 adults and found a positive correlation ($r = 0.22$; $p = .001$) between symptoms of inattention and adverse life events. They also found a positive correlation ($r = 0.21$; $p = .003$) between symptoms of hyperactivity and adverse life events. These significant associations may be a reflection of how the deficits which individuals with ADHD experience in everyday life may result in an increased likelihood of negative life events.

Treatment

Currently, there are both pharmacological and behavioral treatments available for individuals with ADHD. Typically, studies regarding ADHD treatment consider improvement to be a decrease in the total number of ADHD symptoms. There seems to be sufficient evidence that both pharmacological and behavioral treatments decrease the total or average number of ADHD symptoms for individuals with ADHD. Regarding long-term outcomes, Shaw et al. (2012) found in a systematic review that both behavioral and pharmacological treatments decrease drug use, antisocial behavior, suicide, obesity, and use of community services. They also found that adults who had received either type of treatment were found to have better academic achievement, social abilities, and vocational outcomes. However, researchers also found that of the various outcome measures, vocational outcomes were the least likely to improve with treatment. Shaw and colleagues suggest that the failure of treatment to impact vocational outcomes may be due to an accumulation of the effects of symptoms over the lifespan of an individual with ADHD.

Medication. Traditionally, stimulant medications have been the primary treatment for adults and children with ADHD. Stimulant medications have been associated with improvement of symptoms in individuals with ADHD when given over either short-term or long-term periods of time (American Academy of Pediatrics, 2001; Barbaresi et al., 2014; Coghill, 2010; Greenhill, Pliszka, & Dulcan, 2002; Wigal et al., 1999). Stimulant medications decrease symptoms of ADHD by increasing dopamine and norepinephrine in the brain and/or by preventing the re-uptake of these neurotransmitters, depending on the specific stimulant.

Stimulant medication improves executive functioning in adults with ADHD compared to adults with ADHD who have not taken stimulant medication (Biederman et al., 2008). However,

compared to healthy controls, individuals with ADHD taking stimulant medication still demonstrate a significant deficit in executive functioning (Biederman et al., 2008; Maruta, Spielman, Tseretopoulos, Hezghia, & Ghajar, 2014).

Non-stimulant medications have emerged as a viable treatment for symptoms of ADHD in the past decade or so (Michelson et al., 2003). Schulz et al. (2012) tested the effects of stimulant (methylphenidate) and non-stimulant (atomoxetine) medications on adolescents with ADHD. Both medications were associated with significant improvements in mean ADHD symptoms reported on the ADHD Rating Scale-IV (ADHD-RS-IV; DuPaul, Power, Nastopoulos & Reid, 1998). The amount of improvement did not differ significantly between the two types of medication.

There is less literature examining the effect of non-stimulant medication on EF deficits in adult ADHD. Using the Behavior Rating Inventory of Executive Function-Adult Version (BRIEF-A; Roth, Isquith, & Gioia, 2005), Adler, Clemow, Williams, and Durrel (2014) found a significantly lower level of self-reported executive dysfunction in adults with ADHD medicated with atomoxetine when compared to placebo.

Non-pharmacological treatment. Psycho-education and skills training were some of the first non-pharmacological interventions available for adults with ADHD which were found to be effective treatments (Wiggins, Singh, Getz, & Hutchins, 1999). Mindfulness training has also been associated with decreased symptoms of ADHD (Schoenberg et al., 2013). Coaching is a treatment for ADHD that is still currently being developed, but has been regarded as a promising intervention (Kubik, 2010).

Cognitive Behavioral Therapy (CBT) has recently gained support as an efficacious treatment for adults with ADHD compared to other psychological therapies (Chandler, 2013;

Mongia & Hechtman, 2012; Vidal-Estrada, Bosch-Munso, Nogueira-Morais, Casas-Brugue, & Ramos-Quiroga, 2012). Salakari et al. (2010) found after 10 weeks of a CBT-oriented group therapy, 44% of adults with ADHD had an improvement of symptoms. A significant improvement was determined to be any symptom reduction of 20% or higher as rated on the Brown Attention Deficit Disorder Scale (BADDS; Brown, 1996). The content of the CBT groups consisted of topics such as the neurological underpinnings of ADHD, motivation and initiation of activities, organization, attention, emotional regulation, memory, communication, impulsivity, psychiatric comorbidities, and self-esteem. Weiss et al. (2012) completed a study examining CBT and stimulant medications in adults with ADHD. They found that individuals with ADHD receiving CBT and medication had similar improvement in the average amount of ADHD symptoms compared to individuals receiving a placebo and CBT over a 20-week period. They had a similar threshold as Salakari et al. (2010) for what was considered improvement and included scores of at least a 25% decrease on the ADHD Rating Scale-IV (ADHD RS-IV; DuPaul, Power, Anastopoulos, 1998). They found that 63% of the group receiving CBT was considered improved by these standards.

Another study evaluated the effectiveness of CBT for adults stabilized on medication but still reporting residual symptoms of ADHD (Safren et al. 2010). These researchers compared CBT to relaxation techniques with educational support. The CBT participants completed 12 sessions of individual CBT which followed the treatment manual of Safren, Perlman, Sprich, and Otto (2005). The treatment consisted of three modules, the first being psychoeducation and training in organization, planning and problem solving. The first module consisted of techniques such as using a calendar and task list system to address organization and planning deficits and breaking large overwhelming tasks into smaller tasks to address problem solving deficits. The

second module consisted of techniques to reduce distractibility symptoms such as writing down a distraction rather than acting upon it. The third module consisted of cognitive restructuring techniques to improve an individual's ability to adapt to novel situations that they might find overwhelming or distressing. The researchers considered improvement as a 30% improvement on the ADHD Rating Scale-IV (ADHD-RS-IV; DuPaul, Power, Nastopoulos & Reid, 1998). The researchers also either considered a 2-point reduction, or participants being rated as a 1 or 2 on the Clinical Global Impression Scale as improvement. Fifty three percent of participants who received CBT had significantly more improvement on the Clinical Global Impression Scale (CGI; Busner & Targum, 2007) compared to the 23% of participants who improved after receiving relaxation training with educational supports. Examining degree of improvement, the researchers also found that 67% of the CBT group were considered significantly improved compared to 33% of the relaxation group were considered significantly improved.

Based on the behavioral intervention studies reviewed, CBT as a non-pharmacological treatment appears to have the most research support. However, the studies mentioned in this section only looked at total symptom improvement and did not investigate specific symptom improvements as opposed to overall symptom improvement. In addition, they did not address how symptom improvement affected long-term outcomes.

ADHD Specific Symptom Response to Treatment

A review of treatment studies for ADHD revealed that that most studies measured effectiveness of treatment by a decrease in total or average symptoms of ADHD. This becomes problematic as it is hard to distinguish which areas of ADHD-related cognitive impairment are actually improving with treatment. The studies discussed in this section will evaluate the influence that interventions have on the specific symptoms within ADHD. In the studies reported

below, the researchers not only investigated if total ADHD symptoms of individuals were decreasing, but also discuss which specific ADHD symptoms were significantly decreased.

Advokat (2010) completed a systematic review evaluating the effect that stimulant medication has on the cognitive performance of adults with ADHD. Advokat (2010) concluded that there is strong support for an improvement of sustained attention in adults with ADHD while taking stimulant medication. There was also some support for an improvement of retention of information. This review did not find that the common symptom of distractibility and planning deficits in adults with ADHD were consistently improved.

One study that addresses the treatment of EF deficits of adults with ADHD is that of Solanto, Marks, Mitchell, Wasserstein, and Koffman (2008). In this study, participants completed what is described as a metacognitive therapy aimed at decreasing the underlying EF deficits that commonly occur in ADHD. This treatment, lasting 9-12 weeks, targeted time management, behavioral activation, procrastination, organizational skills, and planning. The goal was for the participants to be able to complete the skills they learned in group, such as checking their daily planner, with the goal of those skills becoming automatic processes, occurring without the cues presented in treatment. Their outcome measures included the Conners Adult ADHD Rating Scale- Self Report: Long (CAARS-S:L; Conners, K., Erhardt, D., & Sparrow, E., 2004), BADDS (Brown, 1996), and the On Time Management, Organization and Planning Scale (ON-TOP) a self-report measure specifically designed for this study. Approximately 46% of the participants decreased from the clinical range to below clinical range on the inattentive scores of the CAARS-S:L. Effect sizes of pre and post treatment on the BADDS symptom clusters ranged from .449-.595 (moderate to strong effect size).They also found an effect size of .615 on the ON-

TOP measure. The authors of this study did not evaluate the maintenance of these improvements over time, however.

Another study by Virta et al. (2010) evaluated the effectiveness of CBT and cognitive training (CT) of adults with ADHD who were stable on ADHD medication but reported residual symptoms. In their analysis, they compared three groups, consisting of individuals who received CBT, individuals who received cognitive training, and controls. Their CBT participants completed 10 weekly sessions with topics of goals and symptoms of ADHD, attention, motivation and initiation of activities, organizing and planning, stress management and relaxation, self-esteem, and individually chosen topics. The CT group completed 20 twice-weekly computer sessions designed to train attention, executive functions, and working memory. Post-treatment, they found that compared to controls, participants who received CBT had significantly fewer total symptoms as measured by the Brown Attention Deficit Disorder Scale (BADDS; Brown, 1996). The participants who received CBT also had a significant decrease on the attention and memory subscales of the BADDS compared to controls post treatment. They did not, however, differ significantly on the activation (organizing, planning, prioritizing), effort (processing speed and ability to stay alert), or affect (managing emotions such as frustration tolerance) scales of the BADDS compared to controls.

The CNS Vital Signs (Gualtiere & Johnson, 2006) is a neuropsychological battery comprised of seven tasks that include verbal memory, visual memory, finger tapping, symbol-digit coding, the Stroop test, the shifting attention test, and a continuous performance test. This measure was used to assess changes in cognitive performance in each group in the Virta et al. (2010) study. The CBT participants did not show statistically significant improvements on the CNS Vital Signs (CNSVS; Gualtieri & Johnson, 2006), Clinical Global Impressions (CGI;

Busner & Targum, 2007) or the Quality of Life Enjoyment and Satisfaction Questionnaire (Q-LES-Q; Endicott, Nee, Harrison, & Blumenthal, 1993). On the other hand, the participants who received cognitive training as opposed to CBT did not have any significant improvements on self-report measures, but did have significant improvements on Task 3 and Task 5 of the cognitive training tasks.

Melby-Lervag and Hulme (2013) completed a 23-study meta-analysis to evaluate the effectiveness of working memory (WM) training programs such as CogMed and other non-commercial working memory programs for individuals with ADHD. They evaluated effectiveness based on immediate and long-term improvements of verbal and visuospatial working memory abilities. The researchers also evaluated the effect of working memory training on far-transfer measures such as word decoding, verbal abilities, and inhibitory processes. The researchers found large effect sizes for the improvement of verbal WM abilities and moderate effect sizes for visuospatial WM abilities, immediately after participants completed a WM program. On average, follow-up measures were done at nine months post intervention. The effect sizes for long-term follow-up were non-significant for verbal WM. On the other hand, effect sizes for the follow up at an average of five months after program completion were moderate and significant for visual WM measures. The researchers found that although there was support for an immediate improvement in WM abilities for a few months after completion of the program, improvements were not necessarily sustained long-term. Long-term improvement was defined by the average length of follow-up for the studies included in this meta-analysis, which was 9 months for verbal working memory and 5 months for visuospatial working memory.

Mild Traumatic Brain Injury

The Mild Traumatic Brain Injury Committee of the American Congress of Rehabilitation Medicine (ACRM) developed a definition for mild traumatic brain injury (mTBI) in 1993 and the criteria are listed below.

> (i)Any period of loss of consciousness; (ii) any loss of memory for events immediately before or after the accident; (iii) any alteration in mental state at the time of the accident (eg, feeling dazed, disoriented, or confused); and (iv) focal neurological deficit(s) that may or may not be transient; but where the severity of the injury does not exceed the following: (a) loss of consciousness of approximately 30 minutes or less; (b) after 30 minutes, an initial Glasgow Coma Scale (GCS) of 13-15; and (c) posttraumatic amnesia (PTA) not greater than 24 hours.

This definition was the first to include alteration of consciousness and PTA without requiring loss of consciousness. Furthermore, the ACRM definition was the first which stated that symptoms from a mTBI could be persistent and lead to functional disability. The definition altered the way the medical community perceived mild brain injury, and now mTBI is considered by many to be a potentially serious medical disability (Barth, Freeman, & Broshek, 2002).

The definition of mTBI was expanded and standardized by the World Health Organization (WHO) Collaborative on Mild Traumatic Brain Injury (Carroll, Cassidy, Holm, Kraus, & Coronado, 2004). The expanded WHO definition is listed below.

> MTBI is an acute brain injury resulting from mechanical energy to the head from external physical forces. Operational criteria for clinical identification include: (i) 1 or more of the following: confusion or disorientation, loss of consciousness for 30 minutes or less, post-traumatic amnesia for less than 24 hours, and/or other transient neurological abnormalities such as focal signs, seizure, and intracranial lesion not requiring surgery; (ii) Glasgow Coma Scale score of 13–15 after 30 minutes post-injury or later upon presentation for healthcare. These manifestations of MTBI must not be due to drugs, alcohol, medications,

> caused by other injuries or treatment for other injuries (e.g. systemic injuries, facial injuries or intubation), caused by other problems (e.g. psychological trauma, language barrier or coexisting medical conditions), or caused by penetrating craniocerebral injury. (Carroll et al., 2004, pp. 115)

It is estimated that at least 1.5 million people sustain a traumatic brain injury (TBI) per year in the U.S. and of those 75% are mild (National Center for Injury Prevention and Control, 2003).

Mild traumatic brain injury can result in a wide array of physical symptoms. The physical symptoms following mTBI can consist of headache, nausea, tinnitus, sleep disturbance, vomiting, blurred vision, and dizziness (Barth et al., 2002). Sosnoff, Broglio, Shin, and Ferrara (2011) have found that individuals with a history of mTBI have differences in postural control, or balance, compared to individuals with no previous mTBI.

Cognitive symptoms that are often associated with mTBI are impairments in attention, memory, processing speed, learning abilities, and/or abstract reasoning (Barth et al., 2002). The visuospatial skills of individuals with mTBI have been found to be mildly impaired, as measured by the Block Design task of the WAIS-IV (Kashluba, Hanks, Casey, & Mills, 2008).

Along with these symptoms, individuals who have experienced a mTBI may have lasting deficits in their executive functioning (Lewine et al., 2007; Raskin & Mateer, 2000). Common EF deficits include impairments in prospective memory, processing speed, and working memory (Johansson, Berglund, & Ronnback, 2009; McAllister et al., 2001; Tay, Ang, Lau, Meyyappan, & Collinson, 2010). The impairments in EF may be measured by subjective testing or by neuropsychological testing. However, Ruff (2009) notes the neuropsychological tests for EF do not necessarily find all the deficits that are measured by subjective reports post mTBI.

Mild TBI is also associated with neurobehavioral symptoms that may include depression, anxiety, adynamia, and irritability (Barth et al., 2002). Hartlage, Durant-Wilson, and Patch (2001) reported findings of persistent irritability and depression following mTBI. These

emotional changes may be neurologically based and/or could be responses to the loss of function individuals experience after mTBI.

Physiology of mTBI

The physiological changes that occur during the period soon after a mTBI is sustained are referred to as the neuro-metabolic cascade of concussion (Giza & Hovda, 2001). After the initial injury, potassium and calcium levels are changed drastically. In an attempt to stabilize the changes, additional glucose is metabolized; however, this occurs at the same time that there has been a decrease in blood flow. It seems that basically the brain needs more energy (thus the hyper-metabolism of glucose), but at the same time is trying to conserve energy (less blood flow) which leads to the "energy crisis" which Giza & Hovda (2001) believe leaves the brain particularly vulnerable to a second injury.

There is a period of time immediately after a mTBI when an individual is vulnerable to long-term and/or significant cognitive impairment if he or she sustains another head injury. Should a second injury occur before symptoms of the initial injury subside, there is the possibility of Second Impact Syndrome (SIS; Cantu & Dean, 2010). Second Impact Syndrome is described by Cantu and Dean (2010) as a rapid swelling of the brain after the second injury that cannot be attributed to the severity of the second injury. They suggest that it is due to the dysautoregulation of the brain which began as a result of the initial injury. Since the acutely injured brain has not returned to metabolic homeostasis, the brain's capacity to adequately regulate metabolic function is even further compromised after a second injury, even if it is less serious than the first injury. Additionally, individuals who sustain frequent and repetitive mTBIs are at higher risk for developing chronic traumatic encephalopathy (CTE), a progressive and degenerative brain disease (McKee et al., 2009).

Neuroimaging in mTBI

Typically, standard MRI or CT imaging does not provide physical evidence of cerebral lesions after a mTBI. Historically, this lack of visible intracranial abnormalities led many professionals to assume that the symptoms after mTBI were primarily psychologically-based rather than neurologically-based. It is likely that there are some psychological factors that contribute to the symptoms present after mTBI, but most professionals today consider post-concussive symptoms to be primarily neurologically-based.

Advanced imaging techniques such as diffusion tensor imaging (DTI) can now detect diffuse axonal injury in subcortical white matter tracts after mTBI (Shenton et al., 2012). Rutgers et al., (2008) found reduced fractional anisotropy in many brain regions of individuals with mTBI compared to healthy controls. They reported that most of the regions with reduced fractional anisotropy were in the white matter of all lobes, but most occurred in white matter tracts subserving the frontal lobes, the corpus callosum, and the cingulate gyrus. Another study investigating mTBI using DTI also found significantly lower fractional anisotropy in the left corpus callosum (Lo, Shifteh, Gold, Bello, & Lipton, 2009). Inglese et al. (2005) found reduced fractional anisotropy in three brain structures of participants with mTBI, including the corpus callosum, the internal capsule, and the centrum semiovale. It is clear that abnormalities detected on diffusion tensor imaging vary across studies of mTBI; however, abnormalities in the corpus callosum were consistently noted.

Niogi et al. (2008) completed a DTI study with the purpose of identifying whether specific brain regions are associated with attention and memory functions and whether cognitive impairment following mTBI is associated with a change in DTI images. In this study, the average reduction in fractional anisotropy was significantly correlated with both right and left

uncinate fasciculus among healthy controls. They also found that reduction in fractional anisotropy in the left anterior corona radiata of healthy controls was significantly correlated with attentional performance. They then examined the results of individuals with previous mTBI compared to healthy controls. They found that compared to normal controls, individuals at least one month post mTBI scored significantly lower on the Long Delay Free Recall (LDFR) subtest of the California Verbal Learning Test. In addition, these findings were associated with significantly reduced fractional anisotropy in the uncinate fasciculus. MTBI subjects did not, however, differ significantly from healthy controls on attentional performance measures or fractional anisotropy.

Niogi & Mukherjee (2010) completed a review of DTI studies and the relevance of examining mTBI deficits. The researchers found that DTI has been found to be more sensitive in identifying the neurological changes on a microstructural level compared to other standard imaging techniques. In particular, they found that individuals with chronic post-concussive symptoms typically have reduced fractional anisotropy and increased apparent diffusion coefficient. The researchers recommend that DTI should be a standard along with CT and MRI when assessing mTBI. However, many institutions do not have DTI technology at this time.

Lewine et al. (2007) investigated a subset of individuals with persistent post-concussive symptoms. This neuroimaging study was unique because it used multiple imaging instruments to study participants who had experienced mild brain trauma with three distinct complaint profiles. Participants were grouped by somatic symptoms, psychiatric symptoms, or cognitive symptoms. All patients had undergone neuropsychological testing, and the group with cognitive impairment was identified based on abnormal neuropsychological test results. The other two groups were based on symptom report on a three-domain bioclinical symptom profile. The imaging

instruments used included magnetoencephalography (MEG), single photon emission computed tomography (SPECT), and magnetic resonance imaging (MRI). As predicted, standard MRI had the lowest detection rate (18%) of structural brain abnormalities in individuals who were considered impaired in any of the cognitive domains (attention, memory, executive function, and information processing) examined. However, abnormalities on MEG and SPECT imaging of these subjects were observed much more frequently. Abnormal SPECT findings were found in 40% of the subjects with cognitive impairment. MEG proved to be even more sensitive to mild brain trauma than either SPECT or MRI. Specifically, 86% of mTBI individuals complaining of cognitive deficits demonstrated abnormal activity on MEG. Relationships among slow wave abnormalities from MEG included slowed temporal lobe waves and memory, slowed parietal waves and attentional problems, slowed frontal lobe waves, and problems in executive functioning, and finally, slowed temporal lobe waves and impaired processing speed. It is of note that the same imaging protocol with the mTBI subjects who had a primarily somatic presentation revealed no significant abnormalities on any of these imaging techniques except for mTBI patients complaining of headaches. Among the individuals with headache, only one area (basal ganglia) on one imaging technique (SPECT) was found to be significant for headache symptoms. Overall, the Lewine et al. (2007) study provides more evidence that cognitive symptoms following mTBI have a biological rather than a psychological basis. In addition, it sheds light on the possible differentiation of cognitive and psychological factors with regard to their neurological bases in mTBI. The study also establishes that standard clinical imaging techniques most often used in a hospital setting such as CT and MRI are too insensitive to detect the physiological abnormalities that underlie post-concussive symptomatology in mTBI.

Recovery from mTBI

The majority of individuals who sustain a mTBI typically have a full recovery of cognitive symptoms by 90 days post-injury (Karr, Areshenkoff & Garcia-Barrera, 2014). However, 10-20% experience symptoms long after the initial injury (Barth et al., 2002; Binder, 1997; Erez, Rothschild, Katz, Tuchner, & Hartman, 2009).

It has been suggested that meta-analyses obscure persistent symptoms that occur in some individuals who have sustained a mTBI (Iverson, 2010). Pertab, James, and Bigler (2009) reanalyzed the data from previous meta-analyses (Binder, Rohling, & Larrabee, 1997; Frenchman, Fox, & Maybery, 2005) and have postulated four key reasons that meta-analyses can obscure persistent symptoms.

1. Mild traumatic brain injuries can be produced by many different mechanisms, a factor which is typically not accounted for in meta-analyses. For example, individuals who sustain mTBI by car accident will likely have different areas of the brain affected than those who may have been struck on the head by an object such as a baseball.

2. Diagnostic criteria for mTBI still vary widely among studies, despite the well-accepted ACRM definition.

3. The neuropsychological assessment tools used vary widely among studies included in the meta-analyses.

4. Symptomatic participants are usually not considered separately from other injured participants who do not have persistent symptoms.

Pertab et al. (2009) suggest that meta-analyses should separate individuals into groups of persistent and non-persistent symptoms to develop a better understanding of the subset of

individuals who experience symptoms past the typical three-month recovery time. It is this subset of individuals who experience persistent cognitive symptoms that are relevant to this thesis.

From the literature reviewed, a subset of individuals can be identified who have persistent cognitive and EF impairment after mTBI. It is on this group of individuals with mTBI that the author will focus when reviewing similarities between ADHD and mTBI and when considering viable treatments.

Medication

Maksimowski & Tampi (2016) completed a systematic review of studies examining the effects of stimulant medications on cognitive symptoms after TBI. In their review, they identified 18 randomized controlled studies. The researchers found that the majority of the studies reported a significant improvement in attention and concentration for the treatment group compared to placebo controls. Although this study identified generally positive results using stimulants for TBI in the literature, they also state that these results should be interpreted with caution due to the heterogeneity and small sample sizes among the studies.

Cognitive Rehabilitation

A review of the treatments for TBI-related EF deficits resulted in evidence for mostly bimodal or multimodal cognitive rehabilitative (CR) models. These models include two or more therapeutic interventions occurring concurrently or as one treatment program. Cognitive rehabilitation in general is a process by which medical and therapeutic services are provided to individuals with the intent of improving their cognitive abilities (Cernich, Kurtz, Mordecai, & Ryan, 2010). Interventions for mTBI have been adopted from cognitive rehabilitation for more severe TBI.

Ravi (2008) described cognitive rehabilitation as consisting of two approaches: remediation/restorative interventions and compensatory interventions. The purpose of remediation is to improve impaired areas of cognition to as much as possible via manual activities and/or computerized activities. Remediation activities are intended to activate or build up an area of cognition that may be deficient after injury; these activities typically include sets of drills or repetitive tasks. Compensatory training for an individual intends to provide internal strategies and external aids for cognitive deficits that cannot be fully remediated.

The existing literature examining the efficacy of cognitive rehabilitation varies greatly. Ravi (2008) described a divide in the literature, with some studies supporting the efficacy of CR, but other studies not supporting CR at all. There are a number of issues within the CR literature that may contribute to the conflicting data available. One of the issues is the heterogeneity of participants in studies that examine CR efficacy; specifically, the mechanisms by which participants have acquired their deficits are not necessarily homogeneous within studies. The next issue is that assessment tools and measures also vary greatly from one study to the next. Another reason is that it is often difficult to distinguish which components within CR are actually beneficial because the interventions among studies are not standardized and many interventions address several deficits at once. Finally, a critical issue is whether the improvements gained in treatment will generalize to real-life tasks. Parente and Herrman (2008) describe the generalization of skills in CR as a patient being able to use these new skills in a novel situation.

Typically, CR techniques will vary depending on the deficit being addressed. In regard to attention deficits, a remediation approach is considered to be the most beneficial (Helmick, 2010; Slomine & Locascio, 2009). The first and second versions of Attention Processing Training are

the most commonly recommended remediation tools for attention deficits (APT-II; Palmese & Raskin, 2000).

On the other hand, memory impairments are most often addressed by a compensatory approach, training individuals to use memory strategies such as mnemonic devices to help encode the information or using external aids to enhance memory (Helmick, 2010; Slomine & Locascio, 2009). External aids may include organizers/memory notebooks, but programs commonly found on electronic devices (e.g., calendars, memo applications) are now often utilized in place of paper-and-pencil strategies.

EF deficits are most often addressed in CR by training in problem solving and decision making, as well as increasing an individual's metacognitive functions through self-monitoring and goal setting (Helmick, 2010; Parente & Herrman, 2008; Slomine & Locascio, 2009).

Three systematic reviews of cognitive rehabilitation for TBI were completed from 2000-2011 (Cicerone et al., 2000 Cicerone et. al., 2005; Cicerone et al., 2011). From these systematic reviews, many recommendations were made for the cognitive rehabilitation of TBI. For patients with attentional deficits, the recommendation is that remediation and compensatory strategies should also be developed for these individuals. It was recommended that memory deficits are best addressed by establishing compensatory strategies.Training problem solving skills and interventions directed to increase metacognition are most beneficial for EF deficits.

Psychotherapy has also been noted as helpful for individuals' adjustment to life after a TBI. Psychotherapy is a beneficial addition to cognitive rehabilitation to address the emotional issues that are common after TBI, whether they are due to neurobehavioral changes or they reflect difficulties in coping with the noticeable difference in functioning after injury (Brewer-Mixon & Cullum, 2013).

Models of Cognitive Rehabilitation

There are multiple CR models described in the literature that may use some or all of the techniques described above to address cognitive deficits. Overall, it appears that first and foremost, these programs address attention deficits in order to lay the foundation to address other deficits. Parente and Herrman (2008) recommend a model which includes active, passive, and supportive modes. The active model consists of direct training of strategies and remediation of deficits. The passive mode works on the individual's physical and emotional state. Physically, an individual's nutrition, sleep, exercise, etc. could affect his or her ability to sustain mental activity. Emotionally, other factors such as depression or anxiety could also affect an individual's ability to concentrate and sustain mental activity. These would also need to be addressed. The supportive mode addresses the individual's environment, determining what modifications or external aids are needed to improve function. It is recommended by Parente and Herrman (2008) that all three of these modes be addressed concurrently during rehabilitation in order to have an optimum outcome.

Another CR model is the Executive Plus Model recommended by Gordon, Cantor, Ashman, and Brown (2006). In this model, Gordon and colleagues recommend first addressing attention deficits as a prerequisite to other CR interventions. They then go on to problem-solving training and teach emotional regulation strategies concurrently. They acknowledge that issues with emotional regulation present a possible hindrance to problem-solving in general. In their problem-solving training, they teach patients four steps: orienting to and defining the problem, developing alternatives, making a decision, and getting feedback on the solution. The emotional regulation training consisted of a three-part strategy, including first becoming aware of one's emotions, determining the triggers for the emotion, and changing thoughts that may lead to

negative emotions. The problem-solving training and emotional regulation training include having individuals practice these techniques in a variety of group settings, such as community group, cognitive skills group, psychotherapy group, community access group, computer education group, education group, and life skills group. Opportunities are presented in each of these groups to put into action the skills that the patients learn.

Cognitive Rehabilitation Studies Specific to Mild Traumatic Brain Injury

In some individuals, mTBI may lead to lasting deficits in executive functioning (Erez et al., 2009; Grossman et al., 2012). These deficits often include the sub-processes of prospective memory, processing speed, and working memory (Johansson, Berglund, & Ronnback, 2009; McAllister2001; Tay, Ang, Lau, Meyyappan, & Collinson, 2010). As mentioned previously, the interventions used to address the cognitive deficits after mTBI are adopted from general CR that is often used for moderate and severe TBI. From the literature regarding persistent deficits after mTBI, it would be reasonable to conclude that interventions should focus on attention, working memory, processing speed, and prospective memory. The earlier studies specific to mTBI typically aimed to examine the effect of interventions on specific deficits following mTBI. More recently, there have been studies that evaluate programs that address numerous deficits within mTBI at once.

It is generally recommended that CR programs first remediate attention, or at least concurrently rehabilitate attention and other deficits. Palmese and Raskin (2000) found that the APT-II program improved the functioning of three individuals with mTBI in a case study. One participant improved in sustained and divided attention. The second participant improved in sustained attention only. The last participant did not have as much improvement as the others, but was able to increase rate of work under time constraints.

Cicerone et al. (1996) conducted a study examining the effectiveness of a cognitive rehabilitation program for individuals who experienced a mTBI. The researchers wanted to evaluate which specific cognitive functions were improving when individuals reported a good outcome. They considered a good outcome to treatment as the participant being able to return to his/her work or other primary responsibilities after being injured. Individuals who participated in treatment received cognitive remediation in which they were asked to complete "paper and pencil" or "real-life" tasks while sustaining attention and withstanding distractions. Individuals also received CBT to address their affective symptoms and improve their self-efficacy. Functional skills were addressed for individuals by developing compensatory strategies to improve organization, time management, and higher level activities of daily living such as managing finances. Twenty individuals with a mTBI completed the program, and of those, half were considered to have a good outcome. The researchers then examined what cognitive functions significantly improved from pre- to post-treatment in those individuals who had a good outcome. They found that on measures of attention, the good outcome group had significant improvements on the more complex attention tasks included on the Trail-Making Test, Part B (TMT; Tombaugh, 2004), the Continuous Performance Test of Attention (CPTA; Rosvold, Mirsky, & Sarason, 1956), and the Paced Auditory Serial Addition Test (PASAT; Gronwall, 1977).

Ho and Bennett (1997) examined a program that consisted of compensatory strategy training and cognitive remediation for individuals with mTBI, the Brain Injury Recovery Program (BIRP) at Colorado State University. Their study included only participants who had sustained injury by motor vehicle accident, were at least three months post-injury, and had persistent cognitive impairments, including EF deficits. Their study was a retroactive study of

patients who complete individualized CR programs that lasted approximately 37 weeks. All of these patients received remediation and compensatory strategy training. They found that after completion of the program, participants' performance on neuropsychological tests significantly improved on almost all cognitive domains, as measured by the Halstead-Reitan Neuropsychological Battery (HRB; Reitan & Wolfson, 1985). The cognitive domains they examined from this battery included attention, processing speed, memory skills, executive functioning, conceptual skills, and flexibility of thinking. There were also significant improvements on self-ratings of activities of daily living for the participants. They did not, however, find correlations between the neuropsychological test scores and the behavioral ratings. One reason these did not correlate could be that the neuropsychological measures may be more sensitive to cognitive impairments than the behavioral scales were. Another possible reason is that the behavior ratings may not be measuring the same aspects of neuropsychological functioning as the formal tests. Additionally, the participants may have had reduced insight with regard to their own functioning.

Tiersky et al. (2005) modified general cognitive rehabilitation that is typically used for severe TBI and applied it to individuals with persistent cognitive complaints 1-20 years following a mTBI. The protocol for this study consisted of individual psychotherapy and cognitive remediation. The individual psychotherapy used CBT techniques, with the goal of helping individuals more effectively process feelings of loss they experienced following their injury. The cognitive remediation portion of the treatment consisted of tasks pulled from the Attention Process Training II materials (APT-II; Palmese & Raskin, 2000). Participants were also trained to use compensatory strategies for single task completion, removal of distractions, and planning and problem-solving techniques. Participants had a significant improvement in

processing speed as measured by the Paced Auditory Serial Addition Test (PASAT; Gronwall, 1977). However, participants did not have a significant improvement in community integration as measured by the Community Integration Questionnaire (CIQ; Willer, Rosenthal, & Kreutzer, Gordon, & Rempel, 1993). They did not measure other cognitive areas such as planning and problem solving directly. It is important to note that participants in this study did not subjectively report improvement in functional ability, whereas, the participants of the Ho & Bennett, (1997) did significantly report improvement in functional ability. Tiersky et al. (2005) discuss the possibility that their findings could be due to "psychological feedback loops" that maintain functional disability or an individual's perception of functional disability after mTBI, despite objective improvements after treatment.

Twamley, Jak, Delis, Bondi, and Lohr (2014) created a manualized treatment protocol for individuals with mild to moderate traumatic brain injury. The treatment consists of training compensatory strategies and is called Cognitive Symptoms Management and Rehabilitation Therapy (CogSMART). For prospective memory deficits, use of a daily calendar, prioritizing tasks, and external reminder cues were all taught to individuals. For executive function deficits, they recommend training individuals in a six-step problem-solving method, using self-talk while completing tasks, as well as hypothesis-testing and self-monitoring. They found that self-report of prospective memory abilities were significantly improved in the individuals who completed the training. Twamley et al. (2015) completed a follow-up study. At 12 months, participants continued to score significantly higher on prospective memory measures compared to controls.

Cicerone (2002) completed a prospective case-comparison study to examine the effectiveness of an intervention addressing attention and working memory deficits after mTBI. In this study, they had participants complete n-back procedures using playing cards. An n-back

procedure is a working memory test in which a series of items/objects are presented and the individual has to recognize the stimulus that was presented "n" objects/items ago. N represents how many other stimuli are presented before the target stimuli have to be remembered. Participants were required to complete 3 variants of the n-back procedures and in each step, the working memory load increased. The working memory load was increased by either increasing distractions while completing the task or the task becoming more difficult. In the final stage of each procedure, they were required to complete a secondary task that closely resembled a task they had to do for work while also completing the n-back tasks. The goal of this final task was to simulate a real-world environment in which one's primary task (n-back task) is often interrupted to complete a secondary task. Outcome measures included the Trail-Making Test (TMT; Tombaugh, 2004) the Paced Auditory Serial Addition Test (Gronwall, 1977), The Continuous Performance Test of Attention (CPTA; Rosvold, Mirsky, & Sarason, 1956), and the 2 and 7 Test (Ruff, Niemann, Allen, Farrow, & Wylie, 1992). The treatment group had significant improvements on attention measures. They also had significant improvements on Trial 1 of the PASAT, indicating an improvement in working memory. One non-significant finding was on Trial 4, on which the treatment group and comparison participants did not differ, indicating that the treatment did not have an effect on processing speed. However, the treatment overall was beneficial to the treatment group.

Executive Function

As previously noted, both individuals with Attention-Deficit/Hyperactivity Disorder and those with mild traumatic brain injury commonly have deficits in the various cognitive processes that fall in the executive function (EF) domain. Executive function is the supervisory function of the brain that depends on attention and allows for self-regulation, planning, problem-solving,

reasoning, and judgment. A deficit in any one of the abilities within the domain of executive function may also cause problems in other executive functions.

The definition of EF has expanded over time. Welsh and Pennington (1988) included four processes in their EF definition. These processes included goal-directed behavior, working memory, planning, and inhibition. Denckla (1996) built on this definition and described EF as a large set of mental processes necessary for efficient and productive behavior. Denckla (1996) went on to elaborate that executive functions are separate from intelligence, and that although someone with executive dysfunction may have an average IQ, he or she may struggle with relatively easy tasks. Executive function is now commonly thought of not as a unitary cognitive function, but rather a set of higher-level cognitive sub-processes that together allow an individual to self-regulate and effectively manage his or her day-to-day life (Elliott, 2003; Funahashi, 2001).

The most common executive functions include judgment, attention, organization, planning, decision-making, behavioral disinhibition, set-shifting, and working memory (Carpenter, Just, & Reichle, 2000; Elliott, 2003; Funahashi, 2001; Roberts, Robbins, & Weiskrantz, 1998). Together these higher-level cognitive abilities coordinate to achieve goal-directed behavior and problem-solving in a novel situation. Bewick, Raymond, Malia, and Bennett (1995) have argued that at the core of executive dysfunction is impairment in metacognition, one's ability to monitor his or her own thoughts and performance. An individual with impaired metacognition will not be able to notice the deficits in his or her EF. Improving individuals' ability to monitor their performance will increase insight into their deficits and allow them to implement compensatory strategies for these deficits.

Executive functions can be measured by neuropsychological instruments (Burgess, Alderman, Evans, Emslie, & Wilson, 1998), but testing is often limited to measuring the individual sub-processes of executive function (e.g., set-shifting, attention, and behavioral inhibition). It is less effective in measuring EF as a whole, especially on measures in which all (or even several) sub-processes are working together. Unfortunately, measuring each specific function individually may not give a clear picture of the severity of functional deficits an individual may experience in his or her natural environment because EF is ultimately a coordination of the various processes (Rabbit, 1997). In one study, individuals with frontal lobe damage performed well on the traditional neuropsychological tests of executive function but had trouble planning and organizing daily activities (Shallice & Burgess, 1991). Additionally, Alderman, Burgess, Knight, and Henman (2003) found significant differences between individuals with a previous brain injury and controls in a "real-life" shopping task measuring executive function. Lezak, Howieson, Loving, Hannah, & Fischer (2004) describe in their book a patient who, post brain injury, continued to do extremely well on neuropsychological tests, and from a testing standpoint should have been able to return to his previous lifestyle and career as a surgeon. However, his self-regulating and ability to initiate were essentially non-existent after his injury, resulting in him no longer being able to work as a surgeon and essentially all his personal affairs being handled by family. Studies included in this thesis utilized all the various types of EF assessment tools such as clinical observation, self-report, and formal neuropsychological testing in order to present a more comprehensive picture of EF functioning in individuals.

Similarities in Executive Functioning of Persons with ADHD and Mild Traumatic Brain Injury

There is a significant overlap between the deficits of individuals with ADHD and mTBI. In particular, deficits in processing speed, working memory, prospective memory and metacognition are common to both ADHD and mTBI. Each of these sub-processes will be discussed in depth in the following sections. The studies investigating deficits after mTBI typically measure deficits over time and usually aim to distinguish whether or not cognitive deficits that occur in the acute stage following a mTBI persist. On the other hand, it is generally accepted that the deficits in executive functioning experienced by individuals with ADHD will continue throughout their life, as it is a developmental condition. One does not "recover" from ADHD, whereas, mTBI is an injury event and there is a natural healing period after the injury. Therefore the studies for ADHD are typically designed in a way only to show the deficit exists. Mild TBI studies, however, are designed to show the deficit exists and that it will continue to cause impairment over the course of the individual's life. At this time, the author of this thesis did not find studies of executive functioning deficits in mTBI and ADHD that were comparable in this way. Regardless of how EF deficits are acquired (i.e., injury event or developmental) this paper is focused on how to remediate or compensate for these deficits.

Processing Speed

Processing speed is the measure of an individual's ability to efficiently and automatically process information without intentional effort to make a rapid response (Shanahan et al., 2006). Processing speed appears to be impaired in both ADHD and mTBI. Brown, Reichel, and Quinlan (2009) found in a sample of adults with ADHD and a FSIQ greater than or equal to 120, that 85% of them were impaired on the Processing Speed index of the WAIS-III and 44% were

considered to have severe impairments. A more recent study found that adults with ADHD have impaired processing speed relative to controls measured on the WAIS-IV (Theiling & Petermann, 2014). Individuals with a previous mTBI and persistent cognitive complaints were administered the Coding subtest from the WAIS-III and the Trail Making Test to assess visual processing speed. There were significant differences in the performances of mTBI participants compared to healthy controls on both tests (Johansson, Berglund, & Ronnback, 2009). The researchers of this study speculated that slowed visual processing speed may be due to mental fatigue in individuals with mTBI. Additionally, O'Jile et al. (2006) found that individuals with mTBI do not benefit from repeated exposure to processing speed trials in the way that healthy controls do, as measured by the Paced Auditory Serial-Addition Task.

Clearly, processing speed impairments have been well-documented in both ADHD and mTBI. Deficits in this sub-process of EF likely make it difficult for individuals with either condition to quickly formulate responses to information, including information that is meant to be understood quickly and in an "automatic" manner. Difficulty under time constraints may lead to "careless errors" in work or in school (Solanto, 2014). This is often noticed as simple mistakes in the work of a student or employee who has ADHD or mTBI.

Working Memory

Working memory is the ability to hold information in mind while mentally manipulating it. Working memory has been studied extensively in both ADHD and mTBI. A meta-analysis found moderate to large effect sizes for differences in working memory impairments in children and adolescents with ADHD compared to healthy controls (Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005). Another meta-analytic study involving adults with ADHD resulted in similar findings (Alderson, Kasper, Hudec, & Patros, 2013). Rohling et al. (2011) found that

working memory deficits in adults with mTBI remained impaired at three months. Kumar, Rao, Chandramouli, and Pillai (2013) found similar results in adults with mTBI compared to controls up to six months post-injury.

Neuroimaging studies have shown similar working memory impairments in both ADHD and mTBI. Ko et al. (2013) found differential brain activation in adults with ADHD vs. healthy controls on working memory tasks. Specifically, they found that as the difficulty of working memory tasks increases, brain activation does not increase at the same rate for persons with ADHD compared with healthy controls. Another study using functional near–infrared spectroscopy (fNIRS) found reduced activation in adults with ADHD when completing a higher intensity working memory task compared to healthy controls, specifically in the prefrontal cortex (Ehlis, Bahne, Jaocb, Hermann, & Fallgatter, 2008). McAllister et al. (2001) found similar results for the working memory performance and brain activity of individuals with mTBI. In this study, individuals with mTBI had significantly less brain activation on fMRI while attempting the most rigorous working memory tasks compared to healthy controls. Chen et al. (2012) conducted a similar study in which individuals with mTBI performed working memory tasks during fMRI scanning. They not only found a reduced activation in the parietal and frontal lobes for the most intense working memory task, but also at moderate levels of difficulty.

In summary, the literature supports working memory as a common impairment in both mTBI and ADHD. Essentially, as the intensity of the working memory task increases, an individual with either ADHD or mTBI will have significantly more difficulty compared to healthy controls. The imaging studies for ADHD generally show that this difference in activation is located in the prefrontal cortex. Imaging studies for mTBI also show differences in the

activation of the frontal lobe compared to healthy controls; however, for mTBI, activation differences are more global, as evidenced by the Chen et al. (2012) study,

Prospective Memory & Metacognition

Prospective memory is "remembering to remember" to do something in the future. Metacognition is the ability to monitor one's thoughts and performance. Schnitzspahn, Zeintl, Jager, and Kiegel (2011) suggested that there is a connection between prospective memory abilities and an individual's metacognitive abilities. For example, an individual's prediction of what his or her performance will be (metacognition) determines the amount of effort that will be allocated to remembering. Therefore, better metacognition likely means a better chance of remembering to perform a certain action in the future (prospective memory).

Prospective memory deficits are established in the literature for individuals with ADHD. Adults with ADHD were found to have impaired prospective memory compared to healthy controls (Fuermaier et al., 2013). In this study, they also suggest that prospective memory deficits are likely due to difficulty planning ahead. Prospective memory deficits have also been identified in individuals with a mTBI. Tay, Ang, Lau, Meyyappan, and Collinson (2010) identified impaired prospective memory in adults with mTBI in the acute and post-acute phases of recovery in comparison to healthy controls.

The deficits in prospective memory that individuals with these conditions display likely make it difficult to function in a work environment in which abilities to plan ahead and remember to complete tasks are important. Prospective memory is needed throughout the day for most people. A deficit in this area would make it very difficult to remember to do daily errands or tasks without constant reminders.

CHAPTER THREE

Discussion

It would be reasonable to hypothesize that the cognitive rehabilitation treatments that have been demonstrated to be effective for treating EF in mTBI would also be effective in treating the EF deficits in ADHD. The outcome of this review is that the cognitive rehabilitation techniques used to treat mTBI could also be beneficial in ADHD treatment. Two bodies of literature appear to support this hypothesis. First, the cognitive deficits in individuals with mTBI and ADHD are similar. Second, intervention techniques and programs have been effective in improving the cognitive functioning of individuals with mTBI. Furthermore, many of the current techniques used in both ADHD and mTBI at times overlap.

Executive functioning deficits have been demonstrated to cause significant impairments in the lives of individuals with these disorders. Impaired attention is believed to be at the core of these EF deficits. In a rehabilitation model, it would be crucial to address multiple deficits at once, as a deficit in one EF sub-process would also likely have an effect on another. A deficit in attention will likely have an effect on the other sub-processes of EF including working memory, processing speed, and prospective memory, all of which have been documented to be impaired in ADHD and mTBI (Fuermaier et al., 2013; Brown, Reichel, & Quinlan, 2009; Johansson, Berglund, & Ronnback, 2009; Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; Rohling et al., 2011; Tay, Ang, Lau, Meyyappan, & Collinson, 2010; Theiling & Petermann, 2014). Although attention is at the core of these conditions, metacognition also is an essential ability, allowing the individuals to notice when there is a problem with their functioning.

The non-pharmacological treatments currently available for individuals with ADHD have been shown to be beneficial to varying degrees. CBT appears to be the most efficacious non-

pharmacological treatment available for ADHD at this time. The cognitive portion of this treatment typically addresses the negative thoughts that stem from years of underachievement, caused by deficits experienced by individuals with ADHD. The behavioral component typically addresses compensatory strategies such as organizational and planning skills, problem-solving, and the use of external aids. For example, the treatment manual by Safren, Sprich, Otto, Perlman, and Otto (2005) first has individuals learn problem-solving, organizational, and planning skills. They then move on to the cognitive portion to address maladaptive thoughts which often lead to avoidance. CBT programs such as this have been shown to decrease the total number of symptoms exhibited by an individual with ADHD (Salakari et al., 2010; Weiss et al., 2012). Furthermore, improvements in attention and memory appear to be consistent among CBT studies in adults with ADHD (Solanto, Marks, Mitchell, Wasserstein, and Koffman, 2008; Virta et al., 2010). However, outcomes regarding improvement in organization, planning, and working memory within these same studies are conflicting.

Treatment for mTBI is relatively sparse, due to a shift in the medical perspective away from the assumption that mTBI does not result in lasting cognitive deficits. Treatment for mTBI typically is bimodal or multimodal (two or more therapeutic interventions occurring concurrently), and types of therapeutic services can vary widely depending on the deficits each individual presents (Cernich, Kurtz, Mordecai, & Ryan, 2010; Ravi, 2008). A review of the literature on cognitive rehabilitation for mTBI revealed promising results from these therapeutic interventions. From the studies reviewed, interventions including the components of remediation, compensatory strategies, and psychotherapy all were found to be beneficial in decreasing EF deficits in individuals with mTBI. Table 1 lists the studies reviewed and the techniques used in each one.

Some overlap in interventions already exists among treatments for ADHD and mTBI. Some of the overlapping techniques include training in problem-solving, organizational skills, and planning skills, along with the use of external aids to enhance memory; in addition, emotional regulation is a component of these techniques. The CR models used for mTBI are more likely to be multi-interventional models that include remediation and compensatory strategy training. Typically, individuals in treatment for mTBI are developing compensatory strategies, remediating attention deficits, and participating in psychotherapy or emotional regulation training. The studies that exist for ADHD mostly address compensatory strategy training and psychotherapy rather than remediation. It is hypothesized that optimal outcome could possibly be achieved by concurrently using both. In fact, computer-based remediation programs do exist for ADHD. Melby-Lervag and Hulme (2013) found in their meta-analysis that computer-based remediation programs for ADHD do improve verbal and visual working memory immediately following treatment, but these improvements do not necessarily last, nor do they generalize or have any effect on other sub-processes within executive functioning. Thus, there is room for improvement in remediation techniques for ADHD. The remediation techniques used in mTBI could potentially be beneficial to individuals with ADHD, given the similarities in cognitive deficits these two groups share.

Another concept that stands out in mTBI treatment is the emphasis on real-life therapy tasks. For example, when Cicerone (2002) had participants remediating working memory, they were also completing other activities similar to tasks in their work environment. One of the points of the study was to show that CR tasks have ecological validity, resulting in real-world improvements in TBI patients' lives. The remediation of ADHD deficits has been addressed by

computerized training programs. However, programs focused on remediation for these deficits do not necessarily integrate a realistic component to their regimen.

At this time, the literature available for mTBI-specific treatment is growing rapidly. It seems that behavioral treatments for ADHD are expanding as well; however, whereas CR is the first line of treatment for the cognitive deficits of mTBI, it is an adjunct treatment for ADHD. Literature regarding remediating and/or compensating for deficits after mTBI supports the premise that possibly combining psychopharmacological and cognitive rehabilitative treatment for ADHD could be most beneficial for the similar deficits that are experienced. This could also be true for mTBI and, anecdotally, is already in practice as in some rehabilitation settings individuals with TBI are also being prescribed stimulants to assist with symptoms of inattention. In the systematic review completed by Maksimowski & Tampi (2016), generally positive results for attention and concentration deficits were found after treating TBI with stimulant medication.

CHAPTER FOUR

Suggestions for Future Research

The ADHD treatment literature could be expanded in many ways. The literature regarding treatment for ADHD would benefit from more studies examining the effect of a multimodal cognitive rehabilitation program. Although it is beneficial to see the effects of different components of treatment, we may be missing the benefit of all the modes of treatment working together.

Literature regarding ADHD treatment would also benefit by examining symptom improvement individually as well total number of symptoms. Although decreasing the overall average number of symptoms of individuals with ADHD is important, deficits that remain despite treatment may still be disruptive to these individuals' lives. Focusing more on the specific individual symptoms may also inform the treatment of ADHD.

Future studies could also expand the effects of remediation techniques on ADHD, specifically, remediation techniques focused on attention, working memory, and processing speed. At this point, the literature regarding remediation of ADHD deficits only addresses remediating working memory and typically by computerized programs. When using remediation techniques, ADHD treatment models in general could first address attention, then step-up to other processes as do the mTBI treatment models.

Treatment could also be improved by incorporating real-life tasks into ADHD treatment as is done in CR of mTBI. An increase in the emphasis on real-life tasks could potentially increase the generalization of skills learned in treatment. Compensatory strategies for ADHD are typically developed on an individual basis to suit a person's needs. For example, environmental modifications or external aids are developed depending on what typically occurs in an

individual's daily life. Contrasted with compensatory strategies, it appears the minimal remediation that occurs for individuals with ADHD is computer-based. With a real life emphasis, a therapist could assist individuals in finding realistic tasks that require sustained attention, such as reading. The individuals could be encouraged to approach the task in a hierarchical manner in which they start small and then as attention improves, they increase the volume of work or read more difficult pieces. As difficulty increases, these abilities are strengthened. Furthermore, this example could apply to other cognitive abilities that are deficient.

Incorporating real-life tasks into the CR of ADHD also could tie in with improving metacognition as it would give individuals the opportunity to increase their insight into their functioning in the day-to-day activities. Problem-solving skills could be beneficial as typically the first step in training those skills is teaching individuals how to identify and orient to the problem such as in the treatment model of Gordon, Cantor, Ashman, & Brown (2006). If, for example, an individual is constantly forgetting deadlines but not understanding why, he or she needs first to identify that it is a problem and then can work towards solving the problem. A therapist working with the individual can assist by objectively identifying deficits that an individual may not notice on a day-to-day basis. As an individual improves in treatment, metacognition improves and the individual would be able to identify deficits or problems independently.

Although ADHD and mTBI have similar EF deficits, one is an injury event whereas the other is a lifetime condition. Therefore, not all facets of treatment should be approached in the same manner for these two conditions. The affective components would likely differ between these conditions as an individual who has sustained an mTBI would likely have a sense of loss after their injury that could be addressed in psychotherapy. On the other hand, individuals with

ADHD may have lifelong frustration from their deficits that may manifest as poor self-efficacy. Furthermore, it may be beneficial to approach ADHD treatment with a “habilitation” perspective as opposed to “rehabilitation” perspective.

From the literature reviewed in this thesis it is concluded that treatment options for mTBI could also benefit individuals with ADHD. At this time, some of the techniques of cognitive rehabilitation for mTBI and treatment for ADHD overlap. As the literature increases for both of these conditions, it could be beneficial for one to inform the other.

References

Adler, L.A., Clemow, D.B., Williams, D.W., & Durell, T.M. (2014). Atomoxetine effects on executive function as measured by the BRIEF-A in young adults with ADHD: A randomized, double-blind, placebo-controlled study. *Public Library of Science ONE, 9*(8), 1-8. doi: 10.1371/journal.pone.0104175

Advokat, C. (2010). What are the cognitive effects of stimulant medications? Emphasis on adults with Attention-Deficit/Hyperactivity Disorder (ADHD). *Neuroscience and Rehabilitation Reviews, 34*, 1256-1266.

Alberts-Corush, J., Firestone, P, & Goodman, J.T. (1986). Attention and impulsivity characteristics of the biological and adoptive parents of hyperactive and normal control children. *American Journal of Orthopsychiatry, 56*(3), 413-423.

Alderson, R., Kasper, L., Hudec, K., & Patros, C. (2013). Attention-Deficit/Hyperactivity Disorder (ADHD) and working memory in adults: A meta-analytic review. *Neuropsychology, 27*(3), 287-302. doi: 10.1037/a0032371

Alderman, N., Burgess, P.W., Knight, C., & Henman, C. (2003). Ecological validity of a simplified version of the multiple errands shopping test. *Journal of the International Neuropsychological Society, 9*, 31-44. doi:10.10170S1355617703910046.

American Academy of Pediatrics. (2001). Clinical practice guideline: Treatment of the school aged child with Attention-Deficit/Hyperactivity Disorder. *Journal of the American Academy of Pediatrics, 108*(4), 1033-1044. doi:10.1542/peds.108.4.1033

American Psychiatric Association. (1994). *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.). Washington, DC: Author.

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental*

Disorders (5th ed.). Washington, DC: Author.

Antshel, K., Faraone, S., Maglione, K., Doyle, A., Fried, R., Seidman, L., & Biederman, J. (2010). Executive functioning in high-IQ adults with ADHD. *Psychological Medicine, 40*(11), 1909-1918. doi: 10.1017/S00332

Ashtari, M., Kumra, S., Bhaskar, S., Clarke, T., Thaden, E., Cervellione, K.,. . . .Ardekani, B. (2005). Attention-Deficit/Hyperactivity Disorder: A preliminary diffusion tensor imaging study. *Biological Psychiatry, 57*(5), 448-445. doi: 10.1016/j.biopsych.2004.11.047

Barbaresi, W. J., Colligan, R. C., Weaver, A. L., Voigt, R. G., Killian, J. M., & Katusic, S. K. (2013). Mortality, ADHD, and psychosocial adversity in adults with childhood ADHD: A prospective study. *Pediatrics, 131*(4), 637-644. doi: 10.1542/peds.2012-2354

Barbaresi, W. J., Katusic, S. K., Colligan, R. C., Weaver, A. L., Leibson, C. L., & Jacobsen, S. J. (2014). Long-term stimulant medication treatment of Attention-Deficit/Hyperactivity Disorder: Results from a population-based study. *Journal of Developmental and Behavioral Pediatrics, 35*(7), 448-457. doi: 10.1097/dbp.0000000000000099

Barkley, R. (2006). *Attention Deficit Hyperactivity Disorder: A handbook for diagnosis and treatment.* New York: The Guilford Press.

Barkley, R. A., & Murphy, K. R. (2010). Impairment in occupational functioning and adult ADHD: The predictive utility of executive function (EF) ratings versus EF tests. *Archives of Clinical Neuropsychology*, *25*(3), 157-173. doi: 10.1093/arclin/acq014

Barth, J., Freeman, J., & Broshek, D. (2002). Mild head injury. *Encyclopedia of The Human Brain, 3,* 81-92.

Bewick, K. C., Raymond, M. J., Malia, K. B., & Bennett, T. L. (1995). Metacognition as the ultimate executive: Techniques and tasks to facilitate executive functions. *NeuroRehabilitation, 5*(4), 367-375. doi: 10.3233/nre-1995-5411

Biederman, J., & Faraone, S.V. (2005). Attention-Deficit Hyperactivity Disorder. *Lancet, 366,* 237-248. doi: 10.1016/S0140-6736(05)66915-2

Biederman, J., Monuteaux, M. C., Mick, E., Spencer, T., Wilens, T. E., Silva, J. M., . . . Faraone, S. V. (2006). Young adult outcome of Attention Deficit Hyperactivity Disorder: A controlled 10-year follow-up study. *Psychological Medicine, 36*(2), 167-179. doi: 10.1017/S0033291705006410

Biederman, J., Petty, C., Fried, R., Fontanella, J., Doyle, A.E., & Seidman, L.J. (2006). Impact of psychometrically defined deficits of executive functioning in adults with Attention Deficit Hyperactivity Disorder. *American Journal of Psychiatry, 163,* 1730-1738.

Biederman, J., Petty, C., Fried, R., Kaiser, R., Dolan, C., Schoenfield, S., … Faraone, S., (2008). Educational and occupational underattainment in adults with Attention Deficit/Hyperactivity Disorder: A controlled study. *Journal of Clinical Psychiatry, 69*(8), 1217-1222.

Biederman, J., Seidman, L.J., Petty, C.R., Fried, F., Doyle, A.E., Cohen, D.R., . . . Faraone, S.V. (2008). Effects of stimulant medication on neuropsychological functioning in young adults with Attention-Deficit/Hyperactivity Disorder. *Journal of Clinical Psychiatry, 69*(7), 1150-1156.

Biederman, J., Petty, C.R., Clarke, A., Lomedico, A., & Faraone, S.V. (2011). Predictors of persistent ADHD: An 11-year follow-up study. *Journal of Psychiatric Research, 45*(2), 150-155. doi: 10.1016/j.jpsychires.2010.06.009

Biederman, J., Petty, C. R., Woodworth, K. Y., Lomedico, A., Hyder, L. L., & Faraone, S. V. (2012). Adult outcome of Attention-Deficit/Hyperactivity Disorder: A controlled 16-year follow-up study. *Journal of Clinical Psychiatry, 73*(7), 941-950. doi: 10.4088/JCP.11m07529

Binder, L., Rohling, M., & Larrabee, G. (1997) A review of mild head trauma. Part I: Meta-analytic review of neuropsychological studies. *Journal of Clinical and Experimental Neuropsychology, 19*(3), 421-431. doi: 10.1080/01688639708403870

Binder, L. (1997). A review of mild head trauma. Part II: Clinical implications. *Journal of Clinical and Experimental Neuropsychology, 19*(3), 432-457.

Brewer-Mixon, K.K & Cullum, C.M. (2013). Historical Principles and Foundations of Neuropsychological Rehabilitation. In C.A. Noggle & R.S. Dean with M.T. Barisa (Eds.). *Neuropsychological Rehabilitation* (pp. 1-11). New York: Springer Publishing Company.

Brown, T. (1996). *Brown Attention-Deficit Disorder Scales for Adolescents and Adults.* San Antonio, TX: The Psychological Corporation.

Brown, T.E., Reichel, P.C., & Quinlan, D.M. (2009). Executive function impairments in high IQ adults with ADHD. *Journal of Attention Disorders, 13*(2), 161-167. doi: 10.1177/1087054708326113

Burgess, P. W., Alderman, N., Evans, J., Emslie, H., & Wilson, B. A. (1998). The ecological validity of tests of executive function. *Journal of the International Neuropsychological Society, 4*(6), 547-558.

Busner, J. & Targum, S. (2007). The clinical global impressions scale: Applying a research tool in clinical practices. *Psychiatry, 4*(7), 28-37.

Cantu, R. & Dean, A. (2010). Second-impact syndrome and a small subdural hematoma: An uncommon catastrophic result of repetitive head injury with a characteristic imaging appearance. *Journal of Neurotrauma, 27*(9), 1557-1564. doi: 10.1089/neu.2010.1334.

Carpenter, P. A., Just, M. A., & Reichle, E. D. (2000). Working memory and executive function: Evidence from neuroimaging. *Currrent Opinion in Neurobiology, 10*(2), 195-199.

Carroll, L, Cassidy, D., Holm, L., Kraus, J., & Coronado, V. (2004). Methodological issues and research recommendations for mild traumatic brain injury: The WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *Journal of Rehabilitation Medicine, 43*, 113-125.

Castellanos, F.X., Lee, P.P., Sharp, W., Jeffries, N.O., Greenstein, D.K., Clasen, L.S., . . . Giedd, J.N. (2002). Developmental trajectories of brain volume abnormalities in children and adolescents with Attention Deficit-Hyperactivity Disorder. *The Journal of the American Medical Association, 288*(14), 1740-1748.

Cernich, A., Kurta, S., Mordecai, K., & Ryan, P. (2010). Cognitive rehabilitation in traumatic brain injury. *Current Treatment Options in Neurology, 12,* 412-423. doi: 10.1007/s11940-010-0085-6

Chandler, M. L. (2013). Psychotherapy for adult Attention Deficit/Hyperactivity Disorder: A comparison with cognitive behaviour therapy. *Journal of Psychiatric and Mental Health Nursing, 20*(9), 814-820. doi: 10.1111/jpm.12023

Chen, C., Wu, C., Liao, Y., Hsu, H., Tseng, Y., Liu, H., & Chiu, W. (2012). Working memory in patients with mild traumatic brain injury: Functional MR imaging analysis. *Radiology, 265*(3), 844-851.

Chwastiak, L. & Von Korff, M. (2003). Disability in depression and back pain: Evaluation of the World Health Organization Disability Assessment Schedule (WHO DAS II) in a primary care setting. *Journal of Clinical Epidemiology, 56,* 507–514.

Cicerone, K., Smith, L., Ellmo, W., Mangel, H. Nelson, P., Chase, R., & Kalmar, K. (1996). Neuropsychological rehabilitation of mild traumatic brain injury. *Brain Injury, 10*(4), 277-286.

Cicerone, K., Dahlberg, C., Kalmar, K., Langenbahn, D., Malec, J., Berquist, T.,…Morse, P. (2000). Evidence-based cognitive rehabilitation: Recommendations for clinical practice. *Archives of Physical Medicine and Rehabilitation, 81,* 1596-1615. doi: 10.1053/apmr.2000/19240

Cicerone, K. (2002). Remediation of 'working attention' in mild traumatic brain injury. *Brain Injury, 16*(3), 183-195.

Cicerone, K., Dahlberg, C., Malec, J., Langenbahn, D., Felicetti, T., Keipp, S….Catanese, J. (2005). Evidence-based cognitive rehabilitation: Updated review of the literature from 1998-2002. *Archives of Physical Medicine and Rehabilitation, 86,* 1681-1692. doi: 10.1016/j.ampr.2005.03.024

Cicerone, K., Langenbahn, D., Braden, C., Malec, J., Kalmar, K., Fraas, M.,…Ashman, T. (2011). Evidence-based cognitive rehabilitation: Updated review of the literature from 2003-2008. *Archives of Physical Medicine and Rehabilitation, 92,* 519-530. doi: 10.1016/j.ampr.2010.11.015

Conners, K., Erhardt, D., & Sparrow, E. (2004). *Conners Adult ADHD Rating Scale—Self-Report: Long* [Measurement Instrument]. Retrieved from http://www.mhs.com/product.aspx?gr=cli&id=overview&prod=caars

Coghill, D. (2010). The impact of medications on quality of life in Attention-Deficit Hyperactivity Disorder. *CNS Drugs, 24*(10), 843-866.

de Graaf, R., Kessler, R., Fayyad, J., ten Have, M., Alonso, J., Angermeyer,…Posada-Villa, J. (2008). The prevalence and effects of adult Attention-Deficit/Hyperactivity Disorder (ADHD) on the performance of workers: Results from the WHO World Mental Health Survey Initiative. *Occupational and Environmental Medicine, 65*(12), 835-42. doi:10.1136/oem.2007.038448

Denckla, M. B. (1996). A theory and model of executive function: A neuropsychological perspective. In G. R. Lyon & N. A. Krasnegor (Eds.), *Attention, memory, and executive function* (pp. 263-278). Baltimore, MD: Paul H. Brookes.

Doshi, J., Hodgkins, P., Kahle, J., Sikirica, V., Cangelosi, M., Setyawan, J., …Neumann, P. (2012). Economic impact of childhood and adult Attention-Deficit/Hyperactivity Disorder in the United States. *Journal of the American Academy of Child and Adolescent Psychiatry, 51*(10), 990-1002. doi: 10.1016/j.jaac.2012.07.008

DuPaul G.J., Power T.J., Anastopoulos, A.D., & Reid, R. (1998). *ADHD rating scale-IV: Checklists, norms, and clinical interpretation.* New York, NY: The Guilford Press.

Endicott, J., Nee, J., Harrison, W., & Blumenthal, R. (1993). Quality of life enjoyment and satisfaction questionnaire: A new measure. *Psychopharmacology Bulletin, 29*(2), 321-326.

Ehlis, A., Bahne, C., Jacob, C., Hermann, M., & Fallgatter, A. (2008). Reduced lateral prefrontal activation in adult patients with Attention-Deficit/Hyperactivity Disorder (ADHD) during a working memory: A functional near-infrared spectroscopy (fNIRS) study. *Journal of Psychiatric Research, 42*(13), 1060-1067. doi: 10.1016/j.jpsychires.2007.11.011

Elliott, R. (2003). Executive functions and their disorders. *British Medical Bulletin, 65*, 49-59.

Erez, A., Rothschild, E., Katz, N., Tuchner, M., & Hartman-Maier, A. (2009). Executive functioning, awareness, and participation in daily life after mild traumatic brain injury: A preliminary study. *American Journal of Occupational Therapy, 63,* 634-640.

Faraone, S. V. (2000). Attention Deficit Hyperactivity Disorder in adults: Implications for theories of diagnosis. *Current Directions in Psychological Science, 9*(1), 33-36. doi: 10.1111/1467-8721.00055

Faraone, S.V., Perlis, R.H., Doyle, A.E., Smoller, J.W., Goralnick, J.J., Holmgren, M.A., Sklar, P. (2005). Molecular genetics of Attention-Deficit/Hyperactivity Disorder. *Society of Biological Psychiatry, 57*, 1313–1323. doi:10.1016/j.biopsych.2004.11.024

Faraone, S., Biederman, J., & Mick, E. (2006). The age-dependent decline of Attention Deficit Hyperactivity Disorder: A meta-analysis of follow-up studies. *Psychological Medicine, 36*(2),159-165.

Frenchman, K., Fox, A., & Maybery, M. (2005). Neuropsychological studies of mild traumatic brain injury: A meta-analytic review of research since 1995. *Journal of Clinical and Experimental Neuropsychology, 27*(3), 334-351. doi: 10.1080/13803390490520328

Frick, P., Kamphaus, R., Lahey, B., Loeber, R., Christ, M., Hart, E., & Tannenbaum, L. (1991). Academic underachievement and the disruptive behavior disorders. *Journal of Consulting and Clinical Psychology, 59*(2), 289-294. doi:10.1037/0022-006X.59.2.289

Fuermaier, A.B., Tucha, L., Koerts, J., Aschenbrenner, S., Westermann, C., Weisbrod, M.,…& Oliver, T. (2013) Complex prospective memory in adults with Attention Deficit Hyperactivity Disorder. *Public Library of Science ONE, 8*(3). doi: 10.1371/journal.pone.0058338

Funahashi, S. (2001). Neuronal mechanisms of executive control by the prefrontal cortex. *Neuroscience Research, 39*(2), 147-165.

Galera, C., Bouvard, M. P., Lagarde, E., Michel, G., Touchette, E., Fombonne, E., & Melchior, M. (2012). Childhood attention problems and socioeconomic status in adulthood: 18-year follow-up. *The British Journal of Psychiatry, 201*(1), 20-25. doi: 10.1192/bjp.bp.111.102491

Garcia, C. R., Bau, C. H., Silva, K. L., Callegari-Jacques, S. M., Salgado, C. A. I., Fischer, A. G., . . . Grevet, E. H. (2012). The burdened life of adults with ADHD: Impairment beyond comorbidity. *European Psychiatry, 27*(5), 309-313. doi: 10.1016/j.eurpsy.2010.08.002.

Giza, C.C, & Hovda, D.A. (2001). The neurometabolic cascade of concussion. *Journal of Athletic Training, 36*(3), 228-235.

Gordon, W. A., Cantor, J., Ashman, T., & Brown, M. (2006). Treatment of post-TBI executive dysfunction: Application of theory to clinical practice. *The Journal of Head Trauma Rehabilitation, 21*(2), 156-167.

Greenhill, L., Pliszka, S. & Dulcan, M. (2002). Practice parameter for the use of stimulant medications in the treatment of children, adolescents, and adults. *Journal of the American Academy of Child & Adolescent Psychiatry, 41*, 26S-29S.

Gronwall, D. (1977). Paced auditory serial addition test: A measure of recovery from concussion. *Perceptual and Motor Skills, 44*, 367-373.

Grossman, E, Jensen, J., Babb, J., Chen, Q., Tabesh, A., Fieremans,… & Grossman, R. (2012). Cognitive impairment in mild traumatic brain injury: Longitudinal diffusional kurtosis and perfusion imaging study. *American Journal of Neuroradiology, 34*(5), 951-957.

Gualtiere, C. & Johnson, L. (2006). Reliability and validity of a computerized neurocognitive test battery, CNS Vital Signs. *Archives of Clinical Neuropsychology, 21*(7), 623-643.

Hamilton, L.S., Levitt, J.G., O'Neill, J., Alger, J.R., Luders, E., & Phillips, O.R. (2008). Reduced white matter integrity in Attention-Deficit Hyperactivity Disorder. *NeuroReport, 19*(17). doi: 10.1097/WNR.0b013e3283174415

Hartlage, L.C., Durant-Wilson, D., & Patch, P.C. (2001). Persistent neurobehavioral problems following mild traumatic brain injury. *Archives of Clinical Neuropsychology, 16*(6), 561-570.

Helmick, K. (2010). Cognitive rehabilitation for military personnel with mild traumatic brain injury and chronic post-concussional disordr: Results of April 2009 consensus conference. *NeuroRehabilitation, 26,* 239-255. doi:10.3233/NRE-2010-0560

Hesslinger, B., Terbatz van Elst, L., Thiel, T., Haegele, K., Hennig, J., & Ebert, D. (2002). Frontoorbital volume reductions in adult patients with Attention Deficit Hyperactivity Disorder. *Neuroscience Letters, 328*(3), 319-321. doi: 10.1016/S0304-3940(02)00554-2

Ho, M. & Bennett, T. (1997). Efficacy of neuropsychological rehabilitation for mild-moderate traumatic brain injury. *Archives of Clinical Neuropsychology, 12*(1),1-11.

Hollingshead, A.A. (1975). Four factor index of social status. *Unpublished manuscript.*

Hoogman, M., Rijpkema, M., Janss, L., Brunner, H., Fernandez, G., Buitelaar, J., . . . Arias-Vasquez, A. (2012). Current self-reported symptoms of Attention Deficit-Hyperactivity Disorder are associtated with total brain volume in healthy adults. *Public Library of Science One, 7*(2). doi: 10.1371/journal.pone.0031273.t001

Inglese, M., Makani, S., Johnson, G., Cohen, B., Silver, J., Gonen, O., & Grossman, R. (2005). Diffuse axonal injury in mild traumatic brain injury: A diffusion tensor imaging study. *Journal of Neurosurgery,103*(2), 298-303. doi:10.3171/jns.2005.103.2.0298

Iverson, G.L. (2010). Mild traumatic brain injury meta-analyses can obscure individual differences. *Brain Injury, 24*(10), 1246-1255. doi:10.3109/02699052.2010.490513

Johansson, B., Berglund, P., & Ronnback, L. (2009). Mental fatigue and impaired information processing after mild and moderate traumatic brain injury. *Brain Injury, 23*(13-14), 1027-1040. doi:10.3109/02699050903421099

Karr, J.E., Areshenkoff, C.N., & Garcia-Barrera, M.A. (2014). The neuropsychological outcomes of concussion: A systematic review of meta-analyses on the cognitive sequelae of mild traumatic brain injury. *Neuropsychology, 28*(3), 321-336. doi:10.1037/neu0000037

Kashluba, S., Hanks, R., Casey, J., & Mills, S. (2008). Neuropsychological and functional outcome after complicated mild traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 89*(5), 904-911.

Kay, T., Harrington, D., Adams, R., Anderson, T., Berrol, S., Cicerone, K., . . . Malec, J. . (1993). Definition of mild traumatic brain injury. *Journal of Head Trauma Rehabilitation, 8*(3), 86-87.

Kessler, R.C., Barber, C., Beck, A., Berglund, P., Cleary, P.D., McKenas, D., … Wang, P. (2003). The World Health Organization Health and Work Performance Questionnaire (HPQ). *Journal of Occupational and Environmental Medicine,* 45 (2), 156-174.

Kessler, R., Leonard, A., Minnie, A., Barkley, R., Birnbaum, H., Greenberg, P.,. . .Usten, T. (2005). The prevalence and effects of adult Attention Deficit/Hyperactivity Disorder on work performance in a nationally representative sample of workers. *Journal of*

Occupational and Environmental Medicine, 47(16), 565-572. doi:10.1097/01.jom.0000166863.33541.39

Kessler, R., Adler, L., Barkley, R., Biederman,J., Conners, C.K., Demler, O., . . . Zaslavsky, A.M. (2006). The prevalence and correlates of adult ADHD in the United States: Results from the National Comorbidity Survey Replication. *American Journal of Psychiatry, 163*(4), 716-723.

Ko, C., Yen, J., Yen, C., Chen, C., Lin, W., Wang, P., & Liu, W. (2013). Brain activation deficit in increased-load working memory tasks among adults with ADHD using fMRI. *European Archives of Psychiatry and Clinical Neuroscience, 263*(7), 561-573. doi: 10.1007/s00406-013-0407-2

Kubik, J. A. (2010). Efficacy of ADHD coaching for adults with ADHD. *Journal of Attentional Disorders, 13*(5), 442-453. doi: 10.1177/1087054708329960

Kumar, L., Rao, S., Chandramouli, B., & Pillai, S. (2013). Reduced contribution of executive functions in impaired working memory performance in mild traumatic brain injury patients. *Clinical Neurology and Neurosurgery, 115*(8), 1326-1332. doi: 10.1111/j.1440-1819.2005.01400.x

Lewine, J., Davis, J., Bigler, E., Toma, R., Hill, D., Funke, M.,…& Orrison, W. (2007). Objective documentation of traumatic brain injury subsequent to mild head trauma: Multimodal brain amaging with MEG, SPECT, and MRI. *The Journal of Head Trauma Rehabilitation, 22*(3), 141-155.

Lezak, M., Howieson, D., Loring, D., Hannah, H., & Fischer, J. (2004). *Neuropsychological Assessment.* New York: Oxford University Press

Lo, C., Shifteh, K., Gold, T., Bello, J., & Lipton, M. (2009). Diffusion tensor imaging abnormalities in patients with mild traumatic brain injury and neurocognitive impairment. *Journal of Computer Assisted Tomography, 33*(2), 293-297. doi: 10.1097/RCT.0b013e31817579d1

Makris, N., Biederman, J., Valera, E., Bush, G., Kaiser, J., Kennedy, D., . . . Seidman, L. (2007). Cortical thinning of the attention and executive function networks in adults with Attention-Deficit/Hyperactivity Disorder. *Cerebral Cortex, 17*(6), 1364-1375. doi: 10.1093/cercor/bhl047

Maksimowski, M., & Tampi, R. (2016). Efficacy of stimulants for psychiatric symptoms in individuals with traumatic brain injury. *Annals of Clinical Psychiatry, 28*(3), 156-166.

Martel, M., Nikolas, M., & Nigg, J.T. (2007). Executive function in adolescents with ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry,46*(11), 1437-1444. doi: 10.1097/chi.0b013e31814cf953

Martinussen, R., Hayden, J., Hogg-Johnson, S., & Tannock, R. (2005). A meta-analysis of working memory impairments in children with Attention-Deficit/Hyperactivity Disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 44*(4), 377-384.

Maruta, J., Spielman, L. A., Tseretopoulos, I. D., Hezghia, A., & Ghajar, J. (2014). Possible medication-resistant deficits in adult ADHD. *Journal of Attentional Disorders*. doi: 10.1177/1087054714538659

McAllister, T. W., Sparling, M. B., Flashman, L. A., Guerin, S. J., Mamourian, A. C., & Saykin, A. J. (2001). Differential working memory load effects after mild traumatic brain injury. *Neuroimage, 14*(5), 1004-1012. doi: 10.1006/nimg.2001.0899

McKee, A. C., Cantu, R. C., Nowinski, C. J., Hedley-Whyte, E. T., Gavett, B. E., Budson, A. E., &… Stern, R. A. (2009). Chronic traumatic encephalopathy in athletes: Progressive tauopathy after repetitive head injury. *Journal of Neuropathology and Experimental Neurology, 68*(7), 709-735. doi: 10.1097/NEN.0b013e3181a9d503

Michelson, D., Adler, L., Spencer, T., Reimherr, F., West, S., Allen, A., . . . Milton, D. (2003). Atomoxetine in adults with ADHD: Two randomized, placebo-controlled studies. *Biological Psychiatry, 53*(2), 112-120. doi: 10.1016/S0006-3223(02)01671-2

Melby-Lervag, M. & Hulme, C. (2013). Is working memory training effective? A meta-analytic review. *Developmental Psychology, 49*(2), 270-291.

Mongia, M., & Hechtman, L. (2012). Cognitive behavior therapy for adults with Attention-Deficit/Hyperactivity Disorder: A review of recent randomized controlled trials. *Current Psychiatry Reports, 14*(5), 561-567. doi: 10.1007/s11920-012-0303-x

Murphy, K.R., & Barkley, R.A. (1996). Attention Deficit Hyperactivity Disorder adults: Comorbidities and adaptive impairments. *Comprehensive Psychiatry, 37*(6), 393-401. doi:http://dx.doi.org/10.1016/S0010-440X(96)90022-X

National Center for Injury Prevention and Control. (2003). *Report to Congress on Mild Traumatic Brain Injury in the United States: Steps to Prevent a Serious Public Health Problem*. Atlanta, GA: Centers for Disease Control and Prevention

Niogi, S. & Mukherjee, P. (2010). Diffusion tensor imaging of mild traumatic brain injury. *Journal of Head Trauma Rehabilitation, 24*(4), 241-255.

Niogi, S., Mukherjee, P., Ghajar, J., Johnson, C., Rachel, K., Lee, H.,…McCandliss, B. (2008). Structural dissociation of attentional control and memory in adults with and without mild traumatic brain injury. *Brain, 131*, 3209-3221.

O'Jile, J. R., Ryan, L. M., Betz, B., Parks-Levy, J., Hilsabeck, R. C., Rhudy, J. L., & Gouvier, W. D. (2006). Information processing following mild head injury. *Archives of Clinical Neuropsychology, 21*(4), 293-296. 10.1016/j.acn.2006.03.003

Palmese, C. & Raskin, S. (2000). The rehabilitation of attention in individuals with mild traumatic brain injury, using the APT-II programme. *Brain Injury*, *14*(6), 535-548.

Parente, R., & Herrman, D. (2008). *Retraining cognition*. Austin, TX: Pro-Ed.

Pertab, J., James K., & Bigler, E. (2009). Limitations of mild traumatic brain injury meta-analyses. *Brain Injury,23*(6), 498-508. doi: 10.1080/02699050902927984

Pliszka, S.R. (2003). Psychiatric comorbidities in children with Attention Deficit Hyperactivity Disorder: Implications for management. *Pediatric Drugs, 5*(11), 741-750.

Polanczyk, G., Silva de Lima, M., Horta, B.L., Biederman, J., & Rohde, L.A. (2007). The worldwide prevalence of ADHD: A systematic review and metregression analysis. *American Journal of Psychiatry*, *164*(6), 942-948.

Rabbit, P. (Ed.). (1997). *Methodologies and models in the study of executive function*. East Sussex, UK: Psychology Press Publishers.

Ramtekkar, U.P., Reiersen, A.M., Tdorov, A.A., & Todd, R.D. (2010). Sex and age differences in Attention-Deficit/Hyperactivity Disorder symptoms and diagnoses: Implications for DSM-V and ICD-11. *Journal of the American Academy of Child and Adolescent Psychiatry, 49*(3), 217-228.

Raskin, S. (2000). Executive functions. In Raskin, S. & Mateer, Catherine (Eds.), *Neuropsychological management of mild traumatic brain injury.* (pp. 113-133). New York: Oxford University Press.

Ravi, S. (2008). Cognitive rehabilitation for reversible and progressive brain injury. *Indian Journal of Psychiatry, 50*(4), 282-284. doi: 10.4103/0019-5545.44752

Reitan, R.M. & Wolfson, D. (1985). *The Halstead-Reitan Neuropsychological Test Battery: Theory and clinical interpretation:* Tuscon, TX, Neuropsychology Press.

Roberts, A., Robbins, T.W., & Weiskrantz, L.E. (Eds.). (1998). *The prefrontal cortex: Executive and cognitive functions.* New York, NY: Oxford University Press.

Rohling, M., Binder, L., Demakis, G., Larrabee, G., Ploetz, D., & Langhinrichsen-Rohling, J. (2011). A meta-analysis of neuropsychological outcome after mild traumatic brain injury: Re-analyses and reconsiderations of Binder et al. , Frencham et al. , and Pertab et al. *The Clinical Neuropsychologist, 25*(4), 608-623. doi: 10.1080/13854046.2011.565076

Rosvold, H., Mirsky, A., Sarason, E. (1956). A continuous performance test of brain damage. *Journal of Consulting Psychology, 20*, 343-350.

Roth R.M., Isquith P.K., & Gioia, G.A. (2005). Behavior Rating Inventory of Executive Function–Adult Version (BRIEF-A). Lutz, FL: Psychological Assessment Resources.

Ruff, R. (2009). Best practices guidelines for forensic neuropsychological examinations of patients with traumatic brain injury. *Journal of Head Trauma Rehabilitation, 24*(2), 131-140.

Ruff, R., Niemann, H., Allen, C., Farrow, C., & Wylie, T. (1992). The Ruff 2 and 7 Selective Attention Test: A neuropsychological application. *Perceptual and Motor Skills, 75*, 1311-1319.

Rutgers, D., Toulgoat, F., Cazejust, J., Fillard, P., Lasjuanias, P., & Ducreux, D., (2008). White matter abnormalities in mild traumatic brain injury: A diffusion tensor imaging study. *American Journal of Neuroradiology, 29*(3), 514-519. doi : 10.3174/ajnr.A0856

Safren, S., Perlman, C., Sprich, S., & Otto., W. (2005). *Mastering your adult ADHD : A Cognitive-behavioral treatment program therapist guide.* New York : Oxford University Press.

Safren, S., Sprich, S., Mimiaga, M., Surman, C., Knouse, L., Groves, M., & Otto, M. (2010). Cognitive behavioral therapy vs. relaxation with educational support for medication-treated adults with ADHD and persistent symptoms: A randomized controlled trial. *Journal of the American Medical Association, 304*(8), 875-880.

Salakari, A., Virta, M., Gronroos, N., Chydenius, E., Partinen, M., Vataja, R., . . . Iivanainen, M. (2010). Cognitive-behaviorally-oriented group rehabilitation of adults with ADHD: Results of a 6-month follow-up. *Journal of Attentional Disorders, 13*(5), 516-523. doi: 10.1177/1087054709332069

Sarason, I.G., Johnson, J.H., & Siegel, J.M. (1978). Assessing the impact of life changes: Development of the life experiences survey. *Journal of Consulting and Clinical Psychology, 46*(5), 932-946.

Schnitzspahn, K. M., Zeintl, M., Jager, T., & Kliegel, M. (2011). Metacognition in prospective memory: Are performance predictions accurate? *Canadian Journal of Experimental Psychology, 65*(1),19-26. doi:10.1037/a0022842

Schoenberg, P.L., Hepark, S., Kan, C.C., Barendregt, H.P., Buitelaar, J.K., & Speckens, A.E. (2013). Effects of mindfulness-based cognitive therapy on neurophysiological correlates

of performance monitoring in adult Attention-Deficit/Hyperactivity Disorder. *Clinical Neurophysiology, 125*(7), 1407-16. doi: 10.1016/j.clinph.2013.11.031.

Schulz, K.P., Fan, F.J., Bedard, A., Clerkin, S., Ivanov, I., Tang, C., . . . Newcorn, J. (2012). Common and unique therapeutic mechanisms of stimulant and nonstimulant treatments for Attention-Deficit/Hyperactivity Disorder. *Archives of General Psychiatry, 69*(9), 952-961. doi: 10.1001/archgenpsychiatry.2011.2053

Seidman, L. J. (2006). Neuropsychological functioning in people with ADHD across the lifespan. *Clinical Psychology Review, 26*(4), 466-485. doi: 10.1016/j.cpr.2006.01.004

Seidman, L., Valera, E., Makris, N., Monuteaux, M., Boriel, D., Kelkar, K.,. . . Biederman, J. (2006). Dorsolateral prefrontal and anterior cingulate cortex volumetric abnormalities in adults with Attention-Deficit/Hyperactivity Disorder identified by magnetic resonance imaging. *Biological Psychiatry, 60(10)*, 1071-1080. doi: 10.1016/j.biopsych.2006.04.031

Shallice, T., & Burgess, P. W. (1991). Deficits in strategy application following frontal lobe damage in man. *Brain, 114 (Pt 2)*, 727-741.

Shanahan, M., Pennington, B., Yerys, B., Scott, A., Broada, R., Wilcutt, E.,…& Defries, J. (2006). Processing speed deficits in Attention Deficit/Hyperactivity Disorder and reading disability. *Journal of Abnormal Child Psychology, 34*(5), 584-601. doi: 10.1007/s10802-006-9037-8

Shaw, M. , Hodgkins, P., Caci, H., Young, S., Kahle, J., Woods, A.G., & Arnold, L. (2012). A systematic review and analysis of long-term outcomes in Attention Deficit Hyperactivity Disorder: Effects of treatment and non-treatment. *BMC Medicine, 10*(1), 99-113. doi: 10.1186/1741-7015-10-99

Shenton, M. E., Hamoda, H. M., Schneiderman, J. S., Bouix, S., Pasternak, O., Rathi, Y.,… & Zafonte, R. (2012). A review of magnetic resonance imaging and diffusion tensor imaging findings in mild traumatic brain injury. *Brain Imaging & Behavior, 6*(2), 137-192. doi:10.1007/s11682-012-9156-5

Slomine, B. & Locascio, G. (2009). Cognitive rehabilitation for children with acquired brain injury. *Development Disabilities Research Reviews, 15*(2), 133-143. doi: 10.1002/ddrr.56.

Smalley, S.L., McGough, J.J., Del'Homme, M., NewDelman, J., Gordon, E., Kim, T.,...McCracken, J.T. (2000). Familial clustering of symptoms and disruptive behaviors in multiplex families with Attention-Deficit/Hyperactivity Disorder. *Journal of American Academy of Child and Adolescent Psychiatry, 39*(9), 1135-1143. doi:10.1097/00004583-200009000-00013\

Solanto, M. (2014). Executive function deficits in adults with ADHD. In Barkley, R. (Ed.), *Attention deficit hyperactivity disorder: A handbook for diagnosis and treatment* (pp. 256-267). New York: Guilford Press.

Solanto, M., Marks, D., Mitchell, K., Wasserstein, J., & Koffman, M. (2008). Development of a new psychosocial treatment for adult ADHD. *Journal of Attentional Disorders, 11*(6), 728-736. doi: 10.1177/1087054707305100

Sosnoff, J., Broglio, S., Shin, S., & Ferrara, M. (2011). Previous mild traumatic brain injury and postural-control dynamics. *Journal of Athletic Training, 46*(1), 85-91. doi:10.4085/1062-6050-46.1.85

Sprich, S., Biederman, J., Crawford, M.H., Mundy, E., & Faraone, S.V. (2000). Adoptive and biological families of childern and adolescents with ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry, 39*(11), 1432-1437.

Tay, S. Y., Ang, B. T., Lau, X. Y., Meyyappan, A., & Collinson, S. L. (2010). Chronic impairment of prospective memory after mild traumatic brain injury. *Journal of Neurotrauma, 27(1),* 77-83. doi: 10.1089/neu.2009.1074

Theiling, J., & Petermann, F. (2014). Neuropsychological profiles on the WAIS-IV of ADHD adults. *Journal of Attentional Disorders.* doi: 10.1177/1087054713518241.

Tiersky, L.A., Anselmi, V., Johnston, M.V., Kurtyka, J., Roosen, E., Schwartz, T., & Deluca, J. (2005). A trial of neuropsychologic rehabilitation in mild-spectrum traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 86*(8), 1565-1574.

Tombaugh, T. (2004). Trail Making Test A and B: Normative data stratified by age and education. *Archives of Clinical Neuropsychology, 19*(2), 203-214. doi: 10.1016/S0887-6177(03)00039-8

Twamley, E., Jak, A., Delis, D., Bondi, M., & Lohr, M. (2014). Cognitive Symptom Management and Rehabilitation Therapy (CogSMART) for veterans with traumatic brain injury: Pilot randomized controlled trial. *Journal of Rehabilitation Research and Development, 51*(1), 59-70. doi: 10.1682/JRRD.2013.01.0020

Twamley, E., Thomas, K., Gregory, A., Jak, A., Bondi, M., Delis, D., & Lohr, J. (2015). CogSMART compensatory cognitive training for Traumatic Brain Injury: Effects over 1 year. *Journal of Head Trauma Rehabilitation, 30*(6), 391-401. doi: 10.1097/HTR.0000000000000076

U.S. Department of Health and Human Services, Centers for Disease Control and Prevention. (2012). *Summary health statistics for U.S. children: National health interview survey* (DHHS Publication No. (PHS) 2013-1582). Retrieved from http://www.cdc.gov/nchs/data/series/sr_10/sr10_254.pdf

Vidal-Estrada, R., Bosch-Munso, R., Nogueira-Morais, M., Casas-Brugue, M., & Ramos-Quiroga, J. A. (2012). Psychological treatment of Attention Deficit Hyperactivity Disorder in adults: A systematic review. *Actas Espanolas de Psiquiatria, 40*(3), 147-154.

Virta, M., Salakari, A., Antila, M., Chydenius, E., Partinen, M., Kaski, M., … & Iivanainen, M. (2010). Short cognitive behavioral therapy and cognitive training for adults with ADHD - A randomized controlled pilot study. *Neuropsychiatric Disease and Treatment, 6*, 443-453.

Wechsler, D. (1997) *Wechsler Adult Intelligence Scale -Third Edition (WAIS-III).* [Measurement instrument]. Retrieved from http://www.pearsonclinical.com/psychology/products/100000243/wechsler-adult-intelligence-scale--third-edition-wais-iii.html.

Weiss, M., Murray, C., Wasdell, M., Greenfield, B., Giles, L., & Hechtman, L. (2012). A randomized controlled trial of CBT therapy for adults with ADHD with and without medication. *BioMed Central Psychiatry, 12*(30), 1-8. doi: 10.1186/1471-244x-12-30

Welsh, M. C., & Pennington, B. F. (1988). Assessing frontal lobe functioning in children: Views from developmental psychology. *Developmental Neuropsychology, 4*, 199-230.

Wigal, T., Swanson, J., Regino, R., Lerner, M., Soliman, I., Steinhoff, K., . . . Wigal, S.. (1999). Stimulant medications for the treatment of ADHD: Efficacy and limitations. *Mental Retardation and Developmental Disabilities Research Reviews, 5*(3), 214-224.

Wiggins, D., Singh, K., Getz, H., & Hutchins, D. (1999). Effects of brief group intervention for adults with Attention Deficit/Hyperactivity Disorder. *Journal of Mental Health Counseling, 21*(1), 82-92.

Willer, B., Rosenthal, M., Kreutzer, J, Gordon W., & Rempel, R. (1993). Assessment of community integration following rehabilitation for traumatic brain injury. *Journal of Head Trauma Rehabilitation, 8,* 75-87.

Table 1

Cognitive Deficits and Recommended Treatment Techniques

Study	Deficit	Techniques Used
Cicerone et al. (1996)	Attention	Withstanding distractions while real life or paper and pencil tasks
	EF	Compensatory Strategies
Palmese and Raskin (2000)	Attention	APT-II
Cicerone (2002)	Attention/WM	N-back procedures
Tiersky et al. (2005)	Psychological	CBT
	Attention	APT-II
Gordon et al. (2006)	Attention	APT-II
	EF	Problem Solving Training
	Psychological	Emotional Regulation Training
Parrente & Herrman (2008)	Attention/EF	Mental exercises/remediation Strategy training Organization Skills Problem solving training
	Memory	Environmental modifications External aids
	Psychological	Addressing nutrition, sleep, and emotional issues.
Slomine & Locascio (2009)	Attention	APT-II
	EF	Problem Solving Training
	Psychological	Emotional Regulation Training
Helmick (2010)	Attention	Remediation
	Memory	Memory Strategies External Aids
	EF	Problem Solving Training Metacognitive Strategies
Twamley et al. (2014)	EF	Problem Solving Self Talk Hypothesis Testing Self-Monitoring

BIOGRAPHICAL SKETCH

Ashley Yates
ayates@utexas.edu

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE	YEAR(s)	FIELD OF STUDY
The University of Texas at Austin	B.A.	2012	Psychology
The University of Texas Southwestern School of Health Professions	M.C.R.C.	2016	Rehabilitation Counseling

Positions and Employment

2015-2016 Therapist/Discharge Planner Aspire Health Partners

Clinical Experience

2013-2014 Rehabilitation Counseling Intern-Pate Rehabilitation
2012-2013 Rehabilitation Counseling Intern-Metrocare Services