

Traumatic Brain Injury and Cognitive Remediation

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Purpose of This Paper

The purpose of this paper is to provide an overview of the recent literature regarding traumatic brain injury and the potential opportunities for cognitive remediation. Traumatic brain injury (TBI) is a significant public health concern and a major cause of permanent disability. TBI can have broad effects on physiology, cognition, emotional regulation and social interactions, with long-term outcomes ranging from complete recovery to disability and death. This review of what is known about the brain changes that occur following a TBI and recent the advances in cognitive remediation strategies are offered as a guide to physicians, educators and other professionals in making recommendations to individuals with TBI and their caregivers who are interested in programs informed by neurobiological data to improve cognitive abilities.

Traumatic Brain Injury

Traumatic brain injury (TBI) is caused by a blow or jolt to the head, or by a penetrating head injury that disrupts the normal function of the brain. Each year in the United States, at least 1.7 million TBIs occur either as an isolated injury or in combination with other injuries, according to the Centers for Disease Control (CDC, 2013). An estimated 70-90% of these injuries fall into the mild TBI (mTBI) classification, however, these numbers likely underrepresent TBI incidences, as they do not factor persons treated for TBI in settings other than a hospital or emergency room (Cassidy, 2004; Langlois, 2006). For example, individuals treated in outpatient settings or in primary care offices are not counted. Military personnel treated in military facilities domestically and abroad are also not included in these statistics, although the Department of Defense tracks diagnosed TBI through the Defense and Veterans Brain Injury Center (DVBIC, 2013). Mild cases of TBI may go completely unreported, especially if the injured person does not recognize and report symptoms that would indicate a possible brain injury has occurred.

According to the CDC, the leading causes of TBI are falls (35%), motor vehicle accidents (17.3%), struck by or against events (16.5%), and assaults (10%). Falls cause 50% of the TBIs among children aged 0-14, and among adults aged 65 years and older. While motor vehicle accidents are the second leading cause of TBI, they make up the

largest percentage of TBI-related deaths (31.8%). The very young (four years and younger) and adults over 75 have the highest risk of a TBI, with males diagnosed more frequently (59%) than females.

Soldiers returning from combat in Iraq and Afghanistan have been profoundly affected by TBI with an estimated one in five soldiers experiencing a head injury (Warden, 2006; Okie, 2005). Blasts from improvised explosive devices are a leading cause of TBI among active duty soldiers who suffer the effects of the blast wave and impact of explosion fragments. According to the data published by the DVBIC, the total number of service members with TBI diagnoses has more than doubled in the past five years with the majority categorized in the mild and moderate categories (DVBIC, 2013). Consequently, TBI has been named as the signature wound of the War in Iraq and Afghanistan.

Sports and recreation activities are also a major cause of TBI with the highest rates among males aged 10-19 years and a 62% increase in prevalence between 2001 and 2009 (Gilchrist, 2011). These data and other reports have led to the development and implementation of prevention strategies in schools and professional sports settings to raise public awareness (DeKosky, 2010). The International Conference on Concussion in Sport was formed to examine sport concussion and management issues and assembles every four years to revise and update their recommendations for prevention and management, including time to return to play (McCrorry, 2013).

TBI is classified into mild, moderate and severe categories, typically using three independent grading systems (Saatman, 2008; Moore, 2012). The Glasgow Coma Scale (GCS) grades a person's level of consciousness on a scale from 3-18, based on verbal, motor, and eye-opening reactions to stimuli. Post-traumatic Amnesia (PTA) and Loss of Consciousness (LOC) scales are also considered in the classification process and both are scored on duration; PTA scores number of days and LOC scores in minutes to hours. The overall TBI diagnosis is based on a combination of these scores and other medical information, including neuroimaging data, to determine the severity of the damage. These classification measures tend to be less useful in mild TBI, which make up the majority of TBI incidents, but where the symptoms are much less obvious and there is heavier reliance on self-reporting (Saatman, 2008; DeKosky, 2010).

The Pathology of Traumatic Brain Injury

Direct mechanical trauma causes the sudden movement of the skull on its axis which in turn produces a rotational, acceleration, or deceleration injury to the underlying brain tissue, termed the Primary Traumatic Brain Damage (Dekosky, 2010; Moore, 2012). The primary damage often results in focal and diffuse injuries incurred from the mechanical forces that produce direct damage to cerebral blood vessels, long axons interconnecting brain regions, neurons, and glial cells. Secondary Traumatic Brain Damage refers to the cascade of physiological changes that occur in the brain due to complications from the primary damage and can include brain tissue hypoxia, ischemia, hydrocephalus, raised intracranial pressure, and infection. In cases of mild TBI, patients may lose consciousness briefly and experience headache, dizziness, blurred vision, fatigue and balance problems. Cognitively, a patient may experience confusion and changes in memory, concentration, attention and thinking. Recovery from these symptoms can range from days to weeks or months.

The symptoms resulting from the primary damage depend on the part of the brain affected and whether the injury is focal, diffuse or both. Specific regions that appear to be particularly sensitive to concussive forces include the midbrain and diencephalon in which the reticular activating system is located (Ropper, 2007; Pearce, 2007). Other regions affected include the upper part of the brainstem, the fornix, the corpus callosum, the temporal lobe, and the frontal lobes (Taber, 2006; Schneiderman, 2008). Until recently, most of these regions have been identified based on symptomology rather than clinical imaging results as the diffuse, microscopic injuries are below the level of detection of standard emergency room CT or MRI scans.

The primary damage disrupts neuronal cell membranes and stretches axonal projections, causing a disturbance in the ion conductance across the membranes. This is followed by the widespread release of neurotransmitters, particular glutamate, which further excites surrounding neurons to release additional neurotransmitters (Cernak, 2010; Barkhoudarian, 2011; Helmy, 2011). Traumatic brain injury also induces changes in brain metabolism, as measured by initially increased (sign of metabolic stress) and then decreased levels of the metabolites, N-acetylaspartate and creatine-containing

compounds in the hours and days following the injury (Vagnozzi, 2010; Barkhoudarian, 2011). In mild TBI, these changes are limited, but there is evidence suggesting that this sequence of molecular events temporarily makes the individual more susceptible to subsequent brain injuries and more severe, longer lasting damage (Henry, 2010).

Neuroimaging of Traumatic Brain Injury

As noted above, traditional emergency room neuroimaging techniques typically do not have sufficient resolution to detect the microscopic and functional damage caused in mild traumatic brain injuries. However, techniques measuring functional neurological changes, such as fMRI, have proven to be more effective in elucidating the brain regions affected in mTBI. Current data suggest that the neural activation patterns in TBI subjects have significant differences compared to healthy controls during cognitive tasks (Ptito, 2007; Prabhu, 2011). Regions affected include the right parietal and right dorsolateral frontal regions, in the prefrontal cortex, and in the cingulate region. Reductions in brain region volume have been reported in cortical and subcortical areas crucial for executive functioning, including the hippocampus, amygdala, thalamus, superior frontal cortex, superior parietal cortex, and precuneous cortical areas (for review, see Shenton, 2012).

As a hallmark of mild TBI is axonal stretching and injury, the technique of diffusion tensor imaging (DTI) has been highly informative for the characterization of subtle changes in white matter fiber tracts and for revealing microstructural axonal injuries (Shenton, 2012). DTI reflects myelin integrity and is based on the characteristic of myelin sheaths and cell membranes of white matter tracts that restricts the movement of water molecules. Pathological changes that affect axonal projections and microstructure in turn influence the diffusion of water molecules and these changes can be quantified using DTI. Regions particularly susceptible to injury following TBI include the anterior and posterior corpus callosum, the anterior and posterior limbs of the internal capsule, and the fornix (Kinnunen, 2010; Singh, 2010). The magnitude of the DTI changes appear to be positively correlated with the severity of the damage and provide an explanation for the prolonged symptoms and cognitive difficulties experienced by patients with mild TBI who appear to have otherwise recovered from

their primary brain injury (Wilde, 2008). These techniques have also been utilized to monitor brain activation during the performance of cognitive tasks to ascertain the efficacy of cognitive remediation techniques and these studies will be described in more detail in sections below.

As the consequences of mild traumatic brain injuries can last weeks to months or longer, it is not surprising that these injuries carry a tremendous financial burden, estimated at 60 billion dollars in direct medical costs and indirect costs due to lost productivity (Xu, 2010; CDC, 2013). With the growing incidence of TBI in our escalating aging population, in soldiers returning from combat, in child and adult athletes, and in the general population, there is a critical need for a systematically applied set of therapeutic services that restore the executive functioning that can be restored, improve quality of life, and lessen the societal burden. The most effective interventions will be those that target the root causes of cognitive dysfunction and utilize strategies based on the current knowledge of brain plasticity following injury.

The Learning Model and Cognitive Remediation

Executive function is an overarching term that is inclusive of cognitive abilities such as attention, working memory, problem solving, logic & reasoning, inhibitory control, and multitasking. These and other intellectual functions can be clustered in several different measurable domains based on the Cattell-Horn-Carroll (CHC) theory of intelligence, the most researched and widely accepted of theory of the composition of intellectual abilities (Cattell, 1941; Horn, 1965; Carroll, 1993). The concept of fluid intelligence (Gf), or fluid reasoning, comes from the CHC theory and is defined as the ability to reason abstractly and solve novel problems.

The Woodcock-Johnson Tests of Cognitive Abilities, Third Edition and the Woodcock-Johnson Tests of Achievement are the gold standards for comprehensive assessment of these cognitive abilities. These tests measure strengths of key cognitive skills and help identify areas of weakness that might benefit from cognitive training and improve reading ability:

- *Attention (three types)*: Sustained attention is the ability to stay on task; selective attention is the ability not to be distracted; divided attention is the ability to handle more than one task at a time.
- *Short-term (Working) memory*: The ability to apprehend and hold information in immediate awareness while simultaneously performing a mental operation.
- *Processing speed*: The ability to perform automatic cognitive tasks, particularly when measured under pressure to maintain focused attention.
- *Logic & reasoning*: The ability to reason, form concepts, and solve problems using unfamiliar information or novel procedures.
- *Visual processing*: The ability to perceive, analyze, and think in visual images.
- *Auditory processing (phonemic awareness)*: The ability to analyze, blend, and segment sounds.
- *Long-term memory*: The ability to recall information that was stored in the past.

This testing also generates a General Intellectual Ability (GIA) score, also referred to as general intelligence or IQ (Woodcock, 2001). Improvements specific to reading abilities can be measured using Word Attack, which is the ability to apply phonic and structural analysis skills to pronounce unfamiliar printed words and is a subtest of the Woodcock Johnson III Tests of Achievement (Woodcock, 2011).

Cognitive deficits associated with TBI include impaired attention, disrupted judgment and thought, reduced processing speed, distractibility, and executive functioning deficits including abstract reasoning, planning, problem solving, and multitasking (Kinnunen, 2010; Chen, A., 2012). Memory loss and dysfunction is the most common cognitive impairment and may be associated with deficits in understanding spoken or written language. In the cases of mild TBI, these deficits may be subtle and difficult to detect with standard neuropsychological testing, but still be sufficient to interfere with activities of daily living, driving, and work or school performance (Erez, 2009).

Executive function is based primarily in the prefrontal regions of the frontal lobe, although visual and auditory processing tasks also invoke activity in the occipital and auditory lobes, respectively (Alvarez, 2006). There are substantial amounts of imaging and electrophysiological data correlating cognitive training with physical brain changes

in regions associated with specific cognitive tasks (for reviews, see Musiek, 1995; Fey, 2011; Rabipour and Raz, 2012). Finally, a review of DTI studies indicates that there is a direct correspondence between the clinical manifestations of TBI patients and the damage observed in specific white matter tracts (Zappala, 2012). The extensive nature of these injuries to axonal projections may limit full recovery of cognitive functioning, however, evidence of brain plasticity supports the engagement of behavioral and cognitive interventions to enhance quality of life and improve functioning as much as is physiologically possible following brain injury.

Working Memory and Attention in TBI

Working memory is the capacity to maintain and manipulate information in the presence of distraction and is directly related to the ability to hold attention. It is a system to actively hold information when needed for verbal and nonverbal tasks such as reasoning and reading comprehension, and to make them available for further processing. Neuroimaging studies have identified prefrontal regions of the frontal lobe and superior parietal regions with working memory (Wager, 2003). For spatial working memory, widely distributed neural activation is consistently reported in the dorsolateral prefrontal cortex, as well as in the inferior parietal sulcus, anterior cingulate cortex, basal ganglia, and cerebellum (Curtis, 2004).

Several recent studies in TBI patients indicate cortical activity deficits associated with working memory and sustained attention tasks. In an fMRI study using a two-back task for working memory and a visual sustained attention task, individuals with moderate TBI were less successful in their performance than healthy controls. Brain activation differences were observed both at rest and during task performance in the bilateral superior occipital cortices and the left superior temporal cortex when TBI patients were compared to controls (Kim, 2012). Another study examined the effects of TBI specific to the anterior cingulate cortex (ACC), as ACC activity has been correlated with task difficulty and figures prominently in executive function tasks (Cazalis, 2010). In this study, the task was a spatial-temporal working memory task that required patients to reproduce visual sequences in multiple trials at increasing levels of difficulty. TBI adolescents were compared with controls and then re-evaluated 12 months later. As

expected, the TBI patients exhibited worse performance with higher response times and poorer accuracy than controls on the task. In both groups, the task activated a network including the prefrontal cortex, the cingulate cortex, pre- and post-central gyri, parietal and occipital cortices, temporal gyri, and the head of the caudate nucleus. In the TBI group, the anterior cingulate cortex was significantly activated with increasing difficulty in the working memory task and no change was observed in the left sensorimotor cortex. The opposite activation pattern was observed in the control groups which the authors interpret as a difference in recruitment of cortical areas to perform the task. Upon retesting 12 months later, TBI adolescents demonstrated a decrease in their response time and showed a brain activation pattern more similar to controls, indicative of functional recovery over time.

Similar improvements have been reported in athletes recovering from sports-induced TBI (Ptito, 2007). A group of 16 injured athletes were compared with healthy controls using an externally ordered working memory task. In the more injured athletes, there were weaker task-related activations in the posterior prefrontal cortex and less bilateral activation of the dorsolateral prefrontal cortex than in controls. One subject tested three months later demonstrated both behavioral improvement on the task and augmented prefrontal activation associated with the task that was similar to the control subjects.

Electrophysiological studies have also been effective in detecting brain activation differences in brain activity following mTBI. Event-related potential (ERP) studies utilize noninvasive methods to measure electrophysiological signals associated with cognitive processes during a task. In an externally ordered working memory task, mTBI patients had smaller response amplitudes of frontal and parietal ERP components when compared with controls (Gosselin, 2012). These decreases were associated with slower reaction times and worse accuracy during task performance. Other electrophysiological studies have demonstrated differences in activation and working memory processing resources in mTBI patients compared to controls, even when there were not measurable differences in task performance (McCrea, 2009). Compensatory recruitment of undamaged cortical regions may explain the subtlety of cognitive symptoms in mTBI, as evidenced by sensitive imaging and electrophysiological

techniques. As these activation patterns may return to a more normal pattern during recovery, this time window of neural plasticity may be optimal for cognitive interventions to strengthen and accelerate the recovery process.

Cognitive rehabilitation studies on memory following TBI are limited but suggest that targeted interventions may improve function (Chen, 2012). In one study, TBI patients received six weeks of a group intervention that emphasized internal memory strategy training (Strangman, 2010). The intervention ran twice weekly for six weeks at 90 minutes per session and focused on semantic organization, elaboration, and imagery from encoding, storage and retrieval perspectives. The investigators also examined the volume of cortical regions involved in memory and executive functioning before and after cognitive training. Improvements in memory following the cognitive training were positively correlated with increased hippocampal and ventrolateral prefrontal cortical volumes and with increased volume in the cingulate gyrus. Other studies using computerized working memory training programs indicate that intensive programs can also improve cognitive functioning (Klingberg, 2010; Lundqvist, 2012), although the generalizability of these techniques to improvement of overall cognitive functioning remains questionable and may be more effective in combination with other cognitive remediation programs (Cicerone, 2011; Rabipour, 2012).

TBI and Information Processing

Processing speed, visual processing and logic & reasoning encompass cognitive skills that share the underlying neural circuitry of the prefrontal and parietal brain regions involved in attention and working memory. In addition, fMRI data supports the involvement of the lateral frontopolar cortex, the inferior parietal sulcus area, ventral and premotor cortex in cognitive tasks that involve decision making under pressure and distraction (Bunge and Wendelken, 2009). Visual processing is frequently involved as distinctions must be made between stimuli and visualization and imagery are part of many reasoning tasks and involves activation of occipital cortex (Haxby, 1991; Malach, 1995).

Mental fatigue and slower processing speeds are commonly reported after TBI and are associated with damage to sensory and cognitive processing pathways.

Measures of processing speed, including digit symbol-coding, reading speed, and the trail making test, are all decreased in mTBI (Johansson, 2009). In structural MRI studies analyzing brain volume during tests of processing speed, a distributed pattern of volume loss in TBI patients in temporal, ventromedial prefrontal, and right parietal regions and cingulate regions (Levine, 2013). Inflammation and microglial activation has been reported in similar cortical and subcortical regions following brain injury. These changes also adversely affect processing speed and can last for years following the initial injury (Ramlackhansingh, 2010).

Abnormalities have also been detected using ERP analysis of TBI patients performing auditory and visual processing tasks (Lew, 2004). In simple discrimination tasks, response accuracy was not different between TBI patients and healthy controls; however, the mean reaction time for both auditory and visual modalities was reduced in the TBI group. This difference was correlated with diminished P300 amplitude and delayed peak latency, indicators of processing speed.

Information processing speed is critical for overall cognitive functioning and remediation strategies invoking multiple cognitive domains have had some success in improving processing speed in individuals with TBI. For example, cognitive remediation that incorporated direct attention training along with problem-solving strategies and with cognitive behavioral therapy demonstrated improvements in complex or divided attention tasks (Cicerone, 2011). Time pressure management strategies may also be effective in improving processing speed following brain injuries and have the potential to generalize from task improvement to broader gains in concentration, speed, and memory (Fasotti, 2000). These studies are limited in their reproducibility and the difficulties associated with the variability of TBI. Evidence clearly supports the value of interventions and a recent review of the literature provides recommendations that include tailoring the interventions to the level of injury and impairment and using direct attention training and metacognitive training to promote self-directed strategies and generalization to daily living tasks (Cicerone, 2011).

Auditory Processing and TBI

Auditory processing is the ability to analyze, synthesize, and discriminate auditory stimuli, including the ability to process and discriminate speech sounds that may be presented under distorted conditions. It is a complex ability that includes phonemic awareness, discrimination between tones, and tracking auditory temporal events. Auditory problems are common long-term symptoms following mTBI and can include difficulty processing auditory information, including difficulty listening in background noise, inability to follow oral instructions, and difficulty understanding speech (Vander Werff, 2012). Auditory dysfunction is one of the most prevalent military-related disabilities associated with blast-related TBI (Fausti, 2009). Brain regions associated with auditory processing, such as the temporal lobe, corpus callosum, and thalamus are vulnerable to diffuse axonal damage, which can contribute to observed TBI auditory deficits.

To date, few studies examining central auditory changes following TBI are available, although there are electrophysiological data suggesting abnormal auditory brainstem responses and long-latency auditory cortical evoked potentials in TBI patients (Fliger, 2002; Mazzini, 2001). The auditory P300 event-related potential has been the most frequently studied technique in TBI individuals. As noted in the previous section, this potential is used to assess deficits in attention, memory and processing speed in the auditory modality and significant differences have been noted in amplitude and latency when TBI participants are compared with controls (Lew, 2004).

Evoked potential and auditory P300 techniques have been used to evaluate the efficacy of cognitive rehabilitation on auditory processing (Folmer, 2011). In different populations, auditory evoked potentials have been shown to be sensitive to auditory training and remediation. However, most reports in the literature focused specifically on auditory processing and remediation are limited to single-subject case study and a broad range of injury severity (Murphy, 2011). While encouraging, more research is needed to establish the efficacy of training and whether it is limited to improvements in the auditory modality or generalizable to other modalities and functions.

Behavioral and Cognitive Interventions for Traumatic Brain Injury

In an effort to establish evidence-based standard recommendations for effective cognitive rehabilitation strategies, a comprehensive review of the literature as it pertains to traumatic brain injury and stroke was recently conducted (Cicerone, 2011). Cicerone (2011) built from previous reviews commissioned by The Cognitive Rehabilitation Task Force of the American Congress of Rehabilitation Medicine Brain Injury Interdisciplinary Special Interest Group, which served as the basis for specific recommendations for TBI rehabilitation. Most early studies provided TBI patients with external supportive devices and strategies to enhance functional memory difficulties, but did not address the root causes of the ongoing cognitive deficits. There are a variety of studies on cognitive rehabilitation techniques that range from single subject cases to well-designed randomized control trials. The most consistent findings and strongest recommendations for practice standards from this comprehensive review are the engagement of direct attention training and of meta-cognitive training to increase awareness and enhance compensatory strategies. Other areas emphasized in the study included working memory training, memory strategy training, and self-monitoring and self-regulation for deficits in executive functioning.

Although peer-reviewed scientific evidence supports the effects of cognitive training on brain plasticity, the concept of cognitive training remains controversial. This may be due, in part, to a lack of consistency between research and cognitive training approaches used. Many cognitive training approaches demonstrate efficacy, but it is rarely transferable beyond the skill being trained. To generalize the effects of cognitive training beyond a single skill, the review recommended tailoring the interventions based on the severity of TBI and the cognitive deficits identified (Cicerone, 2011). The desired outcome for these patients is a better quality of life and overall improved functioning that is more likely to be achieved with interventions engaging multiple cognitive domains rather than remediation strategies focusing on a single function.

In addition to the cognitive remediation strategies, cognitive behavioral psychotherapy (CBT) appears to influence recovery from injury and improvement of overall functioning (Tiersky, 2005). In a trial examining the effect of combined cognitive behavioral therapy and cognitive remediation, TBI patients exhibited less anxiety and

depression and improved performance on RAVLT learning trials. This study is somewhat limited as it lacked groups that examined the CBT and cognitive training independently. However, in most 'real world' situations, it is likely that a multiplicity of factors including, but not limited to, cognitive remediation strategies will produce the best outcomes for those recovering from TBI.

LearningRx Programs and TBI

The LearningRx approach to cognitive training strengthens weak cognitive skills by targeting long-term memory, processing speed, logic & reasoning, working memory, visual processing, auditory processing, and attention – all areas recommended by the Cognitive Rehabilitation Task Force as practice standards. Many of the LearningRx strategies include time pressure management approaches to enhance sustained improvements in cognitive function. The focus of LearningRx cognitive training programs is to strengthen underlying brain skills that are essential for learning. This system trains the student to develop the appropriate strategy to complete a given task through the structured experience provided by the training procedures (see www.learningrx.com for more information). A synergistic “drill for skill” and metacognitive approach to developing cognitive skills provides the framework for a successful system achieving sustained results. A brain-based approach to cognitive remediation is used that considers recent literature about neural plasticity and cortical regions that have been shown to be involved in the acquisition and improvement of different cognitive skills. The LearningRx system tailors the program to a level appropriate for the individual, a characteristic particularly salient for TBI participants as injuries and functional deficits vary widely. The training incorporates a broad range of cognitive skills to demonstrate improvements not only in individual components, but also in overall IQ scores.

Unlike other intervention programs, the LearningRx program doesn't make assumptions about the root cause of cognitive difficulties. Instead, the program begins with the gold standard of assessment tools, the Woodcock Johnson Tests of Cognitive Abilities and the Word Attack Test from the Woodcock Johnson III Tests of Achievement. Using these tools, weak areas are identified and targeted with intensive training programs. The LearningRx reading program includes the ThinkRx training

consisting of 24 procedures with over 1,000 levels available based on individual ability, with tasks becoming more difficult as training progresses. All cognitive skills are addressed, but the program can be tailored to meet individual needs and strengthen deficient areas. This program is a comprehensive and intensive program that is continually being informed by the latest neurobiological and cognitive science research. It is not constrained by a bias toward a phonological awareness or a visually-focused approach, but recognizes the interconnectivity and interdependence of these neurobiological systems in the development of cognitive skills.

The value of LearningRx interventions that invoke several cognitive domains is supported by multiple studies utilizing the LearningRx cognitive training system (Carpenter, 2009; Luckey, 2009; LearningRx, 2011). Data collected from more than 2,000 participants indicate that tasks emphasizing auditory or visual processing and requiring attention and reasoning throughout training have profound effects on cognitive abilities (Luckey, 2009). In another study of 1,277 children aged 10-19, significant gains were reported in measures of working memory and processing speed following participation in LearningRx cognitive training programs (Pfister, 2012). Working memory and processing speed are cognitive domains that are profoundly impacted by TBI and that have the potential to benefit from these innovative training programs. Another recent study utilizing the LearningRx cognitive training program supports the importance of strengthening specific cognitive skills with evidence of excellent gains in post-training central auditory testing in children with CAPD (Jedlicka, 2008). Similar gains were reported in adults with Auditory Processing Disorders who also underwent the LearningRx cognitive training tasks with improvements in filtered words, competing words and competing sentences. This is particularly compelling for brain-injured military personnel who consistently exhibit auditory processing deficits following exposure to acute blast trauma (Fausti, 2009).

Indeed, specialized LearningRx cognitive training protocols have been developed for post-deployment soldiers and several pilot studies have been conducted with promising results (LearningRx, 2011). In January 2010, LearningRx in partnership with the Washington State Department of Veterans Affairs and the Warrior Transition Battalion (WTB) in Washington state conducted a pilot program to train and improve the

cognitive functioning of 15 volunteer WTB active duty service men and women who were suffering from TBI. The program format included six hours intensive one-on-one (three hours) and online (three hours) cognitive skills training per week. WTB soldiers who entered and remained in the program (11 of the original 15 volunteers) gained significant improvement in cognitive functioning, including elimination of symptoms such as memory loss, poor concentration and difficulty organizing thoughts. Specifically, the soldiers demonstrated significant improvement from pre- to post-testing with 12 subscales of the Woodcock Johnson III Tests of Cognitive Abilities, tasks measuring working memory, and six subscales of the Woodcock Johnson III Tests of Achievement. Qualitative feedback from the soldiers was exceptionally positive, including one soldier who wanted these resources opened up to more soldiers suffering from post-deployment TBI.

Another study in the Colorado Springs region with 35 soldiers or veterans referred from Fort Carson, Colorado and from VA case workers in Colorado Springs was recently completed with similar improvements in all areas of cognitive functioning tested (Mitchell, 2012). Results from these pilot studies as well as published recommendations indicate that LearningRx cognitive training is a well-established, clearly defined and manualized cognitive training program that could be made immediately available to thousands of soldiers to improve functioning, return to duty and quality of life. Veterans afflicted with TBI could also benefit from cognitive rehabilitation with improved functioning, quality of life, and reduced long-term health care costs.

These initial studies in TBI populations clearly indicate that the LearningRx cognitive training programs have the potential to be highly effective in remediating deficits in the extensive military population and for other populations suffering from TBI. As health care providers, caregivers, and other professionals look for effective behavioral interventions to enhance recovery after brain injury, it is essential to critically evaluate the programs and their basis in scientific knowledge. The LearningRx system provides an excellent resource as a uniquely multidimensional, multisensory program grounded in the most recent neuroscience research, regularly informed by a scientific advisory board, and supported by data from thousands of students who have benefited from the programs.

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