Seton Hall University

eRepository @ Seton Hall

Seton Hall University Dissertations and Theses (ETDs)

Seton Hall University Dissertations and Theses

Fall 9-18-2019

Identifying the Prevalence of Sensory Processing Dysfunction in a Post-Concussion Population and it's Relationship with Quality of Life and Functional Indicators

Stephen Van Lew
stephen.vanlew@student.shu.edu

Follow this and additional works at: https://scholarship.shu.edu/dissertations

 Part of the Occupational Therapy Commons

Recommended Citation

Van Lew, Stephen, "Identifying the Prevalence of Sensory Processing Dysfunction in a Post-Concussion Population and it's Relationship with Quality of Life and Functional Indicators" (2019). *Seton Hall University Dissertations and Theses (ETDs)*. 2713.
https://scholarship.shu.edu/dissertations/2713

IDENTIFYING THE PREVALENCE OF SENSORY PROCESSING DYSFUNCTION IN A POST-CONCUSSION POPULATION AND ITS RELATIONSHIP WITH QUALITY OF LIFE AND FUNCTIONAL INDICATORS

BY

Stephen Van Lew

Dissertation Committee

Genevieve Pinto Zipp, PT, EdD (Chair)

Terrence Cahill, EdD, FACHE

Fortunato Battaglia, MD, PhD

Submitted in partial fulfillment of the
requirements for the degree of Doctor of Philosophy in Health Sciences
Seton Hall University
2019

Copyright © 2019 Stephen Van Lew
All rights reserved

SETON HALL UNIVERSITY
SCHOOL OF HEALTH AND MEDICAL SCIENCES
Department of Interprofessional Health Sciences and Health Administration

APPROVAL FOR SUCCESSFUL DEFENSE
and
COMPLETION OF DISSERTATION MANUSCRIPT

Stephen F. Van Lew has successfully defended and completed the text of the doctoral dissertation for the PhD in Health Sciences degree, during this Fall Semester, 2019.

DISSERTATION COMMITTEE
(please sign and date beside your name)

Chair:
Dr. Genevieve Pinto Zipp Genevieve Pinto Zipp PT, EdD 11/1/19
date

Committee Member:
Dr. Fortunato Battaglia Fortunato Battaglia 11/12/19
date

Committee Member:
Dr. Terrence F. Cahill Terrence F. Cahill 11/1/19
date

ACKNOWLEDGEMENTS

To quote Albert Pine “What we do for ourselves dies with us; what we do for others becomes immortal and lives on”. A powerful quote and one that seems to resonant with me at this particular moment of my life. For me this doctoral journey is truly about trying to find answers to questions that hopefully in some way will benefit society. I am blessed to have had a wonderful mentor and role model, Dr. Pinto-Zipp who first was my Master Degree advisor and continued on with me as my PhD committee chair. I would like to also thank Dr. Cahill and Dr. Battaglia for their support and guidance as my committee members.

This research was inspired by patient experiences that required more exploring and investigating. I am forever thankful to the patients for their willingness to explore their post-concussion symptoms with me. I hope this research will enhance our understanding of post-concussion symptomology especially in the area of human sensory processing. Finally, I wish to thank my team of co-investigators at NYULMC for their dedication and passion for this research project.

DEDICATION

This dissertation is dedicated to my wife, Amy Lyn Van Lew. She has truly been my rock during this journey. When I doubted myself her words inspired me, when I was tired her words lifted me, when I fell, she lifted me back to my feet. Without her this journey would have never been possible, and I will be forever grateful for her unconditional love and support.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ABSTRACT

Background: Approximately 10-20% of individuals who sustain a concussion will experience post-concussion syndrome, defined as symptomology beyond the traditional 7-10-day concussion recovery period. A particular area of symptomology, sensory processing deficits was explored in this study to ascertain the prevalence of these deficits along with possible associations with QOL and everyday functional outcomes. **Methods**: This study was a prospective, cross-sectional, quantitative study using a consecutive and convenience sampling model. The research participants were recruited from an outpatient occupational therapy program at an academic medical center. Normative data from two different sensory processing outcome tools were used in the analysis. **Results:** The total sample size was *N*=28 which included (*n*=9) males and (*n*=19) females. Research participants exhibited statistically mean differences in all four quadrants of the ASP ($p<0.000$) and half of the subgroups for the ASPS when comparing these sensory processing outcome tools to the normative data associated with each outcome tool. There were some levels of moderate to strong correlations between the results from the sensory processing outcome tools and the QOL and everyday function outcome tools. For the ASP outcome tool there was a moderate (*r*=.-356, *p*=0.810), negative correlation between the sensory sensitivity ASP quadrant and the SLWS and a strong positive correlation between this ASP quadrant and the FSE (*r*=.690, p<0.000). ASPS results indicate a range of correlation coefficients from (*r*=.280-.628) for the five ASPS subgroups and the FSE outcome measure. 80% (*n*=4) of these subgroups had a moderate to strong positive coefficient. **Conclusion:** Individuals experiencing post-concussion syndrome may be experiencing greater amounts of

sensory processing dysfunction as compared to normative population data. The results also suggest that there may be some correlation between the presence of sensory processing dysfunction and QOL and everyday function.

Key Words

Concussion

Sensory Processing

Mild Traumatic Brain Injury (MTBI)

Quality of Life (QOL)

Chapter I

INTRODUCTION

Based upon estimates from the Center for Disease Control, approximately 1.6 to 3.8 million sports related concussions (SRCs) occur among athletes annually (Daneshaver, Nowinski, McKee, & Cantu, 2011). This number appears to be an underestimation due to the underreporting of sport concussions (Halstead & Walter, 2010). The total number of individuals sustaining a concussion in the United States annually is closer to over 5 million when non-sports related concussions are added to this total (McCrory et al., 2013). Recently, concussions have been classified as the mildest subset of mild traumatic brain injury (MTBI) and have been defined as a closed head injury due to either a direct blow to or a shaking of the head by an impulsive force, resulting in transient alteration in mental status and brain function (Echemendia, 2006; McCory et al., 2013; Lincoln at al., 2011).

MTBI has been called a silent epidemic because `many of the acute and enduring alterations in cognitive, motor and somatosensory functions may not be readily apparent to external observers (Laskowski, Creed, & Raghupahti, 2015). MTBI has been defined as a closed acute brain injury resulting from mechanical energy to the head from forces with the following; confusion or disorientation and/or loss of consciousness for 30 minutes of less and/or post traumatic amnesia for less than 24 hours and other transient neurological abnormities, such as focal signs, seizures and intracranial lesions not

requiring surgery, Glasgow Coma Scale of 13-15 about 30 minutes after injury, and exclusion of other physical or mental causes (Holm, Cassidy, Carroll, & Borg, 2005). MTBI results from a complex pathophysiological process affecting the brain from an impulsive force transmitted to the head, face, neck or other region of the body (McCrory et al., 2013). During MTBI, linear and rotational accelerations of the brain occur relative to the skull, producing pressure and shear forces throughout the delicate brain tissue (Meaney & Smith, 2011). Despite the label of mild, MTBI injuries directly and negatively impact neurological functioning for individuals and have been found to be associated with cognitive, physical, psychological, and social dysfunction resulting in significant disability (Bay & Mclean, 2007). Unfortunately, as the literature suggests despite an array of deficits in function, individuals with MTBI usually present with an absence of structural lesions using conventional neuro-imaging techniques which continues to create difficulty among health care providers in identifying a proper diagnosis and thus proper clinical management (Holli et al., 2010).

Concussions are the most frequently classified types of MTBI and have been receiving increased attention both from the medical community and the media due to the increase in reporting of SRCs. Although categorized as the mildest form of MTBI, recent studies have demonstrated long-term alterations following concussions may be more severe than previously thought (Chamard, Lefebve, Lassonde, & Theoret, 2016). In regard to gender, females have nearly twice the rate of reported concussions compared to males with some variations in self-reporting symptoms (Dick, 2009; Lincoln el al., 2011). This increase in concussion awareness has resulted in increased reporting of concussions throughout our health care system and thus increased health care

interventions. Individuals sustaining a concussion may have a wide spectrum of clinical signs and symptoms including psychological distress, cognitive impairment, somatic symptoms, physical impairments, nausea, and other neurological symptoms (Mustafi et al., 2018). According to the literature loss of consciousness occurs in about 10% of concussions (Meehan, Hemecourt, & Comstock, 2010; Mansell et al., 2010).

Of particular concern to health care providers and individuals who have sustained a concussion is a condition called post-concussion syndrome (PCS). Individuals diagnosed with this condition often experience persistent post-concussion symptoms. These symptoms continue beyond the traditional 7-10-day concussion recovery period (Marar, Mclvian, & Fields, 2012; Makdissi et al., 2010). Symptoms usually encompass somatic, physical, cognitive and affective complaints, as well as patients commonly reporting headache, dizziness, difficulty concentrating, depression and anxiety (Wojcik, 2014). There appears to be both psychological and structural components to post-concussion syndrome, as patients with a history of migraines, depression or anxiety are more likely to experience post-concussive disorder after sustaining a concussion (Cantu & Register-Mihalik, 2011). While the majority of concussions resolve within 7-10 days, in some instance's symptoms can persist for weeks, months or years beyond the initial injury (Harmon et al., 2015). It is estimated that approximately 10%-20% of concussions do not resolved in this 7-10-day period of time and thus result in post-concussion syndrome (Chong & Schwedt, 2015). In the literature, specifically when a concussion injury transforms into a post-concussion syndrome is ill-defined and poorly understood. There are no exact guidelines except that the classification of post-concussion syndrome involves symptoms and signs that last for weeks to months after the incident

(Jotwani & Harmon, 2010; McCrory, 2009). This lack of diagnostic clarity and consensus for PCS has resulted in confusion among patients and health care providers in regard to proper identification and clinical management of these individuals.

In summary the prevalence of MTBI continues to have a significant impact among the overall population with the Centers for Disease Control estimating that 2% of our population (not including our wounded warriors) currently live with MTBI sequela and disabilities, at a huge cost to the health care system during the acute care phase and more so from long term medical and rehabilitative care (Griffin, 2011). Overall, the fiscal impact that MTBI has on society is staggering, with direct and indirect costs associated with MTBI exceeding 26.4 billion annually (Coronado et al., 2011). A significant portion of this amount is associated with reportable concussions while the actual amounts associated with concussions are probably much higher due to initial underreporting of this injury (McCrory et al., 2013). Concussion, the most common type of MTBI is recognized as a worldwide problem affecting millions of athletes and non-athletes a year (Langlois, Rutland-Brown, & Wald, 2006).

Problem/Purpose of Study

The significant short- and long-term impact that concussions have on individuals and society and the recent increase in societal attention and clinical reporting of such occurrences indicates a need to increase scientific inquiry into this condition. The American Medical Society supports such scientific inquiry in their position paper, with Harmon et al. (2013) suggesting additional research is needed to validate current assessment tools, delineate the role of neuropsychology testing and improve the

identification of those at risk for prolonged post-concussive symptoms or other long-term complications. In addition, given the collection of anecdotal evidence by this investigator, that individuals who sustain concussions may experience additional unique deficits in sensory processing, further investigation into this clinical condition seems warranted. Such sensory processing impairments can affect all aspects of an individual's function in activities of daily living (Figure 1). According to self-reported unpublished data these individuals also sometimes lack the awareness and knowledge that they may be experiencing sensory processing dysfunction and thus can suffer for months and even years before a clinician identifies these deficits. As far as this investigator is aware the clinical identification of global sensory processing deficits resulting from neurological damage to the brain in the adult concussion population has not been clinically substantiated in the scientific community. It is believed that a scientific approach to exploring sensory processing in the concussion population could result in important clinical information that has the potential to create a better understanding and improved identification of this phenomena, along with creating new clinical rehabilitation protocols for the clinical management and rehabilitation of individuals experiencing sensory processing deficits following a concussion.

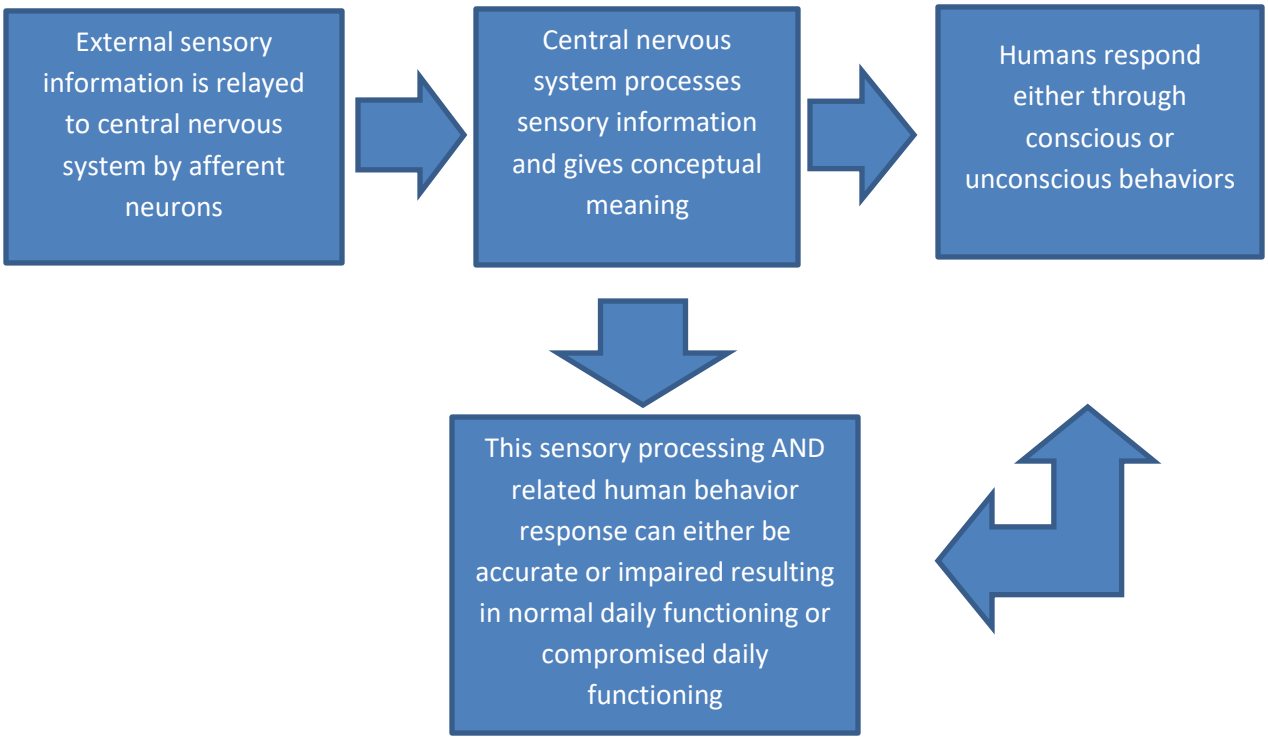

Figure 1. Human sensory processing

Primary Research Question and Hypothesis

RQ1: Do individuals with a diagnosis of post-concussion experience greater amounts of sensory processing dysfunction as compared to the normal population?

Primary Ha1: Individuals with a diagnosis of post-concussion will experience greater sensory processing dysfunction as compared to the normal population.

Secondary Research Question and Hypothesis

RQ2: Is there a relationship between scores on the sensory processing assessments and the scores on the assessments designed to measure quality of life and everyday function in a post-concussion population?

Secondary Ha2: There is a relationship between scores on the sensory processing assessments and the scores on the assessments designed to measure quality of life and everyday function in a post-concussion population

Conceptual Theoretical Framework

The sensory structures of the human brain including both subcortical and cortical areas are quite susceptible to damage following concussions. Such damage can occur in both white and grey matter. Compelling evidence suggests that such damage could be the etiology of sensory system dysfunction in individuals who have sustained concussions. (Alwis, Johnstone, Yan, & Rajan, 2013). Specifically, such sensory system dysfunction can include deficits in sensory processing, which as previously outlined, can impact all areas of function for individuals who have sustained a concussion (Figure 2). The ability to rapidly perceive, integrate, organize and respond to incoming sensory information is critical to adaptive functioning and successful performance in all aspect of activities of daily living. As Johnstone et al. (2015) highlights significant amounts of research investigating the underpinnings of various deficits that occur after concussions have focused on cognitive and emotional disturbances, whereas the sensory system has been largely ignored. These authors suggest that given the extent to which we rely on sensory information to conduct motor, cognitive and social activities, the effects of concussion on sensory systems should be explored and considered when interpreting post-concussion deficits.

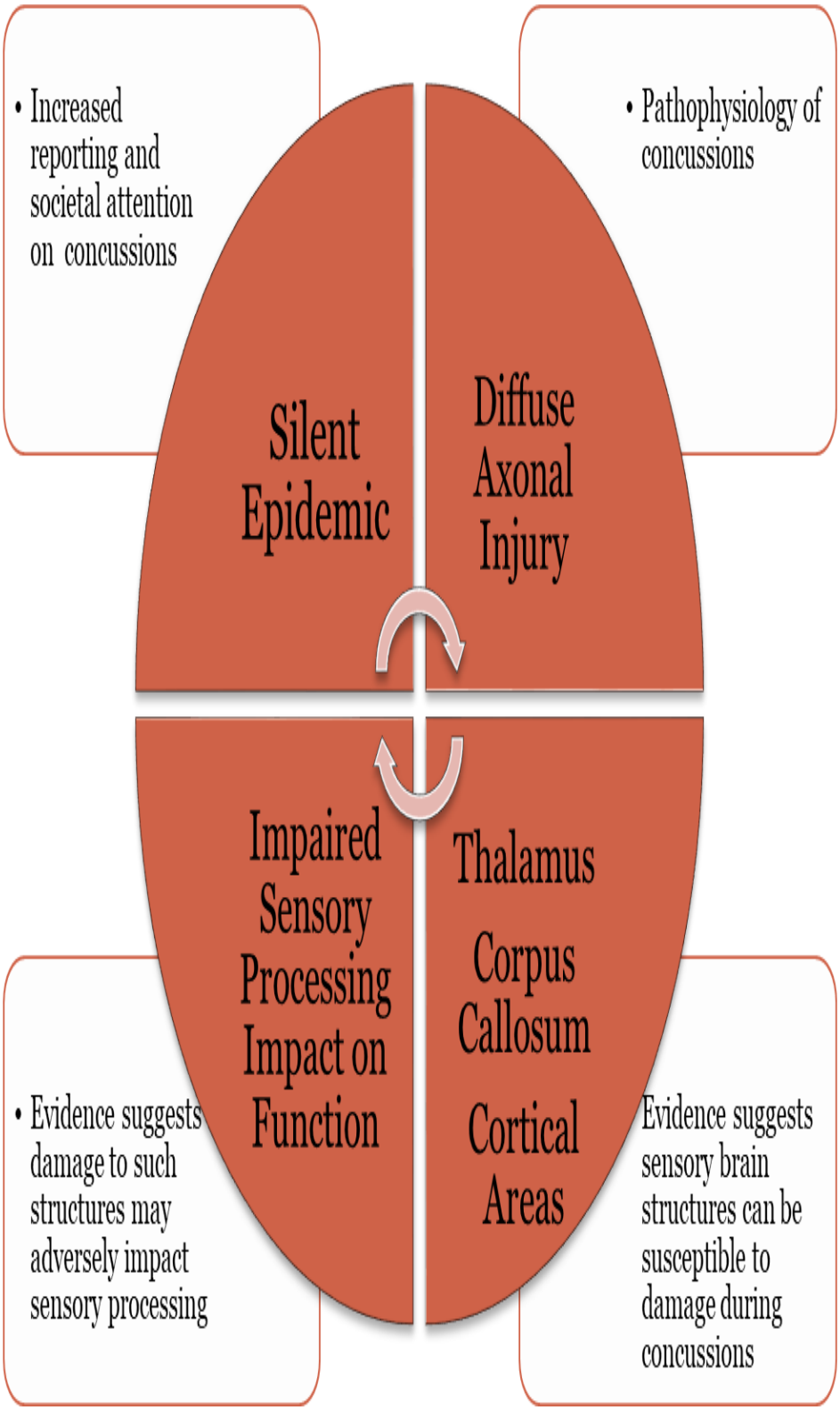

Figure 2. Conceptual Theoretical Framework

Chapter II

REVIEW OF THE LITERATURE

Pathophysiology of Concussion

The human brain is comprised of over 100 billion neurons functioning in a complex hierarchical and stratified manner (Bear, Conners, & Paradiso, 2007). There are two types of tissue located in the brain, grey and white matter. Grey matter, which has a pinkish grey color in the living brain contains cell bodies, dendrites and axon terminals of neurons, glial cells and capillaries. This is where all the synaptic activity occurs between neurons. White matter is made of axons connecting different parts of the grey matter to each other. Myelin, which acts as an insulator, increases the speed of transmission of nerve cells and gives the white matter its white color. Generally, grey matter is the tissue on the outer surface of the cerebral hemispheres along with some located inside the hemispheres. White matter is generally located inside the hemispheres.

It has been suggested that the pathophysiology of concussion can be categorized as a neurometabolic cascade including complex ionic, metabolic and pathophysiology events which is accompanied by microscope axonal damages (Baarkhoudarian, Hovada, & Giza, 2016). Primary diffuse axonal injury (DAI) is thought to be triggered by the inertial forces of trauma to the brain, resulting in subsequent structural and

subcellular changes within the axon cylinder (Buki & Povlishock, 2006; Creed, DiLeonardi, Fox, Tessler, & Raghupathi, 2011). This microstructural damage to the axon is believed to create disruption of both anterograde and retrograde transport resulting in eventual edema in contiguous axons with secondary axotomy (Creed et al., 2011). Such axotomy and eventual lysis and breakdown of damaged axons following trauma may result in accumulating plaque thus interfering with synaptic functioning (Messe et al., 2010). This classical understanding of DAI as morphologically altered axons due to impaired transport may represent just one pathological subset of damaged axons. Morphologically intact axons with disrupted physiology may also contribute to the pathological milieu leading to clinical dysfunction across a wide range of injury severities including concussions (Johnson, Stewart, & Smith, 2013). A multitude of diverse secondary chemical and ionic cascades detrimental to axons have been investigated following concussion. For example, ionic imbalance after axonal trauma is thought to play a central role in post-injury in both axonal degeneration and the persistent dysfunction of otherwise intact axons (Johnson et al., 2013). In addition to such axonal white matter damage, grey matter damage has been observed in neuro-imaging studies supporting the premise that both morphological and structural connectivity analysis can yield complimentary data, with the former usually evaluating changes in grey matter density and thickness and the latter giving a measure of white matter integrity (Dean, Sato, Vieria, McNamara, & Sterr, 2015).

Concussion can have both short- and long-term clinical sequela that can adversely affect all components of daily functioning for individuals who sustain such injuries. The study of concussion using traditional neuroimaging techniques including computer

topography (CT) and magnetic resonance imaging (MRI) have produced very limited amounts of information specifying the actual brain damage following concussion (Shenton et al., 2012). Recently, the use of new neuroimaging techniques including diffusion tensor imaging (DTI) and BOLD resting-state functional MRI (rs-fMRI) has shown promise in providing evidence of structural damage to the brain by measuring the movement of water within the brain and levels of blood oxygenation.

Two commonly reported DTI metrics are fractional anisotropy (FA) and mean diffusivity (MD). FA is a measure of the relative directionality of water diffusion in the brain, which is organized and specific in healthy individuals (Harmon et al., 2013). FA is a scalar measure that ranges from 0 to 1 with 0 being completely isotropic, meaning the water diffuses equally in all directions and 1 depicting the most extreme anisotropic scenario in which water molecules are diffusing along a single axis (Shenton et al., 2012). MD, the second most common measure, is different from FA in that it measures the average distance a water molecule traverses within a given observation time (Delouche et al., 2016). MD measures the overall non-directional mobility of water within the brain. The quantification of pathology using DTI is based on measures that calculate the amount or restriction of water movement in the brain, which is determined to a large extent by the tissue being measured. FA and MD are frequently observed to be inversely related (Niogi & Mukherjee, 2010). In regards to the relationship between FA and healthy white and grey matter, the FA value in grey matter is usually close to 0 secondary to the direction of water being isotropic whereas in white matter the water is free along the axons but restricted perpendicular to the axons, and therefore more anisotropic with FA being closer to 1 (Shenton et al., 2012).

Rs-fMRI uses paradigm specific brain activation patterns that are inferred by subtracting task-based brain activation from baseline brain activity (Greicius et al., 2002). This creates a measurement of functional connectivity with the brain that can be objectively analyzed. Unlike DTI rs-fMRI can image grey matter along with some aspects of white matter. Majority of the concussion literature using this technique has focused on specific neuronal tracks called the default-mode-network (DMN) (Zhou et al., 2012). This network includes the medial prefrontal, medial temporal, posterior cingulate and parietal area of the cerebral cortex.

Most studies using DTI neuroimaging supports the findings that both FA and MD have been shown to change after concussion suggesting axonal white matter damage (Chong & Schwedt, 2015). DTI, however, is somewhat non-specific and it is not clear whether disruptions in FA and MA are the result of damage to axonal membranes, myelin sheath, microtubules, neurofilaments or other white matter factors (Shenton et al., 2012). Specific studies by (Lancaster et al.,2016; Tremblay et al., 2014; Mustafi et al., 2017) have all shown changes in fractional anisotropy following concussion though how these changes correlate with clinical measures of injury and recovery have yet to be universally substantiated. Research supports these findings in both acute studies and long-term studies. Lange et al. (2015), in a recent study, examined a subset of patients experiencing post-concussion symptoms approximately 6-8 weeks following a concussion. The researchers used a control group with no history of concussion and two concussion groups based upon ICD-10 criteria for PCS and PCS absent. For the specific DTI outcomes there were no significant differences in FA and MD when comparing the PSC-present and the PSC absent groups. However, there were

significant differences (p= 0.005) in MD when comparing the PCS-present and control groups thus suggesting a correlation between the presence of post-concussion symptoms and severity of brain damage. In another acute longitudinal study by Meier et al. (2016) the investigators found increases in FA during DTI for a group of concussed collegiate athletes at one day, one week and one-month post-concussion relative to a demographically matched sample of healthy collegiate athletes.

Although there appears to be some level of consensus among researchers that microscopic whiter matter damage is prevalent and may be the cause of the functional deficits experienced by individuals with a concussion, one exception to this is in the results from a study by Ilvesmaki et al. (2014). This study demonstrated no significant differences in white matter changes using DTI when comparing individuals with MTBI and control individuals. These researchers suggested that although many studies have reported white matter changes in individuals with history of MTBI, conclusions from these studies must be viewed with caution secondary to significant methodological differences and difficulty in controlling confounding variables such as temporal variables, sample sizes, patient characteristics including pre-injury heath factors and differing DTI analysis techniques. In this study by Ilvesmaki et al. (2014) the researcher's goal was to reduce or eliminate as many confounding variables as possible through stringent inclusion and exclusion criteria. The researchers performed subgroup analysis based on injury severity characteristics for all 75 individuals with MTBI comparing them to 40 control subjects. This analysis was performed for three different severity measures; loss of consciousness for greater than 5 minutes, post-traumatic

amnesia greater than 3 hours, complicated MTBI with trauma-related structural abnormality on traditional neuroimaging.

As with the outline DTI studies, many rs-fMRI studies examining the brain in the concussion population indicates abnormal functional connectivity in certain areas of the brain specifically the DMN. For example several studies indicate a pattern of abnormal frontal hyper-connectivity and posterior hypo-connectivity in concussed patients (Borich, Babul, & Huang et al., 2014; Johnson, Zhang, & Gay et al., 2012) A study by Borich et al. (2012) found alterations with the DMN including abnormalities in connectivity with the frontoparietal areas of the cortex in a group of adolescent athletes that were imaged less than 2 months post-concussion. These authors suggest that such alterations is likely indicates pathophysiological disturbances in response to the concussion injury. Similar to DTI concussion studies, researchers using rs-fMRI have noted that there is an inherent variability in concussion neurological pathophysiology and that temporal variable of concussion-to-imagining greatly influences certain validity of concussion neuroimaging studies particularly the generalization of certain findings (Narayana, 2017; Chong & Schwedt, 2015).

In a recent review by Chong and Schwedt (2015) of the concussion literature on DTI and rs-fMRI studies it was revealed that the majority of the recent concussion and DTI studies reported some level of DTI abnormalities although their anatomical location did not always converge. The authors of this review also suggest that the lack of convergence is not surprising given the heterogeneity of brain injuries as well as the variability in these studies between time of injury and DTI scan. These authors highlight the importance of the “concussion-to-imaging- interval” as an important variable to keep

in mind when comparing neuroimaging and concussion studies. These authors conclude that with the use of more modern research approaches including the use of tract-based spatial statistics (TBSS) more recent studies have shown some consistency demonstrating increased RD and MD in the concussion population indicating consistent white matter damage.

Functional Implications of Neuro-Anatomical Structural & Functional Connectivity Changes Following MTBI

The significance of both white and grey matter structural changes in concussion is substantial. Traditionally, as previously outlined, white matter damage particularly DAI is thought to be the primary mechanism of injury in concussion whereas grey matter damage is more associated with a more severe TBI (Dean et al., 2015). This does not suggest that individuals sustaining a concussion do not experience both white and grey matter damage. Reduced prefrontal grey matter concentration has been observed in the acute stage of concussion in previous studies using rs-fMRI (Churchill et al., 2017) One recent long term study by Dean et al. (2015) found both grey and white matter alterations when comparing individuals with concussion to a control group with no history of head injury post greater than one year. Specifically, subtle damage to the left frontal and right mid frontal gyrus was observed along with grey matter changes in the precuneus, medial temporal and inferior parietal lobe. Of particular interest was an association between PCS symptom reporting and reduced grey matter which has rarely

been reported in the literature in contrast to the frequency of white matter damage and PCS reporting. This finding supports other research by Ling, Klimaj, Toulouse, & Mayer (2013) that found changes in the superior frontal cortex was associated with cognitive and emotional complaints by individuals with PCS.

It is suggested that such alterations in brain tissue results in the diverse neurological and psychological symptoms that post-concussion individuals experience (Smits et al., 2011). The categorization of functional deficits following concussion include; cognitive, emotional, physical, psychological, visual, vestibular, and sensory. Of particular interest to this investigator is the involvement of the sensory processing system and how concussion affects this system. Not all individuals who sustain such injury will experience any or all of these deficits. While, it would be impossible to summarize the extensive amount of research on concussion and the resulting deficits particularly in the area of neuropsychology in this document the following examples are provided to highlight some major neuro-anatomical structures and their proposed involvement in concussion and seeks to bridge the evidence supporting the theoretical framework of this proposed study.

One prominent area of the brain that is consistently highlighted in the concussion literature as being susceptible to damage is the corpus callosum (CC). Studies by (Warner et al., 2010; Messe et al., 2012; Rutgers et al., 2008; Smits et al., 2011; Singh, Jeong, Hwang, Sungkrant, & Gruen, 2010) all demonstrated FA and MD alterations in the CC using DTI in the concussion population. The CC is a structure that is allows the right and left hemispheres to communicate with one another. It is mainly comprised of white matter and the structure arches around the anterior horn of the lateral ventricles. It

is the largest fiber bundle in the human brain with over 300 million fibers (Hofer & Frahm, 2006). In a study by Hofer and Frahm (2006) using DTI technology, five segments of the CC were identified. These five segments contain fibers that project into the prefrontal, premotor and supplementary motor, primary motor, and primary sensory areas as well as into parietal, temporal and occipital cortical areas. Of particular interest is the role that the CC plays in relaying sensory information to the primary sensory and parietal cortical areas, and how this might be compromised in individuals who have sustained a concussion and the resulting impact on the brain's ability to accurately process sensory information.

Another prominent area of the brain that has been highlighted in the literature as being susceptible to damage is the thalamus. The thalamus is a major relay and processing center for all types of sensory and motor information. There are two thalamic lobes, one in each hemisphere containing 26 pair of nuclei (Hofer & Frahm, 2006). The thalamus is a complex deep grey matter mass consisting of many groups of nuclei and white matter bundles and is vulnerable to damage during sudden acceleration or deceleration movements during head trauma.

In a study by Lange et al. (2015) using DTI technology significant increases of MD were observed in the bilateral posterior thalamic radiations when comparing a concussion group with PCS to a control group without PCS. In addition, two studies by (Tang et al., 2011; Sours et al., 2015) examined the resting state networks of the thalamus in acute concussion patients. In the study by Tang et al. (2011) participants with a concussion where compared to a control group using rs-fMR. The concussion group demonstrated more abnormally distributed functional connectivity between

thalamic and cortical regions during resting state despite the absence of any lesions in the thalamic regions using conventional imaging. These researchers found a significant negative correlation between atypical thalamic resting state activity and neurocognitive testing adding to the argument that subtle changes in neurological physiology following concussion results in functional deficits for individuals. A study by Sours et al. (2015) examined resting state networks as well and found similar results. Specifically, comparing a group of individuals post-concussion with a group on non-concussed individuals these researchers found increased functional connectivity between thalamus and cortical regions associated with primary sensory processing and increased functional connectivity between the thalamus and the DMN.

Traditionally, the thalamus has been thought of mainly a relay station for virtually all sensory information about the external environment via the thalamocortical tract to the neocortex. (Tyll, Budinger, & Noesselt, 2011). This one-way communication now has been recently challenged and has been confirmed to be only part of the overall function of the thalamus. Several studies reveal a much more complex picture of thalamic function (Briggs & Usrey, 2008). Notably, thalamic neurons receive input from the cortex via the corticothalamic pathway thereby allowing the cortex to communicate continuously with the thalamus. Cortical-thalamic axons provide the predominant synaptic input to the sensory thalamus accounting for 30%-44% of all synapses that the thalamocortical cells receive, supporting this complex communication between the thalamus and cortex (Tyll, Budinger, & Noesselt, 2011). In a study by Crandell, Cruikshank, and Conners (2015) examining the somatosensory system of mice, this complex mechanism of cortical influence on the thalamus was found to be very dynamic

with both exhibitory and inhibitory components. As Cappe, Roullier, and Barone (2009) further highlight the evidence supports the presents of such highly specialized corticothalamic pathways providing feed forward projections from one cortical area to other cortical regions via the thalamus.

This supports the concept of thalamic involvement in not only relaying sensory information but in processing sensory stimuli which is vital for human functioning. Clearly, as the literature suggests the complexity of thalamic functioning yields a strong argument that certain amounts of sensory processing occurs not only in the sensory specific areas of the cortex but occurs at subcortical levels of the brain including the thalamus. As Tyll, Budinger, and Noesselt (2011) suggests several animal studies performed in various species identified thalamic nuclei based on their anatomical connections to structures of different sensory modalities, and based upon the multisensory response characteristics of these neurons suggest integration of multisensory information before the information has reached the neocortical areas. Cappe, Roullier, and Barone (2009) further highlight such cortical-thalamo-cortical routing is suitable for multisensory interplay and integration by a convergence of different modalities on the same thalamic nucleus, resulting in integrated sensory information being available to a target cortical region of the brain.

In conclusion, along with the CC and thalamic functional abnormalities following a concussion, somatosensory areas of the cortex are also susceptible to damage from a concussion although not as clearly delineated as related white matter structures (Dean, 2015). The sensory cortex is laminated in a very highly organized fashion with columns of cells in the grey matter stretching from the cortical surface to the white matter and

spanning cells across the laminae (Alwis et al., 2013). These areas of the brain are also significantly involved in sensory processing and can include the premotor cortex and much of the parietal lobe. For example, the premotor cortex is an area of multisensory integration secondary to being a zone of convergence of visual, auditory and somatosensory inputs (Graziano, 2001). In summary and consistent with the proposed theoretical framework of this proposed study, somatosensory areas of the cerebral cortex play a crucial role in sensory processing along with other proposed subcortical areas of the brain both being adversely affected by concussion.

Sensory Processing

Most species, including humans are equipped with highly specialized sensory systems that give them access to numerous types of information from their surrounding environment (Dione-Dostie, Paquette, Lassonde, & Gallagher, 2015). The human body possess eight sensory systems; visual, auditory, tactile, olfactory, gustatory, vestibular, proprioception and interoception. Humans experience two types of sensory experiences; unimodal and multi-sensory experiences. Unimodal experiences refer to sensory experiences derived solely from one sensory system whereas multi-sensory experiences include sensory information from two or more sensory systems. Most of the sensory experiences that humans experience are multi-sensory derived from a combination of information acquired through several sensory modalities often simultaneously (Su, Parham, 2014).

As previously outlined the human brain contains both sensory specific cortices and heteromodal areas where sensory interplay and functional integration occurs (Macaluso & Driver, 2005). This interplay is crucial for higher level sensory processing. For example, numerous functional MRI and EEG/MEG studies have shown that multi-sensory interplay can affect not only established multi-sensory convergence zones, but also brain areas and responses traditionally considered sensory specific (Driver & Noesselt, 2008). In fact some neuroscientists suggest that the entire neocortex is essentially multi-sensory (Ghazanfar & Schroeder, 2006).

Sensory processing refers to the way the nervous system receives messages from the senses and turns them into responses (Miller, Nielsen, Schoen, & Brett-Green, 2009). As an individual engages in life experiences the nervous system evolves and these experiences help shape the nervous system. As part of this process, a balance of habituation and sensitization must be developed to support appropriate adaptive behaviors to everyday environmental demands and sensory stimulus (Brown & Dunn, 2002). Considering that much of this processing occurs with multi-sensory information, the ability to process and integrate sensory information is a fundamental element of our successful engagement in our everyday environment allowing us identify stimulus and apply optimal behavioral responses. Clinically, the term sensory integration was first used by Ayres (1972) to identify a field of study related to individuals especially children with atypical responses to sensory stimulation. In neurosciences, sensory integration is used to specifically describe the combining of signals from two or more senses in the central nervous system (Calvert, Spence, & Stein, 2004). Ayres (1972) defined integration as the “interaction and coordination of two or more functions or processes in

a manner which enhances the adaptiveness of brain response" (pp. 25-26). She further defined the integrative process as filtering, organizing and integrating sensory information and did not specifically limit it to the combination of two or more senses. Thus her definition of sensory integration seem to give a much broader representation of sensory processing than just the integration of several senses, the definition often used by neuroscientists (Davies & Gavin, 2007). It has been suggested by Davies and Gavin (2007) that when conducting research on individuals who have difficulties with processing sensory information using the term sensory processing disorder (SPD) better captures all aspects of the deficits these individuals are experiencing.

(SPD) is a heterogeneous condition which includes 3 subcategories including sensory modulation disorder, sensory-based motor disorder and sensory discrimination disorder. Although a consensus on these subcategories is not unified in the scientific community these subcategories can be further divided into six subtypes (Miller, Anzalone, Lane, Cermak, & Osten, 2007). Individuals with the disorder have impaired responses to, processing of, and/or organization of sensory information that effects participation in functional daily life routines and activities (Miller, Nielsen, Schoen, & Brett-Green, 2009). Sensory modulation disorder (SMD), the most frequently cited of the three sub categories of SPD in the therapy literature is defined as difficulty in regulating and organizing the degree, intensity and nature of responses to sensory input in a graded and adaptive manner, allowing the individual to achieve and maintain optimal range of functional performance and to adapt to challenges in daily life (Miller & Lane, 2000).

Sensory modulation involves both a neurophysiological and behavioral component. Neurophysiologically, modulation involves balancing excitatory and inhibitory sensory inputs within the central nervous system (CNS) (Brown & Dunn, 2002). Modulation of input is accomplished through changes in synaptic transmission at the cellular level (Lane, Lynn &, Reynolds, 2010). Behaviorally, modulation involves an individual's ability to regulate and organize responses reaction to sensory input in an adaptive way (Miller & Lane, 2000). Successful sensory modulation in the CNS involves a type of homeostasis, whereas the ability of the CNS to regulate the excitatory and inhibitory sensory signals from an individual's internal and external environment. For example, the one of the functions of the thalamus is to filter sensory input and ensures that the other areas of the brain are not overwhelmed by sensations, and that the most pertinent sensory information is directed to the intended centers of the brain for further processing and integration. Interestingly, and related to theoretical framework of this proposed research, is the identification of the thalamus as a key area of the brain responsible for sensory modulation, particularly in the suppression of some sensations (Breedlove, Rosenzwig, & Watson, 2007). Damage to the thalamus could produce challenges for concussed individuals including, becoming overwhelmed by sensory stimuli or demonstrating difficulty in prioritizing which sensations, he or she should direct attention to.

The clinical presentation of SMD varies with considerable heterogeneity in symptomatology with three different subtypes' sensory over-responsivity, sensory under-responsivity and sensory seeking (James, Miller, Schaaf, Nielsen, & Schoen, 2011). Miller et al. (2007) define these subtypes as follows: Sensory over-responsive

refers to responding too much, for too long or to stimuli of weak intensity; Sensory under-responsive refers to responding too little, or needs extremely strong stimulation to become aware of the stimulus; Sensory seeking refers to responding with intense searching for more or stronger stimulus. An example of this can be seen clinically with an individual experiencing sensory over-responsivity. They may overreact to sensory stimuli, appear to have higher level of anxiety or arousal, or avoid certain sensory stimuli or environments altogether (Miller et al., 2007).

The majority of SPD scientific investigation and literature has focused on SPD and children. It is believed that SPD affects 5-16% of children within the general population (Ben-Sasson, Carter, & Briggs-Gowan, 2009). Research has demonstrated co-morbidity with attention deficit hyperactivity disorder (ADHD), autism and other psychopathology but also SPD can exist in isolation. (Van Hulle, Schmidt, & Goldsmith, 2012). In regards to the brain both white and grey matter studies have been performed. Historically, the underlying basis for SPD implicates both primary sensory cortical areas and higher level cortical regions sub serving multi-modal sensory integration and processing (Owen et al., 2013). Specifically, the posterior parietal cortex and superior temporal sulcus are involved in auditory-tactile integration, whereas dorsolateral prefrontal cortex helps mediate attentional control (Brett-Green et al., 2008; Chait et al., 2010). Traditionally, these areas of mostly grey matter have been examined in regard to their role in the sensory system and overall their involvement in sensory integration. However, recent research has started to focus on white matter abnormalities and sensory processing.

In one study by Owen et al. (2013) the researchers used DTI to examine certain areas of the brain containing white matter including the corpus callosum and thalamus.

Similar to other DTI studies previously highlighted the researchers used FA and MD as some of the variables associated with examining white mater microstructural integrity. In summary these researchers compared a small group of children with SPD and a small group of normally developing children (TDC). There results indicated significant differences (p= 0.02) after corrections for multiple comparisons in FA and MD between the experimental group and the TDC group in the areas of the splenuim and isthmus of the corpus callosum and the left posterior thalamic radiations, left posterior corona radiate and the left superior longitudinal fasciculus. Of most interest are the white matter abnormalities seen in this study in regard to the thalamus and corpus callosum, considering their previously highlighted roles in the human sensory system and their susceptibleness to damage from a concussion. An additional component of this study also examined correlations of these white matter abnormalities and atypical sensory behavior as extracted by their scores on the parent report Sensory Profile (SP) (Dunn & Westman 1997). The SP for children is a parent report questionnaire which measures behavioral sensory differences, producing scores within individual sensory domains and a total score. Their findings indicated after correction for multiple comparisons significant correlations were found in predominantly posterior white matter areas. Specifically, Owen et al. (2013) noted that in this study DTI microstructural parameters were closely correlated with sensory profile scores in those central white matter pathways known to be involved in primary sensory information transmission, such as the CC sub serving unimodal and multimodal sensory processing.

A second recent study by Chang et al. (2016) suggests similar white matter microstructural abnormalities associated with SPD. In this larger study, the researchers

had a total of 80 subjects divided into two groups, children with SPD and typically developing children. Again using FA and MD as some of the variables, these researchers found strong decreases in white matter microstructural integrity in posterior projections and commissural tracts of the bilateral posterior thalamic radiations and the splenium of the corpus callosum, which contains all the primary sensory projection pathways excluding olfaction and connect homologous sensory cortical regions et al., Additionally, their findings included abnormalities in the retrolenticular area of the right internal capsule, an important structure containing visual and auditory projection fibers. Overall in this study microstructural integrity was noted to be disrupted extensively, but with a posterior bias, throughout the white matter of the subjects identified with SPD (Chang et al., 2016).

Consistent with other studies examining white matter abnormalities and SPD is the results of a study done by Demopoulos et al. (2015) which examined 20 adult subjects with either complete or partial agenesis of the corpus callosum and the presence of atypical sensory processing. The researchers used the Adolescent/Adult Sensory Profile (ASP), which is a 60-item self-report questionnaire which characterizes sensory experiences and behavior and evaluates their impact on functional abilities and daily life (Brown & Dunn, 2002). In summary participants reported measurable differences in multiple aspects of sensory processing using the ASP tool. The largest difference was in the quadrant of low sensory registration, suggesting individuals with agenesis of the corpus callosum may require sensory information to be presented more slowly or at a higher intensity for adequate processing.

In summary the corpus callosum is one of the major white matter areas of the brain that can be damaged during a concussion and evidence indicates its role in the sensory system and specifically sensory processing is well established. (Messe et al., 2010; Messe et al., 2012). Disrupted interhemispheric function likely affects not only the movement of information from one hemisphere to the other but also contralateral inhibition of homologous cortex during sensory processing tasks which are highly lateralized, such as auditory processing leading to speech (Demopoulos et al., 2013). In addition such reduced neuroanatomical capacity for information exchange is thought to have a negative impact on sensory processing and subsequently, the cognitive processes that rely on rapid integration of sensory information. This can affect all aspects of functioning for individuals with such neurologically compromised corpus callosum. Such diffuse damage to the corpus callosum is theorized to be one of the underlying mechanisms for sensory processing dysfunction in the adult concussion population.

Clinically, SPD research can be challenging due to the difficulty in eliminated confounding variables especially possible pre-morbid sensory processing difficulties. For example, in a study with children by Galvin, Froude, and Imms (2009) the researchers used a prospective descriptive design with a convenience sampling of 20 children to investigate atypical sensory processing in a pediatric TBI population. Although strong evidence ($p < .001$) indicated that the percentage of children with TBI who demonstrated sensory processing difficulties was greater than children in the normative sample derived from the Sensory Profile such studies have limitations. First, retrospective measurement of preinjury status is difficult, and parents have been

reported to provide more positive ratings of their child's preinjury behavior after traumatic injuries (Aitken, Mele, & Barrett, 2004). Thus, it is possible that some of the children had atypical sensory processing before the injury. Second, because of convenience sampling and the small number of participants the generalizability of the findings to other children with TBI is limited. Finally, this study had a high percentage of children with a more severe TBI with Glasgow Coma Scale scores ranging from 3-10. For example, 10 of the 20 participants were admitted with the lowest possible score of 3. Thus, these clinical findings would not be unexpected considering the amount of cortical damage that participants experienced with these severe injuries as indicated by their low Glasgow Coma Scale scores.

Similar factors must also be considered when exploring SPD in the adult population. The most important factor is the historical perspective on whether these adults were diagnosed with SPD or experienced difficulties in sensory processing as a child. As May-Benson and Kinnealey (2012) suggest young adults and adults do not "grow out" of their sensory difficulties. An example of this phenomenon was found in a study by (May-Benson & Patane 2010). These researchers used a qualitative approach and found adults seeking occupational therapy sensory integration services for the first time reported remarkably little change in their signs and symptoms of sensory dysfunctions from childhood to adulthood. The effects that these sensory processing challenges have on quality of life and function for these individuals can be substantial. For example, Koomar (2012) used qualitative methods and interviewed adults with SPD and found sensory processing issues negatively affected their quality of life. In addition as May-Benson and Kinnealey (2012) highlights adults with SPD experience a lifetime of living

with adverse sensory functioning which can shape their perceptions of the world and create possible issues with their emotional and mental health. Considering this historical perspective in regards to SPD and the adult population, the methodology of this research study included collecting past medical history pertaining to any previous diagnosis or treatment for SPD or any related sensory processing deficits, although as previously outlined such retrospective analysis of preinjury status can be difficult to accurately ascertain.

Much debate remains regarding the validity of SPD and the lack of uniformity of nomenclature. The use of SPD as a separate diagnosis in children continues to be controversy topic. One study by Davies and Gavin (2007) used EEG technology and found children with SPD demonstrated less sensory gating then children who were typically developing. They found a significant relationship between sensory gating and age for typically developing children but not in children with SPD. In addition, brain activity correctly distinguished children with SPD from children who were typically developing with 86% accuracy. However, SPD was not included in the DSM-5 as a separate diagnostic category; although sensory processing challenges are noted as one of the diagnostic criteria for autism (Critz, Blake, & Nogueira, 2015). The American Academy of Pediatrics (2012) recommends that pediatricians not use sensory processing disorder as a diagnosis secondary to lack of a universally excepted framework for such diagnosis. Secondary to these recommendations the PI for this study did not classify observed sensory processing dysfunction as SPD although patterns of sensory processing deficits were able to be identified and explored scientifically using the proposed research methods.

Chapter III

METHODS

Type of Study

This study used a clinical (non-experimental), prospective, cross-sectional, and quantitative design with descriptive, inferential and correlations components. The descriptive and inferential components examined the phenomena of sensory processing in an adult concussion population. The correlation component examined the relationships between sensory processing, quality of life and everyday function sensory processing. Operationally, the diagnosis of concussion, the independent variable, will be defined in this study by an official diagnosis from a medical physician while the dependent variable of sensory processing will be defined as performance scores on instruments to measure sensory processing. Operationally, the dependent variables of quality of life and everyday function will be defined as performance scores on instruments to measure quality of life and everyday function in the concussion population.

Participants

Participants that were receiving occupational therapy were recruited from the Ambulatory Care Center at NYU Langone Medical Center.

Inclusion Criteria

The following inclusion criteria were used to determine participant selection:

- 18-79 years of age
- Cognitive and language skills sufficient to comprehend multi-step directions
- Cognitively be able to consent to participate in research study
- Medical diagnosis of concussion within 1 year of consenting date (history of multiple concussions will NOT exclude from study)
- Able to speak and read in English

Exclusion Criteria

The following exclusion criteria were used to determine participant selection:

- Past medical history of sensory processing disorder or other related sensory processing deficits
- Past medical history of any neurological condition that would affect the sensory system including but not limited to stroke, spinal cord injury or any peripheral nervous system conditions

Recruitment Procedures

IRB approval for this study was received from both New York University Langone Medical Center (Study # i16-01468) (Appendix A) and Hackensack Meridian Health System (Study # 2018-0260) (Appendix B). Both male and female participants were recruited for the study. Minimal demographic data were collected and was used in the analysis of results. All participants in this study were between the ages of 18-62 years

old. There was no racial or ethnic restriction to this study. Data on racial/ethnic origin was not be collected by the researcher. Patients who were referred to outpatient occupational therapy at the Ambulatory Care Center at NYULMC with a medical diagnosis of concussion was initially evaluated on their first visit by an occupational therapists as standard protocol. These occupational therapists introduced the study to potential subjects at the conclusion of their therapy evaluation. If potential subjects verbally agreed to participate then the PI contacted the potential subjects to confirm initial interest in participation and scheduled a time for consent and screening for the study. If the participant did not meet the inclusion and exclusion criteria then they were considered a screen failure and did not participate in the study.

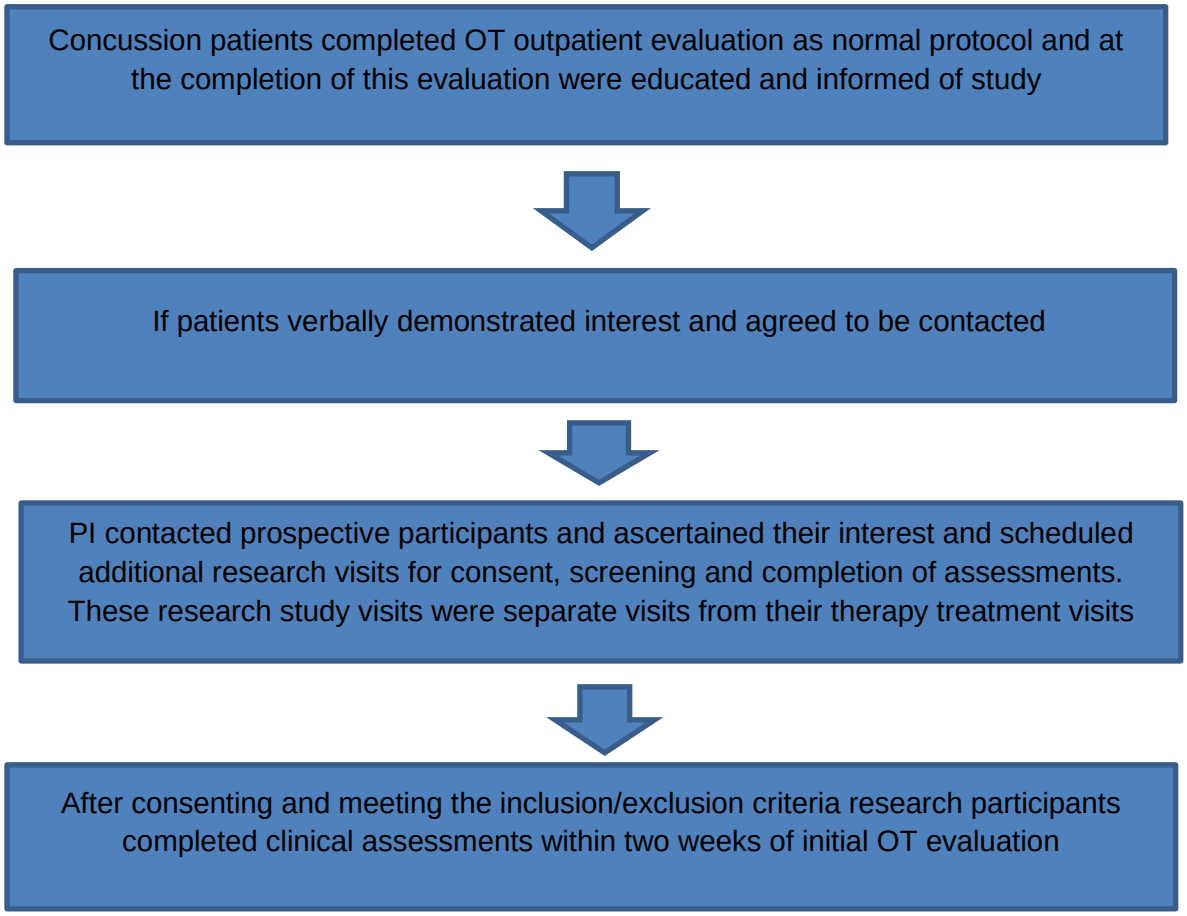

Figure 3. Schematic Design of Research Protocol

Sampling

Sampling for the study was done through a convenience and consecutive model and occurred at NYLMC, a nationally ranked academic medical center. For Hypothesis #1 priori G* power analysis was performed with an effect size of 0.50, and p value of 0.05 (one-tailed test) indicated a sample size of 27 subjects would be needed obtain a power of 0.80.

Outcome Measures

The sensory processing variable was assessed using the Adult Sensory Profile (ASP) and the Adult Sensory Processing Scale (ASPS).The ASP assessment tool was developed by Brown and Dunn (2001) and contains a 60 item self-report scale designed to measure adolescent and adult behaviors and performance in relation to sensory processing. The tool characterizes both the neurological thresholds and behavior responses to sensory information. The items on the ASP are organized and presented as a patient self-reporting assessment tool according to the following sensory processing categories; taste/smell, movement, visual, touch, auditory and activity level. Based upon Dunn's (1997) Model of Sensory Processing the responses are categorized and scored and associated with certain quadrants; low registration, sensation seeking, sensory sensitivity and sensation avoiding. In addition, based upon the scores, a neurological threshold continuum (low and high) and a behavioral/self-regulation continuum (passive or active) is created. The ASP provides cut-off scores for each quadrant and a quadrant profile. The cut-off scores are derived from research data and allows for comparison of an individual's sensory processing skills and responses with

normative population data. A unique characteristic of this tool is the ability to provide cut off scores for comparisons of two age groups, 18-64 and 65 years of age and older.

Several psychometric analyses were performed by Brown, Tollefson, Dunn, and Cromwell (2001) on the ASP including reliability and construct validity. First, item reliability was examined by computing coefficient alpha, an internal consistency estimate for each quadrant with coefficient alpha ranging from (.77 to .82) for the four subscales. This score indicates moderate-strong internal consistency. Second, item reliability was examined using the correlation of each item in a subscale with the total score for each ASP subscale using Pearson product-moment correlations. The results indicated only two items did not have their highest correlation on the intended subscale. Construct validity was examined using physiological skin conductance measures. Amplitude and frequency of a skin conductance response can be used as a measure of attentional response allocation and processing of sensory stimulus (Dawson, Filion, & Schell, 1989). One-way analysis of variance (ANOVA) was used to examine the differences among the four groups of participants with the results indicating significant differences (p=.001) in responsivity across the four groups: sensory sensitivity, sensation avoiding, low registration and sensation seeking suggesting adequate construct validity for these distant subscales.

The second outcome measurement tool that was administered in this study was the Adult Sensory Processing Scale (ASPS). This is a relatively recently developed tool by Blanche, Purham, Chang, & Mallison (2014) designed to measure specific sensory systems and sensory processing. The ASPS is a self-report assessment tool designed to measure behavioral responses that are indicative of sensory processing challenges

in five sensory systems: tactile, proprioceptive, vestibular, auditory and visual (Blanche, Parham, Chang, & Mallinson, 2014). Overall it has been proven to be a valid tool for identifying patterns of sensory responsiveness linked to distinct sensory systems in adult while identifying specific patterns of responsiveness (over responsiveness, under responsiveness and sensory seeking) within sensory systems. During the development of this tool item structure, construct hierarchy and measurement precision was examined using Rasch analysis. Results from this psychometric tool testing reveals good levels person separation reliability (PSR) with a .89 (interpreted similarly to a Cronbach's α) suggesting the tool has good measurement precision (Bond & Fox, 2007). In addition instrument internal consistency was established with a Cronbach's α reliability coefficient for all factors and total factors. Total factor analysis produced α=.87 which is considered strong (Kline, 2000) with α for each tool item comprising the factors ranging from .6 to .8 which is considered acceptable. Overall initial psychometric findings indicate that this tool has adequate internal consistency, strong content validity and acceptable construct validity (Blanche, Parham, Chang, & Mallison, 2014).

The third and fourth outcome tools that used explored quality of life variables. The Quality of Life After Brain Injury (QOLIBRI) and the Satisfaction with Life Scale (SWLS) was administered after the completion of the sensory processing outcomes tools. The QOLIBRI tool was completed by the research participant in a self-report method. The tool provides information about a patient's subjective perception of their well-being and health related quality of life after sustaining a TBI. The QOLIBRI is the first disease-specific scale to assess health related quality of life in the TBI population. It is comprised of 37 items in four satisfaction scales; cognition, self, daily life and autonomy

and social relationships, and two bothered scales, emotions and physical problems. Psychometric testing was performed by von Steinbuchel et al. (2010) on the QOLIBRI scales with it meeting standard psychometric criteria (internal consistency, α = 0.75-0.89, test-retest reliability, α= 0.78-0.85). Test-retest reliability (α= 0.68-0.87) as well as internal consistency (α= 0.81-0.91) were also acceptable in a subgroup of participants with lower cognitive performance. The SWLS was the fourth outcome tool given to the research participants (Diener, Emmons, Larsen, & Griffin, 1985). This tool assessed perceived life satisfaction using a short 5-item questionnaire. The SWLS demonstrated acceptable levels of internal consistency reliability (α=.88) along with good levels of convergent validity and discriminant validity (Kobau, Sniezek, Zack, Lucas, & Burns, 2010). These tools were used to examine possible correlations between performance on the ASP and ASPS and quality of life measurement tools.

The fifth outcome tool used in this study was the Functional Status Examination (FSE). This tool is designed to measure change in activities of everyday life as a function of an event or illness. The tool has a total of 10 domains encompassing physical, social and psychological areas. The tool is administrated by way of self-report and if needed interview. Each domain is scored on a scale of 0-3, with 0 signifying no change from preinjury to a 3 indicating individual is dependent for activities associated with this domain or does not perform activities. The tool has favorable psychometric properties including good test-retest reliability (*r*=0.80) and good levels of construct and criterion validity when compared to the Glasgow Outcome Scale (GOS) and the SF-36 (Dikeman, Machamer, Miller, Doctor, & Temkin, 2001).

Chapter IV

RESULTS AND DISCUSSION

Results

As seen in Figure 4, 127 concussion patients were prescreened by study team members. The prescreening included the following inclusion criteria: age between 18-79 years of age and medical diagnosis of concussion. Out of the 127 patients, 83 of the patients were not referred to the PI see Figure 4. Forty-four of the patients met the prescreening criteria and were referred to the PI for consenting and formal screening. 13 of the 44 did not meet the full inclusion criteria and were deemed screen failures and were dropped from the study. These screen failures consisted of medical chart review resulting in these 13 having previous or current other medical conditions that may have affected their sensory system as is indicated in the formal exclusion criteria. The remaining 31 participants began the research study with 3 of the participants not completing all assessments. Thus 28 participants were included in the final analysis. The data analysis included descriptive, inferential and correlative statistics using SPSS version 25 (IBM Corp., 2017).

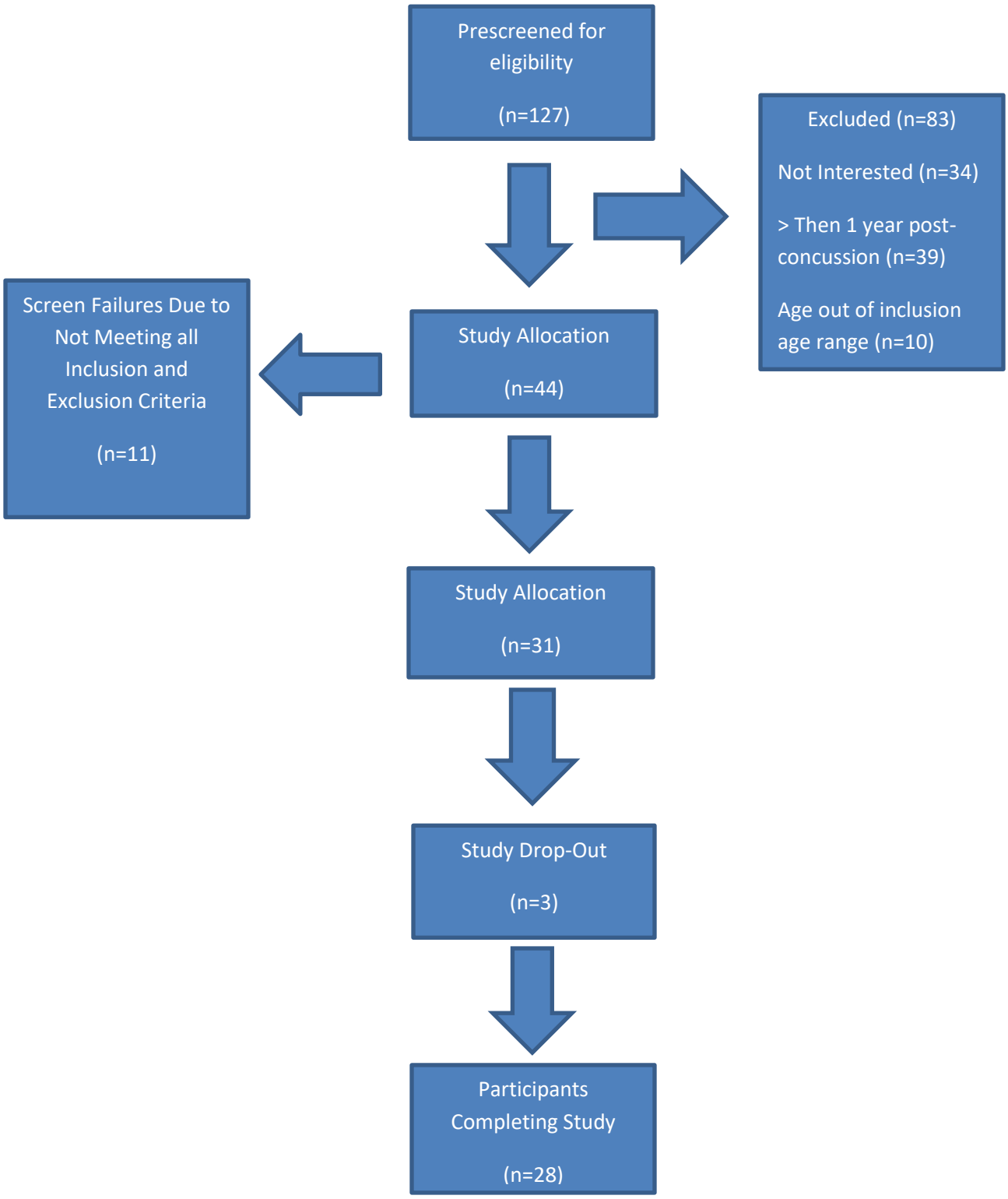

Figure 4. Consort Diagram

Descriptive Analysis

The total sample size was *N*=28, with the average age of 36.4 (*SD*=12.9) and age range of 19-62 years old. 67.8% (*n*=19) of the sample was female and 32.2% (*n*=9) was male. The average time period from concussion to research visit was 98.8 days with a range from 32-300 days. In regard to employment 53.5% (*n*=15) of the participants classified themselves as short-term disabled, 27.5% (*n*=8) were employed, 14.2% (*n*=4) were attending college and 3.5% (*n*=1) were retired. 25.0% (*n*=7) of the participants had a medical history of previous concussions.

A post hoc power analysis was completed using G* power version 3.1.9.4. (Faul, Buchner, Erdfelder, & Lang, 2007). Using the ASP tool as the primary outcome measure for this analysis with an associated Glass Delta effect size of 0.89, *p* value (<0.05) and a one sample *t* test statistical procedure, the post hoc power was .99 greatly exceeding the original power in the a priori power analysis of .80

Table 1

Gender

Gender	Frequency	Percent
Female	19	67.8%
Male	9	32.2%
Total	28	100%

Table 2

Sample Characteristics of Participants

Variable	Mean	Standard Deviation	Min-Max Range
Age	36.14	12.89	19-62 Years
Days from Concussion to Research Participation	98.18	60.09	32-300 days

Primary Research Question #1: Do individuals with a diagnosis of post-concussion experience greater amounts of sensory processing deficits as compared to the normal population? The planned analysis for this research question was a one sample *t* test examining the scores on the ASP and ASPS to normative data for these assessments. An individual one sample *t* test was performed for each sub-group in the assessments as they related to the different types of sensory processing. The key assumption underlining the one sample *t* test is data normality for the research group. (Elliott & Woodward, 2007). Normality was established for 82% (*n*=9) of the sub-groups' categories for the ASPS and 100% of the quadrants for the ASP using Sharpio-Wilk test for normality. The Sharpio-Wilk test is the preferred test for establishing normality for small sample sizes where N is less than 50 (Laerd Statistics, 2019). The Over Responsive to Auditory Input ASPS subgroup had a mild departure from normality, (p=0.033), thus a parametric one sample *t* test was still used for this analysis. Considering the other subgroup, Under Responsive to Auditory (Seeking) had a significant departure from normality, (p<0.000) further analysis was not performed. A

non-parametric analysis was not possible due to the median of the normative data set not available in the literature for comparison.

The results of the ASP one sample *t* test are seen in Table 3, while the results for the ASPS one sample *t* test is seen in Table 4. As Table 3 indicates research participants exhibited statistically mean differences in all four quadrants of the ASP as compared to the normative data. As Table 4 indicates the research participants exhibited statistical mean differences in 50% of the ASPS subcategories (*n*=5)

Table 3

Results for ASP

ASP Quadrant	Sharpio-Wilk *P* Value	ASP Research Group Mean/SD	ASP Normative Group Mean/SD	*P* Value
Low Registration	0.582	40.52 (10.21)	30.29 (6.25)	*p*<0.000
Sensation Seeking	0.501	42.96 (8.29)	49.91 (6.83)	*p*<0.000
Sensory Sensitivity	0.644	40.52 (10.12)	33.71 (7.63)	*p*<0.000
Sensation Avoiding	0.529	45.52 (10.21)	34.57 (7.34)	*p*<0.000

Table 4

Results for ASPS

Sub Groups for ASPS	Sharpio-Wilk *P* Value	ASPS Research Group Mean	ASPS Normative Mean	*P* Value
1. Over Responsive to Vestibular Input	p=0.752	19.89 (5.58)	15.77	p<0.000
2. Over Responsive to Auditory Input	p=0.033	26.55 (6.86)	20.05	p<0.000
3. Over Responsive to Visual Input	p=0.103	18.96 (7.02)	15.53	p=0.007
4. Over Responsive to Tactile	p=0.925	10.57 (3.01)	10.04	p=0.215
5. Proprioceptive Seeking (Under)	p=0.070	12.17 (5.15)	11.80	p=0.350
6. General Under Responsive	p=0.604	12.03	9.03	p<0.000
7. Vestibular-Proprioceptive Motor/Postural	p=0.402	11.89 (3.77)	13.18	p=0.082
8. Under Responsive to Auditory	p<0.000*			
9. Over Responsive to Tactile	p=0.402	6.92 (2.44)	7.33	p=0.196
10. Over Responsive to Vestibular	p=0.164	9.17 (3.33)	7.56	p=0.050
11. Over Responsive to Tactile (Clothing)	p=0.529	7.64 (3.39)	7.48	p=0.403

*Denotes a Sharipo-Wilk *p* value < 0.000 indicates significant departure from data normality

Effect size was calculated for the results of the four quadrants of the ASP from the research group data and compared to the normative data for the ASP as seen in Table 7. Glass Delta was used to calculate these effect sizes secondary to large variances in the N of the samples and differences between the SD of the two sample groups (Laird Statistics, 2018). This was done by calculating the difference between the means of the quadrant scores and normative data associated with the quadrants and dividing it by the SD of the normative data sample group. Effect size was not calculated for the ASPS subgroups secondary to limited normative necessary *SD* data available in the literature.

Table 5

ASP Quadrant Effect Size

ASP Quadrant	Glass Delta
Low Registration	1.63
Sensory Seeking	1.01
Sensory Sensitivity	1.75
Sensory Avoiding	1.49

Secondary Research Question #2: Is there a relationship between scores on the sensory processing assessments and the scores on the outcome tools designed to measure QOL and everyday function? The planned analysis for this is a bivariate correlation examining relationships between the ASP and ASPS scores with the perceived quality of life (as measured by the QOLBRI and SWLS) and everyday function (as measured by the FSE) using Pearson product-moment correlation coefficient. Considering the hypothesis associated with research question #1, these correlation analyses included the individual quadrants scores of the ASP and the 5 subgroups for the ASPS that demonstrated statistical significance. Preliminary analysis was performed to ensure no violation of the assumptions of normality and linearity. Normality was assessed using Sharpio-Wilk test as seen in Table 3 & 4, and linearity was established using visual inspections of scatterplots of the variables.

Table 6

ASP Correlations

ASP Total Score and Quadrants	QOLIBRI	SLS	FSE
Low Registration	*r*= .169 *p*= 0.465	*r*=- .037 *p*= 0.851	*r*= *.625* *p*= 0.001
Sensory Seeking	*r*= -.217 *p*= 0.345	*r*= 0.356 *p*= *0.081*	*r*= -.429 *p*= 0.032
Sensory Sensitivity	*r*=. 235 *p*= 0.305	*r*= -.356 *p*= 0.810	*r*= .690 *p*< 0.000
Sensory Avoiding	*r*=. 240 *p*= 0.295	*r*=- .335 *p*= 0.102	*r*= .510 *p*= 0.009

Note. *r* represents Pearson product-moment correlation coefficient value

Table 7

ASPS Correlations

ASPS Subgroup	QOLIBRI	SWLS	FSE
Over Responsive to Vestibular Input	*r*= .365 *p*= 0.087	*r*= .169 *p*= 0.391	*r*= .579 *p*= 0.002
Over Responsive to Auditory Input	*r*= .546 *p*= 0.007	*r*= .304 *p*= 0.116	*r*= .628 *p*< 0.000
Over Responsive to Visual Input	*r*= .001 *p*= 0.995	*r*= .087 *p*= .659	*r*= .280 *p*= 0.157
General Under Responsive	*r*= .340 *p*= 0.867	*r*= .107 *p*= 0.589	*r*= .339 *p*= 0.84
Over Responsive to Vestibular	*r*= .053 *p*= 0.809	*r*= -.019 *p*= 0.923	*r*= .464 *p*= 0.015

Discussion

The purpose of this study was to explore the prevalence of sensory processing deficits in a concussion patient population along with exploring possible relationships between sensory processing deficits and QOL and everyday function indicators. Driving this line of scientific inquiry was the PI's previously collected anecdotal patient report information indicating the possibility that post-concussion patients were experiencing sensory processing challenges which may have been impacting their quality of life and everyday function. Using this anecdotal information a non-experimental clinical design was used to investigate this phenomenon.

As Table 2 indicates the average time from diagnosis of concussion to actual research sessions was 98 days. This demographic variable is essential in

understanding the chronic nature of concussion recovery and post-concussion syndrome. This demographic also reinforces the rational for this study, as the majority of concussed individuals continue to experience post-concussion syndrome symptomology months after their concussion, including sensory processing deficits. Often these symptoms are the impetuous for them to seek rehabilitative interventions at the Concussion Center at NYULMC.

The final sample size from this study, *N*=28 exceeded the a priori power analysis required size of *N*=27 by one participant for the primary hypothesis. As previously noted, the sample sizes for the normative data were significantly larger than the research sample size for this study. Specifically, normative sample size for the ASP was *N*=496, and normative sample size was *N*=491 for the ASPS. Some levels of homogeneity existed between the normative data and the research group in regard to age of participants. For the ASPS tool the mean age for the participants in the normative data set was 37.1 (*SD*=12.8) whereas the mean age in the research group was 36.4 (*SD*=12.9). In regard to gender, some levels of homogeneity existed between the normative data set for the ASPS tool and research group. In the research group 67.8% (n=19) of the sample were female and 32.2% (*n*=9) were male whereas 64.3% (*n*=298) were female 35.7% (*n*=193) were male in the normative data set for the ASPS.

The literature consistently describes a higher proportion of females experiencing concussions and the resulting symptomology. For example, as compared to their male counterparts, female athletes appear to be at a greater risk of sustaining a concussion and they exhibit a higher number of symptoms in the acute phase, and typically requiring a longer recovery time (Kostyun & Hafeez, 2015; Preiss-Farzanegan,

Chapman, Wong, Wu, & Bazarian, 2009.) Broshek et al. (2005) reported concussed females experienced greater declines in cognitive function and indicated a greater number of symptoms both subjectively and objectively as compared concussed males. Consistent with the literature the proportion of both eligible pre-screened concussed individuals 69.2% (*n*=88) and the research group 67.8% (*n*=19) was heavily skewed to the female gender.

The etiology of such disparities seems to be related to the difference in brain connectivity between males and females. Research has demonstrated that males tend to be more single hemispheric dependent for many tasks, while females tend to more bi-hemispheric dependent (Nowicka & Fersten, 2001). Integrated bi-hemispheric activity requires an intact corpus callosum to relay information to both hemispheres. As previously outlined, the CC is a major structure that has been shown to be susceptible to damage following a concussion. A study done by Charnard, Lefebvre, Lasssonde, and Theort (2016) examined long term abnormalities in the CC of female concussed athlete. Using DTI imaging, the researchers compared a group of 8 healthy unconcussed females to a group of 10 females six months post-concussion. The CC for the concussed females displayed compromised axonal function mainly in the areas that projected into the parietal and temporal lobes of the brain. In conclusion, the CC is the primary structure connecting the left and right hemispheres. Because females rely on greater interhemispheric connections as compared to males, any disruption to this neural network from axonal damage may be the cause of more severe and prolonged symptomology experienced by females who have sustained a concussion (Solomito, Reuman, & Wang, 2019).

For the first research question the primary research hypothesis can be accepted because there was a significant difference between all four of the ASP quadrants scores for the research group as compared to the normative sample group ($p< 0.000$). The effect size for this comparison was quite large ranging from (.89 to 1.63) for the four different ASP quadrants as seen in Table 7. As Sawilowsky (2009) indicates these effect sizes for the ASP analysis is classified as very large and represents a significant amount of standard deviation when measuring the difference between these groups. Examining each quadrant score and their relationship to each other and to overall human sensory processing can further highlight possible sensory system dysfunction. For example, individuals who are experiencing overall sensory sensitivity exceeding their neurological threshold or comfort zone would most likely experience more sensory avoidance to reduce overall sensory input. The study results demonstrate this is indeed what occurred with research participants experiencing both statistically significant differences in sensory sensitivity and sensory avoidance. More importantly is the functional aspect of this dysfunction, whereas such sensory sensitivity and subsequent sensory avoidance could impact individual's engagement in their environment and their everyday function in activities of daily living. Another example can be seen with the ASP quadrants of sensory sensitivity and sensory seeking. If an individual is experiencing sensory hypersensitivity then they may not engage in appropriate levels of sensory seeking behaviors in a desire to limit sensory input that may be interpreted by the central nervous system as excessive or problematic. The study results support this frame of reference as research participants experienced both statistically significant differences in sensory sensitivity and sensory seeking. Specifically, participants

experienced a statistically higher amount of sensory sensitivity while experiencing a statistically significant lower amount of sensory seeking.

Further evidence to support the acceptance of the primary hypothesis is the results of the scores for the ASPS. This tool examined sensory processing related to five of the sensory systems; vestibular, auditory, visual, tactile and proprioception. This tool classified participants as either experiencing over responsive, under responsive or normal levels of responsiveness. The findings of this study identify that participants experienced statistically significant differences in the areas of over responsiveness to vestibular (p<0.000), auditory (p<0.000) and visual (p=0.007) sensory input while also demonstrating significant difference in overall under responsiveness (p<0.000). Although these results on the surface may contradict one another, when we examine how individuals might respond to more chronic over-responsiveness in certain sensory systems these results seem plausible. Individuals experiencing chronic over-responsiveness in a certain sensory system may have dysfunction in the CNS ability to regulate the balance between sensory habituation and sensitization thus producing unconscious or conscious overall under-responsiveness to certain sensory environments as a mechanism to create less sensory input.

In addition, this information is quite valuable because it demonstrates particular areas of sensory processing dysfunction as they relate to specific sensory systems. An example of this can be found in the statistically significant finding in the area of over responsiveness to visual input, most likely resulting from participants' sensitivity with different types of light and glare along with monocular/binocular eye function used in such activities reading and computer use. Such detailed sensory processing

assessment information could be valuable to both the post-concussed individual and to rehabilitation professionals as they may be able to develop specific rehabilitation protocols to address such sensory processing deficits either through compensatory or remediation techniques.

The secondary research question explored in this study sought to determine whether there were any relationships between noted sensory processing dysfunction as indicated by the participant's scores on the ASP and ASPS and the QOLIBRI, SWLS, and the FSE. Based upon the study findings the research hypothesis can be accepted as there were some levels of small correlations between the sensory processing outcome tools and the QOL outcome tools and more moderate to stronger correlations between the sensory processing outcome tools scores and the outcome tool measuring everyday function.

Using Cohen's (1983) correlation guidelines and examining the results of the APS and QOL and everyday function outcomes, 25% (*n*=3) of the *r* coefficient variable fell into the large correlation range, 25% (*n*=3) fell into the medium correlation range, 34% (*n*=4), fell into the small correlation range while 16% (*n*=2) showed no correlations between these variables as seen in Table 5. Interestingly, the strongest ASP correlations were found when comparing to the FSE outcome variable. All four of these correlations, between the four quadrants for the ASP and the FSE demonstrated moderate to strong positive correlations. The higher the score on the FSE the more dependent an individual is in their everyday function thus this positive correlation would be expected. The smallest set of correlations were seen with the APS and the QOLIBRI outcome tool. All correlations between the quadrants of the ASP and QOLIBRI were in

the small range. A most interesting correlation was found between the sensory sensitivity quadrant and the SLWS and FSE outcome measures. There was a moderate (*r*=.-356, *p*=0.810, negative correlation between this ASP quadrant and the SLWS and a strong positive correlation between this ASP quadrant and the FSE (*r*=.690, p<0.000). These would be expected results as increases in sensory sensitivity has the potential to negatively impact QOL for individuals. In addition, increased sensory sensitivity also has the potential to impact levels of assistance needed during everyday function.

When examining the results of the ASPS and the QOL and everyday function outcomes variables similar correlations are present particularly for the ASPS and FSE outcome results. Correlation analyses were performed for the five ASPA subgroups that showed statistical significance for the primary hypothesis as seen in Table 6. These results indicate a range of correlation coefficients from *r*=.280-.628 for the five ASPS subgroups and the FSE outcome measure. 80% (*n*=4) of the subgroups had a moderate to strong positive coefficient. There was one noticeable data outlier demonstrating a moderate positive correlation between the ASPS over-responsiveness to auditory input and the QOLIBRI, (*r*=.546, *p*=0.007) and SWLS, (*r*=.304, *p*=.304) outcome measures. Typically, we would expect such over-responsiveness to possibly negatively impact scores on these two QOL indicators. In summary all of the correlation analyses need to be viewed with caution as the sample size was small for a typical correlation analysis and the *p* values had significant variations among the different Person correlation coefficient analyses, indicating inconsistent levels of confidence that the correlation analysis results would have occurred by chance. Although such caution is warranted clearly there is some level of scientific support that sensory processing dysfunction may

be associated with lower levels of everyday function and dependence as demonstrated by the FSE for individual suffering from post-concussion syndrome? Both sensory processing outcomes tools demonstrated a majority of either moderate to strong correlations to the FSE outcome measure. In addition, although not as frequent or as strong there were some small-moderate correlations between the presence of sensory processing dysfunction and QOL indicators as measured by the QOLIBRI and SWLS. In conclusion considering that 53.5% of the research participants reported experiencing short-term disability resulting from the concussion the association between sensory processing dysfunction and everyday function should be further investigated in future studies.

Chapter V

SUMMARY AND CONCLUSIONS

Summary

This study was grounded in the theoretical model of brain function specificity and resulting brain dysfunction and symptomology following a concussion. The results indicate that post-concussion individuals may be experiencing greater amounts of sensory processing dysfunction as compared to normative population data. In addition, the results suggest that there may be some associations between the presence of such sensory processing dysfunction and QOL and everyday function.

Limitations

Similar to all research this study had limitations in design and analysis. This prospective, non-experimental, cross sectional design had threats to both internal and external validity.

Internal validity considerations. Many extraneous factors could have contributed to this study's findings. First, a convenience and consecutive sampling mechanism was used to recruit study participants. A selection effect could have occurred resulting in confounding variables with the research group that were not able to be controlled. For example although attempts were made to screen individuals who may have had a

conditions affecting sensory processing we cannot rule out that some of the individuals in the research group had previous history of sensory processing challenges that would have impacted the study results.

Second, all of the outcome tools were some variation of self-report. Response bias is a widely discussed phenomenon in behavioral and healthcare research where self-reported data are used; it occurs when individuals offer self-assessed measure of some phenomenon (Rosenman, Tennekon, & Hill, 2011). As Rosenman, Tennekon, and Hill, (2011) suggests there are many reasons individuals might offer biased estimated of self-assessed behaviors, ranging from misunderstanding of what a proper measurement is to social-desirability bias. Such response bias is most concerning to researchers when data collection is done after an intervention. Although certainly a possibility, considering this design was non-experimental, response bias was hopefully minimized by the PI by being present during the completion of the outcome tools and through the detailed instructions the participants received when completing the outcome tools. A third internal validity concern were the possible experimenter effects the PI had on the behavior of the research participants. This was minimized by the PI using a set script to describe each outcome measure and specific instructions for each measure.

External Validity. The research design used could have produced a participant group that does not truly represent a post-concussion syndrome population. For example, the participants may have been biased in their agreement to participate or could have been influenced to participate by other factors. The overall generalizability of the study results is limited to a sample of post-concussion individuals who have post-concussion

syndrome symptomology which have not resolved in the traditional 7-10-day concussion recovery period and who are actively participating in outpatient rehabilitation.

Design Limitations

Research Question 1

A one sample *t* test was used to compare results from the ASP and ASPS to normative data population data. There are no assurances that sample characteristics from the research group were identical to the normative group which may have resulted in sampling error. As a result interpretations from one sample *t* tests should be viewed with caution. For example *SD* normative data is not included in the one sample *t* test thus sampling errors cannot be ruled out when interpreting one sample *t* tests.

Research Question 2

Correlation studies have inherent limitations which is the case with this particular study. Correlation findings in this study do not imply any causation between sensory processing deficits and QOL and everyday function. Clearly, other contributory factors could have influenced these findings. Some of these factors are known and some of these factors may be unknown to the PI. In conclusion, as Portney and Watkins (2010) indicate the application of correlation findings to clinical decision making must be considered carefully secondary to the limited amount of information such analysis yields about the actual nature of the phenomena being studied.

Conclusions

The purpose of this study was to attempt to address a literature gap in understanding post-concussion syndrome sensory processing symptomology and possible associations with individual's QOL and everyday function. The findings from this study, along with the chronic nature of the observed sensory processing deficits reveals the importance for all medical and rehabilitative practitioners to increase their knowledge about the prevalence of such deficits in order to properly identify and provide rehabilitation interventions to address these deficits. This will hopefully result in improved rehabilitation and functional outcomes for this population.

Future Research

This study produces a theoretical foundation for the development of effective compensatory and remediation rehabilitation interventions for individuals experiencing post-concussion syndrome sensory processing deficits. Efficacy research studies for these interventions should be prioritized as part of a post-concussion evidence-based rehabilitation practice model.

It is also recommended for future exploratory studies to use a larger sample size so the generalizability of the findings can be expanded. Within these future exploratory studies expanding the outcome measure to use both self-report and performance-based measures to measure sensory processing may increase the level of study internal validity.

REFERENCES

Aitken, M.E., Mele, N., & Barrett, K.W. (2004). Recovery of injured children: Parent perspectives on family needs. *Archives of Physical Medicine and Rehabilitation*, *85*, 567-573. http://dx.doi.org/10.1016/j.apmr.2003.06.018

Alwis, D.S., Johnstone, V., Yan, E., & Rajan, R. (2013). Diffuse traumatic brain injury and the sensory brain. *Clinical and Experimental Pharmacology and Physiology*, *40* (7), 473-483. http://aups.org.au/Proceedings/44/13-26

American Academy of Pediatrics (2012). Sensory integration therapies for children with developmental and behavioral disorders. http://pediatrics.aapublications.org/content/129/6/1186.long/. Accessed February 2015.

Ayres, A.J. (1972). *Sensory integration and learning disabilities*. Los Angeles: Western Psychological Services.

Barhoudarian, G., Hovda, D. A., & Giza, C.C. (2016). The molecular pathophysiology of concussive brain injury-an update. *Phys Med Rehabil Clin N Am, 27(*2) doi: 10.1016/j.pmr.2016.01.003

Bay, E., & Mclean, S.A. (2007). Mild traumatic brain injury: An update for advanced practice nurses. *Journal of Neuroscience Nursing*, *39*, 43-51. http://dx.doi:10.1097/01376517-2070

Bear, M.F., Conners, B. W., Paradiso, M. A. (2007). *Neuroscience Exploring the Brain* (3rded.) Philadelphia, PA: Lippincott, Williams, and Wilkins

Ben-Sasson, A., Carter, A.S., & Briggs-Gowan, M.J. (2009). Sensory over-responsivity in elementary school: prevalence and social-emotional correlates. *Journal of Abnormal Child Psychology*, *37*, 705-716. http://dx. Die: 10.1007/s10802-008-

9295-8

Bilge, E.D. (2008). Neuropsychology and clinical neuroscience of persistent post-concussive *Journal of International Neuropsychology*, *14* (1), 1-22. http://dx.doi: 10.10170S135561770808017X

Blanche E.I., Parham, D., Chang, M., Mallinson, T. (2014). Development of an Adult Sensory Processing Scale (ASPS). *American Journal of Occupational Therapy*, 68 (5), 531-538.

Borich, M., Babul, A.N., & Huang, P.H. (2014). Alterations in resting state brain networks in concusses adolescent athletes. *Journal of Neurotrauma*, 13, (2), 345- 356.

Breedlove, M.S., Rosenzweig, M.R., & Watson, N.V. (Eds.) (2007). *Biological psychology: An introduction to behavioral, cognitive and clinical neuroscience* (5thed.). Sunderland, MA: Sinauer Associates

Breet-Green, B.A., Miller, L.J., Gavin, W.J., & Davies, P.L. (2008). Multisensory integration in children: a preliminary ERP study. *Brain Research*, *12* (42), 283-290. http://dx. doi:10.1016/j.brainres.2008.03.090

Briggs, F. & Usrey, W.M. (2008). Emerging views of corticothalamic function. *Current Opinion in Neurobiology, 18,* 403-407. http://dx.doi:10.1016/j.conb.2008.09.002

Broshek, D.K., Kaushik, T., Freeman, J.R., Erlanger, D., Webbe, F., & Barth, J.T. (2005) Sex differences in outcome following sports-related concussion. *Journal of Neurosurgery*, 102(5), 856-863.

Brown, C., & Dunn, W. (2002). *Adolescent/Adult Sensory Profile: User's manual*. San Antonio, TX: Psychological Corporation.

Brown, C., Tollefson, N., Dunn, W., Cromwell, R., & Filion, D. (2001). The Adult Sensory Profile: Measuring patterns of sensory processing. *American Journal of*

Occupational Therapy, *55*, 75-82. http://dx. doi:10.5014/ajot.55.1.75

Buki, A., & Povlishock, J. T. (2006). All roads lead to disconnection? Traumatic axonal injury revisited. *Acta Neurochir (Wien)*, *148*, 181-194. http://dx.dio:10.1007/s00701-005-0674-4

Calvert, G.A., Spence, C., & Stein, B. E. (Eds.) (2004). *The handbook on multisensory processes*. Cambridge, MA: MIT Press

Cantu, R. C. & Register-Mehalik J. K. (2011). Considerations for return-to-play and retirement decisions after concussion*. Physical Medicine & Rehabilitation 3,* S440-S444. http://dx.doi:10.1016/j.pmrj.2011.07.013

Cappe, C., Rouiller, E.M., & Barone, P. (2009). Multisensory anatomical pathways*. Hearing Research*, *258*, 28-36. http://dx.doi: 10.1016/j.heares.2009.04.017, 2009

Chang, Y.S., Gratiot, M., Owen, J.P., Brandes-Aitken, A., Desai, S. S., Hill, S., . . . Mukjerjee, P. (2016). White matter microstructure is associated with auditory and tactile processing in children with and without sensory processing disorders. *Frontiers in Neuroanatomy*, *26*, 1- 4. http://journal.frontiersin.org/article/10.3389/fnana.2015.00169/

Churchill, N.W., Hutchison, M.G., Richards, D., Leung, G., Graham, S.J., Schweizer, T. (2017). The first week after concussion: Blood flow, brain function and white matter microstructure. *Neuroimage Clinical,* 14, 480-489.

Chait, M., de Cheveigne, A., Poeppel, D., Simon, J.Z. (2010). Neural dynamics of attending and ignoring in human auditory cortex. *Neuropsychologia, 48*, 3262-3271. http://dx.doi:10.1016/j.neuropsychologia.2010.07.007

Clark, J.M., & Zaidel, E. (1994). Anatomical-behavioral relationships: corpus callosum morphometry and hemispheric specialization. *Behavior and Brain Research*, *64*, 185-202. http://dx.doi:10.1016/0166-4328(94)90131-7

Charmard, E., Lefebvre, G., Lassonde, M., & Theoret, H. (2016). Long-term abnormalities in the corpus callasum of the female concussed athletes. *Journal of Neurotrauma*. 33, 1220-1126. DOI: 10.1089/neu.2015.3948.

Chong, C.D, Schwedt, T.J. (2014). White Matter Damage and Brain Network Alterations in Concussed Patients: A Review of Recent Diffusion Tensor Imaging and Resting-State Functional Connectivity Data. *Curr Pain Headache Rep*, 19: 12 DOI 10.1007/s11916-015-0485-0

Cohen,J.W. (1988). *Statistical power analysis for behavioral sciences. (*2nd edn.). Hilldisde, NJ: Lawerence Erlbaum Associates.

Coronado, V. G., Xu, L., Basavaraju, S.V., McGuire, L.C., Wald, M.M., Faul, M. D., . . . Hemphill, J.D. (2011). Surveillance for traumatic brain injury- related deaths-United States, 1997-2007. *MMWR Surveillance Summary*, *60*, 1-32.

Crandall, S.R., Cruikshank, S. J., & Conners, B. W. (2015). A corticothalamic switch: Controlling the thalamus with dynamic synapses. *Neuron*, *86,* 768-782. http://dx.doi:10.1016/j.neuron.2015.03.040. Epub 2015 Apr 23.

Creed, J.A., DiLeonardi, A.M., Fox, D.P., Tessler, A.R., & Raghupathi R. (2011). Concussive brain trauma in the mouse results in acute cognitive deficits and sustained impairment of axonal function. *Journal of Neurotrauma*, *28*, 547-563. http://dx.doi:10.1089/neu.2010.1729

Critz, C., Blake, K., & Nogueira, E. (2015). Sensory Processing Challenges in Children. *The Journal of Nurse Practitioners*, *11* (7), 710-716. http://dx.doi.org/10.1016/j.nurpra.2015.04.016

Daneshvar, D.H., Nowinski, C.J., McKee, A.C., & Cantu, R.C. (2011). The epidemiology

of sport-related concussion. *Clinical Sports Medicine*, 30, 1-17.

Davies, P.L., & Gavin, W.J. (2007). Validating the diagnosis of sensory processing disorders using EEG Technology. *American Journal of Occupational Therapy,* 61(2), 176-189. http://dx.doi: 10.5014/ajot.61.2.176.

Dawson, M.E., Filion, D.L., & Shell, A. M. (1989). Is elicitation of the autonomic orienting response associated with allocation of processing resources? *Psychophysiology*, *26*, 560-572. http://dx.doi:10.1111/j.1469-8986.1989.tb00710.x

Dean, A.J., Sato, J.R., Viera, G., McNamara, A., & Steer A. (2015). Long term structural changes after mTBI and their relationship to post-concussion symptoms*. Brain Injury*, *29*(10), 1211-1218. http://dx.doi:10.3109/02699052.2015.1035334

Delouche, A., Attye, A., Heck, O., Grand, S., Kastler, A., Lamalle, L., . . . Krainik, A. (2016). Diffusion MRI Pitfalls: Pitfalls, literature review and future directions of research in mild traumatic brain injury. *European Journal of Radiology*, *85*, 25-30. http://dx.doi:10.1016/j.ejrad.2015.11.004

Demopoulos, C., Arroyo, M., Dunn, W., Strominger,Z., Sherr, E.H., & Marco, E. (2015). Individuals with agenesis of the corpus callosum show sensory processing difficulties as measured by the sensory profile. *Neuropsychology*, *29* (5), 751-758. http://dx.doi.org/10.1037/neu0000165

Dick, R.W. (2009). Is there a gender difference in concussion incidence and outcomes? *British Journal of Sports Medicine*, *43*, i46-50. http://dx.doi:10.1136/bjsm.2009.058172

Dickmen, S., Machamer, J., Miller, B., Doctor, J., & Temkin, N. (2001). Functional status examination: a new instrument for assessing outcome in traumatic brain injury. *Journal of Neurotrauma*, *18* (2), 127-140. http://dx.doi:10.1089/08977150150502578

Ding, K., Marquez de Plata, C., Wang J.Y., Mumphrey, M., Moore, C., Harper, C., . . . Diaz-Arrastia R. (2008). Cerebral atrophy after traumatic white matter injury: Correlation with acute neuroimaging and outcome. *Journal of Neuortrauma, 25*, 1433-1440. http://dx.doi: 10.1089/neu.2008.0683

Dickman, S.S., Matchmaker, J.E., Miller, B., Doctor J., Temkin, N. (2001). Functional status examination: a new instrument for assessing outcome in traumatic brain injury. *Neurotrauma*, 18(2), 127-140.

Dionne-Dostie, E., Paquette, N., Lassonde, M., & Gallagher, A. (2015). Multisensory integration and child development. *Brain Sciences*, *5,* 32-57. http://dx. doi:10.3390/brainsci5010032

Driver, J., & Noesselt, T. (2008). Multisensory interplay reveals crossmodal influences on sensory specific brain regions, neural responses and judgments. *Neuron, 57* (1), 11-23. http://dx.doi:10.1016/j.neuron.2007.12.013.

Dunn, W. (1997). The impact of sensory processing abilities on the daily lives of young children and their families: A conceptual model. *Infants and Young Children*, *9*, 23-35. http://dx.doi:10.1097/00001163-199704000-00005

Dunn, W., Westman, H. D. (1997). The sensory profile: the performance of a national sample of children without disabilities. *American Journal of Occupational Therapy*, *51*, 25-34. http://dx.doi:10.5014/ajot.51.1.25

Elliott, A.C. & Woodward, W.A. (2007). *Statistical Analysis Quick Reference Guide* Thousand Oaks, California: Sage Publications.

Echemendia, R.J. (2006). *Sports Neuropsychology*. Guilford Press: New York.

Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, *39*, 175-191.

Faul, M., Xu, L., Wald, M.M., & Coronado, F.G. (2010). *Traumatic brain injury in the United States: emergency department visits, hospitalizations and deaths 2002-2006.* Atlanta: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control.

Galvin, J., Froude, E.H., Imms, C. (2009). Sensory processing abilities of children who have sustained traumatic brain injuries. *American Journal of Occupational Therapy, 63* (6), 701-709. http://dx. doi:10.5014/ajot.63.6.701

Ghazanfar, A.A., & Schroeder, C.E. (2006). Is neocortex essentially multisensory? *Trends in Cognitive Science, 10*, 278-285. http://dx. doi:10.1016/j.tics.2006.04.008

Graziano, M.S. (2001). A system of multimodal areas in the primate brain. *Neuron*, *29*, 4-6. http://dx. doi:10.1016/S0896-6273(01)00174-X

Greicius, M.D., Krasnow, B., Reiss, A.L. (2003). Functional connectivity in the resting brain: a network analysis of the default mod hypothesis. *Pro Natl Aca Science USA*, 100(1), 253-258

Griffin, G. D. (2011). The injured brain: TBI, mTBI, the immune system, and infection: Connnecting the dots. *Military Medicine*, *176* (4), 364-368. http://dx. doi: 10.7205/MILMED-D-10-00021

Halstead, M. E., & Walker, K.D. (2010). Clinical Report-Sport Concussion in children and adolescents. *Pediatrics, 126,* 597-615. http://dx. doi:10.1542/peds.2010-2005

Harmon, K.S., Drezner, J. A., Gammons, M., Guskiewicz, K. M., Halstead, S.A., Herring, S.A., . . . Roberts, W.O. (2013). American medical society for sports medicine position statement: concussion in sport. *British Journal of Sports Medicine*, *47,* 15-26. http://dx. doi: 10.1136/bjsports-2012-091941

Hofer, S., & Frahm, J. (2006). Topography of the human corpus callosum revisited-Comprehensive fiber tractography using diffusion tensor magnetic resonance imaging. *NeuorImage*, *32*, 989-994. http://dx. doi:10.1016/j.neuroimage.2006.05.044

Holli, K. K., Harrison, L. Dastidar, P., Waljas, M., Liimatainen, S., Luukkaais, T., . . . Eskola, H. (2010). Texture analysis of MR images of patient with mild traumatic brain injury. *BMC Medical Imaging*, *8*, 1-10. http://dx.doi**:** 10.1186/1471-2342-10-8

Holli, K. K., Waljas, M., Harrison, L., Liimataninen, S., Luukkaala, T., Ryymin R., . . . Dastidar, P. (2010). Mild traumatic brain injury: Tissue texture analysis correlated to neuropsychological and DTI findings*. Academic Radiology*, *17* (9), 1096-1102. http://dx. doi:10.1016/j.acra.2010.04.009

Holm, L., Cassidy, J., Carroll, L.J., & Borg, J. (2005). Neurotrauma trauma task force on mild traumatic brain injury of the WHJO collaborating centre. Summary of the WHO collaborating centre for neurotrauma task force on mild traumatic brain injury. *Journal of Rehabilitation Medicine, 37* (3), 137-141. http:// dx.doi:10.1080/16501970510027321

Hudak, A.M., Caesar, R. R., Frol, A.B, Krueger, K., Harper, C.R., Temkin, N.R., Dikmen, S.S,. . . Diaz-Arrastia, R. (2005). Functional outcome scales in traumatic brain injury: A comparison of the Glasgow Outcome Scale (Extended) and the Functional Status Examination. *Journal of Neurotrauma, 22* (11), 1319-1326. http://dx.doi: 10.1089/neu.2005.22.1319.

Hung, H., Zhang, J., Juang, H., Wakana, S., Poetscher, L., Miller, M.I., . . .Mori, S. (2005). DTI tractography based parcellation of white matter: application to mid-sagittal morphology of corpus callosum. *Neuroimage, 26*, 195-205. http://dx.doi: 10.1016/j.neuroimage.2005.01.019

Ilvesmaki, T., Luoto, T.M., Hakulinen, U., Brander, A., Ryymin, P., Eskola, H., . . . Ohman, J. (2014). Acute mild traumatic brain injury is not associated with white matter change on diffusion tensor imaging. *Brain,* 137, 1876-1882.

James, K., Miller, L.J., Schaaf, R., Nielsen, D. M., & Schoen, S. A. (2011). Phenotypes with sensory modulation dysfunction. *Comprehensive Psychiatry*, *52*, 715-724. http://dx. doi:10.1016/j.comppsych.2010.11.010

Johnson, V. E., Stewart, W., & Smith, D. H. (2013). Axonal pathology in traumatic brain injury. *Experimental Neurology*, *246*, 35-43. http://dx. doi: 10.1016/j.expneurol.2012.01.013.

Johnson, B., Zhang, K., Gay, M. (2012). Alterations of brain default network in subacute phase of injury in concussed individuals: resting-state fMRI study. *Neuroimage,* 59(1), 511-518.

Johnstone, V.P., Wright, D.K., Wong, K., O'Brien, T.J., Rajan, R., & Shultz, S. R. (2015). Experimental traumatic brain injury results in long-term recovery of functional responsiveness in sensory cortex but persisting structural changes and sensorimotor, cognitive and emotional deficits. *Journal of Neurotrauma*, *32*, 1333-1346. http://dx. doi: 10.1089/neu.2014.3785.

Jotwanni, V., & Harmon, K.G. (2010). Postconcussion syndrome in athletes. *Current Sports Medicine Report*, *9*, 21-26. http://dx.doi: 10.1249/JSR.0b013e3181ccb55e

Komar J. (2012, April). *Examination of qualitative and quantitative aspects of sensory processing in adults*. Paper presented at the American Occupational Therapy Association Annual Conference and Expo, Indianapolis, Indiana.

Kostyun, R.O., & Hafeez, I. (2015). Protracted recovery from a concussion: a focus on gender and treatment interventions in an adolescent population. *Sports Health*, 7,

52-57

Laerd Statistics. (2018). *Statistical tutorials and software guides*. Retrieved from https://statistics.laerd.com/

Lancaster, M.A, Olson, D.V., McCrea, M.A., Nelson, L.D., LaRoche, A.A., & Mufuler, L.T. (2016). Acute white matter changes following sports-related concussion: A serial diffusion tensor and diffusion kurtosis tensor imaging study. *Human Brain Mapping*, 37, 3821-3843.

Lane, S.J., Lynn, J.Z., & Reynolds, S. (2010). Sensory modulation-A neuroscience and behavioral overview*. OT Practice*, *15* (21), CE1-CE8

Laskowski, R.A., Creed, J.A., & Raghupathi, R. (2015). Pathophysiology of Mild TBI. In Kobeissy, F.H. (Ed), *Brain Trauma*, (pp. 35-41). Boston, MA: Taylor-Francis

Lange, R.T., Panenka, W.J., Shewchuk, J.R., Heran, M. K., Brubacher, J.R., Bioux, S., . . . Iverson, G.L. (2015). Diffusion tensor imaging findings and post-concussion symptom reporting six weeks following mild traumatic brain injury. *Archives of Clinical Neuopsychology*, *30*, 7-25. http://dx. doi: 10.1093/arclin/acu060.

Langlosis, J.A., Rutland-Brown, W., & Wald, M.M (2006). The epidemiology and impact of traumatic brain injury: a brief overview. *Journal of Head Trauma Rehabilitation*, 21, 275-378.

Levine, B., Kovacevic, N., Cheung, G., Gao, F., Schwatz, B.L. & Black, S. E. (2008). The Toronto traumatic brain injury study: injury severity and quantified MRI. *Neurology, 70,* 771-778. http://dx doi: 10.1212/01.wnl.0000304108.32283.aa

Lincoln, A. E., Caswell, S. V., Almquist, J.L., Dunn, R.E., Norris, J. B., & Hinton, R. Y. (2011). Trends in concussion incidence in high school sports: A prospective 11-year study. *American Journal of Sports Medicine*, *39,* 958-963. http://dx.doi: 10.1177/0363546510392326

Ling, J.M., Klimaj, S., Toulouse, A. R., & Mayer, A. (2013). A prospective study of gray matter abnormalities in mild traumatic brain injury. *Neurology, 81,* 2121-2127. http://dx.doi.org/10.1212/01.wnl.0000437302.36064.b1

Macaluso, E., & Driver, J. (2005). Multisensory spatial interactions: a window onto functional integration in the human brain. *Trends in Neurosciences*, *28* (5), 264-270. http://dx.doi:10.1016/j.tins.2005.03.008

Makdissi, M., Darby, D., Maruff, P., Ugoni, A., Bruckner, P., & McCrory, P.R. (2010). Natural history of concussion in sport: markers of severity and implications for management. *American Journal of Sports Medicine*, *38*, 464-471. http://dx.doi. 10.1177/0363546509349491

Mansell, J. L., Tienery, R. T., Higgins, M., McDevitt, J., Toone, N., & Glutting, J. (2010). Concussive signs and symptoms following head impacts in collegiate athletes. *Brain Injury*, *24*, 1070-1074. http://dx. doi:10.3109/02699052.2010.494589

Marar, M., Mclvian, N.M., & Fields, S.K. (2012). Epidemiology of concussions among high school athletes in 20 sports. *American Journal of Sports Medicine, 40,* 747-755. http://dx. doi: 10.1177/0363546511435626

May-Benson, T.A., & Kinnealey, C. (2012). Understanding the occupational needs for adults sensory processing disorder. *OT Practice*, 16(10), 13-18.

May-Benson, T.A., & Patane, S. (2010). *Commonalities in sensory processing of adults seeking sensory integration occupational therapy services*: A qualitative analysis. Watertown, MA: The Spiral Foundation.

McCrory, P. (2009). Sport concussion assessment tool. *Scandinavian Journal of Medical Science Sports*, *19*, 452-462.. http://dx.doi: 10.1111/j.1600-0838.2009.00978.x

McCrory, P., Meeuwisse, W., Aubry, M., Cantu, B., Dvorak, J., Echemendia, R. J., . .

Turner, M. (2013). Concussion statement on concussion in sport: the 4th international conference on concussion in sport held in Zurich, November 2012. *British Journal of Sports Medicine*, *43,* i76-90. http://dx.doi:10.4085/1062-6050-44.4.434

Meaney, D. F., & Smith, D. H. (2011). Biomechanics of concussion. *Clinical Sports Medicine*, *30*, 19-31.http://dx.doi: http://dx.doi.org/10.1016/j.csm.2010.08.009

Meehan, W.P. III, d'Hemcourt, P., & Comstock, R.D. (2010). High School Concussion in the 2008-2009 academic year: mechanisms, symptoms, and management. *American Journal of Sports Medicine*, *38*, 2405-2409. http:// dx. doi:10.1177/0363546510376737

Meier, T.B.,Bergamini, M., Bellgowan, S.F., Teague T.K., Ling, J.M., Jeromin, A., & Mayer, A.R. (2016). Longitudinal assessment of white matter abnormalities following a sports-related concussion. *Human Brain Mapping*, 37, 833-845

Meese, A., Caplain, S., Paradot, G., Garrigue, D., Mineo J.F., Sota, . . . Lehericy, S. (2010). Diffusion tensor imaging and white matter lesions at the subacute stage in mild traumatic brain injury with persistent neurobehavioral impairments. *Human Brain Mapping*, *32* (6), 999-1011. http://dx.doi:10.1002/hbm.21092. http://dx. doi: 10.1002/hbm.21092

Messe, A., Caplain, S., Pelegrini-Issac , M., Blancho, S., Montreuil, M., Levy, R., . . . Benali, H. (2012). Structural integrity and post-concussion syndrome in mild traumatic brain injury patients. *Brain Imaging and Behavior*, *6*, 283-292. http://doi:10.1007/s11682-012-9159-2

Miller, L., Anzalone, M., Lane, S., Cermak S., & Osten E. (2007). Concept evolution in sensory integration: a proposed nosology for diagnosis. *American Journal of Occupational Therapy, 61*, 135-140. http://dx. doi:10.5014/ajot.61.2.135

Miller, L.J., & Lane, S.J. (2000, March). Toward a consensus in terminology in sensory integration theory and practice: Part I: Taxonomy of neurophysiological processes. *Sensory Integration Special Interest Section Quarterly*, *23*(1), 1-4.

Miller, L. J., Nielsen, D. M., Schoen, S. A., & Brett-Green, B. (2009). Perspectives on sensory processing disorder: a call for translational research. *Frontiers in Integrative Neuroscience*, *3*, 1-12. http://dx. doi:10.3389/neuro.07.022.2009

Mustafi, S.M., Harezlak, J., Koch, K.M., Nencka, A.S., Meir, T. B., West, J.D. . . . Wu, Y.C. (2018). Acute white-matter abnormalities in sports-related concussion: A diffusion tensor imaging study from the NCAA-DoD CARE consortium. *Journal of Neurotrauma*. DOI: 10.1089/neu.2017.5158

Narayana, P.A. (2017). White matter changes in patients with mild traumatic brain injury: MRI perspective. *Concussion*, 2(2), CNC 35.

Niogi, S.N., & Mukjerjee, P. (2010). Diffusion tensor imaging of mild TBI. *The Journal of Head Trauma Rehabilitation 25* (4), 241-255. http://dx. oi:10.1097/HTR.0b013e3181e52c2a

Nowicka, A., Fersten, E. Sex-related differences in interhemispheric transmission time in the human brain. *Neuroreport,* 12(18), 4171-75.

Owen, J.P., Marco, E.J., Desai, S., Fourie, E., Harris, J., Hill, S, . . . Mukherjee, P. (2013). Abnormal white matter microstructure in children with sensory processing disorders. *NeuroImage: Clinical*, *2*, 844-853. http://dx.doi:10.1016/j.nicl.2013.06.009

Portney, L.G., & Watkins, M.P. (2000). *Foundations of Clinical Research* (2ed). Upper Saddle River River, NJ: Prentice-Hall Inc.

Preiss-Farzanggan, S.J., Chapman, B., Wong, T. M., Wu, J., & Bazarian, J.J. (2009) The relationship between gender and postconcussion symptoms after sports-

related mild traumatic brain injury. *PMR*, 1, 245-253.

Rosenman, R., Tennekon, V., & Hill, L.G. (2011). Measuring bias in self-reported data *International Journal of Behavior Health Research*, 2(4), 320-332.

Rutgers, D.R., Toulgoat, F., Cazejust, J., Fillard, P., Lasjaunais P., & Ducreux, D. (2008). White matter abnormalities in mild traumatic brain injury: A diffusion tensor imaging study. *American Journal of Neuroradiology*, *53* (8), 553-563. http://dx.doi: 10.1007/s11682-012- 9156-5

Sawilowsky, S. (2009). "New effect size rules of thumb". *Journal of Modern Applied Statistical Methods*. **8** (2): 467–474.

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 and Behavior*, 6*,* 137-192. http://dx.doi: 10.1007/s11682-012-9156-5.

Singh, M., Jeong, J., Hwang, D., Sungkarat, W., & Gruen, P. (2010). Novel diffusion tensor imaging methodology to detect and quantify injured regions and affected brain pathways in traumatic brain injury. *Magnetic Resonance Imaging*, *28*, 22-40. http://dx. doi:10.1016/j.mri.2009.05.049

Smits, M., Houston, G.C., Dippel, D.W., Wielopolski, P. A., Vernooji, M. W., Koudstaal, P.J. . .Van der Lugt, A. (2011). Microstructural brain injury in post-concussion Syndrome after minor head injury. *Neuroradiology, 53* (8), 654-671. http://dx. doi:10.1002/brb3.292

Solomito, M.J., Reuman, H., & Wang, D.H. (2019). Sex differences in concussion: a review of brain anatomy, function, and biomechanical response to impact. *Brain Injury*, 33(2), 105-110.

Sours, C., George, E.O., Zhuo, J., Roys, S., & Gullapalli, R. P. (2015). Hyper-

connectivity of the thalamus during early stages following mild traumatic brain injury. *Brain Imaging and Behavior*, *9,* 550-563. http://dx. doi:10.1007/s11682-015-9424-2

Su C. T., & Parham D. (2014). Validity of sensory systems as distant constructs. *American Journal of Occupational Therapy*, 68 (5), 546-554.

related mild traumatic brain injury. *PMR*, 1, 245-253.

Tang, L, Ge, Y., Sodickson, D. K., Miles, L., Zhou, Y., Reaume, J., & Grossman, R. (2011). Thalamic resting state functional networks: Disruption in Patients with Mild Traumatic Brain Injury. *Radiology, 260* (3), 831840.http://dx.doi:10.1148/radiol.11110014

Tremblay, S., Henry, L.C., Bedetti, C., Larson-Dupuis, C., Gagnon, J.F., Evans, A.C. . . . Beaumont, L.D. (2014). Diffuse white matter tract abnormalities on clinically normal ageing retired athletes with a history of sports-related concussions

Tyll, S., Budinger, E., & Noesselt, T. (2011). Thalamic influences on multisensory integration. *Communicative and Integrative Biology*, *4* (4), 378-381. http://dx. doi:10.4161/cib.4.4.15222

Van Hulle, C.A., Shmidt, N.L., & Goldsmith, H.H. (2012). Is sensory over-responsivity distinguishable from childhood behavior problems? A phenotypic and genetic analysis. *Journal of Child Psychology and Psychiatry*, *53*, 64-72. http://dx.doi:10.1111/j.1469-7610.2011.02432

Von Steinbuchel, N., Wilson, L., Gibbons, H., Hawthorne, G., Hofer, S., Schmidt, S., . . & Bullinger, M. (2010). Quality of lifer and brain injury (QOLIBRI): Scale development and metric properties. *Journal of Neurotrauma*, *27,* 1167-1185.

Warner, M.A., Marquez del Plata, C., Spence, J., Wang, J.Y., Harper, C., Morre, C., . . . & Diaz- Arrastia, T. (2010). Assessing spatial relationships between axonal

integrity, regional brain volumes, and neuropsychological outcomes after traumatic axonal injury. *Journal of Neurotrauma, 27*, 2121-2130. http://dx.doi:10.1089/neu.2010.1429

Wojcik, S. M. (2014). Predicting mild traumatic brain injury patients at risk of persistent symptoms in the emergency room. *Brain Injury*, *28* (4), 422-430. http://dx.doi: doi: 10.3109/02699052.2014.884241

Zho, Y., Milham, M.P., Lui, Y.W. (2012). Default-mode network disruption in mild traumatic brain injury. *Radiology*, 265(3), 882-892.

Zitnay, G. A., Zitnay, K.M., Povlishock, J.T., Hall, E.D, Marion, D. W., Trudel, T., . . . Barth, J. T. (2008) Traumatic brain injury priorities: The Conemuagh International Brain Injury Symposium. *Journal of Neurotrauma*, *25*, 1135-1152. http://dx.doi:10.1089/neu.2008.0599

Appendix A

NYULMC IRB Approval

Approval of Submission July 3, 2017

Dear Steve Vanlew:

On 7/3/2017 3:10 PM EDT, the IRB reviewed the submission below: All conditions for approval were met on 7/3/2017.

principal investigator	Steve Vanlew
email	vanles01@nyumc.org
study number	i16-01468
study title	Exploring the Prevalence and Relationship of Atypical Sensory Processing Patterns in the Mild Traumatic Brain Injury Population with Quality of Life and Functional Indicators
performance period	7/3/2017 - to 7/2/2018 inclusive. Before 7/2/2018 or within 30 days of study closure, whichever is earlier, you are to submit a continuing review with required explanations. You can submit a continuing review by navigating to the active study and clicking Create Modification / CR. If continuing review approval is not granted before the expiration date of 7/2/2018, approval of this study expires on that date.
location(s)	Rusk Rehabilitation (34th Street) (NYUMC Locations)
sponsor(s)	Name: Occupational Therapy
review type	Initial Study [Expedited Category 7]
board name	All Boards
materials approved for use	• 16-01468 MTBI Research Protocol 6-30-17 Clean.pdf, Category: IRB Protocol • 16-01468 MTBI Consent IRB 5-15-17.pdf, Category: Consent Form • SWLS Tool.pdf, Category: IRB Protocol • Adolescent-Adult Sensory Profile.pdf, Category: IRB Protocol • Adult Sensory Processing Questionnaire.pdf, Category: IRB Protocol • QOLABI.pdf, Category: IRB Protocol • FSE.pdf, Category: IRB Protocol • Study Demographics Form.pdf, Category: IRB Protocol
#of subjects approved to consent	50
vulnerable populations approved for participation in this study	

The current IRB Status of your study is: Approved. This study was reviewed by the NYU School of Medicine's Institutional Review Board (IRB). During the review of your study, the IRB specifically considered:

1. the risks and anticipated benefits (if any) to your subjects
2. the selection of subjects
3. the procedures for securing and documenting informed consent
4. the safety of your subjects
5. the privacy of your subjects and confidentiality of the data

Your study cannot commence until all ancillary review decisions are complete. In order to determine the state of all ancillary reviews please go the My Studies page of this study in Research Navigator. Ancillary review statuses will be found on the right side of the header section.

Please note; if your study includes a clinical trial agreement or budget you will need to ensure approval has been issued from My Agreements/CRMS and The Office of Clinical Trials before you proceed with any aspects of this study including the enrollment of human subjects.

phone (212) 263-4110 | fax (212) 263-4147 | email irb-info@med.nyu.edu | page 1 of 2

NYU School of Medicine IRB

Review Notes

For NIH Grant funded research: the IRB has found the IRB approved protocol referenced above to be consistent with the NIH grant application.

July 3, 2017

RE: Study#i16-01468
Helen Panageas, Director, Institutional Review Board OHRP #FWA00004952

Notes

- You must submit all changes to this study (e.g., protocol, recruitment materials, consent forms, etc.) via eSubmission to the IRB for review and approval prior to initiation of the change(s), except where necessary to eliminate apparent immediate hazards to the subject(s). Changes made to eliminate apparent immediate hazards to subjects must be reported to the IRB within 24 hours.
- You must report all adverse and/or unanticipated event(s) that occur during the course of this study to IRB via eSubmission in accordance with IRB Policy.
- Use only IRB-approved copies of your consent form(s), questionnaire(s), letter(s), advertisement(s), etc. in your study. Do not use expired consent forms.
- You must inform all research staff listed on this study of changes or adverse events that occur.
- IRB's approval is valid until the end date of the performance period indicated above. A reminder for renewal should be e-mailed to you from the IRB 90, 60 and 30 days before this study's approval is scheduled to expire. However, you are responsible for submitting all renewal materials at least eight weeks before expiration regardless of whether or not you receive a reminder notice.
- All IRB policy documents can be found on our website: http://irb.med.nyu.edu/library
- Prior to initiating an IRB-approved study, you must receive written approval from an authorized representative for each site where your study will take place. Key contacts are:

- **Bellevue Hospital**: when Bellevue Hospital is listed as a site where your study can take place, please note that you may have to complete additional work in BHC's Reason system. Bellevue will be contacting you with any additional needed information. For questions on Bellevue Hospital research, please contact *BellevueResearch@bellevue.nychhc.org*
- CTSI - Clinical and Translational Science Institute, NYU School of Medicine [formerly General Clinical Research Center (GCRC)], ctsi@nyumc.org.
- NYU Langone Medical Center (Tisch Hospital/Rusk Institute/Co-op Care/HJD/Perlmutter Cancer Center) site approval is handled for you automatically (as needed) by the Office of Clinical Trials

• The IRB may terminate studies that are not in compliance with NYU Langone Medical Center/School of Medicine Policies & Procedures and the requirements of the Institution's Federal Wide Assurance with the Federal Government. Direct IRB questions, correspondence and forms (e.g., continuing reviews, amendments, adverse events, etc.) to 212-263-4110 or IRB-INFO@nyumc.org.

• Prior to initiating an IRB-approved study, you must receive written approval from an authorized representative of the Office of Clinical Trials. You may contact the Office of Clinical Trials at 212.263.4210 or clinicaltrials@nyumc.org.

NYU SoM IRB operates in accordance with Good Clinical Practices (GCP) and applicable laws and regulations. The NYU SoM IRB Federal Wide Assurance number is 00004952.

Appendix B

Hackensack Meridian Health IRB Approval

EXPEDITED REVIEW APPROVAL

From: Robert Krugman, MD

To: Stephen Van Lew

CC:

Re: Study# Pro2018-0260
Exploring the Prevalence and Relationship of Atypical Sensory Processing Patterns in the Mild Traumatic Brain Injury Population with Quality of Life and Functional Indicators

Study Expiration Date: 7/23/2019
Study Approval Date: 7/24/2018

This is to advise you that the above Study has been presented to the Institutional Review Board for expedited review.

Please be reminded that all modifications to approved projects must be reviewed and approved by the Institutional Review Board before they may be implemented. Any changes to this protocol must be submitted for IRB approval before initiated.

All serious adverse events and unexpected adverse events must be reported to Institutional Review Board within seven days.

Please do not make any changes to the IRB approved consent without approval of the IRB. Only the IRB stamped approved consent should be used.

If your study meets the definition of a qualifying study that meets the FDAAA 801 definition of an "applicable clinical trial", you are responsible for ensuring that the trial has been registered properly on the Clinical Trials.gov website prior to the enrollment of any subject.

"Applicable clinical trials" generally include controlled clinical investigations, other than phase 1 clinical investigations (with one or more arms) of FDA-regulated drugs, biological products, or devices, that meet one of the following conditions:

- The trial has one or more sites in the United States
- The trial is conducted under an FDA investigational new drug application or investigational device exemption
- The trial involves a drug, biologic, or device that is manufactured in the United States or its territories and is exported for research

For complete statutory definitions and more information on the meaning of "applicable clinical trial," see Elaboration of Definitions of Responsible Party and Applicable Clinical Trial (PDF).

HIPAA Authorization is required.

Important news about our email communications.
Hackensack Meridian Health Network has implemented secure messaging services. If you need assistance with retrieving a secure email, please send an e-mail to postmaster@hackensackmeridian.org

Confidentiality Notice:
This e-mail message and any attachments from Hackensack University Medical Center are confidential and for the sole use of the intended recipient. This communication may contain Protected Health Information ("PHI"). PHI is confidential information that may only be used or disclosed in accordance with applicable law. There are penalties under the law for the improper use or further disclosure of PHI. If you are not the intended recipient of this e-mail or the employee or agent responsible for delivering the communication to the intended recipient, then you may not read, copy, distribute or otherwise use or disclose the information contained in this message. If you received this message in error, please notify us by telephone at 551.996.2000 or by e-mail to postmaster@hackensackmeridian.org. Please indicate that you were not the intended recipient, and confirm that you have deleted the original message. Please do not retransmit the contents of the message. Thank you. Hackensack Meridian Health Network is the proud recipient of Quality New Jersey's Governor's Gold Award for Performance Excellence

Copyright © 2016 Hackensack Meridian Health Network