The Impact Of Dietary Intake On Concussion Recovery In Division I Ncaa Athletes

Matthew Richard Frakes

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THE IMPACT OF DIETARY INTAKE ON CONCUSSION RECOVERY IN DIVISION I
NCAA ATHLETES

A Dissertation
presented in partial fulfillment of requirements
for the degree of Doctor of Philosophy
in the Department of Nutrition and Hospitality Management
The University of Mississippi

By
MATTHEW R. FRAKES
May 2020
ABSTRACT

The purpose of this investigation was to observe the relationship between total calorie and macronutrient intake on return to baseline measurement times in concussions collegiate athletes. Forty division I collegiate athletes (19.83 ± 1.01 years) were randomly assigned to a control group (n = 22) or intervention group (n = 18) once diagnosed with a concussion. Macronutrient intake and daily caloric intake was analyzed using the Nutrition Data System for Research. Concussion related symptoms were assessed using the Sports Concussion Assessment Tool. Statistically significant differences were found on the number of days until symptoms reached baseline based on overall average calorie intake. Statistically significant differences were not found on the number of days until symptoms reached baseline based on the nutrition intervention. Meeting overall average calorie needs throughout the course of concussion recovery resulted in a reduced number of days of symptoms (mean = 5.53 ± 3.87, η²=0.50). There was a statistically significant relationship between carbohydrate, protein, fat and number of days until symptoms reached baseline. Throughout the symptomatic period, when carbohydrates reached the average intake of 475.79 ± 161.72 grams per day, the number of days until concussion recovery reduced by almost 4 days for carbohydrates (t = -4.03, p <.001). These findings suggest that meeting overall energy needs and the intake of carbohydrate may shorten symptom duration post-concussion. Further research is warranted to investigate the extent that macronutrient and energy intake have on concussion recovery time.
DEDICATION

I would like to first thank my committee chair and mentor, Dr. Melinda Valliant, who gave me the opportunity to pursue a challenge that will provide myself, my family, and community with an endless amount of opportunities. I would also like to thank the Medical Director/Sports Medicine Physician, Dr. Marshall Crowther, and my committee members for their support, input, encouragement, and time. Those committee members include Dr. Kathy B. Knight, Dr. Hyun-Woo (David) Joung, and Dr. Martha Ann Bass.

I would like to thank my colleagues and professors of the Nutrition & Hospitality Management Department for their support and encouragement throughout this degree program. I would also like to express my gratitude toward the athletic training staff, sports nutritionist, and graduate assistants of the Ole Miss Health and Sports Performance Department for their time and support. Without it, this project would not be possible.

Finally, I would like to thank the love of my life, my wife, Kassandra Frakes, for her support, sacrifice, encouragement, and patience to see me through this degree and project, while being the anchor of our family at home. I would also like to thank everyone that supported me throughout this journey. It would be impossible without their presence and listening ears. This project and degree is dedicated to my family, friends, faith, and community that had an impact on the man I am today, and to the people I love who are no longer with us, especially:

“It was hard, but it was fair”

– my hero and father, Gregory Lee Frakes.
**LIST OF ABBREVIATIONS AND SYMBOLS**

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Full Form</th>
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<tbody>
<tr>
<td>ADHD</td>
<td>Attention Deficit-Hyperactivity Disorder</td>
</tr>
<tr>
<td>ALA</td>
<td>Alpha-Linolenic Acid</td>
</tr>
<tr>
<td>ATC</td>
<td>Athletic Trainer</td>
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<tr>
<td>ATP</td>
<td>Adenosine Triphosphate</td>
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<td>CCI</td>
<td>Controlled Cortical Impact</td>
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<tr>
<td>DHA</td>
<td>Docosahexaenoic Acid</td>
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<tr>
<td>DoD</td>
<td>U.S. Department of Defense</td>
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<tr>
<td>EPA</td>
<td>Eicosapentaenoic Acid</td>
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<tr>
<td>FP</td>
<td>Fluid Percussion</td>
</tr>
<tr>
<td>GADPH</td>
<td>Glyceraldehyde 3-Phosphate Dehydrogenase</td>
</tr>
<tr>
<td>GSC</td>
<td>Graded Symptom Checklist</td>
</tr>
<tr>
<td>ImPACT</td>
<td>Immediate Post-Concussion Assessment and Cognitive Testing</td>
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<tr>
<td>mBESS</td>
<td>Modified Balance Error Scoring System</td>
</tr>
<tr>
<td>MD</td>
<td>Sports Medicine Director/Physician</td>
</tr>
<tr>
<td>MLB</td>
<td>Major League Baseball</td>
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<tr>
<td>MRI</td>
<td>Magnetic Resonance Imaging</td>
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<tr>
<td>mTBI</td>
<td>Mild Traumatic Brain Injuries</td>
</tr>
<tr>
<td>NAD</td>
<td>Nicotinamide Adenine Dinucleotide</td>
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<tr>
<td>NCAAA</td>
<td>National Collegiate Athletic Association</td>
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<tr>
<td>Acronym</td>
<td>Description</td>
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<tr>
<td>NDS-R</td>
<td>Nutrition Data System for Research</td>
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<tr>
<td>NFL</td>
<td>National Football League</td>
</tr>
<tr>
<td>NMDA</td>
<td>N-Methyl-D-Aspartate</td>
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<tr>
<td>NOS</td>
<td>Nitric Oxide Synthase</td>
</tr>
<tr>
<td>PARP</td>
<td>ADP-Ribose Polymerase</td>
</tr>
<tr>
<td>RDN</td>
<td>Sports Registered Dietitian</td>
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<tr>
<td>RMR</td>
<td>Resting Metabolic Rate</td>
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<tr>
<td>SAC</td>
<td>Standardized Assessment of Concussion</td>
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<tr>
<td>SCAT</td>
<td>Sport Concussion Assessment Tool</td>
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<tr>
<td>TBI</td>
<td>Traumatic Brain Injury</td>
</tr>
<tr>
<td>TEE</td>
<td>Total Energy Expenditure</td>
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CHAPTER I
INTRODUCTION

Approximately 1.6 to 3.8 million mild traumatic brain injuries (mTBI) occur in sports in the United States annually and account for five to nine percent of all sports related injuries (Broglio et al., 2014; Harmon et al., 2013; McCrory et al., 2017). Organized sport participation in American football, basketball, soccer, volleyball, and wrestling increases an individual’s risk of experiencing a mTBI or sports related concussion. The brain metabolizes glucose in order to provide energy to neurons for proper neurological functions, but in concussions, axonal injury and synaptic plasticity occur, decreasing the brain’s ability to adequately manage sufficient glucose metabolism and energy function. Athletes have reported “loss of appetite” as a subjective symptom during concussion protocols lasting longer than 7 days (Casson et al., 2011), or during post concussive syndrome (Johnston et al., 2001; Rees & Bellon, 2007). This loss in appetite can potentially result in calorie intakes that are insufficient for adequate recovery. The neurological damage in concussions creates a transient hypermetabolic state that causes hyperglycemia, protein catabolism, and an increased demand for energy in order to start the recovery process and is followed by a hypometabolic state. The state of an “energy crisis” that occurs immediately following impact can last anywhere from 30 minutes to 4 hours in animals (Barkhoudarian et al 2011; Giza & Hovda, 2014).

Glucose, metabolized to generate ATP, provides energy to maintain cell maintenance, including generation of neurotransmitters. Glycogenolysis during physical activity may spare
glucose utilization for neurotransmitters, so that the brain has adequate fuel for neurons to perform critical functions for the brain to recover. Specific sites of the brain, such as the hypothalamus, sense central and peripheral glucose levels in order to regulate glucose metabolism and metabolite distribution. The axonal injury and synaptic plasticity that occurs in mTBI, decreases the brain's ability to manage sufficient glucose and energy production for neuronal functioning (Bergsneider et al., 2001; Cook et al., 2008; Echemendia, 2006; McCrory et al., 2017; Mergenthaler et al., 2013).

Currently, studies have revealed the value of nutrition therapy in supporting concussion management and recovery. Several animal studies report that meeting adequate nutrition support that includes omega 3 fatty acids, calories, glucose, ketones, and creatine may support assessment measurements return to baseline levels (Yoshino et al., 1991; Sullivan et al., 2000; Hua Li et al., 2004; Cook et al., 2008; McConeghy et al., 2012; Harmon et al., 2013; Weil et al., 2014; Oliver et al., 2016a). Studies by Hartl et al., 2008, Cook et al., 2008, and Lucke-Wold et al., 2016, reported that patients who experienced a severe traumatic brain injury had improved recovery outcomes when consuming calories immediately post injury.

Athletes who have had a concussion are at a 2 to 5.8 times higher risk of experiencing another concussion, heightened symptomatology, and subsequent injury due to increased vulnerability of the post-concussed brain (Harmon et al., 2013). Clinical findings have revealed that the first 10 days post injury, present the highest risk of repeated injury (Broglio et al., 2014; McCrory et al., 2017). Athletes who have had a concussion are exposed to repeating a concussion when they do not fully recover to baseline measurements in reported symptoms. Ensuring that the athlete fully recovers to baseline is not only imperative, but will improve
management of cognitive performance, and ensure the safety and overall well-being of the athlete post athletic career.

Research Questions

1. To what extent of the relationship does meeting overall average calorie needs have on concussion recovery of collegiate athletes?

2. To what extent of the relationship does a nutrition intervention containing a carbohydrate supplement have on concussion recovery of collegiate athletes?

3. To what extent of the relationship does macronutrient intake have on concussion recovery of collegiate athletes?
CHAPTER II
REVIEW OF LITERATURE

Metabolic Cascade of Events

Through a series of chemical reactions and reducing equivalents, the Tricarboxylic Acid Cycle (Krebs Cycle) produces ATP during normal metabolism. In the electron transport chain, 1-2% of the oxygen generated from byproducts of free radicals at Complex I is normally released in the respiratory chain for removal. A sports related concussion decreases the availability of these reducing equivalents and increases the production of oxygen and intracellular accumulation of calcium. Due to this metabolic shift, an energy crisis occurs, where the brain requires more energy than is available. This increase in energy demand creates a hypermetabolic state resulting in tissue damage and a massive ion flux in the brain. Within the acute phase of the energy crises (≤ 1 hour) a large release of glutamate from presynaptic terminals disrupts the ionic equilibrium on postsynaptic membranes, contributing to the hypermetabolic and catabolic state observed (Prins et al., 2013). This hypermetabolic and catabolic state increases systemic and cerebral energy requirements as ATP, in order to promote cell firing that is blocked by the neurotoxin tetrodotoxin, that is increased due to the rapid efflux of potassium and uptake of calcium within the mitochondria (Giza & Hovda, 2014; Hovda et al., 1997; Prins et al., 2013).

An increase in catabolism creates a neurometabolic cascade of events, resulting in an increase demand for energy to reestablish homeostasis due to a decrease in cerebral blood flow and mitochondrial dysfunction (Giza & Hovda, 2014; Harmon et al., 2012; Prins et al., 2013). Glucose is shunted toward the pentose phosphate pathway creating lower levels of nicotinamide
adenine dinucleotide (NAD+) for the glyceraldehyde 3-phosphate dehydrogenase (GADPH) step. The decrease in NAD may be due to the enzymatic activation of poly (ADP-ribose) polymerase (PARP), xanthine dehydrogenase, phospholipase A2 and nitric oxide synthase (NOS), in response to the DNA damage (Lewén et al., 2009). This activation generates less pyruvate, less production of ATP, and increases the production of oxidative damage and free radicals, creating the energy crisis. During this energy crisis, restoration of cellular homeostasis is attempted, causing hyperglycolysis where the ATP sodium-potassium ionic pumps, shift into overdrive. This shift creates a further reduction in cerebral blood flow, causing an uncoupling between energy supply and demand, leading to additional stress. This ion flux builds up lactic acid, and increases extracellular glutamate, binding to the ligand-gated receptor, N-methyl-D-aspartate (NMDA), creating a change in membrane ionic balance. (Barkhoudarian et al., 2011; Cook et al., 2008; Hartl et al., 2008; Hovda et al., 1997; Vagnozzi et al., 2013). The additional stress creates free radicals that contribute to prolonged impairments and vulnerability of the brain to risk a repeated energy.

Using animal models, Yoshino et al. (1991) showed that hyperglycolysis, a depletion of energy reserves, and an increase in ADP occurs in order to restore ionic cellular homeostasis. Hyperglycolysis can be observed within the first 8 days of cerebral glucose metabolism after traumatic brain injury (Bergsneider et al., 1997). This indicates the need for immediate glucose intake to compensate for the heightened demand in glucose catabolism that occurs in hyperglycolysis. After the acute phase of hyperglycolysis, a prolonged period of decreased cerebral glucose metabolism is observed. This period of prolonged decrease in cerebral glucose metabolism has been observed in experimental animal models of fluid percussion and controlled cortical impact. Glucose metabolic depression was found to be resolved within 5, 10, or 14 days
after mild, moderate, or severe fluid percussion injury in adult rats (Hovda et al. 1994; Thomas et al., 2000).

Motor dysfunction is dependent upon the severity of the hypermetabolic state and the level of injury of the concussion. Hyperglycemia, protein catabolism, and an increased demand for glucose, stimulates the excess secretion of metabolic hormones, reducing energy storage and increasing the rate of glycolysis. Giza and Hovda (2014) reported within rat models a state of energy crisis occurs due to a Traumatic Brain Injury (TBI) and concluded that the increase in glucose metabolism seen within the energy crisis, happened immediately at the point of the TBI, lasting anywhere from 30 minutes to 4 hours. After 6 hours of a TBI, a state of glucose hypometabolism can occur. In humans, this glucose hypometabolic state has been reported to recover based on the severity of the TBI. Hypometabolism may range from lasting 7-10 days for mild TBI’s, (most commonly seen in sports related concussions) to months after a moderate to severe TBI (Barkhoudarian et al 2011; Giza & Hovda, 2014).

**Concussion Overview**

A concussion is a brain injury induced by biomechanical forces that create a complex pathophysiological process affecting the brain. Torres (2013) conducted a study using an anonymous survey of collegiate athletes and revealed that 25% of total participants indicated sustaining a sports related concussion during their athletic career. Of those, 46% indicated that concussions negatively impacted academic performance, and 64% reported missing practice or competitions due to concussion symptoms (Torres et al., 2013). Seventy to ninety percent of concussions resolve within 14 days (Weil et al., 2014), but 10% of athletes experience prolonged recovery periods past 7-14 days (McCrea et al., 2012). To provide the framework to further
investigate and understand the immediate, intermediate, long term and cumulative effects of concussions and repetitive head impact exposures, the National Collegiate Athletic Association (NCAA) and U.S. Department of Defense (DoD) established the largest ongoing concussion and repetitive head impact research to date, the Concussion Assessment, Research and Education Consortium (CARE Consortium) - which continues to publish findings in peer reviewed medical journals from its participating institutions and leading researchers (NCAA Sports Science Institute, 2018). Key findings of the CARE Consortium include: acute effects of concussion and repetitive head impact exposure; further investigation using advanced brain imaging techniques; clinical and neurobiological recovery times; gender differences within concussion diagnoses; the influence of age at first concussion exposure; associations of head impact mechanics and concussion symptoms; concussion test-retest reliability; and sleep duration and concussion recovery (NCAA Sports Science Institute, 2018).

Common complications that may occur following a concussion include: acute impairment of neurologic functions, signs and symptoms (i.e. dizziness, headaches, nausea, vomiting, acute gastroenteritis, sleep disturbances); neuropathological changes that reflect functional disturbances; and graded clinical symptoms that potentially include loss of consciousness (McCrory et al., 2017). Several investigators (Ashbaugh & McGrew, 2016; Giza & Hovda, 2014, & Lewis et al., 2016; Prins et al., 2013), describe concussions as a disruption of neuronal cells, that can cause a continuous and random change of potassium efflux, and influx of sodium and calcium ions due to the release of excitatory neurotransmitters that bind to N-methyl-D-aspartate. Axonal and cell membrane disturbances occur due to sudden impact to the head or body, that transfers forces to brain tissue, causing stretching of brain cells, the release of neurotransmitters across the cell membrane, and an increase in utilization of cerebral glucose, activating pumping
mechanisms to restore ionic homeostasis (Giza & Hovda, 2014; Pabian et. al., 2013). When an athlete sustains a concussion, contusions can occur in the direct, and opposite planes of the brain. The prolonged secondary stage of injury in concussion consists of ischemia, axonal injury, cerebral edema, inflammation and regeneration. Figure 1 shows the overall cascade of events that occurs throughout concussions (Lewis, 2016).

Figure 1 (Lewis, 2016). The overall metabolic cascade of events, where TBI is caused by a transfer of mechanical injury to the brain tissue. Secondary injury occurs over minutes to hours to days to weeks and even months. Numerous metabolic and biochemical cascades cause more damage than the initial insult itself.

In order to restore the ionic imbalance that is created by neuronal depression and the increased extracellular potassium, an increased activity of the sodium-potassium pump occurs. Cellular protein homeostasis depends on the functioning system of protein turnover. The energy impairment that occurs activates proteases and cell death, causing oxidative stress that leads to an accumulation of damaged proteins that affect metabolic enzymes and the ubiquitin-proteasome system (Giza & Hovda, 2014; Omalu et al., 2010; Yoshino et al., 1991). In animals, Yoshino et al (1991) has shown cognitive impairments and the increased accumulation of
astrocytes or astrogliosis, where Mannix et al. (2013) revealed a removal of amyloid proteins or tau not occurring in chronic time points in a study of repetitive mTBI. With these continuous and randomized changes, the acute inflammation in the brain occurs at ~3 hours are preceded by the apoptotic response at ~6 to 48 hours post injury (Shojo et al., 2010). This leads to rapid activation of immune cells of the central nervous system, microglia, and astrocytes. These astrocytes are the most abundant cells in the brain, which stimulates the release of various growth factors, cytokines, and chemokines that regulate inflammation creating neuronal cytotoxicity. The inflammatory response, and release of cytokines and chemokines is a neuroprotective mechanism that initiates the recovery process post injury (Patterson & Holahan, 2012).

In humans, the hyperglycolytic state can last from 3 to 5 days (Prins, 2017). While glucose metabolism initially increases, a state of hypometabolism follows for upwards to 10 days in animals (Namjoshi et al., 2013; Yoshino et al., 2001) and can last up to 1 month or longer in humans (Giza & Hovda, 2014; Namjoshi et al., 2013). Along with a metabolic shift in glucose metabolism in mTBI’s, other physiological events associated with concussions include a decrease in serum magnesium, leaky blood brain barrier, activation of microglia (responsible for neuron protection), disruption of cell function, sympathetic nervous system and autonomic nervous system dysfunction, tauopathy where there is a hyperphosphorylation of tau protein becoming ubiquinated, diffuse axonal injury and shearing, prolonged calcium accumulation, neurofibrillary tangles, and changes in neurotransmitter activity (Lowenstein et al., 1992; McConeghy et al., 2012; Omalu et al., 2010; Takashi et al., 1981).

These complications contribute to physical signs and symptoms such as nausea, fatigue, irritability, anxiety, insomnia, loss of concentration, loss of memory, loss of appetite, ringing in
the ears, visual disturbances, headaches, and balance changes that can potentially resolve in 2 to 7 days (Broglio et. al., 2014; Harmon et. al., 2013; McCrory et. al., 2017). The long-term complications described can be considered post-concussive syndrome, lasting longer than the 2 to 7-day period. Long term complications have been described as fatigability, poor concentration, behavior changes, sleep pattern changes, and mood alterations. Cessation of symptoms are not always equivalent to physiological recovery. With an increase in concussion research and improvement in protocols over time, clearance for return to play may be prematurely made prior to complete physiological recovery (McCrea & Guskiewicz, 2014). Athletes may also be hesitant on reporting symptoms authentically (McCrea et al., 2004; Kutcher et al., 2010).

Physiological symptoms commonly subside within 2 to 7 days after injury. In a study by Askens et al. (2018), athletes who sustained a concussion and were immediately removed from sport activity lost significantly less time, an average of 3 fewer days, and reported less severe symptoms when compared to athletes with delayed removal from activity. Immediate removal from athletic activity had a 39% lower likelihood of requiring 14 days of alleviated symptoms, and 47% lower likelihood of requiring more than 21 days to achieve full medical clearance for sport. Studies have shown that athletes who continue to play while experiencing concussion symptoms had longer symptom duration and severer clinical assessment scores, than those who were immediately removed, increasing recovery time to ≥ 14 days (Asken et al., 2018, Elbin et al., 2016; Heyer et al., 2016). Slower recovery rates from concussion has consistently been predicted by the severity of a person’s acute and subacute symptoms (Iverson et al., 2017).

**Loss of Appetite**
The brain must detect energy stores and match energy intake with expenditure to obtain energy balance. Energy homeostasis, glucose metabolism, and the regulation of appetite is controlled by neuronal connections that affect eating behaviors (Ahima & Antwi, 2008) and regulate metabolic pathways (Cornejo et al., 2016). The regions critical for appetite regulation include the arcuate nucleus of the hypothalamus, prefrontal cerebral cortex, and the amygdala (Guyenet, 2017).

Loss of appetite is a common symptom observed in concussions. Experiencing a loss of appetite contributes to the symptom severity score. Visual, olfactory, and gustatory stimuli stimulate an increase in exocrine and endocrine secretions that impact the desire for the individual to eat to meet energy intake (Ahima & Antwi, 2008). In a review of studies using rat models, mechanical damage of the ventromedial nucleus within the hypothalamus was shown to have an effect on appetite hormones and eating behaviors (Hetherington & Ranson, 1940). The neurological damage observed in concussions may affect the regions critical for regulating the individual’s appetite and dietary intake.

Inadequate Energy Intake

In a review of hormonal activity and brain function, Gomez-Pinalla (2008) explored how nutrients and hormones affect neurological function. Adipogenic hormones (such as leptin and ghrelin) that control satiety and hunger, may be affected in sports related concussions if the regions of the brain that effect appetite are impacted. Leptin controls individual satiety and sends signals to tell an individual to decrease food intake. Leptin receptors can be found in several brain areas, including the hypothalamus, the cerebral cortex, and the hippocampus, revealing that this hormone may mediate energy homeostasis, and the thought process behind food intake.
(Gomez-Pinilla, 2008). The hormone ghrelin acts as an appetite stimulant and is released when the stomach is empty. Within the nucleus of the hypothalamus and the hippocampus, ghrelin is found as an endogenous ligand of the receptor that secretes growth hormone (Druce, 2006; Gomez-Pinilla, 2008). With neuronal damage occurring in concussions, the production of these hormones may be affected, and this may potentially be the cause of loss of appetite, and the inability to consume adequate energy intake.

**Baseline Measurements**

Previous studies have reported that athletes ranging from the high school to elite level may hide concussion symptoms from health care professionals to prevent missing time in training and/or competition (Register-Mihalik et al., 2013; Torres et al., 2013). Hiding concussion symptoms poses a risk that the athlete may return to play before they are fully recovered.

Reported symptoms play a significant role in concussion management assessment. Therefore, baseline and postinjury assessment test are often administered to avoid the underreporting of symptoms by athletes and subsequent risk of further neurological injury. In the National Football League (NFL), Major League Baseball (MLB), and most collegiate sport settings, Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT) has been found to be the most used concussion management testing assessment tool (Broglio et al., 2014; Echemendia, 2006). ImPACT evaluates the severity of concussion’s by establishing a comparison between baseline measurements established prior to concussion and the post injury results of the athlete with the concussion. This provides objective data to inform return to play decisions and decrease the likelihood of athletes hiding or underreporting symptoms. Testing
protocols may utilize assessment tools, such as the Sport Concussion Assessment Tool (SCAT5), that are administered by the sports medicine team including athletic trainers (ATC), sports medicine director/physicians (MD), school nurses, sport coaches, and anyone trained to administer the baseline test (Maroon & Bost, 2011).

SCAT was developed in 2005 by the International Conference of Concussion in Sport and incorporates both symptom evaluation, and cognitive and physical evaluation (Broglio et. al., 2014; Echemendia et. al., 2013; Pabian et al., 2013). The latest revisions of the test were made in 2016 by the International Conference of Concussion in Sport, who changed its name to SCAT5 (McCrory et al., 2017). When a player shows any of the signs or symptoms previously described, a stepwise procedure of the on-field or sideline evaluation should be followed. The player is evaluated by a physician or other licensed healthcare provider on site, where the appropriate disposition of the player must be determined by the treating health care provider. The SCAT5 assessment should be made once first aid issues are addressed. If the player is diagnosed with a concussion, the athlete should not be allowed to return to play the day of the injury (McCrory et al., 2017). After this assessment is made, a return to play protocol is initiated, and at this time, nutrition support can be of value to give the athlete the necessary tools for an accelerated and optimized recovery (Sullivan et al., 2000; Hua Li et al., 2004; Cook et al., 2008; McConeghy et al., 2012; Harmon et al., 2013; Weil et al., 2014; Oliver et al., 2016a).

In baseline assessment testing, accurate interpretations of neurocognitive scores from sports medicine practitioners can be difficult to assess due to the various factors associated with performance outcomes that could lower performance and impact the performance validity (Abeare et al., 2018). Factors that have been associated with significantly influenced neurocognitive test include gender differences (Covassin et al., 2006), previous history of
concussions (Collins et al., 2016), history of learning disabilities such as attention deficit-hyperactivity disorder (ADHD) (Collins et al., 2016; Elbin et al., 2013; Houck et al., 2017), psychological stress disorders such as anxiety and depression (Bailey et al., 2010), education level (Jones et al., 2014), and age and race (Houck et al., 2017).

The return to play decision making process should not begin until the athlete no longer reports concussion-based symptoms, has a normal clinical examination with a physician, and demonstrates pre-injury cognitive performance levels and motor control (Broglio et al., 2014; McCrory et al., 2017). Before the athlete can fully participate in sport, the athlete should participate and graduate through the “Graduation Return to Play Protocol”. This protocol implements stages of physical activity that are separated by at least 24 hours (no activity; light exercise, 70% of age predicted maximal heart rate; sport-specific activities without the threat of contact from others; non-contact training involving others, resistance training; unrestricted training; and return to full participation) (Broglio et al., 2014). In a study by Willer et al (2019) aerobic exercise improved recovery time of concussions in adolescent athletes (aged 13-18 years) when compared to a rest group and placebo-like stretching group, revealing that active concussion treatment may improve concussion recovery outcomes when compared to relative rest recommended in concussion treatment.

**Concussion Reocurrence Risk**

Athletes are 2 to 5.8 times at higher risk of experiencing heightened symptoms, subsequent injury, and another concussion due to increased vulnerability of the brain post-concussion (Broglio et al., 2014; Harmon et al., 2013; Hartl et al., 2008; Maroon et al., 2011). Clinical findings reveal that the first 10 days post initial injury has the highest risk of repeated
injury (Broglio et al., 2014; McCrory et al., 2017). Athletes are exposed to repeating a concussion within the first 10 days post-concussion injury (Giza & Hovda, 2014) when they do not fully recover to baseline measurements, or if individuals have experienced the first concussion before the age of 18 (Scmidt et al., 2018).

In animals, the period of reduced glucose metabolism immediately post-concussion is associated with greater neurological impairments if the second injury took place post full metabolic recovery (Giza & Hovda, 2014). Repeated concussions were associated with worsened signs and symptoms, prolonged recovery time, early onset of age-related memory complications, dementia, persistent headaches, chronic traumatic encephalopathy, sleep disturbances, lowered concentration, and irritability. Returning to play and suffering a second concussion before fully recovering to baseline risk second impact syndrome. Second impact syndrome is the effect of cerebral swelling that can result in a coma and severe neurological decline. Suffering second impact syndrome has led to 30 to 40 deaths over the past decade (Barkhoudarian et al., 2011; Harmon et al., 2013; Torres et al., 2013). Ensuring that the athlete fully recovers to baseline measurements is not only imperative to the athlete’s safety, but will improve management of cognitive performance, and the overall health of the athlete post athletic career.

**Nutrition Therapy**

Several studies have investigated the role of nutritional status in concussion management and recovery in animals, and have found a relationship between improved dietary intake and recovery effects on the acceleration of return to baseline assessment measurements (Yoshino et al., 1991; Sullivan et al., 2000; Hua Li et al., 2004; Cook et al., 2008; McConeghy et al., 2012; Harmon et al., 2013; Weil et al., 2014; Oliver et al., 2016a). Studies by Hartl et al. (2008), Hovda
et al. (2009), Cook et al. (2008), and Lucke-Wold et al. (2016), reported calories should be consumed immediately post traumatic brain injury. The pathophysiological cascade of events may suggest a molecular window for nutrition interventions to support adequate recovery.

Within 30 minutes following injury within the hippocampus, an altered expression within a large number of genes shows an acute response of hyperglycolysis that is maintained 24 hours post-concussion (Hua Li et al., 2004). In an analysis of prospective data by Hartl et al. (2008), 797 patients in 22 centers found that mortality following traumatic brain injury improved per 10 to 25kcal/kg increase in energy intake. Current nutritional recommendations in clinical patients with traumatic brain injuries include, consuming 140% above resting metabolic expenditure in caloric intake within the first 5 days, at least 1.0 to 1.5 g/kg of protein for two weeks post injury, and Omega 3 fatty acid and creatine supplementation for the duration of concussion symptoms (Cook et al., 2008). These recommendations assist with meeting the athletes caloric needs to generate enough ATP and anti-inflammatory properties to protect and assist the brain in to regenerate damaged cells throughout the recovery process.

Omega 3 Fatty Acids

The recommendation for daily dietary fat intake is based on individual needs toward health, body composition, training regimen, type of sport, and position relative to sport. Omega 3 fatty acids are recognized for cardiovascular benefits, neurodevelopment properties and functions, and commonly recommended by Sports Registered Dietitians (RDN) to be consumed via the athlete’s normal diet (Wilson & Madrigal, 2016). The Omega 3 fatty acids include docosahexaenoic acid (DHA), eicosapentaenoic acid (EPA), and alpha-linolenic acid (ALA). The human brain primarily consists of 97% DHA, with high concentrations within phospholipids
of neuronal plasma membranes and synaptic vesicles (Bazan, 2005). A recent study from Anzalone et al. (2019) revealed that 34% of collegiate athletes are in a “high risk” category of sport concussions due to low dietary intake of Omega 3 fatty acids assessed by erythrocyte levels of Omega 3. Current findings have reported the protective properties of Omega 3 fatty acids against plasticity of neurons, impaired learning, and oxidative stress resistance created from a concussion (Ashbaugh & McGrew, 2016; Gomez-Pinilla, 2008; Lucke-Wold et al., 2016; Maroon & Bost, 2011; Wilson & Madrigal, 2016). DHA has shown to promote synapse formation and glutamate receptor expression, reducing excitotoxicity (Lucke-Wold et al., 2016). Supplemental DHA on plasma DHA was studied by Oliver et al. (2016), where 6 grams of Omega 3 fatty acids supplemented per day had a substantial increase on plasma DHA in comparison to the 4 grams per day and 2 grams per day tested groups. This supplemental DHA has also shown to improve cognition and reduce cell death of neural tissue (Lucke-Wold et al., 2016). Experimental data within the athletic population is limited, but Omega 3 fatty acid supplementation can potentially contribute to the recovery process of concussion injuries for athletes.

Protein Requirements

In the position of the Academy of Nutrition and Dietetics (Academy), Dietitians of Canada (DC), and the American College of Sports Medicine (ACSM) for nutrition and athletic performance, protein requirements of 1.2 to 2.0g/kg/day are recommended to support metabolic adaptation, repair, remodeling, and protein turnover in athletes (Thomas et al., 2016). In healthy athletic populations, supplying greater amounts of protein between 1.6–2.4 g protein/kg/day may improve the preservation of lean body mass and muscle protein synthetic rates when energy
intake is limited (Hector & Phillips, 2018), where protein doses should be evenly distributed every 3 to 4 hours with 20 to 40 grams per ingestion period across the day (Kerksick et al., 2018). The pathogenic process related to the secondary phase of recovery following concussions include neuroinflammation, increased excitatory amino acids, free radicals and neuronal damage (Barrett et al., 2014). Meeting protein requirements can potentially be a factor in the support of these complications described. Further research is warranted on the implementation, timing, and observation of protein intake and its effect on concussion recovery in the athletic population.

During the energy crises of concussions, creatine may theoretically improve brain function by providing energy during times of accelerated ATP usage. Creatine supplementation may protect the brain against acute physiological symptoms experienced within concussions, while facilitating recovery (Dolan et al., 2018; Kreider, 2003; Sullivan et al., 2000). Experimental data in humans is limited but reveals that the supplementation of creatine may potentially improve cognition, communication, behavior, and reductions in headaches, dizziness, and fatigue in children with traumatic brain injuries (Sakellaris, et al., 2006; Sakellaris, et al., 2008). Dietary intake of sources high in creatine may pose similar benefits in concussion recovery as those seen in supplemental creatine ingestion.

Carbohydrates: Utilization and Restoration

During times of stress, hormones are released to mobilize energy production and utilization. The pancreas is stimulated to release glucagon, where glucocorticoids, glucagon, and the sympathetic nervous system raises blood levels of glucose. Adequate carbohydrate consumption assures sufficient glucose levels in order for the brain to have adequate fuel and activate regions of the brain that can enhance performance when carbohydrates are present in the
mouth (Chambers et al., 2009; Gant, Stinear, & Byblow, 2010; Turner et al., 2014). With carbohydrate consumption, muscle and liver glycogen stores are replenished. In a review by Obel et al. (2012), astrocytes within the brain store glycogen. The glycogen reserved within astrocytes are primarily utilized for functions such as synaptic activity and memory formation during hypoglycemia or ischemia. Glycogen available within muscle allow for glycogenolysis for the athlete to have sufficient energy to utilize during training and competition. Sufficient energy storage and utilization that matches the intensity and duration of the athlete’s specific sport demands can prevent injury and muscle degradation for the overall health and recovery of the athlete.

The brain utilizes an abundant amount of calories and glucose in relation to the rest of the body in order to transfer energy into neurons to properly function. The metabolism of glucose generating ATP, provides energy for the brain to maintain cell maintenance and generation of neurotransmitters. Glycogenolysis during physical activity spares glucose utilization for neurotransmitters, so that the brain has adequate fuel for neurons to provide critical functions. Specific sites of the brain, such as the hypothalamus, sense central and peripheral glucose levels in order to regulate glucose metabolism and metabolite distribution. Axonal injury and synaptic plasticity that occur in concussions, decreases the brain’s ability to manage sufficient glucose and energy production for neuronal functioning (Bergsneider et al., 2001; Cook et al., 2008; Echemendia, 2006; Gomez-Pinalla, 2008; Mergenthaler et al., 2013; McCrory et al., 2017; Shanley et al., 2001; Venyman et al., 2006).

**Dietary Assessment**
Assessing the dietary intake of athletes is important to measure the overall nutrient and calorie intake contributing to concussion recovery. Various dietary assessments and analyses are utilized by RDN’s to observe and assess the dietary intake of athletes. Some of the assessment tools utilized to measure dietary intake include dietary record (DR) and mobile phone image based dietary assessment. The DR method is considered the “gold standard” of dietary methods utilized within research. This method is used as a reference tool in calibration or validation studies and provides the ability to be filled in by another individual other than the RDN. Mobile phone images can assist with the accuracy of recording dietary intake by capturing food and meal images. This method can potentially detect missing data when comparing DR assessments with camera and mobile telephone technology during real time for individuals with disabilities, or in the case of the subject matter, cognitive impairment due to injury (Ortega et al., 2015; Ptomey et. al., 2015). The Nutrition Data System for Research (NDS-R) is used to assess dietary intake, by calculating the amount of nutrients from the foods collected within the dietary assessment tool used to measure dietary intake. The combination of these methods can reduce recall bias by improving the quality of data and compliance of the participants (Martin et. al., 2012; Rollo et al., 2015). An assigned support staff member/athlete/guardian can assist with recording these images when the concussed athlete is not cleared to utilize electronic devices.

There is growing evidence to show the effects of nutrition intervention and supplementation on concussion recovery; however, the direct cause and to what extent these effects have is yet to be determined. Furthermore, in the athletic setting, there is insufficient data regarding the utilization of carbohydrate supplementation, dietary assessment, and the interaction of overall dietary intake and on return to baseline measurements from pre and post-concussion assessments. The purpose of this investigation was to implement a nutrition intervention,
including a carbohydrate supplement, at the time of concussion diagnosis or suspicion of concussion. Additionally, total calorie and dietary intake related to return to baseline measurements from pre and post-concussion assessments was observed. The goal of this study is to contribute to the limited literature on post-concussion recovery and the impact of overall energy intake on return to baseline recovery rates.
CHAPTER III
METHODS

Participants

Participants included 40 division I collegiate athletes who were 18 years of age or older from a southeastern university in the United States (mean age = 19.83, SD = 1.01). Participants were randomly assigned to a control (n = 22) group or intervention group (n = 18) once diagnosed with a concussion. This study was approved by the University of Mississippi Institutional Review Board protocol number 19-005.

Procedure

Pre-participation consent forms were given during preseason physical screenings or preseason team meetings [Appendix A]. Athletes suspected of or diagnosed with concussion were referred to the attending team ATC to follow injury protocol. The athlete was then referred to the team RDN. The RDN met with the athlete to provide the supplement. If the injury occurred out of town or if the RDN was not available to administer the supplement within the initial serving window, the attending team ATC followed the supplement protocol and instructions [Appendix C]. The RDN met with the athlete daily in the training room when the athlete was in for the physician assessment until return to baseline was achieved. One of every 2 athletes with a concussion or suspicion of concussion was assigned to the intervention group, the
others were assigned to the control group. For the Intervention group: single serving packet(s) (26 grams) of a cornstarch based carbohydrate supplement were provided at immediate impact of the suspected concussion or within 30-60 minutes of diagnosis, every other hour within the first 4 hours the day of the diagnosis, and twice per day until the athlete returned to baseline from pre-concussion cognitive assessment test.

**Measures**

The MD and RDN determined the best mode of dietary intake procedure. For both the control and intervention groups, the primary investigator and team RDN, recorded daily dietary intake using 1 of 3 methods depending on recommendations of the MD regarding exposure to electronic devices: mobile phone image based dietary assessment by assigning a roommate/teammate to send a picture of each meal/snack consumed daily, along with times of dietary consumption during concussion protocol if the participant was not cleared to use mobile devices (Martin et al. 2009); met with the student athlete(s) in the on-campus dining facility daily during main meal times (i.e. breakfast/lunch/dinner); or met with the participants during daily injury and symptomology assessment with the team ATC.

Estimated dietary intake for all participants was analyzed using the NDS-R by a trained investigator and RDN of the study. The results from the NDS-R was compared to the results of estimated energy needs using the Nelson equation to determine resting metabolic rate (RMR) (Nelson, Weinsier, Long & Schutz, 1992). To determine total energy expenditure (TEE), an activity factor of 2.07 was multiplied to RMR. This activity factor corresponds to that of a very active population. RMR was expressed as kilocalorie per day (kcal/day) and calculated (RMR = 25.80 x Fat-free mass (kg) + 4.04 x Fat mass (kg)).
Concussion related symptoms were self-reported and selected on the Sport Concussion Assessment Tool (SCAT5) symptom evaluation form by the participating athlete during daily assessments with the team ATC [Appendix B] (Echemendia, et al., 2017). The SCAT5 is a concise and relevant sideline clinical concussion evaluation tool that includes a Graded Symptom Checklist (GSC); the Standardized Assessment of Concussion (SAC); and the 6 surface conditions of the Modified Balance Error Scoring System (mBESS). The GSC assesses 22 symptoms commonly associated with concussion. A 7-point Likert scale is used to rate each symptom as 0 for not present, 1 to 2 for mild, 3 to 4 for moderate, and 5 to 6 being severe. Including a symptom evaluation form such as the Sport Concussion Assessment Tool (SCAT5), improves the model’s discrimination ability when using regression models to combine multiple assessments validity and accuracy (Garcia et al., 2018).

**Statistical Analysis**

A 2 x 2 factorial analysis of variance was conducted on number of days until symptoms reach baseline based on daily average calorie intake and comparing the control and intervention groups of collegiate athletes at a division I southeastern university. A multiple regression analysis was conducted on the number of days until symptoms reached baseline based on daily macronutrient intake of carbohydrate, protein, and fat of collegiate athletes at a division I southeastern university. All statistics were run in SPSS version 26 (IBM, Chicago, IL). An a priori alpha level of 0.05 was used in all analysis.
CHAPTER IV

MANUSCRIPT I
POSITIVE ENERGY BALANCE SIGNIFICANTLY CONTRIBUTES TO RECOVERY FROM CONCUSSION IN COLLEGIATE ATHLETES

To be submitted to the Journal of the International Society of Sports Nutrition
BACKGROUND

Mild traumatic brain injuries or sports related concussions are injuries that can occur in team sports such as American football, basketball, soccer, volleyball, and wrestling. Studies by Hartl et al., 2008, Cook et al., 2008, and Lucke-Wold et al., 2016, have shown that subjects who have experienced a traumatic brain injury, need to consume calories immediately post injury. In hospitalized severe traumatic brain injuries, energy intakes in the range of 25–50 kcal/kg per day are generally recommended (Cerra et al., 1997; Bratton et al., 2007; McClave et al., 2009). Athletes are 2 to 5.8 times at higher risk of being exposed to a repeated concussion, experiencing heightened symptoms, and subsequent injury when they do not fully recover to baseline measurements post-concussion (Harmon et al., 2013).

In concussions, axonal injury and synaptic plasticity creates a metabolic cascade of events. The brains ability to adequately manage and utilize sufficient glucose and energy metabolized for proper neurological function decreases due to the decrease in cerebral blood flow as the demand for glucose metabolism and uptake increase. The neurological damage in concussions create a hypermetabolic state leading to, hyperglycemia, accelerated protein catabolism, and an increased demand for energy in order to start the recovery process. In animals, Yoshino et al. (1991) reported that hyperglycolysis and an increase in adenosine diphosphate occurs in order to restore ionic cellular homeostasis. In humans, this hyperglycolytic state last up to 3 to 5 days (Prins, 2017). During the hypermetabolic state, an “energy crises” is observed immediately following impact of concussions and can last anywhere between 30 minutes to 4 hours (Barkhoudarian et al., 2011; Giza & Hovda, 2014). The hypometabolic state
following can last throughout the duration of the recovery process, lasting longer than 10 days in animals and 1 month or longer in humans (Giza & Hovda, 2014; Namioshi et al., 2013).

These complications contribute to physical signs and symptoms such as nausea, fatigue, irritability, anxiety, insomnia, loss of concentration, loss of memory, loss of appetite, ringing in the ears, visual disturbances, headaches, and alterations to balance that can potentially resolve in 2 to 7 days (Broglio et. al., 2014; Harmon et. al., 2013; McCrory et. al., 2017). These symptoms can decrease calorie intake below what is needed for adequate recovery. The inability to meet calorie needs decreases the overall energy consumed that is needed for cell maintenance and neurotransmission for neurons to initiate proper brain function and recovery. Slower recovery rates from concussion has consistently been predicted by the severity of a person’s acute and subacute symptoms (Iverson et al., 2017).

There is growing evidence to show the effects of nutrition therapy and supplementation on concussion recovery (Ashbaugh & McGrew, 2016; Oliver et al., 2018); however, the direct cause and to what extent these nutrition interventions have is yet to be determined. The purpose of this investigation was to observe the extent of the relationship total calorie intake and a carbohydrate-based nutrition intervention has on return to baseline measurements in concussions.

METHODS

Participants

Participants included 40 division I collegiate athletes who were at least 18 years of age and at a southeastern university in the United States (mean age = 19.83, SD = 1.01). After being diagnosed with a concussion, participants were randomly assigned to a control (n = 22) group or intervention group (n = 18). This study was approved by the University of Mississippi
Institutional Review Board protocol number 19-005. Pre-participation consent forms were given during preseason physical screenings or preseason team meetings.

Procedures

Athletes diagnosed or suspected of a concussion were referred to the attending team athletic trainer (ATC) to follow injury protocol. The athlete was then referred to the team Sports Dietitian (RDN). The RDN met with the athlete to provide the supplement. If the injury occurred out of town or if the RDN was not available to administer the supplement within the initial serving window, the attending team ATC followed the supplement protocol and instructions. The RDN met with the athlete daily to record dietary intake until the athlete was asymptomatic. One of every two athletes with a concussion or suspicion of concussion was assigned to the intervention group; the others were assigned to the control group. For the Intervention group: single serving packet(s) (26 grams) of a cornstarch based carbohydrate supplement was provided at immediate impact of suspected concussion or within 30-60 minutes of diagnosis, every other hour within the first four hours the day of the diagnosis, and twice per day until the athlete returns to baseline from pre-concussion cognitive assessment test.

Measures

Concussion related symptoms were self-reported and selected on the Sport Concussion Assessment Tool (SCAT5) by the athlete during daily visits with the team ATC (Echemendia, et al., 2017). The SCAT5 is a concise and relevant sideline clinical concussion evaluation tool that includes a Graded Symptom Checklist (GSC); the Standardized Assessment of Concussion (SAC); and the 6 surface conditions of the Modified Balance Error Scoring System (mBESS).
The GSC assesses 22 symptoms commonly associated with concussion. A 7-point Likert scale is used to rate each symptom as 0 for not present, 1 to 2 for mild, 3 to 4 for moderate, and 5 to 6 being severe. Including a symptom evaluation form such as the Sport Concussion Assessment Tool (SCAT5), improves the model’s discrimination ability when using regression models to combine multiple assessments validity and accuracy (Garcia et al., 2018).

Estimated calorie intake for all participants was analyzed using the Nutrition Data System for Research (NDS-R) by an RDN trained to use NDS-R. A trained research associate also analyzed the dietary intake using NDS-R while under the supervision of the trained investigator. The trained investigator provided clear instructions to support consistency and reliability in comparing estimated calorie intake assessments. The results from the NDS-R was compared to the results of estimated energy needs using the Nelson equation to determine resting metabolic rate (RMR). (Nelson, Weinsier, Long & Schutz, 1992). To determine total energy expenditure (TEE), an activity factor of 2.07 was multiplied to RMR. This activity factor corresponds to that of a very active population. RMR was expressed as kilocalorie per day (kcal/day) and calculated (RMR = 25.80 x Fat-free mass (kg) + 4.04 x Fat mass (kg)).

The Sports Medicine Director (MD) and RDN determined the best mode of dietary intake procedure. For both the control and intervention groups, the primary investigator or RDN recorded daily dietary intake using 1 of 3 methods depending on recommendations of the MD regarding exposure to electronic devices: mobile phone image based dietary assessment by assigning a roommate/teammate/guardian to send a picture of each meal/snack consumed daily, along with times of dietary consumption during concussion protocol if the participant was not cleared to use mobile devices (Martin et al. 2009); met with student athlete(s) in the On-Campus
dining facility daily during main meal times (i.e. breakfast/lunch/dinner); or met with participants during daily injury and symptomology assessment with the team ATC.

Statistical Analyses

A 2 x 2 factorial analysis of variance was conducted on number of days until asymptomatic based on daily average calorie intake (overall avg calories met; yes =1, no=0) and comparing the control and intervention groups (control/intervention; control=0, intervention=1). All statistics were run in SPSS version 26 (IBM, Chicago, IL). An a priori alpha level of 0.05 was used in all analysis. Shapiro-Wilks was used to test for normality assumption. Levene’s Test of Equality of Error Variances was used to test for homogeneity of variance assumption. Cohen (1973) suggest using $\eta^2$ to determine effect sizes and interpreted using the criteria of small (.01), moderate (.06), large (.14). Thus, the total number of participants used in analysis for the total number of days until asymptomatic was forty (n = 40). Given the sample size of n = 40, statistical significance would be detected for large effect sizes, $\eta^2 > .17$.

RESULTS

A 2 x 2 factorial analysis of variance was conducted on the number of days until the subject was asymptomatic and overall average calories met throughout the duration of the concussion protocol. Descriptive statistics are reported in Table 1. An alpha level of .05 was considered significant. The control and intervention group were normally distributed. Overall average calories met was also normally distributed between the overall calories being met and overall calories not being met group. Variances were homogeneous, $F_{Levene} (3, 36) = 1.91, p = .15.$
There was not a statistically significant interaction between the control/intervention and overall avg calories met, $F(1,36) = .17, p = .69$. Statistically significant differences were found in number of days until asymptomatic between overall avg calories met (yes or no), $F(1,36) = 36.74, p<.001$. Subjects who met overall average calories improved number of days until asymptomatic (see Table 1). A large effect size was noted $\eta^2=.50$ indicating a strong degree of practical significance. Statistical significant differences were not found in number of days until symptoms reached baseline between the control/intervention group, $F(1,36) = .37, p = .54$. A small effect size was noted $\eta^2 = .01$ indicating a weak degree of practical significance.

**Table 1**

*Means and Standard Deviations for Number of Days Until Asymptomatic by Overall Calories Met and Experiment*

<table>
<thead>
<tr>
<th>Overall Avg Calories Met</th>
<th>Control</th>
<th>Intervention</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$N$</td>
<td>$M$</td>
<td>$SD$</td>
</tr>
<tr>
<td>Yes</td>
<td>10</td>
<td>3.10</td>
<td>1.37</td>
</tr>
<tr>
<td>No</td>
<td>12</td>
<td>8.92</td>
<td>3.75</td>
</tr>
</tbody>
</table>
### Table 2

*Analysis of Variance for Number of Days Until Asymptomatic*

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
<th>$p^{\eta^2}$</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control/Intervention (E)</td>
<td>1</td>
<td>.37</td>
<td>.01</td>
<td>.54</td>
</tr>
<tr>
<td>Overall Avg Calories Met (Kcal)</td>
<td>1</td>
<td>36.72</td>
<td>.50</td>
<td>&lt;.001*</td>
</tr>
<tr>
<td>E X Kcal</td>
<td>1</td>
<td>.17</td>
<td>.00</td>
<td>.68</td>
</tr>
<tr>
<td>S within group error</td>
<td>36</td>
<td>(7.46)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. The value enclosed in parentheses is the mean square error (MSw). S=Subjects, * Significant effect on number of days until asymptomatic, $p < .05$

### Table 3

*Multiple Comparisons for Number of Days Until Asymptomatic Among Overall Avg Calories Met & Treatment*

<table>
<thead>
<tr>
<th>Independent Variable</th>
<th>$\Delta M$</th>
<th>SE $\Delta M$</th>
<th>95% CI for $\Delta M$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes-No</td>
<td>-5.45*</td>
<td>.90</td>
<td>-7.27  -3.63</td>
</tr>
<tr>
<td>Control-Intervention</td>
<td>.55</td>
<td>.90</td>
<td>-1.27  2.37</td>
</tr>
</tbody>
</table>

Note. $\Delta M$ = Mean difference. SE$\Delta M$ = Standard error $\Delta M$, $p < .05$
* Significant effect on number of days until asymptomatic, $p < .05$
DISCUSSION

The findings of this investigation suggest that meeting overall energy needs may shorten the length of time symptoms are present post-concussion. Meeting overall average calories ($\Delta M = -5.45, p<.001$) throughout the course of concussion recovery resulted in a reduced number of days by an average of 5 days ($\eta^2=0.50$). Although symptom duration overall had a duration of mean $5.53 \pm 3.87$ days, full clinical recovery still needs to be further investigated. In an analysis of prospective data by Hartl et al. (2008), 797 Patients in in 22 centers found that mortality following traumatic brain injury improved per 10 kcal/kg to 25 kcal/kg increase in nutrition support by the Brain Trauma Foundation. Seventy to ninety percent of concussions resolved within 10-14 days (Weil et al., 2014), where ten percent of athletes experience prolonged recovery periods past 14 days (McCrea et al., 2012).

In a preliminary study, concussed adolescent athletes were observed consuming fewer calories (Bernitt et al., 2017). The loss of appetite following a concussion can prolong recovery time and heighten symptoms of concussions by not allowing the athlete to consume sufficient energy needed to support recovery. Similar to the findings in Frakes et al., 2019, the experimental group did not have a significant decrease in the number of days of symptom alleviation ($p=0.57$), but an average decrease in number of days of symptoms experienced was observed. Even though the results of the experimental group were not statistically significant, this small difference can support nutrition intervention implementation in concussion injury protocol. Optimizing and supporting concussion injury recovery protocols benefits the athlete by integrating a multidisciplinary plan of care, support full neurometabolic recovery, decrease time to return to full participation, improves the management of cognitive performance, and improves the future well-being of the athlete post athletic career.
There is conflicting evidence revealing that energy or calorie restriction can inhibit the recovery of concussions in animal models. Liu et al (2017) observed how calorie restriction diets may ameliorate cognitive dysfunction following concussions in mice by promoting autophagy and inhibiting astrogliosis for 30 days. Further animal models have revealed that calorie restriction correlated with improved recovery outcomes from mTBI when compared to a high fat diet (Mychasiuk et al., 2015), and/or standard diet (Rich et al., 2010; Mychasiuk et al., 2015).

Although the findings of calorie restriction may show positive results of concussion recovery in animal models, the research is lacking in humans, and challenges will present when implementing similar protocols in athletes. Energy is essential in supporting basal metabolism and digestion processes. Energy demands increase as physical activity and psychological stress increases. The methodology used in these animal models may pose an ethical challenge and health risk to the athletic population due to the increase demand of calories needed for performance and recovery. Calorie restriction diets decreases overall food intake, which can risk decreases in lean muscle mass, nutrient deficiency, increased rates of fatigue, decreased sex hormone production, reduced bone formation and increased resorption, and lowered immune system integrity. Further research is warranted in the context of implementing a calorie restriction protocol to mitigate symptomology without conflicting with the athlete’s needs for performance, recovery, and academic success.

Dietary intake can have day to day variability, which may pose a risk of inaccuracy, underreporting, and limitations to the dietary intake data collected. Limitations that may be seen in dietary methods include: unusual dietary intake not natural to the student-athlete, not reporting actual intake and altering dietary behaviors due to the knowledge and amount of foods being recorded, risk of misinformation for athletes with lower literacy levels and language barriers, and
risk of participant and observer burden based on time, resources, and repeating measures. These limitations can increase sources of error between the athletes and the researchers collecting the dietary intake. Sources of error based on the respondents include: motivation, memory, communication skills, over or underestimation, personal characteristics (age, sex, obesity); where the sources of error from the researchers include: insufficient training to instruct individuals on completing the dietary record sufficiently, not thoroughly reviewing the foods collected within the dietary record, and the risk of tabulation and food codification (Ortega, Perez-Rodrigo, & Lopez-Sobaler, 2015).

Consideration with variability in accuracy and validity in this investigation should be noted. The timing of the treatment and dietary assessment may vary due to the travel within the competitive schedule, where access to the RDN may be limited in recording the dietary assessment accurately. Symptom reporting is also a subjective and non-normally distributed measurement reported. The recorded athlete reported symptom evaluation may contain outdated or inaccurate measurements toward full clinical recovery, especially if baseline measurements are not reassessed annually (Broglio et al., 2018). Neurometabolic recovery needs to be further considered and researched when paired with symptom evaluation to assist with the accuracy and validity of clinical recovery. Athletes may potentially be cleared to participate before concussion symptoms take place considering symptomology possibly not occurring until after the symptom assessment is completed in concussions or being hesitant on reporting symptoms authentically (McCrea et al., 2004; Kutcher et al., 2010). This conflicts with the clinical definition of recovery from concussion due to a ceiling effect on the SAC assessment used in the SCAT5 (Echemendia et al., 2017).
Other variables not measured within this study can affect the recovery time or time to being asymptomatic including post injury sleep declines (Hoffman et al., 2017, Howell et al., 2019), dehydration (Weber et al., 2013), gender (Bock et al., 2015; Miller et al., 2016; Zemek et al., 2016; Eisenberg et al., 2013), injury severity (Iverson et al., 2017), and pre-existing neurodevelopmental disorders and health complications such as ADHD, learning disabilities, substance abuse, history of headaches and migraines (Iverson et al., 2016; Iverson et al., 2017). Further research is warranted not only on the previously listed variables, but to study the extent of the relationship energy intake has on concussion recovery within the athletic population to guide nutrition treatment and intervention decisions post injury.

CONCLUSION

The current study shows that consuming adequate energy supports sports related concussion recovery in relation to symptom duration. The experimental group did not reveal a significant effect on symptom duration, but this small difference in the reduced number of days in symptoms can support nutrition intervention development and implementation in concussion injury protocol. While symptoms are a subjective measurement reflecting various phases in the metabolic cascade of events, it is important to consider the relationship and role energy intake has on the recovery of neurological and physiological trauma. Health and athletic performance practitioners make objective return to play decisions by observing symptoms. This allows the athlete to successfully navigate through return to play graduation protocols for full participation clearance. These considerations decrease the likelihood for athletes to hide or underreport symptoms to ensure full recovery prior to the athlete returning to sport. The qualified sports dietitian/nutritionist on staff involved in athlete’s concussion recovery and return to play
graduation protocols can support post-concussion recovery by assisting with the analysis, assessment, and promotion of dietary intake to optimize the overall health and welfare of the athlete.
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HIGH CARBOHYDRATE INTAKE SIGNIFICANTLY RESULTED IN SHORTER RECOVERY TIME FROM CONCUSSIONS IN COLLEGIATE ATHLETES: A RANDOMIZED CONTROL TRIAL

To be submitted to the Journal of Head Trauma Rehabilitation
BACKGROUND

A concussion or mild traumatic brain injury is induced by biomechanical forces that create complex pathophysiological processes affecting the brain. In the United States, approximately 1.6 to 3.8 million sports related concussive injuries occur annually, and account for five to nine percent of all sports related injuries (Broglio et al., 2014; Harmon et al., 2013; McCrory et al., 2017). The brain utilizes glucose to neurons in order to provide energy for proper neurological functions. Athletes have reported “loss of appetite” as a common symptom of concussive diagnoses in concussion lasting longer than 7 days (Casson et al., 2011) or during post concussive syndrome (Johnston et al., 2001; Rees & Bellon, 2007). This loss of appetite can decrease dietary intake below what is needed for adequate recovery.

Carbohydrate consumption provides athletes with sufficient calories and energy in order for the brain to have adequate fuel that can enhance performance. Regions within the brain have been reported to be activated when carbohydrates are present in the mouth (Chambers et al., 2009; Gant, Stinear, & Byblow, 2010; Turner et al., 2014). In a review by Obel et al. (2012) glycogen storage in astrocytes are primarily utilized for neurological functions such as synaptic activity and memory function during hypoglycemia or ischemia. The axonal injury and synaptic plasticity followed by hypoglycemia and ischemia decreases the brains ability to manage sufficient glucose and energy production for neuronal functioning in concussions (Bergsneider et al., 2001; Cook et al., 2008; Echemendia, 2006; Gomez-Pinalla,
The Academy of Nutrition and Dietetics (Academy), Dietitians of Canada (DC), and the American College of Sports Medicine (ACSM), nutrition and athletic performance position paper recommends 1.2 to 2.0g/kg per day of protein to support metabolic adaptation, repair, remodeling, and for protein turnover range from for athletes (Thomas et al., 2016). Supplying greater amounts of protein between 1.6–2.4 g protein/kg/day will improve the preservation of lean body mass and muscle protein synthetic rates when energy intake is limited (Hector & Phillips, 2018), where ingestion of protein should be evenly distributed every 3 to 4 hours across the day with 20-40 grams per ingestion period (Kerksick et al., 2018). Pathogenic processes related to the secondary phase of recovery following concussions includes neuroinflammation, increased excitatory amino acids, free radical production and neuronal damage (Barrett et al., 2014; Tipton et al., 2015). Meeting protein requirements can potentially be a factor in the support of the complications described. In the athletic population, further research is warranted on the implementation, timing, and observation of protein intake and its effect on concussion recovery.

Recommendations of dietary fat intake for athletes are similar to those of who are normally healthy and do not participate in elite and collegiate athletics. Dietary fat intake supports hormone regulation, absorption of fat-soluble vitamins, replenishment of intramuscular tracylglycerol stores, fuel substrate utilization, maintenance of energy balance and storage, consumption of adequate amounts of essential fatty acids, thermoregulation, and essential cell membrane structure. The essential fatty acids that are consistently researched for its relationship and support in concussion recovery are Omega 3 fatty acids. Omega 3 fatty acids consist of docosahexaenoic acid (DHA), eicosapentaenoic acid (EPA), and alpha-linolenic acid (ALA).
The human brain primarily consists of 97% DHA, with high concentrations within phospholipids of neuronal plasma membranes and synaptic vesicles (Bazan, 2005). A recent study from Anzalone et al., 2019 has revealed that 34% of collegiate athletes are in a “high risk” category of sport concussions due to low dietary intake of Omega 3 fatty acids assessed by erythrocyte levels of Omega-3. Omega 3 fatty acids can protect against plasticity of neurons, impaired learning, and provide resistance on oxidative stress created from a concussion (Ashbaugh & McGrew, 2016; Gomez-Pinilla, 2008; Lucke-Wold et al., 2016; Maroon & Bost, 2011; Wilson & Madrigal, 2016). In concussions, dietary intakes high in polyunsaturated fatty acids can make the brain susceptible to lipid peroxidation in the presence of increased reactive oxygen species, and diets high in saturated fat can increase free radical formation that exacerbate neurological consequences of the injury (Oliver et al., 2018). The amount and type of dietary fat recommended for daily intake is based upon the individual’s health, body composition, training regimen, type of sport, and position relative to sport.

In animals, nutritional status in concussion management and recovery have found a significant relationship between dietary intake and return to baseline time in concussion assessment measurements (Yoshino et al., 1991; Sullivan et al., 2000; Hua Li et al., 2004; Cook et al., 2008; McConeghy et al., 2012; Harmon et al., 2013; Weil et al., 2014; Oliver et al., 2016a). In a review by Cook et al. (2008) nutritional considerations and recommendations for clinical patients with traumatic brain injuries include consuming 140% above resting metabolic expenditure in caloric intake within the first 5 days, at least 1.0-1.5 g/kg of protein for two weeks post injury, and Omega 3 fatty acid and creatine supplementation for the duration of concussion symptoms. The purpose of this study is to observe the extent of the relationship macronutrient intake has on return to baseline measurement times in concussions of collegiate athletes.
METHODS

Participants

Participants included 40 division I collegiate athletes who were at least 18 years of age and at a southeastern university in the United States (mean age = 19.83 ± 1.01) Participants were diagnosed with a concussion and randomly assigned in a control (n = 22) group or intervention group (n = 18). This study was approved by the University of Mississippi Institutional Review Board protocol number 19-005.

Procedures

Pre-participation consent forms were given during preseason physical screenings or preseason team meetings. Athletes suspected of or diagnosed with concussion was referred to the Sports Dietitian/Nutritionist (RDN). The RDN or primary investigator met with the athlete daily to record daily dietary intake. The participants were met with daily to record dietary intake until return to baseline was achieved on the SCAT5 assessment.

Measures

Concussion related symptoms were self-reported and selected on the Sport Concussion Assessment Tool (SCAT5) symptom evaluation form by the participating athletes during daily assessments with the team ATC (Echemendia et al., 2017). The SCAT5 is a concise and relevant sideline clinical concussion evaluation tool that includes a Graded Symptom Checklist (GSC); Standardized Assessment of Concussion (SAC); and the 6 surface conditions of the Modified Balance Error Scoring System (mBESS). The GSC assesses 22 symptoms commonly associated with concussion. A 7-point likert scale is used to rate each symptom as 0 for not present, 1 to 2
for mild, 3 to 4 for moderate, and 5 to 6 being severe. Including a symptom evaluation form such as the Sport Concussion Tool (SCAT5), improves the model’s discrimination ability when using regression models to combine multiple assessments validity and accuracy (Garcia et al., 2018). All measures were administered by study personnel (e.g. ATC, RDN, MD) who were fully trained and supervised by the investigators on standardized assessment methods.

The Sports Medicine Director (MD) and RDN determined the best mode for recording dietary intake. The primary investigator and RDN, recorded daily intake using 1 of 3 methods depending on the recommendations of the MD regarding permissibility using electronic devices: mobile phone image based dietary assessment by assigning a roommate/teammate/guardian to send a picture of each meal/snack consumed daily, along with times of dietary consumption during concussion protocol if the participant is not cleared to use mobile devices (Martin et al. 2009); met with student athlete(s) in the On-Campus dining facility daily during main meal times (i.e. breakfast/lunch/dinner); or met with participants during daily injury and symptomology assessment with the team athletic trainer (ATC). Estimated macronutrient intake for all participants were analyzed using Nutrition Data System for Research (NDSR) by a trained investigator and RDN of the study. A trained research associate also analyzed the dietary intake using NDSR while under the supervision of the trained investigator.

Statistical Analyses

A multiple regression analysis was conducted on the number of days until asymptomatic based on daily macronutrient intake of carbohydrate, protein, and fat of collegiate athletes at a division I southeastern university. All statistics were run in SPSS version 26 (IBM, Chicago, IL). An a priori alpha level of 0.05 was used in analysis. Cohen (1992) suggest using $f^2$ to determine
effect sizes and interpreted using the criteria of small (.02), moderate (.15), large (.35). These
effect size values are converted to $R^2$ to measure practical significance with the following
interpretations small (.00-.02), moderate (.02-.13), large (.13-.26). Thus, the total number of
participants used in analysis for the total number of days until asymptomatic was forty (n = 40).

RESULTS

Descriptive statistics are reported in Table 1. Macronutrient intake was normally
distributed. The model of assumptions test for the values of the standardized residuals to be
normally distributed may be violated, but no extreme outliers were evident. Scatterplots were
analyzed, and no curvilinear relationships between the criterion variable and the predictor
variables or heteroscedasticity were evident. There was a statistically significant relationship
between carbohydrate, protein, fat and numbers of days until asymptomatic, $F(3, 36) = 8.16, p < .001$. A large effect size was noted with approximately 40% of the variance accounted for in the
model, $R^2 = .41$. Carbohydrate intake was a statistically significant predictor of number of days
until asymptomatic (see Table 2) uniquely accounting for 27% of the variance, indicating a large
effect size. Fat intake was not statistically significant predictor on the number of days until
asymptomatic (see Table 2) uniquely accounting for 7% of the variance, indicating a moderate
effect size. Protein was not significant and uniquely accounted for 0.2% of the variance. Thus, as
single predictor’s, Protein has a small effect and Fat has a moderate effect but are less
meaningful when included in a model with Carbohydrate. Given the sample size of $n = 40$,
statistical significance would be detected for large effect sizes, $R^2 > .30$.  

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Table 1

Correlations between Macronutrient Intake and Number of Days Until Asymptomatic

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Mean</th>
<th>SD</th>
<th>N</th>
<th>Symptoms (# of days)</th>
<th>Carbohydrate</th>
<th>Protein</th>
<th>Fat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms</td>
<td>5.53</td>
<td>3.87</td>
<td>40</td>
<td>---</td>
<td>475.79</td>
<td>161.72</td>
<td>---</td>
</tr>
<tr>
<td>Carbohydrate</td>
<td>475.79</td>
<td>161.72</td>
<td>40</td>
<td>-.58**</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Protein</td>
<td>115.74</td>
<td>36.03</td>
<td>40</td>
<td>-.35</td>
<td>.65</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Fat</td>
<td>91.99</td>
<td>13.05</td>
<td>40</td>
<td>-.10</td>
<td>.55</td>
<td>.56</td>
<td>---</td>
</tr>
</tbody>
</table>

Note. Statistical significance on the number of days until asymptomatic; *p < .05, **p < .001

Table 2

Multiple Regression Results for Number of Days Until Asymptomatic

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE B</th>
<th>β</th>
<th>t</th>
<th>p</th>
<th>sr²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbohydrate</td>
<td>-.02</td>
<td>.00</td>
<td>-.71</td>
<td>-4.03**</td>
<td>.00</td>
<td>.27</td>
</tr>
<tr>
<td>Protein</td>
<td>-.01</td>
<td>.02</td>
<td>-.07</td>
<td>-.41</td>
<td>.68</td>
<td>.00</td>
</tr>
<tr>
<td>Fat</td>
<td>.10</td>
<td>.05</td>
<td>.33</td>
<td>2.04</td>
<td>.05</td>
<td>.07</td>
</tr>
</tbody>
</table>

Note. Statistical significance on the number of days until asymptomatic; *p < .05, **p < .001

DISCUSSION

The findings of this investigation included that the increased intake of carbohydrate and fat potentially effects the length of time collegiate athletes experience symptoms when diagnosed with a concussion. When carbohydrates reached the average intake of 475.79 ± 161.72 grams per
day throughout the symptomatic period, there was a correlation on concussion recovery (number of days until asymptomatic) where symptoms decreased by 4 days \((t = -4.03, p < .001)\).

Evidence is limited in athletic populations, where further research is warranted on carbohydrate consumption and the number of days until the participants were asymptomatic. Axonal injury and synaptic plasticity decreases the brain's ability to manage sufficient glucose and energy production for neuronal functioning (Bergsneider et al., 2001; Cook et al., 2008; Echemendia, 2006; Gomez-Pinilla, 2008; Mergenthaler et al., 2013; McCrory et al., 2017; Shanley et al., 2001; Venyman et al., 2006). Although Fat was not statistically significant, there was a slight inverse relationship between dietary fat intake and the number of days until asymptomatic, where the number of days increased as dietary fat intake increased (Table 2). As previously discussed, the beneficial effects of Omega 3 fatty acids are presented in previous studies (Ashbaugh & McGrew, 2016; Gomez-Pinilla, 2008; Lucke-Wold et al., 2016; Maroon & Bost, 2011; Wilson & Madrigal, 2016), where this study only measured overall dietary fat intake and not the association between Omega 3 fatty acid consumption and concussion recovery.

Although the current findings in the study observed a reduced time to being asymptomatic by meeting adequate carbohydrate intake, it is worth mentioning the extent high fat low carbohydrate diets have on supporting concussion recovery. High fat low carbohydrate diets are composed of 80 to 90 percent of dietary fat, adequate dietary protein, and ≤ 50 grams of carbohydrates (Gasior et al., 2016). Various human models have shown beneficial results when focusing on the reduction of epileptic seizures in high fat low carbohydrate diets (Beniczky et al., 2010; Kossoff and Rho, 2009; Nathan et al., 2009; Porta et al., 2009; Sharma et al., 2009; Villeneuve et al., 2009). Wolahan et al., 2017 observed the impact of intravenous glycemic control on endogenous ketogenesis in severe traumatic brain injuries of 15 patients ages 16 years
and older using a randomized within-patient crossover study. The results revealed that there was a 60% increase in ketones within the tight glycemic control group when compared to the loose glycemic control group (Wolahan et al., 2017). Rodent models suggest low carbohydrate, high fat diets have a significant effect on rodents with TBI by reducing edema and cellular apoptosis (Prins, 2008; Appelberg et al., 2009; Hu et al., 2009). With respect to risk of participant burden on altering dietary intake, the diet may create food restrictions and dietary needs to fall below recommended amounts. Human clinical trials evaluating the role of ketogenic diets and its effect on loss of appetite following concussions are limited. Further research is warranted on the role of high dietary fat intake and its role of concussion recovery in the athlete population.

In this investigation, protein intake had no effect on concussion symptom recovery (see Table 1). The essential amino acids leucine, isoleucine, and valine, also known as branched chain amino acids (BCAAs), have shown to have an effect on the production of neurotransmitters. Previous studies show the influence of BCAA’s and its influence on improved neurological function on patients with TBI (Aquilani et al., 2005; Aquilani et al., 2008; Ott et al., 1988) and children with epilepsy when paired with a ketogenic diet (Evangeliou et al., 2009). Further research is warranted on the effects of protein intake and branched chain amino acids effect on symptomology recovery in concussions.

There is still not a gold standard to fully observing clinical recovery from concussions. In Broglio et al. (2018), the study evaluated 4,874 participants collected from the Concussion Assessment, Research, and Education Consortium, evaluating baseline assessments on separate occasions. The assessment tools evaluated contained less than optimal reliability for commonly used assessment tools including the SCAT and ImPACT used within this study. Athlete reported symptoms during these assessments may not reveal consistent or accurate clinical recovery
definitions used for those diagnosed with concussions if baseline assessments are not redone annually (Broglio et al., 2018; Vagnozzi et al., 2013). Neurometabolic processes may also be hindered due to other nutrients affected such as N-Acetylaspartate, creatine, and choline from concussions (Vagnozzi et al., 2013). Further research is warranted on the extent macronutrient distribution and intake have on concussion recovery to guide the decision making for nutrition treatment and intervention.

CONCLUSION

The current study shows that as carbohydrate consumption increased, the length of time collegiate athletes experience symptoms decreased post-concussion. Although protein and fat did not prove to have a significant impact on time until being asymptomatic, it is important to consider the value protein has on lean muscle preservation and protein turnover, and the value fat has on cell membrane structure and as an alternative fuel source during hypocaloric or low carbohydrate intakes. While symptomology is a subjective measurement, carbohydrates, protein, and fat have various roles on the overall health of the student athlete and has further beneficial effects that warrants further investigation in concussion. Having an expert sports dietitian/nutritionist available on staff is essential in order to assess and analyze pertinent information regarding adequate dietary intake in athletes, can assist with monitoring the subjective symptoms experienced that may affect dietary intake (i.e. loss of appetite, nausea, vomiting, fatigue) and reflect dehydration (i.e. headache). The coordination of care between the MD, ATC, and RDN will optimize the overall health and welfare of the athlete, while fully supporting the recovery of the athlete post-concussion.
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LIST OF APPENDICES
APPENDIX A

INFORMED CONSENT
Consent to Participate in Research

Study Title: Dietary intake and return to baseline in sports related concussions

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☐ By checking this box I certify that I am 18 years of age or older.

The purpose of this study
The goal of this study is to observe if overall dietary intake including the addition of a carbohydrate rich dietary supplement contributes to recovery from sports related concussions.

What you will do for this study
You will be evaluated for a sports related concussion by the team athletic trainer and medical director at the point of suspicion of a concussion. During this time of evaluation, the nutrition intervention will be provided (if applicable). The sports dietitian will meet with you daily until full recovery, where your daily calorie intake will be assessed.

1. One of every two athletes with concussion or suspicion of concussion will be assigned to the intervention group. The others will be assigned to the control group.
2. Depending on the group that you are assigned to, you may consume a carbohydrate based supplement at the time of concussion or suspicion of concussion. This supplement will be consumed until full recovery/full participation has been cleared by the Medical Director. Directions on supplement consumption will be given by the Sports Dietitian during concussion evaluation and assessment.
3. For both groups, the primary investigator and team sports dietitian, will record daily dietary intake using 1 of 3 methods dependent on recommendations of Medical Director regarding need for decrease in stimuli:
a. Assigning a roommate/teammate to send a picture of each meal/snack consumed daily, along with times of dietary consumption during concussion protocol.

b. Meeting in the On-Campus dining facility daily (Grill 1810, Rebel Market, etc.), during main meal times (i.e. breakfast/lunch/dinner); All meals and snacks consumed outside of the dining facility will be recorded.

c. Bring your prepared meals and assess total dietary intake after each boxed lunch is consumed.

Time required for this study
Daily evaluation of recovery of concussion and dietary intake assessment will take ~1.5 hours daily. Administration of the carbohydrate supplement (if applicable) will assist with total dietary intake and the energy demands needed for full recovery from injury. Total time for the study is dependent on the athletes return to baseline recovery time and when the injury is gone. Time for full recovery from a concussion varies drastically and is completely individualized. Clearance to full participation following concussion diagnosis has been found to be anywhere between 7 to 14 days.

Possible risk from your participation
We anticipate there to be no risk to you in this study.

Benefits from your participation
All student athletes with concussion will receive an in-depth nutrition assessment which is occasionally done as part of the protocol but will now be consistent. The addition of the carbohydrate rich supplement may result in accelerated recovery and return to play post-concussion, and decreased chances of developing second impact syndrome or post-concussion syndrome.

Confidentiality
All information in the study will be collected confidentially. At the end of the study, all identifiable information will be removed so that it will not be possible to associate your name with your responses.

Right to Withdraw
You do not have to volunteer for this study, and there is no penalty if you refuse. If you start the study and decide that you do not want to finish, just tell the sports dietitian, medical director, and/or team athletic trainer. Whether or not you participate or withdraw will not affect your current or future relationship with the Department of Nutrition & Hospitality Management, or with the University, and it will not cause you to lose any benefits to which you are entitled.

Protected Health Information
Protected health information is any personal health information which identifies you in some way. The data collected in this study includes: (name, date, phone number, medical record number). A decision to participate in this research means that you agree to the use of your health information for the study described in this form. This information will not be released beyond the purposes of conducting this study. The information collected for this study will be kept until
May 2019. While this study is ongoing you may not have access to the research information, but you may request it after the research is completed.

**IRB Approval**
This study has been reviewed by The University of Mississippi’s Institutional Review Board (IRB). The IRB has determined that this study fulfills the human research subject protections obligations required by state and federal law and University policies. If you have any questions, concerns, or reports regarding your rights as a participant of research, please contact the IRB at (662) 915-7482.

Please ask the researcher if there is anything that is not clear or if you need more information. When all your questions have been answered, you can decide if you want to be in the study or not.

**Statement of Consent**
I have read the above information. I have been given a copy of this form. I have had an opportunity to ask questions, and I have received answers. I consent to participate in the study.

Furthermore, I also affirm that the experimenter explained the study to me and told me about the study’s risks as well as my right to refuse to participate and to withdraw.

Signature of Participant __________________________________________ Date ________________

Printed name of Participant

**NOTE TO PARTICIPANTS: DO NOT SIGN THIS FORM IF THE IRB APPROVAL STAMP ON THE FIRST PAGE HAS EXPIRED**
APPENDIX B

SPORT CONCUSSION ASSESSMENT TOOL (SCAT5) SYMPTOM EVALUATION FORM
Sport Concussion Assessment Tool (SCAT5)
Symptom Evaluation Form

Name: ___________________ Date of Injury: ___________ Sport: ______
Date of Assessment: ___________ Examiner: ___________________

*Please note that athlete should score their self on the following symptoms based on how he/she feels now.

<table>
<thead>
<tr>
<th>SYMPTOM</th>
<th>NONE</th>
<th>MILD</th>
<th>MODERATE</th>
<th>SEVERE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>&quot;Pressure in head&quot;</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Neck Pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Nausea or Vomiting</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Dizziness</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Blurred vision</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Balance problems</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Sensitivity to light</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Sensitivity to noise</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Feeling slowed down</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Feeling like &quot;in a fog&quot;</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>&quot;Don't feel right&quot;</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Difficulty Concentrating</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Difficulty Remembering</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Fatigue or low energy</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Confusion</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Drowsiness</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>More emotional</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Irritability</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Sadness</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Nervous or Anxious</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Trouble falling asleep</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>(if applicable)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Total Number of Symptoms (Maximum possible 22) /22
Symptom Severity Score (Add all scores in table, maximum possible: 22 x 6 = 132) /132

Do the symptoms get worse with physical activity? □ Y □ N
Do the symptoms get worse with mental activity? □ Y □ N

If 100% is feeling perfectly normal, what percent of normal do you feel? _______
If not 100%, why?
_________________________________________________________________________
_________________________________________________________________________
APPENDIX C

DIETARY INTAKE AND SPORTS RELATED CONCUSSION STUDY PROTOCOL

FOR ATC
DIETARY INTAKE AND SPORTS RELATED CONCUSSION STUDY
PROTOCOL FOR ATC

OBJECTIVE

- To implement a nutrition intervention at the time of concussion diagnosis or suspicion of a concussion.

PROTOCOL

- Time of injury (ATC/MD)
  
  i. Team ATC will notify MD (Dr. Marshall Crowther) of MTBI/concussion immediately at time of suspicion/evaluation of concussion. The MD will notify primary investigator (Matt Frakes), where primary investigator will implement nutrition intervention.

  ii. In times of away events or practices where the primary investigator or team RD cannot be present:

     1. ATC will have a trusted assigned support staff member/student implement the nutrition intervention after approval of the MD or primary investigator.

     2. Nutrition intervention instructions are as follows:

        a. Within 30 to 60 minutes of suspicion/evaluation of concussion, the athlete will consume two single serving packets of UCAN in 16-24oz of water

        b. Following the initial consumption, the athlete will then receive one single serving packet of UCAN in 8-12oz of water every other hour within the first 4 hours of MTBI/concussion suspicion/evaluation.

        c. During ATC/MD follow up sessions until full participation clearance: athlete consumes one single serving UCAN packet with 8-12oz of water at the beginning of evaluation session, and one single serving packet with 8-12oz of water at the end of the session/post nutrition assessment.

  iii. Referral to Sports RD within 24 hours of concussion diagnosis

Any questions and/or concerns, please contact the primary investigator, Matthew Frakes via cell (614-216-8211) or email (mfrakes@go.olemiss.edu)
APPENDIX D

RECRUITMENT SCRIPT
Recruitment to Participate Script

We are doing a study to find out if dietary intake can improve concussion recovery time. Your participation in the study will give you a thorough nutritional assessment throughout your recovery protocol, and may result in accelerated recovery time and return to play post-concussion, although we cannot guarantee this. Your participation can help us learn more about concussion treatment.

If you want to participate, you need to sign a consent to participate form. Your sports dietitian will meet with you daily until you are fully recovered. Any known food allergy will exclude you from participation in this study.
VITA

Name: Matthew R. Frakes, PhD, RD, CSCS

Current Position: Sports Nutritionist, University of Louisville

Academic Preparation

a. Prospective Degrees
University of Mississippi, Oxford, Mississippi
Doctor of Philosophy, 2020; Field of Study: Nutrition and Hospitality Management
Major: Sports Nutrition

b. Degrees Completed
Bowling Green State University, Bowling Green, Ohio
Certificate (Registered Dietitian-Eligible Status), 2015; Field of Study: Dietetics (Distance Dietetic Internship Program)

Bowling Green State University, Bowling Green, Ohio
Master of Food and Nutrition, 2014; Field of Study: Nutrition and Food Science
Area of Concentration: Food and Nutrition Sciences

Bowling Green State University, Bowling Green, Ohio
Bachelor of Science in Dietetics, 2011; Field of Study: Dietetics
Major: Dietetics

Professional Credentials and Experience

a. Licenses/Credentials/Certifications

Certified Strength and Conditioning Specialist (CSCS), National Strength and Conditioning Association 2017-Present.
Licensed Dietitian/Nutritionist (LDN), Louisiana Board of Examiners for Dietetics and Nutrition, State of Louisiana, Lafayette, Louisiana (#3031), 2019-Present.
Licensed Dietitian (LD), Ohio Board of Dietetics, State of Ohio, Columbus, Ohio (LD7955), 2016-2018.
Registered Dietitian (RD)/Registered Dietitian-Nutritionist (RDN), Commission on Dietetic Registration, Chicago, Illinois (86011404) 2016-Present.

Certifications
ISAK-Level 1, International Society for the Advancement of Kinanthropometry, 2018
Basic Life Support Healthcare Professional (CPR) Certified, American Heart Association 2016-Present.

b. Professional Positions
Sports Nutritionist, University of Louisville, Louisville, KY; (2020-Present)
Associate Director of Athletic Performance for Sports Nutrition, University of Louisiana, Lafayette, LA; (2019-2020).
Graduate Instructor, The University of Mississippi, University, MS, School of Applied Sciences; (2018-2019).
Assistant Sports Dietitian, The University of Mississippi, University, MS, Center of Health and Sports Performance; (2017-2019)
Graduate Instructor, Bowling Green State University, Bowling Green, OH, College of Health and Human Services; 1 year (2013-2014).
Weight Management Clinic Registered Dietitian, Mary-Rutan Hospital, Bellefontaine, OH; 3 months (2018)
Nutrition Program Coordinator and Registered Dietitian, Life Time Fitness, Dublin, OH; 6 months (2017-2018)
Nutrition and Physical Education Coordinator, Children’s Hunger Alliance, Columbus, OH; 5 months (2017)
Wellness Coordinator, HealthWorks, Cincinnati, OH; 1 year (2015-2016)

Scholarly and Creative Activities/Accomplishments

a. Abstracts

b. Presentations
Invited (Non-refereed) Platform Presentations
Frakes, MR. Protein and Amino Acids in Sports Nutrition, University of Mississippi, Oxford, MS. February 2019
Frakes, MR. Fueling for Performance. (Invited) Coaches Clinic, Mary-Rutan Hospital, May 2015.
Frakes, MR. Nutrition and Health. (Invited) Men’s Health Week, Marion-Franklin Community Center, October 2014

c. Research Activities

Professional/Community/University/Military Service and Administration

a. Reviewer/Editorships (Includes Service to Refereed Journals)
International Journal of Exercise Science [Reviewer, 2018-Present]

b. Professional Association Memberships and Service
The American College of Sports Medicine (ACSM) (2018-Present)
International Society for the Advancement of Kinanthropometry (ISAK) (2018-Present)
National Strength and Conditioning Association (NSCA) (2017-Present)
National Organization of Blacks in Dietetics and Nutrition (NOBIDAN) (2017-Present)
Academy of Nutrition and Dietetics (Academy) (2016-Present)
Sports, Cardiovascular, and Wellness Nutrition (SCAN-DPG) (2016-Present)
   • 34th Annual SCAN Symposium Poster Session Judge (2018)
   • 31st Annual SCAN Symposium Volunteer (2015)
The Collegiate and Professional Sports Dietitians Association (CPSDA) (2016-Present)

c. Community Memberships and Service
Columbus Young Professionals Club, Columbus, OH (2015-2017)

Clubs and Organizations
Bowling Green State University Student Nutrition Association (2010-2011, 2013-2014)

Other University Service
Bowling Green State University Football (2009, 2010)

Instruction and Advisement

a. Courses Taught
University of Mississippi, Winter Intersession, 2019
NHM 213 Principles of Food Preparation (lab) (12 students) (sole instructor)

University of Mississippi, Fall, 2018 and Spring, 2019
NHM 311 Basic Nutrition (lecture) (106 students) (sole instructor)
Bowling Green State University, Fall, 2013
FN 2120 Food: Preparation, Availability, and Resources (lab) (60 students) (sole instructor)

Bowling Green State University, Spring, 2014
FN 2120 Food: Preparation, Availability, and Resources (lab) (60 students) (sole instructor)

b. Courses Developed
V.E.T. Eating Nutrition Module - Vital Education Through Eating (12-15 students, ages 9-13) (sole nutrition educator), Fall 2017-Spring 2018

Honors and Awards
2019 10 Under 10, University Advancement, Bowling Green State University, August 2019
2018 3 Minute Thesis (3MT) First Place Doctoral Category, Graduate School, University of Mississippi, November 2018
2018 Summer Research Assistantship, Graduate School, University of Mississippi April 2018.
2017 Graduate Assistantship, Center for Health and Sports Performance, University of Mississippi, February 2017
2016 Most Valuable Trainer, Dublin Club, Life Time Fitness, December 2016
2014 Graduate Student Research Support Award, Family and Consumer Sciences, Bowling Green State University, February 2014
2013 Graduate Assistant Teaching Assistantship, Department of Family and Consumer Sciences, Bowling Green State University, May 2013

Updated May 5, 2020