ASSESSMENT OF NUTRIENT INTAKES AND DIETARY BEHAVIOR IN A POPULATION WITH HEAD INJURY

by

JACLYN BARTA

(Under the Direction of Hea Jin Park)

ABSTRACT

Nutrients help regulate inflammation, yet there are no nutrition guidelines for inflammatory traumatic brain injury. We utilized National Health and Nutrition Examination Survey data to understand the diets of US adults with head injury with loss of consciousness (HIC). HIC consumed significantly more calories than those without head injury (No-HIC). After adjusting for energy intakes, HIC consumed less protein, antioxidants, and omega-3 fatty acids than No-HIC. When compared to Dietary Reference Intakes (DRI), HIC consumed more of 10 nutrients than No-HIC, many which were pro-inflammatory nutrients. We investigated the intakes of those who self-reported adhering to a weight loss diet, using nutrients with DRIs. Only Vitamin E intake was different between HIC dieters and non-dieters, yet 9 nutrient intakes were different between No-HIC dieters and non-dieters. Findings from this study provide preliminary data on dietary behavior and dietary inflexibility of US adults with head injury, leading toward dietary recommendations.

INDEX WORDS: NHANES, traumatic brain injury, antioxidants, weight loss, obesity, RDA

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TABLE OF CONTENTS

Page

LIST OF TABLES

Page

LIST OF FIGURES

Page

CHAPTER 1

INTRODUCTION

Traumatic brain injury (TBI) is a state of altered brain functionality as a direct result of a jolt or blow to the head, which can result in memory loss, cognitive impairment, loss of consciousness, and long-term disability.[1] There were 64,000 TBI-related deaths in America in 2020, and approximately 20% of individuals die within 5 years of their TBI.[1, 2] Given the urgency of care for these patients, nutrition intervention is a key treatment. Nutrients which possess anti-inflammatory, reparative, and antioxidant functions may benefit patients with TBI and improve overall health outcomes.

TBI results in a hypermetabolic state, increasing caloric needs, inflammation, and stress hormones, all of which can alter the processing of nutrients.[3] Since TBI triggers an inflammatory response, secondary injuries can occur from the buildup of blood and pressure on the brain, which often causes more damage than the initial injury.[4] The long-term consequences of uncontrolled neuroinflammation include neurodegenerative diseases such as Alzheimer's Disease or dementia.[4] There may be benefits from consuming foods and supplements which promote memory repair, reduce inflammation and oxidative stress, or promote neurogenesis for patients with TBI.[5-7]

Possible dietary interventions for TBI treatment are primarily focused on nutrients which promote tissue repair, cognitive function, and possess anti-inflammatory and antioxidant roles. Energy and protein needs increase immediately after injury, therefore adequate intakes are essential for repair of damaged tissue and prevention of malnutrition in the hypermetabolic

state.[8] Omega-3 fatty acids, particularly DHA, have been observed to possess anti-inflammatory properties, which may help reduce inflammatory cascades post-injury.[9, 10] DHA may also promote blood flow to the brain, protect cognitive function, and improve motor function, after TBI.[11, 12] Antioxidants, such as Vitamin E and selenium, may be neuroprotective, help reduce oxidative stress, improve cognition, and promote synaptic plasticity.[13, 14] Zinc and lutein also may reduce the impact of the injury by mediating the inflammatory response to TBI.[6, 7] Zinc is necessary for a myriad of processes involved in healing from a TBI such as wound healing, neurogenesis, and reduction of oxidants.[15] Lutein may improve outcomes after TBI by reducing neuroinflammation, oxidation, and lesion size.[6] Various nutrients play an important role in regulating secondary injury following TBI to promote long-term recovery of the patients.

Nutrients may also indirectly impact recovery from injury in patients with TBI. A correlation between poor sleep and head injury has been observed, which can impact quality of life in TBI patients.[16] Branched chain amino acids are a possible supplement for the population with head injury, as they have been observed to mitigate some of the sleep disturbances observed in TBI patients.[17] Consumption of essential branched chain amino acids led to reduced insomnia severity index scores, improved sleep efficiency, and decreased the time to fall asleep in a group of veterans with TBI.[17] Improving sleep quality can improve health outcomes, therefore branched chain amino acids could be a key supplement for optimal recovery from TBI.

There are many proposed benefits from consuming a diet rich in nutrients with antioxidant, anti-inflammatory, reparative, and neurogenesis-promoting functions in the recovery from the injury in the patients with TBI.[8, 9, 13, 14, 18] However, despite the many ways these

nutrients could benefit patients with TBI, there are no established dietary guidelines for TBI recovery. More importantly, there is a lack of information on the dietary intakes and diet quality of this population post-injury.

Chronic inflammatory conditions can lead to various chronic diseases such as obesity, diabetes, cardiovascular disease, and neurological disorders. As discussed in Chapter 2, low nutrition knowledge has been observed to associate with poorer health outcomes.[19] Diet educations increase patient nutrition knowledge and self-efficacy in a variety of disease states to allow for improved patient self-care nutrition practices.[20-22] Improving nutrition literacy was observed as critical to improving health outcomes, as nutrition knowledge was correlated with better health and nutrient intake habits across multiple disease states.[19, 23, 24] While neurological injuries in patients with stroke or TBI can induce dysphagia and other post-injury disabilities which impact the ability to feed oneself, dietary intakes most correlated with Recommended Dietary Allowance (RDA) values led to optimal health outcomes among patients with these injuries.[25-27] Nutrition knowledge and diet behaviors are essential for recovery from these diet-related disease states.

In this thesis, Chapter 2 reviews the literature on diet behaviors of populations with diet-related chronic disease states, including obesity, Type 2 Diabetes Mellitus, hypertension, atherosclerosis, and neurological injuries. Chapter 3 assesses the current dietary intakes and diet quality among a population from the National Health and Nutrition Examination Survey (NHANES) with history of head injury, compared to those who reported no history of head injury. Chapter 4 is a study which further investigates the behavioral flexibility of the NHANES participants with and without history of head injury who report adherence to the most reported dietary modification in NHANES, a weight loss diet.

Therefore, given the lack of information available on the current dietary behaviors in the population with TBI, the goal of this thesis was to analyze preliminary data on the current dietary intakes of the population with head injury. We analyzed data from the National Health and Nutrition Examination Survey (NHANES) from 2011-2014, utilizing the question CSQ240, "Have you had a loss of consciousness because of a head injury?". We compared the dietary nutrient intakes from dietary recalls of those who reported a history of head injury with loss of consciousness (HIC) and those who reported no head injury with loss of consciousness (No-HIC). We then compared dietary nutrient intakes to the age- and gender-specific Dietary Reference Intakes, to determine the diet quality of participant diets. Additionally in Chapter 4, we investigated the impact of self-reported diet modification on nutrient intakes in the participants, to assess behavioral flexibility in the participants with TBI. This thesis sought to report preliminary findings on the dietary intakes and capability for dietary behavior change of the population with head injury, to further the field of head injury nutrition toward the development of post-TBI nutrition guidelines.

CHAPTER 2

LITERATURE REVIEW

2.1 BEHAVIOR CHANGE, FOOD CHOICE, AND DIET BEHAVIOR

Dietary patterns have a major impact on health outcomes in a myriad of disease states, yet nutrition knowledge alone may not lead to successful changes long-term. Despite proper dietary education, patients often struggle to change their eating behaviors. Food choices are driven by socioeconomic, psychological, nutritional, and sensory factors.[28] Stress in early life, the modern obesogenic dietary environment, and sensory cues from food can all influence dietary decisions and can shape dietary patterns.[28] Sensory appeal and familiarity were found to be the most and least important aspects when selecting food products, respectively.[29] It is well established that more palatable foods, such as those higher in sugar and fat, induce a greater reward in the neurological circuitry than bland foods.[28] Price and preparation convenience have also been highly rated as important when selecting foods.[29] Similarly, family food preferences and cost were found to strongly influence food choices among low-income women.[30] Increased stress levels were seen to increase preference for sweet, higher calorie foods among women in a study by Zellner et al.[31] 46% of women were found to overeat when stressed, compared to only 17% of men.[31] Perceived level of higher diet quality may also be indicative of higher diet quality.[32]

Therefore, to change dietary behaviors, a variety of factors need to first be addressed. Dietary behaviors can be influenced by food preferences, socioeconomic status, household size, time constraints for food preparation, and food security.[28, 30] Due to the extensive time

employees spend there, the workplace can be a location for a dietary behavior change.[33] If management participates in health promotion, workplace cafeterias serve healthy options, and the program fits the environment with little burden on the employees, workplace dietary interventions can be successful at improving dietary outcomes.[33] Behavior change in patients has been found to be most effective when practitioners utilize the cognitive behavioral theory, which aims to improve both thoughts inhibiting health changes, as well as environmental factors which make healthy lifestyles difficult.[34] Motivational interviewing was found to be another effective tool for helping the client find and resolve barriers to change in their health status.[34] The trans-theoretical model stages of change helps to find barriers which prevent patients from improving their dietary patterns, and may be used to determine when an intervention is appropriate, and when it is acceptable to provide information on the health risks associated with poor dietary quality.[35]

Nutrient-dense dietary patterns can improve health outcomes in a variety of chronic disease states by decreasing inflammatory and oxidative stress cascades, improving the efficacy of the immune system, and helping to maintain optimal blood glucose levels.[36-38] Diet is linked with many chronic diseases, including certain cancers, diabetes, obesity, hypertension, cardiovascular diseases, and others.[36, 38, 39] Dietary behavior is critical to health status, so we reviewed several health conditions with dietary recommendations to review the dietary behaviors in the different populations.

2.1.1 *Diet Behavior in Nutrition-Related Chronic Diseases*

There is a myriad of chronic diseases which can be impacted by dietary intakes and other chronic diseases. Therefore, consuming a dietary pattern aimed at improving one chronic disease may in turn reduce the disease burden in other chronic conditions. Obesity is a risk factor for

developing Type 2 Diabetes Mellitus, as abdominal visceral obesity is associated with insulin resistance.[39] Therefore, adherence to weight loss diets to reduce BMI and the burden of obesity may improve blood sugar control in patients with diabetes.[40] Similarly, the inflammatory states caused by other diet-related chronic diseases can exacerbate the impact of other chronic diseases. These connections between disease states can be utilized by practitioners to motivate behavior change for multiple disease states with fewer interventions.

2.2 CONNECTIONS BETWEEN NUTRITION-RELATED CHRONIC DISEASES

Multiple chronic diseases are strongly impacted by dietary intakes and are often found as comorbidities in patients. Obesity is a disease state which can lead to chronic low-grade inflammation.[41] This chronic inflammation can increase the risk of developing diabetes and cardiovascular disease.[41] Furthermore, diabetes is a risk factor for atherosclerosis, and prolonged hyperglycemia plays a role in the progression of the disease.[42] Elevated LDL cholesterol can increase the risk of developing atherosclerosis, and reduction in LDL may reduce the incidence of stroke occurrence.[42]

Consumption of a high-fat western diet or a diet high in animal products may further lead to chronic inflammation, and increases the risk of developing atherosclerosis, hypertension, diabetes, dementia and other diet-related chronic conditions.[43-45] Chronic neuroinflammation can enhance the development and progression of neurodegenerative diseases, therefore, patients with obesity or atherosclerosis are at higher risk due to their underlying inflammatory conditions.[45]

Like other chronic diseases, hypertension plays a role in inflammation, and can lead to further cardiovascular events.[44] Not only does chronic inflammation play a role in

development of cardiovascular disease, stroke, and neurodegenerative disease, inflammation observed in patients with obesity can negatively impact neurological outcomes after stroke.[46]

Reductions in immunity have also been observed in patients with a state of chronic inflammation.[44] Patients with obesity may experience more frequent infections, higher levels of pro-inflammatory biomarkers, and poorer neurological outcomes post-stroke compared to their counterparts with BMI within normal range.[46] Despite these negative outcomes, the patients with obesity were not found to have any differences in functional outcomes 3 months post-stroke compared to their counterparts.[46] Long-term inflammation can also strain the endothelial muscles, which can lead to cardiovascular events.[44] As chronic diseases are highly correlated with inflammation, which can exacerbate the symptoms of other diseases, dietary interventions to reduce inflammation and mediate the impacts of chronic disease are essential.

2.3 DISEASE BURDEN AND DIETARY BEHAVIORS IN THE POPULATION WITH OBESITY

Obesity is a chronic disease classified by a body mass index of greater than 30 kg/m^2.[47] Symptoms of obesity can include sleep apnea, joint pain, dyspnea, and depression.[48] Obesity can induce chronic low-grade inflammation, often leading to poorer health outcomes.[49] Inflammation from obesity can contribute to development of chronic diseases, such as chronic kidney disease, diabetes mellitus, depression, and cardiovascular disease.[47, 49] Obesity impacts more than 2 billion people globally, with the BMI of nearly 30% of the world population classified as overweight or obese.[50] 42.5% of U.S. adults 20 years old and older have obesity, and nearly 1 in 11 U.S. adults have severe obesity, according to the 2017-2018 National Health and Nutrition Examination Survey.[51] While obesity is common in the United States, the lowest rate of all-cause mortality has been observed within the “normal” BMI range of 20-25 kg/m^2.[47]

High levels of adipose tissue in the body can lead to significant hormone release, such as estrogen, associated with an increased risk of developing breast cancer.[41] Eleven cancers have positive associations with obesity, including endometrial, esophageal, gastric cardia, liver, kidney, multiple myeloma, meningioma, pancreatic, colorectal, gallbladder, breast, ovarian, and thyroid cancer.[52] Obesity induces a state of chronic inflammation, which can impair insulin signaling, causing obesity to be a risk factor for the development of Type 2 Diabetes Mellitus.[39, 41] Obesity is a critical health concern, therefore dietary interventions have been a tool utilized for management of the chronic condition.

2.3.1 *Nutrition Interventions Recommended for Treatment of Overweight and Obesity*

Weight loss diets are commonly practiced among the public for the prevention and reduction of symptoms in overweight and obesity.[40] Common diets attempt to achieve an energy deficit via reduced intake of macronutrient groups, such as a low carbohydrate and low-fat diet.[40] Nutrition educations have been observed as efficacious in the overweight and obese population.[21] Educating overweight and obese individuals on how to increase fruit and vegetable intakes and the health benefits of consumption may lead increased produce intakes.[21] However, the strongest recommendation is to select a dietary pattern that incorporates patient preferences and promotes adherence to achieve optimal weight loss long-term.[40]

2.3.2 *Diet Behaviors in the Population with Overweight and Obesity*

Improved diet quality has been associated with reduced body weight and improved health outcomes in overweight and obesity, but diet behaviors can be impacted by a myriad of factors. Nutrition educations have been effective tools for changing dietary behaviors in the population with overweight and obesity, as educating participants on how to increase fruit and vegetable

consumption and the health benefits of increased intakes was seen to increase intakes in a study by Wagner et al.[21] When participants were provided with detailed educations on the benefits of consumption of produce for a 10 week period, participants increased total weekly fruit and vegetable consumption by almost 1 extra fruit or vegetable per day (6.5 servings per week).[21] Increasing intakes of low-calorie fruits and vegetables can improve health outcomes beyond weight loss, so the educations to increase intakes are clearly beneficial to these patients.

While certain dietary patterns may increase weight loss, patient food preferences are an essential consideration for what diet will be most effective. When participants were given the choice between low-fat and low-calorie and low-carbohydrate diets for weight loss, diet selections were seen to correlate with current dietary patterns and belief that a certain diet was healthier.[53] Dietary approaches for weight loss were found to be selected based on current dietary habits, along with which may be a healthier option, within participants with overweight or obesity attempting weight loss.[53] Interestingly, following a diet similar to baseline food preferences did not always lead to increased weight loss.[54] Among those following a low-carbohydrate diet, increase in low-carbohydrate preferences led to increased weight loss, yet this change was not seen among those on a low-fat diet.[54] Similarly, older veterans with a BMI of greater than 30 kg/m^2 were offered their choice of two diets for weight loss: low fat or low carbohydrate. The comparator group included participants randomly assigned to their diets, low-fat or low carbohydrate. Dietary adherence was not significantly different between those who chose their diet and those who were assigned, and offering the choice of dietary intervention did not increase weight loss compared to their counterparts.[55]

Commonly seen in patients with obesity, metabolic syndrome is a collection of conditions which increase your risk of chronic diseases, such as diabetes, heart disease, and

stroke.[56] Metabolic syndrome is noted by having 3 or more conditions, which may include elevated blood pressure, elevated blood glucose, excess body fat around the waist, or abnormal cholesterol or triglyceride levels.[56] Diets which closely adhere to dietary guidelines, the DASH diet, or the Mediterranean diet may lead to fewer incidences of metabolic syndrome.[57] There is limited evidence to correlate occurrence metabolic syndrome with intakes of saturated fatty acids.[57] Metabolic syndrome may be caused by a combination of factors from dietary patterns, as triglyceride levels, blood sugars, and blood pressure all have established correlations with dietary intakes.[57]

2.4 DISEASE BURDEN AND DIETARY BEHAVIORS IN THE POPULATION WITH TYPE 2 DIABETES MELLITUS

Type 2 Diabetes Mellitus (T2DM) is a disease noted by elevated blood glucose levels, caused by resistance to insulin.[58] In 2018, an estimated 10.2% of the US population aged 18 or older had a diagnosis of diabetes, and an estimated 2.8% of the adult US population met laboratory criteria of diabetes but were not yet diagnosed with diabetes.[59] Risk factors for developing Type 2 Diabetes Mellitus include those who were over 45 years old, overweight or obese, had a family history of DM, physically inactive, or were affected by other health conditions, such as history of gestational diabetes.[58] As Type 2 Diabetes Mellitus can develop over the years, it can be difficult to detect symptoms, but symptoms may include polyuria, polydipsia, unintentional weight loss, increases in hunger, blurred vision, numb or tingling feet or hands, fatigue, having sores that heal slowly, or an increase in infections.[60] Since diabetes is correlated so strongly with dietary intakes, dietary interventions are a common tool for ameliorating the condition.

2.4.1 *Nutrition Interventions Recommended for Treatment of Type 2 Diabetes Mellitus*

Type 2 Diabetes Mellitus is interconnected with dietary patterns, and therefore diet is a primary intervention for the care of patients with diabetes. Dietary interventions for the treatment and management of Type 2 Diabetes Mellitus include carbohydrate counting, low-carbohydrate diets, high-fat low-carbohydrate, and the Mediterranean diet.[61-63] Treatment of Type 2 Diabetes Mellitus includes proper blood sugar control, weight management, and reduction of health risks from comorbidities.[64] The Mediterranean Diet may reduce HbA1c levels, and plant-based diets may ameliorate insulin resistance.[62, 65] Carbohydrate counting is a common method for consistent carbohydrate intakes among those with insulin resistance, and has been shown to be effective at maintaining normalized blood glucose levels.[66, 67] The American Diabetes Association states that there is not one ideal diet which will be appropriate for all patients with diabetes, rather medical nutrition therapy should focus on individual dietary preferences and healthful eating patterns rather than a single nutrient.[64]

2.4.2 *Diet Behaviors in the Population with Type 2 Diabetes Mellitus*

Environmental and internal factors have been shown to influence diet behaviors. Self-efficacy and social support both are beneficial for diabetes diet self-management.[68] Social support utilizing friends, family, medical personnel, and other patient support is important for diet maintenance and adherence long-term in patients with diabetes.[68] However, diet self-efficacy mediates the association between social support and diabetes self-management, indicating that when patients have greater confidence in their ability to consume healthy foods and improve their health, less dependence on social support is necessary.[68]

Dietary educations can likewise improve A1c levels in the patient population with diabetes. Provision of at least 3 months of diet education was found to be an effective means for

controlling A1c in the patient population with Type 2 diabetes mellitus, in a study by Kim et al.[69] A1c levels were impacted most significantly by combined educations on dietary intakes, exercise, and psychological factors.[69] Providing insulin-dependent patients with diabetes education on medication, insulin injection, healthy diet, physical activity, SMBG (blood glucose self-monitoring), and prevention of hypoglycemia and complications was seen to lead to lower A1c levels compared to a control group.[70] While A1c may be decreased from proper adherence to medication, lifestyle changes and dietary patterns may produce further beneficial outcomes from the educations.[70] Therefore, nutrition knowledge plays a key role in dietary choice.

Diabetes knowledge of 304 Thai adults with T2DM was measured using the "Theptarin DM questionnaire" from Theptarin hospital.[20] Within these participants, macronutrient consumption was adequate, but the participants consumed excesses of saturated fat and free sugar, and lower dietary fiber intake than recommended.[20] Interestingly, there were no associations between macronutrient intakes and those who had good glycemic control (A1c <7.0%) and those who had poor glycemic control (A1c >/= 7.0%).[20] Most patients reported having a moderate knowledge of diabetes self-care, including topics such as counting carbohydrates or following a diabetes exchange list, yet there were no differences between self-reported diabetes knowledge and adherence to dietary self-care behaviors.[20]

Food choices can heavily impact diabetes outcomes, and high intakes of red meat and poultry were observed to associate with higher rates of diabetes occurrence in a cohort study.[71] Long-term adherence to a vegetarian diet reduced the odds of developing diabetes by 74% compared to those who followed a non-vegetarian diet throughout the study.[71] This dietary correlation may be caused more by dietary patterns which include meat and processed meat,

rather than simply from the increased intakes of fat and saturated fat in meat, and nitrates and nitrites in processed meats.[71] Another study also found a potentially protective effect in T2DM risk in participants who consumed plant-based diets.[72] Participants with diets higher in plant-based foods and lower in animal products were found to have reduced risk of insulin resistance, prediabetes, and T2DM.[72] Similarly, dietary intakes which adhered more closely to the DASH, Mediterranean, and AHEI (alternative healthy eating index) dietary patterns, which all emphasize consumption of plant-based foods, were shown to reduce risk of developing T2DM.[73] Therefore, a correlation was observed between increased fruit and vegetable intake and adherence to nutrition guidelines and decreased risk and exacerbation of diabetes.

2.5 DISEASE BURDEN AND DIETARY BEHAVIORS IN THE POPULATION WITH HYPERTENSION AND ATHEROSCLEROSIS

Hypertension is defined by elevated blood pressure, systolic BP at ≥ 130 mmHg or diastolic BP ≥ 80 mmHg.[74] The age-adjusted prevalence of hypertension in Americans over 20 years old was estimated as 47.3% per NHANES 2013-2016.[74] In the population over 65 years old, the prevalence of high BP is estimated as 77.0% per NHANES 2015-2018.[74] High blood pressure is a risk factor for a variety of cardiovascular diseases, including heart failure and stroke.[74] Long-term inflammation can strain the endothelial muscles, which can lead to cardiovascular events.[44] Hypertension often does not induce any symptoms.[75] If there are symptoms in a patient with hypertension, they may experience morning headaches, nosebleeds, irregular heart rhythms, vision changes, and buzzing in the ears.[75] Symptoms of severe hypertension can include fatigue, nausea, confusion, chest pain, or muscle tremors.[75] Given the relationship between hypertension and cardiovascular health, dietary treatments are essential to ensure blood pressure reduces to the normal range.

Hypertension is a risk factor for the development of atherosclerosis, the hardening of arteries via plaque buildup.[76] Like hypertension, atherosclerosis has few symptoms until the plaque buildup is severe enough to cause a stroke, blood clot, or heart attack.[76] Other risk factors for atherosclerosis include high cholesterol and triglyceride levels, smoking, obesity, physical inactivity, and a diet high in saturated fat.[76] Atherosclerosis is estimated to be the cause of 50% of all deaths in western society.[77] Due to the correlation between dietary choices, atherosclerosis, and heart disease, dietary interventions in this population are essential.

2.5.1 *Nutrition Interventions Recommended for Treatment of Hypertension and Atherosclerosis*

Dietary treatment of hypertension often includes implementation of a sodium reduction, the Dietary Approaches to Stop Hypertension (DASH) diet, or the Mediterranean Diet.[78-80] A low sodium diet (<1,150 mg Na/day), in combination with adherence to the DASH diet, which is rich in fruits, vegetables, low-fat dairy foods, whole grains, poultry, nuts, fish, and low in sugar sweetened beverages, desserts, and red meats, may reduce blood pressure.[78, 79] The American Heart Association recommends consuming less than 2,300 mg of sodium per day for the average American adult, with an ideal limit of 1,500 mg per day.[81] Similar to the DASH diet, the Mediterranean diet emphasizes fruits, vegetables, nuts, legumes, seeds, and whole grains, with a moderate intake of fish, poultry, and reduced intake of red meat and sweets.[80] The distinguishing characteristics of the Mediterranean diet include an emphasis on moderate intake on red wine and high intake of olive oil.[80] The dietary treatments for atherosclerosis are similar to those for hypertension, with the additional recommendations to increase beneficial fatty acids and nutrients, such as omega-3 fatty acids, fiber, and nutrients with antioxidant properties.[42]

2.5.2 *Diet Behaviors in the Population with Hypertension*

Elevated blood pressure may be correlated with poor dietary patterns and is a risk factor for the development of multiple cardiovascular conditions. When observing the effect of cooking meals at home on hypertension, a correlation may be present between cooking at home, gender, and risk of hypertension.[82] Most female participants cooked daily, and less than one quarter of the male participants cooked daily in a study by Zhang et al.[82] Interestingly, the more frequently men cooked at home, the stronger the odds were of that participant having hypertension.[82] Conversely, daily cooking had a reduction in rates of hypertension among women.[82] Therefore, gender and cooking style may play a role in the correlation between cooking meals at home and the risk of hypertension.

Nutrition knowledge can be a critical factor in how closely patients adhere to antihypertensive diet recommendations, such as the DASH diet. Diet quality was analyzed via DASH score, indicating level of adherence to the DASH dietary patterns in certain nutrient groups in a study by Geaney et al.[23] Nutrition knowledge was likewise analyzed to discover the correlations between nutrition knowledge and DASH adherence score.[23] Women scored in the highest quintile for DASH adherence scores in a larger number than men, indicating better DASH diet adherence among women.[23] Participants who reported “always” adding salt at the table were found to have lower DASH scores than those who reported “never” adding salt to food, as a low sodium intake is a key factor in DASH diet adherence.[23] Diets which adhered more closely to the DASH diet were observed to correlate with increased nutrition knowledge.[23] Presence of hypertension was negatively associated with nutrition knowledge.[23]

Increased adherence to an energy-restricted Mediterranean dietary pattern led to a trend in decreased rates of hypertension, in a study by Álvarez et al.[83] Interestingly, among the male participants, there was an observed association between increased adherence to the Mediterranean dietary pattern and presence of diabetes.[83] Álvarez et al. found that greater adherence to the Mediterranean diet was associated with lower triglyceride and LDL levels, along with adiposity measures, which are risk factors for other cardiovascular diseases.[83] Despite the minimal change to hypertension rates, adhering to the Mediterranean diet may help to decrease cardiovascular risk factors.

2.5.3 *Diet Behaviors in the Population with Atherosclerosis*

Dietary behaviors play a key role in the development of atherosclerosis. Therefore, improving dietary behaviors may impact health outcomes in the population with atherosclerosis. Due to the higher fiber content and lower saturated fat content, plant-based diets may decrease the risk of developing atherosclerosis.[84] Among healthy participants, dietary intakes were assessed via food frequency questionnaire, and risk of developing atherosclerosis was measured by testing blood lipids, leukocytes, and pro-inflammatory metabolites in a study by Yang et al.[84] Although meat products, which are correlated with increased cholesterol and risk of atherosclerosis, are excluded from the vegetarian diet, adhering to the vegetarian diet for less than 10 years was not found to change cardiometabolic health or inflammation compared to their omnivore counterparts.[84] Long term vegetarianism (>10 years) was seen to correlate with lower cholesterol levels.[84] Generally, a diet high in leafy green vegetables, fruits, and water, and which avoided foods high in added sugars and sodium, such as sugar-sweetened beverages and instant noodles, was found to correlate with lower levels of inflammation, and less severe carotid intima-media thickness, an indicator for degree of atherosclerosis.[84]

5-year dietary adherence to either low-fat (<30% of calories from fat) or Mediterranean dietary patterns was measured to determine the efficacy of dietary educations on the long-term maintenance of diets for cardiovascular health.[85] Both diet groups were provided dietary educations from a registered dietitian bimonthly, with the attempt to maintain dietary adherence and reinforce dietary recommendations.[85] After the 5 year dietary intervention, those adhering to the Mediterranean diet followed dietary patterns higher in fiber, monounsaturated fatty acids, polyunsaturated fatty acids, and lower in saturated fats, carbohydrates, and cholesterol.[85] Following the 5-year intervention, those adhering to the low-fat dietary pattern increased their intakes of fiber, complex carbohydrates, and decreased their total fat intakes, along with all types of fatty acids and cholesterol.[85] These maintained dietary changes, such as an increased intake of the anti-inflammatory alpha-linoleic acid among the Mediterranean diet group and a decrease in dietary cholesterol in the low-fat diet group, can lead to a decrease in the risk of atherosclerosis and cardiovascular disease.[85] The benefits of long-term changes to dietary behaviors can contribute to a lower risk of cardiovascular disease.

2.6 DISEASE BURDEN AND DIETARY BEHAVIORS IN THE POPULATION WITH NEUROLOGICAL INJURY

Approximately 7 million Americans 20 years of age and older self-report having experienced a stroke in their lifetime, and prevalence of stroke is increasing in the American population.[86] Stroke occurs when blood supply to the brain is stopped, or a blood vessel in the brain bursts, preventing the brain from receiving nutrients and oxygen from the blood.[87, 88] Symptoms of stroke include the sudden onset of the following symptoms: numbness in the face, arms, or legs, particularly on one side of the body, confusion, trouble speaking or understanding speech, vision problems, trouble walking or dizziness, and severe headache.[89] African

Americans are at greater risk of first-ever stroke occurrence compared to Caucasian Americans, after adjusting for income.[86] 87% of strokes are ischemic, 10% are intracerebral hemorrhage (ICH), and 3% are Subarachnoid hemorrhage (SAH).[86]

Traumatic brain injuries (TBI) are another neurological injury which put enormous stress on the healthcare system. Approximately 176 Americans die from traumatic brain injuries (TBI) every day, and more than 223,000 TBI-related hospitalizations occurred in 2019.[90] TBI occur as a result of a jolt or blow to the head and can induce a myriad of symptoms in the patient.[91] Symptoms may include loss of consciousness, vomiting, confusion, paralysis, dilated pupils, vision changes, lethargy, dizziness, speech difficulties, body numbness or tingling, memory loss, cognitive impairment, headaches, and breathing trouble.[91] Due to the risk of chronic neuroinflammation after injury, dietary intervention to minimize the long-term neurological damage is essential.

2.6.1 *Nutrition Interventions Recommended for the Treatment of Neurological Injury*

The dietary recommendation for the prevention of first and secondary stroke is similar to those for reduction of hypertension: adhere to the Mediterranean diet, avoid excessive intakes of salt, and consume low intakes of processed and red meats.[92] Following a stroke, dietary interventions focus on reducing risk of a second stroke and accommodating any functional losses the patient may have experienced which reduces their ability to feed themself.[26, 92]

While there are no current dietary recommendations for patients with TBI, diets rich in protein and energy are essential immediately after injury to prevent malnutrition due to post-injury elevated energy needs.[8] Protein, vitamins with antioxidant properties, DHA, and minerals such as zinc and selenium have been hypothesized as potentially beneficial to reduce neuroinflammation, improve wound healing, and promote healing after TBI occurrence.[6, 8, 13-

15] Dietary interventions following neurological injuries need to include nutrients which help to maintain cognitive functionality and reduce neuroinflammation.

2.6.2 *Diet Behaviors in the Population with Neurological Injury*

Diet behavior after stroke can be heavily influenced by the environment where the patients recover, as well the decreased functionality resulting from the neurological damage.[26] Stroke patients can have severe physical impairments which require occupational and physical therapy to recover abilities, so their dietary intakes can be directly impacted.[26] Stroke patients were found to experience less satisfaction in hospitals with food choice compared to patients in other wings of the hospital, which may be correlated with their increased physical barriers to food consumption.[26] Physical barriers were noted as needing help eating at meals, being unable to cut food or unwrap packets, and not being in a comfortable position to eat.[26] The functional losses in hand-eye coordinator, swallowing, or other functions necessary for self-feeding may impact the dietary intakes in this population.

While dietary changes may occur after stroke, certain dietary behaviors may reduce the risk of stroke occurring. Consumption of breakfast daily may reduce the risk of developing stroke, and a breakfast of oatmeal instead of eggs or white toast may have a greater reduction in risk.[93, 94] Frequency of breakfast intakes were found to inversely correlate with risk of developing stroke and other cardiovascular diseases, in a Japanese cohort study.[93] Furthermore, what foods are consumed at breakfast may decrease risk of developing stroke.[94] One serving of oatmeal, a cholesterol-lowering whole grain, was observed to correlate with lower risk of stroke, compared to intakes of eggs or white bread in a cohort study.[94] Therefore, dietary choices at breakfast may play a significant role in the risk of stroke.

Like those with stroke, patients with traumatic brain injury may experience dysphagia and difficulty swallowing their food.[25] TBI patients with dysphagia often need altered feeding modes, which may lead to a modified texture diet, increased viscosity of patients' beverages, or feeding via a gastrostomy tube.[25] When patients experience post-injury dysphagia, they are at increased risk of developing malnutrition, as they often are not able to receive adequate protein and calorie intakes if they are hospitalized post-injury.[8] Therefore, dietary changes must include increased protein and calorie intakes to account for the hypermetabolism in these patients.[8]

As there are no dietary guidelines for TBI recovery or long-term management of symptoms, the population with head injury may be following dietary patterns correlated with poorer health outcomes. Adherence to the Recommended Dietary Allowance (RDA) for the most nutrients was observed to correlate with the mildest symptoms of TBI, compared to those who adhered to the RDA of the fewest number of nutrients, among 39 veterans with suspected TBI.[27] Even among the participants whose food frequency questionnaires most closely aligned with nutrient recommendations, no participants were found to meet the RDA of all 14 nutrients analyzed from food frequency questionnaires.[27] No participants reported a dietary intake of Vitamin A at or above the recommendation, despite the function of Vitamin A as an antioxidant.[27] After Vitamin A, Vitamin E and manganese were met the least frequently, which posed a concern due to their antioxidant capacity and role in neurotransmission, respectively.[14, 27, 95] There are few studies which discuss the dietary behaviors in patients with traumatic brain injury, yet the role of nutrition is essential for optimal recovery.

CHAPTER 3

ENERGY-DENSE DIETS LOWER IN PROTEIN, ANTIOXIDANTS, AND OMEGA 3 FATTY ACIDS IN US ADULTS WITH A SELF-REPORTED HEAD INJURY WITH LOSS OF CONSCIOUSNESS[1]

[1] Barta, J., Kim, S., Park, H.J. Submitted to *Nutrition Research*.

3.1 ABSTRACT

Head injury results in thousands of hospitalizations and deaths each year in the US, yet there are no current dietary recommendations after head injury. We assessed the dietary nutrient intakes in the population with self-reported head injury with loss of consciousness (HIC) utilizing National Health and Nutrition Examination Survey (NHANES) data. Secondary data analysis was performed on participants aged 40 and over from the NHANES 2011-2014 surveys with and without head injury with loss of consciousness. The nutrient intake differences were measured between HIC and those who reported no head injury with loss of consciousness (No-HIC) based on average daily nutrient intakes. We further compared these nutrient intakes to nutrition guidelines to determine diet quality. SPSS software was used to perform Mann Whitney U-Tests comparing nutrient intakes of the HIC to No-HIC groups. HIC reported higher intakes of calories, protein, carbohydrates, sugar, fat, and various vitamins and minerals, including many with antioxidant properties, compared to No-HIC. After adjusting for energy intakes, HIC consumed more sugar, Vitamin B6, caffeine, moisture, and multiple saturated fatty acids than No-HIC, and intakes of protein, fiber, many B vitamins, nutrients with antioxidant properties, minerals, and omega-3 fatty acids were lower in HIC compared to No-HIC. HIC consumed higher intakes compared to age- and gender-specific recommendations than No-HIC for 10 nutrients, including calories, carbohydrates, calories from saturated fat, and sodium, further indicating pro-inflammatory energy-dense diets. Therefore, nutrition guidelines are essential to educate the head injury population to improve future health outcomes post-injury.

Keywords: traumatic brain injury, diet, inflammation, antioxidant, NHANES

3.2 INTRODUCTION

Head injuries tax the healthcare system and cause thousands of hospitalizations and deaths per year.[91, 96] Traumatic brain injury (TBI) is a state of altered brain functionality resulting from a jolt or blow to the head, leading to memory loss, cognitive impairment, loss of consciousness, and long-term disability.[1] TBI can cause chronic neuroinflammation, leading to neurodegenerative diseases and further healthcare costs.

The Centers for Disease Control and Prevention conduct the National Health and Nutrition Examination Survey (NHANES) every other year, which collects health information and two days of dietary recalls from Americans.[97] The 2011-2012 and 2013-2014 surveys included a question on whether participants had experienced a head injury with loss of consciousness. In previous NHANES research, head injury with loss of consciousness was more highly correlated with heavy drinking, smoking, taste and smell disturbances, and olfactory alterations compared to those who reported no head injury with loss of consciousness.[16, 98, 99]

Nutrition status is key in recovery from head injury by promoting anti-inflammatory pathways, cognitive improvements, and enhanced tissue repair. Head injury induces a hypermetabolic state from injury recovery, inflammation, and the release of stress hormones. Energy and protein needs can increase by up to 200% during stress.[8] Antioxidants and anti-inflammatory nutrients play a potential role in reducing oxidative damage and helping to maintain cognitive function.[7, 10, 13, 18]

Poor dietary intakes are a risk factor for chronic disease, yet the Americans generally consume diets which deviate from the target dietary patterns. The Dietary Guidelines for Americans reported that the average American scored only a 59 out of 100 on the Healthy Eating

Index, which measures adherence to the Dietary Guidelines in a person's diet.[100] To determine the diet quality of NHANES participants who answered CSQ240, this study analyzed their dietary intakes per age- and gender-specific Dietary Reference Intakes (DRI). DRIs include Recommended Dietary Allowance (RDA), Adequate Intake (AI), Estimated Energy Requirement (EER), and Chronic Disease Risk Reduction Level (CDRR).[100] RDA is an intake level which should meet or exceed the nutrient needs for 97 to 98 percent of individuals within a given group.[101] AI is a measure of the mean nutrient intake from a group of presumably healthy people.[101] EER provides an age- and gender-specific energy recommendation, with estimates established for different activity levels.[100] CDRR is a recommended intake limit to reduce risk of chronic disease.[100] Therefore, comparing nutrient intakes to nutrition guidelines can indicate whether participants are consuming diets which optimize health outcomes post-injury.

However, there are currently no dietary recommendations given to head injury patients, which creates a lack of uniform dietary habits. We aimed to assess the dietary nutrient intakes and diet quality of those who experienced head injury with loss of consciousness and compared it to that of their counterparts. The findings from this study will provide preliminary information on the dietary behaviors and quality in this population.

3.3 METHODS AND MATERIALS

3.3.1 *NHANES datasets*

All data collected for the study was analyzed from the NHANES database, a survey conducted by the National Center for Health Statistics out of the CDC. NHANES data is released every 2 years.[97] Participants included adults aged 40 years or older who participated in the Taste & Smell (CSQ) questionnaire and responded "yes" or "no" to the question CSQ 240, "Have you ever had a loss of consciousness because of a head injury?"[102] All participants who

responded “yes” or “no” to CSQ 240 were included in this study. The datasets of 2 survey cycles, 2011-2012 and 2013-2014, were analyzed. Weighted nutrient intake was analyzed using Day 1 and 2 Total Nutrient Intakes (DR1TOT, DR2TOT). Height and weight were self-reported in the WHQ Weight History (WHQ) questionnaire and used to calculate BMI.

3.3.2 *Diet quality in the population with head injury with loss of consciousness*

Diet quality was measured as a proportion of average daily nutrient intake per Dietary Reference Intake (DRI), sourced from the Dietary Guidelines for Americans.[100] The nutrient intakes were the average of DR1TOT and DR2TOT datasets. 26 nutrient intakes were divided by their age- and gender-specific DRI. The nutrient intakes (% DRI) were reported as mean ± SEM for the two groups. Nutrients were reported as % DRI, which includes RDA, EER, CDRR, and AI. Recommendations from the Dietary Guidelines for Americans were used for fiber (14g/1000 kcals), and saturated fat (<10% calories from saturated fat).[100]

3.3.3 *Statistical analyses*

After noting nutrient differences between groups, the nutrient intakes were adjusted to account for calorie differences by dividing the average nutrient intake by average calorie intake. The participants with a head injury with loss of consciousness (HIC) were compared to those participants with no history of a head injury with loss of consciousness (No-HIC) using Chi-squared or a Mann Whitney U-Test via SPSS software. The significance was determined at $p < .05$.

3.4 RESULTS

3.4.1 *Demographics*

Of the 7,399 Americans aged 40 or older who responded to yes or no to the question CSQ240, 12.8% reported a head injury with loss of consciousness. In **Table 1**, the HIC group

was comprised of 18.5% more males than females, compared to the female-majority in the No-HIC group ($p < .001$). The HIC group was 1.2 years younger than the No-HIC ($p = .015$). The HIC group reported higher body weights ($p < .001$) and heights ($p < .001$) than the No-HIC, however, BMI was not significantly different between the groups ($p = .771$), likely due to the higher male population in the HIC group.

Table 1. Demographic Characteristics of Participants in NHANES 2011-2014 Who Answered CSQ 240 (Weighted n = 7399)

Group	**HIC[1] (Weighted n = 948)**	**No-HIC[2] (Weighted n = 6,451)**	
	Mean (SEM)	**Mean (SEM)**	***p*-Value**
Gender			
Male, n (%)	556 (58.60)	2890 (44.80)	0.000
Female, n (%)	380 (40.10)	3560 (55.20)	
Age (years)	56.78 (0.42)	57.98 (0.24)	0.015
Current self-reported height (in)	67.60 (0.18)	66.47 (0.08)	0.000
Weight			
Current self-reported weight (lb.)	187.04 (2.25)	176.69 (.56)	0.000
Self-reported weight 1 year ago (lb.)	188.35 (2.75)	181.41 (0.90)	0.000
BMI (kg/m^2)	28.68 (0.30)	28.47 (0.11)	0.771

[1]Answered "yes" to the question CSQ 240 "Have you ever had a loss of consciousness because of a head injury?" [2]Answered "no" to the question CSQ 240 "Have you ever had a loss of consciousness because of a head injury?"

3.4.2 *Daily nutrient intakes in the participants with the history of head injury with loss of consciousness*

Nutrient intakes were calculated from two days of 24-hour dietary recall. **Table 2** shows the nutrients that were significantly different between the HIC and No-HIC groups. Average unweighted energy intake was 107.9 kcal per day higher in the HIC group compared to the No-

HIC group ($p < .001$). In addition to energy, intake of protein, carbohydrate, total sugar, fat, and various vitamins and minerals, including Vitamin E, many B vitamins, and retinol, were seen to be significantly different in the two groups. Protein and total fat intakes were significantly higher in the HIC group compared to the No-HIC group. HIC consumed 15.1 more grams of carbohydrates and 11 more grams of sugar per day than No-HIC.

Intakes of many B and fat-soluble vitamins were higher in the HIC population also, as shown in **Table 2**. Intakes of thiamin ($p = .015$), riboflavin ($p < .001$), niacin ($p = .011$), Vitamin B6 ($p = .004$), and choline ($p = .002$) were significantly higher in the HIC group compared to the No-HIC group. Consumption of lipid-soluble vitamins, including Vitamin E ($p < .001$) and added alpha-tocopherol ($p = .047$) were higher in HIC compared to No-HIC. Consumption of retinol was 15.5% higher in HIC than No-HIC.

Similarly, multiple minerals and dietary components were significantly higher in the HIC population compared to the No-HIC population (**Table 2)**. Consumption of calcium ($p < .001$), copper ($p = .010$), iron ($p = .014$), magnesium ($p < .001$), phosphorous ($p = .001$), potassium ($p < .001$), sodium ($p = .009$), and zinc ($p < .001$) were higher in the HIC group than the No-HIC group. HIC consumed 19.9% more caffeine than No-HIC. Intake of the dietary components theobromine ($p = .034$) and moisture ($p < .001$) were also higher in HIC compared to No-HIC group.

Table 2. Dietary Nutrient Intakes by 2011-2014 NHANES Participants Significantly Different Between Head Injury with Loss of Consciousness and No Head Injury with Loss of Consciousness Groups at $p < .05$ (Weighted n = 7399)

Nutrient	**HIC[1] (Weighted n = 948)**	**No-HIC[2] (Weighted n = 6,451)**	
	Mean (SEM)	**Mean (SEM)**	***p*-Value**
Energy (kcal/day)	**2110.42 (31.86)**	2002.50 (18.32)	0.000
Protein (g/day)	**81.72 (1.03)**	79.38 (0.52)	0.048
Carbohydrate (g/day)	**253.74 (6.02)**	238.67 (2.06)	0.000
Total sugars (g/day)	**113.87 (3.98)**	103.03 (1.14)	0.000
Fat (g/day)	**81.91 (1.10)**	77.17 (0.84)	0.000
Thiamin (Vitamin B1) (mg/day)	**1.63 (0.03)**	1.56 (0.01)	0.015
Riboflavin (Vitamin B2) (mg/day)	**2.28 (0.06)**	2.09 (0.02)	0.000
Niacin (mg/day)	**25.97 (0.47)**	24.41 (0.19)	0.011
Vitamin B6 (mg/day)	**2.26 (0.07)**	2.05 (0.02)	0.004
Total choline (mg/day)	**340.99 (5.47)**	330.06 (3.747)	0.002
Vitamin E, alpha-tocopherol (mg/day)	**9.51 (0.25)**	8.74 (0.16)	0.000
Added alpha-tocopherol (Vitamin E) (mg/day)	**0.95 (0.13)**	0.73 (0.07)	0.047
Retinol (mcg/day)	**493.19 (45.43)**	416.76 (6.13)	0.000
Calcium (mg/day)	**968.58 (21.96)**	923.46 (9.24)	0.000
Copper (mg/day)	**1.37 (0. 073)**	1.25 (0.01)	0.010
Iron (mg/day)	**15.09 (0.38)**	14.60 (0.13)	0.014
Magnesium (mg/day)	**318.30 (7.62)**	300.59 (3.44)	0.000
Phosphorus (mg/day)	**1403.57 (22.45)**	1340.768 (11.217)	0.001
Potassium (mg/day)	**2828.78 (45.26)**	2718.02 (31.53)	0.000
Sodium (mg/day)	**3462.86 (44.77)**	3352.74 (26.32)	0.009
Zinc (mg/day)	**11.42 (0.27)**	10.82 (010)	0.000
Theobromine (mg/day)	**42.16 (3.26)**	36.55 (1.41)	0.034
Caffeine (mg/day)	**220.77 (14.35)**	176.93 (4.53)	0.000
Moisture (g/day)	**3161.01 (76.08)**	2889.31 (39.53)	0.000
Saturated fatty acids (g/day)	**26.27 (0.52)**	24.66 (0.30)	0.000
Monounsaturated fatty acids (g/day)	**29.19 (0.45)**	27.38 (0.32)	0.000
Polyunsaturated fatty acids (g/day)	**19.39 (0.27)**	18.38 (0.22)	0.002
SFA 4:0 (Butanoic) (g/day)	**0.53 (0.02)**	0.49 (0.01)	0.003
SFA 6:0 (Hexanoic) (g/day)	**0.32 (0.02)**	0.29 (0.01)	0.005
SFA 8:0 (Octanoic) (g/day)	**0.26 (0.01)**	0.24 (0.00)	0.000
SFA 10:0 (Decanoic) (g/day)	**0.50 (0.02)**	0.46 (0.01)	0.000
SFA 12:0 (Dodecanoic) (g/day)	**0.82 (0.04)**	0.75 (0.02)	0.000
SFA 14:0 (Tetradecanoic) (g/day)	**2.18 (0.08)**	2.04 (0.03)	0.000
SFA 16:0 (Hexadecanoic) (g/day)	**14.19 (0.22)**	13.37 (0.15)	0.000
SFA 18:0 (Octadecanoic) (g/day)	**6.47 (0.12)**	6.07 (0.08)	0.000
MFA 16:1 (Hexadecenoic) (g/day)	**1.06 (0.02)**	1.01 (0.01)	0.004
MFA 18:1 (Octadecenoic) (g/day)	**27.01 (0.42)**	25.18 (0.30)	0.000
MFA 20:1 (Eicosenoic) (g/day)	**0.31 (0.01)**	0.30 (0.01)	0.046
PFA 18:2 (Octadecadienoic) (g/day)	**17.14 (0.24)**	16.18 (0.20)	0.001
PFA 18:4 (Octadecatetraenoic) (dg/day)[3]	0.09 (0.01)	**0.11 (0.00)**	0.013
PFA 20:5 (Eicosapentaenoic) (g/day)	0.026 (0.00)	**0.033 (0.00)**	0.000
PFA 22:6 (Docosahexaenoic) (g/day)	0.06 (0.01)	**0.07 (0.00)**	0.013

Nutrients significant at $p < .05$ were included in this table. Nutrients excluded based on p-value: fiber, cholesterol, Vitamin A, alpha-carotene, beta-carotene, beta-cryptoxanthin, lycopene, lutein

+ zeaxanthin, total folate, folic acid, food folate, folate DFE, Vitamin B12, added Vitamin B12, Vitamin C, Vitamin D, Vitamin K, selenium, alcohol, MFA 22:1, PFA 18:3, PFA 20:4, and PFA 22:5.
[1]Answered "yes" to the question CSQ 240 "Have you ever had a loss of consciousness because of a head injury?"
[2]Answered "no" to the question CSQ 240 "Have you ever had a loss of consciousness because of a head injury?" [3]Octadecatetraenoic acid was converted from g/day to dg/day.

Intakes of most saturated fatty acids, monounsaturated fatty acids (MUFA), and polyunsaturated fatty acids (PUFA) were higher in the HIC group compared to the No-HIC group. HIC consumed 6.1% more saturated fatty acids, 6.2% more MUFA, and 5.2% more PUFA than No-HIC. Further breakdown of the saturated fatty acids in the diets showed higher intakes of butanoic acid ($p = .003$), hexanoic acid ($p = .005$), octanoic acid ($p < .001$), decanoic acid ($p < .001$), dodecanoic acid ($p < .001$), tetradecanoic acid ($p < .001$), hexadecenoic acid ($p < .001$), and octadecanoic acid ($p < .001$) in HIC than No-HIC. Intakes of MUFA followed the same pattern, with higher intakes of hexadecenoic acid ($p = .004$), octadecenoic acid ($p < .001$), and eicosenoic acid ($p = .046$) in HIC compared to No-HIC. HIC also consumed more of the PUFA octadecadienoic acid ($p = .001$) than No-HIC. Intakes of only three fatty acids were lower in HIC compared to No-HIC. HIC consumed 18.8% less octadecatetraenoic acid, 21.2% less eicosapentaenoic acid (EPA), and 14.3% less docosahexaenoic acid (DHA) ($p = .013$) than No-HIC.

Intakes of fiber, cholesterol, Vitamin A, alpha-carotene, beta-carotene, beta-cryptoxanthin, lycopene, lutein + zeaxanthin, folate, folic acid, food folate, folate DFE, Vitamin B12, added Vitamin B12, Vitamin C, Vitamin D, Vitamin K, selenium, alcohol, docosenoic acid, octadecatrienoic acid, eicosatetraenoic acid, and docosapentaenoic acid were not significantly different between the two groups.

Age and gender are the major factors that affect nutrients need and recommendations.[100] We therefore calculated diet quality considering age- and gender-specific dietary reference intake (DRI). The percentage of each nutrient intake per its Dietary Reference Intake (%DRI) was calculated for 26 nutrients whose DRIs are currently available. As seen in **Table 3**, Participants in HIC consumed higher intakes (%DRI) of calories, carbohydrates, percent of calories from saturated fat, calcium, iron, phosphorous, sodium, Vitamin E, Riboflavin, and Vitamin B6, compared to No-HIC. HIC consumed 95.8% of their DRI for calories, and No-HIC consumed 92.4% of their DRI. Both groups were consuming close to double the recommended intake of carbohydrates, as HIC consumed 195.0% of their DRI for carbohydrates and No-HIC consumed 180.9% of their DRI. Although both groups consumed intakes of sodium which were >40% above the recommended intake, intakes among HIC compared to the recommendation were higher than No-HIC. Participants in HIC consumed slightly more Vitamin E compared to No-HIC, but both groups were consuming Vitamin E at only 59-66% of the recommendation.

Table 3. Nutrient Intakes Per Gender- and Age-Specific Dietary Recommendations Among NHANES 2011-2014 Participants Who Answered CSQ 240 (n = 7,399)

	HIC[1] (n = 948)	**No-HIC[2] (n = 6,451)**	
Nutrient (% DRI)[3]	**Mean (SEM)**	**Mean (SEM)**	***p*-Value**
Calories	**95.8 (1.3)**	92.4 (0.5)	**0.028**
Protein	153.3 (2.2)	152.5 (0.8)	0.737
Carbohydrate	**195.0 (3.1)**	180.9 (1.0)	**0.000**
Fiber[4]	72.3 (1.4)	71.1 (0.5)	0.384
Saturated fat[4] (% kcals)	**108.7 (1.1)**	105.6 (0.4)	**0.014**
PFA 18:2	124.2 (2.5)	119.3 (0.9)	0.158
PFA 18:3	128.8 (3.0)	127.0 (1.1)	0.754
Calcium	**90.1 (1.7)**	81.9 (0.6)	**0.000**
Iron	**172.7 (3.3)**	162.3 (1.2)	**0.002**
Magnesium	81.7 (1.2)	79.4 (0.5)	0.105
Phosphorous	**193.9 (2.8)**	183.3 (1.0)	**0.001**
Potassium	89.9 (1.2)	87.7 (0.5)	0.089
Sodium	**149.2 (2.3)**	142.2 (0.8)	**0.009**
Zinc	114.3 (2.0)	110.5 (0.7)	0.151
Vitamin A, RAE	80.1 (2.6)	79.8 (1.0)	0.920
Vitamin E[5]	**66.2 (1.9)**	58.7 (0.6)	**0.001**
Vitamin D	32.5 (1.1)	31.6 (0.5)	0.348
Vitamin C	114.1 (3.5)	111.9 (1.4)	0.981
Thiamin (Vitamin B1)	135.7 (2.1)	131.4 (0.8)	0.074
Riboflavin (Vitamin B2)	**175.7 (2.9)**	162.2 (1.0)	**0.000**
Niacin	163.6 (2.7)	157.9 (1.0)	0.115
Vitamin B6	**143.2 (3.1)**	132.9 (1.0)	**0.017**
Vitamin B12[5]	247.6 (10.4)	226.7 (2.9)	0.106
Choline	67.9 (1.1)	66.4 (0.4)	0.199
Vitamin K	113.0 (4.7)	124.4 (3.6)	0.091
Folate, DFE	132.1 (2.7)	125.1 (1.0)	0.079

[1]Answered "yes" to the question CSQ 240 "Have you ever had a loss of consciousness because of a head injury?"

[2]Answered "no" to the question CSQ 240 "Have you ever had a loss of consciousness because of a head injury?"

[3]Nutrients were calculated as nutrient intakes divided by DRI. DRI used: EER for calories; RDA for protein, carbohydrate, calcium, iron, magnesium, phosphorous, zinc, Vitamin A, Vitamin E, Vitamin D, Vitamin C, thiamin, riboflavin, niacin, Vitamin B6, Vitamin B12, and folate; AI for PFA 18:2, PFA 18:3, potassium, choline, and Vitamin K; CDRR for sodium.

[4]DGA values were used for fiber (14g/1000 kcals), and saturated fat (<10% of calories from saturated fat).

[5]Daily Vitamin E was calculated as a combination of the variables Vitamin E (alpha-tocopherol) + Added alpha-tocopherol. Daily Vitamin B12 was calculated as a combination of the variables Vitamin B12 + Added Vitamin B12.

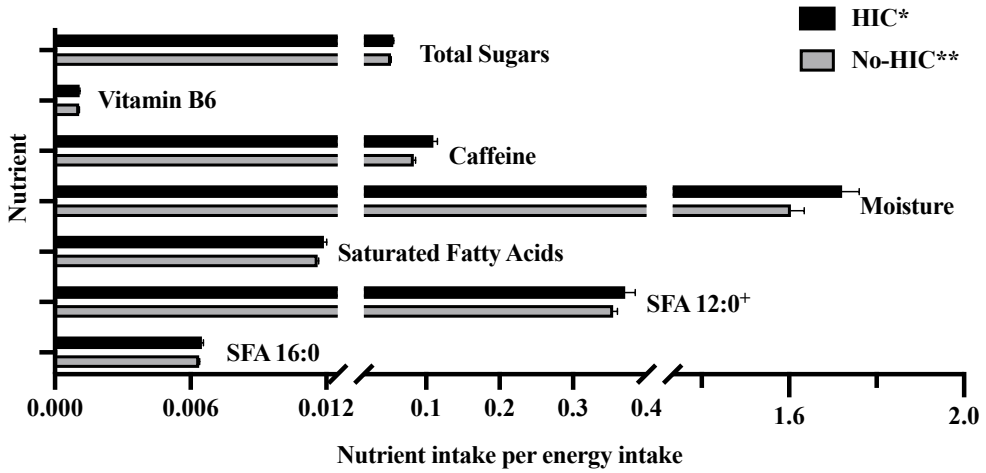
3.4.4 *Dietary nutrient density comparisons between the participants with head injury with loss of consciousness* and those without head injury with loss of consciousness

Nutrient density describes the ratio of nutrients to energy in a product, calculated in this study as nutrient intake divided by calorie intake per day.[103] **Figure 1a** shows nutrients which were consumed more in the HIC group compared to the No-HIC group after adjusting for energy intake. Sugar intake was higher in the HIC group than the No-HIC group after energy adjustment ($p = .042$), as was Vitamin B6 intake ($p = .023$). Caffeine consumption was 24.1% higher in HIC ($p < .001$) and moisture intake was 6.8% higher in HIC ($p = .002$) compared to No-HIC when adjusted for energy intake. Similarly, intake of total saturated fatty acids ($p = .029$), and the saturated fatty acids dodecanoic acid ($p = .013$) and hexadecanoic acid (p = .042) were significantly higher in the HIC group compared to the No-HIC group when adjusted for energy intake.

Figure 1b shows the nutrients which were consumed less in the HIC group than the No-HIC group after energy intake adjustment. Intake of protein ($p < .001$) and dietary fiber ($p = .024$) were lower in the HIC group than the No-HIC group when adjusted for energy intake. Micronutrients consumed less in HIC than No-HIC when adjusted for energy intake included thiamin ($p = .011$), niacin ($p = .009$), folate ($p = .009$), folate DFE ($p = .020$), food folate ($p = .011$), Vitamin C ($p = .045$), alpha-carotene ($p = .026$), beta-carotene ($p = .011$), Lutein + zeaxanthin ($p = .016$), Vitamin K ($p = .003$), copper ($p = .035$), iron ($p = .026$), phosphorous ($p = .041$), selenium ($p < .001$), and sodium ($p = .004$).

Consumption of multiple fatty acids was lower in HIC than No-HIC when adjusted for calories. Intakes of omega-3 fatty acids octadecatrienoic acid ($p = .028$), octadecatetraenoic acid ($p = .001$), and docosapentaenoic acid ($p = .003$) and were lower in the HIC group than the No-HIC group after energy adjustment. Consumption of eicosapentaenoic acid was 33.9% lower ($p < .001$) and docosahexaenoic acid was 24.1% lower ($p < .001$) in the HIC group than the No-HIC group when adjusted for energy intake.

a.


HIC*
No-HIC**
Total Sugars
Vitamin B6
Caffeine
Moisture
Saturated Fatty Acids
SFA 12:0+
SFA 16:0
Nutrient
0.000
0.006
0.012
0.1
0.2
0.3
0.4
1.6
2.0
Nutrient intake per energy intake

b.

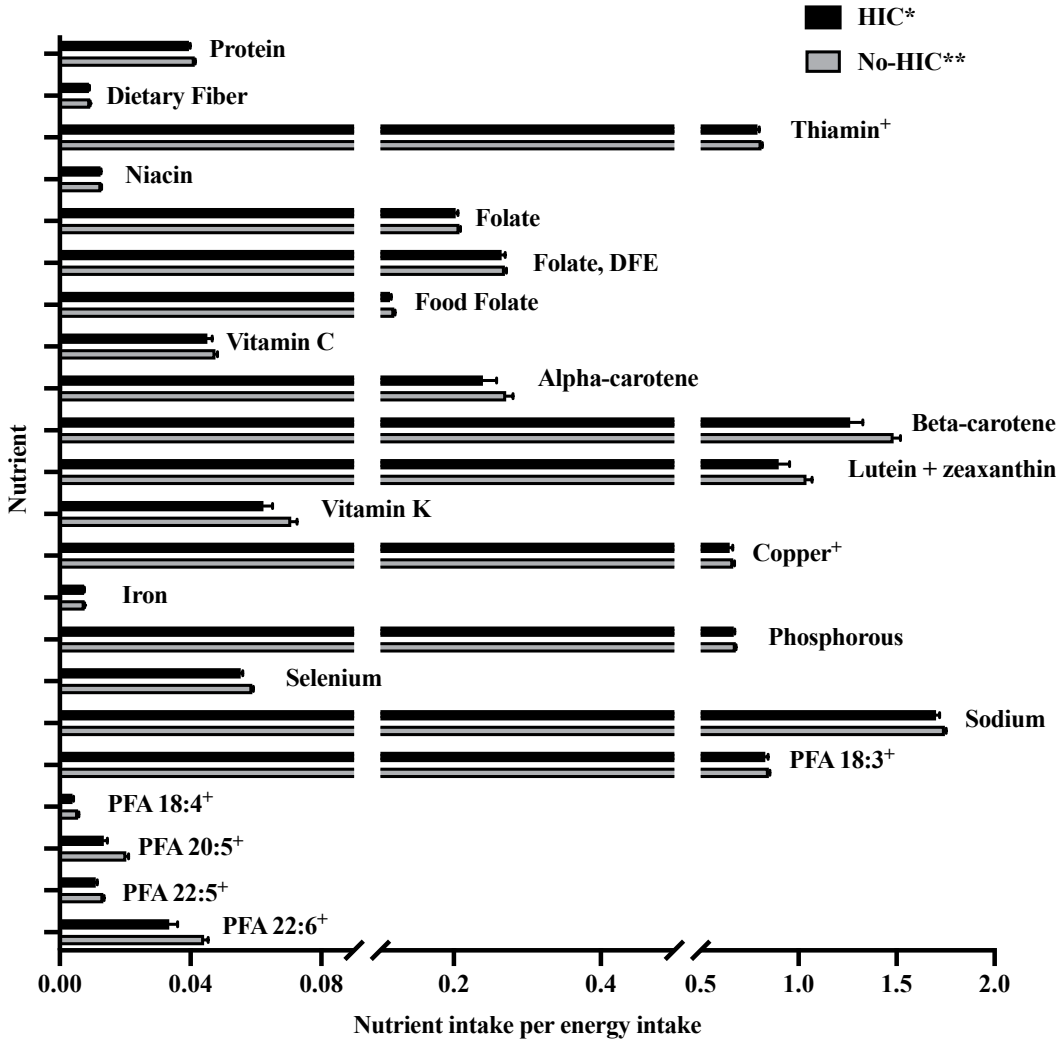

HIC*
No-HIC**
Protein
Dietary Fiber
Thiamin+
Niacin
Folate
Folate, DFE
Food Folate
Vitamin C
Alpha-carotene
Beta-carotene
Lutein + zeaxanthin
Vitamin K
Copper+
Iron
Phosphorous
Selenium
Sodium
PFA 18:3+
PFA 18:4+
PFA 20:5+
PFA 22:5+
PFA 22:6+
Nutrient
0.00
0.04
0.08
0.2
0.4
0.5
1.0
1.5
2.0
Nutrient intake per energy intake

Figure 1. Nutrient Intake Adjusted by Calories in the 2011-2014 NHANES Participants Who Answered CSQ 240 (n = 7399) **a)** Nutrients that were consumed in significantly higher amounts in the HIC group than No-HIC group **b)** Nutrients that were consumed in significantly lower amounts in the HIC group than the No-HIC group.
*Participants in the Head Injury group answered "yes" to the question CSQ 240 "Have you ever had a loss of consciousness because of a head injury?" (n = 948).
**Participants in the "No Head Injury" group answered "no" to the question CSQ 240 "Have you ever had a loss of consciousness because of a head injury?" (n = 6,451).
[+]Units: mg/kcal for SFA 12:0 (Dodecanoic), PFA 18:3 (Octadecatrienoic), PFA 18:4 (Octadecatetraenoic), PFA 20:5 (Eicosapentaenoic), PFA 22:5 (Docosapentaenoic), and PFA 22:6 (Docosahexaenoic), and mcg/kcal for Thiamin (Vitamin B1) and Copper.

3.5 Discussion

Poor diet quality impedes recovery from head injury, but consuming a diet rich in protein, antioxidants, and the anti-inflammatory fatty acids can improve head injury health outcomes. While research remains largely inconclusive, diets high in protein and calories with adequate antioxidant and DHA intake may ameliorate oxidative stress and inflammation after head injury.[104] The hypothesis was proved true, there were significant differences between the groups' nutrient intakes. The HIC group reported higher intake of 40 of all 65 nutrients evaluated compared to the No-HIC group. HIC consumed higher nutrient intakes compared to DRI for calories, carbohydrates, and 8 other nutrients. Once adjusted for energy intakes, the HIC group consumed higher amounts of sugars, Vitamin B6, caffeine, moisture, saturated fatty acids, dodecanoic acid, and hexadecanoic acid compared to the No-HIC group.

Dietary Reference Intakes (DRI) are nutrient intake goals specific to age and gender released by the Food and Nutrition Board of the Institute of Medicine, to determine an appropriate intake of nutrients to avoid both deficiency and toxicity. DRI include estimated average requirements (EAR), recommended dietary allowances (RDA), adequate intakes (AI), and tolerable upper limits (UL). As DRI are specific to the age and gender of a person,

individualized energy and nutrient needs were accounted for in our study when we adjusted nutrient intakes by DRI. Therefore, DRI was used in this study as a standard for the estimated appropriate nutrient intakes each participant should consume. The comparison of participant nutrient intakes to their DRI provided insights into whether participants were consuming optimal diets for head injury recovery and general healthfulness.

The HIC group had higher calorie intakes compared to the No-HIC group, potentially caused by the larger percentage of males in the group, who have higher energy requirements than women.[100] However, the energy difference between the HIC and No-HIC groups was seen even after adjusting by the gender-specific energy recommendations.[100] We divided the individual energy intakes of each participant by their respective calorie needs, based on age and gender. The HIC group consumed 95.8% and the No-HIC group consumed 92.4% of recommended energy intakes. The HIC group did not show a correlation between percent calories consumed per recommended energy intake and BMI ($r = .032$, $p = .374$), while the No-HIC group did show a correlation ($r = .057$, $p < .001$). This may be caused by the small number of participants in the HIC group, indicating a need for further studies to determine whether BMI and percent calories consumed per recommended intake are related.

In the current study, carbohydrate intakes in both HIC and No-HIC were almost twice the recommended intake amount. Intakes of sugar and saturated fat were higher in the HIC group compared to those in No-HIC group, when adjusted for energy intake. While HIC was consuming significantly higher intakes of saturated fat compared to No-HIC, both groups were exceeding the recommended intake levels by 5-8%. Diets higher in saturated fat and sugar have been seen to reduce the beneficial proteobacteria in the gut, which can trigger a pro-inflammatory response in the brain via the brain-gut axis.[105] The HIC group reported higher

intakes of total sugars and lower intakes of fiber, therefore appearing to choose less desirable carbohydrate sources.

Diets high in protein, antioxidants, and omega-3 fatty acids may improve head injury outcomes.[6, 8, 9, 11, 13, 14, 18, 106] Protein is essential after head injury, as increased energy expenditure and protein catabolism occurs during recovery.[8, 107] Adequate nutrition is tied with physical functionality 6 months into recovery, so lower protein intake in the HIC group compared to the No-HIC is not desirable.[107] However, after comparing intakes to dietary recommendations, both groups consumed exceeded the recommendations for protein by over 50%, which should meet the increased needs for protein post-injury in HIC.

Most antioxidant intakes were lower or equal in the HIC group compared to the No-HIC group in our study after adjusting for energy intakes. These equivalent antioxidant intakes may be due to a lack of consumption of fat-soluble vitamins daily, as these vitamins can be stored in the liver and adipose tissue.[108] Antioxidants may be neuroprotective after head injury by reducing lipid peroxidation and improving synaptic plasticity.[13, 14] Selenium may have neuroprotective effects that prevent oxidative damage after injury, yet the HIC group had lower intakes than the No-HIC group after adjusting for energy.[13] Lutein has potential for reducing the post-injury inflammation, yet lutein + zeaxanthin intake was consumed less in HIC compared to No-HIC when adjusted for energy intake.[6] Antioxidants Vitamin C, alpha-carotene, and beta-carotene were all consumed less in HIC compared to No-HIC when adjusted for energy intakes as well. Moreover, intakes of retinol, Vitamin A, and Vitamin E did not differ between the two groups when adjusted for energy intake. Participants in both HIC and No-HIC met the recommendations for the antioxidant Vitamin C, yet HIC consumed only 80% of the Vitamin A recommendation and 66% of the Vitamin E recommendation. Riboflavin can act as an

antioxidant, and intakes in both HIC and No-HIC exceeded recommendations.[109] However, antioxidant recommendations and antioxidant-rich diet educations after TBI are still critical.

Omega 3 fatty acids have been reported to reduce damage from secondary injury by reducing inflammation and enhancing neurogeneration, resulting in improved neurocognition in patients with head injury.[9, 110, 111] Consumption of DHA after head injury may improve health outcomes, reduce apoptosis and long-term oxidative stress, and improve cognition.[10-12] However, the current study shows that intakes of EPA, DHA, and their precursor alpha-linolenic acid (PFA 18:3) were lower in the HIC group than the No-HIC group.[112] Although there is increased public attention on the impact of DHA on neurocognition, the DHA intake in the HIC group was lower than that of the no-HIC group in our analysis, indicating the need for public nutrition education in this population.

In the US, sodium intake has gained attention due to its strong relationship with increased risk of chronic diseases.[44, 78] The US population consumes approximately 48% more sodium than the recommendation regardless age and gender, according to the Food and Drug Administration.[113] Consistently in our study, the sodium intake of both HIC and No-HIC exceeds the recommendations by >40%. Moreover, the sodium intakes were significantly lower in HIC compared to No-HIC after adjusting for energy intake. Considering the strong association between sodium intake and risk of inflammatory chronic diseases, underlying inflammatory conditions may worsen TBI health outcomes with higher intake of dietary sodium in these population.[44]

Similarly, intakes of Vitamin B6 and calcium compared to recommendations were higher in HIC than No-HIC. Dietary intakes of Vitamin B6 or calcium have tangential benefits in brain injury recovery, although they might not directly improve the wound or mitigate secondary

injury. Vitamin B6 is critical for macronutrient metabolism and deficient intakes may increase risk of cognitive decline.[114, 115] Vitamin B6 also plays a key role in decarboxylation of L-amino acids, which can yield amines to function as neurotransmitters.[114] As calcium is critical to coagulation, hypocalcemia may by correlated with increased morbidity and mortality in trauma patients, such as those with TBI.[116] There is a link observed between calcium stores and neuronal functions, which may even correlate with the development of neurodegenerative disease.[117] While there is a need for further studies on the impact of these nutrients on TBI, increased intakes of Vitamin B6 and calcium may benefit recovery in TBI patients.

Alcohol consumption in NHANES participants with head injury was reported previously using data from the questionnaire ALQ, with the question ALQ151, which asked, "Was there ever a time or times in your life when you drank 4-5 or more drinks of any kind of alcoholic beverage almost every day?"[16] Schneider et al. found that 24.7% of those with head injury in this NHANES population reported being a current or former heavy drinker.[16] Consistently, our study showed that the alcohol intakes in those with head injury tended to be higher than counterparts. Our study was the first study to our knowledge which included alcohol consumption from 2-days of 24 hour-dietary recalls in NHANES participants with head injury with loss of consciousness. Alcohol consumption can disrupt the gut microbiota and cause intestinal inflammation.[118] Chronic release of pro-inflammatory cytokines in the body from alcohol consumption can induce oxidative damage in the brain, which could compound with the secondary damage occurring from head injury.[119] Our study also shows that HIC group consumed higher amount of caffeine than their counterparts, based on the 2 days of 24 hour-dietary recalls. Similarly, Schneider et al. reported higher alcohol intakes in the population with HIC, a correlation with sleeping less than 6 hours per night in HIC, and a diagnosis of sleep

disorders in HIC, all of which could drive increased caffeine intakes.[16] Sleep deprivation is common in head injury, and increased caffeine intakes in the HIC group could be correlated to poor sleep.[16, 120, 121] Head injury can increase fatigue throughout the day, potentially causing the HIC group to consume more caffeine.[121] Increased caffeine and a trend in increased alcohol intakes, correlated with inflammation and poor sleep, may inhibit recovery from head injury.

Limitations in this study included the lack of medical history on participant recovery status and injury severity, and an absence of data on participants who experienced a head injury without losing consciousness. The study design further prevented evaluations of causality been occurrence of head injury and food consumption. While we assessed antioxidant intakes, there was no physiological measurement utilized to determine antioxidant status in plasma or tissue. Further, food intakes were acquired via 24-hour recalls, and food intakes in this population may have changed based on day of the week, and may have included recall bias, thus intakes may have been over- or under-estimated. Similarly, dietary recalls may be inaccurate in those with head injury due to the nature of the injury and its impact on memory function. Finally, we only adjusted for age- and gender-specific DRI, and no other confounding factors such as occupation, which may impact our findings.

This study proves the need for dietary recommendations and educations to patients with head injuries with loss of consciousness. The pro-inflammatory diets in HIC, which diverge frequently from dietary guidelines, indicate a lack of awareness of beneficial nutrients for recovery, as there are no guidelines. Future studies are needed to investigate the optimal dietary interventions in the HIC population.

CHAPTER 4

IMPACT OF SELF-REPORTED ADHERENCE TO WEIGHT LOSS DIETS ON NUTRIENT INTAKES IN US ADULTS WITH A SELF-REPORTED HEAD INJURY WITH LOSS OF CONSCIOUSNESS[2]

[2] Barta, J., Kim, S., Park, H.J. To be submitted to *Journal of the Academy of Nutrition and Dietetics*.

4.1 ABSTRACT

Enhanced diet quality has been suggested to play an important role in improved recovery from head injury, but Chapter 3 indicated that those with history of head injury (HIC) follow energy-dense diets which are low in anti-inflammatory nutrients. More importantly, the poor behavioral flexibility in dietary behavior following an intended diet modification has been widely noticed. In this chapter, we analyzed the impact of self-reported adherence to weight loss diets on diet quality within HIC and those without history of head injury (No-HIC) to assess resistance to dietary change in this population. Interestingly, only Vitamin E intake (% DRI) was different between those in HIC who reported adhered to a weight loss diet and those in HIC who did not report adhering to a weight loss diet. There were no calorie or macronutrient intake (% DRI) differences between dieters and non-dieters. Among No-HIC, dieters consumed 9 nutrient intakes (%DRI) at significantly different amounts than non-dieters, indicating a stronger adherence to dietary modifications compared to HIC. Taken together, the population with head injury appears to be resistant to dietary changes, indicating a possibility of poor behavioral flexibility in this population.

4.2 INTRODUCTION

Traumatic brain injury is a major health crisis in America, yet there are no proper guidelines for nutrition interventions post-injury. Nutrients which possess antioxidant functions such as Vitamin E and selenium may reduce oxidative stress and lipid peroxidation, maintain cognitive function, and reduce risk of neuroinflammation-related neurodegenerative disease.[13, 14, 18] Anti-inflammatory nutrients, such as omega-3 fatty acids, similarly help to promote brain repair after TBI.[9] There are many other neuro-regenerative and anti-inflammatory nutrients considered for recovery from TBI. Zinc may help to repair damaged tissue and decreased neural

cell death, magnesium may prevent neuro-excitotoxicity, and choline may act in an anti-inflammatory and antioxidant capacity.[122] Although there are potentially beneficial dietary interventions, resistance to change, environmental or financial hindrances, socioeconomic status, lack of social support, and unawareness of consequences of poor nutrition may be major hindrances to diet implementation.[35, 123] Obesity is a common disease state for the implementation of weight loss behavior change programs, as US adult obesity prevalence was at 41.7% in 2017.[124] Weight loss diets are commonly practiced in US adults to reduce prevalence and severity of obesity.

Therefore, diet quality is a particular concern when considering the American diet, as obesity rates are on the rise.[47] Obesity is an inflammatory state which increases the risk of developing cardiovascular disease, diabetes, and many other chronic diseases.[41] Because obesity is another source of chronic low-grade inflammation, individuals with obesity may have longer periods of recovery from TBI.[125] Inflammation from TBI can also interrupt endocrine hormones and impact weight via pituitary gland dysfunction.[126] Therefore, inflammation from obesity and TBI can combine to exacerbate neuro-inflammation, which may lead to neurodegenerative disease in TBI patients.[127] Weight loss diets are commonly practiced in the public to prevent or reduce the symptoms of overweight and obesity.

Behavioral flexibility is an executive function in the brain which allows for a person to shift from a routine they typically perform to a modified behavior, such as adhering to a healthier dietary pattern.[128] Dietary behavior change is heavily impacted by a myriad of other factors beyond simply having the executive function to set health goals for oneself, such as socioeconomic status, dietary environment, self-efficacy, and social support.[30, 123, 129] In the American diet, the Healthy Eating Index (HEI) can be used as a metric to determine adherence to

Dietary Guidelines for Americans.[100] The American population on average consumes a diet which scores a 59 out of 100 points on the Healthy Eating Index, showing poor behavioral flexibility to diet modification in general.[100] Behavioral flexibility can further be deterred by dementia and increased age, and the HIC population faces both increased risk of neurodegenerative disease and was aged 40 and older in this study.[128] Therefore, dietary adherence and behavioral flexibility in the population with history of head injury may be more difficult than in a healthy population.

We further analyzed the diet quality of NHANES participants with and without history of head injury who self-reported adherence to weight loss diets to determine the impact of dieting on actual intakes and further assess their behavioral flexibility on diet modification.

4.3 METHODS

Data for the participants (n=7,399) with or without head injury with loss of consciousness were collected from the 2011-2014 NHANES survey as previously described in **Section 3.3**. HIC and No-HIC were further divided into 2 groups (4 groups total) based on their response to the question DRQSDT1, which asked "What kind of diet are you on? Is it a weight loss or low-calorie diet?". Those who responded "yes" to DRQSDT1 became the HIC + Diet and No-HIC + Diet groups, based on their head injury status. Those who did not respond to DRQSDT1 were grouped as not adhering to a weight loss diet, HIC + No Diet and No-HIC + No Diet, based on their head injury status. The mean % DRI ± SEM was reported for each group based on 26 nutrients in the Dietary Guidelines for Americans.

4.3.1 *Statistical analyses*

For the analyses of those adhering to a weight loss diet, we built a linear model using CSQ240 and DRQSDT1 as predictors, and the corresponding variable as response. The linear

model was an interactive model since comparisons were made between different levels of CSQ240 conditioning on each level of DRQSDT1, or between different levels of DRQSDT1 conditioning on each level of CSQ240. Based on the linear model, estimated marginal means (EMMs) were used to compute the *p*-values in each comparison using a contrast method. Statistics were performed using SPSS Statistical Software, R, and GraphPad Prism with significance at $p < .05$.

4.4 RESULTS

To further examine diet behaviors in the population with head injury, we analyzed the dietary intakes (% DRI) of participants adhering to a weight loss diet as shown in **Table 4**. Among the participants with the history of head injury with loss of consciousness (HIC), no significant differences in nutrient intakes were observed, except for Vitamin E, between the participants who adhered and who did not adhere to a weight loss diet. Vitamin E intake (%DRI) was 14.4% higher in the HIC participants who reported the adherence on a weight loss diet compared to those who did not report following a weight loss diet ($p = .005$). No other nutrients were significantly different between the HIC groups.

Among the participant without the history of head injury with loss of consciousness (No-HIC), 9 nutrient intakes (% DRI) were significantly different between those who self-reported adhering to a weight loss diet and those who did not report adhering to the diet. No-HIC + Diet consumed 5.3% fewer calories compared to recommendations and 20% less carbohydrates compared to recommendations than No-HIC + No Diet. Within No-HIC, nutrient intake (%DRI) was also lower among those who reported adhering to a weight loss diet for iron ($p = .006$), and thiamin ($p = .045$), compared to No-HIC + No Diet. Within No-HIC, nutrient intake (%DRI) was higher among those who reported adhering to a weight loss diet for fiber ($p = .014$), magnesium

(p = .001), Vitamin E (p < .001), and Vitamin C (p = .008), compared to No-HIC + No Diet. No other nutrients were different between the No-HIC groups.

We further compared the nutrient intakes (% DRI) of the participants who self-reported adhering to a weight loss diet between HIC and No-HIC. There were 3 nutrient intakes (% DRI) that were significantly different between HIC + Diet and No-HIC + Diet. Nutrient intake (% DRI) was higher among those in HIC + Diet for calcium (p = .049) and Folate (p = .021), compared to No-HIC + Diet. Vitamin E intake was significantly higher in HIC + Diet than No-HIC + Diet, but both groups consumed intakes below 80% of the recommendation. When comparing the two groups who did not report adhering to a weight loss diet, 11 nutrient intakes (% DRI) were significantly higher in HIC + No Diet than No-HIC + No Diet.

Table 4. Nutrient Intakes Per Gender- and Age-Specific Dietary Recommendations Among Participants in NHANES 2011-2014 Who Answered CSQ 240 and DRQSDT1 (n = 7,399)

Nutrient (% DRI)[3]	HIC[1] Diet (n = 73)	HIC[1] No Diet (n = 875)		No-HIC[2] Diet (n = 422)	No-HIC[2] No Diet (n = 6,029)			
	Mean (SEM)	Mean (SEM)	*p*-Value[4]	Mean (SEM)	Mean (SEM)	*p*-Value[5]	*p*-Value[6]	*p*-Value[7]
Calories	90.2 (3.8)	96.3 (1.3)	0.149	87.6 (1.6)	92.9 (0.5)	**0.003**	0.548	**0.010**
Protein	149.6 (6.5)	153.6 (2.3)	0.844	154.2 (2.9)	152.3 (0.9)	0.454	0.830	0.377
Carbohydrate	174.5 (8.2)	197.0 (3.2)	0.056	162.4 (3.5)	182.4 (1.1)	**0.000**	0.183	**0.000**
Fiber[8]	70.2 (3.7)	72.5 (1.5)	0.998	76.7 (2.1)	70.6 (0.5)	**0.014**	0.465	0.412
Saturated fat[8] (% kcals)	105.6 (4.1)	109.0 (1.2)	0.401	104.0 (1.7)	105.8 (0.5)	0.280	0.703	**0.014**
PFA 18:2	121.8 (7.6)	124.4 (2.6)	0.914	124.0 (3.6)	118.9 (0.9)	0.124	0.884	0.065
PFA 18:3	133.2 (9.3)	128.4 (3.2)	0.468	132.4 (4.4)	126.6 (1.1)	0.190	0.862	0.981
Calcium	94.0 (7.0)	89.7 (1.7)	0.317	83.9 (2.2)	81.8 (0.6)	0.436	**0.049**	**0.000**
Iron	163.8 (13.9)	173.5 (3.3)	0.687	149.8 (4.3)	163.4 (1.3)	**0.006**	0.148	**0.017**
Magnesium	83.3 (4.5)	81.5 (1.3)	0.397	84.9 (1.8)	78.9 (0.5)	**0.001**	0.937	0.077
Phosphorous	186.0 (9.0)	194.7 (3.0)	0.509	182.3 (3.7)	183.4 (1.1)	0.910	0.553	**0.000**
Potassium	89.8 (4.1)	89.9 (1.3)	0.821	92.8 (1.8)	87.3 (0.5)	**0.003**	0.702	0.059
Sodium	141.5 (6.2)	150.0 (2.4)	0.317	136.8 (3.1)	142.7 (0.9)	0.162	0.553	0.001
Zinc	119.4 (8.8)	113.8 (2.0)	0.182	110.9 (2.6)	110.4 (0.7)	0.891	0.071	0.067
Vitamin A, RAE	88.7 (8.8)	79.3 (2.7)	0.193	83.0 (3.1)	79.5 (1.0)	0.733	0.312	0.713
Vitamin E[9]	79.4 (10.9)	65.0 (1.8)	**0.005**	66.4 (2.8)	58.1 (0.6)	**0.000**	**0.013**	**0.000**
Vitamin D	31.9 (3.5)	32.6 (1.2)	0.869	32.4 (1.6)	31.6 (0.5)	0.530	0.991	0.174
Vitamin C	124.8 (10.1)	113.1 (3.7)	0.348	126.0 (5.3)	110.8 (1.4)	**0.008**	0.905	0.385
Thiamin (Vitamin B1)	133.2 (8.2)	136.0 (2.2)	0.959	125.7 (2.9)	131.9 (0.8)	**0.045**	0.205	0.172
Riboflavin (Vitamin B2)	174.5 (10.4)	175.8 (3.0)	0.864	160.7 (3.4)	162.3 (1.1)	0.623	0.088	**0.000**
Niacin	156.1 (8.9)	164.3 (2.8)	0.602	154.5 (3.3)	158.2 (1.0)	0.307	0.550	**0.024**

Nutrient (% DRI)[3]	HIC[1] Diet (n = 73)	HIC[1] No Diet (n = 875)		No-HIC[2] Diet (n = 422)	No-HIC[2] No Diet (n = 6,029)			
	Mean (SEM)	**Mean (SEM)**	***p*-Value[4]**	**Mean (SEM)**	**Mean (SEM)**	***p*-Value[5]**	***p*-Value[6]**	***p*-Value[7]**
Vitamin B6	136.6 (9.7)	143.8 (3.3)	0.608	132.8 (3.3)	132.9 (1.1)	0.903	0.502	**0.000**
Vitamin B12[9]	261.0 (31.5)	264.4 (11.0)	0.504	223.8 (9.2)	227.0 (3.0)	0.589	0.098	**0.011**
Choline	64.8 (2.9)	68.2 (1.2)	0.537	66.3 (1.4)	66.4 (0.4)	0.797	0.815	0.132
Vitamin K	129.5 (14.5)	111.4 (5.0)	0.470	138.0 (7.4)	123.3 (3.9)	0.323	0.907	0.172
Folate, DFE	137.5 (12.0)	131.6 (2.7)	0.282	118.7 (3.3)	125.6 (1.0)	0.079	**0.021**	0.063

[1]Answered "yes" to the question CSQ 240 "Have you ever had a loss of consciousness because of a head injury?"
[2]Answered "no" to the question CSQ 240 "Have you ever had a loss of consciousness because of a head injury?"
[3]Nutrients were calculated as nutrient intakes divided by DRI. DRI used: EER for calories; RDA for protein, carbohydrate, calcium, iron, magnesium, phosphorous, zinc, Vitamin A, Vitamin E, Vitamin D, Vitamin C, thiamin, riboflavin, niacin, Vitamin B6, Vitamin B12, and folate; AI for PFA 18:2, PFA 18:3, potassium, choline, and Vitamin K; CDRR for sodium.
[4]This value compares HIC + Diet to HIC + No Diet.
[5]This value compares No-HIC + Diet to No-HIC + No Diet.
[6]This value compares HIC + Diet to No-HIC + Diet.
[7]This value compares HIC + No Diet to No-HIC + No Diet.
[8]DGA values were used for fiber (14g/1000 kcals), and saturated fat (<10% of calories from saturated fat).
[9]Daily Vitamin E was calculated as a combination of the variables Vitamin E (alpha-tocopherol) + Added alpha-tocopherol. Daily Vitamin B12 was calculated as a combination of the variables Vitamin B12 + Added Vitamin B12.

4.5 DISCUSSION

It is well known that most Americans do not adhere closely to recommended dietary patterns.[100] We previously found that both HIC and No-HIC were consuming diets which exceeded recommendations for carbohydrates, calories from saturated fat, and sodium, and HIC consumed 34% less Vitamin E than the recommendation, despite its benefits as an antioxidant, as seen in **Table 3**.[14] The current study further analyzed the diet quality of those with head injury to determine behavioral flexibility among those who self-reported adherence to a weight loss diet. Those with history of head injury who reported adhering to a weight loss diet consumed similar nutrient intakes (% DRI) to those of their non-dieting peers, despite their self-reported attempt to lose weight. Baseline dietary patterns which do not adhere to the dietary guidelines combined with possible resistance to dietary change may lead to poor health outcomes in the population with head injury.

Obesity is another prevalent health concern in the American population. Like traumatic brain injury, obesity can induce a state of low-grade inflammation, which can increase risk of chronic neuroinflammation and decrease recovery post-injury.[41] As weight loss diets are commonly practiced in the general population to prevent and reduce symptoms of overweight and obesity, we wanted to further analyze the relationship between head injury and self-reported adherence to weight loss diets.

Despite the claim that participants were adhering to a weight loss diet, there was only one nutrient (% DRI) consumed significantly more among those in HIC + Diet compared to HIC + No Diet in **Table 4**. Vitamin E intake (%DRI) was 14.4% higher in HIC + Diet compared to No-HIC + Diet, which may be beneficial due to its antioxidant properties.[18] Vitamin E is a strong antioxidant and has been observed to reduce oxidative stress post-injury, which can reduce the

risk of neurodegenerative disease and TBI-related dementia.[14, 18] However, Vitamin E intakes in HIC + Diet were still consuming only 80% of the recommended intake. Additionally, there were no other nutrient intake (% DRI) differences observed between those who self-reported adhering to a weight loss diet and those who did not report adhering to the diet for calories or macronutrients, despite the established relationship between reducing energy intake and weight loss.[130]

Alternatively, among those without history of head injury, adhering to a weight loss diet led to changed nutrient intakes (%DRI) of 8 nutrients compared to No-HIC + No Diet. Those in No-HIC + Diet consumed fewer calories and carbohydrates (%DRI) than No-HIC + No Diet, indicating an attempt to create a calorie deficit. A low carbohydrate diet is a common weight-loss method among the population with overweight and obesity.[131] However, No-HIC + Diet was still exceeding the carbohydrate recommendation by 60%, and consumed significantly more fiber than No-HIC + No Diet, so those following the weight loss diet may have instead attempted to select more healthful carbohydrate choices. Overall, those without history of head injury displayed greater dietary flexibility than HIC when adhering to a weight loss diet.

As observed in this study, head injury may correlate with decreased behavioral flexibility in adapting to new diets, yet this may be caused by a myriad of other health conditions resulting from the injury. Head injuries are correlated with increased risk of neurodegenerative disease and depression, both of which can decrease behavioral flexibility and ability to change habits.[18, 127, 128, 132] Depressive disorders are also correlated with negative thinking, which may decrease self-efficacy to improve dietary patterns after head injury.[128] Thus, there is a lack of information on the interest in dietary changes and nutrition knowledge in this population, but the side effects of head injury may correlate with decreased dietary flexibility in this population.

When analyzing the diets of those who self-reported adhering to a weight loss diet, those with history of head injury consumed diets more similar to their non-dieting peers, indicating a possible resistance to dietary changes. Interestingly, adherence to a weight loss diet in No-HIC led to lower intakes (% DRI) of calories, carbohydrates, and higher intakes of beneficial antioxidant nutrients compared to No-HIC + No Diet. This study suggests that the population with head injury may be prone to inflexibility in behavioral modifications, increasing the potential difficulty of implementing post-TBI diet interventions.

CHAPTER 5

CONCLUSIONS

This thesis aimed to determine two things: first, to investigate the current dietary patterns of those with history of head injury and determine the diet quality based on national nutrition recommendations, and second, to determine the behavioral flexibility to diet modifications among the population with history of head injury.

In Chapter 2, our study demonstrated that US adults with history of head injury with loss of consciousness consumed higher intakes of multiple nutrients including calories, compared to those without history of head injury. Even after adjusting for energy intake, the diets of HIC were higher in pro-inflammatory nutrients, and lower in antioxidants and omega-3 fatty acids, compared to those of No-HIC. HIC exceeded the recommendations for carbohydrates, calories from saturated fat, and sodium, and consumed less than 70% of the recommendation for the antioxidant Vitamin E. HIC consumed significantly higher intakes of 10 nutrients compared to the recommendations than No-HIC, indicating larger portions of food in HIC. The poor diet quality in HIC further indicates the urgency of establishing dietary recommendations for the population with history of head injury, to ensure long-term recovery and improvements of the quality of life.

In Chapter 3, we suggest that there appeared to be a resistance to dietary changes among those with history of head injury, using the dietary intakes of those who self-reported adherence to a weight loss diet in this population. In this analysis, those with head injury only altered the intake of Vitamin E, whereas No-HIC participants who adhered a self-reported weight loss diet

altered the intake of calories, carbohydrates, fiber, magnesium, potassium, Vitamin E and Vitamin C, indicating better adherence to their intended modification toward healthful dietary behaviors.

There are no established dietary interventions for patients suffering from head injury, therefore this thesis identified some gaps in dietary intakes, diet quality, and behavioral flexibility on diet modification which can be used as background for establishing TBI nutrition interventions. The findings from this study can further be used as preliminary information for the development of nutrition recommendations post-injury in the population with head injury.

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