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THE NUTRITION AND PHYSICAL ASSESSMENT OF PATIENTS WITH NEUROLOGICAL DISORDERS

Dara Lyn LoBuono
University of Rhode Island, dara_lobuono@my.uri.edu

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THE NUTRITION AND PHYSICAL ASSESSMENT OF
PATIENTS WITH NEUROLOGICAL DISORDERS

BY

DARA LYN LOBUONO

A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF THE
REQUIREMENTS FOR THE DEGREE OF
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OF
DARA LOBUONO

APPROVED:

Thesis Committee:

Major Professor

Ingrid Lofgren

Leslie Mahler

Geoffrey Greene

Nasser H. Zawia

DEAN OF THE GRADUATE SCHOOL

UNIVERSITY OF RHODE ISLAND
2014

ABSTRACT

Objective: To describe cognitive, nutritional and physical functioning status of those with diagnosed neurological disorders for at least one year.

Design: This is a cross-sectional study.

Setting: University of Rhode Island's Speech and Hearing Clinic.

Participants: Community-dwelling adults living with Parkinson's disease (PD) or acquired brain injury (ABI) for ≥ 12 months.

Main Outcome Measure(s): Cognitive-linguistic status was measured via the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS). Nutritional and cardio-metabolic status were measured using the dietary screening tool (DST), anthropometrics (height, weight, body mass index (BMI), body composition), biochemical (lipid and glucose profile), and clinical (blood pressure) measures. Physical activity and functioning were measured via the Physical Activity and Disability Survey (PADS), short physical performance battery (SPPB), and the timed up and go (TUG) test.

Results: Twelve participants were assessed (8 PD, 4 ABI). Participants with PD had significantly higher total RBANS scores than participants with ABI (87.1 ± 14.6 vs. 66.0 ± 12.6 , $p=0.034$, $d=2.455$). Mean BMI ($n=12$) was $26.8 \pm 3.0 \text{ kg/m}^2$ and the majority of participants were overweight. DST scores indicated 58% ($N=7$) at possible nutrition risk and 25% ($N=3$) were at nutrition risk. Mean PADS scores (94.9 ± 89.7) indicated participants were physically active. A positive correlation was found between general activity scores and DST scores ($r=0.697$, $p=0.012$). Fifty percent of participants

reported suboptimal SPPB, balance or TUG scores. Multiple risk factors for development of CVD were present in all participants.

Conclusion: Deficits in cognition, low dietary quality and physical functioning, and cardio-metabolic risk factors are frequently reported in patients with neurological disorders. These findings confirmed the presence of cognitive, dietary and physical decline in our participant group and support the need for an interdisciplinary team to work with patients and caregivers to improve health and prevent further development of comorbidities in this population.

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Lastly, I would like to thank my family and friends. I do not know where I would be today without you. For this, I dedicate this thesis to my parents, for all their love and support, and in hopes I will continue to take them by surprise.

PREFACE

This thesis was written to comply with the University of Rhode Island graduate school Manuscript Thesis Format. This thesis contains one manuscript: *The Nutrition and Physical Assessment of Patients with Neurological Disorders*. This manuscript has been written in a form suitable for publication in *Archives of Physical Medicine and Rehabilitation*.

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CHAPTER 1

The Nutrition and Physical Assessment of Patients with Neurological Disorders

Dara L. LoBuono^b, Amy Taetzsch^b, Matthew J. Delmonico^c, Ingrid E. Lofgren^b,
Leslie Mahler^a

^aDepartment of Communicative Disorders, University of Rhode Island, Independence
Square I, Kingston, Rhode Island, 02881, United States

^bDepartment of Nutrition and Food Sciences, University of Rhode Island, Ranger Hall,
Kingston, Rhode Island, 02881, United States

^cDepartment of Kinesiology, University of Rhode Island, Independence Square II,
Kingston, Rhode Island, 02881, United States

To be submitted to Archives of Physical Medicine and Rehabilitation

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reported suboptimal SPPB, balance or TUG scores. Multiple risk factors for development of CVD were present in all participants.

Conclusion: Deficits in cognition, low dietary quality and physical functioning, and cardio-metabolic risk factors exist in patients with neurological disorders. These findings confirmed the presence of cognitive, dietary and physical decline in our participant group and support the need for an interdisciplinary team to work with patients and caregivers to improve health and prevent further development of comorbidities in this population.

Key words: Parkinson's disease, stroke, traumatic brain injury

INTRODUCTION

Parkinson's disease (PD), traumatic brain injury (TBI), and stroke are neurological disorders that can result in lifelong challenges that impact cognitive, physical, and psychosocial functioning¹⁻⁵, which, in turn, can jeopardize dietary intake and increase likelihood of nutritional risk and cardiovascular disease (CVD).

Parkinson's disease is a progressive, neurodegenerative, movement disorder affecting nearly one million people in the United States⁶. Traumatic brain injury and stroke are types of acquired brain injury (ABI), defined as structural damage to the brain^{7,8}.

Nearly 2,500,000 people experience a TBI or stroke annually in the United States^{8,9}.

While these neurological diseases are each unique and have different long-term effects, they share similar complications that can negatively impact nutritional status and dietary intake, as well as physical function.

Limited research exists examining the interaction between cognition, nutritional status, and physical function for those with PD and ABI one year or more after diagnosis. Unintentional weight loss is typical at the time of PD diagnosis and nutritional status declines as the disease progresses¹. Malnutrition and muscle wasting are consistently reported in the acute stages of ABI¹⁰⁻¹³. Since nutritional status is compromised in the acute phase after an ABI, long-term nutritional status is likely to be poor¹³. However, due to a scarcity of research, little is known about the long-term nutritional status of those with ABI. Cognitive and physical impairment associated with these neurological disorders may contribute to metabolic and nutritional imbalances^{1,3,5,14,15}.

The cardio-metabolic effect of PD seems to be two-fold, research indicates those with PD may have sub-optimal lipid profiles and glucose intolerance¹⁶, while other research indicates there may be a protective cardio-metabolic effect of PD, even in those with PD that are overweight^{17,18}. Cardio-metabolic effects of ABI have been less investigated. Those with TBI are at increased risk for significant weight change, and diseases related to diet and activity, such as coronary heart disease (CHD) and metabolic syndrome^{3,15}. An obesity paradox seems to exist following stroke^{12,19}. Being overweight or obesity increases risk of a first-time stroke, however, those who are overweight or obese post-stroke, have better functional outcomes^{12,19}, and are less likely to have a reoccurring stroke in the years following stroke^{20,21}.

Research has yet to examine and describe the long-term status (a year or more since diagnosis) of the cognitive, nutritional and physical functioning status across all three neurological diagnoses. The purpose of this cross-sectional study is to describe the cognitive function, dietary quality, and physical activity and functioning in those living with PD or ABI for a year or more. A secondary aim is to analyze how compromised cognition, nutrition and physical activity and functioning increase cardiovascular risk in those living with PD or ABI. An exploratory aim is to assess differences between PD and ABI across the areas of cognition, nutrition, and physical activity.

MATERIALS AND METHODS

Design

This cross-sectional study is a sub-study of a five-year longitudinal, observational study examining the nutritional status, cognitive and physical activity levels of those living with neurological disorders, specifically, PD, TBI, and stroke. Data used in this study was obtained during baseline assessments through the University of Rhode Island (URI)'s Department of Communicative Disorders' Clinic, and included study staff from the Departments of Nutrition and Food Sciences and Kinesiology. Study approval was obtained from URI's Institutional Review Board, and written informed consent of all participants was obtained. Two baseline assessments were conducted in November and December 2013 at the URI Kingston's Campus Speech and Hearing Clinic.

Participants, Setting and Recruitment

For the purposes of this sub-study participants had to be diagnosed with PD, TBI or stroke for at least 12 months and be between the ages of 18-85. Participants were recruited via brochures and word-of-mouth to clients currently obtaining services at the Department of Communicative Disorder's Speech and Hearing Clinic. Fifteen people were eligible to participate, 13 completed the informed consent process, and 12 completed baseline assessments. Participants completed evaluations related to cognitive-linguistic status, nutritional status, physical activity and functioning, and cardio-metabolic risk, as well as a past medical history questionnaire and questionnaires related to life satisfaction and resilience.

Assessment Visit and Data Collection

Anthropometrics

Height (cm) and weight (kg) were measured using a stadiometer (Webb City Missouri). Height was rounded to the nearest 0.5 cm and weight was rounded to the closest 0.1 kg. Height and weight were used to calculate BMI (kg of body weight/height in meters²). Predetermined categories classified BMI scores: underweight <18.5 kg/m², normal weight = 18.5-24.9 kg/m², overweight = 25-29.9 kg/m² and obese ≥ 30 kg/m²²². A foot-to-foot bioelectrical impedance analysis device (Tanita BF-556, Arlington Heights Illinois) was used to obtain body composition and report percent (%) body fat. American Council on Exercise²³, any female with $\geq 32\%$ body fat and any male with $\geq 25\%$ body fat was considered obese.

Biochemical

Following a 12 hour fast, serum total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), triacylglycerol, and glucose were assayed using the portable Cholestech[®] (LDX system, Hayward California) machine. Total serum cholesterol <200 mg/dL, LDL-C <100 mg/dL, HDL-C >40 mg/dL, triglycerides <150 mg/dL, glucose <100 mg/dL are considered optimal metabolic-lipid profiles²⁴.

Clinical

Blood pressure was measured after participants sat quietly for five minutes using an automatic blood pressure machine (Omron Healthcare Inc., Bannockburin Illinois). Systolic blood pressure (SBP) <120 mmHg and diastolic blood pressure (DBP) <80 mmHg was considered optimal. Systolic blood pressure >130 mmHg and

DBP >85 mmHg was borderline high, while SBP > 140 mmHg and DBP >90 mmHg was considered hypertensive.

Cognitive Linguistic

Cognitive-linguistic function was assessed using the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS)²⁵. The RBANS includes 12 subtests, with five index scores, summarizing: immediate memory, visuospatial/constructional, language, attention and delayed memory. The total score is the sum of the five index scores. Scoring is based on a standardized mean of 100 and a standard deviation of 15; a score <70 indicates suboptimal cognitive function.

Swallowing Evaluation

The Swallowing SWAL-QOL is a 44-item survey where participants rate factors about 10 quality of life concepts related to swallowing, primarily in regards to eating and diet^{26,27}. The highest possible score is 220, the higher the score the better one's swallowing function.

Dietary Intake and Nutritional Risk Assessment

The Dietary Screening Tool (DST) is a 25-item questionnaire that is used to identify dietary patterns and nutritional risk in older adults^{28,29}. Participants reported how frequently they consumed a food or type of food. A composite score was calculated based on subcomponents of the DST. The sub-components and point distribution included: whole fruit and juices (15 points), vegetables (15 points), lean proteins (10 points), added fats, sugars and sweets (25 points), dairy (10 points) and processed meats (10 points), and use of a dietary supplement (5 points).

These sum-components are composited and total scores are defined for three different nutritional risk levels: (<60) at risk, (60-75) possible risk, and (>75) not at risk²⁸. Categorization into three nutritional risk categories provides a way to determine if a patient needs a follow-up or nutrition assessment²⁸. The DST Dietary intake was broken down based on point classification for the DST sub-components²⁸ to explore specific areas contributing to low dietary quality. These sub-components were created to identify specific areas of the diet that may be problematic on an individual basis²⁸.

Participants with fewer than 50% of total possible points for each sub-component and if the participant did not currently take dietary supplement were considered low sub-scores. Less than 50% was selected as an exploratory marker to indicate infrequent/inadequate intake of whole grains, fruits, vegetables, dairy or lean proteins, and high consumption of processed meats, added sugars, fats and sweets.

Physical Activity and Functioning

The Physical Activity and Disability Survey (PADS)^{30,31} was used to assess physical activity levels. It covers a wide range of activities and includes six sub-scores: exercise, leisure-time physical activity (LTPA), general activity, therapy, employment, and wheelchair use. The PADS is coded based on intensity, type and amount of activity and can detect varying levels of physical activity³². Raw scores of ≤ 27 indicate minimal, 28-42 is minimal to moderate, 43-67 is moderate, and ≥ 68 indicates a high activity³⁰.

The short physical performance battery (SPPB)³³ was completed to assess participants' global physical functioning and included usual gait speed, standing

balance tests, and five repeated chair stands. SPPB scores are on a 12 point scale, the higher the score the better physical function. Scores 8-12 are considered “high functioning³³.”

A timed up and go (TUG) test was used to assess basic functional mobility³⁴. It measures the time it takes for the participant to rise from a chair, walk three meters, turn around and walk back to the chair. Time was recorded in seconds, and the shorter amount of time, the better the score. The test was administered twice and the better score was recorded. A TUG score >13.5 is considered high risk for falls³⁵

Other Questionnaires

A non-validated medical history questionnaire was used to identify health-related conditions and how these conditions may impact health.

Cardio-Metabolic Assessment

Cardiovascular disease is the disease of the heart and/or blood vessels³⁶. Traditional risk factors for CVD include modifiable and non-modifiable risk factors. Non-modifiable risk factors include age, sex and ethnicity. Modifiable risk factors include being overweight/obese, having hypertension, elevated blood glucose levels, and dyslipidemia³⁷. As well lifestyle factors such as physical inactivity, tobacco use and a diet high in saturated fat and processed foods and low in fruits and vegetables and fiber^{37,38}.

Cardiovascular risk factors were selected based on these risk factors but also unique factors that may impact CVD risk for those living with disability. These factors were grouped into eight categories, which included: 1) age (>45 years for men, >55 years for women), 2) anthropometrics (BMI, %BF), 3) clinical (SBP and DBP), 4)

biochemical (TC, HDL-C, TC/HDL-C ratio, LDL-C, TRG and Glucose), 5) diet (DST scores), 6) physical barriers (use of legs, arm or assistive device), activity (PADS scores) and functioning (SPPB and TUG scores), 7) smoking status, and 8) cognition. Those living with neurological disorders suffer physical and cognitive impairment that may disrupt activities of daily living (ADL); as a result physical barriers (such as partial or no use of arms and legs, as well as use of an assistive device) were examined due to the possibility they may promote a sedentary lifestyle. Cognitive function was also included as a CVD risk factor through examining total RBANS scores and three RBANS index scores (immediate memory, attention, delayed memory) <70.

Statistical Analysis

Statistical analysis were not hypothesis driven, but conducted to generate hypotheses and explore the population. Data were analyzed using SPSS, version 22.0 for Windows (IBM Corp. Summers, NY). Descriptive statistics were conducted and distribution of data was assessed through analyzing skewness and kurtosis. For data that were not normally distributed, proper statistical methods were used to account for this. Categorical variables are represented as numbers and percentages and continuous variables are reported as mean \pm standard deviation.

To analyze CVD risk factors, the participants were individually analyzed across the eight categories. The most frequent risk factors were then analyzed based on the variables within the eight CVD categories and reported as top CVD risk factors.

For normally distributed data, independent *t*-tests were conducted to determine if there were significant differences between groups regarding assessment variables. Cohen's *d* was calculated to describe effect size for differences between groups in

normally distributed variables (0.2 = small effect, 0.5 = medium effect, 0.8 = large effect). For data not normally distributed, Mann-Whitney U tests were used to analyze differences between groups and effect size was signified through calculating r (0.1=small effect, 0.3=medium effect, 0.5=large effect). Differences between groups for categorical variables were identified using Fisher Exact Tests. Phi squared or Cramer's V were used to signify effect size (0.1=small effect, 0.3=medium effect, 0.5=large effect). Pearson's R correlations were used with normally distributed variables, and Spearman Rho correlations were used as the non-parametric alternative. Differences between groups were significant when $p < 0.05$.

RESULTS

Demographic characteristics and results from specific assessment measures are summarized in Table 1. Of the four ABI participants two were diagnosed with stroke, one with a TBI and one participant had a diagnosis of TBI and stroke. An independent *t*-test found, those living with ABI were diagnosed significantly longer than those living with PD ($p=0.01$, $d=1.8$). All participants self-identified as white/Caucasian and of non-Hispanic/Latino ethnicity. Two participants (both with ABI) were employed at the time of testing. The age range was 33 through 71. Comorbidities amongst participants included musculoskeletal conditions (osteoporosis, arthritis), cancer, cardiovascular (coronary artery disease), previous head injury, and psychological disorders (bipolar). None reported endocrine dysfunction, such as thyroid disease or diabetes.

No significant correlation was found between RBANS scores and nutrition, cardio-metabolic, or physical assessments. As indicated by DST scores, three participants with PD were classified at nutritional risk and seven participants (4 PD and 3 ABI) were classified at possible nutrition risk (Table 2). Two participants with PD reported diet changes to soft, easy to chew foods, while all other participants reported to follow a “full normal diet,” not modified in texture or consistency. All participants reported drinking thin liquids without any modifications.

Results from 10 DST sub-scores per participant are described in Table 3. When examining dietary components, 75% of participants were not consuming enough dairy products, and 50% were frequently consuming added fats, sugars and sweets and/or not taking a dietary supplement. The majority of participants were consuming fruits,

vegetables and whole grains regularly. On average, participants had inadequate sub-scores in three out of ten DST sub-categories.

Physical barriers, physical activity level and physical functioning for each participated are reported in Table 4. Six participants had partial use of arms and eight had partial use of legs, while seven participants reported to use an assistive device. The range of PADS scores for participants ranged from -12 to 311. Mean total TUG score was 12.1 ± 3.2 seconds. Physical barriers, physical activity level and physical functioning for each participated are reported in Table 4.

The secondary aim of this study was to analyze how compromised cognition, nutrition, and physical activity and functioning may increase CVD risk. Seven participants were taking medication to manage blood pressure and/or lipid profiles. Results of the RBANS revealed that three participants had total index scores <70 (1 PD, 2 ABI), three participants (1 PD, 2 ABI) had <70 Immediate Memory Scores, three ABI participants had <70 Attention scores and two PD and one ABI had <70 delayed memory scores. In total, five participants had cognitive impairment in one of these four areas. Typically, cognitive status is not classified as a CVD risk factor, however these tests have been included to explore the potential impact of impaired cognition associated with neurological disorders may have on CVD risk. Additionally, the presence of possible nutrition risk (Table 3) and compromised physical functioning (Table 4) may worsen overtime and evolve into a worse diet and sedentary lifestyle, factors that in turn can lead to CVD. The presence of nutrition and cardio-metabolic risk factors, including anthropometrics, clinical and biochemical values are reported for each participant in Table 2.

Participants had three to six risk factors. The most common risk factors are highlighted in Figure 1. Body mass index scores ranged from 23.0 kg/m² to 33.3 kg/m². Additionally, 58.3% participants had elevated SBP and/or DBP, and, 72.7% participants had at least one abnormal lipid or glucose level lab value (Table 2).

An exploratory aim of this study was to examine if there are differences between those living with PD and ABI across cognition, nutrition and physical activity and functioning. In addition to significantly higher total RBANS scores, those with PD scored significantly higher in language (96.9±8.7, 80.00±2.3, $p=0.004$, $d=2.66$) and attention (95.0±10.4, 56.00±15.9, $p=0.000$, $d=2.90$). There was no difference in DST or PADS scores between groups.

A Fisher's exact test found those with PD were more likely to have full use of arms ($p=0.061$, $\Phi=0.707$) and legs ($p=0.208$, $\Phi=0.50$), and less likely to use assistive devices ($p=0.061$, $\Phi=0.598$) with a large effect size (Table 4). Overall, TUG performance for those with PD was better, 11.1±2.5 seconds versus 14.2±3.8 seconds ($p=0.112$, $d=0.96$), with a large effect size. Participants with ABI had SPPB scores below eight. There was also no difference in anthropometrics, clinical or biochemical data in those living with PD versus ABI.

DISCUSSION

This baseline study is the first of a 5-year observational study in those with neurological disorders to understand the rate and magnitude of change in this population, as well to help prevent comorbidities. This study is the first to examine cognition, nutrition, and physical activity and functioning together in those living with PD or ABI. Findings indicated those living with one of these neurological diagnoses present with deficits in cognition nutrition, cardio-metabolic and/or physical functioning. Our findings highlight the need for an interdisciplinary team to work with this population to help prevent CVD. The majority of our participants, were overweight/obese (n=9) and had DST scores <75 (n=10). Additionally, 50% performed sub-optimally in physical functioning assessments and all presented CVD risk factors.

A Pearson correlation found a negative correlation between total RBANS scores and months elapsed since diagnosis ($r=-0.655$, $p=0.021$). The association between RBANS scores and time since diagnosis correlated with previous literature regarding cognitive decline in this population^{39,40}. The significantly lower RBANS scores for those with ABI may be associated with the significantly longer time since diagnosis (5.3 ± 4.3 vs. 14.3 ± 5.6 years, $p=0.011$).

While malnutrition with low body weight or eating difficulties may be present in these disease states^{3,5,41,42}, none of our participants were underweight and only two reported to consume a “soft” modified texture diet. Ten out of twelve participants had <75 DST scores. A score between 60 and 75 indicates possible nutritional risk. Those with higher DST scores (a more healthful dietary pattern) have been associated with

more favorable biomarkers, health status, nutrient dense diets and body compositions²⁸. As a result, we felt it was necessary to examine DST scores <75 overtime and identify participants with these scores needed improvements in dietary quality.

The frequent consumption of added fats, sugar and sweets (Table 3) may indicate those with cognitive and physical impairment are choosing convenience foods frequently. Nine out of twelve participants were not consuming dairy frequently, and to optimize bone health, future dietary interventions should consider promoting consumption of low/reduced fat products high in calcium and vitamin D. As well as, encouraging the use of a daily multi-vitamin supplement, if appropriate, and an education of which foods provide appropriate amount of calories and nutrients for this population.

Low DST and general activity scores and their relationship suggests further investigation of disease-related eating barriers, such as ability to buy and prepare meals^{5,14,43}. Altered satiety cues and change in appetite associated with TBI may also be present^{3,42}. It also implies isolated activity time is less of a barrier than performing household tasks. Most participants were married or lived with family, and household work was performed by someone else. These findings are consistent with past research, which found 86% of those with PD were disabled from participating in ADL, but 88.8% had social support, and 64% participated in regular exercise/leisure time activity⁴⁴. Future physical activity interventions should target ways to help increase the ability to independently carry out more ADL, which may improve diet quality.

Evidence shows physical activity can help reduce age-related physical and cognitive decline⁴⁵⁻⁴⁷. The presence of elevated exercise and LTPA activity and their correlation with SPPB scores may imply a protective effect of activity on participant's physical decline and should be examined over the next five years.

Due to the small sample size and the use of pharmacological medications to manage blood pressure and/or cholesterol levels by many participants (58.3%), it is hard to speculate the risk factor with the biggest impact on the development of a cardiovascular event in those with neurological disorders. Despite the use of medication to manage lipid profiles and blood pressure, other CVD risk factors were still present and may lead to a CVD event. This can be highlighted by presence of elevated bodyweight and low dietary scores in many participants and shows the importance to continue to monitor and promote heart health in this population.

Research still needs to better understand the risk of CVD in those with PD and whether these is in fact a protective cardio-metabolic effect present in those with PD or an increased risk of a CVD event^{16-18,48,49}. Both those with ABI and PD the presence of elevated BMI and %BF, suboptimal DST scores and slight abnormality in clinical and biochemical values gives reason to continue to monitor those with one of these neurological disorders. Especially given the association with TBI and the development of cardio-metabolic alterations overtime^{33,42,50}. Due to the small sample size of those living with stroke, it is difficult to compare participants and assess if the notion of the obesity paradox (those obese/overweight at the time of stroke have better functional outcomes^{12,19}) exists a year or more after stroke.

While more research is needed to draw conclusion regarding cardiovascular risk across these disease states, it is still important to monitor and treat cardiovascular risk factors present. Research notes those with multiple marginal risk factors are at risk for a CVD event within 10 years⁵¹. Those currently taking medication to treat hypertension, with suboptimal blood pressure (especially elevated SBP levels) are at more increased risk than those not taking medication with elevated blood pressure levels⁵¹. Four out of twelve of participants were currently taking blood pressure medication and presented with elevated/ pre-hypertensive SBP (≥ 130 mmHg).

Despite the small sample size, the biggest difference between those living with ABI or PD were differences in cognitive-linguistic deficits. Cognitive-linguistic complications are common across diseases^{39,52-56}, however for PD, older age at disease onset, depression, male gender, motor symptom severity, and advanced disease are risk factors for cognitive impairment⁴⁰. Cognitive symptoms usually appear within 15 years of PD diagnosis^{57,58}. Only one participant with PD had a low total RBANS scores. The two participants with stroke had total RBANS scores >70 while the two TBI participants had scores <70 . Both TBI participants had low RBANS index scores for immediate memory, and attention, which is consistent with the nature of the disease³⁹. However, PD progresses, it is likely issues with cognition, nutrition and physical activity and functioning may become more pronounced than those living with ABI.

Study Strengths and Limitations

This is the first study to complete combined assessments of cognition, nutrition, and physical activity and functioning in those living with PD or ABI. This

interdisciplinary study obtained both subjective and objective data via validated tools to assess the study sample and generate hypotheses.

While this study is novel, it is not without limitations. The small sample size and homogeneity of participants does not allow wide generalizability for those living with PD or ABI. Participants were predominantly well educated and of white/Caucasian decent, and recruited on a voluntary basis. As a result, participants likely had an interest and motivation to improve health status. Finally, since there were a larger number of PD participants than anticipated, obtaining Hoehn and Yahr classifications to identify severity of disability in PD⁵⁹ would strengthen results.

Future research should expand recruitment across the three disease states, to compare differences between PD, TBI and stroke, rather than just PD and ABI. This will allow a better understanding of how cognition, nutrition and physical functioning differs across the three diseases. Research should also expand recruitment to include healthy, age-matched controls, to provide insight on how living with neurological disorders compare to the healthy population. Once a more complete understanding of the population is established, well-defined recommendations and interventions can be developed and tested to improve treatment of those with PD and ABI.

CONCLUSION

Suboptimal cognition, dietary quality, physical functioning, and cardio-metabolic risk factors are frequently reported in patients with neurological disorders. These findings confirmed the presence of cognitive, dietary and physical decline in our participant group and support the need for an interdisciplinary team to work with patients and caregivers to improve health and prevent further development of comorbidities in this population.

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TABLES AND FIGURES

Table 1: Characteristics of All Participants and by Neurological Disorder				
	All (n=12)	PD (n=8)	ABI (n=4)	<i>Effect Size</i>
Age (years) [†]	60.0±12.4	65.5±5.0	49.0±16.1	0.54
Months Since Dx.	99.3±75.5	63.4±51.3	171.0±66.7*	1.91
Gender%(n) [‡]				0.250
Men	66.7(8)	75.0(6)	50.0(2)	
Women	33.3(4)	25.0(2)	50.0(2)	
Education Level %(n) [‡]				0.463
HS Diploma	25(3)	12.5(1)	50.0(2)	
Bachelors	16.6(2)	25.0(2)	0(0)	
≥Graduate Degree	58.3(7)	62.5(5)	50.0(2)	
RBANS Total	80.1±17.0	87.1±14.6	66.0±12.6*	2.455
Height (cm)	170.1±9.2	170.5±6.98	169.4±14.1	0.10
Weight (kg)	78.4±15.0	79.7±14.1	75.7±18.7	0.25
BMI (kg/m ²)	26.8±3.0	27.3±3.3	26.0±2.4	0.40
BMI Categories%(n) [‡]				0.217
Normal	25.0(3)	25.0(2)	25.0(1)	
Overweight	66.7(8)	62.5(5)	75.0(3)	
Obese	8.3(1)	12.5(1)	0(0)	
Body Fat (%)	30.0±6.5	29.9±6.7	30.1±7.2	0.02
TC (mg/dL) [†]	182.9±37.3 (11)	169.1±28.9 (7)	207.0±42.0 (4)	0.139
TAG (mg/dL) [†]	131.00±70.0 (11)	132.7±73.8 (7)	128.0±73.6 (4)	0.286
HDL-C (mg/dL) [†]	48.6±20.4(10)	43.7±12.7(7)	60.0±33.5(3)	0.109
LDL-C (mg/dL) [†]	99.6±30.6(10)	98.9±27.6(7)	101.3±43.8(3)	0.03
Glucose (mg/dL) [†]	87.0±12.9(10)	91.2±7.9(6)	80.8±14.5(4)	0.2365
SBP (mmHg) [†]	133.7±17.0	138.6±18.5	124.0±8.8	0.343
DBP (mmHg) [†]	79.0±9.0	80.3±9.9	76.5±7.2	0.196
DST	64.3±12.4	61.1±13.6	70.8±7.0	1.308
SWAL-QOL [*]	188.0±20.6	176.9±15.0	210.3±6.1*	4.188
PADS [†] _n	94.9±89.7	89.7±99.6	108.7±71.5	0.143
SPPB ^d	8.7±1.6	9.3±1.0	7.5±2.1	1.996

*Indicates statistically significant differences between PD and ABI groups (p<0.05). Notes: values provided for total number of participants and by disease state expressed as mean± standard deviation. Differences between groups for all normally distributed variables analyzed using independent t-tests, Cohen's d for effect size (0.2 = small effect, 0.5 = medium effect, 0.8 = large effect). [†]Between group differences conducted using Mann-Whitney U Test, effect size reported as r (0.1 = small effect, 0.3 = medium effect, 0.5= large effect). [‡]Between group differences for categorical variables provided using fisher exact test, effect size for fisher exact test using phi coefficient or Cramer's V (0.10 = small, 0.30 = medium, large = 0.50). Abbreviations: Dx, diagnosis

Table 2: Nutrition & Cardio-Metabolic Risk Factors By Participants

	Diagnosis	Months Since Diagnosis	BMI (>25 kg/m ²)	%BF ^a	DST <75	SBP ≥130 mmHG	DBP ≥85 mmHG	TC >200 mg/dL	HDL-C, <40 mg/dL	TC/HDLC >5:1	LDL-C >100 mg/dL	TRG >150 mg/dL	Glucose >100 mg/dL	Smoking Status	Total # of RF/PPT
PPT1 [†]	ABI	216	X	X	X	X									4
PPT2	PD	96			X							X			2
PPT3 [†]	PD	12	X	X	X	X		X			X				6
PPT4	ABI	240	X		X	X	X				X				5
PPT5	ABI	120	X	N/A				X	X	X	X				5
PPT6	ABI	108			X		X	N/A	N/A	N/A	N/A				2
PPT8 ^{‡§}	PD	36	X	X		X	X								4
PPT9	PD	36	X		X	X									3
PPT10 [†]	PD	36	X	X	X	X	X	N/A	N/A	N/A	N/A	N/A	N/A		5
PPT11 [†]	PD	113	X	X	X			X	X	X	X			X	8
PPT12 [†]	PD	23		X	X							X	N/A	X	4
PPT13 [‡]	PD	155	X	X	X	X		X	X	X	X	X			9
TOTAL RF			9	7	10	5	4	3	4	3	5	4	1		

^aBased on guidelines from American Council of Exercise, females with ≥32%BF and males with ≥25%BF; [†]PPT currently takes blood pressure medication;

[‡]PPT currently takes blood pressure & lipid lowering medication; [§]PPT had history of coronary artery disease. Abbreviations: BMI, Body Mass Index; %BF, percent body fat; BP, blood pressure, TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TRG, triglycerides; PADS, physical activity and disability scale; DST, dietary screening tool; RF, Risk Factors

Table 3: DST Sub-Scores by Participant

PPT	Diagnosis	Fruit & Juice	Vegetables	Whole Grains	Lean Proteins	Added Fats, Sugar, Sweets	Dairy	Processed Meats	Supplement	Total Sub-Categories
PPT1	ABI			X	X				X	3
PPT2	PD					X	X		X	3
PPT3	PD				X	X	X		X	4
PPT4	ABI					X	X	X		3
PPT5	ABI									
PPT6	ABI	X			X		X			3
PPT8	PD						X			1
PPT9	PD		X		X		X			3
PPT10	PD					X	X		X	3
PPT11	PD						X		X	2
PPT12	PD		X	X		X				3
PPT13	PD	X	X	X		X	X	X	X	7
Total:		2	3	3	4	6	9	2	6	

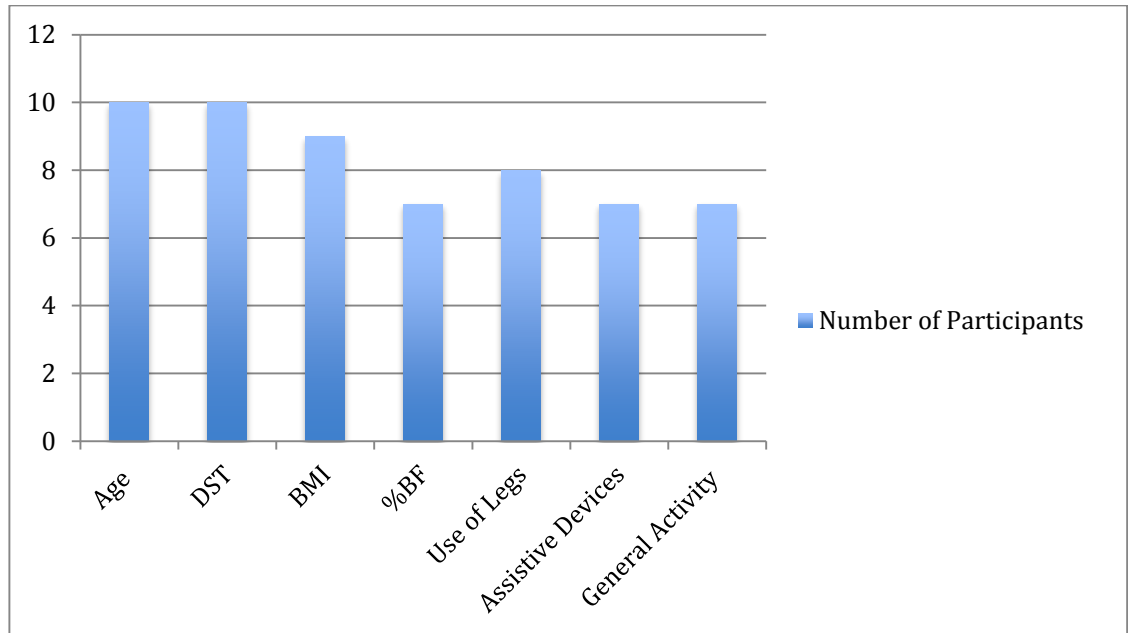
Note: Check marks indicate fruit & juice, vegetables, whole grains scores ≤ 7 out of 15 points, lean protein, dairy, processed meats scores ≤ 5 out of 10, added fats, sugar, sweets ≤ 12 out of 25, and if the participant did not take a supplement. PPT 5 and 8 were not at nutritional risk. Abbreviations: PPT, participants; DST, dietary screening tool

Table 4: Physical Barriers, Physical Activity and Physical Function

PPT	Diagnosi s	PHYSICAL BARRIERS				ACTIVITY LEVELS				PHYSICAL FUNCTION MEASURES			Total RF
		Arms	Legs	Assistive Device	Fall Frequency	PADS ≤ 42	Exercise [†]	LTPA [†]	General Activity [†]	SPPB < 8	Balance ≤ 2	TUG ≥ 13.5 sec	
PPT1	ABI	X	X	X				X			X		5
PPT2	PD		X	X	X			X	X		X		6
PPT3	PD		X	X								X	3
PPT4	ABI	X	X	X		X	X	X	X	X	X	X	10
PPT5	ABI	X	X	X							X	X	5
PPT6	ABI	X	X	X					X				4
PPT8	PD												0
PPT9	PD								X				1
PPT10	PD	X	X						X				3
PPT11	PD	X	X	X	X	X	X	X				X	8
PPT12	PD				X	X	X	X	X				5
PPT13	PD								X				1
Total RF		6	8	7	3	3	3	5	7	2	3	4	

Check marks indicate participants are at risk of poor physical function and activity level. Portray poor physical function and activity level. Physical barriers include if participant has partial use of arms or legs, uses an assistive device, and reports frequent falls. [†]Indicates PPT does not participate in PADS sub-scores (exercise, leisure time, or general activity) and received a score of 0 or below. Abbreviations: PPT, participant; LTPA, Leisure-Time Physical Activity; RF, risk factor

Figure 1: Most Common Nutrition, Cardio-Metabolic and Physical Risk Factors



APPENDICES

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APPENDIX A: Review of Literature

I. Introduction

Parkinson's disease (PD), traumatic brain injury (TBI), and stroke are neurological conditions that can result in lifelong challenges that affect physical, cognitive and psychosocial functioning¹⁻⁵, which in turn can jeopardize dietary intake and increase likelihood of nutritional and cardiovascular disease (CVD) risk. Traumatic brain injury and stroke are subtypes of acquired brain injury (ABI), a term used to describe structural damage to the brain^{6,7}. Approximately 1,700,000 and 795,000 people experience a TBI or stroke, respectively, every year in the United States (US)^{7,8}. Parkinson's disease is a progressive, neurodegenerative, movement disorder that one million people in the US live with⁹. While these neurological diseases are each unique and have different long-term impacts, they have similar complications that can negatively impact nutritional status and dietary intake^{1,3,10}.

Limited research exists examining the relationship between cognition, nutrition and physical activity in those with one of these neurological disorders. Research has examined long-term (a year or more following diagnosis) nutritional status of those living with PD^{1,11,12}. Unintentional weight loss is typical at the time of PD diagnosis and nutritional status declines as the disease progresses¹. Meanwhile, nutritional status of patients in the acute stages of stroke and TBI has been assessed and malnutrition and muscle wasting are consistently reported¹³⁻¹⁶. Evidence indicates up to six months following a stroke and two months following a TBI are a high-risk time for malnutrition^{5,17}, however very little research has examined nutritional status a year after. Since nutritional status is compromised in the acute phase after an ABI, long-

term nutritional status is likely to be poor¹⁶. In addition to risk of poor nutritional status, physical disabilities associated with these neurological disorders^{3,18,19,20} warrant an assessment of physical activity and functioning. An assessment of both nutrition and physical activity will provide a clearer idea of if these neurological disorders are at increased risk of diet and exercise related comorbidities.

Research indicates there may be a protective cardio-metabolic effect of PD, even in those with PD that are overweight^{21,22}. Meanwhile, those with TBI are at increased risk for significant weight change, as well diseases related to physical activity and diet, such as coronary heart disease (CHD) and metabolic syndrome, a disease marked by abdominal obesity, hypertension, dyslipidemia and insulin resistance^{3,23}. Research indicates an obesity paradox seems to exist following stroke, decreasing risk of reoccurring stroke^{15,24}, however a cardiovascular risk assessment of those living with stroke has not been completed.

Due to complications of neurological disorders, it is implied that nutritional imbalances can result in over-nutrition or under-nutrition^{1,3,5,17,23}. While research has examined nutritional status in those with PD, limited research exists analyzing how cognition, nutrition and physical activity and functioning relate. Similar complications of these three disease states suggest research should examine long-term nutritional status of those with stroke and TBI, as well as cognition and physical activity. Due to this lack of research, a cross-sectional study including those living with PD, TBI and stroke is necessary to assess dietary quality, nutritional status, physical activity and physical functioning, as well as cardiovascular risk, and quality of life.

II. Parkinson's Disease

The Definition, Prevalence and Effects of PD

Parkinson's Disease is a neurodegenerative, movement disorder caused by the death of dopamine cells⁹. Dopamine cells are the neurons that regulate motor function²⁵ and are concentrated in the substantia nigra, the area of the brain that controls movement²⁶. In addition to motor symptoms (partial or full functional loss of a body part), those living with PD experience a variety of cognitive deficits, even during early stages of the disease²⁷. The direct and indirect cost of total PD in the US is approximately \$25 billion per year⁹. Up to 60,000 new cases of PD occur annually in the US²⁸ and once motor symptoms are apparent up to 80% of the dopamine-producing cells are dead²⁶. These motor and non-motor symptoms develop overtime and impact daily activity¹ and negatively impact quality of life²⁷.

Parkinson's disease is one of the most common late-life (beginning in the sixth or seventh decade of life) neurodegenerative diseases, impacting up to 2.0% of people 60 years and older²⁹. Men are one and a half times more likely to be diagnosed with PD than women⁹. Common motor impairments include bradykinesia (slowness of movement), akinesia (difficulty initiating movement), tremors, and rigidity³⁰. The three commonly identified subtypes of PD are based on major motor symptoms: 1) tremor dominant, 2) postural instability gait disorder and 3) akinetic-rigid^{31,32}. Postural symptoms are more common in women, while symmetrical upper-disease related motor symptoms are more common in men³³.

Parkinson's can be classified into a variety of different subtypes, usually based upon the dominant motor symptoms present. Parkinson's can also be identified in

stages based on the Hoehn and Yahr scale (HY)³⁴. This one through five scale was developed to describe degree of disability and functional limitations related to PD³⁴. Phase one describes early onset symptoms, while stage five would describe the final phases, where one is confined to a wheelchair or bed. The progression of the disease is usually marked by increased motor impairment⁹. These motor symptoms, as well as, non-motor symptoms, can impact dietary intake and compromise nutritional status^{35,36}. Non-motor symptoms prevalent in PD include: cognitive impairment, neuropsychiatric symptoms, sleep disturbances, fatigue, gastrointestinal symptoms and sensory disturbances^{37,38}. Cognitive dysfunction can occur early in diagnosis³⁹.

Parkinson's Disease and Effects on Cognition

Parkinson's disease is associated with a variety of cognitive deficits that appear in the earliest stages of the disease and continue to worsen as the disease progresses^{27,37,38}. Within 15 years of diagnosis, over 80% of those living with PD experience a form of cognitive impairment (deficiency in brain function that affects memory, language, perception, visuospatial aspects, ability to carry out tasks and judgment)⁴⁰ or dementia⁴¹. These alterations can interfere with everyday functioning²⁷. Older age at disease onset, depression, male gender, severity of motor symptoms and advanced disease stage are risk factors for the development of mild cognitive impairment (MCI) for those with PD¹⁰. Evidence of MCI in the early stages of PD is associated with the incidence of dementia in later stages of PD⁴².

Cognitive symptoms associated with PD are mostly a result of the pathological changes within the brain, including changes in the dopaminergic pathway⁴³. Dopaminergic drugs, such as levodopa (L-dopa), used to synthetically produce

dopamine have been associated with improving cognition in PD patients, as well as negatively impacting cognition^{44,45}. There are multiple reasons for cognitive changes in those with PD and many pathways are disrupted as a result of lack of dopamine deficiency. In addition to lack of dopamine, the presence of Lewy body production (abnormal aggregates of protein that develop inside nerve cells and interfere with signal conduction) and cell loss in the brain can also disrupt cognitive function^{39,46}.

In those newly diagnosed with PD, the most prominent cognitive issues are related to memory and executive functions (EF)⁴⁷. Executive function is the set of mental processes that help one connect past experience with present action⁴⁸. Deficits with EF include difficulty with concept formation, decision-making, cognitive flexibility and selective attention^{37,47,49}. Difficulties with EF can be attributed to damage of the frontal cortex as a result of PD^{47,49}. A cross-sectional study examining the pattern of EF in 34 participants with mild to moderate PD, with a mean duration of 68.2±52.4 months, found nearly 45% of participants had a degree of EF impairment⁴⁹.

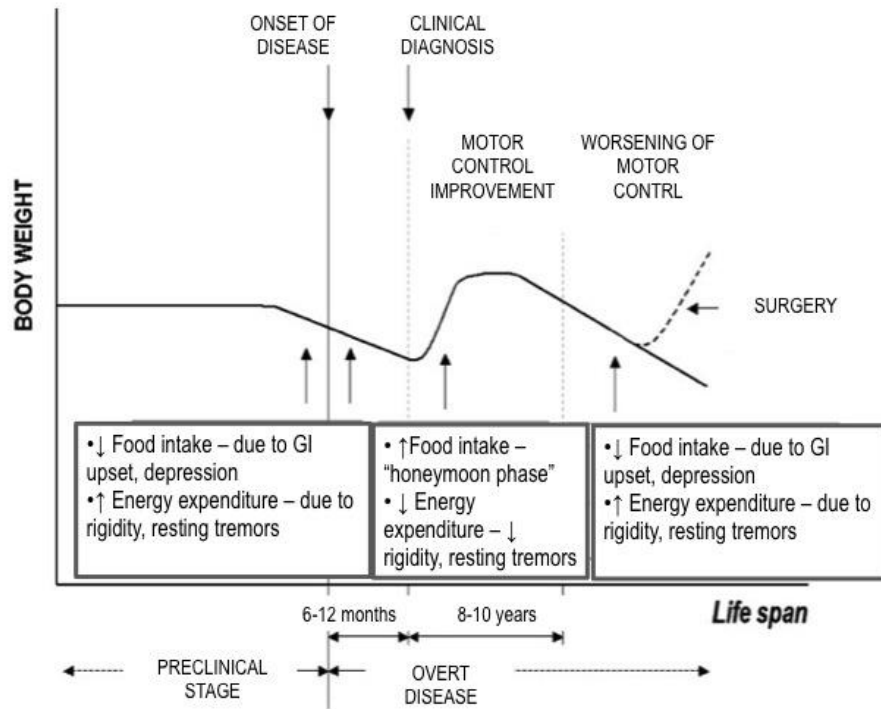
Those living with PD have issues with dual-tasking, which are tasks that require simultaneous performance of at least two cognitive functions along with motor functions⁴³. Wild and colleagues²⁷ examined dual tasking, in those with PD (n=18) versus healthy older adults (n = 18) and found that those with PD compromised the cognitive function tasks to focus on the motor task²⁷. Poor dual tasking is also an indicator of impaired EF. These symptoms may contribute to a compromised quality of life and health status^{39,47}, which in turn may impact one's ability to make proper dietary choices.

In addition to compromised EF and memory, neuropsychiatric conditions, including depression, anxiety and apathy, often precede motor symptoms⁵⁰. These conditions add significant burden to PD⁴³ and impact quality of life⁵¹. Paranoid delusions, hallucinations, impulse control disorder, and addictive symptoms can result due to treatment and medications for PD⁴³. These cognitive and neuropsychiatric impairments, combined with motor impairment, not only impact quality of life but have physiological side effects on the body that impact energy needs¹.

Physiological Side Effects and Energy Needs of PD

Physiological side effects and energy needs vary depending on the stage of the disease^{1,52}. Unintentional weight loss preceding a PD diagnosis is common⁵³, and weight then tends to increase immediately after diagnosis when treatment is initiated^{1,52}. The initial pre-diagnosis weight loss is attributed to the energy imbalance from the increase in energy expenditure as a result of motor symptoms, such as rigidity and dyskinesias (involuntary muscle movements) compared to reduced energy intake. A longitudinal, prospective study examined weight changes of 468 people with PD and found a mean weight loss of 5.2 pounds 10 years preceding diagnosis, but a fairly constant weight two to four years prior to diagnosis⁵³. Physiological changes in PD can also impact desire to eat as a result of hyposmia (reduced ability to smell) and change in eating habits as a result of PD^{1,54}. Generally, nutritional status declines as the disease progresses and resistance to various forms of available treatment develops (Figure A)¹. However, findings have contradicted this decline in weight as a result of treatment⁵².

Figure A:



Malnutrition and PD: Under-nutrition

Malnutrition, or poor nutritional status, is defined as an imbalance of energy, macronutrients and/or micronutrients that results in compromised body shape, size and composition, function and clinical outcomes⁵⁵. It includes under-nutrition, which results from insufficient intake to meet energy expenditure⁵⁶, and over-nutrition, which is caused by excessive calorie intake and/or inadequate activity⁵⁶. Protein-energy malnutrition refers specifically to deficiency rather than an excess of protein and energy³⁶. It also can result in a lower quality of life and poorer health outcomes, which in turn can increase risk of falls, hospitalization and admittance to aged care facilities^{36,57,58}. Assessments of nutritional status determine if one is malnourished

through examining anthropometrics, biochemical and clinical data, and dietary intake. Nutrition assessments of those living with PD typically find compromised nutritional status^{11,12,59}.

The average age of those diagnosed with PD is 60 years old⁶⁰. Change in weight, particularly weight loss, during aging is typical⁶¹, yet it seems to be greater in those with PD⁶². Changed in total body weight in men and woman overtime varies. Typically, men gain weight until about the age of 55 and women until the age of 65; after this point in time weight loss is common. Weight loss in healthy aging adults occurs because of the loss of lean muscle tissue, which is replaced with fat⁶¹.

Under-nutrition is prevalent in those living with PD^{11,12,59}, however it is difficult to determine how much of it is due to PD³⁶. The majority of PD patients are elderly and as a result are also experiencing physiological changes associated with aging⁶¹. In addition to weight loss, changes include reduction of digestive secretions, intestinal absorption, protein synthesis, and factors that decrease energy intake⁶³; these factors include altered sense of smell and taste, loss of teeth and ill-fitting dental prostheses⁶³. A mean weight loss of 7.7 pounds during the 8 years following diagnosis of PD has been reported⁵³. The study found energy intake to be similar between those with PD and those without PD shortly before diagnosis, but saw an increase in energy intake shortly after the diagnosis of PD⁵³. This research implies that weight loss associated with PD can be due to an increase in energy expenditure unique to PD, that healthy adults do not experience⁵³.

Compared to age matched controls, research has found lower body mass index (BMI) scores in those with PD⁶⁴. Other study have also found those with PD to have

significantly lower BMI scores ($24.4 \pm 0.5 \text{ kg/m}^2$) and percent body fat (%BF) ($22.2\% \pm 0.9$) compared to age-matched controls without PD ($26.3 \pm 0.4 \text{ kg/m}^2$, $24.6\% \pm 0.9$)⁶². The majority of these participants were in stage two of PD^{34,62}.

Motor and non-motor symptoms^{1,36} often compromise dietary intake in those living with PD^{36,59} and may also impact malnutrition. As PD progresses, a decreased response to treatment occurs and motor control declines^{1,65}. Decline in motor control, such as the presence of dyskinesia and rigidity raises energy expenditure and can lead to negative energy balance and weight loss¹. Physical changes and motor symptoms such as curve posture, shuffling gait and postural instability¹ make buying, preparing and eating food independently difficult^{1,35,66}. This development of chronic disabilities can interfere with activities of daily living (ADL), which include washing, dressing, eating and walking⁵⁸. Those with PD lose the ability to eat independently due to decreased hand-mouth coordination and decline in fine movements to use utensils^{30,67}. Motor symptoms can lead to a reduction in energy intake and weight^{1,66}.

Non-motor symptoms can also cause a decline in dietary intake³⁶ and includes depression, dementia, confusion, and apathy, as well as gastrointestinal symptoms; typical gastrointestinal symptoms include constipation, dysphagia (difficulty swallowing), early satiety, decreased gastric transit time, nausea, change in taste, loss of appetite and sialorrhea (excessive drooling), also cause a decline in dietary intake^{1,36,68,69}. A qualitative study examined food shopping, cooking and eating habits in older women with PD, and found decrease in sense of smell, appetite and taste in combination with issues transporting food to the mouth and swallowing were major issues associated with altering nutritional status⁷⁰. Due to these barriers to meet

nutrient and energy needs, those with PD are at increased for weight loss and loss of lean body mass³⁶. Weight loss is a natural part of the aging process, yet seems to be more common and severe in those with PD⁶².

Research indicates lower body weight and barriers for inadequate dietary intake in this population, indicating under-nutrition is present in this population. Findings reveal a strong correlation between lower Mini Nutritional Assessment (MNA) scores and time since PD diagnosis⁶⁵. A cross-sectional, population-based study, which included 135 PD cases in two regions of North East England, found that 21 of the 135 (15.4%) participants were classified as malnourished¹¹. The study also found that females with PD, were more likely to be severely malnourished (BMI <18.5) than male counterparts¹¹; this research is consistent to males and females without PD⁷¹.

Sheard and colleagues⁷² found similar results, where 19 of 125 (13%) participants were considered moderately malnourished, and, females were more frequently malnourished and more likely to experience unintentional weight loss compared to males with PD. Malnourished males in this study had a significantly higher median BMI score (21.1 (range:19.5-33.2) kg/m²) compared to malnourished females (19.5 (range: 17.7-21.6) kg/m²)¹². Males also had a significantly higher median waist circumference (86.75 (ranger: 78.9-116.0) cm) compared to females (73.5 (Range: 65.5-88.0) cm)¹². Patient-Generated Subjective Global Assessments in this study suggest muscle wasting in malnourished men and women living with PD¹². Due to the larger BMI scores and waist circumference in malnourished men with PD, accompanied by the presence of muscle wasting, these malnourished participants are

likely suffer from sarcopenic obesity, while malnourished females may suffer from both muscle and fat loss¹². Sarcopenia is the age-related loss of lean muscle mass and muscle function⁷³⁻⁷⁵. These results indicate that nutritional assessments for those living with PD are necessary. More research is needed to understand exactly why malnutrition occurs in this population and to help prevent malnutrition and sarcopenia, as well as to monitor how medications and treatment impact weight in this population.

Medications and treatment for PD may also impact weight and food choices^{1,64,76}. Dopaminergic treatment has been associated with a decrease in weight for those with PD who experience dyskinesia, common complication of L-dopa⁶⁴. Lower BMI scores are associated with advanced PD patients taking higher dosages of L-dopa⁶⁴. A common side effect of L-dopa is nausea and vomiting, to help limit this prescribed with carbidopa as to reduce symptoms of nausea and vomiting and help control PD symptoms. Reducing symptoms of nausea and vomiting, as well as seeking alternative treatments may help those with PD reduce risk of weight loss. While under-nutrition and weight loss are problematic for those with PD, weight gain can also occur and is associated with PD treatment¹.

Malnutrition and PD: Over-nutrition

Time of PD diagnosis is often associated with weight gain, because those living with PD begin treatment to control symptoms that promote weight loss¹. While some research has found weight loss in response to treatment for PD, other research has reported weight gain as a result. For instance, surgical procedures to treat PD, such as deep brain stimulation (DBS) are associated with significant weight gain^{52,77}.

Pharmacological medications can also cause weight gain^{52,76}. Dopamine agonists are medications that stimulate the parts of brain to believe it is receiving dopamine. Since they are not as potent as L-dopa, they are less likely to cause dyskinesia⁷⁸, which in turn would decrease energy expenditure associated with dyskinesia. They also can cause compulsive behaviors, include compulsive eating and undesirable weight gain⁷⁶. In a report by Nirenberg and Waters⁷⁶, seven cases of PD are described that experienced compulsive eating and undesired weight gain related to dopamine agonists, these participants saw an average 15% increase (13 kg \pm 7 kg) in body weight. Five of the seven participants decreased or discontinued dopamine agonists and self-reported a decrease in food cravings and weight⁷⁶. Despite a small sample size findings suggest further research should evaluate the impact of dopamine agonists, weight status and dietary habits on those living with PD.

Another treatment that impacts weight is DBS, a surgical option for those with PD, and utilized by approximately 30,000 people with PD worldwide⁷⁹. In this surgical procedure, an impulse generator is placed under the collarbone that provides an electrical impulse to the substantia nigra⁷⁹. It typically is recommended for those with PD who have complications from medication such as dyskinesia and severe motor fluctuations, as well as those who no longer responding to pharmacological treatment³⁰. The procedure can effectively reduce motor symptoms, which allows for a decrease in pharmacological medications⁸⁰. The effectiveness can reduce L-dopa dosage, in turn reducing negative side effects that impact dietary intake, such as nausea, vomiting, and involuntary movements associated with the medication.

Deep brain stimulation is also associated with an increase in appetite and restoration of hormonal balance. As a result, DBS promotes significant weight gain^{52,76,81}. An investigation of weight gain in 30 PD patients receiving DBS found 29 participants (96.7%) experienced weight gain within one year of treatment⁵²; the mean weight gain was 9.3 ± 6.2 kg⁵². Just over 46% reported increased weight within the first three months⁵². Additionally, 24% of participants reported weighing the most in their lifetime⁵². None of the participants experienced weight loss and there was a clear shift from being underweight and normal body weight to being overweight and obese⁵².

Contradicting previous research, a higher prevalence of overweight and obesity in those with PD seems to exist compared to the general population⁶³. Of 134 participants, 62% of participants were classified as obese or overweight, while 3% were classified as underweight⁶³. The mean duration of PD was 10.6 ± 5.3 years and mean age of participants was 65.9 ± 8.9 years⁶³. In comparing these participants to the general population, there was a higher proportion of obese people living with PD than living without PD (25 vs. 14%)⁶³. However there seems to be an inverse relationship between the course of PD and BMI⁶³. This highlights that while under-nutrition and excessive weight loss are an issue for those living with PD, weight gain and obesity are also present. Excessive weight gain and obesity are risk factors for CVD and other chronic diseases⁸²

Both weight loss and weight gain has been observed in this population and it is understood that nutritional status in those with PD is likely compromised³⁶ and to worsen as the disease progresses¹. Consequently, a nutrition assessment, evaluation,

monitoring and possible treatments should be included in the treatment plan for those with PD¹. Additionally, neurological disorders, such as TBI and stroke, with similar cognitive and physical complications as PD, may also experience malnutrition.

III. Traumatic Brain Injury

The Definition, Prevalence and the Effects of TBI.

Traumatic brain injury occurs from an external mechanical force, such as a jolt or blow to the brain, that causes brain dysfunction⁸³. There are two types of severe TBI's, closed and penetrating⁸⁴. A closed TBI is caused by movement of the brain within the skull and can be caused by falls, motor vehicle accidents, or being struck with an object⁸⁴. Penetrating head injuries are caused when a foreign object enters the skull and includes being struck with a sharp object or a firearm injury⁸⁴.

Each year, approximately 1.7 million people in the US endure a TBI and about 275,000 are hospitalized⁸. In the US, the total annual economic cost of TBI is \$60 billion⁸⁵. Those sustaining a TBI is bimodal, occurring primarily in younger or older adults⁸⁶. Older adults have the highest rates of TBI hospitalizations and deaths^{8,87}. The leading cause of TBI is falls (35%), followed by motor vehicle-related injuries (17%) and a strike or blow to the head (workplace or sports-related injuries) (16.5%)⁸. However, approximately 21% of the causes are unknown⁸. Amongst, military personnel, one in six U.S. service members deployed in support of Operation Enduring Freedom (OEF) or Operation Iraqi Freedom (OIF), sustained a TBI⁸⁸.

Complications following TBI persist as time progresses⁸⁹. Up to 5.3 million people (1.7%) in the US live with long-term disabilities as a result of a TBI⁸⁵, making TBI the leading cause of disabilities. Long-term disability is defined as the loss of one

or more physical or mental functions⁸⁵. A model created to predict long-term disability following TBI hospitalization found about 43.3% of a population based sample sustained long-term disability⁹⁰; findings highlight side-effects of TBI are a serious public health issue in the US⁹⁰.

Following TBI, chronic, late emerging health implications can occur and include neurologic, mobility, endocrine, swallowing and gastrointestinal issues³. Breed and colleagues⁸⁶ examined those who sustained a TBI up to four years ago, and noted that younger and older adults with TBI reported musculoskeletal issues more frequently than those living without TBI. In addition to physical impairment, acquired cognitive impairments as a result of TBI can impede everyday activities and interfere with productivity, independence as well as safety⁹¹. As a result, it is essential to manage the effects of TBI and analyze how cognition, nutritional status and physical function are impacted a year or more after injury.

TBI and Effects on Cognition

The cognitive effects following a TBI can last a life-time⁸⁶. Neurological difficulties following TBI include seizures, spasticity, balance difficulties, loss of urinary control, communicative impairment, and chronic headaches⁸⁹. Individuals who sustain brain injuries frequently have trouble with concentration, memory, perception, attention, social interaction, managing emotions, problem solving, and decision making⁹¹. These complications can interfere with multiple aspects of all aspects of daily activity⁹¹. A study analyzing functional status among OEF/OIF veterans with a history of TBI found reduced functional status was strongly correlated with poorer cognitive function⁹², particularly related to motor function, processing

speed and executive function⁹². These impairments highlight TBI's impacts cognitive functions related to carrying out ADL and to make decisions.

Cognitive complications in those living with TBI change over time. However, research indicates after a mild to severe TBI, cognition improvement occurs during the first two years, but remains impaired beyond this point⁹³. During the early recovery phases, people seem to have problems with arousal, attention and memory⁹¹.

Although problems with attention and memory persist overtime, particular difficulties with executive function, attention and cognitive control mechanisms become more prominent⁹¹. A longitudinal study followed older adults with TBI for 12 months following injury, finding poor processing speed, verbal, memory, language and executive function compared to older adults without TBI⁹⁴. Traumatic brain injury is also associated with increase risk for PD and other neurodegenerative diseases^{95,96}.

Furthermore, older adults with closed TBI tend to have issues with naming and word fluency under timed conditions, as well as with verbal and visual memory⁹⁷. Impaired cognitive function in this population seems to exacerbate other health related issues³ and the Brain Injury Association of American recognizes additional research is necessary to further understand the disease and develop clinical guidelines and modifications⁹¹. Additionally, how these cognitive issues impact dietary intake, nutritional status and whether they promote nutritional risk needs to be further examined.

Physiological Side Effects and Energy Needs of TBI

Research has extensively examined the physiological side effects and impact on nutritional status during the acute phase of TBI, from time of diagnosis to six

months^{10,11}. The body undergoes hyper-metabolic, hyper-catabolic, and hyper-dynamic responses¹³. These physiological responses are not only a result of the injury, but due to unfavorable secondary events from TBI⁹⁸. These secondary events include fever, increased intracranial pressure, and seizures⁹⁸. Consequently, following a TBI the body's energy expenditure and nitrogen excretion increase¹⁴. This increased energy expenditure results in a catabolic state, increasing muscle breakdown as well as negative effects on immune function and morbidity⁹⁹. Energy and protein needs increase and meeting these requirements is imperative to prevent and prolong muscle wasting and excessive weight loss¹³. To treat those with a TBI during the acute phase, early nutrition support decreases risk for morbidity and mortality^{100,101}.

A retrospective study explored nutritional treatment (parenteral and enteral nutrition) and outcomes up to six months after a TBI¹³. A nutrition assessment found 68% participants exhibited signs of malnutrition during this time period, with up to 29% body weight loss¹³. This study revealed that in most participants weight continued to decrease one to two months after injury¹³, highlighting the increase in energy expenditure and importance of adequate nutrition during the immediate stages. However, what happens beyond this point in those living with a TBI needs to be further examined.

Malnutrition and TBI: Under-nutrition

Nutritional status following the acute phase of a TBI has not been extensively examined. Research has been reported immediately following a TBI and found those living with TBI seem to reach lowest weight two months after injury¹³. Research has

also examined nutritional status up to six months after injury, typically this time is the rehabilitation stage¹³.

While research has not examined nutritional status in those living with TBI a year or more, chronic complications following a TBI may compromise dietary intake and lead to poor nutritional status³. Self-reported chronic issues of TBI include changes in weight and appetite, sensory changes, swallowing problems, chronic pain and dystonia (a neurological movement disorder)³. Due to similar chronic symptoms in those living with PD and TBI, there is reason to assess the impact physical and cognitive disabilities, including depression associated with TBI³, has on nutritional status and ability to buy and prepare food. This is especially true since research has shown physical and cognitive disabilities impact dietary intake and promote difficulty preparing meals in those living with PD^{1,36,68,69,70}.

In a systematic review, Murphy and Carmine³ note dysphagia is common immediately following a TBI, and can persist or even worsen overtime. Achieving adequate nutrition and hydration can be a lifelong challenge for those living with TBI and dysphagia³. Compared to the general population, those with TBI are two and a half times more likely to die of digestive disorders¹⁰². The most common digestive disorders include gastro-esophageal reflux disease, which occurs when acid re-enters the esophagus due to irregular relaxation of the lower esophageal sphincter. It can cause heartburn, regurgitation, dysphagia, aspiration pneumonia, chest pain and nausea³. While, gastrointestinal disorders can cause a decrease in dietary intake and weight loss, it is often caused by obesity, use of steroid medication and hiatal hernia³.

Malnutrition & TBI: Over-nutrition

A common late-onset complication of TBI is weight gain³. Excessive weight gain associated with TBI can be due TBI's impact on metabolism and hormone production, memory, self-regulation, mobility, altered satiety cues, and appetite altering medications^{3,86}. Weight gain for those with disabilities significantly increases risk for CVD, diabetes, functional dependence, and immobility³. Weight gain in TBI is associated with metabolic syndrome, which can lead to stroke, retinopathy, peripheral artery disease, diabetes and cardiovascular disease^{3,86}.

Additionally, TBI is associated with post-traumatic hypopituitarism (PTHP) (decrease in the release of pituitary hormones to regulate homeostasis in the body); approximately 25% of long-term survivors have post-traumatic hypopituitarism¹⁰³. The number of people with PTHP after a TBI is a concern because the condition is associated with increase in weight and metabolic imbalances up to two months after a TBI¹⁰⁴. The pituitary gland is a vital link between the nervous system and endocrine system and releases hormones that impact metabolism and other life processes¹⁰⁵. It is often undiagnosed in those with TBI because of the underlying primary diagnosis of TBI, but can be treated with hormone replacement therapy¹⁰³. Klose and colleagues²³ found those with TBI and PTHP have higher body compositions and BMI compared to those without PTHP up to 12 months after injury. An increase in BMI from three months ($27.1 \pm 5.7 \text{ kg/m}^2$) after injury to 12 months ($29.3 \pm 5.7 \text{ kg/m}^2$) was found²³. Those with PTHP were also older, and had a more severe TBI. What happens beyond this point regarding weight and body composition needs to be examined.

Weight change is a common theme in those living with TBI²³. Hibbard and colleagues⁸⁹ found those living with TBI for a year or more reported unintentional weight loss or gain greater than ten pounds. However, to our knowledge, the research has not examined long-term (12 or more months following TBI) weight status and dietary intake in people with TBI. How diet, as well as physical inactivity, plays a role in weight gain and if weight gain continues overtime needs to be examined. Since research indicates nutritional status and weight change occurs in TBI, a form of ABI, it is likely that changes also occur in those living with other forms of ABI, such as stroke.

IV. Stroke

The Definition, Prevalence and Effects of Stroke

A stroke results when the brain's blood supply is blocked or when a blood vessel in the brain bursts¹⁰⁶. The American Heart Association (AHA) estimates that someone in the US has a stroke every 40 seconds⁸². By 2030, nearly 4% of the US is projected to have experienced a stroke, and there will be an additional 3.4 million people with stroke compared to in 2012¹⁰⁷. Stroke is the fourth leading cause of death¹⁰⁶ and 80% of stroke can be prevented¹⁰⁶. Maintaining a healthy diet and exercising are two ways to help prevent a stroke¹⁰⁸. Eating five servings of fruits and vegetables per day has been found to lower risk of stroke by 30%¹⁰⁸. Additionally, a meta-analysis found those who had high physical activity levels had a 27% lower risk of stroke¹⁰⁹. Stroke occurs mostly in adults over the age of 65⁷. Women ages 55-75 have a one in five chance of stroke, while male counterparts have a one in six chance¹¹⁰; each year approximately 55,000 more women than men have a stroke¹¹¹.

Although age-specific stroke rates are high in men, women have much higher incidences at older ages due to the longer life expectancy in women¹¹².

The total annual direct and indirect costs of stroke are forecasted to increase to \$240.67 billion by 2030, an increase of 129%¹⁰⁷. Immediately following a stroke, at least 40% of stroke survivors have moderate disabilities and up to 30% have severe disabilities¹¹³. A stroke may impact psychological, physical, social and cognitive function¹¹⁴. These complications can alter a person's life and increase risk of being institutionalized, becoming psychotic or depressed, or of dying¹¹⁴. Of a 180 people over 65 years old, six months after stroke, 50% had hemiparesis, 46% had altered cognitive function, 35% had symptoms of depression, 30% were unable to walk without assistance, and 19% had aphasia¹⁹. Similar to PD and TBI, side effects of stroke impact physical and cognitive function and may also lead to assistance with ADL¹¹⁵, as well as a decrease in dietary intake and quality overtime.

Stroke and Effects on Cognition

Cognitive-linguistic deficits following a stroke reduces functional outcome for at least five years after stroke in some individuals¹¹⁶. Assessments of cognitive-linguistic function after stroke assess memory, information processing speed, visuoperceptual/construction ability, EF, attention, language and depression^{114,117}. Impaired memory, poor concentration and mental slowness are the most common cognitive complaints after a stroke^{118,119}. The functional status of 126 stroke patients was examined, (mean age 76.9 ± 10 years) at baseline (2-3 weeks after stroke) and 13 months following event of stroke¹²⁰. Findings concluded cognitive function at baseline

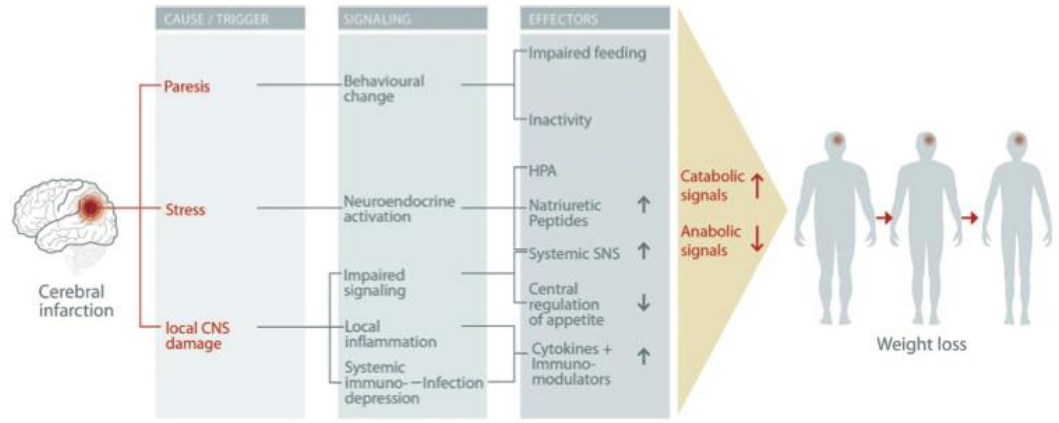
was an independent and significant predictor of long-term functional outcome and degree of disability after stroke¹²⁰.

Subjective cognitive complaints in 25 older adult stroke patients (mean age 67±10 years) were assessed three to nine months after stroke and revealed 90% of participants reported cognitive issues or impairment⁴. Another study found attention, language, construction and reasoning the most affected cognitive limitations, where orientation was least affected¹²¹. Depression was the main variable that made a significant independent contribution to the prediction of subjective cognitive complaint⁴. In general, cognitive decline, is associated with risk for functional decline in ADL¹²², which can impact dietary intake and physical activity level. Whether these cognitive issues in junction with physiological side effects of stroke impact dietary intake and physically activity level, as well as increases risk of reoccurring stroke must be assessed.

Physiological Side Effects and Energy Needs of Stroke

Similar to TBI, the physiological side effects and impact of nutritional status during the acute phase of stroke has been thoroughly examined^{15,123}. Following a stroke the body is in a hyper-catabolic state, which increases energy needs^{15,16}. These energy needs are usually not met and as a result, fat and muscle tissue wasting and weight loss occurs (Figure B)¹⁵. The body's response to stroke leads to functional decline and the depletion of energy stores¹⁵. People who experience tissue wasting may also experience cachexia (dramatic weight loss and muscle wasting)¹²⁴ or sarcopenia, which can contribute to a poorer prognosis after stroke¹⁵.

Figure B:



Malnutrition and Stroke: Under-Nutrition

In addition to weight loss from this hyper-metabolic state, persons in the acute phase of stroke are susceptible to weight loss due to fever, local central nervous system damage, and cytokines and oxygen-free radical build-up due damage of the blood vessel lining¹⁵. Impaired feeding and dysregulation of appetite can also promote weight loss following a stroke¹⁵. Finally, inactivity due to paralysis, can promote weight loss due to a loss of lean muscle mass^{15,125}, and severely interfere with one’s ability to prepare meals. As a result, malnutrition can result from inadequate intake of kilocalories, protein and/or nutrients the body requires for maintenance and repair¹¹⁵. Malnutrition as a result of stroke can negatively impact physical and social well-being and quality of life¹²⁶.

Decline in nutritional status during the acute phase of stroke is highlighted in a prospective, descriptive study examining the risk and prevalence of malnutrition of 23 participants¹²³. At baseline, 21 of 23 participants were considered well-nourished, however significantly more participants were malnourished or at risk of becoming

malnourished ten days later¹²³. Based on MNA scores, after ten days, eight participants (35%) were well-nourished, nine (39%) were at risk of malnutrition and six (26%) were malnourished¹²³. The MNA not only exams anthropometrics, but also includes a dietary questionnaire, as well as a global and subjective assessment. While MNA scores indicated decline in nutritional status, there was no significant change in weight and BMI scores between these two time periods¹²³. Despite a small sample size, this decline in nutritional status highlights rapid changes that occur during the acute phase of stroke.

Impaired feeding during the acute phases of stroke can be due dysphagia, and up to 65% of people with stroke experience dysphagia¹²⁶. Many regain functional swallowing usually within the first month following a stroke¹²⁷, however, for some, dysphagia is still present six months after stroke¹²⁸. Dysphagia can compromise food and fluid intake, which can alter consciousness and promote physical weakness. The physical weakness experienced post-stroke can also negatively impact swallowing. Complications from dysphagia can also increase length of hospital stay, rehabilitation time, and result in a poorer long-term outcome¹²⁶.

Change in consciousness, difficulty swallowing, inadequate food and fluid intake, and an increased in energy expenditure as a result of stroke can result in severe malnutrition^{15,126}. Whether nutritional status improves or further declines after the acute phase needs to be more closely examined. Since nutritional status is likely to decline in the acute stage, where there is closer monitoring over dietary intake, it is necessary to examine what happens to nutritional status in people with stroke as time progresses.

Westergren⁵ found stroke survivors who self-reported low energy levels, were more prone to poor nutritional status, since they may stop eating before daily energy needs are met. Research has examined nutritional status in the month following a stroke, and found chronic disabilities that interfere with ADL related to eating including purchasing groceries, preparing food, and eating⁵. Eating difficulties, barriers that negatively interfere with the preparation and intake of food and fluids¹²⁹, are common among stroke survivors and negatively impact dietary intake, as well as quality of life^{123,130}. Eating difficulties (sitting position, managing food on plate, transporting food to mouth, opening and closing mouth, manipulating food in mouth, swallowing, food consumption, alertness and eating speed) related to stroke were observed.

A comparison of eating difficulties and nutritional status in the acute phase (the days immediately following stroke) was compared with nutritional status three months after stroke. The research found that majority of baseline eating difficulties, observed continued three months later. Inadequate food intake, increased in 22 participants in the acute phase to 32 participants post intervention¹⁷. Despite these observations, nutritional status, based on total MNA scores, significantly improved three months after a stroke¹⁷. The contradiction of improved MNA scores compared to the observed inadequate food intakes, suggests nutritional status in those with stroke needs to be examined more thoroughly, and that the MNA may not be an effective tool in assessing nutritional status overtime in this population.

Other research has highlighted malnutrition to be an issue beyond the rehabilitation phase and up to six months after stroke compared to the acute

phase^{131,132}. Six months after stroke 82% of women and 78% of men with eating disabilities did not meet energy requirements. Nutritional status, male gender, age, and stroke severity at admission were significant risk factors for poorer functional outcomes six months post-stroke¹⁶. Weight loss as little as three kilograms or more following a stroke has been an independent predictor of adverse outcomes following stroke¹³³, this weight loss has been found to occur up to a year¹³⁴. As a result, malnutrition during acute stroke is considered a potentially modifiable factor that should be corrected and monitored to help improve long-term functional¹⁶. Whether weight loss continues to persist after a year following stroke must also be assessed, especially since many eating difficulties are associated with stroke¹³⁵.

Eating difficulties following a stroke highlights that individualized therapy after stroke to improve eating ability is imperative¹³⁵. More research is needed to understand whether malnutrition persists a year or more after stroke, as well as preventing stroke reoccurrence. Additionally, due to the susceptibility of malnutrition in the months following a stroke, an obesity paradox exists. While, being overweight/obese increases risk of stroke, 22% and 64% respectively¹³⁶, research has found an obesity paradox following a stroke¹³⁷. Being overweight or obese at the time of stroke seems to result in better long-term functional outcomes for those living with stroke¹³⁷.

Malnutrition and Stroke: Over-nutrition

An obesity paradox seems to exist with stroke patients, in that mortality seems to be lower in obese patients¹³⁷. Those who are underweight at the time of stroke seem to have more complications and poorer survival than those who are of normal

weight or overweight when assessed six months after stroke¹³⁸. The underlying cause of this paradox is currently unknown, but supported from research with large study populations¹³⁹. The notion of the obesity paradox and stroke is often attributed to the catabolic imbalances and inflammatory state immediately following a stroke that causes weight loss¹⁵. This weight loss also causes muscle tissue wasting, which impairs recovery after stroke¹⁵.

While obesity increases risk of a first-time stroke, however, those who are overweight or obese post-stroke, are less likely to have a reoccurring stroke in the years following stroke^{139,140}. Through examining data from the Danish Stroke Registry, Andersen and Olsen¹⁴⁰ found as BMI increases, the risk of a stroke reoccurring decreased, despite age, sex, stroke severity or cardiovascular risk profile. Underweight patients more likely had a history of a previous stroke¹⁴⁰. However, other research indicates, normal weight patients were more likely to have a reoccurring stroke²⁴. This is a concern since research implicates compromised dietary intake in six months following¹²⁹, which can promote a further decline in weight.

The obesity paradox seems to persist as time following a stroke progresses. A prospective study examined stroke survivors up to 30 months after stroke or a transient ischemic attack, and found overweight and obese participants to be at the lowest risk for the following endpoints: morbidity, mortality, and poor functional outcomes²⁴. Those positive outcome measures were found the most in obese or very obese, while underweight participants were at the highest risk for all endpoints²⁴.

Overall, existing research supports obesity paradox and stroke, however a recent study examining stroke rehabilitation outcomes, slightly contradicts the idea of

obesity paradox¹⁴¹. The relationship between functional independence measurement (FIM) scores, including cognitive and motor sub-scores, and BMI scores for those in stroke rehabilitation was examined and those considered overweight had the highest motor and cognitive sub-scores¹⁴¹. There was no significance differences between motor FIM sub-scores between groups, but the overweight group had significantly higher cognitive sub-scores compared to the obese group¹⁴¹. No significant differences were found between normal weight stroke participants and underweight or overweight participants for cognitive sub-scores¹⁴¹. Whether this holds true beyond the rehabilitation phase has not been assessed, and highlights the need to develop proper guidelines for weight status and dietary intake following a stroke. Especially since research has found an association between obesity and poor cognition in the aging population¹⁴².

Due to mixed results, more research is needed understand how to help optimize health and prevent a reoccurring stroke or other forms of chronic disease. Research regarding the obesity paradox and stroke contradicts current guidelines to reduce body weight after stroke¹⁵, and clearer guidelines are needed to promote optimal weight status in this population, especially long-term (a year or more) after stroke. Stroke is a cardiovascular event, and as a result it is important to continue to assess the cardiovascular risk of this population to prevent the occurrence of another cardiovascular event. Due to the similarities this neurological condition has with TBI and PD, the cardiovascular health of these populations must also be examined.

V. Overall Cardiovascular Risk

Across these three disease states research varies as to whether or not those living with neurological conditions such as PD, TBI and stroke are at increased CVD risk^{3,52,63,143} and this may be due to change in lifestyle following diagnosis as well as physiological impacts of the disease. Cardiovascular disease is the leading cause of death in the United States¹⁴⁴. It is the disease of the heart and/or blood vessels and often results from atherosclerosis, which is the hardening of the arteries¹⁴⁵. Modifiable CVD risk factors include being overweight or obese, having hypertension, elevated blood glucose levels, and dyslipidemia¹⁴⁶. As well as life style choices such as physical inactivity, tobacco use and a diet high in saturated fat and processed foods and low in fruits, vegetables and fiber^{146,147}.

To assess if one is overweight or obese, BMI and %BF are indicators. Body mass index is calculated from weight and height and can serve as an indicator of body fatness¹⁴⁸. Body mass index is classified according to predetermined categories: underweight = $<18.5 \text{ kg/m}^2$, normal weight = $18.5\text{-}24.9 \text{ kg/m}^2$, overweight = $25\text{-}29.9 \text{ kg/m}^2$ and obese $\geq 30 \text{ kg/m}^2$ ¹⁴⁸. According to the American Council on Exercise¹⁴⁹, any female with $\geq 32\%$ BF and any male with $\geq 25\%$ BF are considered obese.

Blood pressure is a clinical risk factor for CVD. Systolic blood pressure (SBP) $>120 \text{ mmHg}$ and diastolic blood pressure (DBP) $>80 \text{ mmHg}$ are considered suboptimal. When diagnosing for metabolic syndrome, SBP $\geq 135 \text{ mmHg}$ and DBP $\geq 85 \text{ mmHg}$ are used; these levels are considered pre-hypertensive¹⁵⁰. Hypertension can be caused by lifestyle factors such as a high sodium diet, obesity and physical inactivity¹⁵¹. Suboptimal serum levels are also a risk factor for cardiovascular disease and can be affected by poor diet and physical activity¹⁵². Total serum cholesterol >200

mg/dL, low density lipoprotein cholesterol (LDL-C) >100 mg/dL, high-density lipoprotein cholesterol (HDL-C) < 40 mg/dL, and triglycerides > 150 mg/dL are used to describe those with dyslipidemia¹⁵³. Dyslipidemia can lead to hardening of the arteries over time, which can lead to CVD¹⁵⁴. Hyperglycemia is marked by fasting blood sugar > 100 mg/dL¹⁵⁰ and can indicate insulin resistance, metabolic syndrome, pre-diabetes (110-125 mg/dL) or diabetes (>126 mg/dL)¹⁵⁵.

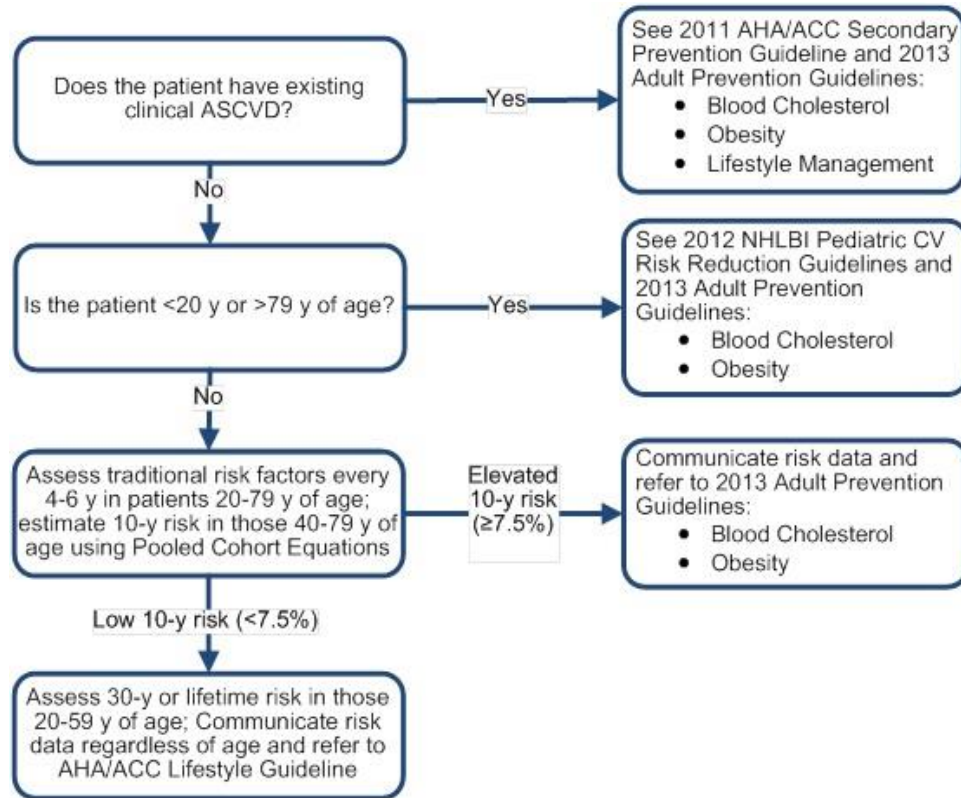
The development of clinical and biochemical CVD risk factors are impacted by diet and physical activity. An inverse relationship has been found with fruit and vegetable consumption and the risk of CVD¹⁴⁷. Consuming fruits and vegetables >3 times a day compared to once a day was associated with a 27% lower stroke incidence, a 42% lower stroke mortality, and a 27% lower cardiovascular disease mortality¹⁴⁷. Increasing fiber intake, which can be found in fruits and vegetables and whole grain products, has also been found to reduce modifiable CVD risk factors such as dyslipidemia, diabetes and being overweight¹⁵⁶⁻¹⁵⁹. Physical inactivity can also negatively impact blood pressure, lipid profiles, fasting blood glucose and weight, which in turn can lead to a cardiovascular event¹⁶⁰.

In addition to modifiable risk factors that can increase risk of CVD, Non-modifiable CVD risk factors include age, gender, race and family history¹⁴⁵. Risk of developing heart disease increases with aging¹⁴⁵. Men are at greater risk of heart disease than pre-menopausal women, however once women are post-menopausal risk is similar across both genders¹⁴⁵. African Americans seem to be at greater risk for developing CVD¹⁶¹. Finally, risk also increases if a first-degree relative had a cardiovascular event before the age of 55¹⁴⁵. To assess and treat these risk factors, the

American Heart Association and American College of Cardiology developed 10-year and lifetime risk estimates using algorithms for the development of atherosclerotic cardiovascular disease (Figure C)¹⁶². As described previously, those living with PD, TBI or stroke experience changes in physical and cognitive function³, which can not only impact nutritional status and dietary, but also promote a sedentary lifestyle, which may increase the risk of CVD in this population.

Research indicates those living with neurological conditions such as PD, TBI and stroke may be at increased CVD risk because they have one or more of these modifiable risk factors, such as a sedentary lifestyle, elevated lipid profiles or inadequate diets^{3,52,63,143}. As described earlier, neurological conditions can impact dietary intake, especially for those living alone. Generally, older men living alone are at risk of malnutrition and need a supportive environment to provide optimal dietary intake and help compromise cardiovascular health¹⁶³. A recent study analyzed the daily eating events among older men living with PD, stroke or rheumatoid arthritis and found that men living alone used more convenience foods such as instant and ready-to-eat foods, compared to those who lived with a significant other¹⁶³. Convenient foods are often higher in sodium, sugar and fat compared to fresh, homemade meals and can increase risk for chronic disease, including CVD¹⁶⁴.

Figure C:



1

While poor dietary intake may increase cardiovascular risk in those with stroke and PD, limited research exists regarding the dietary intake of those living with TBI. A late-onset complication of TBI is weight gain which can be a result from hormone and metabolic imbalances, as well as changes in mobility, self-regulation, altered satiety cues, and from medications that alter appetite^{3,86}. Weight gain can lead to metabolic syndrome, which seems to develop in many of those living with TBI³. Metabolic syndrome is associated with excess food consumption, which can greatly be due to larger portion sizes, convenience foods and the availability of foods high in

¹ Algorithm developed by The American Heart Association and American College of Cardiology to estimate 10-year and lifetime risks for the development of atherosclerotic cardiovascular disease (ASCVD)

saturated and trans fats and added sugars^{165,166}. The prevalence of metabolic syndrome can also increase risk for the development of CVD in this population³. Yet, the prevalence of those with TBI who have CVD or may be at risk for CVD has yet to be assessed.

Meanwhile, the prevalence of overweight and obesity seems to change throughout the course of PD, depending on the stage of PD and side effects of medications¹. Research has found a lower prevalence rate of stroke in those with PD compared to the general population¹⁶⁷. The mechanism behind this is not fully understood. There seems to be a higher prevalence of those overweight and obese with PD compared to the general population, however being overweight/obese with PD does not seem to increase risk for CVD¹⁶⁸. While, research has found this association, the mechanism is still not quite understood, and as result monitoring weight is necessary to help prevent other declines associated with excessive weight gain.

Besides weight, research has also analyzed lipid profiles in those with PD. The relationship between cardio-metabolic risk factors and duration of PD has been examined to find that the longer one is living with PD, the better HDL-C levels are²². An inverse relationship of the TC-to-HDL-C ratio was also observed²². Research has also examined the effect of lipid profiles on those with TBI. Hypopituitarism is a common side effect following TBI and those living with TBI and PTHP frequently present metabolic alterations that include altered glucose levels, insulin resistance and hypertriglyceridemia and dyslipidemia¹⁶⁹.

In a total risk assessment, patients with a TBI appear to be at significantly increased risk for stroke¹⁷⁰, as well as diabetes mellitus, hypertension, atrial fibrillation and CVD^{3,16,89,170-172}. Those with PTHP twelve months after TBI, had a higher total and LDL-cholesterol, as well as significantly higher BMI ($23.9 \pm 3.4 \text{ kg/m}^2$ vs. $27.1 \pm 29.3 \text{ kg/m}^2$), waist circumference ($84 \pm 10 \text{ cm}$ vs. $102 \pm 18 \text{ cm}$) and total and abdominal FM compared to those TBI patients with intact pituitary function²³. Mean BMI score for insufficient pituitary TBI patients increased significantly from three months to 12 months following injury, from 27.1 kg/m^2 to 29.3 kg/m^2 . Additionally, total cholesterol significantly increased from $174.1 \pm 46.4 \text{ mg/dL}$ to 208.8 ± 27.1 in those with insufficient pituitary function¹⁰⁴. For those without PTHP, mean BMI scores remained stable and within normal limits, and total cholesterol levels saw a modest decrease²³. This study highlights the presence of cardiovascular risk in those with PTHP and TBI 12 months following injury, however what happens to weight status in TBI patients without and without PTHP beyond a year needs to be assessed.

When enough cardiovascular risk factors are present, this can lead to a cardiovascular event. Traumatic brain injury is associated with increased risk of stroke, a population based study followed those living with TBI for 5 years after injury, and found TBI patients had 10.21 times, 4.61 times and 2.32 times greater risk of stroke compared to non-TBI patients at 3 months, one year and five years post diagnosis¹⁷⁰. At three months following TBI diagnosis, 675 strokes occurred in those with TBI, while only 207 cases were reported in non-TBI patients¹⁷⁰. This study speculates the increase risk of stroke in those with TBI can be from the disturbance of blood supply to the brain post TBI, the formation of an atherosclerotic blood vessel

due to the sudden impact after a TBI, or an increase in intracranial pressure and blood pressure post TBI¹⁷⁰. Antipsychotic drugs used to treat TBI may also increase risk of stroke¹⁷³. The presence of cardiovascular risk factors previously described in this population^{3,23}, as well as the role diet and physical inactivity plays in developing CVD risk factors may also increase risk of stroke. As a result a more thorough assessment to understand the relationship between TBI and a stroke event is warranted. Additionally, since both TBI and stroke are forms of ABI, the presence of CVD risk factors in TBI give warrant an examination of CVD risk n those living with stroke.

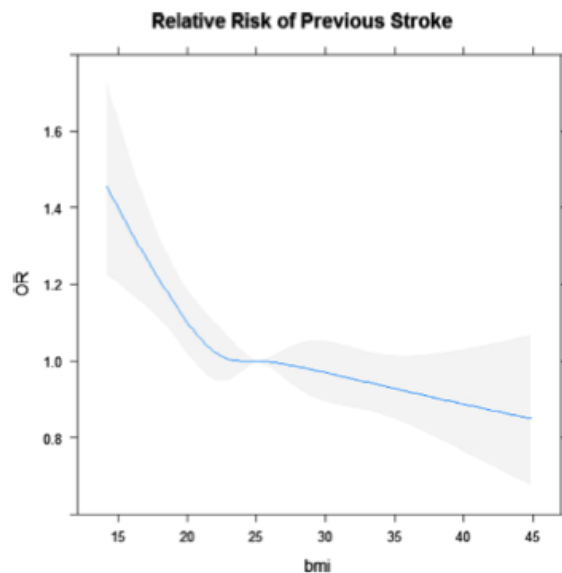
Whether those living with stroke remain at cardiovascular risk following event of stroke needs to be assessed. As described earlier, an obesity paradox seems to exist following stroke, and is associated with a decrease risk in stroke reoccurrence¹⁴³. The AHA proposed guidelines to prevent stroke reoccurrence, target similar modifiable and non-modifiable risk factors associated with CVD prevention¹⁷⁴. The guidelines note that while obesity increases risk of stroke, no study has found weight reduction to reduce risk of stroke recurrence¹⁷⁴. Additionally, Andersen and Olsen¹⁴⁰ found patients with recurrent stroke are more often men, underweight and have a history of heart attack, atrial fibrillation, and hypertension. This study highlights the negative correlation between BMI and stroke recurrence based on an odds ratio (OR) (Figure D)¹⁴⁰. It also highlights that risk factors for stroke and other cardiovascular events continue to exist in those following stroke if lifestyle modifications are not made.

A final modifiable risk factor that increases the chances of developing CVD is smoking tobacco. Smoking increases risk for coronary heart disease and stroke by two to four times¹⁷⁵; it can also reduce blood flow to legs and skin¹⁷⁵. The prevalence

of smoking amongst those with neurological diseases and the influence it has on cardiovascular health in this population needs to thoroughly be examined. In addition to lifestyle choices, such as smoking tobacco, physical activity and function are associated with cardiovascular risk. Due to the nature of these three diseases, physical activity level may be limited. Physical impairment can promote a sedentary lifestyle and weight gain, but also negatively impact lean body mass.

Figure D:

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VI. Physical Activity and Physical Function

Physical activity is bodily movement by the skeletal muscles that results in energy expenditure¹⁷⁶. It includes leisure time activities, such as sports and exercise, and ADL¹⁷⁶. Physical activity curtails risk of mortality and morbidity¹⁶⁰. Health benefits of physical activity have been well documented, including weight management, and preventing and treating cardiovascular disease and musculoskeletal

disorders¹⁶⁰. Physical activity promotes good physical health status, as well as mood and cognitive function, which can improve quality of life¹⁷⁷. Evidence shows physical activity can help reduce age-related physical and cognitive decline¹⁷⁸⁻¹⁸⁰. A recent study examined the impact of a physical and leisure activity programmed on community-dwelling older adults and found the intervention had improvement in areas of cognition, specifically in abstract reasoning ability, a common initial symptom of dementia¹⁸¹, as well as significant in QOL.

Physical activity greatly relates to physical function, which is one's ability to carry out tasks related to activities of daily living, as well as quality of life¹⁸². Limited physical function can be due compromised upper and lower extremity function¹²². Compromised physical function can interfere with performing different tasks such as walking, climbing stairs, lifting heavy objects, picking up an object off the floor¹⁸³, bathing, dressing, eating and cleaning¹⁸⁴. A decline in physical function may lead to sedentary behaviors, which combined with excess calorie intake, can increase risk for obesity¹⁸⁵. Obesity is further associated with functional limitations¹⁸⁶.

Many older adults develop sarcopenic-obesity, and this increase in body fat and decrease in muscle mass has been found to accelerate decline in physical function^{183,187}. To treat and prevent complications of sarcopenic-obesity, incorporating physical activity can increase muscle mass and decrease fat mass, and in turn enhance physical function¹⁸⁸. However, low BMI scores are also associated with compromised physical function, resulting in increased risk of falls and inability to carry out ADL¹⁸⁹.

Guidelines for physical activity have been created to promote optimal health benefits¹⁹⁰. The 2008 Physical Activity Guidelines for Americans¹⁹⁰ recommends adults participate in at least 150 minutes of moderate-intensity or 75 minutes per week of vigorous activity. This includes aerobic and muscle strengthening activity. Those living with chronic disease that follow physical activity guidelines can lower mortality by 46%, meanwhile adults without chronic disease following these guidelines can lower mortality by 27%¹⁹¹. The same guidelines pertain to those adults living with disability, including neurological disorders¹⁹⁰. Research has found exercise to help lessen the burden of mobility related issues of PD¹⁹²; those with PD who exercise have a better quality of life, walking ability, balance, strength, flexibility, and cardiovascular fitness levels¹⁹².

Despite these health benefits, some adults living with disability may not be able to meet these guidelines due to cognitive and physical impairment. For those living with disability, any degree of activity can provide health benefits and curtail functional decline¹⁹³. Physicians can modify physical activity recommendations for those unable to meet guidelines¹⁹³, but realities of these neurological disorders must be recognized to help this population overcome barriers of the disease that may deter participation in physical activity.

Physical and cognitive disabilities associated with neurological diseases can greatly impact physical function, which may decrease physical activity level and can also result in a decline in quality of life and depression^{3,4,39,194}. A decline in physical activity is a risk factor for decline in physical function and can further inhibit mobility¹²². Consequently, those living with PD, TBI or stroke are at risk for sedentary

behaviors and further induced functional decline, especially since these diseases can greatly impact movement of arms and legs¹⁸⁵.

Motor and non-motor motor symptoms associated with PD may impact physical activity and function. . A reduction in physical function can also reduce ADL such as housekeeping, preparing meals, and ability to travel in the community¹⁹⁵. Additionally, Wild and colleague's²⁷ dual task study, revealed people with PD had a shorter gait due to difficulty with performing a cognitive task in conjunction with a motor task. Additionally, those with PD that had postural instability and gait difficulty reported worse health-related quality³⁸. Changes highlight the impact both motor and non-motor symptoms have on PD, resulting in life-style changes.

Physical and cognitive changes experienced in this population may also increase risk for falling. Falls not only impact well-being, but can result in severe injuries¹⁹⁶. Factors that increase risk for falls include older age, body composition, number of chronic diseases, muscle strength, functional mobility, balance, and fear of falling¹⁹⁷⁻¹⁹⁹. Targeting these risk factors by promoting physical activity that targets muscle strength and balance may help to prevent falls in this population, as well as those with other neurological disorders.

Extensive research indicates those with PD and stroke are at increased risk for falls^{200,201}. Approximately 60% of those living with PD fall yearly²⁰². Predicting falls in those with PD are associated with history of previous falls, disease severity, poor balance, reduced mobility, freezing of gait and reduced leg strength^{203,204}. Similar physical limitation following a stroke also increase risk of falling²⁰⁵, and it is one of the most common complications following a stroke²⁰⁶.

Following a stroke, impaired dynamic balance is associated with frequent falls three weeks after stroke²⁰⁵. Those who experience falling after a stroke show lower cognitive and physical functions, compared to those who did not fall, which highlights falling may impact cognitive and physical function²⁰⁵. This loss of balance, combined with fear of falling, may promote a sedentary lifestyle and decline in physical activity level.

Additionally, immediately following a stroke, walking dysfunction is common, as well as fatigue and lack of energy⁵. To treat these symptoms rehabilitation is usually followed after a stroke. Research also reveals rehabilitation post stroke is a key time to assess cognitive function, and that stroke-related cognitive impairment can limit functional gains during stroke rehabilitation²⁰⁰. Also, despite rehabilitation physical limitations can persist or re-occur^{20,130}. Six months following a stroke, among 206 participants, 75% experience upper limb/motor impairment¹³⁰. Research has also found three to nine months following a year-long rehabilitation, those living with chronic stroke seem likely to redevelop symptoms of walking dysfunction²⁰⁷⁻²⁰⁹. Additionally, research has found gait seems to worsen in the later years post stroke²⁰, which can present challenges with balance, an increase in energy expenditure, risk of injury and decrease activity levels²⁰. These findings insinuate rehabilitation exercises beyond the rehabilitation phase may be necessary to promote and maintain optimal physical function in those living with chronic stroke.

The literature also implies those living with TBI may be at increase risk for falling^{3,7}. This can be due to changes in strength, endurance and balance²¹⁰, as well as diminished motor-planning and attention, pain and weight gain³. All of these factors

are inter-related and can promote poor physical function and low physical activity levels. While a decline in mobility is a part of the natural aging process, it seems to happen more rapidly in those living with TBI³.

Those who sustain a TBI are also more likely to experience muscular and orthopedic symptoms compared to nondisabled people⁸⁶. Change in mobility, pelvis, limb and vertebrae fractures and other physical traumas that occur at the time of injury can also have long-term consequence and can re-emerge overtime, thus decreasing physical function and activity level and promoting weight gain³. Pain and stiffness in the joints may also deter physical activity in this population³. If this population continues to be sedentary and obesity develops, pain can become more pronounced, as research has found an association between obesity and joint and muscle pain²¹¹. Since over-nutrition has also been found in those with PD and stroke these issues may also occur in these neurological disorders as well.

As previously described, those with PD, TBI or stroke can suffer from under- and over-nutrition. As a result, both physical inactivity and being obese are associated with decline in physical function and associated risk factors, such as the development of metabolic syndrome, CVD, and diabetes¹⁹³. Being underweight can result in muscle wasting and poor physical function¹⁸. As previously described those with disability that are underweight may have issues with eating and swallowing which can also promote decline in physical function¹²⁹.

Issues of physical function are prevalent in all three of these disease states and can further negatively impact health and quality of life. While, functional decline is a natural part of aging, it is more severe for those living with neurological conditions

and other chronic diseases¹⁹³. A recent cohort study examined factors associated with changes in physical function in older Chinese adults and found the main cause of functional decline was chronic disease; older persons with chronic conditions, such as stroke, PD, diabetes, and osteoporosis were more likely to experience a decline in physical function over the four year study period¹⁹³. This study highlights that while chronic diseases increase the likelihood of disability, information regarding effective management of these disabling conditions is limited^{193,212}. Research has also yet to examine the role of combining nutrition, cognition and physical activity in this population and how it can positively improve health outcomes.

Cognitive decline associated with these disease states can also impact physical activity level and physical functioning. Even mild levels of cognitive impairment have been found to increase disability and overall functional impairment¹⁰. Those with cognitive impairment may not be able to effectively perform meaningful tasks, which is hypothesized to be closely linked to inability to carry out ADL²¹³. Besides poor cognition, psychological issues associated with these neurological disorders, including fear of falling is recognized as a factor that may deter older adults susceptible to falls from being physically active¹⁹⁸.

While existing barriers can lead deter physical activity and promote decline in physical functioning, certain factors in this population have been found to help curtail this decline. A recent study examining factors that impact exercise behavior of 260 participants living with PD contradicts this²¹⁴. The study found that those who participated in exercise had less severe motor impairment, fewer depressive symptoms, and few issues with physical mobility. Through a multivariate logistic

regression, this cross sectional study found the degree of physical impairments, activity limitation and participation restrictions were not significantly associated with exercise behavior, but self-efficacy and belief regarding capabilities to exercise were²¹⁴.

Regardless of disease severity, those with higher self-efficacy were more than twice as likely to participate in routine exercise compared to those with low self-efficacy²¹⁴. A higher education level and older age also was associated with increased likelihood to partake in exercise²¹⁴. The majority of participants (86%) had mobility related issues that disabled them from participating ADL, but 88.8% of participants had a social support system at home to assist them²¹⁴. This highlights that the main source of physical activity for this population is from planned exercise and leisure activity, as opposed through independently carrying out ADL. Findings also imply that promoting an increase activity in this population may be most effective through encouraging exercise and leisure time activity.

A limitation of this study was that the majority of participants only had mild-to-moderate PD. Additionally, due to the cross-sectional study design, causality as to whether exercise promotes high self-efficacy or high self-efficacy promotes exercise needs to be monitored overtime²¹⁴. How the progression of the disease impacts self-efficacy, willingness to exercise overtime, and whether exercise overtime can help delay the progression of the disease must also be assessed²¹⁴.

Never the less, the findings of Ellis and colleagues²¹⁴, reveal an understanding of factors that promotes physical activity can help provide vital information on how to promote physical activity and provide clearer recommendations for those living with

neurological disorders. Physical inactivity can result in a further decline in physical function. Decline in physical function can promote muscle loss, as well as a decline in quality of life. While research has identified barriers that may promote a sedentary lifestyle, research has not examined the physical activity level of those living with one of these neurological conditions overtime. Doing so will help understand how and why ability to participate in exercise changes overtime. An assessment of physical activity levels and physical function of this population overtime can provide a better understanding of the diseases, and help create clearer recommendations and specialized programs for individuals throughout life. Doing so will not only decrease risk of developing other comorbidities associated with a sedentary lifestyle, but also help improve quality of life¹⁶⁰.

VII. Quality of Life

The World Health Organization (WHO) defines quality of life (QOL) as one's perception of their position in life in relation to the culture and value system in which they live²¹⁵. The long-term disabilities of these neurological diseases greatly impact QOL^{3,4,58}. Quality of life has been shown to be lower in those with PD compared to age-matched healthy controls as well as compared to those with other disease entities such as diabetes²¹⁶. A recent study, examined resilience, or one's ability to "bounce back" following an event or diagnosis, in those with PD. Results found that those with higher resilience scores, had better physical and mental quality of life and lesser disability²¹⁷. However, no correlation was found between disease severity and resilience scores²¹⁷. Depression and mild cognitive impairment in those with PD is also correlated with a poorer nutritional status, indicating a worse QOL^{1,36,68,69}. Those

living with TBI and stroke also experience mental and physical side effects, which can also experience changes in QOL⁷².

Those with TBI often experience side-effects that compromise well-being, such a bowel and bladder incontinence, sleep disorders, psychiatric diseases and sexual dysfunction²¹⁸. Additionally, Siponkoski and colleagues²¹⁹ examined the quality of life of those with TBI that received intensive multidisciplinary residential rehabilitation treatment for up to 15 years post injury, and found quality of life was related to age at time of injury, depression, anxiety, amount of assistance needed, and education level²¹⁹. Results also found that QOL remained stable over time and that those with milder injuries had a lower QOL²¹⁹. Traumatic brain injuries can impact long-term functional outcomes in older adults, however more information is needed to identify the specific, long-term post TBI side effects have on ADL, especially in older adults²⁸. Jacobsson and Lexell²²⁰ examined life satisfaction in those living with TBI six to fifteen years after injury and found those with a higher life satisfaction had fewer emotional disturbances and better social participation, as well as a longer time post injury and a more severe injury sustained later in life. It appears cohabiting and higher productivity levels also helps improve life satisfaction²²⁰. Findings indicate life satisfaction can improve with time and highlight factors that can impact life satisfaction. Both studies indicate more severe cognitive impairment seems to be associated with greater QOL and life satisfaction^{219,220}. The reasoning for this, as well as the impact disease severity has on other health parameters, needs to be further investigated.

Quality of life is also impacted in those with stroke, which can be exhibited by eating difficulties and inability to perform activities of daily living⁵. Research also consistently finds women seem to have less favorable outcomes after stroke than men, more difficulty with ADL, depression and overall lower QOL¹¹². This lower QOL in women could be due to worse mobility, pain/discomfort, and anxiety and depression observed up to 12 months after stroke²²¹. Research suggests that quality of life is often compromised in those living with PD, TBI and stroke, and as a result depression is a common side-effect of those living with one of these neurological conditions^{38,37,219}. Fatigue and sleep disturbances are common side effects of these three neurological conditions that can greatly impact quality of life⁸⁶. A decline in quality of life can negatively impact nutritional status, as well as physical activity and function.

VII. Conclusion

Parkinson's disease, TBI and stroke are three neurological diseases that impact cognitive and physical function as well as nutritional status and risk of CVD. Research suggests dietary intake and nutritional status can be compromised due to the complications of these neurological conditions^{3,16,65}. How nutritional status and physical activity level are impacted a year or more after diagnosis in those living with one of these diseases must be assessed to understand the difference between each disease and to acquire an understanding in treating those living long-term with PD, TBI or stroke. Assessing how nutritional status and physical activity level impact risk for cardiovascular disease, physical function and quality of life must also be assessed. Due to the prevalent research of weight loss and eating difficulties associated with one

of these neurological disorders, research also indicates those living with neurological conditions may be at risk for malnutrition^{1,13-16} a year or more after diagnosis. It is necessary to examine how disabilities increase rate of nutritional and cardiovascular risk. It is also necessary to examine how cognition, nutrition and physical functioning interact in those living with neurological disorders and impact quality of life.

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Appendix B: Extended Methodology

Subjects

Twelve participants were included in the study and consisted of eight males and four females between ages 33 and 71. At baseline, a total of 15 subjects were recruited. Participants were eligible if they were diagnosed with Parkinson's disease (PD), traumatic brain injury (TBI) or stroke for at least 12 months and were between the ages of 18 to 85. Participants were required to have a means of transportation to one of the URI Speech and Hearing Clinic locations.

This research was conducted as a sub-study of a longitudinal, observational study examining the cognition, nutritional status, physical activity level and physical functioning of those living with neurological disorders. Both received approval from URI's Institutional Review Board (IRB), Longitudinal Study of Communication, Nutrition and Physical Activity, IRB HU1314-006. Participants were recruited via announcements, brochures, and word-of-mouth. Brochures were provided to physician offices, hospitals, rehabilitation centers and health professionals to aid in recruitment as well as to those clients participating in URI's Department of Communicative Disorder's LOUD Crowd and Gateway Wellness Café. Study staff explained the research project to potential participants. If a potential participant was eligible and interested, they signed two copies of an informed consent. This sub-study only included baseline assessments, but participants consented to participate in a five-year longitudinal study with assessments every six months. Participants were then assigned identification numbers for data management that did not include any identifying numbers or letters.

The study utilized baseline data collected from November and December 2013. Written data was stored in a locked file cabinet in 203 Fernwood on the URI campus. Electronic data with identified information was stored on a computer in 203 Fernwood and password protected and encrypted. De-identified data sets (identifying information like name address, email and telephone numbers removed) were stored on password - protected computers in Ranger 301 (Lofgren's lab) and 214 Independence Square (Delmonico's lab).

Protocol Overview

Participants reported to a URI Speech and Hearing Clinic location for baseline assessments. Baseline assessments were conducted on two separate occasions. After signing informed consent, participants completed cognitive, nutrition, and physical function assessments. Participants fasted 12-hours prior to the assessment, but were encouraged to drink water during this fast. During the assessment, a 12-hour fasting blood sample was collected, as well as blood pressure. Nutritional status was examined using assessment tools that reflect dietary quality, anthropometrics, body composition and biochemical values.

All questionnaires were administered to participants by study staff. The following surveys related to cognitive-linguistic capacity, nutrition and physical functioning and physical activity were completed: The Repeatable Battery for the Assessment of Neuropsychological Status (RBANS)¹, the Swallowing Quality of Life Survey (SWAL-QOL)^{2,3}, the Dietary Screening Tool (DST)^{4,5}, and Physical Activity and Disability Survey (PADS)^{6,7}. Other questionnaires included: a medical history questionnaire, 14-item Resilience Questionnaire^{8,9}, and the Satisfaction with Life

Scale (SWLS)^{10,11}. To assess physical function the short physical performance battery (SPPB)¹² and the timed up and go (TUG)¹³ test was used. Assessments took approximately 3 ½ hours to complete per participant.

Anthropometrics

Trained study staffed obtained all measurements using standard procedures. Height (cm) and weight (kg) were measured using a stadiometer (Webb City, MO, USA). Height was rounded to the nearest 0.5 cm and weight was rounded to the closest 0.1 kg. Height and weight value was used to calculate BMI (kg of body weight/height in meters²). Body mass index was classified according to predetermined categories: underweight <18.5 kg/m², normal weight = 18.5-24.9 kg/m², overweight = 25-29.9 kg/m² and obese ≥ 30 kg/m²¹⁴.

A foot-to-foot bioelectrical impedance analysis device (Tanita BF-556, Arlington Heights, Illinois) was used to obtain body composition. This is a portable, battery-operated device that uses a very low electrical current (battery powered, 50 kHz) to estimate fat mass and percentage body fat. This test is a valid and reliable tool to measure body composition¹⁵. According to the American Council on Exercise¹⁶, any female with ≥32% body fat and any male with ≥25% body fat was considered obese. Even though risk for this test is minimal, participants with a pacemaker were not tested.

Biochemical Markers

Serum total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), triacylglycerol (TRG), and glucose were obtained using a finger stick (Cholestech[®] LDX system, Hayward California). Prior to

obtaining blood markers, participants were instructed to abstain from eating or drinking for 12 hours. However, unlimited water intake was permitted and encouraged during the 12 hour fast. Forty microliters of blood was drawn from the participant's finger by trained personnel using a lancet, collected into a capillary tube, put into a cartridge, and assayed using the portable Cholestech[®] (LDX system, Hayward California) machine. The study staff used universal precautions and standardized protocols. Participants with total serum cholesterol <200 mg/dL, LDL-C < 100 mg/dL, HDL-C > 40 mg/dL, and TRG < 150 mg/dL will have a serum lipid panel within normal limits¹⁷. A clinical diagnosis of high biochemical values include TC \geq 240 mg/dL, HDL-C <40 mg/dL, LDL-C \geq 160 mg/dL, and TRG \geq 200¹⁷. Fasting blood glucose <100mg/dL was considered normal, 100-125mg/dL impaired, and \geq 126mg/dL was considered high.

Blood Pressure

Trained study staff measured blood pressures¹⁸. Participants sat quietly for five minutes and the cuffed arm was supported at heart level. Blood pressure was measured using an automatic blood pressure machine (Omron Healthcare Inc., Bannockburin Illinois). Blood pressure was taken twice or until readings were within 2 mg/dl and the readings were averaged together. Systolic blood pressure <120mmHg and diastolic blood pressure < 80 mmHg were considered within normal limits. Systolic blood pressure > 130 mmHg and diastolic blood pressure > 85 mmHg were considered borderline high, while SBP >140mmHg and DBP >90 mmHg was considered hypertensive.

Questionnaires

Due to cognitive difficulties, the described questionnaires were chosen to decrease labor intensity for the population of interest. To reduce error and inaccuracy, short questionnaires validated in elderly populations, neurological conditions or physical disabilities were selected.

Cognitive-Linguistic

To measure attention, language, memory, constructional and visual-spatial abilities, the RBANS was used¹. It includes 12 subtests, with five index scores, summarizing: immediate memory, visuospatial/constructional, language, attention and delayed memory. The total score is the sum of all the index scores. Scoring is based on a mean of 100 and a standard deviation of 15, and a score <70 indicates suboptimal cognitive function. The test takes approximately 30 minutes to complete and is intended to assess adults ages 20-89.

Swallowing Evaluation

The SWAL-QOL is a 44-item survey where participants rate factors about 10 quality of life concepts related to swallowing^{2,3}. Specifically, participants were asked how swallowing problems affect their daily lives, especially in regards to eating and diet^{2,3}. The highest possible score is 220, the higher the score the better one's swallowing function. The SWAL-QOL took approximately 10 minutes to complete.

Dietary Quality & Nutrition Risk Assessment

Participants completed the DST. This 25-item questionnaire is used to identify dietary patterns and nutritional risk in older adults^{4,5}. Participants reported how frequently they consumed a food or type of food, as well as different dietary practices.

The sub-components and point distribution included: whole fruit and juices (15 points), vegetables (15 points), lean proteins (10 points), Added fats, sugars and sweets (25 points), dairy (10 points) and processed meats (10 points), and use of a dietary supplement (5 points).

Based on these responses a composite score was calculated and correlated with three different nutritional risk levels; (<60) at risk, (60-75) possible risk, and (>75) not at risk⁴. Categorization into three nutritional risk categories provides a way to determine if a patient needs a follow-up or nutrition assessment⁴. The DST Dietary intake was broken down based on point classification for the DST sub-components⁴ to explore specific areas contributing to low dietary quality. These sub-components were created to identify specific areas of the diet that may be problematic on an individual basis⁴. Cut points were set based upon if the participant had a score of approximately 50% or less of possible points for each sub-component. Participants with fewer than 50% of total possible points for each sub-component and if the participant did not currently take dietary supplement were considered low sub-scores. Less than 50% was selected as an exploratory marker to indicate infrequent/inadequate intake of whole grains, fruits, vegetables, dairy or lean proteins, and high consumption of processed meats, added sugars, fats and sweets.

This survey took approximately 10-15 minutes for the study staff to complete with each individual participant. While the DST has not been validated in those living with neurological disorders, it has been validated in older adults and is capable of characterizing the overall dietary patterns of the older adult population⁴.

Physical Activity Survey

The PADS was used to measure activity level. This survey is a validated and reliable source to measure physical activity in those who are sedentary and disabled^{6,19}. This tool covers a wide range of activities and includes six sub-scores: exercise, leisure-time physical activity (LTPA), general activity, therapy, employment, and wheelchair use. Scores increase as physical activity increases. The PADS is coded based on intensity, type and amount of activity, and can detect varying levels of physical activity¹⁹. Raw scores of 0-27 indicate minimal, 28-42 is minimal to moderate, 43-67 is moderate, and > 68 indicates an extreme level of activity⁶. This survey took about 20 minutes complete.

Physical Functioning Assessment

The SPPB¹² was completed to assess participants' global physical functioning and included timed measures of usual gait speed, standing balance tests, and repeated five chair stands. For the balance tests participants were asked to maintain their feet in side-by-side, semitandem, and tandem stances for 10 seconds each. To assess walking speed participants walked four meters at their usual pace. Walking speed was performed twice, and the faster time was recorded. During the chair test participants stood up from sitting position with their arms folded across their chest. If the participant were able to perform this task, they were instructed to stand up and sit down five times as quickly as possible. The time to perform this task was recorded. SPPB scores are reported out of 12, and each sub score ranges from zero to four, and the higher the score the better lower physical function. Scores 8-12 are considered "high functioning"¹².

A timed up and go (TUG) test was used to assess basic functional mobility¹³. It measures the time it takes for the participant to raise from a chair, walk three meters, turn around and walk back to the chair. The shorter amount of time, the better the score. Time was recorded in seconds, the test was administered twice and the better score was recorded. Those with TUG scores >13.5, are considered high risk for falls²⁰

Other Questionnaires

Past Medical History Questionnaire

To identify health-related conditions and how these conditions may impact health, a non-validated medical history questionnaire will be used. Topics covered will include neurological, endocrine and cardiovascular health. Ten minutes were allotted to recite and complete this survey for each participant.

Resilience

The 14-item Resilience Scale^{8,9} measured resiliency. Resiliency is one's ability to recover from a physiological or psychological event^{8,9}. It is a validated and reliable tool that has found a significant correlation between resilience, morale, depression and overall life satisfaction in older adults⁹. Questions are answered on a Likert scale from one to seven, where participants reflect on their belief that their life has meaning, sense of uniqueness, and ability to exist alone and manage their lives; higher scores are associated with greater degrees of resiliency^{8,9}. It took approximately five minutes to complete. Total possible scores range from 14 to 98, and a higher score indicates higher levels of resilience.

Life Satisfaction

The SWLS, a five-item questionnaire, was used to obtain overall life satisfaction¹⁰. It is used to measure global life satisfaction by asking participants to make a broad judgment of their life¹⁰. This questionnaire is answered on a Likert scale that ranges from one to seven^{10,11}. Total possible score ranges from five to 35, and the higher the score, the greater life satisfaction.

Cardio-Metabolic Assessment

Cardio risk factors were selected, and grouped into eight categories based upon modifiable and non-modifiable risk factors. These eight selected categories included: 1.) age (>45 years for men, >55 years for women), 2.) anthropometrics (BMI, %BF) 3.) clinical (SBP and DBP), 4.) biochemical (TC, HDL-C, TC/HDL-C ratio, LDL-C, TRG and Glucose), 5.) diet (DST scores), 6.) physical barriers (use of legs, arm or assistive device), activity (PADS scores) and functioning (SPPB and TUG scores), 7.) smoking status, and 8.) cognition (RBANS scores). Those living with neurological disorders suffer physical and cognitive impairment that may disrupt activities of daily living (ADL); as a result physical barriers (such as partial or no use of arms and legs, as well as use of an assistive device) were examined due to the possibility they may promote a sedentary lifestyle. Cognitive function was also included as a CVD risk factor through examining total RBANS scores and three RBANS index scores (immediate memory, attention, delayed memory) <70.

Statistical Analysis

This is the first analysis of this study, and therefore statistical analysis were not hypothesis driven, but conducted to generate hypothesis and explore the population.

Collected data was analyzed using SPSS, version 22.0 for Windows (IBM Corp. Summers, NY). Descriptive statistics were conducted and distribution of data was assessed through analyzing skewness and kurtosis. For data that was not normally distributed, proper statistical methods were used to account for this. Categorical variables are represented as numbers and percentages and continuous variables are reported as mean \pm standard deviation.

To analyze CVD risk factors, the participants were individually analyzed across the eight categories. The most frequent risk factors were then analyzed based on the variables within the eight CVD categories and reported as top CVD risk factor.

For all normally distributed data, independent t-tests were conducted to determine if there were significant differences between groups regarding assessment variables. Cohen's *d* was calculated to describe effect size for differences between groups in normally distributed variables (0.2 = small effect, 0.5 = medium effect, 0.8 = large effect). For data not normally distributed, Mann-Whitney U tests were used to analyze differences between groups and effect size was signified through calculating *r* (0.1 = small effect, 0.3 = medium effect, 0.5 = large effect). Between group differences for categorical variables were identified using Fisher Exact Tests. Phi coefficient or Cramer's *V* were used to signify effect size (0.1 = small effect, 0.3 = medium effect, 0.5 = large effect). Pearson's *R* Correlations were used for normally distributed variables, and Spearman Rho Correlations were used as the non-parametric alternative. Significance for all analyses was set at $p < 0.05$.

Power Calculation

An A-priori power analysis found that a sample of 28 PD participants and 14 ABI participants would find significant difference between groups and SPPB scores. To find significant differences between groups and DST scores, an A-priori power analysis suggests 63 PD participants and 31 ABI participants are needed.

Resources Required

Funding for this research was provided by URI's Department of Communicative Disorders. Supplies provided by the Department of Nutrition and Food Sciences included: computers, printers, computer software, paper and physical assessment equipment. Personnel to conduct the study were supplied by the Departments of Communicative Disorders, Kinesiology, and Nutrition and Food Sciences. No other university resources will be required for this study.

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Appendix C: Consent Form for Research

Longitudinal Study of Communication, Nutrition and Physical Activity

Leslie A. Mahler, PhD, Principal Investigator
Ingrid Lofgren, PhD, co-Investigator
Matthew Delmonico, PhD, co-Investigator

CONSENT FORM FOR RESEARCH: Participant
Version 2: July 30, 2013

The University of Rhode Island
Department of Communicative Disorders
25 W Independence Square, Suite I
Kingston, RI 02881

Purpose of the Consent:

You have been invited to take part in a research project described below. The purpose of the consent form you are about to read is to provide you with details about the research study and to inform you of your rights if you agree to participate in the study. Your participation is completely up to you. The researcher will explain the project to you in detail. You should feel free to ask questions. If you have more questions later you can call, Dr. Leslie Mahler, the person mainly responsible for this study, at 401-874-2490. You may also contact Dr. Ingrid Lofgren at 401-874-5706 or Dr. Matthew Delmonico at 401-874-5440, who are co-Investigators on the study. You must be at least 18 years old, speak English, and have neurological diagnosis of traumatic brain injury, stroke, or Parkinson disease to be in this research project.

Description of the project:

This is a research project designed to look at communication, nutrition, and physical activity characteristics of adults who have a stroke, traumatic brain injury or Parkinson disease. All evaluations will be conducted at one of two University of Rhode Island locations; in Independence Square on the Kingston Campus at 25 West Independence Way, Kingston or in Independence Square at 500 Prospect Street in Pawtucket.

You are being asked to be in this study because we want to determine the long-term impact of neurological disorders on communication, nutrition, and physical activity. We are looking for 200 people who have a stroke, traumatic brain injury or Parkinson disease to participate in this project. Participation in this study is entirely your choice.

If you decide to take part in this study, you should understand that the evaluations are investigational and you may not experience any benefit from participation. Participation may also involve additional risks as listed in the Potential Risks and Discomforts section. The consent form will help make sure you understand the tasks

included in the study before you decide whether you want to take part in the study. You may also quit the study at any time.

What will be done:

If you agree to take part in this study, you will be asked to complete up to 11 evaluations over five years. Evaluations will take place every six months. The evaluations will include a variety of tasks such as reading sentences and describing a picture, an assessment of how your muscles move, a cognitive screening, an interview, a clinical swallowing evaluation, and questionnaires regarding swallowing, diet and physical activity. The total time for each evaluation will be approximately 3½ hours. All evaluations will be conducted in a quiet private room at one of the University of Rhode Island Speech and Hearing Clinic locations (Kingston or Pawtucket).

With your permission, we will request health information from your physician about the following specific items only:

- Date of diagnosis
- Current medications
- Imaging information about where the brain damage is located (if appropriate)

You will sign a separate form to indicate whether you give your permission to release this health information for the study.

Potential risks and discomforts:

There are minimal foreseeable risks associated with these evaluations. There have been no reported adverse affects from clinical evaluation of speech and swallowing. There may be some unknown or unanticipated risks, but every precaution will be taken to ensure your personal safety. Even though experienced personnel will obtain the blood samples from a finger prick, there is a chance of discomfort and minor bruising from the finger stick. For physical function testing there is a risk of muscle soreness or other muscle injury as well as skeletal injury but we will minimize these risks by using standard safety practices.

Purpose and benefits of the study:

The purpose of this study is to describe communication, nutrition, and physical activity behaviors over time to see how they change and affect quality of life. The information obtained is important because it will help us to understand how to provide services to meet the needs of people with neurological diagnoses. This is an investigational study and there is no guaranteed benefit to your communication or nutrition or physical function as a result of participation in this research study. You will receive personal health information such as your height and weight, physical function determined by a physical assessment, your blood lipids such as cholesterol and triglycerides. In addition, you will receive information about your thinking skills and language skills and dietary choices.

Drugs, devices or instruments to be used:

Drugs will not be used in this study. The equipment for the evaluations include: microphone, sound level meter, tongue blade, a digital tuner, tape recorder, and video cameras. All equipment used to collect cognitive-linguistic and physical function data is considered non-invasive. A lancet and capillary tube will be used to obtain the blood sample from a finger prick and the sample will be analyzed on a small portable machine that is on a table.

Cost to participant:

There is no cost to you for participation in the evaluations. Parking is available for free.

Confidentiality:

Your part in this study is confidential. Your individual privacy will be maintained in all published and written data resulting from this study. No names of participants will be published or included in written data resulting from this study. Results of this study may be used for purposes of research, educational lectures, and/or professional presentations. When you are entered into the study you will be assigned a code that does not include any identifying information. For example, the first participant will be coded as Long01. The code number will be used on all response forms and in the analysis of the data.

Dr. Mahler and her research team will have sole access to all contact information and evaluation results containing your name. This information will be kept in a locked filing cabinet in a locked office. However, the U.S. Department of Health and Human Services, and the University of Rhode Island Institutional Review Board have the right to inspect all of your records relating to this research for the purpose of verifying data. Because of the need to release information to these parties, absolute confidentiality cannot be guaranteed. Following completion of this project, contact information will be destroyed for those participants who wish, for any reason, not to be contacted in the future. All other information will be archived and kept in a locked filing cabinet with the study results at the University of Rhode Island. All research data will be retained for a minimum of three years following completion of the study and then will be destroyed. Research data will be located in a locked filing cabinet in the principal investigator's locked office.

Cognitive-linguistic evaluations will be audio and video recorded to allow for data analyses. At times these recordings can be useful for teaching students or professionals about the disorders of people with a neurological diagnosis such as yours. Please indicate by signing below whether you give your permission to use your samples for lectures and presentations. Audio and/or videotapes may be used for teaching for up to 3 years after completion of the study. If you agree, you will never be identified by name in the presentations or lectures. Your decision to give permission to use audio and/or video samples in lectures has no impact on your participation in the study.

_____ Yes, I give permission to use audio samples in lectures and presentations.

_____ Yes, I give permission to use video samples in lectures and presentations.

_____ No, I do not want audio samples used except for research analysis.

_____ No, I do not want video samples used except for research analysis.

In case there is any injury to you during the study:

If this study causes you any injury, you should immediately contact Dr. Leslie Mahler at (401) 874-2490 or contact the University of Rhode Island Speech and Hearing Clinic at (401) 874-5969. You may also call the office of the Vice President for Research Integrity, 70 Lower College Road, University of Rhode Island, Kingston, RI at (401) 874-4328. If you are injured during an evaluation or during treatment every effort will be made to get you medical attention but you will be responsible for paying for the medical treatment needed.

Decision to quit at any time:

The decision to take part in this study is up to you. You do not have to participate. If you decide to take part in the study, you may quit and stop participating in this study at any time. You have the right to refuse to answer any question(s) or participate in any procedure for any reason. Deciding not to participate will have no effect on your potential to receive services from a speech-language pathologist. If you wish to quit, simply inform Leslie Mahler at 874-2490 of your decision. If you wish to pursue an alternative treatment instead of completing the study you will be provided with information on how to obtain those services.

Rights and complaints:

If you are not satisfied with the way this study is performed, you may discuss your complaints with Dr. Leslie Mahler (lmahler@uri.edu; 401-874-2490), Dr. Ingrid Lofgren (ingridlofgren@uri.edu, 401-874-5706), or Dr. Matthew Delmonico (delmonico@uri.edu; 401-874-5440), or you may contact the office of the Vice President for Research for concerns or any questions about your rights as a research subject at: 70 Lower College Road, University of Rhode Island, Kingston, RI at (401) 874-4328 and speak to them anonymously if you choose.

Authorization:

Your authorization means that you have read this paper and know the purpose of the study and the possible risks and benefits. It also means you know that being in this study is voluntary and you choose to be in this study. You can also withdraw at any time. Your questions have been answered. Your signature on this form means that you understand the information and you agree to participate in this study.

Signature of Participant

Signature of Researcher

Participant Typed/printed Name

Researcher Typed/printed name

Date

Date

Signature of Guardian

Signature of Researcher

Guardian Typed/printed Name

Researcher Typed/printed name

Date

Date

Please sign both consent forms, keeping one for yourself.

Appendix D: Assessment Materials

INTERVIEW
Longitudinal Study of Communication, Nutrition and Physical Activity

Leslie Mahler, PhD, CCC-SLP, Principal Investigator

Ingrid Lofgren, PhD, co-Investigator

Matthew Delmonico, PhD, co-Investigator

Version 2: 2-17-14

Participant Name: _____ **Initials:** _____ **ID#:** _____

Name of Interviewer: _____ **Date:** _____

Emergency contact name and address & phone:

DIRECTIONS: Read the following questions out loud to each prospective volunteer and record the answers. Any answers that require clarification should be written in the space below the question or on the back of the sheet. Indicate whether any follow-up is necessary or if any referrals are appropriate.

What is your neurological diagnosis? _____

When were you diagnosed? _____

What were your symptoms at that time?

What are your symptoms now?

What is your communication like?

What is your diet like?

What is your physical activity like?

Does your speech sound clear to other people?

If not, how does it sound?

Can you think of the words that you want to say?

If you do have trouble, how often does it happen?

Are you experiencing any symptoms of a swallowing disorder? _____

If yes, what is the problem with your swallowing?

What would you say is your most significant problem with speech or swallowing today? _____

Did you experience any changes in your speech or swallowing *before* your diagnosis?

Does medication affect your speech or voice or swallowing? _____

If yes, in what way? _____

Speech

How many hours of speaking do you do in a day? _____

What is a typical day of communicating like for you?

Do you pronounce your words clearly?

Do people ask you to repeat yourself?

Do people have a hard time understanding you?

What do you do when you want to be as easy to understand as possible?

What percent of your speech do you think is understandable?

Has your neurological diagnosis caused you to talk less?

If so, how much less? _____ Why? _____

Swallowing

Do you have any difficulty with swallowing?

Do you cough during mealtimes? _____

If yes, do you cough more with water or solid food? _____

Do you have difficulty making the food go down (need to swallow twice)?

Does it take you longer to finish a meal than before your neurological diagnosis?

Have you experienced any unintentional recent weight loss? _____

Have you ever been diagnosed with pneumonia? _____

If yes, when? _____

Have you changed your diet since your neurological

diagnosis? _____

If yes, what did you modify? _____

Musculoskeletal system:

Has your doctor ever told you that you have: (circle all that apply)

- Osteoarthritis or degenerative arthritis
- Rheumatoid arthritis
- Osteoporosis
- Ankylosing Spondylitis
- Unknown or other type of arthritis
- Any other disease of joint or muscle:
- Comments: _____

Cardiovascular system:

- Has any family member had a heart attack prior to the age of 55?
 - If so, how are they related to you? _____
- Have you ever had frequent cramping in your legs while resting?
 - If yes, is it a current problem? _____
- Have you ever had pain or cramping in your legs while walking?
 - If yes, is it a current problem? _____
- If yes, is this pain relieved by rest or by discontinuing walking? _____
- Have you ever been told that you have high blood pressure
 - If yes, what was the date of onset? _____
 - Were you given any medications? _____
- Did a doctor ever tell you that you had a heart problem?
 - If yes: What was the date of onset? _____
- What did the doctor call it? _____
 - Were you given any medications? _____
- Do you have any history of high cholesterol in your blood as evidenced by a previous blood lipid tests?

Comments: _____

Endocrine system:

Have you ever had any of the following: Thyroid problems, adrenal problems, diabetes mellitus?

• _____

- If yes to diabetes, which type? Type 1 or Type 2

Date of onset- _____

Are/were you on any medication, or is it diet controlled?

Neurological system:

- Do you have any significant problems with your memory? (circle all that apply)
 - When answering the telephone, do you recall what you were doing before it rang?
 - Can you give the directions to your house/apartment?
 - Can you keep appointments without a reminder?
 - Can you remember what clothes you wore yesterday?
- Any problems with vision other than corrective lens changes?
 - If yes, which of the following conditions- Blindness, temporary loss of vision, double vision, glaucoma, cataract, macular degeneration or others.

Do you have and of the following?: (circle all that apply)

- Vertigo (a feeling of spinning, or unsteadiness)
- Seizure or convulsions?
- Migraine or severe headaches?
- Paralysis of arm or leg?
- A head injury with loss of consciousness?
- Pain, numbness or tingling in your limbs?
- Pain in your lower back?

- Do you have pain in any part of body including headaches while exercising?
- Have you been told that you have a peripheral neuropathy?
- Tremors?
- Problems with walking? If yes,
 - Do you fall frequently?
 - Is your walking problem related to pain, weakness or loss of balance?
- Have you ever had an operation on skull or brain?
- Have you ever had meningitis or Brain fever?

Comments: _____

Previous Treatment

Have you had previous speech or swallow treatment, occupational therapy or physical therapy? _____

If yes, please describe (when, what)

Was it beneficial?

If yes, what changes did you notice?

Employment

Are you employed?

Type of employment

How much speaking do you do at your job?

Smoking Status

Do you currently smoke cigarettes? _____

Do you have a history of smoking cigarettes? _____

If you have quit smoking cigarettes, how many months/years ago did you quit?

Other

Have you noticed any difficulty with your memory?

Have you experienced any changes in your mood?

Is it difficult for you to pay attention long enough to finish a task?

Do you have any difficulty reading? _____

Do you have any difficulty writing? _____

Do you have any other health problems or conditions that would affect communication, nutrition or physical activity?

Are you Hispanic or Latino? _____

What race do you identify with? (Check all that apply) _____ I prefer not to provide this information

White/Caucasian _____ African American _____ Asian _____ American

Indian/Alaskan Native _____ Native Hawaiian/Pacific Islander _____

Other _____

Dietary Screening Tool

DIRECTIONS: Please check one response to each question that best describes how you eat.

How often do you usually eat fruit as a snack?

- Never
- Less than once a week
- 1 or 2 times a week
- 3 or more times a week

How often do you usually eat whole grain breads?

- Never **or** less than once a week
- 1 or 2 times a week
- 3 or more times a week

How often do you usually eat whole grain cereals?

- Never **or** less than once a week
- 1 or 2 times a week
- 3 or more times a week

How often do you usually eat candy or chocolate?

- Never
- Less than once a week
- 1 or 2 times a week
- 3 or more times a week

How often do you eat crackers, pretzels, chips, or popcorn?

- Never
- Less than once a week
- 1 or 2 times a week
- 3 or more times a week

How often do you eat cakes or pies?

- Never
- Less than once a week
- 1 or 2 times a week
- 3 or more times a week

How often do you eat cookies?

- Never
- Less than once a week

- 1 or 2 times a week
- 3 or more times a week

How often do you eat ice cream?

- Never
- Less than once a week
- 1 or 2 times a week
- 3 or more times a week

How often do you eat cold cuts, hot dogs, lunchmeats or deli meats?

- Never **or** less than once a week
- 1 or 2 times a week
- 3 or more times a week

How often do you eat bacon or sausage?

- Never **or** less than once a week
- 1 or 2 times a week
- 3 or more times a week

How often do you eat carrots, sweet potatoes, broccoli, or spinach?

- Never
- Less than once a week
- 1 or 2 times a week
- 3 or more times a week

How often do you eat fruit (not including juice)? Please include fresh, canned or frozen fruit.

- Never **or** Less than once a week
- 1 or 2 times a week
- 3 to 5 times a week
- Every day or almost every day

How often do you eat hot or cold breakfast cereal?

- Never
- Less than once a week

- 1 or 2 times a week
- 3 to 5 times a week
- Every day or almost every day

How often do you drink some kind of juice at breakfast?

- Never **or** Less than once a week
- 1 or 2 times a week
- 3 to 5 times a week
- Every day or almost every day

How often do you eat chicken or turkey?

- Never **or** less than once a week
- 1 or 2 times a week
- More than 3 times a week

How often do you drink a glass of milk?

- Never **or** Less than once a week
- 1 or 2 times a week
- 3 to 5 times a week
- Every day or almost every day
- More than once every day

Do you usually add butter or margarine to foods like bread, rolls, or biscuits?

- Yes
- No

Do you usually add fat (butter, margarine or oil) to potatoes and other vegetables?

- Yes
- No

Do you use gravy (when available) at meals?

- Yes
- No

Do you usually add sugar or honey to sweeten your coffee or tea?

- Yes
- No

Do you usually drink wine, beer or other alcoholic beverages?

- Yes
- No

How often do you eat fish or seafood that IS NOT fried?

- Never
- Less than once a week
- Once a week
- More than once a week

How many servings of milk, cheese, or yogurt do you usually have each DAY?

- None
- One
- Two or more

How many different vegetable servings do you usually have at your main meal of the day?

- None
- One
- Two
- Three or more

Which of the following best describes your nutritional supplement use.

- I don't use supplements
- I use supplements other than vitamins and mineral
- I use a multivitamin/mineral preparation (e.g. Centrum)

NCPAD Physical Activity and Disability Scale

DEMOGRAPHICS

NAME: _____ DATE: _____

AGE: _____ years

GENDER

- Male
 Female

TYPE OF DISABILITY _____

ASSISTIVE DEVICES (Check all that apply)

- Walker
 Braces
 Cane
 Wheelchair

USE OF ARMS (Check one)

- Full
 Partial
 No Use

USE OF LEGS

- Full
 Partial
 No Use

Directions: On the following pages are a list of questions related to physical activity and exercise. There are no right or wrong answers and your responses will be kept anonymous. Note that your answers to certain questions in the survey may cause your browser to skip other questions and move to a later item in the survey. Don't worry--this is how the survey was designed in order to save time. Please answer each presented question as accurately and as completely as possible. When you have finished the survey, press the "Submit" button. Your survey responses will be checked and used to create scores reflecting your level of physical activity. A window presenting your scores will then appear.

1. EXERCISE

1.00 Do you currently exercise?

- Yes
- No

↳ IF NO, PLEASE GO TO THE LEISURE ACTIVITY SECTION.

1.01 What kind of exercise do you do?

Directions: List up to four (4) activities below that you do on a regular basis for primary purpose of increasing or maintaining fitness. Aerobics are done for a sustained period of time and result in an increase in your heart rate and breathing rate. Examples include walking, jogging, attending an aerobics class, and bicycling. Strength activities include lifting weights or using elastic bands or weight training machines. Flexibility refers to activities that involve muscle stretching

Activity Type

- | | |
|--------------|----------------------|
| <u>Code:</u> | <u>Description</u> |
| A: | Aerobic Exercise |
| S: | Strength Exercise |
| F: | Flexibility Exercise |

Activity Type (check one)	Activity	Days/Week	Minutes/Day	Months/Year
<input type="checkbox"/> A <input type="checkbox"/> S <input type="checkbox"/> F				
<input type="checkbox"/> A <input type="checkbox"/> S <input type="checkbox"/> F				
<input type="checkbox"/> A <input type="checkbox"/> S <input type="checkbox"/> F				
<input type="checkbox"/> A <input type="checkbox"/> S <input type="checkbox"/> F				

1.02 Have you been exercising for more than one year or less than one year?

- More than one year
- Less than one year

1.03 How would you describe the average intensity of your exercise program?

- Light exercise: Don't sweat or breathe heavily
- Moderate exercise: Breathe a little harder and may sweat
- Vigorous: Breathe hard and sweat

2. LEISURE ACTIVITY

2.0 Do you engage in leisure time physical activity?

- Yes
 No



IF NO, GO TO THE GENERAL ACTIVITY SECTION ON THE NEXT PAGE.

2.1 What type of activities do you do?

Directions: List up to four (4) activities below that you do for leisure or recreation. These activities can be done on a regular or irregular basis and may not necessarily result in sustained increases in heart rate and breathing rate. Examples include hiking, boating, skiing, dancing and sports activities. Please indicate whether the activity is an endurance activity or a Non-Endurance activity. Examples of endurance activities include: hiking, tennis, dancing, skiing. Non-endurance activities include boating, softball and horseback riding. Do not list activities here that you already listed under exercise.

Activity Type

Code	Description
E	Endurance
NE	Non-Endurance

Activity Type (check one)	Activity	Days/Week	Minutes/Day	Months/Year
<input type="checkbox"/> E <input type="checkbox"/> NE				
<input type="checkbox"/> E <input type="checkbox"/> NE				
<input type="checkbox"/> E <input type="checkbox"/> NE				
<input type="checkbox"/> E <input type="checkbox"/> NE				

3. GENERAL ACTIVITY

3.00 From Monday through Friday, how many waking hours a day do you usually spend inside your home?

- Less than 6 hours a day
- 6 to 10 hours a day
- More than 10 hours a day

3.01 On Saturday and Sunday, how many waking hours a day do you usually spend inside your home?

- Less than 6 hours a day
- 6 to 10 hours a day
- More than 10 hours a day

3.02 On average, how many hours a day do you sleep including naps?

_____ hours

3.03 On average, how many hours a day are you sitting or lying down, excluding sleeping?

_____ hours

3.04 Are most of your indoor household activities done by you or someone Else?

- Done by you
- Done by someone else

↳ IF DONE BY SOMEONE ELSE, GO TO QUESTION 3.06.

3.05 Please list up to four (4) indoor household activities you do and the number of minutes a week you spend on each activity.

Activity	Minutes/Week

3.06 Do you do any outdoor household activities such as gardening?

- Yes
- No



IF NO, GO TO QUESTION 3.08.

3.07 Please list up to four (4) outdoor household activities you do and the number of minutes a week you spend on each activity.

Activity	Days/Week	Minutes/Day	Months/Year

3.08 How much assistance do you need to perform activities of daily living such as dressing and bathing?

- No assistance
- Some assistance
- Full assistance

4. THERAPY

4.00 Do you currently receive physical or occupational therapy?

Yes

No



IF NO, GO TO EMPLOYMENT SECTION ON THE NEXT PAGE.

4.01 How many days a week do you receive therapy?

_____ Days.

4.02 How long does each therapy session usually last?

_____ Minutes

6. WHEELCHAIR USERS

6.00 Do you use a wheelchair?

- Yes
- No



IF NO, STOP HERE.

6.01 How many years have you used a wheelchair? _____ years?

6.02 During the time that you are awake, how much time do you spend in your wheelchair?

- All day
- Most of the day
- A few hours

6.03 What type of wheelchair do you primarily use?

- Manual wheelchair
- Powered wheelchair



IF POWERED WHEELCHAIR, STOP HERE.

6.04 Who usually pushes your wheelchair?

- Myself
- Someone else



IF SOMEONE ELSE, STOP HERE.

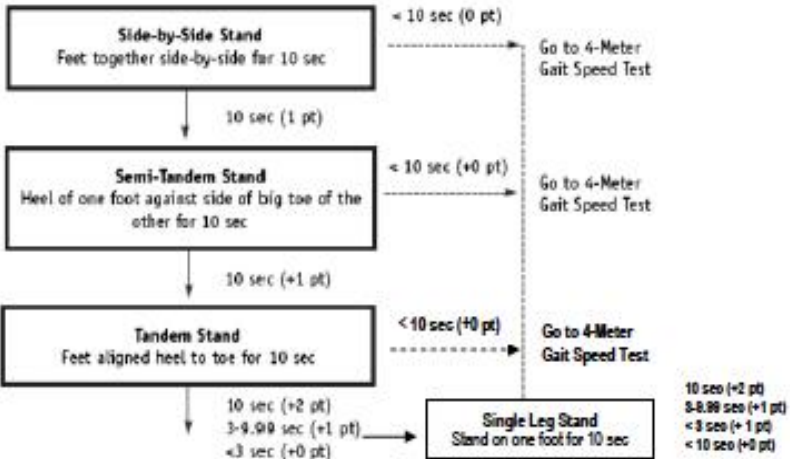
6.05 On average, how many minutes a day do you push yourself in your wheelchair?

- Less than 60 minutes
- Sixty minutes or more

Thank you for completing this survey!

1.

Balance Tests



2.

Gait Speed Test

Measures the time required to walk 4 meters at a normal pace (use best of 2 times)



< 4.82 sec	4 pt
4.82-6.20 sec	3 pt
6.21-8.70 sec	2 pt
> 8.7 sec	1 pt
Unable	0 pt

3.

Chair Stand Test

Pre-test
Participants fold their arms across their chest and try to stand up once from a chair

unable → Stop (0 pt)



5 repeats
Measures the time required to perform five rises from a chair to an upright position as fast as possible without the use of the arms



≤ 11.19 sec	4 pt
11.20-13.60 sec	3 pt
13.70-16.60 sec	2 pt
≥ 16.7 sec	1 pt
> 60 sec or unable	0 pt

Go to 4-Meter

Gait Speed Test