2016

Nutritional Status, Cognition and Cardiometabolic Risk in Individuals with Neurological Disorders

Chelsea Paulin

University of Rhode Island, chelsea_warrell@uri.edu

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NUTRITIONAL STATUS, COGNITION, AND CARDIOMETABOLIC RISK IN
INDIVIDUALS WITH NEUROLOGICAL DISORDERS

BY

CHELSEA PAULIN

A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF THE

REQUIREMENTS FOR THE DEGREE OF

MASTER OF SCIENCE

IN

NUTRITION AND FOOD SCIENCES

UNIVERSITY OF RHODE ISLAND

2016

MASTER OF SCIENCE

OF

CHELSEA PAULIN

APPROVED:

Thesis Committee:

Major Professor

Ingrid Lofgren

Geoffrey Greene

Leslie Mahler

Furong Xu

Nassar H. Zawia

DEAN OF THE GRADUATE SCHOOL

UNIVERSITY OF RHODE ISLAND

2016

ABSTRACT

Background: Limited research has been done to monitor the progression of health outcomes in individuals with Parkinson's disease (PD) and acquired brain injury (ABI).

Objective/Hypothesis: The purpose of this longitudinal study was to assess the natural progression of the disease process and its effects on nutritional status, cognition, and cardiometabolic risk over a two year time period in those living with PD and ABI ≥ 12 months post-diagnosis.

Methods: Thirteen community-dwelling adults (9 with PD and 4 with ABI) were evaluated for nutritional status using the dietary screening tool (DST), cognition using the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS), and cardiometabolic risk factors using anthropometric, biochemical, and clinical assessments. Three evaluations separated by 6-12 month increments for each participant took place. Changes in nutritional status, cognition, and cardiometabolic risk were assessed using RMANCOVA ($p < 0.05$).

Results: Of the 9 participants with PD, 6 (66.7%) did not change nutritional risk categories over time; 2 worsened and 1 improved. Of the 4 participants with ABI, 2 (50%) did not change nutritional risk categories over time; 1 improved and 1 worsened. Time appeared to have a negative effect on cognition for participants with PD and a positive effect on cognition for participants with ABI. Six PD and 2 ABI participants had no change or fewer cardiometabolic risk factors over time, while 1 PD and 1 ABI had more cardiometabolic risk factors over time. Findings however were not statistically significant.

Conclusion: Our data reinforces the notion of outcome heterogeneity in individuals with PD and ABI. Since nutritional status, cognition, and cardiometabolic risk factors are unique to each patient, it is of clinical importance to provide evaluation and treatment on an individual basis. More consistent, long-term evaluations are needed to detect disease progression trends and determine what risk factors occur when to guide intervention development.

ACKNOWLEDGMENTS

I would like to formally recognize several people who have assisted and inspired me in writing my thesis. First and foremost, I would like to thank my major advisor, Dr. Ingrid Lofgren, who I have had the pleasure of working under for a total of five years during my undergraduate and graduate career, for her dedication, mentorship, and encouragement throughout my studies. I would not be where I am today without you. I would also like to thank my committee members, Dr. Leslie Mahler, and Dr. Geoffrey Greene, for their guidance, support, and wisdom.

Thank you to my fellow graduate students and dietetic interns for all of their help and support. Specific thank you to Dara for providing me with guidance throughout my journey. I would also like to thank Nicole, Leah, and Julie for all your continuous help and hard work. As well as a very special thank you to the faculty and students in Communicative Disorders and Kinesiology for helping to make this research project possible. Thank you to the clients of LOUD Crowd and Gateway Café to help me deepen my understanding of my research topic.

Lastly, I would like to thank my family and friends who have supported me every day as I pursued my degree. I proudly dedicate this thesis to my two biggest fans, my mother, Elizabeth Willan, and grandmother Edith Kooker.

PREFACE

This thesis was written to comply with the University of Rhode Island graduate school Manuscript Thesis Format. This thesis contains one manuscript: *Nutritional Status, Cognition, and Cardiometabolic Risk in Individuals with Neurological Disorders*. This manuscript has been written in a form suitable for publication in *Disability and Health Journal*.

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CHAPTER 1

Nutritional Status, Cognition, and Cardiometabolic Risk Factors in Individuals with Neurological Disorders

^bChelsea Paulin, ^bIngrid E Lofgren, ^cFurong Xu, ^cMatthew J Delmonico,

^aLeslie Mahler

^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 Disability and Health Journal

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INTRODUCTION

Parkinson's disease (PD), traumatic brain injury (TBI), and stroke are unique neurological diagnoses that share common symptoms and risk factors for decline in the areas of nutritional status, cognitive function, and cardiometabolic risk.¹⁻⁵

Parkinson's disease is a neurodegenerative movement disorder that worsens over time.⁶ Traumatic brain injury (TBI) and stroke are two types of acquired brain injury (ABI) that cause damage to the brain.⁷ These conditions may decrease life expectancy by as much as 33 years compared to healthy populations,⁸⁻¹⁰ and individuals with these diagnoses experience declines that can negatively impact nutritional status, cognitive function, and cardiometabolic risk earlier in life.^{2, 7, 9-14}

While health status at the time of diagnosis or immediately after diagnosis has been studied, limited research has been done to monitor the progression of nutritional status, cognition, and cardiometabolic risk factors in individuals living with PD or ABI.¹⁻³ Earlier and faster deteriorations in these health outcomes can lead to chronic diseases such as cardiovascular disease (CVD) and can negatively influence quality of life sooner.¹⁵ Consistent, regular monitoring of these outcomes in those with PD and ABI is critical to determine how these populations differ from the general population. Furthermore, knowledge of how and when these specific neurological disorders impact these outcomes will allow for earlier and more targeted interventions to attenuate disease evolution. The purpose of this longitudinal study is to assess the natural evolution of the disease process on nutritional status, cognition, and cardiometabolic risk factors over a time frame of 12-18 months in those living with PD and ABI ≥ 12 months post-diagnosis.

MATERIALS AND METHODS

Design

This study assessing nutritional status, cognitive function, and cardiometabolic risk factors in those with PD or ABI is an ancillary portion of a five-year longitudinal, observational study. Evaluations are administered every six months and use a variety of metrics to assess participants' characteristics. Study approval was obtained from the University of Rhode Island's Institutional Review Board (IRB HU#1314-006). This ancillary study will expand upon the previous cross-sectional assessment performed in this population by LoBuono et al.¹¹

Participants, setting, and recruitment

Participants (18-85 years of age) diagnosed with PD or ABI for at least 12 months and determined to be medically stable by a neurologist were recruited on a rolling basis via brochures and word-of-mouth. Before entering into the study, all participants completed the informed consent process. Participant evaluations occurred once every six months. Specifically, this report will focus on participants who have completed their first time point (T1) and two additional follow-up visits (T2 and T3) in 12-18 months following T1.

Assessment visit and data collection

At each assessment, participants completed a medical history questionnaire and a multidisciplinary assessment in the areas of nutrition, cognition, and cardiometabolic risk factors.

Procedures/Measures

Nutritional Status- Nutritional status was assessed using the Dietary Screening Tool (DST).^{16, 17} The DST identifies dietary patterns and characterizes three levels of nutrition risk based on dietary quality using a 25-item questionnaire. A total of 100 points can be achieved with cutoff scores for nutrition risk as follows: at risk (<60), possible risk (60-75), and not at risk (>75).^{16, 17}

Cognition- Cognition was assessed using the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS).¹⁸ The RBANS is a measurement of neuropsychological status comprised of 12 subtests that measure 5 cognitive domains: 1. immediate memory, 2. visuospatial/constructional, 3. language, 4. attention, and 5. delayed memory. Each of the five index scores are added together for a total score. Scores are scale-corrected based on normative information by age and education level and have a mean of 100 ± 15 , with lower scores indicative of greater cognitive impairment.¹⁹

Cardiometabolic Risk- Cardiometabolic risk factors assessed in this study were the anthropometric, biochemical, and clinical modifiable risk factors identified by the American Diabetes Association and the American College of Cardiology Foundation in the concept of global cardiometabolic risk.²⁰⁻²³ These factors are: 1. overweight or obesity, 2. hypertension, 3. dyslipidemia, and 4. hyperglycemia. Height (cm), measured using a stadiometer (Deteco, Webb City Missouri), and weight (kg), measured using a scale (Tanita BF-556, Arlington Heights, Illinois), were used to calculate body mass index (BMI) ($\text{kg}/\text{height in meters}^2$) and determine weight status. Blood pressure was measured using an automatic blood pressure machine (Omron

Healthcare Inc., Bannockburn Illinois). Following a 12-hour fast, plasma total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), triglycerides (TAG), and glucose were analyzed using the portable Cholestech[®] machine (Cholestech[®] LDX system, Hayward California), while LDL-C was calculated using the Friedewald formula.²⁴⁻²⁶ Participants were considered overweight if BMI $\geq 25\text{kg/m}^2$ and obese if BMI $\geq 30\text{kg/m}^2$. A systolic blood pressure (SBP) $>140\text{mm Hg}$ and diastolic blood pressure (DBP) $>90\text{mm Hg}$ was considered hypertensive. Abnormal biochemical lab values were as follows: TC $>200\text{mg/dL}$, HDL-C $<40\text{mg/dL}$, LDL-C $>100\text{mg/dL}$, TAG $>150\text{mg/dL}$, and glucose $>100\text{mg/dL}$.

Statistical Analysis

Data were analyzed using SPSS, version 23.0 (IBM Corp. Somers, NY). Descriptive statistics were performed and normality assessed using the Shapiro-Wilk test. A log-transformation of HDL-C was used to analyze the data. Age was not transformed and thus non-parametric tests were used. Between group differences at T1 were determined via independent sample t-tests, Mann-Whitney U tests, and Fisher's exact test. The primary aim of assessing nutritional status over time was tested using a repeated measures analysis of covariance (RMANCOVA) looking at the dependent variable of nutritional status as measured by DST score. Analysis of covariance (ANCOVA) was used to assess the relationship at T1 of nutritional status on cardiometabolic risk factors and cognitive function over time using with DST risk category as the independent variable. Cognitive function as RBANS score and the number of cardiometabolic risk factors served as the dependent variables. Covariates included were length of time since diagnosis and age, except for RBANS scores which

are already age adjusted. Neither time since diagnosis nor age were found to be significant covariates in explaining any of the key dependent measures and thus were not used for analysis in results presented here. For analyses in violation of Mauchly's test of sphericity, results were interpreted using the Greenhouse-Geisser correction. Analyses were considered significant when $p < 0.05$.

RESULTS

A total of 16 participants consented to participate in the study. Two passed away after their first assessments and one was unable to attend a third assessment for personal reasons. Thus, 13 participants who completed three assessments separated by 12-18 months were included in data analyses. Participant characteristics are in Tables 1 and 2. Most were male (n=10, 76.9%) and were diagnosed with PD (n=9, 69.2%). Eight participants were taking medication to manage blood pressure and/or lipids, including one participant taking diabetes medication.

Participants with PD were significantly older than participants with ABI (67.7 ± 3.9 vs. 49.0 ± 16.2 , $p=0.02$). Participants with PD scored significantly higher scores on the RBANS indexes of language (97.2 ± 8.5 vs. 80.0 ± 2.3 , $p=0.002$) and attention (93.6 ± 10.7 vs. 56.0 ± 15.9 , $p=0.001$) as well as total RBANS score (85.6 ± 13.7 vs. 66.5 ± 13.2 , $p=0.04$) at T1 than participants with ABI.

Nutritional Status

There were no significant differences in nutritional risk within or between groups over time as measured by the DST (Figure 1). Furthermore, no significant relationship was found between nutritional status and any of the cardiometabolic risk factors or RBANS indexes over time. At T1, 11 (84.6%) participants were “at nutrition risk” or “at possible nutritional risk” (8 PD and 3 ABI).

Eight participants did not change nutritional risk categories over time. Six of the 8 were participants with PD; 2 stayed “at nutritional risk,” 3 stayed “at possible nutritional risk,” and 1 stayed “not at nutritional risk.” The remaining 2 of the 8 were participants with ABI; both stayed “at possible nutritional risk.”

Five participants changed nutritional risk categories over time. Three of the 8 were participants with PD; 2 worsened from “at possible nutritional risk” to “at nutritional risk”, while 1 improved from “at nutritional risk” to “at possible nutritional risk.” One participant with ABI worsened from “not at nutritional risk” to “at possible nutritional risk” and 1 participant with ABI improved from “at possible nutritional risk” to “not at nutritional risk”.

Cognition

There were no significant differences within or between groups over time in any index score or total RBANS score over time.

Although not significant, time appeared to effect measures of cognition for all participants. There was a different trajectory between groups, with a decrease in all RBANS scores for participants with PD and an increase in all RBANS scores for participants with ABI except in the index of attention (Tables 1 and 2).

Cardiometabolic Risk Factors

Total cholesterol, HDL-C, LDL-C, TAG, DBP and glucose concentrations did not significantly differ between or within groups over time. However, there was a significant effect for time on SBP in ABI participants ($p=0.01$) (Table 2). Weight significantly decreased over time in participants with PD ($p=0.001$) and BMI trended toward significance ($p=0.06$).

All participants had at least one cardiometabolic risk factor at T1 and T2 (Table 3). The most prevalent risk factor at T1 was BMI ≥ 25 kg/m² (7 PD and 2 ABI). Seven (53.8%) participants remained overweight or obese at all time points (5 PD and 2 ABI). Six PD participants had no change or fewer risk factors over time, while 1

increased over time. Two PD participants had elevated LDL-C at all time points; 2 remained with elevated SBP, 2 maintained elevated glucose, and 3 maintained low HDL-C. Two ABI participants had no change or fewer risk factors over time, while 1 increased over time. One ABI participant had elevated LDL-C at all time points. Two ABI participants maintained elevated TC, and 2 maintained elevated BMI over all time points.

DISCUSSION

To our knowledge, this study is the first to explore the evolution of nutritional status, cognition, and cardiometabolic risk over time in individuals with PD and ABI. This study showed that there is significant variability within groups of the same disease state and demonstrates the clinical importance of considering patients on an individual basis.

Routine assessments for individuals with PD and ABI remains imperative. Our results showed that a majority of participants remained at poor nutritional status or declined over time. Individuals may be at nutritional risk due to similar deleterious sequelae that can occur across all three disease states including problems with chewing, swallowing, and motor skills.²⁷⁻²⁹ While those with PD may be at increased nutritional risk due to a tendency to crave sweet foods or carbohydrates,³⁰ research on dietary habits of individuals post TBI and stroke are lacking. Current findings establish the importance of using a dietary screening tool such as the DST in these populations. Dietary screening can be an effective strategy for early detection of those with compromised dietary intake to reduce nutritional risk and the burden of neurological disorders.¹⁶ However, validation of the DST is needed in these populations. Consistent with previous literature demonstrating PD patients continuously lose weight despite consuming a diet higher in energy content and lower in diet quality,³¹ our results showed that individuals with PD experienced weight loss while remaining “at” or “possibly at” nutrition risk. This weight loss exemplifies one of the many unexplained burdens of chronic neurological disorders, and may be attributed to the progressive nature of the disease. Additional research is warranted to

determine whether nutrition education can prevent weight loss to maintain or improve quality of life. While previous research on individuals post ABI have shown weight loss and poor nutritional status after the initial injury, data on long term weight and nutritional status has been limited.^{27, 32} The preliminary results of our longitudinal assessments with a small sample size indicate that individuals with ABI appear to maintain weight and nutritional status. The small sample of participants with TBI and stroke in the current study prevented a comparison between types of ABI.

Participants with ABI consistently scored the lowest on the attention index of the RBANS; a deficit consistent with previous research findings.^{33, 34} These deficits result in an inability to focus and self-regulate, which may negatively impact nutritional status by hindering meal planning, food shopping, and meal completion. While participants with PD in this study did better than those with ABI in all of the RBANS indexes, deficits were most prominent in the area of immediate memory. It appears that this area of cognition might be more vulnerable in the PD population.³⁵ Deficits in immediate memory may negatively impact nutritional status by interfering with meal preparation, as well as shopping and eating.³⁶ The non-significant trajectory shown in this study of cognitive decline in PD is consistent with previous literature associating declines with the duration of the disease.² Results showing a non-significant trajectory toward an increase in cognitive status in ABI are novel, but may be related to the average time since diagnosis of over 17 years. It is possible that individuals with ABI may be more likely to seek activities to improve cognitive decline than individuals with PD whose cognitive decline occurs gradually. Continuous evaluation of this trend will occur during the remainder of this five-year

study. Previous research in individuals post stroke found stable cognition two years post injury³ while significant variability in outcomes has been seen up to 5 years post TBI.³³ Large effect sizes suggest that future studies with greater power may find clinically significant improvements over time in cognition in individuals with ABI diagnosed ≥ 10 years prior.

Previous research has shown hypertension to be one of the most frequent cardiometabolic risk factors in participants with PD.⁵ However, our most prominent cardiometabolic risk factor was elevated BMI. This may be due to the use of medications to manage blood pressure and lipids by a majority of participants (61.5%). Two participants with PD maintained impaired fasting glucose across all three time points, one with diagnosed diabetes. While diabetes has been associated with chronic neurodegeneration and it is hypothesized that chronic hyperglycemia may exacerbate the severity of the motor disability^{37, 38}, evidence associating diabetes and PD is inconclusive. Our results lend support to recommendations for individuals with PD to receive routine glucose monitoring.³⁵ Additionally, our results are in agreement with previous studies demonstrating that cardiometabolic risk factors are often suboptimally managed after TBI and stroke, as two of the participants with ABI maintained hypercholesterolemia over all three time points.^{39, 40} More consistent assessments and active involvement by a multidisciplinary team of medical personnel in the management of these modifiable risk factors is necessary to help mitigate disease progression and the risk of recurrent stroke.^{39, 40}

Although it appeared that there were differences over time in total cholesterol and between group differences in LDL-C, HDL-C and RBANS scores, our sample

size may have been too small to detect differences. Studies with larger samples may wish to explore these variables further.

Study strengths and limitations

This is the first study to report on multiple assessments of the effects of the evolution of the disease process in the areas of nutrition, cognition, and cardiometabolic risk factors in those living with PD or ABI. Despite the important results this study presents, several limitations must be taken into consideration. Due to our small sample size, generalizability to larger populations is limited. However, looking at data individually is of clinical importance; since nutritional status, cognition, and cardiometabolic risk factors are unique to each patient, it is necessary to provide evaluation and treatment on an individual basis. Furthermore, this longitudinal study was non-experimental and therefore causation of results cannot be determined. Nonetheless, our longitudinal design was consistent with the goals of this research to observe disease evolution and individualize goals of treatment in the respective populations. Individuals in this study were all community-dwelling, and not representative of the PD or ABI populations as a whole. Future studies should include a larger sample size for a deeper understanding of the relationship of the progression of the disease process and its relationship to nutrition, cognition, and cardiometabolic risk factors. Additional longitudinal assessments with a longer period of follow up are warranted for a deeper understanding of the changes that occur in the disease process over time. Comparing participants with PD or ABI to age-matched healthy controls in future studies would help to see how participants with neurological disorders differ than the general population.

CONCLUSION

Our data reinforces the notion of outcome heterogeneity in individuals with PD and ABI. Nutritional status, cognition, and cardiometabolic risk may change over time in individuals with PD and ABI. Consistent assessments of these health outcomes should be conducted regularly to identify and/or prevent the development of later-onset complications. Identification of which health outcomes are most prominent and who is at greatest risk is necessary to interrupt the disease process as early as possible after diagnosis.⁴¹

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TABLES AND FIGURES

Table 1. PD Participant Characteristics and Within Group Changes Over Time

Time point	PD (n=9)			P-value	Effect Size
	1	2	3		
Age (years)	67.7±3.9*				
Time since Diagnosis (years)	7.1±7.2				
Gender %(n)					
Men	88.9(8)				
Women	11.1(1)				
Height (cm)	170.7±6.0	169.7±6.6	169.9±6.6	0.27	0.150
Weight (kg)	79.2±12.7 ^a	77.9±12.6 ^{ab}	75.7±13.7 ^b	0.001	0.562
BMI (kg/m ²)	27.1±3.2	26.9±3.1	26.2±3.8	0.06	0.298
BMI Categories %(n)					
Normal	33.3(3)	33.3(3)	44.4(4)		
Overweight	55.6(4)	44.4(4)	44.4(4)		
Obese	11.1(1)	22.2(2)	11.1(1)		
DST Total	61.2±12.8	65.6±11.3	58.8±14.1	0.07	0.287
DST Categories %(n)					
At Nutritional Risk	33.3(3)	23.1(3)	44.4(4)		
Possible Nutritional Risk	55.6(4)	44.4(4)	44.4(4)		
Not at Nutritional Risk	11.1(1)	22.2(2)	11.1(1)		
Total cholesterol (mmol/L)	9.1±1.9 [†]	8.5±1.5	8.3±1.2	0.23	0.192
HDL-C (mmol/L) [#]	2.2±0.7 [†]	2.1±0.6	2.1±0.5	0.88	0.018
LDL-C (mmol/L)	5.4±1.6 [†]	5.2±1.3	4.8±1.5	0.31	0.155
Triglycerides (mmol/L)	7.4±3.7 [†]	5.6±1.7	6.9±2.5	0.28	0.165
Glucose (mmol/L)	5.6±1.0 [‡]	5.2±0.9	5.5±1.0	0.24	0.218
Systolic BP (mm Hg)	139.6±22.9	132.3±14.6	144.7±17.4	0.29	0.143
Diastolic BP (mm Hg)	82.0±11.1	79.7±6.2	81.4±13.4	0.88	0.016
RBANS Total	85.6±13.7*	82.8±16.3	80.0±15.1	0.16	0.208
Immediate Memory	85.4±14.6	76.8±17.2	79.4±19.0	0.30	0.141
Visuospatial/ Constructional	86.8±15.1	84.4±24.3	79.6±17.9	0.42	0.102
Language	97.2±8.5**	93.6±8.6	91.1±10.5	0.21	0.179
Attention	93.6±10.7***	88.1±19.6	88.7±15.1	0.33	0.128
Delayed Memory	84.8±22.7	90.4±15.7	82.7±22.1	0.09	0.298

Notes: values expressed as mean± standard deviation. Differences between groups for all normally distributed variables analyzed using independent t-tests. *Difference between PD vs. ABI p < .05; **Difference between PD vs. ABI p < .005; ***Difference between PD vs. ABI p < .0005; [†]n=8; [‡]n=7; [§]n=3; [#]Log-transformed for analysis

Abbreviations: BMI, body mass index; DST, dietary screening tool; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; BP, blood pressure; RBANS, Repeatable Battery for the Assessment of Neuropsychological Status.; BMI: Normal = 18.5-24.9 kg/m², Overweight = 25.0-29.9 kg/m², Obese = ≥30.0 kg/m²; DST: At Nutritional Risk = <60, Possible Nutritional Risk = 60-75, Not at Nutritional Risk = >75

Table 2. ABI Participant Characteristics and Within Group Changes Over Time

Time point	ABI (n=4)			P-value	Effect Size
	1	2	3		
Age (years)	49.0±16.1*				
Time since Diagnosis (years)	17.0±8.7				
Gender %(n)					
Men	50.0(2)				
Women	50.0(2)				
Height (cm)	169.4±14.1	169.0±14.5	169.4±14.7	0.73	0.101
Weight (kg)	75.7±18.7	75.1±18.0	73.8±18.1	0.11	0.528
BMI (kg/m ²)	26.0±2.3	25.2±1.6	25.3±2.2	0.65	0.133
BMI Categories %(n)					
Normal	25.0(1)	25.0(1)	50.0(2)		
Overweight	75.0(3)	75.0(3)	50.0(2)		
Obese	0.0(0)	0.0(0)	0.0(0)		
DST Total	70.8±7.0	66.8±7.9	70.3±6.0	0.40	0.640
DST Categories %(n)					
At Nutritional Risk	0.0(0)	0.00(0)	0.00(0)		
Possible Nutritional Risk	75.0(3)	75.0(3)	75.0(3)		
Not at Nutritional Risk	25.0(1)	25.0(1)	25.0(1)		
Total cholesterol (mmol/L)	11.5±2.3	10.6±1.9	10.8±2.0§	0.33	0.420
HDL-C (mmol/L) [#]	3.3±1.9	3.4±1.7	4.4±1.4§	0.49	0.520
LDL-C (mmol/L)	5.6±2.4	5.6±1.9	5.6±2.5§	0.83	0.073
Triglycerides (mmol/L)	7.1±4.1	7.7±3.9	6.7±4.6§	0.09	0.700
Glucose (mmol/L)	4.5±1.0	5.0±0.5	4.8±0.7§	0.44	0.338
Systolic BP (mm Hg)	124.0±8.8	115.1±4.3	122.8±8.3	0.01	0.757
Diastolic BP (mm Hg)	76.5±7.2	73.6±7.3	73.3±6.3	0.11	0.526
RBANS Total	66.5±13.2*	67.0±16.6	72.5±16.0	0.15	0.472
Immediate Memory	68.8±22.6	66.3±18.2	74.5±24.2	0.33	0.307
Visuospatial/ Constructional	78.0±10.7	80.3±24.3	89.3±22.6	0.17	0.450
Language	80.0±2.3**	70.5±13.8	83.5±4.4	0.64	0.139
Attention	56.0±15.9*	56.8±10.4	53.3±11.3	0.09	0.546
Delayed Memory	79.0±24.6	81.5±26.3	87.0±29.3	0.11	0.526

Notes: values expressed as mean± standard deviation. Differences between groups for all normally distributed variables analyzed using independent t-tests.*Difference between PD vs. ABI p < .05; **Difference between PD vs. ABI p < .005; ***Difference between PD vs. ABI p < .0005; †n=8; ‡n=7; §n=3; #Log-transformed for analysis

Abbreviations: BMI, body mass index; DST, dietary screening tool; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; BP, blood pressure; RBANS, Repeatable Battery for the Assessment of Neuropsychological Status.; BMI: Normal = 18.5-24.9 kg/m², Overweight = 25.0-29.9 kg/m², Obese = ≥30.0 kg/m²; DST: At Nutritional Risk = <60, Possible Nutritional Risk = 60-75, Not at Nutritional Risk = >75

Table 3. Cardiometabolic Risk Factors by Participant Over Time

Time point	BMI			SBP			DBP			TC			HDL-C			LDL-C			TAG			GLU		
	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3	1	2	3
Participant																								
1 ^{a,b}	X	X																						
2	X	X	X							X	X	X				X	X	X		X				X
3	X	X	X										X	X			X		X	X				
4										X	X	X				X	X							
5													X	X								X	X	X
6 ^{a,b}	X	X		X	X	X		X					X	X					X					
7 ^b	X	X	X	X	X																			
8 ^{a,d}	X	X	X	X	X								X	X	X		X		X					X
9 ^{b,d}																	X		X					X
10 ^a	X	X	X	X	X	X				X			X	X	X	X	X	X						
11				X	X	X		X					X	X	X	X	X				X			X
12 ^b	X	X	X										X											
13 ^{b,c}	X	X	X	X	X	X	X	X					X	X	X							X	X	X
Total # RF	9	9	7	5	4	6	3	0	2	3	2	2	6	7	6	5	5	5	4	2	1	3	3	5

^a PPT taking blood pressure medication ^b PPT taking lipid lowering medication ^c PPT taking diabetes medication ^d PPT is a current smoker

BMI, body mass index; SBP, systolic blood pressure, DBP, diastolic blood pressure, TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TAG, triglycerides; RF, risk factors

Note: Participants 1,2,3,4 diagnosed with ABL, remaining are diagnosed with PD

Figure 1. DST Score by Participant Over Time

Note: Participants 1,2,3,4 diagnosed with ABI, remaining are diagnosed with PD

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APPENDIX A: REVIEW OF LITERATURE

I. Introduction

Parkinson's disease (PD) and acquired brain injury (ABI) are unique neurological diagnoses that impact the brain and share common sequelae that affect nutritional status, cognition, and cardiometabolic risk.¹⁻⁵ Parkinson's disease is a neurodegenerative movement disorder.⁶ Traumatic brain injury (TBI) and stroke are two types of ABI that cause temporary or permanent damage to the brain.⁷ Currently, more than 2.5 million individuals have one of these neurological disorders.⁸⁻¹⁰ These conditions may decrease life expectancy by as much as 33 years compared to healthy populations,¹¹⁻¹³ and declines in functionality and health outcomes can reduce quality of life.¹⁴⁻¹⁶ These declines negatively impact nutritional status, cognition, and cardiometabolic risk earlier in life than that of the general population.^{7, 17}

Parkinson's disease is currently the 14th leading cause of death for Americans and the number of diagnosed individuals is predicted to more than double in the next 25 years.^{18, 19} Unintentional injuries such as TBI are currently the 4th leading cause of death for Americans with TBI-related emergency department visits increasing 70% in the past decade.^{10, 18, 19} Part of the increase in TBI prevalence may be due to the recent wars involving the United States (US) with Iraq and Afghanistan.²⁰ Stroke rates have doubled in the past five years, making it the 5th leading cause of death for Americans.^{19, 21} These neurological disorders contribute to the significant cost of prescriptions, health care, and lost productivity.^{3, 14, 22} The total current annual economic impact of PD, TBI, and stroke in the US is \$121.3 billion^{9, 14, 23} In the

future, these costs are expected to rise with the increase in incidence and prevalence of these neurological conditions.

Routine, consistent monitoring of long-term nutritional status, cognition, and cardiometabolic risk factors in those with PD and ABI is lacking but critical to determine how these populations differ from those without these neurological conditions.^{2, 3, 5} Assessing the natural evolution of the disease process can indicate how nutritional status, cognition, and cardiometabolic risk change over time. Earlier and faster deteriorations in these areas can lead to chronic diseases such as cardiovascular disease (CVD) and can negatively influence quality of life sooner.¹⁵ Knowledge of how and when these specific neurological disorders impact these health outcomes will allow for earlier and more targeted interventions to attenuate disease progression. With such little research being performed in these populations and the variability in individual outcomes, more knowledge on individual data and the evolution of the diseases will allow for detection of disease trends, and promote earlier and more targeted interventions to attenuate disease progression.

II. Neurological Disorders

a. Parkinson's Disease: Definition and Description

Parkinson's disease is a neurodegenerative movement disorder.⁶ Up to 60,000 new cases of PD are diagnosed each year in the US, adding to the 1 million people that were already living with the disease in 2014.⁸ While the exact etiology of PD remains unknown, the disease routinely presents itself in adults over the age of 60, with only 4% of the cases occurring in those under the age of 50.²⁴ Males have a significantly higher incidence rate of PD; 1.5 times greater than that of females ($p=0.031$).²⁵ The

pathological trademark of PD is a lack of dopamine-producing neurons in the brain.²⁶ Since dopamine helps regulate body movement, lacking this critical neurotransmitter can cause uncontrollable tremor, postural imbalance, slowness of movement and rigidity, all of which are characteristic motor symptoms of PD.²⁶ Life expectancy of those living with PD can be the same as the general population,²⁷ and the disease can continue for upwards of 20 years or more once motor features manifest.²⁸ Parkinson's disease is chronic and progressive, and although not considered fatal, there is no current cure or treatment that successfully reverses the effects of the disease.⁸ While symptoms of PD gradually result in disability and impairment, a variety of tools have been created to help delay and prevent deterioration.

Two rating scales are predominately used to characterize the disability and impairment level of individuals with PD. The Hoehn and Yahr scale focuses primarily on motor symptoms and is based on the level of clinical disability ranking stages 1 through 5.^{8, 29} Stage 1 of the Hoehn and Yahr scale involves minimal or no functional impairment, while stage 5 involves severe disease progression with confinement to bed or wheelchair unless aided.²⁹ A more recent and comprehensive scale called the Movement Disorder Society-United Parkinson's Disease Rating Scale (MDS-UPDRS) is comprised of four sections that focus on both motor and non-motor symptoms; I: non-motor experiences of daily living; II: motor experiences of daily living; III: motor examination; and IV: motor complications.³⁰ Each section is based on a five-point range of impairment and disability, where 0 = normal, and 4 = severe.³⁰ Non-motor symptoms of PD include cognitive impairment, neuropsychological problems, sleep disturbances, sensory complaints, urinary and gastrointestinal problems, symptomatic

orthostasis, and fatigue.³⁰ Both assessment scales are used routinely to delay or prevent disease progression in individuals with PD by assessing for symptoms that would suggest signs of decline. Disease advancement as measured on these scales can impact nutritional status, cognition, and cardiometabolic risk factors.

b. Traumatic Brain Injury: Definition and Description

A TBI is a type of ABI that is a major cause of disability in the US.¹⁰ Although it is considered an acute injury, individuals can face a variety of chronic complications and life-long challenges in recovery.³¹ Traumatic brain injury occurs as a result of an external mechanical force such as a violent blow or jolt to the head or body which causes impairment of brain function.³² There are approximately 1.7 million new cases annually.¹⁰ Children aged 0 to 4 years, older adolescents aged 15 to 19 years, and adults aged 65 years and older are most likely to sustain a TBI.¹⁰ Rates of TBI are higher for males than for females, with approximately 1.4 times as many TBIs occurring among males than females.¹⁰ Falls are the leading cause of TBI, followed by motor-vehicle traffic injuries and striking injuries.¹⁰ Approximately half of individuals with a severe TBI will need surgery.³³

Symptoms and sequelae resulting from TBI are dependent on multiple variables including the timing of medical attention, severity and location of the injury, age, and general health of the individual at the time of injury.³³ There is no current treatment to reverse the initial brain damage.³¹ Deficits resulting from a TBI are typically seen in cognition, emotional functioning, behavior, communication, sensory functioning, and mental health.^{31, 33} Traumatic brain injury can also lead to increased risk for other health conditions, such as depression, Alzheimer's disease, and stroke.³⁴⁻

³⁶ Depending on age of injury onset, it is estimated that the average length of survival for an individual after experiencing a TBI is 50 years.³⁷ Life expectancy is reduced by approximately four years.¹³ Despite the fact that much has been learned about the effects of TBI on the brain in recent years, there has been no significant improvement in treatment due to the heterogeneity of the injuries and difficulty in identifying which individual are most likely to benefit from different forms of treatment.³⁸

Severity of TBI is typically measured using the Glasgow Coma Scale (GCS), in which injuries are classified as mild, moderate, or severe according to initial level of consciousness after injury.³⁹ Mild TBI may cause temporary dysfunction of brain cells, while severe TBI can result in permanent damage to the brain that can cause long-term complications or death.³² In an article reviewing the epidemiology and impact of TBI, Langlois, Rutland-Brown, and Wald⁴⁰ issue a call to action to further quantify the increased risk of health problems, both short- and long-term after TBI. Specifically, the areas of nutritional status, cognition, and cardiometabolic risk factors requires additional investigation.

c. Stroke: Definition and Description

A stroke is a type of ABI that occurs when blood circulation to the brain fails, resulting in decreased blood and oxygen flow, and ultimately cell death.⁴¹ While an estimated 6.6 million Americans ≥ 20 years of age have already had a stroke, 795,000 new strokes occur each year,⁹ with the expectation of the number of new strokes to double over the next 40 years.⁹ Although stroke can occur at any age, prevalence is higher in older adults, with the risk of stroke doubling each decade after the age of 55.⁴¹ African Americans have nearly twice the risk as Caucasians of having a first

stroke.⁴¹ Additional non-modifiable risk factors for stroke include family history and gender.^{9, 41} Annually, approximately 55,000 more females than males have a stroke.⁹ Modifiable risk factors include cigarette smoking, physical inactivity, poor nutrition, heart disease, high blood pressure (BP), diabetes, dyslipidemia, lower levels of education, and living in the southeastern US.⁹

Strokes are categorized by etiology and severity. Ischemic stroke accounts for approximately 80% of all strokes and are caused by a blockage of blood flow in the brain or neck.⁴¹ Hemorrhagic strokes account for the other 20% of strokes that are caused by bleeding into the brain.⁴¹ Severity of a stroke is typically measured using the National Institute of Health Stroke Scale (NIHSS), which provides a quantitative measure of stroke-related neurologic deficit.^{42, 43} The NIHSS is comprised of the following items; consciousness, language, neglect, visual-field loss, extraocular movement, motor strength, ataxia, dysarthria, and sensory loss. Each item score ranges from 0-4, with higher scores indicative of higher impairment. The item scores are summed for a patient's total NIHSS score.^{42, 43}

Similar to TBI, symptoms and sequelae resulting from stroke are dependent on variables such as the timing of medical attention after the injury, severity and location of the stroke, age, and the general health of the individual at the time of the stroke.⁴⁴ Some individuals are able to make a full recovery after stroke, while others may live with lifelong consequences.⁴¹ After stroke, deficits are typically seen in cognitive, physical, and psychological functioning.^{4, 5, 45} Stroke can also lead to increased risk for other health conditions, such as CVD, recurrent stroke, depression, dementia, and Alzheimer's disease.⁴⁵⁻⁴⁷ There is no current treatment to reverse the initial brain

damage done in stroke, and it is estimated that individuals with stroke live 33% fewer remaining years when compared to matched controls.¹¹ This reduced life expectancy increases the importance of improving quality of life to make the remaining years of life meaningful. Health outcomes of importance include nutritional status, cognition, and cardiometabolic risk factors, with additional research needed to determine how these areas are affected post-stroke.

While outcomes and life expectancy can vary between PD, TBI, and stroke, it is important to look at the three conditions together because they share common symptoms and sequelae impacting nutritional status, cognition, and cardiometabolic risk factors.^{7, 14, 48} For example, similar impacts on nutritional status can be the result of problems with chewing, swallowing, and motor skills that occur across all three disease states.⁴⁹⁻⁵¹ In addition, cognitive deficits in the areas of attention and memory^{12, 17, 52, 53} and cardiometabolic risk factors such as hypertension^{1, 36, 48, 54, 55} have been identified in all individuals post-diagnosis. A thorough understanding of the disease evolution on these health outcomes has yet to be established. Assessing and monitoring nutritional status, cognition, and cardiometabolic risk factors in these individuals can allow for the delineation of a timeline of decline and allow for comparisons to be made within groups. Routine monitoring of these health outcomes will greatly influence life post-diagnosis and allow for earlier and more targeted interventions to be developed to attenuate disease progression.

III. Nutritional Status in Individuals with Neurological Disorders

a. Defining Nutritional Status

“Nutritional status” implies measurement of the responses to nutrients, or lack of nutrients, by an individual, a group, or a community.⁵⁶ Nutritional status is often determined by screenings that identify individuals requiring a more thorough nutrition assessment due to identified possible nutrition risk which can then result in developing an intervention plan.⁵⁷ There is currently no consensus on a definition of nutritional status or evaluation instrument for individuals with neurological disorders. A review of previously used tools to measure nutrition status in individuals with neurological disorders is presented in Table 1.

Table 1: Comparison of Dietary Screening Tools

	<i>Mini-Nutritional Assessment (MNA)</i>	<i>Mini-Nutritional Assessment Short-Form (MNA-SF)</i>	<i>Dietary Screening Tool (DST)</i>	<i>Subjective Global Assessment (SGA)</i>	<i>Patient-Generated Subjective Global Assessment (PG-SGA)</i>	<i>Malnutrition Universal Screening Tool (MUST)</i>
<i>Population</i>	Older adults	Older adults (>65)	Older adults	Adults	Malnutrition in oncology/chronic catabolic disease	Adults
<i>Dietary Intake</i>			X	X	X	
<i>Weight History/Loss</i>	X	X		X	X	X
<i>BMI</i>	X	X				X
<i>Mobility/Function</i>	X	X		X	X	
<i>Acute Disease</i>	X	X			X	X
<i>Physiological Stress</i>	X	X				
<i>Neuro-physiological Problems</i>	X	X				
<i>Physical Exams</i>				X	X	
<i>Gastrointestinal Symptoms</i>				X		
<i>Symptoms Affecting Food Intake</i>	X	X			X	
<i>Metabolic Stress</i>					X	
<i>Mid-Arm Circumference (MAC)</i>	X					

For the most part, each tool has its own strengths and limitations. The Mini Nutrition Assessment (MNA)⁵⁸, used more frequently in the literature, is a complex tool with 18 questions comprised of anthropometrics, a global assessment, a diet

questionnaire, and a subjective assessment. The MNA Short-Form (MNA-SF)⁵⁹ is comprised of 6 of the original 18 questions and has been used less, possibly due to the fact that it was recently validated in 2009. Both tools have only been validated in older adults. The Subjective Global Assessment (SGA),⁶⁰ comprised of a medical history and physical exam, and the Patient-Generated SGA (PG-SGA),⁶¹ an adaptation developed specifically for use in oncology, are both tools used less frequently in the literature. Despite their novelty, they are valuable for their comprehensive appreciation of nutritional and non-nutritional factors. The Malnutrition Universal Screening Tool (MUST)⁶² was created for use in all patients based on the unique variables of current weight status, unintentional weight loss, and presence of acute disease. The Dietary Screening Tool (DST)^{63, 64} determines nutrition risk by scoring questions that assess dietary quality, awarding more points for higher consumption of healthier foods and less points for consumption of less healthy foods. Regardless of each tool's respected strengths and limitations, all tools can be useful to an extent for their ability to quickly identify those at risk- requiring nutrition assessments that provide more detailed information including diet history, weight changes, and a physical examination.⁵⁷

Tool selection is an important consideration when assessing an individual. For example, diet quality, which can be measured by the DST, is a potentially modifiable factor that has been associated with many chronic diseases.⁶⁵ The negative outcomes associated with poor nutritional status are numerous,^{9, 66} and it is well established that diet plays a crucial role in cardiovascular health and CVD prevention.^{9, 67} Low quality dietary patterns are positively associated with chronic disease risks such as obesity,

metabolic syndrome, and CVD, while high quality diets have been inversely associated with major chronic disease.⁹ This tool can provide valuable knowledge on individuals with PD and ABI, as many exhibit compromised intakes of foods and nutrients that place them at nutrition risk without evidence of clinical malnutrition.⁶⁴ Measures of food intake and dietary quality can also help to provide insight into food behaviors describing habitual consumption details of areas that need improvement.

For these reasons, the DST may be a more appropriate tool than others for individuals with cognitive deficits such as those with PD and ABI. The DST is brief, low participant burden, and relatively easy to complete. Those considered at risk using the DST may be recommended for a nutritional assessment that would necessitate interventions specifically aimed at improving dietary intake to improve overall nutrition status. Consistent assessments repeated over time could lead to a better understanding of the relationship between diet and disease.⁶⁴ Therefore, analysis of dietary quality can be an important approach for early identification of those with compromised dietary intake, which may be indicated by a nutritional risk screening. Identification of these individuals can help guide individualized intervention development, directed toward preventing or diminishing the rate of decline. Proactive screening and intervention in older adults has proven successful as a public health strategy to improve dietary intake and quality of life, as well as for primary or secondary prevention of disease.^{68, 69} Similar effects can be expected with proactive screening of individuals with PD and ABI.

b. Nutritional Status of Individuals with PD

Factors Affecting Nutritional Status in Individuals with PD

Multiple functional changes occur in individuals with PD that affect nutritional status. PD is typically seen in the older population, and the mean age of diagnosis of PD is 62 years.⁷⁰ Individuals with PD may be at greater risk for poor nutritional status compared to older adults without the disease due to simultaneous declines associated with aging and disease progression.⁷¹ These physiological changes include reductions in sense of smell and taste, decreased ability to chew and swallow, and alterations in gastrointestinal and bowel function.⁷¹⁻⁷⁴ Possible nutritional complications resulting from PD include loss of appetite, unintentional weight loss, decreased enjoyment of food, alterations in nutrient needs, and ultimately deficits in overall nutritional status.⁷⁵ Changes in body composition like increases in adipose tissue and decreases in muscle mass, strength, and ability to perform activities of daily living are seen as people age and as a sequelae of PD.⁷⁶⁻⁷⁸ Furthermore, individuals with PD can display impaired motor skills hindering food shopping, cooking, and eating due to a reduced ability to safely prepare food, use utensils, and transfer food to the mouth.⁷⁹⁻⁸² Combined with impaired cognition, these deficits can adversely affect nutritional status by altering dietary habits and food intake.⁸⁰⁻⁸²

Physiological changes experienced by those with PD may be exacerbated by additional non-physiological changes. These can include an increase in the number of medical costs and hospital days.¹⁴ In addition, numerous costly pharmacologic medications prescribed for PD come along with negative side effects.^{14, 83} There is a significant financial burden placed on this population from costs related to disability,

therapy, and medicine.¹⁴ As a result, individuals with PD have twice the direct medical costs of those without PD.¹⁴ Medications for PD treatment including Levodopa, the most effective agent in the management of Parkinson's symptoms, which can cause adverse side effects such as nausea, gastrointestinal distress, lack of appetite, confusion,

and impulsive behaviors like binge eating.⁸⁴ Medications for PD may also interact with certain foods, other medications, vitamins, herbal supplements, over the counter cold pills and other remedies. For example, Levodopa competes with protein for absorption from the gut.⁸⁴ These non-physiological changes need to be taken into consideration when assessing nutritional status of individuals with PD, as they can play an important part in the alteration of dietary quality and intake.

Additional complications from PD that can affect nutritional status are anxiety and depression.^{17, 83, 85} Approximately 35% of individuals exhibit clinically significant depressive symptoms while approximately 40% exhibit symptoms of anxiety.^{85, 86} Disease progression can cause symptoms such as loss of independence and low self-esteem by forcing individuals to make changes to their normal habits and routines.⁷⁹ Usual simple tasks such as grocery shopping and cooking meals can become stressful, difficult, and burdensome to accomplish as the disease progresses and as debilities such as hyperkinesia begin to appear.⁷⁹ Ultimately, the entire eating experience can be affected, potentially causing nutritional needs to be unmet.⁷⁹ If not treated in a timely manner, these psychiatric disorders can have significant influences on quality of life and nutritional status in individuals with PD.^{17, 79, 83, 85}

Assessment of Nutritional Status in Individuals with PD

Numerous studies have shown a relationship between nutritional status and PD, with a variety of different tools used in a variety of sub-populations.^{2, 48, 57, 87, 88} A commonly used tool in individuals with PD in the current literature is the MNA. Using the MNA, Barichella et al.⁸⁸ reported malnutrition risk increased from 22.9% to 34.3% over three years in individuals with PD with a mean age of 70.5 ± 5.5 years and a mean disease duration of 9 ± 6.3 years.⁸⁸ The MNA was also used by Vikadhl et al.⁸¹ who found the risk of malnutrition increased from 14% to 20.7% from baseline to three years post-diagnosis in a group of individuals with PD with a mean age of 68.4 ± 8.0 years, compared to an increase from 0% to 8.3% in matched controls. Such different percentages of malnutrition in similar populations using the same tool over the same amount of time could be the result of measuring at different times post-diagnosis. Despite these differences, increases in risk of malnutrition in a timespan of just three years would suggest further deterioration in the future. Continuous monitoring of individuals is necessary in order to identify and/or prevent the development of later-onset complications.

Another tool used in this population recently was the DST.⁴⁸ Using a smaller sample with a disease duration of 5.28 ± 4.28 years, LoBuono et al.⁴⁸ found that 38% of individuals with PD were at nutrition risk and 50% were at possible nutrition risk based on dietary quality criteria. However, this study only looked at one point in time and it has been suggested by Fereshtehnejada et al.⁸⁷ that it is necessary to closely monitor and evaluate aspects of nutrition routinely over the long-term. There is a current lack of longitudinal research being done on individuals more than 12 months

post diagnosis, which is necessary to help better understand not only what factors are influencing nutrition, but also how nutrition is influencing other health and lifestyle factors.

c. Nutritional Status of Individuals with TBI

Factors Affecting Nutritional Status in Individuals with TBI

Similar to individuals with PD, individuals with TBI experience physiological sequelae that affect nutritional status including cognitive deficits, depression, alterations in gastrointestinal function, sleep disturbances, and impaired ability to shop, cook, and eat independently.^{16, 34} In addition, alterations in swallowing ability can impact the nutritional status of individuals with TBI.⁵⁰ Impaired cognition and damage to the part of the brain that involves swallowing can contribute to dysphagia.⁵⁰ Dysphagia after TBI is reported to be as high as 61%, and up to 16% of individual do not regain independent oral feedings even 6 months post-injury.⁴⁹ Additional complications that happen immediately after injury include dental and facial fractures and/or the need to be mechanically ventilated, which may eliminate an oral diet completely and require enteral or parenteral nutrition.⁸⁹ Nutrition is a significant predictor of death in individuals with TBI.⁹⁰ Early and adequate nutrition support immediately post-TBI is important and has been associated with a decrease in mortality rate,⁹⁰ but little is known about the impact of nutrition after. In a review article of the long-term health implications of TBI, Murphy and Carmine¹⁶ emphasize the importance of consistent health screenings to evaluate the individuals comprehensive issues. Tools used at screenings that measure nutritional status are important to identify and/or prevent later-onset complications after injury.¹⁶

Assessment of Nutritional Status in Individuals with TBI

Assessment of nutritional status during and immediately after the acute stages of TBI has been thoroughly examined, with a majority of data showing significant nutrition deficits during this time.^{49, 66, 91, 92} Data looking beyond the first few months post-diagnosis is limited. One of the few studies of the post-acute rehabilitation period was a retrospective study performed in Sweden assessing individuals with a severe TBI.⁴⁹ Using the MUST, Krakau et al.⁴⁹ found that 68% of individuals with TBI exhibited signs of malnutrition extending to the second month after injury. Even less data exists on nutritional status more than six months post-injury. Using the DST, LoBuono et al.⁴⁸ assessed individuals with ABI who were 14.25 ± 5.6 months post-injury and found 75% of individuals were at possible nutrition risk. It is unclear how nutritional status is affected over time once individuals are in the rehabilitation period, as consistent nutritional status assessments that occur a year or more post-diagnosis are difficult to find.

d. Nutritional Status of Individuals with Stroke

Factors Affecting Nutritional Status in Individuals with Stroke

Factors affecting nutritional status in individuals with stroke are similar to those of individuals with PD, since both conditions tend to occur at an older age. The average age of an individual experiencing a stroke is 69, only 4 years greater than the average age of an individual with PD.⁹³ The risk of poor nutritional status after a stroke is increased for those that are older.⁹¹ There is a high level of impairment and disability immediately post-stroke due to brain trauma and as a result adequate

nutritional support can be difficult.⁹⁴ Dietary intake is reduced immediately post-stroke, during hospitalization and rehabilitation, and 6 months after diagnosis, resulting in energy and protein deficits associated with overall loss of body tissue.⁹⁴

Significant functional changes that present after stroke include dysphagia, aphasia, motor weakness, and disturbance of sensory function.⁴ These deficits can alter the ability of the individuals to prepare, shop, and cook food.⁴ Dysphagia is highly prevalent in individuals immediately post-stroke.⁹⁵ According to a systematic review by Martino et al.⁹⁵ dysphagia incidence has been reported as high as 78% depending on the instrument used. Dysphagia is a dangerous consequence of stroke that increases the risk of pneumonia, dehydration, and malnutrition (Figure A).⁹⁶

Individuals who are not diagnosed with dysphagia may still experience sequelae related to the stroke including poor appetite from motor weakness or sensory function disturbances.^{4, 97} As a result, they may eat more slowly or be less inclined to eat because of facial weakness or social embarrassment related to changes in eating skills; all of which will decrease total energy intake.⁹⁷ Fatigue and low energy levels are a frequent burden in individuals after stroke recovery that can affect food purchase, preparation, and appetite.^{4, 98} Sequelae such as chronic fatigue can result in feelings of sadness and depression that can also affect nutritional status; 15% of individuals post-stroke report feeling gloomy and sad at least some of the time.⁴ An individual with these symptoms might experience a decrease in motivation to eat and/or difficulties with food preparation that cause them to reduce their intake.⁴ Undernutrition immediately after a stroke increases the risk of poor outcomes such as reductions in functional status, prolonged hospital stay, and reduced survival-

outcomes that can continue six months post-stroke and beyond.⁹⁹⁻¹⁰² While individualized nutritional support can prevent clinically significant weight loss and improve quality of life three months after stroke in elderly individuals at nutritional risk, the long term effects of poor nutritional status and prevention efforts have yet to be studied.⁹⁹

Figure A:

Assessment of Nutritional Status in Individuals with Stroke

The interaction of nutrition and stroke is well recognized, and variety of tools have been used to classify nutritional status after stroke.^{91, 94, 103} These tools can identify people who may be at nutritional risk. Based off the results of the tool, an intervention can be initiated to help improve nutrition, delay the effects of aging, and possibly reduce the burden of disease.¹⁰³ The prevalence of poor nutritional status immediately post-stroke has been as high as 26.3%.¹⁰⁴ Substantially less research exists on nutritional status after stroke hospitalization. Finestone et al.⁹² evaluated nutritional status using biochemical and anthropometric data to identify the presence of malnutrition. On admission to a stroke rehabilitation service immediately after

hospital discharge, 49% of individuals were considered malnourished, declining to 34%, 22%, and 19% at 1 months, 2 months, and 4 months, respectfully.⁹² Brynningsen et al.¹⁰² evaluated nutritional status using the variables of body weight, BMI, mid upper arm circumference, tricep skinfold thickness, and serum concentrations of albumin and transferrin, with malnutrition being defined as the presence of two or more abnormal variables. One week after stroke, 35% of individuals studied were considered malnourished, with 22% being malnourished six months post-stroke. These results show that deficits are still seen in the population several months after the event, and warrants further investigation into whether deficits continue beyond this period of time. Furthermore, while these results provide important insight into the overall trends of nutritional status in the stroke population, we do not know how many individuals post-stroke improve, decline, or have no change in their nutritional status over time. The lack of research in this population on long-term nutritional status demonstrates the need for more knowledge in this area, so that prevention and intervention programs can be developed and implemented at the appropriate times.

IV. Cognition in Individuals with Neurological Disorders

a. Defining Cognition

Cognition is defined as conscious mental activities such as thinking, understanding, learning, and remembering.¹⁰⁵ A gold standard cognition assessment tool has yet to be established, as different tools provide varying assessment measurements. For example, the Scales for Outcomes in Parkinson's disease-COGnition (SCOPA-COG)¹⁰⁶ was developed specifically for individuals with PD for the purpose of comparing groups in research settings and not as a screening or

diagnostic tool, with indexes for attention, memory and learning, executive functions, visuospatial functions, verbal functions, and thinking and reasoning. The Repeatable Battery for the Assessment of Neuropsychological Status (RBANS)¹⁰⁷ was developed for the dual purposes of identifying and characterizing abnormal cognitive decline in the older adult and as a neuropsychological screening battery for younger individual with indexes for immediate memory, visuospatial/constructional, language, attention, and delayed memory. Repeated measurements of cognitive status over time is vital in individuals with PD and ABI in order to prevent, detect, and monitor cognitive deficits.

b. Cognition in Individuals with PD

Although PD is primarily characterized as a movement disorder, deficits in cognition are also associated with the disease and can greatly impact activities of daily living. Impairments in cognition are common even in newly diagnosed individuals, with worsening levels associated with the duration of the disease.^{3, 12, 106, 108} It appears that there is a spectrum of cognitive impairment, ranging from a lesser severe form known as mild cognitive impairment (MCI), to PD dementia (PDD).¹⁰⁹

Cognitive deficits in this population are seen in a variety of domains, including attention, memory and learning, executive functions, visuospatial functions, verbal functions, and thinking and reasoning.¹⁰⁶ Risk factors for MCI in individuals with PD are older age at disease onset, male gender, severity of motor symptoms, and advanced diseased stage.¹¹⁰ Individuals with PD with MCI have an increased risk of developing dementia, both in general and when compared to individuals with PD with intact cognition.^{3, 111} Thus, it has been suggested that MCI in PD is an early manifestation of

dementia.^{111, 112} Mild cognitive impairment is defined as the transitional state between normality and dementia, with cognitive impairment that is abnormal for age but with little to no impairments in activities of daily living.^{111, 112} Aarsland et al.¹¹³ found that for individuals with PD, the cumulative incidence of dementia was 52% four years after diagnosis and nearly 80% after eight years.

Changes within the brain are the source of cognitive deficits seen in individuals with PD.^{26, 114} The cause appears to be the lack of dopamine-producing neurons, but the exact mechanism responsible for cell death remains unknown.²⁶ Research suggests that the pathogenic trigger could actually be a collection of causes or events, that although harmless when appearing by themselves, may produce deleterious effects upon accumulation.²⁶ Cumulatively, mitochondrial dysfunction, oxidative stress, and altered expression of proteins are thought to play a critical role in the pathogenesis of PD, and it is hypothesized that these processes are induced by non-genetic factors such as environmental toxins in interaction with susceptibility genes.^{114, 115}

Cognitive deficits in individuals with PD are heterogeneous, seen in a variety of domains, and at different stages of PD.¹¹⁶ Deficits in executive function tend to be the most frequently reported cognitive problem.¹¹⁶ These deficits affect the ability to plan, organize, and regulate goal-oriented behavior. Using the Frontal Assessment Battery (FAB) to measure executive functioning, Lima et al.¹¹⁷ found that even after controlling for age and education level, individuals with PD had significantly lower scores than controls (13.7 vs. 14.9; $p < 0.0001$). Consistent with these results are those of Verbaan et al.,¹¹⁸ who found that individuals with PD scored significantly lower on

all cognitive subdomains using the SCOPA-COG compared with controls, with the largest differences occurring in the subdomains of executive functioning and memory. Furthermore, memory, attention and visuospatial functioning are also considered important domains that see decline in individuals with PD.^{106, 108-111, 116, 118} In comparison with controls, Verbaan et al.¹¹⁸ found that all four cognitive domains were impaired in individuals with PD. Cognitive deficits are closely associated with depression in individuals with PD and can negatively affect quality of life.^{17, 79}

c. Cognition in Individuals with TBI

Cognitive deficits are seen in virtually all individuals who sustain a TBI, as the injury directly impacts the brain.^{53, 119} These cognitive deficits often become chronic, negatively affecting nutrition and overall health status by directly impacting the activities of daily living. Impairments in cognition are most prominent in newly diagnosed individuals, with improvements occurring during the rehabilitation period.¹¹⁹ While there is insufficient evidence to determine whether mild TBI is associated with cognitive deficits six months or longer post-injury, cognitive deficits are associated with penetrating, moderate, and severe TBIs in individuals who are six months or more post-injury.⁵³

There is significant variability in cognitive status after TBI, given wide heterogeneity among individuals.^{5, 119} Chronic cognitive sequelae that occur after TBI include problems with attention, memory, problem solving, judgment, new learning, and processing speed.¹¹⁹ While the specific areas of cognition affected by TBI have been studied extensively, there is a lack of evidence on the timeline of recovery of cognitive status in individuals from the community.^{48, 119} Millis et al.¹¹⁹ looked at

neuropsychological status of individuals with TBI at one year and five years post-injury, finding 22% improved and 15% declined. It appears that improvements in the areas of attention, cognitive speed, verbal fluency, verbal memory, and visuoconstruction continue up to around 18 months post-injury, while long-term deficits are more likely to be seen in the areas of memory function, attention, and processing speed.¹¹⁹ Additional population-based studies are required to reliably identify the individuals at risk for cognitive decline, as determinants of progression of cognitive impairment are still unknown.

Individuals with TBI demonstrate short-term and long-term neuropsychological deficits.^{34, 35, 120, 121} Short-term effects have been extensively studied, including reduced capacity for new learning and slowed information processing.^{120, 121} Depression has been seen across the traumatic brain injury timeline. There is an increased risk of depression following head injury, while injury during early adulthood has been associated with a lifetime prevalence of minor and major depression.³⁴ Furthermore, research suggests individuals with TBI have an increased risk of Alzheimer's disease and other forms of dementia years after initial injury.³⁵ Similar to what is seen in the PD population, cognitive deficits including neurophysiological outcomes such as depression in individuals with TBI are closely associated with quality of life and increase the burden of illness, which may significantly affect overall health as a result.³⁴

d. Cognition in Individuals with Stroke

Deficits in cognition are commonly associated with stroke and vary depending on the stroke severity.¹²² Similar to individuals with TBI, impairments in cognition are

most prominent in newly diagnosed individuals, with improvements occurring during the rehabilitation period.¹²³ While some cognitive improvements do occur as individual progress after stroke, a majority continue with some degree of cognitive dysfunction.⁵² This chronic deficit in cognition can negatively influence nutrition and overall health status by directly impacting functionality.¹²⁴ Recovery of cognitive function has been found to be a near-significant factor for return to work.⁵² In a study of 58 individuals age 65 and younger, Hofgren et al.⁵² found that despite significant improvements from discharge to one year, 83% still had cognitive dysfunction. Only 7% had returned to work at one year, with this number improving to 20% after three years.

Cognitive deficits in this population are seen in a variety of domains, including memory, orientation, language, and attention.^{122, 125} The risk and severity of cognitive deficits post-stroke do not appear to be influenced by the type of stroke,^{122, 125} but more influenced by age, mental decline before stroke, number of prescribed drugs, diastolic BP on admission, and episodes of hypotension during hospital admission.⁵ Cognitive recovery in stroke is similar to that of TBI as it is highly dependent on a number of individual variables.^{5, 52, 123} As a result, it has yet to be determined who is more prone to deterioration.⁵

While it appears that cognitive deficits remain permanent and fairly stable over the long term, more individuals tend to deteriorate than improve.^{5, 123} Using the Clinical Dementia Rating (CDR) to define progression of cognitive impairment, Del Ser et al.⁵ found that cognition remained stable for two years after stroke in 78.2% of 193 individuals studied, while 14% declined and 7.8% improved. Using the Mini-

Mental State Examination (MMSE),¹²⁶ Patel et al.¹²³ found similar results in that cognitive impairment remained highly prevalent up to three years post-stroke in 163 individuals. At three months, one, two and three years post-stroke, the prevalence rates of cognitive impairment were 39%, 35%, 30% and 32% respectively. More specific longitudinal research extending beyond three years after stroke is limited. Consistent, long-term monitoring of cognitive status is needed to reliably identify and intervene in individuals at risk for cognitive decline.

Similar to individuals with PD, individuals post-stroke have an increased risk of developing dementia, with approximately 1/6 of individuals having dementia before the stroke event.¹²⁷ The risk of dementia is higher in older individuals and in those with preexisting cognitive deficits.¹²⁷ Approximately 30% of survivors develop post-stroke dementia (PSD) within 12 months of having a stroke, while many others experience indications of early cognitive impairment in the absence of dementia.¹²⁵ Cognitive Impairment, No Dementia (CIND) utilizes a combination of clinical and global cognitive criteria to identify cognitive impairment in the absence of dementia.¹²⁸ Stephens et al.¹²⁹ found that individual three months post-stroke with CIND were impaired in all cognitive domains, but scored significantly lower on tests of executive function, memory, and language compared to those not meeting CIND criteria. Deficits were significantly more pronounced in those with dementia in the areas of orientation and memory.¹²⁹ It remains unknown whether CIND is the precursor to post-stroke dementia, and further research is required to establish if CIND can predict the possibility of further decline toward dementia.¹²⁹ Understanding the

evolution of cognitive decline in individuals post-stroke and identifying individuals at risk could help to guide intervention development and improve overall quality of life.

III. Cardiometabolic Risk Factors in Individuals with Neurological Disorders

a. Defining Cardiometabolic Risk Factors

The assessment of cardiometabolic risk factors is useful across diagnoses to guide treatment. Although CVD is the number one cause of death in the US, few studies have explored cardiometabolic changes in individuals with PD and ABI.¹⁹ Cardiometabolic risk factors contribute to an increased risk for CVD and modifiable factors include: overweight/obesity, insulin resistance, hyperglycemia, dyslipidemia, hypertension, inflammation, smoking, and physical inactivity (Figure B).¹³⁰ Non-modifiable risk factors are age, sex, ethnicity, and family history.

Figure B:

b. Cardiometabolic Risk Factors in Individuals with PD

Current research demonstrates that those with PD may be at decreased CVD risk because of a decrease in sympathetic activity that reduces important cardiometabolic risk factors such as hypertension, diabetes, and dyslipidemia.¹³¹ This is because the sympathetic nervous system is involved in the release of cortisol and catecholamines, which through a variety of steps stimulates glycogenolysis and thus increases plasma glucose and lipids.^{131, 132} In addition, increased sympathetic activity has also been shown to increase blood pressure.¹³² However, many individuals with PD receive treatment that may negate any CVD risk reduction benefits.¹³³ Levodopa, for example, raises levels of homocysteine in the blood.¹³³ Elevated homocysteine levels have been associated with an increased risk of vascular diseases in both the general population and in those with PD.^{133, 134} Despite this association, several studies have demonstrated that the presence of hypertension is similar or lower in individuals with PD than controls.^{1, 131, 135} Although two of three studies had control groups that would not be considered representative of the general population, there may be other unknown factors involved that contribute to this occurrence.^{1, 135} While diabetes has been associated with chronic neurodegeneration and it is hypothesized that chronic hyperglycemia may exacerbate the severity of the motor disability^{136, 137}, evidence associating diabetes and PD is inconclusive.

When looking at individuals with PD, both weight loss and gain are seen over time.^{80, 138, 139} In general, individuals with PD tend to lose weight and be underweight, and PD has been reported as an independent risk factor for being underweight and experiencing weight loss.¹⁴⁰ Beyer et al.¹³⁸ found that individuals with PD were four

times more likely to report weight loss greater than 10 pounds than matched control subjects, and individuals with PD reported a mean weight loss of 7.2 pounds compared to 2.1 pounds in control subjects. Furthermore, Abbott et al.⁸⁰ found that 29% of individuals with PD had a BMI $<20 \text{ kg/m}^2$, compared with only 7% of a matched control group, and that 58% of individuals with PD had lost weight since onset of the disease. Strikingly, this study reported that almost 15% of individuals studied lost more than 20% of their original body weight, with none of the individuals starting with a BMI $>27 \text{ kg/m}^2$. Since analysis of dietary intake has indicated that neither protein nor energy intake of individuals with PD significantly differ from the recommended values, it is hypothesized that weight loss is attributed to increased requirements or energy expenditure rather than reduced food intake.^{80, 141} Although studies show that individuals with PD often experience unintentional weight loss, less knowledge exists on when during the course of the disease this actually begins to occur. To our knowledge, the only current proposed outline of the possible natural history of body weight changes in individuals with PD can be seen in Figure C.²

Figure C:

While a large portion of the research demonstrates individuals with PD lose weight, there is also data to show that some individuals gain weight after diagnosis.^{81, 139} Vikdahl et al.⁸¹ performed a community based case-control study in individuals with PD with a mean age of 68.4 ± 8.0 years. Individuals with PD gained a significant amount of weight three years post-diagnosis (1.62 ± 4.60 kilograms (kg), $p=0.009$). Controls did not experience significant weight gain in the same time period (0.23 ± 3.08 kg, $p=0.724$). Barichella et al.¹³⁹ found 62% of 134 individuals with PD with a mean age of 65.9 ± 8.9 years and a mean disease duration of 10.6 ± 5.3 years were overweight or obese, with a higher proportion of obese individuals living with PD than in the general Italian population (25% vs 14%).

Weight gain has been reported after deep brain stimulation (DBS), a surgical procedure used in treatment of PD.¹⁴² Barichella et al.¹⁴² found that significant weight gain ($14.8 \pm 9.8\%$ of original body weight) occurred one year after the procedure in 29 out of 30 individuals studied. It is clear that weight shifts occur during the course of the disease, with both weight loss and weight gain being observed. Overall, weight loss has been more frequently observed as a continuous process that worsens as the disease progresses, with weight loss actually starting several years before diagnosis (Figure C).^{2, 141} Weight gain appears to be more of a result of surgical treatment procedures and has not been associated with the overt disease progression.

Cereda et al.¹ found that individuals with PD had a more favorable cardiometabolic risk profile when compared to a control group. In individuals in the early stages of PD, mean concentrations of glucose, total cholesterol (TC), and triglycerides (TAG) along with BP were all found to be lower in the individuals with PD.¹ While mean concentrations of low density lipoprotein cholesterol (LDL-C) were still suboptimal in the PD population (113.0 ± 30), previous research has associated lower levels of LDL-C with PD.¹⁴³ This study came with a large limitation in that controls were recruited from a medical center treating excessive body weight, and all were sedentary and obese. Furthermore, 25% of the control participants smokers. Studies looking at cardiometabolic risk factors over the long-term are lacking, but are necessary for the prevention of CVD in individuals with PD. Other neurological disorders, including TBI and stroke, also require further assessment of cardiometabolic risk factors in order to identify which risk factors are most prominent and therefore most necessary to screen for.

c. Cardiometabolic Risk Factors in Individuals with TBI

Experiencing a TBI can effect multiple organ systems and cardiometabolic risk factors, initiating and accelerating the chronic disease process.¹⁴⁴ In an article on the classification of TBI as a chronic process, not an event, Masel and DeWitt¹⁴⁴ encourage an emphasis on the management of TBI as a chronic disease and for it to be defined as such by health care and insurance providers. Current treatment with acute rehabilitation creates the perception that little additional treatment is needed and that no chronic effects exist. This misunderstanding can leave health deficits undetected and can increase the risk for health problems such as metabolic syndrome and CVD.¹⁶

Individuals with TBI are slightly more likely to be diagnosed with traditional cardiometabolic risk factors before or after a TBI compared with unaffected individuals.⁵⁵ Individuals with TBI had significantly higher rates of hypertension, diabetes, and CVD compared to unaffected individuals (16.4% vs 14.3%, 8.8% vs. 7.0%, 9.6% vs. 8.1%), but similar rates of dyslipidemia (7.6 vs. 7.7%).⁵⁵ These results have important clinical implications in the management of individuals post-TBI because they demonstrate that more intensive medical monitoring, support, and interventions are needed.⁵⁵

Weight gain in individuals with TBI may also increase the risk for CVD if additional risk factors are present.¹⁶ It has been noted that individuals with TBI have prominent risk factors for metabolic syndrome, a chronic medical disorder that increases the risk for both CVD and diabetes.¹⁶ Cardiovascular disease is considered to be the primary clinical outcome of metabolic syndrome, characterized by measurements of waist circumference, insulin resistance, elevated BP, and

dyslipidemia.¹⁴⁵ Metabolic syndrome has overlapping risk factors with that of CVD, and both can be deadly if not treated appropriately.¹⁴⁵

Chronic TBI sequelae that could affect cardiovascular health include impulsivity, problems with memory, poor self-regulation, decreased mobility, altered sense of satiety, and prolonged use of anti-psychotic, anti-convulsant, and selective serotonin reuptake inhibitor medications.^{16, 50, 55} Currently, there is a knowledge gap on the status of blood pressure, the lipid profile, and blood glucose levels in individuals post-TBI. Knowledge of how and when cardiometabolic risk factors are affected by a TBI may help to prevent risk factors from occurring in the first place and treat risk factors that are already present.

Weight status of individuals with TBI varies depending on the severity of the injury and the time since diagnosis. Individuals sustaining a TBI lose weight immediately after and approximately 1-2 months after injury, followed by a slow return toward the initial weight.⁴⁹ Considerable metabolic alterations including hypercatabolism and hypermetabolism dramatically increase the demand for energy and protein immediately after injury.^{89, 146} Initial dramatic metabolic responses to trauma and escalation in nutritional demand typically results in weight loss in individuals with TBI.⁴⁹ Using indirect calorimetry, Bruder et al.¹⁴⁷ measured energy expenditure in individuals after severe TBI and found that expenditure increased to 150% of basal energy expenditure 12 hours after lifting sedation. After sedation was lifted for 48 hours, energy expenditure remained elevated to 130% of basal energy expenditure.¹⁴⁷ Overall, energy expenditure increased approximately 700 additional calories per day from the period during sedation to the time period off sedation.¹⁴⁷

Krakau et al.⁴⁹ assessed weight status of 56 individuals with TBI during the intensive care period, and found that within 3 ± 2.9 days of the first recorded measurement, all but four had experienced some initial weight loss.

If individuals with TBI gain weight, it is common to be a later-onset complication more frequently associated with post-traumatic hypopituitarism (PTHP).¹⁴⁸ Functionality of the pituitary gland, which sits at the base of the skull, is altered in individuals after TBI, reducing the amount of hormones normally produced by the gland.¹⁴⁸ The prevalence of PTHP has been estimated at 16% in individuals >12 months post-injury, with likelihood of PTHP increasing with severity of injury.¹⁴⁹ Posttraumatic hypopituitarism has been associated with an unfavorable body composition, lipid profile, and decreased quality of life.¹⁵⁰

Individuals with TBI and PTHP present with a less favorable cardiometabolic profile than those with TBI and without PTHP.^{148, 149} Prodam et al.¹⁵¹ reported higher prevalence of dyslipidemia and altered glucose metabolism in those with PTHP compared to non PTHP individuals >12 months post-injury. Triglycerides were significantly higher in individuals with PTHP than not (161.3 ± 29.3 vs. 98.0 ± 16.8 mg/dL, $p < 0.05$). Although TC levels did not significantly differ between the groups, the mean TC of individuals with PTHP was above the optimal concentration of 200 mg/dL (204.1 ± 14.5 mg/dL). Agha et al.¹⁵² reported 13 of 50 individuals studied in the immediate period following TBI had diabetes, with nine individuals fully recovered by six months and one additional individual recovered by 12 months. While most cases of diabetes appear to recover completely, there are individuals who experience long-term diabetes who require chronic treatment. Although these

cardiometabolic risk factors have been assessed at the time of diagnosis and in the time period immediately after diagnosis, little has been done to monitor their progression over time in individuals living with TBI. Consistent, long-term monitoring of cardiometabolic risk factors is necessary to identify and/or prevent the development of later-onset complications for those that are obligated to live with the sequelae of TBI for years and even decades post-diagnosis with no true cure.

d. Cardiometabolic Risk Factors in Individuals with Stroke

In the US, approximately 185,000 individuals experience recurrent strokes annually, and they are associated with having a greater number of cardiometabolic risk factors.⁹ Survivors beyond one year die at twice the expected rate among the general population over the subsequent four years, and the most common cause of death is CVD.¹⁵³ Therefore, identifying cardiometabolic risk factors in individuals after stroke can help mitigate this risk.¹⁵⁴ Research performed on cardiometabolic risk factors following a stroke demonstrate the need for improvement in the management of secondary prevention strategies of cardiometabolic risk factors.^{155, 156}

Hypertension is an influential risk factor for cardiovascular events, and lower rates of recurrent stroke have been associated with lower BP.¹⁵⁷ While it has been reported that each 10 mmHg increase in DBP increases the risk of first stroke by 50%,¹⁵⁸ decreasing DBP by 5 mmHg can reduce the risk of recurrent stroke by 1/3.¹⁵⁹ Although a target BP range has not yet been established, The Secondary Prevention of Small Subcortical Strokes multi-center international trial showed that recurrent stroke was reduced by 20% (p=0.07) in those at a goal SBP of <130 mmHg relative to those at a goal >130 mmHg.¹⁵⁷ These reductions were statistically non-significant, but the

trends are clinically important in that they demonstrate that reduced blood pressure could reduce recurrent cardiovascular events.¹⁵⁷

Blood pressure, hyperlipidemia, and diabetes are among the risk factors that are often suboptimally managed after stroke.¹⁵⁵ Mouradian et al.¹⁵⁵ found that 1/3 of individuals post-stroke had inadequately managed BP at baseline and 14% at one year follow up, indicating suboptimal secondary prevention. Over 80% of individuals were inadequately managed for hyperlipidemia at baseline, with 51% remaining inadequately managed after one year. In 77% of diabetic individuals post-stroke, baseline glycemic control was considered inadequate. At one year post-stroke, 65% of diabetics continued to have inadequate glycemic control.¹⁵⁵ These results of suboptimal management of multiple cardiometabolic risk factors post-stroke demonstrate the need for more consistent assessments and monitoring this population in order to prevent future damage from occurring and to make a significant impact on reducing the risk of CVD.

Obesity is an important risk factor for stroke, and elevated BMI at the time of stroke is associated with poor outcomes including a lower likelihood of hospital discharge and extended hospital stay.¹⁶⁰ After stroke, weight loss is seen as a result of sequelae such as eating difficulties and loss of independence.¹⁶¹ Weight loss >3 kg was found in 24% of individuals four months post-stroke and in 26% of individuals one year post-stroke by Jonsson et al.¹⁶¹ Weight loss is also brought about by the impaired metabolic signaling affected by stroke.¹⁶² A catabolic/anabolic imbalance may develop and result in wasting of fat, muscle, and eventually weight loss.

Due to the false alterations in plasma lipid concentrations immediately after stroke, it is recommended that lipid measurements should be taken at least three months after stroke.¹⁶³ Ryder et al.¹⁶⁴ found that TC ($p < 0.001$), LDL-C ($p < 0.001$), high density lipoprotein cholesterol (HDL-C) ($p < 0.01$), and glucose ($p < 0.001$) concentrations all showed significant declines between days one and nine post-stroke, with TAG concentrations unchanged. No overall significant differences were seen between plasma lipid concentrations measured during the first 48 hours after stroke and those measured three months later, and more individuals had hypercholesterolemia in the first 48 hours than at three months. In a review that characterizes lipid profile changes after stroke, Rosenson et al.¹⁶⁵ found maximal reductions in TC occur at days four to five post-stroke, with levels 47% below baseline. Both LDL-C and HDL-C concentrations decreased to 48% and 32% below baseline on day 7, respectively. In contrast to results from Ryder et al.¹⁶⁴ TAG concentrations increased up to 58% above baseline on day seven.

Reasons for differences in plasma lipid concentrations post-stroke can be a result of different stroke subtypes and stroke severity.⁵⁴ When looking at the three subtypes of cerebral infarction, lacunar infarction, and intracerebral hemorrhage, Woo et al.⁵⁴ found that individual suffering cerebral infarction had significantly higher concentrations of TC and LDL-C and significantly lower concentrations of TAG <48 hours after stroke than 3 months later. The plasma lipid concentrations of individuals suffering lacunar infarction were similar on both occasions except for significant differences in higher TC and LDL-C <48 hours after stroke. No significant changes

were observed among individual suffering from cerebral hemorrhage except for a significantly higher concentration of HDL-C <48 hours after stroke.

VI. Conclusion

Parkinson's disease, TBI, and stroke are three related neurological disorders that impact nutritional status, cognition, and cardiometabolic risk factors. Current research does not adequately address how natural disease evolution affects these three health outcomes in those living with these neurological disorders for twelve or more months post-diagnosis. Awareness to this gap in knowledge has been called to attention in all three disease states. Fereshtehnejad et al.⁸⁷ stated, "a follow-up examination of PD patients to track longitudinal changes is of great demand," while Masel and DeWitt¹⁴⁴ recommended that TBI research be directed at discovering therapies to interrupt the disease processes after the initiating event as early as possible. Levine et al.¹⁶⁶ concluded that stroke survivors warrant monitoring for impairment over the years after the event, and that strategies and systems will need to be developed in order to manage the long-term needs of this population. Further evaluation is required to understand the difference between each disease state and to improve treatment for those living with these long-term conditions. Further research is also required to determine differences in the rate of change in nutritional status over time between PD and ABI, as well as the impact of nutritional status on cognition and cardiometabolic risk factors over time.

Nutritional status, cognition, and cardiometabolic risk may change over time in individuals with PD and ABI. Repeated evaluations may increase awareness and knowledge of associated health risks, allowing for superior evidence-based treatments

and improved overall outcomes. Therefore, it is critical to perform consistent, long-term evaluations of individuals with PD and ABI to detect disease progression trends and promote earlier and more targeted interventions to attenuate disease advancement. Consistent assessments over time of these health outcomes are vital in order to identify and/or prevent the development of later-onset complications. Furthermore, longitudinal assessments can help identify which consequences of these neurological disorders are most prominent and who is at greatest risk.

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APPENDIX B: EXTENDED METHODOLOGY

Subjects

Individuals diagnosed with PD or ABI were recruited on a rolling basis starting in the fall of 2013. A total of thirteen participants were included in this study and consisted of nine males and four females between the ages of 33 and 71. Participants were eligible if they were between the ages of 18 to 85 and were diagnosed with PD, TBI, or stroke for ≥ 12 months. Participants were required to have a method of transportation to the URI Walter J. Beaupre Speech and Hearing Clinic. This research was conducted as an ancillary portion of a longitudinal, observational study examining the physical, cognitive-linguistic, and dietary characteristics of individuals with PD and ABI. Approval was obtained 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 given to physician offices, hospitals, rehabilitation centers, and health professionals for recruitment. Individuals participating in URI's Department of Communicative Disorder's LOUD Crowd and Gateway Wellness Café also received brochures. Study staff explained the research project to potential participants. If a potential participant was interested and eligible to participate, they signed two copies of an informed consent form. The total time commitment for participants is approximately 3 hours for each evaluation for a total of 33 hours over 5 years. Participants were assigned identification numbers for data management that did not

include any identifying numbers or letters. 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, e-mail, and telephone numbers removed) were stored on password - protected computers in Fogarty 225 (Lofgren's lab).

Protocol Overview

Participants came to the Walter J. Beaupre Speech and Hearing Clinic for anthropometric, biochemical, clinical, nutrition, and cognition assessments once every six months. Participants who have completed three time points will be assessed in this study. Participants fasted for at least 12-hours before each assessment, but were encouraged to drink water and stay adequately hydrated. At each assessment, a 12-hour fasting blood sample was collected first followed by a blood pressure measurement. Specific assessments for this study include: nutritional status measured using the Dietary Screening Tool (DST), cognition measured using the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS), and cardiometabolic risk factors measured using anthropometric, biochemical, and clinical measurements. Descriptions of these and additional assessments are as follows.

Assessments

Nutritional Status

Nutritional status was assessed using the DST.¹ The survey takes approximately 10-15 minutes to administer. The DST has been validated in older adults, and has not yet been validated in individuals living with neurological

disorders.¹ The DST identifies dietary patterns and nutritional risk using a 25-item questionnaire. Seventeen questions relate to the frequency of intake of foods (e.g. how often do you usually eat fruit as a snack?). Two questions are behavior-related (e.g. how many different vegetable servings do you usually have at your main meal of the day?). One final question asks about the use of a multivitamin.¹ The four questions answered either yes or no (e.g. do you usually add butter or margarine to foods like bread, rolls, or biscuits?) are not included in the principal component analysis (PCA) because of the dichotomous response options.^{1,2}

Points are first based on major dietary category.¹ For example, 15 points alone are allotted to two questions about vegetables.¹ Answers to the questions are given points based on the chosen response. Point values are assigned based on higher consumption of a healthier diet.¹ Eating three more servings of different vegetables at the main meal of the day is awarded seven points, while no vegetable servings at the main meal of the day is given a point value of zero.² Questions dealing with less healthy patterns receive higher points for lower reported intake.¹ For example, eating less ice cream is associated with more points received.³ One point is awarded to each of the yes-or-no questions, and five additional bonus points are available for the use of a multivitamin.¹ To make the scores more clinically relevant and easily interpretable, a total point score of 100 was chosen.¹

Based on the DST score, participants are put into one of three risk categories. Individuals with scores of <60 are considered “at risk”.¹ Scores of 60-75 are considered “possible risk” and scores >75 are “not at risk”.¹ The DST has proven sensitivity, specificity, and positive predictive values when compared with nutritional

risk based on the Dietary Reference Intakes.¹ The DST has been validated to identify nutritional risk in older adults when compared to 24-hour food recalls.²

Cognition

Cognition was assessed using the RBANS.⁴ The test takes approximately 30 minutes to complete and was validated in adults ages 20-89.⁴ The RBANS is a measurement of neuropsychological status that includes 12 subtests, with five index scores for immediate memory, visuospatial/constructional, language, attention, and delayed memory. Each of the five index scores are added together for a total score; Scores are based on a standardized mean by age and education level for normative samples of 100±15, with lower scores indicative of greater cognitive impairment. The RBANS was originally designed as a screening tool to assess for dementia and has been validated as a reliable tool to assess the cognitive function in individuals with TBI and PD.^{5, 6}

Cardiometabolic Risk Factors

Cardiometabolic risk factors assessed in this study were the anthropometric, biochemical, and clinical modifiable risk factors identified by the American Diabetes Association and the American College of Cardiology Foundation in the concept of global cardiometabolic risk.⁷⁻¹⁰ This concept represents the overall risk of developing CVD. Risk factor categories were: 1.) anthropometric (BMI) 2.) biochemical (TC, HDL-C, LDL-C, TAG, and glucose) and 3.) clinical (blood pressure). Risk criteria values can be seen in Table 1.

	Anthropometric	Biochemical					Clinical
	BMI	TC	HDL-C	LDL-C	TAG	Glucose	BP

Risk value	$\geq 25 \text{ kg/m}^2$	$> 200 \text{ mg/dL}$	$< 40 \text{ mg/dL}$	$> 100 \text{ mg/dL}$	$> 150 \text{ mg/dL}$	$> 100 \text{ mg/dL}$	SBP $> 140 \text{ mmHg}$ and DBP $> 90 \text{ mmHg}$
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Table 1: Criteria for Cardiometabolic Risk Factors⁷⁻¹⁰

Anthropometrics

Trained study personnel obtained all measurements using standard procedures. Height (cm) was measured using a stadiometer (Webb City, MO, USA). Weight (kg) was measured using a bioelectrical impedance analysis device (Tanita BF-556, Arlington Heights, Illinois). Height was rounded to the nearest 0.5 cm, weight was rounded to the closest 0.1 kg, and both were used to calculate BMI (kg/m^2). Body mass index was 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 $\geq 30 \text{ kg/m}^2$.¹¹

Biochemical

Biochemical assessments of serum TC, LDL-C, HDL-C, TAG were performed to analyze cardiometabolic risk factors. Following a 12-hour fast, a finger stick was performed and analyzed using the portable Cholestech[®] machine (Cholestech[®] LDX system, Hayward California). The Cholestech[®] table top analyzer is a valid and reliable method of analyzing lipid and glucose concentrations when compared to standard venous measures.^{12, 13} Using universal precautions and standardized techniques, study staff used a lancet and capillary tube to put forty microliters of blood from each participant's finger into a cartridge for assay. Values used for cutoff criteria can be seen in Table 3, and were factors identified by the American Diabetes

Association and the American College of Cardiology Foundation in the concept of global cardiometabolic risk.⁷⁻¹⁰ This concept represents the overall risk of CVD.

Clinical

Blood pressure was measured by study staff after participants sat quietly for five minutes using an automatic blood pressure machine (Omron Healthcare Inc., Bannockburn Illinois). Blood pressure was taken twice or until readings were within 3 mg/dL and the readings were averaged together.

Additional Assessments

All questionnaires were administered to participants by study personnel. A number of surveys and tests were administered to the participants at each time point. Additional questionnaires and tests that were administered but not analyzed in this study are as follows: the Swallowing Quality of Life Survey (SWAL-QOL), the Physical Activity and Disability Survey (PADS), a 14-item Resilience Questionnaire, and the Satisfaction with Life Scale (SWLS), the Short Physical Performance Battery (SPPB), and the Timed Up and Go (TUG) test.

Statistical Analysis

Data were analyzed using SPSS, version 23.0 for Windows (IBM Corp. Somers, NY). Descriptive statistics were performed and normality assessed using the Shapiro-Wilk test. Transformations or nonparametric tests were used to analyze non-normally distributed data. Between group differences at T1 were determined via independent sample t-tests, Mann-Whitney U tests, and Fisher's exact test. Hedge's *g* was calculated to describe effect size for differences between groups (0.2 = small effect, 0.5 = medium effect, 0.8 = large effect).¹⁴ Repeated measures analysis of

covariance (RMANCOVA) was used to assess nutritional status over time looking at the dependent variable of nutritional status as measured by DST score. Partial η^2 was calculated to describe effect size for differences between groups over time (0.01 = small effect, 0.06 = medium effect, 0.14 = large effect).¹⁴ Analysis of covariance (ANCOVA) was used to assess the relationship at T1 of nutritional status on cardiometabolic risk factors and cognition using DST risk category as the independent variable. Cognition as RBANS score and the number of cardiometabolic risk factors served as the dependent variables. Covariates included were length of time since diagnosis and age, except for RBANS scores which are already age adjusted. Neither time since diagnosis nor age were found to be significant covariates in explaining any of the key dependent measures and were thus not used for analysis or in results presented here. For analyses in violation of Mauchly's test of sphericity, results were interpreted using the Greenhouse-Geisser correction. Analyses were considered significant when $p < 0.05$.

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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?

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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?

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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? _____

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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: _____

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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?

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- 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

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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