Modifying and Validating a Measure of Chronic Stress for People With Aphasia

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Abstract

Purpose: Chronic stress is likely a common experience among people with the language impairment of aphasia. Importantly, chronic stress reportedly alters the neural networks central to learning and memory—essential ingredients of aphasia rehabilitation. Before we can explore the influence of chronic stress on rehabilitation outcomes, we must be able to measure chronic stress in this population. The purpose of this study was to (a) modify a widely used measure of chronic stress (Perceived Stress Scale [PSS]; Cohen & Janicki-Deverts, 2012) to fit the communication needs of people with aphasia (PWA) and (b) validate the modified PSS (mPSS) with PWA.

Method: Following systematic modification of the PSS (with permission), 72 PWA completed the validation portion of the study. Each participant completed the mPSS, measures of depression, anxiety, and resilience, and provided a sample of the stress hormone cortisol extracted from the hair. Pearson's product–moment correlations were used to examine associations between mPSS scores and these measures. Approximately 30% of participants completed the mPSS 1 week later to establish test–retest reliability, analyzed using an interclass correlation coefficient.

Results: Significant positive correlations were evident between the reports of chronic stress and depression and anxiety. In addition, a significant inverse correlation was found between reports of chronic stress and resilience. The mPSS also showed evidence of test–retest reliability. No association was found between mPSS score and cortisol level.

Conclusion: Although questions remain about the biological correlates of chronic stress in people with poststroke aphasia, significant associations between chronic stress and several psychosocial variables provide evidence of validity of this emerging measure of chronic stress.

People with aphasia (PWA) report greater stress; stress-related emotional challenges such as depression, frustration, and anxiety; and decreased quality of life compared to neurotypical adults (DuBay, Laures-Gore, Matheny, & Romski, 2011; Laures-Gore & Buchanan, 2015; Laures-Gore, Hamilton, & Matheny, 2007; Parr, 1994). In fact, communication itself is often deemed stressful—described as "linguistic anxiety" by Cahana-Amitay et al. (2011). At the same time, PWA may possess fewer resources to cope with perceived stress (DuBay et al., 2011). For example, PWA reported less than optimal acceptance of life changes related to aphasia, as well as diminished ability to monitor and manage tension. Prolonged stress not only impacts quality of life and ability to adjust to new life changes but may also influence the overall trajectory of recovery (Code & Herrmann, 2003).

When we consider life with the language impairment of aphasia, it is easy to acknowledge the stress that may stem from participation in daily activities and conversations (Code & Herrmann, 2003; Cruice, Worrall, Hickson, & Murison, 2003; Laures-Gore & Buchanan, 2015). Importantly, if stress continues over the months and years following aphasia onset, it may have a detrimental impact on the physical, emotional, and cognitive health of a person with aphasia—including his or her ability to learn and remember. In the last decade, research evidence suggests that chronic stress may decrease neuronal activity in networks supporting memory and attention (Bao, Meynen, & Swaab, 2008; Christoffel, Golden, & Russo, 2011; Davidson & McEwen, 2012; Gianaros et al., 2007; McEwen, 2001; Mirescu & Gould, 2006). In other words, chronic stress may limit the neuroplastic capacity of the individual and influence his or her ability to learn. If learning is the central component of rehabilitation, PWA experiencing ongoing stress may not be able to fully benefit from what they experience during treatment.

The recovery and rehabilitation of aphasia depends on both the spontaneous neural recovery that occurs after stroke as well as the dynamic plasticity of the brain that occurs with experience and learning (i.e., aphasia treatment; Nadeau, 2014). Experience-dependent neuroplasticity, the foundation for learning across the life span, is also key to rehabilitation following neural injury (Kleim & Jones, 2008). Over the last several decades, aphasiologists have developed a number of speech and language treatments to improve the impairments of aphasia by capitalizing on the neuroplastic potential of each patient. Aphasia treatments focus on relearning of semantic/conceptual, word form, syllabic, and/or phonological aspects of language, with the assumption that neuroplastic changes to the brain occur dynamically in relation to type, intensity, and saliency of these linguistic treatments (Nadeau, 2014; Raymer et al., 2008).

Because chronic stress appears to interfere with an individual's neuroplastic potential, it may therefore limit the success of rehabilitation. Indeed, aphasia treatment outcomes are variable (Cherney & Robey, 2008), even among individuals with similar lesion and impairment profiles (Hemsley & Code, 1996; McClung, Gonzalez Rothi, & Nadeau, 2010). Although a number of factors may contribute to outcome variability, chronic stress is a modifiable factor that may account for individual response to treatment. However, up until now, clinicians and researchers have not had a valid way to measure chronic stress in PWA to examine its impact on rehabilitation outcomes.

In this article, we will summarize empirical evidence about stress and aphasia, the impact of chronic stress on the brain, and how chronic stress is measured. Next, we will describe the process of modifying and validating a self-report measure of chronic stress for PWA and conclude with what we discovered about chronic stress and aphasia in this process.

About Chronic Stress

In McEwen's work on chronic stress, stress is defined as systemic or psychogenic disturbances that interfere with physiological and psychological homeostasis (McEwen, 1998, 2006). When we perceive a stressor, a number of biological and psychological processes are involved in our reaction (Thiel & Dretsch, 2011). Our body is built to accommodate the regular ebb and flow of the physiological stress response, sometimes called *allostasis* (McEwen, 1998, 2006): The various stress response systems in the body react when a stressor arises and then return to baseline when the stressor is removed. A number of neurochemical messengers are involved in the physiological stress response, including the hormone cortisol. Cortisol is released when stress is perceived (Bao et al., 2008) and is frequently used as a biological measure of an individual's level of stress (e.g., Bay, Sikorskii, & Gao, 2008; Juster, McEwen, & Lupien, 2010; Kalra, Einarson, Karaskov, Van Uum, & Koren, 2007; Rosmond, Dallman, & Björntorp, 1998).

Obviously, stress can have a positive influence on the body and behavior; the acute stress response mobilizes our body systems to "fight or flee." This results in our ability to move our body away from danger, complete a high-stakes test, or manage an acute health crisis. If a stressor continues, however, the resulting adaptations are detrimental to the body and brain (McEwen, 1998, 2006; Segerstrom & Miller, 2004). In addition to stress-related physical consequences (see Bao et al., 2008; Segerstrom & Miller, 2004), adaptations to chronic stress also appear to influence the neural structures and physiology important to memory, attention, executive function, and emotion.

Chronic stress, depression, and anxiety are frequently associated (Mazure, 1998; Pittenger & Duman, 2008; Tafet & Bernardini, 2003). It is commonly accepted that, when an individual experiences certain types of stressful life events, symptoms of depression and anxiety may evolve. Furthermore, McEwen and Gianaros (2011) have described depression and anxiety as "disorders of stress adaptation" (p. 440). As stressors arise and are prolonged over time, individuals often adapt both psychologically and physiologically. If adaptation does not occur, however, higher levels of stress-related hormones may be maintained, negatively influencing the neurophysiological networks that subserve emotional regulation as well as some aspects of cognitive function (Tafet & Bernardini, 2003).

In the past decade, a body of research has yielded evidence of chronic stress leading to neuronal atrophy, particularly in the prefrontal cortex and the hippocampus, where a high volume of cortisol receptors resides (e.g., Davidson & McEwen, 2012; Liston, McEwen, & Casey, 2009; McEwen, 2006; Pittenger & Duman, 2008; Russo, Murrough, Han, Charney, & Nestler, 2012). In other words, the stress-related neurophysiological changes in these regions appear to diminish the neural activity required for optimal memory, attention, and executive function—the same neurophysiology required for successful aphasia rehabilitation.

The Impact of Chronic Stress on Cognition

Importantly, the evidence of a relationship between heightened perceived stress and deficits in memory trigger questions about the relationship between chronic stress and learning ability. Answers to these questions have been borne out in prior research (Alderson & Novack, 2002; Conrad, 2006; Kirschbaum, Wolf, May, Wippich, & Hellhammer, 1996; Lupien et al., 2005). For example, Kirschbaum et al. (1996) found that heightened cortisol levels, attributed to perceived stress, were associated with reduced declarative memory

performance and spatial learning. Other studies have examined the influence of behaviorally measured perceived stress and cognitive ability in a number of populations. For example, Aggarwal et al. (2013) examined perceived chronic stress and changes in cognitive function in more than 6,000 community-based adults, aged 65 years and up. Participants completed the Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983) as well as several assessments of cognitive function, including perceptual speed, immediate and delayed recall, and a measure of global cognition. The authors found that as levels of chronic stress increased, cognitive function declined (Aggarwal et al., 2013). In addition, Zuniga, Mackenzie, Kramer, and McAuley (2016) found an association between heightened levels of perceived stress and greater perceived memory impairment in older (aged 59–81 years) community-based adults. These and other studies of stress and cognitive abilities point to the importance of attending to chronic stress when considering the efficacy of rehabilitation.

Measuring Chronic Stress

Chronic stress is generally measured in two ways: behaviorally and biologically. One frequently used behavioral self-report measure of chronic stress is the PSS (Cohen & Janicki-Deverts, 2012; Cohen et al., 1983; Cohen & Williamson, 1988). The PSS is widely used—it has been translated into approximately 30 languages—and is considered a well-validated measure of self-appraised stress. For example, this 10-item scale's psychometric qualities were measured and validated in the United States among 2,387 community-based adults from various geographic regions, residential communities, and of varied ages, ethnicities, races, and socioeconomic backgrounds (Cohen & Williamson, 1988). This scale asks the respondent to assess his or her stress over the last month with questions such as "Within the last month, how often have you felt nervous and 'stressed'?" and "Within the last month, how often have you felt that you were on top of things?" Although the PSS has been used most frequently with nonclinical populations, it has also been administered to patient populations, including stroke survivors and adults with chronic pain (respectively, Gottlieb, Golander, Bar-Tal, & Gottlieb, 2001; Van Uum et al., 2008), and to assess acute stress in PWA (Laures-Gore, Hamilton, & Matheny, 2007; Laures-Gore, 2012). The construct validity of the PSS has also been demonstrated, including in studies that found significant correlations between PSS score and participant cortisol levels (Kalra et al., 2007; Pruessner, Hellhammer, & Kirschbaum, 1999; van Eck & Nicholson, 1994; Van Uum et al., 2008). However, the PSS in its original form may not be fully understood by people who have communication limitations because of its linguistic complexity and visually distracting format (Laures-Gore & DeFife, 2013; Laures-Gore, Hamilton, & Matheny, 2007).

Common biological measures of stress include tests of the stress hormone cortisol in the blood, urine, saliva, and, more recently, hair. Cortisol is a hormone originating from the adrenal gland, which assists with regulating the body's stress response. Immediately following stroke, there is an organic elevation of stress-related hormones that appears to subside after 1 year (Bustamante et al., 2014). At 1 year post–cerebrovascular accident, an elevated amount of cortisol can be an indicator of non–lesion-related stress. Although cortisol measured in blood, saliva, and urine yields data on short-term cortisol concentrations, hair assays provide long-term data about cortisol concentration: 1 cm length of hair contains approximately 1 month of cortisol concentrations (hair cortisol concentration [HCC]; Stalder et al., 2012). Although somewhat novel, most research reports described HCC to be a reliable and valid measure of cortisol levels over an extended time (Manenschijn, Koper, Lamberts, & van Rossum, 2011; Sauve, Koren, Walsch, Tokmakejian, & Van Uum, 2007; Stalder et al., 2012, 2017). In a meta-analysis by Stalder et al. (2017), HCC and self-report measures of

chronic stress were not significantly associated; however, results showed a significant correlation between participant groups characterized by ongoing chronic stress (e.g., participants engaged in caregiving, currently unemployed) and an average of 43% elevation in HCC levels.

Because chronic stress is likely a common experience for PWA, it is useful to know how it has been measured and explored in this population to date.

Measuring Stress in PWA

Stress has been measured both behaviorally and biologically in PWA. For example, Laures-Gore, Hamilton, & Matheny (2007) used the Coping Resources Inventory for Stress (Matheny, Curlette, Aycock, Pugh, & Taylor, 1987) and the PSS (14-item version; Cohen et al., 1983) to examine self-reports of stress in participants with aphasia and neurotypical controls. Laures-Gore, Hamilton, & Matheny (2007) found an association between fewer coping resources and greater perceived stress for the participants with aphasia compared to control participants. Importantly, the authors also acknowledge the limitations of the participants with aphasia in understanding the self-report measures used (Laures-Gore, Hamilton, & Matheny 2007). In addition, perceived chronic stress has been measured in participants with aphasia and compared against a measure of depression and neurological functioning (Laures-Gore & DeFife, 2013). Although some interesting findings emerged about the significant association between perceived stress and symptoms of depression, the authors acknowledged the limitation of using scales that are not intended for participants with aphasia. In other words, the linguistic complexity and format of these measures are not suited for many people with communication limitations.

Examining stress via the biomarker cortisol in PWA has also been undertaken, more often surrounding acute stress associated with linguistic performance. Results have been mixed. Laures-Gore and colleagues reported that acute cortisol levels (via saliva) were elevated for PWA during tasks of word productivity and appear to relate to reduced speech fluency (Buchanan, Laures-Gore, & Duff, 2014; Laures-Gore, 2012; Laures-Gore, DuBay, Duff, & Buchanan, 2010). Another study found no increase in salivary cortisol levels related to linguistic and nonlinguistic stress tasks in PWA, though cortisol increases were found for healthy control participants (Laures-Gore, Heim, & Hsu, 2007). Importantly, Laures-Gore and Buchanan (2015) point out that stress surrounding linguistic performance is not always a negative factor. Just as in an emergent situation requiring "fight or flight," the body's stress response may provide an increase in attention and motivation and support more successful word retrieval or overall fluency. However, there is also evidence demonstrating that PWA experience stress during language tasks and that stress can negatively impact language production (Buchanan et al., 2014; Cahana-Amitay et al., 2011). Further research will help clarify the complex relationship between stress and linguistic performance.

Studies about stress in PWA have allowed us to begin to understand the complexities of acute stress and language performance, both within and outside aphasia treatment. To understand the unique contribution of chronic stress on aphasia rehabilitation, it is important to accurately measure chronic stress. The purpose of this study was to first modify and then validate a measure of chronic stress (the PSS; Cohen & Janicki-Deverts, 2012, with permission) so that it may be maximally understood by PWA. To undertake this research, we conducted two phases. Phase I involved modifying the PSS, and Phase II involved validating the newly modified measure, as framed by the following research questions:

1. Does the modified PSS (mPSS) show evidence of construct validity, determined through correlations between the mPSS and a biological measure of chronic stress (hair cortisol), in PWA? We hypothesized a minimal to moderate ($r > .30$) correlation between the mPSS and level of hair cortisol found in hair.
2. Does the mPSS show evidence of convergent validity, determined through correlations between the mPSS, a visual analogue stress scale, and a resilience scale in PWA? We hypothesized a moderate ($r > .50$) positive correlation between the mPSS and the visual analogue stress scale and a negative correlation between the mPSS and the resilience scale in PWA.
3. Does the mPSS correlate with measures of depression and anxiety in PWA? We hypothesized a moderate ($r > .50$) positive correlation between the mPSS and scales of depression and anxiety in PWA.
4. Does the mPSS show evidence of test–retest reliability, as measured through repeated administration of the mPSS with PWA? We hypothesize the mPSS will demonstrate good to excellent reliability.

Method

This study comprised two phases. In the first phase, the PSS (Cohen & Janicki-Deverts, 2012, with permission) was modified to meet the needs of people with communication limitations. In the second phase, the modified form of the PSS (mPSS) was validated with 72 PWA using other measures of chronic stress, related psychological constructs, and cortisol levels extracted from hair.

Phase I: Modification of the PSS

The authors sought to modify the widely used PSS (Cohen & Janicki-Deverts, 2012; Cohen et al., 1983; Cohen & Williamson, 1988). This scale consists of 10 questions about the participant's perceived stress within the last month. Participants respond on a five-item Likert scale, with options from *never* to *very often*. Each item is scored according to response option (0 = *never* and 4 = *very often*), with a range of total scores from 0 to 40. Some PWA may not adequately understand instructions, questions, or other elements of the PSS in its original form. Therefore, the PSS was modified to decrease the language burden for use with this population. Permission for this modification was granted via personal communication with S. Cohen's lab (April 2014).

The modification of the PSS was conducted in three steps: (a) initial modification of all questions, response options, instructions, and layout of the PSS by two of the authors; (b) review of the initial modifications by two expert panels and subsequent remodification; and (c) administration, cognitive interviewing with PWA, and subsequent remodification of the scale (see Figure 1).

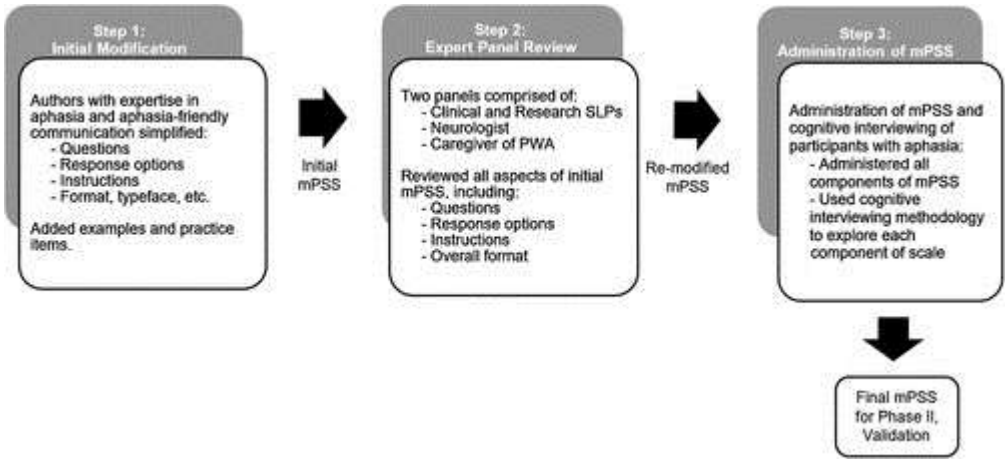

Figure 1. Three steps were used during Phase I: Modification. mPSS = modified Perceived Stress Scale; SLP = speech-language pathologist; PWA = people with aphasia.

In Step 1 of the modification, two of the authors (the first and fourth authors), both of whom possess extensive experience communicating with PWA, reviewed and modified the instructions, questions, response options, and the scale's overall layout. To maintain the validity of the original PSS, the authors avoided altering the tone or intent of the instructions, the number of items, the focus or tenor of each question, and the number or structure of response options.

In Step 2 of the modification, seven experts reviewed and discussed the content and structure of the instrument as part of two review panels, approximately 60 min each. These panels were composed of either three or four members, consisting of a clinical speech-language pathologist, a research speech-language pathologist, a neurologist with expertise in aphasia, and a caregiver of a person with aphasia. Panelists were given a usability heuristic (Couper, 1999) to inspect the mPSS's instructions, layout, example and practice questions, response options, and questions. For example, the usability heuristic included questions such as “Are the words, phrases, and concepts familiar to the user?” and “Is the information presented in a logical order?” (Levi & Conrad, 1995, p. 54).

In Step 3 of the modification process, the mPSS was administered to nine PWA (six women, three men), all at least 1 year poststroke with an average age of 58.67 years and average education of 15.33 years. Participants were selectively recruited from the Northwest Aphasia Registry and Repository based in part on the lead author's familiarity with these participants' ability to engage in relatively abstract discussion. These participants were categorized as mild to mild–moderate in aphasia severity, as determined by Western Aphasia Battery–Aphasia Quotient (scores between 80 and 93.8; Kertesz, 1982). The mPSS was administered to each participant via both silent reading by the participant and reading aloud by the examiner. Following mPSS administration, the examiner conducted a cognitive interview with each of the nine participants. Cognitive interviewing, considered the standard of practice in patient-related outcomes instrument development (DeWalt, Rothrock, Yount, & Stone, 2007; Morgan, Amtmann, Abrahamson, Kajlich, & Hafner, 2014), is used to examine if an instrument functions as intended. Cognitive interviews help instrument developers explore participant cognitive processes used to understand, interpret, make decisions, and respond to each item, response choice, and overall organization of a measure. The purpose of the

interviews was to gather information to help assess the mPSS's face validity and provide ideas for remodification of the scale before administering the instrument more widely. Interviews included questions such as “What is this question about?” “What was your response to Question 1?” and “What were you thinking when you chose your response to Question 1?” These techniques, called think aloud and verbal probing, helped assess participants' understanding and interpretation of each component of the measure (Willis, DeMaio, & Harris-Kojetin, 1999). The results of the audio-recorded interview helped to identify where the participant had difficulty with comprehension, decision making, or responding (see Morgan et al., 2014) and contributed to the final version of the mPSS.

Phase II: Validation of the mPSS

Participants

Seventy-five PWA were recruited from either the VA Health Care System–Puget Sound or the Northwest Aphasia Registry and Repository and screened for the validation phase of the study. Of these, 72 (29 women, 43 men) completed the study; three recruited participants were excluded because they were unable to fully complete the informed consent process. All participants were at least 1 year poststroke and had been diagnosed with aphasia using the McNeil and Pratt (2001) definition: a language-dominant hemisphere lesion resulting in acquired, multimodal language processing deficits. The following characteristics served as exclusion criteria: degenerative neurological disease, dementia, psychiatric disorders, currently uncontrolled substance abuse, diffuse brain injury or disease, pregnancy, adrenocortical dysfunction (e.g., Cushing syndrome or Addison's disease), or use of a systemic glucocorticoid medication (e.g., Prednisone). Participants with concomitant apraxia of speech or dysarthria were permitted into the study. Participants who used hair color or other chemically based hair treatments were included in the study but were excluded from cortisol analyses if the hair treatment occurred more recently than 8 weeks from the time of the study session. The mean age of participants was 64.53 years, the mean duration of education was 16.17 years, and the mean time postonset was 81.10 months. All participants completed the comprehension subtests (spoken and written language) of the Comprehensive Aphasia Test (CAT; Swinburn, Porter, & Howard, 2004), with a mean of 99.89 of 128 (*SD* = 18.65). This mean can be represented as a *T* score of 53 (*M* = 50, *SD* = 10) that references a normative sample of PWA (Swinburn et al., 2004). Participants' scores on the Spoken Language Comprehension subtests ranged from *T* = 35 to *T* = 74; scores on the Written Language Comprehension subtests ranged from *T* = 35 to *T* = 73.

Self-Report Measures

The mPSS was administered to each participant along with several self-report measures of related constructs to provide evidence of validity of the mPSS. The Stress Visual Analogue Scale (Stress VAS; Lesage, Berjot, & Deschamps, 2012) is a single-item measure of current acute stress using a ruler-type scale. The scale includes a single question (“Indicate how stressed you feel on the ruler”) and provides a 100-line ruler with “as bad as it could be” at the top and “none” at the bottom. A maximum score on this scale is 100 (Lesage et al., 2012). The Connor–Davidson Resilience Scale (CD-RISC; Connor & Davidson, 2001) is a 10-question scale developed to measure a person's perceived ability to cope with stressors over the past month. For example, Question 1 states “I am able to adapt when changes occur.” The CD-RISC provides Likert-style response options from “not true at all” to “true nearly all the time,” with a maximum score of 40 for the entire scale.

Because chronic stress is associated with depression and anxiety, these mood disorders were evaluated to gather additional information about the psychological qualities of the sample and the validity of the mPSS. The Patient Health Questionnaire (PHQ-8; Kroenke, Spitzer, & Williams, 2001) and the General Anxiety Disorder Scale (GAD-7; Spitzer, Kroenke, Williams, & Löwe, 2006) were administered to elicit self-appraisal of depression and anxiety, respectively. The PHQ-8 includes eight questions with the lead phrase "How often during the past 2 weeks were you bothered by…" and concluding with a specific phrase about symptoms of depression. For example, Question 1 states "Little interest or pleasure in doing things." Respondents can select one of four response options from "not at all" to "nearly every day," with a maximum score of 24 for the entire scale (Kroenke et al., 2001). The GAD-7 includes seven questions with the lead phrase "How often during the past 2 weeks were you bothered by the following problems?" and concludes with a specific phrase related to symptoms of anxiety. For example, Question 1 states "Feeling nervous, anxious, or on edge." Respondents can select one of four response options from "not at all sure" to "nearly every day," with a maximum score of 21 for the entire scale (Spitzer et al., 2006). Both the PHQ-8 and GAD-7 are well validated, widely used, and simply written for clinical populations (see Gilbody, Richards, Brealey, & Hewitt, 2007; Löwe et al., 2008, respectively).

Biological Measure

To measure the biological stress response, each participant provided a small hair sample from the posterior vertex of the head. The samples measured approximately 1–2 cm in length, clipped proximal to the scalp, representing approximately 1–2 months' worth of cortisol levels (Stalder et al., 2012; Wennig, 2000). The examiner selected a 3-mm-diameter hair sample from the posterior vertex of the scalp. Once selected, the examiner verified the location of the sample with the participant. With the participant's agreement on the sample location, the examiner clipped the sample as close to the scalp as possible using a small electric hair trimmer. Each sample was trimmed to 2 cm (scalp end of sample), placed in a small aluminum foil packet for short-term storage, and then stored at room temperature until it was sent to the University of Washington Biobehavioral Nursing and Health Systems Laboratory for processing. Once in the lab, the hair was weighed and then pulverized into a powder. The powder was mixed with 3 ml of methanol in a vial, sealed, and placed in a tube rotator overnight. The now extracted cortisol (contained within the methanol) was removed from the vial and put into a Speed-Vac lyophilizer. The resulting pellet was resuspended in a cortisol buffer (Salimetrics High-Sensitivity Cortisol EIA kit; Salimetrics), and the assay was carried out using the protocol described in the kit. The resulting cortisol level within each sample was determined through extrapolation of sample values based on a standard curve generated with each EIA test kit. Sample values were compared against a reference range for neurologically typical adults (17.7–153.2 pg/mg; Sauve et al., 2007).

Procedure

After the informed consent process, gathering demographic information, and assessment of language comprehension and fields of vision via the CAT (Swinburn et al., 2004), each participant completed six self-report measures: stress (2), depression, anxiety, resilience, and sleep. The text of each measure was read aloud by the examiner; participants were invited to read along silently as well. To control for order effects, the order of measures within the protocol varied; each participant received one of three protocol orders, depending on their alphanumeric study number (assigned randomly). After the completion of the behavioral self-

report measures, each participant provided a hair sample to assess cortisol level. The total time for the testing session was between 90 and 120 min for each participant.

Although modifications improved the ease of comprehension of the PSS, an additional approach was used to improve understanding of each instrument administered as part of the study protocol: the Communicative Support Hierarchy and Independence Rating Scale created by Tucker, Edwards, Mathews, Baum, and Connor (2012). This was used to (a) provide systematic communicative support for each participant to complete each measure and (b) rate the participant's communicative independence throughout each measure's administration. The Tucker Communicative Support Hierarchy and Independence Rating Scale (Tucker et al., 2012) provided specific steps for the examiner to support the participant during test item administration, such as "3. Re-explain the choice scale" and "4. Combine a yes–no question with the scale" (p. 45). The independence scale is a 7-point rating scale that allowed the examiner to rate the level of communicative support necessary to administer the test for each participant. For example, the examiner may rate communicative participation as 1 = *does not produce response with maximal support* to 7 = *responds with no need for additional support* (Tucker et al., 2012, p. 45). Inclusion of the Tucker Communicative Support Hierarchy and Independence Rating Scale allowed for more complete participation of PWA with more severe communication impairment.

Test–Retest Reliability

Approximately 30% of the participants completed a brief (15–30 min) second study session, approximately 1 week following the first session. The examiner readministered the mPSS and the Stress VAS to 21 of the 72 participants (every third participant based on their randomly assigned alphanumeric study number) to gather data for test–retest reliability.

Data Analyses

Pearson correlational analyses were used to address Research Questions 1, 2, and 3. An interclass coefficient correlation was used to address Research Question 4 regarding measurement of test–retest reliability.

Results

This study comprised two phases: the modification of the PSS was carried out in Phase I, and the validation of the mPSS was conducted in Phase II.

Phase I: Modification

For Phase I of the study, three steps were involved in the modification of the PSS: (a) initial modification by two of the authors, (b) review of the initial modifications by two expert panels and subsequent remodification, and (c) administration of the mPSS, related cognitive interviewing with PWA, and subsequent remodification.

In Step 1 of the modification phase, two of the authors (the first and fourth authors) reviewed and modified the instructions, questions, response options, and the scale's overall layout. Specific modifications resulting from this step included presenting the instructions and each

question on the scale in a large font (Times New Roman, 18 point), with instructions and each question on its own page. A simple image of a calendar month to coincide with the phrase “In the last month…” was added to the instructions. Each question started with the same original carrier phrase: “In the last month, how often have you….” The concluding phrase of each question on the scale was simplified. For example, the original Item 4 on the PSS read “In the last month, how often have you felt confident about your ability to handle your personal problems?” and was simplified to “In the last month, how often have you felt unsure about your ability to handle your problems?” Four questions, including this one, were altered in their direction from negative to positive to ease understanding and prevent confusion, as is carried out in national patient-reported outcome measurement initiatives, such as Patient-Reported Outcomes Measurement Information System. At this point in the modification process, response options were kept the same as the original PSS: “never,” “almost never,” “sometimes,” “fairly often,” and “very often.”

Example and practice items were also added to the PSS. Two example questions allowed the examiner to demonstrate and discuss the format of the test. Example questions allowed each participant an opportunity to observe the scale before answering any questions. One example item read, “In the last month, how often have you worn a watch?” For each example question, the examiner would read the question aloud and then point to and discuss his or her selected response using his or her own experience. After example questions, the examiner could move on to two practice items for the participant to complete. The intention of the practice items was to provide an opportunity for participants to engage in the scale before starting the scale's questions. One original practice item at this step read “In the last month, how often have you watched sports on TV?” The examiner and participant could discuss the item and response options to support the participant's general understanding of the questions and the overall framework of the scale.

In Step 2 of the modification, seven experts reviewed and discussed the content and structure of the instrument as part of two review panels. The results of the expert panel reviews included the following input about the mPSS: Panelists suggested and agreed upon emphasizing (in bold) the “thoughts and feelings” part of the instructions. The panels reviewed and discussed the content of the example and practice questions. For example, panel members suggested omitting the example question “In the last month, how often have you run a mile?” and the practice question “In the last month, how often have you watched sports on TV?” The panelists agreed that practice questions, in particular, should focus on thoughts or feelings to mirror the tone of the scale's questions. The panels both discussed in depth the wording of the response options. For example, “very often” was perceived a clearer choice over “always” or “almost always.” Response options were ultimately altered from “never,” “almost never,” “sometimes,” “fairly often,” and “very often” to “never,” “rarely,” “sometimes,” “often,” and “very often.” Furthermore, the panel members discussed pairing the response options with the calendar month image. Panelists suggested using this calendar month image, the same as the one used in the instructions, with added checkmarks in days of the month to represent frequency corresponding to “never,” “rarely,” “sometimes,” and so forth (see Figure 2).

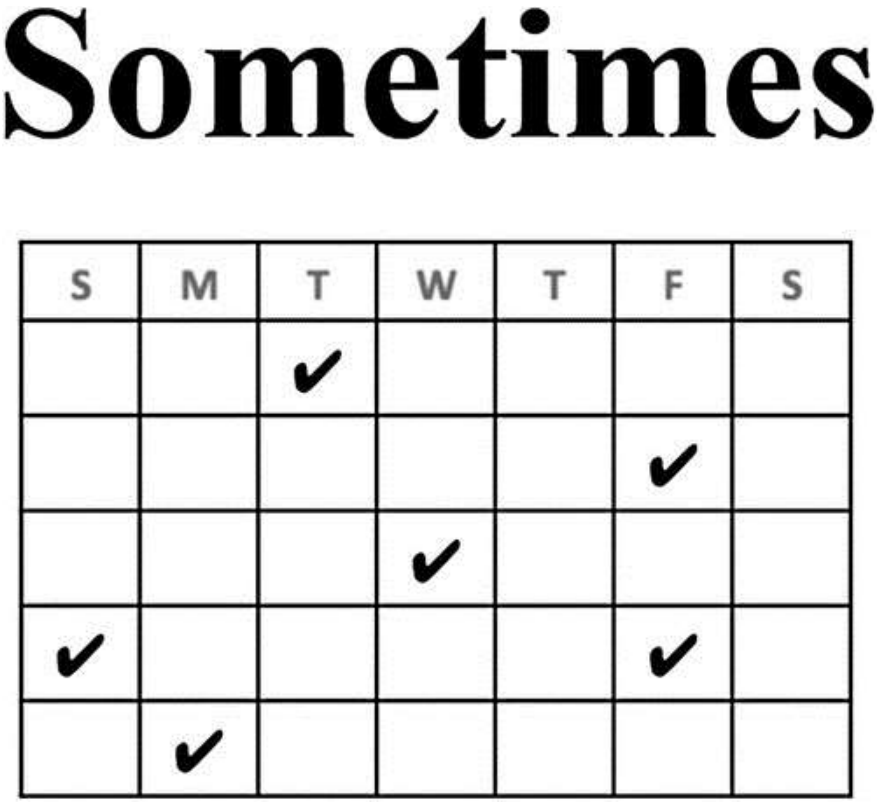

Figure 2. Calendar month graphic used for the response option "sometimes" of the modified Perceived Stress Scale.

In addition, panelists discussed visually separating each question's lead phrase—"In the last month, how often were you…"—from the concluding phrase. The wording of each question was also considered at length. For example, the concluding phrase of Question 2 states "…felt you were unable to control the important things in your life?" Expert panel discussion yielded the alternative phrases "…felt unable to manage the…," "felt things were out of control in your life," and finally "…felt you could not control the important things in your life." Ultimately, feedback from each panel member was reviewed and incorporated into the mPSS by the authors.

In Step 3 of the modification process, the mPSS was administered to nine PWA. Following mPSS administration, the examiner conducted a cognitive interview with each participant. Each cognitive interview was audio-recorded, and the resulting feedback was incorporated into the mPSS: from the wording of the instructions and questions to the response option images, page and paragraph layout, and font size. For example, cognitive interviews yielded modifications such as the orientation of the response options (from vertical to horizontal), the number of checkmarks in the response option calendar month images to represent the response option words, the particular line breaks of several questions (visually shortening phrases in the questions), adding an underline to the key "feeling" word within each question (e.g., "In the last month, how often have you felt you were not on top of things?"), moving the feeling word phrase to the next line, and replacing the word "irritate" with "annoy" in Question 7. Five initial cognitive interviews were conducted. After modifying the items to reflect feedback from the first five interviews, four additional cognitive interviews were conducted. This part of the modification phase concluded when participant feedback no longer contributed any new information about the mPSS, and the mPSS was deemed ready for the next phase: validation. Notably, the Tucker Support Hierarchy (Tucker et al., 2012)

was available for this step of the mPSS modification, but essentially unnecessary for use with these high-level participants.

Phase II: Validation

Phase II results are described related to each research question. See Table 1 for demographic characteristics and Table 2 for study data of each participant.

Table 1. Phase II participant demographic data.

					Comprehensive Aphasia Test		
Pt#	**Age (years)**	**Gender**	**Months postonset**	**Education years**	**Comprehension Subtests**		
					Spoken	**Written**	**Subtotal**
001	72	F	228	17	60	52	112
002	65	F	71	17	59	55	114
003	48	F	39	16	58	58	116
004	72	F	69	18	54	57	111
005	66	F	45	14	62	54	116
006	56	M	31	14	61	62	123
007	66	F	112	12	56	50	106
008	63	M	30	16	60	55	115
009	56	M	94	16	63	60	123
010	67	F	32	18	64	61	125
011	63	M	60	19	33	39	72
012	75	F	18	15	37	39	76
013	64	F	46	15	55	54	109
014	70	F	63	12	53	42	95
015	67	M	23	16	63	54	117
016	66	M	96	13	59	59	118
017	67	F	26	16	50	48	98
019	65	M	15	14	60	46	106
020	79	F	120	20	60	53	113
021	84	F	56	18	58	52	110
022	70	M	45	12	55	47	102
023	68	M	131	16	37	29	66
024	60	M	23	12	37	50	87
025	53	F	92	13	49	45	94
026	61	M	106	14	58	53	111
027	79	F	24	16	63	60	123
028	65	F	168	18	46	24	70
029	70	M	141	16	66	58	124
030	79	M	60	15	54	52	106
031	75	M	63	14	55	48	103
032	56	M	37	16	57	50	107
033	71	M	78	15	63	53	116
034	61	F	132	20	44	35	79
035	78	M	73	13	24	9	33

Table 1. Phase II participant demographic data.

					Comprehensive Aphasia Test		
Pt#	**Age (years)**	**Gender**	**Months postonset**	**Education years**	**Comprehension Subtests**		
					Spoken	**Written**	**Subtotal**
037	71	F	220	16	62	57	119
038	60	F	203	12	55	42	97
039	69	M	128	26	49	41	90
040	63	M	16	14	50	36	86
041	52	F	70	16	56	52	108
042	67	M	124	16	42	43	85
043	79	M	144	18	52	52	104
044	60	M	148	18	47	41	88
045	56	F	75	13	43	42	85
046	65	M	173	12	22	28	50
048	63	M	100	16	51	53	104
049	33	M	114	14	51	38	89
050	64	M	64	16	44	42	86
051	62	M	72	25	58	58	116
052	66	F	153	16	37	41	78
053	59	F	88	14	36	16	52
054	73	M	175	16	55	47	102
055	63	M	139	19	49	53	102
056	55	M	14	16	63	55	118
057	74	M	86	25	66	60	126
058	63	M	116	19	64	61	125
059	79	M	149	25	50	42	92
060	37	M	156	16	61	52	113
061	73	M	48	20	41	36	77
062	70	M	136	13	56	51	107
063	71	M	29	16	55	52	107
064	55	M	22	16	56	51	107
065	46	M	27	14	52	51	103
066	62	M	54	16	54	48	102
067	64	M	34	20	47	54	101
068	84	F	27	19	45	42	87
069	75	M	12	20	44	47	91
070	40	F	61	13	52	40	92
071	57	F	35	12	52	48	100
072	74	F	57	18	62	55	117
073	42	F	38	16	55	47	102
074	40	M	52	12	44	41	85
075	83	F	33	15	52	51	103
Mean	64.53	29 F	81	16.17	52.26	47.63	99.89
(*SD*)	(10.91)	43 M	(54)	(3.16)	(9.45)	(10.13)	(18.65)
Max score					66	62	128

Note. Pt# = Participant no.; F = female; M = male.

Table 2. Phase II participant study data.

Pt#	Hair treatment	Cortisol (pg/mg)	mPSS	Stress VAS	CD-RISC	PHQ-8	GAD-7	Tucker ranking
001		20.00	11	12	35	0	0	7
002		25.00	20	0	18	6	4	7
003		14.29	19	15	19	10	5	7
004	< 8 weeks		17	4	35	3	1	6
005		14.55	15	58	38	3	5	6
006			18	40	25	8	9	7
007		11.25	17	50	24	9	5	7
008		30.00	17	15	28	12	17	7
009	< 8 weeks	30.00	1	0	39	1	1	7
010	< 8 weeks	7.27	16	50	25	6	10	7
011		25.00	15	24	23	6	4	5
012	8+ weeks	13.33	8	17	27	4	0	5
013		8.57	9	35	29	2	3	7
014		8.24	18	1	23	8	6	5
015		28.00	11	10	30	7	4	7
016		11.67	12	20	8	10	10	7
017	< 8 weeks	32.00	13	8	31	2	2	6
019		25.00	19	40	29	8	12	6
020		3.75	17	60	32	7	12	7
021	< 8 weeks	36.36	16	10	33	7	5	7
022		45.45	11	28	22	2	2	5.8
023		30.77	19	20	15	4	10	5
024		8.70	14	30	38	3	2	4.2
025	< 8 weeks	53.31	18	50	23	7	12	6.2
026		28.47	23	70	29	14	12	7
027		5.08	21	15	25	1	3	7
028	8+ weeks	3.45	15	2	35	4	2	5
029		30.00	19	3	36	4	5	7
030		4.00	18	50	24	9	6	7
031		12.70	3	0	37	3	3	7
032		17.50	12	5	25	9	4	7
033			8	3	32	2	0	7
034		5.33	8	8	31	4	1	4
035		38.40	30	75	DNT	17	DNT	3
037		16.67	15	35	33	5	3	7
038	8+ weeks	13.33	11	10	30	10	3	5
039		39.70	12	10	23	2	1	7
040		33.33	17	50	27	10	6	5
041	< 8 weeks	27.50	21	58	18	13	5	7
042		60.00	12	9	DNT	3	4	3
043		26.20	12	5	27	7	6	7
044		30.20	15	12	26	5	7	7
045	8+ weeks	11.20	19	19	29	2	8	5.6

Table 2. Phase II participant study data.

Pt#	Hair treatment	Cortisol (pg/mg)	mPSS	Stress VAS	CD-RISC	PHQ-8	GAD-7	Tucker ranking
046		19.40	10	20	DNT	7	0	
048		10.80	12	13	28	3	3	7
049		19.20	15	40	26	10	5	5.4
050		33.30	10	20	27	11	6	7
051		7.60	23	30	26	8	8	7
052	< 8 weeks	18.30	13	23	35	4	3	7
053		8.40	14	19	DNT	4	2	n/a
054		11.40	13	5	35	3	2	
055		10.00	15	11	35	6	6	7
056		14.40	14	5	26	3	2	7
057		26.96	4	1	36	4	0	6.8
058		22.10	19	25	32	4	7	7
059		9.50	14	25	23	12	7	6.4
060		33.80	28	0	39	7	17	6.4
061		22.77	31	9	36	0	0	7
062		46.41	10	2	32	6	3	6.2
063		73.32	7	5	31	6	2	6.2
064		27.41	24	65	20	9	9	7
065		3.17	19	85	31	6	5	7
066		2.36	3	2	36	1	0	6.4
067		15.98	6	3	34	5	2	7
068		44.75	19	22	28	6	3	5.6
069		20.33	27	14	27	11	5	7
070	< 8 weeks	46.63	14	5	36	7	2	7
071	< 8 weeks	16.98	6	2	34	2	5	6.2
072	< 8 weeks	35.54	7	10	33	5	2	20
073	< 8 weeks	18.88	26	70	18	7	10	6.2
074		3.67	14	25	24	7	3	7
075		15.37	18	28	DNT	3	1	3.8
Mean		22.09	14.96	22.50	28.72	5.92	4.79	6.51
(*SD*)		(14.66)	(6.17)	(21.36)	(6.33)	(3.52)	(3.87)	(1.94)
Max score			40	100	40	30	30	7

Note. Pt# = Participant no.; mPSS = modified Perceived Stress Scale; Stress VAS = Stress Visual Analogue Scale; CD-RISC = Connor–Davidson Resilience Scale; PHQ-8 = Patient Health Questionnaire; GAD-7 = General Anxiety Disorder Scale; DNT = did not test; n/a = not applicable.

Research Question 1 examined the association between mPSS and the biological measure of stress (cortisol found in hair) in 57 of the 72 participants. No significant correlation was found between these variables; in fact, more than 11,000 participants would have been required to detect a significant effect between these variables. Research Question 2 examined the association between the mPSS and the Stress VAS and between the mPSS and the CD-RISC. Analyses found two significant associations between these variables: First, there was a moderate positive correlation between the mPSS and the Stress VAS, $r(70) = .482$, $p < .001$,

and a small inverse correlation between the mPSS and the CD-RISC, $r(70) = -.301$, $p < .05$. In other words, as self-report of stress went up, self-report of coping went down. Research Question 3 examined the association between the mPSS and self-report of depression and anxiety. Analyses found two significant associations between these variables: There were moderate positive correlations for mPSS and the PHQ-8 measuring depression, $r(70) = .408$, $p < .001$, and the GAD-7 measuring anxiety, $r(70) = .520$, $p < .001$, supporting the a priori hypotheses of these research questions. Lastly, Research Question 4 focused on the test–retest reliability of the mPSS. A high degree of reliability was found between the first and second administrations of the mPSS: The average measure interclass correlation coefficient was .900, with a 95% confidence interval from .749 to .959, $F(20) = 10.925$, $p < .001$ (see Figure 3).

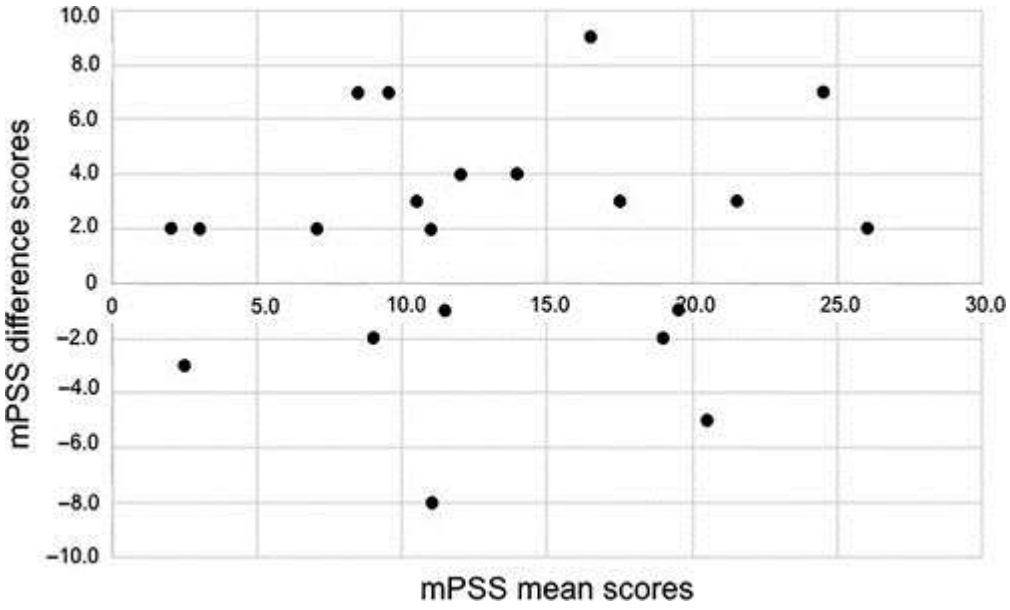

Figure 3. Bland–Altman plot of the test–retest scores for the modified Perceived Stress Scale (mPSS).

Discussion

In this study, we modified the PSS (Cohen & Janicki-Deverts, 2012) using a multistep process and incorporating the input of experts in aphasia. Next, we validated the mPSS with 72 PWA. Overall, the validation phase showed evidence of validity and test–retest reliability. The results of analyses support the convergent validity of the mPSS, as demonstrated by significant correlations between the measure and scales of depression, anxiety, resilience, and a visual analogue scale of stress. There was no apparent association between the mPSS and level of the stress hormone cortisol.

Phase I: Modification

During the modification phase, we used several widely used patient-reported outcome measurement development strategies to identify changes to the PSS that would aid in comprehension and engagement. These changes included simplifying wording of the instructions and questions, finding clear descriptors for the Likert scale response options, providing a graphic representation of each response option, and altering the scale's format

(e.g., font size, one question per page) to help clarify content and minimize distraction on each part of the measure. Many of these changes relied on "expert" opinion—from clinicians and researchers who work with PWA to PWA themselves. Prior research on readability and comprehension of materials for PWA has reported participant preference for relatively shortened phrasing, materials that include pictures or images (Rose, Worrall, Hickson, & Hoffman, 2011), a larger san serif font (e.g., 14-point Verdana), and "generous amounts of white space" (Rose, Worrall, Hickson, & Hoffman, 2012, p. 19). The mPSS addresses most of these readability guidelines. Foremost, the mPSS presents each element of the PSS in simplified language in a large font on a separate page. This formatting style, in particular, provides ample white space to eliminate the distraction of other items or a visually "busy" response option format and allows the respondent to focus only on the question at hand. Interestingly, perceived ease of readability is an important factor in the level of engagement in presented material for people with communicative or cognitive limitations (Rose et al., 2011). Although we did not explicitly ask participants to rate the "overall perceived ease of readability" of the final mPSS, this question was implied throughout the cognitive interviews and all other aspects of the modification phase. However, exploring "overall readability" is an avenue for future research when comparing this measure to other self-report measures used with people with communication limitations.

Phase II: Validation

At this study's inception, we predicted HCC level would correlate to a moderate degree with the self-report of chronic stress and provide evidence of construct validity of the mPSS (Research Question 1) based on prior research (e.g., Kalra et al., 2007). Instead, cortisol levels and mPSS scores showed no association—a finding that fits within a more recent and related meta-analysis (Stalder et al., 2017). Of particular interest, however, is the finding that approximately half of participants in this study showed cortisol levels below the selected reference range published in a report from Sauve et al. (2007). Specifically, 50.8% of this study's participants had HCC levels of < 17.7 pg/mg. In other words, these participants' cortisol levels appeared to be exceedingly low relative to a commonly used range of normal (17.7–153.2 pg/mg; Sauve et al., 2007), even for participants who reported moderate to moderately high levels of chronic stress. This result is interesting when considered with the report from Laures-Gore, Heim, & Hsu (2007), wherein participants with aphasia reported both greater perceived stress and less elevation in cortisol following a linguistic task when compared to the group of neurotypical controls. Taken together, the results from Laures-Gore and colleagues and this study raise questions about the biological correlates of perceived stress for PWA relative to other groups.

Importantly, recent and larger studies of HCCs (Abell et al., 2016; see also meta-analysis by Stalder et al., 2017) conflict with the earlier and widely used reference range by Sauve et al. (2007). Specifically, these more recent reports show a lower range of typical cortisol levels as extracted from hair. Notably, extracting and measuring cortisol from hair is a relatively novel practice, and reported ranges have varied by research site and processing method. Although it is interesting to consider why participants who reported moderate and higher levels of chronic stress had a wide range of cortisol levels, interpreting these results without a local control group is challenging and potentially misleading. Additional research is planned to explore cortisol levels for PWA relative to several control groups. This forthcoming research will help explore the degree of association between behavioral reports of chronic stress and the biological marker cortisol and how these associations may differ for PWA.

Research Questions 2–4 also explored evidence of validity and reliability of the mPSS. First, results of this study found a moderate positive correlation between the mPSS and the Stress VAS (Lesage et al., 2012). This indicates that the mPSS and the Stress VAS appear to be measuring similar constructs and support convergent validity of the mPSS. Prior research has found significant positive correlations between the PSS and the Stress VAS in 360 (Lesage & Berjot, 2011) and 457 (Barré, Brunel, Barthet, & Laurencin-Dalicieux, 2017) neurotypical adults. These results may indicate, according to Barré et al. (2017), that the Stress VAS is a quick and convenient stand-in for more extensive perceived chronic stress measures.

In addition, there appears to be a small but statistically significant inverse correlation between the mPSS and the resilience scale (CD-RISC; Connor & Davidson, 2001). As reported previously (DuBay et al., 2011), as participants report greater perceived stress, they appear to report a lower ability to cope with that stress. Stress and resilience are often discussed as close confederates. For example, Windle (2011) described resilience as a process of successful adaptation to stressors, and it can be observed in effective functioning in the face of adversity or successful recovery from stress. Resilience is believed to comprise a complex combination of personal and neurobiological, social/interpersonal, and community/societal factors that provide a degree of protection from the consequences of chronic stress. Often described as an adaptive and active process, resilience appears to equip an individual with more strategies for coping with stress (Fletcher & Sarkar, 2013; Russo et al., 2012). Resilience may be coupled with a number of individual factors including emotional stability, cognitive flexibility, social support, and conscientiousness (Haglund, Nestadt, Cooper, Southwick, & Charney, 2007; White, Driver, & Warren, 2008). In this study, the measure of resilience, although widely used, has not been adapted to suit the communication needs of PWA. If PWA struggled to comprehend components of the CD-RISC (or any of the scales used in the protocol), the Tucker Support Hierarchy was used to systematically support communication. Ultimately, these results suggest that, although the inverse relationship between reports of chronic stress and reports of resilience is small, it contributes to the convergent validity of the mPSS.

Study results show that the scores on the mPSS correlate with the scores on both the depression and anxiety scales; analyses revealed significant moderate positive correlations between the self-reported level of chronic stress and symptoms of depression and anxiety. The association between stress, depression, and anxiety has been borne out in clinical populations in prior research. For example, previous research reports found significant associations between the PSS and the PHQ scale of depression (Wu & Amtmann, 2013) and the GAD scale of anxiety (Mills et al., 2014). As a psychological impairment hypothetically related to stress adaptation, depression has been noted as a common and disabling challenge that can follow stroke and accompany living with aphasia (Code & Herrmann, 2003; Whyte & Mulsant, 2002). However, studies that focus on poststroke depression often exclude participants with aphasia because of their communication limitations, and at the same time, studies that focus on aphasia often exclude participants who report depression because of depression-related cognitive deficits (Spencer, Tompkins, & Schulz, 1997). Understanding depression and its impact on rehabilitation outcomes in aphasia is almost as little understood as chronic stress or anxiety, and all warrant discussion, best addressed outside the present report (see Hunting Pompon, Smith, Baylor, & Kendall, 2018).

A note about participants' comprehension of the mPSS may be useful to understand this study's results in context. Post hoc analyses revealed, predictably, that there was a significant moderate correlation, $r(70) = .428$; $p < .001$, between scores on the aphasia comprehension

tests (CAT; Swinburn et al., 2004) and the Tucker Independence Rating of Communicative Support (Tucker et al., 2012; see Table 2) for Phase II participants. In other words, participants with greater comprehension ability, as measured by the CAT Comprehension subtests, required less communicative support in their understanding of the self-report measures. However, no associations were apparent when comparing scores on the mPSS to either the CAT Comprehension scores or the Tucker Independence Rankings. Put differently, these results indicate that participants' level of perceived chronic stress using this newly modified measure did not vary depending on severity of comprehension impairment nor degree of communicative support required for completing the self-report measures.

Limitations

This study included several challenges. First, it is widely believed that people with low psychosocial functioning typically do not seek out research opportunities. As a result, this sample was unlikely to be representative of PWA, which limits its generalization. Second, although self-report is the most common way to collect information about perceived stress levels, measuring the attitudes and perspectives by self-report may be prone to the influence of context and the reliability of memory (Schwartz, Vollmer, Lee & the North American Research Consortium on Multiple Sclerosis Outcome Study Group, 1999), as well as difficulties with estimation and self-awareness (Barrett, 2010). Furthermore, although a communicative support hierarchy (Tucker et al., 2012) was used to facilitate understanding and response to each behavioral scale, limits in understanding of these measures may pose a threat to validity of some of these measures—particularly the CD-RISC, which is more linguistically complex than the other scales administered. Next, gathering information on premorbid history of depression and anxiety would have contributed to the interpretation of scores on these psychological measures. In addition, cortisol and the multifaceted system that produces and manages this stress-related hormone are incredibly complex. It would have been helpful to consider other factors that may influence cortisol levels over time, such as body mass index, statin medications, and diagnosis of diabetes (Abell et al., 2016).

Conclusion

In this study, we modified and validated a measure of chronic stress in people with communication limitations such as aphasia. This new tool will allow us to evaluate the level of chronic stress in individuals with aphasia in a way that accommodates limitations in comprehension. Through a process to validate this new measure, we found that, although the mPSS did not correlate with a biological measure of stress, it did correlate significantly with another measure of stress as well as scales describing the related constructs of resilience, depression, and anxiety. These associations are well documented in the literature, lend support for the validity of the mPSS, and point to the potential of the mPSS for clinical use with individuals with aphasia. Although further work is needed to explore the mPSS and how it aligns with the biological stress response system, the results of this study suggest that this aphasia-friendly scale will be a useful tool to explore how chronic stress may influence treatment engagement and response and potentially the arch of recovery for PWA.

Our body is wired to survive and thrive in times of stress, but the sophisticated and multifaceted stress response system may become a barrier to learning and memory if it works overtime. Put succinctly by Mirescu and Gould (2006), “growth inhibition is probably a

reasonable compromise during stress, when energy must be mobilized to insure survival of the organism" (p. 236). Rehabilitation researchers and professionals, in aphasia and across other neurorehabilitation disciplines, are well acquainted with the strain people with aphasia and other acquired impairments may face in day-to-day life. What remains to be uncovered is how these stresses, especially as they are prolonged over time, influence patients' recovery and treatment success.

Acknowledgments

This research was completed with funding from VA Rehabilitation Research & Development Career Development Award (1IK1RX001934). We thank neurologists Kyra Becker and Stephen Nadeau for their thoughtful input at the inception of this project and JoAnn Silkes for her support. We also thank the clinicians, clinical researchers, family members, and especially the participants with aphasia involved with this project for their time and efforts.

References

Abell, J. G., Stalder, T., Ferrie, J. E., Shipley, M. J., Kirschbaum, C.,Kivimaki, M. & Kumari, M. (2016) Assessing cortisol from hair samples in a large observational cohort: The Whitehall II study. *Psychoneuroendocrinology*, 73,148–156.

Aggarwal, N. T., Wilson, R. S., Beck, T. L., Rajan, K. B. Mendes de Leon, C. F., Evans, D. A. & Everson-Rose, S. A. (2013) Perceived stress and change in cognitive function among adults 65 years and older. *Psychosomatic Medicine*,76,80–85.

Alderson, A. L. & Novack, T. A. (2002) Neurophysiological and clinical aspects of glucocorticoids and memory: A review. *Journal of Clinical and Experimental Neuropsychology*, 24(3),335–355.

Bao, A.-M., Meynen, G. & Swaab, D. F. (2008) The stress system in depression and neurodegeneration: Focus on the human hypothalamus. *Brain Research Reviews*, 57,531–553.

Barré, R., Brunel, G., Barthet, P. & Laurencin-Dalicieux, S. (2017) The Visual Analogue Scale: An easy and reliable way of assessing perceived stress. *Quality in Primary Health Care*, 1(1), 1–5.

Barrett, A. M. (2010) Rose-colored answers: Neuropsychological deficits and patient-reported outcomes after stroke. *Behavioral Neurology*, 22,17–23.

Bay, E., Sikorskii, A. & Gao, F. (2008) Functional status, chronic stress, and cortisol response after mild-to-moderate traumatic brain injury. *Biological Research for Nursing*, 10(3), 213–225.

Buchanan, T. W., Laures-Gore, J. S. & Duff, M. C. (2014) Acute stress reduces speech fluency. *Biological Psychology*, 97,60–66.

Bustamante, A., Sobrino, T., Giralt, D., García-Berrocoso, T.,Llombart, V., Ugarriza, I., . . . Montaner, J. (2014) Prognostic value of blood interleukin-6 in the prediction of functional outcome after stroke: A systematic review and meta-analysis. *Journal of Neuroimmunology*, 274(1–2), 215–224.

Cahana-Amitay, D., Albert, M. L., Pyun, S.-B., Westwood, A., Jenkins, T., Wolfard, S. & Finley, M. (2011) Language as a stressor in aphasia. *Aphasiology*, 25(5), 593–614.

Cherney, L. R. & Robey, R. R. (2008) Aphasia treatment: Recovery, prognosis, and clinical effectiveness. In R. Chapey (Ed.), *Language intervention strategies in aphasia and related neurogenic communication disorder* (5th ed., pp. 186–202). New York, NY: Wolters Kluwer-Health/Lippincott Williams & Wilkins.

Christoffel, D. J., Golden, S. A. & Russo, S. J. (2011) Structural and synaptic plasticity in stress-related disorders. *Reviews in the Neurosciences*, 22(5), 535–549.

Code, C. & Herrmann, M. (2003) The relevance of emotional and psychosocial factors in aphasia to rehabilitation. *Neuro-psychological Rehabilitation*, 13(1–2), 109–132.

Cohen, S. & Janicki-Deverts, D. (2012) Who's stressed? Distributions of psychological stress in the United States in probability samples from 1983, 2006, and 2009: Psychological stress in the U.S. *Journal of Applied Social Psychology*, 42(6), 1320–1334.

Cohen, S., Kamarck, T. & Mermelstein, R. (1983) A global measure of perceived stress. *Journal of Health and Social Behavior*,24(4), 385–396.

Cohen, S. & Williamson, G. M. (1988). Perceived stress in a probability sample of the United States. In S. Spacapan & S. Oskamp (Eds.), *The social psychology of health* (pp. 31–67). Newbury Park, CA: Sage.

Connor, K.M. & Davidson, J.R.T. (2001) *Connor–Davidson Resilience Scale (CD-RISC).* Retrieved from http://www.cd-risc.com

Conrad, C. D. (2006) What is the functional significance of chronic stress-induced CA3 dendritic retraction within the hippocampus? *Behavioral and Cognitive Neuroscience Reviews*, 5(1),41–60.

Couper, B. (1999) The application of cognitive science to computer assisted interviewing. In G. B. Willis (Ed.), *Cognition and survey research* (pp. 277–300). New York, NY: Wiley.

Cruice, M., Worrall, L., Hickson, L. & Murison, R. (2003) Finding a focus for quality of life with aphasia: Social and emotional health, and psychological well-being. *Aphasiology*, 17(4),333–353.

Davidson, R. J. & McEwen, B. S. (2012) Social influences on neuroplasticity: Stress and interventions to promote well-being. *Nature Neuroscience*, 15(5), 689–695.

DeWalt, D. A., Rothrock, N., Yount, S. & Stone, A. A. (2007) Evaluation of item candidates: The PROMIS qualitative item review. *Medical Care*, 45(5, Suppl. 1), S12–S21.

DuBay, M. F., Laures-Gore, J. S., Matheny, K. & Romski, M. A. (2011) Coping resources in individuals with aphasia. *Aphasiology*, 25(9), 1016–1029.

Fletcher, D. & Sarkar, M. (2013) Psychological resilience: A review and critique of definitions, concepts, and theory. *European Psychologist*, 18(1), 12–23.

Gianaros, P. J., Jennings, J. R., Sheu, L. K., Greer, P. J., Kuller,L. H. & Matthews, K. A.(2007) Prospective reports of chronic life stress predict decreased grey matter volume in the hippocampus. *NeuroImage*, 35(2), 795–803.

Gilbody, S., Richards, D., Brealey, S. & Hewitt, C. (2007) Screening for depression in medical settings with the Patient Health Questionnaire (PHQ): A diagnostic meta-analysis. J*ournal of General Internal Medicine*, 22(11), 1596–1602.

Gottlieb, A., Golander, H., Bar-Tal, Y. & Gottlieb, D. (2001) The influence of social support and perceived control on handicap and quality of life after stroke. *Aging Clinical and Experimental Research*, 13,11–15.

Haglund, M. E., Nestadt, P. S., Cooper, N. S., Southwick, S. M. & Charney, D. S. (2007) Psychobiological mechanisms of resilience: Relevance to prevention and treatment of stress-related psychopathology. *Development and Psychopathology*,19(3), 889–920.

Hemsley, G. & Code, C. (1996) Interactions between recovery in aphasia, emotional and psychological factors in subjects with aphasia, their significant others and speech pathologists. *Disability and Rehabilitation*, 18(11), 567–584.

Hunting Pompon, R., Smith, A., Baylor, C. & Kendall, D. (2018) *Exploring associations between chronic stress, depression and anxiety in people with aphasia*. Manuscript submitted for publication.

Juster, R.-P., McEwen, B. S. & Lupien, S. J. (2010) Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience & Biobehavioral Reviews*, 35(1), 2–16.

Kalra, S., Einarson, A., Karaskov, T., Van Uum, S. & Koren, G. (2007) The relationship between stress and hair cortisol in healthy pregnant women. *Clinical & Investigative Medicine*,30(2), 103–107.

Kertesz, A. (1982) The Western Aphasia Battery. New York: Grune & Stratton.

Kirschbaum, C., Wolf, O. T., May, M., Wippich, W. & Hellhammer,D. H. (1996) Stress- and treatment-induced elevations of cortisol levels associated with impaired declarative memory in healthy adults. *Life Sciences*, 58(17), 1475–1483.

Kleim, J. A. & Jones, T. A. (2008) Principles of experience-dependent neural plasticity: Implications for rehabilitation after brain damage. *Journal of Speech, Language, and Hearing Research*, 51(1), S225–S223.

Kroenke, K., Spitzer, R. L. & Williams, J. B. (2001) The PHQ-9: Validity of a brief depression severity measure. *Journal of General Internal Medicine*, 16(9), 606–613.

Laures-Gore, J. S. (2012) Aphasia severity and salivary cortisol over time. J*ournal of Clinical and Experimental Neuropsychology*,34(5), 489–496.

Laures-Gore, J. S. & Buchanan, T. W. (2015) Aphasia and the neuropsychobiology of stress. *Journal of Clinical and Experimental Neuropsychology*, 37(7), 688–700.

Laures-Gore, J. S. & Defife, L. C. (2013) Perceived stress and depression in left and right hemisphere post-stroke patients. *Neuropsychological Rehabilitation*, 23(6), 783–797.

Laures-Gore, J. S., Dubay, M. F., Duff, M. C. & Buchanan, T. W. (2010) Identifying behavioral measures of stress in individuals with aphasia. *Journal of Speech, Language, and Hearing Research*, 53,1394–1400.

Laures-Gore, J., Hamilton, A. & Matheny, K. (2007) Coping resources, perceived stress, and life experiences in individuals with aphasia. *Journal of Medical Speech-Language Pathology*,15,423–431.

Laures-Gore, J., Heim, C. M. & Hsu, Y.-S. (2007) Assessing cortisol reactivity to a linguistic task as a mark of stress in individuals with left-hemisphere stroke and aphasia. *Journal of Speech, Language, and Hearing Research*, 50,493–507.

Lesage, F.-X. & Berjot, S. (2011) Validity of occupational stress assessment using a visual analogue scale. *Occupational Medicine*, 61(6), 434–436.

Lesage, F.-X., Berjot, S. & Deschamps, F. (2012) Clinical stress assessment using a visual analogue scale. *Occupational Medicine*,62(8), 600–605.

Levi, M. D. & Conrad, F. G. (1995) A heuristic evaluation of a world wide web prototype. *Interactions*, 3(4), 50–61.

Liston, C., McEwen, B. S. & Casey, B. J. (2009) Psychosocial stress reversibly disrupts prefrontal processing and attentional control. *Proceedings of the National Academy of Sciences*,106(3), 912–917.

Löwe, B., Decker, O., Müller, S., Brähler, E., Schellberg, D., Herzog, W. & Herzberg, P. Y. (2008) Validation and standardization of the Generalized Anxiety Disorder Screener (GAD-7) in the general population. *Medical Care*, 46(3), 266–274.

Lupien, S. J., Fiocco, A., Wan, N., Maheu, F., Lord, C., Schramek,T. & Tu, M. T. (2005) Stress hormones and human memory function across the lifespan. *Psychoneuroendocrinology*, 30(3),225–242.

Manenschijn, L., Koper, J. W., Lamberts, S. W. J. & van Rossum, E. F. C. (2011. Evaluation of a method to measure long term cortisol levels. *Steroids*, 76,1032–1036.

Matheny, K. B., Curlette, W. L., Aycock, D. W., Pugh, J. L. & Taylor, H. F. (1987) *The Coping Resources Inventory for Stress*. Atlanta, GA: Health Prisms.

Mazure, C. M. (1998) Life stressors as risk factors in depression. *Clinical Psychology: Science and Practice*, 5(3), 291–313.

McClung, J. S., Gonzalez Rothi, L. J. & Nadeau, S. E. (2010) Ambient experience in restitutive treatment of aphasia. *Frontiers in Human Neuroscience*, 4,183.

McEwen, B. S. (1998) Protective and damaging effects of stress mediators. *Seminars in Medicine of the Beth Israel Deaconess Medical Center*, 338(3), 171–179.

McEwen, B. S. (2001) Plasticity of the hippocampus: Adaptation to chronic stress and allostatic load. *Annals of New York Academy of Sciences*, 933(1), 265–277.

McEwen, B. S. (2006) Protective and damaging effects of stress mediators: Central role of the brain. *Dialogues in Clinical Neuroscience*, 8(4), 367–381.

McEwen, B. S. & Gianaros, P. J. (2011) Stress- and allostasis-induced brain plasticity. *Annual Review of Medicine*, 62(1),431–445.

McNeil, M. R. & Pratt, S. R. (2001) Defining aphasia: Some theoretical and clinical implications of operating from a formal definition. *Aphasiology*, 15(10–11), 901–911.

Mills, S. D., Fox, R. S., Malcarne, V. L., Roesch, S. C., Champagne,B. R. & Sadler, G. R. (2014) The psychometric properties of the Generalized Anxiety Disorder-7 Scale in Hispanic Americans with English or Spanish language preference. *Cultural Diversity and Ethnic Minority Psychology*, 20(3), 463–468.Mirescu, C., & Gould, E.(2006). Stress and adult neurogenesis.Hippocampus, 16(3), 233–238.

Morgan, S. J., Amtmann, D., Abrahamson, D. C., Kajlich, A. J. & Hafner, B. J. (2014) Use of cognitive interviews in the development of the PLUS-M item bank. *Qualitative Life Research*, 23,1767–1775.

Nadeau, S. E. (2014) Neuroplastic mechanisms of language recovery after stroke. In J. Tracy, B. Hamstead, & K. Sathian (Eds.), *Plasticity of cognition in neurologic disorders*. New York, NY: Oxford University Press.

Parr, S. (1994) Coping with aphasia: Conversations with 20 aphasic people. *Aphasiology*, 8(5), 457–466.

Pittenger, C. & Duman, R. S. (2008) Stress, depression, and neuroplasticity: A convergence of mechanisms. *Neuropsycho-pharmacology*, 33(1), 88–109.

Pruessner, J. C., Hellhammer, D. H. & Kirschbaum, C. (1999) Burnout, perceived stress, and cortisol responses to awakening. *Psychosomatic Medicine*, 61,197–204.

Raymer, A. M., Beeson, P., Holland, A., Kendall, D., Maher, L. M.,Martin, N., . . . Gonzalez Rothi, L. J. (2008) Translational re-search in aphasia: From neuroscience to neurorehabilitation. *Journal of Speech, Language, and Hearing Research*, 51(1),S259–S275.

Rose, T. A., Worrall, L. E., Hickson, L. M. & Hoffman, T. C. (2011) Exploring the use of graphics in written health information for people with aphasia. *Aphasiology*, 25(12), 1579–1599.

Rose, T. A., Worrall, L. E., Hickson, L. M. & Hoffman, T. C. (2012) Guiding principles for printed education materials: Design preferences of people with aphasia. *International Journal of Speech-Language Pathology*, 14(1), 11–23.

Rosmond, R., Dallman, M. & Björntorp, P. (1998) Stress-related cortisol secretion in men: Relationships with abdominal obesity and endocrine, metabolic and hemodynamic abnormalities. *Journal of Clinical Endocrinology & Metabolism*, 83(6),1853–1859.

Russo, S. J., Murrough, J. W., Han, M.-H., Charney, D. S. &Nestler, E. J. (2012) Neurobiology of resilience. *Nature Neuroscience*, 15(11), 1475–1484.

Sauve, B., Koren, G., Walsh, G., Tokmakejian, S. & Van Uum, S. H. (2007) Measurement of cortisol in human hair as a biomarker of systemic exposure. *Clinical & Investigative Medicine*, 30(5),183–191.

Schwartz, C. E., Vollmer, T., Lee, H. & the North American Research Consortium on Multiple Sclerosis Outcomes Study Group (1999) Reliability and validity of two self-report measures of impairment and disability for MS. *Neurology*, 52(1),63–70.

Segerstrom, S. C. & Miller, G. E. (2004) Psychological stress and the human immune system: A meta-analytic study of 30 years of inquiry. *Psychological Bulletin*, 130(4), 601–630.

Spencer, K., Tompkins, C. & Schulz, R. (1997) Assessment of depression in patients with brain pathology: The case of stroke. *Psychological Bulletin*, 122,132–152.

Spitzer, R. L., Kroenke, K., Williams, J. B. & Löwe, B. (2006) A brief measure for assessing generalized anxiety disorders: TheGAD-7. *Archives of Internal Medicine*, 166(19), 1092–1097.

Stalder, T., Steudte, S., Miller, R., Skoluda, N., Dettenborn, L. & Kirschbaum, C. (2012) Intraindividual stability of hair cortisol concentrations. *Psychoneuroendocrinology,* 37(5), 602–610.

Stalder, T., Steudte-Schmiedgen, S., Alexander, N., Klucken, T., Vater, A., Wichmann, S., . . . Miller, R. (2017) Stress-related and basic determinants of hair cortisol in humans: A meta-analysis. *Psychoneuroendocrinology*, 77, 261–274.

Swinburn, K., Porter, G. & Howard, D. (2004) Comprehensive Aphasia Test. Hove, East Sussex: Psychology Press.

Tafet, G. E. & Bernardini, R. (2003) Psychoneuroendocrinological links between chronic stress and depression. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 27(6), 893–903.

Thiel, K. J. & Dretsch, M. N. (2011) The basics of the stress response. In C. J. Conrad (Ed.), *The handbook of stress: Neuro-psychological effects on the brain* (pp. 67–101). Hoboken, NJ: Wiley.

Tucker, F. M., Edwards, D. F., Mathews, L., Baum, C. & Connor, L. T. (2012) Modifying health outcome measures for people with aphasia. *American Journal of Occupational Therapy*, 66,42–50.

Van Eck, M. M. & Nicholson, N. A. (1994) Perceived stress and salivary cortisol in daily life. *Annals of Behavioral Medicine*,16(3), 221–227.

Van Uum, S. H. M., Sauve, B., Fraser, L. A., Morelye-Forster, P., Paul, T. L. & Koren, G. (2008) Elevated content of cortisol in hair of patients with severe chronic pain: A novel biomarker for stress. *Stress*, 11(6), 483–488.

Wennig, R. (2000) Potential problems with the interpretation of hair analysis results. *Forensic Science International*, 107,5–12.

White, B., Driver, S. & Warren, A.-M. (2008) Considering resilience in the rehabilitation of people with traumatic disabilities. *Rehabilitation Psychology*, 53(1), 9–17.

Whyte, E. M. & Mulsant, B. H. (2002) Post stroke depression: Epidemiology, pathophysiology, and biological treatment. *Biological Psychiatry*, 52,253–264.

Willis, G. B., DeMaio, T. J. & Harris-Kojetin, B. (1999) Is the bandwagon headed to the methodological promised land? Evaluating the validity of cognitive interviewing techniques. In G. B. Willis (Ed.), *Cognition and survey research* (pp. 133–153). New York, NY: Wiley.

Windle, G. (2011) What is resilience? A review and concept analysis. *Reviews in Clinical Gerontology*, 21(2), 152–169.

Wu, S. M. & Amtmann, D. (2013) Psychometric evaluation of the perceived stress scale in multiple sclerosis. *ISRN Rehabilitation*, 2013,1–9.

Zuniga, K. E., Mackenzie, M. J., Kramer, A. & McAuley, E. (2016) Subjective memory impairment and well-being in community-dwelling older adults. *Psychogeriatrics*, 16,20–26.