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**STRESS, COGNITION, AND AGING: AN EXAMINATION OF PREDICTORS
AND MECHANISMS**

A Dissertation in
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by

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ABSTRACT

Recent research links exposure to chronic stress with poor cognitive function and decline but these studies have failed to consider how individual differences in subjective or perceived stress are associated with negative cognitive health outcomes. Further, studies on stress and cognitive function are comprised of primarily non-Hispanic white participants precluding considerations of how the observed associations operate among racially and ethnically diverse adults. This dissertation brings together findings from two studies to address these knowledge gaps in the stress and cognitive aging literature.

Using data from a repeated measurement-burst design, Study 1 examined the longitudinal association between perceived stress and cognitive slowing among 116 older adults between the ages of 67 and 96. Bursts of six daily cognitive assessments were repeated every six months over a two-year period with measures of perceived stress provided at the start of every burst. Applying a double-exponential learning model to account for learning effects, two parameters were estimated: 1) asymptotic level (peak performance), and 2) asymptotic change (the rate in which peak performance changed across bursts). A latent growth curve analysis demonstrated that greater reports of perceived stress across the study period were associated with asymptotic cognitive slowing across time.

Using data from a multi-center epidemiological study of Hispanic/Latino adults, Study 2 examined whether the negative stress-cognition association reported in the literature also holds for Hispanics/Latinos ($N = 3,132$) between 45 and 75 years of age. Applying epidemiological data analytic techniques to account for the study's complex sampling design, this study found a negative association between self-reported chronic stress and cognitive performance as well as between self-reported acculturation stress and cognition. Tests of multiple indirect effects showed that negative repetitive thought and depressive affect differentially accounted for the stress-cognition associations.

Overall, this dissertation demonstrates the key role of psychological stress on poor cognitive function and change among middle aged and older adults and shows that Hispanics/Latinos may experience culture-specific stressors that elevate their risk for poor cognitive function. This dissertation also shows that repetitive thought and depressive affect are candidate mechanisms of the association between stress and cognitive function in aging adults.

TABLE OF CONTENTS

LIST OF FIGURES	vi
LIST OF TABLES	vii
ACKNOWLEDGEMENTS	viii
CHAPTER 1. INTRODUCTION	1
1.1 Cognitive Aging	1
1.2 Stress and Cognitive Function	3
1.3 Stress, Cognition, and Aging	4
1.4 Stress, Cognition, and Aging in Hispanic Adults	5
1.5 Negative Repetitive Thought as a Psychological Pathway of Stress and Cognitive Function	6
1.6 Current Studies	8
1.7 References	10
CHAPTER 2. STUDY 1	20
“Global Perceived Stress Predicts Cognitive Change among Older Adults”	
2.1 Introduction	20
2.2 Method	29
2.3 Results	34
2.4 Discussion	40
2.5 References	46
CHAPTER 3. STUDY 2	66
“Stress is associated with Neurocognitive Function in Hispanic/Latino Adults: Results from HCHS/SOL Socio-Cultural Ancillary Study”	
3.1 Introduction	66
3.2 Method	73
3.3 Results	78
3.4 Discussion	81
3.5 References	87
CHAPTER 4. DISCUSSION	105
4.1 Chronic Psychological Stress and Cognitive Function	105
4.2 Negative Repetitive Thought and Depressed Mood as Indirect Pathways between Stress and Cognitive Function	107
4.3 Future Directions	109
4.4 Conclusions	110

APPENDIX A: Stress-Related Terms and their Definitions Derived From the Literature	114
APPENDIX B: Fixed and Random Effects from Double-Exponential Learning Model	115
APPENDIX C: Fixed and Random Effects from Double-Exponential Learning Model	116
APPENDIX D: Reported Severity of Chronic Stress Categories from Study 2	117

LIST OF FIGURES

CHAPTER 1

Figure 1.1: A heuristic model of the stress process adapted from Cohen, Kessler, & Gordon (1995).....	17
Figure 1.2: Pathways linking race/ethnicity and cognitive aging adapted from Glymour and Manly (2008).....	18
Figure 1.3: Adaptation of Gallo and Mathews' (2003) reserve capacity model Stress and Cognitive Function	19

CHAPTER 2

Figure 2.1: Average and predicted response times from dual-exponential learning model. Average and predicted RTs are presented across sessions separated by bursts. RTs are presented in seconds. The y axes for each plot were modified to optimize representation of data.	63
Figure 2.2: Two-factor latent variable model of cognition ($\chi^2(8) = 7.30, p = .50, CFI = 1.00, RMSEA = 0.00$). Standardized parameters are presented and are all statistically significant ($p < .05$).....	64
Figure 2.3: Representation of growth model used to examine the prediction of perceived stress intercept and slope on baseline asymptote and change in asymptote factors. GPS=Global Perceived Stress (denoted by the burst number). Speed = processing speed, WM = working memory, Atten. = attention.	65

CHAPTER 3

Figure 3.1: Illustration of a multiple mediation design in which stress has an effect on neurocognitive performance (direct effect) through depressive affect or repetitive thought (indirect effects) with both indirect effects correlated with each other.	104
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LIST OF TABLES

CHAPTER 2

Table 2.1: Estimated asymptotes and change in asymptotes across bursts.....	57
Table 2.2: Descriptive statistics for observed scores at each burst	58
Table 2.3: Pearson correlation coefficients	59
Table 2.4: Pearson correlations among pooled estimates	61
Table 2.5: Model estimates of GPS predicting baseline asymptote and change in asymptote	62

CHAPTER 3

Table 3.1: Hypothesized factors, items, and resulting standardized loadings from CFA	95
Table 3.2: Weighted sample characteristics (N = 3, 132)	96
Table 3.3: Weighted zero-order and age-partial correlations among stress measures, cognition, and covariates	98
Table 3.4: Weighted multiple linear regression on cognitive tasks regresses onto chronic stress and covariates	100
Table 3.5: Weighted multiple linear regression on cognitive tasks regresses onto acculturation stress and covariates	101
Table 3.6: Individual path and mediation estimates of chronic stress on neurocognitive tasks	102
Table 3.7: Individual path and mediation estimates of acculturation stress on neurocognitive tasks	103

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CHAPTER 1. INTRODUCTION

Cognitive aging research largely converges in the conclusion that there are a multitude of predictors of poor cognitive function that vary between individuals. Stress is an important predictor of cognitive aging in adulthood but the current literature lacks specificity as to which particular component of the stress process is responsible for the reported cognitive decrements. Given the increasing racial and ethnic diversity in the United States, research in cognitive aging will further benefit from determining the applicability of previous research to racial and ethnic minorities. This dissertation aims to extend previous empirical evidence on stress and cognitive aging by addressing critical knowledge gaps in this research area.

1.1 Cognitive Aging

Increasing age in adulthood is associated with declines in functioning in an assortment of cognitive tasks involved in the mechanistic or fluid dimension of intelligence (Baltes, Staudinger, & Lindenberger, 1999; Hertzog & Shing, 2011; Horn & Cattell, 1967). Early theories explained these declines as resulting from age-related decrements in processing resources, such as speed of processing, working memory, inhibitory abilities, and sensory function (Hasher, Zacks, & May, 1999; Lindenberger & Baltes, 1994; Park, 2000; Salthouse, 1996). High correlations between cognitive functioning and sensory mechanisms, such as hearing and visual acuity, gave rise to “common cause” theories of cognitive aging (Baltes & Lindenberger, 1997; Lindenberger & Baltes, 1994). Because age differences in a broad range of functional domains exhibited a high degree of statistical dependence, theorists argued that all cognitive aging effects could be attributed to a small number or even a single common, underlying cause. This “common cause” was proposed to involve age-related decrements in the integrity of brain structure and functioning linked with higher order cognitive functions such as

reductions in white-matter integrity and volume in the prefrontal and medio-temporal regions (Raz et al., 1995; Raz et al., 2005). These forms of changes were considered to be *normative age-graded* influences on development because they are highly correlated with age and similar for all individuals (Baltes & Nesselroade, 1979). *Non-normative* influences, on the contrary, are biological and environmental influences on development that do not occur for everyone and are not strongly correlated with chronological age (Baltes & Nesselroade, 1979).

Subsequent longitudinal investigations questioned the common cause account of cognitive aging by demonstrating that age-related decline in multiple cognitive domains are not as strongly inter-correlated as was suggested based upon cross-sectional findings (Lindenberger & Ghisletta, 2009; Sliwinski, Hofer, & Hall, 2003). There is substantial heterogeneity in rates of cognitive change across multiple cognitive domains, suggesting that there is no one single process driving age-related changes (Wilson et al., 2002). These studies suggested that declines in cognitive function in old age primarily reflect an accumulated influence of a multitude of factors that could be *normative* (e.g., reduced white matter integrity) or *non-normative* (e.g., major life events or Alzheimer's disease; Baltes & Nesselroade, 1979; Sliwinski et al., 2003, Wilson et al., 2002). This is consistent with the lifespan perspective, which proposes that in addition to normative age-graded developmental influences on cognitive change, there are also non-normative influences. Non-normative influences, along with normative age-graded influences, “operate throughout the life course, and their effects accumulate with time, and as a dynamic package, they are responsible for how lives develop” (Baltes, 1987, p. 621). Stress is a non-normative influence on development, as its presence varies across individuals and it is dependent upon an individual's social and psychological context. This dissertation focuses on

individual differences in psychological stress as a regulator of individual differences in cognitive functioning and change in adulthood.

1.2 Stress and Cognitive Function

Theories of stress and health emphasize the adaptive and maladaptive consequences of the stress response (McEwen, 1998a; Selye, 1956; for a list of terms used in this area, see Appendix A). The stress response is adaptive in the short-term, in response to acute stressors, as it helps mobilize energy and increase alertness (Kemeny, 2003). Under optimal conditions, this response is deactivated once the threat has been removed, but it is linked with adverse outcomes if it is activated too often or if it remains activated after the threat has passed (McEwen, 1998b; Smyth, Zawadzki, & Gerin, 2013). Figure 1.1 presents a heuristic model of the stress process by Cohen and colleagues (1995). This model indicates that an environmental demand triggers negative emotional and physiological responses that increase risk of poor health *only* if the demand is perceived to be stressful (Cohen, Kessler, & Gordon, 1995). With chronic or constant perceptions of stress, the emotional and physiological stress response is activated for longer than necessary. This results in allostatic load, defined as the “wear and tear on the body and brain resulting from chronic overactivity of physiological systems that are normally involved in adaptation to environmental challenge” (McEwen, 1998a, p. 38). Chronic stress has been associated with negative mental and physical health outcomes, and more recently, with poor cognitive function and decline.

Biological and psychosocial indicators of chronic stress have both been associated with poor cognitive function and decline. Excess levels of some biological markers, such as glucocorticoids and inflammatory cytokines, are hypothesized to be the end result of chronic stress and have been associated with neuronal death that is in turn linked with poor cognitive

function and cognitive decline (Sapolsky, Krey, & McEwen, 1986; Lupien, McEwen, Gunnar, & Heim, 2009; Yaffe et al., 2003). Some psychosocial indicators of chronic stress linked with poor cognitive functioning include occupying demanding social roles, such as being in a stressful work environment (Andel, Crowe, Kareholt, Wastesson, & Parker, 2011) or providing care for a chronically ill spouse or partner (Vitaliano, et al., 2005), as well as the lingering effects of major life events (Korten, Sliwinski, Comjris, & Smyth, 2014). Although informative, evidence on psychosocial stress and cognitive function is lacking in specificity. This previous evidence addresses the effects of whether or not a person is exposed to demanding psychosocial influences, but individual differences in the perceived stressfulness of these demands are not quantified. Increasing evidence shows that mere exposure to life stress is not predictive of poor cognitive outcomes, but rather that perception of *how stressful* these events are predicts poor cognitive function (Korten et al., 2014; Rosnick, Small, McEvoy, Borenstein, & Mortimer, 2007). Following the stress process model depicted in Figure 1.1, in order to establish a direct connection between stress and cognitive function, it is necessary for subjective or perceived stress to be linked directly with cognitive function.

1.3 Stress, Cognition, and Aging

Some research on emotional reactivity to daily stress suggests that older adults may be affected more by experiences of stress compared to their younger counterparts. Older adults tend to report reduced exposure to everyday stressors, but when they do report a stressor they experience similar stress-related increases in negative affect as their younger counterparts (Mroczek & Almeida, 2004; Stawski, Sliwinski, Almeida, & Smyth, 2008); although this finding has not been consistent across study methodologies (e.g., Uchino, Berg, Smith, Pearce, & Skinner, 2006). Older age is characterized by improved emotional experiences, possibly due to

older adults' ability to reappraise negative situations based on their lived experience, but they may be at equal or greater risk for negative emotional states when they experience chronic stress (Charles, 2010; Sliwinski & Scott, 2014). Examining connections between psychological stress and cognition among adults is critical in order to determining how these associations hold in this developmental stage.

1.4 Stress, Cognition, and Aging in Hispanic Adults

Examining the role of stress on cognitive function in adulthood is essential given the considerable growth in the United States' older population, but the structure of this population is also projected to change such that the proportion of racial minorities will increase compared to the racial non-Hispanic white majority (Wan, Sengupta, Velkoff, & DeBarros, 2005). Because most empirical evidence is derived from primarily non-Hispanic white participants, it is unclear whether relationships between stress and cognitive aging described in the existing literature also apply to racial and ethnic minorities. To address this limitation, this dissertation examines associations among chronic stress and cognitive function in Hispanic adults—a sector of the United States' population projected to comprise the largest proportion of older racial and ethnic minorities by the year 2030 (Wan et al., 2005).

Glymour and Manly (2008) proposed a lifecourse framework that delineates the pathways linking race and ethnicity to poor cognitive function (see Figure 1.2). This model denotes that cognitive function in ethnic and racial minorities is shaped by environmental experiences across the lifespan. According to this model, distal and socially patterned mediators, such as low socioeconomic position, increase “person-specific” mediators that negatively affect cognitive function. Low socioeconomic environments increase risk of exposure to enduring environmental, social, and interpersonal stressors that directly increase racial and ethnic

minorities' risk for adverse cognitive health outcomes. Indirectly, chronic exposure to low socioeconomic environments may wear down a person's ability to physically and psychologically manage stressors increasing their susceptibility to the negative effects of stress. Indeed, Gallo and Matthews's (2003) reserve capacity model postulates that low socioeconomic status (SES) predicts worse health outcomes because of increased stress exposure and limited reserve capacity to manage stressors. According to this model (see Figure 1.3), low-SES individuals are more likely to be exposed to threatening and stressful experiences that have an impact on health outcomes through negative emotions and thoughts (among other mediators). Although this model has yet to be tested in relation to cognitive function, empirical evidence suggests that the same pathways apply with this outcome.

1.5 Negative Repetitive Thought as a Psychological Pathway of Stress and Cognitive Function

Either repeated exposure or prolonged activation of the acute stress response can lead to chronic stress and ultimately, to allostatic load. Continual exposure to the hassles and stressors of everyday life, for example, results in consistent activation of the stress response that can lead to overexposure of stress mediators associated with cognitive decline (McEwen, 1998a). However, individuals exposed to the same number and type of stressors may not be affected the same way. Another way in which acute stress can lead to chronic activation of the stress response is through an inability to terminate the stress response after the stressor has been removed (McEwen, 1998a). The tendency to engage in unconstructive repetitive thought (URT) represents a candidate psychological mechanism through which stress becomes chronic resulting in impaired cognitive function (Brosschot, Gerin, & Thayer, 2006).

The process of thinking repetitively about a negative stressful event even after that event has ceased to exist maintains the stress response activated longer than necessary (Baum, Cohen, & Hall, 1993; Brosschot et al., 2006; Brosschot, Pieper, & Thayer, 2005). This unconstructive form of repetitive thinking is characterized by constructs such as worry and rumination that have been shown to prolong and amplify the affective and physiological stress response. Empirical evidence suggests that URT prolongs physical and emotional reactivity to acute laboratory stress (Brosschot & Thayer, 1998; Gerin, Davidson, Christenfeld, Goyal, & Schwartz, 2006; Glynn, Christenfeld, & Gerin, 2002) and reactivity to daily hassles (Brosschot, Van Dijk, & Thayer, 2007; Moberly & Watkins, 2008; Schlotz, Hellhammer, Schulz, & Stone, 2004). Longitudinally, URT has been associated with adverse cardiovascular health outcomes (Kubzansky et al., 1997) and mental health status (Nolen-Hoeksema & Morrow, 1991). URT may be an important mechanism through which acute stress becomes chronic resulting in negative mental and physical health outcomes. Recent empirical evidence suggests that URT may also be associated with impaired cognitive function.

Repetitive thinking about negative events has been associated with impaired performance in attention-demanding cognitive tasks such as working memory, processing speed, and episodic memory (Klein & Boals, 2001; Stawski, Sliwinski, & Smyth, 2006). Two processes may help explain the effects of URT on cognitive function. First, the attention-depletion hypothesis states that concurrent intrusive thoughts about a negative event impair cognitive performance because these thoughts compete for attentional resources necessary for successful performance in these tasks (Sliwinski et al., 2006). Second, URT about negative events prolongs the emotional and physiological stress response longer than necessary, leading to physiological dysregulation and cognitive decline over time (Baum et al., 1993; Brosschot et al., 2005). Based on these

propositions, intrusive and repetitive thoughts about a negative event may impair cognitive processing by depleting attentional resources, and they may also exacerbate cognitive decline by promoting chronic stress-related to neurological impairment. One study has demonstrated that individual differences in URT among older adults accounts for the link between daily stress reports and self-reported cognitive function (Stawski, Mogle, & Sliwinski, 2013), and another found that this was also the case for objective cognitive function among college students (Klein & Boals, 2001). Additional evidence is needed to corroborate these previous findings on objective cognitive function in middle aged and older adults.

1.6 The Current Studies

The primary goal of this dissertation is to provide further empirical support for the link between psychological stress and cognitive status and change. The two studies that comprise this dissertation critically assess this association by incorporating necessary analytic techniques to explicitly test the proposed hypotheses. In addition to assessing psychological stress-cognition connections, this dissertation aims to examine the role of negative repetitive thought in this link. By incorporating analyses in a racial and ethnic minority sample, this dissertation increases the relevance of results from this area of research to the United States' aging population. The current dissertation aims to meet these goals by testing the hypotheses outlined below:

Hypothesis 1: Greater levels of psychological stress will be associated with cognitive declines in an older adult sample (Study 1).

Hypothesis 2: Increases in psychological stress will be associated with cognitive declines in an older adult sample (Study 1).

Hypothesis 3: Greater chronic stress will be associated with poorer neurocognitive performance in a middle aged and older adult Hispanic/Latino sample (Study 2).

Hypothesis 4: Greater acculturation stress will be associated with lower neurocognitive performance in a middle aged and older adult Hispanic/Latino sample (Study 2).

Hypothesis 5: Unconstructive repetitive thought will comprise an indirect pathway in the association between stress and cognitive function (Study 2).

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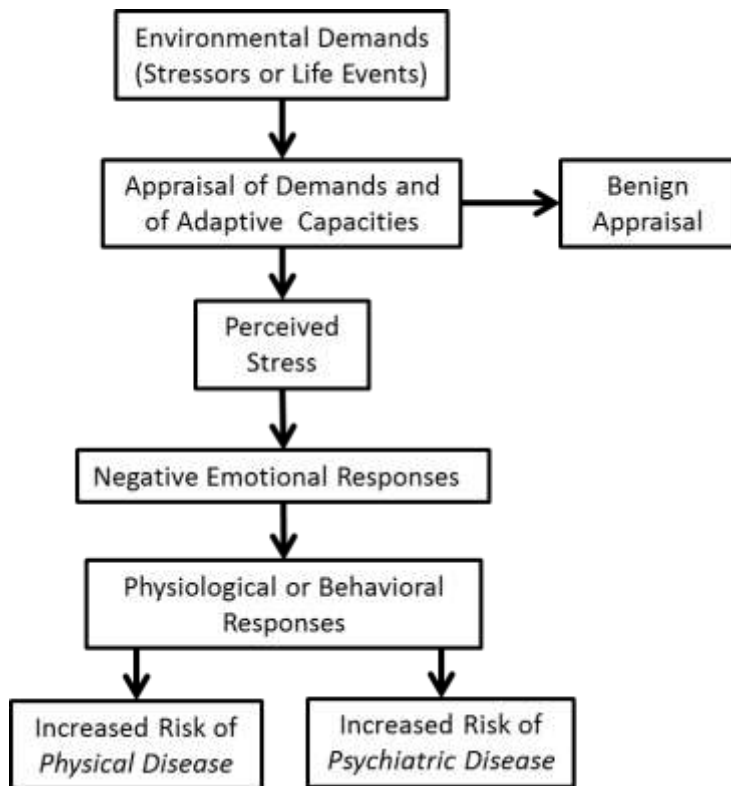


Figure 1.1. A heuristic model of the stress process adapted from Cohen, Kessler, & Gordon (1995).

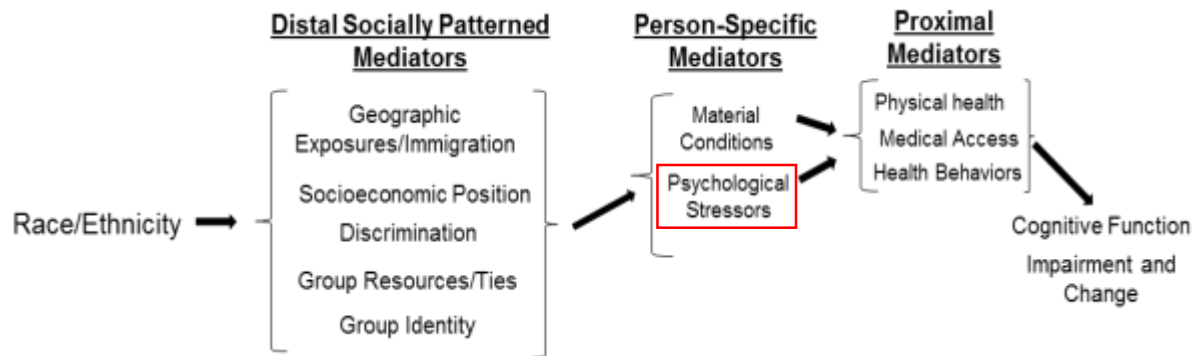


Figure 1.2. Pathways linking race/ethnicity and cognitive aging adapted from Glymour and Manly (2008).

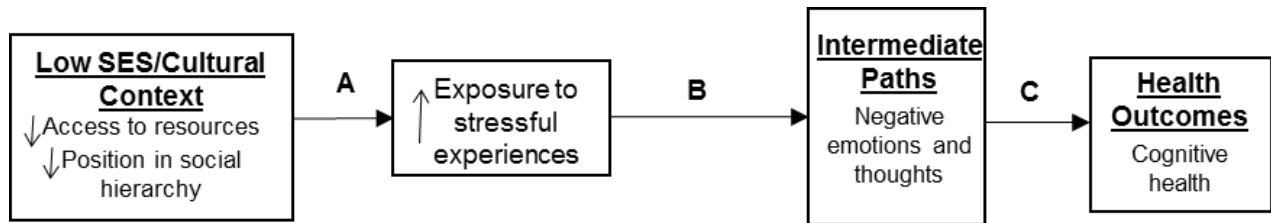


Figure 1.3. Adaptation of Gallo and Mathews's (2003) reserve capacity model with theorized pathways through which low socioeconomic status (SES) results in negative health outcomes (including cognitive health). Arrow A depicts the direct influence of low SES on greater exposure to stressful experiences. Arrow B shows the effects of stress on intermediate pathways hypothesized to predict negative health outcomes (arrow C).

CHAPTER 2. STUDY 1

Global Perceived Stress Predicts Cognitive Change among Older Adults

2.1 Introduction

An important public health goal of cognitive aging research has been to identify modifiable risk-factors of cognitive decline. Identifying factors that contribute to cognitive impairments in old age is imperative for designing interventions that assist people in maintaining their cognitive health and functional independence (e.g., Tomaszewski Farias et al., 2009). Stress represents one potentially modifiable risk-factor of cognitive decline and impairment in old age (McEwen, 2000). Although much research has focused on the biological markers of stress and stressful life events, less is known about the association between the experiential aspects of stress (i.e., perceived stress) and cognition. The purpose of this study is to examine the longitudinal association between perceived stress and cognitive function in older adults.

2.1.1 Stress and Cognitive Function

Two lines of research provide support for the association between stress and cognitive functioning. One line examines putative biomarkers of stress such as cortisol and inflammation. Research in this area suggests that chronic activation of the physiological stress response leads to cumulative “wear and tear” of the brain and body through overexposure to glucocorticoids (e.g., Lupien, McEwen, Gunnar, Heim, 2009; McEwen, 1998) and circulating inflammatory cytokines (e.g., Black, 2002; Yaffe, 2004). Elevated levels of stress biomarkers (e.g., cortisol) are associated with atrophy of brain structures, such as the hippocampus and prefrontal cortex that are essential for cognitive functioning (Lupien & Lepage, 2001). This line of research presupposes that these biomarkers are the end result of events and experiences that elicit psychological stress in individuals, but the psychological component of the stress response is not examined in these studies (e.g., Lupien et al., 1997). Furthermore, examining associations among

biomarkers of stress alone is confounded with their association with other health conditions (e.g., cardiovascular disease; Williamson, Mangos, & Kelly, 2005) that are also associated with impaired cognitive function (e.g., Knopman et al., 2001). It is therefore necessary to examine the effects of the psychological aspects of stress to establish a direct connection between experienced stress and cognitive impairment.

A second line of research has established associations between exposure to stress events and cognitive function in adults. Greater reports of life events or chronic difficulties, for example, are associated with declines in cognitive performance among adults with already compromised cognition (i.e., those who initially exhibited symptoms of mild cognitive impairment; Peavey et al., 2009). Although there have been inconsistent findings in the literature regarding life events and cognitive function (e.g., Comijs, van den Kommer, Minnaar, Penninx, Deeg, 2011), adverse effects of these events on functioning appear to be dependent upon the type of stressor and the perceived implications of the event on an individual's well-being (Cohen, Kessler, & Gordon, 1995; Rosnick, Small, McEvoy, Borenstein, & Mortimer, 2007; Tschanz et al., 2013). This indicates that exposure to events alone does not predict loss of cognitive function.

Although individuals who report experiencing a greater frequency of stressful life events may not exhibit cognitive impairment, they may exhibit poorer cognitive performance at times proximal to reporting having experienced a negative event. For instance, on days when participants reported having experienced a negative stress event, they were more likely to perform worse on attention-demanding cognitive tasks compared to non-stress days and these decrements were more pronounced among older compared to younger adults (Sliwinski, Smyth, Hofer, & Stawski, 2006). Reports of daily negative events have also been associated with memory failures and self-reported changes in memory (Neupert, Almeida, Mroczek, & Spiro,

2006; Stawski, Mogle, & Sliwinski, 2013). Although daily negative experiences may exert immediate effects on cognitive function, long-term ramifications for cognition have not been established. It is possible that the accumulation of these negative experiences have long-term consequences for cognition as has been demonstrated for mental and physical health outcomes (Charles, Piazza, Mogle, Sliwinski, & Almeida, 2013; Piazza, Charles, Sliwinski, Mogle, & Almeida, 2012).

Consistent with this perspective, there is evidence that individuals under chronic stress associated with their social roles are at risk for loss of cognitive function. Individuals who are in a chronically stressful social role, such as an uncontrollable work environment or caring for a family member with Alzheimer's disease, may experience burnout (Maslach, Schaufeli, & Leiter, 2001; Takai et al., 2009). Burnout is a combination of psychophysiological symptoms, including emotional exhaustion, reported as a result of chronic role-related stress (Linden, Keijsers, Eling, & Schaijk, 2005). Indeed, individuals exhibiting symptoms of work-related chronic burnout perform more poorly on tasks of sustained attention as well as immediate and delayed recall compared to controls (Linden et al., 2005; Öhman, Nordin, Bergdahl, Birgander, & Neely, 2007; Sandström, Rhodin, Lundberg, Olsson, & Nyberg, 2005). Middle-aged adults who reported greater job strain, characterized by having low control in ones' occupation, were also at greater risk of cognitive impairment in older adulthood (Andel, Crowe, Kareholt, Wastesson, & Parker, 2011). Caregiving for a spouse with Alzheimer's disease is a source of chronic role stress among older adults that involves exposure to numerous long-term stressors that are often unpredictable and uncontrollable and have been prospectively associated with cognitive decline (Vitaliano et al., 2005).

Overall these studies show that experiences of stress and being in a demanding social role are associated with poorer cognitive function. However, it is likely that not all individuals in a particular social role or who experience a potentially stressful event become psychologically stressed (e.g., Aldwin, Sutton, Chiara, & Spiro, 1996). Chronic exposure to demanding situations and occupying difficult social roles certainly contribute to psychological stress but there are individual differences in how people experience stress in response to similar difficult situations. Additionally, psychological stress can emerge even in the absence of major life events and in persons who do not occupy normatively stressful social roles. Although individual differences in subjective appraisals of stress likely play a critical role in determining the effects of stress, they have seldom been explored in the cognitive aging literature. The current study aims to address this lack of knowledge on the association between individual differences in perceived stress and cognitive function. We examine the longitudinal association between perceived stress and cognitive function in a sample of older adults followed prospectively for two years as part of a measurement burst study (Nesselroade, 1991; Sliwinski, 2008).

2.1.2 Perceived Stress and Cognitive Function

A key component of the stress process is that events increase risk for adverse health outcomes if they are perceived to be stressful (Cohen et al., 1995; Lazarus & Folkman, 1987). Therefore, the psychological perceptions of environmental demands or events are important to examine in relation to health outcomes. Consistent with this view, some evidence indicates that distress resulting from negative events, as opposed to the number of events experienced, has implications for cognitive function. Stawski and colleagues, for example, found that greater affective responses to daily hassles, rather than the frequency of exposure to hassles, were associated with fluid cognitive ability (Stawski, Almeida, Lachman, Tun, & Rosnick, 2010;

Stawski, Mogle, Sliwinski, 2011). These results may indicate that those with greater fluid abilities are better able to regulate their affective reactions to stressors. Stawski's results may also indicate that greater emotional reactions to daily hassles are associated with poorer cognitive abilities. This is in line with Rosnick and colleagues' (2007) findings, which indicated that it was not the total count but the self-reported severity of the events that had implications for task performance.

Perceived stress is a key component of the stress process that triggers emotional, physiological or behavioral responses associated with increased risk of cognitive impairment (Cohen, Kamarck, & Mermelstein, 1983). In a recent longitudinal study, Rönnlund and colleagues examined the association between self-reported stress and cognitive function every five years over three time points among middle aged adults (Rönnlund, Sundström, Sörman, & Nilsson, 2013). Participants responded to one item at each measurement time point: "Do you feel stressed in general?" and were categorized as reporting consistently high levels of stress across each of the measurement occasions or consistently low levels. Responses for the single stress item were consistent with scores in a perceived stress questionnaire such that those who consistently responded yes to the stress item also tended to exhibit greater perceived stress scores on a longer perceived stress measure. Middle aged adults who were categorized as reporting consistently high levels of stress across each of the three assessment occasions, reported greater declines in subjective cognition compared to those who were categorized as reporting consistently low levels of stress (Rönnlund et al., 2013). Among older adults, Aggarwal and colleagues (2014) reported that greater baseline perceived stress was associated with impaired cognitive function as well as with cognitive decline across a six year period.

Consistent with Aggarwal and colleagues (2014), the current study assesses global perceived stress (GPS) measured via the Perceived Stress Scale (Cohen et al., 1983). This scale measures psychological stress by capturing individuals' feelings of overload, unpredictability, and uncontrollability over the previous month (Cohen, Kessler, & Gordon, 1995). We refer to this as "global" perceived stress, because this measure reflects the chronic influence of ongoing life circumstances, concerns about the future, and reactions to events (Cohen et al., 1983).

GPS may result from acute and more common everyday hassles as well as major life events, thus it may be sensitive to the changing demands an individual perceives (Sliwinski, Almeida, Smyth, & Stawski, 2009; van Eck, Nicolson, & Berkhof, 1998). This implies that GPS is not constant or fixed, but will change within people over time to reflect changes in life circumstances and coping resources. Indeed, fluctuations in GPS within people were found to be associated with greater negative affect reactivity to daily events such that older adults tended to report greater negative affect in response to daily events during measurement occasions when they reported greater GPS (Sliwinski et al., 2009). Despite the apparent dynamic nature of GPS, previous work has only examined how GPS status at baseline predicts subsequent change in cognition in older adults (i.e., Aggarwal et al., 2014); no study to date has examined how changes in GPS predict changes in cognitive function. Assessing the extent to which changes in GPS are associated with cognition is important in order to understand the process through which stress affects cognition through fluctuations in perceived demands or chronic and consistent levels of them. In all, it is still unclear what it is about GPS that results in poorer cognitive function. That is, is it the overall amount of burden an individual experiences (i.e., level) or is it the adjustment necessary as a result of increases in environmental demands (i.e., change) that puts individuals at greatest risk for cognitive impairment?

2.1.8 Current Study and Hypotheses

GPS may be associated with poor cognitive function in at least two ways. First, greater levels of GPS may, over time, increase the likelihood of cognitive decline through neurocognitive impairments that result from exposure to stress-related dysregulation of hormones and cytokines such as cortisol and inflammation (Lupien et al., 1998; Sapolsky, Krey, & McEwen, 1986). Second, individuals who experience an increase in their GPS level may display decrements in cognitive function perhaps due to secondary effects of stress such as fatigue, negative mood, and worry (Smyth, Zawadzki, & Gerin, 2013). These two possible pathways lead to two predictions for this study. First, we predict that higher overall levels of GPS reflect chronic or enduring burden and will be associated with greater cognitive slowing in attention, working memory, and speed of processing tasks (Hypothesis 1). Second, we predict that increases in GPS indicate changes in psychological and environmental demands and will be associated with increased rates of cognitive slowing (Hypothesis 2). We account for depressed mood and physical symptoms as they may be potentially confounding variables. Depressed mood is robustly associated with both psychological stress (Öhman, Bergdahl, Nyberg, & Nilsson, 2007; Watson & Pennebaker, 1989; Watson, 1988) and cognitive function (McBride & Abeles, 2000), and health related functional limitations have also been linked with increased perceived stress (Vasunilashorn, Lynch, Gleib, Weinstein, & Goldman, 2014).

Previous studies that have examined the prospective effects of stress on cognition have not considered the influence of potential practice or retest effects (e.g., Wilson, Li, Bienias, & Bennett, 2006). Failure to model retest effects may result in confounding the effects of stress on practice (or learning of the task due to repeated exposure to the stimuli) and its adverse effects on cognition. In the current study, we use a measurement burst design (Nesselroade, 1991;

Sliwinski, 2008) to assess cognition repeatedly (6 assessments) every 6 months for 2 years (30 measurements across 2 years per individual). Practice effects are ubiquitous in longitudinal cognitive research (e.g., Salthouse, Schroeder, & Ferrer, 2004) and may be amplified in intensive repeated measurement designs. Indeed, a previous publication analyzing data from this sample showed retest related performance improvements across sessions within the baseline measurement burst that follow a negative exponential learning function (Sliwinski et al., 2006). Due to the clustered assessments in a measurement burst, conventional approaches to accounting for retest effects, such as dropping the first assessment occasion or statistically adjusting for the number of retest occasions, are not appropriate for addressing retest effects in measurement burst designs (Sliwinski, Hoffman, & Hofer, 2010).

Because measurement bursts consist of clusters of closely spaced measurements (e.g., daily) repeated over longer intervals (i.e., biannually), retest effects likely reflect different processes occurring across these different timescales. Sliwinski and colleagues (2010) described the double-exponential learning model which captures two retest processes: continuous learning and a recovery or ‘warm-up’ effect. Continuous learning effects accrue across all the sessions of a measurement burst; in the case of this present study the maximum number of sessions is 30. However, because there are six month ‘gaps’ between bursts, performance decrements for initial sessions of follow-up bursts might be worse than peak performance after practice during the previous burst. This implies there might be some initial ‘warm-up’ effects during follow-up bursts that reflect a quick return to previous, highly-practiced performance levels, overlaid on continuous processing of learning. This results in a rate of retest improvement during follow-up bursts that is faster than could be predicted by a single learning function. Such warm-up effects

are commonly observed in multisession skill acquisition studies (Newell, Mayer-Kress, & Lui, 2001; Rickard, 2007).

Sliwinski and colleagues. (2010) provided a real life example of the type of learning (retest) effects that are analogous to those one should observe in a measurement burst study. An older adult who takes up cross-country skiing for the first time might display considerable and rapid improvement in her skiing performance during her first season as indexed by the time taken to ski around the lodge. Then spring arrives, the snow melts and 8 months pass before the next snowfall allows her to resume skiing. On her first day out in her second season, she is a bit 'rusty' (out of practice) and a bit slower than she was at the end of the previous season. But after a few skiing sessions, she quickly recovers the skill that was 'lost' during the warmer months and then continues to improve with additional practice. After a prolonged temporal disruption in practice, performance becomes a function not only of the total amount practice, but of how much practice has recently occurred. The double exponential (described more fully in the Analytic Approach to Modeling Retest Effects session) captures the temporal dynamics of clustered bouts of intensive practice (within-bursts) separated by lengthy temporal gaps (between-bursts).

In addition to performance gains attributable to cumulative and recent practice, aging related effects may exert themselves during the interval that separates measurement bursts. To follow the above example, although the older skier is becoming more skilled every season due to her ongoing practice, her potential best speed might be decreasing across seasons because she is aging. To bring this example back to the present study, overt performance on a speeded cognitive task could improve across sessions within bursts, due to the benefit of practice, but that estimate of individuals' latent potential (i.e., the asymptote) could reveal slowing across bursts, due to aging or other long-term processes (such as the cumulative effects of stress). We applied

the double-exponential learning model to obtain estimates of long-term (across burst) declines in asymptotic performance across bursts that are distinguished from short-term (within burst) retest related gains. Therefore, tests of our primary hypotheses involve evaluating whether GPS predicts changes in asymptotic cognitive performance that are not confounded by retest effects.

2.2 Method

2.2.1 Participants

One hundred sixteen older adults from senior residence centers in the Syracuse, NY metropolitan area were recruited for participation in a longitudinal study of health and cognition through advertisements in local newspapers and flyers posted in their residence centers. The average age at baseline was 80.38 years ($SD = 6.40$, range 67-96) and 72% of the sample was female. Participants had on average 14.9 years of education ($SD = 2.40$) and 97% were white, 2% were African American, and 1% Asian American.

2.2.2 Materials

Global perceived stress. We measured global perceived stress using Cohen and colleagues' Perceived Stress Scale (PSS; Cohen et al., 1983). The PSS is a 14-item measure assessing an individual's subjective appraisal of how stressful, overwhelming, and uncontrollable his or her life has been over the past month. Responses to questions such as "In the past month, how often have you felt nervous or 'stressed'?" and "In the past month, how often have you felt difficulties were piling up so high that you could not overcome them?" were made on a 5-point scale (1 = *never* to 5 = *very often*). Positively worded questions were reverse coded, and a total score was obtained by summing the values of all the items, with higher scores reflecting greater levels of perceived stress. Cronbach's alpha for the PSS in this sample was .81.

Depressive symptoms. We used the Center for Epidemiological Studies-Depression scale (CES-D; Radloff, 1977) to assess participants' depressive symptoms. The CES-D is an established measure consisting of 20 items assessing negative mood and depression. Example items include the following: "I feel depressed," and "I am happy". Responses were provided on a 4-point scale (0 = *not at all* to 3 = *very much*) with positively worded items being reverse coded. A total score was calculated by summing the responses on the 20 items, with higher scores indicating greater depressive symptoms. Cronbach's alpha for the CES-D in the current sample was .86.

Health related functional limitations. Participants' physical symptoms reports were assessed daily using a brief version of the Larsen & Kasimatis (1991) physical symptom checklist. This checklist assessed five constellations of symptoms: aches/pain (headaches, backaches, joint pain, and muscle soreness), gastrointestinal symptoms (poor appetite, nausea/upset stomach, constipation/diarrhea), symptoms associated with cardiovascular functioning (chest pain, dizziness, heart pounding), upper respiratory symptoms (cold/flu symptoms, allergy/hay fever symptoms) and a category for "other" physical symptoms or discomforts. Four follow-up questions asked participants whether their physical symptoms limited 1) the amount of time they spent on work or other activities, 2) the extent to which they accomplished less than what they would like, 3) whether they felt limited in the activities they did, and 4) whether they experienced difficulty performing their work or other activities. Participants responded on a three-point scale: "not at all", "slightly", or "very much". Responses were summed across the four items to produce a daily functional limitations score that ranged between 0 to 12 with higher scores indicating greater limitations.

Somatic health. To measure reported somatic health, participants completed the 12-item Health Survey-Short Form (SF-12; Ware, Kosinski & Keller, 1996). The SF-12 assesses the state of individuals' health and limitations they have experienced for a broad range physical and social activities in the past 4 weeks. For the present analysis, we used the physical health component score, which ranges in scores from 0-100 scale, with higher scores indicating greater physical health and fewer health-related limitations.

Number Match. Two variations of Salthouse's (1996) number match–processing speed task were used as indices of processing speed efficiency. In the easy variation, participants had to make a decision as to whether two 3-digit strings were composed of the same numbers or not. Responses were indicated by pressing the “/” key if the digit strings were the same and the “z” key if the digit strings were different. In the difficult variation, participants had to decide whether two 5-digit strings were composed of the same numbers. Responses were indicated in the same manner as stated earlier, and trials were separated by a 500-ms intertrial interval. Thirty-two trials were completed for both the three- and five-item versions of the task. Response times (RTs) for each variation were calculated by averaging across trials on which a correct response was made; RTs served as the dependent variable.

N-Back. A 1-back and a 2-back version of the n-back task (Awh et al., 1996; Smith & Jonides, 1997) were used as markers of working memory performance. Individuals decided whether the currently presented stimulus was the same or different than a stimulus presented one screen back (1-back) or two screens back (2-back). Stimuli (the digits 1 through 9) were presented one at a time in random order in the center of a computer screen. The stimulus digits appeared in white on a black screen. Individuals were instructed to press one of two keys as accurately and quickly as possible indicating whether the current stimuli was the same (“/” key)

or different (“z” key) than the stimulus observed one or two screens back. Stimuli appeared immediately upon a participant’s response; no interstimulus interval (ISI) was included. Half of the trials required a response of same, half required a response of different. Three blocks of 20 items were presented for each version of the n-back for a total of 60 trials for each task. The dependent variables were the RTs for each version of this n-back tasks that were calculated by averaging across trials on which a correct response was made.

Keep-Track. A serial counting task (Keep-Track 1) was used to as an index of simple processing speed efficiency. Participants were presented with one of two geometric shapes (a circle and a diamond) on a computer screen, one at a time, in random order. The task was to count only one of the two objects (the circle) while ignoring the other (the diamond). After a shape was displayed, participants were instructed to press the space bar as quickly as possible after they had counted the shape. A new stimulus would appear immediately after the space bar had been pressed (i.e., no ISI). At the end of each trial participants reported the number of targets they counted. Five trials with between 8 and 14 items per trial were administered (for a total of 60 RTs). The average time to count an object served as the dependent measure, and only RTs from trials on which counting was accurate were included.

The hard version of this task consisted of a variation of Garavan’s (1998) serial-attention task (Keep-Track 2) which was used to assess the accuracy in which individuals could keep separate running counts of two distinct items simultaneously. Participants were presented with one of two geometric shapes (i.e., a rectangle or a triangle) presented in the center of a computer screen, and the task was to press the space bar each time they counted the displayed object. The following item appeared immediately after the space bar was pressed (i.e., no ISI). Count totals

for each shape were reported after each trial ranging between 8 and 16 items in length. Accuracy rates were pooled across trials and were arcsine transformed to normalize the distribution.

2.2.3 Procedure

Participants were given a brief introduction to the study, and the experimenter obtained informed consent as approved by the Syracuse University Institutional Review Board. Participants were told that they were participating in a longitudinal study examining health and cognition in adulthood. Participants were scheduled to visit the research site six times within a 10-day period during which they completed the cognitive tests that were administered by a trained research assistant. These bursts of six daily cognitive assessments were repeated every 6 months for a two-year period, yielding up to five bursts and 30 daily cognitive assessments. Participants completed a GPS and depressive symptoms measure at the start of every burst and they completed a physical symptoms checklist daily within each burst prior to their cognitive assessments. The retention rates were as follows: 78% ($n = 90$) completed all five bursts, 87% ($n = 101$) completed at least four bursts, 88% ($n = 102$) completed three or more bursts and 93% ($n = 108$) completed at least one follow-up burst. Of the 26 participants who missed at least one burst the reason for missing them varied, eight died prior to their next burst, eight dropped out because of serious illness, six canceled appointments because of illness, two canceled appointments because of scheduling conflict, and two moved out of state.

2.2.4 Analytic Approach for Modeling Retest Effects

The following double-negative exponential model, described in detail by Sliwinski, Hoffman, and Hofer (2010), was fit to each cognitive task assessed during the measurement bursts:

$$RT_{ii} = a_i + \Delta a_i(burst_{kj}) + g_i \exp[-r_i(occasion_{ii})] + (Burst_{kj} > 1) \times g_i^* \exp[-r_i^*(occasion_{tki})] + e_{ii}$$

According to this model, response time (RT) for a given individual (i), on a given assessment (t) is a function of the person's initial or baseline asymptotic response time (a_i), the amount by which their asymptote has changed, $\Delta a_i(burst_{kj})$, and two negative exponential learning functions. Learning across all sessions and bursts is accounted for by the term $g_i \exp[-r_i(occasion_{it})]$ where g_i reflects the difference between initial RT and asymptote (i.e., the total gain in performance due to practice), and a second term, $(Burst_{kj} > 1) \times g_i^* \exp[-r_i^*(occasion_{tki})]$ that captures "warm-up" related improvements in each burst subsequent to the first (i.e., $Burst_{kj} > 1$). That is, some of the practice related performance gains are lost between burst and some re-learning or "warm-up" occurs at the beginning of follow-up bursts. The r parameter denotes the rate of practice related improvement across all sessions and bursts, and r^* signifies the rate of "warm-up" related gains at follow-up bursts. PROC NL MIXED (SAS Institute, 2008) was used to fit this double-exponential nonlinear model and we outputted random asymptote (a_i) and delta asymptote (Δa_i) parameters for each individual and for each of the six cognitive tasks. The baseline asymptote (a_i) and change in asymptote (Δa_i) parameters served as the primary outcome variables in the analyses described in the results section.

2.3 Results

2.3.1 Baseline Asymptote and Change in Asymptote

Figure 1 illustrates the sample means for each task on each session across all five measurement bursts (i.e., a total of 30 session means for each task). The fitted curves in each panel were obtained by plotting the average of the predicted RTs across all individuals for each session and burst. Visual inspection of the average RTs across all tasks show practice effects

(faster RTs) across sessions within burst, especially for the baseline burst, as well as some loss of practice gains (slower RTs) during the initial sessions of follow-up bursts. The fitted curves capture both practice related gains as well as the “warm-up” effects at follow-up bursts. In addition to the practice and warm-up effects, parameter estimates indicated that estimates of asymptotic RT increased significantly across bursts for most of the tasks. Table 2.1 shows the average baseline asymptotes, change in asymptotes, and standard errors for each cognitive task obtained by fitting the double-exponential model to the measurement burst data. All fixed and random effect parameter estimates from the double-exponential model are provided in Appendix B. Estimates on Table 2.1 show that asymptotic response time (RT) for the easy version of the speed task (Number Match 3) was 1500 milliseconds at the baseline burst, and that asymptotic RT for this task significantly ($p < .05$) increased (i.e., became slower) by an average of 54 milliseconds per burst. Asymptotic RT for the difficult version of the speed task (Number Match 5) was about 3221 milliseconds at baseline and significantly ($p < .05$) increased by an average of 60 milliseconds per burst. RTs for the working memory tasks exhibited a similar pattern of results, with significant ($p < .05$) slowing of asymptotic RT by an average of 9 milliseconds and 29 milliseconds for the N-Back 1 and N-Back 2, respectively. For the Keep-Track task, only easy version (Keep-Track 1) showed significant asymptotic slowing (7 milliseconds per burst). Although the point estimate of asymptotic change for the difficult version of the attention tasks (Keep-Track 2) was positive (13 milliseconds per burst), this effect did not attain statistical significance ($p = .08$).

To facilitate data reduction described below, person-specific random effects for baseline asymptote (a_i) and change in asymptotic RT (Δa_i) were outputted for each of the tasks. These estimates were then standardized to T-scores with a mean of 50 and standard deviation of 10; our

analyses henceforth will use these standardized asymptote (a_i) and delta asymptote (Δa_i) parameters as the cognitive indicators.

2.3.2 Descriptive Statistics and Correlations

Descriptive statistics for GPS and age are presented on Table 2.2. The average depressive score at baseline was 10.41 ($SD = 7.69$). We averaged participants' total daily functional limitation scores across the six days within each burst to obtain burst-level scores of functional limitation; participants reported an average functional limitation score of 5.46 ($SD = 1.67$) during the baseline burst assessment. Average somatic health at baseline was 44.92 ($SD = 9.77$); Appendix C presents the baseline means and standard deviations of each task. Table 3 presents the correlations between each cognitive estimate (i.e., asymptote, delta asymptote), GPS, baseline age, depressive symptoms, health limitations, somatic health, sex, and education. Age was significantly correlated with greater baseline response time and increases in response time for both processing speed tasks (number match tasks) and the easy version of the working memory task (1-back; r s range from 0.21 to .33, $p < .05$). Age was significantly correlated with increases in response time for the easy version of the attention task (Keep-Track 1; $r = .22$, $p < .05$). Age was also positively, but not significantly, correlated with baseline response times for both Keep-Track attention tasks (r s = .18 and .19, $p < .10$). GPS at each burst was not significantly associated with age at baseline but was positively associated with baseline depressive symptoms (r s range from .41 to .50, $p < .05$) as well as with functional limitations (r s range from .22 to .36, $p < .05$) and somatic health (r s range from -.23 to -.35, $p < .05$). GPS was unrelated to years of education and gender, but sex was associated with cognitive function. Males exhibited faster baseline RTs in the N-Back 1 ($r = -.22$, $p < .05$) and Keep-Track 1 tasks ($r = -.27$, $p < .05$) and exhibited slower cognitive decline in the difficult variation of the processing

speed task (Number Match 5; $r = -.24, p < .05$). GPS at each burst was also more consistently associated with change in asymptote parameters compared to the baseline asymptote parameters.

2.3.3 Data Reduction

To reduce the number of parameters produced from the double-exponential learning models, we first averaged the standardized parameter estimates of baseline asymptote and asymptotic change across the easy and difficult versions of each of the tasks (i.e., number match, n-back, and keep track) producing three asymptote and three change in asymptote parameters. The correlations among the pooled estimates presented on Table 4 show significant intercorrelations among the baseline asymptote scores (r s range from .32 to .49, $p < .05$) and asymptotic change scores (r s range from .25 to .37, $p < .05$). We verified the pattern on intercorrelations by conducting an exploratory factor analysis, which suggested a two factor structure with baseline asymptote scores loading on one factor and change in asymptote scores loading on the second factor. We fit a confirmatory factor analysis to formally assess the fit of this two factor structure presented on Figure 2.2. The first latent factor was comprised by the three baseline asymptote scores and the second factor (labeled as “change in asymptote” in the figure) was comprised by the asymptotic change scores. The two factors were positively correlated with each other ($r = .32, p < .05$) indicating that individuals with slower asymptotic RTs at baseline also experienced more rapid increases in their asymptotic RTs across bursts. These two factors (i.e., baseline asymptote and change in asymptote) were used as our outcomes in the remainder of the analyses.

2.3.4 GPS and Asymptotic Performance

Level and change in GPS. To test our predictions regarding whether both level and change in GPS predicted cognitive change, we first fit a linear growth curve centering data at the

midpoint (i.e., burst 3) to model average level (intercept) and change (slope) in GPS. The intraclass correlation (ICC) of GPS was .68, indicating that GPS scores were relatively stable across bursts. The mean and variance component for the GPS intercept were significant (Intercept = 18.13, Variance = 30.90, $p < .05$). The fixed GPS slope was positive and significant (Slope = 0.12, $p < .05$) showing modest but reliable increases in average GPS across the two-year follow-up period. However, the variance component of the GPS slope was not significant (Slope Variance = 0.06, $p = .30$), indicating that there were no significant individual differences in rates of GPS change. The lack of significance in the slope variance does not necessarily imply that everyone changed at equal rates, but may indicate a lack of power to detect significant individual differences in rates of GPS change¹. In addition to a linear slope, we also explored the possibility for a quadratic slope in GPS that was not statistically significant.

Perceived stress and asymptotic performance. Figure 2.3 provides a schematic of the growth model used to examine whether GPS level predicted baseline asymptote and asymptotic change. We also modeled the association between GPS slope and asymptotic change and accounted for the possible association between lower baseline asymptote to changes in GPS. Residual bootstrapping on 5000 bootstrap samples was used to produce bias-corrected standard errors for tests of statistical significance. An initial model indicated that GPS slope was not predicted by baseline asymptotic performance and that the GPS slope did not predict asymptotic change; this was not surprising given that there was no evidence of reliable individual differences in rates of GPS change. Therefore, subsequent models did not include the GPS slope as an outcome or predictor. Table 2.5 (Model 1) shows results from a model that included average GPS level (i.e., GPS intercept) as a predictor of baseline asymptotic performance and rate of change in asymptotic performance. GPS level was not significantly related to asymptotic

RT at baseline ($B = 0.03 (0.12)$, *ns*), but was positively related to change in asymptotic performance ($B = 0.31 (0.15)$, $p < .05$). These results indicate that those individuals with higher overall stress exhibited more rapid slowing across bursts².

Next, we examined whether the effect of stress level on cognitive slowing remained after adjusting for age, education, sex as well as for baseline levels of depressive symptoms, functional health limitations, and somatic health. We first examined the univariate associations between each of the covariates of interest and baseline asymptotic performance and change in asymptote. Older age was associated with slower baseline performance ($B = 0.27 (0.12)$, $p < .05$) and with greater decline in performance ($B = 0.29 (0.11)$, $p < .05$). Males had faster baseline RTs ($B = -3.49 (1.62)$, $p < .05$) and greater functional limitations were associated with slower RTs at baseline ($B = 0.71 (0.41)$, $p = .08$), though this association was not statistically significant. Poorer somatic health was significantly associated with faster cognitive slowing across bursts ($B = -0.150 (0.07)$, $p < .05$). Education and depressive symptoms were unrelated to baseline asymptote and change in asymptote. We decided to retain these variables as covariates due to their association with GPS. Table 2.5 (Model 2) shows parameter estimates after accounting for demographic variables and baseline depression and health. Older age was significantly associated with more rapid cognitive decline ($B = 0.21 (0.10)$, $p < .05$) and positively (but not significantly) associated with slower asymptotic performance at baseline ($B = 0.22 (0.13)$, $p < .09$). Men exhibited significantly faster asymptotic RTs ($B = -3.21 (1.49)$, $p < .05$), but there were no gender differences in rates of cognitive change. Years of education, depressive symptoms and the health variables were not significantly related either to baseline or rate of change in cognition. GPS remained a significant predictor of increases in the asymptotic slope ($B = 0.27 (0.12)$, $p < .05$) across bursts³. We also examined interactions among demographic and

mental health variables. These supplementary analyses showed that GPS effects on asymptotic change did not varied across age ($B = .01 (.03)$, $p = .97$), gender ($B = .43 (.36)$, $p = .97$), years of education ($B = -.03 (.05)$, $p = .59$), and depressive symptoms ($B = .01 (.02)$, $p = .77$).

Finally, due to the robust conceptual and statistical association among depressive symptom, health, and GPS, we conducted a set of exploratory analyses to determine whether changes in these measures were associated with cognitive change. Neither changes in depressive nor physical symptoms were significantly related to cognitive change.

2.4 Discussion

In this study we examined the prospective association between level and change in GPS and cognitive slowing among 116 adults aged 67 years and older, who participated in a two-year measurement-burst study. In support of our first hypothesis, we found that higher average levels of GPS across the study predicted cognitive slowing in a latent factor of attention, working memory, and speed of processing performance and that this effect remained after accounting for age, education, sex, depressive symptoms, and physical health. Contrary to our second hypothesis, however, changes in GPS did not predict changes in cognitive performance.

2.4.1 Global Perceived Stress and Cognitive Change

Despite the relatively small sample size ($n = 116$) and short follow-up period (2 years), the present study found evidence of significant cognitive slowing on most of the cognitive tasks, and that the rate of decline was positively related to both chronological age and GPS. One reason that we were able to detect significant cognitive slowing was that our sample was relatively old (mean age of 80). Another reason is that our use of an intensive measurement burst design afforded increased precision, resulting from the large number of measurements (up to 30) on each individual, as well as applying a model that allowed distinguishing retest performance gains

from age-related cognitive decline. Specifically, by using a double-exponential learning model which separately modeled practice related gains within bursts and aging related slowing across bursts, we demonstrated significant declines in asymptotic speeded performance on most of the cognitive tasks. Although they present logistical challenges, intensive measurement designs offer potential for addressing some of the important challenges that face longitudinal cognitive aging research, such as accounting for retest effects and sensitive detection of cognitive decline (Sliwinski, 2008; 2011).

The present study is among a few to examine prospective associations between GPS and cognitive performance. Our results are in line with two previous studies that demonstrated a longitudinal association between greater baseline GPS and cognitive decline. Rönnlund and colleagues (2013) examined a decline in self-reported cognition across 15 years and Aggarwal and colleagues (2014) examined declines in objective cognition across a 6 year period. Specifically, Aggarwal and colleagues showed that baseline GPS predicted decline in accuracy performance across various cognitive tasks. Our results extend this previous work in several ways. First, we demonstrated that perceived stress also relates to less efficient (slower) cognitive performance as evidenced by increased reaction time on the attention demanding tasks used in this study. Second, we separately modeled retest effects which allows us to rule out the possibility that stress relates to prospective cognitive decline simply by reducing the ability of individuals to benefit from practice on repeatedly administered cognitive tests. That is, our results indicate that stress predicts cognitive decline independent of retest effects. And third, we examined whether not only level but change in stress predicted cognitive decline.

2.4.2 Global Perceived Stress Level versus Change

We found that average GPS level predicted cognitive decline, whereas increases in GPS did not. This raises the question: why does (average) level but not change in GPS predict cognitive decline? One possible answer is that biannual changes in psychological stress might reflect relatively transient fluctuations that do not exert a substantial cumulative effect on cognitive function. That is, increases or decreases in levels of stress are less important than whether the absolute level of psychological stress is high or low. In contrast, higher levels of GPS may reflect relatively enduring chronic sources of stress that may accumulate overtime and impair cognitive function (e.g., Korten, Penninx, Pot, Deeg & Comjjs, 2014). The relatively high ICC for GPS (.68) implies stable individual differences in stress levels across the study period and is consistent with this interpretation.

Because the measure of perceived stress used in this study asked participants to report on how they perceived their life over “the past month” it is worth considering why individual perceptions of stress during the last 30 days is both stable over time and predicts future cognitive changes. We consider three possible explanations for this question. One possibility is that greater GPS levels reflect enduring influences of a person’s environment. For instance, living in a disadvantaged neighborhood characterized by low socioeconomic status or high levels of violence may promote concerns or worries about an individual’s safety that, if chronic, can contribute to cognitive decline over time (Baum, Garofalo, & Yali, 1999). A second possibility that is specifically related to older adulthood is the experience of bereavement, that entails having to adapt to the loss of a spouse, or caring for a spouse who is chronically ill can increase feelings of stress within individuals that can be enduring and promote cognitive decline (Bonanno & Kaltman, 1999; Pinquart & Sörensen, 2003).

Lastly, higher levels of GPS reports may also reflect individual differences in coping mechanisms that may exacerbate stress responses (e.g., Scott, Sliwinski, & Blanchard-Fields, 2013) and accumulate over time. For example, daily diary studies show that individuals who report greater GPS exhibit greater negative emotional reactivity to daily hassles (van Eck et al., 1998; Sliwinski et al., 2009), which in turn predict long-term negative mental and physical health outcomes (Charles et al., 2013; Piazza et al., 2012), and worse cognitive function (Stawski, Mogle, & Sliwinski, 2013). A measure of personality trait neuroticism was not available and thus not accounted for in this study. Trait neuroticism, operationalized as the tendency to experience emotional distress in previous studies, has indeed been shown to predict cognitive decline over time (Wilson et al., 2005). Aggarwal and colleagues (2014) did account for trait neuroticism and found consistent results with greater GPS level predicting cognitive decline across a six year period. Although emotional distress is a construct that is tightly linked with GPS and should be accounted for, we did account for depressive symptoms which capture the tendency to experience negative mood. We show that individual differences in GPS remained a significant predictor of cognitive decline after adjusting for physical and depressive symptoms. Therefore, we can rule out that the association between GPS and cognitive decline was a by-product of depression or health related functional limitations.

2.4.3 Limitations and Future Directions

Some limitations to this study are worth noting. First, one reason for the lack of association between rates of change in GPS and cognitive decline could include our relatively small sample size and short follow up duration, especially if the longitudinal associations between GPS and cognitive decline are relatively small. However, this lack of association between GPS increases and cognitive slowing argues against the possibility that decreases in

cognitive abilities may lead to increases in feelings of stress and vice versa. Second, our study included only six assessments of cognition within each burst which may have reduced the precision of estimate asymptotic performance. Third, our failure to detect differential sensitivity of stress effects across different cognitive domains could be due to the considerable amount of shared variance among our tasks given the speeded nature of these tasks. Thus, we cannot conclude that there is equivalence across perceptual speed and working memory, for example. Future studies considering both accuracy and response time across various tasks will be informative in determining differential sensitivity of stress on cognitive domains. Lastly, we did not examine local effects of GPS within each burst. A previous study examining effects within and across bursts found that participants experienced more negative affect during burst in which they reported GPS scores that were greater than their average (Sliwinski et al., 2009). Although such analyses were beyond the scope of our study, future studies should examine whether similar within person effects of GPS on cognition exist in this and other measurement-burst studies.

Other future directions include an analysis of the time course through which GPS may change and exert negative effects on cognitive function. It is possible that GPS changes at a slower pace compared to asymptotic performance. Studies that incorporate multiple time-scales (e.g., across decades, years, months, weeks, days), may thus be necessary to facilitate understanding of the time course through which changes in stress may impair cognitive function. Our sample of participants was relatively homogeneous consisting of well-educated, white female participants residing in senior residence centers, which does not allow for a generalization of results to more racially and socioeconomically diverse individuals. In supplementary analyses we found non-significant moderation of the link between GPS level and cognitive slowing by gender and years of education which was not surprising given the sample's homogeneity. Future

studies that incorporate more socioeconomically diverse participants should examine the role of social and demographic moderators of the stress-cognition link.

Identification of modifiable indirect pathways through which perceived stress relates to cognitive function represents an additional future direction. For example, stress-related cognitive interference has been linked with poorer cognitive performance among older adults (Stawski, Sliwinski, & Smyth, 2006). Experiencing intrusive thoughts related to a recent negative event, may momentarily impair cognitive performance by occupying attentional resources (e.g., Sliwinski, Smyth, Hofer, & Stawski, 2006). Over time, a tendency to experience these form of thoughts (i.e., stress-related intrusive and repetitive thoughts), may contribute to chronic stress by prolonging the stress response and result in impaired cognitive function (Brosschot, Gerin, & Thayer, 2006). Individual differences in self-regulatory processes, such as levels of perceived control and mastery may also comprise an important indirect pathway. Greater GPS levels may reduce feelings of control and mastery that may promote cognitive decline possibly due to reduced engagement in health-promoting behaviors such as poor sleep and decreased participation in physical activities (Ballis, Segall, Mahon, Chpperfield, & Dunn, 2001; Lachman & Weaver, 1998).

2.4.4 Conclusion

In spite of its limitations, the present study is the first to demonstrate a prospective association between perceived stress and cognitive slowing. These results were held after controlling for demographic, mental, and physical health variables. The unique association between perceived stress and cognitive slowing emphasizes the utility of measuring perceptions of stress to complete assessments of mere exposure to negative events.

2.5 References

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Footnotes

¹A likelihood ratio test showed that model fit was not statistically different ($p = .23$) between a full model and a model constraining the slope variance to zero.

²It is worth mentioning that the analyses presented on table 2.5 were also conducted while centering the GPS observations at baseline and that the pattern of results did not change—GPS intercept at baseline, and not slope, predicted more rapid cognitive slowing indicating a true prospective association between greater GPS baseline level and cognitive change overtime.

³We examined whether there was differential prediction of GPS intercept on asymptotic slope for the multiple cognitive tasks by fitting a series of models which tested unique paths from stress to each of the cognitive tasks. None of these unique paths were significant, indicating that none of the tasks were differentially sensitive to GPS over and above the shared variance among them.

Table 2.1

Estimated asymptotes and change in asymptotes across bursts

	Baseline Asymptote		Change in Asymptote	
	Estimate	(SE)	Estimate	(SE)
NM3	1500.42	(48.21)*	53.59	(7.44)*
NM5	3220.55	(96.90)*	60.29	(13.22)*
NB1	832.18	(29.71)*	9.06	(4.20)*
NB2	1289.86	(66.14)*	28.69	(10.49)*
KT1	581.61	(20.57)*	6.89	(3.11)*
KT2	984.55	(37.25)*	12.91	(7.34) [†]

Note. Estimates in milliseconds. WM = Working Memory; Atten. =

Attention; NM3 = Number Match 3; NM5 = Number Match 5; NB1=

N-Back 1; NB2 = N-Back 2; KT1 = Keep-Track; KT2 = Keep-Track 2.

*p<.05. [†]p<.10

Table 2.2

Descriptive statistics for observed scores at each burst

	Burst 1	Burst 2	Burst 3	Burst 4	Burst 5
GPS (<i>n</i>)	116	105	104	97	92
<i>M</i>	17.26	18.44	18.77	18.41	18.15
<i>SD</i>	6.68	7.29	7.37	7.12	7.38
Age (<i>n</i>)	116	106	105	97	92
<i>M</i>	80.33	80.66	81.19	81.57	82.08
<i>SD</i>	6.43	6.22	6.20	6.16	6.16

Note. GPS = Global Perceived Stress

15. NB2 (Delta)	.01	.18 [†]	-.05	.08	-.03	-.10	-.01	.10	.04	.11	.01	.27*	.31*	.28	-								
16. KT1 (Delta)	.20*	.21*	.21*	.20*	.18 [†]	.09	.12	.16 [†]	-.06	.14	.14	.25*	.23*	.26*	.14	-							
17. KT2 (Delta)	.08	.14	.09	.06	.10	.17 [†]	.23*	.32*	.04	.33*	.23*	.25*	.18	.43*	.14	.66*	-						
18. Age (T1)	.13	.02	.08	.13	.14	.26*	.33*	.21*	.08	.18 [†]	.19 [†]	.29	.21	.33	-.03	.22*	.12	-					
19. Educ. (T1)	.00	-.04	-.01	.01	-.03	-.06	.06	-.03	.08	.09	.05	-.17 [†]	-.16	.06	.00	.11	.05	-.12	-				
20. Sex (0 = fem)	.04	-.15	-.11	-.04	-.10	-.07	-.11	-.22*	-.15	-.27*	-.17 [†]	-.17 [†]	-.24*	.03 [†]	-.10 [†]	-.06 [†]	-.03 [†]	-.18 [†]	.12	-			
21. Dep. (T1)	.50*	.43*	.46*	.41*	.49*	-.09	-.08	-.10	-.01	-.18 [†]	-.08	.07	.13	-.02	.09	.14	-.01	.05	-.07	.00	-		
22. Funct Lim (T1)	.32*	.36*	.22*	.29*	.29*	.12	.14	.14	.12	.07	.16	.05	.08	.06	.09	.14	.00	.13	-.04	.11	.26*	-	
23. Som Hlth (T1)	-.24*	-.27*	-.35*	-.23*	-.20 [†]	-.06	-.05	-.14	.00	-.16	-.18	-.10	-.21*	-.17 [†]	-.10	-.22*	-.08	-.32*	.04	.06	-.21*	-.45*	-

Note. GPS = Global Perceived Stress; Asymp = Asymptote; NM=Number Match; NB=N-Back; KT=Keep Track; Educ. = Education; Dep. = Depressive Symptoms; Funct Lim = Functional Limitiations; Som Hlth = Somatic Health.

* $p < .05$. [†] $p < .10$.

Table 2.4

Pearson correlations among pooled estimates

	1	2	3	4	5	6
1. Number Match (Asymptote)	-					
2. N-Back (Asymptote)	.32*	-				
3. Keep Track (Asymptote)	.47*	.49*	-			
4. Number Match (Delta)	.10	-.06	.10	-		
5. N-Back (Delta)	.00	.04	.10	.37*	-	
6. Keep Track (Delta)	.21*	.07	.20*	.26*	.25*	-

Note. Speed = Processing Speed; WM = Working Memory.

* $p < .05$.

Table 2.5

Model estimates of GPS predicting baseline asymptote and change in asymptote

	Model 1		Model 2	
	Baseline Asymptote	Change in Asymptote	Baseline Asymptote	Change in Asymptote
	Estimate (SE)	Estimate (SE)	Estimate (SE)	Estimate (SE)
GPS Intercept	0.03 (0.12)	0.31 (0.15)*	-0.06 (0.14)	0.27 (0.12)*
Age			0.22 (0.13) [†]	0.21 (0.10)*
Sex			-3.21 (1.49)*	-1.34 (1.31)
Education Years			0.27 (0.23)	-0.04 (0.24)
Depressive Symptoms (Baseline)			-0.05 (0.11)	-0.03 (0.65)
Functional Limitations (Baseline)			0.73 (0.52)	-0.13 (0.65)
Somatic Health (Baseline)			-0.02 (0.08)	-0.06 (0.07)

Note. GPS=Global perceived Stress. Sex, 0 = female, 1 = male. Residual bootstrap sample size = 5,000.

* $p < .05$. [†] $p < .10$.

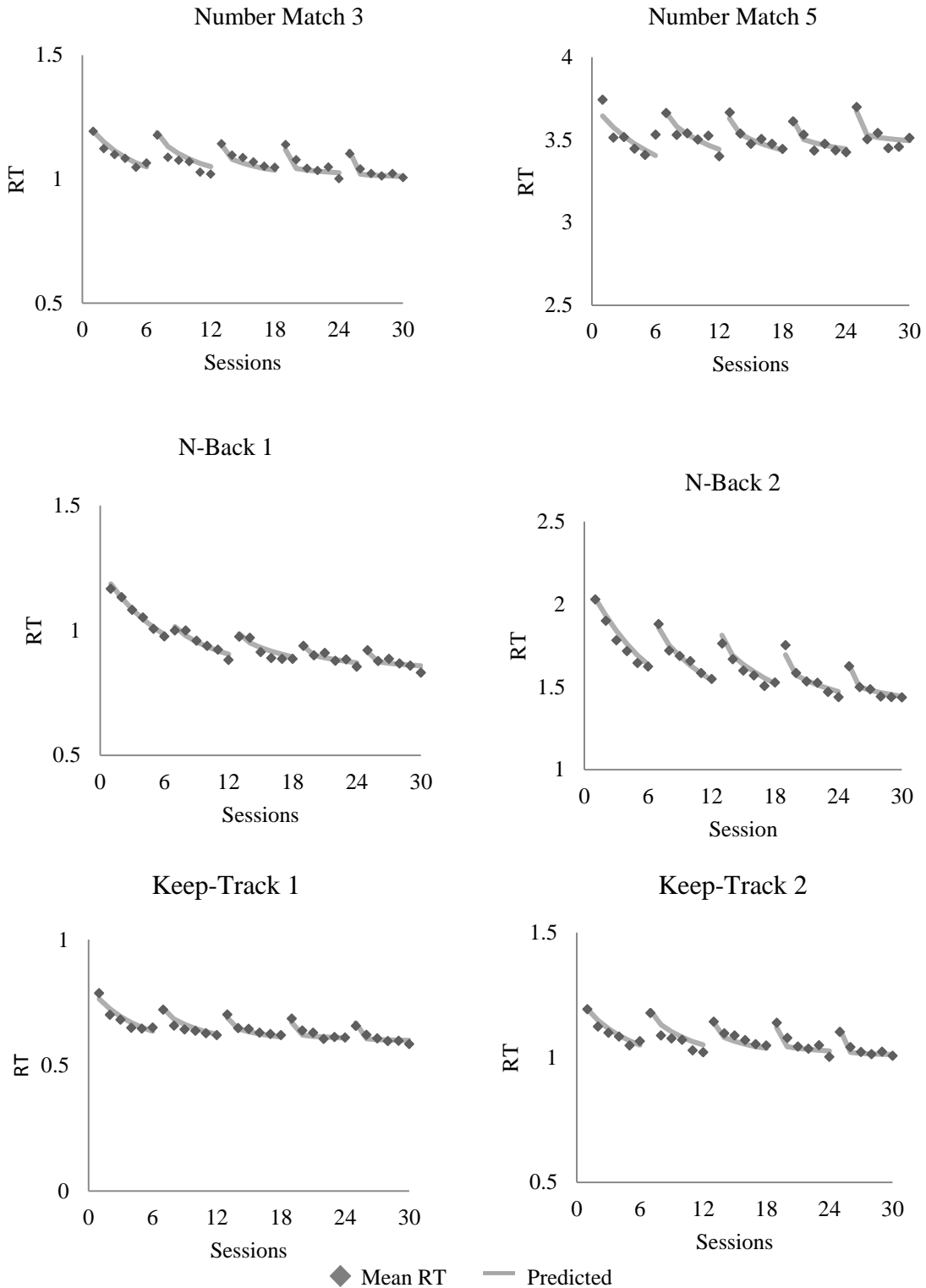


Figure 2.1. Average and predicted response times from dual-exponential learning model. Average and predicted RTs are presented across sessions separated by bursts. RTs are presented in seconds. The y axes for each plot were modified to optimize representation of data.

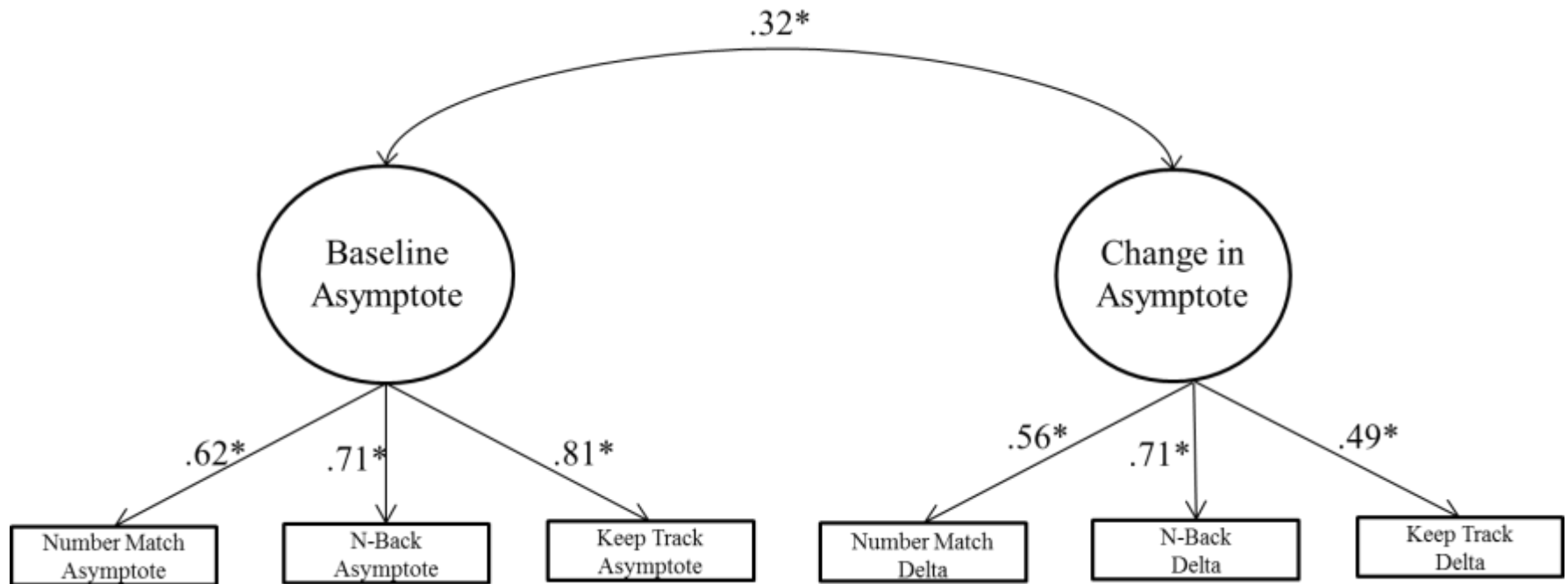


Figure 2.2. Two-factor latent variable model of cognition ($\chi^2(8) = 7.30$, $p = .50$, CFI = 1.00, RMSEA = 0.00). Standardized parameters are presented and are all statistically significant ($p < .05$).

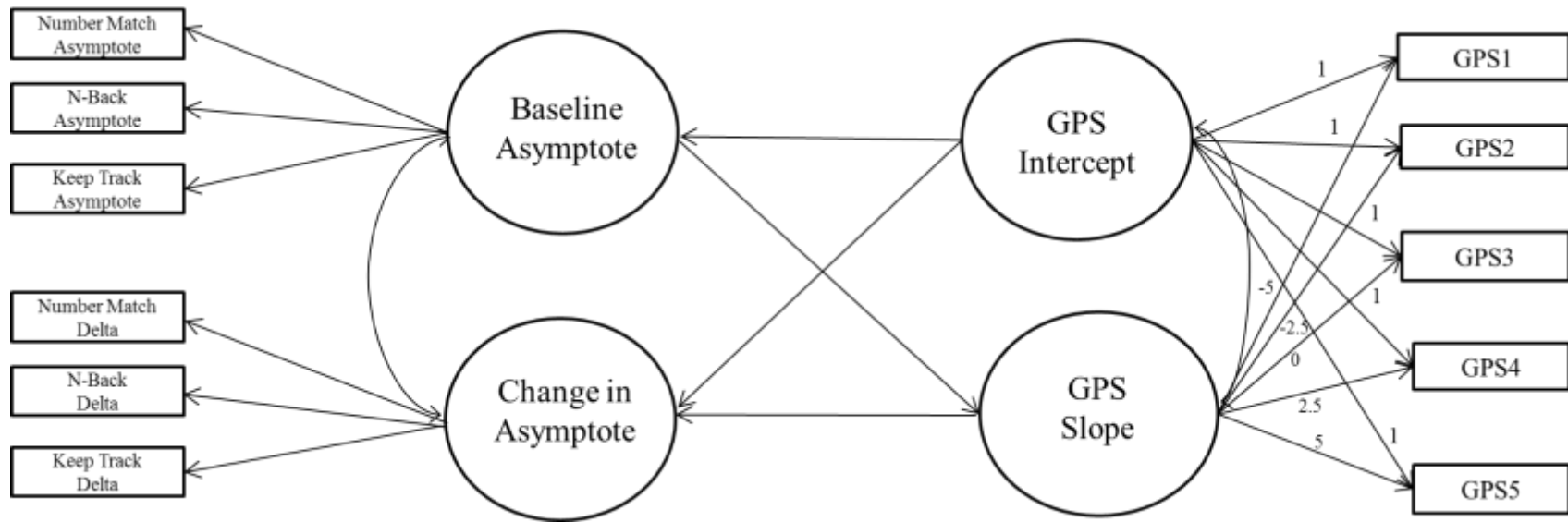


Figure 2.3. Representation of growth model used to examine the prediction of perceived stress intercept and slope on baseline asymptote and change in asymptote factors. GPS=Global Perceived Stress (denoted by the burst number). Speed = processing speed, WM = working memory, Atten. = attention.

CHAPTER 3. STUDY 2

Stress is associated with Neurocognitive Function in Hispanic/Latino Adults: Results from HCHS/SOL Socio-Cultural Ancillary Study

3.1 Introduction

The United States' older population is increasingly becoming more diverse and Hispanics are projected to become the largest older minority in the nation. This demographic shift highlights the need for understanding risk factors for diminished neurocognitive function among Hispanic adults. Accumulating evidence identifies stress as an important risk factor for poor neurocognitive function but studies have yet to determine how this risk factor operates in the Hispanic population. We examined the association between stress and neurocognitive function in a cohort of Hispanic adults and examined depressive affect and repetitive thought as indirect pathways that may account for the stress-neurocognitive function link.

3.1.1 Increasing Diversity of the U.S. Aging Population

It is recognized that the population of older adults in the United States is booming, but it is less often acknowledged that this older population is also becoming more diverse. In 2003, non-Hispanic whites accounted for nearly 83% of the U.S. older (65 years and over) population, followed by 8% blacks and 6% Hispanics. Projections indicate that by 2030 the composition of the older population will be 72% non-Hispanic white, 11% percent Hispanic, and 10% black (Wan, Sengupta, Velkoff, & DeBarros, 2005). Whereas the numbers of the older non-Hispanic white population will be decreasing, the number of older minority individuals will be increasing with older Hispanics becoming the largest older minority. This highlights the need to obtain comprehensive evaluations of factors that may influence the health in this sector of the population.

It is particularly important to understand correlates of health in the Hispanic population because they are likely to undergo unique experiences compared to their counterparts. For example, Hispanics have the lowest educational attainment rates compared to their non-Hispanic white and black counterparts (National Research Council, 2006). This lack of social capital positions Hispanics among the lowest rungs of the occupational spectrum—a factor that can have long-term consequences for their economic and health prospects in older age. Additionally, the “Hispanic Paradox” whereby despite their low social status and economic adversity, Hispanics tend to have lower all-cause mortality rates compared to their counterparts (Franzini, Ribble, & Keddie, 2001; Markides & Coreil, 1986) suggests that different risk processes may be at play in this sector of the population. Due to this difference in social capital and health outcomes, it would be misguided to apply our current knowledge of risk factors of health to Hispanics adults given that most empirical evidence stems from primarily non-Hispanic white participants. The current study aims to establish whether stress—a known risk factor for adverse health outcomes—functions similarly as a risk factor among Hispanic/Latino adults. We focus on neurocognitive health because it is a domain essential for functional well-being in adulthood and old age.

3.1.2 Neurocognitive Function in Hispanics/Latinos

Neurocognitive health is imperative for adults’ ability to retain functional independence and maintain engagement in the workforce and social environments (Krueger et al., 2009). Even before the upcoming demographic changes are apparent, Hispanic adults already tend to perform more poorly on neurocognitive assessments compared to non-Hispanic whites. Generally, studies find that the prevalence of poor neurocognitive function and dementia is higher for Hispanics than non-Hispanic whites (Gurland et al., 1999; Mulgrew et al., 1999; Perkins et al., 1997).

Hispanics tend to score lower on the Mini-Mental State Examination (O'Bryant et al., 2013) and they tend to perform more poorly on domain specific task such as memory, verbal fluency, as well as in executive and visual spatial functioning tasks (Weissberger, Salmon, Bondi, & Gollan, 2013). These associations hold after accounting for age and education suggesting that there may be additional non-demographic risk factors operating in these group differences.

3.1.3 Stress and Neurocognitive Function

Prolonged or chronic stress is associated with poor neurocognitive function and cognitive decline. Physiological markers of persistent or chronic stress, such as elevated cortisol and inflammation, as well as psychosocial markers of stress are associated with impaired neurocognitive function and decline (Lupien et al., 1994). Psychosocial stressors such as perceived stress (Aggarwal et al., 2014), major life events (Rosnick, Small, McEvoy, Borenstein, & Mortimer, 2007), and occupying demanding and stressful social roles, such as being in a stressful work environment (Andel, Crowe, Kareholt, Wastesson, & Parker, 2011), are associated with poorer neurocognitive function. Most of these studies have been carried out among primarily non-Hispanic white individuals and it remains unclear whether stress functions similarly as a risk factor for neurocognitive impairment among racial and ethnic minority groups. One exception is a study by Aggarwal and colleagues who examined the association between perceived stress and cognitive function in a sample that was 65% black and 35% white and found that there were no race differences in the negative association between perceived stress and neurocognitive function between black and white adults (Aggarwal et al., 2014). However, given the reported earlier mortality rates among black adults (Williams, Yu, Jackson, & Anderson, 1997), it is likely that survival effects influenced this result and the possibility exists that this study sampled minority individuals who were sufficiently healthy to live into older adulthood

and were able to participate in the study. It is therefore necessary to examine the stress-neurocognitive function association starting at midlife. To our knowledge, studies have also yet to examine the stress-neurocognitive function association in Hispanic adults to determine whether the same pattern of associations hold in this group as it does for non-Hispanic white and black adults. The proposed research will examine this association beginning at age 45 to age 75 in a national cohort of Hispanics.

3.1.4 Stress and Cognitive Function in Hispanic Adults

It is important to examine the associations between stress and neurocognitive function among Hispanic individuals as they may be more vulnerable to the negative effects of chronic stress compared to their non-Hispanic white counterparts. For example, due to their minority and low socioeconomic status, Hispanic individuals have an increased risk of exposure to enduring environmental, social, and interpersonal stressors that directly increase risks for adverse neurocognitive health outcomes (e.g., Geronimus, 1992). Indirectly, chronic exposure to low socioeconomic status (SES) environments may wear down a person's ability to physically and psychologically manage stressors increasing their susceptibility to the negative effects of stress.

In fact, Gallo and Matthews (2003) propose that low SES predicts worse health outcomes because of increased stress exposure and limited reserve capacity to manage stressors. Although this model has yet to be applied to neurocognitive health, empirical and theoretical evidence suggests that the same pathways may account for racial and ethnic disparities in neurocognitive health. According to the reserve capacity model (Gallo & Matthews, 2003), due to their low SES status, Hispanic individuals are more likely to be exposed to threatening and stressful experiences that have an impact on health outcomes through negative emotions and cognitions (among other mediators). This model theorizes that reserve capacity, comprised of inter- and

intra-personal resources (e.g., family support and optimism) moderates the effects of stress on negative outcomes. Hispanics may have a reduced reserve capacity because of repeated need to adapt to stressors thus amplifying the negative effects of stress. Consistent with this reasoning, using data from the Hispanic Community Health Study/Study of Latinos (HCHS/SOL) used in the current study, Gallo, Isasi, and colleagues found that greater levels of chronic stress are associated with a greater prevalence and risk for cardiovascular disease (Gallo et al., 2014) and that health behaviors could comprise a pathway in these associations as participants in this study who report greater levels of stress were more likely to have poor diet quality and to be obese (Isasi et al., 2015).

3.1.5 Acculturation Stress and Neurocognitive Function

Immigrant racial and ethnic minority individuals undergo unique stressful experiences specific to their racial and ethnic group (e.g., acculturation) that may have additional effects on health. Acculturation stress refers to distress experienced as a result of adapting to a host culture that involves changes in identity, values, behaviors, cognitions, and attitudes (Miranda & Matheny, 2000; Rogler, Cortes, & Malgady, 1991). The process of adapting to a social environment that is new or different from their own is an additional strain for Hispanic/Latino adults that can result in negative health outcomes (Caplan, 2007; Gallo, Penedo, Espinosa de los Monteros, & Arguelles, 2009). Although most foundational work on acculturation stress deals with immigrants adapting to a new culture, we apply this concept to Hispanics/Latinos who might have spent their entire lives living in the United States, often within the same community. Further, although acculturation stress has yet to be examined in relation to neurocognitive function, it is likely that this source of stress is associated with neurocognitive function through

the emotional and cognitive mechanisms delineated in the reserve capacity model (Gallo & Matthews, 2003).

3.1.6 Indirect Pathways between Stress and Neurocognitive Function

Examining indirect pathways that explain the associations between various sources of stress and neurocognitive functioning is important for identifying salient points of intervention for poor neurocognitive function in Hispanics/Latinos. The reserve capacity model postulates that two pathways linking stress to negative outcomes are increases in negative affect and negative cognitions. We investigated the role of dysphoric affect and negative repetitive thought as indirect pathways in the link between stress and neurocognitive function. Empirical evidence demonstrates that experiences of stress resulting from chronic stressors or culture-related stressors are associated with increased levels of depressive symptoms (Kendler, Karkowski, & Prescott, 1999; Kessler, 1997; Snyder, 1987) that are associated with lower neurocognitive performance (Bäckman, Hill, & Forsell, 1996). Indeed, a recent study by Gonzalez and colleagues also using data from the HCHS/SOL reported a negative association between depressive symptoms and neurocognitive performance (Gonzalez et al., 2014). Similarly, psychosocial stress has been linked with greater levels of anxiety-related cognitions (Hovey & Magaña, 2000) that have in turn been associated with poorer neurocognitive performance (Eysenck & Calvo, 1992; Eysenck, Derakshan, Santos, & Calvo, 2007). Studies that have formally tested the indirect pathways between stress and subjective and objective cognitive function find that negative repetitive thought accounts for the stress-cognition link beyond the effects of negative affect (Munoz, Sliwinski, Smyth, Almeida, & King, 2013; Stawski, Mogle, & Sliwinski, 2013). Nonetheless, these associations have yet to be examined in Hispanics/Latinos.

3.1.7 The Current Study

Stress is an important predictor of neurocognitive function, but it remains unclear the extent to which it is associated with neurocognitive function in Hispanics/Latinos. Hispanic/Latino adults may be more susceptible to experience stress (Gallo & Matthews, 2003; Geronimus, 1992; Myers, 2009) that may result in poorer neurocognitive function. Further, because of their unique sociocultural experiences, additional stressors, such as acculturation stress, may also be negatively associated with their neurocognitive functioning. Guided by the reserve capacity model (Gallo & Matthews, 2003) and the empirical evidence reviewed, we predicted that greater chronic stress would be associated with poorer neurocognitive performance in an adult Hispanic/Latino sample (Hypothesis 1). Similarly, we predicted and that greater acculturation stress would be associated with lower neurocognitive performance among Hispanic/Latino adults (Hypothesis 2).

General and culture-related sources of stress are associated with increased depressive affect and negative repetitive thought (Borders & Liang, 2011; Hovey & Magaña, 2000) and these emotional and cognitive factors are associated with poorer neurocognitive performance (Bäckman, Small, & Fratiglioni, 2001; Stawski, Sliwinski, & Smyth, 2006) and dementia risk (Anstey, Cherbuin, & Herath, 2013). A secondary aim of this study was to formally test whether depressive affect or repetitive thought account for the stress-cognition link. Although we hypothesized that depressive affect will comprise a pathway through which stress negatively affects neurocognitive performance (Hypothesis 3), based on previous evidence, we hypothesize that repetitive thought will account for the stress-cognition link beyond the effect of negative affect (Hypothesis 4). We account for socioeconomic and demographic indicators, such as gender, language preferred, education level and income, as well as health status as it has been

linked with increases in stress levels (Vasunilashorn, Lynch, Glei, Weinstein, & Goldman, 2014).

3.2 Method

3.2.1 Participants and Procedure

We used data from the Hispanic Community Health Study/Study of Latinos (HCHS/SOL) and HCHS/SOL Sociocultural Ancillary Study (SCAS). HCHS/SOL is a prospective study that enrolled 16,415 individuals who self-identified as Hispanic/Latino across four field centers in the United States (Bronx, N.Y.; Chicago, IL; Miami, FL; San Diego, CA). HCHS/SOL implemented a two-stage area household probability design (LaVange et al., 2010) and has sample weights and values for the variables to be analyzed. We apply sampling weights to these analyses because they account for unequal probability of selection into the study, adjust for nonresponse, and are calibrated to the target population from the 2010 Decennial Census (LaVange et al., 2010). The target population were all non-institutionalized Hispanic/Latino adults aged 18-74 years (45 -74 years in the current study; LaVange et al., 2010). Applying sampling weights when possible provides a more accurate approximation of our target population estimates. During the baseline examination, participants provided demographic information and were tested in their preferred language (i.e., Spanish or English). Participants who were 45 years or older also completed a neurocognitive battery provided in a face-to-face interview with trained researchers (Gonzalez et al., 2014; Sorlie et al., 2010).

SCAS participants were asked to return within 3 to 9 months after their baseline examination in HCHS/SOL in which they completed a number of psychosocial assessments that included the stress measures used in the current analysis. The current analysis includes data from 3, 278 individuals of the 5, 313 SCAS participants who were 45 years and older and who

provided baseline data from HCHS/SOL. Questionnaires were administered via interview using a standardized approach across all centers. Interviews were between 1 to 2 hours long and participants were compensated \$60. The parent study and SCAS were both conducted with Institutional Review Board approval from all sites (Merz et al., 2014).

3.2.2 Measures

Chronic stress. Assessed with eight items in which participants indicated if an event happened (e.g., “Have you had a serious ongoing health problem?” and “Have you experienced ongoing financial strain?”), whether or not the event has been ongoing for six months or more (e.g., “Has this been a problem for six months or more?”), and the severity of the problem. To indicate the problem’s severity, participants were asked to report on a three-point scale if the problem was “Not very stressful”, “Moderately stressful”, or “Very stressful”. A total count score was calculated for the stressors that were endorsed as ongoing for six months and were reported as being moderately or very stressful whereby greater scores indicated higher chronic stress (Bromberger & Matthews, 1996).

Acculturation stress. Assessed via the Hispanic stress inventory (HSI; Cervantes, Padilla, & De Snyder, 1990), which is a 17-item questionnaire that asked participants to indicate whether (i.e., yes, no) various situations occurred to them during the last three months. Sample items include “Because I am Latino I have had difficulty finding the type of work I want” and “Because of my poor English others have treated me badly.” After each yes/no response, participants rated the situation’s severity by reporting the extent to which they have felt “worried or tense” about it on a 5-point scale from “Not at all worried/tense” (1) to “Extremely worried/tense” (5). A mean severity score was calculated whereby scores from the Likert-type

scale responses were added and divided by the total number of positive endorsements to the items with higher scores indicating greater distress from the problems endorsed.

Depressive symptoms. The 10-item Center for Epidemiological Studies-Depression scale (CES-D; (Radloff, 1977) was used to assess participants' depressive symptoms. This is an established measure for assessing negative mood and depression. Participants responded on a 4-point scale from "Rarely or none of the time" (0) to "All of the time" (3) to items such as "I feel depressed," and "I am happy." Positively worded items were reverse coded and a total sum score was calculated where higher values indicated greater depressive symptoms.

Trait anxiety. Assessed via 10-items from the Spielberger Trait Anxiety Scale (Spielberger, 1983). This measure asked participants to respond to the extent that the items described them generally. Sample items include: "I feel nervous and restless" and "I worry too much over something that really doesn't matter." Participants responded on a 4-point scale from "Almost never" (1) to "Almost always" (4). Items were summed where higher values indicated greater trait anxiety.

Neurocognitive tasks. Cognitive assessments were administered by a trained interviewer in the following fixed order: global mental status, verbal learning and memory, word fluency, and psychomotor speed. Participants were allowed to give their responses in Spanish or English.

Global mental status screener. Six questions derived from the Mini-Mental Status Examination—a short measure used to assess global mental status. The interviewer gave the participant a list of three words and the participant was asked to repeat the list immediately after. Thereafter, the interviewer asked the participant three orientation items (e.g., what year is this?). Lastly, the interviewer asked participant to repeat the list of three words that were presented initially. A total number of words recalled correctly and correct responses to the orientation

items were recorded and a total score calculated with a range from 0 to 6 were greater scores indicate better performance.

Verbal learning and memory. The Spanish English Verbal Learning Test was used to assess new verbal learning and memory. During this task, the interviewer read a list of 15 words and asked the participant to recall as many words as possible over three trials (learning trials). The interviewer then read a distractor list and asked the participant to recall this list. For the last and fifth trial, the interviewer asked the participant to recall the list of words that was presented initially. A total sum score of number of words correctly recalled for the first three trials was calculated for the verbal learning score and a total score of correctly recalled words was calculated for the fifth trail for a memory score where greater scores indicated better performance.

Word fluency. The Word Fluency Test was used to assess verbal functioning. During this task, participants were asked to produce as many words as possible that begin with the letter F and A within a time limit of 60 seconds for each letter while avoiding proper nouns, variations, plurals, and repetitions. A total sum score of admissible words produced by the participant was created were higher scores indicate more words produced.

Psychomotor speed. The Digit Symbol Substitution Test was used in which participants were asked to translate numbers (1-9) to symbols using a key provided at the top of the form. Scoring of this task consisted of assigning one correct point for every symbol that was drawn correctly and in order. Greater scores for this task indicated better performance.

Physical health. A summary score from the physical health domain of quality of life sub-scale form the SF-12 (version 2). This sub-scale score is a norm-based on transformation of a

standardized Z-scores of the constituent items which is scaled to a mean of 50 and standard deviation of 10.

3.2.3 Analytic Strategy

Statistical analysis were conducted for the 3, 278 individuals who participated in the SCAS and were 45 years or older. Individuals who had three or more errors in the six-item screener were excluded from the analyses due to possible cognitive impairment (Callahan, Unverzagt, Hui, Perkins, & Hendrie, 2002). One-hundred and forty-six or 4.45% of individuals made 3 or more errors in the six-item screener and were thus excluded in further analyses resulting in a sample size of 3, 132 individuals. Descriptive statistics, zero-order correlations, and multiple linear regressions were weighted to the known 2010 census target population distribution (LaVange et al., 2010). Tests of indirect effects did not incorporate sampling weights. Descriptive and regression analyses were performed using SAS version 9.4 (SAS Institute, 2013) and statistical tests of mediation applying bootstrap methodology were conducted using Mplus version 6 (Muthen & Muthen, 1998).

Depressive affect and *negative repetitive thought* indicators were calculated based on previous empirical knowledge demonstrating that the CES-D scale has a four-factor structure with one factor representing depressive affect (Hertzog, Van Alstine, Usala, Hultsch, & Dixon, 1990). The trait anxiety scale has been shown to have a two-factor structure with some items loading on depressive affect and another on negative repetitive thought (worry/anxiety; Bieling, Antony, & Swinson, 1998). One item from the CES-D scale: “I had trouble keeping my mind on what I was doing” includes a thought component that we hypothesized would also load on the negative “repetitive thought” factor. Table 3.1 presents the hypothesized factors, the item loadings, and the scales the items originate from. A two factor confirmatory factor analysis with

these items loading in their respective factors resulted in acceptable model fit ($\chi^2(8) = 7.30, p = .50, CFI = 1.00, RMSEA = 0.00$); both factor were positively correlated ($r = .63, p < .01$).

Because response options were provided in identical scales, we averaged scored from the items belonging to each factor. Results henceforth will use these average scores as our depressive affect and repetitive thought indicators.

3.3 Results

3.3.1 Descriptive Statistics and Correlations

Our analytic sample consisted of 1,995 (56.62%) females and 1,137 (43.38%) males with a mean age of 56.69 ($SE = 0.24$; range 45-74; see Table 3.2). The majority of the sample were of low socioeconomic status: 39.42% did not complete high school and over 50% had a yearly household income of 20,000 or less. Spanish was also the language of preference for the majority of the sample. Table 3.3 shows the weighted zero-order correlations among the variables of interest below the diagonal and the correlations controlling for age above the diagonal. The bivariate zero-order correlations show that chronic and acculturation stress are positively correlated ($r = .26, p < .01$). Chronic stress was unrelated to neurocognitive function and acculturation stress was negatively associated with word fluency ($r = -.12, p < .01$) and psychomotor speed ($r = -.11, p < .01$). Both chronic stress and acculturation stress were positively associated with depressive affect ($rs: .37$ and $.31, p < .01$) and repetitive thought ($rs: .32$ and $.35, p < .01$). Both depressive symptoms and repetitive thought were associated with lower neurocognitive performance (rs range between $-.14$ and $-.10, p < .01$) and depressive affect and repetitive thought were robustly correlated with each other ($r = .63, p < .01$).

Age was negatively associated with both stress measures and with neurocognitive performance (rs range: $-.36$ to $-.08, p < .01$). Because age may be obscuring the correlations

between stress and neurocognitive performance, we present the correlations controlling for age above the diagonal (Table 3.3). These age-partial correlations show that chronic stress was not significantly associated with verbal learning ($r = -.05, p = .06$) and verbal memory ($r = -.05, p = .07$) although the direction of these correlations were as expected. Controlling for age, acculturation stress was negatively associated with all four of the neurocognitive tasks (r s range: $-.21$ to $-.08, p < .01$). Age partialled correlations demonstrated a positive association between depressive affect and chronic ($r = .40, p < .01$) and acculturation stress ($r = .35, p < .01$) and negative association with all four neurocognitive tasks (r s range: $-.16$ to $-.11, p < .01$). Similarly, repetitive thought was positively associated with chronic ($r = .36, p < .01$) and acculturation stress ($r = .34, p < .01$) and negatively associated with neurocognitive task performance (r s range: $-.13$ to $-.08, p < .05$). Furthermore, we observed a positive association between income and education and neurocognitive performance—we accounted for both of these indicators in our predictor models as well as for gender, Hispanic background, preferred language (Spanish or English), and self-reported health status.

3.3.2 Chronic Stress Associated with Neurocognitive Performance

Bivariate correlations demonstrated a lack of association between chronic stress and neurocognitive performance. Our next goal was to determine whether chronic stress was independently associated with neurocognitive function while controlling for confounding covariates. Results from our weighted multiple linear regression models of chronic stress predicting neurocognitive function are presented on Table 3.4. These results indicate that chronic stress predicted poorer performance in the verbal learning ($B = -.22, 95\% \text{ CI}, -.37, -.07$) and verbal memory ($B = -.09, 95\% \text{ CI}, -.17, -.01$) tasks. Income and education remained statistically significant predictors of neurocognitive performance (i.e., greater income or education associated

with better performance). Age also remained a statistically significant predictor such that older age was associated with poorer performance with the exception for the word fluency task.

3.3.3 Acculturation Stress Associated with Neurocognitive Performance

We also fit weighted multiple linear regressions with acculturation stress predicting each of the neurocognitive assessments (see Table 3.5) and found that acculturation stress was significantly and negatively associated with verbal learning ($B = -.39$, 95% CI, $-.71, -.07$), word fluency ($B = -.78$, 95% CI, $-1.32, -.24$), and psychomotor speed ($B = -1.64$, 95% CI, $-2.29, -.99$), but was unrelated to verbal memory. As in the model presented on Table 3.4, age, education, and income were associated with neurocognitive performance in the expected direction.

3.3.4 Test of Depressive Affect and Repetitive Thought as Indirect Pathways

We conducted tests of multiple indirect pathways as illustrated on Figure 3.1 to examine the extent to which depressive affect and repetitive thought account for the stress-neurocognitive function associations that were significant in the multiple regression models presented above. Given the robust correlation between depressive affect and repetitive thought, a correlation between these mediators was specified in our models.

Chronic stress. Results from our first set of models that examined the indirect effects of chronic stress on verbal learning and verbal memory through depressive affect and repetitive thought are presented on table 3.6. Results indicated significant indirect effects of chronic stress on verbal learning that were conveyed through both depressive affect ($B = -.07$, 95% CI, $-.14, -.01$) and repetitive thought ($B = -.06$, 95% CI, $-.11, -.01$; CFI = 1.00, TLI = 1.00, RMSEA = 0.00). Although the magnitude of the indirect effect through depressive affect was slightly higher, statistical contrasts of these effects (Preacher & Hayes, 2008) showed that the difference between these two indirect pathways was not statistically different from zero ($B = .12$, 95% CI, -

.10, .12). Repetitive thought accounted for the variance between chronic stress and verbal memory ($B = -.04$, 95% CI, $-.06, -.01$); the indirect effect through depressive affect was not significant ($B = -.01$, 95% CI, $-.04, .02$; CFI = 1.00, TLI = 1.00, RMSEA = 0.00).

Acculturation stress. We also examined the extent to which depressive affect and repetitive thought accounted for the association between acculturation stress and verbal learning, word fluency, and psychomotor speed. Results presented on Table 3.7 demonstrate that, consistent with the chronic stress finding, the association between acculturation stress and verbal learning was accounted for by both depressive affect ($B = -.14$, 95% CI, $-.26, -.02$) and repetitive thought ($B = -.13$, 95% CI, $-.23, -.02$; CFI = 1.00, TLI = 1.00, RMSEA = 0.00) and the difference between these indirect effects was not statistically different from zero ($B = .02$, 95% CI, $-.17, .21$). The association between acculturation stress and word fluency was accounted for by depressive affect ($B = -.17$, 95% CI, $-.32, -.03$; CFI = 1.00, TLI = 1.00, RMSEA = 0.00). A different pattern of associations emerged when we tested the indirect path from acculturation stress to word fluency through depressive affect and repetitive thought. Consistent with the above reported pattern, the indirect effect through depressive affect was negative and statistically significant ($B = -.51$, 95% CI, $-.74, -.29$). However, the indirect effect through repetitive thought was positive and significant ($B = .39$, 95% CI, $.17, .60$; CFI = 1.00, TLI = 1.00, RMSEA = 0.00) indicating a possible suppression effect.

3.4 Discussion

We examined the associations among chronic and acculturation stress and neurocognitive function in 3,132 Hispanic/Latino adults aged between 45 and 75 who participated in HCHS/SOL and SCAS. In support of our first two hypotheses, we found that both chronic and acculturation stress were negatively associated with neurocognitive function. Specifically, we

found that chronic stress predicted poorer functioning in verbal learning and verbal memory. Acculturation stress predicted poorer functioning in verbal learning, word fluency, and psychomotor speed. Contrary to what we expected, both depressive affect and repetitive thought accounted for the stress-cognition associations and these indirect effects varied across tasks.

3.4.1 Stress and Neurocognitive Function

To our knowledge, this is the first study to examine an association between chronic stress and neurocognitive function in Hispanic/Latino adults. Our results are in line with previous literature demonstrating an adverse effect of chronic, ongoing strain on cognitive function (e.g., Aggarwal et al., 2014; Wilson et al., 2005). Our results extend this previous work by demonstrating that chronic stress is also associated with impaired neurocognitive performance in Hispanic/Latino adults. Due to the comprehensive assessment of psychosocial stress in the HCHS/SOL SCAS, we were also able to establish a negative association between acculturation stress and neurocognitive functioning. This shows that stress derived from the acculturation process may be an additional risk factor for poor neurocognitive function in the Hispanic/Latino population. This finding suggests that culturally relevant stressors should be considered in future studies to get a clearer picture of the types of psychosocial stressors influencing cognitive health in the aging population. These two forms of stress may also differentially affect domains of functioning as chronic stress predicted lower performance in the verbal learning and memory whereas acculturation was negatively associated with verbal learning, word fluency, and psychomotor speed.

A question that remains to be addressed is whether acculturation stress contributes to general feelings of stress and how this may affect neurocognitive function. Although both chronic and acculturation were positively and significantly associated with one another, the

magnitude of their correlation was a modest one ($r = .26, p < .01$) suggesting that they are not the same construct. Given the prospective nature of HCHS/SOL, we will be able to explore longitudinal changes in stress domains and how they may contribute to cognitive function and cognitive change in future studies.

3.4.2 Depressive Affect and Repetitive Thought as Indirect Pathways

We also simultaneously tested the indirect effects through depressive affect and repetitive thought and found that these two pathways differentially accounted for the stress-cognition associations. Depressive affect and repetitive thought equally accounted for the associations between both stress domains and verbal learning. Whereas repetitive thought independently accounted for the association between chronic stress and verbal memory and depressive affect independently accounted for the association between acculturation stress and word fluency. Previous studies examining the effects of repetitive thought on neurocognitive performance have primarily focused on attention-demanding response time tasks (Klein & Boals, 2001; Stawski et al., 2006) and not on memory accuracy tasks used in this study. The differential effects of depressive affect and repetitive thought on neurocognitive performance may indicate that these pathways impair important processes essential for successful completion of the tasks at hand. Individual differences in depressive affect may diminish the already limited cognitive resources and motivational capacity in highly stressed individuals resulting in an inability to self-initiate production of words in the Word Fluency task (e.g., Ellis, Thomas, McFarland, & Walter, 1985). Moreover, the tendency to experience negative repetitive thoughts results in inefficient attentional processing thus diminishing individuals' ability to retain information necessary for successful performance in the Verbal Memory task (Eysenck & Calvo, 1992; Sliwinski et al., 2006). Our results showing that both depressive affect and repetitive thought may play a role in

the stress-verbal learning association highlight the need for a fine-grained process-based research approach discussed in the Future Directions section below.

A test of multiple indirect pathways of the association between acculturation stress and psychomotor speed resulted in the expected negative indirect effect through depressive affect such that acculturation stress was associated with greater depressive affect and greater depressive affect was associated with worse performance. The indirect effect through repetitive thought was positive, however. An examination of the individual paths while accounting for the indirect effect through depressive affect showed that acculturation stress was associated with elevated repetitive thought and greater repetitive thought was associated with better performance in the psychomotor speed task. One possible explanation for this result is that depressive affect may be operating as suppressor indirect effect. Although the zero-order weighted correlations showed that repetitive thought was negatively associated with performance in the psychomotor speed task ($r = -.05, p < .01$; see Table 3.3), a supplementary analysis showed that the bivariate correlation between psychomotor speed and repetitive thought was positive and significant when partialled for depressive affect ($r = .04, p = .04$). Supplementary simple indirect effects analyses showed that the indirect effect through repetitive thought was positive but not statistically significant ($B = .01, 95\% \text{ CI}, -.01, .30$), but the simple mediation effect through depressive affect was negative and significant ($B = -.30, 95\% \text{ CI}, -.50, -.12$). This finding may be in line with Altamirano and colleagues' findings suggesting that individual differences in the tendency to experience repetitive thought is associated with improved performance in tasks requiring goal maintenance (Altamirano, Miyake, & Whitmer, 2010). Nonetheless, this result should be interpreted with caution given that the confidence interval of the positive simple indirect effect of acculturation stress to psychomotor speed through repetitive thought crossed zero.

3.4.3 Limitations and Future Directions

We consider three important limitations in the current study. First, the mediator variables operationalized in this study shared a substantial amount of variance as they both originated from two measures of mental health. It was therefore difficult to differentiate the specific mechanistic processes involved in the stress-cognition associations—particularly as it pertained to the verbal learning task. Adopting a process-based approach in future studies through which independent measures of repetitive thought and depressive/negative affect are incorporated, will elucidate the specific processes involved in the stress-cognition link. Second, a key limitation of the current analyses is that they involve a cross-sectional examination and the temporal ordering of stress experiences, to increased depressive affect or repetitive thought, to poorer neurocognitive function cannot be established. Nonetheless, the longitudinal design of HCHS/SOL will allow for future examinations of psychosocial predictors of changes in functioning. Lastly, the clinical implications of stress on neurocognitive health may be questionable given the small effect sizes of chronic and acculturation stress on neurocognitive function. Focusing on chronic stress and verbal learning for example, it is unclear whether a .09 unit decrease in accuracy in verbal memory due to one unit increase in chronic stress (see Table 3.4) indicates transition from healthy neurocognitive function to a diagnosis of cognitive impairment. It is worth pointing out, however, that the effect size for income is a little over two times the effect size of chronic stress and changes in two points on the chronic stress variable represent a change in over 20,000 dollars of a person's annual household income. Attempts to decrease levels of chronic stress are certainly more feasible than attempts to increase a person's household income in order to improve their neurocognitive health.

3.4.4 Conclusion

In spite of its limitations, the current study is among the first to establish that chronic and acculturation stress are differentially associated with impaired neurocognitive performance in a large cohort of Hispanic/Latino adults accounting for key demographic factors. The significant indirect effects through depressive affect and repetitive thought suggest that these may comprise important points of intervention aimed at reducing the burden of neurocognitive impairment in the Hispanic/Latino population.

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Table 3.1

Hypothesized factors, items, and resulting standardized loadings from CFA

Depressive affect		Repetitive thought	
I felt depressed (CES-D)	0.82	I had trouble keeping my mind on what I was doing (CES-D)	0.57
I felt fearful (CES-D)	0.63	I worry too much over something that really doesn't matter (ANX)	0.56
I was happy (R; CES-D)	0.66	I am a steady person (R; ANX)	0.36
I felt lonely (CES-D)	0.54	I get into a state of tension and turmoil as I think over my recent concerns and interests (ANX)	0.61

Note: CES-D = Center for Epidemiological Studies-Depression scale; ANX = Spielberger Trait Anxiety Scale.

Table 3.2

Weighted sample characteristics (N=3, 132)

	Mean (SE)/Freq. (%)	Range
Sex		
Women	1995 (56.62)	
Men	1137 (43.38)	
Age	56.69 (0.24)	45 - 75
Hispanic/Latino Background		
Dominican	304 (10.10)	
Central American	306 (6.41)	
Cuban	525 (27.98)	
Mexican	1151 (29.73)	
Puerto Rican	557 (18.36)	
South American	239 (6.09)	
More than one/Other heritage	50 (1.33)	
Language Preference		
Spanish	2731 (85.45)	
English	401 (14.55)	
Educational attainment		
< High school	1266 (39.42)	

High school education	677 (20.37)	
Some college or more	1181 (40.21)	
Household income		
< \$10,000	578 (22.43)	
\$10,001 – \$20,000	999 (33.57)	
\$20,001 - \$40,000	899 (28.93)	
>\$40,000	398 (15.07)	
Chronic stress	1.38 (0.04)	0 - 8
Acculturative Stress	0.78 (0.02)	0 - 5
Depressive affect	0.78 (0.02)	0 - 3
Repetitive thought	0.90 (0.02)	0 - 3
SEVLT total	22.21 (0.19)	4 - 40
SEVLT delayed	7.97 (0.09)	0 - 15
Word fluency	18.17 (0.25)	0 - 45
Digit symbol substitution	33.83 (0.43)	0 - 83

Table 3.3

Weighted zero-order and age-partial correlations among stress measures, cognition, and covariates

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Chronic stress	-	.29	.40	.36	-.05	-.05	.02	.02	-.08	.07	-.12	-.03	.18	.02
2. Acculturation stress	.26	-	.35	.34	-.11	-.08	-.15	-.21	-.10	-.09	-.25	-.15	-.30	.04
3. Depressive affect	.37	.31	-	.63	-.16	-.13	-.11	-.12	-.12	.00	-.22	-.13	.00	.02
4. Repetitive thought	.32	.35	.63	-	-.13	-.13	-.11	-.08	-.15	-.01	-.18	-.11	.03	-.02
5. Verbal learning	-.03	-.03	-.14	-.10	-	.72	.37	.33	-.19	.03	.23	.29	.01	.00
6. Verbal memory	-.02	-.01	-.11	-.10	.76	-	.31	.31	-.16	.04	.22	.23	-.01	.00
7. Word fluency	.04	-.12	-.10	-.09	.37	.31	-	.43	-.01	.13	.25	.33	.08	.01
8. Psychomotor speed	.05	-.11	-.10	-.05	.42	.38	.46	-	-.05	.25	.33	.43	.29	.03
9. Gender (0 = female)	-.07	-.03	-.13	-.16	-.19	-.15	-.01	-.05	-	.02	.13	-.01	.07	.00
10. Hispanic Background	.08	-.13	.00	-.02	.03	.04	.13	.23	.01	-	.13	.03	.24	.01
11. Income	-.04	-.17	-.23	-.19	.25	.24	.26	.35	.11	.12	-	.23	.10	.01
12. Education	.04	-.07	-.12	-.08	.31	.25	.34	.43	-.01	.04	.23	-	0.05	.00
13. Language (0 = Spanish)	.25	-.16	.03	.00	.04	.02	.10	.30	.05	.29	.19	.11	-	-.01
14. Physical health	.04	.01	.02	.00	-.01	-.02	.00	.02	.02	.03	.03	.01	0.02	-

15. Age	-.08	-.25	-.02	-.07	-.28	-.26	-.10	-.36	-.01	.02	-.14	-.11	-.08	.05
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Note. Bold coefficients significant at $p < .01$. Age partial correlation presented above diagonal

Table 3.4

Weighted multiple linear regression on cognitive tasks regressed onto chronic stress and covariates

	Verbal learning (<i>B</i> , 95% CI)	Verbal memory (<i>B</i> , 95% CI)	Word fluency (<i>B</i> , 95% CI)	Psychomotor speed (<i>B</i> , 95% CI)
Intercept	22.72 (22.41, 23.04)	8.24 (8.08, 8.39)	19.06 (18.55, 19.56)	34.22 (33.67, 34.78)
Chronic stress	-0.22 (-0.37, -0.07)	-0.09 (-0.17, -0.01)	0.14 (-0.11, 0.39)	-0.23 (-0.61, 0.16)
Age	-0.17 (-0.21, -0.13)	-0.08 (-0.10, -0.06)	-0.05 (-0.11, 0.01)	-0.50 (-0.56, -0.43)
Gender	-2.33 (-2.86, -1.80)	-1.02 (-1.28, -0.76)	-0.72 (-1.71, 0.27)	-2.56 (-3.62, -1.50)
Hispanic background	0.07 (-0.08, 0.212)	0.07 (-0.01, 0.15)	0.47 (0.22, 0.71)	1.39 (1.02, 1.77)
Income	0.41 (0.29, 0.53)	0.22 (0.17, 0.28)	0.56 (0.33, 0.80)	1.03 (0.82, 1.23)
Education	1.49 (1.17, 1.81)	0.55 (0.39, 0.71)	2.13 (1.50, 2.77)	4.54 (3.85, 5.22)
Language	-0.11 (-0.82, 0.59)	-0.15 (-0.54, 0.23)	-0.09 (-2.47, 2.28)	6.51 (4.88, 8.15)
Physical health	-0.00 (-0.03, 0.02)	-0.00 (-0.02, 0.01)	-0.00 (-0.05, 0.04)	0.03 (-0.03, 0.09)

Note. Bold coefficients significant at $p < .05$

Table 3.5

Weighted multiple linear regression on cognitive tasks regressed onto acculturation stress and covariates

	Verbal learning (<i>B</i> , 95% CI)	Verbal memory (<i>B</i> , 95% CI)	Word fluency (<i>B</i> , 95% CI)	Psychomotor speed (<i>B</i> , 95% CI)
Intercept	22.79 (22.48, 23.10)	8.27 (8.12, 8.42)	19.02 (18.54, 19.50)	34.33 (33.77, 34.89)
Acculturation stress	-0.39 (-0.71, -0.07)	-0.13 (-0.30, 0.05)	-0.78 (-1.32, -0.24)	-1.64 (-2.29, -0.99)
Age	-0.18 (-0.21, -0.14)	-0.09 (-0.10, -0.07)	-0.07 (-0.13, -0.01)	-0.52 (-0.59, -0.46)
Gender	-2.30 (-2.80, -1.80)	-0.96 (-1.21, -0.71)	-0.77 (-1.74, 0.20)	-2.63 (-3.59, -1.66)
Hispanic background	0.05 (-0.10, 0.20)	0.06 (-0.02, 0.14)	0.44 (0.20, 0.67)	1.28 (0.94, 1.63)
Income	0.39 (0.27, 0.51)	0.22 (0.16, 0.27)	0.48 (0.25, 0.70)	0.95 (0.74, 1.16)
Education	1.44 (1.14, 1.75)	0.54 (0.39, 0.70)	2.14 (1.50, 2.77)	4.47 (3.85, 5.10)
Language	-0.71 (-1.39, -0.02)	-0.47 (-0.83, -0.12)	-0.36 (-2.66, 1.94)	4.95 (3.55, 6.35)
Physical health	-0.00 (-0.03, 0.03)	-0.00 (-0.02, 0.02)	0.01 (-0.03, 0.05)	0.04 (-0.02, 0.09)

Note. Bold coefficients significant at $p < .05$.

Table 3.6

Individual path and indirect effects estimates of chronic stress on neurocognitive tasks

Dependent Variable (DV)	Chronic Stress → DA (B, 95% CI)	DA → DV (B, 95% CI)	Chronic Stress → RT (B, 95% CI)	RT → DV (B, 95% CI)	Chronic Stress → DV (B, 95% CI)	Total (B, 95% CI)	Indirect through:	
							DA (B, 95% CI)	RT (B, 95% CI)
Verbal learning	0.19 (0.17, 0.20)	-0.38 (-0.73, -0.03)	0.15 (0.13, 0.16)	-0.39 (-0.76, -0.03)	-0.11 (-0.24, 0.03)	-0.24 (-0.35, -0.11)	-0.07 (-0.14, -0.01)	-0.06 (-0.11, -0.01)
Verbal memory	0.19 (0.17, 0.20)	-0.05 (-0.23, 0.13)	0.15 (0.13, 0.16)	-0.24 (-0.44, -0.05)	-0.04 (-0.11, 0.03)	-0.09 (-0.15, -0.03)	-0.01 (-0.04, 0.02)	-0.04 (-0.06, -0.01)

Note. Results covaried for age, income, Hispanic background, gender, language preference, and health status. Bold coefficients significant at $p < .05$. Bootstrap sample size=5,000. DA=Depressive affect;

RT = Repetitive thought.

Table 3.7

Individual path and indirect effects estimates of acculturation stress on neurocognitive tasks

Dependent Variable (DV)	Acc. Stress → DA (B, 95% CI)	DA → DV (B, 95% CI)	Acc. Stress → RT (B, 95% CI)	RT → DV (B, 95% CI)	Acc. Stress → DV (B, 95% CI)	Total (B, 95% CI)	Indirect through:	
							DA (B, 95% CI)	RT (B, 95% CI)
Verbal learning	0.32 (0.29, 0.36)	-0.45 (-0.80, -0.11)	0.28 (0.26, 0.32)	-0.44 (-0.80, -0.08)	0.02 (-0.23, 0.26)	-0.25 (-0.48, -0.03)	-0.14 (-0.26, -0.04)	-0.13 (-0.23, -0.02)
Word fluency	0.32 (0.29, 0.36)	-0.53 (-0.98, -0.09)	0.28 (0.26, 0.32)	0.000 (-0.50, 0.48)	-0.25 (-0.60, 0.09)	-0.43 (-0.75, -0.11)	-0.17 (-0.32, -0.03)	0.00 (-0.14, 0.14)
Psychomotor speed	0.32 (0.29, 0.36)	-1.59 (-2.29, -0.91)	0.28 (0.26, 0.32)	1.32 (0.59, 2.07)	-1.10 (-1.64, -0.54)	-1.23 (-1.73, -0.73)	-0.51 (-0.74, -0.29)	0.39 (0.17, 0.60)

Note. Results covaried for age, income, Hispanic background, gender, language preference, and health status. Bold coefficients significant at $p < .05$. Bootstrap sample size=5,000. Acc. = acculturation;

DA=Depressive affect; RT = Repetitive thought.

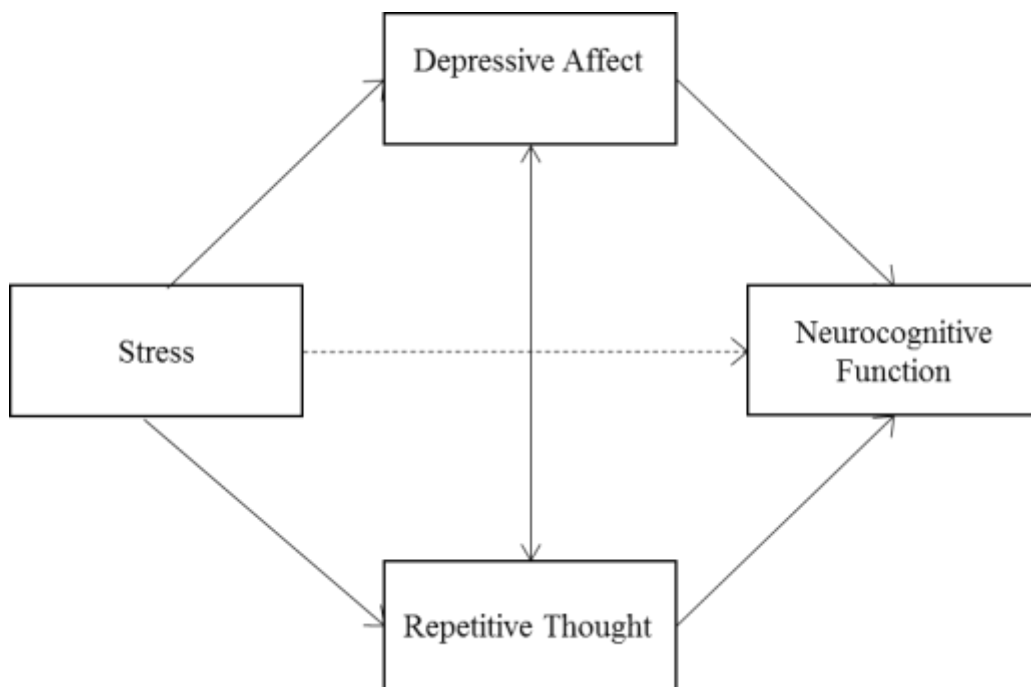


Figure 3.1. Illustration of a multiple indirect effects design in which stress predicts neurocognitive performance (direct effect) through depressive affect or repetitive thought (indirect effects) with both indirect pathways correlated with each other.

CHAPTER 4. DISCUSSION

This dissertation evaluated five hypotheses regarding the association between psychological stress and cognitive function and change as well as the role of negative repetitive thought as a potential mechanism in this association. Consistent with the first hypothesis, results from Study 1 showed that higher levels of psychological stress were associated with more rapid cognitive slowing across a two-year period. However, contrary to the second hypothesis, results from Study 1 did not indicate that changes in stress were associated with changes in cognitive function. Study 2 used data from a multi-center epidemiological study of Hispanic/Latino adults, to demonstrate that higher levels of chronic stress (Hypothesis 3) and acculturation stress (Hypothesis 4) were associated with lower levels of cognitive performance. Finally, negative repetitive thought was an indirect pathway in some stress-cognition associations (Hypothesis 5) but depressive affect also comprised an indirect pathway in some of the associations.

4.1 Chronic Psychological Stress and Cognitive Function

Both dissertation studies found support for a negative association between self-reported stress and cognitive function. Study 2 found evidence that supported the hypothesized associations between greater self-reported severity of chronic and acculturation stress and lower cognitive performance. Study 1 supported the predicted longitudinal association between greater perceived stress reports and prospective cognitive slowing. These findings provide support for the role of psychological stress in cognitive function and change. Although Cohen and colleagues' (1995) stress process model does not include neurocognitive health as an outcome, these studies extend work by others (i.e., Aggarwal et al., 2014; Rönnlund, Sundström, Sörman, & Nilsson, 2013) showing deleterious effects of subjective stress on cognitive function.

4.1.1 Directionality between Stress and Cognitive Function. Despite the longitudinal evidence that level of perceived stress predicts changes in cognition, this does not rule out the possibility that low or declining levels of cognition exacerbate the effects of stress. One inherent assumption across the two studies and the theoretical models they were based on is that higher stress leads to poor cognitive function through various indirect pathways but the possibility exists that this relationship is the reverse and declines in cognition predict stress. One study assessed cognition as a predictor of stress exposure and reactivity and found that individuals with greater fluid abilities reported experiencing more stressors, particularly work and home-related stressors, but they were less emotionally reactive to them (Stawski, Almeida, Lachman, Tun, & Rosnick, 2010). This finding suggests that individuals with fewer abilities may be more likely to be exposed to stressors that negatively impact their emotional well-being. Support for this notion can be further corroborated in future studies with repeated stress and cognitive assessments covering a longer time period to verify if this lack of association holds across time.

4.1.2 Sources of Psychological Stress. One limitation of the perceived stress measure used in Study 1 is that it captures general levels of perceived stress and does not identify actual stressors or sources of stress. Although global measures of stress may usefully predict cognitive outcomes, it is important to identify the “input” or the life domains that generates feelings of stress so that this factor can be targeted for intervention (e.g., Scott, Jackson, & Bergeman). The measure of subjective chronic stress used in Study 2 asked participants to report whether they had experienced a number of stressors and whether they perceived them as “Not very stressful”, “Moderately stressful”, or “Very stressful”. Appendix D provides a breakdown of the type of stressors provided by the chronic stress measure and the degree to which they were reported as being stressful by the study’s participants. It is evident from this table that health-related

stressors were reported as more severe followed by work-related stressors, financial strains, and relationship difficulties. The health domain may contribute to feelings of stress; a recent study has indeed shown that increases in poor self-reported health were prospectively associated with increases in perceived stress (Vasunilashorn, Lynch, Gleib, Weinstein, & Goldman, 2014).

Although the measure used in Study 2 provides greater insight into what contributes to greater feelings of stress, it also limited the categories to which participants could respond. It is possible that sources of perceived stress vary within individuals across the lifespan warranting more detailed assessments of multiple domains. The incorporation of multiple assessments of stress domains across time using repeated momentary assessment methods may be one feasible approach to measure which life domains are more influential for an individual's feelings of stress and how these influences change with time.

Study 2 also incorporated a measure of acculturation stress in which participants were asked to report whether they had experienced a number of difficulties because of their race or ethnicity and how much these experiences made them feel worried or tense. Although this measure was significantly correlated with the self-reported chronic stress score, it was a moderate correlation of .26, given the large sample size, suggesting that this may be a different source of psychological stress. Further investigations into the unique prediction of such culture and race/ethnicity-specific measures above general measures of chronic stress are warranted in order to establish the potential significance of incorporating this type of measure in future studies.

4.2 Negative Repetitive Thought and Depressed Mood as Indirect Pathways between Stress and Cognitive Function.

Study 2 tested the hypothesis that negative repetitive thought would account for the association between psychological stress and cognitive function over and above the effect of depressed mood; this hypothesis was partially supported. Depressive affect and repetitive thought equally accounted for the associations between both chronic and acculturation stress and verbal learning. However, repetitive thought independently accounted for the association between chronic stress and verbal memory and depressive affect independently accounted for the association between acculturation stress and word fluency. The limitations of the study's measures and implications for specific task performance have been discussed previously; broader theoretical implications of these findings are discussed below.

The differential indirect effects of depressive mood and repetitive thought on the stress-cognition associations indicate that further empirical inquiry into the nature of these two mechanisms is needed. Specifically, examinations of the time ordering and directionality of these two processes will be informative for future prevention and intervention efforts. Models of depression propose that ruminative thoughts elevate the dysphoric mood that is associated with impaired memory functioning (McBride & Abeles, 2000; McClintock, Husain, Greer, & Munro, 2010; Nolen-Hoeksema, 1991). This line of reasoning proposes that depressive affect is the source of poor cognitive function because the available cognitive resources are allocated to the thoughts about dysphoric emotions rather than to the tasks being performed (Lyubomirsky, Kasri, & Zehm, 2003; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008; Seibert & Ellis, 1991).

Another line of reasoning is that repetitive thoughts produced by depressed affect are key predictors of poor cognitive performance because they deplete attentional resources necessary for successful completion of the tasks being performed (Eysenck & Calvo, 1992; Smallwood, Fitzgerald, Miles & Phillips, 2009; Sliwinski et al., 2006). Formal tests comparing the indirect

effects of these two processes show that repetitive thoughts account for the stress-cognition link above and beyond the effects of negative affect (e.g., Munoz, Sliwinski, Smyth, Almeida, & King, 2013; Stawski, Mogle, & Sliwinski, 2013). Although repetitive thoughts may indeed be driving the stress cognition relationships, the content of these thoughts may be key to these findings. Watkins (2008) proposed that one component that makes repetitive thoughts unconstructive is the valence of the thoughts; specifically if they are negative. Although previous preliminary evidence demonstrates that the tendency to experience intrusive thoughts accounts for the stress-cognition link, the content of these mediating thoughts has not been investigated. A set of studies incorporating momentary assessments of stress, negative affect, repetitive thoughts, and cognition show that although repetitive thoughts partially account for the increases in negative affect following reports of a negative event (Moberly & Watkins, 2008a), the association between thoughts and negative affect is reciprocal in that thoughts are linked with increases in negative affect at later time-points and vice versa (Moberly & Watkins, 2008b). It may prove to be difficult to disentangle the individual influence of negative affect and repetitive thought in future research. However, studies such as those conducted by Moberly and Watkins suggest that repeated momentary assessments of these processes is an ideal approach to undertake in future investigations.

4.3 Future Directions

This dissertation focused on how the psychological response to stress relates to cognitive performance and change and identified repetitive thought and depressive affect as two feasible mechanisms in this association. However, it is worth acknowledging the critical role of the physiological response to stress as an additional mechanism in the perceived stress-cognition associations. One reason why it is important to examine effects of the physiological stress

response on cognition is because this response originates in the central nervous system. The hypothalamic-pituitary-adrenal (HPA) axis is activated following perceptions of stress during which the cerebral cortex communicates with the hypothalamus to initiate release of corticotropin releasing hormone (CRH). CRH then stimulates the anterior pituitary to secrete adrenocorticotropin hormone, which then triggers the adrenal cortex to release cortisol into the bloodstream (Dickerson & Kemeny, 2004). Once cortisol is released, it binds to receptors in the hippocampus that then inhibit the release of more cortisol. However, if this process is chronically activated due to constant perceptions of stress, this can lead to neuronal death in the hippocampus (Sapolsky, 1999). This HPA axis activity is accompanied by an inflammatory response that is also detrimental for cognitive health if it is prolonged or activated longer than necessary (Solfrizzi et al., 2006; Yaffe et al., 2003). Although studies have linked both elevated cortisol levels and circulating inflammation with cognitive impairment and decline (e.g., Yaffe et al., 2003), future research aimed at understanding the psychological processes that activate and maintain these responses and how they are in turn associated with cognitive functioning is warranted.

4.4 Conclusions

Overall, this dissertation demonstrates the key role of psychological stress on poor cognitive function and change among middle aged and older adults and shows that Hispanics/Latinos may experience culture-specific stressors that elevate their risk for poor cognitive function. This dissertation also shows that repetitive thought and depressive affect constitute possible targets of intervention for reducing the burden of poor cognitive function among aging adults.

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APPENDIX A

Stress-related terms and their definitions derived from the literature

Term	Definition
Stress	"Process in which environmental demands tax or exceed the adaptive capacity of an organism, resulting in psychological and biological changes that may place a person at risk for disease" (Cohen et al., 1995, p.3).
Stressor	<ul style="list-style-type: none"> • Stressful life experiences • Circumstances that threaten a major goal, including the maintenance of physical or psychological well-being
Acute stress	<ul style="list-style-type: none"> • Reversible and relatively short-lived experiences of stress
Chronic stress	<ul style="list-style-type: none"> • Long-term stress <ul style="list-style-type: none"> ○ May stem from a stressor that has not been resolved. If the stressor is resolved, the feelings and thoughts surrounding it may still be present
Stress exposure	<ul style="list-style-type: none"> • Positive endorsement of having experienced a stressor over a given recall period (e.g., over the past hour, day, week, month)
Stress reactivity	<ul style="list-style-type: none"> • The extent to which experiences of stress increases a person's level of negative emotions, cortisol, or cardiovascular activity
Psychological stress	<ul style="list-style-type: none"> • Appraisal of an event as threatening • Occurs between "stimulus presentation" or stress exposure and "stress reaction" or stress reactivity
Distress	<ul style="list-style-type: none"> • Negative psychological response to stressful experiences • Includes a number of emotional and cognitive states, such as sadness, anxiety, frustration, the sense of being overwhelmed, or helplessness

Selected References:

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APPENDIX B

Fixed and random effects from double-exponential learning model

	<u>NM3</u>		<u>NM5</u>		<u>NB1</u>		<u>NB2</u>		<u>KT1</u>		<u>KT2</u>	
	Estimate (SE)		Estimate (SE)		Estimate (SE)		Estimate (SE)		Estimate (SE)		Estimate (SE)	
Fixed effects												
<i>a</i>	1500.42	(48.21)*	3220.55	(96.90)*	832.18	(29.71)*	1289.86	(66.14)*	581.61	(20.57)*	984.55	(37.25)*
Δa	53.59	(7.44)*	60.29	(13.22)*	9.06	(4.20)*	28.69	(10.49)*	6.89	(3.11)*	12.91	(7.34)†
<i>g</i>	559.99	(42.71)*	493.12	(83.37)*	405.62	(34.51)*	892.73	(72.79)*	236.45	(22.24)*	268.45	(34.15)*
<i>r</i>	171.68	(10.56)*	163.97	(19.28)*	169.04	(11.35)*	155.89	(8.52)*	241.23	(24.30)*	233.63	(36.30)*
<i>g*</i>	1060.35	(94.06)*	987.53	(184.33)*	577.27	(65.02)*	1569.64	(149.8)*	684.47	(98.51)*	856.04	(182.13)*
<i>r*</i>	3448.86	(141.11)*	3332.23	(278.42)*	4175.42	(351.79)*	3955.21	(209.10)*	3758.48	(160.21)*	3649.14	(213.10)*
Random effects												
<i>a</i>	199.87	(28.11)*	865.79	(0.12)*	832.18	(29.71)*	1289.86	(66.14)*	31.10	(4.32)*	984.55	(37.25)*
Δa	3.37	(0.66)*	8.88	(2.06)*	9.06	(4.20)*	28.70	(10.49)*	0.25	(0.00)*	3.41	(0.63)*
<i>g</i>	114.61	(23.48)*	452.82	(84.45)*	85.15	(14.89)*	443.40	(81.11)*	32.53	(5.98)*	66.90	(13.83)*
<i>g*</i>	187.20	(56.40)*	378.70	(178.16)*	44.40	(20.36)*	662.29	(161.29)*	140.22	(60.56)*	856.03	(182.13)*
Residual	26.40	(0.77)*	117.47	(3.34)*	16.11	(0.46)*	63.82	(1.84)*	6.60	(0.19)*	16.19	(0.47)*

Note. Estimates in milliseconds. Values correspond to equation parameters described in methods section of original text.

NM3 = Number Match 3; NM5 = Number Match 5; NB1= N-Back 1; NB2 = N-Back 2; KT1 = Keep-Track; KT2 = Keep-Track 2.

* $p < .05$. † $p < .10$.

APPENDIX C

Descriptive statistics for baseline response times

Task	<i>M</i>	<i>SD</i>
Keep Track 1	0.79	0.22
Keep Track 2	1.19	0.35
N-Back 1	1.17	0.38
N-Back 2	2.03	0.66
Number Match 3	2.01	0.56
Number Match 5	3.74	1.20

APPENDIX D

Reported severity of chronic stress categories from study 2

	Frequency	Percent	Cumulative Frequency	Cumulative Percent
Have you had a serious ongoing health problem?				
<i>Not very stressful</i>	486	34.13	486	34.13
<i>Moderately Stressful</i>	392	27.53	878	61.66
<i>Very Stressful</i>	546	38.34	1424	100
Has someone close to you had a serious ongoing health problem?				
<i>Not very stressful</i>	473	37.36	473	37.36
<i>Moderately Stressful</i>	345	27.25	818	64.61
<i>Very Stressful</i>	448	35.39	1266	100
Have you had ongoing difficulties with your job or ability to work?				
<i>Not very stressful</i>	185	22.62	185	22.62
<i>Moderately Stressful</i>	232	28.36	417	50.98
<i>Very Stressful</i>	401	49.02	818	100
Have you experienced ongoing financial strain?				
<i>Not very stressful</i>	795	30.95	795	30.95
<i>Moderately Stressful</i>	769	29.93	1564	60.88
<i>Very Stressful</i>	1005	39.12	2569	100
Have you had ongoing difficulties in a relationship with someone close to you?				
<i>Not very stressful</i>	325	28.91	325	28.91
<i>Moderately Stressful</i>	366	32.56	691	61.48
<i>Very Stressful</i>	433	38.52	1124	100

Has someone close to you had an ongoing problem with alcohol or drug use?

<i>Not very stressful</i>	429	36.92	429	36.92
<i>Moderately Stressful</i>	318	27.37	747	64.29
<i>Very Stressful</i>	415	35.71	1162	100

Have you been helping someone close to you, who is sick, limited or frail?

<i>Not very stressful</i>	1196	60.99	1196	60.99
<i>Moderately Stressful</i>	396	20.19	1592	81.18
<i>Very Stressful</i>	369	18.82	1961	100

Have you had another ongoing problem not listed here?

<i>Not very stressful</i>	47	14.42	47	14.42
<i>Moderately Stressful</i>	83	25.46	130	39.88
<i>Very Stressful</i>	196	60.12	326	100

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EDUCATION

- 2015 **Ph.D.** in Human Development and Family Studies
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- 2009 **B.A.** in Psychology and Social Behavior
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- 2014 Research Assistant, *Psychosocial & Health-Related Influences on Response Time Inconsistency*
- 2014, 2012 Teaching Assistant, *Empirical Inquiry*, Penn State
- 2014 Teaching Assistant, *Intro to Human Development & Family Studies*, Penn State
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- 2010, 2011 Teaching Assistant, *Adult Development and Aging*, Penn State
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- January 2015 Joseph and Jean Britton Graduate Fellowship, Penn State
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SELECTED PRESENTATIONS AND PUBLICATIONS

Hill, L. N., Mogle, J., **Munoz, E.**, Colancecco, E., & Wion, R. (2015). Assessment of subjective cognitive impairment among older adults. *Journal of Gerontological Nursing*. doi:10.3928/00989134-20150309-01

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