1-1-2015

Engagement In Activities And Cognitive Functioning Among Older Adults In The Health And Retirement Study

Pamela Emily May
Wayne State University,

ENGAGEMENT IN ACTIVITIES AND COGNITIVE FUNCTIONING AMONG OLDER ADULTS IN THE HEALTH AND RETIREMENT STUDY

by

PAMELA E. MAY

DISSERTATION

Submitted to the Graduate School

of Wayne State University

Detroit, Michigan

in partial fulfillment of the requirements

for the degree of

DOCTOR OF PHILOSOPHY

2015

MAJOR: PSYCHOLOGY (Clinical)

Approved By:

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Advisor Date

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DEDICATION

I dedicate this dissertation to my family and Saurabh, for your unconditional support and faith in me.

ACKNOWLEDGMENTS

I would like to extend a special thank you to my mentor, John L. Woodard, Ph.D., for his guidance throughout my graduate career and for this dissertation project. It is an honor to work with such an esteemed and learned mentor. In addition, I thank the respondents and staff of the Health and Retirement Study, as they made this project possible.

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CHAPTER 1: Purpose of Dissertation

The goal of this dissertation is to examine the effect of cognitive and social activities on cognitive performance in a national sample of older adults from the Health and Retirement Study (HRS). The current analysis will be the first to examine the effect of activity engagement on cognitive performance with the HRS' latest data collection waves. The HRS assesses engagement in a broad span of activities of cognitive, social, and physical nature. In the current analysis, the focus will be on cognitive and social activities. Many studies previously examined the effect of these types of activities on cognition and concluded that older adults who participate in activities (e.g., reading, socializing, exercising) tend to perform better on cognitive tests (e.g., Lachman, Agrigoroaei, Murphy, & Tun, 2010), show less age-related cognitive decline (e.g., Wilson et al., 2010), and have reduced dementia risk (e.g., Akbaraly et al., 2009). However, there continues to be skepticism regarding the conclusiveness of these studies' findings (e.g., Salthouse, 2006). According to Bielak (2009), there are several gaps in the literature that prevent the field from moving forward, including identification of cognitive domains that benefit the most from activity and determining the directionality of the relationship. The proposed analysis will attempt to address these specific gaps by examining older adults' engagement in cognitive and social activities and their cognitive functioning across four years using latent growth curve modeling. Additional analyses will be conducted to determine if overall health is similarly impacted by activity engagement. The results from these analyses are expected to not only support the hypothesis that engaging in cognitively stimulating activities will reduce decline on cognitive and health measures, but will also identify specific components that contribute to this significant association. A positive activity – cognition association will highlight the impact of

modifiable lifestyle behaviors on health and incentivize preventive strategies for cognitive decline.

1.1 Specific Aims

1.1.1 Identify longitudinal relations between activity (i.e., cognitively stimulating and social) engagement frequency and cognitive functioning. Two hypotheses will be tested, an "engagement-first" hypothesis, such that activity frequency predicts change in cognitive functioning, and a "cognitive-first" hypothesis, such that cognitive performance predicts activity frequency. The former is consistent with the *use it or lose it* hypothesis, such that participating in intellectual and/or social activities in late life will reduce deterioration of cognitive skills by exercising them in different applications and settings. The latter is consistent with the cognitive reserve hypothesis (Stern, 2002), as it proposes that distal and proximal cognitive experiences or activities (e.g., educational pursuits) alter brain function, thereby reducing its vulnerability to brain damage or neurodegeneration. As such, individuals with high cognitive reserve may have always had superior cognitive functioning and engaged in mentally stimulating activities. Although both directions of the association have been supported by the literature (e.g., Hultsch, Hertzog, Dixon, & Small, 1999), it is hypothesized that the "engagement-first" hypothesis will explain the data most accurately, based on theoretical concepts such as successful aging, environmental enrichment, and compensatory scaffolding. If the "engagement-first" hypothesis is statistically supported, it is hypothesized that cognitively stimulating activities and social engagement will predict cognitive performance, yet cognitively stimulating activities will have a statistically stronger effect, as they are intellectually engaging and potentially more complex or demanding than socialization (Park et al., 2014).

1.1.2 Identify links between activity (cognitively stimulating and social) engagement frequency and cognitive domains (i.e., working memory, episodic memory, and semantic memory). In the context of the "engagement-first" hypothesis, it is predicted that lower baseline activity frequency will be associated with greater decline in working memory and episodic memory over time but will not significantly affect semantic memory performance over time. Both working memory (Salthouse & Babcock, 1991) and episodic memory (Rönnlund, Nyberg, Bäckman, & Nilsson, 2005) normally decline with age. Semantic memory generally remains stable or declines in smaller increments than either episodic memory (Rönnlund et al., 2005) or working memory in older adults without neurodegenerative disease. Thus, higher activity engagement is expected to stabilize or buffer against age-related declines in working memory and episodic memory (Lodi-Smith & Park, 2011).

1.1.3 Identify longitudinal relations between activity (cognitively stimulating and social) engagement frequency and overall health, and compare these findings to longitudinal relations between activity engagement frequency and cognitive functioning. Similar to Aim 1.1.1, two hypotheses are proposed, the "engagement-first" hypothesis (activity engagement predicts changes in health) and a "health-first" hypothesis (health predicts activity engagement). It is predicted that the "engagement-first" hypothesis will describe the data the best, based on findings on the positive effect of social ties and activities on subsequent health (e.g., House, Robbins, & Metzner, 1982). In addition, previous cross-sectional research reported significant relations between self-reported health and cognitive performance (e.g., Hultsch, Hammer, & Small, 1993). Changes in cognitive functioning and health will be compared simultaneously over time, to identify causal relations between these constructs. It is hypothesized that declines in health will predict subsequent cognitive decline.

CHAPTER 2: Background

2.1 Overview

With age, older adults may experience multiple significant transitions, including changes in work status, health, and social relations. Changes in these domains may potentially influence trajectories of normal and abnormal age-related cognitive decline. Due to increases in the proportion of older adults in the population (Cohen, 2003) and increased life expectancy, there is a heightened interest to find mechanisms that maintain and promote cognitive functioning in late life (Mitchell et al., 2012). As there are no validated interventions to prevent age-related cognitive impairment or dementia, attention has been turned to finding modifiable, lifestyle behaviors that positively impact cognition. The possibility of engaging in mental activities as a means to influence cognitive functioning and rate of age-related decline is appealing, because it suggests that individuals have some control over their cognitive skills (Salthouse, 2006). In fact, participation in cognitively stimulating activities appears to be beneficial for reducing rates of age-related cognitive decline and for enhancing cognitive functioning among the cognitively intact (e.g., Small, Dixon, McArdle, & Grimm, 2012). In addition, participation in cognitively stimulating activities delay or reduce decline associated with impairment or decline (e.g., Wilson et al., 2010).

The current chapter starts with a general overview of theories that postulate the effects of environment on cognition and neural functioning. Then, cross-sectional and longitudinal research on the association between activities (cognitively stimulating and social) and older adults' cognitive function will be reviewed. Evidence for the directionality of these associations will be reported, followed by a discussion of the limitations of previously conducted research.

2.2 Theories for Lifestyle and Health

In this section, theories of maintaining function in late life generally, and cognition, specifically, are reviewed. The following theories support the association between lifestyle behaviors and health.

2.2.1 Successful aging models. Fries pioneered the concept of prevention and successful aging, through a medical perspective (Swartz, 2008). He coined the term, "compression of morbidity" (Fries, 1980), to reflect the hypothesis that the onset of disabilities resulting from chronic illness could be delayed to a later age, through preventive health efforts (Swartz, 2008). As such, disabilities would be compressed within a shorter timeframe at the end of the lifespan (Swartz, 2008). Initial data supporting this hypothesis were from a 20-year longitudinal study (Vita, Terry, Hubert, & Fries, 1998) examining university alumni's' health risks (e.g., obesity, smoking) and cumulative disability. Disability was delayed by almost eight years for alumni who were in the lowest tertile for health risk, relative to those in the highest tertile (Vita et al., 1998), highlighting the impact of lifestyle behaviors on health. Fries' work historically foreshadowed later contributions towards successful aging.

Rowe and Kahn (1987, 1997) developed one of the most recognized successful aging models. Their model comprised of three components, "avoiding disease and disability," "high cognitive and physical function," and "engagement with life" (p. 434); all three components were deemed necessary for living a healthy late life (Rowe & Kahn, 1997). In their model, avoiding disease not only meant the absence or presence of disease itself, but the absence of severity risk factors for disease (Rowe & Kahn, 1997). Disease risk factors included genetic factors or the psychosocial environment in which an individual was born (Rowe & Kahn, 1997), with the latter further suggesting the importance of environmental factors on health.

The current project is based on the hypothesis that a stimulating environment has a positive effect on cognitive performance and brain health, allowing older adults to age more successfully than older adults who do not engage in stimulating environments. Thus, the current project will focus on "successful cognitive aging." The following section will describe theories specific to protecting cognitive functioning in late life.

2.3 Theories of Neuro-Cognitive Protection in Aging

2.3.1 Environmental enrichment in animal models. Hebb was the first to raise environmental enrichment as an area of scientific interest (van Praag, Kemperman, & Gage, 2000). He reported that when he brought rats home from the laboratory as pets, these rats appeared to make more behavioral advances, relative to their laboratory littermates (Hebb, 1947). Animal experimentation on environmental enrichment began in earnest during the 1970s. Early findings indicated that neither social interaction alone (Rosenzweig, Bennett, Hebert, & Morimoto, 1978) nor indirect contact with enriched environments (i.e., observing other rats in enriched condition; Ferchmin & Bennett, 1975) elicited positive changes in the brain (e.g., increased brain weight). Thus, environmental enrichment was defined as settings with both complex inanimate stimulation and social interaction (Rosenzweig et al., 1978). This definition for environmental enrichment is still utilized in current animal research.

The amount of animal research on the effect of environmental enrichment is substantial. Kemperman, Kuhn, and Gage (1997) revealed through experimental research that adult rats living in an enriched environment had increased survival of newly generated neurons in the hippocampal dentate gyrus, relative to rats living in smaller, standard cages with fewer rats. These findings have been similar to those found with older rats, wherein enrichment increased neurogenesis in the dentate gyrus (Kemperman, Kuhn, & Gage, 1998). In both studies, rats living

in enriched environments performed better on a water maze test, relative to rats in standard cages. More recently, Jankowsky et al. (2005) reported that transgenic mice models of Alzheimer's disease living in an enriched environment completed a water maze task similar to nontransgenic mice living in a non-enriched environment. These results suggested that enrichment allowed the transgenic mice to cognitively withstand neural insults (Jankowsky et al., 2005). Environmental enrichment has also been associated with increased brain nerve growth factor in rats (Pham, Ickes, Albeck, Söderström, & Mohammed, 1999), glutamatergic AMPA receptors necessary for long-term potentiation (Naka, Narita, Okado, & Narita, 2005), and neprilysin, an enzyme that degrades beta-amyloid (Lazarov et al., 2005) in rodents. Overall, such findings from animal models appear promising, in terms of the effect of environmental enrichment on humans' cognition. However, the field of animal research has their own questions that remain unsatisfactorily answered, including the duration of effects from enrichment and identifying elements of enrichment that have specific effects on behavior (van Praag et al., 2000). Such questions foreshadow research issues in the field of "enrichment" for humans. The following sections will provide a theoretical background on the role of the environment on human cognition.

2.3.2 "Use it or lose it." Early researchers on cognitive aging advocated the use of mental stimulation to maintain cognitive functioning or even prevent cognitive decline with age (e.g., Foster & Taylor, 1920). Currently, it is hypothesized that mental stimulation is beneficial to cognitive functioning, as when cognitive skills are used less frequently, functional neural areas dedicated to performing these activities may atrophy (Hultsch et al., 1999). Therefore, abstaining from using certain cognitive skills could exacerbate age-related cognitive decline (Hultsch et al.,

1999). The well-known adage, "use it or lose it," has been applied to explain this hypothesis (Hultsch et al., 1999).

Despite its appeal, Salthouse (2006) criticized the "use it or lose it" hypothesis. He reported that the hypothesis could only be supported when there is an interaction of age and activity, or faster age-related cognitive decline with less cognitive exercise (Salthouse, 2006). He argued that researchers have been quick to conclude that cognitive interventions (e.g., Advanced Cognitive Training for Independent and Vital Elderly [ACTIVE] clinical trial) are related to age-related cognitive changes, when changes are not monitored over the long-term. In addition, he reviewed findings that even older adult experts (e.g., expert chess players; Elo, 1965) and older adults with mentally stimulating occupations (e.g., professors; Christensen, Henderson, Griffiths, & Levings, 1997) experience age-related declines in their performance and cognitive functioning, respectively.

After his critical review of the literature, Salthouse (2006) concluded that the evidence for the "use or lose it" hypothesis was inconclusive. This general remark about the state of the literature stirred a debate. Schooler (2007) reported that he found Salthouse's criteria for evidence to support the "use it or lose it" hypothesis too stringent. He proposed that the hypothesis should be judged against whether individuals function at a greater cognitive level for a greater period of time, due to engaging in mental exercises (Schooler, 2007).

Interestingly, following Salthouse's (2006) review, there have been advances in the literature, including identification of biological mechanisms that could account for the association between mental activities and cognitive performance. For example, Valenzuela and colleagues (2012) examined the association of neuropathology with an index of "cognitive lifestyle" (based on education, occupation, and social engagement). Males with a high cognitive

lifestyle were less likely to have cerebrovascular disease (relative to men with less cognitive lifestyles), and similarly classified women tended to have greater brain weight relative to women with less cognitive lifestyles. For both sexes, a cognitive lifestyle was associated with greater neuronal density and cortical thickness within the frontal lobe. Similarly, previous work (Valenzuela, Sachdev, Wen, Chen, & Brodaty, 2008) indicated that higher degrees of mental activity (based on occupation, education, and leisure on the Lifetime of Experiences Questionnaire; [LEQ]; Valenzuela & Sachdev, 2006), predicted a slower rate of cognitive decline over time, in addition to decreased rate of hippocampal atrophy. Older adults with high LEQ scores lost on average 3.6% of hippocampal volume over three years, while older adults with low LEQ scores lost 8.3% (Valenzuela et al., 2008). Such a finding suggests that the medial temporal lobe is maximally protected by lifelong cognitive stimulation (Valenzuela et al., 2008).

Both animal and human research have found significant findings regarding enrichment or engagement on the hippocampus. The effect of activities on the brain, and thus cognitive functioning, can be further explained with the concept of cognitive reserve.

2.3.3 Cognitive reserve. Cognitive reserve is regarded as a protective aid against the effects of neurodegeneration. Specifically, cognitive reserve is the ability to "optimize or maximize performance through differential recruitment of brain networks, which perhaps reflect the use of alternate cognitive strategies" (Stern, 2002, p. 451). Therefore, individuals who use neural networks efficiently, or are able to use different brain networks or cognitive strategies under increased cognitive demand, have greater cognitive reserve (Stern, 2002). Potential proxies of cognitive reserve are childhood IQ, education level, leisure activities, literacy level, and adult occupation (Stern, 2002, 2009). Thus, the concept of cognitive reserve has been viewed as a dynamic process, as cognitive reserve may fluctuate due to experiential influences such as leisure

and social activities (Sanchez, Torrellas, Martín, & Barrera, 2011). It is hypothesized that cognitive experiences or activities positively alter the brain's structure and function, allowing individuals to be less impacted by brain damage (Stern, 2009).

A recent literature review (Valenzuela & Sachdev, 2006) concluded that higher brain reserve (defined by education level, occupational complexity, premorbid IQ, and mentally stimulating leisure activities) was associated with a reduced risk for incident dementia. A negative, dose-response relationship between the amount of complex cognitive activities in late life and dementia risk has also been reported (Wang, Karp, Winblad, & Fratiglioni, 2002). However, cognitive activity or cognitive reserve does not prevent neuropathology, per se. Abnormal neuropathology develops irrespective of cognitive reserve. Stern (2002) proposed that more neuropathology is needed before memory is affected in individuals with high cognitive reserve. This proposal is consistent with the rate by which AD pathology is clinically observed in older adults with high educational levels; the rate of observable memory decline is steeper and shorter in these older adults relative to older adults with low education (Wilson et al., 2004).

2.3.4 Compensatory scaffolding. Researchers have questioned how older adults function well, despite neural degeneration and cognitive processing inefficiencies (Goh & Park, 2009). There is an expansive pool of research that indicates that the brain's neuroplasticity, or its ability to respond and adapt to changing circumstances, facilitates adequate cognitive performance, despite aging (Goh & Park, 2009). Park and Reuter-Lorenz (2009) proposed the scaffolding theory of aging and cognition (STAC), which suggests that the brain develops "scaffolds" following age-related neural insults. Specifically, with aging losses such as neuronal loss, reduced dopamine receptors, and reduced white matter integrity, the brain responds by scaffolding, or recruiting greater sites of circuitry to process incoming information (Park &

Reuter-Lorenz, 2009; Reuter-Lorenz & Cappell, 2008). Scaffolding occurs throughout the lifespan (Park & Reuter-Lorenz, 2009); it takes place when information is learned, and new circuitry is recruited and used for completing a task (Goh & Park, 2009). However, scaffolding is unique in older adults, such that it occurs not only when new information is learned, but when an older adult faces slightly novel situations or even practices known behaviors, as the previous circuitry deteriorated (Goh & Park, 2009). Park and Bischof (2013) added that it is only when older adults experience significant and sustained demands, that there will be plasticity or scaffolding.

The concept of scaffolding can be broadened further, as compensatory scaffolding. Compensatory scaffolding is the brain's way of maintaining function in the face of neural decline, by increased recruitment of frontal areas, growth and integration of new hippocampus tissue, and greater distribution of processing across various sites (e.g., frontal and/or parietal bilateral activation) (Goh & Park, 2009). The STAC model proposes that older adults can improve their ability to scaffold and develop new circuitry by engaging in new activities, including learning, physical exercise, or even cognitive training (Goh & Park, 2009). The Synapse Intervention Trial (Lodi-Smith & Park, 2011) tested the STAC proposal, as older adults learned a novel, demanding task over the course of three months. Participants were randomly assigned to productive engagement conditions (learning how to quilt and use digital photography), a social control condition (social activities but do not learn new skills), a placebo control (engage in non-challenging tasks, such as listening to music, watching movies), and a no treatment control group. Study results indicated that respondents in the productive engagement condition, but not the social or placebo control conditions, led to improved episodic memory performance.

Projects independent of the Synapse Intervention Trial indicated that engaging in cognitively demanding activities over a period of time improved cognitive functioning and increased intervention-specific increases in brain activity among older adults (e.g., Experience Corps Project; Carlson et al., 2009). However, it has also been found that participation in a variety of tasks, regardless of cognitive demand level, reduces risk of impairment in verbal recall and global cognitive status (Carlson et al., 2012). Thus, Carlson et al.'s (2012) results extend prior theories on cognitive engagement, by raising the importance of variety. Carlson et al. (2012) postulated that the benefit derived from engaging in a variety of activities might result from exercising a greater span of neural circuits.

Overall it is argued through models of successful aging, environmental enrichment, "use it or lose it," cognitive reserve, and compensatory scaffolding that individuals are capable of influencing their cognitive trajectories in late life, through lifestyle choices and activities. The following sections will review more evidence for the effect of cognitive lifestyle and activities on cognitive function.

2.4 Cognitive Lifestyle

A portion of the literature has examined the effect of cognitive reserve, including engaging in activities, under the terms "engaged" or "cognitive lifestyles." For example, early efforts by Schaie's Seattle Longitudinal Study (1984) examined adults' engaged lifestyle (based on job type, social and daily activities, and education) and its association with intellectual changes over seven years. The least intellectual decline was observed by those with high socioeconomic status that were engaged with their environment, while the most intellectual decline was observed in women who were widowed, never employed, and currently exhibited a disengaged lifestyle. However, these findings have been criticized, as Schaie used

socioeconomic status to create different lifestyle levels, and socioeconomic status rather than activity levels per se may have predicted cognitive functioning (Hultsch et al., 1999).

Marioni, Valenzuela, van den Hout, Brayne, and Matthews (2012) examined associations of cognitive lifestyle and older adults' cognitive transitions over time, based on MMSE score (no impairment ≥ 27, slight impairment 23-26, moderate-to-severe impairment ≤ 22). Cognitive lifestyle was defined as education, mid-life occupation complexity (determined by social class and socioeconomic groupings), and late-life social engagement. Findings indicated that a high level of education and complex mid-life occupation were associated with a lowered risk of becoming slightly impaired, from non-impaired, as well as less time with moderate to severe impairment before death. A high level of late-life social engagement was linked to a decreased risk of declining from mild to moderate-to-severe impairment. High education, complex occupation, and social engagement were associated with cognitive recovery (an increased probability of improving from mild impairment to no impairment). Mid-life occupation did not impact cognitive trajectories as much as education. The researchers claimed that an active cognitive lifestyle was associated with compression of cognitive morbidity.

With the same cognitive lifestyle definition, Valenzuela, Brayne, Sachdev, Wilcock, and Matthews (2011) found that the combination of education, occupational complexity, and social engagement, rather than any of these factors alone, predicted dementia risk. Cognitive lifestyle did not predict survival time following diagnosis of dementia, contrary to Stern's (2002) prediction that individuals with higher cognitive reserve experience faster rates of cognitive decline.

2.5 Cognitively Stimulating Activities

The literature on the association between cognitively stimulating activities and cognition is expansive. Much of this research has combined leisurely activities, including hobbies, physical activity for pleasure, social activities, participation in religious organizations, with more intellectually stimulating activities (e.g., reading, writing). For the purposes of the proposed analysis, results from studies that focused on or included cognitively/intellectually stimulating activities will be summarized.

2.5.1 Cognitively stimulating activities and cognitive function in late life. Significant findings will be categorized by three cognitive constructs of interest: working memory, episodic memory, and semantic memory. Working memory represents one's ability to apprehend and hold information in immediate awareness, manipulate this information, and produce a result. Episodic memory, a form of long-term memory, stores information about temporally based events and the temporal relationships among these events (Tulving, 1972). Semantic memory, another form of long-term memory, is the "memory necessary for the use of language," (Tulving, 1972, p. 386), as it stores facts, meanings, concepts, and knowledge that have been acquired. Although these two memory forms are usually studied separately, there is evidence that they are interdependent (for a review, Greenberg & Verfaellie, 2010). Semantic memory facilitates the addition of new episodic memories, and episodic memory facilitates the same process for semantic memory (for a review, Greenberg & Verfaellie, 2010). Similarly, episodic memory facilitates retrieval from semantic memories, and semantic memories are the building blocks for complex episodic memories (for a review, Greenberg & Verfaellie, 2010). Despite their interdependencies, the age trajectories for episodic memory and semantic memory differ. Longitudinal findings indicate that episodic memory gradually deteriorates after age 60, while semantic memory gradually improves until approximately age 55, and deteriorates at a slower rate from this point, relative to episodic

memory (Rönnlund et al., 2005). Working memory, similar to episodic memory, normally declines at a faster rate than semantic memory, with age. These cognitive domains were selected, given their dynamic nature with aging. Early and significant declines in episodic memory may also signal dementia processes.

2.5.1.1 Working memory (or short-term memory). Mitchell et al. (2012) compiled data from four longitudinal studies, the Origins of Variance in the Oldest-Old: Octogenarian Twins Study (Octo-Twin), the Long Beach Longitudinal Study (LBLS), the Seattle Longitudinal Study (SLS), and the Victoria Longitudinal Study (VLS). These studies assessed cognitive performance across similar constructs, including short-term memory (i.e., immediate recall of a story or verbal list). The Octo-Twin Study examined mostly intellectual activities (i.e., playing games and completing puzzles, reading, writing, and doing genealogical research or challenging activities, such as handicraft). Similarly, the LBLS and SLS examined intellectual activities, including participation in educational activities, reading, playing musical instruments, writing, playing games (LBLS only), and engaging in cultural activities. The VLS assessed activities from the Novel Information Processing scale of the VLS Activity Lifestyle Questionnaire, which assessed frequency of engagement in activities such as pursuing further education, writing, studying a second language, completing math calculations, balancing a check book, and playing games (e.g., crosswords, jigsaw puzzles). There was no evidence that baseline intellectual activities predicted change in short-term memory over time. However, across all four studies, changes in intellectual activities from baseline were associated with within-person variability in short-term memory over time.

Wilson et al. (2002) conducted a longitudinal study with 801 Catholic nuns, priests, and brothers without dementia and aged at least 65 years old, at baseline. Cognitive

activities/leisurely activities assessed were "viewing television, listening to radio, reading newspapers, reading magazines, reading books, playing games such as cards, checkers, crosswords, or other puzzles, and going to museums" (p. 743). Engaging in these activities was associated with slower rates of decline in working memory and perceptual speed. Similarly, Bosma et al. (2002) reported that baseline cognitive activity (e.g., playing chess, doing puzzles) predicted subsequent decline on Letter Digit Coding (i.e., respondents presented letter-digit combinations, and later asked to fill in the blanks next to letters, with the correct digits within 90 seconds).

2.5.1.2 Episodic memory. Results from the VLS indicated that declines in cognitive activity (e.g., using the computer, playing bridge) predicted declines in episodic memory (Small et al., 2012). Previously mentioned results from the Synapse Intervention Trial further suggest that practicing and learning a novel task (quilting or using digital photography) over time enhanced episodic memory (Park et al., 2014). Cross-sectional study (Lachman et al., 2010) results revealed that relative to adults that do not perform cognitive activities (i.e., reading, doing games like crosswords, puzzles or scrabble, attending educational lectures or courses, and writing) frequently, adults that do frequent cognitive activities have better episodic memory functioning.

2.5.1.3 Semantic memory. Mitchell et al.'s (2012) multi-study analysis findings also indicated that changes in intellectual activities from baseline were associated with within-person variability on semantic knowledge, in the Octo-Twin, LBLS, SLS, and VLS studies. There was no evidence for baseline level of intellectual activities predicting change in semantic knowledge outcomes. Salthouse (2006) reported results from his own study of cognitively stimulating activities (Salthouse, Berish, & Miles, 2002), in which respondents aged 18 to 97 years old rated

the frequency by which they completed such activities and judged the cognitive demand level of each activity. Participants further completed several tasks, including one that targeted crystallized knowledge or semantic memory (Wechsler Adult Intelligence Scale, Vocabulary subtest; Wechsler, 1955). Participants were divided by the lowest and highest quartiles, in terms of the frequency by which they completed only the most cognitively demanding activities. Performance on the Vocabulary subtest was significantly better for older adults who frequently completed cognitively demanding activities, relative to older adults who completed the least cognitively demanding activities. However, it is important to note that his results were based on cross-sectional data, and therefore, do not reflect cognitive performance over time. Salthouse criticized these results, stating the difference between groups may just reflect different opportunities to learn new information, rather than an actual effect of activities on preserving verbal skills.

Overall, there appears to be evidence for associations between cognitively stimulating tasks and cognitive performance across working memory, episodic memory, and semantic memory domains. However, the amount of evidence is limited and the studies that provide these results vary in methodology and quality. It is unclear if these activity – cognition associations are actually reliable.

2.5.2 Cognitively stimulating activities and dementia risk. There is longitudinal support for the association between engaging in cognitively stimulating activities and reduced or delayed dementia risk (Akbaraly et al., 2009; Paillard-Borg, Fratiglioni, Xu, Winblad, & Wang, 2012; Scarmeas, Levy, Tang, Manly & Stern, 2001; Verghese et al., 2003; Wilson et al., 2002; Wilson et al., 2010). The categorization of activities by Akbaraly et al. (2009) was unique; cognitive activities were classified as "stimulating" (i.e., crosswords, playing cards, attending

organizations, going to movies or theater, and doing artistic activities) and "passive" (i.e., watching television, listening to music or the radio, and knitting/sewing). "Stimulating" activities, when conducted at least twice a week, significantly and independently reduced risk of dementia over four years. "Passive" cognitive activities were not associated with dementia risk. The researchers concluded that "stimulating" activities contribute more to cognitive reserve than "passive" activities.

Wilson et al. (2010) tested Stern's (2002) hypothesis that more cognitive activity would add to cognitive reserve, thus protecting overall cognitive function in spite of developing neuropathology and lead to more rapid cognitive decline following dementia diagnosis. The association between engaging in cognitive activities (i.e., viewing television, listening to the radio, reading newspapers, reading magazines, reading books, playing games, and going to a museum), and a composite cognitive score was examined across three diagnostic groups: no cognitive impairment, mild impairment, and Alzheimer disease (AD). Decline was significantly reduced among older adults without cognitive impairment, when participation in cognitive activities increased. Changes in cognitive decline were not associated with engaging in cognitive activities among older adults with mild cognitive impairment, and cognitive decline was hastened by increases in cognitive activities among older adults with AD. It was concluded that cognitive activity compressed AD morbidity, and helped maintain brain functioning, despite the development of AD.

Despite these appealing findings from Wilson et al. (2010), it is unknown how engagement in activities protects the brain. It is possible that engaging in activities may lead to compensatory scaffolding and neurogenesis (as shown in animal models), greater neuronal density and cortical thickness in the frontal lobe (as shown with high "cognitive lifestyle" scores;

Valenzuela et al., 2012), or decreased risk of hippocampal atrophy (as shown with high "cognitive lifestyle" scores; Valenzuela et al., 2008).

2.5.3 Cognitively stimulating activities predict cognitive performance or vice versa? Research on the direction between activity and cognition teases apart two possibilities: 1) active older adults perform better on cognitive tests because they always had superior cognitive functioning, from 2) older adults perform better cognitively, because they are active. Similarly, Salthouse, Babcock, Skovronek, Mitchell, and Palmon (1990) proposed the hypothesis *differential-preservation*, which predicts that cognitive stimulation has a positive effect on cognitive trajectories, such that people who engage in cognitively stimulating tasks show less cognitive decline relative to inactive individuals. The alternate hypothesis, *preserved-differentiation*, predicts that cognitively active individuals have always performed better than cognitively inactive individuals on cognitive tests (Salthouse et al., 1990). The former would be supported if average activity level significantly predicted rate of cognitive change, while the latter would be supported if activity level did not predict rate of change.

Findings from studies investigating the direction of the relation between cognitive activity and cognitive performance will be organized by those finding a) a reciprocal association, b) associations in which cognitive activity engagement predicted changes in cognitive functioning (supporting *differential-preservation*), and c) associations in which cognitive functioning predicted changes in cognitive activity engagement (supporting *preserved-differentiation*). The findings reported are not limited to working memory, episodic memory, or semantic memory.

2.5.3.1 Reciprocal association. Findings from the six-year Victoria Longitudinal Study (Hultsch et al., 1999) revealed that changes in participation in intellectual engaging activities

("novel information processing activities such as learning a language or playing bridge," p. 248) were associated with changes in working memory. Individuals who decreased their participation in these activities were more likely to experience cognitive changes over time. However, individuals with high intellectual ability also led more intellectually stimulating lives, until they experienced cognitive decline in late life (Hultsch et al., 1999). Findings from the Maastricht Aging Study (Bosma et al., 2002) also indicated that leisure activities and cognitive performance mutually influenced each other in older adults, yet their results were dependent on only two time points, limiting assessment of change over time. Small et al. (2012) found evidence to support that a reduction in cognitive activity (e.g., using the computer, playing bridge) predicted subsequent decline in verbal speed, and that declines in verbal speed also predicted subsequent decline in cognitive activity engagement.

2.5.3.2 Activity predicts cognitive functioning. Continuing with results from Small et al. (2012), greater cognitive activity predicted fewer declines in episodic and semantic memory over time. Wilson et al. (2010) found that higher cognitive activity at baseline was associated with higher baseline composite cognitive score (based on tests of memory, perceptual speed, global cognitive status) as well as more gradual cognitive decline over time. Therefore, cognitive activity was found to be associated with cognitive changes over time. Similarly, Ghisletta, Bickel, and Lövdén (2006) found that increased cognitively stimulating activity reduced decline in perceptual speed over time, but perceptual speed did not have an effect on changes in activity engagement. These findings provide evidence for the protective effect of cognitive activities.

2.5.3.3 Cognitive functioning predicts activity. Bielak, Anstey, Christensen, and Windsor (2012) examined the effect of cognitive and social activities combined. The researchers found that greater activity participation was associated with higher performance for perceptual speed,

short-term memory, working memory, episodic memory, and vocabulary at baseline, for all cohorts (20-24 years old, 40-44 years old, and 60-64 years old). However, changes in activity participation across eight years were not associated to cognitive changes across age groups. The results indicated that active individuals most likely always had good cognitive functioning. It was postulated that it is important have an engaging lifestyle throughout the lifespan, as engaging in activities in late life most likely does not have the same benefit as participating in cognitive activities throughout one's life. It is possible that the difference between Bielak et al.'s (2012) and Wilson et al.'s (2010) findings is due to the fact that the latter study focused on older adults (Bielak et al., 2012).

Overall, evidence for the direction of the activity-cognition association is mixed, and findings may depend on various factors, including the age of the sample and the cognitive domains tested.

2.6 Social Engagement and Cognitive Function

In addition to cognitive stimulation, social engagement is another modifiable factor associated with cognitive trajectories in late life. The following will review literature on associations between social engagement and cognitive functioning. Given the paucity of literature on the direction of this association, results could not be divided by the cognitive constructs of interest (i.e., working memory, episodic memory, and semantic memory), and only research assessing the direction of the association will be reported.

2.6.1 Mechanisms for the social engagement and cognitive functioning association.

An enriched social lifestyle may have a broad effect on health; social contacts can provide assistance through various means (e.g., emotional support, information) (Berkman, Glass, Brisette, & Seeman, 2000), and the act of socializing contributes to purpose and meaning in life

(Krause, 2007). The broad effect of social engagement on health may be observed from epidemiological literature on mortality. For example, Blazer (1982) found that a general lack of social ties with children and siblings was associated with increased mortality risk over a period of 30 months among older adults aged 65 years old and older. Further, in the Evans County Study, mortality risk increased over a 13-year period for older adults with a low number of social ties (Schoenbach, Kaplan, Fredman, & Kleinbaum, 1986). Similarly, men in Tecumseh County, Michigan who reported more social relationships and social activities at baseline were less likely to die nine to 12 years later (House et al., 1982). These mortality studies highlight the significance of social integration for older adults' overall health.

Socializing with others may also be seen as a cognitive exercise as well. In fact, experimental research indicated that executive functioning was positively (yet most likely temporarily) affected by social interactions among young adults (Ybarra, Winkielman, Yeh, Burnstein, & Kavanagh, 2011). Such boosts in executive functioning may result from taking others' perspective, maintaining a plan for the conversation, self-monitoring, and inhibiting oneself from following distractions (Ybarra et al., 2011). It is possible that the positive cognitive effect of socializing with others could occur in older adults (Ybarra et al., 2011).

2.6.3 Social factors and dementia risk. Research has indicated that women with smaller social networks are more likely to develop dementia over time compared to women with larger social networks (Crooks, Lubben, Petitti, Little, & Chiu, 2008). Satisfaction with social networks also had predictive value, as dementia risk was lower among older adults who were "very satisfied" with their networks, relative to those who were "poorly or not satisfied" (Crooks et al., 2008). Cohabitation has been associated with late life cognition; living with a partner may be protective against later cognitive impairment within ages of 65-79 (Hakansson et al., 2009).

Adults who were widowed or divorced during their middle-aged years and continued to have this status in late life were significantly at greater risk for mild cognitive impairment and Alzheimer's disease, relative to adults who cohabited in mid and late life (Hakansson et al., 2009). Similarly, Fratiglioni, Wang, Ericsson, Maytan, and Winblad (2000) found that living alone and having no close personal ties nearly doubled the risk of developing dementia over three years.

2.6.4 Social engagement predicts cognitive performance or vice versa? Research on the direction of the social engagement and cognitive performance association may provide evidence for one or two interpretations. The social transitions (e.g., retirement, loss of a spouse) older adults experience may lead to reduced cognitive stimulation, and thus promote cognitive decline. Alternatively, cognitive decline may also lead to social withdrawal. The few studies on the direction of the social activity-cognition association are reviewed here.

2.6.4.1 Activity predicts cognitive functioning. Longitudinal findings from the Rush Memory and Aging Project (James, Wilson, Barnes, & Bennett, 2011) revealed that older adults who were more socially active were more likely to have higher levels of global cognitive functioning (i.e., composite score of memory, working memory, processing speed, and visuospatial ability) at baseline. Further, greater levels of social activity were associated with less cognitive decline on this global measure following about 5 years. On average, a 1-point increase on a social activity score was linked with a 47% decrease in decline in global cognitive functioning over a year. Social activity was consistently associated with a slower annual rate of change in memory, working memory, perceptual speed, and visuospatial ability. James et al. attempted to rule out reverse causation, or the hypothesis that cognitive decline at baseline may limit social activity, by excluding individuals with MCI. The association between social activity and cognitive functioning persisted in the sample without MCI, indicating that it was unlikely

that poor cognitive function was driving the association. However, it must be noted that this analysis to examine reverse causation (i.e., eliminating individuals with impairment) is not as convincing as other projects that have used advanced statistical models (e.g., latent dual change score models) to assess directionality.

Lövdén, Ghisletta, and Lindenberger's (2005) longitudinal results indicated that changes in social participation predicted changes in perceptual speed, but earlier changes in perceptual speed did not predict subsequent changes in social participation. Lövdén et al.'s (2005) conceptualization of social participation was broad, as it encompassed time spent in leisure activities, instrumental activities, social activities, work activities, and general participation in educational activities and political activities, to name a few. In addition, their research was conducted on the very old (70 years old to 103), and findings regarding perceptual speed in older age groups may not generalize to findings regarding other cognitive abilities in slightly younger age groups (Lövdén et al., 2005).

2.6.4.2 Cognitive functioning predicts activity. Small et al. (2012) found that low episodic memory and low semantic memory predicted declines in social activity, over time. These findings were not in favor of social activities being protective of memory functions; rather, the findings suggest that poor memory predicts social withdrawal.

2.6.4.3 Results incorporating social network. Findings reported by Glei et al. (2005) indicate that social activities, but not social network, were significantly related to cognitive decline in cognitive status (as indicated by the Short Portable Mental Status Questionnaire [SPMSQ]) over time. Older adults who participated in one or two social activities were less likely to perform poorly on the SPMSQ relative to older adults who participated in no social activities. On the contrary, longitudinal analyses by Béland, Zunzunegui, Alvarado, Otero, and

del Ser (2005) revealed that older adults with high levels of family ties and social engagement with relatives maintained better cognitive functioning until age 80, compared to older adults with lower levels of family ties and engagement. Having friends was specifically associated with rate of cognitive change in women. Following age 80, cognitive differences were no longer apparent between groups with high and low family ties and engagement.

In sum, there is evidence that that social activities predict changes in cognitive performance, and vice versa. The paucity of longitudinal research that has examined the association of social activities on cognition prevents one from confidently making conclusions about the direction of the association.

2.8 Limitations of Research on Engagement and Cognition

Despite the large body of research on activity engagement and cognition, there is not enough evidence to understand how activities affect specific cognitive domains and the direction of these specific associations. In addition, the reviewed results are mixed or even controversial. It is believed that differences in the conceptualization of engagement and cognition, methodology, and statistical analysis largely prevent concordant findings across studies. Methodological and analytical limitations of prior research are briefly reviewed below.

2.8.1 Methodological limitations. The definition and operationalization of activity engagement is highly variable. Activity domains are carved in multiple, subjective ways across studies, making it difficult to compare studies and understand the best way activities should be measured. In addition, the activities assessed may not necessarily have much mental stimulation or demand. However, it is difficult to assess the cognitive demand of activities, as the perceived demand may vary by the cognitive ability of the individual (Salthouse, 2006). Questionnaires vary in how many activities are assessed as well as the applicability of the items to most

individuals (Salthouse, 2006). Self-report of participation in activities may also be biased, due to inaccurate memories or social desirability effects (Salthouse, 2006). Further, studies vary on which covariates are measured or included (Ghisletta et al., 2006).

2.8.2 Analytical limitations. According to Small et al. (2012), analytical limitations within the activity-cognition literature include the inability to infer directionality from cross-sectional studies as well as the lack of change data or dynamic models to test directionality and temporal relationships. Although recent research has tended to use longitudinal rather than cross-sectional design to assess activity-cognition associations, longitudinal results have been analyzed in various ways, which may contribute to divergent results across studies. Hierarchical multiple regression, latent longitudinal structural equation models, latent cross-lagged regression models, latent growth models, and dual change score models are all methods previously implemented to analyze longitudinal associations between activity and cognitive performance (Ghisletta et al., 2006). However, despite the similarities between these statistical models, they have different assumptions that could affect result interpretations (Ghisletta et al., 2006). It is also important to examine covariates that may confound the activity-cognition relation.

CHAPTER 3: Methods

3.1 U.S. Health and Retirement Study

This dissertation project is a secondary analysis of data from the U.S. Health and Retirement Study (HRS). The HRS is a nationally representative longitudinal project, funded by the National Institute of Aging and conducted by the University of Michigan Institute for Social Research. The HRS data largely includes variables associated with retirement, economics, and demographics of aging, from about 26,000 initially non-institutionalized adults and spouses aged 50 years old and older. However, the HRS is a multidisciplinary effort, and includes measures of cognitive functioning and activity engagement during its latest years of data collection. HRS data collection began in 1992 from adults aged 51-61 (born between 1931 and 1941). Participants were interviewed biannually, and African Americans and Hispanics were oversampled. Participants that became institutionalized following baseline were contacted and continued in the study, when possible. Data used for this project are from 2008, 2010, and 2012 core surveys, which are available on the HRS website to registered users.

3.2 Participants

Older adults who participated in the HRS in 2008, 2010, and 2012 waves were included if they had data across all three waves and were at least 60 years old. Respondents were not limited to one particular cohort. Respondents were excluded if proxies were used during cognitive testing, if another individual completed their leave behind questionnaire (and it was unknown if the respondent was aware of this), if they reported that a doctor told them they had a "memory-related disease" in 2008, and if they reported that a doctor told them they had "Alzheimer's disease," "dementia, senility or any other serious memory impairment" in 2010

and/or 2012. A total of 3,397 (aged 60+) respondents met inclusion and exclusion criteria. Sample characteristics for this final sample are reported in **Table 1**.

3.3 Procedure and Measures

Interviews were conducted over the phone or face-to-face in respondents' homes. If the respondent had a spouse or partner, he/she was usually interviewed as well. For individuals that could not participate in the interview because of physical or cognitive problems, proxy interviews were conducted. Cognitive tests could not be conducted with proxy respondents, and as such, these respondents were excluded from analyses. Questions regarding individual's psychosocial accounts were provided in a leave-behind questionnaire.

It is important to note that the measures used in the HRS were not consistent across waves, as they were added or removed over time. The following will describe the variables of interest for this project, and indicate the time points during which they were assessed.

3.3.1 Cognition. The HRS provides documentation of the rationale for including tests of specific cognitive domains (see Ofstedal, Fisher, & Herzog, 2005). Psychometric data for the following cognitive measures were not reported in HRS documentation (Ofstedal et al., 2005).

3.3.1.1 Episodic memory (2008, 2010, 2012): Respondents were randomly assigned to one of four possible lists of ten nouns, and were provided a different set of words at each time point to reduce learning effects (Ofstedal et al., 2005). If the respondent's spouse or partner were interviewed as well, both were given different word lists at the same and adjacent time points (Ofstedal et al., 2005). The interviewer read the list of nouns to the respondent once, and the respondent was asked to immediately recall as many as possible in any order (Ofstedal et al., 2005) There was approximately a five minute delay between immediate and delayed recall trials (Ofstedal et al., 2005). Immediate and delayed recall were scored on a 0-10 range.

3.3.1.2 Subjective ratings of memory (2008, 2010, 2012): Respondents were asked to provide a self-rating on their memory, with two items, "First, how would you rate your memory at the present time? Would you say it is excellent, very good, good, fair, or poor?" and "Compared to [the last two years/two years ago], would you say your memory is better now, about the same, or worse now than it was then?"

3.3.1.3 Working memory - Serial 7's (2008, 2010, 2012): The interviewer asked the respondent to subtract 7 from 100, for a total of five trials (Ofstedal et al., 2005). Scores ranged from 0-5.

3.3.1.4 Mental status – Backwards Count, Object Naming, President/Vice President Naming (2008, 2010, 2012): Respondents were asked to count backwards for 10 numbers, starting with the number 20 (scored 0-2). They were asked to name two objects that were described to them in terms of their function or physical characteristics (each scored 0-1; Ofstedal et al., 2005). In addition, respondents were asked to provide the names of the current U.S. President and Vice President (each scored 0-1; Ofstedal et al., 2005). Upon initiating the analyses, it was found that a portion of respondents did not complete items associated with naming. If the respondent's age was less than 65 and they were being re-interviewed, the naming items were skipped. Naming items were administered at every wave, when respondents reached 65 years old and older. As such, not all respondents had responses to the naming items at each wave.

3.3.1.5 Numeracy: Respondents were asked three questions to assess their numerical reasoning skills (Ofstedal et al., 2005). Upon initiating the analyses, it was found that most respondents did not complete Numeracy items in 2008 and 2010, as interview skip logic mandated that the items be skipped if respondents were re-interviewed and 65 years old or older.

As such, Numeracy items were not included in the project analyses.

3.3.2 Psychosocial and Lifestyle Questionnaires (2008, 2012). In addition to the core interviews, respondents were provided self-administered questionnaires on their life circumstances, well-being, and lifestyle (Smith, Fisher, Ryan, Clarke, House, & Weir, 2013). These questionnaires are referred to as Psychosocial and Lifestyle Questionnaires. Information from these questionnaires was obtained at alternate waves, from a rotating and random 50% of the respondents who completed the face-to-face interview (Smith et al., 2013). Therefore, the questionnaire was provided to a random sample in 2008, which was contacted again in 2012 (Smith et al., 2013). For the purposes of this analysis, the 2008 and 2012 questionnaires will be used. Among the randomly selected individuals to complete the psychosocial questionnaire in 2008, the response rate for questionnaire completion was about 89%.

3.3.2.1 Activity engagement. For the current analysis, the 18-item measure *Social Participation – Social Engagement* was of interest, to assess respondents' frequency of engagement in particular activities. The measure was adapted from prior lists of engagement (Hultsch et al., 1999; Jopp & Hertzog, 2010; Levin, 2003; Parslow, Jorm, Christensen, & Mackinnon, 2006; Salthouse et al., 2002). The 2008 activities were ranked on a 6-point scale (1 = *Daily* to 6 = *Not in the last month*), while 2012 activities were ranked on a 7-point scale (1 = *Daily* to 7 = *Never/Not relevant*). To create consistency across time, all activity items were recoded to the following scale, by collapsing response levels: 1 = *Daily to several times a week*, 2 = *Once a week*, 3 = *Several times a month*, 4 = *At least once a month*, 5 = *Not in the last month/Never/Not relevant*.

Scoring for the measure has not been established (Smith et al., 2013). The HRS reported that researchers could create a total sum based on the frequency of activities, or create sum

scores based on varying categories of activities (Smith et al., 2013). As such, no coefficient alpha has been calculated (Smith et al., 2013). For the purposes of the current analyses, single items were implemented to create factors; thus, it was not necessary to create total scores based on multiple items.

3.3.2.1.1 Cognitively stimulating activities. The *Social Participation – Social Engagement* includes several activities that were deemed to be intellectually stimulating. Based on prior ratings of cognitive demand for specific activities from approximately 1,200 adults (Salthouse et al., 2002), items from the *Social Participation – Social Engagement* were selected if the cognitive demand was perceived as mild to high. The selected items along with affiliated cognitive demand ratings from the Salthouse et al. (2002) paper, are reported in **Table 2**.

3.3.2.1.2 Social engagement. Social engagement was also assessed in the 2008 Psychosocial and Lifestyle Questionnaire. Similar to the cognitively stimulating activities, items from the *Social Participation – Social Engagement* were selected if the cognitive demand was perceived as mild to high. The selected items along with the cognitive demand ratings reported from Salthouse et al. (2002) are reported in **Table 3**.

3.3.3 Health (2008, 2010, 2012).

3.3.3.1 Overall health status: Respondents were asked to provide a self-rating on their health, with two items, "Would you say your health is excellent, very good, fair, or poor?" and "Compared with your health when we talked with you in [previous wave month/year] would you say that your health is better now, about the same, or worse?"

3.3.3.2 Lifetime chronic diseases: The HRS asks respondents to indicate if they had a history of a chronic disease during their lifetime, with a list of conditions relevant to older adults. Respondents were asked if they ever had a lifetime history of hypertension, diabetes mellitus,

cancer, chronic lung disease, coronary heart disease, congestive heart failure, stroke, arthritis, or psychiatric problems. To reduce subjectivity or self-diagnosis when asking for self-report data, the core interview asked respondents to report conditions when a doctor told them they had the condition (Fisher et al., 2005).

3.3.3.4 Depressive symptoms – Abbreviated Center for Epidemiologic Studies Depression (CES-D) Scale: The HRS used an abbreviated CES-D scale with nine items. Respondents were asked to consider how much a feeling applied to them during the past week. Responses were coded on a yes/no scale, in addition to "can't do," "don't do," "don't know," and "refused" to respond. No psychometric data were reported on these items. For the current analysis, one of the nine items was used ("felt depressed in the past year"), as respondents were not provided the remaining eight items if they were not depressed in the past year.

3.3.3.5 Sensory functioning: Respondents were asked to rate their current hearing on a 5-point scale ranging from "excellent" to "poor," as well as their vision on a 6-point scale, ranging from "excellent" to "legally blind."

3.3.3.6 Dementia diagnosis: In 2008 core interviews, respondents were asked if a doctor had ever told them they had a "memory-related disease"; responses were coded on a "yes"/"no" scale in addition to "don't know" and "refused" to respond. In 2010 and 2012 core interviews, respondents were asked if a doctor ever told them they had "Alzheimer's disease," "dementia, senility or any other serious memory impairment." Responses were coded on a "yes"/"no" scale in addition to "don't know" and "refused" to respond.

3.4 Data Analysis

3.4.1 Preliminary analyses. The HRS items tended to have additional categories, including "Don't Know" and "Refused." These additional response categories were deleted and

not taken into account for the analyses. Items were selected for latent growth curve models by running principal components analyses (PCAs; on polychoric and Pearson's correlation matrices) with varimax rotation and internal consistencies (Cronbach's alpha, Kuder-Richardson alpha, and ordinal alpha [Gadermann, Guhn, & Zumbo, 2012] where appropriate). PCAs were only conducted when more than two items were considered for a latent factor. PCAs and internal consistency analyses were conducted on Stata, version 13.1 (2013), with the exception of the internal consistency analyses on ordinal data, which were conducted on R (2013). Items were considered to be an indicator for a latent factor if the loading was equal to or greater than .3 on the component explaining the most variance (with an eigenvalue of 1.0 or greater), and if the item's removal did not improve the overall raw alpha.

Selected items were entered into confirmatory factor analyses (CFAs), and then latent growth curve (LGC) models on Mplus 6. LGCs were created with increasing complexity. Intercept models were tested first, followed by intercept and slope models, and lastly the intercept and slope model with time-invariant (years of education) and time-varying covariates (age and sensory functioning).

3.4.2 Latent growth curve analysis. Latent growth curve modeling (LGC) was used to identify the directionality of the relation between activity (cognitive and social) engagement and cognitive functioning (Aim 1.1.1), as well as identify the directionality of the relation between activity (cognitive and social) engagement and overall health (Aim 1.1.3). Path coefficients and standard errors were used to identify statistically significant links between activities and cognitive domains (Aim 1.1.2).

LGC assesses change over time, through two possible routes, structural equation modeling (SEM) or multi-level modeling. The current analysis used SEM to specify LGC

models. The current project aimed to use first- and second-order LGC modeling. First-order LGC estimates change in a particular construct (an observed indicator) that is measured repeatedly across multiple time points. With the first-order LGC modeling framework, the intercept and slope are estimated as latent factors, since they are based on data from multiple time points. Two models are associated with the LGC model framework, including the Level 1 model, which assesses within-person variation across time (i.e., individual trajectories), and the Level 2 model, for between-person variation. The Level 1 model examines trajectories with an intercept (the average level of the construct at the starting time point) and a slope (average change in the construct over at least three time points). The Level 2 model attempts to explain differences in individual growth trajectories by incorporating predictors of change. Since first-order LGC only uses observed indicators for the construct of interest, the analysis is subject to measurement and model estimation errors.

Second-order LGC reduces measurement error, as it uses multiple measurements of the construct of interest to create a latent factor at a particular time point. As such, second-order LGCs assess change in latent factors, rather than observed indicators. The latent factors measured over time are the first-order factors; the intercept and slope are the second-order factors (Preacher, Wichman, MacCallum, & Briggs, 2008).

3.4.2.1 Measurement model. The primary analysis attempted to implement six second-order LGC models assessing activity engagement and cognitive functioning (or health). Separate latent factors were created for cognitive and social engagement, for all six models. The first three LGC models would include latent factors for working memory, episodic memory, and semantic memory across three time points (i.e., 2008, 2010, and 2012). The second three LGC models would attempt to include a single latent factor for health, based on multiple observed variables.

3.4.2.2 Structural model. The first aim of this project was to analyze longitudinal associations between activity engagement and cognitive performance, as well as longitudinal associations between activity engagement and health. The impact of two activity latent factors (i.e., frequency of participation in cognitive and social activities) on the second-order slope and intercept of the three cognitive latent factors would be examined. Similarly, the impact of the two latent factors for activity frequency (cognitive and social) on the slope and intercept of the second-order health latent factor would be observed. Three models explored the cognitive and social activity association, with the first model estimating a correlation between activity latent factors, the second model estimating social engagement as a predictor of cognitively stimulating activities, and a third model estimating participation in cognitively stimulating activities as a predictor of social engagement.

Evidence for the direction of the association between activity engagement and cognitive performance (and health, in other models) would be ascertained from the direction and magnitude of the direct effects between activity and cognitive latent factors. The direct effects would also be used to determine differential links between activity engagement and performance on the three cognitive domains, as well as determine which activity (cognitive vs. social) would predict cognitive and health change.

3.4.3 Latent factor cross-lagged panel model. In addition to identifying longitudinal associations between activity frequency and health, Aim 1.1.3 proposed to identify trends indicative of a causal relation between cognitive functioning and health. A latent factor cross-lagged panel model would be used to estimate the effect of each latent cognitive factor (i.e., working memory, episodic memory, and semantic memory) on a latent health factor (and vice versa) across three time points.

3.4.4 Model evaluation. All models were evaluated based on how well they describe the data, through the chi-square statistic, root mean square error of approximation (RMSEA) and a goodness of fit index (i.e., comparative fit index [CFI]). Both unstandardized and standardized coefficients were reported. When results differed between unstandardized and standardized findings, which occurred infrequently, the unstandardized findings were favored. Given that unstandardized and standardized coefficients are based on different sampling distributions, differences in findings may occur.

Statistics for comparing non-nested models (i.e., Akaike information criterion [AIC]) were reported when possible. No common test statistic could be used to compare all models in the current project, as many models implemented an estimator (i.e., weighted least squares mean and variance adjusted; WLSMV) that does not generate an AIC. Models of varying complexity with similar indicators were generally compared by model fit (e.g., RMSEA, CFI) and significance of model paths.

CHAPTER 4: Results

4.1 Descriptive Statistics

Descriptive statistics for selected items are presented in **Table 4**. **Table 5** presents pairwise correlations between cognitive functioning items and potential covariates (age, education, vision ratings, and hearing ratings). **Table 6** presents pairwise correlations between education and activity level items in 2008.

4.2 PCAs and Internal Consistencies

4.2.1 Episodic memory 2008. Items for immediate recall, delayed recall, and subjective memory ratings (rate memory at present time [LD101], rate change in memory over time [LD102]) were entered into a PCA with three components specified. Two indicators (immediate [LD174] and delayed [LD184] recall in 2008) loaded highly onto the first component (eigenvalue of 1.85, explaining 46% of variance), another indicator ("rate memory at present time" [LD101]) loaded highly onto the first and second (eigenvalue of 1.28, explaining 32% of variance) components, and the last indicator ("rate change in memory over time" [LD102]) loaded highly onto the second and third (eigenvalue of .58, explaining 15% of variance) components. After varimax rotation, immediate recall and delayed recall loaded onto the first component, with loadings of .71, each. Subjective ratings for present memory (LD101) and change in memory over time (LD102) loaded onto the third and second components after varimax rotation, respectively. Consequently, these subjective memory variables were not included in further analyses. The pairwise correlation between immediate recall (LD174) and delayed recall (LD184) was .72 ($p < .001$); Cronbach's alpha for these two items was .83, indicating good internal consistency.

4.2.2 Episodic memory 2010. The pairwise correlation between immediate recall (MD174) and delayed recall (MD184) was .76 ($p < .001$), indicating that 58% of the variance between the two items was shared. The Cronbach's alpha for immediate recall (MD174) and delayed recall (MD184) was .85, indicating good internal consistency.

4.2.3 Episodic memory 2012. The pairwise correlation between immediate recall (ND174) and delayed recall (ND184) was .74 ($p < .001$), indicating that 55% of the variance between the two items was shared. The Cronbach's alpha of these two items was .84, indicating good internal consistency.

4.2.4 Working memory 2008. The pairwise correlation between totals for backwards counting (LD124) and serial 7s was .12 ($p < .001$). The ordinal alpha for backwards counting (LD124) and serial 7s was .41, indicating poor internal consistency. As such, these items were not considered to be indicators for a single latent factor, working memory in 2008.

4.2.5 Working memory 2010. The pairwise correlation between totals for backwards counting (MD124) and serial 7s was .16 ($p < .001$). The ordinal alpha for backwards counting (MD124) and serial 7s was .47, indicating poor internal consistency. As such, these items were not considered to be indicators for a single latent factor, working memory in 2010.

4.2.6 Working memory 2012. The pairwise correlation between totals for backwards counting (ND124) and serial 7s was .18 ($p < .001$). The ordinal alpha for backwards counting (ND124) and serial7s was .49, indicating poor internal consistency. As such, these items were not considered to be indicators for a single latent factor, working memory in 2012.

4.2.7 Semantic memory 2008. Out of the four possible items testing semantic memory, two had little variation across binary response levels (naming "scissors" [LD155] and naming the "President of the United States" [LD157]) and were consequently eliminated from the analyses.

The remaining two items, naming "cactus" (LD156) and the "Vice President" (LD158) were correlated at .15 ($p < .001$). Given that these variables were binary (i.e., correct/incorrect), the Kuder-Richardson coefficient was used as a measure of internal consistency. The Kuder-Richardson coefficient was .23, indicating poor consistency. Thus, these two items were not considered to be indicators for a single latent factor, semantic memory in 2008.

4.2.8 Semantic memory 2010. The correlation between naming "cactus" (MD156) and the "Vice President" (MD158) was .11 ($p < .001$). The Kuder-Richardson coefficient was .15 indicating poor consistency. Thus, these two items were not considered to be indicators for a single latent factor, semantic memory in 2010.

4.2.9 Semantic memory 2012. The correlation between naming "cactus" (ND156) and "Vice President" (ND158) was .16 ($p < .001$). The Kuder-Richardson coefficient was .22, indicating poor internal consistency. Thus, these two items were not likely indicators for a single latent factor, semantic memory in 2012.

4.2.10 Health 2008. Ten items (rate health [LC001], high blood pressure [LC005], diabetes [LC010], cancer [LC018], lung disease [LC030], heart condition [LC036], stroke [LC053], emotional/psychiatric problems [LC065], arthritis [LC070], and depressed in past year [LC150]) were entered into a PCA from a polychoric correlation matrix, with three possible components specified. Seven loaded onto the first component (eigenvalue of 2.67, explaining 27% of variance). Items for cancer (except for skin), stroke, and arthritis had loadings lower than .30 on the first component. After varimax rotation, items for rating health, high blood pressure, diabetes, heart condition, and stroke had their highest loadings (all > .30) on the first component. Cancer's loading on the first component remained < .30 and had a high loading (.71) on the third component (eigenvalue = 1.13). Similarly, lung disease's and arthritis' loading on

the first component was < .30 and had their highest value also on the third component (.44 and .28, respectively). Items for emotional/psychiatric problems and depressed in past year, had low loadings (< .30) on the first component and had high loadings (> .60) on the second component (eigenvalue = 1.36). Given that the initial component's eigenvalue was substantially higher than the remaining components' eigenvalues, all items were considered for the latent health construct, with the exception of cancer, stroke, and arthritis. The Cronbach's alpha of the seven items was .50; the deletion of any of the items resulting in a higher alpha was not indicated.

Given its low internal consistency, it was deemed that the physical health construct would be better achieved as a single indicator, representing the sum of health conditions reported by the respondent. Six physical health indicators were summed, high blood pressure (LC005), diabetes (LC010), lung disease (LC030), heart condition (LC036), emotional/psychiatric problems (LC065), and depressed in past year (LC150), since these items all had the same binary code (yes/no). Further PCAs were not conducted for years 2010 and 2012 given the decision to implement a summed index score rather than a latent variable for physical health.

4.2.11 Cognitive activities 2008. Seven items were entered into a PCA from a polychoric correlation matrix, with three possible components specified. Six of the seven items loaded onto the first component (eigenvalue of 2.43, explaining 35% of variance). Playing chess or cards (LLB001j) had cross loadings on the second (eigenvalue of 1.24, explaining 18% of variance) and third (eigenvalue of .84, explaining 12% of variance) components. After varimax rotation, items pertaining to taking educational courses (LLB001d), writing (LLB001k), using the computer (LLB001l), and doing a hobby (LLB001p) loaded onto the first component, items pertaining to reading (LLB001h) and playing word games (LLB001i) loaded onto the second component, and an item pertaining to playing chess or cards (LLB001j) loaded onto a third

component. For simplicity, the items that loaded onto the first component were considered to be likely indicators of a latent factor for general cognitive activities. The ordinal alpha of the four items was .63, indicating fair internal consistency.

4.2.12 Cognitive activities 2012. The same four items selected from the 2008 cognitive activities for the latent factor, cognitive activities, were selected from the 2012 questionnaire (i.e., "taking educational courses" (NLB001E), "writing" (NLB001M), "using the computer" (NLB001N), and "doing a hobby" (NLB001R) and entered into a PCA from a polychoric correlation matrix with three possible components. The four items loaded onto the first component (eigenvalue = 1.82, explaining 46% of variance) yet all also had high cross-loadings onto either a second or third component (eigenvalues of .77 and .71, respectively). Following varimax rotation, "taking educational courses" and "using the computer" loaded onto the first component, "writing" loaded onto the third component, and "doing a hobby" loaded onto the second component. Given that all of the items loaded onto the first component before varimax rotation, it was deemed that they might have enough similarity to be considered indicators of a single latent factor. The ordinal alpha for the four items was .60, indicating fair internal consistency.

4.2.13 Social activities 2008. Five items were entered into a PCA, from a polychoric correlation matrix, with three components specified. Four of the five items (i.e., "volunteering with youth" [LLB001b], "doing other volunteer or charity work" [LLB001c], "attend sports, social, or other clubs" [LLB001e], and "participating in a non-religious organization" [LLB001f]) loaded onto the first component (eigenvalue of 2.06, explaining 41% of variance). The fifth item pertaining to "taking care of an adult" (LLB001a) loaded onto the second and third components (eigenvalues of 1.07 and .81, respectively). Following varimax rotation, "taking care

of an adult" loaded highly on the third component, "volunteering with youth" and "doing other volunteer or charity work" loaded onto the second component, and "attending sports, social, or other clubs" and "participating in a non-religious organization" loaded onto the first component. However, given the high loadings on the first component, prior to varimax rotation, "volunteering with youth," "doing other volunteer or charity work," "attend sports, social, or other clubs," and "participating in a non-religious organization" were considered to be indicators of a single latent factor, social activities in 2008. The ordinal alpha of the four items was .66, indicating fair consistency.

4.2.14 Social activities 2012. The same four items selected from the 2008 social activities for the latent factor, social activities, were selected from the 2012 questionnaire (i.e., "volunteering with youth" [NLB001C], "other volunteer or charity work" [NLB001D], "attend sports, social, or other clubs" [NLB001F], and "attend non-religious organizations" [NLB001G]) and entered into a PCA from a polychoric correlation matrix with three possible components. All four items loaded onto the first component (eigenvalue of 2.13, explaining 53% of variance), yet all had high cross-loadings onto the second and/or third components (eigenvalues of .87 and .55, respectively). Following varimax rotation, item "volunteering with youth" loaded onto the second component, items "other volunteer or charity work" and "attend non-religious organizations" loaded onto the first component, and item "attend sports, social, or other clubs" loaded onto the third component. Despite the differences in loadings on components following varimax rotation, they were considered to be indicators for a single latent factor, social activities in 2012, given their loadings onto the first component prior to rotation. The ordinal alpha of the four items was .70, indicating an acceptable level of internal consistency.

For the remainder of the analyses, items pertaining to serial 7s and naming the "Vice President," were used as single indicators of working memory and semantic memory, respectively. These items were specifically chosen given their greater level of variability (e.g., serial 7s in 2008 *SD* = 1.60; naming the "Vice President" in 2008 *SD* = .38), relative to either backwards counting (in 2008, *SD* = .20) or naming "cactus" (e.g., in 2008, *SD* = .20) items.

4.3 Test-retest Reliability of Indicators Over Time

Intraclass correlation coefficients (ICCs) were used to assess the test-retest reliability of indicators over time. In the context of longitudinal data, individual ICCs reflect the reliability of individual ratings over time, and average ICCs reflect the average of ratings over time. Although both individual and average are reported here, the individual ICCs are more commonly reported and will be used for making conclusions regarding the test-retest reliability of indicators.

A fair degree of reliability was found across immediate recall performances over time, as the individual intraclass correlation coefficient (ICC) was .43 (95% CI .41-.46) and average intraclass correlation coefficient (ICC) was .70 (95% CI .68-.71). Similarly, the individual ICC for delayed recall performances over time was .48 (95% CI .46-.50) and the average ICC was .73 (95% CI .72-.75). The ICC for serial 7's performance was slightly better than the recall ICCs; the individual ICC was .65 (95% CI .64-.67) and the average ICC was .85 (95% CI .84-.86). The individual ICC for naming was .49 (95% CI .47-.51) and the average ICC was .74 (95% CI .73-.76). Given the two-year lapse between measurement points, the test-retest reliability of the cognitive indicators over time was modest.

4.4 Confirmatory Factor Analyses

The construction of latent factors was considered for cognitive activity frequency (2008, 2012), and social activity frequency (2008, 2012). Latent factors were also considered for

episodic memory, yet CFAs could not be conducted, as they would not be identified with two indicators. Latent factors could not be considered for working and semantic memory, given that these cognitive constructs would be analyzed with single observable indicators. CFAs were next employed to test the structure of potential cognitive and social activity latent factors across time points, 2008 and 2012. Models' chi-square fit indices were reported yet not given much interpretative consideration, as these indices are often statistically significant ($p < .05$) in analyses with large sample sizes.

4.4.1 Cognitive activities 2008 (CA08). A CFA was specified, with "taking educational courses," "writing," "using the computer," and "doing a hobby" as indicators to define the latent factor, cognitive activities in 2008. The mean and variance of the latent factor was fixed to zero and one, respectively, so that the first indicator could be freely estimated. The model fit the data well ($\chi^2 = 8.69$, $df = 2$, $p = 0.01$, RMSEA 90% C.I. = .01-.05, CFI = .99). Unstandardized coefficients are listed by item: "taking educational courses" (b = .49, SE = .04), "writing" (b = .61, SE = .03), "using the computer" (b = .54, SE = .03) and "doing a hobby" (b = .54, SE = .03)

4.4.2 Cognitive activities 2012 (CA12). Indicators "taking educational courses," "writing," "using the computer," and "doing a hobby" from the 2012 wave were entered to define the latent factor, cognitive activities in 2012. The mean and variance of the latent factor was fixed to zero and one, respectively, so that the first indicator could be freely estimated. The model fit the data well ($\chi^2 = 2.46$; $df = 2$; $p = 0.29$; RMSEA 90% C.I. = .00-.04; CFI = .99).

4.4.3 Social activities 2008 (SA08). Indicators "volunteering with youth," "other volunteer or charity work," "attend sports, social, or other clubs," and "attend non-religious organizations" were entered to define the latent factor, social activities in 2008. The mean and

variance of the latent factor was fixed to zero and one, respectively, so that the first indicator could be freely estimated. The model fit with the data was fair (χ^2 = 66.68; *df* = 2; *p* = 0.00; RMSEA 90% C.I. = .08-.12; CFI = .95). Unstandardized coefficients are listed by item: "volunteering with youth" (b = .44, SE = .03), "other volunteer or charity work" (b = .65, SE = .02), "attend sports, social, or other clubs" (b = .51, SE = .03) and "attend non-religious organizations" (b = .76, SE = .03)

4.4.4 Social activities 2012 (SA12). Indicators "volunteering with youth," "other volunteer or charity work," "attend sports, social, or other clubs," and "attend non-religious organizations" from the 2012 wave were entered to define the latent factor, social activities in 2012. The mean and variance of the latent factor was fixed to zero and one, respectively, so that the first indicator could be freely estimated. The model fit with the data was poor (χ^2 = 74.81; *df* = 2; *p* = 0.00; RMSEA 90% C.I. = .09 - .13; CFI = .94).

4.4.5 Cognitive and social activities in 2008. Three models were generated to assess the best structural model for cognitive and social activity latent factors (covarying latent factors vs. SA08 predicting CA08 vs. CA08 predicting SA08). The latent factors, CA08 and SA08, were first entered as covarying factors. The means and variances of the latent factors were fixed to zero and one, respectively, so that their first indicators could be freely estimated. The first indicator for each factor was freed. The model's fit (χ^2 = 205.03 *df* = 19, *p* = .00; RMSEA 90% C.I. = .05-.06; CFI = .94) to the data was modest. The activity latent factors significantly covaried (*cov* = .78, SE = .03, *p* < .001).

The model was repeated with SA08 predicting CA08. The model fit was the same as the previous model (CA08 and SA08 covarying). However, the loadings for CA08 indicators decreased. SA08 significantly predicted CA08 (*cov* = 1.24, SE = .10, *p* < .001). The converse

was also examined, with CA08 predicting SA08. The model fit was identical to the prior models. The individual item loadings for social activities decreased, relative to the initial model. Cognitive activities significantly predicted social activities (*cov* = 1.24, SE = .10, $p < .001$).

Given that the first model had the highest indicator loadings, the latent activity frequency factors will covary within more complex models. All three models had similar standard errors.

4.5 Latent Growth Curve Modeling – Episodic Memory and Activity Frequency

LGC modeling was used to examine the effect of activities on the level and rate of change in cognitive functioning among older adults across three time points.

Measurement invariance for episodic memory (EM) latent factors was first assessed, by specifying three latent factors (EM08, EM10, and EM12) with immediate and delayed recall as indicators. Errors between adjacent, similar indicators covaried over time. Factor loadings of the indicators on the latent factors were non-invariant (no constraints). The resulting chi-square was poor, $\chi^2 = 108.69$, $df = 2$, $p < .001$, as well as the remaining fit indices, RMSEA 90% C.I. = .11-.15; CFI = .99.

The second model was specified to test weak factorial invariance. The factor loadings for the second indicator of each episodic memory latent factor were held equal over time. The resulting fit remained poor, $\chi^2 = 116.84$, $df = 4$, $p < .001$, RMSEA 90% C.I. = .08-.11, CFI = .99. A chi-square difference test revealed that this model significantly differed from the original model, suggesting that the less restrictive model fit significantly better. The relaxation of a loading on one of two indicators for a latent factor could impose difficulties interpreting the latent factor itself. Consequently, a first order-LGC model, rather than a second-order LGC model, was considered, as measurement invariance could not be achieved. Different models were tested for immediate and delayed recall; findings regarding immediate recall are presented first.

4.4.1 Immediate recall. An intercept-only model was specified, using the intercept as a latent factor, and three indicators of immediate recall from each time point (2008, 2010, and 2012). All factor loadings were fixed to one. This model fit the data (χ^2 = 275.02; *df* = 4; *p* = .00; RMSEA 90% C.I. = .13-.16; CFI = .85; AIC = 36386.14) poorly. **Table 7** presents detailed results from this model.

A slope latent factor was added, with linear factor loadings (i.e., 0, 2, 4). The intercept was not permitted to covary with the slope, given that it would become a Heywood case (i.e., negative error variance) if covariation was permitted. The resulting chi-square was unacceptable, χ^2 = 22.58, *df* = 2, *p* = .00, yet the remaining fit indices were acceptable, RMSEA 90% C.I. = .04-.08; CFI = .99 (AIC = 36137.70). The means for the intercept and slope were significant, indicating that they were non-zero. The variance for the intercept, but not the slope, was statistically significant, indicating significant individual variability around the initial immediate recall score, but not around the mean slope or growth rate. All observed indicator residual variances were significant, suggesting further variance to be explained. The model did a fair job in accounting for variance in the observed variables, per R^2 (ranging from 39% - 51%). **Table 8** presents detailed results from this model.

In the third analysis, cognitive and social activities from 2008 were entered as two separate latent factors. Although the chi-square was unacceptable, χ^2 = 437.64, *df* = 41, *p* < .001, the remaining fit indices suggested modest fit, RMSEA 90% C.I. = .05-.06; CFI = .93). The means for the activity latent factors could not be estimated, yet their variances were statistically significant. The unstandardized intercepts for the intercept and the slope were statistically significant. Residual variances for the intercept and observed indicators were statistically significant, indicating that there was additional variance to be explained. Residual variance for

slope was not statistically significant. The model fairly accounted for variance in the observed variables and intercept (R^2 ranging from 37% to 50%), with the exception of slope (R^2 = 9%). The frequency of cognitive activities at 2008 predicted the intercept, such that greater engagement in cognitive activities was associated with higher immediate recall scores at baseline. The frequency of social activities at 2008 also predicted the intercept; however, lower engagement in social activities was associated with higher immediate recall scores at baseline. CA08 and SA08 did not significantly predict the slope. **Table 9** presents detailed results from this model.

In the last model, the intercept was regressed onto years of school. The model fit worsened; (χ^2 = 2115.92; *df* = 51; $p < .001$; RMSEA 90% C.I. = .11; CFI = .66). The intercept was significantly regressed onto years of school. The intercept of the intercept, and the intercept of the slope were statistically significant, indicating that they were non-zero. Residual variances for the intercept and observed indicators were statistically significant, indicating that there was additional variance to be explained; however, the residual variance for the slope was non-significant. Variances for cognitive and social activities were statistically significant. The model explained adequate variance in the observed variables and the intercept, but not the slope (5%). The frequencies of cognitive and social activities covaried (unstandardized estimate = .21, $p < .001$). The frequencies of cognitive activities and social activities at 2008 continued to predict the intercept, in directions specified in the earlier model. The frequency of cognitive and social activities at 2008 did not significantly predict the slope. **Table 10** presents detailed results from this model.

Time-varying covariates, age and sensory ratings, were next added to the model. The addition of covariates substantially altered the means and intercepts of the immediate recall

indicators, implicating issues with multicollinearity. As such, results from these models are not discussed. **Figure 1** graphically displays a template of the final model.

4.4.2 Delayed recall. A similar intercept-only model was specified for delayed recall. The model fit the data (χ^2 = 270.75; *df* = 4; $p < .001$; RMSEA 90% C.I. = .13-.16; CFI = .89; AIC = 39,612.28) poorly. **Table 11** presents detailed results from this model.

A linear slope latent factor was added to improve the model. The intercept and the slope were not permitted to covary, due to issues with a Heywood case (i.e., negative error variance), with covariation. Although the chi-square was unacceptable, the remainder of the fit indices suggested that the model fit the data well (χ^2 = 9.07; *df* = 2; $p = .01$; RMSEA 90% C.I. = .01-.06; CFI = 1.00; AIC = 39,354.60). The means for the intercept and slope were significant, indicating that they were non-zero. There was significant individual variation around the intercept, but not the slope, indicating that not all respondents' had the same initial delayed recall score, yet they had the same growth rate. All observed indicator residual variances were statistically significant, indicating additional variance to be explained. Furthermore, the model fairly accounted for the variance in the observed variables (R^2 ranging from 45% to 57%). **Table 12** presents detailed results from this model.

In the third analysis, CA08 and SA08 were entered as two separate latent factors predicting the intercept and slope of delayed recall. Although the chi-square suggested poor fit, the remainder of the fit indices indicated modest fit (χ^2 = 431.25; *df* = 41; $p < .001$; RMSEA 90% C.I. = .05-.06; CFI = .93). The means for the activity latent factors could not be estimated, yet their variances were statistically significant. The intercepts for the intercept and slope were statistically significant, indicating that they were non-zero. Residual variances for the intercept and observed indicators were statistically significant, with the exception of slope. The model

accounted for a fair portion of variance in the observed variables and intercept, but not in slope (1%). CA08 and SA08 were correlated (standardized estimate = .77, $p < .001$; unstandardized covariation = .19, $p < .001$). The CA08 and SA08 predicted the intercept. Greater CA08 was associated with higher delayed recall scores and lower SA08 was associated with lower delayed recall scores, at baseline. The CA08 and SA08 did not significantly predict the slope. **Table 13** presents detailed results from this model.

In the fourth analysis, a time invariant covariate, years of school, was added to improve model fit. The indicator years of school was specified to predict the intercept of delayed recall. The addition of the covariate worsened model fit, $\chi^2 = 2{,}109.34$, $df = 51$, $p < .001$; RMSEA 90% C.I. = .11, CFI = .65). The intercepts for the intercept and the slope were statistically significant. Variances for CA08 and SA08 were statistically significant. Residual variances for the intercept and observed indicators were statistically significant; the residual variance for the slope was no longer statistically significant, indicating that no further variance could be explained in the slope latent factor. The model accounted for a fair portion of variance in the observed indicators and intercept, but not in slope (<1%). Number of school years significantly predicted the intercept of delayed recall. CA08 and SA08 continued to significantly predict the intercept in the directions specified in the prior model. Neither CA08 nor SA08 predicted slope of delayed recall. **Table 14** presents detailed results from this model.

Time-varying covariates, age and sensory ratings, were next added to the model. The addition of covariates substantially altered the means and intercepts of the delayed recall variables, implicating issues with multicollinearity. As such, results from these models are not discussed. **Figure 1** graphically displays a template of the final model.

4.5 Latent Difference Modeling – Episodic Memory and Activity Frequency

4.5.1 Measurement invariance for CA08 and CA12. To test for measurement invariance across time, a CFA including CA08 and CA12 was specified, with both latent factors covarying. Errors across similar indicators over time covaried. The first model was specified without invariance (no constraints or fixed parameters). The resulting chi-square and remaining fit indices were acceptable, χ^2 = 25.07, *df* = 15, *p* = .05; RMSEA 90% C.I. = .001-.02; CFI = 1.00.

A second model was specified, with factor loadings held equivalent across latent factors. Again, the resulting chi-square and remaining fit indices were acceptable, χ^2 = 27.30, *df* = 18, *p* = .07; RMSEA 90% C.I. = .00-.02; CFI = 1.00. The chi-square (and *df*) difference between these two initial models was not statistically significant, indicating invariance at the factor loading level.

Next, intercepts were made equivalent across latent factors. The intercepts of the first indicator of each factor were set to zero, and the remaining intercepts were set equal over time. An estimation of the latent means was requested, with the latent mean for CA12 freed. The chi-square was unacceptable, χ^2 = 158.63, *df* = 17, *p* < .001, yet the remaining fit indices were modestly acceptable, RMSEA 90% C.I. = .04-.06; CFI = 1.00. The chi-square was considered statistically significant, indicating that the former model with only invariant factor loadings fit the data relatively better.

Partial invariance at the intercept level was assessed, by examining variables with the highest modification indices in terms of intercepts. When the intercept for "hobby" in 2012 was freed, the chi-square lowered (χ^2 = 36.07, *df* = 16, *p* < .001), yet the model still statistically differed from the model with invariant factor loadings. Next, the intercepts for "writing" in 2008, in addition to the intercept for "hobby" in 2012 were freed, resulting in an acceptable chi-square

that did not statistically differ from the model with invariant factor loadings, $\chi^2 = 25.07$, $df = 15$, $p = .05$. The remaining fit indices were also acceptable (RMSEA 90% C.I. = .001-.02; CFI = 1.00). This invariance structure will be used for upcoming models. **Table 15** presents detailed results from this test of measurement invariance.

A SEM model was next specified to test if CA08 predicted CA12. Error variances of similar items across time were allowed to covary. Factor loadings were held invariant and thresholds were held with partial invariance, in tandem, to create strong partial measurement invariance over time. The model fit the data well, $\chi^2 = 25.07$, $df = 15$, $p = .05$, RMSEA 90% C.I. = .001-.02; CFI = 1.00. CA08 significantly predicted CA12 (unstandardized estimate = .72, $p < .001$). Observed indicators significantly covaried over time. The variance of CA08 was significant (unstandardized estimate = .24, SE = .03, $p < .001$), and the residual variance of CA12 was significant (unstandardized estimate = .05, SE = .01, $p < .001$).

4.5.2 Latent difference model for cognitive activities. A latent difference model was next specified (Geiser, 2013). CA08 was specified to predict CA12. A "difference" latent factor was constructed, which predicted CA12 and covaried with CA08. The variance of CA08 and the difference factor were fixed to one, while the variance of CA12 was fixed to zero, allowing the difference factor to reflect the mean difference between CA12 and CA08. The means of CA08 and the difference factor were requested. Error variances of similar activity items across time were allowed to covary. Factor loadings were held invariant and thresholds were held with partial invariance, in tandem, to create strong partial measurement invariance over time. **Figure 2** graphically displays a template of the proposed model.

The model fit the data well, $\chi^2 = 25.07$, $df = 15$, $p < .001$; RMSEA 90% C.I. = .001-.02; CFI = 1.00. The estimated means of CA08 and CA12 were 1.08 and 1.09, respectively, leading

to a mean difference factor of .01, or decrease in activities. The mean for the difference factor was not statistically significant, indicating that it was approaching zero and little to no change had occurred across the two time points. The variance of CA08 and the difference factor were statistically significant. The residual variance for CA12 was fixed and could not be freely estimated. Given that there was no change in cognitive activity frequency from 2008 to 2012, an episodic memory indicator could not be used to predict change. **Table 16** presents detailed results from this model.

4.5.3 Measurement invariance for SA08 and SA12. Testing for measurement invariance was initiated by specifying a CFA including SA08 and SA12, with errors of similar indicators covarying over time. The first model was specified without invariance (no constraints or fixed parameters), and the resulting chi-square and remaining fit indices suggested fair fit, χ^2 = 201.09, df = 15, p = .05; RMSEA 90% C.I. = .05-.07; CFI = .98.

The second model was specified with the factor loadings held equivalent across latent factors. Again, the resulting chi-square was unacceptable, χ^2 = 196.61, df = 18, p < .001, and the remaining fit indices suggested that the fit was modest, RMSEA 90% C.I. = .05-.06; CFI = .98. The difference between the first and second models' chi-squares (and degrees of freedom) was not significant, indicating invariance at the factor loading level.

Next, the intercepts were made equivalent across latent factors. The intercepts of the first indicator of each factor were set to zero, and the remaining intercepts were set equal over time. An estimation of the latent means was requested, with the latent mean for SA12 freed. The chi-square was unacceptable, χ^2 = 200.90, df = 17, p < .001, yet the remaining fit indices were modestly acceptable, RMSEA 90% C.I. = .05-.06; CFI = .98. A chi-square difference test

indicated a significant difference between the current model and the weak factorial invariant model, preferring the latter.

Partial invariance at the intercept level was considered. Modification indices indicated that "volunteer with youth" in 2012 had the highest modification index for intercept; as such, the indicator was freed, yet the chi-square difference test continued to reveal that the less restrictive model, or the weak factorial invariance model, was preferred. Modification indices did not provide further clear suggestions for relaxing the intercepts; however, when any of the remaining intercepts were relaxed, the chi-square and *df* were reduced in an identical manner. Thus, the intercept for "clubs" in 2008 was arbitrarily relaxed (χ^2 = 201.09, *df* = 15, $p < .001$; RMSEA 90% C.I. = .05-.07; CFI = .98). A chi-square difference test indicated that this latest model did not fit the data significantly worse than the model with weak factorial invariance. As such, this invariance structure was used for upcoming models. **Table 17** presents results from the models tested for measurement invariance.

A SEM model was next specified first to test if SA08 predicted SA12. Partial measurement variance was implemented and error variances of similar items across time were allowed to covary. The model fit the data modestly, χ^2 = 201.09; *df* = 15; *p* = .00; RMSEA 90% C.I. = .05-.07; CFI = .98, and SA08 significantly predicted SA12 (unstandardized estimate = .74, $p < .001$). Indictors across time were significantly correlated. The variance of SA08 was significant (unstandardized estimate = .46, $p < .001$), and the residual variance of SA12 was significant (unstandardized estimate = .21, $p < .05$).

4.5.4 Latent difference model for social activities. A second series of latent difference models were specified, for social activities. SA08 was specified to predict SA12. A difference latent factor was constructed, which predicted SA12 and covaried with SA08, and represented

the difference between these two factors. Error variances of similar indicators across time were allowed to covary.

Irrespective of the chi-square, the model fit the data modestly, χ^2 = 201.09, *df* = 15, *p* < .001; RMSEA 90% C.I. = .05-.07; CFI = .98. The estimated means of SA08 and SA12 were .41 and .58, respectively, leading to a mean difference factor of .17, indicating an average decrease in activities. The mean of the difference factor was not statistically significant. Factor variances for SA08 and the difference factor were significant. Given that there was no significant change in social activity frequency from 2008 to 2012, an episodic memory indicator could not be used to predict change. **Table 18** presents detailed results from this model. **Figure 2** graphically displays a template of the final model.

4.6 Latent Growth Curve Modeling – Working Memory and Activity Frequency

In the first analysis, a single indicator linear growth model was specified, using the intercept latent factor (I) and three observed indicators for working memory: serial 7s totals at 2008, 2010, and 2012. Indicator loadings were fixed to one for all time points. The model fit the data poorly (χ^2 = 104.38; *df* = 4; *p* < .001; RMSEA 90% C.I. = .07-.10; CFI = .98; AIC = 34,557.57). All observed indicator residual variances indicated that there were additional significant amounts of variance to be explained. **Table 19** presents detailed findings for this first analysis.

In the second analysis, a linear slope latent factor was added to the model to improve model fit. Slope factor loadings were linear (i.e., 0, 2, 4). The intercept and the slope were not permitted to covary, given that it would become a Heywood case when covariation was permitted. The model with both the intercept and slope latent factors had slightly better fit (χ^2 = 12.34, *df* = 2, *p* < .001; RMSEA 90% C.I. = .02-.06; CFI = 1.00; AIC = 34,469.53). Both the

mean for the intercept and slope were significant, indicating that they were non-zero. There was significant individual variation around the intercept, but not the slope, indicating that not all respondents' had a similar initial serial 7s score, yet they had a similar growth rate. All observed indicator residual variances indicated that there were additional significant amounts of variance to be explained. The model accounted for a fair portion of variance in the observed variables, per R^2 (66-67% variance explained). **Table 20** presents detailed findings for this second analysis.

In the third analysis, CA08 and SA08 were entered as two separate latent factors. Both the intercept and slope factors were regressed onto these activities' latent factors. The model fit slightly worsened (χ^2 = 557.11, df = 41, p < .001; RMSEA 90% C.I. = .06-.07; CFI = .88). The means for the activity latent factors could not be estimated. Latent factors, CA08 and SA08, were correlated (standardized estimate = .77, p < .001). The intercepts for the intercept and the slope were statistically significant, indicating that they were not approaching zero. Variances around the activity latent factors were statistically significant. Residual variances for the observed indicators and intercept contained additional significant amounts of variance to be explained. The residual variance for slope was not statistically significant, indicating that no further variance could be explained. The model accounted for a fair portion of variance in the observed variables and intercept, per R^2 (13-67% variance explained). The model did not adequately account for variance in slope per R^2 (10%). The frequencies of cognitive activities and social activities at 2008 significantly predicted the intercept of serial 7s. Greater frequency in cognitive activity engagement was associated with higher serial 7s performance and greater frequency in social activity engagement was associated with lower serial 7s performance. Neither activity frequency significantly predicted the slope. **Table 21** presents detailed findings for this third analysis.

In the fourth analysis, a time invariant covariate, years of school, was added to improve model fit. This covariate was specified to predict the intercept of serial 7s performance. The addition of the covariate substantially worsened model fit, χ^2 = 2095.35, *df* = 51, $p < .001$; RMSEA 90% C.I. = .11, CFI = .62). The intercepts for the intercept and the slope were statistically significant, indicating that they were non-zero. Variances for CA08 and SA08 were statistically significant. Unstandardized residual variances for the intercept and observed indicators were statistically significant; the unstandardized residual variance for the slope was not statistically significant, indicating that no further variance could be explained in the slope latent factor. Per R^2, the model accounted for a fair portion of variance in the observed indicators and intercept, but not in slope (7% of variance explained). Number of school years significantly predicted the intercept. CA08 and SA08 no longer significantly predicted the intercept. Neither activity frequency significantly predicted the slope of serial 7s performance. **Table 22** presents detailed findings for this fourth analysis. **Figure 1** graphically displays a template of the final model.

4.7 Latent Growth Curve Modeling – Semantic Memory and Activity Frequency

In the first analysis, a single indicator linear growth model was specified, using the intercept latent factor (I) and an indicator of semantic memory (naming the "Vice President" item) at each time point. Due to HRS methodology, there were large sample size differences across time points for respondents who completed this naming item. Relative to years 2010 and 2012, year 2008 had the lowest item completion (*n* = 2,787). Thus, if respondents did not complete the item in 2008, they were excluded from the proposed models. Almost all of the respondents who completed the item in 2008, completed the item in 2010 and 2012, with four missing data points in 2010 and one missing data point in 2012. Indicator loadings were fixed to

one for all time points. The model fit the data poorly (χ^2 = 606.22; *df* = 2; *p* = .00; RMSEA 90% C.I. = .31-.35; CFI = .72). The mean of the intercept was fixed and could not be freely estimated; yet the variance of the intercept was statistically significant (unstandardized estimate = .82, *p* < .001).

When a linear slope latent factor was added, the model was misidentified (observed indicators in 2008 and 2012 had negative residual variances). Further complex models were not computed, given that the model had intrinsic difficulties.

4.8 Post Hoc Latent Growth Curve Models

To determine the effect of years of education on episodic and working memory over time, three post hoc LGC models were specified, with years of education predicting the intercept and slope for each cognitive domain. An LGC model was not specified for semantic memory, given that such a model could not be specified. The purpose of the models was to assess if years of education accounted for initial level and change in cognitive functioning, without the inclusion of activity engagement.

4.8.1 LGC for immediate recall. Years of education were specified to predict the intercept and slope factors for observed immediate recall indicators from 2008, 2010, and 2012. The intercept and slope factors were not permitted to covary. The model fit was good (χ^2 = 25.32, *df* = 3, *p* < .001; RMSEA 90% C.I. = .03-.06; CFI = 99), with the exception of the chi-square. Years of education significantly predicted the intercept (unstandardized estimate = .16, SE = .01, *p* < .001), but not the slope (unstandardized estimate = -.003, SE = .002, *p* = .25).

4.8.2 LGC for delayed recall. The same model was specified for delayed recall. The model fit was good, with the exception of the chi-square (χ^2 = 8.99, *df* = 3, *p* = .03; RMSEA 90% C.I. = .01-.04; CFI = 1.00). Years of education significantly predicted the intercept

(unstandardized estimate = .17, SE = .01, $p < .001$), but not the slope (unstandardized estimate = .00, SE = .003, $p = .86$).

4.8.3 LGC for working memory. The same model was specified for working memory. The model fit was good, with the exception of the chi-square ($\chi^2 = 11.72$, $df = 3$, $p = .01$; RMSEA 90% C.I. = .01-.05; CFI = 1.00). Years of education significantly predicted the intercept (unstandardized estimate = .20, SE = .01, $p < .001$), but not the slope (unstandardized estimate = .003, SE = .002, $p = .22$).

4.9 Latent Growth Curve Modeling – Health and Activity Frequency

An intercept-only model was specified, using the intercept latent factor and three indicators for physical health (summed health indicators at each time point). Indicator loadings were fixed to one for all time points. The model fit the data poorly, $\chi^2 = 327.02$; $df = 4$; $p < .001$; RMSEA 90% C.I. = .15-.18; CFI = .96; AIC = 22,955.77. The mean and variance for the intercept was statistically significant, indicating that they were not approaching zero. All observed indicator residual variances were statistically significant, indicating that there were additional significant amounts of variance to be explained. Per R^2, the model accounted for a good portion of variance in the observed indicators (80-91% of variance explained). **Table 23** presents detailed findings for this analysis.

In the second model, a linear slope latent factor was added to improve model fit. The intercept and slope were permitted to covary. Slope factor loadings were linear (i.e., 0, 2, 4). The model fit the data modestly, ($\chi^2 = 21.93$; $df = 1$; $p < .001$; RMSEA 90% C.I. = .05-.11; CFI = .99; AIC = 22,611.68). Both the means and variances for the intercept and slope were significant, indicating that they were non-zero and there was individual variation around the mean and growth rate for health conditions. All observed indicator residual variances were

statistically significant, indicating that there were additional significant amounts of variance to be explained. Per R^2, the model accounted for a good portion of variance in the observed indicators (86-95% of variance explained). The intercept was negatively associated with the slope (unstandardized estimate = -.04, SE = .01, $p < .001$). **Table 24** presents detailed findings for this analysis.

In the third model, CA08 and SA08 were entered as two separate latent factors. Both the intercept and slope factors were regressed onto these activities' latent factors. The model fit the data well ($\chi^2 = 261.61$; $df = 40$; $p < .001$; RMSEA 90% C.I. = .04-.05; CFI = .96). The means for the activity latent factors could not be estimated. Variances around the activity latent factors were statistically significant. The intercepts of the intercept and slope were significant, indicating that they were non-zero. Residual variances for the observed indicators, intercept, and slope contained additional significant amounts of variance to be explained. Per R^2, the model accounted for a good portion of variance in the observed indicators (86-96% of variance explained), yet a smaller portion of variance in the intercept and slope (1-3% of variance explained). CA08 and SA08 positively covaried (unstandardized estimate = .21, SE = .02, $p < .001$). CA08 significantly and positively predicted the intercept of summed health conditions, such that lower cognitive activity engagement predicted greater sum of health conditions at baseline. SA08 did not significantly predict the intercept. Neither activity frequency significantly predicted the slope. **Table 25** presents detailed findings for this analysis.

In the fourth model, a time invariant covariate, years of school, was added to improve model fit. Years of school were specified to predict the intercept and slope of the summed health indicators. The addition of the covariate substantially worsened model fit, $\chi^2 = 1790.66$, $df = 49$, $p < .001$; RMSEA 90% C.I. = .10 - .11, CFI = .70). The intercepts for the intercept and slope

were statistically significant, indicating that they were non-zero. Variances for CA08 and SA08 were statistically significant. Residual variances for the intercept, slope, and observed indicators were statistically significant, suggesting additional variance to be explained. Per R^2, the model accounted for a good portion of variance in the observed indicators (86-95% of variance explained), yet a smaller portion of variance in the intercept and slope (1-3% of variance explained). Number of school years significantly predicted the intercept of summed health conditions but not the slope of summed health conditions. CA08 and SA08 no longer significantly predicted the intercept. Neither activity frequency significantly predicted the slope of summed health conditions. **Table 26** presents detailed findings for this analysis. **Figure 1** graphically displays a template of the final model.

4.10 Autoregressive Models for Cross-Lagged Panels

4.10.1 Health over time. An autoregressive model was specified for summed health conditions over time. The model fit the data poorly, $\chi^2 = 142.22$, $df = 1$, $p < .001$; RMSEA 90% C.I. = .18-.23; CFI = .99; AIC = 12,149.11. Summed health conditions in 2008 significantly predicted summed health conditions in 2010, which significantly predicted its counterpart in 2012. The intercepts for summed health conditions in 2010 and 2012 were significant, indicating that they were non-zero. In addition, their residual variances were statistically significant, indicating that there was additional variance to be explained. Per R^2, the model accounted for a good portion of variance in the observed indicators (73-76% of variance explained). **Table 27** presents detailed findings for this analysis.

4.10.2 Immediate recall over time. A second autoregressive model was specified for immediate recall over time. The model fit the data poorly, $\chi^2 = 523.33$, $df = 1$, $p < .001$; RMSEA 90% C.I. = .37-.42; CFI = .71; AIC = 24,385.21. However, immediate recall in 2008

significantly predicted performance in 2010, and immediate recall in 2010 significantly predicted performance in 2012. The intercepts and residual variances for immediate recall in 2010 and 2012 were statistically significant, indicating that the means were non-zero and that there was significant portion of variance left to be explained. Per R^2, the model accounted for a small portion of variance in the observed indicators (17-18% of variance explained). **Table 28** presents detailed findings for this analysis.

4.10.3 Delayed recall over time. A third autoregressive model was specified for delayed recall over time. The model fit the data poorly, $\chi^2 = 564.03$, $df = 1$, $p < .001$; RMSEA 90% C.I. = .38-.44; CFI = .76; AIC = 26,416.92. However, delayed recall in 2008 significantly predicted performance in 2010, and delayed recall in 2010 significantly predicted performance in 2012. The intercepts and residual variances for delayed recall in 2010 and 2012 were statistically significant, indicating that the means were non-zero and that there was a significant portion of variance to be explained. Per R^2, the model accounted for a small portion of variance in the observed indicators (23% of variance explained). **Table 29** presents detailed findings for this analysis.

4.10.4 Serial 7s over time. A fourth autoregressive model was specified for serial 7s performance over time. The model fit the data poorly, $\chi^2 = 578.34$, $df = 1$, $p < .001$; RMSEA 90% C.I. = .38-.44; CFI = .87; AIC = 22,195.32. However, serial 7s performance in 2008 significantly predicted performance in 2010, and serial 7s performance in 2010 significantly predicted performance in 2012. The intercepts and residual variances for serial 7s performance in 2010 and 2012 were statistically significant, indicating that the means were non-zero and that there was significant portion of variance left to be explained. Per R^2, the model accounted for a

fair portion of variance in the observed indicators (23% of variance explained). **Table 30** presents detailed findings for this analysis.

4.10.5 Naming over time. A fifth autoregressive model was specified for naming performance over time. The model fit the data poorly, $\chi^2 = 46.04$, $df = 1$, $p < .001$; RMSEA 90% C.I. = .10-.16; CFI = .97. Naming in 2008 significantly predicted performance in 2010, which significantly predicted performance in 2012. The unstandardized thresholds for naming in 2010 and 2012 were statistically significant, indicating that they were non-zero. Per R^2, the model accounted for a fair portion of variance in the observed indicators (18-55% of variance explained). **Table 31** presents detailed findings for this analysis.

4.11 Cross Lagged Panel Modeling – Health and Immediate Recall

Autoregressive and cross-lagged effects were specified, and the indicators were allowed to covary at each time point. The model fit the data poorly, $\chi^2 = 661.41$, $df = 4$, $p < .001$; RMSEA 90% C.I. = .21-.23; CFI = .94; AIC = 59,335.70. All means (summed health conditions and delayed recall in 2008) and intercepts (summed health conditions and delayed recall in 2010 and 2012, respectively) were statistically significant, indicating that they were not approaching zero. Variances for summed health conditions and delayed recall in 2008 were statistically significant. Residual variances for observed indicators in 2010 and 2012 were also statistically significant, indicating that there was additional variance to be explained. Per R^2, the model accounted for a small portion of variance in the observed immediate recall indicators (17-18% of variance explained), and a good portion of variance in the observed summed health indicators (73-76% of variance explained). Immediate recall in 2008 significantly covaried with summed health conditions in 2008 (cov = -.13, SE = .03, p = .000). Immediate recall and summed health conditions did not significantly covary at 2010 or 2012. All autoregressive effects were found to

be statistically significant. In terms of cross-lagged effects, immediate recall in 2008 significantly predicted summed health conditions in 2010, which significantly predicted immediate recall in 2012. **Table 32** presents detailed findings for this analysis.

Covariates were added to improve model fit. First, years of school were added as a time invariant covariate, predicting observed summed health conditions and immediate recall indicators, at each time point. The model continued to fit the data poorly, $\chi^2 = 574.99$, $df = 4$, $p < .001$; RMSEA 90% C.I. = .19-.22; CFI = .95; AIC = 58,717.00. All observed indicator intercepts were statistically significant, indicating that they were non-zero. In addition, all residual variances for observed indicators were significant, indicating extra variance to be explained. Per R^2, the model accounted for a small portion of variance in the observed immediate recall indicators (11-21% of variance explained), and a poor to good portion of variance in the observed summed health indicators (2-76% of variance explained). Immediate recall in 2008 significantly covaried with summed health conditions in 2008 (*cov* = -.07, SE = .03, $p = .01$). Immediate recall and summed health conditions did not significantly covary at 2010 or 2012. All autoregressive coefficients were statistically significant. Number of school years significantly predicted immediate recall at each time point and summed health conditions in 2008 and 2010. Immediate recall in 2008 significantly predicted summed medical conditions in 2010. No other cross-lagged effects were statistically significant. **Table 33** presents detailed findings for this analysis.

The addition of age as a covariate improved model fit slightly, yet the overall fit remained poor, $\chi^2 = 518.07$, $df = 16$, $p < .001$; RMSEA 90% C.I. = .09-.10; CFI = .96; AIC = 58,136.50. All observed indicator intercepts were statistically significant, indicating that they were non-zero. Residual variances were also all significant, indicating extra variance to be

explained. Per R^2, the model accounted for a small portion of variance in the observed immediate recall indicators (16-26% of variance explained), and a poor to good portion of variance in the observed summed health indicators (2-76% of variance explained). Immediate recall in 2008 significantly covaried with summed health conditions in 2008 (*cov* = -.06, SE = .03, p = .02). Immediate recall and summed health conditions did not significantly covary at 2010 or 2012. All autoregressive coefficients were statistically significant. Age at 2008 significantly predicted immediate recall in 2008, age at 2010 significantly predicted immediate recall in 2010, and age at 2012 significantly predicted immediate recall performance in 2012. Age did not significantly predict summed health conditions at any time point. Number of school years significantly predicted immediate recall at each time point and summed health conditions in 2008 and 2010. No cross-lagged effects were significant. **Table 34** presents detailed findings for this analysis. **Figure 3** graphically displays a template of the final model.

4.12 Cross Lagged Panel Modeling – Health and Delayed Recall

Autoregressive and cross-lagged effects were specified, and the indicators were allowed to covary at each time point. The model fit the data poorly, χ^2 = 697.72, *df* = 4, p < .001; RMSEA 90% C.I. = .21-.24; CFI = .94; AIC = 62,602.12. All autoregressive effects were found to be statistically significant. Delayed recall in 2008 significantly covaried with summed medical conditions in 2008 (*cov* = -.15, SE = .04, p < .001); delayed recall and summed health conditions did not significantly covary at 2010 or 2012. All means (summed medical conditions and delayed recall in 2008) and intercepts (summed medical conditions and delayed recall in 2010 and 2012, respectively) were statistically significant, indicating that they were not approaching zero. Variances for summed medical conditions and delayed recall in 2008 were statistically significant. Residual variances for the indicators in 2010 and 2012 were statistically significant,

indicating that there was additional variance to be explained. Per R^2, the model accounted for a small portion of variance in the observed delayed recall indicators (24% of variance explained), and a good portion of variance in the observed summed health indicators (73-76% of variance explained). In terms of cross-lagged effects, delayed recall in 2008 significantly predicted summed medical conditions in 2010, which significantly predicted delayed recall in 2012. **Table 35** presents detailed findings for this analysis.

Covariates were added to improve model fit. First, years of school were added as a covariate, predicting summed medical conditions and delayed recall indicators, at each time point. The model continued to fit the data poorly, $\chi^2 = 639.83$, $df = 4$, $p < .001$; RMSEA 90% C.I. = .20-.23; CFI = .95; AIC = 62,114.21. All intercepts for observed indicators were statistically significant, indicating that they were non-zero. Similarly, residual variances for all observed indicators were significant, indicating extra variance to be explained. Per R^2, the model accounted for a small portion of variance in the observed delayed recall indicators (8-26% of variance explained), and a good portion of variance in the observed summed health indicators (2-76% of variance explained). Delayed recall in 2008 significantly covaried with summed medical conditions in 2008 (cov = -.08, SE = .03, $p < .01$); delayed recall and summed health conditions did not significantly covary at 2010 or 2012. All autoregressive coefficients were statistically significant. Number of school years significantly predicted delayed recall across the three time points, and summed medical conditions in 2008 and 2010. Delayed recall in 2008 significantly predicted summed medical conditions in 2010, and summed medical conditions in 2010 significantly predicted delayed recall in 2012. No other cross-lagged effects were statistically significant. **Table 36** presents detailed findings for this analysis.

The addition of age as a covariate improved model fit slightly, yet the overall fit remained poor, χ^2 = 598.69, *df* = 16, $p < .001$; RMSEA 90% C.I. = .10-.11; CFI = .95; AIC = 61,521.11. All observed indicator intercepts were statistically significant, indicating that they were non-zero. Residual variances were also all significant, indicating extra variance to be explained. Per R^2, the model accounted for a good portion of variance in the observed delayed recall indicators (14-30% of variance explained), and a poor to good portion of variance in the observed summed health indicators (2-76% of variance explained). Delayed recall in 2008 significantly covaried with summed medical conditions in 2008 (*cov* = -.04, SE = .02, $p = .03$); delayed recall and summed health conditions did not significantly covary at 2010 or 2012. All autoregressive coefficients were statistically significant. Age at 2008 significantly predicted delayed recall in 2008, age at 2010 significantly predicted delayed recall in 2010, and age at 2012 significantly predicted delayed recall in 2012. Age did not significantly predict summed medical conditions at any time point. Number of school years significantly predicted delayed recall at each time point and summed health conditions in 2008 and 2010. Delayed recall in 2008 predicted summed medical conditions in 2010, which predicted delayed recall in 2012. No other cross-lagged effects were statistically significant. **Table 37** presents detailed findings for this analysis. **Figure 3** graphically displays a template of the final model.

4.13 Cross Lagged Panel Modeling – Health and Working Memory

Autoregressive and cross-lagged effects were specified for summed health conditions and serial 7s indicators over time. The two constructs were allowed to covary at each time point. The model fit the data poorly, χ^2 = 719.57, *df* = 4, $p < .001$; RMSEA 90% C.I. = .22-.24; CFI = .95; AIC = 57,719.99. All means (i.e., summed medical conditions and serial 7s performance in 2008) and intercepts (i.e., summed medical conditions and serial 7s performance in 2010 and

2012) were statistically significant, indicating that they were not approaching zero. Variances for summed medical conditions and serial 7s performance in 2008 were statistically significant. Residual variances for the indicators in 2010 and 2012 were statistically significant, indicating that there was additional variance to be explained. Per R^2, the model accounted for an adequate portion of variance in the observed serial 7 (43-44% of variance explained) and summed health indicators (73-76% of variance explained). Serial 7s performance in 2008 significantly covaried with summed medical conditions in 2008 (*cov* = -.17, SE = .03, $p < .001$), and serial 7s performance in 2010 significantly covaried with summed medical conditions in 2010 (*cov* = -.03, SE = .01, $p = .02$). Serial 7s and summed health conditions did not significantly covary in 2012. All autoregressive effects were found to be statistically significant. Summed health conditions significantly covaried with serial 7s performance only in 2008 (*cov* = -.17, SE = .03, $p < .001$) and 2010 (*cov* = -.03, SE = .01, $p = .02$). Summed health conditions in 2008 significantly predicted serial 7s performance in 2010, and serial 7s performance in 2008 significantly predicted summed medical conditions in 2010. Summed health conditions in 2010 further predicted serial 7s performance in 2012. **Table 38** presents detailed findings for this analysis.

To improve model fit, covariates were added. First, years of school were added, predicting summed medical conditions and serial 7s indicators, at each time point. The model continued to fit the data poorly, $\chi^2 = 666.15$, $df = 4$, $p < .001$; RMSEA 90% C.I. = .21-.24; CFI = .95; AIC = 57,008.36. With the exception of the intercept for serial 7s performance in 2012, intercepts for all observed indicators were statistically significant, indicating that they were non-zero. Residual variances for all observed indicators were statistically significant, indicating that there was additional variance to be explained. Per R^2, the model accounted for an adequate portion of variance in the observed serial 7s (13-45% of variance explained) and summed health

(2-76% of variance explained) indicators. Serial 7s performance in 2008 significantly covaried with summed medical conditions in 2008 (*cov* = -.09, SE = .03, *p* = .001). Serial 7s and summed health conditions did not significantly covary in 2010 or in 2012. All autoregressive effects were found to be statistically significant. Number of school years significantly predicted serial 7s performance across the three time-points, and summed health conditions in 2008 and 2010. In terms of cross-lagged effects, summed health conditions in 2008 significantly predicted serial 7s performance in 2010, but no other cross-lagged effect was statistically significant. **Table 39** presents detailed findings for this analysis.

Next, age was added in addition to number of school years, as a time varying covariate. The model continued to fit the data poorly, χ^2 = 682.74, *df* = 16, *p* < .001; RMSEA 90% C.I. = .10-.12; CFI = .95; AIC = 57,005.08. All observed indicator intercepts were statistically significant, indicating that they were non-zero. Residual variances for all observed indicators were statistically significant, indicating that there was additional variance to be explained. Per R^2, the model accounted for an adequate portion of variance in the observed serial 7s indicators (13-45% of variance explained) and summed health indicators (2-76% of variance explained). Serial 7s performance in 2008 significantly covaried with summed health conditions in 2008 (*cov* = -.06, SE = .02, *p* = .001). Serial 7s and summed health conditions did not significantly covary in 2010 or in 2012. All autoregressive effects were found to be statistically significant. Age at 2010 significantly covaried with serial 7s performance in 2010; there were no other significant covariations between age and serial 7s performance or summed health conditions. Number of school years significantly predicted serial 7s performance across the three time points as well as summed health conditions in 2008 and 2010. Summed health conditions in 2008 significantly predicted serial 7s performance in 2010, but no other cross-lagged effect was statistically

significant. **Table 40** presents detailed findings for this analysis. **Figure 3** graphically displays a template of the final model.

4.14 Cross Lagged Panel Modeling – Health and Semantic Memory

The following models were specified in a sample of cases that did not have missing data on "Vice President" naming in 2008. Observed indicators for summed health conditions and naming were specified simultaneously over time. Autoregressive and cross-lagged effects were specified, and constructs were allowed to covary at each time point. Parameterization had to be specified to theta for the model to run. The model fit the data modestly, χ^2 = 93.99, *df* = 4, *p* = .00; RMSEA 90% C.I. = .08-.11; CFI = .97. All means (summed health conditions and naming performance in 2008) and intercepts (summed health conditions and naming performance in 2010 and 2012) were statistically significant, indicating that they were not approaching zero. Variances for summed health conditions and naming performance in 2008 were statistically significant. Residual variances for the summed health condition indicators in 2010 and 2012 were statistically significant, indicating that there was additional variance to be explained. Per R^2, the model accounted for an adequate portion of variance in the observed naming (18-56% of variance explained) and summed health indicators (75-79% of variance explained). Naming in 2008 significantly covaried with summed health conditions in 2008 (*cov* = -.03, SE = .01, *p* < .001). Naming and summed health conditions did not significantly covary in 2010 or in 2012. All autoregressive effects were found to be statistically significant. Summed health conditions in 2008 significantly predicted naming performance in 2010, and naming performance in 2008 significantly predicted summed health conditions in 2010. **Table 41** presents detailed findings for this analysis.

To improve model fit, covariates were added. First, years of school were added, predicting summed medical conditions and naming indicators, at each time point. The model continued to fit the data modestly, χ^2 = 62.90, *df* = 4, *p* = .00; RMSEA 90% C.I. = .06-.09; CFI = .98. All observed indicator intercepts were statistically significant, indicating that they were non-zero. Residual variances for all observed indicators were statistically significant, indicating that there was additional variance to be explained. Per R^2, the model accounted for a poor to good portion of variance in the observed naming (7-55% of variance explained) and summed health (1-79% of variance explained) indicators. Naming in 2008 significantly covaried with summed health conditions in 2008 (*cov* = -.02, SE = .01, *p* = .01). Naming and summed health conditions did not significantly covary in 2010 or in 2012. All autoregressive effects were found to be statistically significant. Number of school years significantly predicted naming performance across the three time-points, as well as summed medical conditions in 2008 and 2010. Summed medical conditions in 2008 significantly predicted naming performance in 2010, but no other cross-lagged effect was statistically significant. **Table 42** presents detailed findings for this analysis.

Next, age was added in addition to number of school years, as a time varying covariate. Model fit improved, χ^2 = 23.38, *df* = 16, *p* = .10; RMSEA 90% C.I. = .00-.02; CFI = 1.00. All autoregressive effects were found to be statistically significant. Intercepts (and thresholds) for naming and summed medical conditions across the three time points were statistically significant, indicating that they were non-zero. Residual variances for naming in 2008 and all summed health condition indicators were statistically significant, indicating that there was additional variance to be explained. Per R^2, the model accounted for a poor to good portion of variance in the observed naming (7-55% of variance explained) and summed health (1-79% of variance explained)

indicators. Naming in 2008 significantly covaried with summed health conditions in 2008 (*cov* = -.02, SE = .01, *p* = .01). Naming and summed health conditions did not significantly covary in 2010 or in 2012. Age in 2010 significantly predicted naming performance in 2010; however, there were no further significant associations between age and naming performance at other time points. Number of school years significantly predicted naming performance across the three time-points and summed medical conditions in 2008 and 2010. Summed medical conditions in 2008 significantly predicted naming performance in 2010, but no other cross-lagged effect was statistically significant. **Table 43** presents detailed findings for this analysis. **Figure 3** graphically displays a template of the final model.

CHAPTER 5: Discussion

The overarching goal of this dissertation is to examine the effect of cognitively stimulating and social activities on cognitive functioning and health in a national sample of older adults from the Health and Retirement Study (HRS). The aims of this project were to identify and differentiate longitudinal relations between activity engagement frequency and cognitive domains (episodic memory, working memory, and semantic memory) as well as identify longitudinal relations between activity engagement frequency and overall health, for comparison. Relations between health and cognitive function were further assessed longitudinally. Results were expected to provide greater understanding of the directionality of such longitudinal relations.

5.1 Summary of Findings

It was first hypothesized that baseline activity frequency would predict level and rate of change in cognitive functioning over time, based on the "use it or lose it" model (e.g., Hultsch et al., 1999) and prior findings reporting the positive effects of cognitive and social activity on cognitive functioning over time (Ghisletta et al., 2006; James et al., 2011; Small et al., 2012; Wilson et al., 2010). Thus, it was also hypothesized that there would be less statistical support for the opposing (or reverse) model, in which baseline cognitive performance would predict level and rate of change in activity engagement over time. Findings precluded a comparison between the two contrasting models. Activity frequency (cognitive and social) did not significantly predict rate of cognitive change, and activity frequency did not significantly change over time, rendering a comparison between models as unnecessary. However, frequency of baseline cognitive activity engagement was consistently associated with initial level, or intercept, of episodic memory (immediate and delayed recall). Findings are in line with the "use it or lose it"

hypothesis, as higher engagement in baseline cognitive activities predicted better episodic memory performance at baseline. Interestingly, post hoc latent growth curve models indicated that when education was entered as a single predictor of initial level and rate of change in episodic memory, education significantly predicted the initial level of episodic memory, but not change. As such, it may be understood that level of education, alone, does not predict decline in cognitive skills in late life. The current results do not rule out a protective effect for activity engagement for at least episodic memory, beyond education. In addition, the significant association between initial level of episodic memory and activity engagement may also support the cognitive reserve hypothesis, such that individuals with higher activity engagement may have always had higher cognitive functioning.

Relative to working and semantic memory, episodic memory indicators were consistently associated with baseline activity frequency, in models with and without education as a covariate. It is possible that differences observed between episodic, working, and semantic memory were partly due to indicators' restrictions of range. Indicators of episodic memory had the greatest variability, followed by working memory, then semantic memory. Thus, it is unclear if the association between working memory and activity frequency was attenuated, with the addition of education as a covariate, due to restriction of range.

It was also hypothesized that frequency of cognitive activities would have a statistically stronger effect on the intercept and rate of change of cognitive functioning, relative to frequency of social activities, based on activity differences in cognitive demand (e.g., Park et al., 2014; Salthouse et al., 2002). Findings revealed that cognitive activities had a statistically stronger (and positive) effect on episodic memory intercepts, relative to social activities.

In addition, based on the nature of age-related trajectories for each cognitive domain, it was predicted that greater activity frequency would be associated with reduced changes in working and episodic memory performance over time. It was further predicted that activity frequency would not significantly affect semantic memory performance over time. Unexpectedly, there was no evidence to support these predictions, as activity engagement did not predict change in either working memory or episodic memory over time. Latent growth curve models could not be computed for semantic memory, most likely because of restricted range and limited variability in scores on the items comprising the semantic memory construct (naming items) at each time point and over time. There appeared to be a relatively prominent ceiling effect for these items.

Longitudinal associations between overall health and activity frequency were next examined. Similar to the previous models incorporating cognitive constructs and the "use it or lose it" model, it was hypothesized that activity engagement would predict initial level and rate of change in number of health conditions over time. Thus, it was also hypothesized that there would be less statistical support for the reverse model (i.e., number of health conditions predicting level and rate of change in activity engagement over time). However, the two contrasting models could not be wholly compared, due to a paucity of significant findings.

Results from the latent growth curve models indicated that cognitive activity frequency, but not social activity frequency, predicted the intercept for number of health conditions when no covariates were considered. When education was added as a covariate, neither cognitive nor social activity frequency predicted the intercept for number of health conditions (model fit also worsened with the addition of education). Although education and activity (cognitive and social) level tended to be significantly correlated, the correlations were minimal (*r*'s tending to range from .1 to .2). As such, shared variance between education and activity frequency most likely did

not account for the disappearance of the effect on the intercept, with the inclusion of a covariate. Further, neither cognitive nor social activitiy frequency predicted the slope for number of health conditions, in models with or without education as a covariate. At the very best, it can be concluded that a lower frequency of cognitive activities was associated with a higher number of health conditions at baseline, when education was not accounted for in the model. Results from models focusing on activity frequency and cognitive functioning were similar to results focusing on activity frequency and overall health, such that lower cognitive activity frequency was associated with poorer outcomes.

Longitudinal, causal, associations between physical health and cognitive functioning were assessed over time (similar to Small et al., 2011). It was hypothesized that declines in health would predict subsequent cognitive decline, within the cross-lagged panel analyses. Model fit was poor for cross-lagged analyses for episodic and working memory. However, model fit was adequate for cross-lagged analyses involving health and semantic memory (naming). Number of health conditions in 2008 significantly predicted naming performance in 2010, and naming performance in 2008 significantly predicted number of health conditions in 2010. However, when education and age were added as covariates, only number of health conditions in 2008 significantly predicted naming in 2010, such that a greater number of health conditions at baseline predicted poorer naming performance at the second wave. These results support the hypothesized trend, yet this trend was not indicated over time.

Lastly, in the absence of distinct test statistics to compare non-nested complex models with different estimators (maximum likelihood vs. weighted least squares with mean and variance adjustment), with and without covariates, model comparison was difficult to conduct. However, it must be reiterated that the addition of education as a covariate consistently worsened

model fit, in terms of increasing the chi-square test of model fit, CFI, and RMSEA, for latent growth curve models. Thus, it could be argued that latent growth curve models with a covariate should be given less credence than models without a covariate. The aforementioned latent growth curve findings should be interpreted with this caveat in mind.

5.2 Relation of Results to the Literature

The current findings do not provide clear evidence for the "use it or lose it" hypothesis, as engagement in baseline activities did not have an effect on change in cognitive functioning over time (e.g., lower frequency of activity engagement predicting cognitive decline). Contrary to the current findings, previous studies have found positive associations between activity engagement and cognitive change. For example, results from the Synapse Intervention Trial (Park et al., 2014) revealed that older adults experienced an improvement in episodic memory following completion of learning a novel, cognitively demanding task (i.e., learning digital photography) over time, relative to older adults participating in less cognitively demanding tasks over time (e.g., social activities). It should be noted that the Synapse Intervention Trial was conducted over a 14-week period, involving only two time points (pre- and post-intervention), without follow-up data collection. Similar to findings by Park et al. (2014), Wilson et al. (2002) found that greater baseline frequency of cognitive/leisure activity engagement was associated with lower rates of decline in working memory, yet not in episodic memory, within a longitudinal study (average 4.5 year follow-up) of older Catholic nuns, priests, and brothers. The current analysis assessed cognitive change over five years, at three time points across two-year intervals, without controlled assignment to activity type or activity frequency level. It is possible that the positive findings revealed by Park et al. are partly contributed to the use of only two time points, over a short duration of time. Further, it is possible that Wilson et al.'s (2002) sample of

older adults differ from the national sample used in the current analysis, in terms of education, lifestyle, age, inclusion of participants with dementia, and potentially other factors.

Importantly, the current finding that baseline activity frequency did not predict change in cognitive outcomes is consistent with other published longitudinal findings. For example, Mitchell et al. (2012) reported that changes in cognitive activities from baseline were associated with within-person variability in working memory, yet baseline cognitive activity did not predict change in working memory or semantic knowledge over time. In addition, Vaughan et al. (2014; Women's Health Initiative Memory Study) similarly found that cognitive activity was significantly associated with baseline cognitive performance, but not change in cognitive performance, over three time points in a two-to-three year time period. Consistent with the current methods, Vaughan et al. used structural equation modeling. However, Vaughan et al. created a general cognitive functioning latent factor, rather than examining specific cognitive domains (e.g., episodic memory).

The positive association between baseline episodic memory performance and cognitive activity frequency is largely consistent with prior research examining episodic memory specifically (e.g., Lachman et al., 2010) or global cognitive functioning (e.g., Wilson et al., 2010). Further, the current finding that baseline cognitive activity frequency had a positive impact on initial level of episodic memory indicators, relative to baseline social activity frequency is consistent with research by Park et al. (2014). In their study, Park et al. reported that older adults did not experience cognitive benefit from social engagement relative to more cognitively demanding tasks (undergoing training for quilt making and/or photography), as hypothesized. Results suggest that cognitive activities require sustained or greater activation of working memory, long-term memory, and other processes reflective of executive functioning (Park et al.,

2014). Social activity engagement may require less cognitive activation, relying on passive observation and use of existing knowledge (Park et al., 2014).

The absence of substantial change or variation in activity frequency over time was unexpected. However, this finding may be explained by dispositional traits. Need for cognition (tendency to engage in and prefer challenging cognitive activities [Cacioppo, Petty, Feinstein, & Jarvis, 1999; Salthouse et al., 2002]) and extraversion have been found to explain participation in activities. As such, stability in traits may predict stability in behavior or activity engagement over time.

Longitudinal associations between health conditions and activity frequency were also examined. Unlike prior analyses involving cognitive domains, baseline activity frequency was no longer associated with initial level of health conditions when education was included as a covariate. This finding was unexpected, as there is at least strong support for the effect of social engagement (or contacts) on mortality (e.g., Blazer, 1982; House et al., 1982; Shoenbach et al., 1986).

Furthermore, causal relations between health and cognitive functioning were assessed over three points of time. A number of medical conditions have been associated with cognitive performance, including hypertension (Brady, Spiro, & Gaziano, 2005) and diabetes (Verdelho et al. (2010). Current results did not provide evidence for strong cognition-health trends over time. Unexpectedly, the strongest statistical support and trend was observed for semantic memory, with health conditions in 2008 predicting naming performance in 2010, over and above the effect of age and education. Small et al. (2011) examined the concurrent longitudinal associations between self-reported health and episodic memory, semantic memory, and processing speed. In their analysis, changes in self-reported health were not associated with changes for either

semantic or episodic memory. Thus, it is unclear why semantic memory was strongly associated with health in the current findings, relative to episodic and working memory. The restrictions of range and non-normal distributions for many of these outcome variables may have attenuated their effects in the models tested.

5.3 Significance and Implications

The significant association between baseline activity engagement and initial level of episodic memory does not allow one to rule out the protective effect of engagement in stimulating activities on cognition. This association may also provide support for the cognitive reserve hypothesis, such that respondents who engaged in cognitive activities more frequently may have always had greater cognitive functioning over time. In addition, the current findings reveal that changes in activity engagement were not found over time in a large sample of older adults. Such a finding may indicate that older adults' lifestyles do not significantly change over a five-year period, at least with respect to the indicators used in the HRS. Lastly, health-cognition changes were not consistently indicated over time, suggesting that health conditions, in a broad sense, may not have strong causal effects on age-related changes in cognitive functioning as measured by the indicators used in the HRS.

5.4 Limitations

Methodologically, the HRS is based on observational data, and thus, engagement in mental and social activities was not manipulated. Data regarding health and social engagement were based on self-report, which could be confounded by memory biases and social desirability effects. The number of time points that could be used for this particular analysis was limited to three, as the measures administered across waves were not identical over time, and identical measures were not consistently administered at each time point (based on the study's

questionnaire skip logic). Statistically, there was limited variability within the cognitive items, particularly those reflecting working memory and semantic memory. Variability could not be enhanced through the creation of cognitive latent factors with the limited number of indicators available. Furthermore, second- and third-order factors could not be identified, due to the low number of indicators (three or more indicators necessary per factor). As such, the current analyses were limited to observed indicators, with the exception of latent factors for cognitive and social activities (and the intercept and slope latent factors). A limited number of covariates could be added to the model, as the introduction of further time invariant or time varying covariates led to issues associated with multicollinearity. Analytically, it is possible that the null findings regarding activity frequency predicting cognitive change over time is partly a function of the sample used. The current sample was limited to older adults who denied memory loss or diagnosed disease. As such, the analyses were conducted on cognitively resilient individuals.

Strengths of the current analysis include a large, national sample. The HRS project is ongoing, and as such, there is promise that future waves may have more cognitive items consistently administered over time, which would permit greater variability in responses. The current analysis tapped the strengths of the HRS by implementing repeated measures and latent factors to reduce measurement error.

5.5 Future Directions

The current analyses did not investigate differences among subsamples. It is recommended that future HRS projects conduct mixture analyses examining respondents with low activity frequency relative to respondents with high activity frequency, as well as respondents with memory complaints (or disorders) relative to respondents reporting to be cognitively healthy. In addition, when cognitive functioning and activity engagement data from

future waves are made available (i.e., 2014 and 2016), analyses should be replicated, as change across time is best evaluated with four or more waves. Future HRS analyses, or analyses from other large data sets, are recommended to repeat the current analyses with viable latent factors, where possible.

Table 1

*Final Sample Characteristics (*n = *3,397)*

	Mean (or %)	*SD*
Age		
2008	71.14	6.89
2010	73.50	6.99
2012	75.23	6.90
Gender		
Male	38.56% (*n* = 1,310)	
Female	61.44% (*n* = 2,087)	
Race/Ethnicity		
White/Caucasian	87.25% (*n* = 2,964)	
Black or African American	10.86% (*n* = 369)	
Other	1.88% (*n* = 64)	
Number of Years in School	12.79	2.87

Table 2

Selected Cognitively Stimulating Activities and Associated Cognitive Demands

	Selected items from HRS Psychosocial and Lifestyle Questionnaire	Mean cognitive demand*
1.	Attend an educational or training course	4.0
2.	Do word games such as crossword puzzles or Scrabble	3.9 (for "crossword puzzles")
3.	Play cards or games such as chess	3.7 (for "chess/strategy games")
4.	Use a computer for e-mail, Internet or other tasks	3.5
5.	Writing (such as letters, stories, or journal entries)	3.5
6.	Read books, magazines, or newspapers	2.9 (for "newspapers, magazines") 3.0 (for "novels") 3.6 (for "nonfiction")
7.	Work on a hobby or project	2.8 (for "participating in hobbies and crafts")

*Note: Mean cognitive demands are reported in an independent study (Salthouse et al., 2002). These ratings were from a sample of approximately 1,200 adults. Where there may be slight differences in the content of the item between studies, the content of the Salthouse et al.'s item is specified in parentheses. Ratings of cognitive demands ranged from 1 (low) to 5 (high).

Table 3

Selected Social Engagement Activities and Associated Cognitive Demands

	Selected items from HRS Psychosocial and Lifestyle Questionnaire	Mean cognitive demand*
1.	Care for a sick or disabled adult	3.4 (for "supervising other people")
2.	Do volunteer work with children or young people	3.0 (for "volunteering")
3.	Do any other volunteer or charity work	3.0 (for "volunteering")
4.	Go to a sport, social, or other club	n/a
5.	Attend meetings of non-religious organizations, such as political, community, or other interest groups?	3.3 (for "attending meetings")

*Note: Mean cognitive demands are reported in an independent study (Salthouse et al., 2002). These ratings were from a sample of approximately 1,200 adults. Where there may be slight differences in the content of the item between studies, the content of the Salthouse et al.'s item is specified in parentheses. Ratings of cognitive demands ranged from 1 (low) to 5 (high).

Table 4

*Descriptive Statistics (*n = *3,397; some variables missing values)*

	***M* (or %)**	***SD***	**Range**
Vision rating (recoded)			
2008	2.72	.94	1-5
2010	2.78	.95	1-5
2012	2.82	.97	1-5
Hearing rating (recoded)			
2008	2.63	1.06	1-5
2010	2.69	1.05	1-5
2012	2.78	1.06	1-5
Immediate recall			
2008	5.57	1.46	0-10
2010	5.27	1.65	0-10
2012	5.14	1.61	0-10
Delayed recall			
2008	4.53	1.75	0-10
2010	4.34	2.00	0-10
2012	4.02	1.93	0-10
Serial 7s total			
2008	3.67	1.60	0-5
2010	3.60	1.60	0-5
2012	3.44	1.68	0-5
Vice President naming - restrict to only those with item in 2008 (*n* = 2,787)			
2008	.83	.38	0-1
2010	.58	.49	0-1
2012	.66	.47	0-1
Cognitive activity frequency: Education			
2008	4.73	.79	1-5
2012	4.82	.64	1-5
Cognitive activity frequency: Writing			
2008	3.96	1.45	1-5
2012	4.12	1.40	1-5
Cognitive activity frequency: Computer			
2008	3.00	1.93	1-5
2012	2.97	1.94	1-5
Cognitive activity frequency: Hobby			
2008	3.13	1.70	1-5
2012	3.73	1.60	1-5
Social activity frequency: Volunteer with youth			
2008	4.63	.98	1-5
2012	4.76	.79	1-5
Social activity frequency: Other volunteer/charity			
2008	4.22	1.28	1-5
2012	4.34	1.21	1-5
Social activity frequency: Clubs			
2008	4.08	1.35	1-5
2012	4.22	1.27	1-5

Social activity frequency: Non-religious organization			
2008	4.60	.87	1-5
2012	4.68	.80	1-5

Table 5

*Pairwise Correlations for Cognitive, Health, and Demographic Indicators**

	IR08	DR08	IR10	DR10	IR12	DR12	Serial08	Serial10	Serial12	Naming08	Naming10	Naming12
Age	-.26	-.27	-.29	-.32	-.33	-.34	-.04	-.07	-.07	-.001	-.11	-.12
2008	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p = .01$	$p < .001$	$p < .001$	$p = .96$	$p < .001$	$p < .001$
	n=3,380	n=3,380	n=3,381	n=3,381	n=3,377	n=3,377	n=3,397	n=3,397	n=3,397	n=2,787	n=2,783	n=2,786
Age	-,26	-.27	-.30	-.32	-.33	-.34	-.05	-.08	-.08	-.003	-.10	-.12
2010	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p = .01$	$p < .001$	$p < .001$	$p = .87$	$p < .001$	$p < .001$
	n=3,380	n=3,380	n=3,381	n=3,381	n=3,377	n=3,377	n=3,397	n=3,397	n=3,397	n=2,787	n=2,783	n=2,786
Age	-.26	-.26	-.30	-.32	-.33	-.34	-.04	-.07	-.07	.002	-.10	-.12
2012	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p = .01$	$p < .001$	$p < .001$	$p = .94$	$p < .001$	$p < .001$
	n=3,380	n=3,380	n=3,381	n=3,381	n=3,377	n=3,377	n=3,397	n=3,397	n=3,397	n=2,787	n=2,783	n=2,786
Vision	-.16	-.15	-.14	-.13	-.13	-.13	-.14	-.15	-.15	-.10	-.12	-.10
2008	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$
	n=3,379	n=3,379	n=3,380	n=3,380	n=3,376	n=3,376	n=3,396	n=3,396	n=3,396	n=2,786	n=2,782	n=2,785
Vision	-.16	-.14	-.13	-.11	-.11	-.12	-.16	-.18	-.18	-.12	-.12	-.13
2010	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$
	n=3,371	n=3,371	n=3,372	n=3,372	n=3,368	n=3,368	n=3,388	n=3,388	n=3,388	n=2,779	n=2,775	n=2,778
Vision	-.18	-.18	-.15	-.14	-.14	-.14	-.15	-.17	-.18	-.12	-.14	-.14
2012	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$
	n=3,375	n=3,375	n=3,376	n=3,376	n=3,373	n=3,373	n=3,392	n=3,392	n=3,392	n=2,782	n=2,778	n=2,781
Hearing	-.17	-.16	-.17	-,17	-.15	-.14	-.03	-.04	-.04	-.05	-.05	-.04
2008	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p = .10$	$p = .03$	$p = .02$	$p = .01$	$p = .02$	$p = .04$
	n=3,379	n=3,379	n=3,380	n=3,380	n=3,376	n=3,376	n=3,396	n=3,396	n=3,396	n=2,786	n=2,782	n=2,785
Hearing	-.16	-.14	-.17	-,16	-.15	-.13	-.02	-.03	-.04	-.02	-.02	-.03
2010	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p = .38$	$p = .07$	$p = .04$	$p = .38$	$p = .20$	$p = .09$
	n=3,375	n=3,375	n=3,376	n=3,376	n=3,372	n=3,372	n=3,392	n=3,392	n=3,392	n=2,783	n=2,779	n=2,782
Hearing	-.15	-.15	-.18	-.17	-.17	-.16	-.03	-.07	-.05	-.02	-.06	-.04
2012	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p = .07$	$p = .001$	$p = .004$	$p = .24$	$p = .003$	$p = .02$
	n=3,378	n=3,378	n=3,379	n=3,379	n=3,375	n=3,375	n=3,395	n=3,395	n=3,395	n=2,785	n=2,781	n=2,784
Years of	.33	.29	.25	.24	.28	.26	.36	.36	.36	.26	.28	.28
School	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$	$p < .001$
	n=3,379	n=3,379	n=3,380	n=3,380	n=3,376	n=3,376	n=3,396	n=3,396	n=3,396	n=2,787	n=2,783	n=2,786

*Note: For correlations involving naming in 2008, 2010, and 2012, sample was restricted to only those with item in 2008 (n = 2,787)

Table 6

Pairwise Correlations for Activity Frequency and Education

	Years of Education
Education	-.13 *
	n = 3,189
Writing	-.25*
	n = 3,254
Computer	-.41*
	n = 3,209
Hobby	-.19*
	n = 3,246
Volunteer with youth	-.001ns
	n = 3,219
Other volunteer/Charity	-.18*
	n = 3,229
Club (sports, social)	-.21*
	n = 3,213
Non-religious organization	-.11*
	n = 3,224

Note: ns = $p > .05$; * = $p < .001$.

Table 7

Latent Growth Curve: Intercept-only for Immediate Recall Over Time

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
I | IR08	1.00	.00	--	.69	.01	.000
I | IR10	1.00	.00	--	.62	.01	.000
I | IR12	1.00	.00	--	.65	.01	.000
Mean I	5.35	.02	.000	5.16	.10	.000
I variance	1.07	.04	.000	1.00	.00	--
IR08 Residual variance	1.16	.04	.000	.52	.01	.000
IR10 Residual variance	1.69	.05	.000	.61	.01	.000
IR12 Residual variance	1.48	.05	.000	.58	.01	.000
R^2 IR08				.48	.01	.000
R^2 IR10				.39	.01	.000
R^2 IR12				.42	.02	.000

Note: I = intercept; IR08 = immediate recall in 2008; IR10 = immediate recall in 2010; IR12 = immediate recall in 2012.

Table 8

Latent Growth Curve: Intercept and Slope Only for Immediate Recall Over Time

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
I | IR08	1.00	.00	--	.71	.01	.000
I | IR10	1.00	.00	--	.62	.01	.000
I | IR12	1.00	.00	--	.66	.01	.000
S | IR08	0.00	.00	--	.00	.00	--
S | IR10	2.00	.00	--	.09	.04	.02
S | IR12	4.00	.00	--	.19	.08	.02
Mean I	5.55	.02	.000	5.32	.10	.000
Mean S	-.11	.01	.000	-1.42	.62	.02
I variance	1.09	.04	.000	1.00	.00	--
S variance	.01	.01	.25	1.00	.00	--
IR08 Residual variance	1.05	.04	.000	.49	.01	.000
IR10 Residual variance	1.70	.05	.000	.61	.01	.000
IR12 Residual variance	1.34	.07	.000	.53	.03	.000
R^2 IR08				.51	.01	.000
R^2 IR10				.39	.01	.000
R^2 IR12				.47	.03	.000

Note: I = intercept; S = slope; IR08 = immediate recall in 2008; IR10 = immediate recall in 2010; IR12 = immediate recall in 2012.

Table 9

Latent Growth Curve: Activity Frequency Predicting Intercept and Slope for Immediate Recall Over Time

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
I | IR08	1.00	.00	--	.71	.01	.000
I | IR10	1.00	.00	--	.62	.01	.000
I | IR12	1.00	.00	--	.64	.01	.000
S | IR08	0.00	.00	--	.00	.00	--
S | IR10	2.00	.00	--	.06	.05	.22
S | IR12	4.00	.00	--	.13	.11	.22
I on CA08	-1.48	.19	.000	-.86	.09	.000
I on SA08	.99	.25	.000	.40	.10	.000
S on CA08	-.04	.04	.28	-.45	.68	.51
S on SA08	.05	.05	.33	.41	.63	.52
I of I	5.54	.02	.000	5.40	.11	.000
I of S	-.11	.01	.000	-2.07	1.70	.22
CA08 Variance	.35	.03	.000	1.00	.00	--
SA08 Variance	.17	.03	.000	1.00	.00	--
I Residual Variance	.66	.05	.000	.63	.05	.000
S Residual Variance	.003	.01	.59	.91	.26	.000
IR08 Residual variance	1.08	.04	.000	.51	.01	.000
IR10 Residual variance	1.62	.05	.000	.60	.01	.000
IR12 Residual variance	1.42	.07	.000	.55	.02	.000
R^2 IR08				.50	.01	.000
R^2 IR10				.40	.01	.000
R^2 IR12				.45	.02	.000
R^2 I				.37	.05	.000
R^2 S				.09	.26	.74

Note: I = intercept; S = slope; IR08 = immediate recall in 2008; IR10 = immediate recall in 2010; IR12 = immediate recall in 2012; CA08 = cognitive activities in 2008; SA08 = social activities in 2008.

Table 10

*Latent Growth Curve: Activity Frequency Predicting Intercept and Slope for Immediate Recall, With Education as a Covariate**

	Unstandardized coefficient	S.E.	*p*-value
I | IR08	1.00	.00	--
I | IR10	1.00	.00	--
I | IR12	1.00	.00	--
S | IR08	0.00	.00	--
S | IR10	2.00	.00	--
S | IR12	4.00	.00	--
I on CA08	-.95	.18	.000
I on SA08	.70	.22	.001
S on CA08	-.04	.04	.28
S on SA08	.04	.05	.41
I on Education	.16	.01	.000
I of I	3.48	.10	.000
I of S	-.07	.03	.01
CA08 Variance	.37	.04	.000
SA08 Variance	.20	.03	.000
I Residual Variance	.70	.04	.000
S Residual Variance	.01	.00	.23
IR08 Residual variance	1.05	.04	.000
IR10 Residual variance	1.65	.05	.000
IR12 Residual variance	1.38	.06	.000
R^2 IR08	.50		
R^2 IR10	.40		
R^2 IR12	.46		
R^2 I	.34		
R^2 S	.05		

* Note: The model was conducted with weighted least squares with mean and variance adjustment (WLSMV) estimation; as such, standard errors and *p*-values are not provided for standardized values when the model has covariates and only the unstandardized results are presented here. I = intercept; S = slope; IR08 = immediate recall in 2008; IR10 = immediate recall in 2010; IR12 = immediate recall in 2012; CA08 = cognitive activities in 2008; SA08 = social activities in 2008.

Table 11

Latent Growth Curve: Intercept-only for Delayed Recall Over Time

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
I | DR08	1.00	.00	--	.74	.01	.000
I | DR10	1.00	.00	--	.66	.01	.000
I | DR12	1.00	.00	--	.68	.01	.000
Mean I	4.32	.03	.000	3.26	.06	.000
I variance	1.75	.06	.000	1.00	.00	--
DR08 Residual variance	1.43	.05	.000	.45	.01	.000
DR10 Residual variance	2.22	.07	.000	.56	.01	.000
DR12 Residual variance	1.99	.06	.000	.53	.01	.000
R^2 DR08				.55	.01	.000
R^2 DR10				.44	.01	.000
R^2 DR12				.47	.01	.000

Note: I = intercept; DR08 = delayed recall in 2008; DR10 = delayed recall in 2010; DR12 = delayed recall in 2012.

Table 12

Latent Growth Curve: Intercept and Slope Only for Delayed Recall Over Time

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
I | DR08	1.00	.00	--	.76	.01	.000
I | DR10	1.00	.00	--	.66	.01	.000
I | DR12	1.00	.00	--	.69	.01	.000
S | DR08	0.00	.00	--	.00	.00	--
S | DR10	2.00	.00	--	.11	.03	.000
S | DR12	4.00	.00	--	.24	.06	.000
Mean I	4.54	.03	.000	3.42	.06	.000
Mean S	-.13	.01	.000	-1.10	.30	.000
I variance	1.76	.06	.000	1.00	.00	--
S variance	.01	.01	.06	1.00	.00	--
DR08 Residual variance	1.32	.05	.000	.43	.01	.000
DR10 Residual variance	2.26	.07	.000	.56	.01	.000
DR12 Residual variance	1.69	.10	.000	.46	.03	.000
R^2 DR08				.57	.01	.000
R^2 DR10				.45	.01	.000
R^2 DR12				.54	.03	.000

Note: I = intercept; S = slope; DR08 = delayed recall in 2008; DR10 = delayed recall in 2010; DR12 = delayed recall in 2012.

Table 13

Latent Growth Curve: Activity Frequency Predicting Intercept and Slope for Delayed Recall Over Time

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
I | DR08	1.00	.00	--	.75	.01	.000
I | DR10	1.00	.00	--	.66	.01	.000
I | DR12	1.00	.00	--	.68	.01	.000
S | DR08	0.00	.00	--	.00	.00	--
S | DR10	2.00	.00	--	.10	.03	.001
S | DR12	4.00	.00	--	.22	.07	.001
I on CA08	-1.90	.24	.000	-.88	.10	.000
I on SA08	1.53	.33	.000	.47	.10	.000
S on CA08	-.01	.04	.73	-.08	.25	.75
S on SA08	-.00	.06	.97	-.01	.23	.97
I of I	4.54	.03	.000	3.45	.07	.000
I of S	-.13	.01	.000	-1.20	.38	.01
CA08 Variance	.37	.03	.000	1.00	.00	--
SA08 Variance	.17	.03	.000	1.00	.00	--
I Residual Variance	1.12	.05	.000	.65	.05	.000
S Residual Variance	.01	.01	.12	.99	.02	.000
DR08 Residual variance	1.34	.05	.000	.44	.02	.000
DR10 Residual variance	2.19	.07	.000	.55	.01	.000
DR12 Residual variance	1.76	.10	.000	.47	.02	.000
R^2 DR08				.56	.02	.000
R^2 DR10				.45	.01	.000
R^2 DR12				.53	.02	.000
R^2 I				.36	.05	.000
R^2 S				.01	.02	.69

Note: I = intercept; S = slope; DR08 = delayed recall in 2008; DR10 = delayed recall in 2010; DR12 = delayed recall in 2012; CA08 = cognitive activities in 2008; SA08 = social activities in 2008.

Table 14

*Latent Growth Curve: Activity Frequency Predicting Intercept and Slope for Delayed Recall, With Education as a Covariate**

	Unstandardized coefficient	S.E.	*p*-value
I | DR08	1.00	.00	--
I | DR10	1.00	.00	--
I | DR12	1.00	.00	--
S | DR08	0.00	.00	--
S | DR10	2.00	.00	--
S | DR12	4.00	.00	--
I on CA08	-1.38	.24	.000
I on SA08	1.21	.30	.000
S on CA08	-.01	.04	.90
S on SA08	-.01	.06	.88
I on Education	.17	.01	.000
I of I	2.33	.13	.000
I of S	-.13	.04	.000
CA08 Variance	.38	.04	.000
SA08 Variance	.20	.03	.000
I Residual Variance	1.18	.08	.000
S Residual Variance	.01	.01	.05
DR08 Residual variance	1.33	.05	.000
DR10 Residual variance	2.20	.07	.000
DR12 Residual variance	1.74	.09	.000
R^2 DR08	.57		
R^2 DR10	.45		
R^2 DR12	.53		
R^2 I	.33		
R^2 S	.00		

* Note: The model was conducted with weighted least squares with mean and variance adjustment (WLSMV) estimation; as such, standard errors and *p*-values are not provided for standardized values when the model has covariates and only the unstandardized results are presented here. I = intercept; S = slope; DR08 = delayed recall in 2008; DR10 = delayed recall in 2010; DR12 = delayed recall in 2012; CA08 = cognitive activities in 2008; SA08 = social activities in 2008.

Table 15

Chi-square Difference Tests for Cognitive Activity Measurement Invariance

	Chi-square (*df*)	Absolute difference (*df* difference)
Without invariance	25.07 (15)	
Factor loading invariance	27.30 (18)	2.23 (3)
Factor loading and intercept invariance	158.63 (17)	131.33 (1)*
Factor loading and partial intercept invariance[A]	36.07 (16)	8.77 (1)*
Factor loading and partial intercept invariance[B]	25.07 (15)	2.23 (3)

Note: [A] free "hobby" in 2012 based on modification indices; [B] free "hobby" in 2012 based on modification indices, and free "writing" in 2008 based on modification indices and expected parameter change.

Table 16

Latent Difference: Change in Cognitive Activities from 2008 to 2012

	Unstandardized coefficient	S.E.	*p*	Standardized coefficient	S.E.	*p*
CA08 BY						
Education	1.00	.00	--	.49	.04	.000
Writing	1.22	.11	.000	.60	.03	.000
Use computer	1.12	.10	.000	.55	.03	.000
Hobby	1.10	.10	.000	.54	.03	.000
CA12 BY						
Education	1.00	.00	--	.51	.04	.000
Writing	1.22	.11	.000	.54	.03	.000
Use computer	1.12	.10	.000	.53	.03	.000
Hobby	1.10	.10	.000	.52	.03	.000
Education, 08 with12	.31	.03	.000	.51	.04	.000
Writing, 08 with 12	.37	.05	.000	.58	.02	.000
Use computer, 08 with12	.60	.06	.000	.97	.01	.000
Hobby, 08 with 12	.34	.04	.000	.54	.02	.000
Mean of CA08	1.08	.03	.000	2.19	.17	.000
Mean of Difference Factor	.01	.02	.41	.05	.06	.40
Variance of CA08	.24	.03	.000	1.00	.00	--
Variance of Difference Factor	.07	.01	.000	1.00	.00	--
Residual Variance of CA12	.00	.00	--	.00	--	--
R^2 of Education 08				.24	.03	.000
R^2 of Writing 08				.36	.03	.000
R^2 of Use computer 08				.31	.03	.000
R^2 of Hobby 08				.29	.03	.000
R^2 of Education 12				.26	.04	.000
R^2 of Writing 12				.29	.03	.000
R^2 of Use computer 12				.29	.03	.000
R^2 of Hobby 12				.27	.03	.000

Note: CA08 = cognitive activities in 2008; CA12 = cognitive activities in 2012.

Table 17

Chi-square Difference Tests for Social Activity Measurement Invariance

	Chi-square (*df*)	Absolute difference (*df* difference)
No constraints	201.09 (15)	
Factor loading invariance	196.61 (18)	4.48 (3)
Factor loading and intercept invariance	200.90 (17)	4.29 (1)*
Factor loading and partial intercept invariance[A]	203.09 (16)	6.48 (2)*
Factor loading and partial intercept invariance[B]	201.09 (15)	4.48 (3)

Note: [A] free "volunteer with youth" in 2012; [B] free "volunteer with youth" in 2012 and "clubs" in 2008.

Table 18

Latent Difference: Change in Social Activities from 2008 to 2012

	Unstandardized coefficient	S.E.	*p*	Standardized coefficient	S.E.	*p*
SA08 BY						
Charity work	1.00	.00	--	.67	.02	.000
Volunteer with youth	.64	.05	.000	.43	.03	.000
Club	.75	.04	.000	.51	.03	.000
Non-religious organization	1.09	.06	.000	.74	.03	.000
SA12 BY						
Charity work	1.00	.00	--	.65	.02	.000
Volunteer with youth	.64	.05	.000	.55	.03	.000
Club	.75	.04	.000	.55	.02	.000
Non-religious organization	1.09	.06	.000	.74	.03	.000
Volunteer with youth, 08 with 12	.31	.07	.000	.52	.03	.000
Charity work, 08 with 12	.41	.09	.000	.71	.03	.000
Club, 08 with 12	.38	.08	.000	.58	.02	.000
Non-religious organization, 08 with 12	.14	.03	.000	.31	.05	.000
Mean of SA08	.41	.02	.000	.61	.04	.000
Mean of Difference Factor	.17	.13	.21	.35	.24	.000
Variance of SA08	.46	.03	.000	1.00	.00	--
Variance of Difference Factor	.24	.06	.000	1.00	.00	--
Residual Variance of SA12	.00	--	--	.00	--	--
R^2 of Volunteer with youth 08				.19	.03	.82
R^2 of Charity work 08				.30	.04	.43
R^2 of Non-religious org. 08				.54	.04	.46
R^2 of Club 08				.26	.03	.74
R^2 of Volunteer with youth 12				.46	.03	.55
R^2 of Charity work 12				.42	.03	.63
R^2 of Non-religious org. 12				.55	.04	.44
R^2 of Club 12				.31	.03	.58

Note: SA08 = social activities in 2008; SA12 = social activities in 2012.

Table 19

Latent Growth Curve: Intercept-only for Serial 7s Over Time

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
I | Serial 7s 08	1.00	.00	--	.81	.01	.000
I | Serial 7s 10	1.00	.00	--	.82	.01	.000
I | Serial 7s 12	1.00	.00	--	.79	.01	.000
Mean I	3.58	.03	.000	2.72	.04	.000
I variance	1.73	.05	.000	1.00	.00	--
Serial 7s 08 Residual variance	.90	.03	.000	.34	.01	.000
Serial 7s 10 Residual variance	.86	.03	.000	.33	.01	.000
Serial 7s 12 Residual variance	1.03	.03	.000	.37	.01	.000
R^2 Serial 7s 08				.66	.01	.000
R^2 Serial 7s 10				.67	.01	.000
R^2 Serial 7s 12				.63	.01	.000

Note: I = intercept; S = slope.

Table 20

Latent Growth Curve: Intercept and Slope Only for Serial 7s Over Time

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
I | Serial 7s 08	1.00	.00	--	.82	.01	.000
I | Serial 7s 10	1.00	.00	--	.81	.01	.000
I | Serial 7s 12	1.00	.00	--	.79	.01	.000
S | Serial 7s 08	0.00	.00	--	.00	.00	--
S | Serial 7s 10	2.00	.00	--	.11	.03	.000
S | Serial 7s 12	4.00	.00	--	.21	.06	.000
Mean I	3.68	.03	.000	2.81	.05	.000
Mean S	-.06	.01	.000	-.63	.18	.000
I variance	1.72	.05	.000	1.00	.00	--
S variance	.01	.00	.06	1.00	.00	--
Serial 7s 08 Residual variance	.85	.04	.000	.33	.01	.000
Serial 7s 10 Residual variance	.88	.03	.000	.34	.01	.000
Serial 7s 12 Residual variance	.92	.05	.000	.33	.02	.000
R^2 Serial 7s 08				.67	.01	.000
R^2 Serial 7s 10				.66	.01	.000
R^2 Serial 7s 12				.67	.02	.000

Note: I = intercept; S = slope.

Table 21

Latent Growth Curve: Activity Frequency Predicting Intercept and Slope for Serial 7s Over Time

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
I | Serial 7s 08	1.00	.00	--	.81	.01	.000
I | Serial 7s 10	1.00	.00	--	.81	.01	.000
I | Serial 7s 12	1.00	.00	--	.77	.02	.000
S | Serial 7s 08	0.00	.00	--	.00	.00	--
S | Serial 7s 10	2.00	.00	--	.09	.05	.10
S | Serial 7s 12	4.00	.00	--	.16	.10	.11
I on CA08	-1.01	.19	.000	-.47	.08	.000
I on SA08	.63	.30	.03	.18	.08	.03
S on CA08	-.05	.04	.16	-.46	.44	.29
S on SA08	.04	.06	.47	.22	.35	.53
I of I	3.68	.04	.000	2.84	.05	.000
I of S	-.06	.01	.000	-.83	.63	.19
CA08 Variance	.37	.04	.000	1.00	.00	--
SA08 Variance	.13	.02	.000	1.00	.00	--
I Residual Variance	1.47	.07	.000	.87	.03	.000
S Residual Variance	.004	.01	.47	.90	.17	.000
Serial 7s 08 Residual variance	.88	.05	.000	.34	.02	.000
Serial 7s 10 Residual variance	.83	.06	.000	.32	.02	.000
Serial 7s 12 Residual variance	.99	.08	.000	.35	.03	.000
R^2 Serial 7s 08				.66	.02	.000
R^2 Serial 7s 10				.68	.02	.000
R^2 Serial 7s 12				.65	.03	.000
R^2 I				.13	.03	.000
R^2 S				.10	.17	.54

Note: I = intercept; S = slope; CA08 = cognitive activities in 2008; SA08 = social activities in 2008.

Table 22

*Latent Growth Curve: Activity Frequency Predicting Intercept and Slope for Serial 7s, With Education as a Covariate**

	Unstandardized coefficient	S.E.	*p*-value
I | Serial 7s 08	1.00	.00	--
I | Serial 7s 10	1.00	.00	--
I | Serial 7s 12	1.00	.00	--
S | Serial 7s 08	0.00	.00	--
S | Serial 7s 10	2.00	.00	--
S | Serial 7s 12	4.00	.00	--
I on CA08	-.27	.16	.08
I on SA08	.18	.22	.43
S on CA08	-.05	.04	.22
S on SA08	.04	.05	.51
I on Education	.20	.01	.000
I of I	1.14	.11	.000
I of S	-.09	.03	.000
CA08 Variance	.41	.04	.000
SA08 Variance	.18	.03	.000
I Residual Variance	1.35	.06	.000
S Residual Variance	.01	.01	.22
Serial 7s 08 Residual variance	.87	.04	.000
Serial 7s 10 Residual variance	.83	.05	.000
Serial 7s 12 Residual variance	.98	.06	.000
R^2 Serial 7s 08	.66		
R^2 Serial 7s 10	.68		
R^2 Serial 7s 12	.65		
R^2 I	.21		
R^2 S	.07		

* Note: The model was conducted with weighted least squares with mean and variance adjustment (WLSMV) estimation; as such, standard errors and *p*-values are not provided for standardized values when the model has covariates and only the unstandardized results are

presented here. I = intercept; S = slope; CA08 = cognitive activities in 2008; SA08 = social activities in 2008.

Table 23

Latent Growth Curve: Intercept-only for Health Over Time

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
I | MedSum08	1.00	.00	--	.89	.00	.000
I | MedSum10	1.00	.00	--	.95	.00	.000
I | MedSum12	1.00	.00	--	.91	.00	.000
Mean I	1.59	.02	.000	1.47	.03	.000
I variance	1.17	.03	.000	1.00	.00	--
MedSum08 Residual variance	.30	.01	.000	.21	.01	.000
MedSum10 Residual variance	.12	.01	.000	.09	.01	.000
MedSum12 Residual variance	.25	.01	.000	.18	.01	.000
R^2 MedSum08				.80	.01	.000
R^2 MedSum10				.91	.01	.000
R^2 MedSum12				.82	.01	.000

Note: I = intercept; MedSum08 = summed medical conditions in 2008; MedSum10 = summed medical conditions in 2010; MedSum12 = summed medical conditions in 2012.

Table 24

Latent Growth Curve: Intercept and Slope Only for Health Over Time

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
I | MedSum08	1.00	.00	--	.96	.01	.000
I | MedSum10	1.00	.00	--	.95	.01	.000
I | MedSum12	1.00	.00	--	.94	.01	.000
S | MedSum08	0.00	.00	--	.00	.00	--
S | MedSum10	2.00	.00	--	.27	.01	.000
S | MedSum12	4.00	.00	--	.53	.03	.000
Mean I	1.47	.02	.000	1.33	.03	.000
Mean S	.05	.00	.000	.32	.03	.000
I variance	1.22	.04	.000	1.00	.00	--
S variance	.02	.00	.000	1.00	.00	--
MedSum08 Residual variance	.10	.02	.000	.08	.02	.000
MedSum10 Residual variance	.20	.01	.000	.14	.01	.000
MedSum12 Residual variance	.06	.02	.001	.05	.01	.001
R^2 MedSum08				.92	.02	.000
R^2 MedSum10				.86	.01	.000
R^2 MedSum12				.95	.01	.000

Note: I = intercept; S = slope; MedSum08 = summed medical conditions in 2008; MedSum10 = summed medical conditions in 2010; MedSum12 = summed medical conditions in 2012.

Table 25

Latent Growth Curve: Activity Frequency Predicting Intercept and Slope for Health Over Time

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
I | MedSum08	1.00	.00	--	.96	.01	.000
I | MedSum10	1.00	.00	--	.95	.01	.000
I | MedSum12	1.00	.00	--	.94	.01	.000
S | MedSum08	0.00	.00	--	.00	.00	--
S | MedSum10	2.00	.00	--	.27	.01	.000
S | MedSum12	4.00	.00	--	.53	.03	.000
I on CA08	.26	.11	.02	.15	.06	.02
I on SA08	.02	.18	.91	.01	.06	.91
S on CA08	.01	.02	.56	.05	.08	.56
S on SA08	.02	.03	.56	.04	.08	.56
I of I	1.47	.02	.000	1.34	.03	.000
I of S	.05	.00	.000	.33	.03	.000
CA08 Variance	.41	.04	.000	1.00	.00	--
SA08 Variance	.16	.03	.000	1.00	.00	--
Intercept Residual Variance	1.19	.04	.000	.98	.01	.000
Slope Residual Variance	.02	.00	.000	.99	.01	.000
MedSum08 Residual variance	.10	.02	.000	.08	.01	.000
MedSum10 Residual variance	.19	.01	.000	.14	.01	.000
MedSum12 Residual variance	.0	.02	.001	.05	.01	.001
R^2 MedSum08				.92	.01	.000
R^2 MedSum10				.86	.01	.000
R^2 MedSum12				.96	.01	.000
R^2 I				.03	.01	.001
R^2 S				.01	.01	.12

Note: I = intercept; S = slope; MedSum08 = summed medical conditions in 2008; MedSum10 = summed medical conditions in 2010; MedSum12 = summed medical conditions in 2012. CA08 = cognitive activities in 2008; SA08 = social activities in 2008.

Table 26

*Latent Growth Curve: Activity Frequency Predicting Intercept and Slope for Health, With Education as a Covariate**

	Unstandardized coefficient	S.E.	*p*-value
I | MedSum08	1.00	.00	--
I | MedSum10	1.00	.00	--
I | MedSum12	1.00	.00	--
S | MedSum08	0.00	.00	--
S | MedSum10	2.00	.00	--
S | MedSum12	4.00	.00	--
I on CA08	.11	.12	.33
I on SA08	.09	.17	.60
S on CA08	.01	.02	.59
S on SA08	.01	.03	.62
I on Education	-.05	.01	.000
S on Education	-.00	.00	.09
I of I	2.12	.08	.000
I of S	.08	.02	.000
CA08 Variance	.41	.04	.000
SA08 Variance	.19	.03	.000
I Residual Variance	1.18	.04	.000
S Residual Variance	.02	.00	.000
MedSum08 Residual variance	.11	.02	.000
MedSum10 Residual variance	.19	.01	.000
MedSum12 Residual variance	.07	.02	.001
R^2 MedSum08	.92		
R^2 MedSum10	.86		
R^2 MedSum12	.95		
R^2 I	.03		
R^2 S	.01		

* Note: The model was conducted with weighted least squares with mean and variance adjustment (WLSMV) estimation; as such, standard errors and *p*-values are not provided for standardized values when the model has covariates and only the unstandardized results are presented here. I = intercept; S = slope; MedSum08 = summed medical conditions in 2008;

MedSum10 = summed medical conditions in 2010; MedSum12 = summed medical conditions in 2012. CA08 = cognitive activities in 2008; SA08 = social activities in 2008.

Table 27

Cross Lagged Panel for Summed Health Conditions Over Time

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
MedSum10 on MedSum08	.87	.01	.000	.87	.00	.000
MedSum12 on MedSum10	.87	.01	.000	.85	.01	.000
I of MedSum10	.34	.02	.000	.29	.02	.000
I of MedSum12	.27	.02	.000	.23	.02	.000
MedSum10 Residual Variance	.37	.01	.000	.27	.01	.000
MedSum12 Residual Variance	.33	.01	.000	.24	.01	.000
R^2 MedSum10				.73	.01	.000
R^2 MedSum12				.76	.01	.000

Note: I = intercept; MedSum08 = summed medical conditions in 2008; MedSum10 = summed medical conditions in 2010; MedSum12 = summed medical conditions in 2012.

Table 28

Cross Lagged Panel for Immediate Recall Over Time

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
IR10 on IR08	.47	.02	.000	.41	.01	.000
IR12 on IR10	.41	.02	.000	.42	.01	.000
I of IR10	2.66	.10	.000	1.62	.07	.000
I of IR12	2.97	.08	.000	1.85	.07	.000
IR10 Residual Variance	2.25	.06	.000	.83	.01	.000
IR12 Residual Variance	2.12	.05	.000	.82	.01	.000
R^2 IR10				.17	.01	.000
R^2 IR12				.18	.01	.000

Note: I = intercept; IR08 = immediate recall in 2008; IR10 = immediate recall in 2010; IR12 = immediate recall in 2012.

Table 29

Cross Lagged Panel for Delayed Recall Over Time

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
DR10 on DR08	.55	.02	.000	.48	.01	.000
DR12 on DR10	.46	.02	.000	.48	.01	.000
I of DR10	1.86	.08	.000	.93	.05	.000
I of DR12	2.00	.07	.000	1.04	.04	.000
DR10 Residual Variance	3.05	.07	.000	.77	.01	.000
DR12 Residual Variance	2.85	.07	.000	.77	.01	.000
R^2 DR10				.23	.01	.000
R^2 DR12				.23	.01	.000

Note: I = intercept; DR08 = delayed recall in 2008; DR10 = delayed recall in 2010; DR12 = delayed recall in 2012.

Table 30

Cross Lagged Panel for Serial 7s Over Time

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
Serial10 on Serial08	.65	.01	.000	.65	.01	.000
Serial12 on Serial10	.69	.01	.000	.66	.01	.000
I of Serial10	1.21	.05	.000	.76	.04	.000
I of Serial12	.94	.05	.000	.56	.04	.000
Serial10 Residual Variance	1.48	.04	.000	.58	.01	.000
Serial12 Residual Variance	1.59	.04	.000	.56	.01	.000
R^2 Serial10				.42	.01	.000
R^2 Serial12				.44	.01	.000

Note: I = intercept; Serial08 = Serial 7s in 2008; Serial10 = Serial 7s in 2010; Serial12 = Serial 7s in 2012.

Table 31

*Cross Lagged Panel for Naming Over Time**

	Unstandardized coefficient	S.E.	*p*-value
Naming10 on Naming08	1.22	.07	.000
Naming12 on Naming10	1.01	.06	.000
R^2 Naming10	.18		
R^2 Naming12	.55		

* Note: The model was conducted with weighted least squares with mean and variance adjustment (WLSMV) estimation; as such, standard errors and *p*-values are not provided for standardized values when the model has covariates and only the unstandardized results are presented here.

Table 32

Cross Lagged Panel for Immediate Recall and Summed Health Conditions Over Time

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
MedSum12 on MedSum10	.87	.01	.000	.87	.004	.000
MedSum10 on MedSum08	.86	.01	.000	.85	.01	.000
IR12 on IR10	.41	.02	.000	.42	.01	.000
IR10 on IR08	.47	.02	.000	.41	.01	.000
MedSum12 on IR10	-.01	.01	.26	-.01	.01	.26
MedSum10 on IR08	-.03	.01	.001	-.03	.01	.001
IR12 on MedSum10	-.07	.02	.003	-.05	.02	.003
IR10 on MedSum08	-.05	.02	.02	-.04	.02	.02
Mean for IR08	5.57	.03	.000	3.81	.05	.000
I of IR10	2.74	.11	.000	1.66	.08	.000
I of IR12	3.09	.09	.000	1.92	.07	.000
Mean for MedSum08	1.46	.02	.000	1.27	.02	.000
I of MedSum10	.48	.04	.000	.41	.04	.000
I of MedSum12	.30	.04	.000	.26	.03	.000
IR08 Variance	2.14	.05	.000	1.00	.00	--
IR10 Residual Var.	2.25	.06	.000	.83	.01	.000
IR12 Residual Var.	2.11	.05	.000	.82	.01	.000
MedSum08 Variance	1.32	.03	.000	1.00	.00	--
MedSum10 Residual Var.	.37	.01	.000	.27	.01	.000
MedSum12 Residual Var.	.33	.01	.000	.24	.01	.000
R^2 IR10				.17	.01	.000
R^2 IR12				.18	.01	.000
R^2 MedSum10				.73	.01	.000
R^2 MedSum12				.76	.01	.000

Note: I = intercept; MedSum08 = summed medical conditions in 2008; MedSum10 = summed medical conditions in 2010; MedSum12 = summed medical conditions in 2012; IR08 = immediate recall in 2008; IR10 = immediate recall in 2010; IR12 = immediate recall in 2012.

Table 33

Cross Lagged Panel for Immediate Recall and Health Over Time with Education

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
MedSum12 on MedSum10	.87	.01	.000	.87	.004	.000
MedSum10 on MedSum08	.86	.01	.000	.85	.01	.000
IR12 on IR10	.37	.02	.000	.38	.02	.000
IR10 on IR08	.42	.02	.000	.37	.02	.000
MedSum12 on IR10	-.01	.01	.46	-.01	.01	.46
MedSum10 on IR08	-.02	.01	.04	-.02	.01	.04
IR12 on MedSum10	-.03	.02	.12	-.02	.02	.12
IR10 on MedSum08	-.04	.02	.11	-.03	.02	.11
MedSum12 on Education	-.01	.00	.18	-.01	.01	.18
MedSum10 on Education	-.01	.00	.000	-.04	.01	.000
MedSum08 on Education	-.05	.01	.000	-.12	.02	.000
IR12 on Education	.10	.01	.000	.18	.02	.000
IR10 on Education	.07	.01	.000	.12	.02	.000
IR12 on Education	.17	.01	.000	.33	.02	.000
I of IR08	3.44	.11	.000	2.35	.09	.000
I of IR10	2.06	.14	.000	1.25	.09	.000
I of IR12	1.95	.13	.000	1.21	.09	.000
I of MedSum08	2.08	.09	.000	1.81	.08	.000
I of MedSum10	.61	.06	.000	.53	.05	.000
I of MedSum12	.35	.05	.000	.30	.05	.000
IR08 Residual Var.	1.91	.05	.000	.89	.01	.000
IR10 Residual Var.	2.22	.05	.000	.81	.01	.000
IR12 Residual Var.	2.03	.05	.000	.79	.01	.000
MedSum08 Residual Var.	1.30	.03	.000	.99	.00	.000
MedSum10 Residual Var.	.37	.01	.000	.27	.01	.000
MedSum12 Residual Var.	.33	.01	.000	.24	.01	.000
R^2 IR08				.11	.01	.000
R^2 IR10				.19	.01	.000
R^2 IR12				.21	.01	.000
R^2 MedSum08				.02	.00	.000
R^2 MedSum10				.73	.01	.000
R^2 MedSum12				.76	.01	.000

Note: I = intercept; MedSum08 = summed medical conditions in 2008; MedSum10 = summed medical conditions in 2010; MedSum12 = summed medical conditions in 2012; IR08 = immediate recall in 2008; IR10 = immediate recall in 2010; IR12 = immediate recall in 2012.

Table 34

Cross Lagged Panel for Immediate Recall and Health Over Time with Education and Age

	Unstandardized coefficient	S.E.	*p*	Standardized coefficient	S.E.	*p*
MedSum12 on MedSum10	.87	.01	.000	.87	.00	.000
MedSum10 on MedSum08	.86	.01	.000	.85	.01	.000
IR12 on IR10	.30	.02	.000	.31	.02	.000
IR10 on IR08	.36	.02	.000	.32	.02	.000
MedSum12 on IR10	-.01	.01	.38	-.01	.01	.38
MedSum10 on IR08	-.02	.01	.05	-.02	.01	.05
IR12 on MedSum10	-.03	.02	.14	-.02	.02	.14
IR10 on MedSum08	-.03	.02	.14	-.02	.02	.14
MedSum12 on Education	-.01	.00	.18	-.01	.01	.18
MedSum10 on Education	-.01	.00	.000	-.04	.01	.000
IR12 on Education	.11	.01	.000	.19	.02	.000
IR10 on Education	.07	.01	.000	.13	.02	.000
MedSum08 on Education	-.05	.01	.000	-.12	.02	.000
IR08 on Education	.16	.01	.000	.31	.02	.000
MedSum12 on Age12	-.00	.00	.53	-.01	.01	.53
MedSum10 on Age10	.00	.00	.80	.002	.01	.80
IR12 on Age12	-.05	.00	.000	-.22	.02	.000
IR10 on Age10	-05	.00	.000	-.21	.02	.000
MedSum08 on Age08	.00	.00	.19	.02	.02	.19
IR08 on Age08	-.05	.00	.000	-.24	.02	.000
I of IR08	7.13	.27	.000	4.88	.18	.000
I of IR10	5.92	.33	.000	3.58	.20	.000
I of IR12	6.19	.32	.000	3.85	.20	.000
I of MedSum08	1.81	.23	.000	1.57	.20	.000
I of MedSum10	.58	.14	.000	.50	.12	.000
I of MedSum12	.43	.13	.001	.37	.11	.001
IR08 Residual Var.	1.79	.04	.000	.84	.01	.000
IR10 Residual Var.	2.11	.05	.000	.77	.01	.000
IR12 Residual Var.	1.92	.05	.000	.74	.01	.000
MedSum08 Residual Var.	1.30	.03	.000	.99	.00	.000
MedSum10 Residual Var.	.37	.01	.000	.27	.01	.000
MedSum12 Residual Var.	.33	.01	.000	.24	.01	.000
R^2 IR08				.16	.01	.000
R^2 IR10				.23	.01	.000

R^2 IR12	.26	.01	.000
R^2 MedSum08	.02	.00	.000
R^2 MedSum10	.73	.01	.000
R^2 MedSum12	.76	.01	.000

Note: I = intercept; MedSum08 = summed medical conditions in 2008; MedSum10 = summed medical conditions in 2010; MedSum12 = summed medical conditions in 2012; IR08 = immediate recall in 2008; IR10 = immediate recall in 2010; IR12 = immediate recall in 2012; Age08 = age in 2008; Age10 = age in 2010; Age12 = age in 2012.

Table 35

Cross Lagged Panel for Delayed Recall and Health Over Time

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
MedSum12 on MedSum10	.87	.01	.000	.87	.004	.000
MedSum10 on MedSum08	.86	.01	.000	.85	.01	.000
DR12 on DR10	.46	.02	.000	.48	.01	.000
DR10 on DR08	.55	.02	.000	.48	.01	.000
MedSum12 on DR10	-.00	.01	.55	-.01	.01	.55
MedSum10 on DR08	-.03	.01	.000	-.04	.01	.000
DR12 on MedSum10	-09	.03	.000	-.06	.02	.000
DR10 on MedSum08	-.03	.03	.21	-.02	.02	.21
Mean of DR08	4.52	.03	.000	2.58	.04	.000
I of DR10	1.90	.09	.000	.95	.05	.000
I of DR12	2.16	.08	.000	1.12	.05	.000
Mean of MedSum08	1.46	.02	.000	1.27	.02	.000
I of MedSum10	.47	.03	.000	.40	.03	.000
I of MedSum12	.28	.03	.000	.24	.03	.000
DR08 Variance	3.08	.08	.000	1.00	.00	--
DR10 Residual Var.	3.06	.08	.000	.77	.01	--
DR12 Residual Var.	2.84	.07	.000	.76	.01	.000
MedSum08 Variance	1.32	.03	.000	1.00	.00	--
MedSum10 Residual Var.	.37	.01	.000	.27	.01	.000
MedSum12 Residual Var.	.33	.01	.000	.24	.01	.000
R^2 DR10				.24	.01	.000
R^2 DR12				.24	.01	.000
R^2 MedSum10				.73	.01	.000
R^2 MedSum12				.76	.01	.000

Note: I = intercept; MedSum08 = summed medical conditions in 2008; MedSum10 = summed medical conditions in 2010; MedSum12 = summed medical conditions in 2012; IR08 = immediate recall in 2008; IR10 = immediate recall in 2010; IR12 = immediate recall in 2012.

Table 36

Cross Lagged Panel for Delayed Recall and Health Over Time with Education

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
MedSum12 on MedSum10	.87	.01	.000	.87	.00	.000
MedSum10 on MedSum08	.86	.01	.000	.85	.01	.000
DR12 on DR10	.43	.02	.000	.44	.01	.000
DR10 on DR08	.51	.02	.000	.45	.01	.000
MedSum12 on DR10	-.00	.01	.84	-.00	.01	.84
MedSum10 on DR08	-.02	.01	.000	-.03	.01	.000
DR12 on MedSum10	-.06	.03	.02	-.04	.02	.02
DR10 on MedSum08	-.01	.03	.61	-.01	.02	.61
MedSum08 on Education	-.05	.01	.000	-.12	.02	.000
MedSum12 on Education	-.01	.00	.14	-.01	.01	.14
MedSum10 on Education	-.01	.00	.001	-.03	.01	.001
DR12 on Education	.10	.01	.000	.15	.02	.000
DR10 on Education	.08	.01	.000	.12	.02	.000
DR08 on Education	.17	.01	.000	.29	.02	.000
I of DR08	2.29	.13	.000	1.31	.08	.000
I of DR10	1.02	.15	.000	.51	.08	.000
I of DR12	.97	.15	.000	.50	.08	.000
I of MedSum08	2.08	.09	.000	1.81	.08	.000
I of MedSum10	.61	.05	.000	.52	.05	.000
I of MedSum12	.34	.05	.000	.29	.04	.000
DR08 Residual Var.	2.83	.07	.000	.92	.01	.000
DR10 Residual Var.	3.01	.07	.000	.75	.01	.000
DR12 Residual Var.	2.76	.07	.000	.74	.01	.000
MedSum08 Residual Var.	1.30	.03	.000	.99	.00	.000
MedSum10 Residual Var.	.37	.01	.000	.27	.01	.000
MedSum12 Residual Var.	.33	.01	.000	.24	.01	.000
R^2 DR08				.08	.01	.000
R^2 DR10				.25	.01	.000
R^2 DR12				.26	.01	.000
R^2 MedSum08				.02	.00	.000
R^2 MedSum10				.73	.01	.000
R^2 MedSum12				.76	.01	.000

Note: I = intercept; MedSum08 = summed medical conditions in 2008; MedSum10 = summed medical conditions in 2010; MedSum12 = summed medical conditions in 2012; DR08 = delayed recall in 2008; DR10 = delayed recall in 2010; DR12 = delayed recall in 2012.

Table 37

Cross Lagged Panel for Delayed Recall and Health Over Time with Education and Age

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
MedSum12 on MedSum10	.87	.01	.000	.87	.00	.000
MedSum10 on MedSum08	.86	.01	.000	.85	.01	.000
DR12 on DR10	.36	.02	.000	.38	.02	.000
DR10 on DR08	.45	.02	.000	.40	.02	.000
MedSum12 on DR10	.00	.01	.74	.00	.01	.74
MedSum10 on DR08	-.02	.01	.001	-.03	.01	.001
DR12 on MedSum10	-.05	.02	.03	-.03	.02	.03
DR10 on MedSum08	-.01	.03	.72	-.01	.02	.72
MedSum08 on Education	-.05	.01	.000	-.12	.02	.000
MedSum12 on Education	-.01	.00	.14	-.01	.01	.14
MedSum10 on Education	-.01	.00	.001	-.03	.01	.001
DR12 on Education	.10	.01	.000	.15	.02	.000
DR10 on Education	.08	.01	.000	.12	.02	.000
DR08 on Education	.17	.01	.000	.27	.02	.000
MedSum12 on Age12	.00	.00	.63	-.004	.01	.63
MedSum10 on Age10	.00	.00	.89	-.001	.01	.89
MedSum08 on Age08	.00	.00	.19	.02	.02	.19
DR12 on Age12	-.06	.00	.000	-.21	.02	.000
DR10 on Age10	-.06	.00	.000	-.20	.02	.000
DR08 on Age08	-.06	.00	.000	-.25	.02	.000
I of DR08	6.93	.32	.000	3.95	.18	.000
I of DR10	5.52	.37	.000	2.76	.19	.000
I of DR12	5.69	.37	.000	2.95	.19	.000
I of MedSum08	1.81	.23	.000	1.57	.20	.000
I of MedSum10	.63	.13	.000	.54	.11	.000
I of MedSum12	.40	.13	.000	.34	.11	.002
DR08 Residual Var.	2.63	.06	.000	.86	.01	.000
DR10 Residual Var.	2.86	.07	.000	.72	.01	.000
DR12 Residual Var.	2.61	.06	.000	.70	.01	.000
MedSum08 Residual Var.	1.30	.03	.000	.99	.00	.000
MedSum10 Residual Var.	.37	.01	.000	.27	.01	.000
MedSum12 Residual Var.	.33	.01	.000	.24	.01	.000
R^2 DR08				.14	.01	.000
R^2 DR10				.28	.01	.000

R^2 DR12	.30	.01	.000
R^2 MedSum08	.02	.00	.000
R^2 MedSum10	.73	.01	.000
R^2 MedSum12	.76	.01	.000

Note: I = intercept; MedSum08 = summed medical conditions in 2008; MedSum10 = summed medical conditions in 2010; MedSum12 = summed medical conditions in 2012; DR08 = delayed recall in 2008; DR10 = delayed recall in 2010; DR12 = delayed recall in 2012; Age08 = age in 2008; Age10 = age in 2010; Age12 = age in 2012.

Table 38

Cross Lagged Panel for Serial 7s and Health Over Time

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
MedSum12 on MedSum10	.87	.01	.000	.87	.004	.000
MedSum10 on MedSum08	.86	.01	.000	.85	.01	.000
Serial12 on Serial10	.70	.01	.000	.66	.01	.000
Serial10 on Serial08	.65	.01	.000	.65	.01	.000
MedSum12 on Serial10	.00	.01	.88	.00	.01	.88
MedSum10 on Serial08	-.02	.01	.01	-.03	.01	.01
Serial12 on MedSum10	-.04	.02	.03	-.03	.01	.03
Serial10 on MedSum08	-.07	.02	.000	-.05	.01	.000
Mean of Serial08	3.67	.03	.000	2.29	.03	.000
I of Serial10	1.33	.06	.000	.83	.04	.000
I of Serial12	1.02	.06	.000	.61	.04	.000
Mean of MedSum08	1.46	.02	.000	1.27	.02	.000
I of MedSum10	.41	.03	.000	.35	.03	.000
I of MedSum12	.26	.03	.000	.22	.03	.000
Serial08 Variance	2.56	.06	.000	1.00	.00	---
Serial10 Residual Var.	1.47	.04	.000	.57	.01	.000
Serial12 Residual Var.	1.59	.04	.000	.56	.01	.000
MedSum08 Variance	1.32	.03	.000	1.00	00	--
MedSum10 Residual Var.	.37	.01	.000	.27	.01	.000
MedSum12 Residual Var.	.33	.01	.000	.24	.01	.000
R^2 Serial10				.43	.01	.000
R^2 Serial12				.44	.01	.000
R^2 MedSum10				.73	.01	.000
R^2 MedSum12				.76	.01	.000

Note: I = intercept; MedSum08 = summed medical conditions in 2008; MedSum10 = summed medical conditions in 2010; MedSum12 = summed medical conditions in 2012; Serial08 = Serial 7s in 2008; Serial10 = Serial 7s in 2010; Serial12 = Serial 7s in 2012.

Table 39

Cross Lagged Panel for Serial 7s and Health Over Time with Education

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
MedSum12 on MedSum10	.87	.01	.000	.87	.004	.000
MedSum10 on MedSum08	.86	.01	.000	.85	.01	.000
Serial12 on Serial10	.64	.01	.000	.61	.01	.000
Serial10 on Serial08	.60	.01	.000	.60	.01	.000
MedSum12 on Serial10	.01	.01	.45	.01	.01	.45
MedSum10 on Serial08	-.01	.01	.22	-.01	.01	.22
Serial12 on MedSum10	-.02	.02	.27	-.01	.01	.27
Serial10 on MedSum08	-.05	.02	.003	-.04	.01	.003
MedSum12 on Education	-.01	.00	.08	-.02	.01	.08
MedSum10 on Education	-.02	.00	.000	-.04	.01	.000
Serial12 on Education	.08	.01	.000	.13	.01	.000
Serial10 on Education	.08	.01	.000	.15	.01	.000
MedSum08 on Education	-.05	.01	.000	-.12	.02	.000
Serial08 on Education	.20	.01	.000	.36	.02	.000
I of Serial08	1.12	.12	.000	.70	.08	.000
I of Serial10	.45	.10	.000	.28	.07	.000
I of Serial12	.17	.11	.13	.10	.07	.13
I of MedSum08	2.08	.09	.000	1.81	.08	.000
I of MedSum10	.57	.05	.000	.49	.05	.000
I of MedSum12	.33	.05	.000	.28	.04	.000
Serial08 Residual Var.	2.24	.05	.000	.87	.01	.000
Serial10 Residual Var.	1.43	.04	.000	.56	.01	.000
Serial12 Residual Var.	1.54	.04	.000	.55	.01	.000
MedSum08 Residual Var.	1.30	.03	.000	.99	.00	.000
MedSum10 Residual Var.	.37	.01	.000	.27	.01	.000
MedSum12 Residual Var.	.33	.01	.000	.24	.01	.000
R^2 Serial08				.13	.01	.000
R^2 Serial10				.45	.01	.000
R^2 Serial12				.45	.01	.000
R^2 MedSum08				.02	.00	.000
R^2 MedSum10				.73	.01	.000
R^2 MedSum12				.76	.01	.000

Note: I = intercept; MedSum08 = summed medical conditions in 2008; MedSum10 = summed medical conditions in 2010; MedSum12 = summed medical conditions in 2012; Serial08 = Serial 7s in 2008; Serial10 = Serial 7s in 2010; Serial12 = Serial 7s in 2012.

Table 40

Cross Lagged Panel for Serial 7s and Health Over Time with Education and Age

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
MedSum12 on MedSum10	.87	.01	.000	.87	.004	.000
MedSum10 on MedSum08	.86	.01	.000	.85	.01	.000
Serial12 on Serial10	.64	.01	.000	.61	.01	.000
Serial10 on Serial08	.60	.01	.000	.60	.01	.000
MedSum12 on Serial10	.01	.01	.47	.01	.01	.47
MedSum10 on Serial08	-.01	.01	.22	-.01	.01	.22
Serial12 on MedSum10	-.02	.02	.28	-.01	.01	.28
Serial10 on MedSum08	-.05	.02	.004	-.04	.01	.004
MedSum12 on Education	-.01	.00	.08	-.02	.01	.08
MedSum10 on Education	-.02	.00	.000	-.04	.01	.000
Serial12 on Education	.08	.01	.000	.13	.01	.000
Serial10 on Education	.08	.01	.000	.14	.01	.000
MedSum08 on Education	-.05	.01	.000	-.12	.02	.000
Serial08 on Education	.20	.01	.000	.36	.02	.000
MedSum12 on Age12	.00	.00	.72	-.00	.01	.72
MedSum10 on Age10	.00	.00	.46	.01	.01	.46
Serial12 on Age12	-.01	.00	.08	-.02	.01	.08
Serial10 on Age10	-.01	.00	.004	-.04	.01	.004
MedSum08 on Age08	.004	.00	.19	.02	.02	.19
Serial08 on Age08	-.01	.00	.17	-.02	.02	.17
I of Serial08	1.50	.30	.000	.93	.19	.000
I of Serial10	1.09	.24	.000	.68	.15	.000
I of Serial12	.59	.26	.03	.35	.16	.03
I of MedSum08	1.81	.23	.000	1.57	.20	.000
I of MedSum10	.49	.12	.000	.42	.11	.000
I of MedSum12	.37	.12	.002	.32	.10	.002
Serial08 Residual Var.	2.24	.05	.000	.87	.01	.000
Serial10 Residual Var.	1.42	.04	.000	.55	.01	.000
Serial12 Residual Var.	1.54	.04	.000	.55	.01	.000
MedSum08 Residual Var.	1.30	.03	.000	.99	.00	.000
MedSum10 Residual Var.	.37	.01	.000	.27	.01	.000
MedSum12 Residual Var.	.33	.01	.000	.24	.01	.000

R^2 Serial08	.13	.01	.000
R^2 Serial10	.45	.01	.000
R^2 Serial12	.45	.01	.000
R^2 MedSum08	.02	.00	.000
R^2 MedSum10	.73	.01	.000
R^2 MedSum12	.76	.01	.000

Note: I = intercept; MedSum08 = summed medical conditions in 2008; MedSum10 = summed medical conditions in 2010; MedSum12 = summed medical conditions in 2012; Serial08 = Serial 7s in 2008; Serial10 = Serial 7s in 2010; Serial12 = Serial 7s in 2012; Age08 = age in 2008; Age10 = age in 2010; Age12 = age in 2012.

Table 41

Cross Lagged Panel for Naming and Health Over Time

	Unstandardized coefficient	S.E.	*p*-value	Standardized coefficient	S.E.	*p*-value
MedSum12 on MedSum10	.91	.01	.000	.89	.00	.000
MedSum10 on MedSum08	.87	.01	.000	.87	.00	.000
Naming12 on Naming10	1.02	.05	.000	.74	.02	.000
Naming10 on Naming08	1.21	.07	.000	.42	.02	.000
MedSum12 on Naming10	.00	.01	.78	.00	.01	.78
MedSum10 on Naming08	-.07	.03	.02	-.02	.01	.02
Naming12 on MedSum10	-.05	.03	.08	-.04	.02	.08
Naming10 on MedSum08	-.08	.02	.000	-.08	.02	.000
Mean of Naming08	.83	.01	.000	2.18	.05	.000
Threshold for Naming10	.39	.04	.000	.58	.06	.000
Threshold for Naming 12	.21	.03	.000	.12	.06	.04
Mean for MedSum08	1.47	.02	.000	1.31	.03	.000
I of MedSum10	.39	.04	.000	.35	.03	.000
I of MedSum12	.21	.03	.000	.18	.03	.000
Naming08 Variance	.14	.01	.000	1.00	.00	--
MedSum08 Variance	1.25	.04	.000	1.00	.00	--
MedSum10 Residual Var.	.31	.01	.000	.25	.01	.000
MedSum12 Residual Var.	.27	.01	.000	.21	.01	.000
R^2 Naming10				.18	.02	.000
R^2 Naming12				.56	.03	.000
R^2 MedSum10				.75	.01	.000
R^2 MedSum12				.79	.01	.000

Note: I = intercept; MedSum08 = summed medical conditions in 2008; MedSum10 = summed medical conditions in 2010; MedSum12 = summed medical conditions in 2012.

Table 42

*Cross Lagged Panel for Naming and Health Over Time with Education**

	Unstandardized coefficient	S.E.	*p*-value
MedSum12 on MedSum10	.91	.01	.000
MedSum10 on MedSum08	.87	.01	.000
Naming12 on Naming10	.90	.05	.000
Naming10 on Naming08	1.04	.07	.000
MedSum12 on Naming10	.01	.01	.50
MedSum10 on Naming08	-.04	.03	.15
Naming12 on MedSum10	-.04	.03	.15
Naming10 on MedSum08	-.07	.02	.003
MedSum12 on Education	-.01	.00	.24
MedSum10 on Education	-.01	.01	.01
Naming12 on Education	.06	.01	.000
Naming10 on Education	.11	.01	.000
MedSum08 on Education	-.03	.01	.000
Naming08 on Education	.04	.00	.000
I of Naming08	.39	.03	.000
Threshold for Naming10	1.86	.14	.000
Threshold for Naming12	1.94	.19	.000
I of MedSum08	1.88	.10	.000
I of MedSum10	.52	.07	.000
I of MedSum12	.26	.06	.000
Naming08 Residual Var.	.39	.03	.000
MedSum08 Residual Var.	1.88	.10	.000
MedSum10 Residual Var.	.52	.07	.000
MedSum12 Residual Var.	.26	.06	.000
R^2 Naming 08	.07		
R^2 Naming 10	.24		
R^2 Naming 12	.55		
R^2 MedSum08	.01		
R^2 MedSum10	.75		
R^2 MedSum12	.79		

* Note: The model was conducted with weighted least squares with mean and variance adjustment (WLSMV) estimation; as such, standard errors and *p*-values are not provided for standardized values when the model has covariates and only the unstandardized results are

presented here. I = intercept; MedSum08 = summed medical conditions in 2008; MedSum10 = summed medical conditions in 2010; MedSum12 = summed medical conditions in 2012.

Table 43

*Cross Lagged Panel for Naming and Health Over Time with Education and Age**

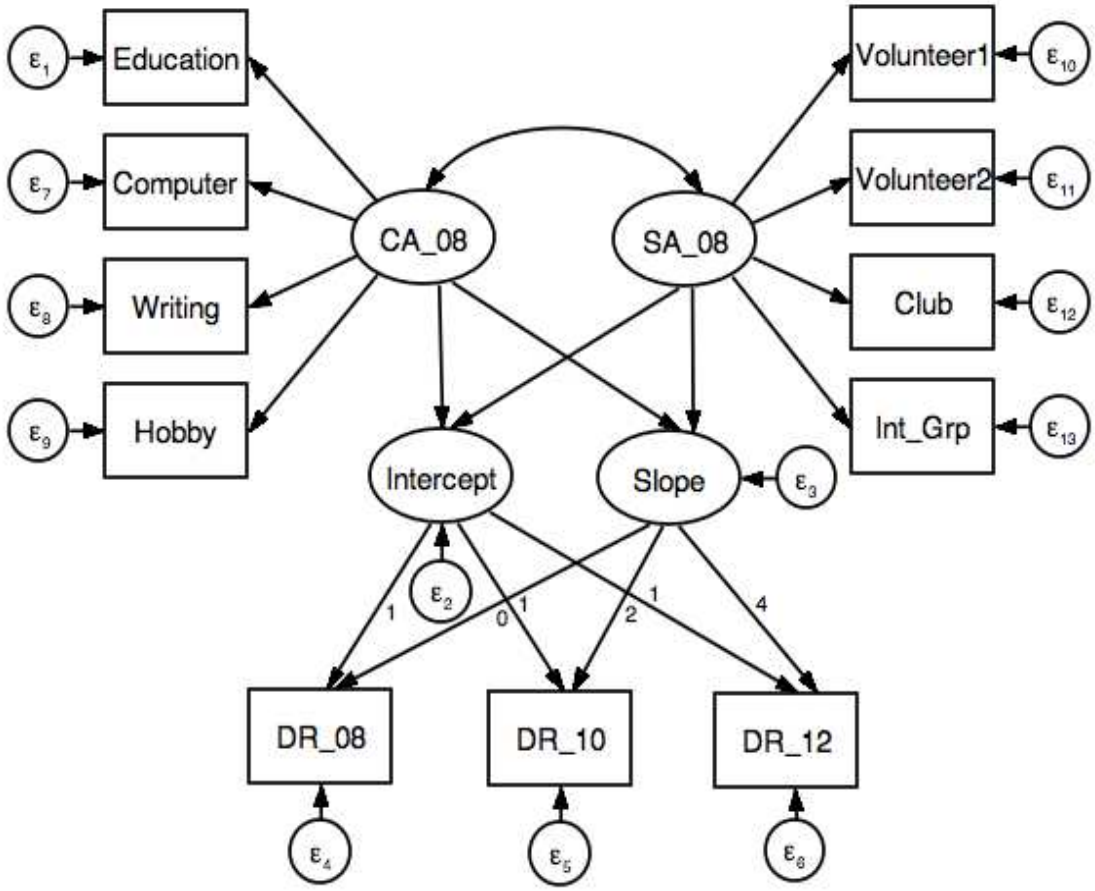
	Unstandardized coefficient	S.E.	*p*-value
MedSum12 on MedSum10	.91	.01	.000
MedSum10 on MedSum08	.87	.01	.000
Naming12 on Naming10	.89	.05	.000
Naming10 on Naming08	1.05	.07	.000
MedSum12 on Naming10	.01	.01	.59
MedSum10 on Naming08	-.04	.03	.17
Naming12 on MedSum10	-.04	.03	.15
Naming10 on MedSum08	-.06	.02	.004
MedSum12 on Education	-.01	.00	.26
MedSum10 on Education	-.01	.01	.01
Naming12 on Education	.06	.01	.000
Naming10 on Education	.11	.01	.000
MedSum08 on Education	-.03	.01	.000
Naming08 on Education	.04	.00	.000
MedSum12 on Age12	-.02	.06	.69
MedSum10 on Age10	.06	.05	.17
Naming12 on Age12	.10	.09	.26
Naming10 on Age10	-.12	.06	.03
MedSum08 on Age08	-.01	.06	.91
Naming08 on Age08	.00	.02	.84
I of Naming08	.36	.11	.001
Threshold for Naming10	.40	.43	.36
Threshold for Naming12	-.65	.55	.24
I of MedSum08	1.49	.36	.000
I of MedSum10	.47	.21	.03
I of MedSum12	.36	.20	.08
Naming08 Residual Var.	.13	.01	.000
MedSum08 Residual Var.	1.23	.04	.000
MedSum10 Residual Var.	.31	.01	.000
MedSum12 Residual Var.	.27	.01	.000
R^2 Naming08	.07		
R^2 Naming10	.45		
R^2 Naming12	.55		
R^2 MedSum08	.01		

R^2 MedSum10	.78
R^2 MedSum12	.80

* Note: The model was conducted with weighted least squares with mean and variance adjustment (WLSMV) estimation; as such, standard errors and *p*-values are not provided for standardized values when the model has covariates and only the unstandardized results are presented here. I = intercept; MedSum08 = summed medical conditions in 2008; MedSum10 = summed medical conditions in 2010; MedSum12 = summed medical conditions in 2012; Age08 = age in 2008; Age10 = age in 2010; Age12 = age in 2012.

Figure 1

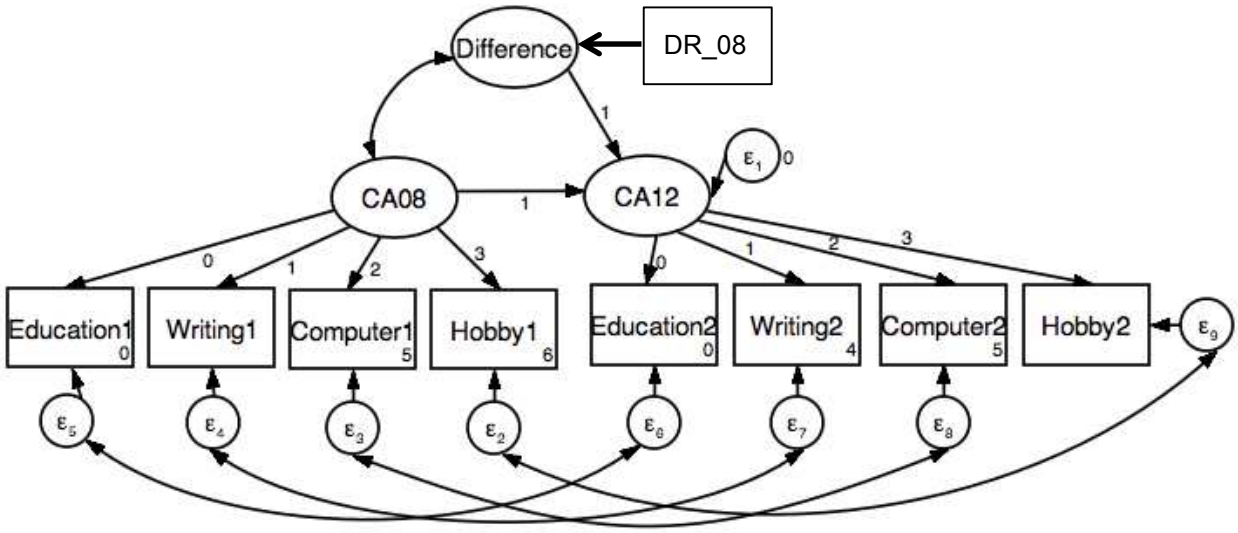
Proposed Latent Growth Curve Model with Activity Frequency Predicting Delayed Recall Over Time



Note: DR = delayed recall by year; CA08 = latent factor for cognitive activities in 2008; SA08 = latent factor for social activities in 2008. All intercept factor loadings set to 1; slope factor loadings set to 0, 2, and 4.

Figure 2

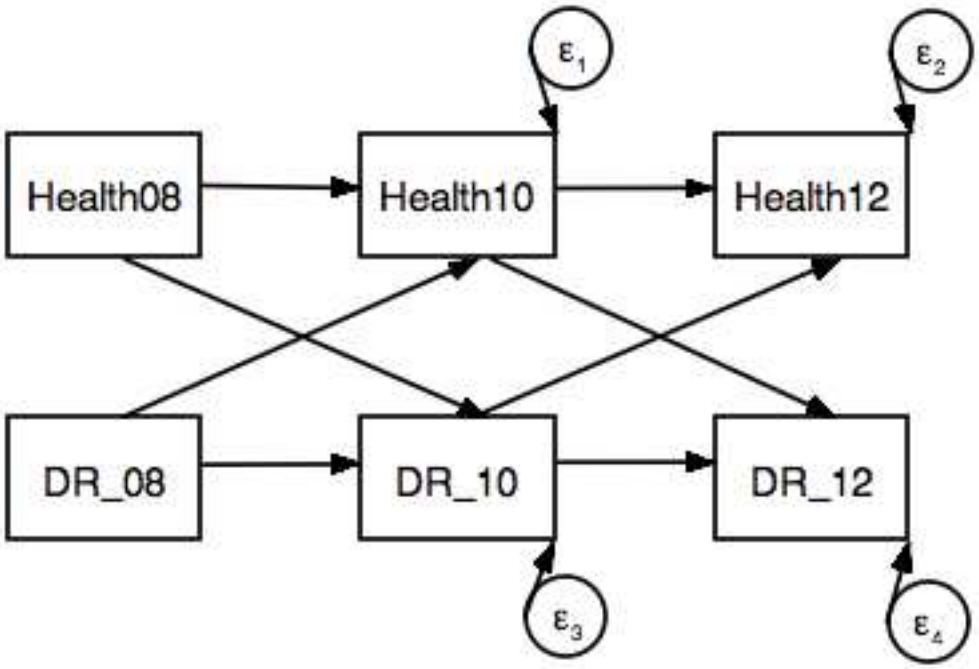
Proposed Latent Difference Model: Baseline Delayed Recall Predicting Change in Activity Frequency



Note: DR_08 = delayed recall in 2008; CA08 = latent factor for cognitive activities in 2008; CA12 = latent factor for cognitive activities in 2012; Difference = latent factor for the difference between CA12 and CA08. Final factor loadings reflect partial measurement invariance. Latent difference model adapted from Geiser (2013).

Figure 3

Sample Cross-Lagged Panel for Health Conditions and Delayed Recall



Note: Correlations between indicators of each time point are not depicted here. Health = observed indicator, reflecting sum of physical health conditions by year; DR = observed indicator for delayed recall by year.

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ABSTRACT

ENGAGEMENT IN ACTIVITIES AND COGNITIVE FUNCTIONING AMONG OLDER ADULTS IN THE HEALTH AND RETIREMENT STUDY

by

PAMELA E. MAY

August 2015

Advisor: Dr. John L. Woodard

Major: Psychology (Clinical)

Degree: Doctor of Philosophy

The goal of this dissertation is to examine the effect of cognitive and social activities on cognitive performance and health conditions in a national sample of older adults from the Health and Retirement Study (HRS). This dissertation first aimed to identify longitudinal relations between activity frequency and cognitive functioning. Two hypotheses were tested, baseline activity frequency predicts change in cognitive functioning over time, and baseline cognitive performance predicts change in activity frequency over time. The dissertation's second aim was to identify links between activity frequency and cognitive trajectories. The third aim was to identify longitudinal relations between activity frequency and overall health. Changes in cognitive functioning and health were also compared simultaneously over three time points, to identify causal relations. The sample included 3,397 respondents aged ≥ 60 years old from the Health and Retirement Study's 2008, 2010, and 2012 waves. Respondents completed brief cognitive tests and items regarding their health during each wave, as well as items ranking frequency of engagement in cognitive and social activities in 2008 and 2012. A series of structural equation models were implemented to test the aforementioned aims. A paucity of significant findings precluded a comparison between the two hypothesized models on activity

frequency and cognitive functioning, as well as between the two contrasting models for activity frequency and overall health. Activity frequency did not significantly predict rate of change in cognitive performance or health conditions over time. Activity frequency also did not significantly change over time. However, frequency of baseline cognitive activity was associated with initial level of episodic memory. Further, a lower frequency of cognitive activities was associated with a higher number of health conditions at baseline, when education was not included in the model. Relations between health and cognition were not consistently indicated over time, suggesting that health conditions may not have strong causal effects on age-related changes in cognitive functioning. Significant associations between baseline activity engagement and initial level of episodic memory and health conditions does not allow one to rule out the protective effect of activity engagement on cognition and overall health.

AUTOBIOGRAPHICAL STATEMENT

Pamela E. May is from Massapequa, New York, and received her Bachelor of Arts at State University of New York at Geneseo College. During her undergraduate career, she became involved in several areas of research, including sexual victimization in women, sleep, and cognitive and daily functioning of older adults with macular degeneration. After four-years in the upstate New York area, she moved to Detroit, Michigan, to pursue a Ph.D. in Psychology at Wayne State University with an emphasis in clinical neuropsychology. She was a pre-doctoral trainee at the Institute of Gerontology, studying cognitive impairment in older adults, for most of her graduate career, under the supervision of John L. Woodard, Ph.D. Additionally, she completed neuropsychology practica at the Detroit Medical Center and John D. Dingell VA Medical Center, was a teaching assistant for multiple courses, and worked as a statistician assistant in a corporate setting focused on health psychology. She currently is a clinical psychology intern at the John D. Dingell VA Medical Center, and will be beginning a postdoctoral fellowship in clinical neuropsychology at Nebraska Medicine in Omaha, Nebraska in late 2015.