COGNITIVE ACTIVITY AND INTELLIGENCE:

IMPLICATIONS FOR THE COGNITIVE RESERVE MODEL

A thesis presented to

the faculty of

the College of Arts and Sciences of Ohio University

In partial fulfillment

of the requirements for the degree

Master of Science

Erin M. Mark

March 2007

This thesis entitled

COGNITIVE ACTIVITY AND INTELLIGENCE:

IMPLICATIONS FOR THE COGNITIVE RESERVE MODEL

by

ERIN M. MARK

has been approved for

the Department of Psychology

and the College of Arts and Sciences by

Julie A. Suhr

Associate Professor of Psychology

Benjamin M. Ogles

Dean, College of Arts and Sciences

Abstract

MARK, ERIN M., M.S., March 2007, Department of Psychology

COGNITIVE ACTIVITY AND INTELLIGENCE: IMPLICATIONS FOR THE COGNITIVE RESERVE MODEL (106 pp.)

Director of Thesis: Julie A. Suhr

Evidence suggests cognitive activity (CA) in older adulthood slows or prevents cognitive decline and neurodegenerative disease, after controlling for the effects of IQ and education. Despite media messages and public health campaigns endorsing CA as a main prevention strategy, there are limitations to studies using self-report, composite measures to estimate CA. In the present cross-sectional study we examined the association of education, self-reported CA, and estimated IQ (Vocabulary) with neuropsychological performance in a sample of 66 community-dwelling adults.

As predicted, IQ and education were associated with memory, global cognitive function, and letter fluency after controlling for CA. Contrary to predictions, self-reported CA *was not* related to neuropsychological performance. This study supports the use of IQ to predict neuropsychological performance, but fails to find support for the relation between self-reported CA and performance. Exploratory analyses suggested that education and CA interact to moderate cognitive performance, especially in adults with low education.

Approved: _____

Julie A. Suhr

Associate Professor of Psychology

Table of Contents

Page

List of Tables

Table Page

List of Figures

Figure Page

Clinicians have long observed differences in the way patients respond to central nervous system damage and progressive brain disease. For example, two strokes of a similar magnitude occurring in a similar area of the brain may produce severe dysfunction in one patient, while resulting in minimal dysfunction in another patient (Stern, 2002). Similarly, when assessing individuals with dementia, clinicians note there is often a discrepancy between actual disease pathology (e.g., quantified by cerebral blood flow) and expression of cognitive and behavioral deficits (e.g., poor performance on a delayed recall task). The theory most widely used to conceptualize the capacity of the brain to absorb disease pathology, as well as to help explain the basis for individual differences, is the *reserve theory*. Reserve theory posits that individuals possess a capacity to withstand or absorb a certain amount of neural insult (i.e. a reserve capacity) (Satz, 1993; Stern et al., 1994). This capacity is unique to each individual and is likely a product of the interaction of several factors. One of the most exciting areas of the reserve theory debate seeks to answer the question: What accounts for the phenomenon of individual differences shown in response to brain damage? Are individual differences due to genetic endowment and early development (i.e. general intellect) or instead, is reserve accumulated and maintained through a lifetime of mental stimulation (i.e. cognitive activity)?

The present study examined the association between general intellect factors (i.e. education and IQ score), lifestyle factors (i.e. remaining cognitively active in old age), and cognitive function within the construct of reserve. Specifically, the goals of the present study were: 1) To examine the relation between factors commonly used as

markers for cognitive reserve (i.e., cognitive activity, level of education, and general intellect) in an effort to ascertain the factor or factors that are most related to neuropsychological performance; and 2) To address methodological weaknesses in the cognitive activity and cognitive reserve literature. To this end, the broader construct of reserve will be presented first, followed by: A review of the Alzheimer's disease (AD) model; a presentation of known risk factors for AD; the genetic versus the environmental contributions to general intellect; and the evidence regarding the influence of general intellect and lifestyle factors on the formation of reserve. Within the review of the cognitive activity literature, the weaknesses of this research will be highlighted and methodological limitations will be addressed.

Cognitive Reserve Theory

The concept of reserve was first advanced by Roth, Tomlinson, & Blessed (1967) and Blessed, Tomlinson, & Roth (1968). They observed that the "senile plaques" and "neurofibrillary tangles" previously described by Alois Alzheimer were present in normal (i.e. non-demented) adults that had not exhibited cognitive deficits before death. When the plaques and tangles were present in large enough numbers, the deceased was nearly always demented. Blessed et al. described this phenomenon as a "threshold effect" that mediated the development of dementia. Roth et al. made the observation that "a certain amount of damage estimated by plaque counts may be accommodated within the reserve capacity of the cerebrum without causing manifest intellectual change" (1967, p.258). Thus, the concept of reserve against brain damage originated from the observation that there is *not* a direct relation between brain injury and subsequent manifestation of the

clinical symptoms of pathology (Blessed et al., 1968; Roth et al., 1967; Katzman et al., 1989; Stern, 2002). Instead, a disease or damage threshold that is unique to each individual appears to mediate the advent of symptom manifestation.

Consider the following analogy. A balloon slowly fills with liquid. The capacity of the balloon to hold a given quantity of liquid depends on several factors (e.g., the strength of the balloon, the elasticity of the balloon, the weight of the liquid, etc.). There is a point after which the balloon's ability to stretch and receive liquid is exhausted—a breaking threshold. When this breaking threshold is met, additional liquid causes the balloon to break, exposing all of the liquid which was previously tolerated. In this analogy, the balloon is an individual's brain and the liquid is disease or damage. The theoretical construct that describes how a threshold is set is cognitive reserve. The question of interest to the present study was: Which factors contribute to the formation of an individual's breaking threshold and what are the relative contribution of the various factors?

Within the last decade, the main proponent of the cognitive reserve theory has been a research group led by the neuropsychologist, Yaakov Stern. In his 2002 review of the cognitive reserve construct, Stern divides reserve theory into two groups: *passive reserve* and *active reserve*. Passive reserve describes the amount of damage the brain can sustain before reaching a "threshold for clinical expression" (Stern, 2002, p.448). Several passive reserve models have been articulated such as the *brain reserve model* (Katzman, 1993), the *neuronal reserve model* (Mortimer, Schuman, & French, 1981), and the *threshold model* (Blessed et al., 1968; Roth et al., 1967; Satz, 1993) of reserve. The

threshold model is arguably the most understood model of passive reserve and was critically reviewed by Satz (1993). In the threshold model, *brain reserve capacity*, which may be quantified as synaptic density or intracranial volume, is the central construct. According to the model, once brain reserve capacity is exhausted past a certain threshold (e.g., due to disease pathology), clinical impairment becomes evident. Although Stern acknowledges the relevance of passive models of reserve, he does not believe that passive models account for the individual differences in how the brain continues to function after damage (Stern, 2002).

Alternatively, *cognitive reserve* is an active model of reserve. Active reserve processes reflect the ability of the brain to "actively compensate for brain damage" (Stern, 2002, p. 449). Unlike earlier passive models, the cognitive reserve model rejects the idea of a universal "threshold" that acts as a cut-off for impairment across individuals. Cognitive reserve assumes that individual brains respond differently to damage, and thus acknowledges neuroanatomical variability (Stern, 2002). According to Stern, cognitive reserve is the "ability to optimize or maximize performance through differential recruitment of brain networks, which perhaps reflects the use of alternate cognitive strategies" (Stern, 2002, p. 449).

According to Stern (2002), when an individual uses a brain network efficiently, activates alternate brain networks, or employs alternate cognitive strategies, they are demonstrating cognitive reserve (p. 449). Functional imaging studies of neural processing efficiency show that when presented with increasingly difficult tasks, normal individuals typically respond with increased activation of neural networks (Grady et al.,

1996; Stern et al., 2003). Cognitive reserve is believed to explain discrepancies between disease pathology and clinical symptom manifestation for most forms of neurodegenerative disease, dementia, and traumatic brain injury. For example, individuals with more advanced Alzheimer's disease pathology *and* high levels of cognitive reserve (using level of education as a proxy for cognitive reserve) express similar symptoms and perform equally well on cognitive measures (i.e. neuropsychological tests) as individuals with less advanced Alzheimer's disease pathology (e.g., measured by cerebral blood flow) *and* low levels of cognitive reserve (Stern, 2002). Furthermore, individuals with high levels of cognitive reserve will worsen more quickly than individuals with lower cognitive reserve. This second observation, though counterintuitive, reflects cognitive reserve at work. In other words, higher levels of cognitive reserve allow individuals to withstand a great amount of damage until a threshold, that is unique to the individual, is met. Subsequent damage beyond this threshold causes a rapid decline in cognitive function. Cognitive reserve helps explain why it is more difficult to detect Alzheimer's disease in highly educated and highly intelligent individuals (Scarmeas et al., 2003; Stern, 2002).

The Alzheimer's Disease (AD) Model

The disease model most often discussed in the context of cognitive reserve theory is dementia and more specifically, Alzheimer's disease (AD). According to Stern (2002, p. 448), Alzheimer's disease is an often-used disease model for three main reasons. First, AD induces deficits within numerous cortical circuits that underlie an array of cognitive functions, allowing discussion of global features of reserve versus more focal abilities

(e.g., short term memory recall, p. 448). Second, AD typically affects similar neuroanatomical areas across individuals, allowing generalization. Third, the progressive course of AD provides a gauge of the severity of pathology required before associated neural networks are disrupted (Stern, 2002, p. 448). In addition to the three advantages of studying AD in the context of cognitive reserve, AD is presently a leading public health concern in the United States.

AD is the most common form of dementia, accounting for 50% of dementia cases in adults over age 65 (Cummings & Cole, 2002). In 2000, there were approximately 4.5 million Americans with AD (Hebert, Scherr, Bienias, Bennett, & Evans, 2003). Although the statistics vary depending on sampling methods and inclusion criteria, 4% - 10% of adults over 65 years old, and nearly 50% of adults over 85 years old are affected with AD (American Psychiatric Association [APA], 2000; Cummings & Cole, 2002; Evans et al., 1989). The prevalence rates are alarming and will grow in the coming decades as the baby-boomer generation ages. Brookmeyer, Gray, and Kawas (1998) estimated that if the current aging trend continues, there could be at least 360,000 new AD cases per year. By 2050, the prevalence of AD in the U. S. is expected to quadruple, resulting in more than 13 million people with the disease (Hebert et al., 2003).

Much of the recent AD research is focused on prevention and early detection. Research on early detection, however, has yet to yield a method for detecting significant cognitive decline among highly intelligent individuals. According to Rentz et al. (2004), the lack of sensitivity may, in part, be due to the inadequate norms against which neuropsychological performance is compared. Standardized scores based on age or

education may not be the best method to estimate premorbid ability. For example, education is viewed as an adequate marker for cognitive reserve and is widely used as an estimate of general intellect, despite conflicting data (Rentz et al., 2004). This is one of the areas addressed in the present study. Regarding the prevention of AD, promising research has emerged that may provide strategies for reducing one's risk for AD.

Individuals with AD have deficits in multiple cognitive domains. Furthermore, the presence of cognitive decline in some cognitive domains may serve as an early indicator of AD (APA, 2000). For example, some prospective studies (Fabrigoule et al. 1998; Jacobs et al. 1995) have shown that individuals experience subclinical declines in cognitive function in the years preceding AD diagnosis (diagnosis of AD includes clinical manifestation of characteristic symptoms according to DSM-IV and NINCDS-ADRDA criteria). Although decline in recent memory is the most common deficit detected in subclinical individuals, slight declines in multiple areas may be observed. Memory impairment, which is a required symptom for diagnosis according to DSM-IV and National Institute of Neurological and Communicative Disorders and Stroke/Alzheimer's Disease and Related Disorders Association (NINCDS-ADRDA) criteria, actually represents deficits in both learning and encoding new information. Learning and encoding deficits, manifest as declines in recent memory, may be measured by tests such as list-learning and other immediate and delayed recall tasks (Knopman & Selnes, 2003).

A deficit in language function is another major symptom of AD (APA, 2000). Most language disturbances are subtle and are detected even in mild stages of the disease. Some of the more common language disturbances include dysnomia or word finding

difficulty and decreased verbal fluency. Verbal fluency is thought to be an executive function task. Dysnomia and verbal fluency may be tested with tasks requiring an individual to quickly generate words relating to a specified condition (e.g., words that begin with the letter "b") and are referred to as verbal fluency tasks (Knopman & Selnes, 2003).

Visuospatial function is another area often affected by AD. Although deficits in visual perception are not typically evidenced during early stages of the disease, perception of spatial relationships (e.g., angles) is commonly affected early in the course of the disease. For example, performance on a measure such as a line orientation task can be negatively affected early in the disease process. Performance on tests of more complex visual construction abilities (e.g., Rey Complex Figure) also tend to be affected in early AD (Knopman & Selnes, 2003).

Executive function deficits are also detectable in mild AD. Indeed, even in *mildly* demented individuals, abstract reasoning and judgment are often impaired. Specifically, working memory, mental dexterity, and set-shifting are areas in which impairment is commonly observed. Neuropsychological tests such as verbal fluency tasks (e.g., COWA) and trail-making tasks (Trail Making Test – Part B) are good measures of executive function and are sensitive to mild AD-induced impairment (Knopman & Selnes, 2003).

Numerous risk factors for AD have been identified within the last two decades including: Increased age (Cummings, Vinters, Cole, & Khachaturian, 1998, Lindsay, et al., 2002), lower level of education (Lindsay et al., 2002; Mortimer, Snowdon, &

Markesbery, 2003; Stern et al., 1994), infrequent participation in cognitively stimulating activities (Wilson, Bennet, et al., 2002; Wilson, Mendez de Leon, et al., 2002), genetic mutation (Cummings, Vinters, Cole, & Khachaturian, 1998; Podewils, 2005), limited social engagement (Fratiglioni, Paillard-Borg, & Winblad, 2004; Zunzunegui et al., 2003), and low levels of physical activity (Podewils, et al., 2005). Of the six risk factors identified above, level of education and cognitive activity are the two most often considered in the larger construct of cognitive reserve. As mentioned above, education (instead of an IQ measure) is often used as an estimate of general intellect. The most likely reason that education is often used as an estimate of intellect in research is convenience. It has been shown that education and IQ are highly correlated. For example, according to a review conducted by Neisser et al. (1996), the correlation between IQ scores and education was approximately 0.55. As will be discussed below, the use of education as a stand-in for intelligence is a weakness of the cognitive reserve literature generally and the cognitive activity literature, specifically.

General Intellect: Genetic versus Environmental Influence

As noted in the opening paragraph of this manuscript, one way to conceptualize cognitive reserve is as the protective effect of overall brain fitness, or *g* (i.e. general intellect). One method of estimating *g* is to use a standardized IQ measure. One such measure is the Verbal Comprehension Index (VCI) on WAIS-III (Wechsler, 1997). Of the four WAIS-III indices, the Verbal Comprehension Index (VCI) tends to hold the best measure of *g.* The VCI is also a good measure of crystallized intelligence. In the Cattell-Horn model of intelligence, crystallized intelligence represents the fact-based and

declarative knowledge that is acquired throughout one's life and remains relatively stable despite increased age, disease, or injury (Horn & Cattell, 1967). The general stability of crystallized intelligence during old age stands in marked contrast to fluid intelligence (e.g., working memory, attention, and processing speed), which has been shown to decline with age and insult. Thus, measures of verbal IQ such as subtests within the Verbal Comprehension Index of the WAIS-III (i.e. crystallized intelligence), may offer a good estimate of general intellect (Neisser et al., 1996). In other words, because of the stability of crystallized intelligence across time, an older adult's performance on tests of verbal IQ may be viewed as a good estimate of their verbal abilities earlier in life. Longitudinal studies also support the lifelong stability of test scores that estimate intelligence, such as the WAIS-III Vocabulary subtest specifically, and the VCI, in general (Neisser et al., 1996; Sands, Terry, & Meredith, 1989; Schaie, 2002).

Further, although intelligence and performance on standardized intelligence tests are strongly related to multiple cognitive abilities and overall cognitive function, intelligence remains a theoretically unique construct. Table 1 presents the correlations of scores on the WAIS-III Verbal Comprehension Index with scores on common neuropsychological measures. Notice the higher correlations between VCI and 1) measures with a high verbal loading (e.g., verbal ability, verbal memory) and 2) composite measures (e.g., WAIS-III Full Scale, Stanford Binet Intelligence Scales Composite score), versus the lower correlations with nonverbal and performance measures (e.g. TMT, Complex Figure Test). As expected, naming and other language

Table 1

Correlations of WAIS-III Verbal Comprehension Index (VCI) with Neuropsychological Tests

Neuropsychological Test	Correlations* with VCI
WAIS-III	
VIQ	.79
PIQ	.60
Full Scale IQ	.76
WISC-IV	
Full Scale	.83
VCI	.87
Stanford Binet Scales-IV	
Verbal Reasoning	.87
Visual Reasoning	.57
Composite	.85
COWA (F, A, S)	.57
Category Naming (Animals)	.62
Boston Naming Task	.48
Wechsler Memory Scale	
Visual immediate memory	.29
Auditory immediate memory	.57
Trail-Making Test	
Part A	-.12
Part B	-.40
CVLT	
Trials 1 – 5	.38
Short delay	.58
Long delay	.58
Complex Figure Test	
Copy	.27
Delayed recall	.01

Note. From Wechsler (1997). * Significance for all correlations is $p < .05$. WAIS-III = Wechsler Adult Intelligence Scales; RBANS = Repeatable Battery for the Assessment of Neuropsychological Status; CVLT = California Verbal Learning Test; COWA = Controlled Oral Word Association with letters F, A, and S.

tasks, such as COWA and the Boston Naming Test, have moderate to strong associations with VCI. It is notable, however, that the correlations with language and naming tasks are significantly lower than the correlations with other *intellect* tests (e.g., $r \geq .8$). This pattern of association provides evidence for the concurrent and discriminant validity of

VCI as a verbal measure that is highly associated with general intellect and not as highly associated with other neuropsychological domain measures (Wechsler, 1997). Thus, there is good evidence that intelligence, while associated with specific cognitive abilities, is a distinct construct.

General intellect includes genetic contributions from both parents, as well as factors that influence early child development. Indeed, general intellect appears to have a strong familial component. Studies of monozygotic twins reared apart provide the truest estimate of *heritability* for a given trait because monozygotic twins are genetically identical. According to Neisser et al. (1996), the heritability (h^2) of a given trait represents the proportion of variation in that trait that is attributable to genetic contribution. The remaining variation ($1 - h^2$) is associated with environmental factors and error variance. Heritability estimates vary with age. The relationship between inherited and environmental influence is complex. For example, Neisser et al. (1996) discuss *vocabulary* as an ability that while "substantially" heritable, is also highly influenced by environment. In other words, while the size of one's vocabulary tends to be highly heritable, words must be *learned* through interaction with the environment. Further complicating the discussion of environmental versus genetic contribution is the idea that individuals actively shape their environment. This last point is germane to the cognitive activity debate. Namely, do brighter individuals seek out more complex and stimulating environments, and thus remain cognitively healthy, longer?

According to the review by Neisser et al. (1996), the heritability of IQ increases with age, while the variance attributable to *shared environment* (e.g. early home life)

decreases with age (McGue, Bouchard, , & Lykken, 1993). In childhood, h^2 for IQ is approximately .45 and by late adolescence h^2 is approximately .75 (Neisser et al., 1996). Large studies in the U.S. and Europe found that adult IQ scores for monozygotic twins raised apart are highly correlated, ranging from .68 and .78 (McGue et al., 1993; Lee, 2003).

Cognitive ability in general appears to be highly heritable. According to a literature review conducted by Lee (2003), most studies found heritability estimates of cognitive function ranging from 0.4 to 0.6., with some variability in the heritability estimates for various cognitive abilities. For example, McClearn et al. (1997) studied the heritability of different cognitive abilities in healthy older monozygotic (MZ; n = 110 pairs) and dizygotic (DZ; n = 130 pairs) same-sex twins raised apart (Median age= 82.3). McClearn et al. (1997) found the heritability estimate for general cognitive function was .53 using a short form of the Wechsler Adult Intelligence Scale. Heritability estimates for other cognitive domains are listed in descending order (with 95% CI): Processing speed =.62 (.29 - .73), verbal ability = .55 (.24 - .81), memory = .52 (.07 - .67), spatial ability = .32 (0 - .58).

A later study by Swan & Carmelli (2002) reported similarly high estimates for general cognitive function (but not for memory). This study attempted to tease apart the genetic contributions for various cognitive domains. Specifically, they adjusted for age and education and found that heritability explained 79% of executive function variability. Swan & Carmelli's findings (2002) suggest that that both cognitive ability and rate of decline of cognitive function are highly heritable. In other words, despite environmental

differences, one's cognitive or intellectual ability, as well as one's ability to stave off brain disease, are largely a product of what one is born with.

The above studies also demonstrated that approximately 40% of the variance in cognitive ability is due to environment. Interestingly, most of the environmental variance is attributable to *unshared* factors versus shared environmental factors (e.g., early home life) mentioned earlier. In other words, individual lifestyle differences (even among MZ twins) explain most of the non-genetic variance in cognitive ability. A logical next question is: Does an individual possess the ability to affect the "fitness" or reserve capacity of his or her brain through lifestyle (e.g., engaging in mentally stimulating activity)? This lifestyle hypothesis is often referred to with the popular expression "use-it-or-lose-it". For example, a recent study found that the more cognitively active an individual was, the less likely that individual was to develop Alzheimer's disease (Wilson & Bennett, 2003). As it is likely evident to the reader, it is difficult to distinguish the degree to which *g* is influenced by genetics versus environment. The same conundrum plagues the cognitive reserve debate. Namely, how much of reserve is the result of what one is born with versus the lifestyle one adopts?

The Influence of Education on Cognitive Reserve

Studies have repeatedly found education to be related to dementia risk (Le Carret et al., 2003; Lindsay et al., 2002; Mortimer, Snowdon, & Markesbery, 2003; Stern et al., 1994). Education has been found to have a protective effect against the development of dementia and cognitive decline, in general. Indeed, increased cognitive reserve is the most widely held explanation for the protective effects of education. Individuals with

more education are believed to possess greater amounts of reserve than individuals with less education, due to the observation that those with more education become demented significantly later in life. The mechanism by which education contributes to cognitive reserve, however, remains unclear.

One possible explanation is that education provides environmental enrichment and cognitive stimulation to the young and developing brain, resulting in better, more efficient brains. According to Katzman (1993), education may increase synaptic density and help establish elaboration of neural networks. Support for the beneficial effects of education come from human and animal studies.

In animal studies, environmental stimulation in young rats leads to anatomic improvements such as increased synaptic density, increased glial production, increased production of synaptic vesicles (e.g., Kempermann, Gast, & Gage, 2002), as well as improved performance on learning tasks (e.g., Jankowsky et al., 2005, Saari, Armstrong, Nobrega, Pappas, & Coscina, 1990). In short, animal studies have shown early environmental stimulation is beneficial to brains of young rats (for a review see van Praag, Kempermann, and Gage, 2000).

Human studies also support the beneficial effects of education on cognitive status. Staff et al. (2004) conducted a study of 92 individuals born in 1921 to investigate the influence of the three main hypothesized proxies of cognitive reserve: Education, head size (MRI measure of intracranial volume), and occupation. The participant's cognitive function had previously been assessed at age 11 with the Moray House Test. Staff et al. assessed cognitive function at age 79 using Ravens' Progressive Matrices (Raven, 1960)

and the Auditory Verbal Learning Test (Rey, 1958). The Raven's Progressive Matrices is a nonverbal reasoning measure while the Auditory Verbal Learning Test taps immediate and delayed verbal memory. Both memory and reasoning have been shown to decline with age (Salthouse, 2003) and show decline in early AD (Fabrigoule et al. 1998; Jacobs et al. 1995).

Staff et al. (2004) found that brain size (defined as intracranial volume) made no significant contribution to memory or reasoning ability at age 79. Education and occupation, however, were significantly related to cognitive function at age 79. Specifically, education accounted for 5% to 6% ($p < .05$) of the variance in memory, but was not significantly related to reasoning. They also reported (although they did not view it as a major finding) that childhood cognitive ability, as measured by Moray House Test scores, accounted for 6% of the variance in verbal memory at age 79 ($p = 0.02$) and 14% ($p < 0.001$) of the variance in nonverbal reasoning at age 79. From these results, Staff et al. (2004) concluded that education and occupational status contribute to cognitive reserve. Specifically, they concluded that "more education and higher occupational status predicted higher cognitive ability in old age than would be expected for a person's childhood ability and accumulated brain burden" (Staff et al., 2004, p. 1198).

However, another explanation of the findings is possible. An alternative explanation for the results is that brighter, smarter individuals (i.e. those who have more reserve from birth) achieve higher levels of education and are more resistant to decline than less bright individuals. According to this explanation, one's cognitive ability in old-

age is less a result of how active one has been in life and is more related to one's general intellect (i.e. what you are born with). Interestingly, Staff et al. did not conceptualize childhood ability as a potential proxy of cognitive reserve processes. Instead, they incidentally reported the significant correlation between childhood ability and old-age verbal memory and nonverbal reasoning.

The significant correlation between childhood ability and old-age ability found in Staff et al. (2004) speaks to one of the foremost issues within the cognitive reserve debate. Childhood ability is an indicator of general intellect which likely influences later achievement and performance on ability measures. Unfortunately, due to the statistical design of the Staff et al. study (highlighted here as a limitation) partial correlations (i.e. measures of unique variance accounted for) for childhood ability in the presence of education and occupation (and vice versa), were not examined. Therefore we are unable to draw any conclusions about the unique contribution of childhood ability to later cognitive function. The first order correlations between childhood ability, education, and adult occupation were also not reported, representing an additional limitation of the Staff et al. study. Another limitation of the Staff et al. (2004) study relates to sample bias. As the authors acknowledged, survival bias is one of the most salient issues related to sample characteristics in this study (and any other studies of older adults). In other words, the cognitive variability of the sample was restricted due to the fact that only healthy survivors were included. Arguably, individuals who were excluded due to poor health or mortality had likely aged less "successfully" than the study participants.

In a prospective population-based study, Richards & Sacker (2003) employed path analysis to model lifetime trajectories and antecedents of cognitive reserve (n > 4000) using a 1946 British birth cohort. They analyzed parental occupation, cognitive ability at age eight, educational attainment, occupation at age 53, National Adult Reading Test (NART; Nelson, 1982) score at current age, verbal memory, and timed visual search. The latter three variables were the cognitive outcome variables in the study. Verbal and nonverbal cognitive ability at age 8 was measured by tests devised by the National Foundation for Educational Research (Pigeon, 1964). These tests included a reading comprehension task, a word reading and pronunciation task, a vocabulary task, and a picture intelligence (i.e. nonverbal reasoning) task.

The NART, which served as an outcome measure in this study, is a pronunciation test that taps accumulated verbal knowledge and is mostly resistant to age-related decline. The NART is often used as a brief measure of general intellect and is predictive of full scale IQ (Nelson & Willison, 1991; Richards & Sacker, 2003). Educational attainment was classified using the Burnham Scale (Department of Education & Science, 1972). Using this classification system, an individual's highest education or "training" achieved by age 26 was designated as belonging to one of the following groups: No qualification, vocational, ordinary secondary qualifications, advanced secondary qualifications, or higher qualifications (i.e. degree or equivalent; Richards and Sacker, 2003, p. 616). The range of educational attainment in the Richards and Sacker (2003) sample ranged from individuals with no qualifications (39.8%) to individuals with higher qualifications (9.3%).

An analysis of path trajectories demonstrated significant, independent (standardized regression weights) paths from childhood cognition (.50), level of education (.22), and occupation (.13) to adult NART scores (Richards & Sacker, 2003). The strongest association was from childhood cognition. Similar, though somewhat weaker paths were found from childhood cognition to verbal memory and psychomotor processing. In general, the literature has previously shown a moderate association between education and cognitive function. The finding of the Richards & Sacker study suggest that childhood verbal and non-verbal ability is more predictive of later ability than is level of education or occupation. One interpretation of the Richards & Sacker finding (i.e. childhood cognition is the most predictive variable of adult cognition) is that it lends support to the argument that general intellect is the major factor in the generation of cognitive reserve.

Cognitive Activity: Influence on Cognitive Reserve

In addition to education, an individual's lifestyle may convey some protective affects against brain disease. For example, there is evidence suggesting that participation in cognitively stimulating occupational and leisure activities during one's "post-educational years" may further build-up cognitive reserve and enhance the effect of education (Capurso et al., 2000; Cockburn, Smith, & Wade, 1990; Fratiglioni, Paillard-Borg, & Winblad, 2004; Le Carret et al., 2003). One particularly intriguing area of cognitive reserve research is this relationship between lifestyle and cognitive decline (e.g., development of AD). This argument, as noted before, is popularly termed "use-it-or-lose-it". The basic premise of the "use-it-or-lose-it" argument is this: The more an

individual uses his brain, the less likely it is he will develop a degenerative brain disease, such as AD (Cassel, 2002; Wilson & Bennett, 2003). Support for this argument comes primarily from a research group led by Robert Wilson, at the Rush Alzheimer's Disease Center in Chicago. Wilson and his colleagues assert that one of the primary mechanisms for building up cognitive reserve is through a lifetime of cognitively stimulating activities (Wilson, Barnes, & Bennett, 2003). In a related finding, Capurso et al. (2000; as cited in Le Carret et al. 2003, p. 319) found that a low-complexity occupation is a risk factor for age-related cognitive decline, thus lending support for the lifestyle position.

There is evidence suggesting that remaining cognitively active and continuing to learn across the lifespan makes an important contribution to brain health and may serve to slow and even prevent neurodegenerative disorders in late life (Stern et al., 1994). Recent animal studies suggest that even in mid-life, environmental enrichment can be beneficial to *adult* rat and mice brains. Neurogenesis, especially in the hippocampus, has been documented in adult mice moved from a standard laboratory cage without "stimulation" to an enriched environment with toys and "stimulating objects" (Kempermann, Gast, & Gage, 2002). Neurogenesis has also been documented in adult mice that underwent "associative learning tasks" (Gould et al., 1999). Kemperman, Gast, & Gage found that moving the mice to an enriched environment exerted not only an acute, but also a sustained effect on brain fitness. In general, enriched mice (and rats) have bigger brains, generate more neurons (five times as many in the hippocampal region of the brain), develop less brain disease, and maintain mental acuity longer than their "un-enriched" counterparts (Kempermann, Gast, & Gage, 2002).

Studies with adult humans suggest similar benefits of enrichment and cognitively stimulating environments. One of the ways in which individuals may enrich their environment is to engage their mind in information processing tasks (i.e., cognitively stimulating activities) and leisure activities. Many activities in which individuals engage may be considered cognitively stimulating. Examples of common cognitively stimulating activities include reading, working crossword puzzles, writing, and playing cards and other games.

In 2002, Wilson, Mendes de Leon, and colleagues published results from a longitudinal study of 801 older adults from the Religious Orders Study. All of the participants were community dwelling members of a religious order and were followed for an average of 4.5 years. Of the 801 participants who began the study, 111 individuals developed AD and 622 did not (the rest were lost due to attrition, death, or other medical complication). Of those individuals who developed AD, the mean age was 81.1 (*SD* = 6.2) and the mean level of education was 18.1 years (*SD* = 3.6). Of those who did not develop AD, the mean age was 74.3 (*SD* = 6.3) and the mean level of education was 18.2 years (*SD* = 3.2).

Wilson, Mendes de Leon et al. (2002) employed a self-report questionnaire (originally validated in Wilson, Bennett, et al., 1999) to measure frequency of cognitive activity. The questionnaire inquired about the time the participant typically spent in seven common activities. All of the activities involved an information processing component and included watching television, listening to the radio, reading newspapers, reading magazines, reading books, playing games, and going to museums. Frequency of

participation in each activity was rated on a 5-point scale (5 = every day or nearly every day, 4 = several times per week, 3 = several times per month, 2 = several times per year, and 1 = once per year or less). The activity responses were averaged to create a composite activity measure.

At enrollment in the study, the participants were examined by a board-certified neurologist using NINCDS/ADRDA criteria and were judged *not* to have dementia. At baseline, participants were given the cognitive activity questionnaire and were also administered 19 tests to assess cognitive function: Immediate and delayed recall of the East Boston Story, Logical Memory Recall I-a and II-a, Word List Memory/Recall/ Recognition, Boston Naming Test, Extended Range Vocabulary, Verbal Fluency, National Adult Reading Test, Digits Forward and Digits Backward, Digit Ordering, Alpha Span, Symbol Digit Modalities Test, Judgment of Line Orientation, and Standard Progressive Matrices. At annual follow-up evaluations, participants were re-evaluated using the same measures. In addition, incident dementia and AD at follow-up were diagnosed yearly by a board certified neurologist using NINCDS/ADRDA criteria.

Composite scores for five cognitive domains (i.e. episodic memory, semantic memory, perceptual speed, working memory, and visuospatial ability), as well as for general cognitive function, were generated by standardizing the scores on the 19 cognitive tests. The composite scores were used to describe change in cognitive function (baseline minus follow-up). The relationship between decline in neuropsychological test performance and time spent participating in cognitively stimulating activities was analyzed producing a relative risk probability for AD (as diagnosed by a board certified

neurologist using NINCDS/ADRDA criteria) via a Cox proportional hazards model (Wilson, Mendes de Leon et al., 2002, p. 743). In random-effects models that controlled for age, sex, education, and baseline cognitive function, a one-point increase in cognitive activity was associated with reduced rate of decline in global cognitive function (reduced by 47%; b = .020, *SE* not given, $p < .05$), working memory (reduced by 60%; b = 0.021, *SE* = .008, $p < .007$), and perceptual speed (reduced by 30%; b = 0.026, *SE* = .012, $p < .02$). In addition, using AD diagnosis, they found each one-point increase (i.e. more frequent participation) in cognitive activity as measured at baseline was associated with a 33% reduction in AD risk (hazard ratio, 0.67; 95% CI, 0.49 – 0.92). On average, (at mean follow-up of 4.5 years) individuals reporting frequent participation in cognitively stimulating activities (90^{th} percentile) had nearly *half* the risk of developing AD compared to those individuals reporting less frequent participation (10^{th} percentile).

In a separate longitudinal study of older adults, Wilson, Bennett and colleagues (2002) examined whether cognitive activity could explain the association previously found between educational attainment and AD risk (Lindsay et al., 2002; Stern et al., 1994). They examined baseline cognitive activity using a questionnaire that inquired about an individual's current engagement in mentally stimulating activities in community-dwelling older adults (n = 842). Wilson, Bennett et al. compared baseline cognitive activity to risk of AD at four year follow-up. They conducted a series of logistic regression models adjusted for age, education, sex, race, and *APOE 4*-allele status. They found that level of education was associated with AD risk at four-year follow-up. However, when frequency of participation in cognitively stimulating

activities was added to the analysis, the associations between education and AD risk were reduced and no longer statistically significant. Specifically, they found that a one-point increase in the *current* cognitive activity (as measured at baseline) score was associated with a 64% reduction in risk of incident AD (OR = 0.36; 95% C.I. = 0.20 to 0.65). According to Wilson, Bennett et al. (2002), these findings suggest that the association between educational attainment and the risk of AD may be due to the fact that individuals with more education tend to be more cognitively active.

What accounts for the association between greater cognitive activity and reduced AD risk? Wilson & Bennett (2003) offered three possible explanations. One possible explanation is that cognitive activity and cognitive function are positively correlated. Cognitively active older adults are likely to enter advanced age with a higher level of cognitive function. Likewise, individuals with greater cognitive function are likely to be more cognitively active than individuals with reduced cognitive function. According to reserve theory, this individual would need to experience more disease pathology before expressing symptoms of dementia or perceptible cognitive decline.

Another explanation for the way in which cognitive activity may affect AD risk is "through an association with the primary manifestation of the disease" namely "progressive cognitive decline" (Wilson & Bennett 2003, p. 89). According to Wilson and Bennett, older adults who are more cognitively active enter old age with better cognitive skills and these skills may decline less quickly (2003). As support for this idea, Wilson and Bennett cite evidence that cognitive training programs have been shown to have substantial, long lasting, and beneficial effects on *specific* cognitive domains

(e.g., Ball et al., 2002). For example, Wilson and Bennett (2003) noted that cognitive activity appears to be primarily associated with reduced decline in processing skills like perceptual speed and working memory (e.g., Wilson, Mendes de Leon et al., 2002). As they remarked, processing speed and working memory skills are components of most cognitive or intellectual activities. Other evidence of the specificity of the protective effects of cognitive abilities includes an earlier study (Wilson, Bennett et al., 2000) where premorbid reading level was related to decline in *verbal* but not *nonverbal* abilities (as cited in Wilson & Bennett, p. 89).

A third explanation offered for the association between cognitive activity and cognitive function may be that individuals with subclinical AD, and other forms of neural degeneration, engage in and seek out less activity. Thus, reduced cognitive activity is perhaps an "early sign of the disease rather than an independent risk factor" (Wilson & Bennett, 2003, p. 90).

Wilson, Barnes, and Bennett (2003) examined cognitive activity and education, a widely used proxy or marker of general intellect and premorbid intellect. In this cross-sectional study of healthy older adults (n = 141, Mean age= 83.5, *SD* = 5.5; Mean education = 14.7, *SD* = 3.0), the authors examined the relation between *lifetime* cognitive activity, education, and cognitive function (i.e. performance on MMSE in addition to 17 commonly used neuropsychological tests) within five cognitive domains: Episodic memory, semantic memory, working memory, perceptual speed, and visuospatial ability. To this end, the authors created a Lifetime Cognitive Activity Assessment (LCAA) which is a questionnaire designed to ascertain the amount of time an individual has participated

in cognitive activities at various time points (hereafter referred to as age epochs) throughout life.

In the Wilson, Barnes, and Bennett (2003) study, community dwelling participants were asked to rate the frequency with which they engaged in cognitively stimulating activities at age 6 (three items), 12 (six items), and 18 (six items), 40 (five items), and current age (five items). Responses were rated on a 5-point scale, similar to the scale described above. Table 2 presents the items from the LCAA questionnaire.

Table 2

Lifetime Cognitive Activity Assessment items

Age epoch	Items
6	Read to; Play game; Tell story
12	Visit library; Read newspaper; Read magazine; Read Book; Write letter; Play game
18	Visit library; Read newspaper; Read magazine; Read Book; Write letter; Play game
40	Read newspaper; Read magazine; Read Book; Write letter; Play game
Present	Read newspaper; Read magazine; Read Book; Write letter; Play game

A composite cognitive activity score, calculated as a weighted sum of the responses on all the items, was used in linear regression analyses while controlling for age and sex. First, they examined the relation of lifetime cognitive activity and years of education. Next, they constructed a series of models: Model 1 = lifetime cognitive activity score and five domain measures of cognitive function (i.e. episodic memory, semantic memory, working memory, perceptual speed, and visuospatial ability); Model 2

= level of education and five domain measures of cognitive function; and Model 3 = education, LCAA score, and cognitive function. Wilson, Barnes, and Bennett (2003) found that education was positively related to lifetime cognitive activity, but only accounted for 6% of the variance in the cognitive activity score (b = 0.05, *SE* = 0.02, *p* = .002). They also found that LCAA score was significantly related to semantic memory, perceptual speed, and visuospatial ability (but not working memory) when education was in the model (b = 0.299, *SE* = 0.102, *p* < .01; b = 0.539, *SE* = 0.137, *p* < .001; and b = 0.400, *SE* = 0.119, *p* < .001, respectively). In the presence of LCAA score, however, education was only related to episodic memory (b = 0.047, *SE* = 0.022, *p* < .05).

Wilson and colleagues' results were limited in both studies. First, survival bias was an issue in the selection of participants. In other words, by choosing healthy older adults, a bias toward presumably better cognitive function is unavoidable. As such, it is impossible to truly test the limits of the cognitive reserve construct.

Second, the lack of diversity in the two samples created an issue relating to the generalizability of results. For example, in the Wilson, Bennett, et al (2002) study the religious sample was extremely homogenous with respect to education, lifestyle, and race (e.g., Mean education = 18 years, occupational status = 100% teacher or administrative, race > 94% Caucasian). Similarly, the Wilson, Barnes, and Bennett (2003) study was also highly homogeneous with respect to race (i.e. 95% White non-Hispanic) and education (i.e. range of education was not reported, Mean education = 14.7, *SD* = 3.0). Indeed, although the range of education was not reported in either study, the high means (i.e. Mean education = 18 years, and 14.7 years, respectively) suggest that at least 68% of

participants in the Wilson, Bennett, et al (2002) study had at least 15 years of school while 68% of participants in the Wilson, Barnes, and Bennett (2003) study had at least 11 years of education. It is possible that both studies lacked individuals with low to very low levels of education which could have produced a considerable floor effect. In other words, the relation between lower levels of education and activity may still be unknown. If the range of education was restricted in both studies, by lacking individuals with low levels of education, than it would have been impossible to fully test the association between education and cognitive function or the usefulness of education as a proxy for reserve.

Third, Wilson and colleagues used education as a stand-in for intelligence which they then used as a proxy for cognitive reserve. Their studies tested the association between 1) *education* and cognitive function and between 2) cognitive activity and cognitive function. They concluded, however, that 1) *intellect* had been adequately tested against cognitive activity and 2) cognitive activity accounted for more variance in cognitive function than *intellect.* Using education primarily as a measure of general intellect, however, is problematic. Indeed, the use of education as a measure of intellect is one of the major limitations of the cognitive reserve literature and will be addressed in detail below.

Review of the Cognitive Reserve Model

To review, the concept of cognitive reserve was developed in response to the clinical observation that individuals respond differently to similar levels of brain insult or pathology. Cognitive reserve is one explanation for the individual differences observed

clinically. The concern of the present study was to more closely examine the question: What factors contribute to the formation and maintenance of cognitive reserve? Accepting the widely employed practice of using education and general intellect as markers of cognitive reserve, the present study sought to compare the two most likely arguments for the formation of cognitive reserve: "Use-it-or-lose-it" versus general intellect. Figure 1 presents an illustration of these two arguments.

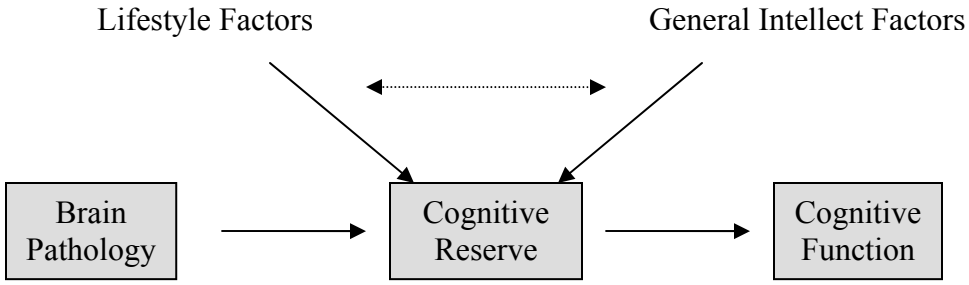


Figure 1. Cognitive reserve: Lifestyle versus general intellect factors

Recall that general intellect not only includes the genetic contribution to ability and intelligence, it also includes the early environmental influences that help shape the young brain (e.g., pre-natal and post-natal health, infant nutrition, early environmental enrichment, etc.). In this way, the general intellect argument is not purely genetic or biological. However, the general intellect argument differs from the "use-it-or-lose-it" lifestyle argument by maintaining that cognitive reserve is *mostly* a function of what you are born with. Further, the general intellect argument does not argue against the relationship between engagement in mentally stimulating activity and better cognitive function. Instead, the general intellect stance provides an explanation for the observed

correlation between increased cognitive activity and reduced dementia risk. In other words, the general intellect model contends that individuals with better brains (e.g., more efficient, more synaptic density, etc.) tend to engage in and remain engaged in, mentally stimulating activity even with advanced age. The lifestyle or "use-it-or-lose-it" argument maintains that engaging in mentally stimulating activity may convey additional benefits over simply possessing a better intellect.

Limitations of the Cognitive Reserve Literature

A major limitation in the cognitive reserve literature is the imprecise operationalization of the constructs of intellect and cognitive activity. Much of the reserve literature uses education as an estimate of general intellect. The present study contends that a more precise representation of the constructs (e.g., the use of an IQ measure to estimate intellect) is needed to adequately address the foremost question within the cognitive reserve debate described elsewhere in this manuscript: What accounts for the formation and maintenance of cognitive reserve, intellect or lifestyle?

Several methodological issues arise when education is used as an estimate for general intellect. According to Stern (2002) education (as well as occupation) is a reasonable proxy for cognitive reserve. Although scores on intelligence tests and level of education are highly related (Neisser et al., 1996), education may not be the best estimate of general intellect. In the present study we suggested that using a more direct measure of general intelligence, such as an IQ measure, is a better estimate of general intellect than education.

According to Stern (2003), estimates of Verbal IQ also serve as proxy measures for general intellect and thus, for cognitive reserve. Verbal IQ has been shown to be highly correlated with measures of Full Scale IQ and thus, general intelligence or *g* (Spearman, 1927). In the present study, we used a measure of Verbal IQ as an estimate of general intellect, thereby improving on Wilson and colleagues' methodological design. No studies of which we are aware have specifically tested the relation between a valid and reliable estimate of IQ, cognitive activity, and cognitive function.

A second measurement issue concerns the way in which cognitive activity has been assessed. It is difficult to separate the effects of cognitive activity from intelligence in existing studies. For example, do more intelligent individuals stay cognitively active longer than individuals with less intelligence? If so, perhaps the protective effects of cognitive reserve are not due to cognitive activity per se, but instead simply due to higher intelligence from birth. In other words, perhaps some individuals are simply born with better brains and thus, have more reserve to begin with. Perhaps these individuals can "afford" to lose more brain capacity than other individuals. Wilson and Bennett (2003) acknowledged this confound as a study limitation.

The Lifetime Cognitive Activity Assessment (LCAA) questionnaire, the primary measure used to assess cognitive activity, is problematic for several reasons. First, because it is a self-report measure, response may be confounded by negative affect. For example, it has been shown that self-reported memory complaints are related to self-reported depression and anxiety and not related to actual memory performance or future cognitive decline in older adults (Derouesné et al., 1999; Levy-Cushman & Abeles, 1998;

Ponds et al., 2000). This is one issue that has not been addressed or acknowledged in the existing cognitive reserve or cognitive activity literature. The present study suggested that responses on the LCAA self-report measure may be related to responses on other self-report measures, and that this potentially confounding relation should be examined.

Second, as a self-report measure, it is also vulnerable to response biases such as social desirability and experiment demand characteristics (Whitley, 2002). In other words, the participant may respond in ways that he or she believes will portray himself or herself in the best possible light or the participant could respond in a manner dependent on the participant's beliefs and feelings about the experiment and experimenter. Third, the LCAA is a *retrospective* measure that calls for individuals to report on activities that occurred during childhood, adolescence, and young adulthood. Retrospective reporting has often been shown to be inaccurate (Whitley, 2002). A fourth potential problem of the LCAA is that because the measure inquires about activities at different age epochs, it is unclear which epoch truly represents the "use-it-or-lose-it" argument. Fifth, activity is affected by many factors including mood, physical health, and cognitive ability in general. For example, an individual's current activity may be an indication of brain disease instead of a predictive factor for it. In other words, individuals may engage in less cognitive activity in the years immediately prior to dementia diagnosis due to the onset of progressive disease. A final problem with the LCAA, as Wilson and Bennett have acknowledged, the psychometrics of the questionnaire are not well established. Wilson et al.'s (2002, 2003) conclusions based on the LCAA have not yet been replicated in a longitudinal study. One important piece of psychometric data that is absent from the

literature, is test-retest reliability. As of the writing of this paper, there is no published data on the test-retest reliability of the LCAA, of which this author is aware.

A last measurement issue within the cognitive reserve literature is the choice of tests used to estimate cognitive function. Most studies use AD diagnosis and global cognitive decline (e.g., Wilson et al., 2003; Wilson, Barnes, & Bennett, 2003). Only a few studies have examined specific cognitive domains. As noted above, some cognitive processes are more sensitive to AD-associated pathology and thus, are better predictors of AD-associated decline especially in preclinical AD (i.e. learning and encoding, visuospatial, language, and executive function). An examination of the relation between cognitive reserve markers and domain measures of cognitive function is arguably a more informative endeavor than simply reporting on global ability.

The present study attempted to improve upon these limitations in several ways. First, the present study utilized an arguably more direct measure of intellect (i.e. WAIS-III Vocabulary subtest score). Second, the present study examined the age epochs of the LCAA in an effort to expand the knowledge of the psychometrics of the LCAA, as well as to attempt to replicate the findings of Wilson, Barnes, and Bennett (2003). Third, the present study attempted to increase the range of education heretofore sampled in the cognitive reserve literature, in an effort to more adequately test the relation between education, LCAA, and cognitive function. Lastly, the present study employed neuropsychological tests with adequate validity and reliability, as well as ecological validity in the context of cognitive processes known to decline in AD. Specifically, we

included tests of immediate verbal and nonverbal memory, delayed verbal and nonverbal memory, visuospatial ability, letter fluency, and executive function.

Present Study

To review, cognitive reserve theory attempts to explain the individual differences seen in response to brain injury, aging, or disease. In the current view, cognitive reserve is what accounts for the observation that two strokes of a similar magnitude in the same brain location may produce profound deficits in one individual while only producing mild impairments in another individual. The more cognitive reserve an individual has will allow him or her to absorb or tolerate greater amounts of brain injury or disease.

The goal of the present study was to examine factors typically used as markers of cognitive reserve, specifically; self-reported cognitive activity, education, and IQ, in an effort to discover which factor or factors are most related to neuropsychological performance. A secondary goal was to address the limitations of the cognitive reserve and cognitive activity literature, including an examination of the Lifetime Cognitive Activity Assessment (Wilson, Barnes, & Bennett, 2003) questionnaire. The present study attempted to address limitations of the literature in four ways, by: 1) Analyzing each age epoch of the LCAA (Wilson et al., 2002) in an effort to find the epoch or combination of epochs that is most related to current neuropsychological function in multiple cognitive domains; 2) examining the relation of the LCAA to self-reported symptoms of distress, 3) Utilizing a test of crystallized intelligence (i.e. Vocabulary) as a measure of general intellect in an effort to ascertain whether intellect is more related to current neuropsychological performance than education; and 4) Increasing the range of level of

education in the sample by actively recruiting individuals with twelve or less years of education.

The present study contained four basic hypotheses. First, the various proxies of cognitive reserve, both lifestyle factors and general intellect factors, will be associated with one another. Second, the level of cognitive reserve, whether estimated by a lifestyle or general intellect factor, will be significantly associated with measures of cognitive function (i.e. immediate memory, delayed memory, visuospatial ability, processing speed, mental flexibility/working memory, letter fluency, and overall cognitive function). Third, the level of self-reported distress (i.e. depression, stress, and memory function) will be significantly associated with lifestyle proxies of cognitive reserve (i.e. self-reported levels of cognitive activity as measured by the Lifetime LCAA). However, self-reported distress will not be significantly associated with general intellect proxies of cognitive reserve (i.e. level of education or intelligence). Fourth, lifestyle factors such as participation in stimulating mental activities will be positively associated with cognitive function, even after controlling for general intellect factors.

Methods

Power Analysis

Results of the Wilson, Mendes de Leon, and colleagues (2002) study demonstrated significant associations between cognitive function (defined as performance on neuropsychological tests of episodic memory, semantic memory, working memory, perceptual speed, and visuospatial ability), self-reported cognitive activity, and level of education. For the present study, the software program *Sample*

Power was used to calculate power and estimate sample size (using "beta per unit estimate" as the estimate of effect size) for a test of the association between self-reported cognitive activity and cognitive function (based on the associations reported in Wilson, Mendes de Leon, et al., 2002). According to logistic regression models reported by Wilson, Mendes de Leon, et al., the estimates ("b") reported between self-reported cognitive activity and cognitive function were as follows: Episodic memory, b = 0.1 (SE = 0.04, $p = .007$); semantic memory, b = 0.22 (SE = 0.04, $p < .001$); working memory, b = 0.08 (SE = 0.04, $p = .05$); perceptual speed, b = 0.21 (SE = 0.05, $p < .001$); and visuospatial ability, b = 0.13 (SE = 0.05, $p = .005$). Using a simple linear regression, continuous variable design based on the above estimates of effect, we needed a sample size between 25 and 45 individuals to achieve power = 0.80 with alpha = 0.05 for the first step of the analyses (i.e. first-order correlations). The second step of the analyses consisted of a series of hierarchical regression models using a continuous variable design based on the above estimates of effect. According to *Sample Power 2.0*, we needed a sample size of between 45 and 73 individuals to achieve power = .80 with alpha = 0.05.

Study Overview

From the existing study, 43 participants from Project S.C.O.R.E. were used. Project S.C.O.R.E. (Screening Cognition in Older Adults with Repeated Evaluations) is an ongoing longitudinal study conducted by the Clinical Neuropsychology Research Laboratory at Ohio University. The purpose of Project S.C.O.R.E. is to examine the current and predictive relation between cognitive performance and demographic, psychological, and cognitive variables. Participants were recruited through newspaper

advertising and fliers. Interested individuals contacted the Clinical Neuropsychology Research Laboratory by phone to schedule a testing session. Trained psychology graduate students administered all of the tests. Data from individuals deemed ineligible during the testing session were not included in analysis. Participants previously enrolled in Project S.C.O.R.E. were not paid for their participation. Instead, they received clinical feedback regarding their performance from the Study Director. Figure 2 illustrates the sequence of recruitment and testing procedures undertaken in the present study.

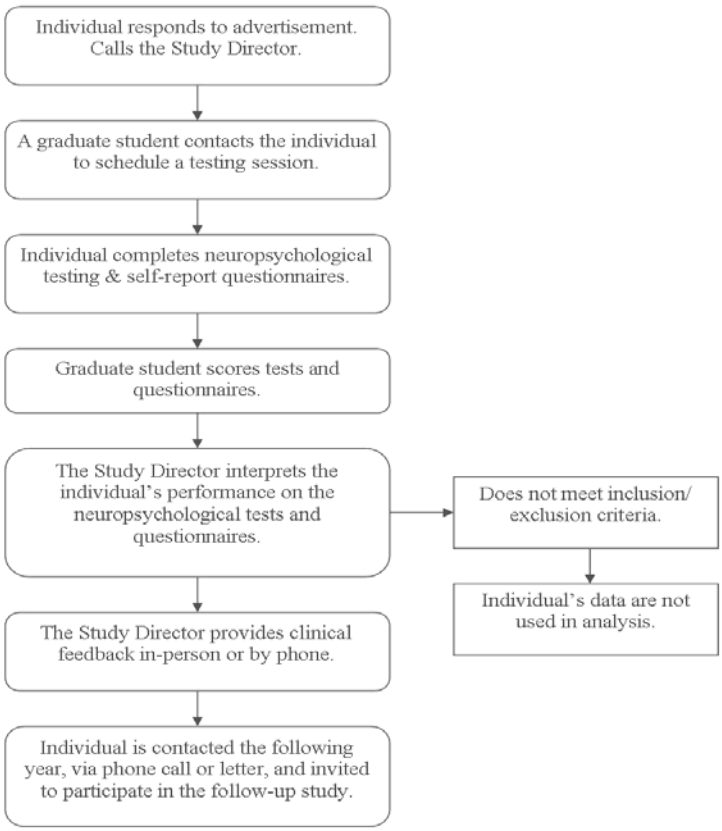


Figure 2. Overview of Recruitment and Testing Procedures for Project S.C.O.R.E.

In addition to the participants previously enrolled in the Project S.C.O.R.E. study, the present study recruited 23 new participants, with the total sample in the present study equaling 66 participants. Eighteen individuals (27 % of the sample) had 12 or less years of education. During the last eight months of the study recruitment, we began to offer $20 compensation for participants with 12 or less years of education.

Participants and Setting

Sixty-six healthy older adults from Southeast Ohio participated in the Project S.C.O.R.E. study. Inclusion criteria for Project S.C.O.R.E. and thus, the present study included: 1) at least 50 years of age; 2) the absence of neurological or psychiatric disorder including learning disability, major stroke, or diagnosed dementia; 3) no reported current drug or alcohol abuse; and 4) no reported history of head injury, defined as a "blow to the head followed by a loss of consciousness for at least 30 minutes". Testing was conducted at one of two locations, the Ohio University Psychology and Social Work Clinic or the Ohio University Clinical Neuropsychology Research Laboratory.

General Procedures

Each participant completed the standard S.C.O.R.E. test battery, which includes psychological, neuropsychological, and behavioral tests and takes 1 ½ to 2 hours to complete. Not all of the tests administered in the S.C.O.R.E. battery were used in the present study. Therefore, only the measures used in the present study are reviewed in detail here.

After informed consent was obtained the participants completed a biographical and health status interview, administered by a trained examiner. After the interview, half of the participants received self-report questionnaires before receiving the cognitive and intelligence measures and the other half of the participants received the self-report psychological questionnaires after the cognitive and intelligence measures. The order of was counter-balanced in an effort to account for possible confounds related to order of test administration (see Appendix A for a diagram of the counter-balanced order of administration of measures).

The questionnaires were administered in the following order: 1) the Perceived AD Threat Scale (Roberts et al., 2000), 2) the Memory Controllability Inventory (MCI; Lachman, Bandura, Weaver, & Elliot, 1995), 3) The Geriatric Depression Scale (GDS; Yesavage, Brink, Rose, & Adey, 1983), 4) the Perceived Stress Scale (PSS; Cohen, Kamarck, and Mermelstein, 1983), and 5) the Lifetime Cognitive Activity Assessment (LCAA; Wilson, Barnes, & Bennett, 2003). The neuropsychological measures were administered in the following order: The Repeatable Battery for the Assessment of Neuropsychological Status (RBANS; Randolph, 1994), Controlled Oral Word Association (COWA) from the Multilingual Aphasia Examination (Benton, Hamsher, & Sivan, 1994), the Trail-Making Test (TMT; Reitan, 1958), and the Benton Visual Retention Test (BVRT; Sivan, 1992). At the conclusion of the testing session, participants received 1) a "Post-Study" form which provided the participant with contact information for psychological and geriatric health resources and 2) a "Permission to Contact" form which gave participants the option of being contacted for follow-up or

additional studies (see Appendices B – K for the forms and non-copyrighted measures and questionnaires listed above). Participants were also offered clinical feedback by the Study Director, a licensed clinical psychologist.

Measures

Biographical and Health Status Interview

Participants were asked to provide information regarding age, handedness, marital status, ethnicity, education level (including Major or area of concentration), involvement in community/volunteer/church work, involvement in social or recreational groups, learning disability history, medical history and current status, mental health history and current status, alcohol and nicotine habits, experience with AD, and their reason for participating in the study. The interview always preceded any testing (i.e. the self-report questionnaires or neuropsychological measures). For the present study, this information was used to follow inclusion/exclusion rules for Project S.C.O.R.E. and to document educational level. *Level of education* was defined as the number of self-reported years of education completed.

Neuropsychological Measures

Although four cognitive tests were given in the Project S.C.O.R.E battery, only the variables of interest to the present study are described in detail below, which include RBANS age-corrected Index scores, a RBANS Composite score, COWA score, and TMT - Part B score.

Repeatable Battery for the Assessment of Neuropsychological Status (RBANS). The RBANS served as both a dementia screen and an estimate of the participant's current

level of functioning in various cognitive domains. There are two forms of the RBANS (Form A and B). The RBANS is comprised of five indices that assess distinct cognitive domains or processes: Immediate memory, visuospatial, language, attention, and delayed memory (Randolph, 1994). Of the five indices, only age-adjusted scores for immediate memory, delayed memory, and visuospatial ability were analyzed for the present study. The total RBANS composite score was also be used in the analysis.

The internal consistency of the RBANS subtests is high, ranging from .78 - .95. The ranges of internal consistency coefficients for the five indices (for adults aged 50-89) are as follows: Immediate memory .85 to .90, visuospatial .78 to .84; language .81 to .87; attention .83 to .88; and delayed memory .81 to .85. The reliability coefficient for the RBANS composite score is .93 to .95. Test-retest reliability for Form A was examined using 40 participants. The test-retest interval ranged from 33 - 43 weeks (Mean interval = 38.7 weeks, SD = 2.8 weeks). Form A test-retest reliability (n= 40) for the five subtests were: Immediate memory = .78, visuospatial = .69, language = .55, attention = .75, and delayed memory = .65. The test-retest reliability coefficient for the total scale score was .88 (Randolph, 1994). Re-test reliability was also examined for Form A to Form B (i.e. Form A administration followed by Form B administration at follow-up, or vice versa) using 100 participants with a counter-balanced design. The retest interval ranged from one to seven days. The Form A – Form B retest reliability for each subtest is as follows: Immediate memory = .68, visuospatial ability = .65, language = .46, attention = .80, and delayed memory = .64. The Form A - Form B retest reliability coefficient for the total scale score is .82 (Randolph, 1994).

Construct validity, or the extent to which a test measures the theoretical construct of interest, has been demonstrated for the RBANS from 1) the pattern of inter-correlation of subtest scores and 2) comparison studies with the RBANS and external measures. The pattern of inter-correlation of RBANS subtest scores (n = 540) suggests that the subtests indeed measure distinct cognitive constructs. As expected, the highest correlation is found between immediate and delayed memory (r = .63). The inter-correlations between the memory indices and other indices range from .28 (immediate memory with visuospatial) to .44 (immediate memory with language). The inter-correlations between the visuospatial, language, and attention index scores range from .20 to .40.

If the participant was administered RBANS – Form A at their previous screening (i.e. approximately one year prior), they were administered Form B at the current screening, and vice versa. Raw scores on each subtest were converted to age-adjusted scaled scores (Mean = 100, *SD* = 15). A total scale score and five subtest scores are generated. Lower scores indicate greater impairment (Randolph, 1994). Administration time for Form A or Form B is 20 to 30 minutes.

C*ontrolled Oral Word Association (COWA).* COWA is a letter fluency task in which participants are asked to generate as many words as possible that begin with a specified letter of the alphabet, within one minute (Benton, Hamsher, & Sivan, 1994). Participants are instructed to refrain from offering 1) proper nouns and 2) words that differ only by suffix (e.g., If “eat” is followed by “eating”, then “eating” is ineligible). Participants were presented with one of two three-letter combinations; C-F-L or P-R-W. Each of the three letters was presented in a separate trial.

Inter-rater reliability is near perfect. One-year retest reliability in older adults is .70, with shorter testing intervals yielding coefficients as high as .88 (Spreen & Strauss, 1998). A participant's score on COWA is the combined number of eligible words for the three letters presented. COWA has been found to be sensitive to neurological impairments such as dementia (Spreen & Strauss, 1998). Fewer words generated indicate greater impairment (Benton, Hamsher, & Sivan, 1994). Administration time is approximately five minutes.

Trail Making Test (TMT). The TMT (Part B) is a timed test that measures visual scanning ability, mental flexibility, sequencing ability, and attention (Reitan, 1958). Part B contains both numbers and letters that participants must connect in order (e.g., 1 – A – 2 – B – 3 – C). Retest reliability, up to one year, ranges from .66 to .86 (Spreen & Strauss, 1998). Factor analytic studies indicate that the TMT – Part B loads on factors of rapid visual search and visuospatial sequencing. Part B also loads on focused mental processing speed and has been shown to be sensitive to various types of neurological damage (Spreen & Strauss, 1998). TMT performance is measured by time (in seconds) to complete each part. Longer time (measured in seconds) to complete the task indicates greater impairment (Reitan, 1958). Administration time is approximately five minutes.

Self-report Measures

Participants completed five paper-and-pencil questionnaires. All questionnaires were self-administered. The Perceived AD Threat Scale, although part of the S.C.O.R.E. battery, was not used in the present study and will not be discussed further.

The Memory Controllability Inventory (MCI). The MCI taps an individual's beliefs about their everyday memory, their memory function, and the likelihood they will develop AD. The MCI also taps the participant's beliefs about memory changes that occur with age. There are six subscales on the MCI: Present ability, potential improvement, effort utility, inevitable decrement, independence, and Alzheimer's disease likelihood (Lachman, Bandura, Weaver, & Elliot, 1995). For the present study, only the present ability subscale was used. Internal consistency coefficients for the present ability scale has ranged from .58 - .70. Inter-item correlations ranged from .30 - .68. Nine day retest reliability ranged from .50 - .65 and three-month retest coefficients ranged from .46 - .57. Administration time takes less than five minutes.

The Geriatric Depression Scale (GDS.). The GDS measures depressive symptoms in older adults and consists of 30 yes/no questions designed for self-administration. The item-total correlation ranges from .32 to .83 with a mean of .56. Internal consistency is .94 and split-half reliability is .94 (Brink et al., 1982). One-week retest reliability was found to be .85. Factor analysis established *dysphoria* (e.g., unhappiness, dissatisfaction with life, helplessness, etc.) as a major factor and worry/dread/obsessive thoughts and apathy/withdrawal as minor factors. Scores on the GDS correlate strongly with scores on the Beck Depression Inventory (.73) indicating good concurrent validity. Further, the GDS has been shown to discriminate well between dementia and depression (Spreen & Strauss, 1998). The cut-off points are: Normal (0-9), mild depression (10-19), and moderate to severe depression (20-30) (Yesavage, Brink, Rose, & Adey, 1983). Administration time is approximately five minutes.

The Perceived Stress Scale (PSS). The PSS is a 10-item scale designed to measure the degree to which an individual has perceived the events in his or her life as stressful, within the last month. The items measure how unpredictable, uncontrollable, and overloaded an individual views his or her life. The scale was designed for use with community-dwelling individuals with at least a middle school education (Cohen, Kamarck, and Mermelstein, 1983). There is no gender effect on scores. Cronbach's coefficient alpha ranges from .67 (Remor and Carrobles, 2001) to .86 (Cohen, Kamarck, and Mermelstein, 1983). Test-retest reliability for a two-day interval has been found to be .85, while six-week interval retest reliability was found to be .55. It has been found to be moderately to strongly correlated with a range of behavioral and self-report measures of stress (e.g., negative life events; Cohen, Kamarck, and Mermelstein, 1983). Higher scores indicate greater perceived stress. Administration time is less than five minutes.

The Lifetime Cognitive Activity Assessment (LCAA). The LCAA queries the participant about his or her involvement in common cognitive activities (e.g., reading, playing games, etc.). The questions are designed to assess five time-periods (i.e. age epochs) in the participant's life: Age 6, age 12, age 18, age 40, and present day. A total score consists of a weighted sum of the responses from the five different epochs on a five-point scale (e.g. every day = 4 points, several times per week = 3 points, several times per month = 2 points, several times per year = 1 point, once a year or less = 0 points). Cronbach's coefficient alpha was 0.88, indicating high internal consistency among the items. There is no published data on the test-retest reliability of the LCAA. Higher scores on the LCAA indicate reports of more frequent participation in cognitive

activity (Wilson, Barnes, & Bennett, 2003). For the present study, participants' response to LCAA items were divided according to the five epochs specified on the measure (i.e. age 6, 12, 18, 40, present day) in addition to a new level created for the purposes of the present study. This new level (i.e. cognitive activity – adult) is the sum of age 40 and present day epoch responses. Due to the restricted range within some of the age epochs on the LCAA, it was hoped that creating a composite score that only includes adult activity would prove useful for statistical analysis by increasing the range and variance, compared to that of a single epoch. Further, a recent study by Wilson et al. (2005), suggested that self-reported activity for current and recent age epochs is most associated with cognitive function. Higher scores indicate more participation in cognitive activities. Administration time takes less than five minutes.

The Wechsler Adult Intelligence Scale – III (WAIS-III) Vocabulary Subtest. The WAIS-III Vocabulary Subtest is a test of verbal comprehension and acquired knowledge. For this subtest, participants are presented a word both orally and visually and asked to provide the meaning of that word. Perfect responses earn 2-points, while 1-point is given for partially correct responses. Administration is discontinued after 6 consecutive scores of zero. Administration time is approximately 10 to 15 minutes.

At least two studies have shown the stability of Vocabulary Subtest scores (i.e. Weins, Bryan, & Crossen, 1993; Yates, 1954) across time, even among individuals with mental illness. The most recent study Weins, Bryan, and Crossen (1993), sampled 24 individuals with schizophrenia. The WISC-R or the WAIS-R (depending on the participant's age) was administered at Time 1 and the WAIS-R was administered at Time

2. The average length of follow up was 23 years (SD = 10.6). They found that WISC-R or WAIS-R Vocabulary subtest scores measured at Time 1 was highly correlated with Full Scale IQ at Time 1 ($r = .80$, $p < .01$) and Time 2 ($r = .80$, $p < .01$). Further, Vocabulary score is highly related to WAIS index and scale scores which have been shown to remain stable over time. For example, the Vocabulary subtest has been shown to be highly correlated with Verbal IQ ($r = .83$), the Verbal Comprehension Index ($r = .83$), and Full Scale IQ ($r = .80$) on the WAIS-III (Wechsler, 1997). The split-half reliability for the Vocabulary subtest was .93 and the retest (average retest interval was five weeks) reliability was .91 (Kaufman & Lichtenberger, 1999).

Hypotheses

Hypothesis 1. Lifestyle and general intellect factors will be significantly associated with one another. Specifically, it was hypothesized that Vocabulary score, education, and LCAA score at age 6, 12, 18, 40, Present, total, and adulthood (40 + present day) would be related to each other. To test this hypothesis, zero order correlations were examined.

Hypothesis 2. Lifestyle and general intellect factors will be significantly associated with cognitive function.

Specifically, it was hypothesized that Vocabulary score, education, and LCAA score at age 6, 12, 18, 40, Present, total, and adulthood (40 + present day) would be related to performance on:

2(a) RBANS Immediate Memory Index,

2(b) RBANS Delayed Memory Index,

2(c) RBANS Visuospatial Index,

2(d) TMT B,

2(e) COWA, and

2(f) the RBANS composite score.

To test this hypothesis, zero-order correlations were examined. As stated above, the Lifetime Cognitive Activity questionnaire has not been examined in detail. As such, it was unclear which epoch would be most highly associated with cognitive function or which epoch component truly isolates the construct of mental activity. Therefore, after zero-order correlations were analyzed, the cognitive activity epoch which was the most highly related to neuropsychological performance would be used in hierarchical regression analyses.

Hypothesis 3. Self-reported depressive symptoms (i.e. Geriatric Depression Scale), perceived stress (i.e. Perceived Stress Scale), and memory function (i.e. Memory Controllability Inventory) will be significantly associated with *lifestyle* proxies of cognitive reserve (i.e. Lifetime Cognitive Activity Assessment). Specifically, it was hypothesized that scores on the three self-report measures would be significantly related to:

4(a) Lifetime Cognitive Activity Assessment- Age 6 epoch score,

4(b) Lifetime Cognitive Activity Assessment- Age 12 epoch score,

4(c) Lifetime Cognitive Activity Assessment- Age 18 epoch score,

4(d) Lifetime Cognitive Activity Assessment- Age 40 epoch score,

4(e) Lifetime Cognitive Activity Assessment- Present Age epoch score.

4(f) Lifetime Cognitive Activity Assessment- Total composite score

4(g) Lifetime Cognitive Activity Assessment- Adult composite score.

To test this hypothesis, zero-order correlations were examined among all of the independent variables. If zero-order correlations showed a significant relation between cognitive activity and any of the self-report measures, then a series of hierarchical regression analyses would be performed.

Hypothesis 4. Lifestyle factors such as self-reported cognitive activity will be associated with cognitive function even after controlling for general intellect factors.

Specifically, it was hypothesized that after accounting for Vocabulary score and education, self-reported cognitive activity would continue to be related to neuropsychological performance on:

3(a) RBANS Immediate Memory index,

3(b) RBANS Delayed Memory index,

3(c) RBANS Visuospatial ability index,

3(d) RBANS composite score,

3(e) TMT B, and

3(f) COWA.

These hypotheses were tested with hierarchical regression analyses. The cognitive activity variable used was to be the epoch that was most highly related with neuropsychological performance in a zero-order correlation matrix.

Results

Statistical Analyses

Prior to conducting statistical analyses, exploratory analyses were conducted to assess for normality, linearity, homoscedasticity, and the presence of outliers. Only education had a non-normal, bimodal distribution. All statistical tests were two tailed, with $\alpha = .05$.

The current study utilized multiple data analytic techniques. First, Pearson product-moment correlation coefficients were conducted on all of the variables collected in the study and were used to investigate 1) the relationship among proxies of cognitive reserve and neuropsychological performance; 2) to identify which epoch of the LCAA is most strongly related to neuropsychological performance, and thus should be used in hierarchical regression analyses; and 3) to examine the relationship between scores on self-report psychological measures, self-reported cognitive activity, vocabulary, and education. Next, hierarchical regression analyses were conducted to determine the amount of unique variation in neuropsychological performance accounted for by self-reported cognitive activity, vocabulary score, and education. Finally, exploratory correlation analyses were conducted.

Participant Characteristics

Sixty-six individuals participated in the study. All participants were administered the biographic health interview, four neuropsychological measures, and five self-report psychological measures. Vocabulary scores were also obtained for 50 of the 66

participants. Vocabulary subtest score and RBANS indices scores were corrected for age. The sample was comprised of 46 females and 20 males, 100% of whom reported Caucasian as their race. Participants ranged in age from 45 to 85 years old, with an average age of approximately 63 years (M = 62.98, SD = 8.6). Participants' self-reported level of education ranged from 10 to 20 years, with an average level of education of approximately 16 years (M = 15.68, SD = 3.04). Table 3 presents the participant characteristics.

Table 3

Participant Characteristics: Gender, Age, and Education

	N
Gender	
Male	20
Female	46
Total	66
Age	
45 – 55 years	10
56 – 65 years	32
66 – 75 years	19
76 – 85 years	5
Education	
0 – 12 years	18
13 – 16 years	21
17 + years	27

Table 4 shows the participant's performance on the vocabulary subtest and the LCAA. Vocabulary scaled scores were adjusted for age. Higher scores indicated a better vocabulary. Scores on the LCAA are divided into the five age-epochs, a total score combining responses for all age-epochs, and an adult score which is the sum of the age

40 and present age epochs. Women performed better than men on vocabulary subtest, the Immediate and Delayed Memory indices on the RBANS, and on COWA.

Table 4

Lifetime Cognitive Activity Assessment (LCAA) and Vocabulary Scaled Score

	N	Range	M (*SD*)
LCAA			
Age 6	65	0 – 12	8.1 (3)
Age 12	66	0 – 23	13.6 (4.7)
Age 18	66	5 – 22	15 (4.1)
Age 40	66	6 – 20	13.3 (3)
Present Age	66	5 – 21	13.3 (3.5)
Total Score	65	27 – 65	63.2 (13.3)
Adult Score	66	12 – 40	26.6 (6.0)
Vocabulary Scaled Score	50	9 – 18	13.5 (2.4)

Pearson Product-moment Correlations

Pearson product-moment correlation coefficients were calculated to investigate the relationship among the independent variables (education, vocabulary, and LCAA age 6, 12, 18, 40, present, total, and adult) thought to be related to neuropsychological performance. The results, presented in Table 5, revealed that more education was associated with higher vocabulary scores but *not* with self-reported cognitive activity on the LCAA. Conversely, higher vocabulary score was associated with more self-reported cognitive activity at age 40, present age, total cognitive activity, and cognitive activity in adulthood. Vocabulary score was *not* related to self-reported cognitive activity at age 6, age 12, or age 18.

Table 5

Pearson Product-Moment Correlations among Education, Vocabulary, Cognitive Activity, and Neuropsychological Measures

	Ed	Voc	CA-6	CA-12	CA-18	CA-40	CA-P	CA-T	CA-Ad	TMT	COWA	VS	IM	DM	RBAN
Ed	1.0	.46**	.16	.13	.18	.15	.13	.20	.15	-.05	.27*	.18	.32*	.25*	.31*
Voc		1.0	.08	.05	.14	.37*	.46**	.31*	.45**	-.01	.23*	.12	.40*	.40*	.49**
CA-6			1.0	.38*	.38*	.16	.26*	.58**	.23	-.22	.15	.16	.09	-.03	.12
CA-12				1.0	.57**	.28*	.18	.73**	.24*	-.02	-.12	.18	.12	.01	.07
CA-18					1.0	.58**	.45**	.85**	.55**	.04	-.06	.12	.08	.03	-.02
CA-40						1.0	.73**	.73**	.92**	.20	-.14	-.14	-.09	-.08	-.18
CA-P							1.0	.69**	.94**	.08	.07	-.13	.03	.07	-.03
CA-T								1.0	.76**	.03	-.001	.07	.04	.01	-.004
CA- Ad									1.0	.14	-.02	-.15	-.03	.004	-.11
TMT										1.0	-.40**	- . 3 5 *	-.43**	-.41**	-.58**
COWA											1.0	.25*	.45**	.34*	.57**
VS												1.0	.36*	.40**	.68**
IM													1.0	.76**	.82**
DM														1.0	.81**

Note. *N = 48 – 66,* * $p < .05$, ** $p \leq .001$. Ed = Education, Voc = Vocabulary SS, CA-6 = LCAA age 6, CA-12 = LCAA age 12, CA-18 = LCAA age 18, CA-40 = LCAA age 40, CA-P = LCAA present age, CA-T = total LCAA, CA-Ad = LCAA age 40 + present age, TMT = Trail-Making Test – Part B, COWA = Controlled Oral Word Association, VS= Visuospatial Index on the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS), IM = RBANS Immediate Memory Index, DM = RBANS Delayed Memory Index, RBAN = RBANS composite score.

It was hypothesized that all LCAA epochs would be significantly associated with measures of immediate memory, delayed memory, visuospatial ability, processing speed, mental flexibility/working memory, and global cognitive function. However, there were no significant correlations found between *any* time epoch from the LCAA and *any* neuropsychological measure (see Table 5). It was also hypothesized that education and vocabulary would be significantly associated with neuropsychological performance. The zero-order correlations revealed that more education was associated with better letter fluency, better immediate memory, better delayed memory, and better overall cognitive function (see Table 5). Similarly, higher vocabulary scores were associated with better letter fluency, better immediate memory, better delayed memory, and better overall cognitive function. There was not a significant correlation between education or vocabulary and visuospatial ability. Both education and vocabulary were modestly associated with COWA, a speeded letter fluency task. However, neither vocabulary nor education was correlated with performance on the TMT-B, a speeded task tapping mental flexibility and working memory.

As there were gender effects found on tests of immediate memory, delayed memory, COWA, and vocabulary (i.e. women performed better than men), we re-ran correlations with cases split by gender. Although the general pattern of the correlations remained the same, the relationship magnitudes between vocabulary and education and performance on immediate memory and delayed memory Index decreased. Two possible explanations for the change in the magnitude of the association for immediate and

delayed memory include a loss of variance and a detrimental loss of N when the male cases were not included in the analyses.

Pearson product-moment correlation coefficients were calculated to investigate the relationship between self-report instruments (including LCAA), education, and vocabulary. It was hypothesized that self-reported depressive symptoms, stress, and memory function would be significantly associated with LCAA scores. It was further hypothesized that self-reported depressive symptoms, stress, and memory function would *not* be significantly associated with level of education or vocabulary score. The results, presented in Table 6, indicated that contrary to prediction, self-reported ratings of depressive symptoms, perceived stress, and memory function were *not* associated with self-reported cognitive activity, at any age epoch. Further, contrary to prediction, more education was significantly related to lower ratings of self-reported depressive symptoms and perceived stress. However, as predicted, vocabulary score was *not* related to depressive symptoms, perceived stress, or memory function. Similarly, as predicted, level of education was also *not* related to memory function.

A series of hierarchical linear regression analyses were proposed to examine the relative contribution of vocabulary, education, and self-reported cognitive activity to variance in neuropsychological performance. Specifically, we predicted that even after controlling for education and vocabulary, self-reported cognitive activity would still account for a significant amount of variance in immediate memory, delayed memory, processing speed, mental flexibility/working memory, visuospatial ability, and a measure of general cognitive function. However, contrary to expectations, self-reported cognitive

activity was not related to performance on *any* of the neuropsychological tests. Although there is no statistical reasoning for conducting hierarchical regression when zero order correlations are insignificant; we ran all analyses *as proposed*.

Table 6

Pearson Product-Moment Correlations among Self-report Measures, Education, Vocabulary Scaled Score, and Cognitive Activity

	Voc	CA-6	CA-12	CA-18	CA-40	CA-P	CA-T	CA-Ad	GDS	MCI	PSS
Ed	.46**	16	.13	.18	.15	.13	.20	.15	-.30*	.22	-.27*
Voc	1.0	.08	.05	.14	.39*	.46**	.31*	.45**	.000	-.12	.05
CA-6		1.0	.38*	.38*	.16	.26*	.58**	.23	-.20	-.09	-.06
CA-12			1.0	.57**	.28*	.18	.73**	.24*	.06	-.13	.02
CA-18				1.0	.58**	.45**	.85**	.55**	-.17	.002	-.14
CA-40					1.0	.73**	.73**	.92**	-.11	-.17	-.03
CA-P						1.0	.70**	.94**	-.13	-.07	-.10
CA-T							1.0	.76**	-.15	-.12	-.09
CA-Ad								1.0	-.13	-.13	-.07
GDS									1.0	.55**	.55**
MCI										1.0	-.48**

Note. * $p < .05$, ** $p \leq .001$. Ed = Education, Voc = Vocabulary SS, CA-6 = Lifetime Cognitive Activity Assessment (LCAA) score age 6, CA-12 = LCAA score age 12, CA-18 = LCAA score age 18, CA-40 = LCAA score age 40, CA-P = LCAA score present age, CA-T = total LCAA score, CA-Ad = LCAA age 40 + present age, GDS= Geriatric Depression Scale, MCI = Memory Controllability Inventory, PSS = Perceived Stress Scale.

For the present study, we created a composite measure of self-reported cognitive activity in adulthood (i.e. cognitive activity – adult = the sum of responses to LCAA items for age 40 and present) to stand as the independent variable for the hierarchical

regression analyses because 1) the composite measure includes activity at age 40 which, if validly reported, should moderate the potentially negative effects of age or health on the participation in cognitive activities; and 2) the lifestyle argument for "use-it-or-lose-it" is centered on the beneficial effects of being active *currently,* which is also included in the composite, and 3) Wilson, et al. (2005) recently found that self-reported current cognitive activity was more related to cognitive function than earlier activity.

When conducting the hierarchical regression analyses, each neuropsychological measure was tested 1) in a model with education entered first, self-reported cognitive activity in adulthood entered second, and the interaction term entered third; 2) in a model with vocabulary entered first, and self-reported cognitive activity in adulthood entered second. Consistent with Wilson, Barnes and Bennett (2003) and Wilson, Mendes de Leon and colleagues (2002), we re-ran hierarchical regression analyses with self-reported cognitive activity in adulthood entered first, and education or vocabulary entered second. The results indicated that in the final model after controlling for level of education, self-reported cognitive activity in adulthood did *not* add a significant contribution to the variance in immediate memory ($R^2\Delta = .005$, $F (1, 63) = .37$, $p = .55$), delayed memory ($R^2\Delta = .001$, $F (1, 61) = .07$, $p = .79$), visuospatial ability ($R^2\Delta = .03$, $F (1, 63) = 2.06$, $p = .16$), general cognitive function ($R^2\Delta = .02$, $F (1, 60) = 1.67$, $p = .20$), TMT-B ($R^2\Delta = .02$, $F (1, 61) = 1.42$, $p = .24$), or COWA ($R^2\Delta = .004$, $F (1, 63) = .28$, $p = .60$) performance. However, when controlling for self-reported cognitive activity in adulthood, education accounted for an additional significant portion of the variance in immediate memory ($R^2\Delta = .11$, $F (1, 63) = 7.38$, $p = .009$), delayed memory ($R^2\Delta = .06$,

F (1, 61) = 4.09, p = .05), general cognitive function ($R^2\Delta$ = .11, F (1, 60) = 7.53, p =.008), and COWA ($R^2\Delta$ = .08, F (1, 63) = 5.34, p = .02). Education did not account for an additional significant amount of variance when accounting for self-reported cognitive activity, in visuospatial ability ($R^2\Delta$ = .04, F (1, 63) = 2.89, p = .09) or TMT-B performance ($R^2\Delta$ = .005, F (1, 61) = .32, p = .57). See Table 7 for final models.

Table 7

Hierarchical Regression Analyses Predicting Neuropsychological Performance from Education and Cognitive Activity in Adulthood: Final Models

Dependent Variable	Independent Variable	*B* (SE)	Beta	*p* value
Immediate Memory Index				
(N = 66)	Education	1.75 (.64)	.33	.009*
	Cognitive Activity Adult	-.20 (.33)	-.07	.55
Delayed Memory Index				
(N = 64)	Education	1.1 (.54)	.25	.05*
	Cognitive Activity Adult	-.07 (.28)	-.33	.79
Visuospatial Index				
(N = 66)	Education	1.2 (.68)	.21	.09
	Cognitive Activity Adult	-.50 (.34)	-.18	.16
RBANS Composite Score				
(N = 63)	Education	1.83 (.67)	.34	.008*
	Cognitive Activity Adult	-.44 (.34)	-.16	.20
COWA				
(N = 64)	Education	1.11 (.48)	.28	.02*
	Cognitive Activity Adult	-.13 (.24)	-.06	.60
TMT-B				
(N = 64)	Education	-.75 (1.3)	-.07	.57
	Cognitive Activity Adult	.80 (.67)	.15	.24

Note. * indicates a significant correlation. RBANS = Repeatable Battery for the Assessment of Neuropsychological Status, TMT - B = Trail-Making Test – Part B, COWA = Controlled Oral Word Association.

The results of the final model that predicted neuropsychological performance from vocabulary score and self-reported cognitive activity in adulthood are presented in Table 8.

Table 8

Hierarchical Regression Analyses Predicting Neuropsychological Performance from Vocabulary Scaled Score and Cognitive Activity in Adulthood: Final Models

Dependent Variable	Independent Variable	*B* (SE)	Beta	*p* value
Immediate Memory Index				
(N = 50)	Vocabulary SS	3.4 (.97)	.52	.001*
	Cognitive Activity Adult	-.69 (.39)	-.26	.08
Delayed Memory Index				
(N = 48)	Vocabulary SS	2.7 (.81)	.50	.002*
	Cognitive Activity Adult	-.48 (.33)	-.22	.15
Visuospatial Index				
(N = 50)	Vocabulary SS	1.6 (1.1)	.23	.15
	Cognitive Activity Adult	-.69 (.44)	-.25	.12
RBANS Composite Score				
(N = 48)	Vocabulary SS	4.6 (.90)	.67	.000*
	Cognitive Activity Adult	-1.1 (.36)	-.41	.003*
COWA				
(N = 50)	Vocabulary SS	1.9 (.75)	.38	.02*
	Cognitive Activity Adult	-.38 (.30)	-.19	.21
TMT-B				
(N = 49)	Vocabulary SS	-.24 (2.12)	-.10	.60
	Cognitive Activity Adult	.97 (.86)	.18	.26

Note. * indicates a significant correlation. RBANS = Repeatable Battery for the Assessment of Neuropsychological Status, TMT – Part B = Trail-Making Test - B, COWA = Controlled Oral Word Association.

The results indicated that after controlling for vocabulary scaled score, self-reported cognitive activity in adulthood did *not* add a significant contribution to the variance in immediate memory ($R^2\Delta$ = .05, F (1,47) = 3.12, p = .08), delayed memory ($R^2\Delta$ = .04, F (1,45) = 2.18, p = .15), visuospatial ability ($R^2\Delta$ = .05, F (1,47) = 2.46, p = .12), TMT-B ($R^2\Delta$ = ..03, F (1,46) = 1.29, p = .26), or COWA ($R^2\Delta$ = .03, F (1,47) = 1.60, p = .21). However, when controlling for self-reported cognitive activity in adulthood, vocabulary accounted for a significant additional portion of the variance in immediate memory ($R^2\Delta$ = .21, F (1,47) = 12.65, p = .001), delayed memory ($R^2\Delta$ = .20, F (1,45) = 11.22, p =

.002), general cognitive function ($R^2\Delta$ = .36, F (1,45) = 25.99, p = .000), and COWA ($R^2\Delta$ = .12, F (1,47) = .6.21, p = .02). Vocabulary did *not* account for an additional amount of variance, when accounting for self-reported cognitive activity, in visuospatial ability ($R^2\Delta$ = .04, F (1, 47) = 2.06, p = .16) or TMT-B ($R^2\Delta$ = .007, F (1, 46) = .34, p = .56) performance. Final models are presented in Table 8.

Exploratory Analyses

The general absence of significant correlations between the Lifetime Cognitive Activity Assessment and neuropsychological performance was unexpected and inconsistent with the findings in other large sample studies conducted by Wilson and colleagues. As the mean education level in the Wilson studies was 14.6 to 18.1 years, we wondered if the inclusion of individuals with lower education in the present study had affected the relationship between LCAA scores and neuropsychological performance. Therefore, we conducted an exploratory hierarchical regression, entering the interaction between education and cognitive activity in adulthood (EdCA) as the final step. This model tested whether the interaction between education and cognitive activity in adulthood accounts for significant variance in neuropsychological performance after accounting for the main effects of education and self-reported cognitive activity in adulthood (i.e. Step 1: Education; Step 2: Cognitive activity in adulthood; Step 3: EdCA).

The results of the exploratory regression analyses indicated that after controlling for main effects, EdCA was significantly associated with performance on TMT – B, ($R^2\Delta$ = .08, F (1, 60) = 5.0, p = .03), COWA ($R^2\Delta$ = .06, F (1, 62) = 3.99, p = .05), and visuospatial ability ($R^2\Delta$ = .11, F (1, 62) = 8.52, p = .005). The interaction term did *not*

account for an additional amount of variance, after accounting for main effects, in immediate memory ($R^2\Delta = .006$, $F (1, 62) = .39$, $p = .53$), delayed memory ($R^2\Delta = .001$, $F (1, 60) = .05$, $p = .83$), or general cognitive function ($R^2\Delta = .02$, $F (1, 59) = 1.26$, $p = .27$).

To further investigate the nature of the significant interaction between education and self-reported cognitive activity in adulthood, we divided the data set into three categories according to years of education completed. Individuals with 10 – 12 years of education (10 was the minimum number of years in the sample) were placed in the first category. Individuals with 13 – 16 years of education were placed in the second category. Individuals with 17 – 20 years of education were placed in the third category. Next, we ran three sets of Pearson product-moment correlations that examined the relationship between cognitive activity in adulthood (i.e. cognitive activity – adult = the sum of responses to LCAA items for age 40 and present) and neuropsychological performance for each educational category (i.e. 1, 2, or 3). The results of the Pearson product moment correlations for the three categories of education are presented in Table 9.

The results indicated that in the group with the lowest level of education (i.e. 10 – 12 years, n = 18), more self-reported cognitive activity in adulthood was associated with *worse* performance on visuospatial tasks and on the TMT-B (i.e. a speeded task that measures mental flexibility and working memory). However, in the group with the lowest level of education, self-reported cognitive activity in adulthood was not associated with performance on measures of immediate or delayed memory (Immediate and

Delayed Memory Indices), letter fluency (i.e. COWA), or general cognitive function (i.e. RBANS Composite Index) In the group with a moderate level of education (i.e. 13 – 16 years, n = 21), self-reported cognitive activity in adulthood was not associated with performance on any neuropsychological measures. In the group with the highest level of education (i.e. 17 – 20 years, n = 18), more self-reported cognitive activity in adulthood was associated with *better* performance on TMT-B, but was not associated with performance on any other neuropsychological measures. Interpretation of the analyses is problematic, however, due to small sample size. Therefore, these results are merely exploratory.

Table 9

Pearson Product-moment Correlations between Cognitive Activity in Adulthood and Neuropsychological Performance by Education Level

	Education		
Neuropsychological Measure	10 – 12 years (N = 18)	13 – 16 years (N = 21)	17 – 20 years (N = 25 – 27)
Immediate Memory Index	- .02	.25	-.10
Delayed Memory Index	-.05	.27	.07
Visuospatial Index	-.66**	-.12	.36
RBANS Composite Index	-.41	.04	.26
COWA	-.30	-.15	.32
TMT – B	.49 * [A]	.09	-.42*

Note. * $p < .05$, ** $p < .01$. RBANS = Repeatable Battery for the Assessment of Neuropsychological Status, TMT-B = Trail-Making Test – Part B, COWA = Controlled Oral Word Association, [A] TMT – B and Cognitive Activity n = 17.

Discussion

The present study examined markers of cognitive reserve (i.e. self-reported cognitive activity, education, and IQ) in an effort to discover which factor or factors were most related to neuropsychological performance, while attempting to address limitations in the cognitive reserve and cognitive activity literature. In so doing, the present study also examined the Lifetime Cognitive Activity Assessment (Wilson, Barnes, & Bennett, 2003). Not surprisingly, we found that more education was related to better vocabulary. This is consistent with prior studies and reviews (Neisser et al., 1996; Richards & Sacker, 2003; Verhaeghen, 2003). In general, however, our first hypothesis predicting that lifestyle and intellect markers of cognitive reserve would be significantly related *was not* fully supported. First, education was not related to the amount of self-reported cognitive activity at any age in this study. This finding is inconsistent with prior research demonstrating a positive relationship between education and LCAA scores (Wilson, Barnes, & Bennett, 2003, Wilson & Bennett, 2003; Wilson, Mendes de Leon et al., 2002) and other questionnaires tapping self-report of cognitive activity, such as the Florida Cognitive Activities Scale (Schinka et al., 2005). However, other studies have also failed to find a significant relationship between education and self-reported activity (e.g., Scarmeas, et al., 2003). Further, while vocabulary was related to self-reported cognitive activity at age 40 and beyond; it *was not* related to reports of activity during childhood and adolescence in this study. This finding is consistent with Wilson et al.'s recent study

that found *current* and recent activity was more related to neuropsychological performance than childhood or midlife activity (2005).

There are several possible explanations for the lack of significant correlations found between self-reported cognitive activity and intellect markers of cognitive reserve. One reason may be that individuals are inaccurately reporting their cognitive activity. This is particularly a concern with the childhood and adolescence section of the LCAA. Indeed, as Whitley (2002) suggests, retrospective self-report measures are traditionally unreliable. Another possible explanation for the lack of significant results may be due to statistical issues related to the bimodal distribution of education in the present study. Further, the lack of participants with very low education (i.e. less than 10 years of school) may have upheld a floor effect that has also been a weakness in other studies (e.g., Wilson, Barnes, & Bennett, 2003, Wilson & Bennett, 2003; Wilson, Mendes de Leon et al., 2002). Additionally, studies that have previously reported an association between education and self-reported cognitive activity (Wilson, Barnes, & Bennett, 2003, Wilson & Bennett, 2003; Wilson, Mendes de Leon et al., 2002) have had very large samples (i.e. > 400 – 1000). It is possible that although these studies found a statistically significant association; it was not a *clinically meaningful* association. As Wilson and colleagues have not commented on the clinical significance of their findings, one is left to speculate on the meaningfulness of the association between self-reported cognitive activity and cognitive performance. Statistical significance as an artifact related to the large sample size may explain the inability of the present study to replicate Wilson and colleagues' studies in general. According to *Sample Power*, the Pearson correlations found in the

present study between most of the LCAA epochs and vocabulary or education were so small (e.g., r = .08, .05, .14, .13, .16, etc.) that a sample of at least 301 - 1221 would be needed to find a significant result for power = .80, alpha = .05.

Another possible explanation for the lack of significant results in this study may be related to the instrument used to assess cognitive activity. As discussed previously, the Lifetime Cognitive Activity Assessment (LCAA) questionnaire, which is the measure used by Wilson and colleagues to assess cognitive activity, is problematic for several reasons. As a self-report measure, it is vulnerable to response biases such as social desirability and experiment demand characteristics (Whitley, 2002). An additional problem with the measure is that it is *retrospective*, requiring individuals to report on activities that occurred during childhood, adolescence, and young adulthood. As mentioned above, retrospective reporting has often been shown to be inaccurate (Whitley, 2002). Another potential problem of the LCAA is that because the measure inquires about activities at different age epochs, it is unclear which epoch truly represents the "use-it-or-lose-it" argument. Another problem with the measure is that cognitive activity is affected by many factors including mood, physical health, and ability. For example, an individual's current activity may be an indication of brain disease instead of a predictive factor for it. In other words, individuals may engage in less cognitive activity in the years immediately prior to dementia diagnosis due to the onset of progressive disease. A final problem with the LCAA, as Wilson and Bennett have acknowledged, the psychometrics of the questionnaire are not well established. Wilson et al.'s (2002, 2003) conclusions based on the LCAA have not yet been replicated in a longitudinal study.

We particularly question the *construct validity* of the LCAA measure. As we have discussed previously, there are so many problems inherent in the design of the LCAA, that it is not clear that high scores on the LCAA reflect high levels of actual activity (e.g., retrospective, self-report). When discussing ecological validity, it is not known how LCAA scores compare to behavioral or verifiable measures of activity (e.g. number of hobbies, number of club memberships) or to functional measures of activity such as evaluation of instrumental activities of daily living. Lastly, it could be that education is truly not related to self-reported cognitive activity. Additional studies that incorporate multiple raters and multiple methods for assessing cognitive activity will help clarify this relationship.

Our hypothesis that vocabulary and education would be related to neuropsychological performance had mixed results. In general, better vocabulary and more education were related to better performance on neuropsychological tests, except for visuospatial tasks and a speeded task of mental flexibility and working memory (i.e. TMT – B). This finding is not surprising, given the fact that vocabulary is a verbal task and the TMT –B and visuospatial index have very low to insignificant verbal loading. Further, 83% of our sample had at least some college, while 40% attended graduate school. In general, our findings were consistent with the literature, which shows that vocabulary and education are related to cognitive ability (e.g. Neisser et al., 1996; Richards & Sacker, 2003; Scarmeas et al., 2003; Staff et al., 2004; Wilson, Barnes, & Bennett, 2003)

Our hypothesis that the LCAA scores would be related to performance on neuropsychological tests was generally not supported. Contrary to prior research (e.g., Wilson et al., 2005; Wilson, Barnes, & Bennett, 2003, Wilson & Bennett, 2003; Wilson, Mendes de Leon et al., 2002), no part or composite of the LCAA was significantly related to any neuropsychological measure in the present study. A closer examination of the zero-order correlations showed generally low *r* values coupled with p-values that did not even approach significance. In fact, the largest association was between self-reported cognitive activity at age 40 and performance on the TMT-B (n = 66, r = .20, p = .12), which is in the opposite direction of the hypothesis (i.e., higher self-reported cognitive activity is related to slower performance on this task). Most of the other Pearson correlations ranged between r = -.001 and .18. Not only were the Pearson correlations very small, many were in the wrong direction. According to *Sample Power 2.0*, a sample of 237 participants would be required to find a significant result at that level of effect (r = .18), with power = .80 and alpha = .05. Because we did not find an association between LCAA and neuropsychological tests as we expected, exploratory Pearson product-moment correlations were conducted. Potential explanations for this lack of significant results will be discussed below, as part of the discussion of the exploratory analysis.

Consistent with the literature, we found that self-reported ratings of memory function, depression, and memory were significantly related to one another. The hypothesis that general intellect factors would not be related to self-report measures had mixed results. While more education was associated with less depression and stress, vocabulary score was not related to any self-report ratings. One explanation may be that

individuals with more education have a better quality of life than individuals with lower levels of education. This finding is particularly interesting because 83% of the sample attended some college. In other words, one would not expect such an education effect among such highly educated individuals.

In general, results did not confirm our hypothesis that self-report instruments would be significantly associated with LCAA in the present study. This finding is inconsistent with research showing that self-reported depression is related to performance on cognitive tests, as well as other measures of self-report. However, the results support the specificity of the LCAA measure as not reflecting mood state or perceived distress, adding some evidence for the discriminant validity of the measure.

We hypothesized that after accounting for the effect of general intellect, self-reported cognitive activity in adulthood would still account for a significant amount of the variation in neuropsychological performance. Contrary to our hypothesis and previous studies (Wilson, et al., 2005; Wilson, Barnes, & Bennett, 2003; Wilson & Bennett, 2003; Wilson, Mendes de Leon et al., 2002), we found that self-reported cognitive activity in adulthood was generally not related to test performance after controlling for the effect of education or vocabulary. Indeed, Pearson product-moment correlations did not reveal a significant association between *any* performance on any neuropsychological test and any epoch of the LCAA. However, the result of our hierarchical regression analyses revealed that self-reported cognitive activity in adulthood, which is a composite score designed and used by and for this study, explained significant additional variance in RBANS composite score, after controlling for the effect

of vocabulary score. This finding is consistent with the findings of Wilson et al. (2005), who found self-reported *current* cognitive activity is more predictive of better cognitive function than is the report of early life activity. It is notable that the Wilson studies discussed above, that found that LCAA scores accounted for significant variance in test scores of multiple psychological domains (e.g., episodic memory, semantic memory, working memory, perceptual speed, and visuospatial ability) even after accounting for education or IQ, used the NART as the estimate of IQ. At least one study (Russell et al., 2000) found that the NART was not as good of an estimate of "premorbid" intellectual ability when compared to the Vocabulary subtest from the WAIS-R. This finding was particularly true when IQ was not in the average range, at both the upper and lower ends of the range of education. Further, Russell et al. (2000) found that the NART overestimated premorbid Full Scale IQ by an average of 15 points (p. 303).

Although the Russell et al. study was small (n = 24) and from a clinical population, its findings are consistent with other studies that have found that the use of NART was less accurate at predicting IQ at the high and low ranges of education (Nelson & Willison, 1991; Wiens, Bryan, & Crossen, 1993). Therefore, perhaps the fact that Wilson and colleagues found that LCAA score accounted for significant variation in cognitive function despite "intellect" or "IQ" reflected the use of a poor IQ measure instead of reflecting the actual relation between IQ and cognitive function. This explanation does not, however, address the fact that the present study did not replicate Wilson and colleagues' multiple and consistent finding that LCAA scores are associated with performance on neuropsychological tests even accounting for education. There are

at least two possible explanations for this. First, it is possible that the present study revealed the true association between LCAA score, education, and neuropsychological performance. Second, it is possible that the very large samples used in the Wilson and colleagues' studies artificially inflated the clinical significance of the partial correlation between self-reported cognitive activity in adulthood and neuropsychological performance, while controlling for education.

The exploratory analyses continued to reveal unexpected findings. When we examined the relationship between neuropsychological functioning and self-reported cognitive activity in adulthood at three levels of education, we did not find support for Wilson and colleagues' findings. Pearson correlations ranged from -.02 to -.66 in the group with 12 or less years of education, from .09 to .27 in the group with some college, and from .07 to -.42 in the group with post-bachelor and graduate training. Not only did the associations vary greatly in magnitude, but the correlations also varied greatly and unexplainably in direction, sometimes within the same measure across educational groups (i.e. relation of self-reported cognitive activity to TMT – B performance in persons with lower education $r = .49$, $p < .05$, with moderate education $r = .09$, $p = .69$, with higher education $r = -.42$, $p < .05$). Caution is required when interpreting the exploratory findings as the samples were small (n = 18 – 27). However, the inconsistent pattern of association across different levels of education, suggests the possibility of an interaction effect among education and self-reported cognitive activity that could be addressed in future studies.

Weaknesses and Strengths of the Present Study

Potential weaknesses of the present study are related to the cross-sectional design, the educational and cultural composition of the sample, and sample size. First, due to the cross-sectional design, it is possible that performance on the self-report measures (including the LCAA) and neuropsychological tests was influenced by very sub-clinical cognitive decline, thus confounding the results. Second, the sample was entirely comprised of Caucasian, non-Hispanic individuals, which creates an issue of the generalizability of the results of the present study to individuals of diverse ethnic and racial backgrounds. Third, despite attempts to recruit individuals with very low levels of education for the present study, the sample does not represent individuals with less than 10 years of education. Therefore, one could make the argument that the present study did not fully examine the relationship between education and self-reported cognitive activity. Fourth, the size of the sample (n = 66) did not have adequate power to detect very low correlations among variables. However, the findings of the Pearson product-moment correlations (i.e. p values approaching one and correlation coefficients nearing zero) indicated that 100 additional participants would not be enough to detect the correlations between LCAA and neuropsychological test scores. A final criticism of many studies investigating cognitive reserve, including the present study, is survival and exclusion bias. In general, older adults included in research studies are likely to be healthier than adults excluded or simply not recruited for studies. This results in an unavoidable bias toward including adults who have presumably aged "better" than other adults and thus have better cognitive function.

There are many strengths of the present study including the use of vocabulary score to estimate IQ, the similarity of the present sample to the Wilson, Bennett, et al. (2002) and the Wilson, Barnes, and Bennett (2003) samples, the examination of the LCAA questionnaire, and the inclusion of adults from an under-represented geographic area (i.e. rural Appalachia). First, the present study used vocabulary score instead of education or the NART to estimate IQ. Multiple studies have shown that neither education nor the NART are as accurate an estimate of IQ as vocabulary score, especially at the lower and higher ranges of intelligence (Nelson & Willison, 1991; Rentz et al., 2004; Richards & Sacker, 2003, Staff et al., 2004; and Weins, Bryan, and Crossen, 1993). Second, the present sample was similar to the racial, age, and educational composition of the samples in the Wilson, Bennett, et al. (2002) study (i.e. Mean education = 18 years, race > 94% Caucasian) and the Wilson, Barnes, and Bennett (2003) study (i.e. 95% White, non-Hispanic, Mean education = 14.7, *SD* = 3.0). Thus, although the present study lacked diversity in general, it did reflect the composition of the Wilson samples that found that LCAA scores predicted cognitive performance and Alzheimer's disease risk. Third, the present study is only the second study to examine each epoch of the LCAA measure and analyze each epoch's relation to neuropsychological performance. Finally, the present sample is comprised of individuals from a rural, Appalachian community, a region and culture which are under-represented in the psychological literature.

Implications and Future Directions

In general, we were unable to replicate Wilson and colleagues (e.g., Wilson, Barnes, and Bennett, 2003; Wilson et al, 2005; Wilson, Bennett, et al., 2002, and Wilson, Mendes de Leon, et al., 2002) findings that LCAA is associated with cognitive function, in our rural sample of healthy older adults. In general, we found that a participant's response on the LCAA was not related to his or her performance on most cognitive tests, except for a measure of global cognitive function. Our sample, though smaller than the Wilson, Mendes de Leon, and colleagues (2002) study, had adequate power to detect the small to moderate effects previously described for the cognitive domains examined in the present study. Further, the lack of significant findings cannot be attributed to differences in the educational composition of the sample in the present study versus the educational composition of the samples in the Wilson, Mendes de Leon, et al. (2002), and Wilson, Barnes, and Bennett (2003) studies, as it was generally similar.

In the present study the best non-neuropsychological indicator of neuropsychological performance was vocabulary score. As a marker of cognitive reserve, vocabulary score explained more of the variance in cognitive performance than did the "report" of anther proposed marker of reserve (i.e. cognitive activity). As noted in the introduction, although verbal reasoning and comprehension (e.g., as measured on the Verbal Comprehension Index on the WAIS-III) are strongly related to performance on composite measures of cognition, as well as neuropsychological tests with high verbal loading; verbal reasoning and comprehension tests are weakly associated with other neuropsychological tests (e.g., tests of processing speed). Therefore, the strong

association between vocabulary score and cognitive performance in the present study may not be attributed solely to the fact that the vocabulary subtest is a simply another neuropsychological test. Further, the findings of the present study lend support for the general intellect argument of the cognitive reserve debate, as vocabulary score has been shown to be a reliable estimate of intellect for individuals at all ranges of intelligence and education. At the very least, the results of our study fail to support the lifestyle argument, in that we did not find a correlation between self-reported activity and neuropsychological function.

The question for the hundreds of thousands of older adults interested in strategies that may stave off the onset of dementia is: Can I positively impact the fitness of my brain with mental exercise? As noted above, the present study does not provide evidence for, nor does it provide evidence against, the protective effect of cognitive activity. Fear of Alzheimer's disease and feelings of helplessness over one's cognitive decline remains high among older adults (Kleinfield, 2002). Evidence of the protective effects of mental exercise has been touted in the mainstream media as an effective tool against brain decline and Alzheimer's disease in particular. The Alzheimer's Association urges older adults to "Maintain Your Brain" as a primary method to protect against cognitive decline (in addition to reduced stress, heart healthy diet, social/emotional exercise, etc.) and offers several specific ways to do so. For example, the Alzheimer's Association advises individuals to: "stay curious and involved; commit to lifelong learning; read; write; work crossword or other puzzles; attend lectures and plays; enroll in courses at your local adult education center; community college or other community group; play games; garden; and

try memory exercises" (http://www.alz.org/maintainyourbrain/mactive.asp, accessed May 30, 2006 at 11:21 pm). The AARP website displays similar information about prevention strategies and recommends various games and activities to "Engage Your Brain". The AARP even makes mention of "neurobics", which is a play on the word *aerobic*, to indicate exercise for your brain. Specifically, the AARP website features information on crossword puzzles and Sudoku, among other mentally challenging leisure activities (http://www.aarp.org/fun/puzzles/, accessed May 30, 2006 at 11:36 p.m.).

Given prior evidence that a better intellect confers a protective effect against cognitive decline and disease, perhaps a more pertinent question is whether or not individuals with average or lower than average intellect are able to "make up" for the protective benefits their brains do not receive as a result of greater intellect, by engaging in mental exercise. The present study is unable to answer this question as the LCAA measure used to estimate cognitive activity demonstrated questionable validity in the present study.

In future studies, the problems associated with a self-report measure may be countered by the design and use of a collateral reporter version of the LCAA. It may not be enough to rely on self-report of past activity. Future research should incorporate verifiable evidence of activity, as well as collateral raters (e.g. spouse, children) on the LCAA to counter the potential effects of social desirability biases in participant reporting. Also, Wilson and colleagues' findings need to be replicated in a longitudinal study, with a period of several years between assessments. If possible, participants who have been

administered intellectual tests should be followed up and re-evaluated while also completing multiple activity questionnaires.

References

American Psychiatric Association. (2000). *Diagnostic and Statistical Manual of Mental Disorders*. (4th ed., Text Revision). Washington, DC: American Psychiatric Association.

Ball, K., Berch, D. B., Helmers, K. F., Jobe, J. B., Leveck, M. D., Marsiske, M., Morris, J. N., Rebok, G. W., Smith, D. M., Tennstedt, S. L., Unverzagt, F. W., & Willis, S. L. (2002). Effects of cognitive training interventions with older adults: A randomized controlled trial. *Journal of the American Medical Association, 288*(18), 2271-2281.

Benton, A. L., Hamsher, K. de S., & Sivan, A. B. (1994). *Multilingual Aphasia Examination* (3rd ed.). Iowa City, IA: AJA Associates.

Blessed, G., Tomllinson, B. E., & Roth, M. (1968). The association between quantitative measures of dementia and of senile change in the cerebral grey matter of elderly subjects. *British Journal of Psychiatry, 114*, 797-811.

Brookmeyer, R., Gray, S., & Kawas, C. (1998). Projections of Alzheimer's disease in the United States and the public health impact of delaying disease onset. *American Journal of Public Health, 88*, 1337-1342.

Capurso, A., Panza, F., Solfrizzi, V., Capurso, C., Mastroianni, F., & Del Parigi, A. (2000). Age-related cognitive decline: Evaluation and prevention strategy. *Recenti Progressi in Medicina, 91*(3), 127-134.

Cassel, C. K. (2002). Use it or lose it: Activity may be the best treatment for aging. *Journal of the American Medical Association, 288*(18), 2333-2335.

Cockburn, J. , Smith, P. T., & Wade, D. T. (1990). Influence of cognitive function on social, domestic, and leisure activities of community-dwelling older people. *International Disabilities Studies, 12*(4), 169-172.

Cohen, S., Kamarck, T., Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior, 24*, 386-396.

Cummings, J. L., & Cole, G. (2002). Alzheimer's disease. *Journal of the American Medical Association, 287*, 2335- 2338.

Cummings, J. L., Vinters, H. V., Cole, G. M., & Khachaturian, Z. S. (1998). Alzheimer's Disease: Etiologies, pathophysiology, cognitive reserve, and treatment opportunities. *Neurology, 51*(Suppl 1: S2-17, discussion S65-7.).

Department of Education & Science (1972). *Burnham further education committee grading courses.* London: HMSO.

Derouesné, C., Lacomblez, L., Thibault, S., & Leponcin, M. (1999). Memory complaints in young and elderly subjects. *International Journal of Geriatric Psychiatry,14*, 291- 301.

Evans, D. A., Funkelstein, H. H., Albert, M. S., Scherr, P. A., Cook, N. R., Chown, M. J., Hebert, L. E., Hennekens, C. H., & Taylor, J. O. (1989). Prevalence of Alzheimer's disease in a community population of older persons. Higher than previously reported. *Journal of the American Medical Association, 262*, 2551-2556.

Fratiglioni, L., Paillard-Borg, S., & Winblad, B. (2004). An active and socially integrated lifestyle in late life might protect against dementia. *Lancet Neurology, 3*(6), 343-353.

Fabrigoule, C., Rouch, I., Taberly, A., Letenneur, L., Commenges, D., Mazaux, J. M., Orgogozo, J. M., & Dartigues, J. F. (1998). Cognitive Processes in preclinical phase of dementia. *Brain, 121*, 135-141.

Gould, E., Beylin, A., Tanapat, P., Reeves, A., & Shors, T. J. (1999). Learning enhances adult neurogenesis in the hippocampal formation. *Nature Neuroscience, 2*(3), 260-265.

Grady, C. L., Horwitz, B., Pietrini, P., Mentis, M., Ungerleiter, L., Rapoport, S. I., & Haxby, J. (1996). The effect of task difficulty on cerebral blood flow during perceptual matching of faces. *Human Brain Mapping, 4*, 227-239.

Hebert, L. E., Scherr, P. A., Bienias, J. L., Bennett, D. A., & Evans, D. A. (2003). Alzheimer Disease in the U.S. Population: Prevalence Estimates Using the 2000 Census. *Archives of Neurology, 60*(8), 1119-1122.

Horn, J. L. & Cattell, R. B. (1967). Age differences in fluid and crystallized intelligence. *Acta Psychologica, 26*, 107-129.

Jacobs, D. M., Sano, M., Dooneief, G., Marder, K., Bell, K. L., & Stern, Y. (1995). Neuropsychological detection and characterization of preclinical Alzheimer's disease. *Neurology, 45*, 957- 962.

Jankowsky, J. L., Melnikova, T., Fadale, D. J., Xu, G. M., Slunt, H. H., Gonzales, V., Younkin, L. J., Younkin, S. G., Borchelt, D. R., & Savonenko, A. V. (2005).

Environmental enrichment mitigates cognitive deficits in a mouse model of Alzheimer's disease. *The Journal of Neuroscience, 25*(21), 5217-5224.

Katzman, R. (1993). Education and the prevalence of dementia and Alzheimer's disease. *Neurology, 43*, 13-20.

Katzman, R., Aronson, M., Fuld, P., Kawas, C., Brown, T., Morgenstern, H., Frishman, W., Gidez, L., Eder, H., & Ooi, W. L. (1989). Development of dementing illnesses in an 80-year-old volunteer cohort. *Annals of Neurology, 25*, 317-324.

Kaufman, A. L., & Lichtenberger, E. O. (1999). *Essentials of WAIS-III Assessment.* New York: John Wiley & Sons, Inc.

Kempermann, G., Gast, D., & Gage, F. H. (2002). Neuroplasticity in old age: sustained fivefold induction of hippocampal neurogenesis by long-term environmental enrichment. *Annals of Neurology, 52*(2), 135-143.

Kleinfield, N.R. (2002, November 11). More than death, fearing a muddled mind. *New York Times* [Electronic version].

Knopman, D. & Selnes, O. (2003). Neuropsychology of dementia. In K. M. Heilman & E. Valenstein (Eds.), *Clinical Neuropsychology* (pp. 574-616). New York: Oxford University Press.

Lachman, M. E., Bandura, M., Weaver, S. L., & Elliot, E. (1995). Assessing memory control beliefs: The memory controllability inventory. *Aging & Cognition, 2*, 67-84.

Le Carret, N., Lafont, S., Mayo, W., & Fabrigoule, C. (2003). The effect of education on cognitive performances and its implication for the constitution of the cognitive reserve. *Developmental Neuropsychology, 23*(3), 317-337.

Lee, J. H. (2003). Genetic evidence for cognitive reserve: Variations in memory and related cognitive functions. *Journal of Clinical and Experimental Neuropsychology, 25*(5), 594-613.

Levy-Cushman, J., & Abeles, N. (1998). Memory complaints in the able elderly. *Clinical Gerontologist, 19*, 3-24.

Lindsay, J., Laurin, D., Verreault, R., Hébert, R., Helliwell, B., Hill, G. B., & McDowell, I. (2002). Risk factors for Alzheimer's disease: A prospective analysis from the Canadian study of health and aging. *American Journal of Epidemiology, 156* (5), 445-453.

McGue, M., Bouchard, T. J., Jr., Iacono, W. G. & Lykken, D. T. (1993). Behavioral genetics of cognitive ability: A lifespan perspective. In R. Plomin & G. E. McClearn (Eds.). *Nature, nurture, & psychology.* (pp. 59-76). Washington, DC: American Psychological Association.

McClearn, G. E., Johansson, B., Berg, S., Pedersen, N. L., Ahern, F., Petrill, S. A., & Plomin, R. (1997). Substantial genetic influence on cognitive abilities in twins 80 or more years old. *Science, 276,* 1560-1563.

Mortimer, J. A., Schuman, L., & French, L. (1981). Epidemiology of dementing illness. In J. A. Mortimer & L. Schuman (Eds.), *The epidemiology of dementia:*

Monographs in epidemiology and biostatistics (pp. 323-333). New York: Oxford University Press.

Mortimer, J. A., Snowdon, D. A., & Markesbery, W. R. (2003). Head circumference, education and risk of Dementia: Findings from the Nun Study. *Journal of Clinical and Experimental Neuropsychology, 25*(5), 671-679.

Neisser, U., Boodoo, G., Bouchard, T. J., Jr, Boykin, A. W., Brody, N., Ceci, S. J., Halpern, D., Loehlin, J. C., Perloff, R., Sternberg, R. J., & Urbina, S. (1996). Intelligence: Knowns and unknowns. *Amercian Psychologist, 51*(2), 77-101.

Nelson, H. E. (1982). *The National Adult Reading Test (NART)*. Windsor, England: NFER.

Nelson, H. E., & Willison (1991). *The National Adult Reading Test (NART)*. Windsor, England: NFER

Podewils, L. J., Guallar, E., Kuller, L. H., Fried, L. P., Lopez, O. L., Carlson, M., & Lyketsos, C. G. (2005). Physical activity, APOE genotype, and dementia risk: Findings from the Cardiovascular Health Cognition Study. American Journal of Epidemiology, *161*(7), 639 - 651.

Pigeon, D. A. (1964). Tests used in the 1954 and 1957 surveys. In J. B. W. Douglas (Ed.), *The home and the school.* London: Macgibbon & Key, Appendix I.

Ponds, R. W. H. M., Van Boxtel, M. P. J., & Jolles, J. (2000). Age-related changes in subjective cognitive functioning. *Educational Gerontology, 26,* 67-83.

Randolph, C. (1994). *Repeatable Battery for the Assessment of Neuropsychological Status*: The Psychological Corporation.

Raven, J. C. (1960). *Guide to the standard progressive matrices*. London: H. K. Lewis.

Reitan, R. (1958). Validity of the Trail Making Test as an indicator. of organic brain damage. *Perceptual and Motor Skills*, *8*, 271–276.

Remor, E. A., & Carrobles, J. A. (2001). The Perceived Stress Scale: Psychometric study with Spanish HIV+ sample. *22nd International Conference STAR,* 12-14.

Rentz, D. M., Huh, T. J., Faust, R. R., Budson, A. E., Scinto, L. F. M., Sperling, R. A., & Daffner, K. R. (2004). Use of IQ-adjusted norms to predict progressive cognitive decline in highly intelligent older individuals. *Neuropsychology, 18*(1), 38-49.

Rey, A. (1958). *L'examen clinique en psychologie*. Paris: Presses Universitaire de France.

Richards, M. & Sacker, A. (2003). Lifetime antecedents of cognitive reserve. *Journal of Clinical and Experimental Neuropsychology, 25*(5), 614-624.

Roth, M., Tomlinson, B. E., & Blessed, G. (1967). *The relationship between quantitative measures of dementia and of degenerative changes in the cerebral grey matter of elderly subjects.* Paper presented at the Proceedings of the Society of Medicine.

Russell, A. J., Munro, J., Jones, P. B., Hayward, P., Hemsley, D. R., & Murray, R. M. (2000). The National Adult Reading Test as a measure of premorbid IQ in schizophrenia. *British Journal of Clinical Pyschology, 39,* 297-305.

Saari, M. J., Armstrong, J. N., Norbrega, J. N., Pappas, B. A., & Coscina, D. V. (1990). Neonatal 6-hydroxydopamine alters the behavior of enriched-impoverished rats in a novel test environment. *Behavioral Neuroscience, 104,* 430-437.

Salthouse, T. A. (2003). Memory aging from 18 to 80. *Alzheimer's Disease Association, 17,* 162-167.

Sands, L., Terry, H., & Meredith, W. (1989). Change and stability in adult intellectual functioning assessed by Wechsler Item responses. *Psychology and Aging, 4(1),* 79-87.

Satz, P. (1993). Brain reserve capacity on symptom onset after brain injury: A formulation and review of evidence for threshold theory. *Neuropsychology, 7*(3), 273-295.

Scarmeas, N., & Stern, Y. (2003). Cognitive reserve and lifestyle. *Journal of Clinical and Experimental Neuropsychology, 25*(5), 625-633.

Scarmeas, N., Zarahn, E., Anderson, K. E., Habeck, C. G., Hilton, J., Flynn, J., Marder, K. S., Bell, K. L., Sackeim, H. A., Van Heertum, R. L., Moeller, J. A., & Stern, Y. (2003). Association of life activities with cerebral blood flow in Alzheimer's Disease: Implications for the Cognitive Reserve Hypothesis. *Archives of Neurology, 60*, 359-365.

Schaie, K. W. (2002). Intellectual Development in Adulthood. In J. E. Birren & K.W. Schaie (Eds.), *Handbook of the psychology of aging* (pp. 266 – 286). New York: Academic Press.

Schinka, J. A., McBride, A., Vanderploeg, R. D., Tennyson, K., Borenstein, A. R., & Mortimer, J. A. (2005). Florida cognitive activities scale: Initial development and validation. *Journal of the International Neuropsychological Society, 11,* 108-116.

Sivan, A. B. (1992). *Benton Visual Retention Test* (5th ed.). San Antonio: The Psychological Corporation.

Spearman, C. (1927). *The abilities of man.* New York: Macmillan.

Spreen, O., & Strauss, E. (1998). *A compendium of neuropsychological tests: Administration, norms, and commentary* (2nd ed.). New York: Oxford University Press.

Staff, R. T., Murray, A. D., Deary, I. J., & Whalley, L. J. (2004). What provides cerebral reserve? *Brain, 127,* 1191-1199.

Stern, Y. (2002). What is cognitive reserve? Theory and research application of the reserve concept. *Journal of the International Neuropsychological Society, 8*, 448-460.

Stern, Y. (2003). The concept of cognitive reserve: A catalyst for research. *Journal of Clinical and Experimental Neuropsychology, 25*(5), 589-593.

Stern, Y., Gurland, B., Tatemaichi, T. K., Tang, M. X., Wilder, D., & Mayeux, R. (1994). Influence of education and on the incidence of Alzheimer's disease. *Journal of the American Medical Association, 271*(13), 1004-1010.

Stern, Y., Zarahn, E., Hilton, H. J., Flynn, J., DeLaPaz, R., & Rakitin, B. (2003). Exploring the neural basis of cognitive reserve. *Journal of Clinical and Experimental Neuropsychology, 25*(5), 691-701.

Swan, G. E., & Carmelli, D. (2002). Evidence for genetic mediation of executive control: A study of aging male twins. *Journal of Gerontology Series B – Psychological Sciences and Social Sciences, 57,* P133-P143.

van Praag, H., Kempermann, G., & Gage, F. H. (2000). Neural consequences of environmental enrichment. *National Review of Neuroscience, 1,* 191-198.

Verhaeghen, P. (2003). Aging and vocabulary scores: A meta-analysis. *Psychology and Aging, 18*(2)*,* 332-339.

Wechsler, D. (1997). *Wechsler Adult Intelligence Scale-III*. New York: The Psychological Corporation.

Whitley, B. E. (2002). *Principles of Research in Behavioral Science* (2nd ed.). New York: McGraw-Hill.

Weins, A. N., Bryan, J. E., & Crossen, J. R. (1993). Estimating WAIS-R FSIQ from the NART-R in normal subjects. *The Clinical Neuropsychologist, 7,* 70-84.

Wilson, R. S., Barnes, L. L., & Bennett, D. A. (2003). Assessment of lifetime participation in cognitively stimulating activities. *Journal of Clinical and Experimental Neuropsychology, 25*(5), 634-642.

Wilson, R. S., Barnes, L. L., Krueger, K. R., Hoganson, G., Bienias, J. L., & Bennett, D. A. (2005). Early and late life cognitive activity and cognitive systems in old age. *Journal of the International Neuropsychological Society, 11,* 400-407.

Wilson, R. S. & Bennett, D. A. (2003). Cognitive Activity and Risk of Alzheimer's Disease. *Psychological Science, 12*(3), 87-91.

Wilson, R. S., Bennett, D. A., Beckett, L. A., Morris, M. C., Gilley, D. W., Bienias, J. L., Scherr, P. A., & Evans, D. A. (1999). Cognitive activity in older persons from a geographically defined population. *Journal of Gerontology: Psychological Sciences, 54B,* P155-P160.

Wilson, R. S., & Bennett, D. A, Bienias, J. L., Aggarwal, N. T., Mendes de Leon, C. F., Morris, M. C., Schneider, J. A., & Evans, D. A. (2002). Cognitive activity and incident AD in a population-based sample of older persons. *Neurology, 59,* 1910-1915.

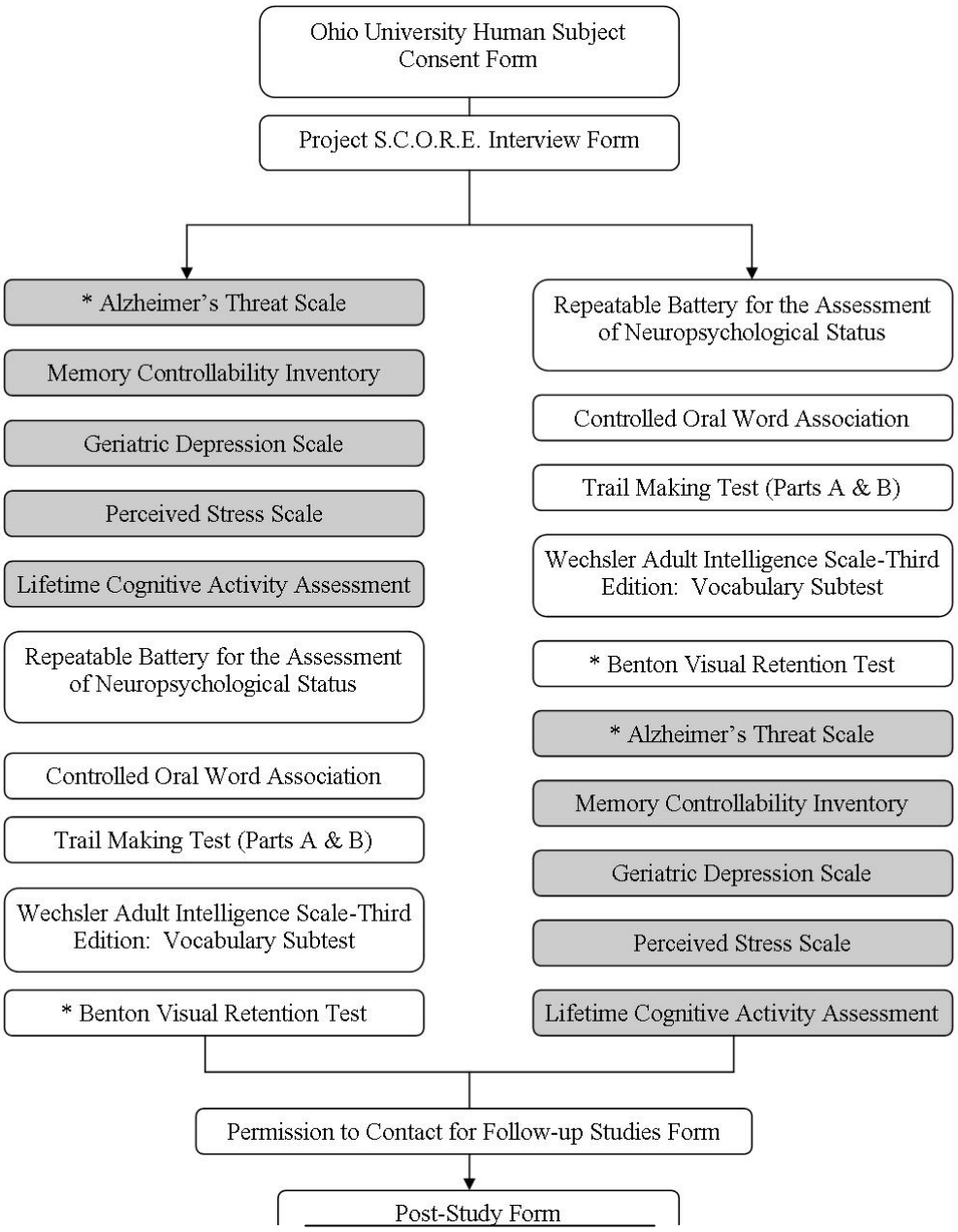
Wilson, R. S., Bennett, D. A., Gilley, D. W., Beckett, L. A., Barnes, L. L., & Evans, D. A. (2000). Premorbid reading activity and patterns of cognitive decline in Alzheimer's disease. *Archives of Neurology, 57*, 1718-1723.

Wilson, R. S., Mendes de Leon, C. F., Barnes, L. L., Schneider, J. A., Bienias, J. L., Evans, D. A., & Bennett, D. A. (2002). Participation in cognitively stimulating activities and risk of incident Alzheimer's Disease. *Journal of the American Medical Association, 287*(6), 742-748.

Yesavage, J. A., Brink, T. L., Rose, T. L., & Adey, M. (1983). The Geriatric Depression Rating Scale: Comparison with other self-report and psychiatric rating scales. In T. Crook, S. Ferris, & R. Bartus (Eds.), *Assessment in Geriatric Psychopharmacology*. New Canaan, CT: Mark Powley Associates.

Zunzunegui, M. V., Alvarado, B. E., Teodoro, D. S., & Otero, A. (2003). Social networks, social integration, and social engagement determine cognitive decline in community dwelling Spanish older adults. *Journal of Gerontology: Social Sciences, 58B*(2), S93-S100.

Appendix A: Overview of Procedures



Note. Shaded boxes are self-report measure. * Indicates measures that were *not* of interest to the present study.

Appendix B: Unpaid Participant Informed Consent

OHIO UNIVERSITY
HUMAN SUBJECT CONSENT FORM I

TITLE OF RESEARCH: Project SCORE NEW WAVE (Screening Cognition in Older Adults with Repeated Evaluations)
PRINCIPAL INVESTIGATOR: Julie Suhr, Ph.D.
DEPARTMENT: Psychology

Federal and university regulations require signed consent for participation in research involving human subjects. After reading the statements below, please indicate your consent by signing this form.

EXPLANATION OF STUDY

Purpose of the research: You are invited to participate in a research study assessing the cognitive performance of persons 50 and above. A person's cognitive performance is how well they do on tests of memory and thinking. In this study, we are specifically interested in how cognitive performance is related to several other factors, including your beliefs about your everyday thinking and memory skills, psychological symptoms you may be experiencing, your beliefs about changes in cognition as we age, and experience with and feelings about cognitive disorders in aging, such as Alzheimer's disease. A secondary purpose of the study is to gather baseline data on the cognitive performance of a large group of healthy older adults, to determine what factors best predict future cognitive decline.

Procedures to be followed: In this study, you will complete several measures of thinking and memory, and you will also be asked to complete several self-report questionnaires. The thinking and memory tests will be administered in a one-on-one format by a trained examiner. Most of them require either an oral response or are paper-and-pencil tasks. The self-report questionnaires that you will complete are also paper-and-pencil tasks, and ask questions about your medical and personal history, your level of current distress (depression, anxiety, general health concerns), ratings of your everyday memory problems, and opinions about events sometimes experienced by older adults.

At the completion of all of the tests, you will be asked to sign a form giving us permission to contact you about any follow-up research studies we might conduct in the future. For example, in order to understand what factors are related to future cognitive decline, we might wish to re-assess individuals who participate in this study in a year or two. We are also planning to explore the relation of stress and stress hormones to cognitive performance, and may wish to invite The permission form does not mean that you are agreeing to participate in future studies, but just that you agree to be contacted about those studies and invited to participate. After you hear about the follow-up project, you can decide whether or not to participate.

Duration of participation: Participation will take approximately 1 to 1½ hours.

RISKS AND DISCOMFORTS

The risks involved in the study are minimal. However, questions about memory, anxiety, and depression may be distressing for some people. No one performs perfectly on the cognitive tasks we administer, and this can create anxiety. Your examiner can answer questions about the tests you are completing if you have any concerns. In addition, you will be able to receive clinical feedback about your performance on the tests by the Study Director, who is a licensed psychologist. Finally, we will be providing you with information on health and counseling/ psychological services in the area, should you have any continued concerns.

BENEFITS

Benefits of the research will primarily be for others, as we hope to gain a better understanding of the relation between reported cognitive difficulties and actual performance, and how that relates to conditions that cause cognitive change during aging. However, you can receive feedback about your performance as described above.

CONFIDENTIALITY OF RECORDS

Records of your participation will be maintained in the locked confidential research files of the Study Director's laboratory at Ohio University. Your data will be identified by a code (your initials plus the last four digits of your social security number, or another combination of numbers and letters of your choosing) rather than your name. We plan to use part of your social security number in the event that you later wish to participate in a follow-up study; this number will be easier for you to remember, but will not violate the confidential nature of the data as no one will be able to identify you on the basis of that number alone. The consent form and the permission to contact for future research studies form (which have your name on them) will be stored separately from the rest of your data. Because you will receive $20 as compensation for your participation today, Ohio University's accounting practices require that we report your name and social security number for payment purposes. No other information about you whatsoever will be shared with university accounting personnel, and no one will know what study you participated in. The only other people who have access to your files are the Study Director and the research assistants involved in the study.

Results of the research may be used for the purposes of teaching, publication in professional journals, or presentation at professional meetings, but your individual identity will not be revealed as a part of these activities.

COMPENSATION

You will not receive any compensation for participation in this project.

CONTACT PERSON

If you have any questions, contact Dr. Julie Suhr, Ph.D., at (740) 593-1091. If you have any questions regarding your rights as a research participant, please contact Jo Ellen Sherow, Director of Research Compliance, Ohio University, (740)593-0664.

I certify that I have read and understand this consent form and agree to participate as a subject in the research described. I agree that known risks to me have been explained to my satisfaction and I understand that no compensation is available from Ohio University and its employees for any injury resulting from my participation in this research. I certify that I am 18 years of age or older. My participation in this research is given voluntarily. I understand that I may discontinue participation at any time without penalty or loss of any benefits to which I may otherwise be entitled. I certify that I have been given a copy of this consent form to take with me.

Signature_____ Date_____

Printed Name_____

Appendix C: Paid Participant Informed Consent

OHIO UNIVERSITY
HUMAN SUBJECT CONSENT FORM II

TITLE OF RESEARCH: Project SCORE NEW WAVE (Screening Cognition in Older Adults with Repeated Evaluations)
PRINCIPAL INVESTIGATOR: Julie Suhr, Ph.D.
DEPARTMENT: Psychology

Federal and university regulations require signed consent for participation in research involving human subjects. After reading the statements below, please indicate your consent by signing this form.

EXPLANATION OF STUDY

Purpose of the research: You are invited to participate in a research study assessing the cognitive performance of persons 50 and above. A person's cognitive performance is how well they do on tests of memory and thinking. In this study, we are specifically interested in how cognitive performance is related to several other factors, including your beliefs about your everyday thinking and memory skills, psychological symptoms you may be experiencing, your beliefs about changes in cognition as we age, and experience with and feelings about cognitive disorders in aging, such as Alzheimer's disease. A secondary purpose of the study is to gather baseline data on the cognitive performance of a large group of healthy older adults, to determine what factors best predict future cognitive decline.

Procedures to be followed: In this study, you will complete several measures of thinking and memory, and you will also be asked to complete several self-report questionnaires. The thinking and memory tests will be administered in a one-on-one format by a trained examiner. Most of them require either an oral response or are paper-and-pencil tasks. The self-report questionnaires that you will complete are also paper-and-pencil tasks, and ask questions about your medical and personal history, your level of current distress (depression, anxiety, general health concerns), ratings of your everyday memory problems, and opinions about events sometimes experienced by older adults.

At the completion of all of the tests, you will be asked to sign a form giving us permission to contact you about any follow-up research studies we might conduct in the future. For example, in order to understand what factors are related to future cognitive decline, we might wish to re-assess individuals who participate in this study in a year or two. We are also planning to explore the relation of stress and stress hormones to cognitive performance, and may wish to invite The permission form does not mean that you are agreeing to participate in future studies, but just that you agree to be contacted about those studies and invited to participate. After you hear about the follow-up project, you can decide whether or not to participate.

Duration of participation: Participation will take approximately 1 to 1½ hours.

RISKS AND DISCOMFORTS

The risks involved in the study are minimal. However, questions about memory, anxiety, and depression may be distressing for some people. No one performs perfectly on the cognitive tasks we administer, and this can create anxiety. Your examiner can answer questions about the tests you are completing if you have any concerns. In addition, you will be able to receive clinical feedback about your performance on the tests by the Study Director, who is a licensed psychologist. Finally, we will be providing you with information on health and counseling/ psychological services in the area, should you have any continued concerns.

BENEFITS

Benefits of the research will primarily be for others, as we hope to gain a better understanding of the relation between reported cognitive difficulties and actual performance, and how that relates to conditions that cause cognitive change during aging. However, you can receive feedback about your performance as described above.

CONFIDENTIALITY OF RECORDS

Records of your participation will be maintained in the locked confidential research files of the Study Director's laboratory at Ohio University. Your data will be identified by a code (your initials plus the last four digits of your social security number, or another combination of numbers and letters of your choosing) rather than your name. We plan to use part of your social security number in the event that you later wish to participate in a follow-up study; this number will be easier for you to remember, but will not violate the confidential nature of the data as no one will be able to identify you on the basis of that number alone. The consent form and the permission to contact for future research studies form (which have your name on them) will be stored separately from the rest of your data. Because you will receive $20 as compensation for your participation today, Ohio University's accounting practices require that we report your name and social security number for payment purposes. No other information about you whatsoever will be shared with university accounting personnel, and no one will know what study you participated in. The only other people who have access to your files are the Study Director and the research assistants involved in the study.
Results of the research may be used for the purposes of teaching, publication in professional journals, or presentation at professional meetings, but your individual identity will not be revealed as a part of these activities.

COMPENSATION

You will be paid $20 for your participation in this research project.

CONTACT PERSON

If you have any questions, contact Dr. Julie Suhr, Ph.D., at (740) 593-1091. If you have any questions regarding your rights as a research participant, please contact Jo Ellen Sherow, Director of Research Compliance, Ohio University, (740)593-0664.

I certify that I have read and understand this consent form and agree to participate as a subject in the research described. I agree that known risks to me have been explained to my satisfaction and I understand that no compensation is available from Ohio University and its employees for any injury resulting from my participation in this research. I certify that I am 18 years of age or older. My participation in this research is given voluntarily. I understand that I may discontinue participation at any time without penalty or loss of any benefits to which I may otherwise be entitled. I certify that I have been given a copy of this consent form to take with me.

*Signature*_____ *Date*_____

*Printed Name*_____

Appendix D: Project S.C.O.R.E. Interview Form

PROJECT SCORE INTERVIEW FORM

1) ID NUMBER:_____ 2) DATE :_____
3) STUDY YEAR:_____

4) Sex: M F 5) Age: _____ 6) Date of birth: _____

7) Reason for participating in the study:
_____
_____

8) Marital Status: Single Married Divorced Divorced/Remarried Widowed

9) How many years of education have you completed? Degree? Major or Area of Concentration?

10) What is/was your occupation (if more than one, please list them and include the approximate duration of each job)?

11) Retired (include approximate date of retirement)?

12) Are you currently involved in any volunteer or community service or church work (If yes, please list and describe the type of activities you perform.)?

13) Do you currently participate in any social or recreational groups such as card playing, book club, exercise class, art class, bingo, etc? Please list.

14) Have you ever been diagnosed with a learning disability? If so, what type (e.g. math, reading, etc)?

15) Have you ever lost consciousness due to a blow to the head or other head injury? Y N
a) If yes, for how long did you lose consciousness?
b) Did you see a doctor?
c) Were you hospitalized?
d) What was your diagnosis, if any?
e) Did you have any form of treatment?

16) Have you ever had any of the following: (If yes, get details including date & type of treatment received, such as hospitalization)
a) Seizures? Y N (details here:
_____)
b) Brain tumor? Y N (details here:
_____)
c) Stroke? Y N (details here:
_____)
d) Heart attack? Y N (details here:
_____)

17) Do you have any other neurological/medical problems? (Please list with approximate date of diagnosis, if known. Write none for none)

18) What current medications do you take? Any vitamins or supplements? (Please list med name, if known. If not known, describe what the medication is supposed to treat – antihypertensive medication, for example. Write none for none.)

19) Do you currently smoke? Y N
a) If yes, how many cigarettes per day on average?
b) How long have you smoked?

20) Are you a prior smoker? Y N
a) If yes, how many years did you smoke?
b) When did you quit?
c) How many cigarettes per day on average did you smoke?

21) Do you currently drink alcohol? Y N
a) If yes, how many drinks do you consume on average per day
or per week _____?

22) Did you previously drink alcohol and now have quit drinking? Y N
a) If yes, when did you quit?

23) Have you ever seen a mental health professional (psychiatrist, psychologist, counselor?) Y N

24) If yes, when did you see a mental health professional (including currently)

25) For what diagnosis(es)?

26) Has a biologically related family member of yours ever been diagnosed with Alzheimer's disease? Y N
a) If yes, specify who (degree of relation is important) _____
b) If yes, specify how close on scale of 1 to 3, 1 being not close, 3 being very close _____
c) If yes, specify how frequently you see that person, 1 being not often 3 being very often _____
Repeat if more than 1 relative:
26d)

26e)

26f)

27) Has a non-biological family member (by marriage) ever been diagnosed with Alzheimer's disease? Y N
a) If yes, specify who _____
b) If yes, specify how close on scale of 1 to 3, 1 being not close, 3 being very close _____
c) If yes, specify how frequent you see that person, 1 being not often 3 being very often _____
Repeat if more than 1 non-biological relative:
27d)

27e)

27f)

28) Has a friend/acquaintance of yours ever been diagnosed with Alzheimer's disease? Y N
a) If yes, specify how close on scale of 1 to 3, 1 being not close, 3 being very close _____
b) If yes, specify how frequent you see that person, 1 being not often 3 being very often _____
Repeat if more than 1 friend/acquaintance:
28c)

28d)

28e)

29) Are you or have you ever been a caregiver for someone with Alzheimer's disease? Y N
a) If yes, specify who (friend, family member, professional via work)

30) Do you believe that Alzheimer's disease is inherited?

Definitely not Maybe not Unsure Maybe yes Definitely yes

Appendix E: Permission to Contact Participant for Follow-up Studies

Permission to contact for follow-up studies:

We are interested in maintaining a list of individuals who are willing to be contacted for follow-up memory screening studies that we plan to conduct annually in the Ohio University Clinical Neuropsychology Research Laboratory. Some potential projects are:

1) annual memory screening with feedback (including feedback about how your performance differs from your performance today);
2) memory intervention studies (in which some of you might be invited to attend workshops about memory management)
3) examining the role of stress in memory and other cognitive changes associated with aging (which might include measures of stress hormones while completing cognitive tasks)

If you provide us permission to contact you about future studies, this does NOT mean you have to participate. You are just giving us permission to call you or write you a letter describing the future study, and at that time you can decide whether you are interested in participating or not.

Note that this form will be stored separately from the data gathered from your research visit today, and thus will in no way provide a link to your research data (this is why we ask you to create a unique identifying number to track your performance over time if you wish to participate in the follow-up studies). This form simply provides us with a list of those interested in being contacted for future studies, and how to reach them.

I, the below signed, provide permission to be contacted by researchers in the Ohio University Clinical Neuropsychology Research Laboratory in the next 1-2 years regarding any follow-up studies to the memory study I just completed. I understand that this does not obligate me to participate in any future studies, but merely gives researchers in the OUCNRL permission to contact me and provide information about the follow-up studies for which I might qualify.

/s/ _____ Date

Printed Name _____

Permanent Address _____

_____

_____

Telephone Number _____

E-mail address _____

Preferred mode of future contact (please circle):

telephone letter e-mail doesn't matter

Appendix F: Participant Post-study Form

Post-study Form

Sometimes brief cognitive screens such as the ones your just completed suggest a need for further testing. We will give you feedback about your performance if you wish. You can either receive feedback right now, if you wait a few minutes for the Study Director, Dr. Julie Suhr, to score and interpret your test results, or, if you do not have time to wait, let your examiner know and he or she will pass your contact information on to Dr. Julie Suhr, who will contact you by phone and provide you with feedback about your performance.

If you continue to have cognitive concerns and would like to have a more comprehensive evaluation free of charge, please contact Dr. Julie Suhr at (740) 593-1091 to schedule an appointment in the Ohio University Psychology and Social Work Clinic. Another resource if you have continued concerns is the Ohio University College of Medicine's Geriatric Department at (740) 593-2482.

For some people, memory changes can be distressing. If you feel that you need counseling or psychological services, please contact:

Ohio University Psychology and Social Work Clinic: 593-0902

Tri-County Mental Health: 592-3091

If you have any questions concerning the study, contact Julie Suhr at 593-1091 or e-mail at suhr@ohiou.edu.