심리학석사학위논문

Cognitive reserve as a protective factor moderating the effect of depressive mood on memory impairment

우울감에 따른 인지기능 감퇴와 인지 보유고의 조절효과

2016년 2월

서울대학교 대학원

심리학과 임상신경심리학 전공

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ABSTRACT

Cognitive reserve as a protective factor moderating the effect of depressive mood on memory impairment

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The cognitive reserve hypothesis holds that older adults show individual differences in their flexibility and efficiency in using available neural resources. Those with higher cognitive reserve show better cognitive functioning, including memory, and can tolerate higher levels of brain pathology before displaying clinical symptoms (Stern, 2002; Scarmeas & Stern, 2004). Studies have shown that depressed patients perform comparatively poorly on memory tasks, and many have associated depression with a significantly increased risk of dementia (Cohen et al., 1982; Burt et al., 1995). This study was conducted to test whether cognitive reserve, measured by years of education and premorbid IQ, can moderate the negative effect of depressive mood on memory performance. 79 healthy, non-demented elderly individuals aged 61 to 87 were recruited from eight different senior centers in Seoul, South Korea. The Geriatric Depression Scale (GDS) was used to measure depressive mood, the Elderly Verbal Learning Test (EVLT) was used to measure short-term recall, long-term recall, and recognition memory, and the

K-WAIS-IV Vocabulary subtest was used as an index of premorbid IQ. The results showed that receiving more years of education, but not having a higher premorbid IQ, significantly reduced the negative association between depressive mood and memory performance. Subjects with low education had declining memory test scores depending on level of depressive mood. However, subjects with high education displayed relatively stable memory function despite level of depressive mood, implying that education has greater protective effects against memory impairment than premorbid intelligence does.

Keywords: cognitive aging, cognitive reserve, depressive mood, memory, education

Student Number: 2013-22823

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INTRODUCTION

Aging is one of the most challenging public health issues that many developed countries, including South Korea, are facing today. With the growth of an aging society, there has been a great amount of increase in initiatives and interventions to promote successful aging (Depp, Harmell, & Vahia, 2012). Successful aging refers to physical, mental, and social well-being in older age. Researchers aim to define what differentiates successful aging from pathological aging in order to design effective strategies and treatments that can potentially protect the health and well-being of the elderly people. The current study discusses cognitive reserve as a potential target for intervention, even in the presence of depressive mood, which has been associated with accelerated aging.

1. Age-associated memory impairment

There are universal declines that accompany aging with the exception of a few cognitive functions, including vocabulary knowledge. But other than verbal intelligence that tends to remain stable throughout the lifespan, the basic mechanisms of cognitive information processing, such as processing speed, spatial visualization, reasoning ability, and memory tend to decline across the adult lifespan (Salthouse, 2010).

One of the key concerns of older adults is the experience of memory loss. Normal aging is associated with a decline in various memory abilities across many cognitive tasks, in a phenomenon known as age-associated memory impairment. The

ability to encode new memories of events or facts and working memory shows decline in both cross-sectional and longitudinal studies (Hedden & Gabrieli, 2004). Older adults demonstrate deficits, relative to young adults, in episodic or explicit tests of memory that require the conscious retrieval of previously experienced events, such as free recall, cued recall, and recognition tests (Light, 1991; Grady & Craik, 2000). Studies comparing the effects of aging on episodic memory, semantic memory, short-term memory and priming find that episodic memory is especially impaired in normal aging.

Brain imaging studies have established that the human brain shrinks with age. Even in the absence of a clinically significant neurodegenerative disease, there are consistent reductions in whole brain volume and increases in cerebrospinal volume (Fox & Schott, 2004). In the presence of cognitive impairment that is not severe enough to meet criteria for a dementia diagnosis, hippocampal atrophy appears to be the most consistent observation (Raz, Rodrigue, Head, Kennedy, & Acker, 2004; Du et al., 2001; Wolf et al., 2001; Jack et al., 2000). Wolf et al. (2001) suggest that the point of transition from normal cognitive aging to the initial stage of Alzheimer's disease may be detected by hippocampal atrophy.

2. Depressive mood on memory impairment

2.1 Depressive mood as a public health problem among elderly

Along with aging, late life depression is likely to become one of the most significant public health problems in Asian countries, including Korea, where the

numbers of elderly people are increasing at a remarkable rate. Although depression is one of the most common and treatable mental illnesses, late-life depression can oftentimes go unrecognized and untreated due to its sub-threshold features and complicated etiologies. The burden of inadequately treated late-life depression is substantial and can be detrimental to well-being, despite it being sub-threshold. Researchers say that it may be just as disabling as major depressive disorder (Beekman, Deeg, Braam, Smit, & Van Tilburg, 1997). Recently, researchers and clinicians have been paying more attention to late-life depression due to a steep rise in the suicide rate among elderly Koreans (Korea National Statistical Office, 2010).

One estimate gave the prevalence of major depressive disorder among the elderly as 5.37% in Korea, which is higher than the prevalence in most Western countries and other Asian countries (Park et al., 2010). However, they say that this may display only a small proportion of the situation, since a considerable amount of the elderly people with clinically significant depressive symptoms do not meet the rigorous diagnostic criteria of the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (American Psychiatric Association, 2013) for major depressive disorder. Park and colleagues (2012) estimated the prevalence rates for possible and probable depression in Korean elders as 10.1% and 17.8%, respectively, making the health condition a significant issue for the country.

2.2 Depressive mood as a risk factor for memory impairment

It has long been observed that depression is associated with memory impairment. Cohen and colleagues (1982) claimed that depressed patients perform comparatively poorly on memory tasks, while Sternberg & Jarvik (1976) have discovered that depressed patients significantly improve their memory performance after treatment with antidepressants or other therapies, suggesting that the association between the two phenomenon may not merely be psychological, in that individuals who begin to experience memory impairments develop depressive mood, but rather that there may be biochemical mechanisms that relate the two. Indeed, more recent evidence from longitudinal studies showed that late-life depression was associated with a 2 to 5-fold increased risk of dementia, for which memory impairment is a large component (Saczynski, Beiser, Seshadri, Auerbach, Wolf, & Au, 2010; Andersen, Lolk, Kragh-Sørensen, Petersen, & Green, 2005; Gatz, Tyas, St John, & Montgomery, 2005; Chen, Hu, Wei, Qin, McCracken, & Copeland, 2008). Another 7-year longitudinal study found that even one additional depressive symptom in older adults increased the risk of dementia by 20 percent (Wilson et al., 2002). This suggests that the severity of late-life depression may be important to the risk of dementia.

There have been discrepancies where some studies found non-significant results at follow-up (Becker et al., 2009; Lindsay et al., 2002). Variations in the results and the amount of risk depressive mood may pose on dementia and memory decline might be explained by variations in sample size, measures used to assess depressive mood, as well as the frequency and severity of depression. Saczynski and colleagues covered 17 years

of follow-up and indicated a 70 percent increase in risk of developing dementia with depression, suggesting that a longer follow-up process may allow for better assessment of depression as a risk factor for dementia (Saczynski, Beiser, Seshadri, Auerbach, Wolf, & Au, 2010).

2.3 Potential mechanisms explaining memory impairment

A prominent mechanism that may link depressive mood and memory impairment is the increase in glucocorticoids, which are stress hormones that can lead to hippocampal atrophy and cognitive deficits if secreted in excessive amounts (Sapolsky, 1992; Butters et al., 2008; Sierksma, van den Hove, Steinbusch, & Prickaerts, 2010). Since stressful life events are highly related to depressive episodes (Pittenger & Duman, 2008; Caspi et al., 2003; Charney & Manji, 2004) and the HPA axis is hyperactive in depressed patients (Wolkowitz, Burke, Epel, & Reus, 2009; Swaab, Bao, & Lucassen, 2005), it can be predicted that depressed patients would have increased glucocorticoid levels that lead to hippocampal neuronal damage and memory impairment. Indeed, reductions in hippocampal volume have been consistently found among individuals with recurrent depression and longer duration of illness, suggesting support for the cortisol-hippocampal pathway (Colla et al., 2007; Sheline, Gado, & Kraemer, 2003; MacQueen et al., 2003).

Another proposed link is a decrease in the levels and activities of brain-derived neurotrophic factors (BDNF) (Caraci, Copani, Nicoletti, & Drago, 2010). BDNF plays a

key role in maintaining neuronal health and modulation of synaptic plasticity in the hippocampus, and is therefore, presumed to be important to the integrity of the hippocampus and memory processing (Fumagalli, Molteni, Calabrese, Maj, Racagni, & Riva, 2008; Tyler, Alonso, Bramham, & Pozzo-Miller, 2002; Alonso et al., 2002; Yamada & Nabeshima, 2003; Bekinschtein, Cammarota, Igaz, Bevilaqua, Izquierdo, & Medina, 2007). However, impairment in BDNF signaling has been detected in stress-induced animal models of depression and individuals with depression (Krishnan & Nestler, 2008; Karege, Vaudan, Schwald, Perroud, & La Harpe, 2005; Angelucci, Brenè, & Mathé, 2005). Studies on depressed patients and dementia patients have demonstrated decreased levels of BDNF in the hippocampus (Sierksma, van den Hove, Steinbusch, & Prickaerts, 2010; Karege, Vaudan, Schwald, Perroud, & La Harpe, 2005), which lead to neuronal losses and impaired neuroplasticity, ultimately resulting in cognitive impairment.

Lastly, studies have shown that a lifetime history of depression corresponds to increases in amyloid-β and tau neurofibrillary tangles within the hippocampus, which plays a role in the neurodegeneration seen in Alzheimer's disease. Both beta-amyloid and tau tangles accumulate more in the hippocampus of Alzheimer's disease (AD) patients with depression compared to AD patients without depression, suggesting an interaction between depression and AD neuropathology (Rapp et al., 2006; Rapp, Schnaider-Beeri, Purohit, Perl, Haroutunian, & Sano, 2008). Green and colleagues reported that stress-level glucocorticoid administration increased amyloid formation and accelerated tau accumulation in mice (Green, Billings, Roozendaal, McGaugh, & LaFerla, 2006), which suggests that high levels of glucocorticoids may play a role in the development and

progression of AD through accumulation of beta-amyloid and tau tangles.

3. Cognitive reserve as a protective factor

The cognitive reserve hypothesis asserts that there are individual differences in the way older adults exhibit cognitive functioning and that some are able to tolerate higher levels of brain pathology before displaying clinical symptoms than others. Cognitive reserve allows for more neural efficiency, capacity, and flexible strategy use in face of aging and pathology. In other words, it indicates the ability to optimize performance through differential recruitment of brain networks or alternative cognitive strategies (Scarmeas & Stern, 2004; Stern, Alexander, Prohovnik, & Mayeux, 1992; Tucker & Stern, 2011). Assuming this hypothesis is true, individuals with higher cognitive reserve will have a delayed onset of cognitive decline, and clinical diagnostic criteria for dementia will be reached later, when the pathology is more severe. Also, at any given level of memory performance, pathology will be more severe for those with higher reserve than those with low reserve.

Cognitive reserve is not fixed and continues to develop across the lifespan. Childhood cognition, educational attainment, and adult occupation all contribute to cognitive reserve independently, as well as synergistically (Stern, Gurland, Tatemichi, Tang, Wilder, & Mayeux, 1994). The concept of cognitive reserves offers an explanation to why many studies have demonstrated that individuals with higher levels of intelligence and educational and occupational attainment are able to sustain greater brain

pathology before demonstrating functional deficit.

As stated above, one of the most well-established proxy measures of reserve capacity in the elderly is educational attainment, which is thought to reflect more effective use of brain networks or cognitive paradigms (Stern, 2009). In line with the hypothesis of cognitive reserve, many studies in both North America and Europe have suggested that educational attainment is associated with better cognitive performance and reduced risk for cognitive impairment and dementia in late life (Brayne & Calloway, 1990; Evans et al., 1997; Fratiglioni et al., 1991; Katzman, 1993; Mortel, Meyer, Herod, & Thornby, 1995; Stern, Gurland, Tatemichi, Tang, Wilder, & Mayeux, 1994).

Similar findings have been reported with respect to memory decline and cognitive decline in general, indicating that the evidence is not only limited to reducing risk of disease (Blum & Jarvik, 1974; Colsher & Wallace, 1991; Evans et al. 1993; Snowdon, Kemper, Mortimer, Greiner, Wekstein, & Markesbery, 1996; Schmand, Smit, Geerlings, & Lindeboom, 1997a). One longitudinal study examined memory test data that was collected at regular intervals from healthy elders who were followed until they became demented. It was found that higher education delayed the onset of accelerated cognitive decline, and that the rate of memory decline after decline onset was more rapid in those with higher education due to increased pathology burden (Hall, Derby, LeValley, Katz, Verghese, & Lipton, 2007).

Results have been mixed on whether years of education moderates the trajectory of age-related cognitive decline (Antsey & Christensen, 2000). Yet, a great amount of

research reports that education attenuates cognitive decline among non-demented older adults (Albert et al., 1995; Bosma et al., 2003; Butler et al., 1996; Evans et al., 1993; Farmer et al., 1995; Lyketsos et al., 1999). The results from these studies support an active cognitive reserve hypothesis in which education promotes more efficient cognitive processing and use of brain networks, which results in smaller cognitive declines in the face of neuropathology, ultimately slowing the process of age-related cognitive decline (Stern, 2002). Although there are other proxy measures of cognitive reserve, education has been thought to be the most important factor (Carret et al., 2003).

According to the hypothesis, more pathology is necessary to bring about cognitive decline in people who are highly educated, compared to those who received less education. As stated previously, amyloid plaques and tau tangles are related to level of cognition. Studies have found that education dampens the association of amyloid plaques with the level of cognition, suggesting that education is related to factors that reduce the deleterious effect of amyloid accumulation on cognitive performance (Bennet, Schneider, Wilson, Bienias, & Arnold, 2005; Roe, Mintun, D'Angelo, Xiong, Grant, & Morris, 2008).

Still, there are some studies that suggest that an estimate of IQ, or premorbid intelligence, might actually be a more powerful measure of reserve (Albert & Teresi, 1999; Alexander et al., 1997; Schmand et al., 1997). In Valenzuela & Sachdev's review of reserve (2005), the combined odds ratio for individuals with high premorbid IQ compared to low was 0.58 (95% CI 0.44-0.77)—a decreased risk of approximately 42 percent (Schmand et al., 1997; Elias et al., 2000). Both studies reported significant

protective effects of premorbid intelligence in the likelihood of developing dementia.

Other indices of cognitive reserve include occupation, which has constantly shown a significant protective effect (Bickel & Cooper, 1994; Stern et al. 1994; Evans et al. 1997; Schmand et al. 1997; Helmer et al. 2001; Scarmeas et al. 2001; Anttila et al., 2002; Karp et al. 2004), as well as mentally stimulating leisure activities, which have been found to have a significant protective effect both before and after controlling for education and occupation (Fabrigoule et al. 1995; Fratiglioni et al. 2000; Scarmeas et al. 2001; Wang et al. 2002; Wilson et al. 2002; Verghese et al. 2003).

4. Objectives and Hypothesis

The aim of this study is to investigate whether cognitive reserve moderates the negative effect of depressive mood on memory impairment and thus, acts as a protective factor against cognitive decline, and if so, to discuss possible implications it has for the delay of memory impairment, especially in the context of South Korea where the society is quickly aging and the rate of late-life depression is high. There are many studies that have explored depression and memory, as well as cognitive reserve and memory. However, there is not much research on the psychosocial factors that may moderate the relationship between the depressive mood and memory decline and create the individual differences that are actually observed. This paper is one of the first to examine the protective effects of cognitive reserve against memory impairment in the context of depressive mood. A sub-goal is to identify which aspects of memory—short-term verbal

recall, long-term verbal recall, or verbal recognition memory—are moderated by cognitive reserve.

The hypothesis is that (1) an increase in depressive mood will result in lower verbal memory performance, particularly for long-term recall and recognition memory, which depend on the functioning of the hippocampus, and (2) subjects with high cognitive reserve will show a significantly smaller decline in memory performance, whereas subjects with low cognitive reserve will display the significant declines in memory that are expected with higher depressive mood levels. In other words, cognitive reserve will serve as a moderator by dampening the negative effect that depressive mood has on memory impairment.

METHODS

1. Subjects

The subjects of this study were recruited from eight different community-based senior centers throughout different districts of Seoul, South Korea in order to gather a diverse sample of the elderly population in the city. A total of 101 elderly women were recruited and administered a demographic, social and economic questionnaire. The subjects' years of education was determined by the number of years of actual school attendance. The subjects were then screened for multiple exclusion criteria. The exclusion criteria were as follows: psychiatric disorders, neurological history, use of psychiatric drugs, medical conditions such as hypertension or diabetes uncontrolled by medication, serious head injury and unconscious experience, and alcohol-drug misuse.

Participants with cognitive impairment were also excluded using scores from Elderly Memory disorder Scale (EMS) and Korean Dementia Rating Scale-2 (K-DRS-2), which are neuropsychological measures that assess memory and cognitive status of older adults (Chey, 2007; Chey, 2010). Exclusion due to cognitive impairment was discussed in a case conference with two licensed clinical psychologists and three master's students in clinical neuropsychology. None of the participants had a disruption of functional ability and were able to maintain an independent daily life.

The final subjects for this study were 79 healthy, non-demented women between the ages of 61 and 87 (mean age: 73.89; SD: 5.85). The mean education was 6.63 years (SD: 4.06; range: 0 to 16) and mean depressive mood rating based on the GDS was 10.38

(SD: 6.51; range: 0 to 30).

After the nature of the study was explained, all participants provided written informed consent and were given financial remuneration for their participation at the end of the session. This study was approved by the Institutional Review Board (IRB) at Seoul National University (SNU).

2. Procedure

All subjects participated in an approximately 50-minute demographic, socio-economic, and social activity background survey in a one-on-one interview, and were screened using exclusion criteria for healthy, non-demented elderly women. Then the participants were scheduled to partake in the second phase of the study approximately 5.97 months later (SD: 1.78, range: 4 to 9 months). Here, the subjects participated individually in an approximately 70-minute neuropsychological test session in a quiet environment. The order of the cognitive tasks that were administered was invariant for all the participants. The current study uses data from a depression scale that was administered in the first data collection phase as well as from an elderly memory scale administered in phase two of data collection.

3. Neuropsychological Assessments

3.1 Depressive mood

The Geriatric Depression Scale (GDS; Yesavage et al., 1982) is one of the most used depression self-reports that was created for the detection of depression among the elderly. Researchers and clinicians have often used this questionnaire for depressive symptoms to capture sub-syndromal depression as well. The 30 items included in the GDS are based on characteristics of depression in the elderly (Montorio & Izal, 1996). The GDS's relevance is also seen in its capacity for measuring the same overall construct of depression as the most universal and widely studied scale for the assessment of depression in adults, the BDI. Also, the simplicity of the response procedure and brief average length enhance understanding, diminish the anxiety generated by the administration of a psychological test, and produce a high completion rate (Pfeiffer, 1987; Zarit, Eiler, & Hassinger, 1985).

Item number 14 ('Do you feel you have more problems with memory than most?') of the GDS was excluded from the analysis in this study since it correlates with memory performance.

3.2 Cognitive impairment

The Korean Dementia Rating Scale-2 (K-DRS-2) was administered to screen for dementia. The DRS-2 is an assessment tool that examines an individual's overall level of cognitive functioning and yields five subscale scores. The five subscales provide addition information on specific abilities, including Attention, Initiation/Perseveration, Construction, Conceptualization, and Memory. Within each subscale, the most difficult

tasks are presented first. If the first one or two tasks in a subscale are performed well, subsequent tasks in the subscale are credited with a correct performance and the examiner proceeds to the next subscale, which allows for a significantly shortened total testing time. The K-DRS-2 is the adapted and standardized version of the DRS-2 (Chey, 2010). Age- and education-corrected normative tables are provided for all K-DRS-2 subscales and total score.

Three subtests of the Elderly Memory disorder Scale (EMS) were conducted: Elderly Verbal Learning Test (EVLT), Simple Rey Figure Test (SRFT), and Korean Boston Naming Test (K-KBNT). The EMS is an optimized scale for the Korean elderly population, developed to diagnose dementia, especially Alzheimer's disease, and mainly focuses on assessing episodic memory (Chey, 2007). The EMS consists of various tests to assess learning/memory, visuospatial abilities, language, and conceptualization abilities. The three subtests used in this study are described below.

The EVLT is a word list learning task for elderly adults that provides measurement of verbal learning and memory, by using the paradigm of the California Verbal Learning Test (Chey et al., 2006; Delis et al., 1987). The EVLT uses a list consisting of 9 words from three categories presented over five trials. Throughout the five trials, the subjects are told to repeat as many of the words as they can remember from the list. This allows for the calculation of immediate recall memory. After the first five trials, an interference list of 9 different words is presented and the subjects are told to repeat this new list. This is followed by short-delay recall of the first, original list and then long-delay recall after 15 to 30 minutes. A recognition trial is also administered at

the end of the test, in which the subjects are given 30 words one at a time, and are asked to indicate whether each word was a part of the original list. Thus, the EVLT provides information about acquisition, recall, retention, and retrieval of verbal information.

The SRFT is a simplified version of the Rey-Osterrieth Complex Figure Test (RCFT) that was modified for the elderly population. The SRFT was developed to assess the ability of visuospatial construction and visual memory; thus, a wide range of cognitive processes are needed for this test, such as perception, motor function, memory, executive function, and problem solving (Chey, 2007). The SRFT is comprised of the copy task, immediate/delayed recall task, and recognition task.

The 15-item short version of the K-BNT, which was modified from the standard 60-item K-BNT (Kim & Na, 1997), was developed to easily assess the naming ability of the patients with dementia and severe cognitive deficit (Chey, 2007). The K-BNT is the most representative visual confrontation naming test for assessing the ability to recall words.

In regards to cognitive impairment, only data from the EVLT were analyzed in this study, since the objective is to examine aspects of memory impairment only, and in particular, impairment in verbal memory recall and recognition.

3.3 Premorbid intelligence

The Korean Wechsler Adult Intelligence Scale-Fourth Edition (K-WAIS-IV) Vocabulary subtest was administered in this study to provide an index of premorbid

intelligence. WAIS is a psychological assessment that has been frequently used to measure cognitive abilities, especially general intelligence (Benson et al., 2010; Weiss et al., 2013). The K-WAIS-IV is an adapted and standardized Korean version of the Wechsler Adult Intelligence Scale-Fourth Edition (WAIS-IV) (Wechsler, 2008a), which is the most recent edition of the Wechsler intelligence scales (Hwang et al., 2012).

4. Cognitive Reserve Index

A vast amount of literature on cognitive reserve derived a cognitive factor score from three or more proxies, including years of education and two indices of premorbid intelligence—the New Adult Reading Test (NART; Nelson, 1982) and the WAIS Vocabulary subtest (Wechsler, 1981)—using factor analysis (Stern et al., 2005). The present study used two variables as proxy measures for each subject's level of cognitive reserve: years of education and one index of intelligence, scores on the K-WAIS-4 vocabulary subtest. The latter test is considered as a good estimate of premorbid verbal intelligence. Using a factor analysis, a cognitive reserve factor score was derived which summarizes the two reserve variables. The cognitive reserve factor accounted for 72% of the common variance in these two measures. This factor score was used to represent cognitive reserve in the subsequent analyses.

5. Analysis

Level of education, premorbid verbal IQ, depression scale scores, and test scores from all neuropsychological assessments were all analyzed using SPSS 18.0. Descriptive statistics of the subjects' demographic data are provided. The effect of depressive mood on memory impairment after controlling for education and premorbid verbal IQ was tested using multiple regression.

The moderation model was used to test whether the prediction of memory impairment from depressive mood differed across levels of cognitive reserve as measured by years of educational attainment and premorbid intelligence, as years of educational alone, and as premorbid intelligence alone. This allowed the investigation of whether cognitive reserve affects the strength of the association between depressive mood and memory function. The moderation effect was seen as an interaction between the cognitive reserve factor and GDS score, where the effects of the GDS score were dependent on the cognitive reserve factor. The model was investigated using a multiple regression analysis, where all variables and their interaction term were centered prior to model estimation.

An independent-samples t-test was conducted in order to compare the differential effect of depressive mood on EVLT short-term recall, long-term recall, and recognition memory scores between subjects with low education and high education. Low education was determined by 6 or less years of formal educational attainment, while high education was determined by more than 6 years of formal educational attainment. The rationale for this criterion is that graduation from elementary school (6 years of

formal education) results in significant differences among Korean elderly (Suk, 2008; Suk, Chey, & Kim, 2010). Individuals in both the low and high education groups were placed in an above the mean GDS score group (M=9.57) and below the mean GDS score group in order observe the effect of depressive mood on memory. Although a GDS cut-off score of 11 is often used to determine those with mild depression (Brink, Yesavage, Owen, Heersema, Adey, & Rose, 1982), this paper examines depressive mood as having a significant effect on memory, and thus, the mean score was used to determine high depressive mood and low depressive mood. Statistical significance for all analysis was determined by a p-value of less than or equal to 0.05.

RESULTS

The descriptive statistics are shown in Table 1. The mean age of the 79 participants was 73.89 years (SD: 5.85, range: 61 to 87). The mean years of formal educational attainment was 6.63 years (SD: 4.06, range: 0 to 16), and the mean K-WAIS-IV Vocabulary subtest score was 18.90 out of a total score of 57 points (SD: 10.55, range: 6 to 46). The mean GDS score was 10.38 out of a total score of 30 (SD: 6.52, range: 0 to 30).

Table 1. Demographic data of the sample

	Min.	Max.	Mean	SD
Age	61	87	73.89	5.85
Years of Education	0	16	6.63	4.06
WAIS Vocab Score	6	46	18.90	10.55
GDS Score	0	30	10.38	6.52
GDS Score (w/o item14)	0	29	9.57	6.40
EVLT short-term recall	0	8	5.77	1.66
EVLT long-term recall	2	9	5.96	1.57
EVLT recognition	17	30	26.77	2.69

WAIS: Korean Wechsler Adult Intelligence Scale- 4th ed. vocabulary subtest score; GDS: Geriatric Depression Scale score; EVLT: Elderly Verbal Learning Test score

In order to examine the association between depressive mood and memory impairment, a regression analysis was conducted after controlling for years of education and WAIS vocabulary scores. Age was not controlled since its correlation with scores on the GDS was not significant (p=.77). Depressive mood was not significantly correlated to short-term memory free-recall scores on the EVLT after controlling for educational attainment (p=.105). On the other hand, as shown in Table 2, depressive mood was significantly correlated to both long-term memory free-recall and recognition scores (p=.002 and p=.010 respectively).

Table 2. Memory impairment predicted by depressive mood

	Model		**B**	**β**	**t**	**p-value**	**R Square**
Short-term recall	1	Edu	.017	.043	.241	.810	
		WAIS	.007	.048	.271	.787	.007
	2	Edu	.006	.016	.090	.928	
		WAIS	.013	.081	.461	.646	.042
		GDS	-.048	-.187	-1.641	.105	
Long-term recall	1	Edu	.008	.022	.128	.899	
		WAIS	.039	.258	1.518	.133	.076
	2	Edu	-.010	-.026	-.162	.872	
		WAIS	.047	.318	1.966	.053	.186
		GDS	-.082	-.335	-3.194	.002**	
Recognition	1	Edu	.029	.044	.260	.796	
		WAIS	.065	.254	1.498	.138	.084
	2	Edu	.002	.004	.022	.982	
		WAIS	.078	.305	1.854	.068	.162
		GDS	-.119	-.283	-2.656	.010**	

Edu: years of education; WAIS: Korean Wechsler Adult Intelligence Scale- 4th ed. vocabulary subtest score; GDS: Geriatric Depression Scale score
**p<.05

When the moderation effect was tested with multiple regression analysis, the cognitive reserve factor score derived from educational attainment and premorbid verbal intelligence did not significantly moderate the association between depressive mood and memory impairment. (p=.09 for long-term recall and p=.11 for recognition). The moderation effect of cognitive reserve was not analyzed for short-term recall since short-term recall did not significantly correlate with depressive mood.

Table 3. Moderation effect of CR measured by education and premorbid IQ

	Model		**B**	**β**	**t**	**p-value**	**R Square**
Long-term recall	1	GDS	-.079	-.322	-3.082	.003	
		compositeCR	.430	.274	2.621	.011	.173
	2	GDS	-.090	-.366	-3.442	.001	
		compositeCR	.384	.244	2.338	.022	.204
		GDSxCR	.044	.184	1.709	.092	
Recognition	1	GDS	-.114	-.271	-2.564	.012	
		compositeCR	.777	.289	2.732	.008	.152
	2	GDS	-.132	-.314	-2.914	.005	
		compositeCR	.700	.260	2.456	.016	.181
		GDSxCR	.074	.179	1.643	.105	

GDS: Geriatric Depression Scale score; compositeCR: composite cognitive reserve score
**p<.05

However, when examining the moderation effect of the two cognitive reserve proxies separately, the results showed that education had a significant effect on the relationship between depressive mood and memory impairment (p=.049 for long-term recall and p=0.050 for recognition). Premorbid intelligence measured by K-WAIS-4 vocabulary subtest scores, on the other hand, did not show a significant moderation effect (p=.14 and p=.17 for long-term recall and recognition respectively).

Table 4. Moderation effect of CR measured by years of education

	Model		B	β	t	p-value	R Square
Long-term recall	1	GDS	-.076	-.311	-2.932	.004	
		Edu	.084	.216	2.040	.045	.145
	2	GDS	-.090	-.366	-3.402	.001	
		Edu	.073	.188	1.788	.078	.188
		GDSxEdu	.014	.217	2.003	.049**	
Recognition	1	GDS	-.109	-.260	-2.420	.018	
		Edu	.156	.236	2.196	.031	.124
	2	GDS	-.132	-.315	-2.892	.005	
		Edu	.137	.207	1.947	.055	.168
		GDSxEdu	.024	.219	1.989	.050*	

GDS: Geriatric Depression Scale score; Edu: years of education
*p<.1, **p<.05

Table 5. Moderation effect of CR measured by premorbid IQ

	Model		B	β	t	p-value	R Square
Long-term recall	1	GDS	-.082	-.333	-3.214	.002	
		WAIS	.044	.298	2.874	.005	.186
	2	GDS	-.090	-.367	-3.482	.001	
		WAIS	.042	.282	2.727	.008	.210
		GDSxWAIS	.004	.158	1.495	.139	
Recognition	1	GDS	-.119	-.283	-2.687	.009	
		WAIS	.078	.307	2.920	.005	.162
	2	GDS	-.132	-.314	-2.938	.004	
		WAIS	.074	.292	2.778	.007	.184
		GDSxWAIS	.006	.150	1.396	.167	

GDS: Geriatric Depression Scale score; WAIS: Korean Wechsler Adult Intelligence Scale- 4th ed. cocabulary subtest score
**p<.05

The results from an independent-samples t-test (refer to Table 6) that was conducted in order to compare educational group differences in the effects of depressive mood on recall and recognition memory showed that in the low education group, there was a significant difference between in long-term recall and recognition between individuals with high depressive mood (long-term recall score: M=4.89, SD=1.68; recognition score: M=25.33, SD=2.38) and low depressive mood (long-term recall score: M=6.36, SD=1.10; recognition score: M= 27.21, SD=2.44); t(44)=3.60, p=.001 and t(44)=2.58, p=.013 for long-term recall and recognition, respectively. Differential performance for short-term memory was not observed.

However, in the high education group, there was not a significant difference in memory performance between those with high depressive mood (long-term recall score: M=5.75, SD=1.36; recognition score: M=26.58, SD=2.91) and low depressive mood (long-term recall score: M=6.48, SD=1.75; recognition score: M=27.52, SD=2.82) individuals; t(31)=1.24, p=.23 and t(31)=.91, p=.37 for long-term recall and recognition, respectively.

Table 6. Educational group differences in effects of depressive mood

		GDS group	M	SD	t	df	p-value
Low Education Group	Short-term recall	Low	6.071	1.359	1.729	44	.091
		High	5.333	1.495			
	Long-term recall	Low	6.357	1.096	3.600	44	.001**
		High	4.889	1.676			
	Recognition	Low	27.214	2.440	2.578	44	.013**
		High	25.333	2.376			
High Education Group	Short-term recall	Low	6.048	1.883	1.143	31	.262
		High	5.250	2.006			
	Long-term recall	Low	6.476	1.750	1.238	31	.225
		High	5.750	1.357			
	Recognition	Low	27.524	2.822	.911	31	.369
		High	26.583	2.906			

**p<.05

Figures 1 and 2 illustrate the results of the independent-samples t-test. Depressive mood had a significant effect on both long-term recall (p=.001) and recognition memory scores (p=.013) in subjects with low educational attainment, whereas in those with high educational attainment, depressive mood did not have a significant effect on neither long-term recall (p=.23) nor recognition memory (p=.37).

Figure 1. Differential effects of depressive mood on long-term recall

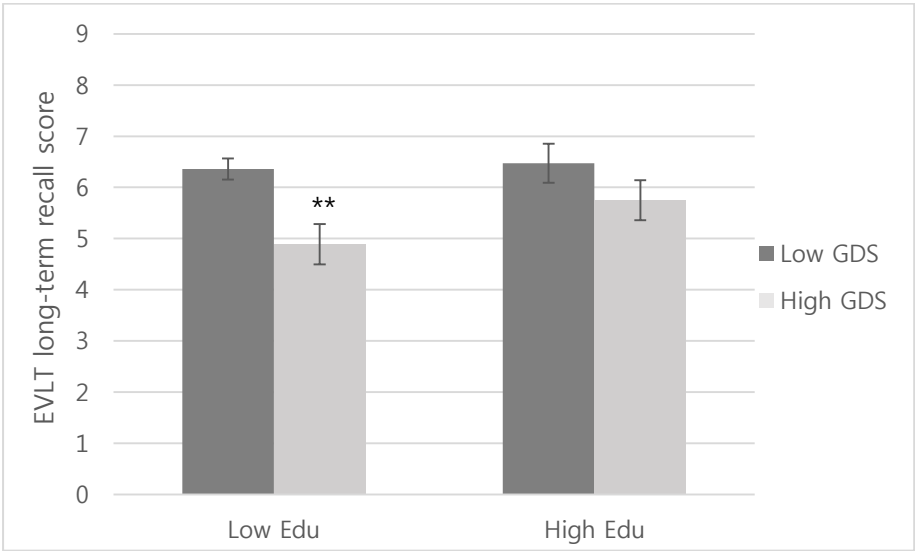
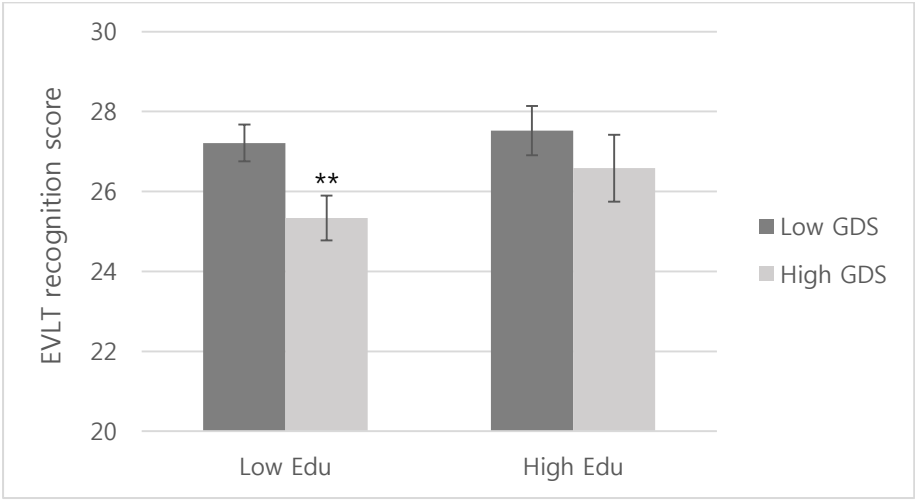


Figure 2. Differential effects of depressive mood on recognition



DISCUSSION

This paper is the first study, to our knowledge, investigating cognitive reserve as a protective factor moderating the effect of depressive mood on memory impairment. There are two main findings: (1) an increase in depressive mood resulted in significantly lower verbal memory performance for long-term recall and recognition memory, but not for short-term recall memory, and (2) higher cognitive reserve significantly reduced the negative effect of depressive mood on memory impairment, but only when cognitive reserve was measured by years of formal education. Cognitive reserve did not serve as a significant moderating factor when it was measured by a composite score of years of education and premorbid intelligence, nor when it was measured by premorbid intelligence alone.

1. Depressive mood is a risk factor of memory impairment

Many studies have raised the question regarding the nature of the close relationship between depression and dementia, of which memory impairment is a highlighting characteristic. Saczynski and colleagues, as well as Andersen and colleagues found that late-life depression was associated with a 2 to 5-fold increase in dementia risk (Saczynski, Beiser, Seshadri, Auerbach, Wolf, & Au, 2010; Andersen, Lolk, Kragh-Sørensen, Petersen, & Green, 2005). Also, patients with a higher magnitude of depression were more prone to memory deficits compared to less depressed patients (Burt, Zembar, & Niederehe, 1995). Parallel to these findings, the results from this study

reveal that an increased level of depressive mood was associated with increased levels of memory impairment.

This observation was significant for long-term recall and recognition memory, but was not significant for short-term memory (p=.002, .010, .105, respectively). This may be because long-term recall and recognition depend on hippocampal functioning, whereas short-term memory is associated with other regions of the brain, such as the perirhinal cortex. Researchers have long known that the medial temporal region of the brain is important for long-term memory. Squire, Cohen, Eichenbaum, and others have claimed that the medial temporal lobes, including the hippocampus and the medial temporal cortices—perirhinal, entorhinal, and parahippocampal cortices—form a declarative memory system that supports recall and recognition memory (Squire, 1992b; Cohen & Eichenbaum, 1993; Cohen et al., 1997; Eichenbaum, 2000). Supporting this claim, patients with medial temporal lobe lesions typically show impaired recall and recognition but intact performance on other memory tests (Squire, 1992a).

Neuroimaging studies involving hippocampal structure have time and time again offered informative findings for this area of research, since the hippocampus is involved in both memory performance and mood disorders. Longitudinal studies using magnetic resonance imaging (MRI) have found that hippocampal volume loss was associated with cognitive decline (Jack et al., 2000), giving support for the well-established claim of hippocampal involvement in cognition. Other MRI studies evaluating hippocampus in late-life depression have indicated that older depressed patients have decreased hippocampal volumes compared to non-depressed individuals

(Steffens et al., 2000; Lloyd, Ferrier, Barber, Gholkar, Young, & O'Brien, 2004; Hickie et al., 2005). Sheline and colleagues claimed that recurrent depression and longer duration of depressive episodes have been associated with hippocampal volume reduction (Sheline et al., 1996; Sheline et al., 1999; Sheline et al., 2003). Others have examined hippocampal volume in late-life depression and subsequent cognitive decline. In one study of depressed patients, declines in hippocampal volume were significantly associated with continuing memory deficits after 6 months (O'Brien et al., 2004). Hence, hippocampal morphology in depressive mood and memory function may play a role in the associative link between the two.

Burt and colleagues conducted a meta-analysis on the association between depression and memory impairment, particularly for recall and recognition memory. According to their study, both recall and recognition were significantly impaired among the depressed subjects. However, contrary to the current study, they found significant results for short-term recall in addition to long-term recall (Burt et al., 1995). This may be due to the fact that the studies included in their meta-analysis involved individuals meeting diagnostic criteria for depression or those described as being clinically depressed. The greater magnitude in depressive mood compared to the subjects included in this study may explain the greater memory deficit that includes short-term recall impairment. Indeed, recent studies have shown that the hippocampus may be critical for short-term memory for associative information (Jonides et al., 2008). In recent years, there has been accumulating evidence suggesting that short-term memory and long-term memory, rather than being qualitatively distinct, may in fact share similar underlying neural mechanisms.

Also contrary to the current results, Fossati and colleagues found deficits in free recall among depressed inpatients, but preserved recognition memory (Fossati et al., 2002). Ilsley and colleagues found congruent results, with impairments on immediate and delayed recall among patients, but intact recognition (Ilsley et al., 1995). These results are difficult to interpret, and further studies examining neuroimaging data may be helpful in providing an explanation. The subjects in the current study may have had more damage to the medial temporal lobe cortex (MTLC), whereas the subjects from Fossati and Ilslley's studies had focal lesions in the hippocampus, but this is only a presumption and further studies will be necessary.

There is widespread agreement that the hippocampus is critical for recall, with focal hippocampal lesions leading to significantly impaired recall performance. However, the data is more controversial regarding effects of focal hippocampal damage on recognition. Some studies have found approximately equal impairments in recall and recognition (Reed & Squire, 1997; Manns & Squire, 1999; Rempel-Clower, Zola, Squire, & Amaral, 1996), while others have found relatively spared recognition after focal hippocampal lesions (Vargha-Khadem et al., 1997; Holdstock et al., 2002; Mayes, Holdstock, Isaac, Hunkin, & Roberts, 2002). It has been said that spared recognition, following hippocampal lesions, depends critically on MTLC; whereas recognition is sometimes spared by focal hippocampal lesions, it is never spared after lesions in both MTLC and the hippocampus (Aggleton & Shaw, 1996). In other words, recognition may involve the hippocampus, but not depend solely on it. This may explain the discrepancy seen between the current results and others.

2. Cognitive reserve moderates the effect of depressive mood

From the literature, it can be hypothesized that individuals with more cognitive reserve would be more successful at coping with the same amount of brain pathology, which may be accelerated by depressive mood. This study was conducted in order to investigate whether cognitive reserve can act as a protective factor by moderating the deleterious effects that depressive mood has, especially on the hippocampus.

The results of this study did not support my second hypothesis that a composite factor score of cognitive reserve, including high educational attainment and premorbid intelligence, will moderate the effect between depressive mood and memory impairment. However, when taking a look at the two proxies of cognitive reserve separately, it appeared to be that the level of educational attainment had a significant moderating effect between depressive mood and memory impairment for both long-term recall and recognition, whereas premorbid verbal intelligence did not have a significant moderating effect. Thus, education serves as a greater protective factor of memory impairment than premorbid IQ.

This is consistent with the abundance of studies claiming that education reduces the risk of cognitive decline (Blum & Jarvik, 1974; Colsher & Wallace, 1991; Evans et al., 1993; Snowdon, Kemper, Mortimer, Greiner, Wekstein, & Markesbery, 1996; Schmand et al., 1997a). It also further supports Carret and colleagues' (2003) claim that education is the most important factor in reducing decline. However, not finding significant results for the protective role of premorbid intelligence contradicts the several studies that indicated premorbid IQ as a good predictor, if not a more powerful one, of

reduced risk of functional decline (Albert & Teresi, 1999; Alexander et al., 1997; Schmand et al., 1997).

What this implies is that education, or other experiences involving cognitively stimulating activity, contributes more to cognitive reserve than that obtained from intelligence. Studies have showed separate and synergistic effects for higher educational and occupational attainment and leisure activities, which also involve activation of the brain (Evans et al., 1993; Mortel, Meyer, Herod, & Thornby, 1995; Rocca et al., 1990; Stern, Gurland, Tatemichi, Tang, Wilder, & Mayeux, 1994; Stern et al., 1995), suggesting that cognitive reserve is not fixed, and that at any point in one's lifetime it results from a combination of exposures rather than simply intelligence itself.

The non-significant findings of premorbid intelligence as a moderating factor may have been due to the fact that vocabulary knowledge does not accurately represent premorbid intelligence among my sample. Although vocabulary knowledge has been claimed to be a good indicator of intelligence, recent findings have suggested that there may be a limit to how well vocabulary knowledge represents general cognitive functioning and intelligence among individuals with low educational attainment. In individuals with 6 or less years of education, vocabulary test scores did not correlate with the individuals' overall cognitive ability, and therefore may not be an appropriate measure of premorbid IQ, whereas in those with more than 6 years of education, vocabulary test scores significantly correlated with overall cognitive ability (Park, Chey, Kim, & Lee, 2016).

Still, the accumulation of cognitive activity and mental stimulation can significantly reduce the risk of developing memory impairments later on in life, even in the context of depressive mood. This in turn suggests that encouraging more learning and mental activity has the potential to produce significant results in the health and well-being of the elderly population. This is one of the first findings regarding cognitive reserve, particularly educational attainment, as a protective factor against the effects of depressive mood on memory impairment.

3. Implications

In line with the cognitive reserve hypothesis, results from a number of studies suggest that an enriched environment may promote brain development or sustainability since it keeps the brain active and neurons constantly stimulated (Swaab, 1991; Valenzuela et al., 2008). This reserve allows individuals greater neural efficiency, greater neural capacity, and the ability for compensation, and in turn, alters the rate of cognitive decline in elderly people. The concept of cognitive reserve implies that a thorough education and other means of cognitive activity results in having a greater cognitive reserve, and that mental stimulation during later life maintains or further increases the reserve as well. Cognitive reserve is not fixed but continues to evolve across the lifespan. Thus, even late-stage interventions hold promise to boost cognitive reserve and thus reduce the prevalence of Alzheimer's disease and other age-related problems.

It is important to keep in mind that higher cognitive reserve delays the onset of

clinical presentation rather than truly decreasing dementia incidence. Yet, from a public health perspective, a delay of 5 years could potentially halve the prevalence of the disease (Katzman, 1993), and in turn, lead to significant personal, social, and economic benefits. Furthermore, targeting cognitive reserve for interventions provides the advantage of having minimal potential for harm, as opposed to using drug treatments. It could also increase the general quality of life for elderly people, by encouraging social engagement and more activity.

Evidence from previous studies show support for the reduction of cognitive decline from the enhancement of reserve through cognitive training. Schaie (1994) found that a program based on problem solving reversed 14-year decline in cognitive ability in 40 percent of the participants. Another team trained a group of healthy elderly in verbal memory to supra-normative levels and showed that the advantage persisted for at least several weeks (Kliegl et al., 1989). Ball and colleagues (2002) showed that 10 sessions of cognitive training in healthy elders could lead to cognitive improvement over 2 years follow-up as opposed to an expected cognitive decline.

Researchers are also beginning to discover possible neurobiological mechanisms behind the apparent advantage of mental stimulation. Valenzuela and colleagues (2003) have shown that 5 weeks of memory-based mental exercise increased resting phosphocreatine levels—which usually declines in early dementia—in the medial temporal lobe of healthy elders. Animal studies have revealed a diverse range of structural brain changes when environmental complexity was enriched, including neurogenetic, synaptic and dendritic changes (van Praag et al., 2000). Furthermore, the

capacity to use compensatory networks, when the typical brain networks fail to activate, have been shown to correlate with cognitive reserve measures such as education and leisure activities (Habeck et al., 2003; Scarmeas et al., 2003; Springer et al., 2005). Thus, focusing our efforts into developing and improving cognitive training programs can have significant effects for the cognitive well-being of our aging society, even for those who display subthreshold depressive mood.

4. Limitations

This study has its limitations in that the subjects are limited to only women. However, in South Korea, a country that has been through much poverty and political turmoil, as well as a tradition of reprimanding female education until the second half of the 20th century, the discrepancy in formal educational attainment was significantly different between genders. Such unfavorable socioeconomic circumstances have limited an individual's opportunities to receive appropriate education, which resulted in males tending to have a higher level of education compared to females. Hence, when dividing the sample into a high education group and low education group, as this study did, it would result in a group almost between genders and not just years of formal education. Including only elderly Korean women in this sample has allowed for this factor to be controlled, but at the cost of only representing half of the population.

The current study also has the inevitable limitations of a cross-sectional study. The level of depressive mood and cognitive performance were assessed within a short

time frame, so the issue of the temporal relationship between the two variables remains unaddressed from this study. Evaluating clinical change, through longitudinal studies, rather than status would provide stronger evidence of the claims being made. The nature of the association between depression and cognitive decline or dementia is not simple, with the possibility that depression may be a prodromal symptom of dementia rather than a risk factor for the disorder. Several researchers have discussed the uncertainty that exists on this issue and concluded that there is insufficient evidence to determine whether depression represents an independent risk factor or an early symptom in dementia (Jorm, 2001; Dal Forno, Palermo, Donohue, Karagiozis, Zonderman, & Kawas, 2005). However, a meta-analysis from Ownby and colleagues evaluated whether the risk for developing dementia was related to the interval between diagnoses of depression and dementia and found that this interval was positively and significantly related to the odds of developing dementia (Ownby, Crocco, Acevedo, John, & Loewenstein, 2006). It can be interpreted that the occurrence of depression is a risk factor rather than a prodrome of the disorder. Another study found that in individuals whose depression symptoms first occurred more than 25 years before the onset of AD, there was still a modest association (Green et al., 2003), further strengthening the observation that depressive symptoms are indeed a risk factor for later development of dementia. However, it should be recognized that these findings do not rule out the possibility that depression may be both a remote risk factor for dementia as well as a prodromal feature of it.

It should also be mentioned that the GDS and EVLT were not administered together. All subjects were administered the GDS first, and then several months later

were given cognitive testing. The results of this study could have been affected by any mood changes or cognitive impairment that may have occurred in-between the two tests. The time gap between the two assessments, which should have been nonexistent or at least controlled, varied between subjects, ranging from 4 months to as long as 9 months. Test-retest correlations for the GDS are high (r=.80 to .98; Ingraham, 1996; Parmelee, Katz, & Lawton, 1989; Laprise & Vezina, 1998; Lyons et al., 1989) over intervals of 1 week to 2 months. Yet, test-retest reliability is still reasonable after intervals of about 6 months (>.70) for cognitive intact individuals (Bedard et al., 2003).

5. Future Considerations

The current study investigated the protective effect of cognitive reserve in moderating association between depressive mood and memory impairment among elderly women in Seoul. However, in order to address the protective effect more thoroughly, a study including both genders should be conducted. Doing so would provide stronger support for the generalization to be made. Also, the subjects in this study did not display a wide variation in regards to socioeconomic status. The majority of the subjects described themselves as being in the lower or lower-middle class. Future studies including elders from the middle and upper class as well should be conducted.

Only years of education and premorbid verbal intelligence were used as proxies of cognitive reserve in this study. There was not enough variety in types of occupations held, with the majority being housewives or farmers throughout most of their lives, so

occupation could not be included as a measurement of reserve. Cognitive lifestyle was not included in the analysis as well, even though stimulating mental activity during later life maintains or further increases cognitive reserve. As previously mentioned, an active life-style characterized by complex social functioning and diverse leisure activities has been found to protect against cognitive decline and dementia (Fabrigoule, Letenneur, Dartigues, Zarrouk, Commenges, & Barberger-Gateau, 1995). Thus, if we want to investigate the protective role of cognitive reserve, it would be desirable to get a more comprehensive account of cognitive reserve by taking into account the quality of mental activity during the post-education years, especially since this factor could vary greatly between subjects. An individual may have received little formal education but continued to engage in mentally stimulating activities throughout life, in which case his or her cognitive reserve could still be high.

Also, stronger support can be drawn regarding the moderating effect of cognitive reserve using brain imaging data. The results of this study would be strengthened if we saw that brain volumes for associated regions, such as the hippocampus and other medial temporal lobe cortices, are affected by depression and also correlate with memory function.

All in all, the present study provides one of the first findings regarding cognitive reserve as a protective factor against the effects of depressive mood on memory impairment. Multiple factors may affect brain development and aging; some of them act as accelerators of age-related declines, and others display a potential for slowing age-related deterioration and delaying its advancement to pathological levels. This study

suggests good news—cognitive reserve—that may reduce the effects of the bad—depressive mood, and what this means for potential interventions.

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국문 초록

우울감에 따른 기억기능 감퇴와 인지 보유고의 조절효과

이 지 윤

임상신경심리학 전공

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노화에 따른 변화는 다양한 양상으로 나타나며 인지기능에서도 개인차가 존재한다. 인지 보유고 가설에 따르면 더 높은 수준의 인지 보유고를 가진 노인이 그렇지 않은 노인들 보다 기억력을 포함하여 더 나은 인지 기능을 보인다. 그리고 임상 증상을 나타내기 전에 뇌 병리를 더 잘 견딜 수 있다(Scarmeas & Stern, 2004). 즉, 동일한 뇌 병리를 가지고 있어도, 인지 보유고가 높은 노인은 신경 효율성이 높으므로 병리에 잘 대처 할 수 있으며, 치매 발병이 수 년씩 늦추어진다.

우울증 환자들은 기억 과제에서 수행능력이 비교적 저조하며, 우울증은 치매 위험을 상당히 증가시킨다(Cohen et al., 1982; Burt et al., 1995). 그리고 노년기 우울 증상을 지닌 사람들은 그렇지 않은 사람들에 비해 더

많은 인지기능 감퇴를 나타났다. 또한, 노년기 우울증 환자들에게 기억 감퇴가 특징인 치매가 발생할 확률이 2배에서 5배까지 높다(Saczynski et al., 2010; Andersen et al., 2005).

이 연구는 교육과 병전 지능에 의해 측정된 인지 보유고가 우울한 기분의 기억 기능에 미치는 부정적인 영향을 조절함으로써 이를 완화 할 수 있는지 여부를 검토하고자 한다. 서울에 소재하는 8개의 사회복지관에서 모집한 79명의 건강한 비치매성 노인들을 대상으로 연구를 하였다. 노인 우울 척도(GDS)로 우울한 기분을 측정 하였고, 노인용 언어 학습 검사(EVLT)로 단기 회상 기억, 장기 회상 기억 및 재인 기억을 측정하였으며, 한국판 웩슬러 성인용 지능검사 제4판(K-WAIS-IV) 어휘 소검사에서의 점수를 병전 지능의 지표로 하였다. 인지 보유고 지표를 만들기 위해 교육 연한과 병전 지능 지표를 측정치로 사용하였다.

교육 및 K-WAIS-IV 어휘 소검사를 통해 얻은 인지 보유고 지수를 기초로 다중 회귀 분석을 사용하여 조절 효과를 검토하였다. 종합적 측정치를 통하여 조절 효과를 검토한 결과, 우울한 기분과 기억손상 사이에 유의미한 조절 효과가 나타나지 않았다. 그러나 인지 보유고의 각각 측정치가 가지는 조절 효과를 살펴보았을 때, 교육 효과는 우울한 기분과 기억손상 사이의 관계를 유의미하게 조절하는 것으로 나타났다. 한편 어휘 소검사로 추정한 병전 지능은 우울한 기분과 기억기능 감퇴의 관계를 유의미하게 조절하지

않았다.

따라서 정규 교육이 노화와 관련된 인지기능 저하를 유의미하게 감소시킴으로써 보호 요인으로 작용한다. 그런데 병전 지능은 그 같은 효과를 나타내지 않았다. 우울한 기분의 정도가 기억 수행에 유의미한 영향을 미치며, 특히 저교육자들에게 더 큰 영향을 미친다. 그러나 고교육자들에서는 우울한 기분에도 불구하고 상대적으로 기억 수행이 안정적으로 유지되는 형상을 볼 수 있다. 이는 교육이 기억 손상을 크게 완화하는 보호 요인으로 작용한다는 것을 시사한다.

주요어: 인지 노화, 인지 보유고, 우울감, 기억기능, 교육

학 번: 2013-22823