To Remember or To Not Remember: Successful Memory Aging

Melissa Johnson

University of North Texas

To Remember or To Not Remember: Successful Memory Aging

**Introduction**

What is memory and why is it important? What areas of the brain affect memory? What happens in people’s brains as they age? Why do some people age better than others? What is different about the successfully aging older people compared to those who do not age well? Are there outside factors that affect aging and memory or is it only a neurological process? This paper will focus on research that explains the concept of memory, where it occurs in the brain, what happens to people’s brains as they age, why some people exhibit successful memory aging and how they differ from those who do not, and some factors that have been proven to affect how people age.

**Memory overview**

Memory is a complex cognitive function. Essential to people’s lives, it has a profound effect on them from the day they are born to the day that they die. Memory can be defined as “the processes involved in retaining, retrieving, and using information about stimuli, images, events, ideas, and skills after the original information is no longer present” (Goldstein, 2011, p. 116). Memory involves both conscious and unconscious thought processes. Without memory, many of the everyday functions that people take for granted would no longer be possible, such as recalling the name of the current president or remembering how to drive home (Goldstein, 2011).

Multiple classification systems exist for memory. This paper will briefly discuss two systems as they both cover the concept of memory in detail including its history. One system divides memory into four main areas: episodic memory, which includes both short-term memory (STM) and long-term memory (LTM); semantic memory; working memory (WM); and procedural memory (Warsi, Lyubkin, & Kales, 2008). Another system divides memory into the two main areas of LTM and STM (Brickman & Stern, 2009; Goldstein, 2011). It includes WM as part of STM. LTM is further subdivided into the two areas of declarative and nondeclarative memory. Declarative memory includes the subdivisions of semantic, episodic, and source memory. Nondeclarative memory includes the subdivisions of procedural memory and priming.

The first model of memory, the “modal model of memory,” proposed by Richard Atkinson and Richard Shiffrin (1971) has provided the foundation for which all succeeding research on memory derives. This model divided memory into three main areas: sensory memory, STM, and LTM. Sensory memory is the beginning memory stage that interacts with a person’s environment, holds incoming information for seconds or less and then transmits that information into STM. STM has a limited capacity of around five to seven items and a limited duration of fifteen to thirty seconds. LTM, unlike STM, can hold vast amounts of information for extended and even unlimited time periods (Atkinson & Shiffrin, 1971; Brickman & Stern, 2009; Goldstein, 2011).

More recent research shows that the capacity of STM is more limited than originally thought and is about four items (Brickman & Stern, 2009; Goldstein, 2011; Warsi et al., 2008). Due to the limited capacity and duration of STM, some information may be forgotten and so is not transferred, or encoded, into LTM. For information to be successfully retrieved, or remembered, it has to travel from LTM back into STM through conscious awareness. Although STM and LTM are separate parts of memory, they must interact with each other to successfully encode and retrieve information (Brickman & Stern, 2009; Goldstein, 2011; Warsi et al., 2008).

Somewhat limited, the model above did not take into account how people can both store items in their memory and actively perform tasks at the same time, such as reading a paragraph while remembering a phone number. Alan Baddeley and Graham Hitch (1974) proposed the working memory (WM) model to account for that shortcoming. WM is defined as a “limited-capacity system for temporary storage and manipulation of information for complex tasks such as comprehension, learning, and reasoning” (Baddeley, 2000, p. 418).

WM differs from STM because it involves more than just storing a single piece of information. WM involves the manipulation of multiple pieces of information at the same time. The working memory model consists of the phonological loop, which processes verbal and auditory information, the visuospatial sketch pad, which processes visual and spatial information, and the central executive, which mediates information between the phonological loop and the visuospatial sketch pad while ignoring irrelevant information (Baddeley & Hitch, 1974; Brickman & Stern, 2009; Goldstein, 2011; Warsi et al., 2008). Baddeley (2000) modified his working memory model to add the episodic buffer, which mediates the retrieval of information from LTM to the central executive and can also store information that temporarily increases WM capacity. Due to similarity of WM and attention, some recent studies cited by Goldstein (2011, p. 137) debate the actual existence of WM as they consider it to be the same as attention.

LTM is divided into the overarching areas of declarative, or conscious memory, and nondeclarative, or unconscious memory. Declarative memory is further divided into the areas of episodic, semantic, and source memory. Episodic memory involves people’s consciously recalled memories for personal experiences or events. Semantic memory involves people’s consciously recalled memories for facts and their stored knowledge (Brickman & Stern, 2009; Goldstein, 2011; Warsi et al., 2008). Source memory includes people’s episodic memories along with the context in which the memories were acquired (Brickman & Stern, 2009).

Nondeclarative memory is further divided into the areas of procedural memory and priming (Brickman & Stern, 2009; Goldstein, 2011; Warsi et al., 2008). Procedural memory includes the unconscious skills that exist in our memories, such as driving a car or walking. Priming includes a person’s unconscious change in response to one stimulus, the test stimulus, due to the presence of another stimulus, the priming stimulus, such as preferring one brand of clothing, the test stimulus, over another due to repeated viewings of a commercial featuring that brand, the priming stimulus (Goldstein, 2011). Priming can take the form of repetition priming, in which the test stimulus is the same or similar the priming stimulus; or it can take the form of conceptual priming, in which the meaning of the test stimulus is influenced by the meaning of the priming stimulus (Brickman & Stern, 2009; Goldstein, 2011).

Where do the different types of memory occur in the brain? Memory is a complex process. While some types of memory more heavily involve certain areas, the entire memory system as a whole is distributed across various areas of the brain. STM and WM both mainly involve the prefrontal cortex area of the brain (Goldstein, 2011; Warsi et al., 2008); however, studies cited by Goldstein (2011, p. 140) show that other areas, such as the frontal lobe, the primary visual cortex, the temporal lobe, the parietal lobe, and the cerebellum, are involved as well.

LTM, specifically episodic memory, largely involves the medial temporal lobe (MTL), which includes the perirhinal cortex, parahippocampal cortex, entorhinal cortex, and the hippocampus (Goldstein, 2011; Warsi et al., 2008). The hippocampus is especially important with regard to the LTM as damage to this area inhibits new LTM formations; the parahippocampal cortex involves spatial recognition, and the entorhinal and perirhinal cortices involve recognition memory (Goldstein, 2011, pp. 192-93). Other areas of the brain that involve LTM formation are the frontal lobe, the amygdala, for emotional memories, the parietal lobe, the inferior lateral temporal lobe, for semantic memories and the basal ganglia, the cerebellum, and the supplemental motor area for procedural memories (Goldstein, 2011; Warsi et al., 2008). As the research shows, although specific types of memory are localized to certain areas, the entire memory process as a whole involves multiple areas of the brain.

**Memory and Aging**

“By age 2030, 72 million Americans will be aged 65 or older, a two-fold increase from 2000” (Ramscar, Hendrix, Shaoul, Milin, & Baayen, 2014, p. 5). As people age, their cognitive abilities, including memory, decline. Studies that focus on intelligence divide it into two broad areas, that of fluid and crystallized. Fluid intelligence, i.e., episodic and WM, includes biologically derived knowledge and skills, such as abstract reasoning, spatial orientation, and perceptual speed. Crystallized intelligence, i.e., semantic memory, includes experiential and educational knowledge, such as verbal comprehension, word association, and social judgment (Diggs, 2008). Timothy Salthouse (1999) defines fluid cognition as “the efficiency or effectiveness of processing at the time of assessment and is typically evaluated with task of learning memory, reasoning, and spatial abilities” (p. 196). Studies comparing the cognitive abilities of older adults show that fluid intelligence abilities decrease, specifically that of WM, processing speed, and the ability to impede irrelevant information; however, crystallized knowledge abilities do not decrease with age (Diggs, 2008).

Not all types of memory decline at the same rate or at the same age for people. Age-related memory decline varies from person to person as well as across studies. Cross-sectional studies, which are studies that compare both young and older adults, as well as longitudinal studies, which are studies that follow the same group over a period of years, have all been undertaken to offer some insight into this area.

Some cross-sectional research shows that WM and episodic memory, specifically the ability to form new episodic memories, start to decline in the twenties and continues declining throughout the remainder of people’s lives; whereas, more accurate longitudinal research that controls for cohort and practice effects shows WM and episodic memory do not start declining until the sixties (Nyberg, Lövdén, Riklund, Lindenberger, & Bäckman, 2012). Semantic memory, which can even increase with age, and STM, with regard to the number items people are able to hold in their brains, tends to remain the same across the lifespan and does not decline until the late seventies or older. Autobiographical memory, emotional memory, and procedural memory show no declines at all (Brickman & Stern, 2009; Hedden & Gabrieli, 2004).

What happens in the brain as people age? Brain imaging techniques, such as functional magnetic resonance imaging (fMRI), positron emission tomography (PET), and diffusion tensor imaging (DTI), show that for healthy, older adults the biggest areas of difference appear in structural decreases in both grey matter and white matter in the brain in the prefrontal cortex (PFC), minimal volume loss in the hippocampus and parahippocampal gyrus, and a decrease in dopamine receptors. All of which are areas that serve WM processes and the formation of new episodic memories (Hedden & Gabrieli, 2004). In contrast, the brain images of adults displaying signs of mild cognitive impairment (MCI), which later progresses into dementia and then, Alzheimer’s disease, show the largest decline in the MTL, particularly in the hippocampus and the entorhinal cortex areas (Hedden & Gabrieli, 2004). Diseases such as hypertension, cardiovascular disease, and Type 2 diabetes are more aligned with early onset of MCI and consequent memory issues (Aine et al., 2011; Brickman & Stern, 2009; Hedden & Gabrieli, 2004).

A suggestion stated by multiple authors (Aine et al., 2011; Brickman & Stern, 2009; Hedden & Gabrieli, 2004) is that more research needs to be undertaken on middle-aged and older adults as opposed to research that compares young adults to older adult. In addition, studies that focus solely on older adults needs to weed out the adults showing signs of pathological decline and undertake more intensive research on those adults who seem impervious to aging as the mixed aging levels skew results and do not provide an accurate view of normal aging (Hedden & Gabrieli, 2004).

**Successful Memory Aging**

Why do some adults show little or no cognitive decline, specifically with regards to memory, as they age? The answer in short is that no definitive answer exists while much debate does. Summaries of some of the conflicting reviews are discussed below.

A large volume of research (Cabeza, Anderson, Locantore, & McIntosh, 2002; Manenti, Cotelli, & Miniussi, 2010; Reuter-Lorenz, 2002; Reuter-Lorenz & Lustig, 2005) agree that successful memory agers “counteract age-related cognitive decline by reorganizing brain functions” (Cabeza et al., 2002, p. 1394). Within this focus of research a debate exists on whether the reorganization, or plasticity, is due to compensatory activity or due to dedifferentiation with a significant amount of research supporting compensatory activity (Cabeza et al., 2002; Manenti et al., 2010; Reuter-Lorenz, 2002; Reuter-Lorenz & Lustig, 2005).

The compensation hypothesis is based upon the “Hemispheric Asymmetry Reduction in Old Adults” (HAROLD) model and states that older adults recruit additional regions in their brains to help compensate for more difficult WM and episodic memory tasks (Cabeza et al., 2002, p. 1394). The dedifferentiation hypothesis states that age-related cognitive declines cause older adults to recruit additional brain regions regardless if those regions help them or not with difficult WM and episodic memory tasks, mostly due to their inability to selectively recruit and efficiently use the necessary brain regions (Reuter-Lorenz & Lusting, 2005).

Using a variety of brain imaging techniques during studies focusing on verbal WM and episodic memory tasks, the authors measured what happens in high-performing older adults’ brains, i.e., successful memory agers, compared to low-performing older adults. The imaging results proved the HAROLD model in that the high-performing older adults showed bilateral activation in the PFC area of the brain; whereas, the low-performing older adults used only one side of the PFC, lateralization, to perform the memory tasks. These studies disproved dedifferentiation in that the low-performing adults did not display bilateral brain activations (Cabeza et al., 2002; Manenti et al., 2010; Reuter-Lorenz, 2002; Reuter-Lorenz & Lustig, 2005). This theory shows that successful memory agers use more of their brain to help them accomplish difficult memory tasks, i.e., compensatory activity.

One study using fMRI imaging compared a group of “SuperAgers” in their eighties with a group of normal, same-aged elderly adults, and a middle-aged group using verbal episodic memory tasks (Harrison, Weintraub, Mesulam, & Rogalski, 2012). This study showed that the cortical area of the “SuperAgers” brains was much thicker than those of the normal elderly adults and that compared to the brains of the middle-aged group, the brains of the “SuperAgers” did not show any atrophy, which is in contrast to typical reports of decreased grey matter volume (Harrison et al., 2012; Hedden & Gabrieli, 2004).

Furthermore, the “SuperAgers” brains also showed an increased volume in the left anterior cingulate area of the brain compared to both the middle-aged and normal elderly groups. Harrison et al. (2012) state that it is currently unknown why there are differences in the size of the cingulate area and propose it could be a feature they were born with or that developed over time. This study also found that the memory abilities of the “SuperAgers” was considered normal when they were younger and that the education level of this group was not unusually high as only four of the twelve “SuperAgers” actually earned a college degree (Harrison et al., 2012); however, as this is a small sample the research may not accurately represent the population as a whole. This research suggests that a healthy brain due to genetics, environment, or a combination of both plays a role in why some people age better than others.

Another study using a variety of imaging techniques, fMRI, PET, DTI, and blood oxygen level dependent signal testing reviewed the concept of brain maintenance as the deciding factor that affects how people’s brains age (Nyberg et al., 2012). This study began by discussing the concepts of brain reserve and cognitive reserve. Brain reserve states that the ability to cope effectively with pathological changes is dependent upon the amount of intact brain. Cognitive reserve states that how people actively approach tasks is what permits them to cope effectively with pathological changes, similar to the compensation theory (Nyberg et al., 2012).

Nyberg et al. (2012) proposed the concept of brain maintenance as complementary topic to the idea of cognitive reserve. They defined brain maintenance as “individual differences in the manifestation of age-related brain changes and pathology allow some people to show little or no age-related cognitive decline” (Nyberg et al., 2012, p. 295). The main idea behind brain maintenance is that little or no neural changes in the brain combined with healthy aging are the best predictor of successful memory aging. Their imaging results showed that the participants classified as successful agers showed no pathological-related issues, recruited appropriate brain regions, specifically that of the PFC area, to accomplish episodic and WM tasks, and did not have decreases in grey and white matter volume in either the PFC or the hippocampal area in the MTL (Nyberg et al., 2012).

How did the study participants maintain their brains? The research undertaken by Nyberg et al. (2012) showed that brain maintenance ultimately relies on a combination of genetics and environment. Some people are simply born with better genes. An example they shared is the brain-derived neurotropic factor, or BDNF gene, that works with learning and memory by increasing brain plasticity. The older adults’ brains that had less of this gene performed more poorly on the memory tasks.

Another factor is the environment in which the successful agers currently live. Surprisingly, environment was not dependent upon the education of the successful agers as the research showed that higher educational levels may help with brain reserve but not with cognitive reserve or brain maintenance. The main part of the environment that predicted successful memory aging was the ability of the older adults to continue participating in socially, mentally, and physically engaging activities. Particularly those activities that mimicked the higher-cognitive skills that they engaged in while still participating in the workforce (Nyberg et al., 2012). Essentially, older adults who maintain a socially, mentally, and physically active lifestyle during retirement are the ones who show little or no cognitive decline and who also maintain their cognitive levels from earlier years.

A completely contrary point of view to all the research that exists on cognitive decline (Ramscar et al., 2014) states older adults’ cognitive abilities do not in fact decline with age, unless they experience pathological-related changes in the brain. This study states that because older adults have lived longer they have amassed more knowledge and memories. When presented with episodic and WM tasks, older adults take longer to process the information because they have more information in their brains to sift through, e.g., a larger vocabulary and more prior knowledge.

For example, they address the common issue of adults forgetting people’s names as caused by an increase in the number of available names due to a larger population plus the added complexity of using common words now as names, e.g., Apple, and that older adults have met more people over their lifetime (Ramscar et al., 2014). These authors make the argument that all the research that exists on cognitive decline is inhibiting older adults’ abilities to function and that new research should continue to focus on debunking declines in cognitive abilities. In addition, the positive outlook from this research should be shared with more people, especially those who interact with elderly people (Ramscar et al., 2014). Perhaps more research in this vein will help conclusively prove that cognitive decline is in fact due to pathological changes in the brain and not age.

**Strategies for reducing cognitive decline in memory**

Some common suggestions to combat memory decline that appeared in the research are briefly discussed. Studies performed on elderly adults who engaged in cardiovascular exercise over a period of weeks showed PFC neural activity that was similar to young adults in their twenties (Aine et al., 2011; Nyberg et al., 2012; Reuter-Lorenz & Lustig, 2005). A study undertaken on a group of healthy middle-aged adults demonstrated that using Lumosity, an online brain training program, for just twenty minutes a day for five weeks with targeted training for visual attention and WM caused significant improvements in their cognitive abilities that translated to other tasks requiring divided visual attention and spatial WM skills. A suggestion from this study is that by implementing focused cognitive training early in life, healthy adults not only improve their current cognitive abilities but could also combat cognitive decline in later ages (Hardy, Drescher, Sarkar, Kellett, & Scanlon, 2011).

Other suggestions to prevent memory decline are to minimize stress and to focus attention by using strategies, in the form of instructions (Brickman & Stern, 2009; Hedden & Gabrieli, 2004; Warsi et al., 2008). Staying intellectually engaged, such as learning a new language or studying new topics, and maintaining a good social network helps older adults to combat cognitive decline because it elicits an environment that requires high-levels of functioning (Nyberg et al., 2012). Maintaining a healthy diet can also help prevent cognitive decline as research shows that older adults who develop cardiovascular disease, hypertension or Type 2 diabetes are more likely to show signs of MCI and later progressions into Alzheimer’s disease (Aine et al., 2012; Brickman & Stern, 2009; Hedden & Gabrieli, 2004).

**Conclusion**

Memory is a complicated process that involves multiple areas of the brain. Cognitive decline as it affects memory occurs during aging; however, the amount of decline as well as the age of onset varies by individual. Longitudinal research studies show that cognitive decline does not begin to occur until the sixties, and both episodic and working memory are the structures most affected as healthy people age with decreases in volume of both grey and white matter tracts in the prefrontal cortex and to some extent in the volume of the hippocampus.

Some older adults, however, demonstrate little or no cognitive decline in advanced ages and are considered to be successful memory agers. Some researchers believe it is due to compensatory activity, in which older adults recruit multiple areas of the brain to successfully perform tasks; some take the view that older adults who had a thicker cortical area with no signs of atrophy were able to maintain brain performance; some believe that brain maintenance in the form of genetics and the environment determine successful aging; whereas, others deny the existence of cognitive decline outside of any pathological issues.

The main finding from the research is that although each person is unique and will age differently, everyone has the ability to take control of their lifestyle and proactively implement changes that will prevent or combat memory decline by maintaining a healthy lifestyle through diet and cardiovascular exercise along with an environment that is mentally and socially stimulating. In this way, everyone has the chance to be a successful memory ager.

References

Aine, C. J., Sanfratello, L., Adair, J. C., Knoefel, J. E., Caprihan, A., & Stephen, J. M. (2011).

Development and decline of memory functions in normal, pathological and healthy successful aging. *Brain Topography, 24*, 323-339. doi:10.1007/s10548-011-0178-x

Atkinson, R. C., & Shiffrin, R. M. (1971). *The control processes of short-term memory*

(IMSSS Technical Report No. 173). Retrieved from Stanford University, Institute

for Mathematical Studies in the Social Sciences, Collected Works of Patrick Suppes website: http://suppescorpus.stanford.edu/techreports/IMSSS\_173.pdf

Baddeley, A. (2000). The episodic buffer: a new component of working memory? *Trends in*

*Cognitive Sciences, 4*, 417-423. doi:10.1016/S1364-6613(00)01538-2

Baddeley, A. D., & Hitch, G. J. (1974). Working memory. In G. A. Bower (Ed.), *The psychology*

*of learning and motivation* (pp. 47-89). New York: Academic Press.

Brickman, A. M., & Stern, Y. (2009). Aging and memory in humans. In L. R. Squire

(Ed.), *Encyclopedia of neuroscience*(pp. 175-180). Oxford: Academic Press.

Cabeza, R., Anderson, N. D., Locantore, J. K., & McIntosh, A. R. (2002). Aging gracefully:

Compensatory brain activity in high-performing older adults. *NeuroImage*, *17*, 1394-

1402. doi:10.1006/nimg.2002.1280

Diggs, J. (2008). Cognitive theory of aging. In S. Loue & M. Sajatovic (Eds.),

*Encyclopedia of aging and public health* (pp. 216-218). New York: Springer.

doi:10.1007/978-0-387-33754-8\_92

Goldstein, E. B. (2011). *Cognitive psychology: Connecting mind, research, and everyday*

*experience* (3rd ed.). Belmont, CA: Wadsworth, Cengage Learning.

Hardy, J. L., Drescher, D., Sarkar, K., Kellett, G., & Scanlon, M. (2011). Enhancing visual

attention and working memory with a web-based cognitive training program. *Mensa Research Journal, 42*(2), 13-20. Retrieved from http://www.us.mensa.org/read/mrj/

Harrison, T. M., Weintraub, S., Mesulam, M. M., & Rogalski, E. (2012). Superior memory

and higher cortical volumes in unusually successful cognitive aging. *Journal of the*

*International Neuropsychological Society, 18,* 1081-1085.

doi:10.1017/S1355617712000847

Hedden, T., & Gabrieli, J. D. E. (2004). Insights into the ageing mind: A view from cognitive

neuroscience. *Nature Reviews Neuroscience, 5*, 87-96. doi:10.1038/nrn1323

Manenti, R., Cotelli, M., & Miniussi, C. (2010). Successful physiological aging and episodic

memory: A brain simulation study. *Behavioural Brain Research, 216*, 153-158. doi:10.1016/j.bbr.2010.07.027

Nyberg, L., Lövdén, M., Riklund, K., Lindenberger, U., & Bäckman, L. (2012). Memory aging

and brain maintenance. *Trends in Cognitive Sciences, 16*, 292-305.

doi.org/10.1016/j.tics.2012.04.005

Ramscar, M., Hendrix, P., Shaoul, C., Milin, P. and Baayen, H. (2014). The myth of cognitive

decline: Non-Linear dynamics of lifelong learning. *Topics in Cognitive Science, 6,*5–42. doi:10.1111/tops.12078

Reuter-Lorenz, P. A. (2002). New visions of the aging mind and brain. *Trends in Cognitive*

*Sciences, 6*, 394-400. doi:10.1016/S1364-6613(02)01957-5

Reuter-Lorenz, P. A., & Lustig, C. (2005). Brain aging: reorganizing discoveries about the aging

mind. *Current Opinion in Neurobiology*, *15*, 245-251. doi:10.1016/j.conb.2005.03.016

Salthouse, T. A. (1999). Theories of cognition. In V. L. Bengston & K. W. Schaie (Eds.),

*Handbook of theories of aging* (pp. 196-208)*.* New York: Springer.

Warsi, M. K., Lyubkin, M., & Kales, H. C. (2008). Memory. In S. Loue & M. Sajatovic (Eds.),

*Encyclopedia of aging and public health* (pp. 549-553). New York: Springer.

doi:10.1007/978-0-387-33754-8\_295