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Theories of Memory and Aging: A Look at the Past and a Glimpse of the Future

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Abstract

The present article reviews theories of memory and aging over the past 50 years. Particularly notable is a progression from early single-mechanism perspectives to complex multifactorial models proposed to account for commonly observed age deficits in memory function. The seminal mechanistic theories of processing speed, limited resources, and inhibitory deficits are discussed and viewed as especially important theories for understanding age-related memory decline. Additionally, advances in multivariate techniques including structural equation modeling provided new tools that led to the development of more complex multifactorial theories than existed earlier. The important role of neuroimaging is considered, along with the current prevalence of intervention studies. We close with predictions about new directions that future research on memory and aging will take.

Keywords: Cognition—Memory—Review—Theory

One of the most striking aspects of aging is that memory processes show decline. This was recognized as early as 700 BC by Solon, a Greek philosopher who, in his *Elegy on the Ages of Men*, noted that intellectual capacities began to diminish around age 56–63 (see Cokayne, 2003). Moreover, Virgil, a Roman poet, alluded to the degradation in memory over time in *Eclogues IX*, writing, "Time robs us of all, even of memory" (Cokayne, 2003, p. 67). Although some level of cognitive frailty has always been viewed as an aspect of aging, the study of cognitive aging came into its own right as the science of human behavior advanced and as significant increases in human longevity were realized. Today the study of memory and aging has taken on particular significance and is a center-stage issue due primarily to the increased longevity resulting from the heightened ability to extend life in the face of major diseases. Indeed, due to longevity, Alzheimer's disease has become a major cause of death and disability for society today, sparking a quest to develop early interventions to prevent or delay neuropathological aging.

In the present article, we review major theories of aging and memory and how they have emerged over the past 50 years (see Figure 1 for a depiction of the exponential growth in research on memory and aging from 1965 to 2013). We start by discussing some of the earliest empirical findings on aging and memory and follow by reviewing initial theoretical explanations for these findings. These early theories were surprisingly insightful, and many versions of them are still viable today. Contemporary models of memory decline build upon these early influential theories, and many have also begun to show a shift toward the brain. Hence, we summarize prominent mechanisms that have been hypothesized to explain why memory declines as we get older, noting early theories that are still dominant today along with newer theories that place additional focus on neural contributions to memory impairment. We also discuss specific memory domains that appear to be more
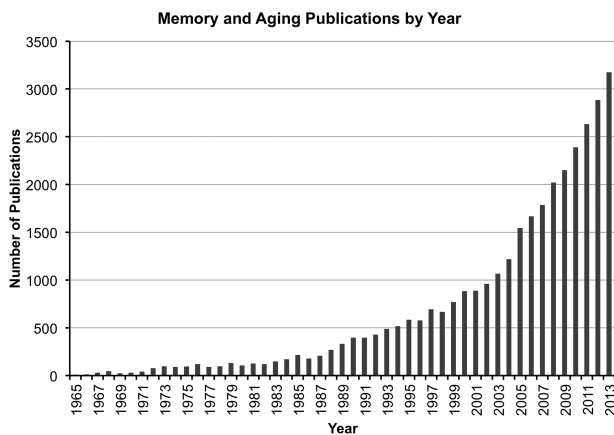
Figure 1. Depiction of the exponential increase in research on memory and aging. The number of articles published each year is plotted, after using Medline trend: Automated yearly statistics of PubMed results for any query (Corlan, 2004) and the query terms "memory" and "age". Website: http://dan.corlan.net/medline-trend.html

age invariant. Finally, we close with current trends in the field of memory and aging, providing reflections on what we expect for future research.

Early Evidence of Memory Impairment with Age

Some of the earliest findings about age differences in memory stem directly from verbal learning paradigms, which dominated the study of human memory when psychology was in the grip of behaviorism. In 1929, Willoughby documented a gradual age-related differences in incidental memory for digit–symbol pairs, such that recall decreased from age 20 through age 70. Moreover, using an intentional paired-associate learning paradigm, Ruch (1934) demonstrated that adults aged 60–82 years exhibited worse memory than adults aged 34–59 years, and that 12- to 17-year-olds displayed the best performance (for a detailed review, see Kausler, 1991). Some early work also assessed strategy use and noted that, compared with younger adults, older adults were less likely to use imagery or to create verbal mnemonics when attempting to remember paired associates (Hulicka & Grossman, 1967). Furthermore, older adults took longer to learn newly re-paired paired associates to criterion in an A–B A–C list-learning paradigm, indicating either greater negative transfer (i.e., interference) for older participants or greater positive transfer from repeated cue presentations in younger adults (Arenberg, 1967).

Another major early finding was that memory effects associated with age were typically larger when participants were asked to recall a list of words, compared with merely recognize them (e.g., Schonfield, 1965; Smith, 1977). Schonfield (1965) reported that older adults had equivalent recognition performance to younger adults but markedly impaired recall (but see e.g., Erber, 1974; Harwood & Naylor, 1969, who report both impaired recall and impaired recognition in older adults). Also of interest was

that picture memory appeared to be protected from dramatic age effects, as older adults recalled and recognized pictures better than words (e.g., Park, Puglisi, & Sovacool, 1983). Perhaps the most well-recognized finding of all was that on virtually any task that had a speed component, participants became slower with age (e.g., Brinley, 1965). Given these empirical results, scientists began proposing mechanistic theories to account for the commonly observed age differences in memory.

Theoretical Models of Aging and Memory Decline

Speed of Processing as a Mechanism of Memory Decline

The initial theories on speed were pioneered by James Birren, and then by John Cerella and Timothy Salthouse (e.g., Birren, 1965; Birren, Woods, & Williams, 1980; Cerella, 1985; Salthouse, 1996). In an early study, Birren noted that participants showed increasingly slower processing time for a broad range of cognitive tasks as a function of age (Birren, 1965), resulting in the hypothesis that slowed processing speed was a fundamental mechanism that governed many age deficits, including memory. Cerella suggested that slowing resulted from deletion of random links in the memory network, which created longer, more circuitous memorial processing paths (Cerella, 1990). In a large corpus of work beginning in the 1980s, Salthouse further validated and expanded this notion that processing speed was fundamental to explain age differences in memory (Salthouse, 1985a, 1985b, 1996). His view is best summarized in an article where he proposes that older adults are deficient in two important mechanisms that account for age-related differences in attention, memory, and reasoning (Salthouse, 1996). He posited a *limited time mechanism*, in which older adults have greater difficulty performing higher-level operations because it takes them longer to process early operations, and a *simultaneity mechanism*, in which older adults cannot consider as many task-relevant components together compared with younger adults because the products of earlier processing may not be available once ongoing processing is completed. Salthouse repeatedly showed that most age-related variance in cognitive tasks, including memory, could be accounted for by measures of speed.

The Processing Resource Model of Memory Deficits in Cognitive Aging

Age differences in levels of processing
In 1972, Craik and Lockhart presented the levels of processing theory of memory, which marked an important transition between basic stimulus–response verbal learning to the study of mental models. They provided evidence that an intention to learn was not the most critical component for remembering. Rather, it was the quality of the encoding

operation—not the time on task—that best predicted memory. Specifically, they reported that guiding participants to engage in deep semantic processing, even when they were not intentionally trying to remember, resulted in memory recall that was equivalent or superior to that of participants who were actively studying (see also Hyde & Jenkins, 1969). The finding that quality of processing could be more important than intention to learn was highly influential in the research community and led to the notion that older adults were deficient in spontaneously engaging in deep processing, as age effects were particularly large when learning was intentional. The inefficiency of older adults spontaneously performing higher-level encoding strategies was termed the *production deficit hypothesis* (Kausler, 1970). If, however, older adults were guided to process meaning and engage in elaborative encoding, memory could be repaired to be more similar to that of younger adults.

Environmental support

The notion that cognition could be repaired led logically to the concept of environmental support. Environmental support, also termed contextual support, involved the presentation of external cues or processing guidance, which provided "mental crutches" that made stimuli easier to remember, especially for older adults (e.g., Craik, 1986). For example, if during a list-learning experiment one was presented with the word "feather" to remember, the provision of the cue word "chicken" at encoding would be a type of environmental support that would facilitate recall of the studied word at test. Experimental research has demonstrated that older adults tend to have better memory when external cues are provided (e.g., Craik & McDowd, 1987; Smith, 1977). However, even when environmental support is afforded, age differences may be reduced but not entirely alleviated (e.g., Park & Shaw, 1992). There is a lengthy literature suggesting that the amount of mental effort required to effectively utilize environmental support determines its effectiveness in minimizing age deficits. For instance, fewer age deficits are observed in familiar situations, whereas novel experiences that require substantial self-initiated processing are more difficult for older adults (e.g., see Craik, 1994; Park & Gutchess, 2000).

Age-limited processing resources

The discovery that external cues could facilitate memory for older adults contributed to two further theoretical mechanisms of age-related memory decline. First was the hypothesis that older adults possessed fewer *processing resources*, also termed *attentional resources* and *mental energy*, and that the limited availability of processing resources served to restrict the quality and quantity of memory operations (Craik & Byrd, 1982). Older adults' limited resources were subsequently proposed to induce *general processing* that led to retention of broad semantic information but left a deficit in specificity (e.g., Rabinowitz & Ackerman, 1982; Rabinowitz, Craik, & Ackerman, 1982). Under conditions

of divided attention (i.e., thought to mimic the inherent impoverished processing capacity of older adults), younger adults were argued to show more general encoding because they exhibited better memory with general as opposed to specific retrieval cues (Rabinowitz et al., 1982). However, additional research questioned the validity of this general encoding theory due to inconsistent and incompatible findings (see Light, 1991). For example, Park, Puglisi, Smith, and Dudley (1987) varied the presence or absence of pictorial cues at encoding and retrieval and found that younger and older adults were equally aided by specific encoding and retrieval cues, indicating similar usage of contextual information, and that even when resources were limited by divided attention, older and younger adults still showed equivalent patterns of memory facilitation from specific cues.

Shortly following this general encoding debate, there was burgeoning interest in false memories and the increased susceptibility of older adults to remember information that was never presented but that was semantically similar to the studied material (e.g., Norman & Schacter, 1997). Despite the concerns about the validity of general encoding in describing verbal learning, the false memory literature, nevertheless, made it clear that older adults tended to rely on gist at the expense of detail.

The issue of whether age-limited processing resources served as an important mechanism of age-related memory deficits reached a head after publication of a chapter by Leah Light (1988), where she criticized the circularity of the theory. The limited resource explanation was routinely invoked when older adults showed poor memory performance, yet there was no independent measure or evidence that processing resource was actually depleted. This argument triggered a paradigm shift in the study of cognitive aging. Researchers began focusing on the measurement of individual resource pools that participants possessed, examining candidate cognitive primitives that could be the instantiation of the "limited resource" (i.e., working memory, attention, processing speed, executive function).

This movement gained further momentum by the great success Timothy Salthouse had in using speed as an individual differences variable that accounted for considerable age-related variance on memory tasks (Salthouse, 1985b, 1996). That is, processing speed could be the limited resource sought by researchers. Serendipitously, around the same time, Baddeley and Hitch (1974) developed their model of working memory, which posited two separate resource pools (visuospatial and verbal), which were controlled by a central executive system. This model became a prominent way to envision the structure of the mind. Working memory, which was a combination of storage and processing capacity, quickly became viewed as an excellent way to measure processing resource, independent of any specific experimental manipulations. Arthur Wingfield and Elizabeth Stine conducted some of the earliest work on this topic. They reported that older adults had poorer verbal

working memory than younger adults, and that better verbal working memory was correlated with better vocabulary (Wingfield, Stine, Lahar, & Aberdeen, 1988). In a follow-up study, they demonstrated that working memory capacity was also associated with simple text recall, confirming its potential explanatory power (Stine & Wingfield, 1990). This research was followed by other articles that offered a broad perspective of additional cognitive mechanisms that could affect memory decline, such as frontal lobe function and executive control (e.g., Moscovitch & Winocur, 1995; West, 1996).

Inhibitory Theory of Memory Deficits With Age

Like Craik and Lockhart (1972)'s article, Hasher and Zacks (1988)'s theorizing on inhibition represented an important innovation that forever changed the way memory was conceptualized. Reminiscent of Rabbitt (1965)'s finding that older adults had difficulty ignoring irrelevant information, Hasher and Zacks proposed the hypothesis that the ability to suppress attention to irrelevant thoughts within working memory was an important predictor of episodic memory. Inhibition, they argued, served to reduce the activation level of off-goal-path thoughts in working memory, and facilitated efficient memorial processing. Older adults were thought to be deficient in inhibition, and, consequently, to be easily distracted and to focus on contextual information at the expense of target information. Poor inhibition was posited to lead to a cluttered working memory that also had limited capacity for the entrance of new relevant information. Moreover, this "mental clutter" amplified competition during memory retrieval, which contributed to higher intrusion rates and heightened memorial interference in older adults. This inhibitory theory was very influential for the field, and it still plays a big role today.

Disuse, Motivational, and Other Noncognitive Theories

Although not the most prominent, certain noncognitive theories were also hypothesized to explain age-related memory deficits. It was suggested that perhaps older adults exhibited differential memory performance due to (a) lower motivation, (b) reduced memory self-efficacy, (c) greater test anxiety, (d) different performance goals, (e) greater time out of school, (f) lower formal education, and (g) poorer health. Although these factors may have influenced performance under certain conditions, there was little support that these noncognitive factors contributed entirely to age-related memory differences, especially because not all domains of memory exhibited similar deficits (i.e., implicit memory, semantic memory; see Burke & Light, 1981; Light, 1991) and because age effects were observed in samples matched on health and education (see Kausler, 1991). Consequently, these noncognitive hypotheses have not played a major role in theories of memory and aging.

Age Invariance in Specific Memory Domains

Not all research was focused on mechanisms of memory differences. Some research sought to identify and explain age-invariant memory domains, that is, domains in which memory performance was equivalent for younger and older adults. In an early instantiation of this concept, Hasher and Zacks (1979) suggested that certain stimulus attributes were encoded automatically and, consequently, showed little effect of age. They reported that both younger and older adults had equivalent memory for the frequency and spatial location of words, whereas older adults demonstrated decreased memory for the words themselves. Later work suggested, however, that age differences could be observed in spatial memory if the tasks were sufficiently demanding (e.g., Park, Puglisi, & Lutz, 1982; Puglisi, Park, Smith, & Hill, 1985).

Semantic memory was also initially considered to be relatively age invariant. Darlene Howard and colleagues showed comparable semantic memory as a function of age (e.g., Howard, Lasaga, & McAndrews, 1980), as did Lars Nyberg, who found age differences for episodic but not semantic memory (Nyberg, Bäckman, Erngrund, Olofsson, & Nilsson, 1996). Nevertheless, under conditions of higher demand, semantic memory deficits appeared. For example, Bowles and Poon (1985) found no evidence for age effects during easy lexical decisions, but when the task required active retrieval of words based on provided definitions, age differences were observed. Similarly, event-based prospective memory was originally claimed to be age invariant (e.g., Einstein & McDaniel, 1990), but later studies called this into question (e.g., Park, Hertzog, Kidder, Morrell, & Mayhorn, 1997). An analogous pattern of results occurred for picture memory. Early work in this domain suggested that old and young performed similarly on tests of complex picture recognition of real-world scenes (Park, Puglisi, & Smith, 1986). However, later work showed that age effects were evident when the pictures were abstract (Smith, Park, Cherry, & Berkovsky, 1990) and when they required active integration of target and context (Park, Smith, Morrell, Puglisi, & Dudley, 1990).

There are two interesting points about the appealing possibility that some types of memory are protected from the effects of aging. First and most importantly, in retrospect, the question of whether age effects could be observed in each of these domains was not the most crucial question. Rather, the critical focus should have been whether the magnitude of the age effects differed for various types of memory (it did). Second, if so, what mechanism(s) contributed to the observed differences? For each of the domains just discussed, subsequent work yielded evidence that the demands of a given task were more important than the domain of the task, providing strong support for the notion of cognitive resource limitations being the basis for age-related memory dysfunction. That is, when the difficulty of these tasks was increased, age effects were apparent (i.e., Bowles & Poon, 1985; Puglisi et al., 1985). The findings from each of these

memory domains posed perplexing results for current memory theories, and ultimately pointed toward lower resource requirements for tasks where age invariance was observed.

Inadequacy of Single-mechanism Theories of Age-related Memory Decline

At this point, the field of memory and aging was confronted with multiple rich theoretical viewpoints (i.e., speed, limited resources, inhibition) that plausibly accounted for age-related memory decline. The research was dominated by experimental psychology paradigms that relied on systematic manipulation of variables and analysis of variance techniques to compare performance between younger and older adults. Based on this approach, troubling studies were presented by each theorist that could be explained by their mechanism of choice but not by other models. Discussion of these competing theories was at a fever pitch in the research community, with resolution of which theory was actually correct seemingly unresolvable. Auspiciously, an alternate data analysis approach emerged that simultaneously considered multiple possible antecedents of age-related memory decline and offered a fresh viewpoint—one that allowed for multiple theories to be correct.

There were a number of research groups working on theories of intelligence and aging led by luminaries such as K. Warner Schaie, John Horn, Earl Hunt, and Christopher Hertzog. They began developing nonexperimental, interrelated models of the aging mind that relied on individual differences, multiple constructs, and structural equation modeling to predict cognitive performance (e.g., Hertzog, 1985; Horn, 1989). Individual differences research made it increasingly obvious that the mechanisms underlying memory and aging were multifactorial, and these new modeling approaches allowed for complex multicausal views of age-related deficits in memory. For the next several years, many influential articles in cognitive aging would take a broad individual differences approach, measuring numerous mechanisms purported to underlie memory function and using structural equation models to predict memory performance.

One article that illustrates this transition was by Park and colleagues (1996). In this article, multiple measures of speed and working memory were used to predict three types of memory that varied in their degree of environmental support: spatial recall (which was hypothesized to be more automatic), cued recall (which provided some environmental support), and free recall (which required the most mental effort). Speed and working memory constructs provided independent measures of two types of cognitive resource. The resulting model demonstrated that speed contributed to all three types of memory. However, working memory explained additional variance in the two more effortful memory tasks: cued recall and free recall. This result provided support both for the fundamental nature of speed (see also e.g., Lindenberger, Mayr, & Kliegl, 1993) and

for the additional role that working memory contributed for demanding memory tasks. In a later article, Hedden, Lautenschlager, and Park (2005) demonstrated that both processing resource and knowledge were important mechanisms for successful memory, but their relative contributions varied as a function of task and age. Processing resource (i.e., speed + working memory) explained significant variance in free recall, cued recall, and verbal fluency, whereas knowledge was only related to verbal fluency and cued recall. Moreover, knowledge was more important for older than younger adults in explaining variance in cued recall, suggesting that older adults increasingly rely on knowledge to compensate for processing declines.

Overall, this multifaceted theoretical approach helped identify particular constructs that were most critical in accounting for large amounts of age-related variance in memory, while also delineating the inadequacy of single-mechanism theories of memory. Unquestionably, the construct that controlled the most age-related variance in cognition was speed, even when modeled with measures of resource (i.e., working memory) and measures of crystallized knowledge (i.e., vocabulary). Thus, along with the realization that multiple factors could influence observed age-related memory effects, speed was confirmed as perhaps the most important contributor to age differences in memory.

Insights From Neuroimaging

The next innovation that transformed the conceptualization of memory was the introduction of structural and functional neuroimaging. In the following sections, we briefly note how theories of memory and aging were influenced by neuroimaging.

The advent of magnetic resonance imaging allowed researchers to measure the volume of brain structures in older adults and to relate these measures to memory performance. Naftali Raz conducted influential work in this domain and demonstrated that older adults with smaller brain volume (i.e., hippocampal, parahippocampal) tended to have impaired explicit memory (e.g., Raz, Gunning-Dixon, Head, Dupuis, & Acker, 1998; although Raz & Rodrigue, 2006 note that these effects are modest). Analysis of white matter integrity further revealed poorer memory in older adults with white matter hyperintensities (e.g., DeCarli et al., 1995; Van Petten et al., 2004). Starting in the 2000s, researchers were additionally able to examine the quality of specific white matter tracts in the brain with diffusion tensor imaging, revealing some associations between white matter connectivity and memory in older adults (see Madden, Bennett, & Song, 2009). Thus, structural imaging methods enabled links to be drawn between brain structure and memory performance, helping identify physiological factors that were related to age-related memory differences.

Functional neuroimaging also offered insights into how the aging brain performed encoding and retrieval processes. One influential finding was that, under certain task

conditions, older adults exhibited greater levels of neural activity than younger adults (e.g., Cabeza et al., 1997; Reuter-Lorenz et al., 2000). Given that most other age effects documented decrements in older versus younger groups, this somewhat counterintuitive finding led to the hypothesis that older adults might be recruiting additional neural resources to compensate for other neural inefficiency and to boost performance (e.g., Cabeza, 2002; Reuter-Lorenz & Cappell, 2008). Questions remain about whether this pattern of activation truly reflects compensation (e.g., see Kalpouzos, Persson, & Nyberg, 2012). Nevertheless, the ability to track changes in neural reactivity in response to task demands gave cognitive neuroscientists the ability to posit brain-based functional theories of memory and aging.

Other biological factors such as altered neurotransmission and vascular dysfunction have been proposed to contribute to age-related memory differences (e.g., see Bäckman, Nyberg, Lindenberger, Li, & Farde, 2006; Braver et al., 2001; Buckner, 2004). Moreover, the development of in vivo β-amyloid and tau imaging has allowed researchers to examine the relationship between neuropathological insults and memory, even in cognitively normal older adults. Greater levels of amyloid have been associated with worse episodic memory (Hedden, Oh, Younger, & Patel, 2013), deficits in other domains of cognition (e.g., Rodrigue et al., 2012), and altered patterns of functional activation during memory encoding (e.g., Kennedy et al., 2012; Mormino et al., 2012). Although tau imaging is still very new, theories posit that greater levels of tau may also be linked with impaired memory (see Villemagne & Okamura, 2016). Ongoing work in this field will help characterize the neuropathology associated with memory performance and the transition from normal aging to Alzheimer's disease.

The Future

In addition to continued research utilizing neuroimaging, which will help characterize biological underpinnings of memory impairment in old age, and to experimental psychology methods, which will continue to critically inform theories of memory and aging, additional focus has turned to applied methods for improving memory across the adult life span. Considerable attention has been given to intervention techniques (i.e., cognitive training, lifestyle adjustments) to boost cognitive function and delay the onset of memory decline. We note, however, that the field of cognitive training is still very young, and continued rigorous scientific studies are needed to determine the reliability, breadth, and duration of training effects.

One of the earliest and most influential intervention studies was an offshoot of the Seattle Longitudinal Study, when Sherry Willis imbedded a cognitive training program within the fifth cycle (see Schaie & Willis, 2010). This work provided the foundation for later training efforts, including the ACTIVE trial, which reported improvements in speed, reasoning, and memory after 10 sessions of targeted cognitive training (Ball et al., 2002). Additional intervention studies were founded upon models of cognitive reserve, which posit that certain lifestyle and health factors can influence current cognition and longitudinal cognitive change (e.g., Reuter-Lorenz & Park, 2014; Tucker & Stern, 2011). As such, some interventions have been developed to determine whether increasing protective factors can heighten memory. Work from our own lab documented increases in episodic memory in older adults who learned complex new skills, such as digital photography or how to use an iPad (Chan, Haber, Drew, & Park, 2014; Park et al., 2014). We envision that cognitive aging researchers will maintain their interest in training studies and that additional experimental work will help characterize the conditions under which neural plasticity can be exploited to improve memory.

Related to cognitive reserve is the concept of brain maintenance. Nyberg, Lovden, Riklund, Lindenberger, and Bäckman (2012) propose that successful agers maintain brains that are similar to those of young adults. Whereas cognitive reserve theories center more on characteristics that enable people to retain cognitive function in the face of neuropathological changes, brain maintenance considers what factors reduce functional, anatomical, and neurochemical brain changes in the first place. Future research will undoubtedly consider predictors of brain maintenance and how they relate to preservation of memory with age.

Neurostimulation has also garnered recent interest as a possible method to enhance memory. Researchers have begun examining the influence of transcranial direct current stimulation (tDCS), a noninvasive technique involving the passage of a small electrical current (i.e., 1–2 mA) through the brain, traveling between two electrodes placed on the surface of the head. Thus far, several studies have documented memorial benefits of tDCS in young adults and at least one older adult sample (see Coffman, Clark, & Parasuraman, 2014). Moreover, one study noted improved verbal recognition memory in Alzheimer's patients following tDCS (Ferrucci et al., 2008). If these findings prove to be replicable and reliable, it is conceivable that such brain stimulation may become popular. Needless to say, further research is needed to determine whether this is a viable technique to abate memory decline in older adults and whether the anatomical loci of these effects will help inform theories of memory and aging.

Finally, we predict that future research will place greater emphasis on genetic risk factors and epigenetic environmental triggers that predispose individuals for memory disturbances. At present, the field has identified certain genes with influences on cognition, such as APOE, BDNF, COMT, and KIBRA (e.g., Laukka et al., 2013), and evidence suggests that the effects of disadvantageous genes are amplified in old age (see Papenberg, Salami, Persson, Lindenberger, & Bäckman, 2015). Additional research will further elucidate these relationships, tracking genetic impacts on memory and cognition throughout the life span.

Conclusions

This brief review highlights major theoretical issues in the study of memory and aging and how they changed over time. No doubt this review is selective and somewhat subjective, but it offers organizing principles for how memory and aging research unfolded. One broad theme evident in the article is that, as research developed, a multitude of variables were recognized to contribute to age-related memory deficits. The shift from early single-mechanism views to involved multifactorial models is striking, and these intricate cognitive models mirror the vast complexity of the aging brain. Advances in neuroimaging clearly delineate that there are age differences in neural structure and function that contribute to memory performance, and contemporary theorists are becoming increasingly interested in using both neural and behavioral measures to differentiate between pathological and normal age-related decline in memory. Additionally, an increased focus has been placed on identifying practical techniques to maintain memory function for life. This research on memory preservation has relied heavily on the rich depth of information generated in earlier research about basic memory function. The investment in memory and aging research over the last 50 years has provided the knowledge base needed to develop increasingly effective interventions and to identify early markers of pathological aging. Continued and sustained investment in this critically important research domain is likely to yield advances that have the potential to enhance the quality of life of both sufferers of age-related memory disorders and their families.

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