11 Episodic Memory Decline in Aging

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Abstract

Age-related changes in memory are a common but worrisome occurrence in many people's lives. However, these changes are not ubiquitous. Healthy aging appears to impact memory for associative/relational details, i.e., the ability to recollect, more so than memory for item information. We propose that alterations in the recruitment of prefrontally mediated cognitive control processes, such as strategy use and inhibitory control, underlie these age-related memory deficits in healthy adults. These processes are particularly critical for remembering specific relational details and for being able to resolve interference between competing memories. Critically, evidence suggests that while there are large individual differences in the impact of aging on memory, various methods of support/intervention can improve memory performance in healthy older adults. We discuss how recent developments in neuroscience analysis methods have enhanced our understanding of how aging affects the control processes that support episodic learning and retrieval. We further suggest that future studies should test more diverse samples of adults and assess the role of lifestyle factors on individual differences in patterns of episodic memory performance and supporting brain activity and structure.

Introduction

Many aspects of cognition decline as people age, even in the absence of disease (e.g., Craik & Bialystok, 2006; Lindenberger, 2014). One of the most common and arguably most distressing such declines in aging is in episodic memory, in large part because it is also an early sign of Alzheimer's disease. We define episodic memory as the ability to encode and retrieve the details and associations that make up the events of our lives. Episodic memory can be tested in multiple ways. Tests of item recognition require participants to determine whether they recognize an item from a previous learning episode or believe it is new. These simple "old/new" recognition decisions may be based upon one or more memory processes. Specifically, in dual-process models of recognition memory, stimuli experienced previously can be recognized either by *recollection* of relational/associative details of the original learning episode (e.g., seeing a person on the bus and remembering their name and where you met them) or by *familiarity* for the item in the absence of retrieval of such details (e.g., recognizing you know the

person on the bus but being unable to place them; Mandler, 1980; Yonelinas, 2002). One way to separate these processes is to use subjective reports of an individual's memory experience, as with the "remember-know" procedure (Tulving, 1985). Participants respond "remember" when they recollect details associated with a previously studied item and "know" when the item seems familiar but they cannot recollect any specific details. A benefit of this technique is that participants can recollect any detail in order to respond "remember," be it something about the stimulus or an internal thought or feeling they had related to it. Another approach is to measure these processes objectively, as in a source memory task. Participants are asked to determine which experimentally manipulated context (e.g., spatial location, color, size of the stimulus, which speaker said the word, etc.) was associated with an item during initial study. Another commonly used objective memory task is paired-associate learning, in which participants learn and later have to remember pairs of stimuli (e.g., words, objects, names, faces). Critically, in contrast to subjective measures of memory, both of these tasks require the participants to recollect specific details.

Numerous studies have shown that older adults demonstrate greater difficulty remembering event-defining details and associations, i.e., relational/associative information, compared to simple old/new item recognition (Mitchell & Johnson, 2009a; Old & Naveh-Benjamin, 2008). Further, when compared directly, age-related episodic memory deficits are more pronounced for objective measures of recollection, namely source memory, than for subjective measures (Ciaramelli & Ghetti, 2007; Duarte, Henson, & Graham, 2008; Duarte et al., 2006; Mark & Rugg, 1998). Interestingly, these age-related episodic memory impairments have been shown for virtually every stimulus modality and testing procedure. One exception to these age-related associative memory impairments seems to be episodic memory for emotional or social information, as discussed in more detail in Chapter 13. Nonetheless, given the otherwise fairly ubiquitous nature of these impairments, it is likely that they may be explained by age-related changes in cognitive processes and brain regions that support episodic memory in a domain-general manner. As we discuss below, we believe that this discrepancy between subjective and objective episodic memory results is related to the differential dependence of these measures on the cognitive control processes of the prefrontal cortex (PFC).

Neural Contributions to Age-Related Episodic Memory Impairments

Numerous structural and functional imaging studies have investigated the underlying neural factors that contribute to older adults' episodic memory impairments. Evidence from numerous fMRI studies has revealed that episodic memory success is dependent on several regions, including the medial temporal lobe (MTL: hippocampus and parahippocampus) and the highly interconnected posterior parietal cortex (Davachi, Mitchell, & Wagner, 2003; Diana, Yonelinas,

& Ranganath, 2009; Uncapher & Wagner, 2009; Vilberg & Rugg, 2008). Furthermore, patient studies suggest that these "core episodic network" regions are necessary for episodic memory function, with MTL or parietal damage producing amnesia (reviewed in Milner, 2005), particularly for associative/relational information as opposed to item information (Horecka et al., 2018; Konkel et al., 2008). Several theories have been proposed to explain the functional role of this network in episodic memory. A common view is that the MTL facilitates the binding of multiple features, regardless of domain, into unique episodic representations during encoding and in the comparison of those representations with retrieval cues during retrieval (Eichenbaum, Yonelinas, & Ranganath, 2007; Monti et al., 2015; Sestieri, Shulman, & Corbetta, 2017; Simons & Spiers, 2003). Regions within the posterior parietal cortex serve a less direct role in memory, such as accumulating mnemonic evidence for memory decisions (i.e., male or female voice? intact or rearranged pair?; Wagner et al., 2005), guiding attention to memory (Cabeza, 2008; Ciaramelli, Grady, & Moscovitch, 2008), and/or maintaining the representation of recollected information (Vilberg & Rugg, 2008). Similarly to the binding operations of the MTL, the processes ascribed to the posterior parietal cortex are thought to support memory in a domain-general manner. Consequently, a reasonable prediction is that dysfunction in this network is a major contributor to the episodic memory impairments in healthy older adults. Indeed, functional imaging studies have shown age-related differences, primarily under-recruitment of these regions, concomitant with older adults' memory impairments (Cansino et al., 2015b; Dennis, Kim, & Cabeza, 2008b).

Yet, there are a few points of evidence that argue against a principal role of MTL dysfunction in age-related episodic memory impairments. First, MTL atrophy is not pronounced until the eighth or ninth decade of life in healthy older adults, but the majority of episodic memory studies have demonstrated age-related deficits in older adults between the ages of sixty and eighty (Salami, Eriksson, & Nyberg, 2012). Furthermore, numerous studies have shown age-related sparing of successful encoding- and retrieval-related activity in the MTL and parietal cortex (Duarte et al., 2008; Dulas & Duarte, 2012; S. L. Miller et al., 2008; Morcom, Li, & Rugg, 2007), particularly when memory performance is experimentally or statistically equated between age groups (Angel et al., 2013; de Chastelaine et al., 2015, 2016; Duverne, Motamedinia, & Rugg, 2009; Rugg & Morcom, 2005). The idea behind equating performance is that group differences in episodic memory activity may be related to dilution by guessing or poor task comprehension rather than aging per se (Rugg & Morcom, 2005). Some studies have implemented study repetitions (Leshikar & Duarte, 2014; Morcom et al., 2007) or reduced memory load (Dulas & Duarte, 2013, 2014) to boost memory performance for older adults to the level of the young, while others have investigated the relationship between neural activity and age after partialing out individual differences in memory performance (de Chastelaine et al., 2015, 2016). Regardless of the method used to account for performance effects, these studies have collectively demonstrated that age-

related changes in the core episodic memory network are unlikely to be the major contributor to healthy older adults' episodic memory impairments.

In addition to the core episodic network, the prefrontal cortex has also been demonstrated to be critical for episodic memory success across a number of material domains (i.e., visual, auditory, words, objects) and task procedures (Blumenfeld et al., 2011; Blumenfeld & Ranganath, 2007; Mitchell & Johnson, 2009). The PFC supports the cognitive control processes that allow information to be processed and flexibly acted upon in ways that are consistent with one's current task goals (E. K. Miller & Cohen, 2001). Cognitive control functions are diverse and include evaluation of relationships between stimuli or concepts, selection between competing memory representations, and monitoring retrieved information (Badre, 2008; Badre & Nee, 2018). While damage to the MTL, and potentially also the posterior parietal cortex, results in profound amnesia, damage to the PFC produces subtler episodic memory deficits (Janowsky, Shimamura, & Squire, 1989; Kopelman, Stanhope, & Kingsley, 1997). Specifically, patients with damage to the lateral PFC show source memory impairments but relatively spared item recognition, as well as spared subjective reports of recollection and familiarity (Duarte, Ranganath, & Knight, 2005). Patients with ventromedial PFC damage show source memory impairments but only for temporal and not spatial source features (Duarte et al., 2010). Moreover, the ventromedial PFC has been shown to be critical for integrating information across time points and making associative inferences (Spalding et al., 2018), highlighting its role in associative memory across episodes.

Several factors point to PFC dysfunction as a principal source of episodic memory impairments in aging. First, healthy older adults, like patients with focal lateral or medial PFC lesions (Ciaramelli & Ghetti, 2007; Duarte et al., 2005), can show intact subjective recollection despite impaired objective recollection of experimentally manipulated details (Duarte et al., 2006, 2008). These results fall in line with the suggestion that objective measures of episodic memory place larger demands upon PFC-mediated cognitive control processes compared to subjective measures. Second, structural imaging studies show disproportionate declines in PFC gray (Raz & Kennedy, 2009) and white matter volume (Nyberg et al., 2010) compared to non-PFC regions across the life span. Furthermore, older adults with larger gray matter volumes in lateral PFC regions show better episodic memory performance (Becker et al., 2015). Third, fMRI studies show age-related reductions in PFC activity during encoding (Dennis et al., 2008a; Dulas & Duarte, 2011) and retrieval (Dulas & Duarte, 2012; McDonough & Gallo, 2013; Rajah, Languay, & Valiquette, 2010) despite age-equivalent MTL recruitment. Further, some fMRI studies have demonstrated age-related increases in PFC recruitment (over-recruitment; Davis et al., 2008; Reuter-Lorenz & Cappell, 2008; Spaniol & Grady, 2012). While such over-recruitment has sometimes been argued to reflect age-related compensation, recent work employing multivariate methods to directly assess the contributions of over-recruitment of the PFC to memory has demonstrated that over-recruitment likely reflects

dedifferentiation and reduced specificity of cognitive functions, underlying age-related deficits (Morcom & Henson, 2018). Lastly, even when memory performance is equated between age groups, PFC activity differences may persist during encoding (de Chastelaine et al., 2016; Dulas & Duarte, 2014) and retrieval (Dulas & Duarte, 2016; Wang et al., 2016). Collectively, these findings are consistent with the "frontal aging hypothesis," which posits that PFC dysfunction underlies many cognitive impairments, including but not limited to episodic memory in aging (West, 1996).

Other influential cognitive aging theories, like the "inhibitory deficit hypothesis," discussed further in Chapter 8, suggest that older adults' cognitive impairments arise from dysfunction in a particular cognitive control function: the ability to reduce interference from task-irrelevant information (Hasher & Zacks, 1988). Importantly, this theory is not inconsistent with the frontal aging hypothesis. Indeed, it is probable that PFC dysfunction underlies poor inhibitory control and that episodic memory tests that place high demands on inhibition will be particularly difficult for older adults (Jacoby et al., 2005). The ability to selectively attend to task-relevant and away from task-irrelevant information depends upon *interference resolution*. Interference resolution deficits contribute to episodic memory failures in a variety of situations, such as when external, distracting stimuli are present or when internally generated memories compete with our ability to encode or retrieve new ones (i.e., proactive interference; Dey, Sommers, & Hasher, 2017; Gazzaley et al., 2005; Postman & Underwood, 1973). Having trouble ignoring other patrons' conversations in a restaurant, or having the strong memory of your former email password impair your ability to remember your new one, are two everyday examples.

Difficulty resolving interference manifests as worse memory for task-relevant information, as well as integration of the distracting information into memory, i.e., "hyper-binding," both of which increase with age (see Healey, Campbell, & Hasher, 2008, for review). Two proposed mechanisms are hypothesized to mediate interference resolution: *Suppression* involves actively inhibiting distractors while *facilitation* involves enhanced processing of targets (see Hasher, Lustig, & Zacks, 2007, for review). These mechanisms are not mutually exclusive, but suppression may be the strongest contributor to interference resolution and disproportionately affected by aging (Dey et al., 2017; Gazzaley & D'Esposito, 2007; Healey et al., 2010). Suppression and facilitation are likely mediated through top-down modulation of activity within sensory (e.g., visual/auditory association cortices) regions representing the relevant or irrelevant external or mnemonic information. Through functional coupling with sensory regions, which demonstrates the connectivity between regions via correlations of their activity patterns, lateral and medial PFC regions have been implicated in neural facilitation and suppression in episodic memory tasks in young adults (Campbell et al., 2012; Chadick & Gazzaley, 2011; Dulas & Duarte, 2016; Gazzaley et al., 2008; Stevens et al., 2008). Few studies have investigated the effect of age on PFC-dependent interference resolution functions in episodic memory tasks. However, extant evidence points to under-recruitment

of lateral PFC regions associated with proactive interference resolution during paired-associate memory retrieval (Dulas & Duarte, 2016).

Compelling evidence suggests that although cognitive control dysfunction may underlie episodic memory decline in aging, it may be reparable. Older adults do not spontaneously generate effective memory strategies to the same extent as young adults. When task instructions facilitate semantic elaboration (Logan et al., 2002), attention toward inter-item associations (Braver et al., 2009), or self-referential thought (Gutchess et al., 2015; Leshikar & Duarte, 2014), age-related memory impairments are reduced and left ventrolateral, dorsolateral, and medial PFC under-recruitment, respectively, are reversed. In other words, older adults can recruit at least some PFC-mediated cognitive control functions to support memory performance when directed to do so by the demands of the task. Moreover, event-related potential (ERP) results have demonstrated that encoding support, in this case self-referential processing, not only serves to reduce age-related under-recruitment, but also ameliorates age-related slowing in the onset of retrieval processes (Dulas, Newsome, & Duarte, 2011). However, even after providing environmental support, age-related deficits often persist (Dulas & Duarte, 2012, 2014). Interestingly, evidence suggests that while environmental support can ameliorate age-related under-recruitment of PFC processes, it does not impact over-recruitment in the PFC related to dedifferentiation, i.e., the decrease in region-specific distinct functioning/processing (Logan et al., 2002). Thus, some of the persistent age-related deficits may be tied to the PFC regions that are over-recruited. In addition, evidence also suggests that even when directed to attend to the critical experimentally manipulated details, older adults may be less likely/able than young adults to inhibit attention toward noncriterial information, such as internal and/or self-referential information (Dulas & Duarte, 2014; Hashtroudi, Johnson, & Chrosniak, 1990; Leshikar, Dulas, & Duarte, 2015). While it is possible that with additional environmental support, e.g., support at both encoding and retrieval, such as having people generate a sentence to relate word pairs during study and then cuing them to retrieve that sentence at test (Naveh-Benjamin, Brav, & Levy, 2007), these deficits could be further ameliorated, these results suggest that some, but not all, age-related changes in the PFC are amenable to intervention.

Individual Differences in Episodic Memory Performance

While the previous sections have discussed age-related episodic memory decline and PFC dysfunction as trends across the aging population, there is substantial interindividual variability. Emerging cognitive neuroscience studies suggest that some high-performing older adults show similar levels of episodic memory performance and patterns of fMRI and EEG brain activity to those of well-performing young adults (de Chastelaine et al., 2015, 2016; Duarte et al., 2006, 2008; Lindenberger, 2014; Nyberg et al., 2012). Several neurocognitive theories have been proposed to explain these individual differences. *Cognitive reserve*

refers to the accumulation of neural resources (e.g., brain volume, blood flow, neurotransmitter levels) that mitigate against age-related pathology and forestall Alzheimer's disease (Barulli & Stern, 2013). Genetic factors, as well as more malleable lifestyle ones like sleep, physical activity, and education, contribute to reserve through a variety of cellular and neurochemical mechanisms. By contrast, *brain maintenance* refers to the preservation of neural resources across the life span, such that individuals who maintain "youth-like" patterns of neural structure and function as they age show better cognitive performance (Lindenberger, 2014; Nyberg et al., 2012). A review of the cognitive reserve framework can be found in Chapter 2. Similarly to reserve, genetic and lifestyle factors contribute to brain maintenance. Although these theories are commonly invoked to interpret results, cognitive neuroscience studies have almost never directly assessed the impact of lifestyle factors on patterns of brain activity and related memory performance. Furthermore, very few studies have sampled the continuous adult life span or a racially diverse sample. This is necessary both for generalizability and for determining whether lifestyle factors impact PFC functioning and episodic memory performance similarly across age or differentially by age, as would be predicted by maintenance and reserve theories, respectively.

The majority of aging studies have been cross-sectional and have not examined memory performance and related neural activity in middle-aged adults. Consequently, we know relatively little about what underlies episodic memory changes in middle age or when many of the aforementioned age-related changes in emotional memory begin to emerge. Evidence from life-span cross-sectional studies suggests that changes in PFC recruitment during encoding (Cansino et al., 2015a) and retrieval (Ankudowich, Pasvanis, & Rajah, 2016; Cansino, Hernandez-Ramos, & Trejo-Morales, 2012; Cansino et al., 2015b; Kwon et al., 2016) contribute to episodic memory decline even in midlife (~40–60 years of age). It is not clear from this work whether the mechanisms underlying memory decline in middle age are the same as those in old age. Arguably the best approach to investigate neural changes underlying episodic memory decline across the life span would be longitudinal assessments of individuals over an extended period of time. Such studies are too few in nature, as they are very costly and face issues including subject attrition and practice effects (Salthouse, 2014). However, those longitudinal studies that have been conducted have revealed some very important findings regarding aging and memory. For example, longitudinal evidence suggests that findings of age-related PFC over-recruitment in cross-sectional studies may be overestimated and that PFC under-recruitment during encoding or retrieval is a more typical response (Nyberg et al., 2010). Furthermore, PFC over-recruitment may be more likely related to declining memory function than to a compensatory mechanism that supports memory performance in older adults (Pudas et al., 2017).

Longitudinal research is needed to more accurately characterize the neural basis of age-related episodic memory decline and the factors that contribute to these changes (i.e., lifestyle, genetics, etc.). Importantly, cross-sectional aging studies

still have important value. Indeed, cross-sectional research is arguably the best method for dissociating cognitive mechanisms that contribute to episodic memory performance (including various cognitive control operations) and identifying the brain areas and networks that support them. A combination of cross-sectional and longitudinal research may be the strongest approach to tackling questions about life-span changes in episodic memory function.

Future Research Directions

Most neuroimaging and EEG studies have used univariate analyses, which are excellent for determining whether a particular brain region or EEG signal contributes to memory or is affected by age, but do not allow for assessments of brain networks. Multivariate analyses, by contrast, can be used to characterize how aging affects functional coherence within and between networks. Thus, while univariate analyses might suggest reduced availability of PFC regions to support episodic memory functioning in older adults, multivariate analyses show how functional communication between these regions is affected by age. Analyses applying graph theory of resting-state data have shown that aging is associated with reduced segregation of functional brain networks supporting distinct cognitive functions such as attention and episodic memory, which in turn is related to impaired memory performance (Chan et al., 2014). For example, age-related reductions in connectivity strength within a network of frontal and parietal regions that support cognitive control functions together with greater connectivity with regions that are normally part of other networks are associated with worse associative memory performance (Antonenko & Floel, 2014). This pattern of reduced intra-network connectivity together with greater inter-network connectivity is consistent with the dedifferentiation model of neurocognitive aging. More recent evidence has shown that this pattern of reduced intra-network connectivity, particularly in high-order control networks, is evident even in middle age (Siman-Tov et al., 2016). It remains to be determined whether age-related desegregation of cognitive control networks affects the recruitment of these networks during performance of episodic memory tasks and the extent to which reduced segregation contributes to age-related memory decline.

Machine learning–based analyses like multivariate pattern analysis (MVPA) have become very popular in recent years for their ability to detect patterns of activity that are indicative of different cognitive states (reviewed in Norman et al., 2006). MVPA has been used in young adults to reveal the specificity, measured as the degree of matching between encoding and retrieval brain activity patterns, with which episodic memories are retrieved (Liang & Preston, 2017; Staresina et al., 2012; Xiao et al., 2017). Specifically, MVPA results show that successful retrieval of previous events is associated with neural reactivation of category (e.g., scene, face, word) or context (e.g., orienting tasks) representations that were observed during encoding. Even more interestingly, MVPA evidence shows that successful

retrieval of word-scene pairs (e.g., "apple"-nature scene) is accompanied by reactivation of encoding activity associated with that *specific* pair above and beyond reactivation of the category-level representations common to all retrieved pairs. Only a handful of studies have applied MVPA to data from older adults but some interesting findings have begun to emerge. For example, in a recent study, young and older adults studied words and pictures of objects. At test, they were shown only words and indicated if they remembered or were familiar with each item. Contrary to the idea that the specificity of recollected memories is affected by age, no age-related differences were shown in category-level reinstatement (i.e., reinstatement of word vs. object processing) for recollected items (Wang et al., 2016). This finding supports the idea that memory for the broad perceptual or conceptual information, i.e., gist, is spared by age, which can contribute to elevated false memory in aging (Brainerd & Reyna, 2001; Schacter, Koutstaal, & Norman, 1997). An interesting question for future studies would be to determine whether the reinstatement of event-level details, such as the specific memory pair, is also spared by age.

In everyday situations, we often have multiple features competing for our attention, and our ability to encode some may depend on our ability to successfully ignore others. As discussed above, aging is well known to increase susceptibility to interference, and it is conceivable that episodic memory impairments may be particularly evident in the presence of task-irrelevant information. MVPA allows for the covert monitoring of one's attentional focus during encoding, which would be difficult to surmise from behavioral responses alone. Recently, we used MVPA to better understand whether age-related difficulties in selectively attending to relevant episodic information contribute to older adults' reduced context memory accuracy and tendency to hyper-bind relevant and irrelevant context features (e.g., scenes, colors; Powell et al., 2018). Capitalizing on the millisecond temporal resolution of EEG, we used MVPA to track what participants were attending to in real time while they encoded pairs of objects and contexts (color or scene) while asked to ignore another context feature. For example, for a particular trial, participants might be asked to decide if an object was associated with a presented color ("Is red likely for this apple?") but ignore the scene (e.g., kitchen) also presented at the same time. We found that moment-to-moment fluctuations of attention between relevant and irrelevant context features predicted individual differences in context memory accuracy and hyper-binding. These results provide strong evidence that age-related declines in episodic memory are attributable, at least in part, to impoverished focused attention during new event learning.

Of course, understanding age-related changes in episodic memory is all well and good, but what can we do about it? While still nascent, there is a growing body of literature examining the impact of interventions, including cognitive, nutritional, and other lifestyle factors, as well as brain training and brain stimulation on age-related memory declines. As discussed previously, environmental support has been shown to be effective at improving memory in older adults and reducing age-related differences in PFC recruitment. Larger-scale interventions, including

cognitive training and social engagement interventions (Park & Bischof, 2013; Stine-Morrow et al., 2008, 2014), have shown promise in supporting memory performance. Interventions that have shown improvements in cognitive function in older adults have been quite varied in nature, including dancing (Kattenstroth et al., 2013), mindfulness training (Banducci et al., 2017), acting (Noice, Noice, & Kramer, 2015), sewing and/or photography (McDonough et al., 2015; Park et al., 2014), and more. In general, evidence suggests that these interventions provide enriched environments which may serve to supplement and bolster, or "scaffold," intact cognitive function and potentially buoy performance (Park & Reuter-Lorenz, 2009; Reuter-Lorenz & Park, 2014). Potentially more promising is evidence that exercise can improve cognitive function, and in particular, episodic memory (Ahlskog et al., 2011; Erickson et al., 2009, 2011; Kramer, Erickson, & Colcombe, 2006), and can result in slowing of age-related declines in gray matter volume loss (Erickson et al., 2011; Jonasson et al., 2016). Moreover, levels of cardiovascular fitness are correlated with episodic memory in older adults, suggesting that cardiovascular fitness may facilitate the activation of cognitive processes, including those involved in cognitive control (Wong et al., 2015). In addition to supporting memory itself, cardiovascular issues are tied to an array of age-related disorders, including stroke and Alzheimer's disease. Thus, improved cardiovascular function may not only improve memory, but prevent/delay the onset of age-related pathological disorders. Because there is some debate over the efficacy of exercise interventions (Northey et al., 2018; Nyberg & Pudas, 2018; Young et al., 2015), it will be important for future studies to compare the effect of different kinds of interventions on episodic memory performance at different points across the life span.

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