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Changes of Neural Connectivity with Aging and its Impact on Memory Decline

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ABSTRACT

Memory loss is one of the most common cognitive concerns associated with aging, but the underlying neural mechanisms remain an important area of investigation. Emerging evidence suggests that age-related changes in brain connections, particularly within networks critical for memory and cognition, play a significant role in memory loss. This systematic review examines how brain connectivity changes with aging and how these alterations impact memory performance in older adults. The PubMed database was used to collect relevant literature from the past two years, focusing on studies that explored natural aging processes, brain connectivity, and memory loss. The review finds that aging is associated with structural and functional weakening of key brain networks, notably the default mode network (DMN), frontoparietal control network (FPCN), and hippocampal-prefrontal pathways. These changes contribute to slower processing speeds, impaired memory formation, and reduced executive function. However, the review also highlights that the aging brain retains notable plasticity. Physical activity, cognitive training, emotional stability, and metabolic health were found to preserve or enhance brain connectivity, supporting memory maintenance despite age-related decline. Contrary findings suggest that factors such as central obesity or emotional resilience may sometimes outweigh structural brain changes in determining memory outcomes. A key limitation of current research is the scarcity of longitudinal studies directly mapping how specific brain connections evolve with healthy aging independent of neurodegenerative disease. Future research should aim to clarify which neural pathways are most vulnerable over time and explore interventions targeting specific brain regions to preserve memory in aging populations.

Keywords: *Aging, Memory Loss, Brain Connectivity, Default Mode Network (DMN), Frontoparietal Control Network (FPCN), Functional Connectivity, Structural Connectivity*

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INTRODUCTION

Aging is universally accompanied by changes in cognitive abilities, with memory decline being one of the most commonly reported concerns among older adults. Cognitive impairment without dementia affects up to 35.9% of individuals aged over 65, and over 10% develop dementia (Qian et al., 2021). While mild forgetfulness or slower recall may be considered a normal part of aging, understanding the biological mechanisms behind this cognitive decline remains an essential area of research. Evidence suggests that one of the key contributors to age-related memory loss lies in changes within the brain's structural and functional connections (Glisky, 2007).

Memory relies on the brain's ability to transmit and process information across a vast network of interconnected neurons. These connections, known as synapses, form highly specialized communication points between neurons, allowing for the transfer of electrical or chemical signals. Over time, the strength and number of these synapses can deteriorate, leading to less efficient communication between brain regions, which may contribute to cognitive decline (Ackerman, 1992).

Synaptic health is not only determined by physical connections but also regulated by various neurochemical factors. Proteins such as brain-derived neurotrophic factor (BDNF) play a crucial role in maintaining synaptic plasticity, which is the ability of synapses to strengthen or weaken over time in response to activity. Reduced levels of BDNF and other neuroprotective biomarkers have been linked to impaired synaptic function and may be implicated in age-related memory decline (Hola et al., 2024).

Within the brain, certain networks are particularly essential for memory and everyday cognition. Two of the most well-studied among them are the default mode network (DMN) and the frontoparietal control network (FPCN). The DMN is responsible for internal thought processes like self-reflection and recalling past experiences, while the FPCN governs externally directed tasks that require focused attention, working memory, and executive function (Anthony et al., 2024; Gozdas et al., 2024). The hippocampus, deeply involved in memory formation, connects extensively with these networks to coordinate the encoding and retrieval of memories. The strength and integrity of these connections between different brain regions are therefore vital for maintaining healthy memory function.

Given the central role of brain connectivity in supporting memory, understanding how these connections change naturally with age independent from disease-specific pathology is crucial. While much existing research like Macfarlane et al. (2025) and Cummings et al. (2025) focuses on memory loss in the context of neurodegenerative diseases like Alzheimer’s disease, other studies have also isolated how normal aging itself alters brain connections. This distinction is important for identifying early biomarkers of cognitive decline and for developing interventions that target healthy aging specifically.

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This systematic review aimed to examine the current literature on how brain connections change with age and to explore how these alterations contribute to memory loss in older adults. Furthermore, this review evaluated the role of neuroprotective factors, such as physical activity, cognitive training, and synaptic-supporting proteins, in preserving brain connectivity and mitigating the effects of aging on memory.

METHODOLOGY

An advanced search was conducted on the PubMed database using the keywords 'aging brain changes' to identify studies examining how brain connections are altered with age and how these changes influence memory loss. Filters were applied to limit the results to full free-text articles, and the article types included were case reports, classical articles, clinical trials, and randomized controlled trials. All of the research was published within the past two years (up to March 29, 2025), and this search yielded a total of 74 results. Each article was then individually screened for relevance to the research topic of how brain connections change with natural aging and affect memory loss. Articles were excluded if they focused primarily on Alzheimer's disease or other specific neurodegenerative disorders rather than general aging, centered on other parts of the human body rather than the brain, investigated unrelated topics, such as non-cognitive symptoms, unrelated diseases, or molecular studies not tied to brain connectivity or function, or provided insufficient information about natural aging and changes in brain connectivity or memory loss. After this screening process, 49 articles were excluded for the reasons listed above. Ultimately, 25 articles were deemed relevant and included for analysis within this systematic review

RESULTS

As individuals age, structural and functional brain changes can significantly impact memory and cognitive performance. Research consistently shows that aging leads to alterations in brain connectivity—particularly within the default mode network (DMN), frontoparietal networks, and hippocampal circuits—that play a critical role in memory formation and retrieval (Glisky, 2007; Ackerman, 1992).

Neuroimaging studies have revealed that both gray and white matter deteriorate with age, contributing to disrupted communication between brain regions (Boccuni et al., 2024). These connectivity changes are especially pronounced in areas responsible for episodic memory and executive function, such as the hippocampus and prefrontal cortex [Cullen et al., 2024; Aksu et al., 2024). This neural degradation is associated with compensatory mechanisms; however, these mechanisms often become less effective over time, particularly in the presence of neuropathological factors like beta-amyloid and tau (Cummings et al., 2025; Wagemann et al., 2024).

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Studies utilizing fMRI and other imaging techniques show a decline in within-network coherence and an increase in between-network interference in older adults, which correlates with declines in working memory and processing speed (Gozdas, et al., 2024; Dimitriadis, et al., 2023**)**. This disintegration of network efficiency partly explains the onset of mild cognitive impairment and increased risk of dementia (Macfarlane et al., 2025; Zhou et al.,. 2025).

Yet there is growing evidence that the brain remains relatively "plastic" while aging, meaning it is able to reroute neural connections to adapt to new challenges and tasks. A recurring finding across numerous studies (Hola et al., 2024; Dupuy et al., 2024) was that physical activity plays a vital role in supporting memory and brain health in aging populations. Regular physical activity has been shown to enhance neuroplasticity, strengthen synaptic connections, and improve cerebral blood flow, all of which contribute to maintaining cognitive function (Walker et al., 2025). Specifically, engaging in moderate-to-vigorous physical activity (MVPA) has been linked to greater activation in brain areas responsible for executive control, while sedentary behavior (SB) was associated with reduced brain engagement (Falck et al., 2024).

Certain forms of physical activity, such as dance and martial arts, were found to increase brain-derived neurotrophic factor (BDNF) levels, a protein essential for synaptic plasticity and memory. The dance group also exhibited significant improvements in cognitive flexibility and attention (Hola et al., 2024). Similar benefits were found in a study examining combined physical exercise and cognitive training, where participants demonstrated notable improvements in executive function and working memory (Dupuy et al., 2024). Additionally, neurophysiological markers of memory processing, such as mismatch negativity (MMN) – an early, automatic brain response to auditory deviations – and memory-P3 event-related potentials (ERPs) – linked to conscious recognition and working memory updating, showed improvement following multidomain interventions that combined both physical and cognitive activities (Cheng et al., 2024).

Interventions targeting both aerobic exercise and cognitive training led to observable changes in brain network connectivity, particularly within the DMN, a region closely tied to memory function (Dimitriadis et al., 2023). However, another study suggested that while exercise improved overall fitness and sleep, reductions in body fat — specifically central obesity, also known as abdominal and truncal obesity — were more strongly correlated with healthier brain aging than fitness levels alone (Wing et al., 2023). Outside of exercise, year-to-year variability in cholesterol levels was associated with accelerated cognitive decline, linking metabolic health to memory outcomes (Zhou et al., 2025).

Beyond physical activity, interventions such as meditation were also shown to positively impact brain function. A study found that 18 months of meditation reduced vulnerability to distractions and improved attentional control, a function associated with frontoparietal brain networks (Requier et al., 2024).

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Additional studies examined the influence of gut microbiota on brain health. The consumption of *Lacticaseibacillus rhamnosus* CBT-LR5 improved tasks related to memory and recall, suggesting that gut health may support synaptic plasticity (Jung et al., 2025). Similarly, movement-based interventions like Tai Chi Chih were associated with increased DMN connectivity, correlating with improved memory performance (Chao et al., 2025).

Neuromodulation techniques were also explored. Transcranial direct current stimulation (tDCS), when paired with cognitive training, led to short-term improvements in executive function and conflict monitoring, although the effects were not sustained over the long term (Aksu et al., 2024). Another study demonstrated that long-term cognitive training increased resting-state functional connectivity within both the DMN and the frontoparietal control network (FPCN), regions vital for memory and executive function (Gozdas et al., 2024). Similarly, HD-tDCS targeting the right inferior frontal lobe mitigated memory decline, while stimulation of other areas produced mixed effects (Schmidt et al., 2024).

Evidence of brain adaptability was further supported by a case study in a patient undergoing brain tumor prehabilitation. Non-invasive neuromodulation shifted language processing away from tumor-affected areas, strengthening alternative functional pathways (Boccuni et al., 2024). This highlights the capacity of brain connections to reorganize in response to damage.

On a molecular level, anti-amyloid therapies like gantenerumab were found to reduce markers of synaptic damage and neuroinflammation, suggesting the preservation of brain connections. In contrast, solanezumab was associated with biomarkers indicating continued axonal injury (Wagemann et al., 2024). Additional findings showed that maintaining stable positive emotions and strong DMN connectivity buffered against neurodegeneration's impact on brain plasticity and executive function (Anthony et al., 2024).

Elevated homocysteine levels were also linked to cortical and subcortical brain atrophy, reduced white matter, and enlarged ventricles in older adults, emphasizing the role of metabolic factors in brain aging (Sah et al., 2024). Similarly, tau-targeting immunotherapy (AADvac1) in Alzheimer's patients slowed cognitive and functional decline, preserved brain structure, and reduced biomarkers of neurodegeneration (Cullen et al., 2024).

DISCUSSION

The findings of this review suggest that age-related memory loss is not solely the result of widespread neuron loss but rather of gradual and dynamic changes in brain connectivity. Natural aging appears to alter the structural and functional integrity of key brain networks, particularly those involved in memory, executive

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functioning, and attention. Most notably, changes in the default mode network (DMN), frontoparietal control network (FPCN), and language networks emerged as central to this process (Anthony et al., 2024; Gozdas et al., 2024).

A consistent trend across the literature is that advancing age is associated with reduced functional connectivity within these networks. Several studies reported physical markers of this disruption, including increased brain atrophy, reduced white matter volume, and weakened synaptic signaling (Sah et al., 2024; Cullen et al., 2024; Zhou et al., 2025). These effects were particularly pronounced in individuals with elevated risk factors such as high homocysteine levels or lipid variability, suggesting that metabolic health directly contributes to structural and functional brain decline (Sah et al., 2024; Zhou et al., 2025).

However, the results also emphasize that while brain connections naturally weaken with aging, particularly within memory and executive function networks, the aging brain maintains significant plasticity. Many studies indicated that functional connectivity within the DMN and FPCN, networks essential for memory retrieval, self-referential thinking, and attentional control, tends to decline as individuals age. This weakening of long-range connectivity can contribute to slower cognitive processing and memory lapses commonly observed in older adults. Yet, interventions such as physical activity and cognitive training were consistently found to counteract this trend by preserving or strengthening these same networks (Gozdas et al., 2024; Dimitriadis et al., 2023). Additionally, increased levels of Brain-Derived Neurotrophic Factor (BDNF), a key biomarker associated with synaptic plasticity, were linked to improved connectivity between regions like the hippocampus, prefrontal cortex, and parietal lobes, which are areas critical for learning and memory (Hola et al., 2024). Non-invasive brain stimulation techniques, such as transcranial direct current stimulation (tDCS), further enhanced connectivity within the dorsolateral prefrontal cortex (DLPFC), supporting executive function and attentional regulation (Aksu et al., 2024). Collectively, these findings illustrate that while aging disrupts functional connectivity within critical brain networks, targeted interventions may promote adaptive reorganization.

In clinical contexts, therapies targeting amyloid and tau pathologies were found not only to reduce hallmark disease biomarkers but also to preserve synaptic integrity and reduce neuroinflammation, further supporting the role of brain connectivity in cognitive preservation (Wagemann et al., 2024; Cullen et al., 2024). Additionally, emotional regulation and positive affect appeared to buffer the negative effects of neurodegeneration on plasticity, reinforcing the multifaceted nature of cognitive resilience in aging (Anthony et al., 2024).

While many of the studies reviewed like Gozdas et al. (2024) and Dimitriadis et al. (2023) emphasize the role of maintaining brain connectivity in mitigating memory loss during aging, some findings suggest that structural brain changes may not be the sole or primary determinant of cognitive decline. For instance, Wing et

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al. (2023) proposed that reductions in central obesity, rather than direct changes in brain connectivity or structure, were more closely associated with healthier brain aging and slower memory deterioration. Their findings indicate that factors such as body fat and metabolic health may exert substantial influence on memory outcomes, potentially through mechanisms involving vascular health or inflammation, rather than through alterations in brain network organization alone.

In addition, Anthony et al. (2024) demonstrated that positive emotional stability and affect regulation might counteract the cognitive impacts of neurodegeneration in older adults. This suggests that while structural changes in brain connectivity do occur with age, they may not inevitably lead to memory decline. Instead, non-structural factors such as emotional resilience could play a protective role, preserving cognitive function despite underlying neurobiological changes. Ultimately, these contrary perspectives highlight that the relationship between brain connectivity, aging, and memory loss is complex and multifactorial.

As a general finding throughout this review, aging appears to increase susceptibility to neurodegenerative diseases such as Alzheimer's, which put more weight on memory loss than the typical, gradual decline observed in healthy aging.

A key limitation of this review was the scarcity of studies that directly investigated how brain connections structurally or functionally change with aging. While many of the included studies focused on strategies to improve memory in older adults, relatively few provided detailed insight into the specific neural mechanisms or connectivity alterations underlying age-related memory decline. This limitation is likely a result of the specific methodologies employed in the reviewed studies, as existing literature does directly explain how brain connections change with aging. For example, the Columbia University Mailman School of Public Health (2021) notes that aging leads to structural brain changes impacting memory, including shrinkage of the frontal lobes and hippocampus, cortical thinning, and reduced white matter. These changes weaken synaptic connectivity and slow communication between brain regions, ultimately contributing to memory decline. There may also be a slight gap in literature that could stem from the inherent challenges of studying brain connectivity in naturally aging populations. Unlike short-term intervention studies that can measure improvements in memory through behavioral tests, tracking how neural connections evolve requires longitudinal studies, advanced imaging techniques, and large participant samples. Furthermore, memory loss in aging is influenced by a multitude of factors, including neurodegeneration, vascular health, and lifestyle habits. This makes it difficult to isolate connectivity changes as the sole cause. As a result, while intervention studies provide valuable insight into strategies for preserving memory, there remains a need for research that directly examines the structural and functional changes in brain networks that occur with age. Additionally, while many studies focused on neurodegenerative diseases, which offer valuable insight into pathological brain changes, they often failed to provide clear information about how brain connections naturally evolve with healthy aging.

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While existing research provides valuable insight into potential interventions that mitigate memory loss in aging, there remains a significant gap in understanding how brain connections naturally evolve independent of disease or targeted treatment. Future research should focus on longitudinal studies that directly track changes in specific brain networks within healthy aging populations. Rather than simply assessing whether interventions improve memory, these studies could map how distinct brain regions change in connectivity over decades. Identifying which neural pathways are most vulnerable to aging and when these changes typically begin would offer a clearer picture of natural cognitive decline.

Additionally, future studies could explore the potential of targeted stimulation or intervention to preserve specific connections known to deteriorate with age. For instance, examining whether region-specific stimulation of memory-critical areas can prevent or slow down disconnection between regions may advance intervention strategies beyond generalized training or exercise. Another important direction may be adopting a more holistic, population-wide approach. Much of the existing literature isolates variables like physical activity or cognitive training, but comprehensive studies that incorporate genetics, lifestyle factors, metabolic health, and environmental exposures could provide a more complete understanding of what preserves memory across diverse aging populations. Given the complexity of aging, understanding shared patterns of deterioration despite differences in genetics or disease susceptibility could help identify universal protective factors.

Finally, future research could investigate the overlap between natural aging processes and early neurodegenerative changes. While many studies treat these as separate phenomena, the presence of similar biomarkers or connection changes in both healthy aging and early-stage disease could suggest that neurodegeneration may, in part, reflect an acceleration or exaggeration of natural aging patterns. Exploring this potential overlap could provide valuable insight into the earliest and most critical changes in brain connectivity that underlie memory loss.

CONCLUSION

In examining how brain connections change with age and how these changes contribute to memory loss, this systematic review found that aging influences both structural and functional alterations within the brain's key memory-related networks. Most notably, the default mode network (DMN), frontoparietal control network (FPCN), and hippocampal-prefrontal pathways exhibit declines in connectivity and efficiency as individuals grow older (Anthony et al., 2024; Gozdas et al., 2024). These weakened connections are closely tied to declines in memory performance, particularly in working memory, episodic recall, and executive functioning.

The aging brain shows reduced synaptic plasticity, lower levels of neuroprotective biomarkers such as BDNF, and increasing vulnerability to neurodegeneration, all of which impair communication between brain regions critical for memory. However, the reviewed studies also suggest that these changes are not entirely

inevitable nor uniform across individuals. Physical activity, cognitive engagement, emotional stability, and metabolic health all emerged as important modifiers that can preserve or even strengthen brain connectivity, potentially mitigating memory loss.

Ultimately, while aging naturally leads to the weakening of specific neural pathways crucial for memory, the extent and impact of this decline are shaped by both biological and lifestyle factors. Understanding how to best preserve these connections, whether through behavioral interventions or targeted therapies, remains essential for addressing memory loss in an aging population.

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