Neural Mechanisms of Successful Episodic Memory Aging

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Abstract

With advancing age, most older adults experience decline in episodic memory, yet some exhibit successful aging of episodic memory, characterized by relatively good memory performance or minimal age-related decline. The brain maintenance theory, neural dedifferentiation theory, cognitive reserve theory, and neural compensation theory each explain the neural mechanisms underlying successful aging of episodic memory from different perspectives. Based on the selective optimization with compensation model to integrate existing theories, it has been found that successful aging of episodic memory may be directly related to an individual' s level of cognitive reserve: older adults with high cognitive reserve can optimize brain regions and networks associated with episodic memory and possess stronger neural compensation capabilities, thus their brain functions (e.g., the specificity of neural representations and neural processing pathways) may be better preserved. Future research should adopt more longitudinal designs to examine the relationships among these theories and their influencing factors, thereby better explaining the neural mechanisms of successful aging of memory and providing support for promoting brain and cognitive health in older adults.

Full Text

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Abstract

Healthy aging is generally associated with a decline in episodic memory. However, older adults demonstrate notable individual differences in episodic memory. While most older adults show a normal or pathological decline in episodic memory, some indicate successful episodic memory aging. It is essential to investigate the neural mechanisms of individual differences in episodic memory aging to demystify the determinants of successful memory aging. To date, four critical theories have been proposed to explain why some older adults exhibit successful memory aging: brain maintenance, neural dedifferentiation, cognitive reserve, and neural compensation. Based on these theories and the SOC model, we speculate that some older adults display successful memory aging because they have higher cognitive reserve shaped by several lifestyle factors throughout their lifespans. Older adults with higher cognitive reserve can optimize the function of the brain regions and networks related to episodic memory and more successfully compensate for age-related neural decline. Ultimately, the benefits of the optimization and compensation processes are reflected in maintaining a higher level of brain function (e.g., the fidelity of neural representation or functional segregation of brain networks). Future research should incorporate more longitudinal studies to investigate the relationship between these theories and their impact factors, which would be beneficial for understanding the neural mechanisms of successful memory aging and providing support for improving brain and cognitive health in older adults.

Keywords: episodic memory, successful aging, brain maintenance, neural dedifferentiation, cognitive reserve, neural compensation

1. Introduction

Episodic memory refers to an individual' s memory of personally experienced events that occurred at specific times and places (Tulving, 2002). The intentional recollection of event-related details constitutes the core characteristic of the retrieval phase in episodic memory (Rugg et al., 2015). In laboratory settings, researchers primarily employ associative memory paradigms to examine the recollection process in episodic memory. In these paradigms, participants typically learn item-item pairings (e.g., word-word or face-name pairs) or item-context pairings (e.g., word-color or face-spatial location pairs) during the encoding phase. Subsequently, they intentionally retrieve relational information between items or between items and context through cue-recall or associative recognition tasks (Naveh-Benjamin, 2000).

With advancing age, older adults experience significant declines in cognitive function, with episodic memory impairment being one of the most prominent aspects (Dodson, 2017; Park et al., 2002). This decline primarily manifests as difficulty in intentionally recollecting relational information between events or between events and context (Alghamdi & Rugg, 2020; Old & Naveh-Benjamin, 2008). Moreover, accurate recollection of event-related details requires the in-

volvement of executive control processes (Shing et al., 2010). Accordingly, structural and functional deterioration in brain regions (e.g., medial temporal lobe and prefrontal cortex) and brain networks (e.g., default mode network and frontoparietal network) responsible for recollection and executive control is considered the main cause of age-related episodic memory decline (Liem et al., 2021; Wang et al., 2017).

Previous research has typically treated older adults as a homogeneous group, primarily comparing age differences in episodic memory performance through group-averaged methods. However, substantial individual differences exist in episodic memory function among older adults (Lindenberger, 2014; Nyberg & Pudas, 2019; Tucker-Drob & Salthouse, 2013). For instance, the Betula project, a large-scale Swedish cohort study, found through cross-sectional comparisons that among 663 older adults (aged 70-85 years), 55 individuals (approximately 8.3%) exhibited significantly higher episodic memory performance than the average of a middle-aged older adult group (aged 50-65 years) (Habib et al., 2007). Furthermore, the project' s longitudinal follow-up of over 1,500 participants (aged 35-85 years) across 15 years revealed that approximately 18% maintained high and stable memory performance (Josefsson et al., 2012). These findings suggest that while most older adults experience significant age-related declines in episodic memory function, a subset demonstrates successful memory aging, characterized by relatively preserved memory performance or slower rates of age-related decline.

The most influential explanation for successful aging comes from Baltes and colleagues' Selective Optimization with Compensation (SOC) model (Baltes & Baltes, 1990; Baltes & Carstensen, 1996; Baltes & Smith, 2003). Integrating lifespan developmental theory (Baltes et al., 1999), the SOC model proposes that older adults can achieve a state of maximizing gains and minimizing losses through three fundamental processes—selection, optimization, and compensation—and their interactions (Han Buxin & Zhu Liqi, 2012). Specifically, as individuals age, those who can narrow their goal range, selectively focus resources on a few important objectives (e.g., dedicating leisure time to expanding knowledge systems), optimize these goals through resource application (e.g., enrolling in senior education courses), and compensate for emerging losses or declines (e.g., using hearing aids to counteract hearing loss) are more likely to achieve successful development. The SOC model provides a general theoretical framework for understanding successful aging across various domains (e.g., cognitive, social, or physiological functioning).

Over the past two decades, advances in neuroimaging technology have propelled rapid development in cognitive neuroscience research on aging. Episodic memory, being one of the cognitive functions most affected by aging, has attracted considerable attention. The neural mechanisms underlying individual differences in episodic memory among older adults have remained a central research focus, giving rise to four key theories: brain maintenance theory, neural dedifferentiation theory, cognitive reserve theory, and neural compensation theory.

Although these theories emerged within cognitive neuroscience, they can be interpreted using core concepts from the SOC model (e.g., optimization and compensation). First, both brain maintenance theory and cognitive reserve theory reflect the optimization process in successful aging, as both posit that older adults with successful memory aging can utilize existing resources (e.g., formal education received in youth) or acquire new resources (e.g., cognitive activities like reading) to optimize memory-related brain regions or networks. Specifically, classic brain maintenance theory emphasizes that factors such as education, occupation, physical exercise, and cognitive activities can help older adults maintain "youthful" brain structure and function—meaning their brain structural morphology and functional levels more closely resemble those of younger adults—resulting in less age-related impact on memory function (Nyberg et al., 2012; Nyberg & Lindenberger, 2020). Following the same logic, neural dedifferentiation theory proposes that older adults with higher neural differentiation levels—those whose neural representation and processing pathway specificity more closely match younger adults—exhibit better episodic memory function, and can thus be viewed as another form of brain maintenance theory (Li et al., 2002; Koen & Rugg, 2019; Koen et al., 2020). Cognitive reserve theory, in contrast, emphasizes that life experiences throughout the lifespan (including the aforementioned education and occupational factors) contribute to the accumulation of cognitive reserve, enabling individuals to better cope functionally with structural brain aging constraints on cognitive function, thereby slowing the rate of episodic memory decline (Barulli & Stern, 2013; Stern, 2002, 2009). While both brain maintenance and cognitive reserve theories reflect the optimization process, brain maintenance theory focuses on the outcome of optimization, whereas cognitive reserve theory emphasizes the pathways to optimization. Second, successful episodic memory aging also requires the involvement of compensation processes. For instance, neural compensation theory posits that older adults with successful memory aging can utilize additional neural pathways to compensate for existing structural or functional brain decline (Cabeza & Dennis, 2013; Reuter-Lorenz & Park, 2014). Finally, optimization and compensation processes may interact closely. For example, older adults' cognitive reserve levels may influence their neural compensation capacity, while neural compensation ability may affect their brain maintenance levels. Integrating these theories suggests that successful episodic memory aging in older adults may result from their ability to accumulate cognitive reserve through various life experiences and resources, which in turn enables optimization of brain regions and networks directly related to episodic memory function and allows compensation for existing brain decline, ultimately leading to better preservation of brain function (e.g., specificity of neural representation and processing pathways). However, the core concepts within each theory and the complex relationships between theories require further investigation.

Examining individual differences in episodic memory aging and investigating the neural mechanisms of successful memory aging not only helps reveal the neural basis of human cognitive function and understand why some older adults

maintain cognitive resilience but also holds significant importance for developing effective interventions to improve episodic memory function and delay cognitive aging. This article first details the core tenets and supporting evidence of each theory, then discusses current issues and future research directions.

2. Brain Maintenance Theory and Evidence

Since aging is typically accompanied by declines in brain structure and function (Spreng & Turner, 2019), individual differences in the degree of such decline may contribute to individual differences in memory function among older adults. From this perspective, brain maintenance theory posits that since brain decline leads to episodic memory impairment, older adults with less brain decline or whose brain structural morphology and functional levels more closely resemble those of younger adults should exhibit better episodic memory function (Nyberg et al., 2012; Nyberg & Lindenberger, 2020; Nyberg & Pudas, 2019). The degree of brain maintenance in older adults can predict the extent of memory decline; higher brain maintenance with aging corresponds to smaller memory decline. Brain maintenance can manifest at different levels, including brain structure (e.g., gray matter volume and white matter integrity) and brain function (e.g., brain activation or functional connectivity). Therefore, according to brain maintenance theory, older adults with better episodic memory function maintain better preservation of brain structural or functional characteristics.

Consistent with brain maintenance theory, cross-sectional studies have demonstrated significant associations between the structural integrity of the medial temporal lobe (particularly the hippocampus/parahippocampus) and episodic memory function in older adults, such that higher structural integrity predicts better memory performance (Köhncke et al., 2021). However, the primary evidence for brain maintenance theory comes from longitudinal studies linking structural or functional changes in the medial temporal lobe with changes in memory performance. For example, Persson et al. (2012) and Gorbach et al. (2017) both found that the degree of hippocampal structural preservation in older adults significantly predicted their episodic memory preservation level —smaller hippocampal volume atrophy corresponded to less episodic memory decline. Functionally, Salami et al. (2014) found that older adults who maintained stable episodic memory performance over two decades showed better preservation of hippocampal resting-state functional connectivity compared to those with memory decline. Using task-based fMRI designs, Pudas et al. (2013) found that memory maintainers showed stronger hippocampal activation during associative memory encoding than memory decliners, with activation patterns more similar to younger adults. Moreover, when controlling for gray matter volume, these effects remained significant, indicating that preserved hippocampal functional activation independently predicts successful memory aging.

Beyond the medial temporal lobe, longitudinal changes in prefrontal cortex activation patterns also provide important support for brain maintenance theory. In episodic memory tasks, the left prefrontal cortex primarily participates in mem-

ory encoding, while the right prefrontal cortex is mainly involved in memory retrieval—a phenomenon known as hemispheric encoding/retrieval asymmetry (Tulving et al., 1994). Johansson et al. (2020) reported that with increasing age, older adults showed a declining trend in hemispheric encoding/retrieval asymmetry during associative memory tasks (i.e., increasing overlap between prefrontal regions activated during encoding and retrieval), and this decline in prefrontal asymmetry was accompanied by decreases in episodic memory performance.

These findings demonstrate that older adults who maintain better memory function also show relatively better preservation of structural and functional characteristics in brain regions directly related to episodic memory function (e.g., hippocampus and prefrontal cortex), suggesting that maintaining a youthful brain may be key to successful memory aging. Brain maintenance theory offers a straightforward perspective supported by longitudinal evidence, earning considerable recognition within the field. However, the theory lacks in-depth exploration of the neural mechanisms underlying preserved functional characteristics in specific brain regions. Neural dedifferentiation theory provides more specific elaboration in this regard.

3. Neural Dedifferentiation Theory and Evidence

The concept of dedifferentiation originates from behavioral research showing that correlations between different cognitive domains increase with age (Lindenberger & Baltes, 1994; Lindenberger et al., 2001), suggesting that older adults increasingly rely on similar neural substrates to support different cognitive functions. Subsequent neurocomputational models proposed that age-related declines in neurotransmitter system function reduce neural differentiation levels, leading to neural dedifferentiation phenomena that consequently impair cognitive function—this constitutes the neural dedifferentiation theory of cognitive aging (Li & Lindenberger, 1999; Li et al., 2001). This theory effectively explains individual differences in memory function among older adults: higher neural dedifferentiation corresponds to more severe declines in the specificity of neural representations and processing pathways, resulting in poorer memory function; conversely, lower neural dedifferentiation, indicating neural representation and processing pathway specificity more similar to younger adults, predicts better memory function. In this sense, neural dedifferentiation theory can be viewed as a more in-depth brain maintenance theory explaining successful episodic memory aging.

Neural dedifferentiation in older adults manifests primarily in two aspects (Li & Sikström, 2002; Li & Rieckmann, 2014). First, the specificity or fidelity of neural representations decreases, leading to reduced distinctiveness between neural representations. With age, the representational specificity and distinctiveness of different events and their contextual background information decline, causing events to become more easily confused. Park et al. (2004) used functional magnetic resonance imaging to demonstrate reduced neural representation specificity or fidelity in older adults' ventral visual cortex during perceptual tasks,

providing early evidence for neural dedifferentiation. Subsequent studies found that neural dedifferentiation in the ventral visual cortex occurs during both episodic memory encoding (Koen et al., 2019; Srokova et al., 2020; Zheng et al., 2018) and retrieval phases (Abdulrahman et al., 2017; Trelle et al., 2020). Additionally, numerous studies have employed multivoxel pattern analysis methods (e.g., representational similarity analysis) to examine neural dedifferentiation during memory retrieval by assessing the strength of neural pattern reinstatement related to recollected information (e.g., Abdulrahman et al., 2017; Folville et al., 2020; Johnson et al., 2015; Trelle et al., 2020). These studies consistently find weaker neural activation pattern reinstatement in core default network regions (including hippocampus, angular gyrus, posterior cingulate cortex, middle temporal gyrus, and medial prefrontal cortex) during recollection in older adults. Specifically, older adults show lower encoding-retrieval similarity in neural activation patterns during recollection, indicating neural dedifferentiation of episodic recollection information. Moreover, neural dedifferentiation levels in older adults correlate significantly and negatively with episodic memory performance—lower dedifferentiation predicts better memory function (Koen et al., 2019; Trelle et al., 2020). These results suggest that neural dedifferentiation in the ventral visual cortex and default network may constitute an important source of individual differences in episodic memory function among older adults.

The second manifestation of neural dedifferentiation involves decreased functional specificity of neural processing pathways and enhanced synchronous changes between different pathways. Younger adults selectively employ specific neural pathways to perform memory tasks, whereas older adults, due to reduced specificity in neural pathways responsible for memory processing, show stronger covariation between different pathways. The human brain can be functionally organized into multiple large-scale brain networks with strong functional segregation or specificity. Recent studies have found that older adults exhibit dedifferentiation in large-scale brain functional networks, showing higher synchronization or interaction between networks and lower functional segregation compared to younger adults (Damoiseaux, 2017; Koen et al., 2020; Wig, 2017). Specifically, multiple lifespan developmental studies have found that functional segregation of brain networks decreases with age (Cao et al., 2014; Chan et al., 2014; Geerligs et al., 2015; Malagurski et al., 2020; Chong et al., 2019; Han et al., 2018). Notably, Chong et al. (2019) and Malagurski et al. (2020) both reported longitudinal evidence of decreased functional segregation in older adults' brain networks, providing solid support for neural dedifferentiation in large-scale brain networks. Furthermore, the degree of brain network dedifferentiation in older adults correlates significantly with cognitive function. Cross-sectional studies show that higher functional segregation (lower dedifferentiation) predicts better episodic memory performance (e.g., Chan et al., 2014; Geerligs et al., 2015), while longitudinal research indicates that increased brain network dedifferentiation leads to cognitive decline (Malagurski et al., 2020; Ng et al., 2016). These associations are particularly evident in large-scale brain networks responsible for higher-order cognitive functions (e.g.,

default mode network and frontoparietal network) (Chan et al., 2014; Geerligs et al., 2015; Malagurski et al., 2020; Ng et al., 2016).

Integrating brain maintenance and neural dedifferentiation theories suggests that older adults who maintain better structural and functional integrity in brain regions (e.g., hippocampus and prefrontal cortex) and networks (e.g., default mode network) related to episodic memory function are more likely to exhibit successful memory aging. As previously noted, brain maintenance can be viewed as the result of older adults optimizing memory-related brain regions or networks using existing or newly acquired resources. Consistent with this view, studies have found that life experiences such as education, occupation, physical exercise, and cognitive activities provide protective effects on the hippocampus and prefrontal cortex in older adults (Nyberg & Pudas, 2019; Nyberg et al., 2020). In addition to these experiential factors, genetics may represent an innate factor influencing brain maintenance. For example, older adults carrying the catechol-O-methyltransferase (COMT) Met genotype are more likely to maintain stable memory function (Josefsson et al., 2012). These findings suggest that brain maintenance levels in older adults may be influenced by both innate (e.g., genetics) and experiential (e.g., education, occupation, lifestyle) factors. However, because verifying brain maintenance theory requires longitudinal evidence, and longitudinal studies are more challenging than cross-sectional ones, research examining factors influencing brain maintenance remains relatively scarce. Particularly, evidence is lacking regarding which factors enable older adults to maintain high levels of specificity in neural representations and processing pathways.

It is important to note that many older adults exhibit insufficient brain structural preservation or age-related structural decline. Can this population still demonstrate good memory performance or slower memory decline? Cognitive reserve theory emphasizes that neural resources accumulated throughout the lifespan can help mitigate the adverse effects of age-related structural decline on memory function from a functional perspective, representing another pathway to achieving successful memory aging.

4. Cognitive Reserve Theory and Evidence

Early brain reserve theory proposed that individual differences in neuroanatomical resources (e.g., neuron number and synaptic density) enable some individuals to better cope with the adverse effects of brain aging. The amount of neuroanatomical resources reflects brain reserve level, which can be quantified through structural or morphological brain features (e.g., total intracranial volume, gray matter volume, cortical surface area, cortical thickness, and white matter tract integrity). According to this theory, when facing structural decline, older adults with higher brain reserve have more available neuroanatomical resources and thus better memory performance (Blessed et al., 1968; Katzman et al., 1988; Satz, 1993; Schofield et al., 1997). However, researchers observed that two older adults with similar neuroanatomical resources could still show

different memory performance (Stern, 2002), suggesting that brain reserve theory cannot effectively explain individual differences in memory function among older adults.

In recent years, cognitive reserve theory has become one of the most influential theories in cognitive aging neuroscience. This theory posits that older adults with high cognitive reserve can more adaptively cope with structural brain aging and maintain high-level episodic memory function. Cognitive reserve refers to the adaptability of cognitive processes accumulated throughout an individual' s lifespan (Barulli & Stern, 2013; Stern, 2002). Various life experiences contribute to cognitive reserve accumulation, with social-behavioral factors such as education, intelligence, occupational complexity, physical exercise, and cognitive activities commonly used to indirectly estimate cognitive reserve levels (Boyle et al., 2021; Clouston et al., 2020; Li et al., 2021; Xu et al., 2016).

The core hypothesis of cognitive reserve theory is that cognitive reserve moderates the relationship between brain structure and cognitive function, meaning that cognitive reserve and brain structure interact to influence cognitive function. In practice, researchers can test this hypothesis by measuring cognitive reserve levels, brain structural features, and episodic memory function (Stern et al., 2020; Varangis & Stern, 2020). The moderating effect of cognitive reserve manifests in three aspects. First, given the same level of brain structure, older adults with higher cognitive reserve may show better memory performance. For example, in subcortical regions closely related to memory function such as the putamen and nucleus accumbens, older adults with higher cognitive reserve demonstrate superior memory performance compared to those with similar gray matter volume (Steffener et al., 2014). Second, cognitive reserve level can moderate the strength of association between brain structural features and episodic memory performance. For older adults with low cognitive reserve, brain structure level substantially impacts memory function; for those with high cognitive reserve, memory function is less dependent on brain structure because cognitive reserve better protects memory function. Studies have found weaker correlations between brain structure levels (e.g., hippocampal gray matter volume) and episodic memory performance in high cognitive reserve older adults (Vuoksimaa et al., 2013; Zahodne et al., 2019). Third, significant associations between cognitive reserve level and episodic memory performance persist even after controlling for normal or pathological brain structural features. For instance, Li et al. (2021) found that high cognitive reserve older adults still showed high-level episodic memory performance after controlling for Alzheimer' s disease-related pathological features (e.g., neurofibrillary tangles).

Cognitive reserve is fundamentally a cognitive concept that should theoretically protect cognitive function through adaptive brain function. Stern and colleagues therefore proposed the concept of neural reserve, which can be expressed through brain function efficiency, capacity, and flexibility to reflect the neural mechanisms of cognitive reserve (Barulli & Stern, 2013; Stern, 2017). High efficiency means that older adults with high cognitive reserve show lower activation levels

in task-related brain regions despite similar behavioral performance. High capacity is reflected in increased activation strength in task-related brain regions as task difficulty or cognitive load increases. High flexibility refers to the ability of high cognitive reserve older adults to use additional brain regions or networks to compensate for age-related brain deterioration to maintain or improve cognitive function. To interpret these three forms of neural reserve as neural mechanisms of cognitive reserve, they must demonstrate significant correlation with cognitive reserve estimates. Unfortunately, the protective mechanisms of neural reserve on episodic memory function in older adults remain theoretical constructs lacking empirical support. For example, Elshiekh et al. (2020) found that older adults with excellent episodic memory performance showed stronger activation in prefrontal and medial temporal regions during memory encoding, suggesting possible neural compensation. However, the activation strength in these regions did not significantly correlate with cognitive reserve indices, indicating that this compensation mechanism does not reflect cognitive reserve' s neural flexibility mechanism. Therefore, using neural reserve to explain how cognitive reserve protects episodic memory function in older adults requires further empirical support.

The advantage of cognitive reserve theory lies in its ability to effectively explain why two older adults with similar neuroanatomical resources may still show different episodic memory performance. However, the theory faces a critical challenge: it lacks a clear operational definition. In practice, some researchers use single social-behavioral indicators to estimate cognitive reserve (Mousavi-Nasab et al., 2014), while others combine multiple indicators into a composite index (Elshiekh et al., 2020). Which indicator better measures individual cognitive reserve levels remains unresolved, raising concerns about the replicability of studies examining how cognitive reserve protects episodic memory in older adults. Using different indicators to estimate cognitive reserve may yield inconsistent results. Second, both cognitive reserve theory and brain maintenance theory can be viewed as reflecting the optimization process in successful memory aging, raising the question of whether these theoretical concepts are closely related. According to cognitive reserve theory, high cognitive reserve enables older adults to better cope functionally with structural brain decline' s adverse effects on memory, potentially resulting in slower memory decline (Stern et al., 2020). From this perspective, cognitive reserve may be an important reason why older adults maintain good brain function. However, this speculation currently lacks support from longitudinal neuroimaging studies. Finally, cognitive reserve theory posits that neural compensation depends on high cognitive reserve and represents one mechanism of neural reserve (Barulli & Stern, 2013). However, neural compensation constitutes a relatively independent theory in the cognitive neuroscience of memory aging, used to explain successful memory aging phenomena (Cabeza et al., 2018). Particularly, this theory effectively embodies the compensation process in the SOC model and can thus be considered another pathway to achieving successful memory aging.

5. Neural Compensation Theory and Evidence

Since the prefrontal cortex is responsible for executive control processes in episodic memory function (e.g., encoding strategy use, retrieval monitoring), functional decline in this region has long been considered a major cause of memory impairment in older adults (Shing et al., 2010). Consistent with this view, numerous studies have found reduced prefrontal activation in older adults during both episodic memory encoding and retrieval (Duarte & Dulas, 2020). However, the actual situation is more complex, as an interesting pattern of results has emerged in research on the neural mechanisms of episodic memory aging: older adults with relatively better memory performance typically show enhanced prefrontal activation or more distributed brain activation patterns (Grady, 2012). Two common neural distribution patterns are the Hemispheric Asymmetry Reduction in Older Adults (HAROLD) and the Posterior-Anterior Shift in Aging (PASA). HAROLD manifests as significant right prefrontal activation during episodic memory retrieval in younger adults, whereas memory-successful older adults show bilateral prefrontal activation (Cabeza, 2002). PASA manifests as enhanced prefrontal activation and reduced posterior visual cortex activation in older adults during episodic memory tasks (Davis et al., 2008). These altered brain activation patterns in older adults are generally considered to reflect functional reorganization, indicating that older adults can use other neural resources to "successfully compensate" for their memory performance when facing cognitive challenges (Cabeza & Dennis, 2013). This compensatory characteristic has been metaphorically termed "compensatory scaffolding" (Park & Reuter-Lorenz, 2009; Reuter-Lorenz & Park, 2014). Therefore, according to neural compensation theory, older adults with successful memory aging can adaptively use compensatory scaffolding to resist the adverse effects of structural or functional brain decline on memory function.

As previously mentioned, cognitive reserve theory suggests that only older adults with high cognitive reserve exhibit neural compensation mechanisms. However, neural compensation theory places less emphasis on the influence of personal life experiences on compensation processes. For example, three compensation mechanisms directly related to memory function (Cabeza et al., 2018) appear unaffected by individual cognitive reserve levels. The first is compensation by upregulation, which manifests as enhanced prefrontal activation in older adults to support cognitive performance when task difficulty increases and required processing resources escalate. In this scenario, older adults activate the same brain regions as younger adults, differing only in activation magnitude (Reuter-Lorenz & Cappell, 2008; Cappell et al., 2010). This compensation mechanism is regulated by cognitive load; when task demands exceed available neural resources, older adults recruit additional neural resources to achieve memory performance similar to younger adults. The second is compensation by selection, where older adults use different neural pathways than younger adults to perform the same task. For instance, younger adults use recollection processes (dependent on the hippocampus) that are more effective but require more ef-

fort, whereas older adults use familiarity processes (dependent on the perirhinal cortex) that are less effective but require less effort (Daselaar et al., 2006). The third mechanism is compensation by reorganization, where memory-successful older adults develop neural pathways not present in younger populations to compensate for their own structural or functional decline. The aforementioned HAROLD pattern represents a typical example of reorganization-based compensation. Reorganization-based compensation involves older adults using new neural pathways (e.g., contralateral prefrontal cortex) to achieve compensation, while the other two forms involve using existing cognitive processes (upregulation mechanism) or cognitive strategies (selection mechanism).

In summary, according to neural compensation theory, older adults with successful memory aging use prefrontal regions similar to but more strongly activated than younger adults, employ different cognitive processes (e.g., familiarity dependent on perirhinal cortex), or even develop new neural pathways (e.g., contralateral prefrontal cortex) to compensate for their memory performance. Neural compensation theory originated from research on age differences in episodic memory (Cabeza, 2002) and holds an important position in the cognitive neuroscience of memory aging. However, using this theory to explain successful memory aging has encountered considerable opposition in recent years. First, enhanced prefrontal activation in older adults must be associated with better memory performance to confirm that it reflects compensation. However, recent studies using multivariate brain imaging analysis have shown that enhanced prefrontal activation patterns in older adults do not necessarily correlate significantly with better episodic memory performance, suggesting that such activation may reflect reduced prefrontal efficiency or even neural dedifferentiation rather than compensation (Morcom & Henson, 2018; Roe et al., 2020). Second, current evidence supporting neural compensation theory primarily comes from cross-sectional studies (Cabeza, 2002; Davis et al., 2008; Reuter-Lorenz & Cappell, 2008; Cappell et al., 2010), with relatively few longitudinal studies. Existing longitudinal studies do not fully support compensation theory (Persson et al., 2006; Pudas et al., 2018). For example, Pudas et al. (2018) found that stronger prefrontal activation existed in older adults showing memory decline rather than those maintaining stable performance, suggesting that enhanced prefrontal activation may not necessarily reflect compensation. Finally, compensation theory emphasizes that neural compensation processes shown by memory-successful older adults are primarily influenced by cognitive load or individual cognitive processes and strategies but fails to specify which characteristics enable older adults to better utilize compensation mechanisms to protect episodic memory function, leaving the theory incomplete in explaining individual differences in memory function among older adults.

6. Discussion and Outlook

In recent years, the neural mechanisms underlying individual differences in cognitive aging have gradually become a research hotspot in cognitive neuroscience.

A critical question requiring explanation is why some older adults exhibit successful episodic memory aging. Investigating the neural mechanisms through which older adults actively cope with age-related brain decline can provide important theoretical insights for enhancing cognitive resilience in older adults and scientific evidence for public health policy formulation. This article, integrating the optimization and compensation concepts from the SOC model, 详细介绍了脑保持、神经去分化、认知储备以及神经补偿四种理论观点及其相应的支持证据。上述理论从不同角度解释了老年人情景记忆功能成功年老化的原因。然而，从理论建构和实际研究层面出发，未来仍有诸多问题值得探讨。

Theoretical Construction Issues

Three important theoretical issues warrant attention. First, the definition of reserve concepts remains controversial. Early researchers typically distinguished between brain reserve and cognitive reserve. Brain reserve focuses on structural aspects, representing the brain' s "hardware," whereas cognitive reserve emphasizes functional flexibility and adaptability, metaphorically representing the brain' s computational "software." Compared to brain reserve, cognitive reserve highlights the influence of various life experiences and represents a more active, dynamic reserve type. However, some researchers argue that artificially distinguishing brain reserve from cognitive reserve is unnecessary, as brain function must depend on structural foundations, and all cognitive functions ultimately rely on complex interactions between structure and function (Cabeza et al., 2018). Moreover, both brain structure and function in older adults show plasticity (Gutchess, 2014), making the distinction between passive brain reserve and active cognitive reserve unreasonable. Therefore, some propose simplifying the terminology to a single "reserve" concept, viewing it as a process of accumulating neural resources that can include gray matter volume of a brain region, integrity of a fiber tract, or functional levels of a brain region or network. Stern and Chételat et al. (2019) maintain the traditional view that a single reserve concept cannot fully capture the research landscape. Brain reserve and cognitive reserve have different emphases: brain reserve can describe the brain' s physiological state, while cognitive reserve focuses on moderating the relationship between brain structure and cognitive function, thus better explaining individual differences in memory function. However, Stern et al. (2020) acknowledge that brain reserve and cognitive reserve are not mutually exclusive but differ primarily in quality rather than quantity. Current research technology limitations constrain clarification of the complex relationship between brain structure and function, restricting integration of these concepts. Future technological advances may resolve this conceptual opposition and provide important support for explaining successful memory aging.

Second, the relationship between cognitive reserve and neural compensation remains controversial. The cognitive reserve perspective views compensation as dependent on cognitive reserve, representing a manifestation of neural reserve flexibility. That is, cognitive reserve level determines whether individuals can

exhibit neural compensation, with high cognitive reserve providing the foundation for older adults to use additional brain regions or networks to compensate for cognitive function (Barulli & Stern, 2013; Stern, Barnes, et al., 2019; Stern, Chételat, et al., 2019). Therefore, theoretically, if two older adults experience the same degree of brain decline, the one with higher cognitive reserve should show better episodic memory performance due to neural compensation. Unfortunately, this theoretical view currently lacks experimental support. Cabeza et al. (2018) argue that cognitive reserve may be a necessary but not sufficient condition for neural compensation; it can create conditions for compensation but does not necessarily lead to it. For example, when cognitive load exceeds available neural resources, memory-successful aging individuals may use other brain regions or networks to support memory function (Cabeza & Dennis, 2013). Thus, neural compensation theory can independently explain the neural mechanisms of individual differences in episodic memory. We propose that these two theoretical views may not be entirely mutually exclusive. Based on current experimental studies supporting compensation theory, whether older adults exhibit neural compensation does not depend on individual cognitive reserve levels but is influenced by task difficulty, cognitive processes, and strategies. However, among memory-successful older adults who do show neural compensation, cognitive reserve level and neural compensation capacity likely have a positive relationship—higher cognitive reserve probably corresponds to greater neural compensation capacity. Further evidence is needed to support this latter point.

Third, explaining successful episodic memory aging requires organic integration of different theoretical perspectives to improve the current theoretical system. Based on existing theories and SOC model' s optimization and compensation concepts, we speculate that some older adults show successful memory aging because they can use various resources to optimize memory-related brain regions and networks and compensate for existing brain decline through neural compensation. However, this view requires discussion of many details. First, according to brain maintenance theory, some older adults show better memory performance or slower decline because they can use resources like education, occupation, physical exercise, and cognitive activities to optimize memory-related brain regions or networks, thus maintaining better brain function. However, cognitive reserve theory also emphasizes that older adults can use various resources to optimize memory-related brain function (Barulli & Stern, 2013; Nyberg et al., 2012). Cognitive reserve theory explains this more specifically, emphasizing that these resources contribute to cognitive reserve accumulation, which functionally protects episodic memory and slows decline or maintains better function (Stern et al., 2020). We can infer that, at least from a functional perspective, brain maintenance may be the direct result of older adults optimizing memory-related brain regions or networks, while cognitive reserve may be the primary pathway to achieving this optimization outcome. That is, memory-successful older adults can use cognitive reserve to optimize brain function, leading to better functional preservation and ultimately good memory performance or slower decline. This inference has received support from behavioral research. For example, Li et

al. (2021) found that high cognitive reserve older adults indeed maintained better episodic memory performance with age. Second, although cognitive reserve may not be a prerequisite for neural compensation, individuals with high cognitive reserve likely show greater neural compensation capacity. Therefore, older adults with high cognitive reserve may demonstrate not only better optimization ability but also greater skill in using neural compensation processes. In summary, from a functional perspective, successful episodic memory aging in older adults may result from better preservation of functional characteristics (e.g., specificity of neural representations or processing pathways) in brain regions and networks directly related to episodic memory function. This high level of functional preservation occurs because life experiences such as education, occupation, physical exercise, and cognitive activities help build high cognitive reserve, enabling better optimization of relevant brain regions and networks. Additionally, these older adults may be more adept at using neural compensation mechanisms to counteract existing brain decline. This speculation requires verification through future neuroimaging research. Exploring interconnections between different concepts will greatly advance the development of more comprehensive theoretical systems explaining successful memory aging.

Empirical Research Issues

Three key issues require attention at the empirical research level. First, both innate (e.g., genetics) and experiential (e.g., education, occupation, lifestyle) factors may influence brain maintenance, neural dedifferentiation, cognitive reserve, and neural compensation levels, thereby contributing to individual differences in memory function among older adults (Nyberg et al., 2020). For example, older adults carrying the apolipoprotein E (APOE) 4 allele show higher risk of hippocampal atrophy and lower brain maintenance levels (Cacciaglia et al., 2018). Additionally, life experiences such as education, occupation, and cognitive activities may help maintain youthful brain function and reduce memory decline by decreasing brain deterioration (e.g., less gray matter volume reduction, fewer cerebral microvascular lesions, or less amyloid aggregation or tau burden) and promoting neural repair processes (Nyberg & Pudas, 2019). These experiential factors may also contribute to cognitive reserve levels, with low education typically associated with low cognitive reserve (Stern, 2009). However, current understanding remains limited regarding how innate and experiential factors differentially modulate brain maintenance, neural dedifferentiation, cognitive reserve, and neural compensation levels. For cognitive reserve specifically, researchers often use social-behavioral factors like education, occupational complexity, physical exercise, and cognitive activities to indirectly estimate cognitive reserve levels, but no consensus exists on which indicator best measures cognitive reserve. Moreover, interactions between different factors complicate quantifying and separating their contributions to cognitive reserve. Future research should further examine how innate and experiential factors influence brain maintenance, neural dedifferentiation, cognitive reserve, and compensation capacity. Investigating how these factors help older adults optimize brain

structure and function is crucial for improving memory function and cognitive health.

Second, among various experiential factors, education level has long been considered an important factor influencing individual differences in cognitive function (especially memory) among older adults, but its mechanism of action lacks consistent explanation. Education level serves as a core social-behavioral indicator for measuring cognitive reserve and is considered an important factor in slowing memory decline (Stern et al., 2020). Additionally, education may help reduce age-related brain deterioration and stimulate repair processes, thereby promoting better preservation of brain and memory function (Nyberg & Pudas, 2019; Nyberg et al., 2020). However, recent evidence suggests the relationship between education and cognitive aging rate is minimal, posing challenges to brain maintenance and cognitive reserve theories of successful memory aging. For instance, through systematic review of longitudinal studies and meta-analyses examining education level and cognitive aging, Seblova et al. (2020) found that although education positively affects cognitive function (higher education associated with better cognitive function), education level does not significantly relate to age-related cognitive decline rate. That is, regardless of education level, the degree of cognitive decline with age is similar, suggesting that education does not slow cognitive decline or constitute a major contributor to maintaining cognitive function. Similarly, Nyberg et al. (2021) recently found that education level does not affect age-related structural brain decline rate. Lövdén et al. (2020) argue that, at least regarding how education influences cognitive function, a simple threshold model can explain education' s effects on cognitive function in older adulthood without invoking cognitive reserve. Specifically, older adults with higher education generally have better cognitive function than those with lower education. If two individuals of the same age experience similar age-related brain decline rates, the more educated person requires more time to reach the threshold for cognitive impairment and thus shows cognitive decline later. Therefore, education may reduce the risk of episodic memory decline in older adults not by slowing memory decline rate but by maintaining cognitive advantages from early adulthood into older age, delaying the onset of memory decline. Future research should continue examining relationships between experiential factors (including education) and rates of age-related decline in brain and memory function.

Finally, investigating how multiple factors influence brain maintenance, neural dedifferentiation, cognitive reserve, and neural compensation capacity urgently requires longitudinal research support. Longitudinal studies represent the best method for examining neural mechanisms of individual differences in episodic memory function among older adults but remain scarce due to cost constraints. For example, our speculation that high cognitive reserve leads to better optimization of brain function, manifested as better functional preservation and slower memory decline, particularly requires longitudinal neuroimaging support. Currently, only brain maintenance theory has received substantial longitudinal research support, and these studies have revealed many other important findings

about memory aging. For instance, longitudinal results suggest that enhanced prefrontal activation during memory tasks found in cross-sectional studies may be exaggerated; in fact, age-related prefrontal activation decreases during memory encoding or retrieval are more typical (Nyberg et al., 2010). Furthermore, increased prefrontal activation with age may be associated with declining rather than compensated memory function (Pudas et al., 2018). These findings challenge neural compensation theory. In summary, more longitudinal studies are needed to accurately characterize the neural basis of individual differences in memory function and its influencing factors and to test core assumptions of each theory.

In recent years, increasing evidence suggests that many factors affecting cognitive function in older adults are modifiable (Mukadam et al., 2019), and the aging brain retains considerable plasticity (Gutchess, 2014; Huo Lijuan et al., 2018). Therefore, interventions targeting life experience-related factors may positively influence episodic memory function in older adults. For example, studies have found that sustained physical exercise or cognitive activity can reduce brain deterioration, enhance neural repair mechanisms, and slow memory decline (Köhncke et al., 2016; Köhncke et al., 2018). Other research shows that cognitive interventions (Lövdén et al., 2010) and exercise interventions (Soshi et al., 2021) can promote recovery of brain structural features and slow age-related gray matter volume decline (Erickson et al., 2011; Jonasson et al., 2017). Additionally, cognitive (Erickson et al., 2007) and exercise interventions (Colcombe et al., 2004; Voss et al., 2019) can make older adults' brain activation patterns more similar to those of younger adults. These results align with brain maintenance theory, suggesting that effective interventions should aim to help older adults repair damaged neural pathways and restore brain structure or function to levels more similar to younger adults, thereby improving memory function and enhancing cognitive resilience. Researchers could also design interventions based on cognitive reserve or neural compensation theory to improve episodic memory function by enhancing cognitive reserve levels or improving compensatory scaffolding. Thus, despite current theoretical controversies, these theories provide important theoretical foundations for research on brain and cognitive plasticity in older adults.

Regarding the neural mechanisms of successful episodic memory aging, multiple theories offer explanations from different perspectives. From the optimization process perspective, brain maintenance theory emphasizes that maintaining "youthful" structure and function in memory-related brain regions (e.g., hippocampus and prefrontal cortex) and networks (e.g., default mode network and frontoparietal network) is key to successful memory aging. Building on this, neural dedifferentiation theory suggests that this functional maintenance primarily manifests in the specificity of neural representations and processing pathways. Cognitive reserve theory posits that cognitive processes accumulated throughout life enable older adults to functionally modulate the adverse effects of structural brain decline on episodic memory, thereby slowing decline or maintaining better function. These theories differ in that brain maintenance

represents the outcome of optimization, while cognitive reserve represents the pathway or means to achieve this outcome. From the compensation process perspective, neural compensation theory emphasizes that older adults can use compensatory scaffolding (e.g., contralateral prefrontal cortex) to compensate for existing structural or functional brain decline, forming the neural basis for good memory performance. Integrating these theories with the SOC model's optimization and compensation processes suggests that successful episodic memory aging may result from life experiences such as education, occupation, physical exercise, and cognitive activities contributing to high cognitive reserve. High cognitive reserve enables older adults to better optimize memory-related brain regions and networks and more skillfully use neural compensation processes, thereby better preserving brain functional states (e.g., specificity of neural representations and processing pathways). Future research should design more longitudinal observational and intervention studies to explore relationships between theoretical concepts and their influencing factors, providing important support for explaining successful memory aging and improving brain and cognitive health in older adults.

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Note: Figure translations are in progress. See original paper for figures.